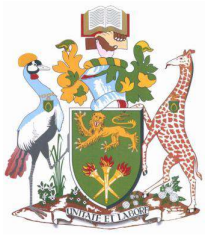

UNIVERSITY OF NAIROBI

COLLEGE OF BIOLOGICAL AND PHYSICAL SCIENCES

SCHOOL OF MATHEMATICS

TITLE: A MATHEMATICAL MODEL OF RIFT VALLEY FEVER IN LIVESTOCK IN KENYA

Submitted by,

MARILYN CHEPKURUI RONOH

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Supervisor

DR. JOSEPHINE KAGUNDA

A Dissertation Submitted to the School of Mathematics

in Partial Fulfillment for a Degree of Master of Science in Applied Mathematics

JULY, 2014

DECLARATION

I, the undersigned, declare that this dissertation is my original work and has not been presented for a degree in any other university.

Signature . . . . . Date . . . . . .

Ronoh Marilyn Chepkurui

I56/81613/2012

This dissertation has been submitted for examination with approval by my supervisor.

Signature . . . . . Date . . . . . .

Dr Josephine Kagunda

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Chapter 1

INTRODUCTION

1.1 Rift Valley Fever

Rift Valley fever, (RVF), is a vector borne disease transmitted by mosquitoes to livestock and wild animals. It is spread by the bite of infected mosquitoes, typically the Aedes, Culex and Anopheles genera. RVF virus is a member of the Phlebovirus genus, one of the five genera in the family Bunyaviridae. Aedes and Culex are believed to be the main vectors. Rift Valley fever virus can be transferred vertically from females to their eggs in some species of the Aedes mosquitoes.While humans can be infected with RVF, we restrict our focus in this study to livestock populations. The virus was first identified in 1931 during an investigation into an epidemic among sheep on a farm in the Rift Valley of Kenya. Since then, outbreaks have been reported in sub-Saharan and North Africa. In 1997-98, a major outbreak occurred in Kenya, Somalia and Tanzania and in September 2000, RVF cases were confirmed in Saudi Arabia and Yemen, marking the first reported occurrence of the disease outside the African continent and raising concerns that it could extend to other parts of Asia and Europe. A major outbreak of this fever occurred again in Kenya in 2006 Nov- 2007 March. The estimated number of cases among humans was

75,000 out of which 700 were reported and 158 numbers of deaths confirmed. Rift Valley Fever is associated with the Great Rift Valley System that runs from Zambezi River in Malawi to Lebanon. Most of this valley falls within the former Rift Valley Province.

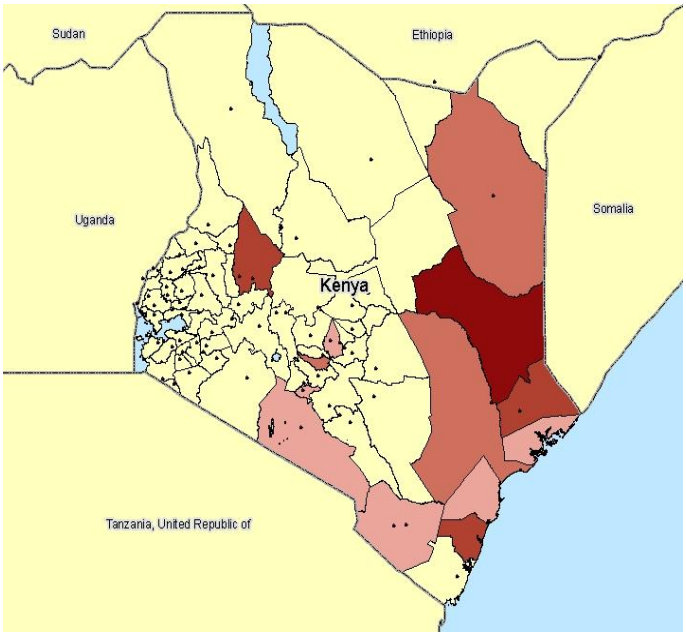

Figure 1.1: Rift Valley Fever Distribution in Kenya,[7]

1.2 Impact of Rift Valley Fever

While Rift Valley fever was originally associated with livestock, recent outbreaks in Kenya have resulted in increased fatality rates among humans, thereby presenting an increased threat to public health. It primarily affects animals but it also has the capacity to infect humans. Infection can cause severe disease in both animals and humans. The disease also results in significant economic losses due to death and abortion among RVF-infected livestock, not to mention morbidity and mortality in humans.

Epidemics of this disease usually emerge after above average and widespread rainfall. Below is the transmission cycle of the Rift Valley Fever.

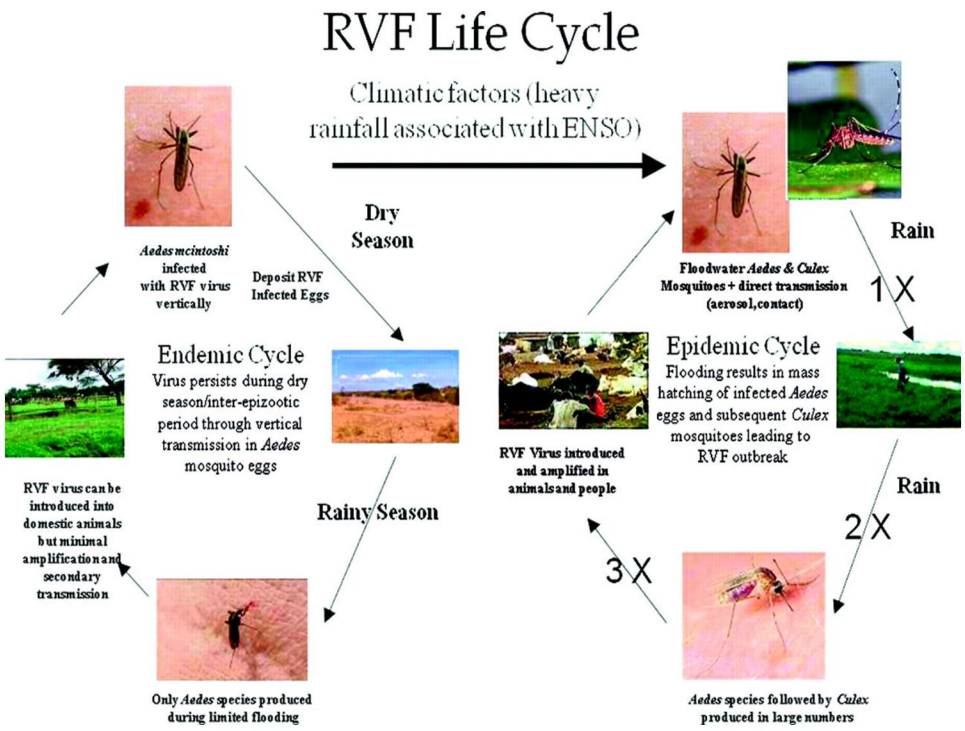

Figure 1.2: Rift Valley Fever Life Cycle,[8]

In this research we will develop a mathematical model that will best capture how this disease interacts with livestock. It is known that livestock particularly living in the high risk areas have in one way or the other through interaction with infected vectors, been infected with the Rift Valley Fever. Several deaths have been reported in livestock and to some extent humans. The disease causes serious effects on rural people's food security, particularly those communities that depend on their livestock for food and household nutrition and on direct and indirect losses to livestock producers in the country. Psycho-social distress that communities go through is enormous, which involves the thinking about the loss of their livestock and crop production. Socially, the status of most livestock producers is eroded in their communities.

Cessation of lucrative trade in ruminants results in serious economic losses to the populations who totally depend upon this income. Therefore, there is need to reduce these kinds of deaths by understanding how this disease is transmitted to livestock and what can be done to ensure there is no further loss of life.

This model will be used to simulate the impact of prevention and control options for the disease. Hopefully, the health and economic costs associated with Rift Valley fever virus can be understood and contained.

1.3 Definition of Terminologies

The Basic Reproduction Number

The reproduction number $\mathcal{R}_0$ is defined as the average number of secondary cases arising from an average primary case in an entirely susceptible population. The reproduction number is used to predict whether the epidemic will spread or die out. Any epidemiological model has a disease free equilibrium (DFE) at which the population remains in the absence of the disease. The basic reproduction number is such that if $\mathcal{R}_0 < 1$ then the DFE is locally asymptotically stable and the disease dies out but if $\mathcal{R}_0 > 1$ then the DFE is unstable and the epidemic spreads. At the endemic equilibrium, the average replacement number is one.

Metzler Matrices

A Metzler matrix is a matrix in which all the off-diagonal components are non negative (equal to or greater than zero). Many non-linear systems , $\dot{x} = Ax$ are modeled by a system of ordinary differential equations with constant coefficients. A necessary condition to keep $x_i(t) \geq 0$ for all i and all t is that $\dot{x} \geq 0$ when $x_i = 0$ and $x_j \geq 0$ for all i not equal to j. The condition will be fulfilled for the linear system above if and only if a $i_j \geq 0$ for all i not equal to j. A matrix that satisfies this condition is called a Metzler matrix. Many compartmental models with constant coefficients are a special subset of Metzler matrices.

Stability

Consider the differential equation $\dot{x} = f(t, x), x \in R^n$ then a point x is **Liaponouv stable** if and only if for all $\epsilon > 0$ there exists $\delta > 0$ such that if $|x-y| < \delta$ then $|f(x,t)-f(y,t)| < \epsilon$ for all $t \geq 0$. A point x is **quasi-asymptotically stable** iff there exists $\delta > 0$ such that if $|x - y| < \delta$ then $|\varphi(x,t) - \varphi(y,t)| \to 0$ as $t \to \infty$. A point x is **asymptotically stable** if it is both liaponouv stable and quasi-asymptotically stable.

