Mathematical Analysis of Vector-Host Diseases Models

by

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A thesis submitted to the

School of Natural Sciences,
National University of Sciences and Technology,
H-12, Islamabad, Pakistan

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Abstract

The emphasis of this dissertation lies on the theoretical study of different models of vector-borne diseases in order to get better understanding of the transmission and spread of these diseases. The patterns of infection in the host population can be understood more precisely if we comprehend those factors that influence the transmission of the disease. Five mathematical models are presented in this dissertation. Four of these explore the dynamics of the disease in relation to human population and mosquitoes. One model is dedicated to pine wilt disease in which hosts are pine trees and vectors are bark beetles. The dynamics of vector-borne diseases are explored on three scales.

First, various mathematical models are constructed by using ordinary differential equations. These models are developed by considering bilinear contact rates, nonlinear incidence rates and standard incidence rates. The models explore direct as well as vector mediated transmission. In mathematical model of pine wilt disease, it is considered that susceptible beetles (vectors of pine wilt disease) receive infection directly from infectious ones through mating.

Next, the global behavior of equilibria of models are analyzed. The analytical expressions for the basic reproduction number $\mathfrak{R}_o$ are obtained and global dynamics of the models are completely described by this number. Using Lyapunov functional theory it is proved that the disease-free equilibria are globally asymptotically stable whenever $\mathfrak{R}_o \leq 1$. The geometric approach is utilized to study the global stabilities of endemic equilibria whenever the basic reproduction number exceeds unity.

Finally, in order to assess the effectiveness of disease control measures, the sensitivity analysis of the basic reproductive number $\mathfrak{R}_o$ and the endemic proportions with respect to epidemiological and demographic parameters is provided. This sensitivity analysis provide an aid to design effective control strategies. It may be an important tool in the decision support system.

Dedicated to

My Parents and my wife Zaib

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List of publications from this thesis

[1] M. Ozair, A. A. Lashari, I. H. Jung and K. O. Okosun, *Stability analysis and optimal control of a vector-borne disease with nonlinear incidence*, Discrete Dynamics in Nature and Society, vol. 2012, 21 pages, 2012.

[2] M. Ozair, A. A. Lashari, I. H. Jung, Y. I. Seo and B. N. Kim, *Stability Analysis of a Vector-Borne Disease with Variable Human Population*, Abstract and Applied Analysis vol. 2013, 12 pages, 2013.

[3] M. Ozair, *Analysis of Pine Wilt Disease Model with Nonlinear Incidence and Horizontal Transmission*, Journal of Applied Mathematics vol. 2014, 9 pages, 2014.

[4] M. Ozair and T. Hussain, *Analysis of Vector-Host Model with Latent Stage Having Partial Immunity*, Applied Mathematical Sciences, **8**, (2014), 1569 - 1584.

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$\mathfrak{R}_o$	basic reproductive number
E_0	disease free equilibrium
E^*	endemic equilibrium
Ω	positively invariant set for the models
Γ	positively invariant set for the reduced models
b_1	per capita birth rate of humans
Λ_h	recruitment rate of humans
Λ_v	increase rate of vectors
Π_h	input rate of pines
Π_v	increase rate of bark beetles
μ_h	natural death rate of humans
δ_h	disease induced death rate of humans
μ_v	mortality rate of vectors
μ_1	The exploitation rate of pine trees
ω	Felling rate of infected pines
η_h	Transfer rate of humans from exposed to infectious class
η_v	Transfer rate of vectors from exposed to infectious class
γ_h	Immunity (temporary or permanent) rate of humans
β_1	The infection rate of susceptible individuals (vectors) which results from effective contact with infectious individuals (vectors).
β_2	The infection rate of susceptible humans as a result of biting effect of infectious vectors
β_3	The infection rate of susceptible vectors as a result of biting effect of infectious humans

Contents

Chapter 1

Introduction

Diseases can be classified into two groups, infectious and noninfectious. Infectious diseases are those diseases that can be passed among individuals for example influenza, whereas noninfectious diseases are one that may develop over individual's lifespan for example arthritis. The epidemiology of noninfectious diseases is mainly concerned with the risk factors that involve in the enhancement of the disease(for example, smoking is a major cause to increase the risk of lung cancer). In contrast transmission of infectious disease depends on the presence of infectious individuals in the population. Infectious diseases can be further divided on the basis of infecting pathogen.i.e., microparacite or macroparcite. Microparasites are small usually single-cell organisms for example viruses, protoza and bacteria where as macroparacites are bigger form of pathogen for example flukes and nematodes.

Infectious diseases including micro-and macroprasitic can also be classified on the basis of transmission (direct or indirect). If the infection is caught by the close contact with the infectious individual then it is called direct transmission. Directly transmitted diseases are influenza, measles and HIV etc. Indirectly transmitted diseases are those in which parasites are transferred to the hosts by the environment. For example diseases caused by helminths and schistosomes. The parasites of these diseases spend part of their life outside the hosts. There is a class of diseases in which transmission occurs via secondary hosts or vectors. These secondary hosts or vectors are usually insects such as mosquitoes, ticks or tsetse flies. However this transmission route can be thought of as the sum of two sequential direct transmission i.e., from primary host to insect and from the insect to another primary host. The models in this dissertation are focused on the indirectly microparasitic diseases. However direct transmission (among hosts) for human diseases and direct transmission (among vectors) for pine tree disease is also considered.

"Vector-borne disease" as a phrase refers to the illness which is carried and transmitted

through a vector. The term vector is any agent that is capable of carrying and transmitting the infectious pathogen from an infected or infectious individual to uninfected individual. The transmission mechanism usually involves three living organisms, the pathological agent, the vector and the host. The pathological agent may either be bacteria, virus, or protozoa and vectors are generally blood feeding arthropods such as mosquitoes, fleas and ticks.

Vector-borne diseases such as malaria, dengue fever, plague, and West Nile fever are infectious diseases caused by the influx of viruses, bacteria, protozoa, or rickettsia. For the spread of these diseases, the infectious agents adapt their life cycle so that part of it is harboured in the host and the other part in the vector, with the vector being the vehicle that transports the disease agent from one host to another [1].

Vector-borne diseases represent a major public health concern in most tropical and subtropical areas, and an emerging threat for more developed countries [2]. These diseases have affected many countries, mainly those who are poor, but due to global warming, there is a real risk of these diseases to appear in regions where they have already been eradicated or even in those where the normal environmental conditions would never have allowed its existence [3]. These diseases have posed problems to national economies especially in countries in the tropical and subtropical regions of the worlds. For example, the vector-borne disease, malaria, caused by the plasmodium parasite and transmitted from one human to another by the female anopheles vector mosquito, continues to plague the world especially the developing nations. The WHO World malaria report [4], reports the parasite, and hence malaria, caused an average of nearly 900,000 deaths in 2006, of which 85% were of children under the age of five. Also, dengue fever, yellow fever, trypanosomiases, and leishmania are all highly prevalent tropical and subtropical diseases. Some vector-borne diseases, such as malaria, dengue, and yellow fever, that used to be common in some developed nations of the world have been successfully put under control. However, these diseases are still a threat to developing nations and hence a potential threat to many regions of the world. The reason is the recent trends in climate change, global warming, increased movement between different nations, disease-transmitting vectors may be able to (re)-colonize and survive in zones not formerly possible.

1.1 Ecological Factors to Enhance Vector-Borne Diseases

Many ecological factors are responsible to increase vector densities or vector-host interactions. Some of these are as follows:

1.1.1 Rapid Urbanization

There have been profound increases in the magnitude of vector-borne disease problems as the result of urbanization. Experts recognize urbanization as one of the most important drivers of global change, and predict that rapid increases in urban populations throughout the world will have major implications for human health in general and vector-borne diseases specifically [5]. Densely packed housing in shanty towns or slums and inadequate drinking-water supplies, garbage collection services, and surface-water drainage systems combine to create favorable habitats for the proliferation of vectors and reservoirs of communicable diseases [6]. As a consequence, vector-borne diseases such as malaria, lymphatic filariasis and dengue are becoming major public health problems associated with rapid urbanization in many tropical countries. The problems in controlling these diseases and eliminating vectors and pests can be resolved by decision-makers and urban planners by moving away from the concept of "blanket" and applications of pesticides towards integrated approaches. Sound environmental management practices and community education and participation form the mainstay can provide some of the most outstanding successes in the area.

1.1.2 Deforestation

Deforestation may bring about whole-scale ecosystem reconstitution. This in turn may influence vector-borne disease transmission through altered vegetation, introduction of livestock, development of human settlements [7]. Forest-related activities, such as mining and logging, have been associated with increased exposure to the vectors of yellow fever, malaria, and leishmaniasis. Deforestation may create ecological niches favoring proliferation of vectors and parasites. For example, water puddles in deforested areas tend to have lower salinity and acidity than puddles in forests. Such deforested water puddles may be more conducive for the larval development of certain Anopheles mosquito species (the vectors of malaria).

1.1.3 Animal Husbandry

Animal husbandry may also increase the transmission of some vector-borne diseases. Farm animals are potential reservoir hosts, thus making pathogens more widespread. Livestock may contribute to the emergence of vector-borne diseases by facilitating the exchange of a pathogen from nonhuman reservoirs to humans while grazing. Transmission of Japanese encephalitis is increasing in parts of Southeast Asia and the western Pacific, largely because of increased irrigated agriculture (especially rice paddies) and pig husbandry (an important

natural host of the virus).

1.1.4 Movement

Within a given population internal movements and migration can have a major influence both on population density and likely contact between infected and susceptible individuals [8]. The movement of domestic animals has led to the extension of a variety of species such as the ticks *Boophilus microplus and Rhipicephalus sanguineus*, both of which are parasites that can cause direct damage to their hosts as well as acting as vectors of a variety of major viral, rickettsial and protozoan pathogens. In addition to the movement of humans, the movement of goods by humans can also lead to the dissemination of parasites and vectors. For example introduction of *Aedes albopictus* from Southeast Asia to North America in water pools in the rims of used tyres.

1.1.5 Direct Transmission

Vector-borne diseases can be transmitted directly through vertical transmission from mother to fetus, transfusion-related transmission, transplantation related transmission, and needle-stick-related transmission [9]. For example dengue virus can be classified as a blood pathogen, there is a stage of viremia in dengue. If blood is donated in this condition, infection of the recipients of the contaminated blood can be expected.

1.2 Epidemiology

Epidemiology studies the frequency and distribution in space and time of diseases in a defined population, as well as the role of determining factors, and their eventual control [10]. Epidemiology can also be defined as the ecology of diseases. There are in fact four types of epidemiology. Descriptive epidemiology consists in collecting and describing data that may be relevant a priori, and basically consists in establishing rates by rationing the number of individuals presenting one particular pathological condition to the population size. Analytical epidemiology investigates the relationships between causes and effects, and evaluates risk factors. Experimental epidemiology tests hypothesis by developing experimental models to handle one or several factors: for example a prophylactic try. Finally, ecological epidemiology, also called mathematical epidemiology identifies the factors and processes affecting the transmission and persistence of pathogens and uses mainly mathematical models. This work reports on epidemiological studies belonging to the second and fourth categories. In this thesis it is attempted to find the important

factors by which vector-borne diseases are highly influenced.

1.3 Mathematical Modeling

In almost all branches of science, research questions are answered from planned repeated experiments. But for infectious diseases, conducting experiments in communities is not ethical or possible [11]. The epidemiological data may not help predict the future trends of the disease. Realistic mathematical models of the transmission of infectious diseases add a new dimension of information to assist in public health policy for control of the disease. These models provide a dynamic picture of disease transmission and are useful to predict the future trends of the disease. Dynamical methods can show the transmission rules of infectious diseases from the mechanism of transmission of the disease, so that people may know some global dynamic behavior of the transmission process [12]. The popular epidemic dynamic models are still so called compartmental models which were constructed by Kermack and Mckendrick in 1927 [13] and is developed by many other biomathematicians. In the K-M model, the population is divided into three compartments: susceptible compartment S, in which all individuals are susceptible to the disease; infected compartment I, in which all individuals are infected by the disease and have infectivity; removed compartment R, in which all the individuals recovered from the class I and have permanent immunity. They did following assumptions.

- The disease spreads in a closed environment, no emigration and immigration, and is no birth and death in population, so the total population remains a constant k, i.e. $S(t) + I(t) + R(t) = k$.
- The infective rate of an infected individual is proportional to the number of susceptible, the coefficient of the proportion is a constant β, so that the total number of new infected at time t is $\beta S(t)I(t)$.
- The recovered rate is proportional to the number of infected, and the coefficient of proportion is a constant γ. So that the recovered rate at time t is $\gamma I(t)$.

Under the above three assumptions, the following model was constructed.

$$\begin{aligned} \frac{dS}{dt} &= -\beta S(t)I(t), \\ \frac{dI}{dt} &= \beta S(t)I(t) - \gamma I(t), \\ \frac{dR}{dt} &= \gamma I(t). \end{aligned}$$

where

$$S(t) + I(t) + R(t) = k.$$

Now we shall explain some basic concepts of epidemiological dynamics.

Contact rate It is defined as the number of times an infective individual contacts the other members in unit time. It often depends on the number N of individuals in the total population, and is denoted by function $U(N)$.

Adequate contact rate If the individuals contacted by an infected individual are susceptible, then they may be infected. Assume that the probability of infection by every time contact is β_0, then function $\beta_0 U(N)$ is called the adequate contact rate. It shows the ability of an infected individual infecting others. It depends on the environment, the toxicity of the virus or bacterium, etc.

Infection rate The susceptible individuals may be infected when they contact with the infectives, and the fraction of the susceptibles in total population is $\frac{S}{N}$, so the mean adequate contact rate of an infective to the susceptible individuals is $\beta_0 U(N)\frac{S}{N}$. It is called the infection rate.

Incidence function The number of new infected individuals yielding in unit time at time t is $\beta_0 U(N) S\frac{I}{N}$. It is called incidence function.

Bilinear incidence or simple mass-action incidence If the contact rate is proportional to the size of total population.i. e. $U(N) = kN$, the incidence is $\beta_0 kSI = \beta SI$. It is called bilinear incidence or simple mass-action incidence. $\beta = \beta_0 k$ is called transmission coefficient.

Standard incidence If the contact rate is a constant i. e. $U(N) = k_1$, then the incidence is $\frac{\beta_0 k_1 SI}{N} = \beta\frac{SI}{N}$. It is called standard incidence.

1.3.1 Basic Reproduction Number

Basic reproduction number is usually denoted by $\mathfrak{R}_0$. It is defined as the average number of secondary infectious infected by an individual of infectives during whose whole course of disease in the case that all the members of the population are susceptible. According to this definition, we can easily understand that if $\mathfrak{R}_0 < 1$, then the infectives will decrease so that the disease will go to extinction. If $\mathfrak{R}_0 > 1$, then the infectives will increase so that the disease can not be eliminated and usually develops into an endemic.

From the mathematical point of view, usually when $\mathfrak{R}_0 < 1$, the model has only disease

free equilibrium and it is globally asymptotically stable. When $\mathfrak{R}_o > 1$, the disease free equilibrium becomes unstable and usually positive endemic equilibrium appears which becomes stable. Hence, if all the members of a population are susceptible in the beginning, then $\mathfrak{R}_o = 1$ is usually a threshold whether the disease go to extinction or go to an endemic.

1.4 Some Vector-Borne Diseases Models

Since the pioneering work of Ross in the late 19th and early 20th centuries, the classical approach for controlling vector-borne diseases involves the eradication or strict population control of the vectors [14]. Sir Ronald Ross, while working at the Indian Medical Service in 1890s, demonstrated the life-cycle of the malaria parasite in mosquito [15]. He published a series of papers using mathematical functions to study transmission of Malaria in early 1900. He developed a simple model, now known as the classical "Ross model" in which the relationship between the number of mosquitoes and incidence of malaria in humans was explained [16]. Ronald Ross used the word "pathometry" in his first mathematical model. This means"quantitative study of a disease either in the individual or in the community". He showed that the reduction of mosquito numbers "below a certain figure" (Transmission threshold) was sufficient to counter malaria.

This simple model was no longer satisfactory when new data became available, and more complexities of interactions were considered. Therefore, several models have been developed by which Ross's model was extended by considering different factors, such as latent period of infection in mosquitoes and human, age-related differential susceptibility to malaria in human population, acquired immunity etc. George Macdonald [17] in the 1950s, reasserted the usefulness of mathematical epidemiology based on 20 years of fieldwork. He modified Ross's model by integrating biological information of latency in the mosquito due to malaria parasite development, and implicated the survivorship of adult female mosquito as the weakest element in the malaria cycle. Latency of infection in humans was introduced by Anderson and May [18] in Macdonald's model making the additional "Exposed" class in humans. Aron and May [19] proposed an age-specific immunity model with a new compartment Immune R_h in humans. This model, thus, consists of three compartments in humans: Susceptible S_h, Infected I_h and Immune R_h, and is a $SIRS$ model. The model, proposed by Koella and Antia [20], further divides the host population infected by drug-sensitive strain into two compartments - treated and untreated. So this model consists of five compartments of human: susceptible S_h, sensitive, infected, and treated I_{h1}, sensitive, infected, and untreated I_{h2}, infected with the resistant strain I_{h3} and the recovered R_h.

The effects of migration and visitation on transmission of malaria were shown by Torres-Sorando and Rodriguez [21] by modifying the basic Ross model. In a recent study, Parham and Michael [22] proposed a model, to study the dynamics of the mosquito population by considering simultaneous effects of rainfall and temperature. The model consists of three compartments in humans (S_h, I_h, R_h) with fixed duration of latency, and three compartments in mosquitoes (S_m, E_m, I_m). Different environmental factors are introduced in this model through parameters related to mosquitoes.

Immunity can be included in a model in two ways - by considering a separate Immune class (R_h) in humans, and by incorporating an Immunity function in existing models. Some models (Dietz et al [23], Aron [24], Ngwa and Shu [25], Chitnis et al [26]) have introduced a separate immune class in their models, whereas, Fillipe et al [27] have used complex immunity functions in their model. Ngwa and Shu proposed an immunity model in which disease related death rate is considered to be significantly high, and the total population is not constant. The Ngwa-Shu model consists of four compartments in humans - Susceptible (S_h), Exposed (E_h), Infected (I_h) and Immune (R_h) and three compartments in mosquitoes - Susceptible (S_m), Exposed (E_m), and Infected (I_m). Mathematical analysis of the model shows that the Basic Reproductive Number, $\mathfrak{R}_o$, can describe the malaria transmission dynamics of the disease, where a globally stable disease-free state exists if $\mathfrak{R}_o < 1$, while for $\mathfrak{R}_o > 1$, the endemic equilibrium becomes globally stable. This model explicitly shows the role of inclusion of demographic effects (net population growth) in predicting the number of fatalities that may arise as a result of the disease. In a similar theme, Chitins et al. included constant immigration of susceptible human population. Considering immigration of people and excluding direct human recovery from the infectious to susceptible class, they showed that the population approaches the locally asymptotically stable endemic equilibrium point, or stable disease-free equilibrium point, depending on the initial size of the susceptible class.

1.5 Some Important Definitions

In this section some important definitions are given that are used in mathematical models.

Equilibrium Point Suppose that $\mathbf{x} = [x_1(t), x_2(t), ..., x_n(t)]$ and $f(\mathbf{x}) = [f_1(\mathbf{x}), f_2(\mathbf{x}), ..., f_n(\mathbf{x})]$ are $n-$vectors, that $f(\mathbf{x})$ is continuously differentiable and that $f(0) = 0$. Then zero is an equilibrium point of the system

$$\mathbf{x}' = f(\mathbf{x}), \tag{1.5.1}$$

where "$\prime$" denotes the derivative with respect to "t".

If $f(\mathbf{p}) = 0$ for $\mathbf{p} \neq 0$, translate $\mathbf{p}$ to the origin , so there is no harm in assuming that the equilibrium point whose stability we will test is at the origin.

Positive Definite Function A real valued function $V(x)$ is positive definite on an open ball B centered at the origin if it has only positive values on B except at the origin, where $V(0) = 0$.

Lyapunov Function Let $V : G^* \to R$, where G^* is an open set in R^n. Let G be any subset of G^*. V is said to be a Lyapunov function of system (1.5.1) on G if
(i) V is continuous.
(ii) $V'(x) \leq 0$ for all $x \in G$.

Strong Lyapunov Function A continuously differentiable function $V(x)$ is a strong Lyapunov function for system (1.5.1) on B if V is positive definite and the derivative V' following the motion is negative definite on B.

Lyapunov's First Theorem If there is a strong Lyapunov Function $V(x)$ for system (1.5.1) on an open ball centered at the origin, then system (1.5.1) is asymptotically stable at the origin.

Invariant Set The set $B \subset R^n$ is said to be invariant with respect to the system (1.5.1) if for any initial value $x_0 \in B$ implies that the solution $x(x_0, t) \in B$ for all time t in the domain of the solution $x(t)$. It is said to be positively invariant if $x_0 \in B$ implies $x(x_0, t) \in B$ for $t > 0$. It means that every solution starting in B remains in B for all t.

LaSalles Invariance Principle Let $\Delta(t) \subset B$ be a compact set that is positively invariant with respect to the system (1.5.1). Let $V : D \to R$ be a continuously differentiable function such that $V'(Y) \leq 0$ on Δ. Let E be the set of all points in Δ such that $V'(Y) = 0$. Let M be the largest invariant set in E. Then every solution starting in Δ approaches M as $t \to \infty$.

Lozinskiĭ Measures The Lozinskiĭ measure for any matrix A is written as

$$\mu(A) = \lim_{h \to 0^+} \frac{\|I + hA\| - 1}{h}.$$

The values of $\|A\|$ and $\mu(A)$ corresponding to the most commonly used norms:

$$\|A\|_\infty = \sup_{1 \leq i \leq n} \sum_{j=1}^{n} |a_{ij}| \qquad (Row\ sum\ Norm),$$

$$\mu(A) = \sup_{1\leq i\leq n} [Re(a_{ii}) + \sum_{j=1,\ j\neq i}^{n} |a_{ij}|],$$

$$\|A\|_1 = \sup_{1\leq j\leq n} \sum_{i=1}^{n} |a_{ij}| \qquad (Column\ sum\ Norm),$$

$$\mu(A) = \sup_{1\leq j\leq n} [Re(a_{jj}) + \sum_{i=1,\ j\neq i}^{n} |a_{ij}|],$$

Second Additive Compound Matrices For an $n \times n$ matrix $A = [a_{ij}]$, the second additive compound $A^{[2]}$ is the $\binom{n}{2} \times \binom{n}{2}$ matrix defined as follows [28]:

For any integer $i = 1, 2, ..., \binom{n}{2}$, let $i = (i_1, i_2)$ be the ith member in the laxicographic ordering of integer pairs (i_1, i_2) such that $1 \leq i_1 < i_2 \leq n$. Then the element in the $i - row$ and the $j - column$ of $A^{[2]}$ is

$a_{i_1 i_1} + a_{i_2 i_2}$,	if $(j) = (i)$
$(-1)^{r+s} a_{i_r j_s}$,	if exactly one entry i_r of (i) does not occur in (j) and j_s does not occur in i
0,	if neither entry from (i) occurs in (j).

For $n = 3$,

$$A = \begin{pmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{pmatrix}$$

$$A^{[2]} = \begin{pmatrix} a_{11} + a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11} + a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22} + a_{33} \end{pmatrix}$$

For $n = 4$,

$$A = \begin{pmatrix} a_{11} & a_{12} & a_{13} & a_{14} \\ a_{21} & a_{22} & a_{23} & a_{24} \\ a_{31} & a_{32} & a_{33} & a_{34} \\ a_{41} & a_{42} & a_{43} & a_{44} \end{pmatrix}$$

$$A^{[2]} = \begin{pmatrix} a_{11}+a_{22} & a_{23} & a_{24} & -a_{13} & -a_{14} & 0 \\ a_{32} & a_{11}+a_{33} & a_{34} & a_{12} & 0 & -a_{14} \\ a_{42} & a_{43} & a_{11}+a_{44} & 0 & a_{12} & a_{13} \\ -a_{31} & a_{21} & 0 & a_{22}+a_{33} & a_{34} & -a_{24} \\ -a_{41} & 0 & a_{21} & a_{43} & a_{22}+a_{44} & a_{23} \\ 0 & -a_{41} & a_{31} & -a_{42} & a_{32} & a_{33}+a_{44} \end{pmatrix}$$

1.6 Thesis Organization

The rest of the dissertation contains five vector-borne disease models. The significant part of this thesis has a theoretical flavor. However, numerical simulation is carried out to verify the analytical results and draw some conclusions. The sharp conditions are found and it is shown by using Lyapunov function theory and geometric approach that the disease either goes to extinction or approach the endemic level. By using the data available in the literature for malaria disease the relative importance of the parameters is found and it is shown that which parameter are the most sensitive for the eradication of the disease or to reduce the endemic level of the disease. One chapter is dedicated to the analysis of vector-borne disease in plants. The control measures has been found.

Most of the literature on disease modeling deals with constant or asymptotically constant total population. This assumption holds true for diseases having short duration (influenza, SARS, etc.) and also for diseases with negligible mortality rate (West Nile virus in human or livestock). However, for endemic diseases such as malaria or diseases with high mortality rate (HIV/AIDS in poor countries), the changes in population size is not negligible. The total population changes with disease-induced deaths, as well as with natural births and deaths. These factors imbalance the inflow and outflow of a given population and thus cause the total population to vary with time.

The incidence of a disease plays an important role in the study of mathematical epidemiology. The simple mass action law ξSI, with ξ as a mass action coefficient, is sometimes used for the horizontal incidence. The parameter ξ has no direct epidemiological interpretation, but comparing it with the standard formulation shows that $\beta = \xi N$, so that this form implicitly assumes that the contact rate β increases linearly with the population size. Using an incidence of the form $\xi\frac{N^v SI}{N}$, data for five human diseases in communities with population sizes from 1,000 to 400,000 ([29], p. 157) imply that v is between 0.03 and 0.07. This strongly suggests that the standard incidence corresponding to $v = 0$ is more realistic for human diseases than the simple mass action incidence corresponding to $v = 1$. The behavior of vector-borne disease model with variable human population and

direct transmission by considering standard incidence is discussed in chapter 2.

Dietz et al. [23] showed that the duration of immunity to malaria depends on repeated exposure. Niger and Gumel [30] discussed the role of the partial immunity on the transmission dynamics of malaria by including multiple infected and recovered classes. Under these assumptions, the model discussed in chapter 3 is extended by including exposed classes in human as well as in vector populations. The partial immunity instead of permanent immunity has been assumed. The stability analysis is analyzed.

In 1978, Capasso and Serio [31] introduced a saturated incidence rate $g(I)S$ in an epidemic models. This is important because the number of effective contact between infective and susceptible individual may saturate at high infective levels due to overcrowding of infective individuals or due to protective measures endorsed by susceptible individuals. A variety of nonlinear incidence rates have been used in epidemic models [32–37]. In [37], an epidemic model with nonlinear incidences is proposed to describe the dynamics of diseases spread by vectors(mosquitoes), such as malaria, yellow fever, dengue and so on. Chapter 4 is based on a model for the transmission dynamics of a vector-borne disease with nonlinear incidence rate. It is proved that the global dynamics of the disease is completely determined by the basic reproduction number. In order to assess the effectiveness of disease control measures, the sensitivity analysis of the basic reproductive number $\mathfrak{R}_o$ and the endemic proportions with respect to epidemiological and demographic parameters is provided.

Chapter 5 is devoted to the stability and sensitivity analysis of pine wilt disease. Pine wilt, a fatal disease of commonly planted pines brought on by the pinewood nematode (Bursaphelenchus xylophilus), causes changes to ecosystem and destructs the variety of ecosystem. Pine trees affected by pine wilt disease usually die within few months. Symptoms of pine wilt disease normally appear in late spring or summer. The most prominent symptom is the lack of resin exudation from barks wounds. The foliage becomes light grayish green, then yellow, and finally it becomes reddish brown. The tree succumbs to the disease at this stage. The affected trees totally lacks resin and their wood becomes dry.

The long-horned pine sawyer beetles (Monochamus alternatus) are the main culprits for the spread of pinewood nematodes from infected pines to healthy or stressed pines. When new adult beetles emerge in spring, they locate a living host tree to feed on the bark of the young branches and transfer nematodes to the healthy trees through the feeding wounds produced by these sawyers. This transmission is referred to primary transmission. The transmission of the nematodes during egg-laying activities in freshly cut timber or dying trees is referred to secondary transmission. Nematodes, introduced during primary

transmission, migrate to the resin canals of their hosts and kill these cells rendering them ineffective due to which a susceptible host can wilt and die within weeks of being infested upon the availability of favorable conditions to disease development. The principle of the Bursaphelenchus xylophilus transmission and disease dissemination is reviewed by Evans et al. [38]. Pine wilt particularly kills Scots pine within few weeks to few months. Some other pine species as Austrian(Pinus nigra), jack(P. banksiana), mogo(P. mugo), red (P. resinosa) pines are occasionally killed by pine wilt.

The incidence of pine wilt disease depends on beetles density because pine sawer beetles are the source of transmission of pinewood nematode. This incidence may approach its saturation level at very high beetle densities. The adult female pine sawyer attempts to avert from erstwhile oviposition scants. It approaches another tree before the saturation point of ovipostion is reached. Thus the isolation of infected individuals result the decrease in the number of contacts between the susceptible and infected individuals at high infective levels. These observations inspire to consider nonlinearities in the incidence rates. It is not meaningful to consider the saturation level when transmission occurred during mating. Thus bilinear incidence has been considered.

The dynamics of many communicable diseases have been extensively analyzed under the assumption that the duration of immunity is independent of exposure to infection [18]. However, the immunity to malaria appears to be sustained by continuing exposure [39]. Hence, the conventional definition of immunity as absolute refractoriness to infection may be too restrictive, as immunity may confer protection against severe illness without eliminating chronic, mild infections [24]. Incomplete immunity to malaria not only complicates the disease control strategies but also partially immune individuals having mild infections become the source of continuous transmission of the parasite in the community. Following the ideas advanced in [25] and [40], the model is investigated in chapter 6 by assuming that the persons who are partially immune to the disease may be infectious. Concluding remarks of the whole thesis are given in chapter 7.

Chapter 2

Stability Analysis of Vector-Host Model with Variable Human Population

This chapter is aimed at the analysis of mathematical model of vector-borne diseases with variable human population. The varying population size includes a term for disease-related deaths. The assumption of direct transmission has been included because direct contacts of infective and susceptible individuals such as blood transfusion, organ transplantation and needle sticks injury can cause the main source of spreading many vector borne diseases. The conscientious analysis of the model exhibits that the control parameter for the stability of the system is the threshold number $\mathfrak{R}_o$. The global asymptotic stability of the disease free equilibrium, when the threshold number $\mathfrak{R}_o$ is less than unity, is proved by using Lyapunov function theory. In this case, the endemic equilibrium does not exist. If threshold number $\mathfrak{R}_o$ exceeds unity, then the disease persists. The unique endemic equilibrium is then globally asymptotically stable and this stability is proved by the geometric approach.

2.1 Model Formulation

We formulate a continuous mathematical model for the transmission of vector-borne disease according to the basic rules of mathematical modeling in epidemiology. We develop the model under the following hypotheses.

1. The total population $N_h(t)$ is split into three compartments:

    - Susceptibles $S_h(t)$: Individuals of the human population who may receive

infection.

- Infected $I_h(t)$: Individuals of the human population infected by the disease.
- Recovered $R_h(t)$: Individuals of the human population recovered from infection.

2. The total population of vectors $N_v(t)$ is divided into two subpopulations:
   - Susceptibles $S_v(t)$: members of vector population who may get infection as a result of biting the infectious humans.
   - Infected $I_v(t)$: members of vector population infected by the parasite.
3. The susceptible humans can be infected through the direct contact with the infectious vectors and transfer to the infected subpopulation.
4. The susceptible humans can also be infected through the direct contact with infectious individuals (for example, transfusion, transplantation, or needle-stick related transmission etc).
5. The recovered humans are assumed to acquire permanent immunity.
6. The humans leave the population either by disease induced mortality or through natural death.
7. The susceptible vectors can be infected through the effective contact with the infectious humans and move to the infected subpopulation.
8. The vectors that get infected once remain infectious throughout their life.
9. The vectors leave the population through natural death.
10. The vector population has constant size with equal birth and death rate.

The total human population is given by

$$N_h(t) = S_h(t) + I_h(t) + R_h(t),$$

and the total vector population is given by

$$N_v(t) = S_v(t) + I_v(t).$$

The dynamics of the disease model for human and vector populations under the above mentioned assumptions is depicted graphically in the following flow diagram.

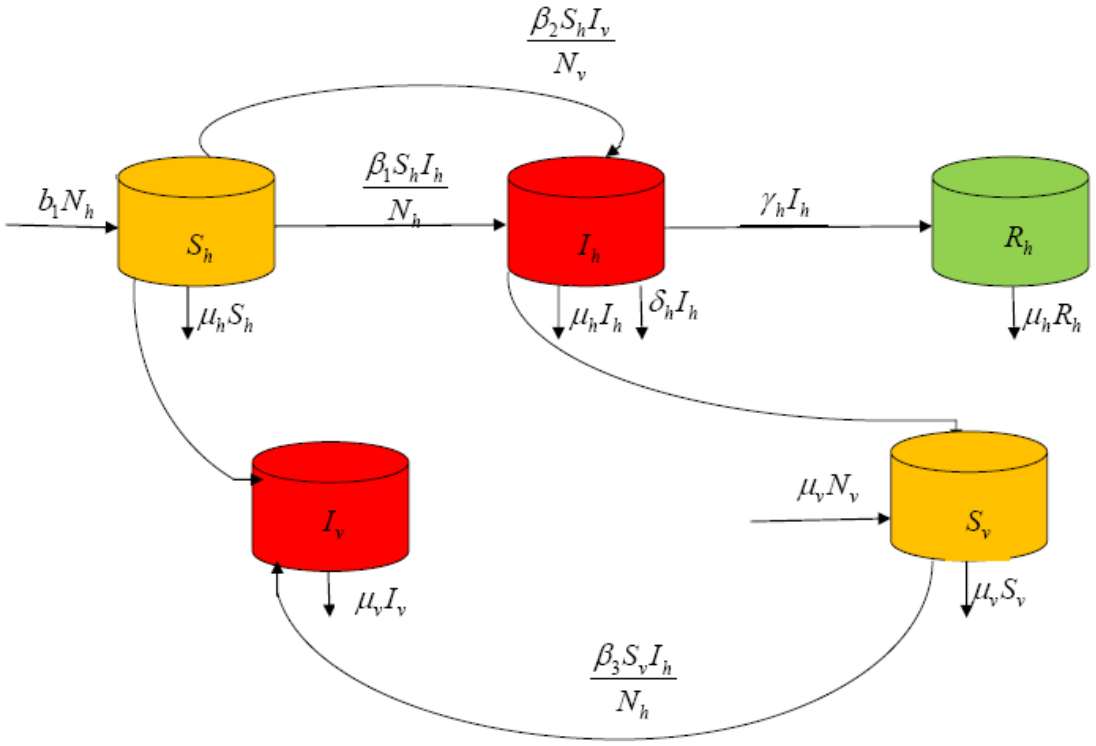

Figure 2.1: Flow diagram of Vector-Host Model with variable human population

From the transfer diagram and assumptions given above the first order nonlinear system of ordinary differential equations, is analytically given as follows

$$\begin{aligned}
\frac{dS_h}{dt} &= b_1 N_h - \frac{\beta_1 S_h I_h}{N_h} - \frac{\beta_2 S_h I_v}{N_v} - \mu_h S_h, \\
\frac{dI_h}{dt} &= \frac{\beta_1 S_h I_h}{N_h} + \frac{\beta_2 S_h I_v}{N_v} - \mu_h I_h - \gamma_h I_h - \delta_h I_h, \\
\frac{dR_h}{dt} &= \gamma_h I_h - \mu_h R_h \qquad (2.1.1) \\
\frac{dS_v}{dt} &= \mu_v N_v - \frac{\beta_3 S_v I_h}{N_h} - \mu_v S_v, \\
\frac{dI_v}{dt} &= \frac{\beta_3 S_v I_h}{N_h} - \mu_v I_v.
\end{aligned}$$

The parameter used in the model are given in Table(2.1) and all these parameters are assumed to be strictly positive.

Table 2.1: Parameter descriptions for the model (2.1.1)

parameter	description
b_1	Birth rate of humans.
β_1	The infection rate of susceptible individuals which results from effective contact with infectious individuals.
β_2	The infection rate of susceptible humans resulting due to the biting of infected vectors.
β_3	The infection rate of susceptible vectors as a result of biting effect of infectious humans.
γ_h	The acquired immunity rate. $\frac{1}{\gamma_h}$ is the average infectious period.
δ_h	Disease related death rate of humans.
μ_h	Natural death rate of humans.
μ_v	The death rate of vectors.

The incidence of new infections via direct (effective contact of susceptible humans with infectious humans) and indirect (biting effect of infected vectors to susceptible humans) routes of transmission is given by the standard incidence form $\frac{\beta_1 S_h I_h}{N_h}$ and $\frac{\beta_2 S_h I_v}{N_v}$, respectively. The incidence of newly infected vectors (biting effect of susceptible vectors to infectious humans) is again given by standard incidence form $\frac{\beta_3 S_v I_h}{N_h}$. The total human population is governed by the following equation:

$$\frac{dN_h}{dt} = b_1 N_h - \mu_h N_h - \delta_h I_h. \tag{2.1.2}$$

2.2 Analysis of Mathematical Model

To examine the model (2.1.1) more conveniently, we shall work with the normalized model by scaling each class of both populations. Suppose

$$s_h = \frac{S_h}{N_h},\ i_h = \frac{I_h}{N_h},\ r_h = \frac{R_h}{N_h},\ s_v = \frac{S_v}{N_v}\ and\ i_v = \frac{I_v}{N_v}. \tag{2.2.1}$$

Differentiating the scaling equations (2.2.1), we get

$$\begin{aligned}
\frac{ds_h}{dt} &= \frac{1}{N_h}\frac{dS_h}{dt} - \frac{S_h}{{N_h}^2}\frac{dN_h}{dt}, \\
\frac{di_h}{dt} &= \frac{1}{N_h}\frac{dI_h}{dt} - \frac{I_h}{{N_h}^2}\frac{dN_h}{dt}, \\
\frac{dr_h}{dt} &= \frac{1}{N_h}\frac{dR_h}{dt} - \frac{R_h}{{N_h}^2}\frac{dN_h}{dt}, \\
\frac{ds_v}{dt} &= \frac{1}{N_v}\frac{dS_v}{dt} - \frac{S_v}{{N_v}^2}\frac{dN_v}{dt}, \\
\frac{di_v}{dt} &= \frac{1}{N_v}\frac{dI_v}{dt} - \frac{I_v}{{N_v}^2}\frac{dN_v}{dt}.
\end{aligned} \tag{2.2.2}$$

We obtain a new 5-dimensional system which is given by

$$\begin{aligned}
\tfrac{ds_h}{dt} &= b_1(1-s_h) - \beta_1 s_h i_h - \beta_2 s_h i_v + \delta_h s_h i_h, \\
\tfrac{di_h}{dt} &= \beta_1 s_h i_h + \beta_2 s_h i_v - (b_1 + \gamma_h + \delta_h) i_h + \delta_h i_h^2, \\
\tfrac{dr_h}{dt} &= \gamma_h i_h - b_1 r_h + \delta_h i_h r_h, \\
\tfrac{ds_v}{dt} &= \mu_v(1-s_v) - \beta_3 s_v i_h, \\
\tfrac{di_v}{dt} &= \beta_3 s_v i_h - \mu_v i_v
\end{aligned} \tag{2.2.3}$$

Suppose there exists a set Ω in which the system (2.2.3) is mathematically and epidemiologically well-posed. This domain is given by $\Omega = D_h \times D_v$, where

$$D_h = \{(s_h, i_h, r_h) \in R^3 : s_h \geq 0, i_h \geq 0, r_h \geq 0, s_h + i_h + r_h = 1\},$$

and

$$D_v = \{(s_v, i_v) \in R^2 : s_v \geq 0, i_v \geq 0, s_v + i_v = 1\}.$$

The set, Ω, is authentic because normalized populations, s_h, i_h, r_h, s_v and i_v are all nonnegative having sums over the species type that are equal to 1. For simplicity we shall denote $\frac{df}{dt}$ by f'. Since the model (2.2.3) characterize the human and vector population, therefore, it will be necessary to prove that all state variables of the populations are positive and the domain, Ω, is positively invariant.

Theorem 2.2.1. *Suppose that the initial conditions for the system (2.2.3) are nonnegative. Then the solutions* $(s_h, i_h, r_h, s_v, i_v)$ *of the system (2.2.3) are positive* $\forall t > 0$. *Furthermore, the feasible set* Ω *is positively invariant.*

Proof. The right hand side of (2.2.3) is continuous having continuous partial derivatives in Ω. We observe from (2.2.3) that if $s_h = 0$, ${s_h}' > 0$; if $i_h = 0$, ${i_h}' \geq 0$; if $r_h = 0$, ${r_h}' \geq 0$; if $s_v = 0$, ${s_v}' > 0$ and if $i_v = 0$, ${i_v}' \geq 0$. We also see that if $s_h + i_h + r_h = 1$ then ${s_h}' + {i_h}' + {r_h}' = 0$; and if $s_v + i_v = 1$ then ${s_v}' + {i_v}' = 0$. Hence a unique solution exists $\forall t > 0$ and none of the orbits can leave Ω.

