Analysis of stochastic vector-host epidemic models

by

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A dissertation submitted to the Graduate Faculty of
Auburn University
in partial fulfillment of the
requirements for the Degree of
Doctor of Philosophy

Auburn, Alabama
August 5, 2017

Keywords: vector-host epidemic model, stochastic model, basic reproductive number, stochastic stability, stationary distribution, regime switching model

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Abstract

In this dissertation, deterministic and stochastic mathematical models are proposed to study vector-host epidemic models with direct transmission. The total population of the host and the vector is divided into different compartments as susceptible hosts, infected hosts, susceptible vectors and infected vectors. In the first chapter, we model and study the deterministic vector-host epidemics with direct transmission using a nonlinear system of differential equations. First we obtain the disease-free equilibrium point E_0 and the endemic equilibrium point E_1. After that we derive the basic reproductive number $\mathcal{R}_0$, and study the local and global stabilities of E_0 and E_1 in relation to $\mathcal{R}_0$. Using the perturbation of fixed point estimation, we investigate the sensitivity of the basic reproductive number in relation to the parameters used in the model. Next by adding environmental fluctuations to the deterministic model, we obtain a nonlinear system of stochastic differential equation that describes the dynamics of the stochastic vector-host epidemic model. By defining a stochastic Lyapunov function, we prove the existence of a unique nonnegative global solution to the stochastic model. Moreover, we show that the solution of the stochastic model is stochastically ultimately bounded and stochastically permanent. Similar to the deterministic case, we obtain the basic reproductive number for the stochastic model $\mathcal{R}_0^s$ and we show that the infection will die out or persist depending on the value of $\mathcal{R}_0^s$. In particular, we show that random effects may lead to extinction in the stochastic case while the deterministic model predicts persistence. We also present necessary conditions for the infection to be persistent in the stochastic model. Finally we present a stochastic vector-host epidemic model with direct transmission in random environment, governed by a system of stochastic differential equations with regime-switching diffusion. We first examine the existence and uniqueness of a nonnegative global solution. Then we investigate stability properties of

the solution, including almost sure and pth moment exponential stability and stochastic asymptotic stability. Moreover, we provide conditions for the existence and uniqueness of a stationary distribution. In all the chapters, we provide numerical simulations and examples to illustrate some of the theoretical results.

Acknowledgments

I would like to express my deepest appreciation and gratitude to my advisors Dr. Yanzhao Cao and Dr. Xiaoying Han, who gave me immense guidance and support during my graduate studies at Auburn University. I am very grateful for their time and enthusiasm, encouragement, and support throughout the years. They continually and convincingly conveyed a spirit of hard work in regard to research and an excitement in regard to teaching. Without their guidance and persistent help this dissertation would not have been possible. I would also like to thank Dr. Wenxian Shen, Dr. Paul G. Schmidt, and Dr. Sang-Jin Suh for serving on my dissertation committee and their help in revising my work.

I also want to express my gratitude to all members of the Department of Mathematics at Auburn University that were an active part of my graduate life. The faculty members at the department have been very kind enough to extend their help at various phases of my study, whenever I approached them, and I do hereby acknowledge all of them. I am very glad to have been part of this Department.

I wish to express my unqualified thanks and love to my wife Tizita and my son Barakel. I could never have accomplished my study without your love, support, and understanding.

I would also like to acknowledge the immense impact and support that my family has given me, including my father, mother, brother and sisters whose encouragement was beyond description. I am very much indebted to you, who encouraged and helped me at every stage of my personal and academic life, and longed to see this achievement come true.

I also thank my friends for providing support and friendship that I needed, too many to list here but you know who you are! Above all, I owe it all to Almighty God for granting me the wisdom, health and strength to undertake my study and enabling me to its completion.

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Chapter 1

Introduction

According to medical dictionary, a disease is a pathologic process with a characteristic set of Signs and Symptoms. It may affect the whole body or any of its parts, and its etiology or cause, Pathology, and Prognosis may be known or unknown. There are different ways of classifying diseases. One such classification is infectious versus noninfectious. Infectious diseases are disorders caused by organisms such as bacteria, viruses, fungi or parasites and can be passed between individuals. On the other hand, noninfectious diseases are medical conditions that are not caused by infectious agents. Noninfectious diseases such as obesity, down syndrome, kidney disease can last for long periods of time and progress slowly, while other noninfectious diseases such as autoimmune diseases, heart diseases, stroke and some cancers may result in rapid death. The epidemiology of noninfectious diseases is mainly related to risk factors such as a person's background, lifestyle and environment which includes age, gender, genetics and some behaviours such as smoking, unhealthy diet and lack of physical exercise. Contrary to the noninfectious diseases, the main risk factor for acquiring an infectious disease is the presence of infectious cases in the population.

Infectious diseases can be classified into two categories based on the way how the infection is transmitted from an infected organism to a susceptible one. Generally an infectious disease can be transmitted in two ways; horizontally or vertically. Horizontal transmission is when the disease is transmitted from one individual to another in the same generation or peers in the same age group, while vertical transmission is passing the agent causing the disease from parent to offspring, such as in prenatal or perinatal transmission [6, 25].

Another way of classifying infectious diseases is based on whether the transmission of infection is direct or indirect. A transmission is called direct if the infection is spread when

disease-causing microorganisms pass from the infected person to the healthy person via direct physical contact with blood or body fluids. Examples of direct contact are touching, kissing, sexual contact, contact with oral secretions, or contact with body lesions. With some exceptions, most of the microparasitic diseases, such as influenza, measles, and HIV, are directly transmitted from an infected person to a healthy one. On the other hand, indirect transmission involves the transfer of an infectious agent through a contaminated intermediate object or person. Some of the indirect transmission mechanisms include the following [6, 25].

1. Airborne transmission: Some bacteria or viruses travel on dust particles or on small respiratory droplets that may become aerosolized when people sneeze, cough, laugh, or exhale. Many common infections such as TB, measles, chickenpox, smallpox can spread by airborne transmission.

2. Contaminated objects: Touching an object, such as a doorknob, soon after an infected person, or through contaminated blood products and medical supplies one might be exposed to infection. Contaminated food and water can also be included in this group.

3. Animal-to-person contact: Some infectious diseases can be transmitted from an animal to a person. This can happen when an infected animal bites or scratches or when one handles animal waste.

4. Insect bites (vector-borne disease): Some zoonotic infectious agents are transmitted by insects, especially those that suck blood. These include mosquitos, fleas, and ticks.

5. Animal reservoirs: There are many diseases that can be transmitted to humans from other vertebrate hosts (zoonoses). Some of the Zoonotic diseases include anthrax (from sheep), rabies (from rodents and other mammals), West Nile virus (from birds) and plague (from rodents).

According to the Center for Strategic and International Studies (CSIS), infectious diseases are the leading cause of death of children, adolescents and adults. For example in 2013, among older children ages 5 to 9, the most common cause of death was diarrheal disease, followed by lower respiratory tract infections, road injuries, intestinal infectious diseases (mainly typhoid and paratyphoid), and malaria. These five causes accounted for 39% of all deaths among children 5 to 9. In the same year, among adolescents 10 to 19, the leading cause of death was road injuries, followed by HIV/AIDS, self-harm, drowning, and intestinal infectious diseases. These five leading causes accounted for 34% of all deaths in this age group [27].

A large proportion of infectious diseases are spread through vector transmission. Vector-borne diseases are infections transmitted by the bite of blood-feeding arthropods such as mosquitoes, ticks, triatomine bugs, sandflies, and blackflies or through contaminated urine, tissues or bites of infected animals such as rats or dogs. Some vector-borne diseases may also be transmitted directly through blood transfusions, organ transplantation, exposure in a laboratory setting, or from mother to baby during pregnancy, delivery and breast feeding. Direct transmission has an impact on the dynamics of many vector-borne diseases [6].

Vector-borne diseases provide unique challenges to public health because the epidemiology is so closely tied to external environmental factors such as climate, landscape, and population migration, as well as the complicated biology of vector-transmitted pathogens.

One bigger problem faced by vector-borne diseases is their effect on livestock and crops. They have the potential to cause a serious economic harm to a country and even can affect trade relation among different countries. For example, according to FAO, bluetongue, a viral disease transmitted among sheep and cattle by biting midges, results in annual losses of approximately $3 billion due to morbidity and mortality of animals, trade embargoes, and vaccination costs [28].

Despite great advances in public health worldwide, insect vector-borne infectious diseases remain a leading cause of morbidity and mortality. While significant advances are

currently being made in interventions to prevent and treat most of these diseases such as zika, malaria, lymphatic filariasis and Chagas disease, other diseases such as dengue continue to spread and increase their number of cases at an alarming pace. The silent expansion of mosquito vectors and their ability to develop resistance to insecticides threatens the gains made through vector control and calls for concerted planning and collaboration across sectors including health, agriculture and the environment. The spread of some vector-borne diseases in rural areas is also aggravated by environmental changes [49].

One of the main reasons for studying infectious diseases is to improve control and ultimately to eradicate the infection from the population. The results from the study are very helpful to predict the developing tendency of the infectious disease, to determine the key factors of the spread of infectious disease and to seek the optimum strategies of preventing and controlling the spread of infectious diseases. In this respect, mathematical models can be very important in providing a unique approach to gain basic insights into the dynamics of infectious diseases and for understanding the underlying mechanisms that influence the spread of disease to suggest control strategies.

For an effective prevention and intervention strategies against infectious diseases, understanding of the fundamental mechanism in the disease transmission is crucial. In order to achieve this goal, many mathematical models have been used to investigate how to more effectively control emerging and reemerging infectious diseases such as SARS, zika, malaria, dengue fever and West Nile virus, via various disease control measures including vaccination, quarantine, and isolation [46].

According to Matt J. Keeling [25], models have two distinct roles, prediction and understanding, which are related to the model properties of accuracy and transparency, and therefore can often be in conflict. We usually require a high degree of accuracy from any predictive model, whereas transparency is a more important quality of models used to improve our understanding. Prediction is the most obvious use of models. It requires that the model is as accurate as possible and therefore includes all of the known complexities and

population-level heterogeneities. Predictive models can have great power in specific situations, guiding difficult policy decisions where a trade-off between two (or more) alternative control strategies exists.

One of the early triumphs of mathematical epidemiology was the formulation of a simple model by Kermack and McKendrick in 1927 whose prediction was very important in analyzing the spread, and control of infectious diseases qualitatively and quantitatively. The Kermack–McKendrick model is a compartmental model based on relatively simple assumptions on the rates of flow between different classes of members of the population. In epidemiology, these models are known as compartmental models, and they serve as a base mathematical framework for understanding the complex dynamics of these systems, which hope to model the main characteristics of the system [10].

In order to model some infectious diseases, we divide the population being studied into different compartments and put some assumptions about the nature and time rate of transfer from one compartment to another. The independent variable in the compartmental model is time t, and the rates of transfer between compartments are expressed mathematically as derivatives with respect to time of the sizes of the compartments. As a result our models are formulated initially as differential equations.

We denote the number of individuals who are susceptible to the disease but not yet infected by $S(t)$. At time $t = 0$, the host encounters an infectious individual and becomes infected. Initially, the individual may exhibit no obvious signs of infection and the abundance of pathogen may be too low to allow further transmission. Individuals in this phase are said to be in the exposed class and are denoted by $E(t)$. Once the level of parasite is sufficiently large within the host, the potential to transmit the infection to other susceptible individuals exists and the host becomes infectious and we denote it by $I(t)$. Finally, once the individual's immune system has cleared the parasite and the host is therefore no longer infectious, they are referred to as recovered and denoted by $R(t)$.

As an example, let us discuss on how to model a communicable disease such as influenza, measles or ebola using the mass-action principle as suggested by Kermack and McKendrick in the 1927. Although the epidemiology of each disease is unique, the models presented in this example provides a framework that captures the common features across many other diseases. The population of the host is categorized as susceptible (if previously unexposed to the pathogen), infected (if currently colonized by the pathogen), and recovered (if they have successfully cleared the infection) and the population of each compartments at time t is denoted by $S(t), I(t), R(t)$ respectively. For simplicity, we assume a closed population (no births, deaths or migration) and thus we have only the transitions from susceptible to infected ($S \to I$) and from infected to recovery ($I \to R$). The flow chart below shows the different compartments and how the infection is transmitted from one group to another.

$$S \to I \to R$$

The following assumptions are considered in deriving the SIR (susceptible-infectious-recovered) model:

(1) The disease spread in a closed environment, no emigration and immigration, and no birth and death in the population, so the total population remains a constant N, i.e. $S(t) + I(t) + R(t) = N$.

(2) 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 $\beta S(t)I(t)$.

(3) 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 $\gamma I(t)$.

From the above assumptions it follows that the susceptible group will decrease at a rate of $\beta S(t)I(t)$ and due to infection, and due to recovery, the infected group will decrease at a rate of $\gamma I(t)$. Thus the transition of individuals from susceptible to infected and from

infected to recovery group can be expressed as a system of nonlinear differential equation as follows.

$$\begin{aligned} \frac{dS(t)}{dt} &= -\beta S(t)I(t) \\ \frac{dI(t)}{dt} &= \beta S(t)I(t) - \gamma I(t) \\ \frac{dR(t)}{dt} &= \gamma I(t) \end{aligned} \tag{1.1}$$

Even though the above system looks simple, it is not possible to find an explicit solution. That is we cannot obtain an exact analytical expression for the dynamics of $S(t), I(t)$ and $R(t)$. However, given some initial conditions $S(0) > 0$, $I(0) > 0$, $R(0) = 0$, the system can be solved numerically.

Many important infections have significant incubation period during which the individual has been exposed and infected but is not yet infectious themselves. For some disease, it takes certain time for an infective agent to multiply inside the host up to the critical level so that the disease actually manifest itself in the body of the host. Thus we include an exposed compartment E and we will end up with a SEIR model.

SI (susceptible - infectious) model is the most appropriate way of modeling some plant infection, since the host is infectious soon after it is infected, such that the exposed period can be safely ignored, and remains infectious until its death. On the other hand, some sexually transmitted infectious diseases such as gonorrhoea and air-borne infections such as influenza and common cold, are better modeled by an SIS (susceptible-infectious-susceptible) technique. This is due to the fact that these infections do not confer any long lasting immunity and thus they do not give immunization upon recovery from infection, and individuals become susceptible again.

One important question in epidemiology is to determine whether or not an infectious disease can spread through a population. Thus in order to determine if the infection will eventually die out or persist in the system, we should know the average number of secondary infections caused by a single infectious individual during their entire infectious lifetime. This fundamental concept is called the basic reproduction number or basic reproductive ratio and

usually denoted by $\mathcal{R}_0$. The idea of basic reproductive number was first developed in 1886 by Alfred Lotka, Ronald Ross, and others to study demographics but its first modern application in epidemiology was by George MacDonald in 1952, who constructed population models of the malaria [21, 29].

The basic reproductive number is affected by several factors including the duration of infectivity, the infectiousness of the organism and the number of susceptible people with whom the infected patient comes in contact. Even though the basic reproductive number can be used as a threshold to determine whether a disease will die out or it may become epidemic, it cannot be used to compare risks associated with different pathogens.

Hefferman et al. (2005) has provided a method for calculating $\mathcal{R}_0$ for any structured population model using the concept of the next generation matrix [22]. The next generation matrix G is defined by $G = FV^{-1}$, where

$$F = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right]$$

represents the rate at which secondary infections are produced in compartment i by an index case in compartment j, and

$$V = \left[\frac{\partial V_i(x_0)}{\partial x_j}\right]$$

is the transfer of infections from compartment i to j and x_0 is the disease free equilibrium. Finally $\mathcal{R}_0$ is defined to be the dominant eigenvalue of the matrix $G = FV^{-1}$. The basic reproductive number of some of the well-known diseases is given in table 1.1 [17].

All the above epidemic models considered are deterministic, that is, the output of the model is fully determined by the parameter values and the initial conditions. When dealing with large populations, as in the case of tuberculosis or HIV, deterministic models often provide useful ways of gaining sufficient understanding about the dynamics of populations. Although deterministic models have contributed much to the understanding of the biological

Table 1.1: Basic reproductive number of some of the well-known diseases.

Disease	Transmission	$\mathcal{R}_0$
Measles	Airborne	12-18
Diphtheria	Saliva	6-7
Smallpox	Airborne droplets	5-7
Polio	Fecal-oral route	5-7
Mumps	Airborne droplets	4-7
HIV/AIDS	Sexual contact	2-5
SARS	Airborne droplets	2-5
Influenza	Airborne droplets	2-3
Ebola (2014 outbreak)	Bodily fluids	1.5-2.5

processes which underlie the spread of disease, the importance of random effects and fluctuations in determining population dynamic patterns of disease incidence and persistence is not reflected.

Generally, fluctuations in the epidemic model arises either from demographic stochasticity or environmental stochasticity. Demographic stochasticity describes the randomness that results from the inherently discrete nature of individuals. As a result of this fluctuation, small populations will be impacted highly. On the other hand, environmental stochasticity describes the randomness resulting from any change that impacts an entire population such as changes in the environment.

The epidemicity of some infection can be critically influenced by environmental variations and fluctuations. For example, some pathogenic nematodes that cause severe disease in staple crops exhibit a critical sensitivity to soil moisture content, becoming inactive at low levels. Also a variety of insect pests and parasites are strongly influenced by environmental switching, most notably by diapause, a suspension of development often triggered by changes in temperature, light levels, or humidity. Environmental variables such as temperature and moisture levels have both a predictable mean trend over time and a short time-scale random component. Both aspects of this variation can be transmitted to the disease process

through the sensitivity of the organisms involved. Hence the variability of the environment is fed through to the state of the epidemic. Thus in the stochastic model, randomness is present, and variable states are not described by unique values, but rather by probability distributions [51].

Thus stochastic models are concerned with approximating this random or probabilistic element by incorporating of effects of secondary factors for which a detailed knowledge is missing. Also if the initial population size is small then a stochastic model is more appropriate, since the likelihood that the population becomes extinct due to chance must be considered. One other important differences between the deterministic and stochastic epidemic models is their asymptotic dynamics. Eventually stochastic solutions (sample paths) may converge to the disease-free state even though the corresponding deterministic solution converges to an endemic equilibrium.

Most of the time, stochastic epidemic models can be derived from their corresponding deterministic ones by incorporating randomness into the system. There are several ways to include these fluctuations in the deterministic model. For example, [13, 19, 48] introduced parametric perturbations, since the parameters in the model are always altered due to continuous environmental fluctuations. Another approach, pioneered in the works of May and Beddington [39], assumes that the environmental noise is generated by an m-dimensional standard Brownian motion. Some other authors used this idea to study the properties of stochastic epidemic models in order to find a more efficient way to reduce infections [9, 13, 45, 54, 61, 62].

The random environments and random factors such as intrinsic growth rates and inter/intra-specific growth rates in ecological systems can be modeled by a continuous-time Markov chain, as the switching between different environments is memory-less and the waiting time is exponentially distributed [11, 20, 32, 60]. Similarly, in vector-host epidemic models the transmission rates of vector-borne diseases and the reproduction rates of vectors vary with

respect to changes of environments. The resulting model is a system of stochastic differential equations with regime switching.

Deterministic vector-host epidemic models have been studied by several authors. As mentioned above, the development of vector-host epidemic models with indirect transmission can be traced back to Ross malaria model in 1911. In his malaria model, Ronald Ross has captured the basic features on the transmission of malaria and argued that malaria can be eradicated if the population of mosquito can be reduced below a certain threshold. Later in 1957, Macdonald has modified the Ross malaria model by including an exposed group and developed a more comprehensive model which led to a better understanding on malaria transmission. All other models that exist for malaria dynamics are developed from the basic models explained earlier by incorporating different factors to make them biologically more realistic in explaining disease prevalence and prediction [35].