Local Asymptotic Stability

A point x^* is an equilibrium point of the system if $f(x^*) = 0$. x^* is locally stable if all solutions which start near x^* (meaning that the initial conditions are in a neighborhood of x^*) remain near x^* for all time. The equilibrium point x^* is said to be **locally asymptotically stable** if x^* is locally stable and, furthermore, all solutions starting near x^* tend towards x^* as $t \to \infty$.

Global Asymptotic Stability

The system $\dot{x} = f(t, x)$ is **globally asymptotically stable** if for every trajectory $x(t)$, we have $x(t) \to x^*$ as $t \to \infty$ (implies x^* is the unique equilibrium point).

Positively Invariant

Consider the system $\dot{x} = f(t, x)$ and let $x(t, x_0)$ denote the trajectory of the system with an initial point x_0. Further let $\Omega = \{x \in R^n | \varphi(x) = 0\{$, where $\varphi(x)$ is real-valued function that characterizes the set Ω. The set Ω is said to be positively-invariant if $x_0 \in \Omega$, then $x(t, x_0) \in \Omega \forall t \geq 0$.

Compartmental Models

Compartmental models are often used to describe transport of material in biological systems. A compartment model contains a number of compartments, each containing well mixed material. Compartments exchange material with each other following certain rules. Compartments are represented by boxes and the connections between the compartments are represented by arrows. Every compartment (that is every box) has a number of connections leading to the box (inflows) and a number of arrows leading from the box (outflows). Material can either flow from one compartment to another, it can be added from the outside through a source like birth or new infection, or it can be removed through a drain where the drain in our case is death. Modeling of dynamical systems plays a very important role in applied science, and compartment models are among the most important tools used for analyzing dynamical systems. A few examples of compartmental models are listed below:

- SIR Model: The SIR model labels these three compartments S = number susceptible, I = number infectious, and R = number recovered. This is a good and simple model for many infectious diseases.

  Birth$\longrightarrow$ $\boxed{\text{S}}$ $\longrightarrow$ $\boxed{\text{I}}$ $\longrightarrow$ $\boxed{\text{R}}$ $\longrightarrow$ $Death$

- SEIR Model: The SEIR model labels four compartments S = number susceptible, E = number exposed, I = number infectious, and R = number recovered. For many important infections there is a significant incubation period during which the individual has been infected but is not yet infectious themselves. During this period the individual is in compartment E (for exposed).

Birth$\longrightarrow$ $\boxed{\text{S}}$ $\longrightarrow$ $\boxed{\text{E}}$ $\longrightarrow$ $\boxed{\text{I}}$ $\longrightarrow$ $\boxed{\text{R}}$ $\longrightarrow$ *Death*

- SIRS Model: The SIRS model labels these four compartments S = number susceptible, I = number infectious, R = number recovered and back to S, that is the recovered become susceptible.

Birth$\longrightarrow$ $\boxed{\text{S}}$ $\longrightarrow$ $\boxed{\text{I}}$ $\longrightarrow$ $\boxed{\text{R}}$ $\longrightarrow$ $\boxed{\text{S}}$

1.4 Statement of the Problem

This model will focus on the interaction between vectors(Aedes and Culex mosquito) and livestock hosts. We will use ordinary differential equations to describe the propagation of the disease in livestock host and the mosquitoes (Aedes and Culex). We shall define the basic reproduction ratio and analyze the local and global stability for both the Disease Free Equilibrium and Endemic Equilibrium using dynamical systems approach.
Below is the compartmental model showing how this vector borne disease interacts with livestock and vectors(Aedes and Culex mosquito). From which we will derive the differential equations that governs the transmission process.

Then analysis and simulation of this disease will follow.

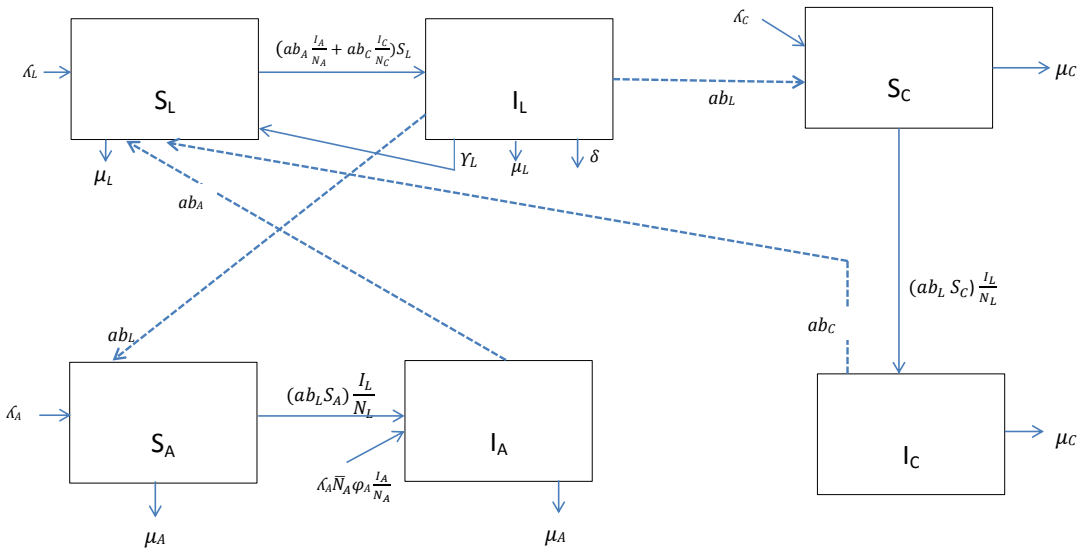

Figure 1.3: Proposed Compartmental Model

1.5 Objective of the Study

The objectives of this study are listed as follows:

- Formulate a deterministic model that describes the transmission of Rift Valley Fever in Livestock in Kenya.

- Show that the system is positively invariant.
- Find the basic reproduction number.
- Investigate and study the stability of the model at the Disease Free Equilibrium and Endemic Equilibrium.
- Numerical Simulation using MATLAB.

1.6 Significance of the Study

This study will have the following significance:

- Minimize deaths that results from Rift Valley Fever hence saving lives of livestock population as a result of a much more predictable model for future cases.
- Give me an opportunity to apply and learn more mathematical theories and their application in models biological systems.
- The model will provide a foundation for further research in epidemiology.

Chapter 2

LITERATURE REVIEW

Holly D. Gaff, David M. Hartley and Nicole P. Leahy presented and explored a novel mathematical model of the epidemiology of Rift Valley Fever (RVF). Their model was an ordinary differential equation model for two populations of mosquito species, those that could transmit vertically and those that could not, and for one livestock population. They analyzed the model to find the stability of the disease-free equilibrium and tested particular model parameters which affected the stability most significantly. This model was the basis for future research into the predication of future outbreaks. One population of vectors represented the Aedes mosquitoes which could be infected either vertically or via a blood meal from an infectious host. The other vector population was able to transmit RVFV to hosts but not to their offspring; here they considered it to be a population of Culex mosquitoes. Once infected, mosquito vectors remain infectious for the remainder of their lifespan. Infection was assumed not to affect mosquito behavior or longevity significantly. Hosts, which represented various livestock animals, could become infected when fed upon by infectious vectors. Hosts would then die from RVFV infection or recover, where upon they would have lifelong immunity to reinfection.

They computed the basic reproduction ratio and proved that when the reproduction number is less than 1, if the disease was introduced, there were insufficient new cases per case, and the disease could not invade the population. But when the reproduction number was greater than one, there exists an endemic equilibrium, which was also globally asymptotically stable.

Since the model incorporates both vertical and horizontal transmission the reproduction number for the system is the sum of the reproduction ratio values for each mode of transmission determined separately,$R_0 = R_{0,V} + R_{0,H}$ where the first term on the Left Hand Side is the reproduction number for the vertical transmission route and the second term in the Left Hand Side is the reproduction number for the horizontal transmission route.Using the concept of Metzler matrices and further simplification of the system they found:

$$R_0 = \frac{b_1 q_1}{d_1} + \sqrt{(\frac{\epsilon_2}{(d_2K_2 + \epsilon_2)(d_2K_2 + \gamma_2 + \mu_2)}(\frac{\epsilon_1\beta_{12}\beta_{21}}{d_1(d_1 + \epsilon_1)} + \frac{\epsilon_3\beta_{32}\beta_{23}}{d_3(d_3 + \epsilon_3)})}$$

For this study they analyzed the resulting model by computing the fundamental reproduction ratio and sensitivity of model output to variation or uncertainty in biological parameters using the technique of Latin hypercube sampling to test the sensitivity of the model to each input parameter in an approach successfully applied in the past to many other disease models.

However their model was too complicated to perform rigorous mathematical analysis.