As we have supposed the permanent immunity so r_h does not involve in s_h and i_h classes. Hence, we can study the following reduced system

$$\begin{aligned} \frac{ds_h}{dt} &= b_1(1 - s_h) - \beta_1 s_h i_h - \beta_2 s_h i_v + \delta_h s_h i_h, \\ \frac{di_h}{dt} &= \beta_1 s_h i_h + \beta_2 s_h i_v - (b_1 + \gamma_h + \delta_h) i_h + \delta_h i_h^2, \\ \frac{di_v}{dt} &= \beta_3(1 - i_v) i_h - \mu_v i_v, \end{aligned} \tag{2.2.4}$$

determining r_h from

$$\frac{dr_h}{dt} = \gamma_h i_h - b_1 r_h + \delta_h i_h r_h, \tag{2.2.5}$$

or from $r_h = 1 - s_h - i_h$ and s_v from $s_v = 1 - i_v$, respectively. Throughout this work, we study the reduced system (2.2.4) in the closed, positively invariant set $\Gamma = \{(s_h, i_h, i_v) \in {R^3}_+, 0 \leq s_h + i_h \leq 1, 0 \leq i_v \leq 1\}$, where R^3_+ denotes the non-negative cone of R^3 with its lower dimensional faces.

The dynamics of the disease is described by the basic reproduction number $\mathfrak{R}_\circ$. The threshold quantity $\mathfrak{R}_\circ$ is called the reproduction number, which is defined as "the average number of secondary infections produced by an infected individual in a completely susceptible population". The basic reproduction number of model (2.2.4) is given by the expression

$$\mathfrak{R}_\circ = \frac{\beta_1}{b_1 + \gamma_h + \delta_h} + \frac{\beta_2 \beta_3}{\mu_v (b_1 + \gamma_h + \delta_h)}. \tag{2.2.6}$$

2.3 Existence of Equilibria

In this section, we seek the conditions for the existence of the disease-"free" equilibrium (DFE) $E_0(s_{h0}, 0, 0)$ and the endemic proportion equilibrium $E^*(s_h^*, i_h^*, i_v^*)$ for system

(2.2.4). We equate the right hand side of (2.2.4) to obtain the steady states. We have

$$\begin{cases} s_h^* &= \dfrac{b_1(\mu_v+\beta_3 i_h^*)}{(\mu_v+\beta_3 i_h^*)(b_1+(\beta_1-\delta_h)i_h^*)+\beta_2\beta_3 i_h^*}, \\ i_v^* &= \dfrac{\beta_3 i_h^*}{\mu_v+\beta_3 i_h^*}, \\ (\beta_1 i_h^*+\beta_2 i_v^*)s_h^* &= (b_1+\gamma_h+\delta_h)i_h^*-\delta_h {i_h^*}^2. \end{cases} \tag{2.3.1}$$

From (2.3.1), we get

$$i_h^*(A_3 {i_h^*}^3 + A_2 {i_h^*}^2 + A_1 i_h^* + A_0) = 0, \tag{2.3.2}$$

with

$$\begin{aligned} A_3 &= \beta_3\delta_h(\beta_1-\delta_h), \\ A_2 &= \beta_2\beta_3\delta_h + (\mu_v\delta_h - \beta_3(b_1+\gamma_h+\delta_h))(\beta_1-\delta_h) + b_1\beta_3\delta_h, \\ A_1 &= b_1(\mu_v\delta_h+\beta_1\beta_3) - (b_1+\gamma_h+\delta_h)(\beta_2\beta_3+b_1\beta_3+\mu_v(\beta_1-\delta_h)), \\ A_0 &= (b_1+\gamma_h+\delta_h)b_1\mu_v(\mathfrak{R}_\circ-1). \end{aligned} \tag{2.3.3}$$

We observe that the solution $i_h^*=0$ of (2.3.2) corresponds to the disease free equilibrium E_0 of (2.2.4), which is given by

$$E_0 = (1,0,0).$$

The other roots of (2.3.2), when exists, corresponds to the endemic equilibrium. From right hand side of (2.2.5) we have $\gamma_h i_h^* = (b_1-\delta_h i_h^*)r_h^* > 0$ and first equation of (2.3.1) $\beta_1\mu_v+\beta_2\beta_3-\mu_v\delta_h > \beta_3\delta_h i_h^*$, which means that

$$0 < i_h^* < min\{1, \frac{b_1}{\delta_h}, (\frac{\beta_1\mu_v+\beta_2\beta_3}{\mu_v\delta_h}-1)\frac{\mu_v}{\beta_3}\}. \tag{2.3.4}$$

If $\dfrac{\beta_1\mu_v+\beta_2\beta_3}{\mu_v\delta_h} \le 1$, there is no positive i_h^* and therefore only equilibrium point in Γ is E_0. Note that this is a special case of $\mathfrak{R}_\circ < 1$. Now we shall discuss the roots of (2.3.2) other than 0. Let us denote

$$f(i_h^*) = A_3 {i_h^*}^3 + A_2 {i_h^*}^2 + A_1 i_h^* + A_0. \tag{2.3.5}$$

Assume that $\mathfrak{R}_\circ > 1$.

(1) If $\beta_1 > \delta_h$, then $A_3 > 0$, we have $f(-\infty) < 0$, $f(\infty) > 0$ and $f(0) = A_0 > 0$. Further, $f(1) < 0$ $(if \frac{b_1}{\delta_h} \ge 1)$ and $f(\frac{b_1}{\delta_h}) < 0$. Thus, there exists unique i_h^* such that $f(i_h^*) = 0$ (see Fig. 2.2).

(2) If $\beta_1 = \delta_h$, then $A_3 = 0$ and $f(i_h^*) = A_2 {i_h^*}^2 + A_1 i_h^* + A_0$, where $A_2 = \beta_2\beta_3\delta_h + b_1\beta_3\delta_h > 0$. $f(-\infty) > 0$, $f(\infty) > 0$ and $f(0) = A_0 > 0$. Moreover, $f(1) < 0$ $(if \frac{b_1}{\delta_h} \geq 1)$ and $f(\frac{b_1}{\delta_h}) < 0$. Therefore, there exists unique i_h^* such that $f(i_h^*) = 0$ (see Fig. 2.3).

(3) If $\beta_1 < \delta_h$, then $A_3 < 0$, we have $f(-\infty) > 0$, $f(\infty) < 0$ and still $f(0) = A_0 > 0$,

$f(1) < 0$ $(if \frac{b_1}{\delta_h} \geq 1)$, $f(\frac{b_1}{\delta_h}) < 0$. In this case, we can say that there is only one root or three roots in the interval $(0,1)$ if $\frac{b_1}{\delta_h} \geq 1$ and if $\frac{b_1}{\delta_h} < 1$ then there is only one root or three roots in the interval $(0, \frac{b_1}{\delta_h})$.

We know that $f(i_h^*) = 0$ has three real roots if and only if

$\frac{q^2}{4} + \frac{p^3}{27} \leq 0$,

where

$p = \frac{A_1}{A_3} - \frac{(A_2)^2}{3(A_3)^2}, \qquad q = \frac{A_0}{A_3} - \frac{A_1A_2}{3(A_3)^2} + \frac{2(A_2)^3}{27(A_3)^3}$,

or

$\hat{R_1} = \frac{18A_0A_1A_2A_3 - 4A_0(A_2)^3 - 4(A_1)^3A_3 + (A_1)^2(A_2)^2}{27(A_0)^2(A_3)^2} \geq 1.$

If $\hat{R_1} < 1$, there is unique i_h^* such that $f(i_h^*) = 0$ in the feasible interval.

If $\hat{R_1} > 1$, there are three different real roots for $f(i_h^*) = 0$ say $i_{h1}^*, i_{h2}^*, i_{h3}^* (i_{h1}^* < i_{h2}^* < i_{h3}^*)$. Note that, differentiating with respect to i_h^*, we obtain

$$f'(i_h^*) = 3A_3 {i_h^*}^2 + 2A_2 i_h^* + A_1. \tag{2.3.6}$$

The three different real roots for $f(i_h^*) = 0$ are in the feasible interval if and only if the following inequalities are satisfied

$$\begin{aligned} 0 &< \tfrac{-A_2}{3A_3} < 1, \\ f'(0) &= A_1 < 0, \\ f'(1) &= 3A_3 + 2A_2 + A_1 < 0 \ (if \tfrac{b_1}{\delta_h} \geq 1), \\ f'(\tfrac{b_1}{\delta_h}) &= 3A_3(\tfrac{b_1}{\delta_h})^2 + 2A_2(\tfrac{b_1}{\delta_h}) + A_1 < 0 \ (if \tfrac{b_1}{\delta_h} < 1). \end{aligned} \tag{2.3.7}$$

If $\hat{R_1} = 1$, there are three real roots for $f(i_h^*) = 0$, in which at least two are identical. Similarly, if inequalities (2.3.7) are satisfied, then there are three real roots for $f(i_h^*) = 0$ in the feasible interval, say $i_{h1}^*, i_{h2}^*, i_{h3}^* (i_{h1}^* = i_{h2}^*)$.

Assume that $\mathfrak{R}_o = 1$.

(1) If $\beta_1 = \delta_h$, then $A_3 = 0$ and (2.3.5) reduces to $i_h^*(A_2 i_h^* + A_1) = 0$, which implies that $i_h^* = 0$ or $i_h^* = \frac{-A_1}{A_2}$, which is negative and lies outside the interval $(0,1)$ if $\frac{b_1}{\delta_h} \geq 1$ or $(0, \frac{b_1}{\delta_h})$ if $\frac{b_1}{\delta_h} < 1$.

(2) If $\beta_1 > \delta_h$, then $A_3 > 0$, we have $i_h^*(A_3 {i_h^*}^2 + A_2 i_h^* + A_1) = 0$ which implies that $i_h^* = 0$ or i_h^* is the solution of the equation

$$g(i_h^*) = A_3 {i_h^*}^2 + A_2 i_h^* + A_1 = 0.$$

$g(-\infty) > 0$, $g(\infty) > 0$ and $g(0) = A_1 < 0$. Moreover, $g(1) < 0$ $(if \frac{b_1}{\delta_h} \geq 1)$ and $g(\frac{b_1}{\delta_h}) < 0$ $if \frac{b_1}{\delta_h} < 1$. Therefore, there exists no i_h^* such that $g(i_h^*) = 0$ in the interval $(0, 1)$ if $\frac{b_1}{\delta_h} \geq 1$ or $(0, \frac{b_1}{\delta_h})$ if $\frac{b_1}{\delta_h} < 1$. In summary, regarding the existence and the number of the "endemic" equilibria, we have

Theorem 2.3.1. *Suppose that $\beta_1 \geq \delta_h$. There is always a disease "free" equilibrium for system (2.2.4); if $\mathfrak{R}_o > 1$, then there is a unique "endemic" equilibrium $E^*(s_h^*, i_h^*, i_v^*)$ with coordinates satisfying (2.3.1) besides the disease "free" equilibrium.*

2.4 Stability of Disease "Free" Equilibrium

We shall analyze local stability as well as global stability of disease "free" equilibrium.

2.4.1 Local Stability

The Jacobian matrix of (2.2.4) at an arbitrary point $E(s_h, i_h, i_v)$ takes the form:

$$J(E) = \begin{pmatrix} -b_1 - \beta_1 i_h - \beta_2 i_v + \delta_h i_h & -(\beta_1 - \delta_h) s_h & -\beta_2 s_h \\ \beta_1 i_h + \beta_2 i_v & \beta_1 s_h - (b_1 + \gamma_h + \delta_h) + 2\delta_h i_h & \beta_2 s_h \\ 0 & \beta_3(1 - i_v) & -\beta_3 i_h - \mu_v \end{pmatrix}.$$

To analyze the stability of DFE, we calculate the characteristic equation of J(E) at $E = E_0$ as follows:

$$(\lambda + b_1)(\lambda^2 + \lambda(\mu_v + b_1 + \gamma_h + \delta_h - \beta_1) + \mu_v(b_1 + \gamma_h + \delta_h)(1 - \mathfrak{R}_o)) \tag{2.4.1}$$

where

$$\mathfrak{R}_o = \frac{\beta_1}{b_1 + \gamma_h + \delta_h} + \frac{\beta_2 \beta_3}{\mu_v(b_1 + \gamma_h + \delta_h)}.$$

By Routh Hurwitz criteria [45] all roots of the equation (2.4.1) have negative real parts if and only if $\mathfrak{R}_o < 1$. So, E_0 is locally asymptotically stable for $\mathfrak{R}_o < 1$. If $\mathfrak{R}_o > 1$, the characteristic equation (2.4.1) has positive eigenvalue, E_0 is thus unstable. We established the following theorem.

Theorem 2.4.1. *The disease free equilibrium is locally asymptotically stable whenever $\mathfrak{R}_o < 1$ and unstable for $\mathfrak{R}_o > 1$.*

2.4.2 Global Stability

In this subsection, we analyze the global behavior of disease-free equilibrium E_0 for system (2.2.4). The following theorem provides the global property of the system.

Theorem 2.4.2. *If $\mathfrak{R}_\circ \leq 1$, then the infection-free equilibrium E_0 is globally asymptotically stable in the interior of Γ.*

Proof. To establish the global stability of the disease-free equilibrium, we construct the following Lyapunov function:

$$L(t) = \mu_v i_h(t) + \beta_2 i_v(t).$$

Calculating the time derivative of L along (2.2.4), we obtain

$$\begin{aligned}
L'(t) &= \mu_v i_h'(t) + \beta_2 i_v'(t) \\
&= \mu_v[\beta_1 s_h i_h + \beta_2 s_h i_v - (b_1 + \gamma_h + \delta_h) i_h + \delta_h i_h^2] + \beta_2(\beta_3 s_v i_h - \mu_v i_v) \\
&= \mu_v[\beta_1(1 - i_h) i_h + \beta_2(1 - i_h) i_v - (b_1 + \gamma_h + \delta_h) i_h + \delta_h i_h^2] + \beta_2[\beta_3(1 - i_v) i_h - \mu_v i_v] \\
&= \mu_v[\beta_1 i_h - \beta_1 i_h^2 + \beta_2 i_v - \beta_2 i_h i_v - (b_1 + \gamma_h + \delta_h) i_h + \delta_h i_h^2] + \beta_2(\beta_3 i_h - \beta_3 i_v i_h - \mu_v i_v) \\
&= \mu_v \beta_1 i_h - \mu_v \beta_1 i_h^2 + \mu_v \beta_2 i_v - \mu_v \beta_2 i_h i_v - \mu_v(b_1 + \gamma_h + \delta_h) i_h + \mu_v \delta_h i_h^2 + \beta_2 \beta_3 i_h - \beta_2 \beta_3 i_v i_h \\
&\quad - \beta_2 \mu_v i_v \\
&= -\mu_v(b_1 + \gamma_h + \delta_h)(1 - \mathfrak{R}_\circ) i_h - \mu_v(\beta_1 - \delta_h) i_h^2 - \mu_v \beta_2 i_h i_v - \beta_2 \beta_3 i_v i_h.
\end{aligned}$$

Thus $L'(t)$ is negative if $\mathfrak{R}_\circ \leq 1$ and $L' = 0$ if and only if $i_h = 0$. Consequently, the largest compact invariant set in $\{(S_h, I_h, I_v) \in \Gamma, L' = 0\}$, when $\mathfrak{R}_\circ \leq 1$, is the singelton $\{E_0\}$. Hence, LaSalle's invariance principle [46] implies that "E_0" is globally asymptotically stable in Γ. This completes the proof.

2.5 Global Stability of "Endemic" Equilibrium

we use the geometrical approach of Li and Muldowney [47] to investigate the global stability of the endemic equilibrium E^* in the feasible region Γ. The detailed introduction of this approach can be seen in [47]. We write the summary of this approach below:
Consider a C^1 map $f : x \mapsto f(x)$ from an open set $D \subset R^n$ to R^n such that each solution $x(t, x_0)$ to the differential equation

$$x' = f(x), \tag{2.5.1}$$

is uniquely determined by the initial value $x(0, x_0)$. We have the following assumptions:
(H_1) D is simply connected;
(H_2) There exists a compact absorbing set $K \subset D$;
(H_3) (2.5.1) has unique equilibrium $\bar{x}$ in D.
Let $P : x \mapsto P(x)$ be a nonsingular $\binom{n}{2} \times \binom{n}{2}$ matrix-valued function which is C^1 in D and a vector norm $|\,.\,|$ on R^N, where$N = \binom{n}{2}$. Let μ be the Lozinskiĭ measure with respect to the $|\,.\,|$. Define a quantity $\bar{q}_2$ as

$$\bar{q}_2 = \limsup_{t\to\infty} \sup_{x_0\in K} \frac{1}{t} \int_0^t \mu(B(x(s, x_0)))ds,$$

where $B = P_f P^{-1} + PJ^{[2]}P^{-1}$, the matrix P_f is obtained by replacing each entry p of P by its derivative in the direction of f, $(p_{ij})_f$, and $J^{[2]}$ is the second additive compound matrix of the Jacobian matrix J of (2.5.1). The following result has been established in Li and Muldowney [47].

Theorem 2.5.1. *Suppose that* (H_1), (H_2) *and* (H_3) *hold, the unique endemic equilibrium* E^* *is globally stable in* Γ *if* $\bar{q}_2 < 0$.

Obviously Γ is simply connected and E^* is unique endemic equilibrium for $\mathfrak{R}_o > 1$ in Γ. To apply the result of above theorem for global stability of endemic equilibrium E^*, we first prove the uniform persistence of (2.2.4) when the threshold parameter $\mathfrak{R}_o > 1$, by applying the acyclicity Theorem (see [48], p. 18).
Definition. [49] The system (2.2.4) is uniformly persistent i. e. there exists $c > 0$ (independent of initial conditions), such that $\liminf_{t\to\infty} s_h \geq c$, $\liminf_{t\to\infty} i_h \geq c$, $\liminf_{t\to\infty} i_v \geq c$.
Let X be a locally compact metric space with metric d and let Γ be a closed nonempty subset of X with boundary Γ and interior Γ°. Clearly, Γ° is a closed subset of Γ. Let Φ_t be a dynamical system defined on Γ. A set B in X is said to be invariant if $\Phi(B, t) = \mathrm{B}$.
Define $M_\partial := \{x \in \Gamma : \Phi(t, x) \in \Gamma, for\ all\ t \geq 0\}$.
The following lemma has been proved in [49].

Lemma 2.5.2. *Assume that*

(a) Φ_t *has a global attractor;*

(b) *There exists* $M = \{M_1, ..., M_k\}$ *of pair-wise disjoint , compact and isolated invariant set on* $\partial\Gamma$ *such that*

1. $\bigcup_{x\in M_\partial} \omega(x) \subseteq \bigcup_{j=1}^k M_j$;

2. *No subsets of* M *form a cycle on* $\partial\Gamma$;

3. *Each* M_j *is also isolated in* Γ*;*

4. $W^s(M_j)\bigcap\Gamma^\circ = \phi$ *for each* $1 \leq j \leq k$*, where* $W^s(M_j)$ *is stable manifold of* M_j*. Then* Φ_t *is uniformly persistent with respect to* Γ.

We shall prove all the conditions of above lemma for our system. We have $\Gamma = \{(s_h, i_h, i_v) \in R^3{}_+, 0 \leq s_h + i_h \leq 1, 0 \leq i_v \leq 1\}$, $\Gamma^\circ = \{(s_h, i_h, i_v) \in R^3{}_+ \; s_h, i_h > 0\}$, $\partial\Gamma = \Gamma/\Gamma^\circ$. Obviously $M_\partial = \partial\Gamma$. Since Γ is bounded and positively invariant so there exists a compact set M in which all solutions of system (2.2.4) initiated in Γ ultimately enter and remain forever. On s_h-axis we have $s_h' = b_1(1 - s_h)$ which means $s_h \to 1$ as $t \to \infty$. Thus E_0 is the only omega limit point on $\partial\Gamma$ i.e., $\omega(x) = E_0$ for all $x \in M_\partial$. Furthermore $M = E_0$ is a covering of $\Omega = \bigcup_{x\in M_\partial}\omega(x)$ because all solutions initiated on the s_h-axis converge to E_0. Also E_0 is isolated and acyclic. This verifies that hypothesis (1) and (2) hold. When $\mathfrak{R}_\circ > 1$, the disease-"free" equilibrium (DFE)E_0 is unstable from theorem (2.4.1) and also $W^s(M) = \partial\Gamma$. Hypothesis (3) and (4) hold. There always admits a global attractor due to ultimate boundedness of solutions. □

The boundedness of Γ and the above lemma imply that (2.2.4) has a compact absorbing set $K \subset \Gamma$ [49]. Now we shall prove that the quantity $\bar{q}_2 < 0$. We choose a suitable vector norm $|.|$ in R^3 and a 3×3 matrix valued function

$$P(x) = \begin{pmatrix} 1 & 0 & 0 \\ 0 & \dfrac{i_h}{i_v} & 0 \\ 0 & 0 & \dfrac{i_h}{i_v} \end{pmatrix}. \tag{2.5.2}$$

Obviously P is C^1 and non singular in the interior of Ω. Linearizing system (2.2.4) about an endemic equilibrium E^* gives the following Jacobian matrix

$$J(E^*) = \begin{pmatrix} -\dfrac{b_1}{s_h} & -(\beta_1 - \delta_h)s_h & -\beta_2 s_h \\ \beta_1 i_h + \beta_2 i_v & \beta_1 s_h - (b_1 + \gamma_h + \delta_h) + 2\delta_h i_h & \beta_2 s_h \\ 0 & \beta_3(1 - i_v) & -\beta_3 i_h - \mu_v \end{pmatrix}.$$

The second additive compound matrix of $J(E^*)$ is given by

$$J^{[2]} = \begin{pmatrix} M_{11} & \beta_2 s_h & \beta_2 s_h \\ \beta_3(1-i_v) & M_{22} & -(\beta_1-\delta_h)s_h \\ 0 & \beta_1 i_h + \beta_2 i_v & M_{33} \end{pmatrix},$$

where

$$\begin{aligned} M_{11} &= -\frac{b_1}{s_h} + \beta_1 s_h - (b_1+\gamma_h+\delta_h) + 2\delta_h i_h, \\ M_{22} &= -\frac{b_1}{s_h} - \beta_3 i_h - \mu_v, \\ M_{33} &= \beta_1 s_h - (b_1+\gamma_h+\delta_h) + 2\delta_h i_h - \beta_3 i_h - \mu_v. \end{aligned} \tag{2.5.3}$$

The matrix $B = P_f P^{-1} + P J^{[2]} P^{-1}$ can be written in block form as

$$B = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix},$$

with

$$\begin{aligned} B_{11} &= -\frac{b_1}{s_h} + \beta_1 s_h - (b_1+\gamma_h+\delta_h) + 2\delta_h i_h, \\ B_{12} &= \big(\beta_2 s_h \frac{i_v}{i_h}, \beta_2 s_h \frac{i_v}{i_h}\big), \\ B_{21} &= \begin{pmatrix} (\frac{i_h}{i_v})\beta_3(1-i_v) \\ \\ 0 \end{pmatrix}, \\ B_{22} &= \begin{pmatrix} Q_{11} & Q_{12} \\ \\ Q_{21} & Q_{22} \end{pmatrix}, \end{aligned} \tag{2.5.4}$$

where

$$\begin{aligned} Q_{11} &= -\frac{b_1}{s_h} - \beta_3 i_h - \mu_v, \\ Q_{12} &= -(\beta_1 - \delta_h) s_h, \\ Q_{21} &= \beta_1 i_h + \beta_2 i_v, \\ Q_{22} &= \beta_1 s_h - (b_1 + \gamma_h + \delta_h) + 2\delta_h i_h - \beta_3 i_h - \mu_v, \\ \frac{i_v}{i_h}(\frac{i_h}{i_v})_f &= \frac{i_h'}{i_h} - \frac{i_v'}{i_v}. \end{aligned} \tag{2.5.5}$$

Consider the norm in R^3 as: $\mid (u, v, w) \mid = max(\mid u \mid, \mid v \mid + \mid w \mid)$ where (u, v, w) denotes the vector in R^3. The Lozinskiĭ measure with respect to this norm is defined as

$$\mu(B) \leq sup(g_1, g_2),$$

where

$$g_1 = \mu_1(B_{11}) + \mid B_{12} \mid, \quad g_2 = \mu_1(B_{22}) + \mid B_{21} \mid .$$

From system (2.2.4) we can write

$$\begin{aligned} \frac{i_h'}{i_h} &= \beta_1 s_h + \beta_2 s_h \frac{i_v}{i_h} - (b_1 + \gamma_h + \delta_h) + \delta_h i_h, \\ \frac{i_v'}{i_v} &= \beta_3 (1 - i_v) \frac{i_h}{i_v} - \mu_v. \end{aligned} \tag{2.5.6}$$

Since B_{11} is a scalar, its Lozinskiĭ measure with respect to any vector norm in R^1 will be equal to B_{11}. Thus

$$B_{11} = -\frac{b_1}{s_h} + \beta_1 s_h - (b_1 + \gamma_h + \delta_h) + 2\delta_h i_h,$$

$$\mid B_{12} \mid = \beta_2 s_h \frac{i_v}{i_h},$$

and g_1 will become

$$\begin{aligned} g_1 &= -\frac{b_1}{s_h} + \beta_1 s_h - (b_1 + \gamma_h + \delta_h) + 2\delta_h i_h + \beta_2 s_h \frac{i_v}{i_h} \\ &= \frac{i_h'}{i_h} - \frac{b_1}{s_h} + \delta_h i_h \\ &\leq \frac{i_h'}{i_h} - b_1 + \delta_h i_h. \end{aligned} \tag{2.5.7}$$

Also $\mid B_{21} \mid = (\frac{i_h}{i_v})\beta_3(1-i_v)$, $\mid B_{12} \mid$ and $\mid B_{21} \mid$ are the operator norms of B_{12} and B_{21} which are mapping from R^2 to R and from R to R^2 respectively, and R^2 is endowed with the l_1 norm. $\mu_1(B_{22})$ is the Lozinskiĭ measure of 2×2 matrix B_{22} with respect to l_1 norm in R^2.

$$\begin{aligned}\mu(B_{22}) &= Sup\{\frac{i_v}{i_h}(\frac{i_h}{i_v})_f - \frac{b_1}{s_h} - \beta_3 i_h - \mu_v + \beta_1 i_h + \beta_2 i_v, \frac{i_v}{i_h}(\frac{i_h}{i_v})_f + (\beta_1-\delta_h)s_h + \beta_1 s_h - \\ &\quad (b_1+\gamma_h+\delta_h) + 2\delta_h - \beta_3 i_h - \mu_v i_h\} \\ &\leq \frac{i_h'}{i_h} - \frac{i_v'}{i_v} - b_1 + \delta_h i_h - \beta_3 i_h - \mu_v, \end{aligned} \tag{2.5.8}$$

if $\beta_1 \leq \frac{\gamma_h}{2}$. Hence

$$\begin{aligned} g_2 &\leq \tfrac{i_h'}{i_h} - \tfrac{i_v'}{i_v} - b_1 + \delta_h i_h - \beta_3 i_h - \mu_v + (\tfrac{i_h}{i_v})\beta_3(1-i_v) \\ &= \tfrac{i_h'}{i_h} - b_1 + \delta_h i_h - \beta_3 i_h. \end{aligned} \tag{2.5.9}$$

Thus,

$$\begin{aligned}\mu(B) &= Sup\{g_1, g_2\} \\ &\leq \frac{i_h'}{i_h} - b_1 + \delta_h \\ &\leq \frac{i_h'}{i_h} - \bar{\beta}_1, \end{aligned} \tag{2.5.10}$$

where $\bar{\beta}_1 = min(\frac{\gamma_h}{2}, \frac{b_1}{2})$. Since (2.2.3) is uniformly persistent when $\mathfrak{R}_o > 1$, so for $T > 0$ such that $t > T$ implies $i_h(t) \geq c$, $i_v(t) \geq c$ and $\frac{1}{t}\log i_h(t) < \frac{\bar{\beta}_1}{2}$ for all $(s_h(0), i_h(0), i_v(0)) \in K$. Thus

$$\frac{1}{t}\int_0^t \mu(B)dt < \frac{\log i_h(t)}{t} - \bar{\beta}_1 < -\frac{\bar{\beta}_1}{2}$$

for all $(s_h(0), i_h(0), i_v(0)) \in K$, which further implies that $\bar{q}_2 < 0$. Therefore all the conditions of Theorem (2.5.1) are satisfied. Hence unique endemic equilibrium E^* is globally stable in Γ.

2.6 Discussions and Simulations

In this section we shall solve the model with the help of Runge-Kutta fourth order scheme. The model has a globally asymptotically stable disease-"free" equilibrium whenever $\mathfrak{R}_o \leq 1$ (Figs. 2.4, 2.5). When $\mathfrak{R}_o > 1$, the disease persists at an"endemic" level (Figs.2.6, 2.7)

if $\beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$.(Figs. 2.8, 2.9, 2.10, 2.11) describes numerically "endemic" level of infectious individuals and infectious vectors under the condition $\beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$. We here question that what are the dynamics of the proportionate system (2.2.4) even if the condition $\beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$ is not satisfied? We see numerically that if $\delta_h, \frac{\gamma_h}{2} < \beta_1 < \frac{b_1}{2}$ or $\frac{\gamma_h}{2} < \beta_1 = \delta_h < \frac{b_1}{2}$, then infectious individuals and infectious vectors will also approach to endemic level for different initial conditions (Figs. 2.12, 2.13, 2.14, 2.15).

Now we shall discuss the epidemiological correlations between the two systems normalized and unnormalized models.

If $\delta_h = 0$ and $b_1 = \mu_h$ then $N_h(t)' = 0$ and so $N_h(t)$ remains fixed at its initial value N_{h0}. In this case the system (2.1.1)becomes the model with constant population whose dynamics are the same as the proportionate system (2.2.2). Hence the solutions with initial conditions $S_{h0} + I_{h0} + R_{h0} = N_{h0}$, tend to $(N_{h0}, 0, 0)$ if $\mathfrak{R}_o \leq 1$ and to $N_{h0}({s_h}^*, {i_h}^*, {r_h}^*)$ if $\mathfrak{R}_o > 1$. In the rest of this section we suppose that $\delta_h > 0$. From system (6.1.1) and (5.5) the trivial equilibrium $E = (0, 0, 0, 0, 0)$ can be easily obtained. Assume that $E_* = ({N_h}^*, {S_h}^*, {I_h}^*, {R_h}^*, {I_v}^*)$ is the endemic equilibrium of system (2.1.1) and (2.1.2), where ${N_h}^* = {S_h}^* + {I_h}^* + {R_h}^*$. This equilibrium exists if and only if the following equations are satisfied

$$\begin{aligned}
\frac{{S_h}^*}{{N_h}^*} &= \frac{Q(\beta_3\alpha_h + \mu_v\delta_h)}{\beta_1(\beta_3\alpha_h + \mu_v\delta_h) + \beta_2\beta_3\delta_h}, \\
\frac{{I_h}^*}{{N_h}^*} &= \frac{\alpha_h}{\delta_h}, \\
\frac{{R_h}^*}{{N_h}^*} &= \frac{\gamma_h\alpha_h}{\mu_h\delta_h}, \\
\frac{{I_v}^*}{{N_h}^*} &= \frac{\beta_3\alpha_h N_v}{(\beta_3\alpha_h + \mu_v){N_h}^*},
\end{aligned}$$

where $\alpha_h = b_1 - \mu_h$ and $Q = \mu_h + \gamma_h + \delta_h$. We introduce the parameters

$$R_1 = \begin{cases} \dfrac{b_1}{\mu_h}, & \text{if } \mathfrak{R}_o \leq 1 \\ \dfrac{b_1}{\mu_h + \delta_h {i_h}^*}, & \text{if } \mathfrak{R}_o > 1. \end{cases}$$

$$R_2 = \begin{cases} \dfrac{\beta_1}{\mu_h + \gamma_h + \delta_h} + \dfrac{\beta_2\beta_3}{\mu_v(\mu_h + \gamma_h + \delta_h)}, & \text{if } \mathfrak{R}_o \leq 1 \\ \dfrac{\beta_1 s_h^*}{\mu_h + \gamma_h + \delta_h} + \dfrac{\beta_2\beta_3 s_h^*(1 - {i_v}^*)}{\mu_v(\mu_h + \gamma_h + \delta_h)}, & \text{if } \mathfrak{R}_o > 1. \end{cases}$$

From (2.1.2) we have for $t \to \infty$

$$\frac{dN_h}{dt} = N_h(b_1 - \mu_h - \delta_h i_h) \to \begin{cases} N_h(b_1 - \mu_h), & \text{if } \mathfrak{R}_o \leq 1 \\ \\ N_h(b_1 - \mu_h - \delta_h {i_h}^*), & \text{if } \mathfrak{R}_o > 1. \end{cases}$$

By the definition of R_1 we have following threshold result.

Theorem 2.6.1. *The total population $N_h(t)$ for the system (2.1.1) decreases to zero if $R_1 < 1$ and increases to ∞ if $R_1 > 1$ as $t \to \infty$. The asymptotic rate of decrease is $\mu_h(R_1 - 1)$ if $\mathfrak{R}_o \leq 1$, and the asymptotic rate of increase is $(\mu_h + \delta_h {i_h}^*)(R_1 - 1)$ when $\mathfrak{R}_o > 1$.*

Theorem 2.6.2. *Suppose $R_1 > 1$, for $t \to \infty$, $(S_h(t), I_h(t), R_h(t))$ tend to $(\infty, 0, 0)$ if $R_2 < 1$ and tend to (∞, ∞, ∞) if $R_2 > 1$.*

Proof. Since ${i_v}' \to 0$ as $t \to \infty$, so in the limiting case the proportion of infectious mosquitoes is related to the proportion of infectious humans as

$$i_v = \frac{\beta_3(1 - i_v)i_h}{\mu_v},$$

thus, the equation for $I_h(t)$ has limiting form

$$\frac{dI_h}{dt} = (\mu_h + \gamma_h + \delta_h)(R_2 - 1)I_h,$$

which shows that $I_h(t)$ decreases exponentially if $R_2 < 1$ and increases exponentially if $R_2 > 1$.

The solution $R_h(t)$ is given by

$$R_h = R_{h0}e^{-\mu_h t} + \gamma_h e^{-\mu_h t} \int_0^t I_h(s)e^{\mu_h s} ds,$$

From the exponential nature of I_h, it follows that I_h declines exponentially if $R_2 < 1$, and grows exponentially if $R_2 > 1$.

Suppose $R_1 = 1$, then $b_1 = \mu_h$ corresponding to $\mathfrak{R}_o < 1$ and the differential equation for $N_h(t)$ will have form

$$\frac{dN_h}{dt} = -\delta_h I_h.$$

which means that $N_h(t)$ is bounded for all $t > 0$, the equilibria $({N_h}^*, 0, 0, 0)$ have one eigenvalue zero and the other eigenvalues have negative real parts.Therefore, each orbit approaches an equilibrium point.

If $\mathfrak{R}_o > 1$, the disease becomes "endemic". From the global stability of E^* and the equation

$$\frac{dN_h}{dt} = \delta_h[(\frac{b_1 - \mu_h}{\delta_h} - {i_h}^*) - (i_h - {i_h}^*)]N_h,$$

we observe that $(N_h, S_h, I_h, R_h, I_v)$ approaches to $(0,0,0,0,0)$ or $(\infty,\infty,\infty,\infty,\infty)$ if $R_1 < 1$ or $R_1 > 1$. From the global stability of ${i_h}^*$, we have $N_h(t)$ converges to some ${N_h}^*$ as t approaches to ∞. Since $s_h = \frac{S_h}{N_h}$, $i_h = \frac{I_h}{N_h}$, $r_h = \frac{R_h}{N_h}$, so we have ${S_h}^* = {s_h}^*{N_h}^*$, ${I_h}^* = {i_h}^*{N_h}^*$, ${R_h}^* = {r_h}^*{N_h}^*$. All the above discussion is summarized in the following Table.

Table 2.2: Asymptotic behavior with threshold criteria

$\Re_o$	R_1	R_2	N_h	$(s_h, i_h, r_h, i_v) \to$	$(S_h, I_h, R_h) \to$
≤ 1	$= 1, \delta_h = 0$	≤ 1	$N_h = N_{h0}$	$(1,0,0,0)$	$(N_{h0},0,0)$
> 1	$= 1, \delta_h = 0$	$= 1$	$N_h = N_{h0}$	$({s_h}^*, {i_h}^*, {r_h}^*, {i_v}^*)$	$N_{h0}({s_h}^*, {i_h}^*, {r_h}^*)$
≤ 1	< 1	< 1	$N_h \to 0$	$(1,0,0,0)$	$(0,0,0)$
> 1	< 1	< 1	$N_h \to 0$	$({s_h}^*, {i_h}^*, {r_h}^*, {i_v}^*)$	$(0,0,0)$
≤ 1	> 1	< 1	$N_h \to \infty$	$(1,0,0,0)$	$(\infty,0,0)$
≤ 1	> 1	> 1	$N_h \to \infty$	$(1,0,0,0)$	(∞,∞,∞)
< 1	$= 1$	< 1	$N_h \to N_h^*$	$(1,0,0,0)$	$(N_h^*,0,0)$
> 1	> 1	> 1	$N_h \to \infty$	$({s_h}^*, {i_h}^*, {r_h}^*, {i_v}^*)$	(∞,∞,∞)
> 1	$= 1$	$= 1$	$N_h \to N_h^*$	$({s_h}^*, {i_h}^*, {r_h}^*, {i_v}^*)$	$({S_h}^*, {I_h}^*, {R_h}^*)$

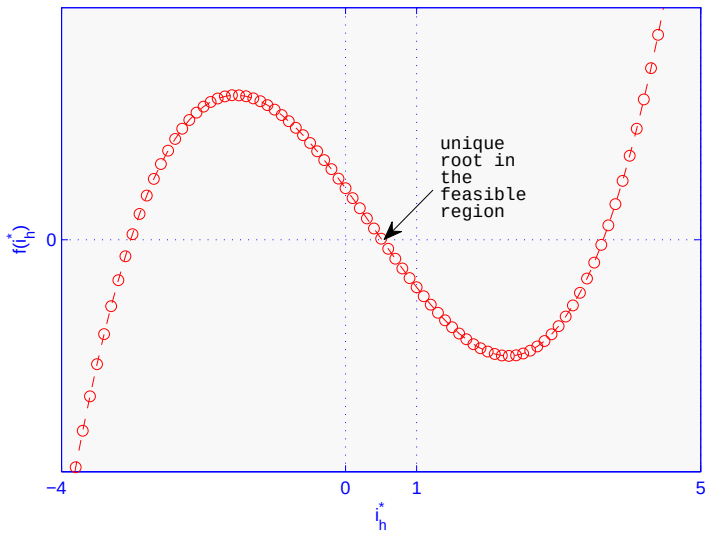

Figure 2.2: Plot of $f(i_h^*)$ showing that unique value of i_h^* in the feasible region when $\beta_1 > \delta_h$.

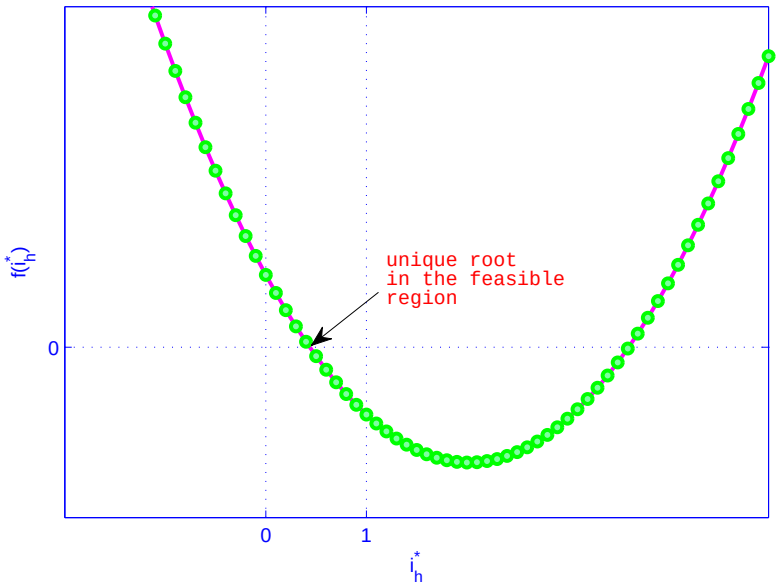

Figure 2.3: Plot of $f(i_h^*)$ showing that unique value of i_h^* in the feasible region when $\beta_1 = \delta_h$.