Since then, due to an increased knowledge of the disease and the availability of data, many researchers have extended these models for a better understanding of vector-borne diseases such as malaria. For example, a number of researchers have studied malaria model by including a recovered group which incorporates a time dependent immunity developed on recovery from infection in humans [4, 15, 40]. Moreover, some models have integrated other factors such as environmental effects [30, 55, 56]; mosquitos resistance to insecticides and resistance of some parasitic strains to anti-malaria drugs [36, 41, 50]. Cai and Li proposed and analyzed vector-host epidemic models with direct transmission. They showed that the stability of the equilibria in the proposed models can be controlled by the basic reproduction number and, moreover, they provided conditions for the global asymptotical stability of the equilibria [8]. Also, optimal control problems related to different infections such as malaria have generated a lot of interest from researchers. For example, Rafikov, Bevilacqua and Wyse formulated a continuous model for malaria vector control with the aim of studying how genetically modified mosquitoes should be introduced in the environment using optimal control problem strategies [43]. Similarly, Kbenesh Blayneh, Yanzhao Cao, and Hee-Dae

Kwon presented an autonomous ordinary differential equation model with vector control and treatment model, and a time dependent counter part of the model involving an optimal control of vector-borne diseases with treatment and prevention as control measures [5].

Similarly, there are several mathematical models that studied HIV/AIDS. Antiviral therapy - strategy and drug resistance at the population and individual level [3, 18, 12]; comparison of introducing a time delay in the disease infection term [42]; prediction of reduction of incidence based on strategies of early detection and widely available antiviral therapy; through both deterministic and stochastic models [18]; a model of vertical HIV transmission including treatment and drug-resistance [23, 44].

Dengue fever is a major international health problem, especially in the tropical and sub-tropical regions of the world including Puerto Rico and in many popular tourist destinations in Latin America, Southeast Asia and the Pacific islands. Due to this fact and the complexity of its transmission, dengue fever has gathered a lot of attention from mathematical epidemiologists throughout the years. Feng and Velasco-Hernandez presented a vector-host dynamics in a two-strain epidemiological system and derived the basic reproductive number $\mathcal{R}_0$. They showed that whenever $\mathcal{R}_0 > 1$, there is an unstable endemic equilibrium and concluded that the system's long-term behaviour under this condition is unpredictable [16]. Recent studies on modeling dengue fever transmission have taken various directions. For example, Yang and Ferreira extended the basic SIR model of dengue fever by testing different vector-control strategies (insecticide or larvicide application, removal of breeding containers) [57], Derouich et al. proposed a mathematical model to simulate the succession of two dengue fever epidemics with variable human populations and studied the stability analysis of the equilibrium points [14], Wei et al. developed a dengue transmission model including direct transmission (which is strictly only expected through blood transfusion, bone marrow transplantation or needle sticks) in addition to the vector-mediated transmission. They also represented the extrinsic incubation period using a time delay and derived the threshold conditions for the existence of an endemic equilibrium [53].

While deterministic epidemic models have made a major contribution to the understanding of dynamics of infectious diseases, they are merely restricted to stationary environments and lose their validity while the environment is fluctuating. To reflect the multiplicities of interactions among organisms and their fluctuating environments, stochastic epidemic models have been studied extensively during the past years.

For example, in [1, 2] Allen presented different methods for formulating stochastic epidemic models that relate directly to their deterministic counterparts and compared the two models for the SIS and SIR cases in discrete time. In [19], Gray et al. extended the classical SIS epidemic model from a deterministic framework to a stochastic one, and formulated it as a stochastic differential equation for the number of infectious individuals $I(t)$. The authors proved the existence of a unique global solution for the stochastic model and established conditions for extinction and persistence of $I(t)$. Yang and Mao considered a class of multi-group SEIR epidemic models with stochastic perturbations and by the method of stochastic Lyapunov functions, they studied their asymptotic behavior in terms of the intensity of the stochastic perturbations and the reproductive number $\mathcal{R}_0$ [58]. Similarly, Jovanovic and Krstic have studied a stochastic vector-host epidemic model with direct transmission by introducing random perturbations around the endemic equilibrium state. Using Lyapunov functions and functionals, they obtained stability conditions for the stochastic model and studied the effect of the delay on the stability of the endemic equilibrium [24].

This dissertation includes the analysis of both deterministic and stochastic vector-host epidemic model with direct transmission. In chapter 2, we present a deterministic vector-host epidemic model with direct transmission. The model consists of a system of non-linear differential equations, that uses the SIS structure for the host and the SI type of structure for the vector. The basic reproductive number is derived and the local and global stabilities of both the disease-free equilibrium point E_0 and the endemic equilibrium point E_1 are discussed. In chapter 3, we present a stochastic vector-host epidemic model with direct transmission to study the effect of adding environmental fluctuations on the corresponding deterministic

model. We analyze the stochastic model including the existence and uniqueness of solution of the system and conditions for the extinction and persistence of the infection. In chapter 4, we consider a regime switching vector-host epidemic model with direct transmission. We construct conditions for the extinction of the disease and investigate the different stability conditions of the solution. In each chapter, numerical simulations are conducted to support the analytical conclusions.

Chapter 2

Analysis of the Deterministic Vector-host Epidemic Model with Direct Transmission

2.1 Derivation of vector-host epidemic model

In this section, we formulate and discuss the transmission and spread of disease in vector-host model. The host population is divided into two compartments: susceptible host ($S_h(t)$) and infected host ($I_h(t)$). The vector population is also divided into two classes: susceptible vector ($S_v(t)$) and infected vector ($I_v(t)$). Susceptible hosts can be infected directly through a contact with an infected host, such as blood transfusion, as well as indirectly by a bite from an infected arthropod, such as mosquito and tick. On the other hand, if a susceptible vector bites an infected host, it will acquire the disease. Using the SIS type of structure for the host and the SI type of structure for the vector results in the following set of nonlinear ODEs [8, 24]

$$\frac{dS_h}{dt} = b_1 - \mu_1 S_h - \beta_2 S_h I_v - \beta_1 S_h I_h + \phi I_h \tag{2.1}$$

$$\frac{dI_h}{dt} = \beta_1 S_h I_h + \beta_2 S_h I_v - (\mu_1 + \phi) I_h, \tag{2.2}$$

$$\frac{dS_v}{dt} = b_2 - \mu_2 S_v - \beta S_v I_h, \tag{2.3}$$

$$\frac{dI_v}{dt} = \beta S_v I_h - \mu_2 I_v, \tag{2.4}$$

where μ_1 and μ_2 are the mortality rates of the host and vector, respectively, ϕ is the recovery rate of infected hosts, β_1 is the direct transmission rate from an infected host to susceptible host, β_2 is the indirect transmission rate from an infected vector to a susceptible host, β is the transition rate from infected host to susceptible vector, and b_1 and b_2 are the recruitment rates of the host and the vector, respectively.

Define the region Γ by

$$\Gamma := \left\{ (S_h, I_h, S_v, I_v) \in \mathbb{R}^4 : S_h + I_h = \frac{b_1}{\mu_1},\ S_v + I_v = \frac{b_2}{\mu_2} \right\}.$$

Denote by $N_h(t) = S_h(t) + I_h(t)$ and $N_v(t) = S_v(t) + I_v(t)$ the total population of the host and the vector, respectively. Then it follows directly from equations (2.1)–(2.4) that

$$\frac{dN_h(t)}{dt} = b_1 - \mu_1 N_h(t) \qquad \text{and} \qquad \frac{dN_v(t)}{dt} = b_2 - \mu_2 N_v(t).$$

Note that for any initial conditions $N_h(0) = b_1/\mu_1$ and $N_v(0) = b_2/\mu_2$,

$$N_h(t) \equiv \frac{b_1}{\mu_1} \qquad \text{and} \qquad N_v(t) \equiv \frac{b_2}{\mu_2}.$$

In addition, it can be easily shown that for any arbitrary initial condition,

$$\lim_{t\to\infty} N_h(t) = \frac{b_1}{\mu_1} \qquad \text{and} \qquad \lim_{t\to\infty} N_v(t) = \frac{b_2}{\mu_2}.$$

Hence the set Γ is invariant and attracting. Similar to [6, 8, 24], throughout this chapter we restrict our model on the set Γ where system (2.1)–(2.4) can be reduced to the following equivalent two dimensional system:

$$\frac{dI_h}{dt} = \frac{\beta_1 b_1}{\mu_1} I_h - \beta_1 I_h^2 + \frac{\beta_2 b_1}{\mu_1} I_v - \beta_2 I_h I_v - (\mu_1 + \phi) I_h, \tag{2.5}$$

$$\frac{dI_v}{dt} = \frac{\beta b_2}{\mu_2} I_h - \beta I_h I_v - \mu_2 I_v. \tag{2.6}$$

2.2 Equilibrium points and basic reproductive numbers

In this section, we derive an explicit expression for the basic reproduction number of the epidemiological model and calculate the equilibrium state solutions. One way to see what will happen to the population eventually is to explore when the system is at equilibrium.

By setting

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

we get the following two equilibrium points. The disease-free equilibrium $E_0 = (\frac{b_1}{\mu_1}, 0, \frac{b_2}{\mu_2}, 0)$ and the endemic-equilibrium $E_1 = (S_h^*, I_h^*, S_v^*, I_v^*)$, where

$$S_v^* = \frac{b_2}{\mu_2 + \beta I_h^*}, \quad I_v^* = \frac{\beta b_2}{\mu_2} \frac{I_h^*}{\mu_2 + \beta I_h^*}, \quad S_h^* = \frac{\mu_2(\mu_1 + \phi)(\mu_2 + \beta I_h^*)}{\beta_1 \mu_2(\mu_2 + \beta I_h^*) + \beta\beta_2 b_2} \tag{2.7}$$

and I_h^* is the positive solution of the equation

$$k_2(I_h^*)^2 + k_1 I_h^* + k_0 = 0, \tag{2.8}$$

with

$$\begin{aligned} k_0 &= -\mu_1\mu_2^2(\mu_1 + \phi)(\mathcal{R}_0 - 1), \qquad k_2 = \beta\mu_2\beta_1\mu_1 \\ k_1 &= \phi\beta\mu_2\mu_1 + \mu_2^2\beta_1\mu_1 + \beta b_2\beta_2\mu_1 + \beta\mu_2\mu_1^2 - \beta b_1\mu_2\beta_1. \end{aligned}$$

The constant $\mathcal{R}_0$ is the basic reproductive number of the model and is defined below. The disease-free equilibrium is the case where the pathogen has suffered extinction and, in the long run, everyone in the population is susceptible, while endemic equilibrium is the state where the disease cannot be totally eradicated but remains in the population.

As described in the introduction part, $\mathcal{R}_0$ can be calculated using the next generation matrix. For the system (2.1)–(2.4), we have that

$$\begin{bmatrix} F_1 \\ F_2 \end{bmatrix} = \begin{bmatrix} \beta_1 S_h I_h + \beta_2 S_h I_v \\ \beta S_v I_h \end{bmatrix} \; and \; \begin{bmatrix} V_1 \\ V_2 \end{bmatrix} = \begin{bmatrix} (\mu_1 + \phi) I_h \\ \mu_2 I_v \end{bmatrix}.$$

Thus

$$F(E_0) = \begin{bmatrix} \frac{\beta_1 b_1}{\mu_1} & \frac{\beta_2 b_1}{\mu_1} \\ \\ \frac{\beta b_2}{\mu_2} & 0 \end{bmatrix} \text{ and } V(E_0) = \begin{bmatrix} \mu_1 + \phi & 0 \\ 0 & \mu_2 \end{bmatrix}.$$

Now we have

$$G = FV^{-1} = \begin{bmatrix} \frac{\beta_1 b_1}{\mu_1(\mu_1+\phi)} & \frac{\beta_2 b_1}{\mu_2 \mu_1} \\ \\ \frac{\beta b_2}{\mu_2(\mu_1+\phi)} & 0 \end{bmatrix}.$$

Hence $\mathcal{R}_0$ is the dominant eigenvalue of G and it is given by

$$\mathcal{R}_0 = \frac{\beta_1 b_1}{\mu_1(\mu_1+\phi)} + \frac{\beta\beta_2 b_1 b_2}{\mu_2^2 \mu_1(\phi+\mu_1)}. \tag{2.9}$$

2.3 Global and local stability of the equilibrium points

In this section, we study the local and global stabilities of both the disease-free and endemic equilibrium points. An equilibrium point $\tilde{x}$ is globally stable or globally asymptotically stable for a model if for all positive initial values, the solution of the model approaches $\tilde{x}$ as t increases. An equilibrium point $\tilde{x}$ is locally stable or locally asymptotically stable if for some neighbourhood of $\tilde{x}$, the solution of the model approaches $\tilde{x}$ as the time increases for all initial values in the neighbourhood of $\tilde{x}$.

2.3.1 Stability of disease-free equilibrium

For the proof of local stability, we use the following theorem found in [47].

Theorem 2.3.1. *Given the differential equation on* $\mathbb{R}^n$

$$x' = f(x),$$

let x_0 *be an equilibrium point of the above equation and* $A = Df(x_0)$ *be the Jacobian matrix of* f *at the point* x_0*. If all the eigenvalues of* A *have strictly negative real part, then* x_0 *is locally asymptotically stable.*

Now we prove the local stability of disease-free equilibrium point.

Theorem 2.3.2. *The disease-free equilibrium point E_0 is locally asymptotically stable if $\mathcal{R}_0 < 1$.*

Proof. Let $J(E_0)$ be the Jacobian matrix corresponding to system (2.1)–(2.4) evaluated at E_0, then

$$J(E_0) = \begin{bmatrix} -\mu_1 & -\frac{\beta_1 b_1}{\mu_1} + \phi & 0 & -\frac{\beta_2 b_1}{\mu_1} \\ 0 & \frac{\beta_1 b_1}{\mu_1} - \mu_1 - \phi & 0 & \frac{\beta_2 b_1}{\mu_1} \\ 0 & -\frac{\beta b_2}{\mu_2} & -\mu_2 & 0 \\ 0 & \frac{\beta b_2}{\mu_2} & 0 & -\mu_2 \end{bmatrix}$$

Now $|J(E_0) - \lambda I| = 0$ if and only if

$$(\lambda + \mu_1)(\lambda + \mu_2)\left[\lambda^2 + \lambda(\mu_2 + \mu_1 + \phi - \frac{\beta_1 b_1}{\mu_1}) + \mu_2(\mu_1 + \phi - \frac{\beta_1 b_1}{\mu_1}) - \frac{\beta\beta_2 b_1 b_2}{\mu_1\mu_2}\right] = 0$$

or equivalently

$$(\lambda + \mu_1)(\lambda + \mu_2)[\lambda^2 + \lambda(\mu_2 + \mu_1 + \phi - \frac{\beta_1 b_1}{\mu_1}) + \mu_2(\mu_1 + \phi)(1 - \mathcal{R}_0)] = 0.$$

Define $h(\lambda) =: (\lambda + \mu_1)(\lambda + \mu_2)(\lambda^2 + k_1\lambda + k_0)$, where

$$k_0 = \mu_2(\mu_1 + \phi)(1 - \mathcal{R}_0), \text{ and } k_1 = \mu_2 + \mu_1 + \phi - \frac{\beta_1 b_1}{\mu_1}.$$

If $\mathcal{R}_0 < 1$ then $k_0 > 0$ and also $\frac{\beta_1 b_1}{\mu_1(\mu_2+\phi)} \leq \mathcal{R}_0 < 1$. This implies $\phi + \mu_2 - \frac{\beta_1 b_1}{\mu_1(\mu_2+\phi)} > 0$ and therefore $k_1 > 0$.

Hence all the real roots of $h(\lambda)$ are negative and by theorem 2.3.1 we conclude that E_0 is locally asymptotically stable. □

The global stability of E_0 follows from the following Lasalle's invariance principle [47].

Theorem 2.3.3. *Let $\Omega \subset D$ be a compact set that is positively invariant with respect to $x' = f(x)$. Let $V : D \to \mathbb{R}$ be a C^1-function such that $V'(t) \leq 0$ on Ω.*
Let $E = \{x \in \Omega : V'(t) = 0\}$ and M be the largest invariant set in E. Then every solution starting in Ω approaches M as $t \to \infty$.

Now we show the disease-free equilibrium point E_0 is globally asymptotical stable.

Theorem 2.3.4. *If $\mathcal{R}_0 < 1$ then E_0 is globally asymptotical stable in Γ. The disease-free equilibrium point E_0 is unstable if $\mathcal{R}_0 > 1$ and the solutions to the system (2.1)–(2.4) starting sufficiently close to E_0 in Γ move away from E_0, except that those starting on the invariant S_h and S_v axis approach E_0 along these axes.*

Proof. Consider the Lyapunov function

$$L(S_h, I_h, S_v, I_v) = \left(S_h - \frac{b_1}{\mu_1} - \frac{b_1}{\mu_1} \ln S_h\right) + I_h + \frac{\mu_1 + \phi}{\beta}\left[(S_v - \frac{b_2}{\mu_2} - \frac{b_2}{\mu_2} \ln S_v) + I_v\right].$$

We can write the derivative as follows

$$\frac{dL}{dt} = -c_1 \frac{(S_h - \frac{b_1}{\mu_1})^2}{S_h} - c_2 \frac{(S_v - \frac{b_2}{\mu_2})^2}{S_v} - c_3(1 - \mathcal{R}_0) I_v,$$

where c_1, c_2 and c_3 are all positive constants. Thus if $\mathcal{R}_0 < 1$ then $\frac{dL}{dt} \leq 0$ and also $\frac{dL}{dt} = 0$ if and only if $S_h = \frac{b_1}{\mu_1}$, $S_v = \frac{b_2}{\mu_2}$ and $I_v = 0$. Therefore the largest compact invariant set in

$$E = \{(S_h, I_h, S_v, I_v) \in \Gamma : \frac{dL}{dt} = 0\}$$

is E_0. Thus by theorem 2.3.3, we conclude that the disease-free equilibrium is globally asymptotically stable in Γ. □

2.3.2 Stability of endemic equilibrium

Consider the endemic equilibrium $E_1 = (S_h^*, I_h^*, S_v^*, I_v^*)$ where S_h^*, I_h^*, S_v^* and I_v^* are given by equations (2.7) and (2.8). In this subsection, we study the local and global stability of the endemic equilibrium.

Theorem 2.3.5. *If $\mathcal{R}_0 > 1$ then E_1 is locally asymptotically stable.*

Proof. The proof is similar to that of theorem 2.3.2. Let $J(E_1)$ be the Jacobian matrix of system (2.5)–(2.6) at E_1, then

$$J(E_1) = \begin{bmatrix} \beta_2 I_v + \beta_1 I_h - \frac{\beta_1 b_1}{\mu_1} + \beta_1 I_h + \mu_1 + \phi & \beta_2(\frac{b_1}{\mu_1} - I_h) \\ \\ \beta(\frac{b_2}{\mu_2} - I_v) & \beta I_h + \mu_2 \end{bmatrix}.$$

Then the characteristics polynomial is given by $|J(E_1) - \lambda I| = \lambda^2 + a_1\lambda + a_0$, where

$$a_1 = \beta_2 I_v + \beta_1 I_h - \frac{\beta_1 b_1}{\mu_1} + \beta_1 I_h + \mu_1 + \phi + \beta I_h + \mu_2 \quad \text{and}$$

$$a_0 = (\beta I_h + \mu_2)\left(\beta_2 I_v + \beta_1 I_h - \frac{\beta_1 b_1}{\mu_1} + \beta_1 I_h + \mu_1 + \phi\right) - \beta\beta_2\left(\frac{b_1}{\mu_1} - I_h\right)\left(\frac{b_2}{\mu_2} - I_v\right).$$

Now using equation (2.7) and (2.8), we can simplify a_0 as follows

$$a_0 = \mu_1\beta\mu_2\beta_1 I_h^{*2} + \mu_1\mu_2^2\beta_1 I_h^* + \beta b_1\mu_2\beta_2 I_v + \mu_1\mu_2^2\beta_2 I_v^* > 0.$$

Also from $\frac{dI_h}{dt} = 0$, it follows that $\mu_1 + \phi = \beta_1 S_h + \beta_2 S_h \frac{I_v}{I_h}$.