Ling Xue, H. M. Scott, Lee W. Cohnstaedt, Caterina Scoglio used a network-based meta population approach to model Rift Valley fever epidemics.They proposed a new compartmentalized model of RVF and the related ordinary differential equations to assess disease spread in both time and space; with the latter driven as a function of contact networks. Humans and livestock hosts and two species of vector mosquitoes are included in the model. The model is based on weighted contact networks, where nodes of the networks represent geographical regions and the weights represent the level of contact between regional pairings for each set of species. The inclusion of human, animal, and vector movements among regions is new to RVF modeling. The benefit of their proposed model was twofold: not only could their model differentiate the maximum number of infected individuals among different provinces, but also it could reproduce the different starting times of the outbreak in multiple locations. The exact value of the reproduction number was numerically computed and upper and lower bounds for the reproduction number were derived analytically. Here is a brief description of their model: The main vectors are the Aedes and Culex mosquitoes and the main hosts are the livestock and humans. They use an SEI compartmental model in which individuals are either in a susceptible (S) state, an exposed (E) state, or an infected state (I) for both Aedes and Culex mosquitoes, and an SEIR compartmental model in which individuals are either in a susceptible (S) state, an exposed (E) state, an infected state (I), or a recovered (R) state for both livestock and human populations.

Infectious Aedes mosquitoes can not only transmit RVFV to susceptible livestock and humans but also to their own eggs. Culex mosquitoes acquire the virus during blood meals on an infected animal and then amplify the transmission of RVFV through blood meals on livestock and humans. Direct livestock-to-human contact is the major (though not only) way for humans to acquire the infection. The mosquitoes will not spontaneously recover once they become infectious. Livestock and humans either perish from the infection or recover. All four species have a specified incubation period. The model is based on a daily time step.

Using the concept of Metzler matrices and further simplification of the system they found:

$$R_0 = \sqrt{(\frac{\epsilon_2}{(d_2K_2+\epsilon_2)(d_2K_2+\gamma_2+\mu_2)}(\frac{\epsilon_1\beta_{12}\beta_{21}}{d_1(d_1+\epsilon_1)}+\frac{\epsilon_3\beta_{32}\beta_{23}}{d_3(d_3+\epsilon_3)})}$$

For this study they analyzed the resulting model by computing the fundamental reproduction ratio and the infection spreads due to movement of the four populations. The sensitivity analysis was estimated using the least square approach. However their model was too complicated to perform rigorous mathematical analysis.

Tianchan Niu(et al) described the foundations of a mathematical approach to access the spatial spread of an introduced RVF. Their approach was based on a previous model of RVF transmission in a small local population and multispecies epidemic models incorporating spatial structure more generally. A single Aedes mosquito was used to represent initial infection. Their RVF model considered Aedes mosquitoes, livestock (e.g., cattle, sheep, and goats), and Culex mosquitoes on a single patch. They identified the need to include spatial variation. This was accomplished within the framework of their compartmental model which models the epidemiological dynamics of arbitrary numbers of species occupying an arbitrary number of patches. Their approach included patch-specific contact

rates, incubation periods, and other biological factors. They also described a method for computing the stability of the disease-free equilibrium in terms of the basic reproduction ratio. They constructed and analyzed a mathematical model of RVF that includes both pathogen propagation within and spreading across different regions via the movement of humans, livestock, and mosquitoes. They analyzed their model to determine the stability and sensitivity of disease-free equilibrium. They used numerical methods to determine the reproduction number.

Egil AJ Fischer (et al) developed a mathematical model that captured the probability of a RVFV outbreak and the probability of persistence of the infection during consecutive years. They applied their model to create risk maps of the Netherlands showing high risk areas for RVF outbreak and for persistence of RVF in livestock. For these maps they considered host species to be cattle, sheep and goats, and considered vector species to be Aedes Mosquitoes and Culex Mosquitoes. They conducted an uncertainty analysis of the input parameters, which yielded knowledge about influential input parameters and data gaps, which could help focus future research and improve the accuracy of the model predictions. Their model described the local spread of the infection in a predefined small area in which all hosts and vectors mix homogeneously. In this study 5 by 5 kilometre area grids were used, based on the highest possible resolution for modelled mosquito abundances. They assumed constant host population sizes and no effect of temperature on host related parameters. Given that activity and survival of mosquitoes during winter months and especially how that affects the virus is poorly understood they assumed a period of stasis during winter, i.e. the number of susceptible and infected vectors and the number of susceptible, infected and recovered hosts at the beginning of the vector season is equal to the situation at the end of the previous vector season implying that the infection cannot die out during the winter in their model. For convenience they assumed stasis of the host as well. To test the effect

of stasis of the host they performed a sensitivity analysis to evaluate its impact and they found a reappearing epidemic after a stasis period very quickly returning to the pattern of a continued epidemic.

Chapter 3

THE MODEL

3.1 Compartmental Model and Differential Equations

We will use an SIS Compartmental Model in which individuals are either in a susceptible (S) state or an infected (I) state for livestock populations. Livestock either die from the infection or recover. The infected host(livestock) who recover become susceptible again as Rift Valley Fever has no permanent immunity. We will use an SI Compartmental Model for Aedes and Culex mosquitoes (vector) populations. Infectious Aedes and Culex mosquitoes transmit Rift Valley Fever vector to susceptible livestock. We assume that once mosquitoes become infected with Rift Valley Fever they do not recover. They remain infected until death. Aedes mosquito will have vertical transmission to their eggs whereas Culex Mosquito will not.

The size of host populations is $N_L = S_L + I_L$ for livestock host. The size of vector population is $N_A = S_A + I_A$ for Aedes Mosquito and $N_C = S_C + I_C$ for Culex Mosquito.

The compartmental model showing the interaction of Rift Valley Fever in humans and livestock is given in figure 3.1.

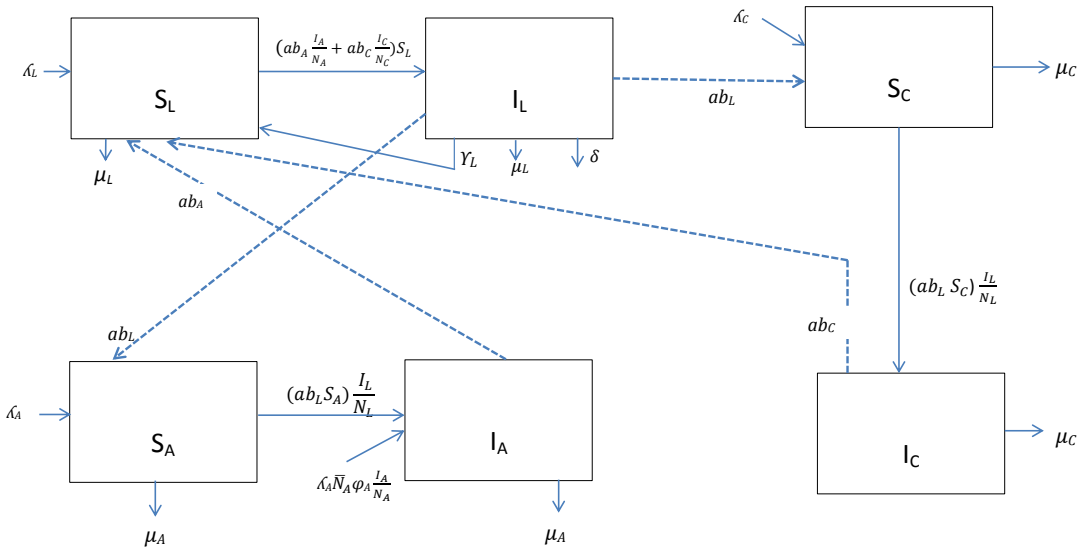

Figure 3.1: Compartmental Model

The system of Ordinary Differential Equations representing the populations is given below:

Livestock Population Model

$$\frac{dS_L(t)}{dt} = \lambda_L - ab_A S_L(t)\frac{I_A(t)}{N_A} - ab_C S_L(t)\frac{I_C(t)}{N_C} - \mu_L S_L(t) + \gamma_L I_L(t)$$

$$\frac{dI_L(t)}{dt} = ab_A S_L(t)\frac{I_A(t)}{N_A} + ab_C S_L(t)\frac{I_C(t)}{N_C} - \mu_L I_L(t) - \gamma_L I_L(t) - \sigma I_L(t)$$

Aedes Mosquito Population Model

$$\frac{dS_A(t)}{dt} = \lambda_A - ab_L S_A(t)\frac{I_L(t)}{N_L} - \mu_A S_A(t)$$

$$\frac{dI_A(t)}{dt} = \lambda_A \bar{N}_A \varphi_A \frac{I_A(t)}{N_A} + ab_L S_A(t)\frac{I_L(t)}{N_L} - \mu_A I_A(t)$$

Culex Mosquito Population Model

$$\frac{dS_C(t)}{dt} = \lambda_C - ab_L S_C(t)\frac{I_L(t)}{N_L} - \mu_C S_C(t)$$

$$\frac{dI_C(t)}{dt} = ab_L S_C(t)\frac{I_L(t)}{N_L} - \mu_C I_C(t)$$

The parameters are described in the table below:

Table 3.1: Description of Parameters

	Parameter	Description
1	S_L	Susceptible Livestock
2	S_A	Susceptible Aedes Mosquitoes
3	S_C	Susceptible Culex Mosquitoes
4	I_L	Infected Livestock
5	I_A	Infected Aedes Mosquitoes
6	I_C	Infected Culex Mosquitoes
7	μ_L	Death rate of livestock in a population
8	μ_A	Death rate of Aedes Mosquitoes in a population
9	μ_C	Death rate of Culex Mosquitoes in a population
10	λ_L	Birth rate of Livestock
11	λ_A	Number of Aedes Mosquitoes eggs laid per day
12	λ_C	Number of Culex Mosquitoes eggs laid per day
13	b_L	Probability a susceptible vector gets infected after biting an infected livestock
14	b_A	Probability a susceptible livestock gets infected after an infectious bite by Aedes M
15	b_C	Probability a susceptible livestock gets infected after an infectious bite by Culex M
16	γ_L	Recovery rate of infected livestock
17	σ	Disease induced death in Livestock
18	φ_A	Probability of vertical infection of Aedes Mosquito eggs