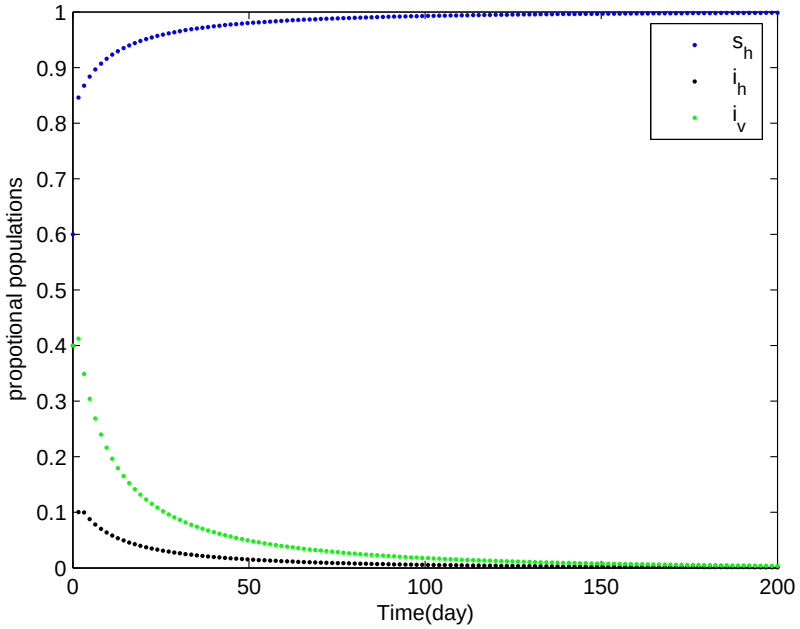

Figure 2.4: The proportional population approaches disease free equilibrium $(1, 0, 0)$ when $\mathfrak{R}_o < 1$ and $\beta_1 = \delta_h$. The parameter values are $b_1 = 1, \beta_1 = 0.02, \beta_2 = 0.4, \beta_3 = 0.6, \gamma_h = 0.3, \delta_h = 0.02, \mu_v = 0.2, \mathfrak{R}_o = 0.92$.

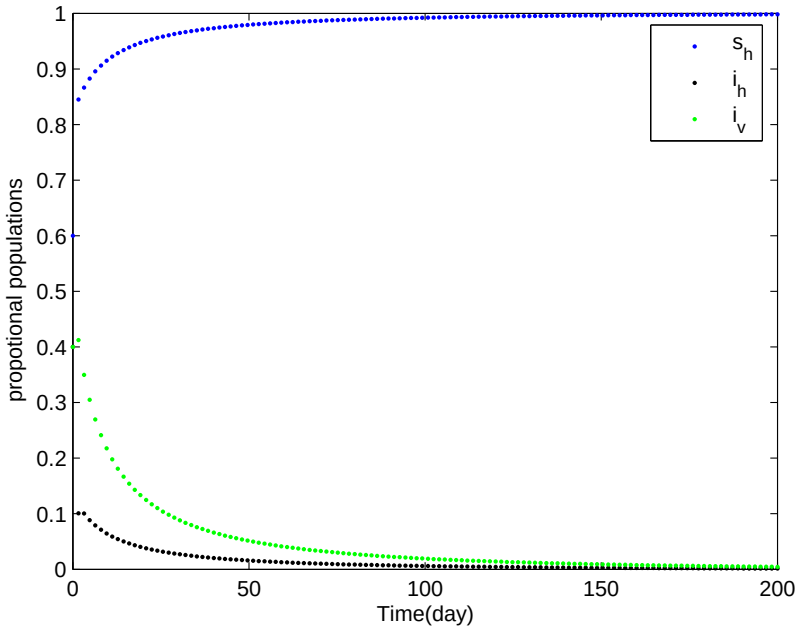

Figure 2.5: The proportional population approaches disease free equilibrium $(1, 0, 0)$ when $\mathfrak{R}_o < 1$ and $\beta_1 > \delta_h$. The parameter values are $b_1 = 1, \beta_1 = 0.02, \beta_2 = 0.4, \beta_3 = 0.6, \gamma_h = 0.3, \delta_h = 0.01, \mu_v = 0.2, \mathfrak{R}_o = 0.92$.

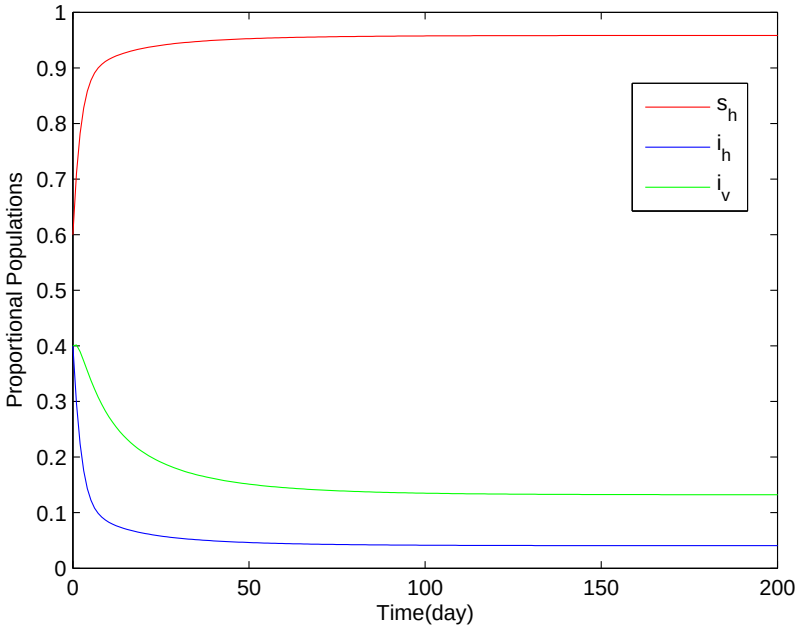

Figure 2.6: The proportional population approaches endemic equilibrium (s_h^*, i_h^*, i_v^*) when $\mathfrak{R}_o > 1$ and $\beta_1 > \delta_h$. The parameter values are $b_1 = 2, \beta_1 = 0.025, \beta_2 = 0.65, \beta_3 = 0.75, \gamma_h = 0.051, \delta_h = 0.000025, \mu_v = 0.2, \mathfrak{R}_o = 1.9$.

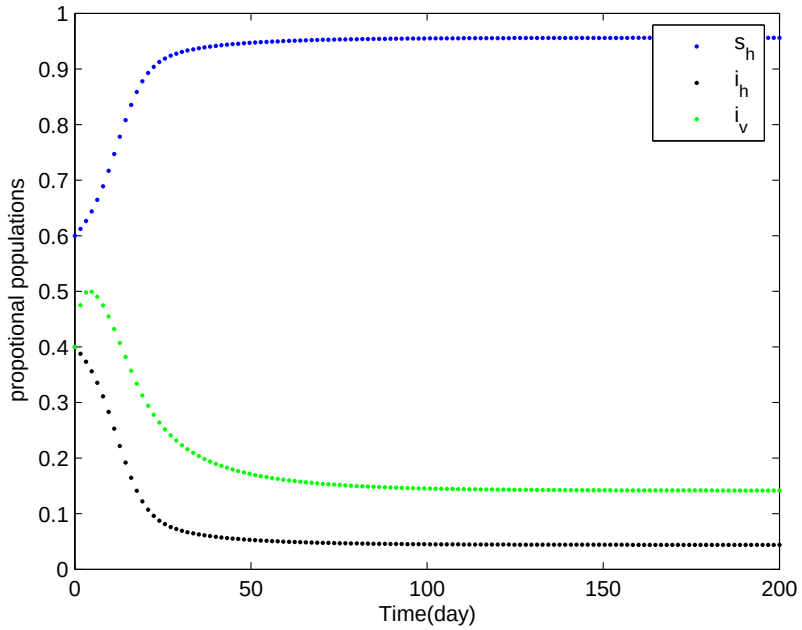

Figure 2.7: The proportional population approaches endemic equilibrium (s_h^*, i_h^*, i_v^*) when $\mathfrak{R}_o > 1$ and $\beta_1 = \delta_h$. The parameter values are $b_1 = 2, \beta_1 = 0.0025, \beta_2 = 0.65, \beta_3 = 0.75, \gamma_h = 0.051, \delta_h = 0.0025, \mu_v = 0.2, \mathfrak{R}_o = 1.19$.

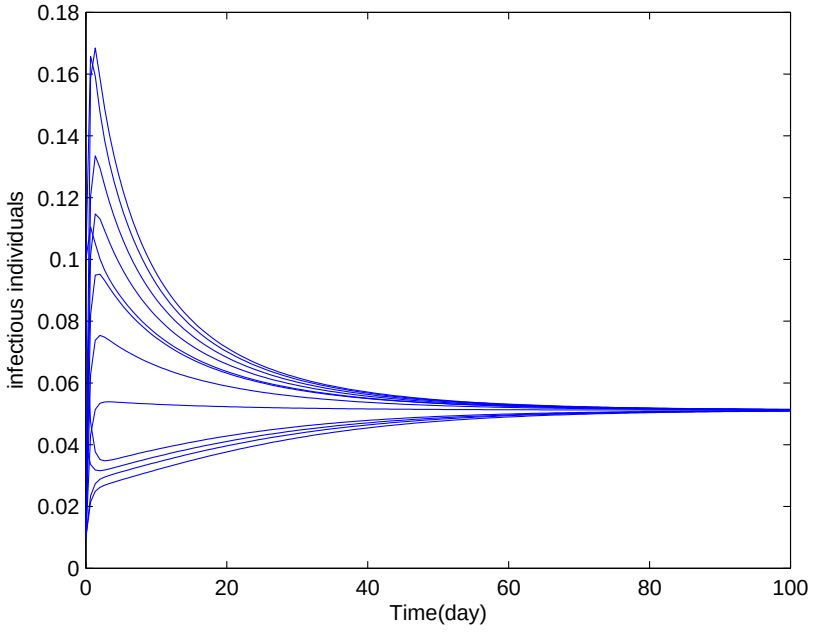

Figure 2.8: Infectious individuals approach unique endemic equilibrium (s_h^*, i_h^*, i_v^*) for different initial conditions when $\mathfrak{R}_o > 1$ and $\delta_h < \beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$. The parameter values are $b_1 = 2, \beta_1 = 0.4, \beta_2 = 0.85, \beta_3 = 0.75, \gamma_h = 0.85, \delta_h = 0.0000001, \mu_v = 0.2, \mathfrak{R}_o = 1.26$.

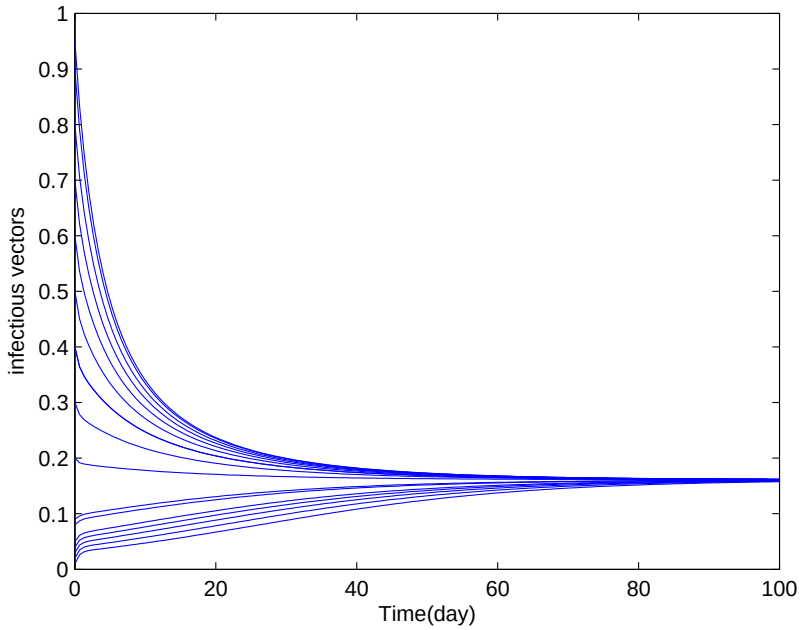

Figure 2.9: Infectious vectors approach unique endemic equilibrium (s_h^*, i_h^*, i_v^*) for different initial conditions when $\mathfrak{R}_o > 1$ and $\delta_h < \beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$. The parameter values are $b_1 = 2, \beta_1 = 0.4, \beta_2 = 0.85, \beta_3 = 0.75, \gamma_h = 0.85, \delta_h = 0.0000001, \mu_v = 0.2, \mathfrak{R}_o = 1.26$.

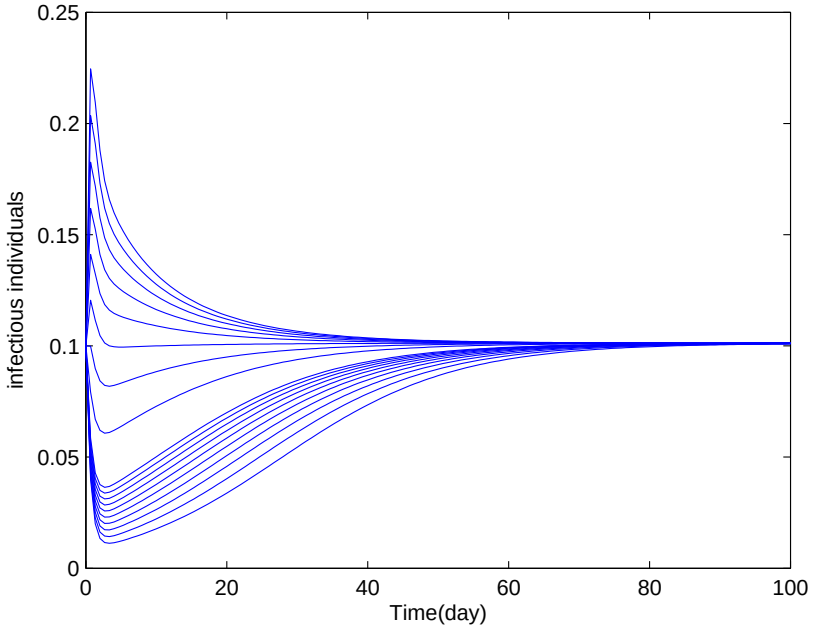

Figure 2.10: Infectious individuals approach unique endemic equilibrium (s_h^*, i_h^*, i_v^*) for different initial conditions when $\mathfrak{R}_o > 1$ and $\delta_h = \beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$. The parameter values are $b_1 = 2, \beta_1 = 0.04, \beta_2 = 0.85, \beta_3 = 0.65, \gamma_h = 0.85, \delta_h = 0.04, \mu_v = 0.1, \mathfrak{R}_o = 1.92$.

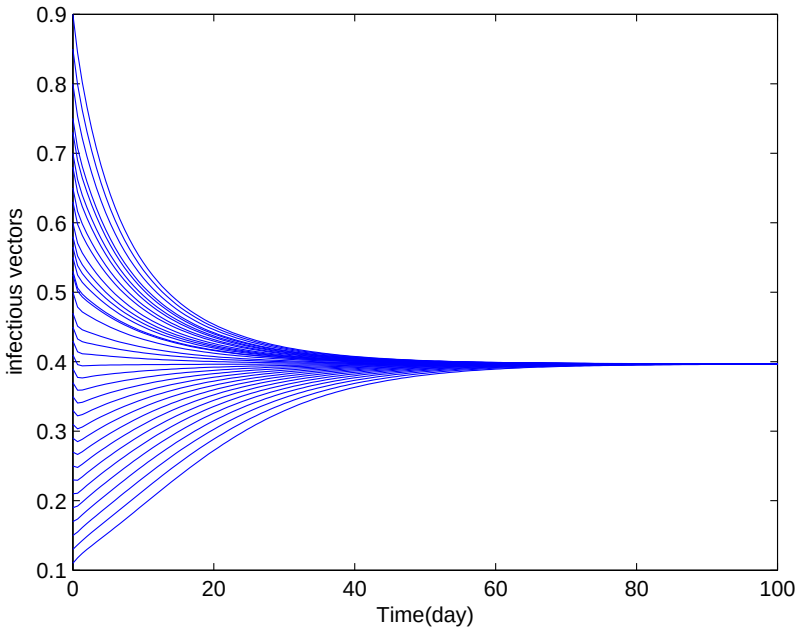

Figure 2.11: Infectious vectors approach unique endemic equilibrium (s_h^*, i_h^*, i_v^*) for different initial conditions when $\mathfrak{R}_o > 1$ and $\delta_h = \beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$. The parameter values are $b_1 = 2, \beta_1 = 0.04, \beta_2 = 0.85, \beta_3 = 0.65, \gamma_h = 0.85, \delta_h = 0.04, \mu_v = 0.1, \mathfrak{R}_o = 1.92$.

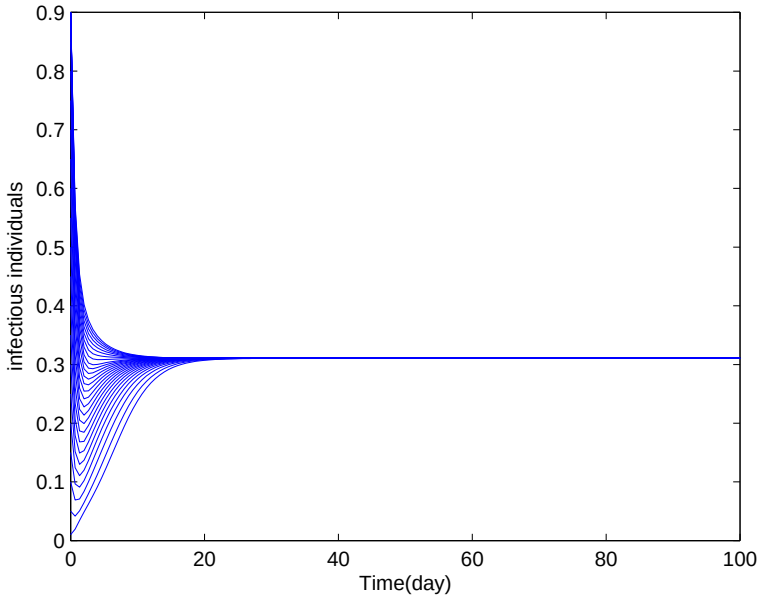

Figure 2.12: Infectious individuals approach unique endemic equilibrium (s_h^*, i_h^*, i_v^*) for different initial conditions even if the condition $\beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$ is voilated.i.e, $\delta_h, \frac{\gamma_h}{2} < \beta_1 < \frac{b_1}{2}$. The parameter values are $b_1 = 1, \beta_1 = 0.01, \beta_2 = 0.85, \beta_3 = 0.95, \gamma_h = 0.015, \delta_h = 0.009, \mu_v = 0.25, \mathfrak{R}_o = 3.16$.

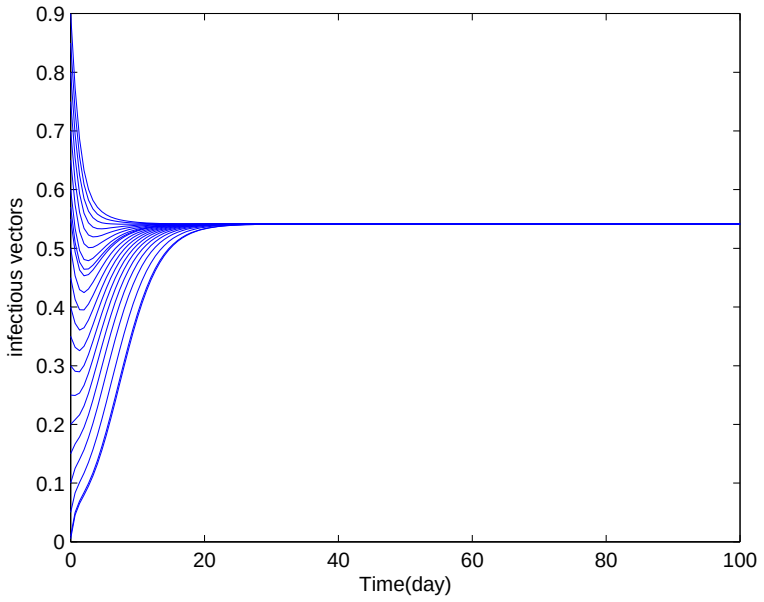

Figure 2.13: Infectious vectors approach unique endemic equilibrium (s_h^*, i_h^*, i_v^*) for different initial conditions even if the condition $\beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$ is voilated.i.e, $\delta_h, \frac{\gamma_h}{2} < \beta_1 < \frac{b_1}{2}$. The parameter values are $b_1 = 1, \beta_1 = 0.01, \beta_2 = 0.85, \beta_3 = 0.95, \gamma_h = 0.015, \delta_h = 0.009, \mu_v = 0.25, \mathfrak{R}_o = 3.16$.

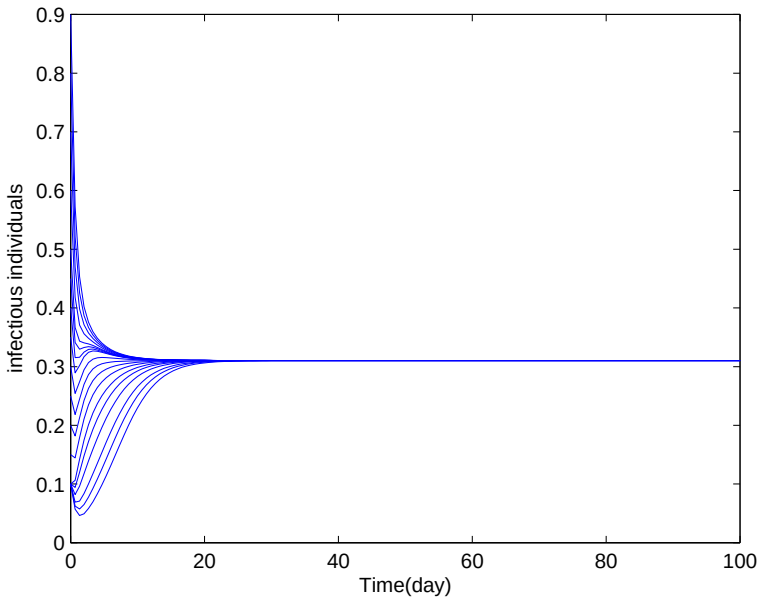

Figure 2.14: Infectious individuals approach unique endemic equilibrium (s_h^*, i_h^*, i_v^*) for different initial conditions even if the condition $\beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$ is voilated.i.e, $\frac{\gamma_h}{2} < \beta_1 = \delta_h < \frac{b_1}{2}$. The parameter values are $b_1 = 1, \beta_1 = 0.01, \beta_2 = 0.85, \beta_3 = 0.95, \gamma_h = 0.015, \delta_h = 0.01, \mu_v = 0.25, \mathfrak{R}_o = 3.16$.

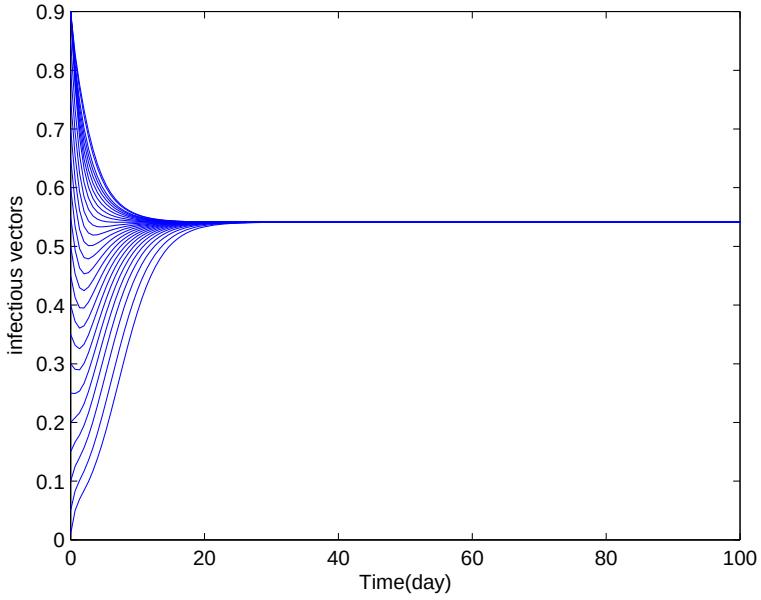

Figure 2.15: Infectious vectors approach unique endemic equilibrium (s_h^*, i_h^*, i_v^*) for different initial conditions even if the condition $\beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$ is voilated.i.e, $\frac{\gamma_h}{2} < \beta_1 = \delta_h < \frac{b_1}{2}$. The parameter values are $b_1 = 1, \beta_1 = 0.01, \beta_2 = 0.85, \beta_3 = 0.95, \gamma_h = 0.015, \delta_h = 0.009, \mu_v = 0.25, \mathfrak{R}_o = 3.16$.

It is also numerically shown that the same is true for the case $\delta_h, \frac{b_1}{2} < \beta_1 < \frac{\gamma_h}{2}$ or $\frac{b_1}{2} < \beta_1 = \delta_h < \frac{\gamma_h}{2}$ (Figs. 2.16, 2.17, 2.18, 2.19). This implies that the condition $\beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$ is weak for the global stability of unique "endemic" equilibrium.

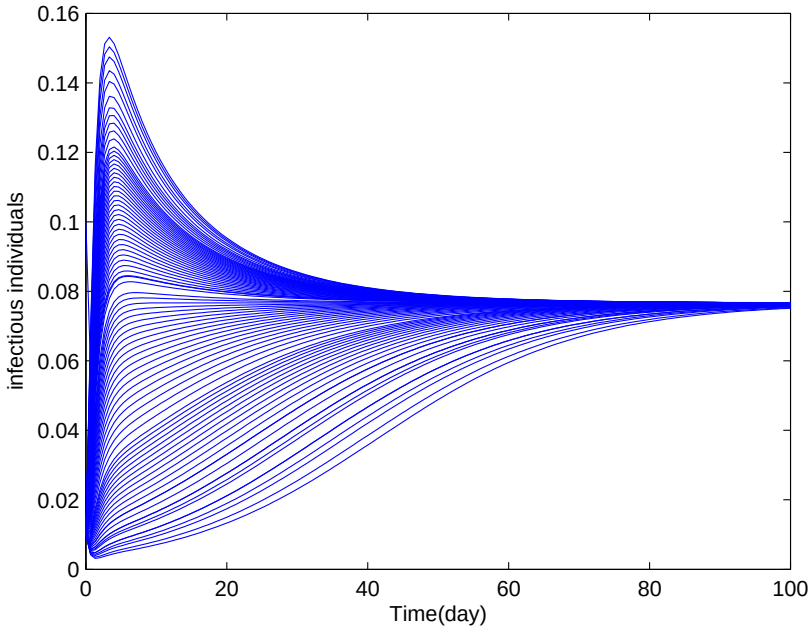

Figure 2.16: Infectious individuals approach unique endemic equilibrium (s_h^*, i_h^*, i_v^*) for different initial conditions if $\frac{b_1}{2} < \beta_1 < \frac{\gamma_h}{2}$. The parameter values are $b_1 = 0.78, \beta_1 = 0.4, \beta_2 = 0.65, \beta_3 = 0.55, \gamma_h = 0.8, \delta_h = 0.35, \mu_v = 0.15, \mathfrak{R}_o = 1.44$.

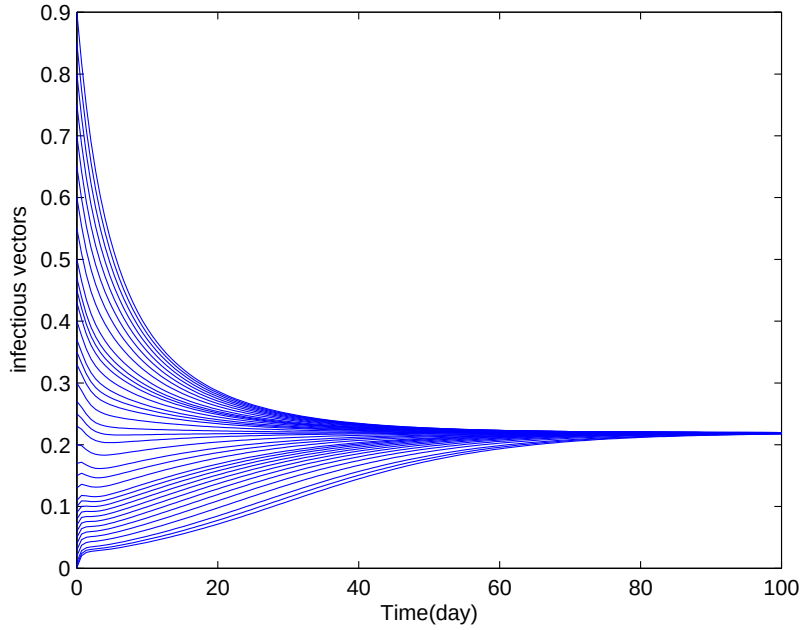

Figure 2.17: Infectious vectors approach unique endemic equilibrium (s_h^*, i_h^*, i_v^*) for different initial conditions if $\frac{b_1}{2} < \beta_1 < \frac{\gamma_h}{2}$. The parameter values are $b_1 = 0.78, \beta_1 = 0.4, \beta_2 = 0.65, \beta_3 = 0.55, \gamma_h = 0.8, \delta_h = 0.35, \mu_v = 0.15, \mathfrak{R}_o = 1.44$.

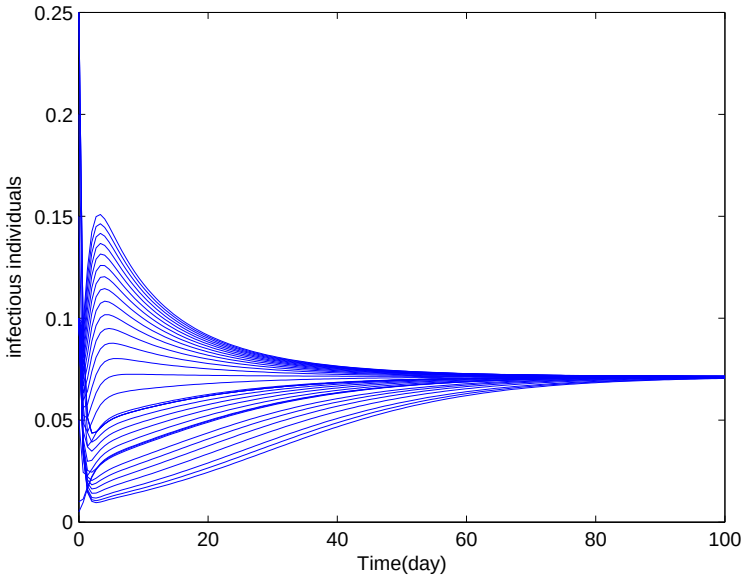

Figure 2.18: Infectious individuals approach unique endemic equilibrium (s_h^*, i_h^*, i_v^*) for different initial conditions if $\frac{b_1}{2} < \delta_h = \beta_1 < \frac{\gamma_h}{2}$. The parameter values are $b_1 = 0.78, \beta_1 = 0.4, \beta_2 = 0.65, \beta_3 = 0.55, \gamma_h = 0.8, \delta_h = 0.4, \mu_v = 0.15, \mathfrak{R}_o = 1.40$.

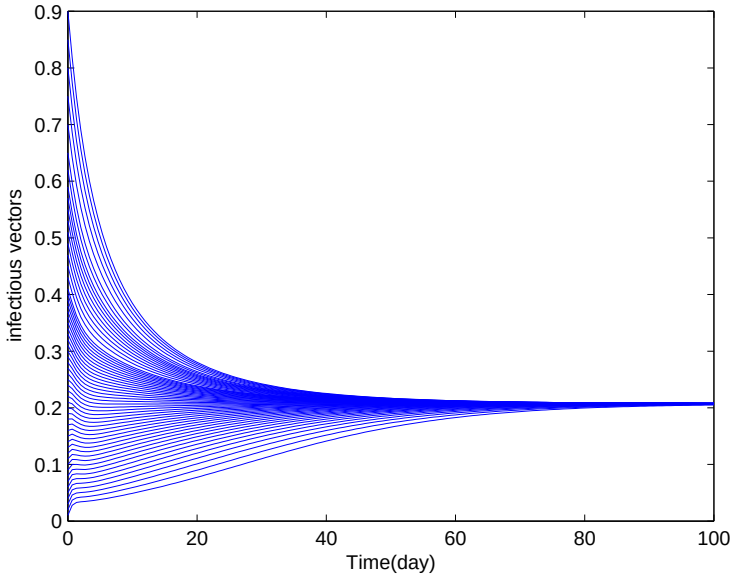

Figure 2.19: Infectious vectors approach unique endemic equilibrium (s_h^*, i_h^*, i_v^*) for different initial conditions if $\frac{b_1}{2} < \delta_h = \beta_1 < \frac{\gamma_h}{2}$. The parameter values are $b_1 = 0.78, \beta_1 = 0.4, \beta_2 = 0.65, \beta_3 = 0.55, \gamma_h = 0.8, \delta_h = 0.4, \mu_v = 0.15, \mathfrak{R}_o = 1.40$.

Chapter 3

Stability Analysis of Vector-Host Model with Latent Hosts and Vectors

Mathematical model discussed in this chapter is the extension of model (2.1.1). The following features are involved in the proposed model:

- The exposed class is introduced in human as well as in vector population.
- The recovered individuals do not acquire permanent immunity they again become susceptible after some time.

The global behavior of disease free equilibrium and the "endemic" equilibrium of the given model is discussed. The following techniques are used to prove the global stability.

- By constructing Lyapunov functional it is proved that the disease-free equilibrium is globally asymptotically stable whenever $\mathfrak{R}_o \leq 1$.
- By using compound matrices and geometric approach it is shown that the disease persists at the "endemic" level if $\mathfrak{R}_o > 1$.

3.1 Model Description and Dimensionless Formulation

The total host population $N_h(t)$, described by SEIS model, is partitioned into three distinct compartments, susceptibles $S_h(t)$, exposed or infected $E_h(t)$ and infectious $I_h(t)$. The vector population $N_v(t)$ is described by SEI model and it is also divided into three subclases namely susceptible $S_v(t)$, exposed $E_v(t)$ and infectious $I_v(t)$ classes. The schematic

diagram of the considered model is as follows:

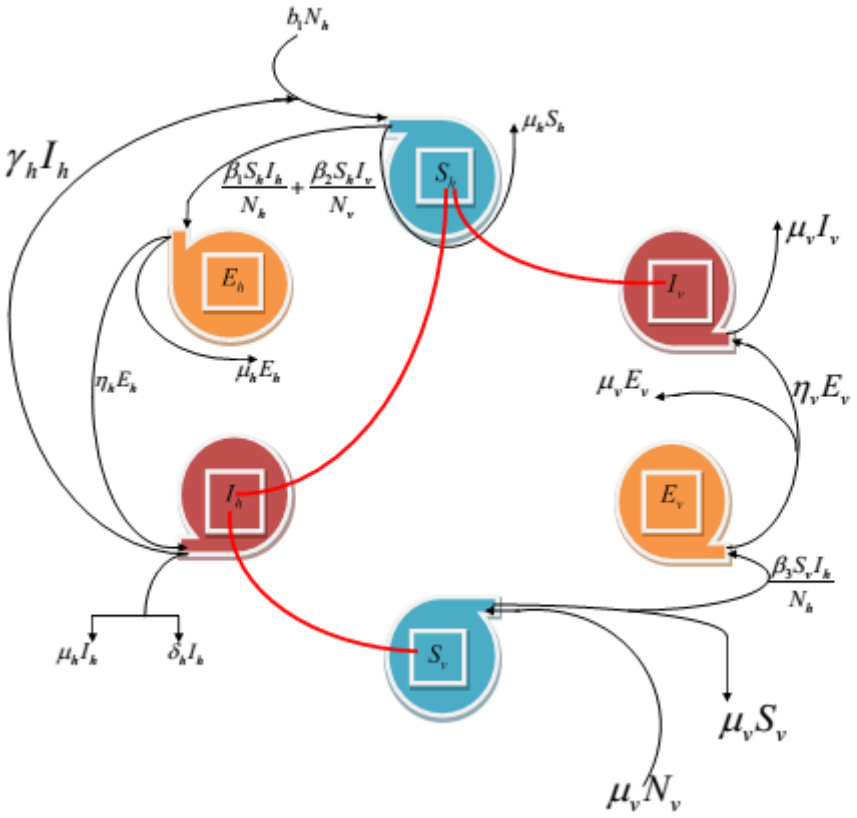

Figure 3.1: Flow diagram of Vector-Host Model with exposed class

The analytical expression of the above model shown in flow diagram is given by

$$\begin{aligned}
\frac{dS_h}{dt} &= b_1 N_h - \beta_1 \frac{S_h I_h}{N_h} - \beta_2 \frac{S_h I_v}{N_v} - \mu_h S_h + \gamma_h I_h, \\
\frac{dE_h}{dt} &= \beta_1 \frac{S_h I_h}{N_h} + \beta_2 \frac{S_h I_v}{N_v} - \eta_h E_h - \mu_h E_h, \\
\frac{dI_h}{dt} &= \eta_h E_h - \gamma_h I_h - \mu_h I_h - \delta_h I_h \\
\frac{dS_v}{dt} &= \mu_v N_v - \beta_3 \frac{S_v I_h}{N_h} - \mu_v S_v, \\
\frac{dE_v}{dt} &= \beta_3 \frac{S_v I_h}{N_h} - \eta_v E_v - \mu_v E_v \\
\frac{dI_v}{dt} &= \eta_v E_v - \mu_v I_v.
\end{aligned} \tag{3.1.1}$$

In the above, b_1 is the per capita birth rate of humans that are assumed to be susceptible, μ_h is natural mortality rate of humans and δ_h is the disease induced death rate. Susceptible humans can be infected through contact with an infected individual and the effective infection rate is represented by β_1. The infectious individuals do not acquire permanent immunity and become susceptible again at the rate γ_h. If the vector is infectious, then the average number of contacts per day that results in infection is β_2. Similarly the effective contact rate between susceptible vectors and infectious humans is β_3. Newly-infected individuals develop clinical symptoms of the disease and move to the infectious class at the rate η_h and exposed vectors progress to the infectious class at the rate η_v. We assume that the birth and death rates of the vector population is equal to μ_v so that it has constant size.

Taking

$$s_h = \frac{S_h}{N_h}, e_h = \frac{E_h}{N_h}, i_h = \frac{I_h}{N_h}, s_v = \frac{S_v}{N_v}, e_v = \frac{E_v}{N_v}, i_v = \frac{I_v}{N_v}, \tag{3.1.2}$$

we arrive at the following normalized model

$$\begin{aligned}
\frac{ds_h}{dt} &= b_1(1-s_h) - \beta_1 s_h i_h - \beta_2 s_h i_v + \gamma_h i_h + \delta_h s_h i_h, \\
\frac{de_h}{dt} &= \beta_1 s_h i_h + \beta_2 s_h i_v - \eta_h e_h - b_1 e_h + \delta_h e_h i_h, \\
\frac{di_h}{dt} &= \eta_h e_h - \gamma_h i_h - \delta_h i_h - b_1 i_h + \delta_h {i_h}^2, \\
\frac{ds_v}{dt} &= \mu_v(1-s_v) - \beta_3 s_v i_h, \\
\frac{de_v}{dt} &= \beta_3 s_v i_h - \eta_v e_v - \mu_v e_v, \\
\frac{di_v}{dt} &= \eta_v e_v - \mu_v i_v.
\end{aligned} \tag{3.1.3}$$

Since

$$s_h + e_h + i_h = 1, s_v + e_v + i_v = 1, \tag{3.1.4}$$

we can study the following subsystem

$$\begin{aligned}
\frac{de_h}{dt} &= \beta_1(1-e_h-i_h)i_h + \beta_2(1-e_h-i_h)i_v - \eta_h e_h - b_1 e_h + \delta_h e_h i_h,\\
\frac{di_h}{dt} &= \eta_h e_h - \gamma_h i_h - \delta_h i_h - b_1 i_h + \delta_h {i_h}^2,\\
\frac{de_v}{dt} &= \beta_3(1-e_v-i_v)i_h - \eta_v e_v - \mu_v e_v\\
\frac{di_v}{dt} &= \eta_v e_v - \mu_v i_v.
\end{aligned} \tag{3.1.5}$$

This system is defined in the subset $\Gamma \times [0,\infty)$ of ${R^5}_+$, where $\Gamma = \{e_h, i_h, e_v, i_v : 0 \leq e_h, i_h, e_v, i_v \leq 1, 0 \leq e_h + i_h \leq 1, 0 \leq e_v + i_v \leq 1\}$ and the original quantities can be determined from the proportions through (3.1.3) and (3.1.4). The Jacobian matrix at disease free equilibrium DFE E_0 given by (e_h, i_h, e_v, i_v)=$(0,0,0,0)$ is

$$J = \begin{pmatrix}
-(b_1+\eta_h) & \beta_1 & 0 & \beta_2\\
\eta_h & -(b_1+\gamma_h+\delta_h) & 0 & 0\\
0 & \beta_3 & -(\eta_v+\mu_v) & 0\\
0 & 0 & \eta_v & -\mu_v
\end{pmatrix}.$$

The characteristic equation for the above Jacobian matrix is given by

$$f(\lambda) = \lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0,$$

where

$$\begin{aligned}
a_1 &= (2\mu_v + \eta_v) + (2b_1 + \eta_h + \gamma_h + \delta_h),\\
a_2 &= (b_1+\eta_h)(b_1+\gamma_h+\delta_h) - \beta_1\eta_h + (2\mu_v+\eta_v)(2b_1+\eta_h+\gamma_h+\delta_h) + \mu_v(\eta_v+\mu_v),\\
a_3 &= (2\mu_v+\eta_v)((b_1+\eta_h)(b_1+\gamma_h+\delta_h) - \beta_1\eta_h) + \mu_v(\eta_v+\mu_v)(2b_1+\eta_h+\gamma_h+\delta_h),\\
a_4 &= \mu_v(\eta_v+\mu_v)(b_1+\eta_h)(b_1+\gamma_h+\delta_h)(1-\mathfrak{R}_o),
\end{aligned} \tag{3.1.6}$$

and

$$\mathfrak{R}_o = \frac{\beta_1\eta_h}{Q_1Q_3} + \frac{\beta_2\beta_3\eta_h\eta_v}{\mu_v Q_1 Q_2 Q_3}.$$

In the definition of $\Re_o$, we have used the symbols $Q_1 = b_1 + \eta_h$, $Q_2 = \eta_v + \mu_v$, $Q_3 = b_1 + \gamma_h + \delta_h$.