Since $S_h = \frac{b_1}{\mu_1} - I_h$ we have,

$$\begin{aligned} a_1 &= \beta_2 I_v + \beta_1 I_h - \frac{\beta_1 b_1}{\mu_1} + \beta_1 I_h + \mu_1 + \phi + \beta I_h + \mu_2 \\ &= \beta_2 I_v + \beta_1 I_h - \beta_1 S_h + \mu_1 + \phi + \beta I_h + \mu_2 \\ &= \beta_2 I_v + \beta_1 I_h + \beta_2 S_h \frac{I_v}{I_h} + \beta I_h + \mu_2 > 0. \end{aligned}$$

Thus all the real part of the solutions of the characteristics polynomial are negative and by theorem 2.3.1 we conclude that E_1 is locally asymptotically stable. □

Next, we discuss the global stability of the endemic equilibrium point E_1. One way to prove this, as suggested by Cai and Li, is by defining a Lyapunov function $L(S_h, I_h, S_v, I_v)$ on Γ as follows [8].

$$L(t) = k_1(S_h - S_h^* - S_h^* \ln S_h) + k_2(I_h - I_h^* - I_h^* \ln I_h) + k_3(S_v - S_v^* - S_v^* \ln S_v) + k_4(I_v - I_v^* - I_v^* \ln I_v),$$

where

$$k_1 = k_2 = \beta S_v^* I_h^*, \quad k_3 = k_4 = \beta_2 S_h^* I_v^* + \beta_1 S_h^* I_h^*.$$

Then they showed that $\frac{dL}{dt} = 0$ if and only if $S_h = S_h^*, I_h = I_h^*, S_v = S_v^*$ and $I_v = I_v^*$. Thus by theorem 2.3.3, they concluded that E_1 is globally asymptotically stable.

Since constructing and computing the derivative of the Lyapunov function to prove the global stability of the endemic equilibrium E_1 is not easy, we provide another way to prove this assertion. For that purpose we use the Poincare-Bendixon Theorem. Before stating the main result, we give some definitions and examples that will be used later.

Definition 2.3.6. *Let A be an $n \times n$ matrix, the second additive compound matrix of A is an $\binom{n}{2} \times \binom{n}{2}$ matrix, denoted by $A^{[2]}$ and is defined as follows:*

$$A^{[2]}_{(i)(j)} = \begin{cases} A_{i_1 i_1} + A_{j_2 j_2} & \text{if } (i) = (j), \\ (-1)^{r+s} A_{i_r j_s} & \text{if exactly one entry } i_r \text{ of } (i) \text{ does not} \\ & \text{occur, in } (j) \text{ and } j_s \text{ does not occur in} \\ & (i) \text{ for some } r, s \in \{1, 2\}, \\ 0 & \text{if } (i) \text{ differs from } (j) \text{ in both entries.} \end{cases}$$

Example 2.3.7. *Let $A = (a_{ij})_{i,j}$ be a 3×3 matrix, then its second additive compound matrix is given by*

$$A^{[2]} = \begin{bmatrix} a_{11} + a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11} + a_3 & a_{12} \\ -a_{31} & a_{21} & a_{22} + a_{33} \end{bmatrix}.$$

Definition 2.3.8. *Let $x \to f(x) \in \mathbb{R}^n$ be a C^1 function for x in an open set $D \in \mathbb{R}^n$ and consider the following system of differential equations*

$$x' = f(x).$$

Then

1. *A set K is called **absorbing** in D if $x(t, K_1) \in K$ for each compact set $K_1 \subset D$ and t sufficiently large.*

2. *The system is said to have a **Poincare-Bendixon property (PBP)** if any nonempty compact omega limit set that contains no equilibria is a closed orbit (periodic orbit).*

3. *The above system is said to be **competitive** in D if for some diagonal matrix $H = diag\,(\epsilon_1, \epsilon_2, \ldots, \epsilon_n)$, $H\left(\frac{df}{dx}\right)H$ has non-positive off-diagonals where $\epsilon_i \in \{1, -1\}$.*

For more detail, please refer to [7, 31].

Definition 2.3.9. *Let X be a metric space with metric d. A map $f : X \to X$ defines a discrete semi-dynamical system $T : Z_+ \times X \to X$ by $T(n, x) = f^n(x)$. Let Y be a subspace of X, we say that f is uniformly persistent with respect to Y if there exists $\mu_2 > 0$ such that for all $x \in X \backslash Y$, $\liminf_{t \to \infty} d(f^n(x), Y) > \mu_2$ [7, 31].*

Uniform persistent captures the idea of non-extinction of the system. The following lemma can be used to check if the given system is uniformly persistent.

Lemma 2.3.10. *Let M be the maximal compact invariant set in Y then f is uniformly persistent with respect to Y if and only if*

1. *The set M is isolated in X and*

2. *The set $W^s(M) \subset Y$ with $W^s(M) := \{x \in X : f^n(x) \to M \quad as \quad n \to +\infty\}$ is the stable set of M.*

Next, we state the Poincare-Bendixon Theorem [7, 31].

Theorem 2.3.11. *Let $x \to f(x) \in \mathbb{R}^n$ be a C^1 function for $x \in D \subset \mathbb{R}^n$ and consider the system of differential equations $x' = f(x)$. Assume that*

1. *there exists a compact absorbing set $K \subset D$ and the above system has a unique equilibrium $\bar{x} \in D$;*

2. *the system satisfies the poincare-bendixon property;*

3. *any periodic orbit of the system is asymptotically orbitally stable and*

4. *the inequality $(-1)^n det(\frac{\partial f}{\partial x}(\bar{x})) > 0$ holds.*

Then the unique equilibrium $\bar{x}$ is globally asymptotically stable in D.

Note that system (2.1)–(2.4) can be reduced to the following equivalent system by replacing $S_v = \frac{b_2}{\mu_2} - I_v$.

$$\frac{dS_h}{dt} = b_1 - \mu_1 S_h - \beta_2 S_h I_v - \beta_1 S_h I_h + \phi I_h \tag{2.10}$$

$$\frac{dI_h}{dt} = \beta_1 S_h I_h + \beta_2 S_h I_v - (\mu_1 + \phi) I_h, \tag{2.11}$$

$$\frac{dI_v}{dt} = \beta \frac{b_2}{\mu_2} I_h - \beta I_v I_h - \mu_2 I_v. \tag{2.12}$$

Let

$$\tilde{\Gamma} := \left\{ (S_h, I_h, I_v) \in \mathbb{R}^3 : S_h + I_h = \frac{b_1}{\mu_1},\ 0 \leq I_v \leq \frac{b_2}{\mu_2} \right\}.$$

Then $\tilde{\Gamma}$ is invariant and attracting under system (2.10)–(2.12). Also the disease-free equilibrium and endemic equilibrium points of system (2.10)–(2.12) are $\tilde{E_0} = (\frac{b_1}{\mu_1}, 0, 0)$ and $\tilde{E_1} = (S_h^*, I_h^*, I_v^*)$, respectively, where S_h^*, I_h^*, I_v^* are given by equation (2.7) and (2.8).

Since system (2.1)–(2.4) and (2.10)–(2.12) are equivalent, we prove the global stability of the endemic equilibrium $\tilde{E}_1$ for the later system. Before that we state the following remark which will be used later [7, 31].

Remark 2.3.12. (i) In $\mathbb{R}^2$, any autonomous system satisfies the **PBP**, while in $\mathbb{R}^3$ a system satisfies the **PBP** if it is competitive and is defined on a convex region.

(ii) If $\mathcal{R}_0 > 1$, then $k_0 = -\mu_1\mu_2^2(\mu_1+\phi)(\mathcal{R}_0-1) < 0$. Hence $I_h^* = \frac{-k_1+\sqrt{k_1^2-4k_1k_0}}{2k_2} > 0$. Thus from equation (2.7) and (2.8) it follows that $S_h^* > 0$, $I_h^* > 0$ and $I_v^* > 0$. In conclusion, if $\mathcal{R}_0 > 1$, then system (2.10)–(2.12) has a unique equilibrium point $\tilde{E}_1$.

Lemma 2.3.13. *System* (2.10)–(2.12) *is uniformly persistent if* $\mathcal{R}_0 > 1$.

Proof. The set $\tilde{\Gamma}$ is positively invariant and globally attractive in $\mathbb{R}^3_+$. Also, $\{\tilde{E}_0\}$ is the maximum invariant set on $\partial\tilde{\Gamma}$ which is isolated. Now we need to show that $W^s(\{\tilde{E}_0\}) \subset \partial\tilde{\Gamma}$.

By contradiction suppose there exists a solution $S_h(t), I_h(t), I_v(t)$ of system (2.10)–(2.12) such that, $S_h(0) > 0, I_h(0) > 0, I_v(0) > 0$ and

$$\lim_{t\to\infty} S_h(t) = \frac{b_1}{\mu_1}, \quad \lim_{t\to\infty} I_h(t) = 0, \quad \lim_{t\to\infty} I_v(t) = 0.$$

Since $\mathcal{R}_0 = \frac{\beta\beta_2 b_1 b_2}{\mu_2^2\mu_1(\mu_1+\phi)} + \frac{\beta_1 b_1}{\mu_1(\mu_1+\phi)} > 1$ there is $\epsilon > 0$ such that,

$$\beta\beta_2\left(\frac{b_1}{\mu_1}-\epsilon\right)\left(\frac{b_2}{\mu_2}-\epsilon\right) + \beta_1\mu_2^2\left(\frac{b_1}{\mu_1}-\epsilon\right) > \mu_2^2\mu_1(\phi+\mu_1).$$

Also, for $\epsilon > 0$ there is $t_0 > 0$ such that, for any $t \geq t_0$

$$\frac{b_1}{\mu_1} - \epsilon < S_h(t) < \frac{b_1}{\mu_1} + \epsilon, \quad I_h(t) < \epsilon, \text{ and } \quad I_v(t) < \epsilon.$$

Now for any $t \geq t_0$, we have that

$$\begin{aligned}\frac{dI_h}{dt} &= \beta_2 S_h I_v + \beta_1 S_h I_h - (\mu_1 + \phi) I_h \\ &\geq \beta_2 \left(\frac{b_1}{\mu_1} - \epsilon\right) I_v + \mu_2 \left(\beta_1 (\frac{b_1}{\mu_1} - \epsilon) - \mu_1 - \phi\right) I_h,\end{aligned}$$

and

$$\frac{dI_v}{dt} = \frac{\beta b_2}{\mu_2} I_h - \beta I_v I_h - \mu_2 I_v \geq \beta (\frac{b_2}{\mu_2} - \epsilon) I_h - \mu_2 I_v.$$

Consider the following system of equations

$$\begin{cases} \frac{dx}{dt} = \mu_2 \left(\beta_1 (\frac{b_1}{\mu_1} - \epsilon) - \mu_1 - \phi\right) x + \beta_2 \left(\frac{b_1}{\mu_1} - \epsilon\right) y \\ \frac{dy}{dt} = \beta (\frac{b_2}{\mu_2} - \epsilon) x - \mu_2 y \\ x(t_0) = I_h(t_0), \quad y(t_0) = I_v(t_0). \end{cases} \tag{2.13}$$

Let

$$B = \begin{bmatrix} \mu_2 \left(\beta_1 (\frac{b_1}{\mu_1} - \epsilon) - \mu_1 - \phi\right) & \beta_2 \left(\frac{b_1}{\mu_1} - \epsilon\right) \\ \beta (\frac{b_2}{\mu_2} - \epsilon) & -\mu_2 \end{bmatrix},$$

then B is a quasi-positive matrix and

$$det(B) = -\left(\beta \beta_2 (\frac{b_1}{\mu_1} - \epsilon)(\frac{b_2}{\mu_2} - \epsilon) + \mu_2^2 \beta_1 (\frac{b_1}{\mu_1} - \epsilon) - \mu_2^2 \mu_1 (\mu_1 + \phi)\right) < 0.$$

Thus by Perron-Frobenius theorem, the spectral bound $S(B) \in \sigma(B)$ and there is a vector $v > 0$ corresponding to the positive eigenvalue λ such that $Bv = \lambda v$. Since $x(t_0) > 0$ and $y(t_0) > 0$ the solution of system (2.10)–(2.12) is unbounded. That is $I_h(t) \to \infty$ and $I_h(t) \to \infty$ as $t \to \infty$ which is a contradiction. Therefore $W^s(\{\tilde{E}_0\}) \subset \partial\tilde{\Gamma}$, and by lemma 2.3.10, we conclude that system (2.10)–(2.12) is uniformly persistent. □

Remark 2.3.14. The fact that $\tilde{\Gamma}$ is bounded and system (2.10)–(2.12) is uniformly persistent implies that this system has a compact absorbing set in $\tilde{\Gamma}$ [52, 31].

Lemma 2.3.15. *Assume $\mathcal{R}_0 > 1$ then system (2.10)–(2.12) is competitive in $\tilde{\Gamma}$.*

Proof. The Jacobian matrix of system (2.10)–(2.12) is given by

$$J = \begin{bmatrix} -\mu_1 - \beta_2 I_v - \beta_1 I_h & -\beta_1 S_h + \phi & -\beta_2 S_h \\ \beta_2 I_v + \beta_1 I_h & \beta_1 S_h - \mu_1 - \phi & \beta_2 S_h \\ 0 & \beta(\frac{b_2}{\mu_2} - I_v) & -\beta I_h - \mu_2 \end{bmatrix}.$$

Let $H = diag\,(-1, 1, -1)$. Then,

$$HJH = \begin{bmatrix} -\mu_1 - \beta_2 I_v - \beta_1 I_h & -\beta_1 S_h - \phi & -\beta_2 S_h \\ -\beta_2 I_v - \beta_1 I_h & \beta_1 S_h - \mu_1 - \phi & -\beta_2 S_h \\ 0 & -\beta(\frac{b_2}{\mu_2} - I_v) & -\beta I_h - \mu_2 \end{bmatrix}.$$

The off-diagonal elements of HJH are non-positive and thus, system (2.10)–(2.12) is competitive. □

In order to prove that any periodic orbit of system (2.10)–(2.12), if it exists, is asymptotically stable we use the following theorem [31].

Theorem 2.3.16. *A periodic orbit $\Omega = \{p(t) : 0 \leq t < \omega\}$ of the differential equation $x' = f(x)$ is orbitally asymptotically stable with asymptotic phase if the linear system*

$$z'(t) = \frac{\partial f^{[2]}}{\partial x}(p(t))z(t)$$

is asymptotically stable, where $\frac{\partial f^{[2]}}{\partial x}$ is the second additive compound matrix of the Jacobian matrix $\frac{\partial f}{\partial x}$.

Lemma 2.3.17. *Any periodic solution to system (2.10)–(2.12) if it exists, is asymptotically orbitally stable.*

Proof. The Jacobian matrix of system (2.10)–(2.12) is given by

$$J = \begin{bmatrix} -\mu_1 - \beta_2 I_v - \beta_1 I_h & -\beta_1 S_h + \phi & -\beta_2 S_h \\ \beta_2 I_v + \beta_1 I_h & \beta_1 S_h - \mu_1 - \phi & \beta_2 S_h \\ 0 & \beta\left(\frac{b_2}{\mu_2} - I_v\right) & -\beta I_h - \mu_2 \end{bmatrix}.$$

Then the corresponding second additive compound matrix $J^{[2]}$ will be

$$J^{[2]} = \begin{bmatrix} -2\mu_1 - \beta_2 I_v - \beta_1 I_h + \beta_1 S_h - \phi & \beta_2 S_h & \beta_2 S_h \\ \beta\left(\frac{b_2}{\mu_2} - I_v\right) & -\mu_1 - \beta_2 I_v - \beta_1 I_h - \beta I_h - \mu_2 & -\beta_1 S_h + \phi \\ 0 & \beta_2 I_v + \beta_1 I_h & \beta_1 S_h - \mu_1 - \phi - \beta I_h - \mu_2 \end{bmatrix}.$$

Suppose $(S_h(t), I_h(t), I_v(t)) \in \tilde{\Gamma}$ is a periodic solution of (2.10)–(2.12) of period τ, then its second compound system along the periodic solution is

$$\begin{aligned} \frac{dX}{dt} &= (-2\mu_1 - \beta_2 I_v - \beta_1 I_h + \beta_1 S_h - \phi)X + \beta_2 S_h(Y + Z) && (2.14) \\ \frac{dY}{dt} &= \beta\left(\frac{b_2}{\mu_2} - I_v\right)X - (\mu_1 + \beta_2 I_v + \beta_1 I_h + \beta I_h + \mu_2)Y - (\beta_1 S_h - \phi)Z && (2.15) \\ \frac{dZ}{dt} &= (\beta_2 I_v + \beta_1 I_h)Y - (-\beta_1 S_h + \mu_1 + \phi + \beta I_h + \mu_2)Z. && (2.16) \end{aligned}$$

We need to show that system (2.14)–(2.16) is asymptotically stable.

Define a Lyapunov function as follows

$$V(X, Y, Z, S_h, I_h, S_v, I_v) := \sup\{|X|, \frac{I_h}{I_v}(|Y| + |Z|)\}.$$

Calculating the right derivatives leads to the following inequalities:

$$\begin{aligned} D_+|X(t)| &\leq -(2\mu_1 + \beta_2 I_v + \beta_1 I_h - \beta_1 S_h + \phi)|X(t)| + \beta_2 S_h(|Y| + |Z|) && (2.17) \\ D_+|Y(t)| &\leq \beta\left(\frac{b_2}{\mu_2} - I_v\right)|X| - (\mu_1 + \beta_2 I_v + \beta_1 I_h + \beta I_h \mu_2)|Y| - (\beta_1 S_h - \phi)|Z| && (2.18) \\ D_+|Z(t)| &\leq (\beta_2 I_v + \beta_1 I_h)|Y| - (-\beta_1 S_h + \mu_1 + \phi + \mu_2 + \beta I_h)|Z|. && (2.19) \end{aligned}$$

Also

$$D_+\left\{\frac{I_h}{I_v}(|Y|+|Z|)\right\} = \frac{I_h}{I_v}(D_+|Y|+D_+|Z|) + (|Y|+|Z|)\left(\frac{I_h' I_v - I_h I_v'}{I_v^2}\right), \tag{2.20}$$

and

$$\begin{aligned}\frac{I_h}{I_v}(D_+|Y|+D_+|Z|) &= \frac{I_h}{I_v}(\beta(\frac{b_2}{\mu_2}-I_v)|X|) + \frac{I_h}{I_v}(-\mu_1-\beta I_h-\mu_2)|Y| - \frac{I_h}{I_v}(\mu_1+\mu_2+\beta I_h)|Z| \\ &= I_h\beta(\frac{b_2}{\mu_2}\frac{1}{I_v}-1)|X| - (\mu_1+\beta I_h+\mu_2)\left(\frac{I_h}{I_v}\right)(|Y|+|Z|). \end{aligned}\tag{2.21}$$

From equations (2.17)–(2.21) it follows that,

$$\begin{aligned}D_+\left\{\frac{I_h}{I_v}(|Y|+|Z|)\right\} &= I_h\beta(\frac{b_2}{\mu_2}\frac{1}{I_v}-1)|X| - (\mu_1+\beta I_h+\mu_2)\left(\frac{I_h}{I_v}\right)(|Y|+|Z|) \\ &\quad + \left(\frac{I_h'}{I_h}-\frac{I_v'}{I_v}\right)\left(\frac{I_h}{I_v}\right)(|Y|+|Z|). \end{aligned}\tag{2.22}$$

Now let

$$f_1 = -2\mu_1 - \beta_2 I_v - \beta_1 I_h + \beta_1 S_h - \phi + \frac{\beta_2 S_h}{I_h} I_v$$

$$f_2 = I_h\beta\left(\frac{b_2}{\mu_2}\frac{1}{I_v}-1\right) + \left(-\mu_1-\beta I_h-\mu_2+\frac{I_h'}{I_h}-\frac{I_v'}{I_v}\right).$$

Then

$$D_+V(t) \le Sup\{f_1, f_2\}V(t). \tag{2.23}$$

From equations (2.11) and (2.12) we obtain that

$$\frac{\beta b_2}{\mu_2}\frac{I_h}{I_v} - \beta I_h - \mu_2 - \frac{I_v'}{I_v} = 0 \quad \text{and} \quad \frac{\beta_2 S_h I_v}{I_h} + \beta_1 S_h - \mu_1 - \phi - \frac{I_h'}{I_h} = 0. \tag{2.24}$$

Using equation (2.24), we can simplify f_1 and f_2 as follows:

$$f_1 = \frac{I_h'}{I_h} - \mu_1 - \beta_2 I_v - \beta_1 I_v \quad \text{and} \quad f_2 = \frac{I_h'}{I_h} - \mu_1 - \beta I_h.$$

In conclusion we have

$$f_1 \le \frac{I_h'}{I_h} - \mu_1, \quad f_2 \le \frac{I_h'}{I_h} - \mu_1$$

and thus $\sup\{f_1(t), f_2(t)\} \leq \frac{I_h'}{I_h} - \mu_1$. Integrating both sides from 0 to τ we get

$$\begin{aligned}\int_0^\tau \sup\{f_1(t), f_2(t)\}dt &\leq \int_0^\tau \left(\frac{I_h'}{I_h} - \mu_1\right) dt \\ &= (ln(I_h(\tau)) - ln(I_h(0))) - \mu_1\tau \\ &= -\mu_1\tau.\end{aligned}$$

Now from inequality (2.23) it follows that $V(t) \leq ce^{-\mu_1\tau}$ and thus $V(t) \to 0$ as $t \to \infty$. This further implies that $X(t), Y(t), Z(t) \to 0$ as $t \to \infty$. In conclusion, the second compound system (2.14)–(2.16) is asymptotically stable and thus by theorem 2.3.16 the periodic orbit of system (2.10)–(2.12) is asymptotically orbitally stable. □

Lemma 2.3.18. *If $\mathcal{R}_0 > 1$ then $(-1)^n det(J(\tilde{E}_1)) > 0$ where $J(\tilde{E}_1)$ is the Jacobian of system* (2.10)–(2.12).