We assume that births and deaths are equal. Given that we have disease induced death in the livestock population model, the livestock population then cannot be constant. The total mosquito population is constant. To prove this we will add up the differential equations in the Livestock population model and the vector (Aedes and Culex Mosquito) population model. We shall omit the independent variable t, for brevity in the preceding sections.This yields the following:

Livestock Population Model:

$$\frac{dS_L}{dt} + \frac{dI_L}{dt} = \lambda_L - N_L\mu_L - \sigma I_L \ldots\ldots\ldots\ldots\ldots (3.1)$$

Aedes Mosquito Population Model:

$$\frac{dS_A}{dt} + \frac{dI_A}{dt} = \lambda_A + \lambda_A\varphi_A\frac{I_A}{N_A}\bar{N_A}) - N_A\mu_A \ldots\ldots\ldots\ldots\ldots (3.2)$$

Culex Mosquito Population Model:

$$\frac{dS_C}{dt} + \frac{dI_C}{dt} = \lambda_C - N_C\mu_C \ldots\ldots\ldots\ldots\ldots (3.3)$$

3.2 Disease Free State

The disease free state implies that $I_L = 0$, $I_A = 0$ and $I_C = 0$. This also implies that $N_L = S_L$, $N_A = S_A$ and $N_C = S_C$. From equation (3.1) we have that $\frac{dN_L}{dt} = \lambda_L - \mu_L N_L$ which implies that $\frac{dN_L}{dt} + \mu_L N_L = \lambda_L$. The integrating factor is $e^{\int \mu_L dt} = e^{\mu_L t}$ therefore $\frac{d}{dt}(N_L e^{\mu_L t}) = \lambda_L e^{\mu_L t}$ thus $\int \frac{d}{dt}(N_L e^{\mu_L t}) = \int \lambda_L e^{\mu_L t} dt$ implying that $N_L e^{\mu_L t} = \frac{\lambda_L e^{\mu_L t}}{\mu_L} + K$ where K is any arbitrary constant. Thus $N_L^* = \frac{\lambda_L}{\mu_L} + Ke^{-\mu_L t}$. At $t = 0, N_L(t) = N_L^0$. This implies that $N_L^0 = \frac{\lambda_L}{\mu_L} + K$. Hence $K = N_L^0 - \frac{\lambda_L}{\mu_L}$. Therefore $N_L^* = \frac{\lambda_L}{\mu_L} + e^{-\mu_L t}(N_L^0 - \frac{\lambda_L}{\mu_L})$.

Similarly from equation (3.2) we have that $\frac{dN_A}{dt} = \lambda_A - \mu_A N_A$ which implies that $\frac{dN_A}{dt} + \mu_A N_A = \lambda_A$. The integrating factor is $e^{\int \mu_A dt} = e^{\mu_A t}$ therefore $\frac{d}{dt}(N_A e^{\mu_A t}) = \lambda_A e^{\mu_A t}$ thus $\int \frac{d}{dt}(N_A e^{\mu_A t}) = \int \lambda_A e^{\mu_A t} dt$ implying that $N_A e^{\mu_A t} = \frac{\lambda_A e^{\mu_A t}}{\mu_A} + K$ where K is any arbitrary constant. Thus $N_A^* = \frac{\lambda_A}{\mu_A} + Ke^{-\mu_A t}$. At $t = 0, N_A(t) = N_A^0$. This implies that $N_A^0 = \frac{\lambda_A}{\mu_A} + K$. Hence $K = N_A^0 - \frac{\lambda_A}{\mu_A}$. Therefore $N_A^* = \frac{\lambda_A}{\mu_A} + e^{-\mu_A t}(N_A^0 - \frac{\lambda_A}{\mu_A})$.

Finally from equation (3.3) we have that $\frac{dN_C}{dt} = \lambda_C - \mu_C N_C$ which implies that $\frac{dN_C}{dt} + \mu_C N_C = \lambda_C$. The integrating factor is $e^{\int \mu_C dt} = e^{\mu_C t}$ therefore $\frac{d}{dt}(N_C e^{\mu_C t}) = \lambda_C e^{\mu_C t}$ thus $\int \frac{d}{dt}(N_C e^{\mu_C t}) = \int \lambda_C e^{\mu_C t} dt$ implying that $N_C e^{\mu_C t} = \frac{\lambda_C e^{\mu_C t}}{\mu_C} + K$ where K is any arbitrary constant. Thus $N_C^* = \frac{\lambda_C}{\mu_C} + Ke^{-\mu_C t}$. At $t = 0, N_C(t) = N_C^0$. This implies that $N_C^0 = \frac{\lambda_C}{\mu_C} + K$. Hence $K = N_C^0 - \frac{\lambda_C}{\mu_C}$. Therefore $N_C^* = \frac{\lambda_C}{\mu_C} + e^{-\mu_C t}(N_C^0 - \frac{\lambda_C}{\mu_C})$.

At $t = 0$, $N_L^* = \frac{\lambda_L}{\mu_L}$, $N_A^* = \frac{\lambda_A}{\mu_A}$ and $N_C^* = \frac{\lambda_C}{\mu_C}$.

3.3 Positively Invariant

We have a dynamic population thus S_L can be obtained once we know I_L and likewise for S_A and S_C given that $N_L = S_L + I_L$, $N_A = S_A + I_A$ and $N_C = S_C + I_C$. Thus we will analyze the dynamics of I_L, I_A and I_C. Also of importance, all parameters are non negative. Let $\delta_L = \mu_L + \gamma_L + \sigma$ We will be looking at the following system of differential equations:

$$\frac{dI_L}{dt} = (ab_A\frac{I_A}{N_A} + ab_C\frac{I_C}{N_C})(N_L - I_L) - \delta_L I_L \ldots\ldots\ldots\ldots\ldots (3.4)$$

$$\frac{dI_A}{dt} = \lambda_A\bar{N_A}\varphi_A\frac{I_A}{N_A} + ab_L\frac{I_L}{N_L}(N_A - I_A) - \mu_A I_A \ldots\ldots\ldots\ldots\ldots (3.5)$$

$$\frac{dI_C}{dt} = ab_L\frac{I_L}{N_L}(N_C - I_C) - \mu_C I_C \ldots\ldots\ldots\ldots\ldots (3.6)$$

We define the region K as follows:

$$K = \{(I_L, I_A, I_C) | 0 \leq I_L \leq N_L,\ 0 \leq I_A \leq N_A,\ 0 \leq I_C \leq N_C\}$$

We want to show that K is positively invariant.

Proof

When $I_L = 0$ equation (3.4) becomes $\frac{dI_L}{dt} = (ab_A\frac{I_A}{N_A} + ab_C\frac{I_C}{N_C})N_L \geq 0$ and the livestock population can only increase. When $I_L = N_L$ equation (3.4) becomes $\frac{dI_L}{dt} = -\delta_L N_L \leq 0$ and the livestock population can only decrease. Similarly when $I_A = 0$, equation (3.5) becomes $\frac{dI_A}{dt} = ab_L\frac{I_L}{N_L}N_A \geq 0$ hence an increase in the Aedes mosquito population. When $I_A = N_A$ equation (3.5) becomes $\frac{dI_A}{dt} = -(\mu_A N_A - \lambda_A \bar{N_A}\varphi_A) \leq 0$ implying a decrease in the Aedes Mosquito population. Finally, when $I_C = 0$, equation (3.6) becomes $\frac{dI_C}{dt} = ab_L\frac{I_L}{N_L}N_C \geq 0$ indicating an increase in the Culex mosquito population. When $I_C = N_C$ equation (3.6) becomes $\frac{dI_C}{dt} = -\mu_C N_C \leq 0$ indicating a decrease in the Culex Mosquito Population. The vector fields cannot cross the positive orthant to the negative hence positively invariant as shown in the region K of figure 3.2.

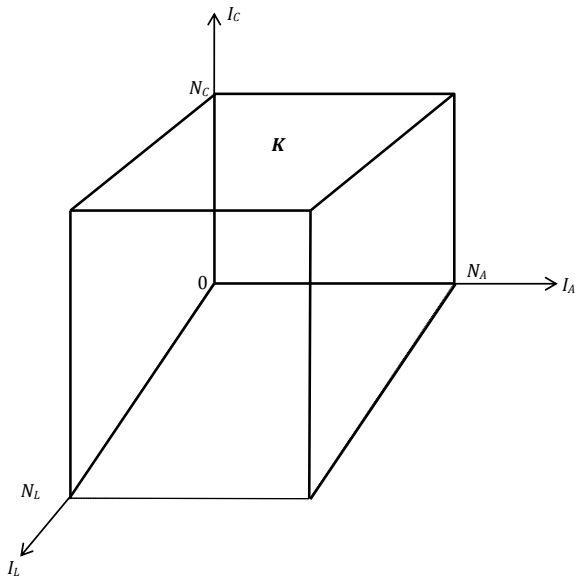

Figure 3.2: Domain of Study

3.4 Calculation of the Basic Reproduction Number

The basic reproduction number, $\mathcal{R}_0$ is defined as the average number of secondary cases arising from an average primary case in an entirely susceptible population[10]. $\mathcal{R}_0$ is used to predict whether the epidemic will spread or die out. In this section we will analyze the dynamics of I_L, I_A and I_C so as to be able to obtain $\mathcal{R}_0$. Let us thus look at the following system of differential equations. Let $\Lambda_A = \lambda_A \bar{N_A} \varphi_A \frac{1}{N_A}$. Thus

$$\frac{dI_L}{dt} = (ab_A \frac{I_A}{N_A} + ab_C \frac{I_C}{N_C})(N_L - I_L) - \delta_L I_L$$

$$\frac{dI_A}{dt} = ab_L \frac{I_L}{N_L}(N_A - I_A) - (\mu_A - \Lambda_A) I_A$$

$$\frac{dI_C}{dt} = ab_L \frac{I_L}{N_L}(N_C - I_C) - \mu_C I_C$$

The above system can be represented in matrix form as $\dot{I} = fI + vI$ where f is the matrix of the infection rates and v is the matrix of the transition rates so that;

$$f = \begin{pmatrix} (ab_A \frac{I_A}{N_A} + ab_C \frac{I_C}{N_C})(N_L - I_L) \\ ab_L \frac{I_L}{N_L}(N_A - I_A) \\ ab_L \frac{I_L}{N_L}(N_C - I_C) \end{pmatrix}, \; v = \begin{pmatrix} -\delta_L I_L \\ -(\mu_A - \Lambda_A) I_A \\ -\mu_C I_C \end{pmatrix}$$