The four eigenvalues of the above Jacobian matrix have negative real parts if they satisfy the Routh-Hurwitz Criteria [45], i.e. $a_i > 0$ for $i = 1, 2, 3, 4$, with $a_1a_2a_3 > a_3^2 + a_1^2a_4$. For $\Re_o < 1$, $(b_1 + \eta_h)(b_1 + \gamma_h + \delta_h) - \beta_1\eta_h > 0$ and so $a_i > 0$ for $i = 1, 2, 3, 4$. It can also be easily verified that $a_1a_2a_3 > a_3^2 + a_1^2a_4$. Thus all the eigenvalues of the above characteristic equation have negative real parts if and only if $\Re_o < 1$, which shows that the disease-free equilibrium E_0 is locally asymptotically stable.

Remark: If $\Re_o > 1$, we have $f(0) < 0$ and $f(\lambda) = +\infty$ as $\lambda \to +\infty$. Thus there exists at least one $\lambda^* > 0$ such that $f(\lambda^*) = 0$ which proves instability of disease free equilibrium.

3.2 Endemic Equilibria

Let $E^* = (e_h^*, i_h^*, e_v^*, i_v^*)$ represents any arbitrary endemic equilibrium of the model (3.1.3). Solving the equations of the system (3.1.3) at steady state gives

$$\begin{aligned} e_h^* &= \frac{(Q_3 - \delta_h i_h^*)i_h^*}{\eta_h}, \\ e_v^* &= \frac{\beta_3 d i_v^*}{Q_2(\beta_3 i_h^* + \mu_v)}, \\ i_v^* &= \frac{\beta_3\eta_v i_h^*}{Q_2(\beta_3 i_h^* + \mu_v)}, \end{aligned} \tag{3.2.1}$$

where i_h^* is any root of the following cubic equation

$$g(i_h^*) = m_3 {i_h^*}^3 + m_2 {i_h^*}^2 + m_1 i_h^* + m_0 = 0, \tag{3.2.2}$$

with

$$\begin{aligned} m_3 &= Q_2\beta_3\delta_h(\beta_1 - \delta_h), \\ m_2 &= \beta_1 Q_2\mu_v\delta_h + \beta_2\beta_3\eta_v\delta_h + b_1Q_2\delta_h\beta_3 - (\beta_1 - \delta_h)(\eta_h Q_2\beta_3 + Q_2Q_3\beta_3), \\ m_1 &= (\beta_3 Q_2\eta_h - \eta_h Q_2\mu_v - Q_2Q_3\mu_v)(\beta_1 - \delta_h) - b_1Q_2Q_3\beta_3 - \beta_2\beta_3\eta_v\eta_h - \beta_2\beta_3\eta_v Q_3 + b_1Q_2\delta_h\mu_v, \\ m_0 &= \mu_v Q_1Q_2Q_3(\Re_o - 1). \end{aligned} \tag{3.2.3}$$

Assuming $\Re_o > 1$,

(1) If $\beta_1 > \delta_h$, then $m_3 > 0$, we have $g(-\infty) < 0$, $g(\infty) > 0$ and $g(0) = m_0 > 0$. Further,

$g(1) < 0$ if $(\frac{b_1}{2} > \beta_1 > \delta_h + \eta_h)$, so there exists **unique** $i_h^* \in (0,1)$ such that $g(i_h^*) = 0$(see Fig. 3.2).

(2) If $\beta_1 = \delta_h$, then $m_3 = 0$ and $g(i_h^*) = m_2 {i_h^*}^2 + m_1 i_h^* + m_0$, where $m_2 = \beta_1 Q_2 \mu_v \delta_h + \beta_2 \beta_3 \eta_v \delta_h + b_1 Q_2 \delta_h \beta_3 > 0$.

$g(-\infty) > 0$, $g(\infty) > 0$ and $g(0) = m_0 > 0$. Moreover, $g(1) < 0$ if $(\frac{b_1}{2} > \beta_1 = \delta_h)$. Therefore, there exists **unique** $i_h^* \in (0,1)$ such that $g(i_h^*) = 0$(see Fig. 3.3).

(3) If $\beta_1 < \delta_h$, then $m_2 > 0$, $m_3 < 0$, we have $g(-\infty) > 0$, $g(\infty) < 0$ and $g(0) = m_0 > 0$. Thus there exists at least one positive root or three positive roots according as m_1 positive or negative. We know that $g(i_h^*) = 0$ has three real roots if and only if

$$\frac{a^2}{4} + \frac{b^3}{27} \leq 0,$$

where

$$a = \frac{m_1}{m_3} - \frac{(m_2)^2}{3(m_3)^2}, \qquad b = \frac{m_0}{m_3} - \frac{m_1 m_2}{3(m_3)^2} + \frac{2(m_2)^3}{27(m_3)^3},$$

or

$$\hat{\mathfrak{R}_o} = \frac{18 m_0 m_1 m_2 m_3 - 4 m_0 (m_2)^3 - 4 (m_1)^3 m_3 + (m_1)^2 (m_2)^2}{27 (m_0)^2 (m_3)^2} \geq 1.$$

If $\hat{\mathfrak{R}_o} < 1$, there is unique i_h^* such that $g(i_h^*) = 0$ in the feasible interval.

If $\hat{\mathfrak{R}_o} > 1$, there are three different real roots for $g(i_h^*) = 0$ say $i_{h1}^*, i_{h2}^*, i_{h3}^* (i_{h1}^* < i_{h2}^* < i_{h3}^*)$.

$g'(i_h^*) = 3 m_3 {i_h^*}^2 + 2 m_2 i_h^* + m_1$.

The three different real roots for $g(i_h^*) = 0$ are in the feasible interval if and only if the following inequalities are satisfied

$$\begin{aligned} 0 &< \frac{-m_2}{3m_3} < 1, \\ g'(0) &= m_1 < 0, \\ g'(1) &= 3m_3 + 2m_2 + m_1 < 0. \end{aligned} \tag{3.2.4}$$

If $\hat{\mathfrak{R}_o} = 1$, there are three real roots for $g(i_h^*) = 0$, in which at least two are identical. Similarly, if inequalities (3.2.4) are satisfied, then there are three real roots for $g(i_h^*) = 0$ in the feasible interval, say $i_{h1}^*, i_{h2}^*, i_{h3}^* (i_{h1}^* = i_{h2}^*)$.

Assume that $\mathfrak{R}_o = 1$.

(1) If $\beta_1 = \delta_h$, then $m_3 = 0$ and (3.2.2) reduces to $i_h^* \bar{g}(i_h^*) = 0$, where $\bar{g}(i_h^*) = (m_2 i_h^* + m_1)$. This implies that $i_h^* = 0$ or $i_h^* = \frac{-m_1}{m_2}$, which is positive but it lies outside the interval $(0,1)$ if $(\frac{b_1}{2} > \beta_1 = \delta_h)$ because $\bar{g}(1) = (m_2 + m_1)$.

(2) If $\beta_1 > \delta_h$, then $m_3 > 0$, we have $i_h^* (m_3 {i_h^*}^2 + m_2 i_h^* + m_1) = 0$ which implies that $i_h^* = 0$

or i_h^* is the solution of the equation

$$\tilde{g}(i_h^*) = m_3 {i_h^*}^2 + m_2 i_h^* + m_1 = 0.$$

$\tilde{g}(-\infty) > 0$, $\tilde{g}(\infty) > 0$, $\tilde{g}(0) = m_1 < 0$ and $\tilde{g}(1) < 0$ if $(\frac{b_1}{2} > \beta_1 > \delta_h + \eta_h)$. Therefore, there exists no i_h^* such that $\tilde{g}(i_h^*) = 0$ in the interval $(0, 1)$ if $(\frac{b_1}{2} > \beta_1 > \delta_h + \eta_h)$. We summarize the discussion below.

Theorem 3.2.1. *Suppose that* $\frac{b_1}{2} > \beta_1 > \delta_h + \eta_h$ *or* $\frac{b_1}{2} > \beta_1 = \delta_h$. *There is always a disease "free" equilibrium for system (3.1.5); if* $\mathfrak{R}_\circ > 1$, *then there is a unique "endemic" equilibrium* $E^*(s_h^*, i_h^*, i_v^*)$ *with coordinates satisfying (3.2.1) and (3.2.2) besides the disease "free" equilibrium.*

3.3 Global Dynamics

3.3.1 Global Stability of Disease-"Free" Equilibrium

In this subsection, we analyze the global behavior of the equilibria for system (3.1.3). The following theorem provides the global property of the disease-free equilibrium E_0 of the system.

Theorem 3.3.1. *If* $\mathfrak{R}_\circ \leq 1$, *then the infection-free equilibrium* E_0 *is globally asymptotically stable in the interior of* Γ.

Proof. To establish the global stability of the disease-free equilibrium, we construct the following Lyapunov function:

$$L(t) = e_h(t) + i_h(t) + \frac{\beta_2}{\mu_v} e_v(t) + \frac{\beta_2}{\mu_v} i_v(t).$$

Calculating the time derivative of L along (3.1.5), we obtain

$$
\begin{aligned}
L'(t) &= e_h'(t) + i_h'(t) + \frac{\beta_2}{\mu_v} e_v'(t) + \frac{\beta_2}{\mu_v} i_v'(t) \\
&= \beta_1(1 - e_h - i_h)i_h + \beta_2(1 - e_h - i_h)i_v - \eta_h e_h - b_1 e_h + \delta_h e_h i_h + \eta_h e_h - \gamma_h i_h - \delta_h i_h \\
&\quad -b_1 i_h + \delta_h {i_h}^2 + \frac{\beta_2}{\mu_v}[\beta_3(1 - e_v - i_v)i_h - \eta_v e_v - \mu_v e_v] + \frac{\beta_2}{\mu_v}[\eta_v e_v - \mu_v i_v] \\
&= \beta_1 i_h - \beta_1 e_h i_h - \beta_1 {i_h}^2 + \beta_2 i_v - \beta_2 e_h i_v - \beta_2 i_h i_v - \eta_h e_h - b_1 e_h + \delta_h e_h i_h + \eta_h e_h - \gamma_h i_h \\
&\quad -\delta_h i_h - b_1 i_h + \delta_h {i_h}^2 + \frac{\beta_2}{\mu_v}[\beta_3 i_h - \beta_3 e_v i_h - \beta_3 i_v i_h - \eta_v e_v - \mu_v e_v] + \frac{\beta_2}{\mu_v}[\eta_v e_v - \mu_v i_v] \\
&= \beta_1 i_h - (\beta_1 - \delta_h) e_h i_h - (\beta_1 - \delta_h) {i_h}^2 + \beta_2 i_v - \beta_2 e_h i_v - \beta_2 i_h i_v - b_1 e_h - Q_3 i_h + \\
&\quad \frac{\beta_2\beta_3}{\mu_v} i_h - \frac{\beta_2\beta_3}{\mu_v} e_v i_h - \frac{\beta_2\beta_3}{\mu_v} i_v i_h - \frac{\beta_2}{\mu_v}\eta_v e_v - \beta_2 e_v + \frac{\beta_2}{\mu_v}\eta_v e_v - \beta_2 i_v \\
&= (\beta_1 + \frac{\beta_2\beta_3}{\mu_v} - Q_3) i_h - (\beta_1 - \delta_h) e_h i_h - (\beta_1 - \delta_h) {i_h}^2 - \beta_2 e_h i_v - \beta_2 i_h i_v - b_1 e_h - \frac{\beta_2\beta_3}{\mu_v} e_v i_h \\
&\quad -\frac{\beta_2\beta_3}{\mu_v} i_v i_h - \beta_2 e_v \\
&= Q_3(\frac{\beta_1}{Q_3} + \frac{\beta_2\beta_3}{\mu_v Q_3} - 1) i_h - (\beta_1 - \delta_h) e_h i_h - (\beta_1 - \delta_h) {i_h}^2 - \beta_2 e_h i_v - \beta_2 i_h i_v - b_1 e_h \\
&\quad -\frac{\beta_2\beta_3}{\mu_v} e_v i_h - \frac{\beta_2\beta_3}{\mu_v} i_v i_h - \beta_2 e_v.
\end{aligned}
$$

We can see that L' is negative if $\frac{\beta_1}{Q_3} + \frac{\beta_2\beta_3}{\mu_v Q_3} < 1$, which implies $\frac{\beta_1\eta_h}{Q_1 Q_3} + \frac{\beta_2\beta_3\eta_h\eta_v}{\mu_v Q_1 Q_2 Q_3} < 1$. Again $L' = 0$ if and only if $e_h = 0, i_h = 0$ and $e_v = 0$. Therefore the largest compact invariant set in $\{(e_h, i_h, e_v, i_v) \in \Gamma, L' = 0\}$, when $\mathfrak{R}_o \leq 1$, is the singelton $\{E_0\}$. Hence, LaSalle's invariance principle [46] implies that "E_0" is globally asymptotically stable in Γ. This completes the proof.

3.3.2 Global Stability of Endemic Equilibrium

Here we apply the result given on page 59 of [50] to establish the global asymptotic stability of the unique "endemic" equilibrium $E^*(s_h^*, i_h^*, i_v^*)$. The Lozinskiĭ measure for an $n \times n$ matrix A is defined as

$$\tilde{\mu}(A) = inf\{\rho : D_+\|Z\| \leq \rho\|Z\| \ for\ all\ solutions\ of\ Z' = AZ\},$$

where D_+ is the right hand derivative [51]. The unique endemic equilibrium is globally asymptotically stable if there exists a norm on R^6 which is associated with the Lozinskiĭ measure which satisfies $\tilde{\mu}(A) < 0$ for all $x \in int(\Gamma)$ if $\Re_o > 1$. The Jacobian matrix at endemic equilibrium point is given by

$$J = \begin{pmatrix} g_{11} & \beta_1(1-e_h-i_h) - \beta_1 i_h - \beta_2 i_v + \delta_h e_h & 0 & \beta_2(1-e_h-i_h) \\ \eta_h & -(b_1+\gamma_h+\delta_h) + 2\delta_h i_h & 0 & 0 \\ 0 & \beta_3(1-e_v-i_v) & -\beta_3 i_h - (\eta_v+\mu_v) & -\beta_3 i_h \\ 0 & 0 & \eta_v & -\mu_v \end{pmatrix}$$

where $g_{11} = -\beta_1 i_h - \beta_2 i_v - (b_1 + \eta_h - \delta_h i_h)$.

The second compound matrix [52] is

$$J^{[2]} = \begin{pmatrix} j_{11} & 0 & 0 & 0 & -\beta_2(1-e_h-i_h) & 0 \\ \beta_3(1-e_v-i_v) & j_{22} & -\beta_3 i_h & j_{24} & 0 & -\beta_2(1-e_h-i_h) \\ 0 & \eta_v & j_{33} & 0 & j_{35} & 0 \\ 0 & \eta_h & 0 & j_{44} & -\beta_3 i_h & 0 \\ 0 & 0 & \eta_h & \eta_v & j_{55} & 0 \\ 0 & 0 & 0 & 0 & \beta_3(1-e_v-i_v) & j_{66} \end{pmatrix}$$

where

$j_{11} = -\beta_1 i_h - \beta_2 i_v - (b_1 + \eta_h - \delta_h i_h) - (b_1 + \gamma_h + \delta_h) + 2\delta_h i_h$

$j_{22} = -\beta_1 i_h - \beta_2 i_v - (b_1 + \eta_h - \delta_h i_h) - \beta_3 i_h - (\eta_v + \mu_v)$

$j_{33} = -\beta_1 i_h - \beta_2 i_v - (b_1 + \eta_h - \delta_h i_h) - \mu_v$

$j_{44} = -(b_1 + \gamma_h + \delta_h) + 2\delta_h i_h - \beta_3 i_h - (\eta_v + \mu_v)$

$j_{55} = -(b_1 + \gamma_h + \delta_h) + 2\delta_h i_h - \mu_v$

$j_{66} = -\beta_3 i_h - (\eta_v + \mu_v) - \mu_v$

$j_{24} = \beta_1(1 - e_h - i_h) - \beta_1 i_h - \beta_2 i_v + \delta_h e_h$

$j_{35} = \beta_1(1 - e_h - i_h) - \beta_1 i_h - \beta_2 i_v + \delta_h e_h.$

Let $P = diag(\frac{1}{i_h}, \frac{1}{i_v}, \frac{1}{i_v}, \frac{1}{i_v}, \frac{1}{i_v}, \frac{1}{i_v})$. Then we have

$$K = P_f P^{-1} + P J^{[2]} P^{-1}$$

where

$$K = \begin{pmatrix} j_{11} - \frac{i_h'}{i_h} & 0 & 0 & 0 & -\beta_2(1-e_h-i_h)\frac{i_v}{i_h} & 0 \\ \beta_3(1-e_v-i_v)\frac{i_h}{i_v} & j_{22} - \frac{i_v'}{i_v} & -\beta_3 i_h & j_{24} & 0 & -\beta_2(1-e_h-i_h) \\ 0 & \eta_v & j_{33} - \frac{i_v'}{i_v} & 0 & j_{35} & 0 \\ 0 & \eta_h & 0 & j_{44} - \frac{i_v'}{i_v} & -\beta_3 i_h & 0 \\ 0 & 0 & \eta_h & \eta_v & j_{55} - \frac{i_v'}{i_v} & 0 \\ 0 & 0 & 0 & 0 & \beta_3(1-e_v-i_v) & j_{66} - \frac{i_v'}{i_v} \end{pmatrix}.$$

Let $Z = (Z_1, Z_2, Z_3, Z_4, Z_5, Z_6)^T$ be the solution of the linear homogeneous system $\frac{dZ}{dt} = KZ$, where

$$\begin{aligned} Z_1{}' &= (j_{11} - \frac{i_h'}{i_h})Z_1 + (-\beta_2(1-e_h-i_h)\frac{i_v}{i_h})Z_5, \\ Z_2{}' &= (\beta_3(1-e_v-i_v)\frac{i_h}{i_v})Z_1 + (j_{22} - \frac{i_v'}{i_v})Z_2 - \beta_3 i_h Z_3 + j_{24}Z_4 - \beta_2(1-e_h-i_h)Z_6, \\ Z_3{}' &= \eta_v Z_2 + (j_{33} - \frac{i_v'}{i_v})Z_3 + j_{35}Z_5 \\ Z_4{}' &= \eta_h Z_2 + (j_{44} - \frac{i_v'}{i_v})Z_4 - \beta_3 i_h Z_5, \\ Z_5{}' &= \eta_h Z_3 + \eta_v Z_4 + (j_{55} - \frac{i_v'}{i_v})Z_5 \\ Z_6{}' &= \beta_3(1-e_v-i_v)Z_5 + (j_{66} - \frac{i_v'}{i_v})Z_6. \end{aligned}$$

It can be easily seen from (3.1.5) that

$$\begin{aligned}
\frac{e_h{}'}{e_h} &= \beta_1(1-e_h-i_h)\frac{i_h}{e_h}+\beta_2(1-e_h-i_h)\frac{i_v}{e_h}-(\eta_h+b_1-\delta_h i_h),\\
\frac{i_h{}'}{i_h} &= \eta_h\frac{e_h}{i_h}-\gamma_h-\delta_h-b_1+\delta_h i_h,\\
\frac{e_v{}'}{e_v} &= \beta_3(1-e_v-i_v)\frac{i_h}{e_v}-\eta_v-\mu_v\\
\frac{i_v{}'}{i_v} &= \eta_v\frac{e_v}{i_v}-\mu_v.
\end{aligned} \tag{3.3.1}$$

Theorem 3.3.2. *Suppose that $\mathfrak{R}_\circ > 1$. The unique endemic equilibrium E^* is globally asymptotically stable in Γ^o if the following inequalities are satisfied:*

$$\begin{aligned}
b_1 &> \delta_h+\eta_h,\\
\beta_3 &< \eta_v+\mu_v,\\
b_1+\mu_v &> \beta_1+\eta_v.
\end{aligned} \tag{3.3.2}$$

Proof. We consider the following norms on Z [53]

$$\|Z\| = \begin{cases} max\{|Z_1|, i_v(|Z_2|+|Z_3|), i_v(|Z_4|+|Z_5|), i_v|Z_6|\}, & \text{if } sgn(Z_1)=sgn(Z_2)=sgn(Z_3), \\ & sgn(Z_4)=sgn(Z_5)=sgn(Z_6) \\ max\{i_h|Z_1|, |Z_2|+|Z_3|, |Z_4|+|Z_5|, |Z_6|\}, & \text{if } -sgn(Z_1)=sgn(Z_2)=sgn(Z_3), \\ & sgn(Z_4)=sgn(Z_5)=sgn(Z_6) \\ max\{|Z_1|, i_v|Z_2|, |Z_3|, |Z_4|+|Z_5|, |Z_6|\}, & \text{if } sgn(Z_1)=-sgn(Z_2)=sgn(Z_3), \\ & sgn(Z_4)=sgn(Z_5)=sgn(Z_6) \\ max\{|Z_1|, i_v|Z_2|, |Z_3|, |Z_4|+|Z_5|, |Z_6|\}, & \text{if } sgn(Z_1)=sgn(Z_2)=-sgn(Z_3), \\ & sgn(Z_4)=sgn(Z_5)=sgn(Z_6) \\ max\{|Z_1|, i_v(|Z_2|+|Z_3|), |Z_4|, |Z_5|, |Z_6|\}, & \text{if } sgn(Z_1)=sgn(Z_2)=sgn(Z_3), \\ & -sgn(Z_4)=sgn(Z_5)=sgn(Z_6) \\ max\{i_h|Z_1|, i_v|Z_2|, i_v|Z_3|, i_v|Z_4|, |Z_5|, |Z_6|\}, & \text{if } sgn(Z_1)=sgn(Z_2)=sgn(Z_3), \\ & sgn(Z_4)=-sgn(Z_5)=sgn(Z_6) \\ max\{|Z_1|, i_v(|Z_2|+|Z_3|), i_v(|Z_4|+|Z_5|), |Z_6|\}, & \text{if } sgn(Z_1)=sgn(Z_2)=sgn(Z_3), \\ & sgn(Z_4)=sgn(Z_5)=-sgn(Z_6). \end{cases} \tag{3.3.3}$$

We discuss four cases here.

Case1. $sgn(Z_1)=sgn(Z_2)=sgn(Z_3), sgn(Z_4)=sgn(Z_5)=sgn(Z_6)$

Then $\|Z\| = max\{|Z_1|, i_v(|Z_2|+|Z_3|), i_v(|Z_4|+|Z_5|), i_v|Z_6|\}$.

Case1a. $|Z_1| > \{i_v(|Z_2|+|Z_3|), i_v(|Z_4|+|Z_5|), i_v|Z_6|\}$.

Then $\|Z\| = |Z_1| = Z_1$ and

$$\begin{aligned} D_+\|Z\| &= Z_1' \\ &= (j_{11} - \frac{i_h'}{i_h})Z_1 - \beta_2(1-e_h-i_h)\frac{i_v}{i_h}Z_5 \\ &= (-\beta_1 i_h - \beta_2 i_v - (b_1+\eta_h-\delta_h i_h) - (b_1+\gamma_h+\delta_h) + 2\delta_h i_h - \eta_h\frac{e_h}{i_h} + (b_1+\gamma_h+\delta_h) \end{aligned}$$

$$
\begin{aligned}
& -\delta_h i_h)Z_1 - \beta_2(1-e_h-i_h)\frac{i_v}{i_h}Z_5 \\
\leq\ & (-(\beta_1-\delta_h)i_h - \beta_2 i_v - (b_1-\delta_h) - (\eta_h i_h + \eta_h\frac{e_h}{i_h}))|Z_1| - \beta_2(1-e_h-i_h)\frac{i_v}{i_h}|Z_5| \\
<\ & (-(\beta_1-\delta_h)i_h - \beta_2 i_v - (b_1-\delta_h) - (\eta_h i_h + \eta_h\frac{e_h}{i_h}))|Z_1| \\
=\ & -\rho_1\|Z\|
\end{aligned}
$$

where

$$\rho_1 = (\beta_1-\delta_h)i_h + \beta_2 i_v + (b_1-\delta_h) + (\eta_h i_h + \eta_h\frac{e_h}{i_h}).$$

Case1b. $i_v(|Z_2|+|Z_3|) > \{|Z_1|, i_v(|Z_4|+|Z_5|), i_v|Z_6|\}$. Then
$\|Z\| = i_v(|Z_2|+|Z_3|) = i_v(Z_2+Z_3)$ and

$$
\begin{aligned}
D_+\|Z\| &= i_v(\frac{i_v'}{i_v}Z_2 + \frac{i_v'}{i_v}Z_3 + Z_2' + Z_3') \\
&= i_v[(\beta_3(1-e_v-i_v)\frac{i_h}{i_v})Z_1 + j_{22}Z_2 - \beta_3 i_h Z_3 + j_{24}Z_4 - \beta_2(1-e_h-i_h)Z_6 + \eta_v Z_2 + j_{33}Z_3 \\
&\quad +j_{35}Z_5] \\
&= \beta_3 i_h(1-e_v-i_v)|Z_1| + j_{22}i_v|Z_2| - \beta_3 i_h i_v|Z_3| + j_{24}i_v|Z_4| - \beta_2 i_v(1-e_h-i_h)|Z_6| \\
&\quad +\gamma_v i_v|Z_2| + j_{33}i_v|Z_3| + j_{35}i_v|Z_5| \\
&< \beta_3 i_h(1-e_v-i_v)|Z_1| + j_{22}i_v|Z_2| - \beta_3 i_h i_v|Z_3| + j_{24}i_v|Z_4| + \eta_v i_v|Z_2| + j_{33}i_v|Z_3 i_v \\
&\quad +j_{35}i_v|Z_5| \\
&= \beta_3 i_h|Z_1| - \beta_3 i_h(e_v+i_v)|Z_1| + (-\beta_1 i_h - \beta_2 i_v - (b_1+\eta_h-\delta_h i_h) - \beta_3 i_h - (\eta_v+\mu_v))i_v|Z_2| \\
&\quad -\beta_3 i_h i_v|Z_3| + (\beta_1(1-e_h-i_h) - \beta_1 i_h - \beta_2 i_v + \delta_h e_h)i_v|Z_4| + \eta_v i_v|Z_2| \\
&\quad +(-\beta_1 i_h - \beta_2 i_v - (b_1+\eta_h-\delta_h i_h) - \mu_v)i_v|Z_3| \\
&\quad +(\beta_1(1-e_h-i_h) - \beta_1 i_h - \beta_2 i_v + \delta_h e_h)i_v|Z_5| \\
&< (\beta_3 i_h - \beta_1 i_h - \beta_2 i_v - (b_1+\eta_h-\delta_h i_h) - \beta_3 i_h - (\eta_v+\mu_v) + \eta_v)i_v|Z_2| \\
&\quad +(\beta_1 + (\beta_1-\delta_h)e_h - \beta_1 i_h - \beta_1 i_h - \beta_2 i_v)i_v|Z_4| + (\beta_3 i_h - \beta_1 i_h - \beta_2 i_v - (b_1+\eta_h-\delta_h i_h) \\
&\quad -\mu_v - \beta_3 i_h)i_v|Z_3| + (\beta_1 + (\beta_1-\delta_h)e_h - \beta_1 i_h - \beta_1 i_h - \beta_2 i_v)i_v|Z_5| \\
&= (-\beta_1 i_h - \beta_2 i_v - (b_1+\eta_h-\delta_h i_h) - \mu_v)i_v|Z_2| \\
&\quad +(-\beta_1 i_h - \beta_2 i_v - (b_1+\eta_h-\delta_h i_h) - \mu_v)i_v|Z_3| + \\
&\quad \beta_1(i_v|Z_4| + i_v|Z_5|) - (\delta_h e_h + \beta_1 i_h + \beta_1 i_h + \beta_2 i_v)(i_v|Z_4| + i_v|Z_5|)
\end{aligned}
$$

$$\begin{aligned} &< (-(\beta_1-\delta_h)i_h-\beta_2 i_v-(b_1-\beta_1)-\eta_h-\mu_v)i_v|Z_2| \\ &\quad +(-(\beta_1-\delta_h)i_h-\beta_2 i_v-(b_1-\beta_1)-\eta_h-\mu_v)i_v|Z_3| \\ &= -\rho_2(i_v|Z_2|+i_v|Z_3|) \\ &= -\rho_2\|Z\| \end{aligned}$$

where

$$\rho_2=(\beta_1-\delta_h)i_h+\beta_2 i_v+(b_1-\beta_1)+\eta_h+\mu_v.$$

Case1c. $i_v(|Z_4|+|Z_5|)>\{|Z_1|,i_v(|Z_2|+|Z_3|),i_v|Z_6|\}$. Then $\|Z\|=i_v(|Z_4|+|Z_5|)=i_v(Z_4+Z_5)$ and

$$\begin{aligned} D_+\|Z\| &= i_v(\frac{i_v'}{i_v}Z_4+\frac{i_v'}{i_v}Z_5+Z_4'+Z_5') \\ &= i_v(\eta_h Z_2+j_{44}Z_4-\beta_3 i_h Z_5+\eta_h Z_3+\eta_v Z_4+j_{55}Z_5) \\ &= i_v(\eta_h Z_2+(-(b_1+\gamma_h+\delta_h)+2\delta_h i_h-\beta_3 i_h-(\eta_v+\mu_v))Z_4-\beta_3 i_h Z_5+\eta_h Z_3+\eta_v Z_4 \\ &\quad +(-(b_1+\gamma_h+\delta_h)+2\delta_h i_h-\mu_v)Z_5) \\ &= \eta_h i_v(|Z_2|+|Z_3|)+(-(b_1+\gamma_h+\delta_h)+2\delta_h i_h-\beta_3 i_h-(\eta_v+\mu_v)+\eta_v)|Z_4| \\ &\quad +(-(b_1+\gamma_h+\delta_h)+2\delta_h i_h-\mu_v-\beta_3 i_h)|Z_5| \\ &\le -[(b_1-\delta_h-\eta_h)+\gamma_h+\beta_3 i_h+\mu_v]i_v(|Z_4|+|Z_5|) \\ &= -\rho_3\|Z\| \end{aligned}$$

where

$$\rho_3=(b_1-\delta_h-\eta_h)+\gamma_h+\beta_3 i_h+\mu_v.$$

Case1d. $i_v|Z_6|>\{|Z_1|,i_v(|Z_2|+|Z_3|),i_v(|Z_4|+|Z_5|)\}$. Then $\|Z\|=i_v|Z_4|=i_v Z_6$ and

$$\begin{aligned} D_+\|Z\| &= i_v(\frac{i_v'}{i_v}Z_6+Z_6') \\ &= i_v(\beta_3(1-e_v-i_v)Z_5+j_{66}Z_6) \\ &\le \beta_3 i_v|Z_5|-\beta_3(e_v+i_v)|Z_5|+(-\beta_3 i_h-(\eta_v+\mu_v)-\mu_v)i_v|Z_6| \end{aligned}$$

$$< \quad (\beta_3 - \beta_3 i_h - (\eta_v + \mu_v) - \mu_v) i_v |Z_6|$$

$$= \quad -\rho_4 \|Z\|$$

where

$$\rho_4 = \beta_3 i_h + (\eta_v + \mu_v) - \beta_3 + \mu_v.$$

Applying the same technique for other cases, after some calculation, we get $\rho_5, \rho_6, ..., \rho_{31}, \tilde{\rho}_{32}, \tilde{\rho}_{33}$. Take $\rho = min\{\rho_1, \rho_2, \rho_3, ..., \rho_{31}, \tilde{\rho}_{32}, \tilde{\rho}_{33}\}$ and $\rho > 0$ under conditions in (3.3.2) and we have the Lozinskiĭ measure $\tilde{\mu}(K) < 0$. By applying the result on page 59 of [50], the unique "endemic" equilibrium is globally asymptotically stable which completes the proof.

Since this chapter concerns diseases with long duration and substantial mortality rate (e.g., malaria), therefore we got the typical solution of the model (3.1.1) for malaria disease. These solutions are shown graphically in figures (3.4) and (3.5). We have used the parameter values used in [26] for low malaria transmission.

We discussed the global dynamics of the normalized model and it has been analytically shown that unique "endemic" equilibrium is globally asymptotically stable under some conditions. We check whether these conditions are necessary or sufficient? We see numerically that if $b_1 < \delta_h + \gamma_h$ then exposed and infectious individuals and vectors will also approach to endemic level for different initial conditions (Figs.3.6,3.7,3.8,3.9). It is also investigated that the infected classes will also approach the endemic level if $\beta_3 > \eta_v + \mu_v$ (Figs.3.10,3.11,3.12,3.13). Same phenomena has been observed even if $b_1 + \mu_v < \beta_1 + \eta_v$(Figs.3.14,3.15,3.16,3.17). From these observations we conclude that the conditions given in (3.3.2) are **not** the necessary conditions for global asymptotic stability. One can take other forms of $\|Z\|$, which may lead to sufficient conditions different from (3.3.2).

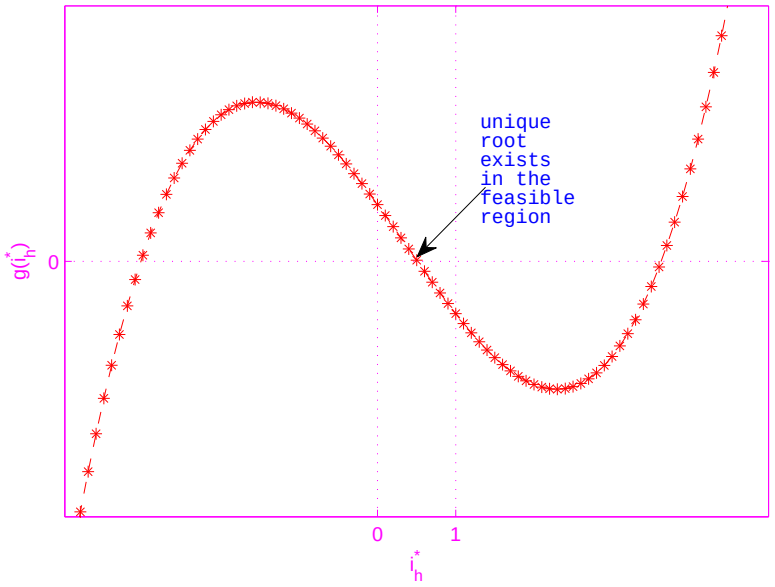

Figure 3.2: Plot of $g(i_h^*)$ showing that unique value of i_h^* in the feasible region when $\frac{b_1}{2} > \beta_1 > \delta_h + \eta_h$

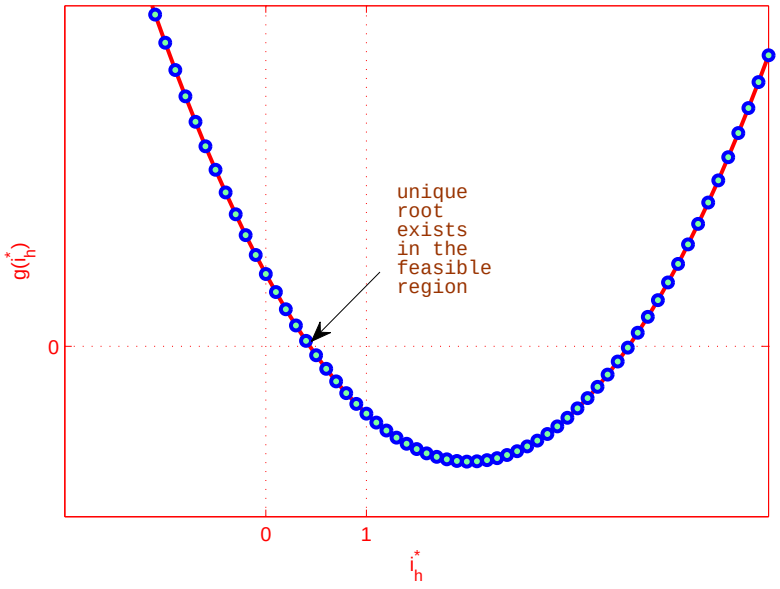

Figure 3.3: Plot of $g(i_h^*)$ showing that unique value of i_h^* in the feasible region when $\frac{b_1}{2} > \beta_1 = \delta_h$

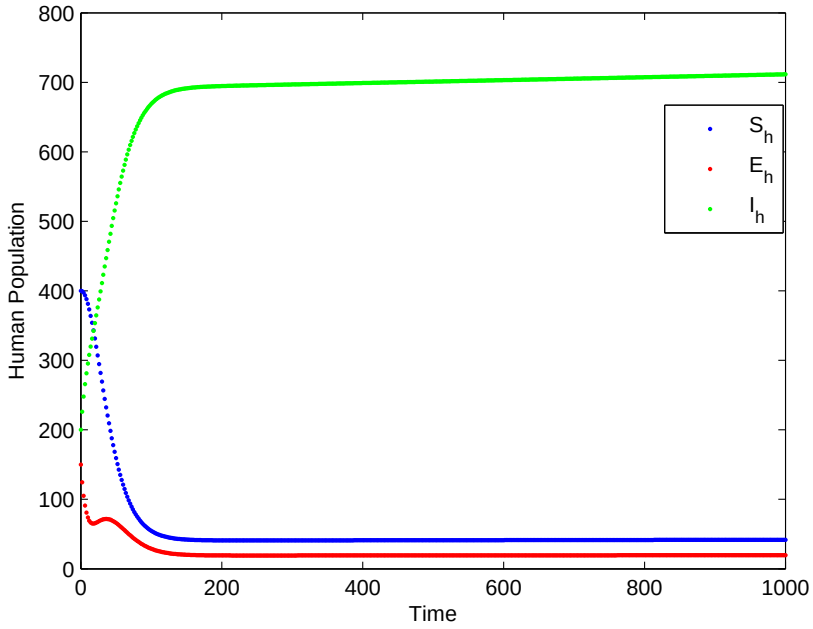

Figure 3.4: The human population approach unique endemic equilibrium for variables given in (3.1.1).

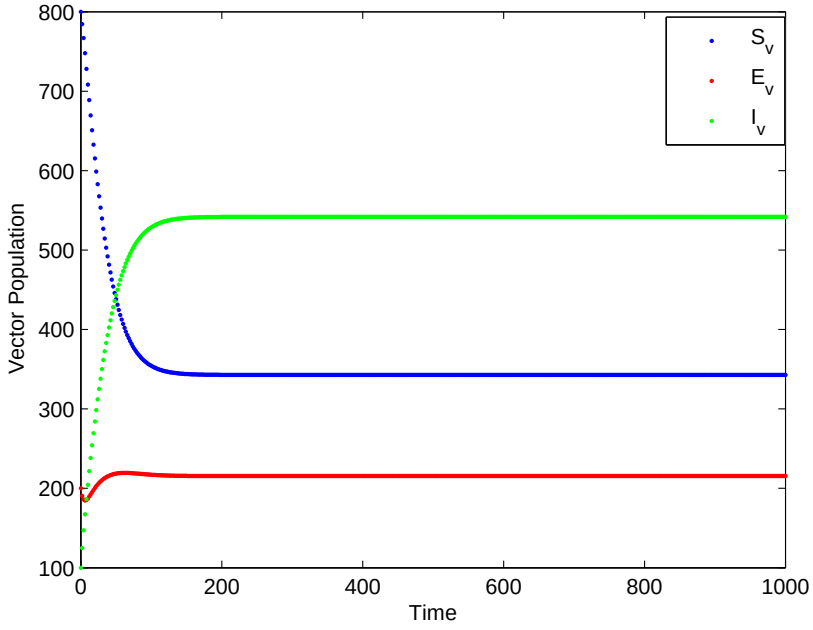

Figure 3.5: The vector population approach unique endemic equilibrium for variables given in (3.1.1).

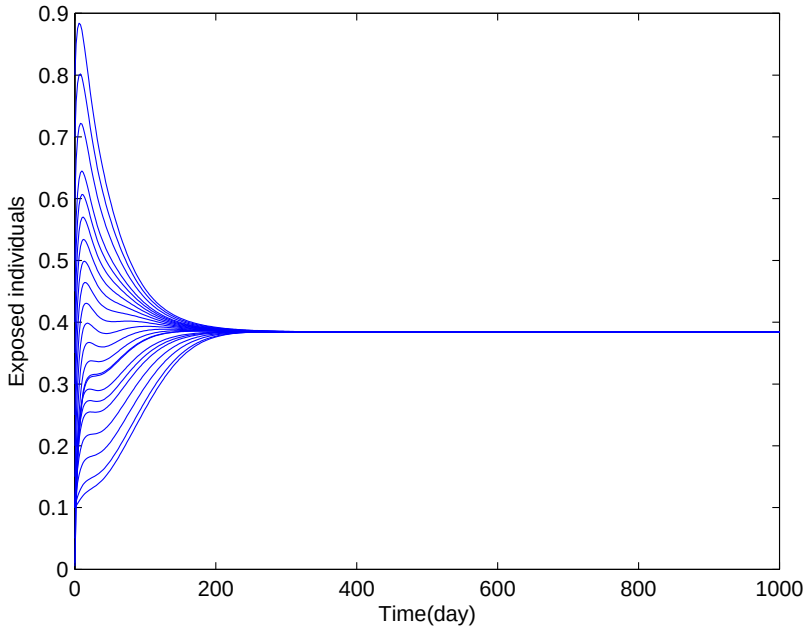

Figure 3.6: Exposed individuals approach unique endemic level for different initial conditions when $b_1 < \delta_h + \eta_h$. The parameter values are $b_1 = 0.015, \beta_1 = 0.1, \beta_2 = 0.74, \beta_3 = 0.1, \mu_v = 0.11, \eta_h = 0.016, \eta_v = 0.01, delta_h = 0.00000018, \gamma_h = 0.0000027$.