Proof. Consider the Jacobian matrix J of system (2.10)–(2.12),

$$det(J) = -\mu_1\beta_2\beta S_h(\frac{b_2}{\mu_2} - I_v) - \mu_1(\beta I_h + \mu_2)(\mu_1 + \beta_2 I_v + \beta_1 I_h + \beta_1 S_h - \phi).$$

Hence evaluating $det(J)$ at $\tilde{E}_1$ we get,

$$det(J(\tilde{E}_1)) = -\frac{\mu_1\beta\beta_2\mu_2 b_2(\mu_1 + \phi)}{\beta_1\mu_2(\mu_2 + \beta I_h^*) + \beta\beta_2 b_2} - h(I_h^*) < 0,$$

where $h(I_h^*) > 0$. In conclusion we have $(-1)^n det(J(\tilde{E}_1)) > 0$. □

The above discussions are summarized in the following theorem, which gives the global stability of the endemic equilibrium.

Theorem 2.3.19. *If $\mathcal{R}_0 > 1$ then $\tilde{E}_1$ is globally asymptotically stable.*

Proof. The first assumption of theorem 2.3.11 follows from lemma 2.3.13 and remark 2.3.14. The second assumption is concluded from remark 2.3.12 and lemma 2.3.15. Finally, the

third and fourth assumptions of the theorem follow from lemma 2.3.17 and lemma 2.3.18, respectively. □

2.3.3 Numerical simulation

To illustrate how the disease dynamics in the deterministic model is influenced by the value of the basic reproductive number $\mathcal{R}_0$, we carried out numerical simulations. In figure 2.1 (a), the value of $\mathcal{R}_0$ is 0.71, thus by theorem 2.3.4 the trajectories of the solution will approach the disease-free equilibrium point $E_0 = (72.30, 0, 40, 0)$ as shown in figure 2(a). In (b), $\mathcal{R}_0 = 34.20$ and hence by theorem 2.3.19 we conclude that the endemic equilibrium point is stable. Using (2.7) and (2.8) we have $E_1 = (83.48, 36.52, 2.42, 97.58)$ which agrees with the numerical result. The parameter values used for the simulation are given in table 2.1.

Table 2.1: Parameter values to determine the basic reproductive number.

Parameter	β_1	β_2	μ_2	β	ϕ	μ_1	b_1	b_2
Figure 2.1 (a)	0.005	0.003	0.02	0.0011	0.35	0.83	60	0.8
Figure 2.1 (b)	0.005	0.003	0.001	0.0011	0.35	0.83	100	0.1

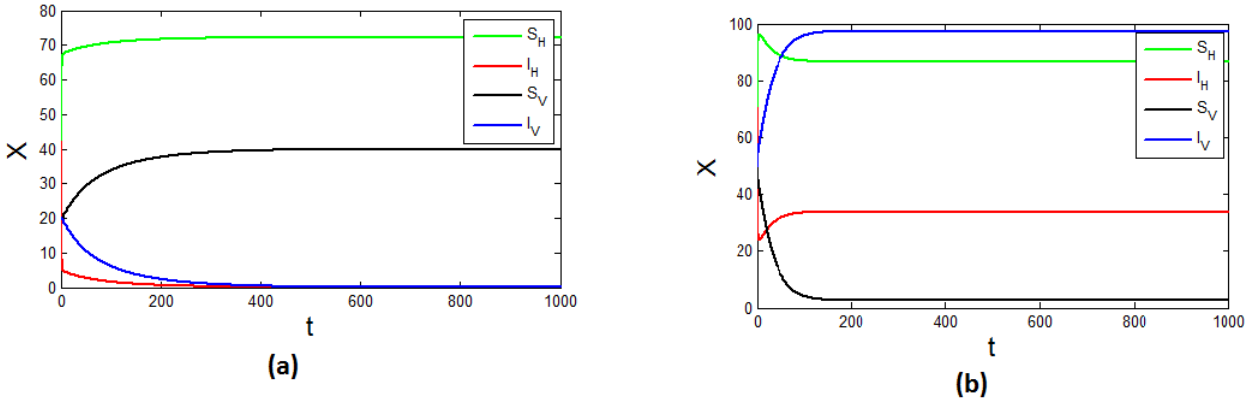

Figure 2.1: Trajectories of solution of the deterministic model (2.1)–(2.4), when $\mathcal{R}_0 = 0.71$ and $\mathcal{R}_0 = 34.20$ respectively.

2.4 Sensitivity analysis of $\mathcal{R}_0$

In this section we provide the sensitivity analysis of the basic reproductive number, that is, we study the effect of each parameter on the values of $\mathcal{R}_0$. In deterministic epidemic study the basic reproductive number and the endemic equilibrium are the two most important values. The first one tells us about the disease transmission or incidence rate, while the second describes how wide spread the disease is or the prevalence rate. In determining how best to reduce human mortality and morbidity due to the disease, it is necessary to know the relative importance of the different factors responsible for its transmission and prevalence. Estimation of parameters and initial conditions while modeling an epidemic is often subject to variation. The pre-fixed parameters are selected from a range, and consequently, the parameters may vary in a range. Varying the parameters varies the output of the model, but which parameters have the most significant impact on that output? There are different methods to study the sensitivity of parameters. One of such methods is the perturbation of fixed point estimation.

2.4.1 Sensitivity analysis of $\mathcal{R}_0$

The sensitivity analysis will determine the relative importance of the different parameters in relation to $\mathcal{R}_0$. For example consider β which is one of the parameters in $\mathcal{R}_0$. Let $\delta > 0$ be a small perturbation corresponding to β. We have

$$\delta_{\mathcal{R}_0} = \mathcal{R}_0(\beta + \delta) - \mathcal{R}_0(\beta) = \frac{\mathcal{R}_0(\beta + \delta) - \mathcal{R}_0(\beta)}{\delta}\delta \approx \delta \frac{\partial \mathcal{R}_0}{\partial \beta}.$$

Thus we define the normalized sensitivity index (si) as

$$si_\beta = \frac{\delta_{\mathcal{R}_0}}{\mathcal{R}_0} \Big/ \frac{\delta}{\beta} = \frac{\beta}{\mathcal{R}_0} \frac{\partial \mathcal{R}_0}{\partial \beta}.$$

Now using this formula we compute the following

$$
\begin{aligned}
si_{\beta} &= \tfrac{\beta\beta_2 b_1 b_2}{\beta\beta_2 b_1 b_2+\beta_1 b_1 \mu_2^2}, & si_{\beta_1} &= \tfrac{\beta_1 b_1 \mu_2^2}{\beta_1 b_1 \mu_2^2+\beta\beta_2 b_1 b_2} \\
si_{\beta_2} &= \tfrac{\beta\beta_2 b_1 b_2}{\beta\beta_2 b_1 b_2+\beta_1 b_1 \mu_2^2}, & si_{\mu_1} &= -\tfrac{2\mu_1+\phi}{\mu_1+\phi} \\
si_{\phi} &= -\tfrac{\phi}{\phi+\mu_1}, & si_{\mu_2} &= -\tfrac{2\beta\beta_2 b_1 b_2}{\beta_1 b_1 \mu_2^2+\beta\beta_2 b_1 b_2} \\
si_{b_1} &= 1, & si_{b_2} &= \tfrac{\beta\beta_2 b_1 b_2}{\beta_1 b_1 \mu_2^2+\beta\beta_2 b_1 b_2}
\end{aligned}
$$

Consider the following value of parameters for malaria epidemic. For more details about the data, please refer to [24].

$$
\begin{aligned}
b_1 &= 9\times 10^{-5},\ b_2 = 3.3\times 10^{-2},\ \beta_1 = 4\times 10^{-5},\ \beta_2 = 0.3, \\
\mu_2 &= 0.0033,\ \phi = 0.0035\,\beta = 0.48,\ \mu_1 = 9\times 10^{-5}
\end{aligned}
$$

Using the above values of parameters, the sensitivity indices of $\mathcal{R}_0$ is given in table 2.2.

The table shows the sensitivity indices of each parameter and the corresponding percentage change for a 1% change in $\mathcal{R}_0$. From the table we conclude that μ_2 is the most important parameters for $\mathcal{R}_0$ and in order to decrease $\mathcal{R}_0$ by 1% we need to increase μ_2 by 0.5%. Similarly the table shows that μ_1 is the second sensitive parameter, followed by b_1, β, β_2 and b_2.

Table 2.2: Sensitivity analysis of $\mathcal{R}_0$

Parameter	Sensitivity indices of $\mathcal{R}_0$	corresponding % change
β	0.9999	1
β_1	0.00001	100000
β_2	0.9999	1
μ_1	-1.0250	-0.9756
ϕ	-0.9749	-1.0257
μ_2	-1.9999	-0.5
b_1	1	1
b_2	0.9999	1

Chapter 3

Dynamics of the Stochastic Vector-host Epidemic Model with Direct Transmission

3.1 Derivation of the stochastic vector-host epidemic model

As described in the introduction part, for a better understanding of the spread of infections, we will include stochastic influences that are concerned with approximating the random or probabilistic element.

In this section we derive the stochastic vector-host epidemic model from the corresponding deterministic one by including a random effect in the deterministic case.

Let $\Delta t > 0$ be fixed and let $X^{(\Delta t)}(t) = \left(S_H^{(\Delta t)}(t), I_H^{(\Delta t)}(t), S_V^{(\Delta t)}(t), I_V^{(\Delta t)}(t)\right)$ be a discrete time Markov chain (DTMC) for $t \in \{0, \Delta t, 2\Delta t, \dots\}$, such that $X^{(\Delta t)}(0) \in \mathbb{R}_+^4$. Also let

$$\left\{R_{S_h}^{(\Delta t)}(k)\right\}_{k=0}^{\infty}, \quad \left\{R_{I_h}^{(\Delta t)}(k)\right\}_{k=0}^{\infty}, \quad \left\{R_{S_v}^{(\Delta t)}(k)\right\}_{k=0}^{\infty}, \quad \left\{R_{I_v}^{(\Delta t)}(k)\right\}_{k=0}^{\infty}$$

be sequences of random variables which are jointly independent to each other and each sequence is identically distributed such that, for any $k \in \{0, 1, 2, \dots\}$

$$E[R_{S_h}^{(\Delta t)}(k)] = E[R_{I_h}^{(\Delta t)}(k)] = E[R_{S_v}^{(\Delta t)}(k)] = E[R_{I_v}^{(\Delta t)}(k)] = 0, \tag{3.1}$$

and

$$\begin{aligned} E\left[R_{S_h}^{(\Delta t)}(k)\right]^2 &= \sigma_{S_h}^2 \Delta t, \;\; E\left[R_{I_h}^{(\Delta t)}(k)\right]^2 = \sigma_{I_h}^2 \Delta t, \\ E\left[R_{S_v}^{(\Delta t)}(k)\right]^2 &= \sigma_{S_v}^2 \Delta t, \;\; E\left[R_{I_v}^{(\Delta t)}(k)\right]^2 = \sigma_{I_v}^2 \Delta t. \end{aligned} \tag{3.2}$$

where E is the expectation and $\sigma_{S_h}, \sigma_{I_h}, \sigma_{S_v}, \sigma_{I_v}$ are some non-negative constants which show the intensity of the fluctuations.

Each sequence of random variables measures the effects of random influence on each compartment during $[k\Delta t, (k+1)\Delta t]$ for $k \in \{0, 1, 2, \dots\}$. Thus, during $[k\Delta t, (k+1)\Delta t]$,

each compartment changes according to the deterministic equation (2.1)–(2.4) and by a random amount. That is, for $k \in \{0, 1, 2, \dots\}$

$$
\begin{aligned}
S_h^{(\Delta t)}((k+1)\Delta t) &= S_h^{(\Delta t)}(k\Delta t) + \Delta t(b_1 - \mu_1 S_h - \beta_2 S_h I_v - \beta_1 S_h I_h + \phi I_h) + R_{S_h}^{(\Delta t)}(k) S_h^{\Delta t}(k\Delta t) \\
I_h^{(\Delta t)}((k+1)\Delta t) &= I_h^{(\Delta t)}(k\Delta t) + \Delta t(\beta_1 S_h I_h + \beta_2 S_h I_v - (\mu_1 + \phi) I_h) + R_{I_h}^{(\Delta t)}(k) I_h^{\Delta t}(k\Delta t) \\
S_v^{(\Delta t)}((k+1)\Delta t) &= S_v^{(\Delta t)}(k\Delta t) + \Delta t(b_2 - \mu_2 S_v - \beta S_v I_h) + R_{S_v}^{(\Delta t)}(k) S_v^{\Delta t}(k\Delta t) \\
I_v^{(\Delta t)}((k+1)\Delta t) &= I_v^{(\Delta t)}(k\Delta t) + \Delta t(\beta S_v I_h - \mu_2 I_v) + R_{I_v}^{(\Delta t)}(k) I_v^{\Delta t}(k\Delta t)
\end{aligned}
$$

We claim that $X^{\Delta t}(t)$ converges to a diffusion process $X(t) = (S_h, I_h, S_v, I_v)$ as $\Delta t \to 0$. For that purpose let $\Pi^{(\Delta t)}(k\Delta t, x; (k+1)\Delta t, A)$ be the transition probability of the homogenous Markov chain $\left\{X^{(\Delta t)}(k\Delta t)\right\}_{k=0}^{\infty}$. That is

$$
\Pi^{(\Delta t)}(k\Delta t, x; (k+1)\Delta t, A) = \mathbb{P}\left\{X^{(\Delta t)}((k+1)\Delta t) \in A : X^{(\Delta t)}(k\Delta t) = x\right\}
$$

for all $x = (x_1^0, x_2^0, x_3^0, x_4^0) \in \mathbb{R}^4$ and all Borel set $A \subset \mathbb{R}^4$.

First, we determine the drift coefficient of the diffusion process.

Let $y = (y_1^0, y_2^0, y_3^0, y_4^0) \in \mathbb{R}^4$ then for any $\epsilon > 0$ we have:

$$
\begin{aligned}
\frac{1}{\Delta t}\int_{|y-x|\le\epsilon} (y_1^0 - x_1^0)\Pi^{(\Delta t)} &= \frac{1}{\Delta t}\left\{E\left[\Delta t(b_1 - \mu_1 S_h - \beta_2 S_h I_v - \beta_1 S_h I_h + \phi I_h) + R_{S_h}^{(\Delta t)}(0) x_1^0\right]\right\} \\
&= b_1 - \mu_1 S_h - \beta_2 S_h I_v - \beta_1 S_h I_h + \phi I_h + \frac{1}{\Delta t} E\left(R_{S_h}^{\Delta t} x_1^0\right) \\
&= b_1 - \mu_1 S_h - \beta_2 S_h I_v - \beta_1 S_h I_h + \phi I_h.
\end{aligned}
$$

Similarly,

$$
\begin{aligned}
\tfrac{1}{\Delta t}\textstyle\int_{|y-x|\le\epsilon} (y_2^0 - x_2^0)\Pi^{(\Delta t)} &= \beta_1 S_h I_h + \beta_2 S_h I_v - (\mu_1 + \phi) I_h. \\
\tfrac{1}{\Delta t}\textstyle\int_{|y-x|\le\epsilon} (y_3^0 - x_3^0)\Pi^{(\Delta t)} &= b_2 - \mu_2 S_v - \beta S_v I_h. \\
\tfrac{1}{\Delta t}\textstyle\int_{|y-x|\le\epsilon} (y_4^0 - x_4^0)\Pi^{(\Delta t)} &= \beta S_v I_h - \mu_2 I_v.
\end{aligned}
$$

In conclusion the drift coefficient of the diffusion process is given by

$$f(X(t),t) = \begin{bmatrix} b_1 - \mu_1 S_h - \beta_2 S_h I_v - \beta_1 S_h I_h + \phi I_h \\ \beta_1 S_h I_h + \beta_2 S_h I_v - (\mu_1 + \phi) I_h \\ b_2 - \mu_2 S_v - \beta S_v I_h \\ \beta S_v I_h - \mu_2 I_v \end{bmatrix} \tag{3.3}$$

Next we determine the diffusion coefficient matrix of the process.

Let $g_{ij}^{\Delta t}(x) = \frac{1}{\Delta t}\int_{|y-x|\leq\epsilon}(y_i - x_i)(y_j - x_j)\Pi^{(\Delta t)}$ be the moment for $i, j = 1, 2, 3, 4$.