Now let:

$$f_1 = (ab_A\frac{I_A}{N_A} + ab_C\frac{I_C}{N_C})(N_L - I_L)$$

$$f_2 = ab_L\frac{I_L}{N_L}(N_A - I_A)$$

$$f_3 = ab_L\frac{I_L}{N_L}(N_C - I_C)$$

Next we define $\mathcal{F}$ the Jacobian of f at $(0,0,0)$. Let $ab_A = \beta_A$, $ab_C = \beta_C$ and $ab_L = \beta_L$. Thus

$$\mathcal{F}=\begin{pmatrix} \frac{\partial f_1}{\partial I_L} & \frac{\partial f_1}{\partial I_A} & \frac{\partial f_1}{\partial I_C} \\ \frac{\partial f_2}{\partial I_L} & \frac{\partial f_2}{\partial I_A} & \frac{\partial f_2}{\partial I_C} \\ \frac{\partial f_3}{\partial I_L} & \frac{\partial f_3}{\partial I_A} & \frac{\partial f_3}{\partial I_C} \end{pmatrix}$$

This implies that:

$$\mathcal{F} = \begin{pmatrix} 0 & \frac{\beta_A}{N_A} & \frac{\beta_C}{N_C} \\ \frac{\beta_L}{N_L} & 0 & 0 \\ \frac{\beta_L}{N_L} & 0 & 0 \end{pmatrix}$$, and we obtain $\mathcal{V}$ the Jacobian of v at $(0,0,0)$. That is:

Let:

$v_1 = -\delta_L I_L$

$v_2 = -(\mu_A - \Lambda_A)I_A$

$v_3 = -\mu_C I_C$

Thus:

$$\mathcal{V} = -\begin{pmatrix} \frac{\partial v_1}{\partial I_L} & \frac{\partial v_1}{\partial I_A} & \frac{\partial v_1}{\partial I_C} \\ \frac{\partial v_2}{\partial I_L} & \frac{\partial v_2}{\partial I_A} & \frac{\partial v_2}{\partial I_C} \\ \frac{\partial v_3}{\partial I_L} & \frac{\partial v_3}{\partial I_A} & \frac{\partial v_3}{\partial I_C} \end{pmatrix}$$

This implies that:

$$\mathcal{V} = -\begin{pmatrix} \delta_L & 0 & 0 \\ 0 & \mu_A - \Lambda_A & 0 \\ 0 & 0 & \mu_C \end{pmatrix}$$

We will now use Gauss Jordan elimination method to obtain $\mathcal{V}^{-1}$

Thus we have:

$$-\left(\begin{array}{ccc|ccc} \delta_L & 0 & 0 & 1 & 0 & 0 \\ 0 & \mu_A - \Lambda_A & 0 & 0 & 1 & 0 \\ 0 & 0 & \mu_C & 0 & 0 & 1 \end{array}\right)$$

Dividing row 1 by δ_L, and row 2 by $\mu_A - \Lambda_A$ and row 3 by μ_C yields the following:

$$-\left(\begin{array}{ccc|ccc} 1 & 0 & 0 & \frac{1}{\delta_L} & 0 & 0 \\ 0 & 1 & 0 & 0 & \frac{1}{\mu_A - \Lambda_A} & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{1}{\mu_C} \end{array}\right)$$

Thus:

$$\mathcal{V}^{-1} = \begin{pmatrix} -(\frac{1}{\delta_L}) & 0 & 0 \\ 0 & -(\frac{1}{\mu_A - \Lambda_A}) & 0 \\ 0 & 0 & -(\frac{1}{\mu_C}) \end{pmatrix}$$

Now we are going to obtain the metzler matrix that is $(-\mathcal{F}\mathcal{V}^{-1})$.

$$-\mathcal{F}\mathcal{V}^{-1} = \begin{pmatrix} 0 & \frac{\beta_A}{N_A(\mu_A - \Lambda_A)} & \frac{\beta_C}{N_C \mu_C} \\ \frac{\beta_L}{N_L \delta_L} & 0 & 0 \\ \frac{\beta_L}{N_L \delta_L} & 0 & 0 \end{pmatrix}$$

The spectral radius of the Metzler Matrix, $\rho(-\mathcal{F}\mathcal{V}^{-1})$, is defined as the largest eigenvalue of the Metzler Matrix[10].

Thus:

$$\rho(-\mathcal{F}\mathcal{V}^{-1}) = |(-\mathcal{F}\mathcal{V}^{-1}) - \lambda| = \begin{vmatrix} -\lambda & \frac{\beta_A}{N_A(\mu_A - \Lambda_A)} & \frac{\beta_C}{N_C \mu_C} \\ \frac{\beta_L}{N_L \delta_L} & -\lambda & 0 \\ \frac{\beta_L}{N_L \delta_L} & 0 & -\lambda \end{vmatrix} =$$

$$-\lambda \begin{vmatrix} -\lambda & 0 \\ 0 & -\lambda \end{vmatrix} - \frac{\beta_A}{N_A(\mu_A - \Lambda_A)} \begin{vmatrix} \frac{\beta_L}{N_L\delta_L} & 0 \\ \frac{\beta_L}{N_L\delta_L} & -\lambda \end{vmatrix} + \frac{\beta_C}{N_C\mu_C} \begin{vmatrix} \frac{\beta_L}{N_L\delta_L} & -\lambda \\ \frac{\beta_L}{N_L\delta_L} & 0 \end{vmatrix} = 0$$

Thus

$$-\lambda^3 - \frac{\beta_A}{N_A(\mu_A - \Lambda_A)} \left[-\frac{\beta_L}{N_L\delta_L} \right] \lambda + \frac{\beta_C}{N_C\mu_C} \left[\frac{\beta_L}{N_L\delta_L} \right] \lambda = 0$$

which yields the following

$$-\lambda \left[\lambda^2 - \left(\frac{\beta_A\beta_L}{(N_A(\mu_A - \Lambda_A))(N_L\delta_L)} + \frac{\beta_C\beta_L}{N_C\mu_C(N_L\delta_L)} \right) \right] = 0$$

The $\rho(-\mathcal{F}\mathcal{V}^{-1}) = EV = \sqrt{\frac{\beta_L}{N_L\delta_L} \left[\frac{\beta_A}{N_A(\mu_A - \Lambda_A)} + \frac{\beta_C}{N_C\mu_C} \right]}$. Thus the basic reproduction number, $\mathcal{R}_0 = (EV)^2$ and it will be given as

$$\mathrm{R}_0 = \frac{\beta_L}{N_L\delta_L} \left[\frac{\beta_A}{N_A(\mu_A - \Lambda_A)} + \frac{\beta_C}{N_C\mu_C} \right] \ldots\ldots\ldots\ldots\ldots (3.7)$$

From the expression above for $\mathcal{R}_0$, $\frac{\beta_L}{N_L\delta_L}$ is the Livestock contribution to the $\mathcal{R}_0$ whereas $\frac{\beta_A}{N_A(\mu_A - \Lambda_A)}$ is the Aedes contribution to $\mathcal{R}_0$ and finally $\frac{\beta_C}{N_C\mu_C}$ is the Culex contribution to the $\mathcal{R}_0$.

3.5 Global Stability of the Disease Free Equilibrium

The local dynamics of a general SIS and SI model is determined by the reproduction number $\mathcal{R}_0$. If $\mathcal{R}_0 \leq 1$, then each infected individual in its entire period of infectiousness will produce less than one infected individual on average. This means that the disease will be wiped out of the population. If $\mathcal{R}_0 > 1$, then each infected individual in its entire infectious period having contact with susceptible individuals will produce more than one infected individual implying that the disease persists in the population. If $\mathcal{R}_0 = 1$, and this is defined as the disease threshold, then one individual infects one more individual. For $\mathcal{R}_0 \leq 1$ the disease free equilibrium is locally asymptotically stable while for $\mathcal{R}_0 > 1$ the disease free equilibrium becomes unstable. By using the theory of Lasalle-Lyapunov function V, we will show the global asymptotic stability. The disease free equilibrium point is $(I_L, I_A, I_C) = (0, 0, 0)$. ,

Theorem 3.1

If $\mathcal{R}_0 \leq 1$, then the disease-free equilibrium $(I_L, I_A, I_C) = (0, 0, 0)$ of the system is globally asymptotically stable on K.