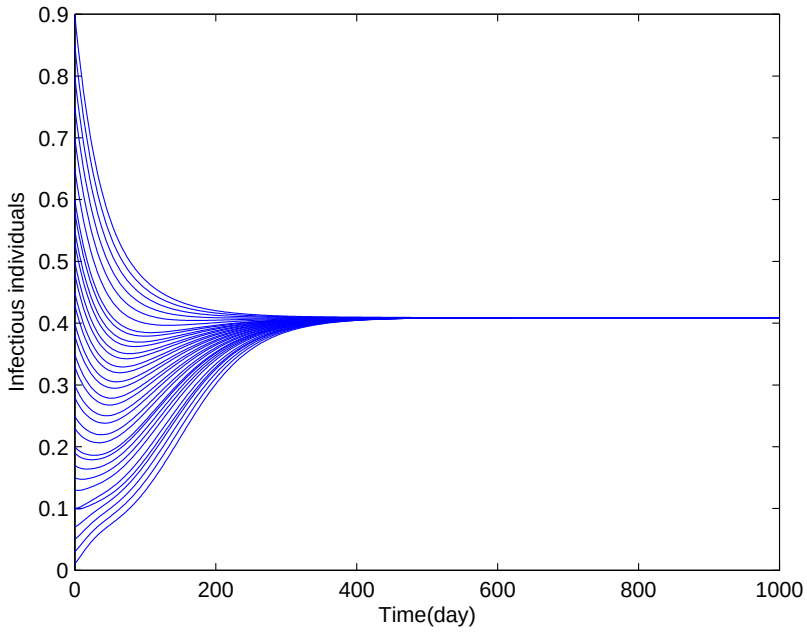

Figure 3.7: Infectious individuals approach unique endemic level for different initial conditions when $b_1 < \delta_h + \eta_h$. The parameter values are $b_1 = 0.015, \beta_1 = 0.1, \beta_2 = 0.74, \beta_3 = 0.1, \mu_v = 0.11, \eta_h = 0.016, \eta_v = 0.01, delta_h = 0.00000018, \gamma_h = 0.0000027$.

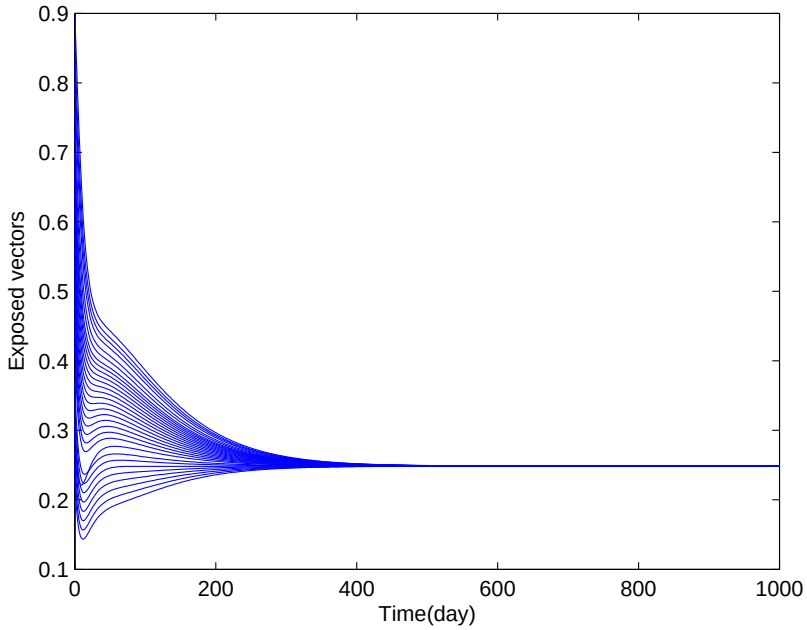

Figure 3.8: Exposed vectors approach unique endemic level for different initial conditions when $b_1 < \delta_h + \eta_h$. The parameter values are $b_1 = 0.015, \beta_1 = 0.1, \beta_2 = 0.74, \beta_3 = 0.1, \mu_v = 0.11, \eta_h = 0.016, \eta_v = 0.01, delta_h = 0.00000018, \gamma_h = 0.0000027$.

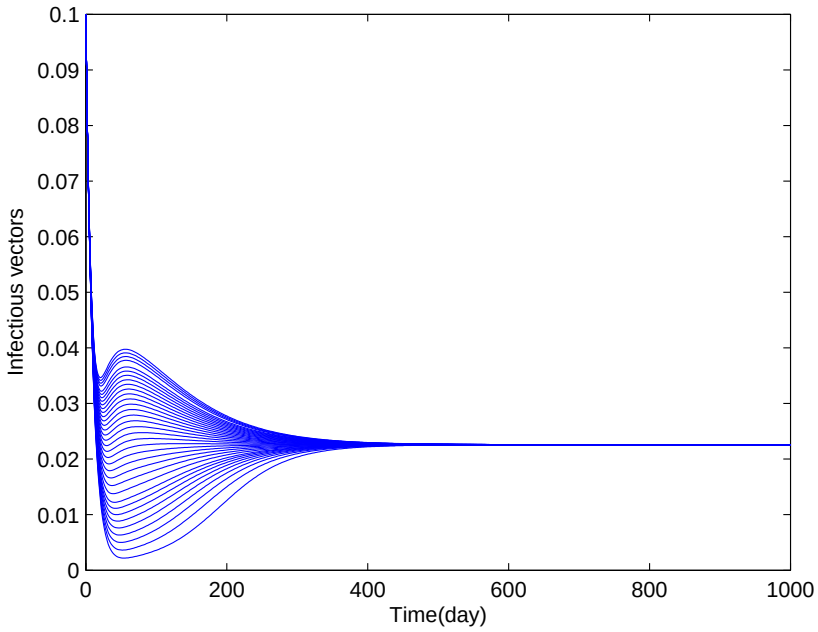

Figure 3.9: Infectious vectors approach unique endemic level for different initial conditions when $b_1 < \delta_h + \eta_h$. The parameter values are $b_1 = 0.015, \beta_1 = 0.1, \beta_2 = 0.74, \beta_3 = 0.1, \mu_v = 0.11, \eta_h = 0.016, \eta_v = 0.01, delta_h = 0.00000018, \gamma_h = 0.0000027$.

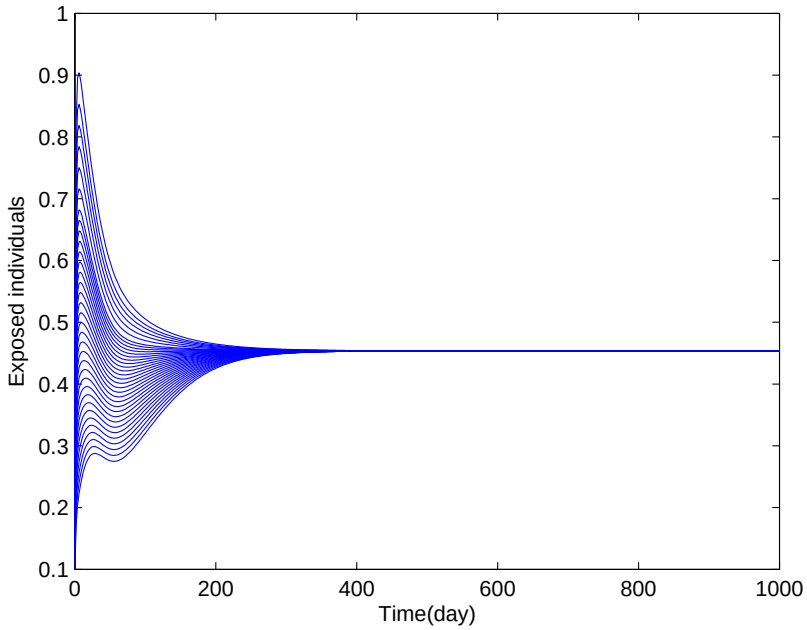

Figure 3.10: Exposed individuals approach unique endemic level for different initial conditions when $\beta_3 > \eta_v + \mu_v$. The parameter values are $b_1 = 0.015, \beta_1 = 0.1, \beta_2 = 0.74, \beta_3 = 0.13, \mu_v = 0.11, \eta_h = 0.01, \eta_v = 0.01, delta_h = 0.00000018, \gamma_h = 0.0000027$.

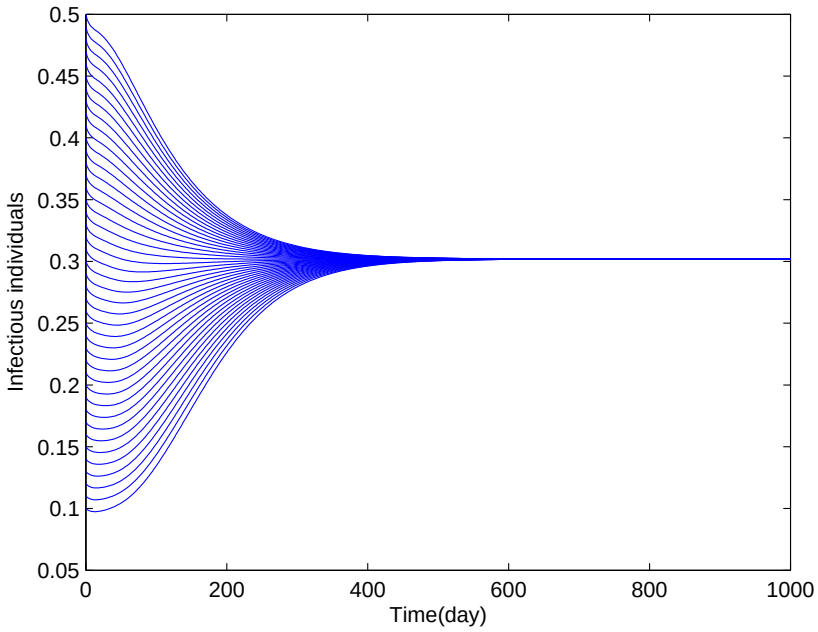

Figure 3.11: Infectious individuals approach unique endemic level for different initial conditions when $\beta_3 > \eta_v + \mu_v$. The parameter values are $b_1 = 0.015, \beta_1 = 0.1, \beta_2 = 0.74, \beta_3 = 0.13, \mu_v = 0.11, \eta_h = 0.01, \eta_v = 0.01, delta_h = 0.00000018, \gamma_h = 0.0000027$.

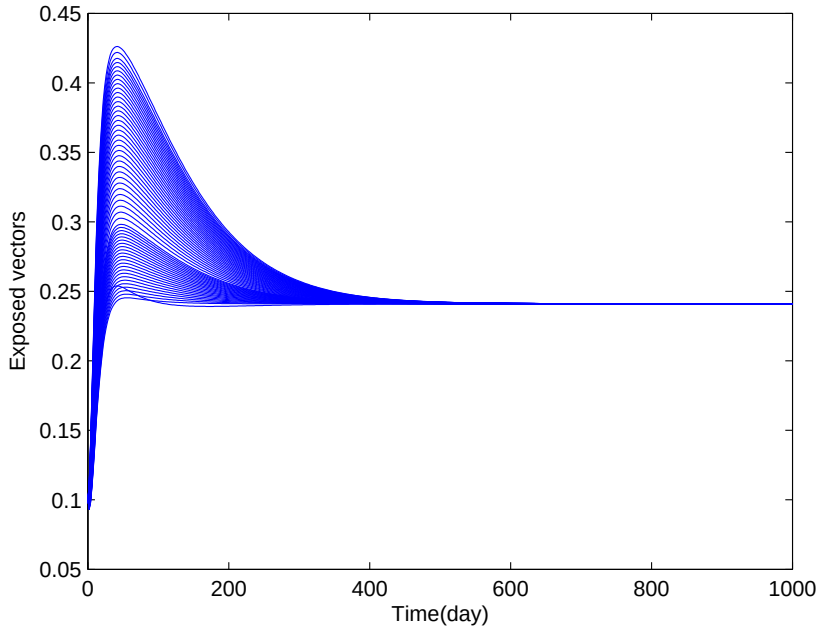

Figure 3.12: Exposed vectors approach unique endemic level for different initial conditions when $\beta_3 > \eta_v + \mu_v$. The parameter values are $b_1 = 0.015, \beta_1 = 0.1, \beta_2 = 0.74, \beta_3 = 0.13, \mu_v = 0.11, \eta_h = 0.01, \eta_v = 0.01, delta_h = 0.00000018, \gamma_h = 0.0000027$.

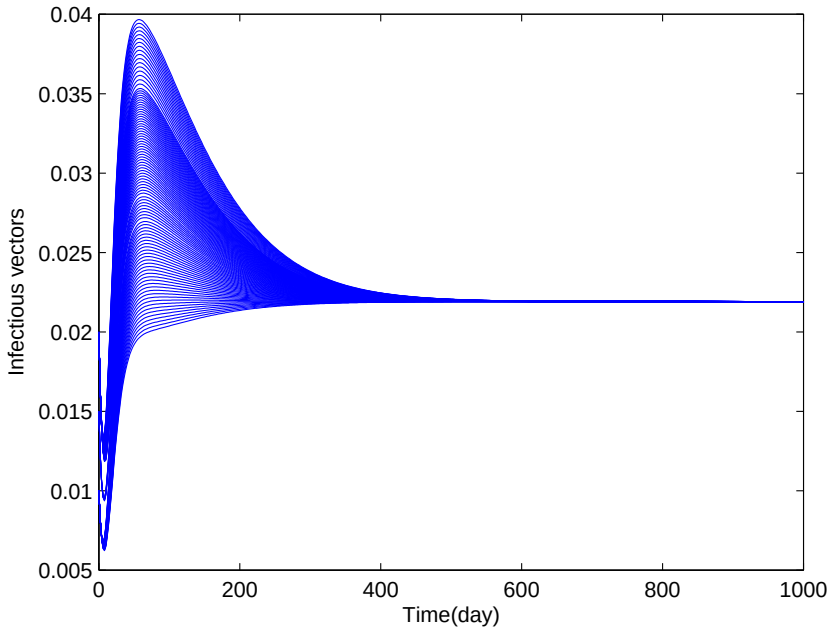

Figure 3.13: Infectious vectors approach unique endemic level for different initial conditions when $\beta_3 > \eta_v + \mu_v$. The parameter values are $b_1 = 0.015, \beta_1 = 0.1, \beta_2 = 0.74, \beta_3 = 0.13, \mu_v = 0.11, \eta_h = 0.01, \eta_v = 0.01, delta_h = 0.00000018, \gamma_h = 0.0000027$.

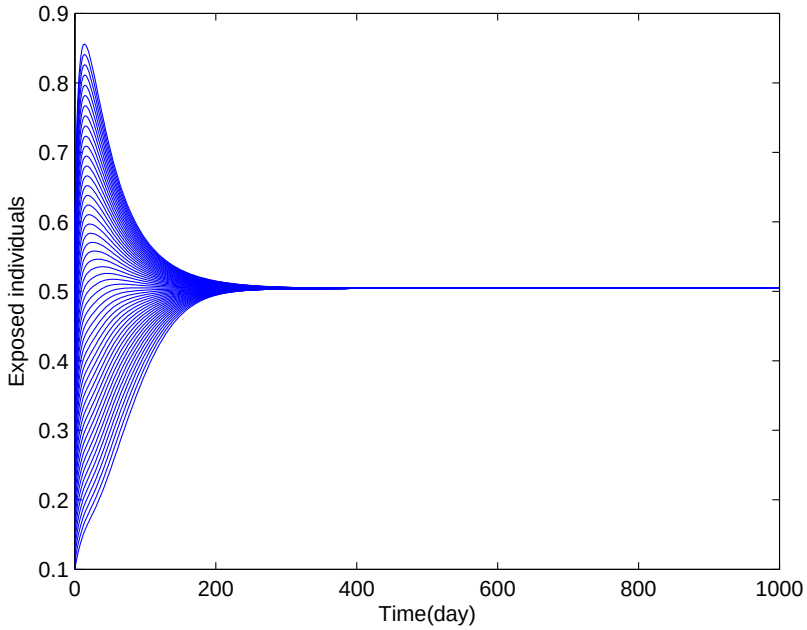

Figure 3.14: Exposed individuals approach unique endemic level for different initial conditions when $b_1 + \mu_v < \beta_1 + \eta_v$. The parameter values are $b_1 = 0.015, \beta_1 = 0.1, \beta_2 = 0.74, \beta_3 = 0.1, \mu_v = 0.11, \eta_h = 0.01, \eta_v = 0.03, delta_h = 0.00000018, \gamma_h = 0.0000027$.

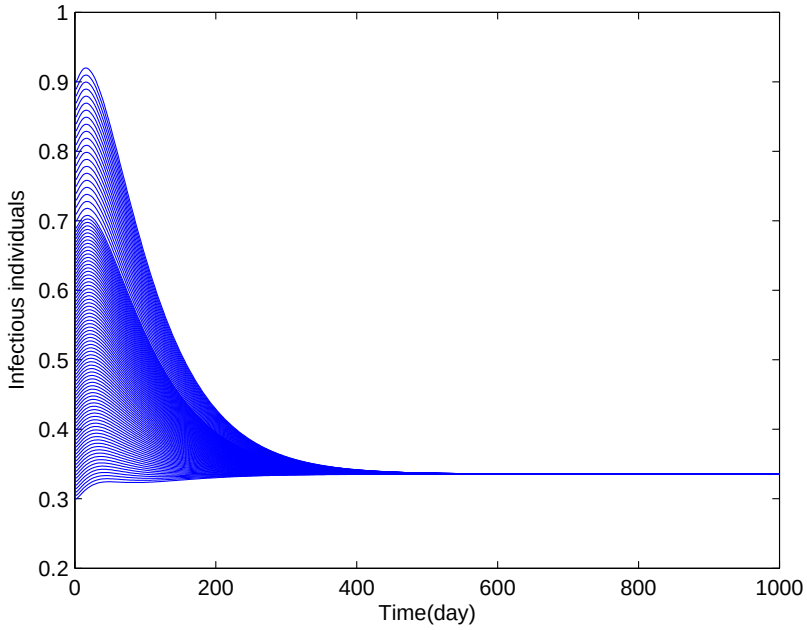

Figure 3.15: Infectious individuals approach unique endemic level for different initial conditions when $b_1 + \mu_v < \beta_1 + \eta_v$. The parameter values are $b_1 = 0.015, \beta_1 = 0.1, \beta_2 = 0.74, \beta_3 = 0.13, \mu_v = 0.11, \eta_h = 0.01, \eta_v = 0.03, delta_h = 0.00000018, \gamma_h = 0.0000027$.

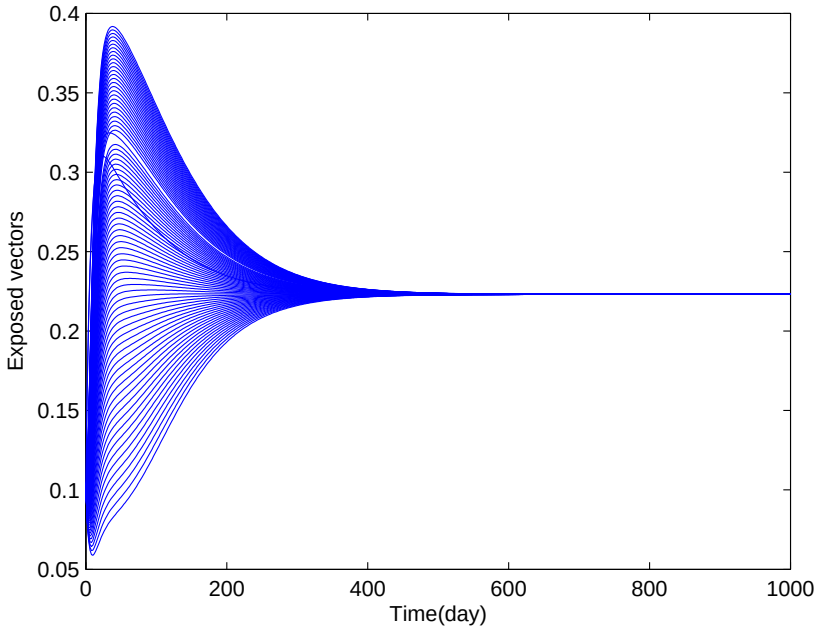

Figure 3.16: Exposed vectors approach unique endemic level for different initial conditions when $b_1 + \mu_v < \beta_1 + \eta_v$. The parameter values are $b_1 = 0.015, \beta_1 = 0.1, \beta_2 = 0.74, \beta_3 = 0.13, \mu_v = 0.11, \eta_h = 0.01, \eta_v = 0.03, delta_h = 0.00000018, \gamma_h = 0.0000027$.

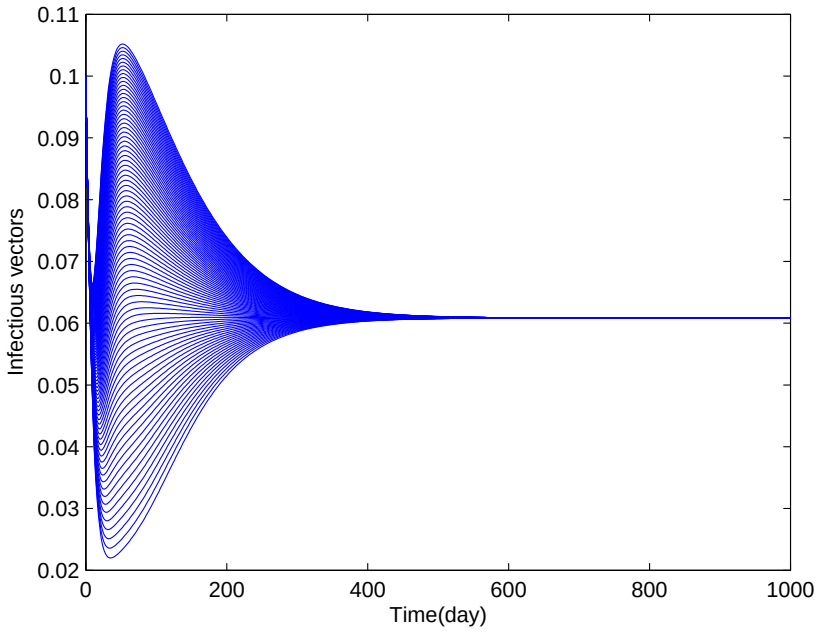

Figure 3.17: Infectious vectors approach unique endemic level for different initial conditions when $b_1 + \mu_v < \beta_1 + \eta_v$. The parameter values are $b_1 = 0.015, \beta_1 = 0.1, \beta_2 = 0.74, \beta_3 = 0.13, \mu_v = 0.11, \eta_h = 0.01, \eta_v = 0.03, delta_h = 0.00000018, \gamma_h = 0.0000027$.

Chapter 4

Stability Analysis of Vector-Host Model with Nonlinear Incidence

In this chapter, the vector host model with nonlinear incidence rate is considered. The purpose of this chapter is to carry out qualitative behavior and present a rigorous analysis of a vector host epidemic model to investigate the parameters to show how they affect the vector-borne disease transmission. The sensitivity analysis of the basic reproductive number and the endemic equilibrium with respect to epidemiological and demographic parameters is performed. From the sensitivity analysis, it is found that the reproductive number is most sensitive to the biting and mortality rates of mosquito. Further, the treatment rate of infectious humans is also sensitive parameter for equilibrium proportion of infectious humans.

4.1 Model Formulation

The total human population, denoted by $N_h(t)$, is split into susceptible individuals ($S_h(t)$) and infected individuals ($I_h(t)$) so that $N_h(t) = S_h(t) + I_h(t)$. Whereas, the total vector population, denoted by $N_v(t)$, is subdivided into susceptible vectors ($S_v(t)$) and infectious vectors ($I_v(t)$). Thus $N_v(t) = S_v(t) + I_v(t)$. The model is shown schematically by the subsequent diagram:

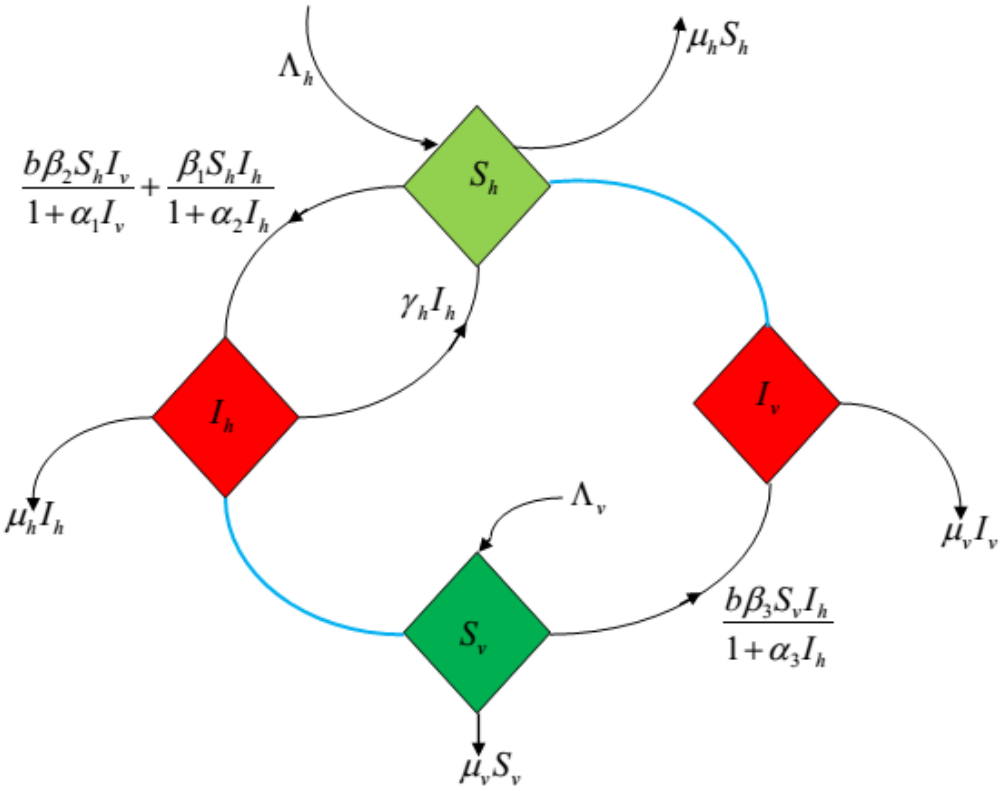

Figure 4.1: Flow diagram of Vector-Host Model with nonlinear incidence

The analytical expression of the model is given by the following system of differential equations:

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \frac{b\beta_2 S_h I_v}{1+\alpha_1 I_v} - \frac{\beta_1 S_h I_h}{1+\alpha_2 I_h} - \mu_h S_h + \gamma_h I_h, \\
\frac{dI_h}{dt} &= \frac{b\beta_2 S_h I_v}{1+\alpha_1 I_v} + \frac{\beta_1 S_h I_h}{1+\alpha_2 I_h} - \mu_h I_h - \gamma_h I_h, \\
\frac{dS_v}{dt} &= \Lambda_v - \frac{b\beta_3 I_h S_v}{1+\alpha_3 I_h} - \mu_v S_v, \\
\frac{dI_v}{dt} &= \frac{b\beta_3 I_h S_v}{1+\alpha_3 I_h} - \mu_v I_v.
\end{aligned} \tag{4.1.1}$$

Susceptible humans are recruited at a rate Λ_h, whereas susceptible vectors are generated by Λ_v. We assume that the number of bites per vector per host per unit time is φ, the proportion of infected bites that gives rise to the infection is r and the ratio of vector numbers to host numbers is ξ. Let $b = \varphi r \xi$, β_2 be the transmission rate from vector to

human, and β_3 be the transmission rate from human to vector. β_1 is the transmission probability from human to human. μ_h is natural death rate of human, μ_v is death rate of vectors, respectively. We assume that infectious individuals do not acquire permanent immunity and become susceptible again by the rate γ_h. Further we assume that incidence terms for human population and vector population that transmit disease are saturation interactions and are given by $\frac{b\beta_2 S_h I_v}{1+\alpha_1 I_v}$, $\frac{\beta_1 S_h I_h}{1+\alpha_2 I_h}$, $\frac{b\beta_3 I_h S_v}{1+\alpha_3 I_h}$, where α_1, α_2 and α_3 determine the level at which the force of infection saturates.

Obviously, $\Omega = \{(S_h, I_h, S_v, I_v) \in R^4 : S_h + I_h = \frac{\Lambda_h}{\mu_h}, S_v + I_v = \frac{\Lambda_v}{\mu_v}\}$, is positively invariant and system (4.1.1) is dissipative and the global attractor is contained in Ω.

The total dynamics of vector population is $\frac{dN_v}{dt} = \Lambda_v - \mu_v N_v$. Thus we can assume without loss of generality that $N_v = \frac{\Lambda_v}{\mu_v}$ for all, $t \geq 0$ provided that $S_v(0) + I_v(0) = \frac{\Lambda_v}{\mu_v}$. On Ω, $S_v = \frac{\Lambda_v}{\mu_v} - I_v$. Therefore, we attack system (4.1.1) by studying the subsystem

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \frac{b\beta_2 S_h I_v}{1+\alpha_1 I_v} - \frac{\beta_1 S_h I_h}{1+\alpha_2 I_h} - \mu_h S_h + \gamma_h I_h, \\
\frac{dI_h}{dt} &= \frac{b\beta_2 S_h I_v}{1+\alpha_1 I_v} + \frac{\beta_1 S_h I_h}{1+\alpha_2 I_h} - \mu_h I_h - \gamma_h I_h, \qquad (4.1.2) \\
\frac{dI_v}{dt} &= \frac{b\beta_3}{\mu_v}\frac{(\Lambda_v - \mu_v I_v) I_h}{1+\alpha_3 I_h} - \mu_v I_v.
\end{aligned}$$

From biological considerations, we study system (4.1.2) in the closed set $\Gamma = \{(S_h, I_h, I_v) \in R^3_+ : S_h + I_h = \frac{\Lambda_h}{\mu_h}, I_v \leq \frac{\Lambda_v}{\mu_v}\}$, where $R^3{}_+$ denotes the non-negative cone of R^3 including its lower dimensional faces. It can be easily verified that Γ is positively invariant with respect to (4.1.2).

4.2 Mathematical Analysis of the Model

The dynamics of the disease is described by the basic reproduction number $\Re_o$. The threshold quantity $\Re_o$ is called the reproduction number, which is defined as the average number of secondary infections produced by an infected individual in a completely susceptible population. The basic reproduction number of model (4.1.2) is given by the expression

$$\Re_o = \frac{\beta_1 \Lambda_h}{\mu_h(\mu_h + \gamma_h)} + \frac{b^2\beta_2\beta_3\Lambda_h\Lambda_v}{\mu_v^2\mu_h(\mu_h+\gamma_h)}. \qquad (4.2.1)$$

Direct calculation shows that system (4.1.2) has two equilibrium states: for $\Re_o \leq 1$, the only equilibrium is disease-free equilibrium $E_0 = (\Lambda_h/\mu_h, 0, 0)$. For $\Re_o > 1$, there is an

additional equilibrium $E^*(S_h^*, I_h^*, I_v^*)$ which is called endemic equilibrium, where

$$\begin{aligned} S_h^* &= \frac{\Lambda_h - \mu_h I_h^*}{\mu_h}, \\ I_v^* &= \frac{b\beta_3 \Lambda_v I_h^*}{\mu_v^2 + (\alpha_3 \mu_v^2 + b\beta_3 \mu_v) I_h^*}, \end{aligned} \tag{4.2.2}$$

and I_h^* is the root of the following quadratic equation

$$a_1 I_h^{2*} + a_2 I_h^* + a_3 = 0, \tag{4.2.3}$$

with

$$\begin{aligned} a_1 &= \alpha_2 \mu_h b^2 \beta_2 \beta_3 \Lambda_v + (\alpha_3 \mu_v^2 + b\beta_3 \mu_v + \alpha_1 b \beta_3 \Lambda_v)[\beta_1 \mu_h + \alpha_2 \mu_h (\mu_h + \gamma_h)], \\ a_2 &= \mu_h (b^2 \beta_2 \beta_3 \Lambda_v + \beta_1 \mu_v^2) + \alpha_2 (\mu_v^2 \mu_h (\mu_h + \gamma_h) - b^2 \beta_2 \beta_3 \Lambda_h \Lambda_v) \\ &\quad + (\alpha_3 \mu_v^2 + b\beta_3 \mu_v + \alpha_1 b \beta_3 \Lambda_v)(\mu_h (\mu_h + \gamma_h) - \Lambda_h \beta_1), \\ a_3 &= \mu_h (\mu_h + \gamma_h) \mu_v^2 (1 - \mathfrak{R}_\circ). \end{aligned} \tag{4.2.4}$$

From (4.2.4), we see that $\mathfrak{R}_\circ > 1$ if and only if, $a_3 < 0$. Since $a_1 > 0$, Eq.(4.2.3) has a unique positive root in feasible region. If $\mathfrak{R}_\circ < 1$, then $a_3 > 0$. Also, it can be easily seen that $a_2 > 0$ for $\mathfrak{R}_\circ < 1$. Thus, by considering the shape of the graph of Eq.(4.2.3) (and noting that $a_3 > 0$), we have that there will be zero (positive) endemic equilibrium in this case. Therefore, we can conclude that if $\mathfrak{R}_\circ < 1$, (4.2.3) has no positive root in the feasible region. If, $\mathfrak{R}_\circ > 1$, (4.2.3) has a unique positive root in the feasible region. This result is summarized below.

Theorem 4.2.1. *System (4.1.2) always has the infection-free equilibrium E_0. If $\mathfrak{R}_0 > 1$, system (4.1.2) has a unique endemic equilibrium $E^* = (S_h^*, I_h^*, I_v^*)$ defined by (4.2.2) and (4.2.3).*

4.2.1 Global Stability of Disease-Free Equilibrium

In this subsection, we analyze the global behavior of the equilibria for system (4.1.2). The following theorem provides the global property of the disease-free equilibrium E_0 of the system.

Theorem 4.2.2. *If $\mathfrak{R}_\circ \leq 1$, then the infection-free equilibrium E_0 is globally asymptotically stable in the interior of Γ.*

Proof. To establish the global stability of the disease-free equilibrium, we construct the following Lyapunov function:

$$L(t) = I_h(t) + b\beta_2 \frac{\Lambda_h}{\mu_h \mu_v} I_v(t). \tag{4.2.5}$$

Calculating the time derivative of L along the solutions of system (4.1.2), we obtain

$$\begin{aligned} L'(t) &= I_h'(t) + b\beta_2 \frac{\Lambda_h}{\mu_h \mu_v} I_v'(t) \\ &= \frac{b\beta_2 S_h I_v}{1+\alpha_1 I_v} + \frac{\beta_1 S_h I_h}{1+\alpha_2 I_h} - (\mu_h + \gamma_h) I_h + b\beta_2 \frac{\Lambda_h}{\mu_h \mu_v} \{ \frac{b\beta_3 \Lambda_v}{\mu_v (1+\alpha_3 I_h)} I_h - \frac{b\beta_3 I_v I_h}{1+\alpha_3 I_h} - \mu_v I_v \} \\ &\leq \frac{b\beta_2 \Lambda_h}{\mu_h} I_v + \frac{\beta_1 \Lambda_h}{\mu_h} I_h - (\mu_h + \gamma_h) I_h + b\beta_2 \frac{\Lambda_h}{\mu_h \mu_v} \{ \frac{b\beta_3 \Lambda_v}{\mu_v} I_h - \frac{b\beta_3 I_v I_h}{1+\alpha_3 I_h} - \mu_v I_v \} \\ &= -(\mu_h + \gamma_h) I_h (1 - \mathfrak{R}_\circ) - b^2 \beta_2 \beta_3 \frac{\Lambda_h}{\mu_h \mu_v} \frac{I_v I_h}{1+\alpha_3 I_h}. \end{aligned} \tag{4.2.6}$$

Thus $L'(t)$ is negative if $\mathfrak{R}_\circ \leq 1$. When $\mathfrak{R}_\circ < 1$, the derivative $L' = 0$ if and only if $I_h = 0$, while in the case $\mathfrak{R}_\circ = 1$, the derivative $L' = 0$ if and only if $I_h = 0$ or $I_v = 0$. Consequently, the largest compact invariant set in $\{(S_h, I_h, I_v) \in \Gamma, L' = 0\}$, when $\mathfrak{R}_\circ \leq 1$, is the singelton E_0. Hence, LaSalle's invariance principle [46] implies that E_0 is globally asymptotically stable in Γ. This completes the proof.

4.2.2 Global Stability of Endemic Equilibrium

Here, we use the geometrical approach as applied in Chapter 2 to investigate the global stability of the endemic equilibrium E^* in the feasible region Γ. To apply the result of theorem 2.5.1 for global stability of endemic equilibrium E^*, we first state and prove the following result.