If the compartments are the same, that is if $i = j$ we have,

$$\left| g_{S_h S_h}^{(\Delta t)}(x) - \sigma_{S_h}^2 S_h^2 \right| = E[R_{S_h}^{(\Delta t)}(0)]^2 \frac{S_h^2}{\Delta t} - \sigma_{S_h}^2 S_h^2 = 0.$$

Thus for any $k > 0$ if follows that

$$\limsup_{\Delta t \to 0 \quad \|x\| \leq k} \left| g_{S_h S_h}^{(\Delta t)}(x) \right| = \sigma_{S_h}^2 S_h^2.$$

Similarly we have

$$\limsup_{\Delta t \to 0 \quad \|x\| \leq k} \left| g_{I_h I_h}^{(\Delta t)}(x) \right| = \sigma_{I_h}^2 I_h^2, \qquad \limsup_{\Delta t \to 0 \quad \|x\| \leq k} \left| g_{S_v S_v}^{(\Delta t)}(x) \right| = \sigma_{S_v}^2 S_v^2$$

and

$$\limsup_{\Delta t \to 0 \quad \|x\| \leq k} \left| g_{I_v I_v}^{(\Delta t)}(x) \right| = \sigma_{I_v}^2 I_v^2.$$

If we take any two different compartments, that is if $i \neq j$ we have

$$\limsup_{\Delta t \to 0 \quad \|x\| \leq k} \left| g_{ij}^{(\Delta t)}(x) \right| = 0.$$

To give an example, consider two compartments S_h and I_v. Then,

$$\begin{aligned}|g^{(\Delta t)}_{S_h I_v}(x)| &= \frac{1}{\Delta t}|E([\Delta t(b_1 - \mu_1 S_h - \beta_2 S_h I_v - \beta_1 S_h I_h + \phi I_h) + R^{(\Delta t)}_{S_h}(k)S_h(k\Delta t)] \\ &\quad [\Delta t(\beta S_v I_h(k\Delta t) - \mu_2 I_v(k\Delta t)) + R^{(\Delta t)}_{I_v}(k)I_v(k\Delta t)])| \\ &= \Delta t(b_1 - \mu_1 S_h - \beta_2 S_h I_v - \beta_1 S_h I_h + \phi I_h)(\beta S_v I_h - \mu_2 I_v) \\ &\quad + E(R^{(\Delta t)}_{I_v}(k)I_v)(b_1 - \mu_1 S_h - \beta_2 S_h I_v - \beta_1 S_h I_h + \phi I_h) \\ &\quad + E(R^{(\Delta t)}_{S_h}(k))S_h)(\beta S_v I_h - \mu_2 I_v) + E(R^{(\Delta t)}_{S_h}(k))E(R^{(\Delta t)}_{I_v}(k))S_h I_v\end{aligned}$$

Using equations (3.1) and (3.2), for any $x \in \mathbb{R}^4$ such that $\|x\| \leq k$ it follows that

$$\limsup_{\Delta t \to 0 \quad \|x\| \leq k} \left| g^{(\Delta t)}_{S_h I_v}(x) \right| = 0.$$

Thus the diffusion coefficient matrix is given by

$$g(X(t), t) = diag\,(\sigma_{S_h} S_h,\, \sigma_{I_h} I_h,\, \sigma_{S_v} S_v,\, \sigma_{I_v} I_v). \tag{3.4}$$

In conclusion as $\Delta t \to 0$, $X^{\Delta t}(t)$ converges to a diffusion process $X(t) = (S_h, I_h, S_v, I_v)$ which satisfies the stochastic differential equation

$$dX(t) = f(X, t)dt + g(X, t)dB(t)$$

where $B(t) = (B_{S_h}, B_{I_h}, B_{S_v}, B_{I_v})$ is a $4-$ dimensional standard Brownian motion such that $B_{S_h}, B_{I_h}, B_{S_v}, B_{I_v}$ are independent to each other. Using equations (3.3) and (3.4) the above

stochastic differential equation can be written as

$$\begin{aligned} dS_h &= (b_1 - \mu_1 S_h - \beta_2 S_h I_v - \beta_1 S_h I_h + \phi I_h)dt + \sigma_{S_h} S_h dB_{S_h}(t), && (3.5)\\ dI_h &= (\beta_1 S_h I_h + \beta_2 S_h I_v - (\mu_1 + \phi) I_h)dt + \sigma_{I_h} I_h dB_{I_h}(t), && (3.6)\\ dS_v &= (b_2 - \mu_2 S_v - \beta S_v I_h)dt + \sigma_{S_v} S_v dB_{S_v}(t), && (3.7)\\ dI_v &= (\beta S_v I_h - \mu_2 I_v)dt + \sigma_{I_v} I_v dB_{I_v}(t). && (3.8) \end{aligned}$$

Throughout this chapter, let $(\Omega, \mathcal{F}, \mathbb{P})$ be a complete probability space with a filtration $\{\mathcal{F}\}_{t\geq 0}$ satisfying the usual conditions, that is, it is right continuous and increasing with $\mathcal{F}_0$ containing all $\mathbb{P}$-null sets. Let $X(t) = (S_h(t), I_h(t), S_v(t), I_v(t))$ be the solution of system (3.5)–(3.8) and define

$$\|X(t)\| = (S_h(t)^2 + I_h(t)^2 + S_v(t)^2 + I_v(t)^2)^{\frac{1}{2}}.$$

In this section we provide some basic preliminaries from [38] and [60] that will be used throughout the remaining chapters.

Definition 3.1.1. *1. Let $\mathbb{L}^1(0,T)$ and $\mathbb{L}^2(0,T)$ denotes the space of all real valued, progressively measurable stochastic processes $G(\cdot)$ such that*

$$E\left(\int_0^T G dt\right) < \infty \quad \textit{and} \quad E\left(\int_0^T G^2 dt\right) < \infty.$$

2. Let $X(t)$ satisfies the stochastic differential equation

$$dX(t) = f(X,t)dt + g(X,t)dB(t),$$

and let $U : \mathbb{R}^d \times \mathbb{R}_+ \to \mathbb{R}^d$ have continuous partial derivatives $\frac{\partial U}{\partial t}$, $\frac{\partial U}{\partial x_k}$, $\frac{\partial^2 U}{\partial x_k \partial x_i}$ for $k, i = 1, 2, \ldots, d$. Define the process $Y(t) = U(X,t)$, then the stochastic differential is

given by $dY(t) = \mathcal{L}U(X,t) + \nabla U^T g(X,t)\, dB(t)$ *where,*

$$\mathcal{L}U(X,t) = \left\{ \frac{\partial U}{\partial t} + f(X,t)\nabla U + \frac{1}{2} tr\left(g(X,t)g(X,t)^T \nabla[\nabla U]\right) \right\} dt. \tag{3.9}$$

3.1.1 Existence of a global solution

In this subsection we show that the stochastic system (3.5)–(3.8) has a unique nonnegative global solution. Since f and g satisfies the local Lipschitz and Linear growth conditions, there exists a unique local solution $X(t)$ on $[0, \tau_e)$ where τ_e is the explosion time, refer to theorem 3.15 [38]. Next, we show that $\tau_e = \infty$, that is, this solution is in fact global.

Let $k_0 > 0$ such that $X(0) \in \left(\frac{1}{k_0}, k_0\right)^4$. For any $k \in \mathbb{N}$ such that $k > k_0$ define
$\tau_k = inf\left\{t \in [0, \tau_e] : (S_h(t), I_h(t), S_v(t), I_v(t)) \notin \left(\frac{1}{k}, k\right)^4\right\}$.
Then $\{\tau_k\}_k$ is an increasing sequence and denote $\tau := \lim_{k\to\infty} \tau_k$. Clearly $\tau \leq \tau_e$.

Next, we show that $\tau = \infty$. This implies that the explosion time is infinity and thus we conclude that system (3.5)–(3.8) has a unique non-negative solution and will remain in $\mathbb{R}^4_+$ with probability 1.

By contradiction suppose $\tau < \infty$. Then there exists $T > 0$ such that
$\mathbb{P}(\tau \leq T) > \epsilon$ for all $\epsilon \in (0,1)$. This implies that there exists $k_1 > k_0$ such that $\mathbb{P}(\tau_k \leq T) \geq \epsilon$ for all $k \geq k_1$. For $X(t) \in \mathbb{R}^4_+$ define

$$V(X(t)) = (S_h - 1 - \ln S_h) + (I_h - 1 - \ln I_h) + (S_v - 1 - \ln S_v) + (I_v - 1 - \ln I_v).$$

Using equation (3.9) we have

$$dV = \mathcal{L}V dt + \sigma_{S_h}(S_h - 1)dB_{S_h} + \sigma_{I_h}(I_h - 1)dB_{I_h} + \sigma_{S_v}(S_v - 1)dB_{S_v} + \sigma_{I_v}(I_v - 1)dB_{I_v},$$

where

$$\begin{aligned}\mathcal{L}V &= \left(1-\frac{1}{S_h}\right)dS_h + \left(1-\frac{1}{I_h}\right)dI_h + \left(1-\frac{1}{S_v}\right)dS_v + \left(1-\frac{1}{I_v}\right)dI_v \\ &\quad + \frac{1}{2}(\sigma_{S_h}^2 + \sigma_{I_h}^2 + \sigma_{S_v}^2 + \sigma_{I_v}^2) \\ &\leq b_1 - \mu_1 S_h - \mu_1 I_h + b_2 - \mu_2 I_v - \mu_2 S_v + \frac{1}{2}(\sigma_{S_h}^2 + \sigma_{I_h}^2 + \sigma_{S_v}^2 + \sigma_{I_v}^2) \\ &\leq C_1 = b_1 + b_2 + \frac{1}{2}(\sigma_{S_h}^2 + \sigma_{I_h}^2 + \sigma_{S_v}^2 + \sigma_{I_v}^2),\end{aligned}$$

where the last inequality follows from (3.5)–(3.8). In conclusion we have,

$$dV \leq C_1 dt + \sigma_{S_h}(S_h - 1)dB_{S_h} + \sigma_{I_h}(I_h - 1)dB_{I_h} + \sigma_{S_v}(S_v - 1)dB_{S_v} + \sigma_{I_v}(I_v - 1)dB_{I_v}.$$

Integrating both sides on $(0, \tau_k \wedge T)$, taking the expectation and noting that for any $G \in \mathbb{L}^2(0,T)$, $E\left(\int_0^T G dB\right) = 0$, we get

$$EV(X(\tau_k \wedge T)) \leq V(X(0)) + C_1 T.$$

For $k \in \mathbb{N}$ such that $k \geq k_0$ let $A_k = \{\tau_k \leq T\}$. Then, $\mathbb{P}(A_k) \geq \epsilon$. If $t \in A_k$, then at least one of the following will hold true:

$$S_h(t) \notin \left(\frac{1}{k}, k\right), \quad I_h(t) \notin \left(\frac{1}{k}, k\right), \quad S_v(t) \notin \left(\frac{1}{k}, k\right), \quad I_v(t) \notin \left(\frac{1}{k}, k\right).$$

Generally, since $f(x) = x - 1 - \ln x$ is increasing on $(1, \infty)$ and decreasing on $(0, 1)$ it follows

$$V(X(\tau_k \wedge t)) \geq (k - 1 - \ln k) \wedge (\frac{1}{k} - 1 - \ln(\frac{1}{k})).$$

Now we have

$$V(X(0)) + C_1 T \geq EV(X(\tau_k \wedge T)) \geq \epsilon \left((k - 1 - \ln k) \wedge (\frac{1}{k} - 1 - \ln \frac{1}{k})\right).$$

Finally, letting $k \to \infty$ we conclude that

$$\infty > V(X(0)) + C_1 T = \infty \text{ a.s.}$$

Hence, we have $\tau = \infty$. We summarize the above result in the following theorem.

Theorem 3.1.2. *For any initial value $X(0) \in \mathbb{R}^4_+$ the system (3.5)–(3.8) has a unique global solution on $t \geq 0$ and the solution will remain in $\mathbb{R}^4_+$ with probability 1.*

3.2 Stochastic boundedness and permanence

Theorem 3.1.2 shows that for any initial condition $X(0) \in \mathbb{R}^4_+$ the solution of model (3.5)–(3.8) is always positive and remains in $\mathbb{R}^4_+$. Next we exam how $X(t)$ varies in $\mathbb{R}^4_+$. First, we give the definition of a stochastically ultimately bounded solution.

Definition 3.2.1. *[37] The solution $X(t)$ of system (3.5)–(3.8) is called stochastically ultimately bounded or ultimately bounded in probability if for any $\epsilon \in (0, 1)$ there is a constant $\chi = \chi(\epsilon) > 0$ such that for any initial solution $X(0) \in \Gamma$, the solution $X(t)$ to system (3.5)–(3.8) has the property that*

$$\limsup_{t \to \infty} P\{\|X(t)\| > \chi\} \leq \epsilon.$$

Lemma 3.2.2. *For any initial value $X(0) \in \Gamma$ and $\theta > 1$, there exists $\kappa = \kappa(\theta) > 0$ such that the solution of system (3.5)–(3.8) satisfies*

$$\limsup_{t \to \infty} E\{\|X(t)\|^\theta\} < \kappa(\theta).$$

Proof. Define $V_1(S_h(t), I_h(t)) = S_h^\theta + I_h^\theta$. Then, we have

$$\begin{aligned}
\mathcal{L}V_1 &= \theta S_h^{\theta-1}\, dS_h + \frac{1}{2}\theta(\theta-1)S_h^{\theta-2}\,\sigma_{S_h}^2 S_h^2 + \theta I_h^{\theta-1}\, dI_h + \frac{1}{2}\theta(\theta-1)I_h^{\theta-2}\sigma_{I_h}^2 I_h^2 \\
&= \theta S_h^{\theta-1}(b_1 - \mu_1 S_h - \beta_2 S_h I_v - \beta_1 S_h I_h + \phi I_h) + \frac{1}{2}\theta(\theta-1)S_h^\theta \sigma_{S_h}^2 \\
&+ \theta I_h^{\theta-1}(\beta_2 S_h I_v + \beta_1 S_h I_h - \mu_1 I_h - \phi I_h) + \frac{1}{2}\theta(\theta-1)I_h^\theta \sigma_{I_h}^2 \\
&\leq \theta(\frac{b_1}{\mu_1})^{\theta-1}(b_1 - \mu_1 S_h - \beta_2 S_h I_v - \beta_1 S_h I_h + \phi I_h + \beta_2 S_h I_v + \beta_1 S_h I_h - \mu_1 I_h - \phi I_h) \\
&+ \frac{1}{2}\theta(\theta-1)(S_h^\theta \sigma_{S_h}^2 + I_h^\theta \sigma_{I_h}^2) \\
&= \theta(\frac{b_1}{\mu_1})^{\theta-1}(b_1 - \mu_1 S_h - \mu_1 I_h) + \frac{1}{2}\theta(\theta-1)(S_h^\theta \sigma_{S_h}^2 + I_h^\theta \sigma_{I_h}^2) \\
&\leq \theta(\frac{b_1}{\mu_1})^{\theta-1} b_1 + \frac{1}{2}\theta(\theta-1)(\frac{b_1}{\mu_1})^\theta(\sigma_{S_h}^2 + \sigma_{I_h}^2) \\
&= \left(\frac{b_1}{\mu_1}\right)^{\theta-1}\left(\theta b_1 + \frac{b_1\theta(\theta-1)(\sigma_{S_h}^2 + \sigma_{I_h}^2)}{2\mu_1}\right) := M_1.
\end{aligned}$$

Similarly, letting $V_2(S_v, I_v) = S_v^\theta + I_v^\theta$ we have $\mathcal{L}V_2 \leq M_2$, where

$$M_2 = \left(\frac{b_2}{\mu_2}\right)^{\theta-1}\left(\theta b_2 + \frac{b_2\theta(\theta-1)(\sigma_{S_v}^2 + \sigma_{I_v}^2)}{2\mu_2}\right).$$

Let $V = V_1 + V_2$. Then $V \leq M_3 = \left((\frac{b_1}{\mu_1})^\theta + (\frac{b_2}{\mu_2})^\theta\right) < \infty$ and by 3.9 we have

$$\begin{aligned}
dV = dV_1 + dV_2 &= (\mathcal{L}V_1 + \mathcal{L}V_2)dt + \theta(\sigma_{S_h} S_h^\theta dB_{S_h} + \sigma_{I_h} I_h^\theta dB_{I_h} + \sigma_{S_v} S_v^\theta dB_{S_v} + \sigma_{I_v} I_v^\theta dB_{I_v}) \\
&\leq (M_1 + M_2)dt + \theta(\sigma_{S_h} S_h^\theta dB_{S_h} + \sigma_{I_h} I_h^\theta dB_{I_h} + \sigma_{S_v} S_v^\theta dB_{S_v} + \sigma_{I_v} I_v^\theta dB_{I_v})
\end{aligned}$$

and

$$\begin{aligned}
d(e^t V) &= e^t(V + \mathcal{L}V)dt + e^t\theta(\sigma_{S_h} S_h^\theta dB_{S_h} + \sigma_{I_h} I_h^\theta dB_{I_h} + \sigma_{S_v} S_v^\theta dB_{S_v} + \sigma_{I_v} I_v^\theta dB_{I_v}) \\
&\leq M e^t dt + e^t\theta(\sigma_{S_h} S_h^\theta dB_{S_h} + \sigma_{I_h} I_h^\theta dB_{I_h} + \sigma_{S_v} S_v^\theta dB_{S_v} + \sigma_{I_v} I_v^\theta dB_{I_v}),
\end{aligned}$$

where $M = M_1 + M_2 + M_3$.

Let $k_0 > 0$, such that $X(0) \in (\frac{1}{k_0}, k_0)^4$ and for $k > k_0$ let

$$\tau_k = inf \left\{ t > 0 : (S_h(t), I_h(t), S_v(t), I_v(t)) \notin \left(\frac{1}{k}, k\right)^4 \right\}.$$

Integrating and taking the expectation on both sides, we get

$$\begin{aligned} E(e^{t\wedge\tau_k} V(X(t \wedge \tau_k))) &\leq ME(\int_0^{t\wedge\tau_k} e^s ds) + V(X(0)) \\ &= ME(e^{t\wedge\tau_k} - 1) + V(X(0)), \end{aligned}$$

Letting $k \to \infty$ we obtain

$$EV(X(t)) \leq e^{-t} V(X(0)) + M(1 - e^{-t}).$$

Note that $\|X(t)\|^\theta \leq 2^\theta V(X(t))$. Thus

$$E\|X(t)\|^\theta \leq 2^\theta EV(X(t)) \leq 2^\theta \left(e^{-t} V(X(0)) + M(1 - e^{-t})\right).$$

and it follows that

$$\limsup_{t\to\infty} E\{\|X(t)\|^\theta\} \leq \kappa(\theta),$$

where $\kappa(\theta) = 2^\theta M$. □

Using the above lemma we show that the solution of system (3.5)–(3.8) is stochastically ultimately bounded.