Proof

We construct the following Lasalle-Lyapunov function $V(I_L, I_A, I_C)$ on the positively invariant compact set K. Thus on K, $V(I_L, I_A, I_C)$ is continuous and non negative.

We define

$$V(I_L, I_A, I_C) = \mu_C(\mu_A - \Lambda_A)I_L + \frac{\beta_A}{N_A}\mu_C I_A + \frac{\beta_C}{N_C}(\mu_A - \Lambda_A)I_C$$

Note that $\mu_A > \Lambda_A$

The system of ordinary differential equations given by equations 3.4, 3.5 and 3.6 can be written as

$$\begin{pmatrix} \dot{I}_L \\ \dot{I}_A \\ \dot{I}_C \end{pmatrix} = \begin{pmatrix} -\delta_L & \frac{\beta_A}{N_A} & \frac{\beta_C}{N_C} \\ \frac{\beta_L}{N_L} & -(\mu_A - \Lambda_A) & 0 \\ \frac{\beta_L}{N_L} & 0 & -\mu_C \end{pmatrix} \begin{pmatrix} I_L \\ I_A \\ I_C \end{pmatrix}$$

This can be written as $\dot{I} = A(I)$ where $A = \begin{pmatrix} -\delta_L & \frac{\beta_A}{N_A} & \frac{\beta_C}{N_C} \\ \frac{\beta_L}{N_L} & -(\mu_A - \Lambda_A) & 0 \\ \frac{\beta_L}{N_L} & 0 & -\mu_C \end{pmatrix}$ and $I = \begin{pmatrix} I_L \\ I_A \\ I_C \end{pmatrix}$. If we define $v^T = \left[\mu_C(\mu_A - \Lambda_A), \frac{\beta_A}{N_A}\mu_C, \frac{\beta_C}{N_C}(\mu_A - \Lambda_A)\right]$, then the derivative along the trajectories is given by $\dot{V} = v^T A(I)$ as

$$v^T A(I) = \left[\mu_C(\mu_A - \Lambda_A), \frac{\beta_A}{N_A}\mu_C, \frac{\beta_C}{N_C}(\mu_A - \Lambda_A)\right] \begin{bmatrix} -\delta_L & \frac{\beta_A}{N_A} & \frac{\beta_C}{N_C} \\ \frac{\beta_L}{N_L} & -(\mu_A - \Lambda_A) & 0 \\ \frac{\beta_L}{N_L} & 0 & -\mu_C \end{bmatrix} =$$

$$\begin{bmatrix} -\mu_C\delta_L(\mu_A - \Lambda_A) + \frac{\beta_L}{N_L}\frac{\beta_A}{N_A}\mu_C + \frac{\beta_L}{N_L}\frac{\beta_C}{N_C}(\mu_A - \Lambda_A) \\ \frac{\beta_A}{N_A}\mu_C(\mu_A - \Lambda_A) - \mu_C(\mu_A - \Lambda_A)\frac{\beta_A}{N_A} \\ \frac{\beta_C}{N_C}(\mu_A - \Lambda_A)\mu_C - \mu_C\frac{\beta_C}{N_C}(\mu_A - \Lambda_A) \end{bmatrix} =$$

$$\begin{bmatrix} -\mu_C\delta_L(\mu_A-\Lambda_A)+\mu_C\delta_L(\mu_A-\Lambda_A)\left(\dfrac{\beta_L\beta_A}{N_L\delta_L N_A(\mu_A-\Lambda_A)}\right)+\mu_C\delta_L(\mu_A-\Lambda_A)\left(\dfrac{\beta_C\beta_L}{N_C\mu_C N_L\delta_L}\right) \\ 0 \\ 0 \end{bmatrix}$$

$$v^T A(I)^T = \left[\mu_C\delta_L(\mu_A-\Lambda_A)\left(-1+\frac{\beta_L\beta_A}{N_L\delta_L N_A(\mu_A-\Lambda_A)}+\frac{\beta_C\beta_L}{N_C\mu_C N_L\delta_L}\right),0,0\right]$$

$$v^T A(I)^T = \mu_C\delta_L(\mu_A-\Lambda_A)\left[(\mathcal{R}_0^2-1),0,0\right]$$

$v^T A(I)^T = \mu_C\delta_L(\mu_A-\Lambda_A)(\mathcal{R}_0+1)\left[(\mathcal{R}_0-1),0,0\right]$ which is strictly decreasing when $\mathcal{R}_0<1$. Thus $\dot{V}\leq\mu_C\delta_L(\mu_A-\Lambda_A)(\mathcal{R}_0+1)(\mathcal{R}_0-1)I_L$

We define the set $E=\left\{(I_L,I_A,I_C)\in K|\dot{V}(I_L,I_A,I_C)=0\right\}$. The largest invariant set is contained in the set E for which $I_L=0$ or $I_A=0$ or $I_C=0$. Thus $\dot{V}<0$ when $\mathcal{R}_0<1$. If $I_L=0$ or $\mathcal{R}_0=1$ then $\dot{V}=0$. Thus by Lasalle's invariance principle the disease free equilibrium is globally asymptotically stable on K.

3.6 Stability of the Endemic Equilibrium

When $\mathcal{R}_0 > 1$ then the unique endemic equilibrium exists and is asymptotically stable. Let (I_L^*, I_A^*, I_C^*) denote the endemic equilibrium. In order to obtain the expressions for I_L^*, I_A^*, I_C^* we equate the RHS of equations 3.4, 3.5 and 3.6 to zero and express I_L^*, I_A^*, I_C^* as shown below. For N_L, N_A, N_C we substitute them with the expressions of N_L^*, N_A^*, N_C^* obtained in section 3.2.

$$(\frac{\beta_A I_A^*}{N_A^*} + \frac{\beta_C I_C^*}{N_C^*})(N_L^* - I_L^*) = \delta_L I_L^* \ldots\ldots\ldots\ldots\ldots (3.8)$$

$$\frac{\beta_L I_L^*}{N_L^*}(N_A^* - I_A^*) = (\mu_A - \Lambda_A) I_A^* \ldots\ldots\ldots\ldots\ldots (3.9)$$

$$\frac{\beta_L I_L^*}{N_L^*}(N_C^* - I_C^*) = \mu_C I_C^* \ldots\ldots\ldots\ldots\ldots (3.10)$$

We can express equation 3.10 as follows

$$I_C^* = \frac{\beta_L I_L^* N_C^*}{\beta_L I_L^* + N_L^* \mu_C} \tag{3.11}$$

Equation 3.9 can be written as

$$I_A^* = \frac{\beta_L I_L^* N_A^*}{\beta_L I_L^* + N_L^*(\mu_A - \Lambda_A)} \tag{3.12}$$

Substituting the expressions of equations 3.11 and 3.12 in equation 3.8 yields the following expression

$$\left[\frac{\beta_A \beta_L I_L^* N_A^*}{\beta_L I_L^* + N_L^*(\mu_A - \Lambda_A)} + \frac{\beta_C \beta_L I_L^* N_C^*}{\beta_L I_L^* + N_L^* \mu_C}\right](N_L^* - I_L^*) - \delta_L I_L^* = 0 \ldots\ldots\ldots\ldots\ldots \tag{3.13}$$

which can be expressed as

$$[\delta_L \beta_L^2 + \beta_A \beta_L^2 N_A^* + \beta_C \beta_L^2 N_C^*] I_L^{*2} +$$
$$[\delta_L \beta_L N_L^* \mu_C + \delta_L \beta_L N_L^*(\mu_A - \Lambda_A) + \beta_A \beta_L N_A^* N_L^* \mu_C + \beta_C \beta_L N_C^* N_L^*(\mu_A - \Lambda_A) - \beta_A \beta_L^2 N_A^* N_L^* -$$
$$\beta_C \beta_L^2 N_C^* N_L^*] I_L^* +$$
$$\delta_L N_L^{*2} \mu_C(\mu_A - \Lambda_A) - \beta_A \beta_L N_A^* N_L^{*2} \mu_C - \beta_C \beta_L N_C^* N_L^{*2}(\mu_A - \Lambda_A) = 0 \ldots\ldots\ldots\ldots\ldots, \tag{3.14}$$

and equation 3.14 becomes

$$\left[I_L^* + \frac{\delta_L\beta_L N_L^*\mu_C + \delta_L\beta_L N_L^*(\mu_A-\Lambda_A) + \beta_A\beta_L N_A^* N_L^*\mu_C + \beta_C\beta_L N_C^* N_L^*(\mu_A-\Lambda_A) - \beta_A\beta_L^2 N_A^* N_L^* - \beta_C\beta_L^2 N_C^* N_L^*}{2(\delta_L\beta_L^2 + \beta_A\beta_L^2 N_A^* + \beta_C\beta_L^2 N_C^*)}\right]^2$$
$$= \frac{\beta_A\beta_L N_A^* {N_L^*}^2\mu_C + \beta_C\beta_L N_C^* {N_L^*}^2(\mu_A-\Lambda_A) - \delta_L {N_L^*}^2\mu_C(\mu_A-\Lambda_A)}{\delta_L\beta_L^2 + \beta_A\beta_L^2 N_A^* + \beta_C\beta_L^2 N_C^*} +$$
$$\left[\frac{\delta_L\beta_L N_L^*\mu_C + \delta_L\beta_L N_L^*(\mu_A-\Lambda_A) + \beta_A\beta_L N_A^* N_L^*\mu_C + \beta_C\beta_L N_C^* N_L^*(\mu_A-\Lambda_A) - \beta_A\beta_L^2 N_A^* N_L^* - \beta_C\beta_L^2 N_C^* N_L^*}{2(\delta_L\beta_L^2 + \beta_A\beta_L^2 N_A^* + \beta_C\beta_L^2 N_C^*)}\right]^2$$
$$\ldots\ldots\ldots\ldots\ldots\ldots (3.15)$$