Lemma 4.2.3. *If $\mathfrak{R}_\circ > 1$, then the system (4.1.2) is uniformly persistent i. e. there exists $c > 0$ (independent of initial conditions), such that $\liminf_{t\to\infty} S_h \geq c$, $\liminf_{t\to\infty} I_h \geq c$, $\liminf_{t\to\infty} I_v \geq c$.*

Proof. Let Φ be semi-dynamical system (4.1.2) in $(R^+)^3$, $\digamma$ be a locally compact metric space and $\Gamma_0 = \{(S_h, I_h, I_v) \in \Gamma : I_v = 0\}$. Γ_0 is a compact subset of Γ and Γ/Γ_0 is positively invariant set of system (4.1.2). Let $P : \digamma \to R^+$ be defined by $P(S_h, I_h, I_v) = I_v$ and set $S = \{(S_h, I_h, I_v) \in \Gamma : P(S_h, I_h, I_v) < \phi\}$, where ϕ is sufficiently small so that

$$\frac{\beta_1 \Lambda_h}{\mu_h(\mu_h + \gamma_h)(1+\alpha_2\phi)} + \frac{b^2\beta_2\beta_3\Lambda_h\Lambda_v(1 - \frac{\mu_v}{\Lambda_v}\phi)}{\mu_v^2 \mu_h(\mu_h + \gamma_h)(1+\alpha_3\phi)} > 1.$$

Assume that there is a solution $x \in S$ such that for each $t > 0$ $P(\Phi(x,t)) < P(x) < \phi$. Let us consider

$$L(t) = \frac{b\beta_2\Lambda_h}{\mu_h\mu_v}(1-\delta^*)I_v + I_h,$$

where δ^* is sufficiently small so that

$$\frac{\beta_1\Lambda_h}{\mu_h(\mu_h+\gamma_h)(1+\alpha_2\phi)} + \frac{b^2\beta_2\beta_3\Lambda_h\Lambda_v(1-\frac{\mu_v}{\Lambda_v}\phi)(1-\delta^*)}{\mu_v^2\mu_h(\mu_h+\gamma_h)(1+\alpha_3\phi)} > 1.$$

By direct calculation we have

$$\begin{aligned} L'(t) &\geq (\mu_h+\gamma_h)(\frac{\beta_1\Lambda_h}{\mu_h(\mu_h+\gamma_h)(1+\alpha_2\phi)} + \frac{b^2\beta_2\beta_3\Lambda_h\Lambda_v(1-\frac{\mu_v}{\Lambda_v}\phi)(1-\delta^*)}{\mu_v^2\mu_h(\mu_h+\gamma_h)(1+\alpha_3\phi)} - 1)I_h + \frac{b\beta_2\Lambda_h}{\mu_h}\delta^* I_v, \\ L'(t) &\geq \alpha L(t), \end{aligned} \tag{4.2.7}$$

where

$$\alpha = min\{(\mu_h+\gamma_h)(\frac{\beta_1\Lambda_h}{\mu_h(\mu_h+\gamma_h)(1+\alpha_2\phi)} + \frac{b^2\beta_2\beta_3\Lambda_h\Lambda_v(1-\frac{\mu_v}{\Lambda_v}\phi)(1-\delta^*)}{\mu_v^2\mu_h(\mu_h+\gamma_h)(1+\alpha_3\phi)} - 1), \frac{\mu_v\delta^*}{1-\delta^*}\}.$$

This implies that $L(t) \to \infty$ as $t \to \infty$. However $L(t)$ is bounded on Γ. According to Theorem 1 in [55] the proof is completed. The boundedness of Γ and the above lemma imply that (4.1.2) has a compact absorbing set $K \subset \Gamma$ [49]. Now we shall prove that the quantity $\bar{q}_2 < 0$. We choose a suitable vector norm $|.|$ in R^3 and a 3×3 matrix valued function

$$P(x) = \begin{pmatrix} 1 & 0 & 0 \\ 0 & \frac{I_h}{I_v} & 0 \\ 0 & 0 & \frac{I_h}{I_v} \end{pmatrix}. \tag{4.2.8}$$

Obviously P is C^1 and non singular in the interior of Γ. Linearizing system (4.1.2) about an endemic equilibrium E^* gives the following Jacobian matrix

$$J(E^*) = \begin{pmatrix} -\frac{b\beta_2 I_v}{1+\alpha_1 I_v} - \frac{\beta_1 I_h}{1+\alpha_2 I_h} - \mu_h & -\frac{\beta_1 S_h}{(1+\alpha_2 I_h)^2} + \gamma_h & -\frac{b\beta_2 S_h}{(1+\alpha_1 I_v)^2} \\ \frac{b\beta_2 I_v}{1+\alpha_1 I_v} + \frac{\beta_1 I_h}{1+\alpha_2 I_h} & \frac{\beta_1 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h+\gamma_h) & \frac{b\beta_2 S_h}{(1+\alpha_1 I_v)^2} \\ 0 & \frac{b\beta_3}{\mu_v}\frac{\Lambda_v - \mu_v I_v}{(1+\alpha_3 I_h)^2} & -\frac{b\beta_3 I_h}{(1+\alpha_3 I_h)} - \mu_v \end{pmatrix}.$$

The second additive compound matrix of $J(E^*)$ is given by

$$J^{[2]} = \begin{pmatrix} M_{11} & \frac{b\beta_2 S_h}{(1+\alpha_1 I_v)^2} & \frac{b\beta_2 S_h}{(1+\alpha_1 I_v)^2} \\ \\ \frac{b\beta_3}{\mu_v}\frac{\Lambda_v - \mu_v I_v}{(1+\alpha_3 I_h)^2} & M_{22} & -\frac{\beta_1 S_h}{(1+\alpha_2 I_h)^2} + \gamma_h \\ \\ 0 & \frac{b\beta_2 I_v}{1+\alpha_1 I_v} + \frac{\beta_1 I_h}{1+\alpha_2 I_h} & M_{33} \end{pmatrix},$$

where

$$\begin{aligned} M_{11} &= -\tfrac{b\beta_2 I_v}{1+\alpha_1 I_v} - \tfrac{\beta_1 I_h}{1+\alpha_2 I_h} - \mu_h + \tfrac{\beta_1 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h + \gamma_h), \\ M_{22} &= -\tfrac{b\beta_2 I_v}{1+\alpha_1 I_v} - \tfrac{\beta_1 I_h}{1+\alpha_2 I_h} - \mu_h - \frac{b\beta_3 I_h}{(1+\alpha_3 I_h)} - \mu_v, \\ M_{33} &= \tfrac{\beta_1 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h + \gamma_h) - \frac{b\beta_3 I_h}{(1+\alpha_3 I_h)} - \mu_v. \end{aligned} \tag{4.2.9}$$

The matrix $B = P_f P^{-1} + P J^{[2]} P^{-1}$ can be written in block form as

$$B = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix},$$

with

$$\begin{aligned} B_{11} &= -\tfrac{b\beta_2 I_v}{1+\alpha_1 I_v} - \tfrac{\beta_1 I_h}{1+\alpha_2 I_h} - \mu_h + \tfrac{\beta_1 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h + \gamma_h), \\ B_{12} &= (\tfrac{b\beta_2 S_h}{(1+\alpha_1 I_v)^2}\frac{I_v}{I_h}, \frac{b\beta_2 S_h}{(1+\alpha_1 I_v)^2}\frac{I_v}{I_h}), \\ B_{21} &= \begin{pmatrix} (\frac{I_h}{I_v})\frac{b\beta_3}{\mu_v}\frac{\Lambda_v - \mu_v I_v}{(1+\alpha_3 I_h)^2} \\ \\ 0 \end{pmatrix}, \\ B_{22} &= \begin{pmatrix} Q_{11} & Q_{12} \\ \\ Q_{21} & Q_{22} \end{pmatrix}, \end{aligned} \tag{4.2.10}$$

where

$$\begin{aligned} Q_{11} &= \frac{I_v}{I_h}(\frac{I_h}{I_v})_f - \frac{b\beta_2 I_v}{1+\alpha_1 I_v} - \frac{\beta_1 I_h}{1+\alpha_2 I_h} - \mu_h - \frac{b\beta_3 I_h}{(1+\alpha_3 I_h)} - \mu_v, \\ Q_{12} &= -\tfrac{\beta_1 S_h}{(1+\alpha_2 I_h)^2} + \gamma_h, \\ Q_{21} &= \frac{b\beta_2 I_v}{1+\alpha_1 I_v} + \frac{\beta_1 I_h}{1+\alpha_2 I_h}, \\ Q_{22} &= \frac{I_v}{I_h}(\frac{I_h}{I_v})_f + \frac{\beta_1 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h+\gamma_h) - \frac{b\beta_3 I_h}{(1+\alpha_3 I_h)} - \mu_v, \\ \frac{I_v}{I_h}(\frac{I_h}{I_v})_f &= \frac{I_h'}{I_h} - \frac{I_v'}{I_v}. \end{aligned} \tag{4.2.11}$$

Consider the norm in R^3 as: $\mid (u,v,w) \mid = max(\mid u \mid, \mid v \mid + \mid w \mid)$ where (u,v,w) denotes the vector in R^3. The Lozinskiĭ measure with respect to this norm is defined as

$$\mu(B) \leq sup(g_1, g_2),$$

where

$$g_1 = \mu_1(B_{11}) + \mid B_{12} \mid, \quad g_2 = \mu_1(B_{22}) + \mid B_{21} \mid .$$

From system (4.1.2) we can write

$$\begin{aligned} \frac{I_h'}{I_h} &= \frac{b\beta_2 S_h}{1+\alpha_1 I_v}\frac{I_v}{I_h} + \frac{\beta_1 S_h}{1+\alpha_2 I_h} - (\mu_h+\gamma_h), \\ \frac{I_v'}{I_v} &= \frac{b\beta_3}{\mu_v}\frac{(\Lambda_v - \mu_v I_v)}{1+\alpha I_h}\frac{I_h}{I_v} - \mu_v. \end{aligned} \tag{4.2.12}$$

Since B_{11} is a scalar, its Lozinskiĭ measure with respect to any vector norm in R^1 will be equal to B_{11}. Thus

$$B_{11} = -\frac{b\beta_2 I_v}{1+\alpha_1 I_v} - \frac{\beta_1 I_h}{1+\alpha_2 I_h} - \mu_h + \frac{\beta_1 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h+\gamma_h),$$

$$\mid B_{12} \mid = \frac{b\beta_2 S_h}{(1+\alpha_1 I_v)^2}\frac{I_v}{I_h},$$

and g_1 will become

$$\begin{aligned} g_1 &= -\tfrac{b\beta_2 I_v}{1+\alpha_1 I_v} - \tfrac{\beta_1 I_h}{1+\alpha_2 I_h} - \mu_h + \tfrac{\beta_1 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h+\gamma_h) + \tfrac{b\beta_2 S_h}{(1+\alpha_1 I_v)^2}\frac{I_v}{I_h} \\ &\leq -\tfrac{b\beta_2 I_v}{1+\alpha_1 I_v} - \tfrac{\beta_1 I_h}{1+\alpha_2 I_h} - \mu_h + \tfrac{\beta_1 S_h}{(1+\alpha_2 I_h)} - (\mu_h+\gamma_h) + \tfrac{b\beta_2 S_h}{(1+\alpha_1 I_v)}\frac{I_v}{I_h} \\ &\leq \frac{I_h'}{I_h} - \mu_h - \frac{b\beta_2 I_v}{1+\alpha_1 I_v} - \frac{\beta_1 I_h}{1+\alpha_2 I_h}. \end{aligned} \tag{4.2.13}$$

Also $\mid B_{21} \mid = (\frac{I_h}{I_v})\frac{b\beta_3}{\mu_v}\frac{\Lambda_v - \mu_v I_v}{(1+\alpha_3 I_h)^2}$, $\mid B_{12} \mid$ and $\mid B_{21} \mid$ are the operator norms of B_{12} and B_{21} which are mapping from R^2 to R and from R to R^2 respectively, and R^2 is endowed with the l_1 norm. $\mu_1(B_{22})$ is the Lozinskiĭ measure of 2×2 matrix B_{22} with respect to l_1 norm in R^2.

$$\begin{aligned}
\mu(B_{22}) &= Sup\{\frac{I_v}{I_h}(\frac{I_h}{I_v})_f - \frac{b\beta_2 I_v}{1+\alpha_1 I_v} - \frac{\beta_1 I_h}{1+\alpha_2 I_h} - \mu_h - \frac{b\beta_3 I_h}{(1+\alpha_3 I_h)} - \mu_v + \frac{b\beta_2 I_v}{1+\alpha_1 I_v} + \\
&\quad \frac{\beta_1 I_h}{1+\alpha_2 I_h}, \frac{I_v}{I_h}(\frac{I_h}{I_v})_f + \frac{\beta_1 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h + \gamma_h) - \frac{b\beta_3 I_h}{(1+\alpha_3 I_h)} - \mu_v - \frac{\beta_1 S_h}{(1+\alpha_2 I_h)^2} + \gamma_h\}, \\
&= \frac{I_v}{I_h}(\frac{I_h}{I_v})_f - \mu_h - \frac{b\beta_3 I_h}{(1+\alpha_3 I_h)} - \mu_v.
\end{aligned} \tag{4.2.14}$$

Hence

$$\begin{aligned}
g_2 &= \frac{I_h'}{I_h} - \frac{I_v'}{I_v} + (\frac{I_h}{I_v})\frac{b\beta_3}{\mu_v}\frac{\Lambda_v - \mu_v I_v}{(1+\alpha_3 I_h)^2} - \mu_h - \frac{b\beta_3 I_h}{(1+\alpha I_h)} - \mu_v \\
&\leq \frac{I_h'}{I_h} - \frac{I_v'}{I_v} + (\frac{I_h}{I_v})\frac{b\beta_3}{\mu_v}\frac{\Lambda_v - \mu_v I_v}{(1+\alpha_3 I_h)} - \mu_h - \frac{b\beta_3 I_h}{(1+\alpha_3 I_h)} - \mu_v \\
&\leq \frac{I_h'}{I_h} - \frac{I_v'}{I_v} + \frac{I_v'}{I_v} - \mu_h - \frac{b\beta_3 I_h}{(1+\alpha_3 I_h)} \\
&\leq \frac{I_h'}{I_h} - \mu_h - \frac{b\beta_3 I_h}{(1+\alpha_3 I_h)}.
\end{aligned} \tag{4.2.15}$$

Thus,

$$\mu(B) = Sup\{g_1, g_2\} \leq \frac{I_h'}{I_h} - \mu_h. \tag{4.2.16}$$

Since (4.1.2) is uniformly persistent when $\Re_o > 1$, so for $T > 0$ such that $t > T$ implies $I_h(t) \geq c$, $I_v(t) \geq c$ and $\frac{1}{t}\log I_h(t) < \frac{\mu}{2}$ for all $(S_h(0), I_h(0), I_v(0)) \in K$. Thus

$$\frac{1}{t}\int_0^t \mu(B)dt < \frac{\log I_h(t)}{t} - \mu < \frac{-\mu}{2}$$

for all $(S_h(0), I_h(0), I_v(0)) \in K$, which further implies that $\bar{q}_2 < 0$. Therefore all the conditions of Theorem (2.5.1) are satisfied. Hence unique endemic equilibrium E^* is globally stable in Γ.

4.3 Sensitivity Analysis

We would like to know different factors that are responsible for the disease transmission and prevalence. In this way we can try to reduce human mortality and morbidity due

to disease. Initial disease transmission depends upon the reproductive number whereas disease prevalence is directly related to the endemic equilibrium point. The class of infectious humans is the most important class because it represents the persons who may be clinically ill, and is directly related to the disease induced deaths. We will calculate the sensitivity indices of the reproductive number, $\mathfrak{R}_\circ$, and the endemic equilibrium point with respect to the parameters given in Table(4.1) for the model.

Table 4.1: Values of parameters used for sensitivity analysis

parameter	value	reference
Λ_h	0.00011	[56]
Λ_v	0.13	[56]
b	0.5	[56]
γ_h	0.7	assumed
β_2	0.022	[56]
β_3	0.48	[56]
β_1	0.004	assumed
α	5	assumed
μ_h	0.000016	[56]
μ_v	0.033	[56]

By the analysis of these indices we could determine which parameter is more crucial for disease transmission and prevalence.

Definition 4.3.1. The normalized forward sensitivity index of a variable, h, that depends differentiably on a parameter, l, is defined as: $\gamma_l^h = \dfrac{\partial h}{\partial l} \times \dfrac{l}{h}$.

Table 4.2 represents sensitivity indices of model parameters to $\mathfrak{R}_\circ$.

Table 4.2: Sensitivity indices of $\mathfrak{R}_o$ to parameters for the model, evaluated at the parameter values given in Table 4.1

Parameter	Description	Sensitivity index
b	rate of biting of a host by mosquito	1.97493
γ_h	loss of imunity	-0.999977
β_2	probability of transmission from mosquitoes to host	0.987467
β_3	probability of transmission from host to mosquitoes	0.987467
β_1	probability of transmission from infectious human to susceptible human	0.0125332
Λ_h	recruitment rate of susceptible hosts	1
Λ_v	recruitment rate of susceptible mosquitoes	0.987467
μ_v	death rate of mosquitoes	-1.97493
μ_h	death rate of hosts	-1.00002

4.3.1 Sensitivity Indices of Endemic Equilibrium

We have numerically calculated the sensitivity indices at the parameter values given in Table (4.1). The most sensitive parameter for I_h^* is mosquito biting rate. Change in mosquito biting rate is directly related to change in I_h^* and inversely related to change in γ_h. This suggests strategies that personal protection and human treatment can lead to marvelous decrease in I_h^*. The most sensitive parameter for I_v^* is mosquito death rate μ_v, followed by mosquito biting rate. We observe that I_v^* can be reduced by personal protection, larvacide and adulticide etc.

The analysis of the sensitivity indices of $\mathfrak{R}_o$, I_h^* and I_v^*, suggests us that three controls personal protection, larvacide and adulticide and treatment of infectious humans can play an effective role to control the disease. The sensitivity indices for S_h^*, I_h^*, and I_v^* with respect to all parameters are given in Table (4.3).

Table 4.3: The sensitivity indices of the state variables at the endemic equilibrium, x_i , to the parameters ,p_j, for parameter values given in Table 4.1

	S_h^*	I_h^*	I_v^*
Λ_h	0.998946	1.50275	0.00011
Λ_v	-0.00108296	0.516688	1.45019
b	-0.00401088	1.91363	2.57621
γ_h	0.00314305	-1.49958	-1.30657
β_2	-0.00302661	1.44402	1.25817
β_3	-0.000984278	0.469608	1.31805
β_1	-0.000116517	0.0555912	0.0484363
α_1	0.00194365	-0.927333	-0.80798
α_2	6.33734×10^{-6}	–0.0030236	-0.00263445
α_3	0.0000407045	-0.0194205	-0.0545075
μ_h	-0.998946	-1.50279	-1.30937
μ_v	0.00206723	-0.986295	-2.76823

Chapter 5

Analysis of Pine Wilt Disease Models

This chapter explains the dynamics of a Pine Wilt Disease. The deterministic pine wilt models with vital dynamics to determine the equilibria and their stability by considering nonlinear incidence rates, standard incidence rates with horizontal transmission is analyzed. The complete global analysis for the equilibria of the models is discussed. Those factors are explained which are responsible in order to eradicate or to lower the endemic level of infectious pines and pine sawyer beetles. On the basis of sensitive parameters we can design the control strategies.

5.1 Model with nonlinear incidence

The pine population, with total population size denoted by $N_h(t)$, is sub-divided into two mutually exclusive compartments: susceptible pine trees $S_h(t)$ and infectious pine trees $I_h(t)$. Thus, $N_h(t) = S_h(t) + I_h(t)$. The emission of oleoresin from susceptible host pines behaves like a physical barrier for beetle oviposition. Beetles can oviposit on the infected pine trees because these trees cease oleoresin. Since there are no cures for pine wilt once a susceptible tree becomes infested with pinewood nematodes, so the recovered class $R_h(t)$ has not been considered.

The total vector population at any time t is denoted by $N_v(t) = S_v(t) + I_v(t)$, where $S_v(t)$ denotes the susceptible adult beetles that do not have any pinewood nematode at time t and $I_v(t)$ denotes the infected adult beetles carrying pinewood nematode at time t when they emerge from dead pine trees. After emergence from the dead tree, beetles choose healthy tree for sufficient feeding and transmit nematodes into the tree. These nematodes move through the feeding wounds and approach the xylem of the tree. When beetles are

in oviposition they choose dying or dead tree and transmit nematode when they lay eggs in slits in bark. Nematodes enter these slits, feed on wood cells or fungi and reproduce themselves. Before beetle's emergence from dead tree the nematodes attach with the tracheae of its respiratory system. The following assumptions are made in formulating the mathematical model.

- The exploitation rate of those pine trees which have infected Bursaphelenchus xylophilus is greater than the normal and susceptible pine trees.
- The susceptible beetles receive nematodes directly from infectious ones through mating.
- Adult beetles emerging from infected trees have pinewood nematode.
- The infected vectors transmit the nematode during maturation feeding as well as via oviposition.

Under these assumptions the model is designed in the following diagram.

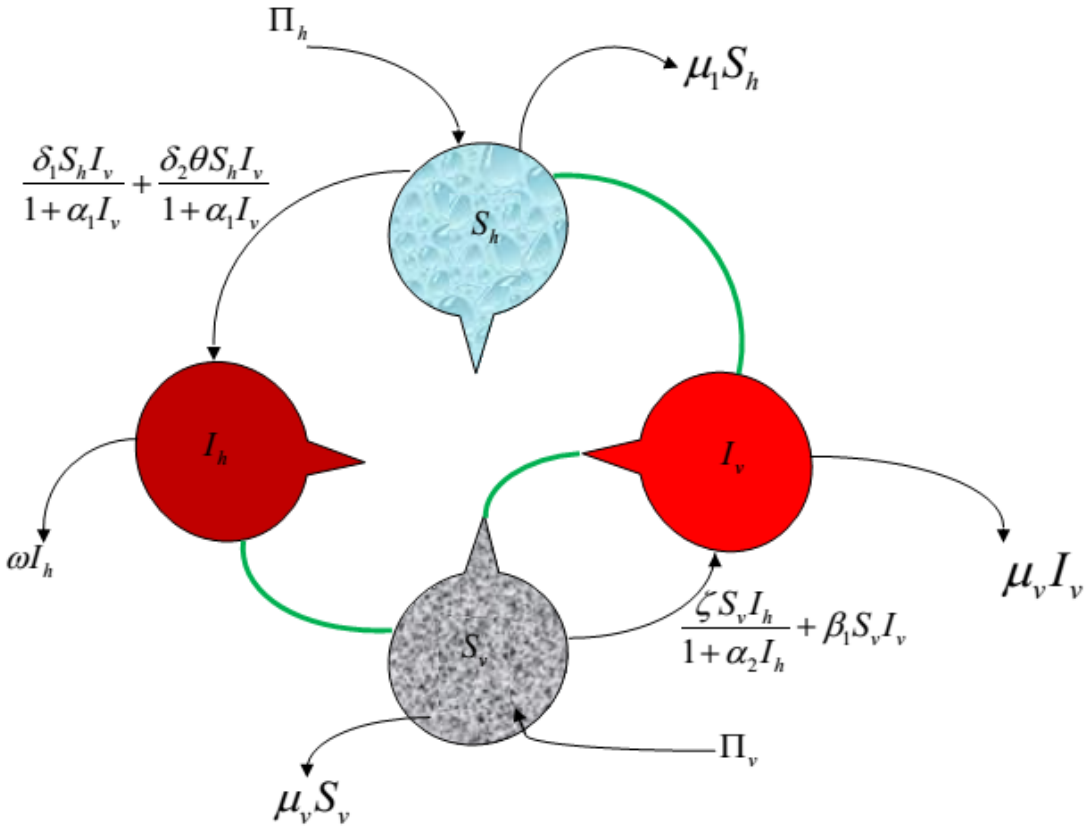

Figure 5.1: Flow diagram of pine wilt disease model with nonlinear incidence

Mathematical description of the model is given by the following system of differential equations.

$$\begin{aligned}
\frac{dS_h}{dt} &= \Pi_h - \frac{\delta_1 S_h I_v}{1+\alpha_1 I_v} - \frac{\delta_2 \theta S_h I_v}{1+\alpha_1 I_v} - \mu_1 S_h, \\
\frac{dI_h}{dt} &= \frac{\delta_1 S_h I_v}{1+\alpha_1 I_v} + \frac{\delta_2 \theta S_h I_v}{1+\alpha_1 I_v} - \omega I_h, \\
\frac{dS_v}{dt} &= \Pi_v - \frac{\zeta S_v I_h}{1+\alpha_2 I_h} - \beta_1 S_v I_v - \mu_v S_v, \\
\frac{dI_v}{dt} &= \frac{\zeta S_v I_h}{1+\alpha_2 I_h} + \beta_1 S_v I_v - \mu_v I_v,
\end{aligned} \tag{5.1.1}$$

where Π_h is the constant input rate of pines, Π_v is the constant increase rate of vectors and μ_v is the mortality rate of vectors. The exploitation rate of susceptible pines is μ_1 where as the percent isolated and felled of pine which has infected Bursaphelenchus xylophilus is ω. The transmission between susceptible pines and infected vectors occurs when infected beetles lay eggs on those dead pines that die of natural causes or through the maturation feeding of infected vectors, the incidence terms for these transmissions are $\frac{\delta_2 \theta S_h I_v}{1+\alpha_1 I_v}$ and $\frac{\delta_1 S_h I_v}{1+\alpha_1 I_v}$, respectively. The parameters, θ is the probability by which

susceptible pines die of natural causes and cease oleoresin exudation without being infected by the nematode, δ_2 indicates the rate at which infected vectors transmit the nematode via oviposition whereas δ_1 denotes transmission rate per contact during maturation feeding. The transmission between susceptible vectors and infected hosts occurs when adult beetles emerge from dead pine trees. This transmission is denoted by $\frac{\zeta S_v I_h}{1+\alpha_2 I_h}$, where ζ is the rate at which adult beetles carry the pinewood nematode when they emerge from dead trees. The parameters α_1 and α_2 determine the level at which the infection saturates. The beetles transmit nematodes directly through mating. The incidence term for this transmission is $\beta_1 S_v I_v$, where β_1 is the transmission rate among beetles during mating. All parameters are assumed to be positive.

The total dynamics of vector population satisfy the following equation:

$$\frac{dN_v}{dt} = \Pi_v - \mu_v N_v. \tag{5.1.2}$$

This leads to $N_v \to \frac{\Pi_v}{\mu_v}$ as $t \to \infty$. Thus, the system (5.1.1) is reduced to the following system of differential equations:

$$\begin{aligned}
\frac{dS_h}{dt} &= \Pi_h - \frac{\delta_1 S_h I_v}{1+\alpha_1 I_v} - \frac{\delta_2 \theta S_h I_v}{1+\alpha_1 I_v} - \mu_1 S_h, \\
\frac{dI_h}{dt} &= \frac{\delta_1 S_h I_v}{1+\alpha_1 I_v} + \frac{\delta_2 \theta S_h I_v}{1+\alpha_1 I_v} - \omega I_h, \\
\frac{dI_v}{dt} &= \zeta(\frac{\Pi_v}{\mu_v} - I_v)\frac{I_h}{1+\alpha_2 I_h} + \beta_1(\frac{\Pi_v}{\mu_v} - I_v)I_v - \mu_v I_v.
\end{aligned} \tag{5.1.3}$$

Considering ecological significance, we study system (5.1.3) in the closed set

$$\Omega = \{(S_h, I_h, I_v) : \frac{\Pi_h}{\omega} \le S_h + I_h \le \frac{\Pi_h}{\mu_1}, 0 \le I_v \le \frac{\Pi_v}{\mu_v}\}. \tag{5.1.4}$$

It can be easily verified that Ω is positively invariant with respect to (5.1.3).

5.2 Existence of Equilibria

The basic reproduction number of model (5.1.3) is given by

$$\mathfrak{R}_o = \frac{\beta_1 \Pi_v}{\mu_v^2} + \frac{\zeta \Pi_v}{\mu_v^2}\frac{\Pi_h}{\mu_1 \omega}(\delta_1 + \theta\delta_2). \tag{5.2.1}$$

Direct calculation shows that for $\mathfrak{R}_o \le 1$, there is only disease-free equilibrium $E_0(\frac{\Pi_h}{\mu_1}, 0, 0)$ and for $\mathfrak{R}_o > 1$, there is an additional equilibrium $E^*(S_h^*, I_h^*, I_v^*)$ which is called endemic equilibrium, with

$$\begin{aligned}
S_h^* &= \frac{\Pi_h - \omega I_h^*}{\mu_1}, \\
I_h^* &= \frac{\Pi_h I_v^*[\delta_1 + \delta_2\theta + (\delta_1\alpha_1 + \delta_2\theta\alpha_1)I_v^*]}{[(\alpha_1\mu_1 + \delta_1 + \delta_2\theta)\alpha_1\omega I_v^{*2} + (\delta_1 + \delta_2\theta + 2\alpha_1\mu_1)\omega I_v^* + \omega\mu_1]},
\end{aligned} \tag{5.2.2}$$

and I_v^* is the root of the following equation

$$AI_v^{*3} + BI_v^{*2} + CI_v^* + D = 0, \tag{5.2.3}$$

where,

$$\begin{aligned} A &= \Pi_h \mu_v \alpha_2 \beta_1 \left(\alpha_1 \delta_1 + \theta \alpha_1 \delta_2\right), \\ B &= \theta\omega\alpha_1\beta_1\delta_2\mu_v + \theta\alpha_1\zeta\delta_2\Pi_h\mu_v + \theta\alpha_2\beta_1\delta_2\Pi_h\mu_v + \omega\alpha_1\alpha_1\beta_1\mu_1\mu_v, \\ C &= \omega\beta_1\mu_1\mu_v + \alpha_1\beta_1\delta_1\Pi_h\Pi_v + \theta\alpha_1\zeta\delta_2\Pi_h\Pi_v, \\ D &= \omega\mu_1\mu_v^2(1 - \mathfrak{R}_\circ). \end{aligned} \tag{5.2.4}$$

From (5.2.4), we see that $\mathfrak{R}_\circ > 1$ if and only if $D < 0$. Since A, B and C are always positive, so there will be zero or unique positive endemic equilibrium according as $\mathfrak{R}_\circ \leq 1$ or $\mathfrak{R}_\circ > 1$. Thus we have following theorem.

Theorem 5.2.1. *System (5.1.3) always has the infection-free equilibrium E_0. If $\mathfrak{R}_\circ > 1$, system (5.1.3) has a unique endemic equilibrium $E^*(S_h^*, I_h^*, I_v^*)$ defined by (5.2.2) and (5.2.3).*

5.3 Stability of Disease-Free Equilibrium

Here, we analyze stability of disease-free equilibrium $E_0(\frac{\Pi_h}{\mu_1}, 0, 0)$ for system (5.1.3). The linearization of the system (5.1.3) at E_0 results the following characteristic equation:

$$(-\mu_1 - \lambda)[\lambda^2 + \lambda(\omega + \mu_v - \frac{\beta_1\Pi_v}{\mu_v}) + \omega\mu_v(1 - \mathfrak{R}_\circ)] = 0. \tag{5.3.1}$$

The characteristic equation (5.3.1) has one eigenvalue $-\mu_1$. The other eigenvalues can be found by the equation

$$\lambda^2 + a\lambda + b = 0, \tag{5.3.2}$$

where,
$a = \omega + \mu_v - \dfrac{\beta_1\Pi_v}{\mu_v}$,
$b = \omega\mu_v(1 - \mathfrak{R}_\circ)$.

We observe that the roots of the quadratic equation (5.3.2) have negative real parts if $\mathfrak{R}_\circ < 1$. If $\mathfrak{R}_\circ = 1$, one root of Eq. (5.3.2) is 0. This fact does not guarantee that all eigenvalues have negative real parts. It will only be possible in case of real roots. If $\mathfrak{R}_\circ > 1$, one of the root of (5.3.2) has positive real part. The above discussion leads to the following theorem.

Theorem 5.3.1. *The disease-free equilibrium of system (5.1.3) is locally asymptotically stable in Ω if $\mathfrak{R}_\circ < 1$ and, it is unstable if $\mathfrak{R}_\circ > 1$.*

Now, we analyze the global behavior of the disease-free equilibrium E_0. The following theorem provides the global property of the system.

Theorem 5.3.2. *If $\mathfrak{R}_\circ \leq 1$, then the infection-free equilibrium E_0 is globally asymptotically stable in the interior of Ω.*

Proof. The following Lyapunov function is proposed to establish the global stability of disease-free equilibrium.

$$L = \zeta \frac{\Pi_v}{\omega} I_h + \mu_v I_v.$$

Taking the time derivative of L along the solutions of (5.1.3), we have

$$\begin{aligned}
L' &= \zeta \frac{\Pi_v}{\omega} I_h' + \mu_v I_v' \\
&= \zeta \frac{\Pi_v}{\omega} \left(\frac{\delta_1 S_h I_v}{1+\alpha_1 I_v} + \frac{\delta_2 \theta S_h I_v}{1+\alpha_1 I_v} - \omega I_h\right) + \mu_v \left[\zeta \left(\frac{\Pi_v}{\mu_v} - I_v\right) \frac{I_h}{1+\alpha_2 I_h} + \beta_1 \left(\frac{\Pi_v}{\mu_v} - I_v\right) I_v - \mu_v I_v\right] \\
&\leq \zeta \frac{\Pi_v}{\omega} (\delta_1 S_h I_v + \delta_2 \theta S_h I_v - \omega I_h) + \mu_v \left[\left(\zeta \frac{\Pi_v}{\mu_v} \frac{I_h}{1+\alpha_2 I_h} - \zeta \frac{I_v I_h}{1+\alpha_2 I_h}\right) + \left(\beta_1 \frac{\Pi_v}{\mu_v} I_v - \beta_1 I_v I_v\right)\right] \\
&\quad -{\mu_v}^2 I_v \\
&< (\delta_1 + \delta_2 \theta) \zeta \frac{\Pi_v}{\omega} \frac{\Pi_h}{\mu_1} I_v + \mu_v \beta_1 \frac{\Pi_v}{\mu_v} I_v - {\mu_v}^2 I_v - \zeta \Pi_v I_h + \zeta \Pi_v I_h - \mu_v \zeta I_v \frac{I_h}{1+\alpha_2 I_h} - \mu_v \beta_1 I_v^2 \\
&= I_v \left[\mu_v^2 (\mathfrak{R}_\circ - 1) - \mu_v \zeta \frac{I_h}{1+\alpha_2 I_h} - \mu_v \beta_1 I_v\right] \leq 0.
\end{aligned}$$

Thus $L'(t)$is negative if $\mathfrak{R}_\circ \leq 1$. When $\mathfrak{R}_\circ < 1$, the derivative $L' = 0$ if and only if $I_v = 0$, while in the case $\mathfrak{R}_\circ = 1$, the derivative $L' = 0$ if and only if $I_h = 0$ or $I_v = 0$. Consequently, the largest compact invariant set in $\{(S_h, I_h, I_v \in \Omega), L' = 0\}$, when $\mathfrak{R}_\circ \leq 1$, is the singleton E_0. Hence, by LaSalle's invariance principle [46], E_0 is globally asymptotically stable in Ω. This completes the proof. □

5.4 Stability of Endemic Equilibrium

The global stability of endemic equilibrium is proved by the method discussed in chapter 2. The uniform persistence of the system (5.1.3) can be easily proved by lemma (2.5.2). We choose a suitable vector norm $|.|$ in R^3 and a 3×3 matrix valued function $P(x)$ as defined in (4.2.4). Linearizing system (5.1.3) about an endemic equilibrium E^* gives the following Jacobian matrix

$$J = \begin{pmatrix} a_{11} & 0 & a_{13} \\ I_v \dfrac{\delta_1}{1+\alpha_1 I_v} + I_v \theta \dfrac{\delta_2}{1+\alpha_1 I_v} & -\omega & a_{23} \\ 0 & \dfrac{\zeta}{\mu_v \left(1+\alpha_2 I_h\right)^2} \left(\Pi_v - I_v \mu_v\right) & a_{33} \end{pmatrix},$$

where,

$$\begin{aligned}
a_{11} &= -\mu_1 - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v}\\
a_{13} &= -S_h\frac{\delta_1}{(1+\alpha_1 I_v)^2} - S_h\theta\frac{\delta_2}{(1+\alpha_1 I_v)^2}\\
a_{23} &= S_h\frac{\delta_1}{(1+\alpha_1 I_v)^2} + S_h\theta\frac{\delta_2}{(1+\alpha_1 I_v)^2}\\
a_{33} &= \frac{\beta_1}{\mu_v}(\Pi_v - 2I_v\mu_v) - \mu_v - I_h\frac{\zeta}{1+\alpha_2 I_h}
\end{aligned}$$

The second additive compound matrix of $J(E^*)$ is given by

$$J^{[2]} = \begin{pmatrix}
b_{11} & S_h\frac{\delta_1}{(1+\alpha_1 I_v)^2} + S_h\theta\frac{\delta_2}{(1+\alpha_1 I_v)^2} & b_{13}\\
\frac{\zeta}{\mu_v(1+\alpha_2 I_h)^2}(\Pi_v - I_v\mu_v) & b_{22} & 0\\
0 & I_v\frac{\delta_1}{1+\alpha_1 I_v} + I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} & b_{33}
\end{pmatrix},$$

where,

$$\begin{aligned}
b_{11} &= -\mu_1 - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} - \omega,\\
b_{13} &= S_h\frac{\delta_1}{(1+\alpha_1 I_v)^2} + S_h\theta\frac{\delta_2}{(1+\alpha_1 I_v)^2}\\
b_{22} &= -\mu_1 - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} + \frac{\beta_1}{\mu_v}(\Pi_v - 2I_v\mu_v) - \mu_v - I_h\frac{\zeta}{1+\alpha_2 I_h},\\
b_{33} &= -\omega + \frac{\beta_1}{\mu_v}(\Pi_v - 2I_v\mu_v) - \mu_v - I_h\frac{\zeta}{1+\alpha_2 I_h}.
\end{aligned}$$

The matrix $B = P_f P^{-1} + PJ^{[2]}P^{-1}$ can be written in block form as $B = \begin{pmatrix} B_{11} & B_{12}\\ B_{21} & B_{22}\end{pmatrix}$, with

$$B_{11} = -\mu_1 - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} - \omega,$$

$$B_{12} = \left(\frac{I_v}{I_h}(S_h\frac{\delta_1}{(1+\alpha_1 I_v)^2} + S_h\theta\frac{\delta_2}{(1+\alpha_1 I_v)^2}), \frac{I_v}{I_h}(S_h\frac{\delta_1}{(1+\alpha_1 I_v)^2} + S_h\theta\frac{\delta_2}{(1+\alpha_1 I_v)^2})\right),$$

$$B_{21} = \begin{pmatrix} \frac{I_h}{I_v}\frac{\zeta}{\mu_v(1+\alpha_2 I_h)^2}(\Pi_v - I_v\mu_v)\\ 0\end{pmatrix},$$

$$B_{22} = \begin{pmatrix} L_{22} + \frac{I_h'}{I_h} - \frac{I_v'}{I_v} & 0\\ I_v\frac{\delta_1}{1+\alpha_1 I_v} + I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} & L_{33} + \frac{I_h'}{I_h} - \frac{I_v'}{I_v}\end{pmatrix},$$

where

$$L_{22} = -\mu_1 - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v} + \frac{\beta_1}{\mu_v}(\Pi_v - 2I_v\mu_v) - \mu_v - I_h\frac{\zeta}{1+\alpha_2 I_h},$$

$$L_{33} = -\omega + \frac{\beta_1}{\mu_v}(\Pi_v - 2I_v\mu_v) - \mu_v - I_h\frac{\zeta}{1+\alpha_2 I_h}.$$

Consider the norm in R^3 as: $\mid (u,v,w) \mid = max(\mid u \mid, \mid v \mid + \mid w \mid)$ where (u,v,w) denotes the vector in R^3. The Lozinskiĭ measure with respect to this norm is defined as

$$\mu(B) \leq sup(f_1, f_2),$$

where

$$f_1 = \mu_1(B_{11}) + \mid B_{12} \mid, \quad f_2 = \mu_1(B_{22}) + \mid B_{21} \mid .$$

From system (5.1.3) we can write

$\frac{I_h'}{I_h} = \frac{I_v}{I_h}(S_h \frac{\delta_1}{1+\alpha_1 I_v} + S_h \theta \frac{\delta_2}{1+\alpha_1 I_v}) - \omega,$

$\frac{I_v'}{I_v} = \frac{I_h}{I_v} \frac{\zeta}{\mu_v(1+\alpha_2 I_h)}(\Pi_v - \mu_v I_v) + \frac{\beta_1}{\mu_v}(\Pi_v - \mu_v I_v) - \mu_v.$

Since B_{11} is a scalar, its Lozinskiĭ measure with respect to any vector norm in R^1 will be equal to B_{11}. Thus

$$B_{11} = -\mu_1 - I_v \frac{\delta_1}{1+\alpha_1 I_v} - I_v \theta \frac{\delta_2}{1+\alpha_1 I_v} - \omega,$$

$$\mid B_{12} \mid = \frac{I_v}{I_h}(S_h \frac{\delta_1}{(1+\alpha_1 I_v)^2} + S_h \theta \frac{\delta_2}{(1+\alpha_1 I_v)^2}),$$

and f_1 will become

$f_1 = -\mu_1 - I_v \frac{\delta_1}{1+\alpha_1 I_v} - I_v \theta \frac{\delta_2}{1+\alpha_1 I_v} - \omega + \frac{I_v}{I_h}(S_h \frac{\delta_1}{(1+\alpha_1 I_v)^2} + S_h \theta \frac{\delta_2}{(1+\alpha_1 I_v)^2})$

$f_1 = \frac{I_h'}{I_h} - \mu_1 - I_v \frac{\delta_1}{1+\alpha_1 I_v} - I_v \theta \frac{\delta_2}{1+\alpha_1 I_v}.$

Also $\mid B_{21} \mid = \frac{I_h}{I_v} \frac{\zeta}{\mu_v (1+\alpha_2 I_h)^2} (\Pi_v - I_v \mu_v)$, $\mid B_{12} \mid$ and $\mid B_{21} \mid$ are the operator norms of B_{12} and B_{21} which are mapping from R^2 to R and from R to R^2 respectively, and R^2 is endowed with the l_1 norm. $\mu_1(B_{22})$ is the Lozinskiĭ measure of 2×2 matrix B_{22} with respect to l_1 norm in R^2.

$\mu_1(B_{22}) = \sup\{L_{22} + \frac{I_h'}{I_h} - \frac{I_v'}{I_v} + I_v \frac{\delta_1}{1+\alpha_1 I_v} + I_v \theta \frac{\delta_2}{1+\alpha_1 I_v}, L_{33} + \frac{I_h'}{I_h} - \frac{I_v'}{I_v}\}$

$= \sup\{-\mu_1 - I_v \frac{\delta_1}{1+\alpha_1 I_v} - I_v \theta \frac{\delta_2}{1+\alpha_1 I_v} + \frac{\beta_1}{\mu_v}(\Pi_v - 2 I_v \mu_v) - \mu_v - I_h \frac{\zeta}{1+\alpha_2 I_h} + \frac{I_h'}{I_h} - \frac{I_h}{I_v} \frac{\zeta}{\mu_v(1+\alpha_2 I_h)}(\Pi_v - \mu_v I_v) - \frac{\beta_1}{\mu_v}(\Pi_v - \mu_v I_v) + \mu_v + I_v \frac{\delta_1}{1+\alpha_1 I_v} + I_v \theta \frac{\delta_2}{1+\alpha_1 I_v},$

$-\omega + \frac{\beta_1}{\mu_v}(\Pi_v - 2 I_v \mu_v) - \mu_v - I_h \frac{\zeta}{1+\alpha_2 I_h} + \frac{I_h'}{I_h} - \frac{I_h}{I_v} \frac{\zeta}{\mu_v(1+\alpha_2 I_h)}(\Pi_v - \mu_v I_v) - \frac{\beta_1}{\mu_v}(\Pi_v - \mu_v I_v) + \mu_v\}$

$\mu_1(B_{22}) = \frac{I_h'}{I_h} - \frac{\beta_1}{\mu_v}(I_v \mu_v) - \frac{I_h}{I_v} \frac{\zeta}{\mu_v(1+\alpha_2 I_h)}(\Pi_v - \mu_v I_v) - \widetilde{\zeta},$

where

$\widetilde{\zeta} = \min\{I_h \frac{\zeta}{1+\alpha_2 I_h} + \mu_1, \omega + I_h \frac{\zeta}{1+\alpha_2 I_h}\}$. Hence $f_2 \leq \frac{I_h'}{I_h} - \frac{\beta_1}{\mu_v}(I_v \mu_v) - \widetilde{\zeta}.$

Thus

$$\mu(B) = \sup\{f_1, f_2\} \leq \sup\{\frac{I_h'}{I_h} - \mu_h - I_v\frac{\delta_1}{1+\alpha_1 I_v} - I_v\theta\frac{\delta_2}{1+\alpha_1 I_v}, \frac{I_h'}{I_h} - \frac{\beta_1}{\mu_v}(I_v\mu_v) - \widetilde{\zeta}\},$$

$$\mu(B) \leq \frac{I_h'}{I_h} - \widetilde{\beta}_1,$$

where

$\widetilde{\beta}_1 = \min\{\mu_1 + I_v\frac{\delta_1}{1+\alpha_1 I_v} + I_v\theta\frac{\delta_2}{1+\alpha_1 I_v}, \frac{\beta_1}{\mu_v}(I_v\mu_v) + \widetilde{\zeta}\}$. Since (5.1.3) is uniformly persistent when $\mathfrak{R}_o > 1$, so for $T > 0$ such that $t > T$ implies $I_h(t) \geq c$, $I_v(t) \geq c$ and $\frac{1}{t}\log I_h(t) < \frac{\widetilde{\beta}_1}{2}$ for all $(S_h(0), I_h(0), I_v(0)) \in K$.