Theorem 3.2.3. *For any initial value $X(0) \in \Gamma$, the solution of system (3.5)–(3.8) is stochastically ultimately bounded.*

Proof. By the previous lemma, there exist a positive constant $\zeta > 0$ such that

$$\limsup_{t\to\infty} E\{\|X(t)\|^{\frac{1}{2}}\} < \zeta.$$

For any $\epsilon > 0$ put $\chi(\epsilon) = \frac{\zeta^2}{\epsilon^2}$. Then, by Chebyshev's inequality we get

$$\mathbb{P}\{\|X(t)\| > \chi\} \leq \frac{E(\|X\|^{\frac{1}{2}})}{\chi^{\frac{1}{2}}}.$$

This concludes $\limsup_{t\to\infty} \mathbb{P}\{\|X(t)\| > \chi\} \leq \frac{\zeta}{\chi^{\frac{1}{2}}} = \epsilon$. □

Lemma 3.2.4. *Let $k := min\{\mu_1, \mu_2\}$, $\sigma^2 := max\{\sigma^2_{S_h}, \sigma^2_{I_h}, \sigma^2_{S_v}, \sigma^2_{I_v}\}$ and assume that $b_1 + b_2 - k > 0$. Then, for any initial value $X(0) \in \Gamma$ the solution $X(t)$ of system (3.5)–(3.8) satisfies*

$$\limsup_{t\to\infty} E\left(\frac{1}{\|X(t)\|^\nu}\right) \leq M,$$

where

$$M = \frac{4^\nu}{\theta}\frac{a_2^2 + 4a_1\theta}{4a_1}\, max\left\{1, \left(1 + \frac{a_2 + \sqrt{a_2^2 + 4a_1\theta}}{2a_1}\right)^{\nu-2}\right\},$$

$$a_1 = -\theta + \nu(b_1 + b_2 - k - \frac{\nu+1}{2}\sigma^2),$$

$$a_2 = 2\theta + \nu k + \nu\sigma^2.$$

Here $\nu > 0$ and $\theta > 0$ are any constants satisfying the following conditions:

$$k + \frac{\nu+1}{2}\sigma^2 - b_1 - b_2 < 0, \text{ and } \theta < \nu(b_1 + b_2 - k - \frac{\nu+1}{2}\sigma^2)$$

Proof. Let $U(S_h, I_h, S_v, I_v) = \frac{1}{S_h + I_h + S_v + I_v}$. Then, using (3.9)

$$dU = \mathcal{L}U dt - U^2(\sigma_{S_h} S_h dB_{S_h} + \sigma_{I_h} I_h dB_{I_h} + \sigma_{S_v} S_v dB_{S_v} + \sigma_{I_v} I_v dB_{I_v}),$$

where

$$\mathcal{L}U = -U^2(b_1 + b_2 - \mu_1(S_h + I_h) - \mu_2(S_v + I_v)) + U^3(\sigma^2_{S_h} S_h^2 + \sigma^2_{I_h} I_h^2 + \sigma^2_{S_v} S_v^2 + \sigma^2_{I_v} I_v^2).$$

Let ν be as in the assumption, then

$$\begin{aligned}\mathcal{L}((1+U)^\nu) &= \nu(1+U)^{\nu-1}\mathcal{L}U + \frac{\nu(\nu-1)}{2}(1+U)^{\nu-2}U^4(\sigma_{S_h}^2 S_h^2 + \sigma_{I_h}^2 I_h^2 + \sigma_{S_v}^2 S_v^2 + \sigma_{I_v}^2 I_v^2)) \\ &= \nu(1+U)^{\nu-2}\psi,\end{aligned}$$

where

$$\begin{aligned}\psi &= (1+U)\mathcal{L}U + \frac{\nu-1}{2}U^4(\sigma_{S_h}^2 S_h^2 + \sigma_{I_h}^2 I_h^2 + \sigma_{S_v}^2 S_v^2 + \sigma_{I_v}^2 I_v^2) \\ &\leq -U^3(b_1+b_2-\frac{k}{U}) - U^2(b_1+b_2-\frac{k}{U}) + (U^3 + \frac{\nu+1}{2}U^4)(\sigma_{S_h}^2 S_h^2 + \sigma_{I_h}^2 I_h^2 + \sigma_{S_v}^2 S_v^2 + \sigma_{I_v}^2 I_v^2) \\ &\leq -U^2(b_1+b_2-k) + kU + (U^3 + \frac{\nu+1}{2}U^4)(\sigma_{S_h}^2 S_h^2 + \sigma_{I_h}^2 I_h^2 + \sigma_{S_v}^2 S_v^2 + \sigma_{I_v}^2 I_v^2) \\ &\leq -U^2(b_1+b_2-k-\frac{\nu+1}{2}\sigma^2) + U(k+\sigma^2).\end{aligned}$$

The last inequality follows from the fact that

$$U^3(\sigma_{S_h}^2 S_h^2 + \sigma_{I_h}^2 I_h^2 + \sigma_{S_v}^2 S_v^2 + \sigma_{I_v}^2 I_v^2) \leq U\sigma^2$$

and

$$U^4(\sigma_{S_h}^2 S_h^2 + \sigma_{I_h}^2 I_h^2 + \sigma_{S_v}^2 S_v^2 + \sigma_{I_v}^2 I_v^2) \leq U^2\sigma^2.$$

Also, let θ satisfy the assumption of the lemma. Then,

$$\begin{aligned}\mathcal{L}(e^{\theta t}(1+U)^\nu) &= \theta e^{\theta t}(1+U)^\nu + e^{\theta t}L(1+U)^\nu \\ &= e^{\theta t}(1+U)^{\nu-2}(\theta(1+U)^2 + \nu\psi).\end{aligned}$$

Now,

$$\theta(1+U)^2+\nu\psi \leq \theta U^2+2\theta U+\theta+\nu\left(-U^2(b_1+b_2-k-\frac{\nu+1}{2}\sigma^2)+U(k+\sigma^2)\right)$$
$$= U^2\left(\theta-\nu(b_1+b_2-k-\frac{\nu+1}{2})\right)+U(2\theta+\nu k+\nu\sigma^2)+\theta.$$

Thus,

$$\mathcal{L}(e^{\theta t}(1+U)^\nu) \leq e^{\theta t}(1+U)^{\nu-2}(-a_1U^2+a_2U+\theta),$$

where $a_1=\nu(b_1+b_2-k-\frac{\nu+1}{2})-\theta$ and $a_2=2\theta+\nu k+\nu\sigma^2$.

Note that $f(U)=-a_1U^2+a_2U+\theta$ has a maximum value of $f\left(\frac{a_2}{2a_1}\right)=\frac{a_2^2+4a_1\theta}{4a_1}$ and also

$$(1+U)^{\nu-2} \leq max\left\{1,\left(1+\frac{a_2+\sqrt{a_2^2+4a_1\theta}}{2a_1}\right)^{\nu-2}\right\}.$$

In conclusion, we have

$$\mathcal{L}(e^{\theta t}(1+U)^\nu) \leq M_1e^{\theta t},$$

where

$$M_1=\frac{a_2^2+4a_1\theta}{4a_1}\,max\left\{1,\left(1+\frac{a_2+\sqrt{a_2^2+4a_1\theta}}{2a_1}\right)^{\nu-2}\right\}.$$

Finally, using (3.9)

$$d(e^{\theta t}(1+U)^\nu)=\mathcal{L}(e^{\theta t}(1+U)^\nu)+\nu(1+U)^{\nu-1}e^{\theta t}(\sigma_{S_h}S_hdB_{S_h}(t)+\sigma_{I_h}I_hdB_{I_h}(t)$$
$$+\sigma_{S_v}S_vdB_{S_v}(t)+\sigma_{I_v}I_vdB_{I_v}(t)).$$

Integrating both sides and taking the expectation will result

$$E(e^{\theta t}(1+U)^\nu-(1+U(0))^\nu) \leq M_1E(\frac{e^{\theta t}}{\theta}-1).$$

Simplifying it further, we get

$$E((1+U)^{\nu}) \leq e^{-\theta t}(1+U(0))^{\nu} + M_1(\frac{1}{\theta} - e^{-\theta t}).$$

Letting $t \to \infty$, we conclude

$$\limsup_{t\to\infty} E(U)^{\nu} \leq \limsup_{t\to\infty} E(1+U)^{\nu} \leq \frac{M_1}{\theta}.$$

Now, for any $(S_h, I_h, S_v, I_v) \in \mathbb{R}^4_+$, we have

$$(S_h + I_h + S_v + I_v)^{\nu} \leq 4^{\nu}(S_h^2 + I_h^2 + S_v^2 + I_v^2)^{\frac{\nu}{2}} \leq 4^{\nu}\|X(t)\|^{\nu}.$$

Thus, it follows that

$$\limsup_{t\to\infty} E\left(\frac{1}{\|X(t)\|^{\nu}}\right) \leq 4^{\nu} \limsup_{t\to\infty} E(U)^{\nu} \leq M,$$

where $M = \frac{4^{\nu}M_1}{\theta}$. □

One other important property of stochastic epidemic models is the stochastic permanence, which indicates how the total population in the model changes in the long run. First, we give its definition and under some conditions we show that system (3.5)–(3.8) is stochastically permanent [62].

Definition 3.2.5. *System* (3.5)–(3.8) *is said to be stochastically permanent, if for any* $\epsilon \in (0,1)$, *there exist positive constants* $\lambda_1 = \lambda_1(\epsilon)$ *and* $\lambda_2 = \lambda_2(\epsilon)$ *such that for any initial value* $X(0) \in \Gamma$, *the solution* $X(t)$ *satisfies the following conditions:*

$$\liminf_{t\to\infty} \mathbb{P}\{\|X(t)\| \leq \lambda_1\} \geq 1-\epsilon, \qquad \liminf_{t\to\infty} \mathbb{P}\{\|X(t)\| \geq \lambda_2\} \geq 1-\epsilon.$$

Theorem 3.2.6. *Under the assumptions of lemma 3.2.4, system* (3.5)–(3.8) *is stochastically permanent for any initial value* $X(0) \in \Gamma$.

Proof. Now by theorem 3.2.3 for any $\epsilon \in (0,1)$, there exists $\lambda_1 > 0$ such that $\mathbb{P}\{\|X(t)\| > \lambda_1\} \leq \epsilon$, which is equivalent to $\mathbb{P}\{\|X(t)\| < \lambda_1\} > 1 - \epsilon$. Thus, it follows that

$$\liminf_{t\to\infty} \mathbb{P}\{\|X(t)\| \leq \lambda_1\} \geq 1 - \epsilon.$$

Also, assume that all the hypotheses in lemma 3.2.4 hold. Then, we have

$$\limsup_{t\to\infty} E\left(\frac{1}{\|X(t)\|^{\nu}}\right) \leq M.$$

For any $\epsilon \in (0,1)$, let $\lambda_2 = \frac{\epsilon^{\nu}}{M^{\nu}}$. Then,

$$\mathbb{P}\{\|X(t)\| < \lambda_2\} = \mathbb{P}\{\frac{1}{\|X(t)\|} > \frac{1}{\lambda_2}\} \leq \frac{E\left(\frac{1}{\|X(t)\|^{\nu}}\right)}{\lambda_2^{-\nu}} = \lambda_2^{\frac{1}{\nu}} E\left(\frac{1}{\|X(t)\|^{\nu}}\right).$$

Taking the limit, we get

$$\limsup_{t\to\infty} \mathbb{P}\{\|X(t)\| < \lambda_2\} \leq \lambda_2^{\frac{1}{\nu}} M = \epsilon.$$

Therefore, we conclude that

$$\liminf_{t\to\infty} \mathbb{P}\{\|X(t)\| \geq \lambda_2\} \geq 1 - \epsilon.$$

□

3.3 Extinction of infection and stochastic stability

In section 2.2 we defined the basic reproductive number $\mathcal{R}_0$ for the deterministic epidemic model. From theorem 2.3.2 and 2.3.4 it follows that the number of infected hosts I_h

and vectors I_v will tend to zero in the long run provided that $\mathcal{R}_0 < 1$. In this section we provide a similar condition for the extinction of infection for the stochastic model.

Theorem 3.3.1. *Let* $\mathcal{R}_0^s := \mathcal{R}_0 - \frac{\sigma_{I_h}^2}{2(\mu_1+\phi)}$. *If* $\mathcal{R}_0^s < 1$ *then for any initial value* $X(0) \in \Gamma$, $I_h(t)$ *will tend to zero exponentially almost surely. That is,* $\limsup_{t\to\infty} \frac{\ln I_h(t)}{t} < 0$ *a.s.*

Proof. From system (3.5)–(3.8), and using $S_h = \frac{b_1}{\mu_1} - I_h$ we get

$$dI_h = (\beta_1(\frac{b_1}{\mu_1} - I_h)I_h + \beta_2(\frac{b_1}{\mu_1} - I_h)I_v - (\mu_1 + \phi)I_h)dt + \sigma_{I_h} I_h dB_{I_h}(t).$$

Now, using (3.9) we have

$$\begin{aligned} d(\ln(I_h(t))) &= \left\{ \frac{1}{I_h} \left(\beta_1(\frac{b_1}{\mu_1} - I_h)I_h + \beta_2(\frac{b_1}{\mu_1} - I_h)I_v - (\mu_1 + \phi)I_h \right) + \frac{1}{2} \frac{-1}{I_h^2} \sigma_{I_h}^2 I_h^2 \right\} dt \\ &\quad + \frac{1}{I_h} \sigma_{I_h} I_h dB_{I_h}(t) \\ &= \left(\beta_1(\frac{b_1}{\mu_1} - I_h) + \beta_2(\frac{b_1}{\mu_1} - I_h)\frac{I_v}{I_h} - \mu_1 - \phi - \frac{1}{2}\sigma_{I_h}^2 \right) dt + \sigma_{I_h} dB_{I_h}(t) \\ &\leq \left(\frac{\beta_1 b_1}{\mu_2} + \frac{\beta_2 b_1 b_2}{\mu_2 \mu_1} - \mu_1 - \phi - \frac{1}{2}\sigma_{I_h}^2 \right) dt + \sigma_{I_h} dB_{I_h}(t). \end{aligned}$$

Integrating both sides on $[0, t]$, we get

$$\frac{ln(I_h(t)) - ln(I_h(0))}{t} \leq \left[\frac{\beta_1 b_1}{\mu_1} + \frac{\beta_2 b_1 b_2}{\mu_1 \mu_2} - \mu_1 - \phi - \frac{1}{2}\sigma_{I_h}^2 \right] + \frac{1}{t} \int_0^t \sigma_{I_h} dB_{I_h}(s).$$

Let $M(t) = \int_0^t \sigma_{I_h} dB_{I_h}(s)$. Then, M is a martingale [37], with a quadratic variation given by

$$\langle M, M \rangle_t = \int_0^t \sigma_{I_h}^2 ds = \sigma_{I_h}^2 t.$$

Since $\limsup_{t\to\infty} \frac{\langle M,M\rangle_t}{t} = \sigma_{I_h}^2 < \infty$, by the strong law of large numbers, it follows that $\limsup_{t\to\infty} \frac{M(t)}{t} = 0$.

Thus,

$$\limsup_{t\to\infty} \frac{lnI_h(t)}{t} \leq \frac{\beta_1 b_1}{\mu_1} + \frac{\beta_2 b_1 b_2}{\mu_1 \mu_2} - \mu_1 - \phi - \frac{1}{2}\sigma_{I_h}^2.$$

Now if $\mathcal{R}_s^0 < 1$ we have

$$\frac{\beta_1 b_1}{\mu_1} + \frac{\beta_2 b_1 b_2}{\mu_1 \mu_2} - \mu_1 - \phi - \frac{1}{2}\sigma_{I_h}^2 < 0.$$

Thus,

$$\limsup_{t\to\infty} \frac{ln I_h(t)}{t} \leq \frac{\beta_1 b_1}{\mu_1} + \frac{\beta_2 b_1 b_2}{\mu_1 \mu_2} - \mu_1 - \phi - \frac{1}{2}\sigma_{I_h}^2 < 0 \ a.s.$$

□

Using the above theorem and the following lemma, we conclude that the number of infected vectors I_v will also tend to zero exponentially almost surely.

Lemma 3.3.2. *Given a stochastic differential equation*

$$dX(t) = f(X(t), t)dt + g(X(t), t)dB(t).$$

Assume that there exists a function $V \in C^{2,1}(R^d \times [t_0, \infty); R_+)$, *and constants* $p > 0, c_1 > 0, c_2 \in R, c_3 \geq 0$, *such that for all* $X \neq 0$ *and* $t \geq t_0$,

1. $c_1\|X\|^p \leq V(X, t)$,
2. $\mathcal{L}V(X, t) \leq c_2 V(X, t)$,
3. $\|V_X(X, t)g(X, t)\|^2 \geq c_3 V^2(X, t)$.

Then,

$$\limsup_{t\to\infty} \frac{\ln \|X(t; t_0, X_0)\|}{t} \leq -\frac{c_3 - 2c_2}{2p} \ a.s.$$

Please refer to [37] for more detail.

Corollary 3.3.3. *If* $\mathcal{R}_0^s < 1$, *then for any initial values* $X(0) \in \Gamma$, $I_v(t)$ *will tend to zero exponentially almost surely. That is* $\limsup_{t\to\infty} \frac{\ln I_v(t)}{t} < 0$ *a.s.*

Proof. From theorem 3.3.1, if $\mathcal{R}_0^s < 1$, then $I_h(t)$ will tend to zero exponentially almost surely and since exponential stability implies asymptotic stability, we have that

$$\lim_{t\to\infty} I_h(t) = 0 \ a.s.$$

Thus, for any $\epsilon > 0$, there exists $k_1 > 0$ and a set $\bar{\Gamma} \subset \Gamma$ such that

$$\mathbb{P}(\bar{\Gamma}) > 1 - \epsilon, \ \ 0 \leq I_h(t) < \frac{\mu_2}{\beta b_2}\epsilon \ \text{ for all } \ t > k_1.$$

Now $(-\epsilon - \mu_2 I_v)\, dt + \sigma_{I_v} I_v dB_{I_v} \leq dI_v \leq (\epsilon - \mu_2 I_v)\, dt + \sigma_{I_v} I_v dB_{I_v}$, and since ϵ is arbitrary, we have

$$dI_v(t) = -\mu_2 I_v\, dt + \sigma_{I_v} I_v dB_{I_v}.$$

Let $V(I_v, t) = I_v^2$, then $\mathcal{L}V = (-2\mu_2 + \sigma_{I_v}^2)V$.

Also, we have $\|V_{I_v} g\|^2 = 4\sigma_{I_v}^2 \|I_v^4\|$, thus by lemma 3.3.2 it follows

$$\limsup_{t\to\infty} \frac{\ln I_v(t)}{t} \leq -\mu_2 - \frac{\sigma_{I_v}^2}{2} < 0 \ \text{ a.s.}$$

□

Remark 3.3.4. Note that in the deterministic case, if $\mathcal{R}_0 < 1$ then the system has a disease-free equilibrium point. Since $\mathcal{R}_0^s = \mathcal{R}_0 - \frac{\sigma_{I_h}^2}{2(\mu_1+\phi)}$, if $\mathcal{R}_0 < 1$ then $\mathcal{R}_0^s < 1$, and by theorem 3.3.1 and corollary 3.3.3, it follows that the number of infected hosts and vectors will go to zero exponentially almost surely. On the contrary, we might have cases where $\mathcal{R}_0^s < 1$, but $\mathcal{R}_0 > 1$. That is, a large environmental fluctuation can suppress the number of infected hosts (see figure 3.2 in section 3.5).

Definition 3.3.5. *Given the stochastic differential equation*

$$dX(t) = f(X(t), t)\, dt + g(X(t), t) dB(t) \ \ on \ \ t \geq t_0.$$

Assume that $f(0,t) = 0,\ g(0,t) = 0$ *for all* $t > 0$ *such that* $X(t) = 0$ *is a trivial solution. Then, the trivial solution is called*

1. *Stochastically stable if for all* $\epsilon \in (0,1)$ *and* $r > 0$ *there exist* $\delta = \delta(\epsilon, r, t_0) > 0$ *such that*

$$\mathbb{P}\{\|X(t; t_0, x_0)\| < r \text{ for all } t \geq t_0\} \geq 1 - \epsilon,$$

   for $X_0 \in \mathbb{R}^d$ *such that* $\|X_0\| < \delta$.

2. *Stochastically asymptotically stable if it is stochastically stable and for every* $\epsilon \in (0,1)$*, there exists a* $\delta_0 = \delta_0(\epsilon, t_0) > 0$ *such that,*

$$\mathbb{P}\{\lim_{t\to\infty} X(t; t_0, x_0) = 0\} \geq 1 - \epsilon$$

   whenever $\|X_0\| < \delta_0$.

Please refer to [37] for more information.

The standard method of studying stability is through a Lyapunov function. However, generally it is not easy to construct such a function for nonlinear system of stochastic differential equations. Therefore we use a different approach to study the stability of system (3.5)–(3.8). First, under some conditions we show that the corresponding linear system of (3.5)–(3.8) is asymptotically stable. Then we use theorem 7.1 [26], to claim the nonlinear system is also asymptotically stable.

Theorem 3.3.6. *If the linear system*

$$dX(t) = MX(t)\,dt + \sigma X(t)dB(t)$$

with constant coefficients is stochastically asymptotically stable, and the coefficients of the nonlinear system

$$dX(t) = b(X,t)\,dt + \sigma(X,t)dB(t)$$

satisfy an inequality

$$\|b(x,t) - M\,x\| + \|\sigma(x,t) - \sigma\,x\| < \gamma\|x\|,$$

in a sufficiently small neighborhood of the point $x = 0$, and with a sufficiently small constant γ , then the solution $X = 0$ of the nonlinear system is stochastically asymptotically stable.