Let

$$\omega = \delta_L\beta_L^2 + \beta_A\beta_L^2 N_A^* + \beta_C\beta_L^2 N_C^*$$

$$\omega_1 = \beta_A\beta_L N_A^* {N_L^*}^2\mu_C + \beta_C\beta_L N_C^* {N_L^*}^2(\mu_A-\Lambda_A) - \delta_L {N_L^*}^2\mu_C(\mu_A-\Lambda_A)$$

$$\omega_2 = \delta_L\beta_L N_L^*\mu_C + \delta_L\beta_L N_L^*(\mu_A-\Lambda_A) + \beta_A\beta_L N_A^* N_L^*\mu_C + \beta_C\beta_L N_C^* N_L^*(\mu_A-\Lambda_A) - \beta_A\beta_L^2 N_A^* N_L^* -$$
$$\beta_C\beta_L^2 N_C^* N_L^*$$

Therefore
$$I_L^* = -\left[\frac{\delta_L\beta_L N_L^*\mu_C + \delta_L\beta_L N_L^*(\mu_A-\Lambda_A) + \beta_A\beta_L N_A^* N_L^*\mu_C + \beta_C\beta_L N_C^* N_L^*(\mu_A-\Lambda_A) - \beta_A\beta_L^2 N_A^* N_L^* - \beta_C\beta_L^2 N_C^* N_L^*}{2\omega}\right]$$
$$+\left[\frac{4\omega\omega_1 + \omega_2^2}{4\omega^2}\right]^{\frac{1}{2}}$$

$$= \frac{1}{2\omega}\left[-\delta_L\beta_L N_L^*\mu_C - \delta_L\beta_L N_L^*(\mu_A-\Lambda_A) - \beta_A\beta_L N_A^* N_L^*\mu_C - \beta_C\beta_L N_C^* N_L^*(\mu_A-\Lambda_A) + \beta_A\beta_L^2 N_A^* N_L^* + \beta_C\beta_L^2 N_C^* N_L^*) + \sqrt{4\omega\omega_1+\omega_2^2}\right]$$

Let $\omega_3 = -\delta_L\beta_L N_L^*\mu_C - \delta_L\beta_L N_L^*(\mu_A-\Lambda_A) + \sqrt{4\omega\omega_1+\omega_2^2}$

$$I_L^* = \frac{1}{2\omega}\left[\omega_3 - {N_L^*}^2\delta_L\mu_C(\mu_A-\Lambda_A)\left(\frac{\beta_A\beta_L N_A^* N_L^*\mu_C + \beta_C\beta_L N_C^* N_L^*(\mu_A-\Lambda_A)}{{N_L^*}^2\delta_L\mu_C(\mu_A-\Lambda_A)}\right) + \beta_A\beta_L^2 N_A^* N_L^* + \beta_C\beta_L^2 N_C^* N_L^*)\right]$$

$$I_L^* = \frac{1}{2\omega}\left[\omega_3 - N_L^{*2}\delta_L\mu_C(\mu_A - \Lambda_A)R_0^2 + \beta_A\beta_L^2 N_A^* N_L^* + \beta_C\beta_L^2 N_C^* N_L^*)\right]$$

$$I_L^* = \frac{1}{2\omega}\left[\omega_3 + (\beta_A\beta_L^2 N_A^* N_L^* + \beta_C\beta_L^2 N_C^* N_L^*)\left[-\frac{N_L^{*2}\delta_L\mu_C(\mu_A - \Lambda_A)}{\beta_A\beta_L^2 N_A^* N_L^* + \beta_C\beta_L^2 N_C^* N_L^*)}R_0^2 + 1\right]\right]$$

Let $\alpha^2 = \dfrac{N_L^{*2}\delta_L\mu_C(\mu_A - \Lambda_A)}{\beta_A\beta_L^2 N_A^* N_L^* + \beta_C\beta_L^2 N_C^* N_L^*)}$

Thus

$$I_L^* = \frac{1}{2\omega}\left[\omega_3 + (\beta_A\beta_L^2 N_A^* N_L^* + \beta_C\beta_L^2 N_C^* N_L^*)(1 - \alpha^2 R_0^2)\right]$$

$$\mathrm{I}_L^* = \frac{1}{2\omega}\left[\omega_3 + (\beta_A\beta_L^2 N_A^* N_L^* + \beta_C\beta_L^2 N_C^* N_L^*)(1 + \alpha R_0)(1 - \alpha R_0)\right] \ldots\ldots\ldots\ldots\ldots (3.16)$$

The endemic equilibrium will thus be

$$\mathrm{I}_L^* = \frac{1}{2\omega}\left[\omega_3 + (\beta_A\beta_L^2\frac{\lambda_A}{\mu_A}\frac{\lambda_L}{\mu_L} + \beta_C\beta_L^2\frac{\lambda_C}{\mu_C}\frac{\lambda_L}{\mu_L})(1 + \alpha R_0)(1 - \alpha R_0)\right] \ldots\ldots\ldots\ldots\ldots (3.17)$$

$$\mathrm{I}_A^* = \frac{\beta_L I_L^* \mu_L \lambda_A}{\mu_A(\mu_L \beta_L I_L^* + \lambda_L(\mu_A - \Lambda_A))} \ldots\ldots\ldots\ldots\ldots (3.18)$$

$$\mathrm{I}_C^* = \frac{\beta_L I_L^* \lambda_C \mu_L}{\mu_C(\mu_L \beta_L I_L^* + \lambda_L \mu_C)} \ldots\ldots\ldots\ldots\ldots (3.19)$$

Theorem 3.2

The endemic equilibrium given by equations 3.17, 3.18 and 3.19 is locally asymptotically stable on K.

Proof

We first obtain the Jacobian computed at the endemic equilibrium using the relations given by equations 3.8, 3.9 and 3.10. Thus

$$J(I_L^*, I_A^*, I_C^*) = \begin{bmatrix} -(\beta_A \frac{I_A^*}{N_A^* I_L^*} + \beta_C \frac{I_C^*}{N_C^* I_L^*}) & \delta_L \frac{I_L^*}{I_A^*} & \delta_L \frac{I_L^*}{I_C^*} \\ (\mu_A - \Lambda_A)\frac{I_A^*}{I_L^*} & -\beta_L \frac{I_L^*}{N_L^* I_A^*} & 0 \\ \mu_C \frac{I_C^*}{I_L^*} & 0 & -\beta_L \frac{I_L^*}{N_L^* I_C^*} \end{bmatrix}$$

To determine the stability of the endemic equilibrium (I_L^*, I_A^*, I_C^*), we use the Routh-Hurwitz stability criteria on the characteristic equation of a third degree polynomial given by $P(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3$. We say that $J(I_L^*, I_A^*, I_C^*)$ is Hurwitz iff $a_1, a_2, a_3 > 0$ and $a_1a_2 - a_3 > 0$.

The coefficient $a_1 = -\text{trace}(J(I_L^*, I_A^*, I_C^*))$, $a_2 = $ sum of all the principal minors of $J(I_L^*, I_A^*, I_C^*)$ and $a_3 = -\text{determinant}(J(I_L^*, I_A^*, I_C^*))$

The trace of J will be given as

trace $J = -a_1 = -(\beta_A \frac{I_A^*}{N_A^* I_L^*} + \beta_C \frac{I_C^*}{N_C^* I_L^*}) - \beta_L \frac{I_L^*}{N_L^* I_A^*} - \beta_L \frac{I_L^*}{N_L^* I_C^*} < 0$, a_2 will be

$$a_2 = \begin{vmatrix} -(\beta_A \frac{I_A^*}{N_A^* I_L^*} + \beta_C \frac{I_C^*}{N_C^* I_L^*}) & \delta_L \frac{I_L^*}{I_C^*} \\ \mu_C \frac{I_C^*}{I_L^*} & -\beta_L \frac{I_L^*}{N_L^* I_C^*} \end{vmatrix} + \begin{vmatrix} -(\beta_A \frac{I_A^*}{N_A^* I_L^*} + \beta_C \frac{I_C^*}{N_C^* I_L^*}) & \delta_L \frac{I_L^*}{I_A^*} \\ (\mu_A - \Lambda_A)\frac{I_A^*}{I_L^*} & -\beta_L \frac{I_L^*}{N_L^* I_A^*} \end{vmatrix} + \begin{vmatrix} -\beta_L \frac{I_L^*}{N_L^* I_A^*} & 0 \\ 0 & -\beta_L \frac{I_L^*}{N_L^* I_C^*} \end{vmatrix}$$

$$= \left[\beta_A \frac{I_A^*}{N_A^* I_L^*} + \beta_C \frac{I_C^*}{N_C^* I_L^*}\right] \left[\beta_L (\frac{I_L^*}{N_L^* I_C^*} + \frac{I_L^*}{N_L^* I_A^*})\right] + \beta_L^2 \frac{I_L^{*2}}{N_L^{*2} I_A^* I_C^*} - \delta_L(\mu_c + (\mu_A - \Lambda_A))$$

$$a_2 > 0 \text{ iff } \left[\beta_A \frac{I_A^*}{N_A^* I_L^*} + \beta_C \frac{I_C^*}{N_C^* I_L^*}\right] \left[\beta_L (\frac{I_L^*}{N_L^* I_C^*} + \frac{I_L^*}{N_L^* I_A^*})\right] + \beta_L^2 \frac{I_L^{*2}}{N_L^{*2} I_A^* I_C^*} > \delta_L(\mu_c + (\mu_A - \Lambda_A))$$

and finally we obtain a_3 we have the upper 2×2 block of $J(I_L^*, I_A^*, I_C^*)$ given by

$$M = \begin{bmatrix} -(\beta_A \frac{I_A^*}{N_A^* I_L^*} + \beta_C \frac{I_C^*}{N_C^* I_L^*}) & \delta_L \frac{I_L^*}{I_A^*} \\ (\mu_A - \Lambda_A)\frac{I_A^*}{I_L^*} & -\beta_L \frac{I_L^*}{N_L^* I_A^*} \end{bmatrix}$$