Thus

$$\frac{1}{t}\int_0^t \mu(B)dt < \frac{\log I_h(t)}{t} - \widetilde{\beta}_1 < -\frac{\widetilde{\beta}_1}{2},$$

for all $(S_h(0), I_h(0), I_v(0)) \in K$, which further implies that $\bar{q}_2 < 0$. Therefore all the conditions of theorem (2.5.1) are satisfied. Hence unique endemic equilibrium E^* is globally stable in Ω.

The variation of infected hosts and infected vectors is shown in Fig.5.2.

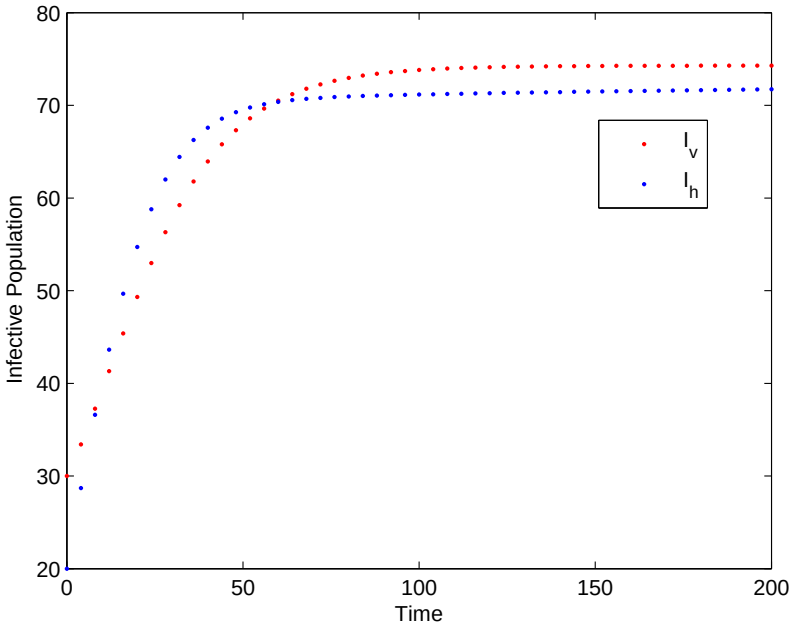

Figure 5.2: The infected population approaches endemic equilibrium for $\mathfrak{R}_o > 1$. The Parameter values are given in the following table.

Parameter	Value	Parameter	Value
Π_h	0.22	Π_v	0.32
μ_1	0.00002	ω	0.003
μ_v	0.004	ζ	0.0004
β_1	0.00034	δ_1	0.0016
δ_2	0.00016	θ	0.00301
α_1	0.001	α_2	0.001

5.5 Model with standard incidence

The model (5.1.1) is modified by considering standard incidence and is given by the following diagram:

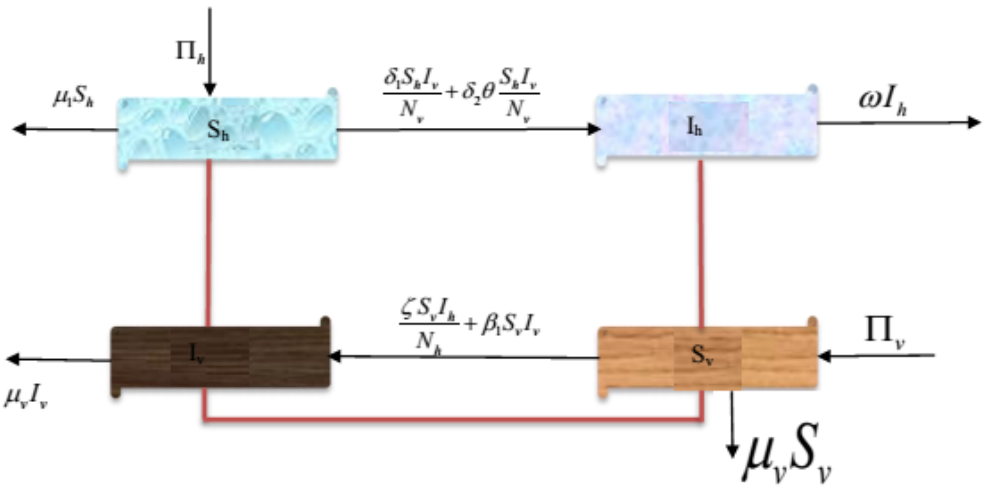

Figure 5.3: Flow diagram of pine wilt disease model with standard incidence

Mathematical model is given by

$$\frac{dS_h}{dt} = \Pi_h - \frac{\delta_1 S_h I_v}{N_v} - \frac{\delta_2 \theta S_h I_v}{N_v} - \mu_1 S_h$$

$$\begin{aligned}
\frac{dI_h}{dt} &= \frac{\delta_1 S_h I_v}{N_v} + \frac{\delta_2 \theta S_h I_v}{N_v} - \omega I_h \\
\frac{dS_v}{dt} &= \Pi_v - \frac{\zeta S_v I_h}{N_h} - \beta_1 S_v I_v - \mu_v S_v \\
\frac{dI_v}{dt} &= \frac{\zeta S_v I_h}{N_h} + \beta_1 S_v I_v - \mu_v I_v
\end{aligned} \tag{5.5.1}$$

The total vector population is again satisfied by the equation (5.1.2) and reduced model is given by

$$\begin{aligned}
\frac{dS_h}{dt} &= \Pi_h - \frac{\delta_1 S_h I_v}{N_v} - \frac{\delta_2 \theta S_h I_v}{N_v} - \mu_1 S_h \\
\frac{dI_h}{dt} &= \frac{\delta_1 S_h I_v}{N_v} + \frac{\delta_2 \theta S_h I_v}{N_v} - \omega I_h \\
\frac{dI_v}{dt} &= \zeta(\frac{\Pi_v}{\mu_v} - I_v)\frac{I_h}{N_h} + \beta_1(\frac{\Pi_v}{\mu_v} - I_v)I_v - \mu_v I_v
\end{aligned} \tag{5.5.2}$$

The system (5.5.2) is studied in the set given in (5.1.4).

5.5.1 Existence of Equilibria

The basic reproduction number of model (5.5.2) is given by

$$\mathfrak{R}_\circ = \frac{\beta_1 \Pi_v}{\mu_v^2} + \frac{\zeta}{\omega \mu_v}(\delta_1 + \theta\delta_2). \tag{5.5.3}$$

The disease free equilibrium is $E_0(\frac{\Pi_h}{\mu_1}, 0, 0)$ and for $\mathfrak{R}_\circ > 1$, endemic equilibrium $E^*(S_h^{**}, I_h^{**}, I_v^{**})$, with

$$\begin{aligned}
S_h^{**} &= \frac{\Pi_h - \omega I_h^{**}}{\mu_1}, \\
I_h^{**} &= \frac{\mu_v(\delta_1 + \delta_2\theta)\Pi_h I_v^{**}}{\omega[\mu_h \Pi_v + \mu_v(\delta_1 + \delta_2\theta)I_v^{**}]}
\end{aligned} \tag{5.5.4}$$

and I_v^{**} is the root of the following equation

$$AI_v^{**2} + BI_v^{**} + CI_v^{**} = 0, \tag{5.5.5}$$

where

$$\begin{aligned}
A &= \beta_1 \Pi_h \mu_v (\delta_1 + \theta\delta_2), \\
B &= \frac{\omega}{\mu_1}\Pi_h[(\delta_1 + \theta\delta_2)(\mu_v^2 - \beta_1\Pi_v) + \beta_1\mu_1\Pi_v],
\end{aligned}$$

$$C = \Pi_h \Pi_v \omega \mu_v (1 - \Re_o). \tag{5.5.6}$$

From (5.5.6), we see that $\Re_o > 1$ if and only if, $C < 0$. Since $A > 0$, Eq.(5.5.5) has a unique positive root in feasible region. If $\Re_o < 1$, then $C > 0$. Also, it can be easily seen that $B > 0$ for $\Re_o < 1$. Thus, by considering the shape of the graph of Eq.(5.5.5) (and noting that $C > 0$), we have that there will be zero (positive) endemic equilibrium in this case. Therefore, we can conclude that if $\Re_o < 1$, Eq.(5.5.5) has no positive root in the feasible region. If, $\Re_o > 1$, Eq.(5.5.5) has a unique positive root in the feasible region. This result is summarized below.

Theorem 5.5.1. *System* (5.5.2) *always has the infection-free equilibrium* E_0. *If* $\Re_o > 1$, *system (5.5.2) has a unique endemic equilibrium* $E^*(S_h^{**}, I_h^{**}, I_v^{**})$ *defined by (5.5.4) and (5.5.5).*

5.6 stability analysis

Now, we analyze the global behaviour of the disease-free equilibrium E_0 and endemic equilibrium $E^*(S_h^{**}, I_h^{**}, I_v^{**})$.

5.6.1 Global stability of disease-free equilibrium

The following theorem provides the global property of the system for the disease-free equilibrium E_0.

Theorem 5.6.1. *If* $\Re_o \leq 1$, *then the infection-free equilibrium* E_0 *is globally asymptotically stable in the interior of* Ω.

Proof. The following Lyapunov function is proposed to establish the global stability of disease-free equilibrium.

$$L = \zeta \frac{\mu_1}{\Pi_h} \frac{\Pi_v}{\omega \mu_v} I_h + I_v$$

Taking the time derivative of L along the solutions of (5.5.2), we have

$$\begin{aligned}
L' &= \zeta \frac{\mu_1}{\Pi_h} \frac{\Pi_v}{\omega \mu_v} I_h' + I_v' \\
L' &= \zeta \frac{\mu_1}{\Pi_h} \frac{\Pi_v}{\omega \mu_v} \left(\frac{\mu_v}{\Pi_v} \delta_1 S_h I_v + \frac{\mu_v}{\Pi_v} \delta_2 \theta S_h I_v - \omega I_h\right) + \left[\zeta \left(\frac{\Pi_v}{\mu_v} - I_v\right) \frac{I_h}{S_h + I_h} + \beta_1 \left(\frac{\Pi_v}{\mu_v} - I_v\right) I_v - \mu_v I_v\right] \\
&\leq \zeta \frac{\mu_1}{\Pi_h} \frac{\Pi_v}{\omega \mu_v} \left[\frac{\mu_v}{\Pi_v} (\delta_1 + \delta_2 \theta) S_h I_v - \omega I_h\right] + \left[\left(\zeta \frac{\Pi_v}{\mu_v} \frac{\mu_1}{\Pi_h} I_h - \zeta I_v \frac{I_h}{S_h + I_h}\right) + \left(\beta_1 \frac{\Pi_v}{\mu_v} I_v - \beta_1 I_v^2\right)\right.
\end{aligned}$$

$$-\mu_v I_v]$$

$$< \quad (\delta_1 + \delta_2\theta)\frac{\zeta}{\omega} I_v - \beta_1 I_v \frac{I_h}{S_h + I_h} + \beta_1 \frac{\Pi_v}{\mu_v} I_v - \beta_1 I_v^2 - \mu_v I_v$$

$$= \quad \mu_v(\mathfrak{R}_\circ - 1) I_v - \zeta I_v \frac{I_h}{S_h + I_h} - \beta_1 I_v^2$$

□

Thus $L'(t)$ is negative if $\mathfrak{R}_\circ \leq 1$. The derivative $L' = 0$ if and only if $I_v = 0$ and $I_h = 0$ whenever $\mathfrak{R}_\circ \leq 1$. Consequently, the largest compact invariant set in $\{(S_h, I_h, I_v \in \Omega), L' = 0\}$, when $\mathfrak{R}_\circ \leq 1$, is the singelton E_0. Hence, LaSalle's invariance principle [46] implies that E_0 is globally asymptotically stable in Ω. This completes the proof.

5.6.2 Global stability of endemic equilibrium

We shall discuss the global stability of the endemic equilibrium E^*. To show the global stability we shall follow the same approach as described in the previous section. The following Jacobian matrix is obtained by linearizing system (5.5.2) about an endemic equilibrium E^*.

$$J = \begin{pmatrix} -\frac{1}{\Pi_v}(\mu_1\Pi_v + I_v\delta_1\mu_v + I_v\theta\delta_2\mu_v) & 0 & -S_h\frac{\mu_v}{\Pi_v}(\delta_1 + \theta\delta_2) \\ I_v\frac{\mu_v}{\Pi_v}(\delta_1 + \theta\delta_2) & -\omega & S_h\frac{\mu_v}{\Pi_v}(\delta_1 + \theta\delta_2) \\ -I_h\frac{\zeta}{\mu_v}\frac{\Pi_v - I_v\mu_v}{(S_h + I_h)^2} & S_h\frac{\zeta}{\mu_v}\frac{\Pi_v - I_v\mu_v}{(S_h + I_h)^2} & a_{33} \end{pmatrix},$$

where,

$$a_{33} = -\frac{1}{\mu_v}(\mu_v^2 - \beta_1\Pi_v + 2I_v\beta_1\mu_v) - \frac{I_h\zeta\mu_v}{\mu_v(S_h + I_h)}.$$

The second additive compound matrix of $J(E^*)$ is given by

$$J^{[2]} = \begin{bmatrix} b_{11} & S_h\frac{\mu_v}{\Pi_v}(\delta_1 + \theta\delta_2) & S_h\frac{\mu_v}{\Pi_v}(\delta_1 + \theta\delta_2) \\ S_h\frac{\zeta}{\mu_v}\frac{\Pi_v - I_v\mu_v}{(S_h + I_h)^2} & b_{22} & 0 \\ I_h\frac{\zeta}{\mu_v}\frac{\Pi_v - I_v\mu_v}{(S_h + I_h)^2} & I_v\frac{\mu_v}{\Pi_v}(\delta_1 + \theta\delta_2) & b_{33} \end{bmatrix},$$

where,

$$b_{11} = -\frac{1}{\Pi_v}(\mu_1\Pi_v + I_v\delta_1\mu_v + I_v\theta\delta_2\mu_v) - \omega$$

$$b_{22} = -\frac{1}{\Pi_v}(\mu_1\Pi_v + I_v\delta_1\mu_v + I_v\theta\delta_2\mu_v) - \frac{1}{\mu_v}(\mu_v^2 - \beta_1\Pi_v + 2I_v\beta_1\mu_v) - \frac{I_h\zeta\mu_v}{\mu_v(S_h + I_h)}$$

$$b_{33} = -\omega - \frac{1}{\mu_v}(\mu_v^2 - \beta_1\Pi_v + 2I_v\beta_1\mu_v) - \frac{I_h\zeta\mu_v}{\mu_v(S_h + I_h)}.$$

The matrix $G = P_f P^{-1} + PJ^{[2]}P^{-1}$ can be written in block form as

$$G = \begin{pmatrix} G_{11} & G_{12} \\ G_{21} & G_{22} \end{pmatrix}$$

with

$$G_{11} = -\frac{1}{\Pi_v}\left(\mu_1\Pi_v + I_v\delta_1\mu_v + I_v\theta\delta_2\mu_v\right) - \omega$$

$$G_{12} = \left(\frac{S_hI_v}{I_h}\frac{\mu_v}{\Pi_v}\left(\delta_1 + \theta\delta_2\right), \frac{S_hI_v}{I_h}\frac{\mu_v}{\Pi_v}\left(\delta_1 + \theta\delta_2\right)\right)$$

$$G_{21} = \begin{pmatrix} \frac{I_h}{I_v}S_h\frac{\zeta}{\mu_v}\frac{\Pi_v - I_v\mu_v}{\left(S_h + I_h\right)^2} \\ \frac{I_h}{I_v}I_h\frac{\zeta}{\mu_v}\frac{\Pi_v - I_v\mu_v}{\left(S_h + I_h\right)^2} \end{pmatrix}$$

$$G_{22} = \begin{pmatrix} M_{22} + \frac{I_h'}{I_h} - \frac{I_v'}{I_v} & 0 \\ I_v\frac{\mu_v}{\Pi_v}\left(\delta_1 + \theta\delta_2\right) & M_{33} + \frac{I_h'}{I_h} - \frac{I_v'}{I_v} \end{pmatrix}$$

where

$$M_{22} = -\frac{1}{\Pi_v}\left(\mu_1\Pi_v + I_v\delta_1\mu_v + I_v\theta\delta_2\mu_v\right) - \frac{1}{\mu_v}(\mu_v^2 - \beta_1\Pi_v + 2I_v\beta_1\mu_v) - \frac{I_h\zeta\mu_v}{\mu_v\left(S_h + I_h\right)}$$

$$M_{33} = -\omega - \frac{1}{\mu_v}(\mu_v^2 - \beta_1\Pi_v + 2I_v\beta_1\mu_v) - \frac{I_h\zeta\mu_v}{\mu_v\left(S_h + I_h\right)}$$

Consider the norm in R^3 as: $\mid (u,v,w) \mid = max(\mid u \mid, \mid v \mid + \mid w \mid)$ where (u,v,w) denotes the vector in R^3. The Lozinskiĭ measure with respect to this norm is defined as $\mu(B) \leq sup(g_1, g_2)$, where

$$g_1 = \mu_1(G_{11}) + \mid G_{12} \mid, \quad g_2 = \mu_1(G_{22}) + \mid G_{21} \mid .$$

From system (5.5.2) we can write $\frac{I_h'}{I_h} = \frac{I_v}{I_h}(\frac{\mu_v}{\Pi_v}\delta_1 S_h + \frac{\mu_v}{\Pi_v}\delta_2\theta S_h) - \omega$

$\frac{I_v'}{I_v} = \frac{I_h}{I_v}\frac{\zeta}{\left(S_h + I_h\right)}(\frac{\Pi_v}{\mu_v} - I_v) + \beta_1(\frac{\Pi_v}{\mu_v} - I_v) - \mu_v$

Since G_{11} is a scalar, its Lozinskiĭ measure with respect to any vector norm in R^1 will be equal to G_{11}. Thus

$$G_{11} = -\frac{1}{\Pi_v}\left(\mu_1\Pi_v + I_v\delta_1\mu_v + I_v\theta\delta_2\mu_v\right) - \omega,$$

$$\mid G_{12} \mid = \frac{S_hI_v}{I_h}\frac{\mu_v}{\Pi_v}\left(\delta_1 + \theta\delta_2\right),$$

and g_1 will become

$$g_1 = -\frac{1}{\Pi_v}\left(\mu_1\Pi_v + I_v\delta_1\mu_v + I_v\theta\delta_2\mu_v\right) - \omega + \frac{I_v}{I_h}(S_h\frac{\mu_v}{\Pi_v}\left(\delta_1 + \theta\delta_2\right)),$$

$$= \frac{I_v}{I_h}(S_h\frac{\mu_v}{\Pi_v}\left(\delta_1 + \theta\delta_2\right)) - \omega - \frac{1}{\Pi_v}\left(\mu_1\Pi_v + I_v\delta_1\mu_v + I_v\theta\delta_2\mu_v\right),$$

$$g_1 = \frac{I_h'}{I_h} - \frac{1}{\Pi_v}\left(\mu_1\Pi_v + I_v\delta_1\mu_v + I_v\theta\delta_2\mu_v\right).$$

$\mid G_{12} \mid$ and $\mid G_{21} \mid$ are the operator norms of G_{12} and G_{21} which are mapping from R^2 to R and from R to R^2 respectively, and R^2 is endowed with the l_1 norm. $\mu_1(G_{22})$ is the Lozinskiĭ measure of 2×2 matrix G_{22} with respect to l_1 norm in R^2.

$$\mu(G_{22}) = \sup\{M_{22} + \frac{I_h'}{I_h} - \frac{I_v'}{I_v} + I_v\frac{\delta_1}{I_v\alpha_1 + 1} + I_v\theta\frac{\delta_2}{I_v\alpha_2 + 1}, M_{33} + \frac{I_h'}{I_h} - \frac{I_v'}{I_v}\}$$

$= \sup\{-\frac{1}{\Pi_v}(\mu_1\Pi_v + I_v\delta_1\mu_v + I_v\theta\delta_2\mu_v) - \frac{1}{\mu_v}(\mu_v^2 - \beta_1\Pi_v + 2I_v\beta_1\mu_v) - \frac{I_h\zeta\mu_v}{\mu_v(S_h+I_h)} + \frac{I_h'}{I_h} - \frac{I_h}{I_v}\frac{\zeta}{(S_h+I_h)}(\frac{\Pi_v}{\mu_v} - I_v) - \beta_1(\frac{\Pi_v}{\mu_v} - I_v) + \mu_v,$

$-\omega - \frac{1}{\mu_v}(\mu_v^2 - \beta_1\Pi_v + 2I_v\beta_1\mu_v) - \frac{I_h\zeta\mu_v}{\mu_v(S_h+I_h)} + \frac{I_h'}{I_h} - \frac{I_h}{I_v}\frac{\zeta}{(S_h+I_h)}(\frac{\Pi_v}{\mu_v} - I_v) - \beta_1(\frac{\Pi_v}{\mu_v} - I_v) + \mu_v\}$

$\mu(G_{22}) = \frac{I_h'}{I_h} - \frac{I_h}{I_v}\frac{\zeta}{(S_h+I_h)}(\frac{\Pi_v}{\mu_v} - I_v) - \frac{I_h\zeta}{(S_h+I_h)} - \beta_1 I_v - \widetilde{\gamma_1}$

where

$\widetilde{\gamma_1} = \min\{\frac{1}{\Pi_v}(\mu_1\Pi_v + I_v\delta_1\mu_v + I_v\theta\delta_2\mu_v), \omega\}$ Hence

$g_2 \leq \frac{I_h'}{I_h} - \frac{I_h\zeta}{(S_h+I_h)} - \beta_1 I_v - \widetilde{\gamma_1}$

Thus

$\mu(G) = \sup\{g_1, g_2\} \leq \sup\{\frac{I_h'}{I_h} - \frac{1}{\Pi_v}(\mu_1\Pi_v + I_v\delta_1\mu_v + I_v\theta\delta_2\mu_v), \frac{I_h'}{I_h} - \frac{I_h\zeta}{(S_h+I_h)} - \beta_1 I_v - \widetilde{\gamma_1}\}$

$\mu(G) \leq \frac{I_h'}{I_h} - \widetilde{\gamma_2}$ where

$\widetilde{\gamma_2} = \min\{\frac{1}{\Pi_v}(\mu_1\Pi_v + I_v\delta_1\mu_v + I_v\theta\delta_2\mu_v), \frac{I_h\zeta}{(S_h+I_h)} + \beta_1 I_v + \widetilde{\gamma_1}\}$. Since (5.5.2) is uniformly persistent when $\mathfrak{R}_\circ > 1$, so for $T > 0$ such that $t > T$ implies $I_h(t) \geq c$, $I_v(t) \geq c$ and $\frac{1}{t}\log I_h(t) < \frac{\widetilde{\gamma_2}}{2}$ for all $(S_h(0), I_h(0), I_v(0)) \in K$. Thus

$$\frac{1}{t}\int_0^t \mu(G)dt < \frac{\log I_h(t)}{t} - \widetilde{\gamma_2} < -\frac{\widetilde{\gamma_2}}{2}$$

for all $(S_h(0), I_h(0), I_v(0)) \in K$, which further implies that $\bar{q}_2 < 0$. Hence unique endemic equilibrium E^* is globally stable in Ω.

5.7 Discussions and Simulations

In this chapter, pine wilt disease transmission models with nonlinear incidence rates, standard incidence rates and horizontal transmission are proposed and analyzed. Bilinear incidence has been considered during mating. The basic reproduction numbers of the models are obtained and with the help of these reproduction numbers the asymptotic behaviour of the models are discussed. The variation of total population of model (5.5.1) is shown in Fig.5.4.

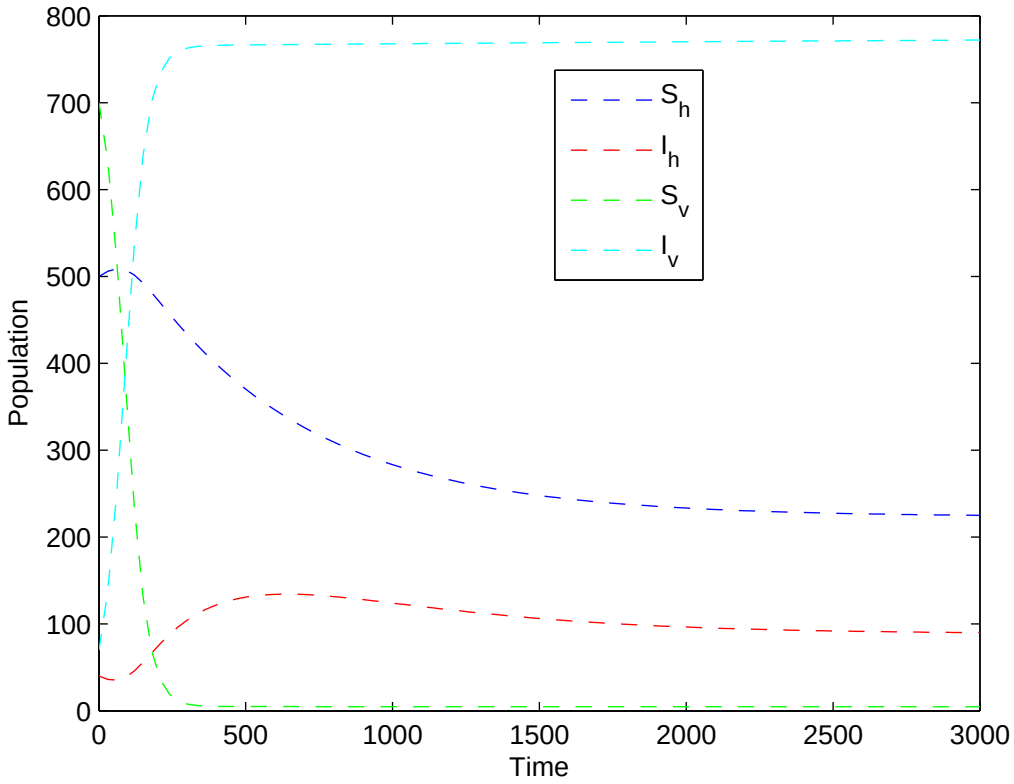

Figure 5.4: The total population approaches endemic equilibrium for $\mathfrak{R}_o > 1$. The parameter values are given in the following table.

Parameter	Value	Parameter	Value
Π_h	0.4	Π_v	0.13
μ_1	0.0002	ω	0.0035
μ_v	0.000165	ζ	0.0004
β_1	0.000034	δ_1	0.0016
δ_2	0.00016	θ	0.00301

We know that the pine wilt disease will disappear whenever reproductive number is less than unity. We shall identify which factors involve to reduce the reproductive number. From the expression given in (5.5.3) we see that,

$$\begin{aligned}\frac{\partial \mathfrak{R}_o}{\partial \mu_v} &= -\frac{1}{\omega \mu_v^3}\left(2\omega\beta_1\Pi_v + \zeta\delta_1\mu_v + \theta\zeta\delta_2\mu_v\right) < 0,\\ \frac{\partial \mathfrak{R}_o}{\partial \omega} &= -\frac{1}{\omega^2}\frac{\zeta}{\mu_v}\left(\delta_1 + \theta\delta_2\right) < 0.\end{aligned}$$

Thus the reproductive number $\mathfrak{R}_o$ is a decreasing function of μ_v and ω. The question

arises which parameter is more sensitive in order to decrease the reproductive number. By using the definition given in [57] and paramter values $\Pi_h = 100, \Pi_v = 400, \beta_1 = 0.00034, \mu_v = 0.00054, \zeta = 0.4, \mu_h = 0.000274, \omega = 0.000137, \delta_1 = 0.01, \theta = 0.00304, \delta_2 = 0.01$, we see that the sensitivity index of the reproductive number with respect to μ_v is -1.997 and with respect to ω is -0.1042. It means that the most sensitive parameter for $\mathfrak{R}_o$ is μ_v. Increasing the mortality rate of Monochamus alternatus by 10%, $\mathfrak{R}_o$ decreases almost 20%. Thus increasing the death rate of Monochamus alternatus is the efficient way to control the disease. Different strategies can be applied to increase the mortality rate of pine sawyer beetles. For example, setting out beetle traps, setting vertical wood traps, using chemicals to kill sawyer beetles, by cutting down dead pine trees and disposing off before the emergence of beetles.

The above mentioned measures are very effective to control pine wilt disease but they have not yet been practiced to eradicate pine wilt disease ultimately because these measures require more cost and labor and even entail danger of forest fires that most owners of forests hesitate to use these measures.

It has been shown that the endemic equilibrium E^* is globally asymptotically stable whenever $\mathfrak{R}_o > 1$. It means that if we do not control the pine wilt disease and allow it to spread at will, then the disease will be prevalent, and finally it will achieve a balance in the ecological environment. It will establish large economic losses if we do not control the parameters well which plays significant role to increase or decrease the endemic level of infected vectors and infected pine trees.

We can increase mortality rate of Monochamus alternatus by using chemicals and establishing beetle traps. In this way we can reduce the endemic level of infected vectors and infected pine trees. Figure5.5 shows different endemic levels of infective pines with respect to the parameter μ_v.

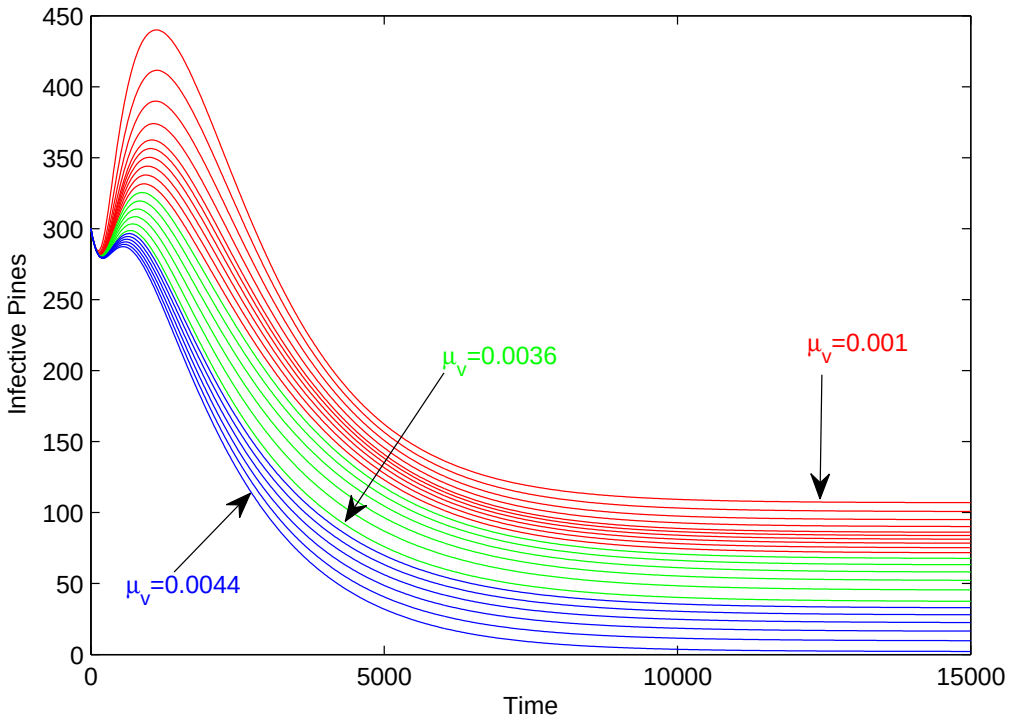

Figure 5.5: The effect of μ_v on infected pine trees. The endemic level of infected pine trees decreases with the increase of mortality rate of vectors. The parameter values are given in the following table.

Parameter	Value	Parameter	Value
Π_h	0.1	Π_v	0.7
μ_1	0.0002	ω	0.00637
μ_v	0.001-0.0044	ζ	0.00002
β_1	0.000034	δ_1	0.0011
δ_2	0.000001	θ	0.0003

We see that by increasing the mortality rate of Monochamus alternatus from 0.001 to 0.0044, the endemic level of infected pines reduces 105 to almost 5. Figure 5.6 shows different endemic levels of infective vectors with respect to the parameter μ_v.

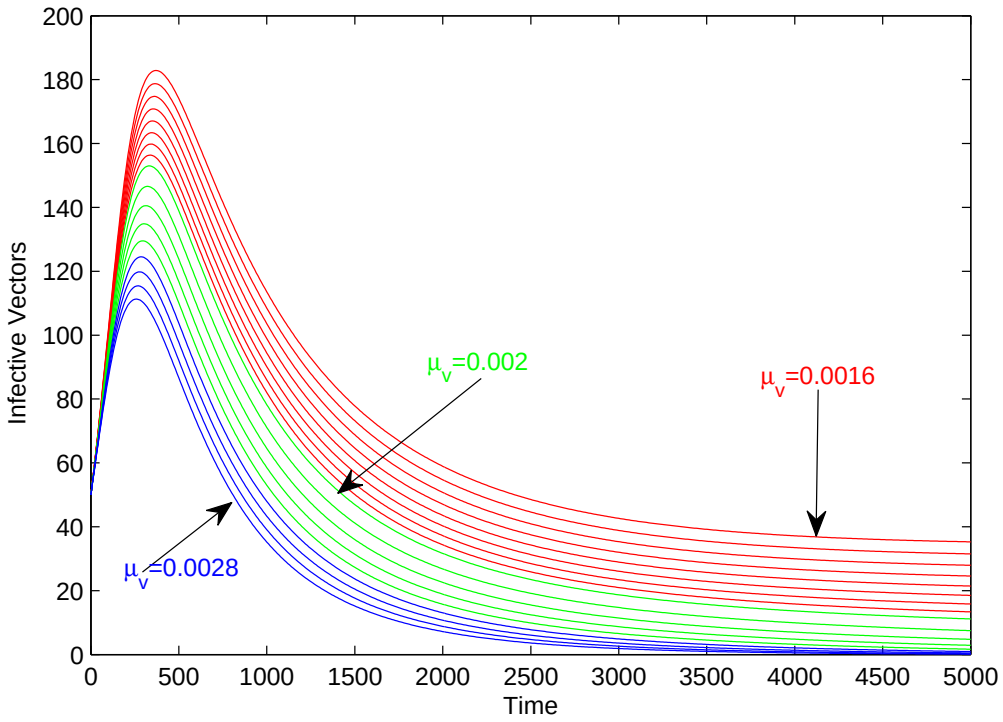

Figure 5.6: The effect of μ_v on infected vectors. The endemic level of infected vectors decreases with the increase of mortality rate of vectors. The parameter values are given in the following table.

Parameter	Value	Parameter	Value
Π_h	0.4	Π_v	0.13
μ_1	0.0002	ω	0.07
μ_v	0.0016-0.0028	ζ	0.0004
β_1	0.000034	δ_1	0.0016
δ_2	0.00016	θ	0.00301

We see that by increasing the mortality rate of vectors from 0.0013 to 0.002, the endemic level of infected vectors reduces from 345 to 190. Since the infected wood can not be used as wood products so the loss of afforestation will be small by increasing the death rate of Monochamus alternatus. The endemic level of infected pines can also be decreased by increasing the removal rate of infected wood. Fig.5.7 shows that by increasing the removal rate of infected pines from 0.0035 to 0.007, the endemic level of infected pines is decreased from 140 to 45.

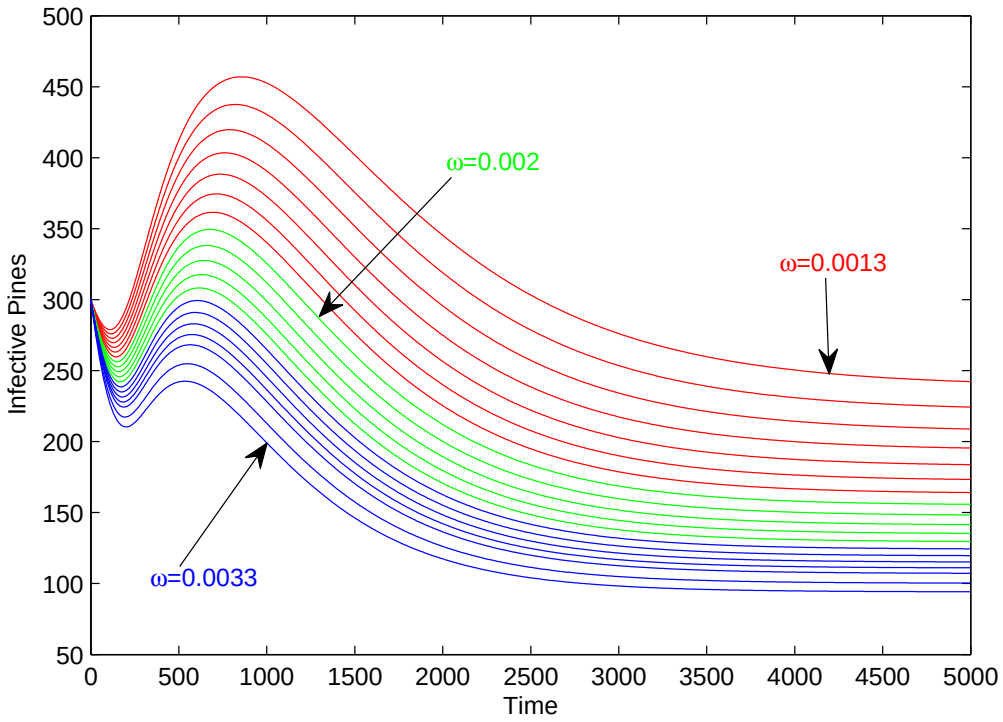

Figure 5.7: The effect of ω on infected pines. The endemic level of infected pines decreases with the increase of felling rate of infected pines. The parameter values are given in the following table.

Parameter	Value	Parameter	Value
Π_h	0.4	Π_v	0.13
μ_1	0.0002	ω	0.0013-0.0033
μ_v	0.000165	ζ	0.0004
β_1	0.000034	δ_1	0.0016
δ_2	0.00016	θ	0.00301

If we dispose off the infected pines before the emergence of bark beetles, it may also help us to reduce the endemic level of infected vectors.

Chapter 6

Vector-Host Model with Latent Stage having Partial Immunity

In this chapter a deterministic SEIRS epidemiological model is developed and analyzed. The complete global analysis for the equilibria of the model is analyzed by constructing Lyapunov functions. The explicit formula for the reproductive number is obtained and it is shown that the disease- free equilibrium always exists and is globally asymptotically stable whenever $\mathfrak{R}_{\mathfrak{o}}$ is below unity. Furthermore, it is proved that under suitable condition the disease persists at an endemic level when the reproductive number exceeds unity. The sensitivity analysis is also performed in order to determine the relative importance of model parameters to disease transmission and prevalence.

6.1 Model Description

The mathematical formulation of our model consists of the following contact parameters:
β_1 = The rate of direct transmission (possibly as a result of transfusion, transplantation, and use of needle–stick) of the disease.
β_2 = The transmission probability as a result of biting by an infected mosquito to the susceptible human.
$\beta_3(\beta)$= The transmission probability of transfering the infection from an infected nonimmune (partially immune) human to the susceptible mosquito.
The total human population denoted by $N_h(t)$ is sub-divided, into four mutually exclusive compartments according to the status of the disease:
Susceptible individuals $S_h(t)$, individuals possessing latent stage $E_h(t)$, infectious individuals $I_h(t)$ and recovered individuals having protective immunity $R_h(t)$. Thus $N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t)$. Similarly the total mosquito population at any time t is

denoted by $N_v(t) = S_h(t) + E_v(t) + I_v(t)$ where $S_v(t)$, $E_v(t)$ and $I_v(t)$ denote Susceptible, Exposed and Infectious vectors, respectively. In contrast to the human population, the vectors once infected remain microparasite carriers throughout their life. The model below is based on the following features:

1. Both humans and vectors are born susceptible.
2. Immunity in human population is temporary and lasts only for some time. Then they become susceptible to infection.
3. The class of persons who are partially immune to the disease may be infectious. We also assume that the infection acquired by a vector from an immune host is less infective than the infection acquired from a non-immune host.

The model can be illustrated in the following diagram.