Using $S_h = \frac{b_1}{\mu_1} - I_h$ and $S_v = \frac{b_2}{\mu_2} - I_v$, system (3.5)–(3.8) reduces to

$$dI_h = (\frac{\beta_1 b_1}{\mu_1} I_h - \beta_1 I_h^2 + \frac{\beta_2 b_1}{\mu_1} I_v - \beta_2 I_h I_v - (\mu_1 + \phi) I_h)dt + \sigma_{I_h} I_h dB_{I_h}(t), \quad (3.10)$$

$$dI_v = (\frac{\beta b_2}{\mu_2} I_h - \beta I_h I_v - \mu_2 I_v)dt + \sigma_{I_v} I_v dB_{I_v}(t). \quad (3.11)$$

The corresponding linearized system is given by

$$dI_h = \left(\left(\frac{\beta_1 b_1}{\mu_1} - \mu_1 - \phi\right) I_h + \frac{\beta_2 b_1}{\mu_1} I_v\right) dt + \sigma_{I_h} I_h dB_{I_h}(t), \quad (3.12)$$

$$dI_v = \left(\frac{\beta b_2}{\mu_2} I_h - \mu_2 I_v\right) dt + \sigma_{I_v} I_v dB_{I_v}(t). \quad (3.13)$$

Finally, we state the condition for the asymptotic stability of the system (3.10)–(3.11) as follows.

Theorem 3.3.7. *Let $X(t) = (I_h(t), I_v(t))$ be the solution of* (3.10)–(3.11)*. Suppose that*

$$\Theta = min\{\mu_1 + \phi - \frac{\beta_1 b_1}{\mu_1} - \frac{c\beta b_2}{\mu_2} - \frac{1}{2}\sigma_{I_h}^2,\ \mu_2 - \frac{1}{2}\sigma_{I_v}^2 - \frac{c\beta_2 b_1}{\mu_1}\} > 0$$

and

$$\mu_1 + \phi + \mu_2 > \frac{\beta_1 b_1}{\mu_1} + \sigma_{I_h}\sigma_{I_v},$$

where

$$c = \frac{\frac{\beta_2 b_1}{\mu_1} + \frac{\beta b_2}{\mu_2}}{\mu_2 + \phi + \mu_1 - \frac{\beta_1 b_1}{\mu_1} - \sigma_{I_h}\sigma_{I_v}} > 0.$$

Then for any initial value $X(0)$, the trivial solution $X(t) = 0$ of system (3.10)–(3.11) is stochastically asymptotically stable.

Proof. Define $V = \frac{1}{2}I_h^2 + \frac{1}{2}I_v^2 + cI_hI_v$. Then, using (3.9), we get

$$\begin{aligned}\mathcal{L}V &= \left(\frac{\beta_1 b_1}{\mu_1} - \mu_1 - \phi + \frac{1}{2}\sigma_{I_h}^2 + \frac{c\beta b_2}{\mu_2}\right) I_h^2 + \left(\frac{1}{2}\sigma_{I_v}^2 - \mu_2 + \frac{c\beta_2 b_1}{\mu_2}\right) I_v^2 \\ &\leq -\Theta\|X\|.\end{aligned}$$

Thus, the trivial solution $X(t) = 0$ of system (3.12)–(3.13) is stochastically asymptotically stable. Also let $\|X\| < \xi$, where $\xi > 0$ is sufficiently small,

$$\begin{aligned}&\|b(X,t) - MX\| + \|\sigma(X,t) - \sigma X\| = \|(-\beta_1 I_h^2 - \beta_2 I_h I_v,\ -\beta I_h I_v)\| \\ &\leq \beta_1 I_h^2 + \beta_2 I_h I_v + \beta I_h I_v \\ &\leq \beta_1 I_h^2 + \frac{(\beta + \beta_2)}{2}(I_h^2 + I_v^2) \\ &\leq (\beta_1 + \frac{\beta + \beta_1}{2})\|X\|^2 \ \leq \gamma\|X\|,\end{aligned}$$

where $\gamma = \xi(\beta_1 + \frac{\beta\beta_2}{2})$ and by theorem 3.3.6 the trivial solution of the nonlinear system (3.10)–(3.11) is stochastically asymptotically stable. □

3.4 Persistence in mean

One of the most fundamental questions in epidemiology and population biology is to know the necessary conditions to ensure the long-term persistence of a population or a collection of interacting populations. In the analysis of stochastic epidemic models, persistence in mean, which captures the idea of non-extinction of the system is frequently used.

Definition 3.4.1. *[19] The system given by* (3.10)–(3.11) *is called persistence in mean if*

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t I_h(s)\,ds > 0 \ \text{ and } \ \liminf_{t\to\infty} \frac{1}{t}\int_0^t I_v(s)\,ds > 0.$$

Note that if $\mathcal{R}_0^s > 1$, then $\mathcal{R}_0 > 1$ and by theorem 2.3.19, the deterministic model (2.1)–(2.4) has endemic equilibrium $E_1 = (S_h^*, I_h^*, S_v^*, I_v^*)$. The following theorem will provide conditions for system (3.10)–(3.11) to be persistence in mean.

Theorem 3.4.2. *Let $\mathcal{R}_0^s > 1$ and assume that*

$$\frac{\sigma_{I_h}^2}{2} < l_1 \ \text{ and } \ \frac{\sigma_{I_v}^2}{2} < l_2,$$

where $l_1 = \frac{\beta_1 b_1}{\mu_1} + \frac{\beta_2 b_1 I_v^*}{\mu_1 I_h^*}$ and $l_2 = \frac{\beta b 2 I_h^*}{\mu_2 I_v^*}$. Then for any initial value $X(0)$, the solution of system (3.10)–(3.11) *satisfies*

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t [(I_h(s) - (1+\frac{\sigma_{I_h}^2}{2l_1})I_h^*)^2 + (I_v(s) - (1+\frac{\sigma_{I_v}^2}{2l_2})I_v^*)^2]ds \le \frac{k_2}{k_1},$$

where $k_1 = min\{l_1, l_2\}$ and $k_2 = \frac{1}{2}\left(1+\frac{\sigma_{I_h}^2}{2l_1}\right)\sigma_{I_h}^2 {I_h}^{*2} + \frac{1}{2}\left(1+\frac{\sigma_{I_v}^2}{2l_2}\right)\sigma_{I_v}^2 {I_v}^{*2}$.

Proof. Let $V_1 = \frac{1}{2}(I_h - I_h^*)^2$ and $V_2 = \frac{1}{2}(I_v - I_v^*)^2$. Then,

$$\mathcal{L}V_1 = (I_h - I_h^*)\left(\frac{\beta_1 b_1}{\mu_1} I_h - \beta_1 I_h^2 + \frac{\beta_2 b_1}{\mu_1} I_v - \beta_2 I_h I_v - (\mu_1 + \phi) I_h\right) + \frac{1}{2}\sigma_{I_h}^2 I_H^2.$$

Using (2.7) and (2.8) we get

$$\begin{aligned}
\mathcal{L}V_1 &\le (I_h - I_h^*)\left(\frac{\beta_1 b_1}{\mu_1}(I_h^* - I_h) + \frac{\beta_2 b_1 I_v^*}{\mu_1} {I_h}^*(I_h^* - I_h)\right) + \frac{1}{2}\sigma_{I_h}^2 I_h^2 \\
&\le -\left(\frac{\beta_1 b_1}{\mu_1} + \frac{\beta_2 b_1 I_v^*}{\mu_1 I_h^*}\right)(I_h - I_h^*)^2 + \frac{1}{2}\sigma_{I_h}^2 I_h^2 \\
&= -l_1\left(I_h - (1+\frac{\sigma_{I_h}^2}{2l_1})I_h^*\right)^2 + \frac{1}{2}(1+\frac{\sigma_{I_h}^2}{2l_1})\sigma_{I_h}^2 {I_H^2}^*.
\end{aligned}$$

Similarly, we have $\mathcal{L}V_2 \leq -l_2\left(I_v - (1+\frac{\sigma_{I_v}^2}{2l_2})I_v^*\right)^2 + \frac{1}{2}(1+\frac{\sigma_{I_v}^2}{2l_2})\sigma_{I_v}^2 {I^2}_V{}^*$.

Now letting $V = V_1 + V_2$, we have

$$\begin{aligned}\mathcal{L}V = \mathcal{L}V_1 + \mathcal{L}V_2 &\leq -l_1\left(I_h - (1+\frac{\sigma_{I_h}^2}{2l_1})I_h^*\right)^2 - l_2\left(I_v - (1+\frac{\sigma_{I_v}^2}{2l_2})I_v^*\right)^2 + k_2 \\ &\leq -k_1\left[\left(I_h - (1+\frac{\sigma_{I_h}^2}{2l_1})I_h^*\right)^2 + \left(I_v - (1+\frac{\sigma_{I_v}^2}{2l_2})I_v^*\right)^2\right] + k_2.\end{aligned}$$

Using equation (3.9),

$$dV = \mathcal{L}V\,dt + \sigma_{I_h} I_h dB_{I_h} + \sigma_{I_v} I_v dB_{I_v}.$$

Integrating both sides, we get

$$\begin{aligned}&k_1\int_0^t \left(\left(I_h(s) - (1+\frac{\sigma_{I_h}^2}{2l_1})I_h^*\right)^2 + \left(I_v(s) - (1+\frac{\sigma_{I_v}^2}{2l_2})I_v^*\right)^2\right) ds \\ &\leq k_2 t - V(I_h(t), I_v(t)) + V(I_h(0), I_v(0)).\end{aligned}$$

Thus, taking the limit we get

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t [(I_h(s) - (1+\frac{\sigma_{I_h}^2}{2l_1})I_h^*)^2 + (I_v(s) - (1+\frac{\sigma_{I_v}^2}{2l_2})I_v^*)^2]ds \leq \frac{k_2}{k_1}.$$

□

Corollary 3.4.3. *Assume that the conditions on Theorem 3.4.2 hold, and suppose that*

$$(1+\frac{\sigma_{I_h}^2}{2l_1})I_h^* > \sqrt{\frac{k_2}{k_1}} \quad \textit{and} \quad (1+\frac{\sigma_{I_v}^2}{2l_2})I_v^* > \sqrt{\frac{k_2}{k_1}}.$$

Then, system (3.5)–(3.8) *is persistent in the mean and*

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t I_h(s)\,ds \geq \frac{1}{2}(1+\frac{\sigma_{I_h}^2}{2l_1})I_h^* - \frac{l_1}{I_h^*(2l_1+\sigma_{I_h}^2)}\frac{k_2}{k_1} > 0, \tag{3.14}$$

and

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t I_v(s)\,ds \geq \frac{1}{2}(1+\frac{\sigma_{I_v}^2}{2l_2})I_v^* - \frac{l_2}{I_v^*(2l_2+\sigma_{I_v}^2)}\frac{k_2}{k_1} > 0. \tag{3.15}$$

Proof. We will prove inequality (3.14) and the other inequality can be proved using a similar procedure. From theorem 3.4.2, we have

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t \left(I_h(s) - (1+\frac{\sigma_{I_h}^2}{2l_1})I_h^*\right)^2 ds \leq \frac{k_2}{k_1}$$

and

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t \left(I_v(s) - (1+\frac{\sigma_{I_v}^2}{2l_2})I_v^*\right)^2 ds \leq \frac{k_2}{k_1}.$$

For any constant $\xi > 0$, we have

$$I_h \geq \frac{1}{2}I_h^*\xi - \frac{1}{2I_h^*\xi}(I_h - I_h^*\xi)^2.$$

Thus,

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t I_h(s)\,ds \geq \frac{1}{2}I_h^*\xi - \frac{1}{2I_h^*\xi}\limsup_{t\to\infty}\frac{1}{t}\int_0^t (I_h - I_h^*\xi)^2\,ds.$$

Let $\xi = 1 + \frac{\sigma_{I_h}^2}{2l_1}$. Then

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t I_h(s)\,ds \geq \frac{1}{2}I_h^*(1+\frac{\sigma_{I_h}^2}{2l_1}) - \frac{l_1}{I_h^*(2l_1+\sigma_{I_h}^2)}\frac{k_2}{k_1} > 0.$$

□

3.5 Numerical simulations

In this section we conduct numerical simulations to demonstrate some of the theoretical results discussed in this chapter. The discretization scheme for the stochastic system and MATLAB code used for the simulation purpose can be found in the appendix 1.

First consider the following values of parameters

$$\begin{aligned} \beta_1 &= 0.005,\ \beta_2 = 0.003,\ \mu_2 = 0.02,\ \beta = 0.0011,\ \phi = 0.35,\ \mu_1 = 0.83, \\ b_1 &= 60,\ b_2 = 0.8,\ \sigma_{S_h} = 0.1,\ \sigma_{I_h} = 0.1,\ \sigma_{S_v} = 0.1,\ \sigma_{I_v} = 0.1 \end{aligned}$$

Then $\mathcal{R}_0^s = 0.6428$, $\mathcal{R}_0 = 0.7106$. Thus by theorem 2.3.2 and 2.3.4 the solution of the deterministic model will converge to a disease-free equilibrium point. Similarly by theorem 3.3.1 and 3.3.3 it follows that the number of infected hosts and vectors will approach to zero exponentially. Thus extinction of infection in the deterministic model implies extinction in the stochastic case as can be see in figure 3.1.

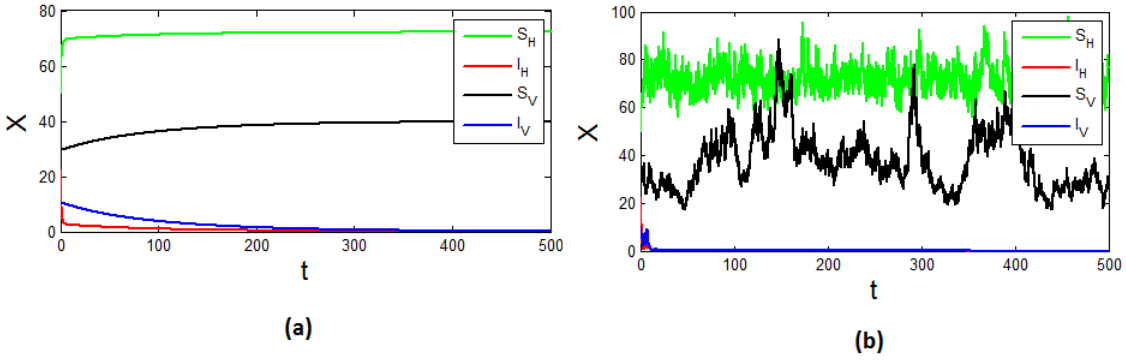

Figure 3.1: Extinction of infection in the deterministic model implies extinction in the stochastic case,$\mathcal{R}_0 = 0.71$ and $\mathcal{R}_0^s = 0.64$ respectivelly.

Now consider the following set of parameters;

$$\begin{aligned} \beta_1 &= 0.09,\ \beta_2 = 0.3,\ \mu_2 = 0.3,\ \beta = 0.14,\ \phi = 0.35,\ \mu_1 = 0.83, \\ b_1 &= 2,\ b_2 = 1,\ \sigma_{S_h} = 0.1,\ \sigma_{I_h} = 0.88,\ \sigma_{S_v} = 0.1,\ \sigma_{I_v} = 0.1 \end{aligned}$$

The we get $\mathcal{R}_0 = 1.1368 > 1$ while $\mathcal{R}_0^s = 0.8086 < 1$. Thus according to theorem 2.3.2 and 2.3.4 the deterministic model has an endemic equilibrium and the infection will persist. On

the contrary, by theorem 3.3.1 and 3.3.3 we conclude that the infection will be extinct from the stochastic model as shown in figure 3.2.

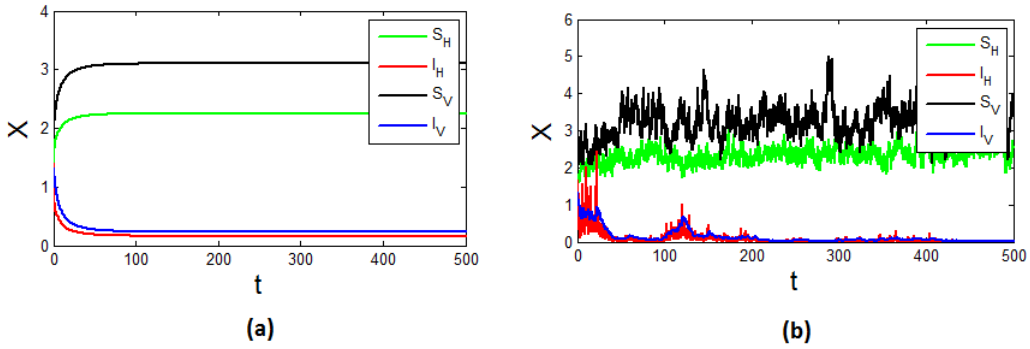

Figure 3.2: Existence of Endemic equilibrium in the deterministic model ($\mathcal{R}_0 = 1.1368$), while extinction in the stochastic case ($\mathcal{R}_0^s = 0.8086$).

Next consider the following parameter values.

$$\begin{aligned} \beta_1 &= 0.01,\ \beta_2 = 0.3,\ \mu_2 = 0.3,\ \beta = 0.06,\ \phi = 0.35,\ \mu_1 = 0.83, \\ b_1 &= 5,\ b_2 = 4,\ \sigma_{S_h} = 0.1,\ \sigma_{I_h} = 0.1,\ \sigma_{S_v} = 0.1,\ \sigma_{I_v} = 0.1 \end{aligned}$$

The values of $\mathcal{R}_0$ and $\mathcal{R}_0^s$ are 4.1352 and 4.1309 respectively. Thus the infection will persist on both the deterministic as well as stochastic cases. The endemic equilibrium is given by $E_1 = (2.47, 3.55, 7.79, 5.54)$. Thus, by theorem 3.4.2, the solution of system (3.5)–(3.8) oscillates about E_1. Now, if we fix all the values of the parameters but increase the noise intensities to $\sigma_{S_h} = 0.5,\ \sigma_{I_h} = 0.5,\ \sigma_{S_v} = 0.5,\ \sigma_{I_v} = 0.5$ then, the new solution will still oscillate about the same endemic equilibrium E_1 with a larger amplitude as can be seen in figure 3.3.

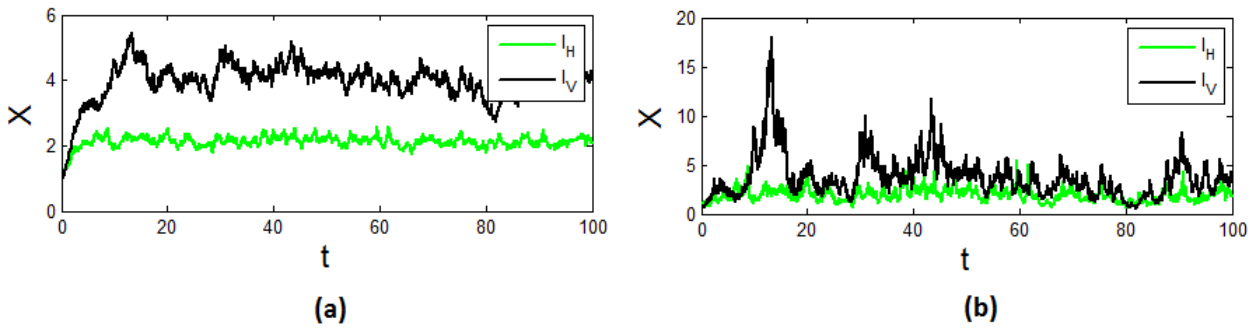

Figure 3.3: The effect of noise intensity on the trajectories of $I_h(t)$ and $I_v(t)$. In both cases the solution oscillates about the endemic equilibrium E_1 with different amplitudes. In (a) the noise intensity is $\sigma_{S_h} = \sigma_{I_h} = \sigma_{S_v} = \sigma_{I_v} = 0.1$, and in (b) $\sigma_{S_h} = \sigma_{I_h} = \sigma_{S_v} = \sigma_{I_v} = 0.5$.

In order to simulate the long-term persistence of the disease, we consider the same set of parameters as above. Then $(1 + \frac{\sigma_{I_h}^2}{2l_1})I_h^* = 3.56$, $(1 + \frac{\sigma_{I_v}^2}{2l_2})I_v^* = 5.59$ and $\sqrt{\frac{k_2}{k_1}} = 0.65$. With this values of parameters, we can easily verify that all the assumptions on theorem 3.4.2 and corollary 3.4.3 are satisfied. Thus we conclude that system (3.5)–(3.8) is persistence in mean with $\liminf_{t\to\infty} \frac{1}{t}\int_0^t I_h(s)\,ds = 1.72 > 0$ and $\liminf_{t\to\infty} \frac{1}{t}\int_0^t I_v(s)\,ds = 2.76 > 0$. This is verified in figure 3.4.