Hence the determinant of M is given by

$det(M) = \frac{\beta_L \beta_A}{N_L^* N_A^*} + \beta_L \beta_C \frac{I_C^*}{N_L^* N_C^* I_A^*} - \delta_L(\mu_A - \Lambda_A)$, and $det(J(I_L^*, I_A^*, I_C^*)) = -a_3 =$

$$-\beta_L \frac{I_L^*}{N_L^* I_C^*}\left[\frac{\beta_L \beta_A}{N_L^* N_A^*} + \beta_L \beta_C \frac{I_C^*}{N_L^* N_C^* I_A^*} - \delta_L(\mu_A - \Lambda_A)\right] + \mu_C \frac{I_C^*}{I_L^*}\left[\beta_L \delta_L \frac{I_L^{*2}}{N_L^* I_A^* I_C^*}\right]$$

$$-a_3 = -\beta_L \frac{I_L^*}{N_L^* I_C^*}\left[\frac{\beta_L \beta_A}{N_L^* N_A^*} + \beta_L \beta_C \frac{I_C^*}{N_L^* N_C^* I_A^*} - \delta_L([\mu_A - \Lambda_A] - \mu_C \frac{I_C^*}{I_A^*})\right] < 0$$

To prove the Routh-Hurwitz stability criteria we compute $a_1 a_2 - a_3$ as

$$a_1 a_2 - a_3 = \frac{\beta_L \beta_A^2 I_A^2}{N_L^* N_A^{*2} I_C^* I_L^*} + \frac{\beta_L \beta_A \beta_C I_C^*}{N_L^* N_A^* N_C^* I_L^*} + \frac{\beta_A \beta_L^2}{N_A^* N_L^{*2} I_C^*} + \frac{\beta_L \beta_A \beta_C I_A^*}{N_L^* N_A^* N_C^* I_L^*} + \frac{\beta_L \beta_C^2 I_C^2}{N_L^* N_C^{*2} I_A^* I_L^*} + \frac{\beta_C \beta_L^2}{N_C^* N_L^{*2} I_A^*} + \frac{\beta_C \beta_L^2 I_L^* I_C^*}{N_C^* N_L^{*2} I_A^{*2}} + \frac{\beta_L^3 I_L^{*2}}{N_L^{*3} I_A^{*2} I_C^*} + \frac{\beta_A \beta_L^2 I_L^* I_A^*}{N_A^* N_L^{*2} I_C^{*2}} + \frac{\beta_L^3 I_L^{*2}}{N_L^{*3} I_A^* I_C^{*2}} + \frac{\beta_L I_L^*}{N_L^* I_A^*}(\frac{\beta_L \beta_C}{N_L^* N_C^*} + \frac{\beta_L \beta_A}{N_L^* N_A^*} - \delta_L(\mu_A - \Lambda_A)) + \frac{\beta_L I_L^*}{N_L^* I_C^*}(\frac{\beta_L \beta_C}{N_L^* N_C^*} + \frac{\beta_L \beta_A}{N_L^* N_A^*} - \delta_L \mu_C) + [\frac{\beta_L \beta_C}{N_L^* N_C^*} + \frac{\beta_L \beta_A}{N_L^* N_A^*} - \delta_L(\mu_c + (\mu_A - \Lambda_A)][\frac{\beta_A I_A^*}{N_A^* I_L^{*2}} + \frac{\beta_C I_C^*}{N_C^* I_L^*}] > 0$$

The requirements of Routh-Hurwitz stability criteria are satisfied hence this proves that the endemic equilibrium is locally asymptotically stable.

Chapter 4

NUMERICAL ANALYSIS

In this section we shall explore the behavior of RVF when introduced into a naive environment and conduct numerical simulations of an isolated system (that is, no immigration or emigration). The model uses a daily time step and is solved by a fourth order Runge-Kutta scheme. For each simulation, we start with 100 susceptible livestock animals, 100 susceptible Culex mosquitoes, 99 susceptible Aedes mosquitoes, 1 infected Aedes mosquito and 1 infected Culex mosquito. We will run simulations to assess the expected vector and host species prevalence. We start by defining the the values of the parameters as

Table 4.1: Parameters with estimated values for numerical simulations

Parameter	Value
μ_L	0.0028
μ_A	0.05
μ_C	0.05
λ_L	0.0028
λ_A	0.05
λ_C	0.05
b_L	0.0021
b_A	0.02
b_C	0.0003
γ_L	0.14
σ	0.0312
a	10000
$\bar{N_A}$	15
φ_A	0.05

$\mathcal{R}_0 = 0.0020544 < 1$ indicating that the epidemic will be wiped out of the population. $\mathcal{R}_0 = 0$ when $b_A = b_C = 0$. When $\varphi_A = 0$, $\mathcal{R}_0 = 0.0020679$. The numerical solution of the system with initial conditions $I_L = 0$, $I_A = 1$, $I_C = 1$, $N_L = 100$, $N_A = 100$ and $N_C = 101$ over a period of two years is graphically shown below

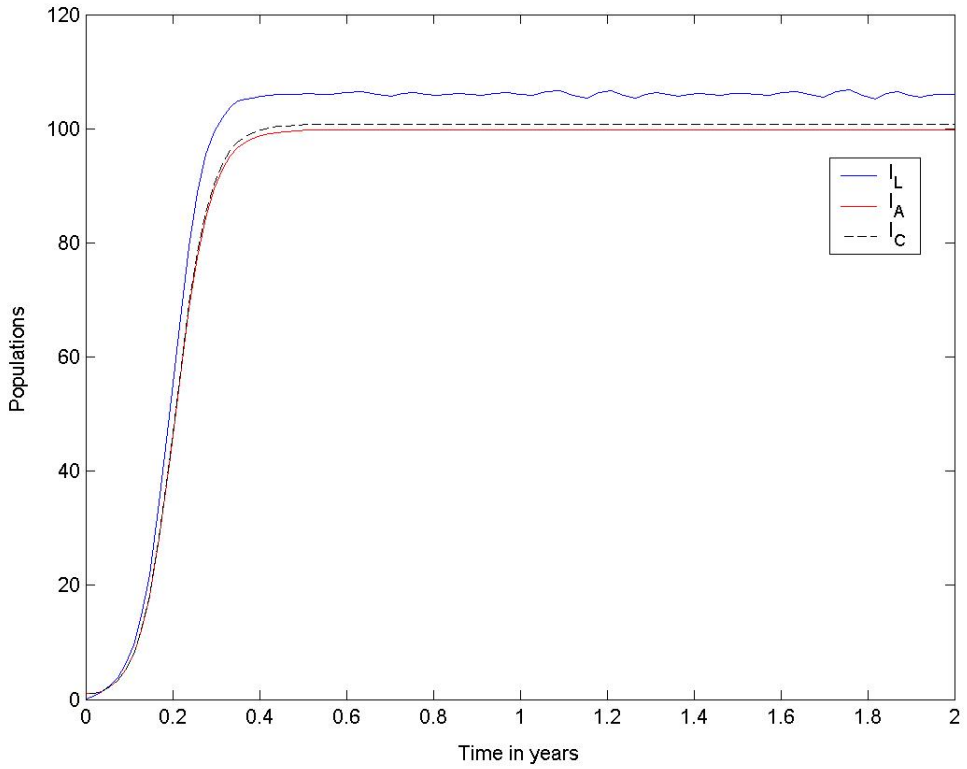

Figure 4.1: Numerical Solution of the System Showing (I_L, I_A, I_C) vs time

Chapter 5

CONCLUSION

In this study, we developed a mathematical model of Rift Valley Fever in Livestock in Kenya. Our model captures the disease induced deaths in Livestock as RVF is known to cause deaths in Livestock. Mathematical analysis was done and it was established that in the absence of the disease a disease free equilibrium will always exist if $\mathcal{R}_0 \leq 1$. We also established that the endemic equilibrium exists in the presence of the disease that is when $\mathcal{R}_0 > 1$ with the infectious population greater than zero. Reducing the vertical transmission in the Aedes Mosquito population $\mathcal{R}_0$ increases slightly. Reducing the infection in the vector population reduces $\mathcal{R}_0$ greatly. Thus the best methods of controlling RVF is to target the Aedes Mosquito and the Culex Mosquito. $\mathcal{R}_0$ is a threshold that completely determines the global dynamics of disease transmission.

This model is a simplified representation of the complex biology involved in the epidemiology of RVF. There is still a lot of details that could be incorporated into our model. Some of the details that we could include in the model to make it better are the exposed compartment and recovered compartment. Seasonal effects on mosquito population may also be incorporated. Data for disease, vector and animal migration from RVF endemic

regions need to be collected so that we can further test the validity of our model. Further the global stability of the endemic equilibrium is in general unclear hence much studies need to be done in order to understand it. We hope this model and these results will act as a base for further investigation on this disease in Kenya.

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