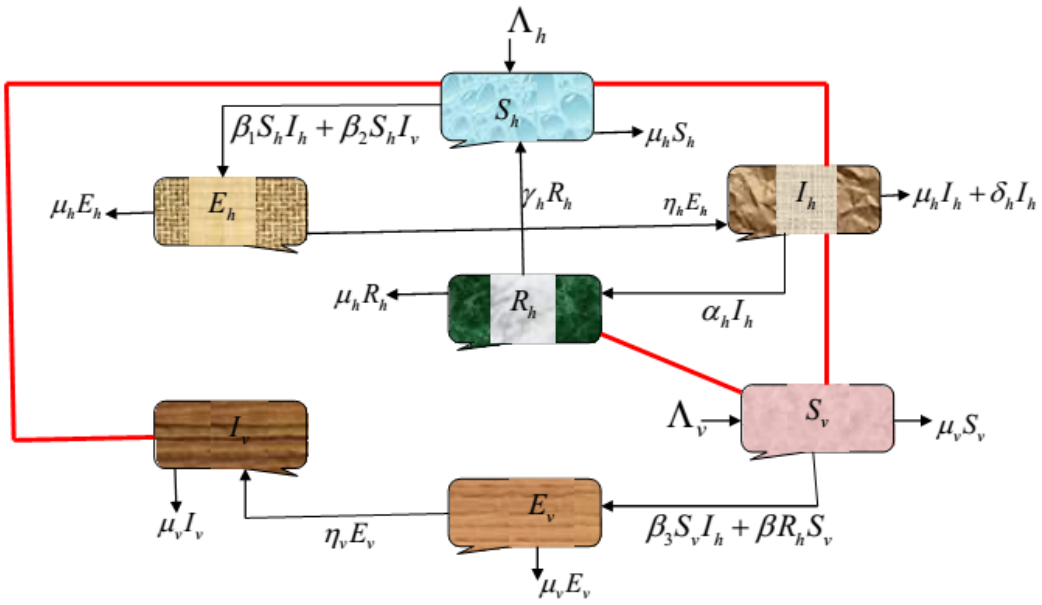

Figure 6.1: Flow diagram of Vector-Host model with partial immunity

Mathematical framework of the model is given in the following system of differential equations:

$$\frac{dS_h}{dt} = \Lambda_h - \beta_1 S_h I_h - \beta_2 S_h I_v - \mu_h S_h + \gamma_h R_h,$$

$$\frac{dE_h}{dt} = \beta_1 S_h I_h + \beta_2 S_h I_v - \eta_h E_h - \mu_h E_h,$$

$$\begin{aligned}
\frac{dI_h}{dt} &= \eta_h E_h - \alpha_h I_h - \mu_h I_h - \delta_h I_h, \\
\frac{dR_h}{dt} &= \alpha_h I_h - \gamma_h R_h - \mu_h R_h, \\
\frac{dS_v}{dt} &= \Lambda_v - \beta_3 S_v I_h - \beta R_h S_v - \mu_v S_v, \\
\frac{dE_v}{dt} &= \beta_3 S_v I_h + \beta R_h S_v - \eta_v E_v - \mu_v E_v, \\
\frac{dI_v}{dt} &= \eta_v E_v - \mu_v I_v. \qquad (6.1.1)
\end{aligned}$$

The model also satisfies the initial conditions,

$$S_h(0) \geq 0, E_h(0) \geq 0, I_h(0) \geq 0, R_h(0) \geq 0, S_v(0) \geq 0, E_v(0) \geq 0, I_v(0) \geq 0. \qquad (6.1.2)$$

In the above model Λ_h and Λ_v are the recruitment rates of humans and vectors respectively. Similarly μ_h and μ_v are the natural mortality rates of humans and vectors respectively. We assume that a disease may be fatal to some infectious host. As a result deaths due to the disease can be included in the model using the disease related death rate, δ_h from infectious class. Exposed humans develop clinical symptoms of the disease and move to the infectious class at rate η_h. The parameter α_h, is the recovery rate of humans. It is assumed that immune human individuals loose their immunity at a rate γ_h. The total human population is then governed by the following equation:

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta_h I_h. \qquad (6.1.3)$$

The given initial conditions (6.1.2) make sure that $N_h(0) \geq 0$. Thus the total population $N_h(t)$ remains positive and bounded for all finite time $t > 0$. Again the dynamics of the total vector population is governed by the equation:

$$\frac{dN_v}{dt} = \Lambda_v - \mu_v N_v. \qquad (6.1.4)$$

It follows from (6.1.3) and (6.1.4) that $lim_{t\to\infty} Sup N_h \leq \frac{\Lambda_h}{\mu_h}$ and$N_v = \frac{\Lambda_v}{\mu_v}$ provided that $S_v(0) + E_v(0) + I_v(0) = N_v(0) = \frac{\Lambda_v}{\mu_v}$ for all, $t \geq 0$. Thus the feasible region for the system (6.1.1) is

$$\Omega = \{(S_h, E_h, I_h, R_h, S_v, E_v, I_v) \in R_+^7, S_h + E_h + I_h + R_h \leq \frac{\Lambda_h}{\mu_h}, S_v + E_v + I_v = \frac{\Lambda_v}{\mu_v}\}.$$

6.1.1 Disease-free equilibrium

Steady state solutions of the system when there is no disease are called "disease-free "equilibrium points. The"diseased" classes containing either exposed, infectious or recovered, the human or mosquito populations, are denoted by E_h, I_h, R_h, E_v, and I_v. Simple calculations shows that the system (6.1.1) has a "disease-free"equilibrium point given by $E_0 = (\frac{\Lambda_h}{\mu_h}, 0, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0)$, which exists for all positive values of the parameters. The dynamics of the disease is described by the basic reproduction number R_0, which is defined as the average number of secondary infections produced by an infected individual in a completely susceptible population. The basic reproduction number of model (6.1.1) is given by the expression

$$\mathfrak{R}_o = \frac{\beta_1 \eta_h \Lambda_h}{\mu_h Q_1 Q_2} + \frac{\beta_2 \beta \eta_h \eta_v \alpha_h \Gamma_h b_v}{\mu_h d_v \mu_v Q_1 Q_2 Q_3 Q_4} + \frac{\beta_2 \beta_3 \eta_h \eta_v \Gamma_h b_v}{\mu_h d_v \mu_v Q_1 Q_2 Q_4}, \tag{6.1.5}$$

where $Q_1 = \eta_h + \mu_h, Q_2 = \mu_h + \alpha_h + \delta_h, Q_3 = \mu_h + \gamma_h, Q_4 = \mu_v + \eta_v$.

Theorem 6.1.1. *If $\mathfrak{R}_o < 1$, then the "disease-free" equilibrium point $E_0(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0)$ of the model (6.1.1) is locally asymptotically stable, otherwise it is unstable.*

Proof. By linearizing the system (6.1.1) around $E_0(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0)$, the Jacobian matrix J is given by:

$$J = \begin{pmatrix} -\lambda - \mu_h & 0 & -\beta_1 \frac{\Lambda_h}{\mu_h} & \gamma_h & 0 & 0 & -\beta_2 \frac{\Lambda_h}{\mu_h} \\ 0 & -\lambda - Q_1 & \beta_1 \frac{\Lambda_h}{\mu_h} & 0 & 0 & 0 & \beta_2 \frac{\Lambda_h}{\mu_h} \\ 0 & \eta_h & -\lambda - Q_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_h & -\lambda - Q_3 & 0 & 0 & 0 \\ 0 & 0 & -\beta_3 \frac{\Lambda_v}{\mu_v} & -\beta \frac{\Lambda_v}{\mu_v} & -\lambda - \mu_v & 0 & 0 \\ 0 & 0 & \beta_3 \frac{\Lambda_v}{\mu_v} & \beta \frac{\Lambda_v}{\mu_v} & 0 & -\lambda - Q_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & \eta_v & -\lambda - \mu_v \end{pmatrix}.$$

The characteristic equation of the above matrix is

$$(\lambda + \mu_h)(\lambda + \mu_v)(\lambda^5 + m_1\lambda^4 + m_2\lambda^3 + m_3\lambda^2 + m_4\lambda + m_5) = 0 \tag{6.1.6}$$

where

$$m_1 = \mu_v + Q_1 + Q_2 + Q_3 + Q_4$$

$$m_2 = Q_1 Q_2 (1 - \frac{\beta_1 \eta_h \Lambda_h}{\mu_h Q_1 Q_2}) + Q_2 Q_3 + 2 Q_3 Q_4 + (Q_1 + Q_2) Q_4 + \mu_v (Q_1 + Q_2 + Q_3 + Q_4)$$

$$
\begin{aligned}
m_3 &= Q_1Q_2(\mu_v + Q_3 + Q_4)(1 - \frac{\beta_1\eta_h\Lambda_h}{\mu_h Q_1 Q_2}) + (Q_1 + Q_2)Q_3Q_4 \\
&\quad +\mu_v(Q_2Q_3 + 2Q_3Q_4 + Q_1Q_4 + Q_2Q_4) \\
m_4 &= Q_1Q_2Q_3(\mu_v + Q_4)(1 - \frac{\beta_1\eta_h\Lambda_h}{\mu_h Q_1 Q_2}) + \mu_v Q_1Q_2Q_4(1 - \frac{\beta_1\eta_h\Lambda_h}{\mu_h Q_1 Q_2} \\
&\quad -\frac{\beta_2\beta_3\eta_h\eta_v\Gamma_h b_v}{\mu_h d_v \mu_v Q_1 Q_2 Q_4}) + \mu_v(Q_2Q_3Q_4 + Q_1Q_3Q_4) \\
m_5 &= \mu_v Q_1Q_2Q_3Q_4(1 - \mathfrak{R}_\circ).
\end{aligned}
$$

Two of the eigenvalues are $-\mu_h$ and $-\mu_v$, which are obviously negative. The remaining five eigenvalues are roots of the equation

$$g(\lambda) = \lambda^5 + m_1\lambda^4 + m_2\lambda^3 + m_3\lambda^2 + m_4\lambda + m_5 = 0. \tag{6.1.7}$$

The necessary and sufficient condition for local asymptotic stability follows from the Routh-Hurwitz conditions applied to the above equation [45], i.e. $m_i > 0$ for $i = 1, 2, 3, 4, 5$ with $m_1m_2m_3 > m_3^2 + m_1^2m_4$ and $(m_1m_4 - m_5)(m_1m_2m_3 - m_3^2 - m_1^2m_4) > m_5(m_1m_2 - m_3)^2 + m_1m_5^2$. For $\mathfrak{R}_\circ < 1$, we see that $m_i > 0$ for $i = 1, 2, 3, 4, 5$. The straightforward but rather lengthy calculations shows that $m_1m_2m_3 > m_3^2 + m_1^2m_4$ and $(m_1m_4 - m_5)(m_1m_2m_3 - m_3^2 - m_1^2m_4) > m_5(m_1m_2 - m_3)^2 + m_1m_5^2$. Hence all the eigenvalues of the characteristic equation (6.1.6) have negative real parts if and only if $\mathfrak{R}_\circ < 1$, which shows that the "disease-free" equilibrium E_0 is locally asymptotically stable. □

Observation: If $\mathfrak{R}_\circ > 1$, we have $g(0) < 0$ and $g(\lambda) = +\infty$ as $\lambda \longrightarrow +\infty$. Thus there exists at least one $\lambda > 0$ such that $g(\lambda) = 0$ which proves instability of "disease-free" equilibrium.

6.1.2 Endemic Equilibrium

Let $E^* = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*)$ represents any arbitrary "endemic" equilibrium of the model (6.1.1). Equating the right hand sides of all the equations in model (6.1.1) to zero,

we have

$$\begin{aligned}
E_h^* &= \frac{Q_2}{\eta_h I_h^*} \\
R_h^* &= \frac{\alpha_h I_h^*}{Q_3} \\
S_v^* &= \frac{\Lambda_v Q_3}{(\beta_3 Q_3 + \beta\alpha_h) I_h^* + \mu_v Q_3} \\
E_v^* &= \frac{\Lambda_v(\beta_3 Q_3 + \beta\alpha_h) I_h^*}{Q_4[(\beta_3 Q_3 + \beta\alpha_h) I_h^* + \mu_v Q_3]} \\
I_v^* &= \frac{\Lambda_v \eta_v (\beta_3 Q_3 + \beta\alpha_h) I_h^*}{(\mu_v Q_4[(\beta_3 Q_3 + \beta\alpha_h) I_h^* + \mu_v Q_3])} \\
S_h^* &= \frac{Q_1 Q_2 \mu_v Q_4[(\beta_3 Q_3 + \beta\alpha_h) I_h^* + \mu_v Q_3]}{\beta_1 \mu_v Q_4 \eta_h[(\beta_3 Q_3 + \beta\alpha_h) I_h^* + \mu_v Q_3] + \Lambda_v \eta_v \beta_2 \eta_h (\beta_3 Q_3 + \beta\alpha_h)}. \qquad (6.1.8)
\end{aligned}$$

In the above I_h^*, is a positive solution of this equation

$$A_1 I_h^{*2} + A_2 I_h^* + A_3 = 0, \qquad (6.1.9)$$

where

$$\begin{aligned}
A_1 &= [\beta_1 \mu_v Q_1 Q_2 Q_3 Q_4 (\beta_3 Q_3 + \beta\alpha_h) + \gamma_h \alpha_h \beta_1 \mu_v Q_4 \eta_h (\beta_3 Q_3 + \beta\alpha_h)] \\
A_2 &= [\beta_1 \mu_v Q_1 Q_2 Q_3 Q_4 \mu_v Q_3 + \beta_2 \Lambda_v \eta_v Q_1 Q_2 Q_3 (\beta_3 Q_3 + \beta\alpha_h) + \gamma_h \beta\alpha_h \beta_1 \mu_v Q_4 \eta_h \mu_v Q_3 \\
&\quad + \gamma_h \beta\alpha_h \Lambda_v \eta_v \beta_2 \eta_h (\beta_3 Q_3 + \beta\alpha_h) + \mu_h d_v Q_1 Q_2 Q_3 Q_4 (\beta_3 Q_3 + \beta\alpha_h)(1 - \frac{\Lambda_h \beta_1 \eta_h}{\mu_h Q_1 Q_2})] \\
A_3 &= \mu_h d_v Q_1 Q_2 Q_3 Q_4 \mu_v Q_3 - \Lambda_h \beta_1 \mu_v Q_3 Q_4 \eta_h \mu_v Q_3 - \Gamma_h b_v \eta_v \beta_2 \eta_h Q_3 (\beta_3 Q_3 + \beta\alpha_h) \\
&= \mu_h d_v \mu_v Q_1 Q_2 Q_3 Q_4 Q_3 (1 - \mathfrak{R}_\circ). \qquad (6.1.10)
\end{aligned}$$

From (6.1.10), we see that $\mathfrak{R}_\circ > 1$ if and only if, $A_3 < 0$. Since $A_1 > 0$, Eq.(6.1.9) has a unique positive root in the feasible region Ω. If $\mathfrak{R}_\circ < 1$, then $A_3 > 0$. Also, it can be easily seen that $A_2 > 0$ for $\mathfrak{R}_\circ < 1$.Thus there will be no (positive) endemic equilibrium in this case. The above conclusion result is summarized below:

Theorem 6.1.2. *System (6.1.1) always has the "infection-free" equilibrium E_0. If $\mathfrak{R}_\circ > 1$, system (6.1.1) has a unique "endemic" equilibrium $E^* = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*)$ defined in (6.1.8) and (6.1.9).*

6.2 Analysis of Global Stability

6.2.1 Global Stability of "Disease−Free" Equilibrium:

We analyze the global behavior of the equilibria for system (6.1.1). The following theorem provides the global property of the "disease-free" equilibrium E_0 of the system.

Theorem 6.2.1. *If $\Re_o < 1$, then the infection–"free" equilibrium E_0 is globally asymptotically stable in the interior of Ω.*

Proof. To prove the global stability of the "disease−free" equilibrium, we construct the following Lyapunov function L and calculate its derivative L' and these are given below :

$$
\begin{aligned}
L &= \frac{\eta_h}{Q_1}E_h + \frac{\eta_h}{Q_1}I_h + \frac{\Gamma_h b_v \beta_2 \beta \eta_h}{\mu_h d_v \mu_v Q_1 Q_3}R_h + \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}E_v + \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}I_v.\\
L' &= \frac{\eta_h}{Q_1}E_h' + \frac{\eta_h}{Q_1}I_h' + \frac{\Gamma_h b_v \beta_2 \beta \eta_h}{\mu_h d_v \mu_v Q_1 Q_3}R_h' + \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}E_v' + \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}I_v'\\
L' &= \frac{\eta_h}{Q_1}(\beta_1 S_h I_h + \beta_2 S_h I_v - Q_1 E_h) + \frac{\eta_h}{Q_1}(\eta_h E_h - Q_2 I_h) + \frac{\Gamma_h b_v \beta_2 \beta \eta_h}{\mu_h d_v \mu_v Q_1 Q_3}(\alpha_h I_h - Q_3 R_h) +\\
&\quad \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}(\beta_3 S_v I_h + \beta R_h S_v - Q_4 E_v) + \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}(\eta_v E_v - \mu_v I_v)\\
&\leq \frac{\eta_h}{Q_1}(\frac{\Lambda_h \beta_1}{\mu_h} I_h + \frac{\Lambda_h \beta_2}{\mu_h} I_v - Q_1 E_h) + \frac{\eta_h}{Q_1}(\eta_h E_h - Q_2 I_h) + \frac{\Gamma_h b_v \beta_2 \beta \eta_h}{\mu_h d_v \mu_v Q_1 Q_3}(\alpha_h I_h - Q_3 R_h) +\\
&\quad \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}(\frac{\Lambda_v \beta_3}{\mu_v} I_h + \frac{\Lambda_v \beta}{\mu_v} R_h - Q_4 E_v) + \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}(\eta_v E_v - \mu_v I_v)\\
&= (\frac{\eta_h}{Q_1}\frac{\Lambda_h \beta_1}{\mu_h} + \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}\frac{\Lambda_v \beta_3}{\mu_v} + \frac{\Gamma_h b_v \beta_2 \beta \eta_h}{\mu_h d_v \mu_v Q_1 Q_3}\alpha_h \frac{\eta_h}{Q_1} Q_2) I_h + (\frac{\eta_h}{Q_1}\frac{\Lambda_h \beta_2}{\mu_h} - \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}\mu_v) I_v +\\
&\quad (-\frac{\eta_h}{Q_1}Q_1 + \frac{\eta_h}{Q_1}\eta_h) E_h + (-\frac{\Gamma_h b_v \beta_2 \beta \eta_h}{\mu_h d_v \mu_v Q_1 Q_3} Q_3 + \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}\frac{\Lambda_v \beta}{\mu_v}) R_h +\\
&\quad (-\frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1} Q_4 + \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}\eta_v) E_v\\
&= (\frac{\eta_h}{Q_1}\frac{\Lambda_h \beta_1}{\mu_h} + \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}\frac{\Lambda_v \beta_3}{\mu_v} + \frac{\Gamma_h b_v \beta_2 \beta \eta_h}{\mu_h d_v \mu_v Q_1 Q_3} - \frac{\eta_h}{Q_1 Q_2}) I_h\\
&\quad +(\frac{\eta_h}{Q_1}(\Lambda_h \beta_2)/\mu_h - (\frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}\mu_v) I_v + (-\frac{\eta_h}{Q_1}Q_1 + \frac{\eta_h}{Q_1}\eta_h) E_h\\
&\quad +(-\frac{\Gamma_h b_v \beta_2 \beta \eta_h}{\mu_h d_v \mu_v Q_1 Q_3}) Q_3 + \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}\frac{\Lambda_v \beta}{\mu_v}) R_h + (-\frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1} Q_4 + \frac{\Lambda_h \beta_2 \eta_h}{\mu_h d_v Q_1}\eta_v) E_v\\
&= \frac{\eta_h Q_2}{Q_1}(\frac{\Lambda_h \beta_1}{\mu_h Q_2} + \frac{\Gamma_h b_v \beta_2 \beta_3}{\mu_h d_v \mu_v Q_2} + \frac{\Gamma_h b_v \beta_2 \beta\ \alpha_h}{\mu_h d_v \mu_v Q_2 Q_3} - 1) I_h + (\frac{\eta_h}{Q_1}\frac{\Lambda_h \beta_2}{\mu_h} - \frac{\Lambda_h \beta_2 \eta_h}{\mu_h Q_1}) I_v +\\
&\quad (-\frac{\eta_h}{Q_1}\eta_h + \frac{\eta_h}{Q_1}\eta_h) E_h + (-\frac{\eta_h \mu_h}{Q_1}) E_h + (-\frac{\Gamma_h b_v \beta_2 \beta \eta_h}{\mu_h d_v \mu_v Q_1} + \frac{\Gamma_h b_v \beta_2 \beta \eta_h}{\mu_h d_v \mu_v Q_1}) R_h + (-\frac{\Lambda_h \beta_2 \eta_h\ \eta_v}{\mu_h d_v Q_1}\\
&\quad +\frac{\Lambda_h \beta_2 \eta_h\ \eta_v}{\mu_h d_v Q_1}) E_v + (-\frac{\Lambda_h \beta_2 \eta_h \mu_v}{\mu_h d_v Q_1}) E_v\\
L' &= \frac{\eta_h Q_2}{Q_1}(\frac{\Lambda_h \beta_1}{\mu_h Q_2} + \frac{\Gamma_h b_v \beta_2 \beta_3}{\mu_h d_v \mu_v Q_2} + \frac{\Gamma_h b_v \beta_2 \beta\ \alpha_h}{\mu_h d_v \mu_v Q_2 Q_3} - 1) I_h - \frac{\eta_h \mu_h}{Q_1} E_h - \frac{\Lambda_h \beta_2 \eta_h}{\mu_h Q_1} E_v
\end{aligned}
$$

We see that L' is negative if $\frac{\Lambda_h \beta_1}{\mu_h Q_2} + \frac{\Gamma_h b_v \beta_2 \beta_3}{\mu_h d_v \mu_v Q_2} + \frac{\Gamma_h b_v \beta_2 \beta\ \alpha_h}{\mu_h d_v \mu_v Q_2 Q_3} < 1$, which implies $\frac{\Lambda_h \beta_1 \eta_h}{\mu_h Q_2 Q_1} + \frac{\Gamma_h b_v \beta_2 \beta_3 \eta_h\ \eta_v}{\mu_h d_v \mu_v Q_2 Q_1 Q_4} + \frac{\Gamma_h b_v \beta_2 \beta \eta_h\ \eta_v\ \alpha_h}{\mu_h d_v \mu_v Q_1 Q_2 Q_3 Q_4} < 1$. Again $L' = 0$ if and only if $I_h = 0 = E_h = E_v$. Therefore the largest compact invariant set in $\{(E_h, I_h, E_v, I_v) \in \Omega, L' = 0\}$, when $\Re_o < 1$, consists of the singelton $\{E_0\}$. Hence, LaSalle's invariance principle [46] implies that E_0 is globally asymptotically stable in Ω. This completes the proof. □

6.2.2 Global Stability of "Endemic" Equilibrium

We shall prove Global stability of the "endemic" equilibrium $E^* = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*)$ where $S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*$ and I_v^* satisfy the following equations:

$$\begin{aligned}
\Lambda_h - \beta_1\ S_h^*\ I_h^* - \beta_2\ S_h^*\ I_v^* - \mu_h\ S_h^* + \gamma_h\ R_h^* &= 0,\\
\beta_1\ S_h^*\ I_h^* + \beta_2\ S_h^*\ I_v^* - Q_1 E_h^* &= 0,\\
\eta_h\ E_h^* - Q_2 I_h^* &= 0,\\
\alpha_h\ I_h^* - Q_3 R_h^* &= 0,\\
\Lambda_v - \beta_3 S_v^*\ I_h^* - \beta\ R_h^*\ S_v^* - \mu_v\ S_v^* &= 0,\\
\beta_3\ S_v^*\ I_h^* + \beta\ R_h^*\ S_v^* - Q_4\ E_v^* &= 0,\\
\eta_v\ E_v^* - \mu_v\ I_v^* &= 0. \qquad (6.2.1)
\end{aligned}$$

We have following theorem. [59]

Theorem 6.2.2. *The unique "endemic" equilibrium E^* is globally asymptotically stable in Ω/Ω_0 whenever $\Re_o > 1$ and $1 + \frac{R_h S_h^*}{R_h^* S_h} - \frac{R_h}{R_h^*} - \frac{S_h^*}{S_h} \geq 0$.*

Proof. The proposed Lyapunov function is given by:

$$\begin{aligned}
L &= a_1(S_h - S_h^* - S_h^* \log\frac{S_h}{S_h^*}) + a_2(E_h - E_h^* - E_h^* \log\frac{E_h}{E_h^*}) + a_3(I_h - I_h^* - I_h^* \log\frac{I_h}{I_h^*})\\
&\quad + a_4(R_h - R_h^* - R_h^* \log\frac{R_h}{R_h^*}) + a_5(S_v - S_v^* - S_v^* \log\frac{S_v}{S_v^*}) + a_6(E_v - E_v^* - E_v^* \log\frac{E_v}{E_v^*})\\
&\quad + a_7(I_v - I_v^* - I_v^* \log\frac{I_v}{I_v^*})
\end{aligned}$$

where $a_1, a_2, a_3, a_4, a_5, a_6$, and a_7 will be chosen later. Differentiating L with respect to t along the solutions of (6.1.1) , we have

$$\begin{aligned}
L' &= a_1(1 - \frac{S_h^*}{S_h})S_h' + a_2(1 - \frac{E_h^*}{E_h})E_h' + a_3(1 - \frac{I_h^*}{I_h})I_h' + a_4(1 - \frac{R_h^*}{R_h})R_h'\\
&\quad + a_5(1 - \frac{S_v^*}{S_v})S_v' + a_6(1 - \frac{E_v^*}{E_v})E_v' + a_7(1 - \frac{I_v^*}{I_v})I_v'
\end{aligned}$$

Substituting the expressions from system (6.1.1) at the endemic steady state, we have

$$\begin{aligned}
L' &= a_1[\beta_1 S_h^* I_h^*(1 - \frac{S_h^*}{S_h} - \frac{S_h I_h}{S_h^* I_h^*} + \frac{I_h}{I_h^*}) + \beta_2 S_h^* I_v^*(1 - \frac{S_h^*}{S_h} - \frac{S_h I_v}{S_h^* I_v^*} + \frac{I_v}{I_v^*}) + d_1 S_h^*(2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*})\\
&\quad - \gamma_h R_h^*(1 - \frac{S_h^*}{S_h} - \frac{R_h}{R_h^*} + \frac{R_h S_h^*}{R_h^* S_h})] + a_2[\beta_1 S_h^* I_h^*(\frac{S_h I_h}{S_h^* I_h^*} - \frac{S_h I_h E_h^*}{S_h^* E_h I_h^*} - \frac{E_h}{E_h^*} + 1) + \beta_2 S_h^* I_v^*(\frac{S_h I_v}{S_h^* I_v^*}\\
&\quad - \frac{S_h I_v E_h^*}{S_h^* E_h I_v^*} - \frac{E_h}{E_h^*} + 1)] + a_3[\eta_h E_h^*(\frac{E_h}{E_h^*} - \frac{E_h I_h^*}{I_h E_h^*} - \frac{I_h}{I_h^*} + 1)] + a_4[\alpha_h I_h^*(\frac{I_h}{I_h^*} - \frac{I_h R_h^*}{I_h^* R_h} - \frac{R_h}{R_h^*} + 1)]\\
&\quad + a_5[\beta_3 S_v^* I_h^*(1 - \frac{S_v^*}{S_v} - \frac{I_h S_v}{I_h^* S_v^*} + \frac{I_h}{I_h^*}) + \beta R_h^* S_v^*(1 - \frac{S_v^*}{S_v} - \frac{R_h S_v}{R_h^* S_v^*} + \frac{R_h}{R_h^*}) + d_2 S_v^*(2 - \frac{S_v^*}{S_v} - \frac{S_v}{S_v^*})]
\end{aligned}$$

$$+a_6[\beta_3 S_v^* I_h^*(\frac{I_h S_v}{I_h^* S_v^*} - \frac{I_h S_v E_v^*}{I_h^* E_v S_v^*} - \frac{E_v}{E_v^*} + 1) + \beta R_h^* S_v^*(\frac{R_h S_v}{R_h^* S_v^*} - \frac{R_h S_v E_v^*}{R_h^* S_v^* E_v} - \frac{E_v}{E_v^*} + 1)]$$
$$+a_7[\eta_v E_v^*(\frac{E_v}{E_v^*} - \frac{E_v I_v^*}{E_v^* I_v} - \frac{I_v}{I_v^*} + 1)] \quad (6.2.2)$$

Setting the values of coefficients

$$\begin{aligned} a_1 &= a_2 = \frac{\beta_3 S_v^* I_h^* + \beta R_h^* S_v^*}{\beta_2 S_h^* I_v^*} \\ a_3 &= \frac{(\beta_1 S_h^* I_h^* + \beta_2 S_h^* I_v^*)(\beta_3 S_v^* I_h^* + \beta R_h^* S_v^*)}{\eta_h E_h^* \beta_2 S_h^* I_v^*} \\ a_4 &= \frac{\beta R_h^* S_v^*}{\alpha_h I_h^*} \\ a_5 &= a_6 = 1 \\ a_7 &= \frac{\beta_3 S_v^* I_h^* + \beta R_h^* S_v^*}{\eta_v E_v^*} \end{aligned}$$

in (6.2.2), and after some calculation, we have

$$\begin{aligned} L' &= \frac{d_1 S_h^*(\beta_3 S_v^* I_h^* + \beta R_h^* S_v^*)}{\beta_2 S_h^* I_v^*}(2 - \frac{S_h}{S_h^*} - \frac{S_h^*}{S_h}) \\ &+\frac{\beta_1 S_h^* I_h^*(\beta_3 S_v^* I_h^* + \beta R_h^* S_v^*)}{\beta_2 S_h^* I_v^*}(3 - \frac{S_h I_h E_h^*}{S_h^* E_h I_h^*} - \frac{S_h^*}{S_h} - \frac{E_h I_h^*}{I_h E_h^*}) + d_2 S_v^*(2 - \frac{S_v}{S_v^*} - \frac{S_v^*}{S_v}) \\ &+\beta R_h^* S_v^*(7 - \frac{R_h S_v E_v^*}{R_h^* E_v S_v^*} - \frac{E_v I_v^*}{I_v E_v^*} - \frac{S_h I_v E_h^*}{S_h^* E_h I_v^*} - \frac{E_h I_h^*}{I_h E_h^*} - \frac{I_h R_h^*}{R_h I_h^*} - \frac{S_v^*}{S_v} - \frac{S_h^*}{S_h}) \\ &+\beta_3 S_v^* I_h^*(6 - \frac{I_h S_v E_v^*}{I_h^* E_v S_v^*} - \frac{E_v I_v^*}{E_v^* I_v} - \frac{S_h I_v E_h^*}{S_h^* I_v^* E_h} - \frac{E_h I_h^*}{I_h E_h^*} - \frac{S_h^*}{S_h} - \frac{S_v^*}{S_v}) \\ &-\frac{\gamma_h R_h^*(\beta_3 S_v^* I_h^* + \beta R_h^* S_v^*)}{\beta_2 S_h^* I_v^*}(1 + \frac{R_h S_h^*}{R_h^* S_h} - \frac{R_h}{R_h^*} - \frac{S_h^*}{S_h}) \end{aligned}$$

The following inequalities hold:

$$\begin{aligned} 2 - \frac{S_h}{S_h^*} - \frac{S_h^*}{S_h} &\leq 0 \\ 3 - \frac{S_h I_h E_h^*}{S_h^* E_h I_h^*} - \frac{S_h^*}{S_h} - \frac{E_h I_h^*}{I_h E_h^*} &\leq 0 \\ 2 - \frac{S_v}{S_v^*} - \frac{S_v^*}{S_v} &\leq 0 \\ 7 - \frac{R_h S_v E_v^*}{R_h^* E_v S_v^*} - \frac{E_v I_v^*}{I_v E_v^*} - \frac{S_h I_v E_h^*}{S_h^* E_h I_v^*} - \frac{E_h I_h^*}{I_h E_h^*} - \frac{I_h R_h^*}{R_h I_h^*} - \frac{S_v^*}{S_v} - \frac{S_h^*}{S_h} &\leq 0 \\ 6 - \frac{I_h S_v E_v^*}{I_h^* E_v S_v^*} - \frac{E_v I_v^*}{E_v^* I_v} - \frac{S_h I_v E_h^*}{S_h^* I_v^* E_h} - \frac{E_h I_h^*}{I_h E_h^*} - \frac{S_h^*}{S_h} - \frac{S_v^*}{S_v} &\leq 0 \end{aligned} \quad (6.2.3)$$

Now, the condition $1 + \frac{R_h S_h^*}{R_h^* S_h} - \frac{R_h}{R_h^*} - \frac{S_h^*}{S_h} \geq 0$ and (6.2.3) imply that $L' \leq 0$. Hence, by Lyapunov's first theorem the endemic equilibrium $E^* = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*)$ is globally asymptotically stable. □

6.3 Sensitivity Analysis

By analyzing different factors that are responsible for the disease transmission and prevalence, we can try to reduce human mortality and morbidity due to disease. Initial disease transmission depends upon the reproductive number whereas disease prevalence is directly related to the endemic equilibrium point. We examine that

$$\frac{\partial \mathfrak{R}_o}{\partial \mu_v} = -\eta_h \eta_v \frac{\Lambda_h}{\mu_h} \frac{\Lambda_v}{\mu_v^3} \frac{\beta_2(2\eta_v + 3\mu_v)}{(\eta_h + \mu_h)(\alpha_h + \delta_h + \mu_h)((\eta_v + \mu_v)^2)} \left(\frac{\beta\alpha_h + \beta_3(\gamma_h + \mu_h)}{(\gamma_h + \mu_h)}\right).$$

So $\mathfrak{R}_o$ is a decreasing function of μ_v. Also we observe that $\mathfrak{R}_o$ is inversely related to the parameters $\alpha_h, \mu_h, \gamma_h$ and δ_h. We want to determine the most crucial parameter in order to decrease the reproductive number less than unity. We can also estimate that this parameter is how much reducing the reproductive number.

Definition 6.3.1. The normalized forward sensitivity index of a variable, h, that depends differentiably on a parameter, l, is defined as $\Gamma_l^h = \frac{\partial h}{\partial l} \times \frac{l}{h}$.

We will calculate the sensitivity indices of the reproductive number, $\mathfrak{R}_o$, with respect to the parameter values given in Table (6.1) for the model. These values are given in Table (6.2).

Table 6.1: Values of the parameters used for sensitivity analysis.

Parameter	Value	Reference
Λ_h	0.00011	[57]
Λ_v	0.13	[57]
γ_h	0.7	[57]
β_2	0.022	[57]
β_3	0.48	[57]
β_1	0.004	[56]
μ_h	0.000016	[57]
μ_v	0.03	[57]
η_h	0.10	[56]
β	0.048	Assumed
η_v	0.091	[56]
α_h	0.0035	[56]
δ_h	0.00009	[56]

Table 6.2: Sensitivity indices of $\mathfrak{R}_o$ to parameters for the model, evaluated at the parameter values given in Table(6.1).

Parameter	Description	Sensitivity index
Λ_h	Recruitment rate of humans	1
Λ_v	Recruitment rate of vectors	0.999973
γ_h	Rate of loss of immunity	-0.000499714
β_2	The transmission probability as a result of biting by an infected mosquito to the susceptible human.	0.999973
β_3	The transmission probability of transfering the infection from an infected human to the susceptible mosquito.	0.999473
β_1	The probability of direct transmission of the disease.	0.000027074
μ_h	Death rate of humans	-1.0046
μ_v	Death rate of vectors	-2.03186
η_h	Rate of progression of humans from exposed class to infectious class	0.000159974
β	The transmission probability of transfering the infection from a partially immune human to the susceptible mosquito.	0.000499725
η_v	Rate of progression of vectors from exposed class to infectious class	0.031914
α_h	Recovery rate of humans	-0.970105
δ_h	Disease related death rate of humans	-0.0249584

By analyzing the sensitivity indices we observe that the most sensitive parameter for the reproductive number is the death rate of mosquitoes μ_v. We can say that an increase or decrease in death rate of mosquitoes by 10% decreases or increases $\mathfrak{R}_o$ by 20%. But it is difficult to make $\mathfrak{R}_o < 1$ by increasing the death rate of mosquitoes μ_v or other parameters dramatically in practice. Although all these measures given above are very effective to control and eradicate the disease but these measures require more cost and labor.

In theorem (6.2.2) it has been proved that the endemic equilibrium E^* is globally asymptotically stable whenever $\mathfrak{R}_o > 1$. We can dicrease the endemic level of the diseased classes besides in making the reproductive number less than unity. The sensitivity indices corresponding to all the parameter values given in Table (6.1) for the infectious vectors and infectious humans are given in Table (6.3).

Table 6.3: The sensitivity indices of the state variables at the endemic equilibrium, x_i, to the parameters, p_j , for parameter values given in Table(6.1).

Parameter	I_h^*	I_v^*
Λ_h	0.999632	0.125391
Λ_v	0.000376465	1.00005
γ_h	-0.999605	-0.125451
β_2	0.000376465	0.0000472229
β_3	4.797×10^{-6}	0.125376
β_1	-0.000372916	-0.0000467777
μ_h	-0.00462088	-0.000579633
μ_v	-0.00039328	-1.1574
η_h	0.000159886	0.0000200557
β	-1.25778×10^{-6}	0.0000625283
η_v	0.0000120148	0.0319164
α_h	-0.970253	-0.121643
δ_h	-0.0249446	-0.00312899

We analyze that the endemic level of infectious vectors is most sensitive to the mortality rate of vectors and also to the recruitment rate of infectious vectors. The endemic level of infectious humans is most sensitive to the rate of loss of immunity. This suggest that the strategies that can be applied in controlling the disease are to target the mosquito biting rate to the partially immuned persons and death rate of the mosquitoes such as the use of insecticide-treated bed nets and indoor residual spray.

Chapter 7

Conclusions

In this dissertation we have analyzed first a vector-host disease model which allows a direct mode of transmission and varying human population. This model concerns diseases with long duration and substantial mortality rate (for example, malaria). Our main results are concerned with the global dynamics of transformed proportionate system. We have constructed Lyapunov function to show the global stability of disease-"free" equilibrium and geometric approach to prove the global stability of "endemic" equilibrium. The epidemiological correlations between the two systems (normalized and unnormalized) has also been discussed. The dynamical behavior of the proportionate model is determined by the basic reproduction number of the disease. The model has a globally asymptotically stable disease-"free" equilibrium whenever $\mathfrak{R}_o \leq 1$. When $\mathfrak{R}_o > 1$, the disease persists at an"endemic" level if $\beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$. We described numerically "endemic" level of infectious individuals and infectious vectors under the condition $\beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$. We have shown numerically that infectious individuals and infectious vectors will also approach to endemic level for different initial conditions even if $\delta_h, \frac{\gamma_h}{2} < \beta_1 < \frac{b_1}{2}$ or $\frac{\gamma_h}{2} < \beta_1 = \delta_h < \frac{b_1}{2}$. We have also shown numerically that the same is true for the case $\delta_h, \frac{b_1}{2} < \beta_1 < \frac{\gamma_h}{2}$ or $\frac{b_1}{2} < \beta_1 = \delta_h < \frac{\gamma_h}{2}$. Thus we conclude that the condition $\beta_1 < min(\frac{b_1}{2}, \frac{\gamma_h}{2})$ is weak for the global stability of unique "endemic" equilibrium. In this model we have assumed permanent immunity. We extend the model by including the exposed class in human and vector population and assumed partial immunity of individuals. We have used compound matrices and the geometric approach to prove the global stability of "endemic" equilibrium. Many researchers discussed the $3-$ dimensional vector-host models by using geometric approach [60–63]. However, to the best of our knowledge, the problem of $4-$ dimensional vector-host models, to prove the global asymptotic stability of endemic equilibrium by using this approach, is being discussed for the first time. We have defined some suitable norms and proved that the Lozinskiĭ measure of homogeneous system is negative under

some conditions. However, the conditions

$$\begin{aligned} b_1 &> \delta_h + \gamma_h, \\ \beta_3 &< \gamma_v + \mu_v, \\ b_1 + \mu_v &> \beta_1 + \gamma_v. \end{aligned} \tag{7.0.1}$$

are **not** the necessary conditions for global asymptotic stability. One can take other forms of $\|Z\|$, which may lead to sufficient conditions different from conditions 7.0.1. We studied a vector host epidemic model with saturated incidence rate. We discussed the sensitivity analysis because we are interested to know which parameters are more crucial to control the disease. We calculated the sensitivity of the reproductive number $\mathfrak{R}_o$ with respect to the parameters given in [26] for malaria disease. We observed that the reproductive number is most sensitive to the mosquito biting rate and death rate. It means that three control efforts personal protection, larvacide and adulticide can be applied to eredicate the disease. However, practically it is not possible to eredicate the disease. So we can reduce the endemic level of infectious classes. We have discussed the sensitivity analysis of the infectious individuals and vectors. From this analysis we concluded that personal protection, larvacide and adulticide and treatment of infectious individuals are the control efforts that can be applied in order to reduce the endemic level of infectious classes. In this dissertation, pine wilt disease transmission model with nonlinear incidence rates and horizontal transmission in vector population is proposed and analyzed. It is not meaningful to consider the saturation level when transmission occurred during mating. Thus bilinear incidence has been considered. We performed stability and sensitivity analysis. By the sensitivity analysis it has been observed that by killing pine sawyer beetles by applying different strategies as using chemicals, setting out beetle traps, setting vertical wood traps, cutting down and disposing off dead pine trees are useful to eredicate the disease completely. But these control measures require more cost and labor and keep danger of forest fire. However we can decrease infectious beetles and infectious and dead pines to some extent by applying these control strategies. Hence from the available data of any vector-borne disease we can identify that which factors are responsible for the enhancement of the disease. On the basis of the sensitivity analysis we can plan more effective control strategies to eredicate the disease completely or at least to reduce the endemic level.

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