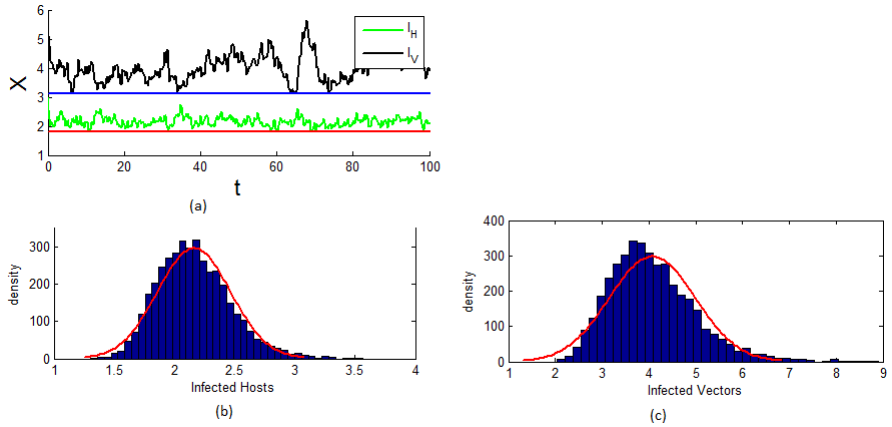

Figure 3.4: Persistence in mean of the stochastic epidemic model and histogram of I_h and I_v.

Chapter 4

Regime Switching Vector-host Epidemic Model with Direct Transmission

4.1 Description of the model

As mentioned in the introduction, in this section we will include another type of environmental noise. The noise considered in system (3.5)–(3.8) is the multiplicative white noise, which has been widely used in applications from engineering and physics. In this chapter we introduce a different type of noise, the Markovian noise, into the vector-host model, based on the assumption that the switching between different environments is memory-less and the waiting time for switching is exponentially distributed. The underlying model relies on a continuous time Markov chain $r(t), t \geq 0$ with a finite state space $\mathcal{M} = \{1, 2, \ldots, m\}$, generated by the transition matrix $Q = (q_{ij})_{m\times m}$, i.e.,

$$\mathbb{P}\{r(t+\Delta t) = j | r(t) = i\} = \begin{cases} q_{ij}\Delta t + o(\Delta t) & \text{if } i \neq j \\ 1 + q_{ii}\Delta t + o(\Delta t) & \text{if } i = j. \end{cases}$$

where $q_{ij} \geq 0$ is the transition rate from i to j if $i \neq j$ and $q_{ii} = -\sum_{i\neq j} q_{ij}$. Thus we model the stochastic vector-host epidemic in random environment using the following system of stochastic differential equation under regime switching.

$$\begin{aligned} dI_h &= \left(\frac{\beta_1^{r(t)} b_1}{\mu_1} I_h - \beta_1^{r(t)} I_h^2 + \frac{\beta_2^{r(t)} b_1}{\mu_1} I_v - \beta_2^{r(t)} I_h I_v - (\mu_1 + \phi) I_h \right) dt \\ &+ \sigma_{I_h}^{r(t)} I_h dB_{I_h}(t), \end{aligned} \tag{4.1}$$

$$dI_v = \left(\frac{\beta^{r(t)} b_2}{\mu_2} I_h - \beta^{r(t)} I_h I_v - \mu_2 I_v \right) dt + \sigma_{I_v}^{r(t)} I_v dB_{I_v}(t). \tag{4.2}$$

The above system (4.1)-(4.2) can be explained as follows. Suppose that the epidemic is initially in environment $i \in \mathcal{M}$, that is $r(0) = i$. Then the Markov chain $r(t)$ rests in the state i for an exponentially distributed random time and the system will have the following form:

$$dI_h = (\tfrac{\beta_1^i b_1}{\mu_1} I_h - \beta_1^i I_h^2 + \tfrac{\beta_2^i b_1}{\mu_1} I_v - \beta_2^i I_h I_v - (\mu_1 + \phi) I_h)dt + \sigma_{I_h}^i I_h dB_{I_h}(t),$$

$$dI_v = (\tfrac{\beta^i b_2}{\mu_2} I_h - \beta^i I_h I_v - \mu_2 I_v)dt + \sigma_{I_v}^i I_v dB_{I_v}(t),$$

where β^i, β_1^i and β_2^i are the transmission rates, while $\sigma_{I_h}^i, \sigma_{I_v}^i$ are the noise intensities in state i. Then the environment will switch and the Markov chain $r(t)$ will jump to another state j. Here the new system will have the form:

$$dI_h = (\tfrac{\beta_1^j b_1}{\mu_1} I_h - \beta_1^j I_h^2 + \tfrac{\beta_2^j b_1}{\mu_1} I_v - \beta_2^j I_h I_v - (\mu_1 + \phi) I_h)dt + \sigma_{I_h}^j I_h dB_{I_h}(t),$$

$$dI_v = (\tfrac{\beta^j b_2}{\mu_2} I_h - \beta^j I_h I_v - \mu_2 I_v)dt + \sigma_{I_v}^j I_v dB_{I_v}(t),$$

where β^j, β_1^j and β_2^j are the transmission rates, while $\sigma_{I_h}^j, \sigma_{I_v}^j$ are the noise intensities in state j.

4.1.1 Notations and remark.

Let $(x(t), r(t))$ be the diffusion process described by the stochastic differential equation with Markovian switching of the form

$$dx(t) = f(x(t), t, r(t))dt + g(x(t), t, r(t))dB(t), \tag{4.3}$$

where $f : \mathbb{R}^n \times \mathbb{R}_+ \times \mathcal{M} \to \mathbb{R}^n$, $\quad g : \mathbb{R}^n \times \mathbb{R}_+ \times \mathcal{M} \to \mathbb{R}^{n \times m}$ and $B(t) = (B_1(t), B_2(t), \ldots, B_m(t))$ is an m-dimensional Brownian motion such that $B_i(t)$ for $i = 1, 2, \ldots, m$ are independent. Denote by $E := \mathcal{C}^{2,1}(\mathbb{R}^n \times \mathbb{R}_+ \times \mathcal{M}; \mathbb{R}_+)$ the family of all nonnegative functions $V(x, t, i)$ which are continuously twice differentiable in x and differentiable in t.

For any $V \in E$, define the operator $\mathcal{L}V : \mathbb{R}^n \times \mathbb{R}_+ \times \mathcal{M} \to \mathbb{R}$ by

$$\mathcal{L}V(x,t,i) = V_t(x,t,i) + V_x(x,t,i)f(x,t,i) + \frac{1}{2}trace[g^T(x,t,i)V_{xx}(x,t,i)g(x,t,i)] + \sum_{j=1}^{m} q_{ij}V(x,t,i). \tag{4.4}$$

Let $(\Omega, \mathcal{F}, \mathbb{P})$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ satisfying the usual conditions, that is, it is right continuous and increasing with $\mathcal{F}_0$ containing all $\mathbb{P}$-null sets. Throughout the rest of this chapter we assume that both the Brownian motion $B(t)$ and the Markov chain $r(t)$ for $t \geq 0$ are independent and are defined on the complete probability space.

Also let $\mathcal{K}$ denote the family of all continuous increasing functions $k : \mathbb{R}_+ \to \mathbb{R}_+$ such that $k(0) = 0$ while $k(u) > 0$ for $u > 0$, and $\mathcal{K}_\infty$ contains the family of all functions $k \in \mathcal{K}$ such that $k(\infty) = \infty$ [38].

Remark 4.1.1. Since we assumed that the system can switch from one state to any other, the Markov chain $r(t), t \geq 0$ is irreducible, thus it has a unique stationary distribution $\pi = (\pi_i)_{i=1}^m \in \mathbb{R}^{1\times m}$ obtained by solving

$$\pi Q = 0, \ \sum_{k=1}^{m} \pi_k = 1.$$

4.2 Existence and uniqueness of the solution

In this section, we show that system (4.1)–(4.2) has a unique nonnegative global solution and also discuss some properties of the solution. For simplicity of notations, denote the solution to (4.1)–(4.2) by $X(t) = (I_h(t), I_v(t))$, and let $\mathbb{R}^2_+ := \{(x,y) : x > 0, y > 0\}$.

Theorem 4.1. For any initial value $X(0) \in \mathbb{R}^2_+$ and $r(0) \in \mathcal{M}$, system (4.1)–(4.2) has a unique global solution on $t \geq 0$ and the solution will remain in $\mathbb{R}^2_+$ with probability 1.

Proof. The idea of the proof is similar to what is used in [34, 60]. For each $i \in \mathcal{M}$, the vector field associated with system (4.1)–(4.2) is local Lipschitz. Thus a unique local positive solution exists on $[0, \tau_e)$, where $\tau_e < \infty$ is the explosion time. Now we show that $\tau_e = \infty$ a.s., thus the existence of a unique positive global solution is guaranteed.

Let $k_0 > 0$ be large enough such that $X(0) \in \left(\frac{1}{k_0}, k_0\right)^2$.

$$\tau_k = inf\left\{t \in [0, \tau_e] : X(t) \notin \left(\frac{1}{k}, k\right)^2\right\}, \quad k > k_0.$$

Thus $\{\tau_k\}_k$ is an increasing sequence. Let $\tau = \lim_{k\to\infty} \tau_k$, then $\tau \leq \tau_e$ a.s. By contradiction, we show $\tau = \infty$ a.s. and we conclude system (4.1)–(4.2) has a unique positive global solution.

Suppose $\tau < \infty$, then there exists $K > 0$ such that $\mathbb{P}(\tau \leq K) > \epsilon$ for all $\epsilon \in (0, 1)$. This implies that there exists $k_1 > k_0$ such that $\mathbb{P}(\tau_k \leq K) \geq \epsilon$ for all $k \geq k_1$.

For $c > 0$ and $(X(t), i) \in \mathbb{R}^2_+ \times \mathcal{M}$, define

$$V(X(t), i) = (I_h - 1 - \ln I_h) + c(I_v - 1 - \ln I_v).$$

Then

$$\begin{aligned}
\mathcal{L}V &= -\beta_1 I_h^2 + \left(\frac{\beta_1 b_1}{\mu_1} + \frac{a\beta b_2}{\mu_2} - (\mu_1 + \gamma) + \beta_1 + a\beta_1\right) I_h - (\beta_2 + a\beta) I_h I_v \\
&+ \left(\frac{\beta_2 b_1}{\mu_1} - a\mu_2 + \beta_2\right) I_v - \left(\frac{\beta_2 b_1}{\mu_1}\frac{I_v}{I_h} + \frac{a\beta b_2}{\mu_2}\frac{I_h}{I_v}\right) + A, \\
&\leq -\beta_1 I_h^2 + \left(\frac{\beta_1 b_1}{\mu_1} + \frac{a\beta b_2}{\mu_2} - (\mu_1 + \gamma) + \beta_1 + a\beta_1\right) I_h + \left(\frac{\beta_2 b_1}{\mu_1} - a\mu_2 + \beta_2\right) I_v + A
\end{aligned}$$

where

$$A = \mu_1 + \gamma + a\mu_2 + \frac{1}{2}\sigma_1^2 + \frac{a}{2}\sigma_2^2.$$

Pick a such that $a > \frac{\beta_2}{\mu_2} + \frac{\beta_2 b_1}{\mu_1 \mu_2}$, then there exists a constant M such that

$$\mathcal{L}V \leq -\beta_1 I_h^2 + \left(\frac{\beta_1 b_1}{\mu_1} + \frac{a\beta b_2}{\mu_2} - (\mu_1 + \gamma) + \beta_1 + a\beta_1\right) I_h + A \leq M.$$

Using equation (3.9) it follows that

$$dV = \mathcal{L}V dt + \sigma_{I_h}(I_h - 1)dB_{I_h} + \sigma_{I_v}(I_v - 1)dB_{I_v}.$$

Integrating both sides of the above inequality on $(0, \tau_k \wedge K)$ and taking the expectation, we have

$$EV(X(\tau_k \wedge K)) \leq V(X(0)) + MK.$$

For $k \geq k_0$ let $A_k = \{\tau_k \leq K\}$, then $P(A_k) \geq \epsilon$. If $t \in A_k$, then either

$$I_h(t) \notin \left(\frac{1}{k}, k\right) \text{ or } I_v(t) \notin \left(\frac{1}{k}, k\right).$$

Thus for $t \in A_k$

$$V(X(\tau_k \wedge t)) \geq (k - 1 - \frac{1}{k}) \wedge (\frac{1}{k} - 1 - \ln(\frac{1}{k})).$$

Now we have

$$V(X(0)) + MK \geq EV(X(\tau_k \wedge K)) \geq \epsilon \left((k - 1 - \ln k) \wedge (\frac{1}{k} - 1 - \ln \frac{1}{k})\right).$$

Finally, letting $k \to \infty$ we have

$$\infty > V(X(0)) + MK = \infty$$

which is a contradiction. Thus we conclude $\tau = \infty$ a.s. □

4.3 Exponential stability and pth moment stability

4.3.1 Almost sure exponential and pth moment exponential stability

The following theorem gives conditions for the extinction of infection in the regime switching model.

Theorem 4.2. For any initial value $X(0) \in \mathbb{R}^2_+$ and $r(0) \in \mathcal{M}$, the solution of system (4.1)–(4.2), satisfies

$$\limsup_{t\to\infty} \frac{1}{t}(\ln I_h(t) + \ln I_v(t)) \leq \sum_{i=1}^{m} \pi_i F(i)$$

where

$$F(i) = \frac{\beta_1^i b_1}{\mu_1} + (\beta_2^i + \beta^i)\frac{b_1 b_2}{\mu_1 \mu_2} - \mu_1 - \phi - \frac{1}{2}(\sigma_{I_h}^i)^2 - \frac{1}{2}(\sigma_{I_v}^i)^2. \tag{4.5}$$

Proof. For any $i \in \mathcal{M}$, define $V(X(t), i) = \ln I_h(t) + \ln I_v(t)$. Then,

$$\begin{aligned}
\mathcal{L}V &= \frac{1}{I_h}\left(\frac{\beta_1^r b_1}{\mu_1} I_h - \beta_1^r I_h^2 + \frac{\beta_2^r b_1}{\mu_1} I_v - \beta_2^r I_h I_v - (\mu_1 + \phi) I_h\right) - \frac{1}{2}(\sigma_{I_h}^r)^2 \\
&+ \frac{1}{I_v}(\frac{\beta^r b_2}{\mu_2} I_h - \beta^r I_h I_v - \mu_2 I_v) - \frac{1}{2}(\sigma_{I_v}^r)^2 \\
&\leq \frac{\beta_1^r b_1}{\mu_1} + (\beta_2^r + \beta^r)\frac{b_1 b_2}{\mu_1 \mu_2} - \mu_1 - \phi - \frac{1}{2}(\sigma_{I_h}^r)^2 - \frac{1}{2}(\sigma_{I_v}^r)^2.
\end{aligned}$$

Using equation (3.9), we have

$$dV \leq \frac{\beta_1^r b_1}{\mu_1} + (\beta_2^r + \beta^r)\frac{b_1 b_2}{\mu_1 \mu_2} - \mu_1 - \phi - \frac{1}{2}(\sigma_{I_h}^r)^2 - \frac{1}{2}(\sigma_{I_v}^r)^2 + \sigma_{I_h}^r dB_{I_h}(t) + \sigma_{I_v}^r dB_{I_v}(t)$$

Integrating both sides on $[0, t]$, we get

$$V(t) \leq V(0) + \int_0^t F(r(s)) + \int_0^t \sigma_{I_h}^{r(s)} dB_{I_h}(s) + \sigma_{I_v}^{r(s)} dB_{I_v}(s), \tag{4.6}$$

where $F(r(t)) = \frac{\beta_1^r b_1}{\mu_1} + (\beta_2^r + \beta^r)\frac{b_1 b_2}{\mu_1 \mu_2} - \mu_1 - \phi - \frac{1}{2}(\sigma_{I_h}^r)^2 - \frac{1}{2}(\sigma_{I_v}^r)^2$.

Let $M(t) = \int_0^t \sigma_{I_h}^{r(s)} dB_{I_h}(s) + \sigma_{I_v}^{r(s)} dB_{I_v}(s)$, then $M(t)$ is a martingale with $M(0) = 0$ and a quadratic variation given by

$$\langle M, M\rangle_t = \int_0^t (\sigma_{I_h}^{r(s)})^2 + (\sigma_{I_v}^{r(s)})^2 ds \leq 2\sigma^2 t,$$

where

$$\sigma^2 = \max\{(\sigma_{I_h}^i)^2, (\sigma_{I_h}^i)^2\}, \quad 1 \leq i \leq m.$$

Since

$$\limsup_{t\to\infty} \frac{\langle M, M\rangle_t}{t} \leq 2\sigma^2 < \infty,$$

by the strong law of large numbers, it follows that $\limsup_{t\to\infty} \frac{M(t)}{t} = 0$. Thus we have,

$$\limsup_{t\to\infty} \frac{V(t)}{t} \leq \limsup_{t\to\infty} \frac{1}{t} \int_0^t F(r(s))ds.$$

Finally by the ergodic property of Markov chain [38],

$$\limsup_{t\to\infty} \frac{1}{t} \int_0^t F(r(s))ds = \sum_{i=1}^{m} \pi_i F(i).$$

Thus it follows that

$$\limsup \frac{1}{t}(\ln I_h(t) + \ln I_v(t)) = \limsup_{t\to\infty} \frac{V(t)}{t} \leq \sum_{i=1}^{m} \pi_i F(i).$$

□

Corollary 4.3.1. *If*

$$\sum_{i=1}^{m} \pi_i F(i) < 0,$$

then the disease-free equilibrium point $E_0 = (\frac{b_1}{\mu_1}, 0, \frac{b_2}{\mu_2}, 0)$ *is exponentially stable almost surely.*

Proof. From theorem 4.2 we have the following results.

$$\limsup_{t\to\infty} \frac{\ln I_h(t)}{t} \leq \limsup \frac{1}{t}(\ln I_h(t) + \ln I_v(t)) < 0$$

and

$$\limsup_{t\to\infty} \frac{\ln I_v(t)}{t} \leq \limsup \frac{1}{t}(\ln I_h(t) + \ln I_v(t)) < 0.$$

Thus the disease-free equilibrium point $E_0 = (\frac{b_1}{\mu_1}, 0, \frac{b_2}{\mu_2}, 0)$ is exponentially stable almost surely. □

Theorem 4.3. For any $p > 0$ and any initial value $X(0) \in \mathbb{R}^2_+$ and $r(0) \in \mathcal{M}$, the solution of system (4.1)–(4.2), satisfies

$$\limsup_{t\to\infty} \frac{1}{t}\mathbb{E}(\ln I_h(t)^p + \ln I_v(t)^p) \leq \sum_{i=1}^{m} \pi_i(pF(i) + p^2\sigma_i^2),$$

where $F(i)$ is given by (4.5) and $\sigma_i^2 = \max\{(\sigma_{I_h}^i)^2, (\sigma_{I_v}^i)^2\}$.

Proof. From (4.6) for any $p > 0$, we have

$$pV(t) \leq pV(0) + p\int_0^t F(r(s)) + p\int_0^t \sigma_{I_h}^{r(s)} dB_{I_h}(s) + \sigma_{I_v}^{r(s)} dB_{I_v}(s).$$

By the ergodic property of Markov chain it follows that for any $\epsilon > 0$

$$\int_0^t F(r(s))ds \leq (\sum_{i=1}^{m} \pi_i\, F(i) + \frac{\epsilon}{p})t.$$

Thus

$$\mathbb{E}(I_h(t)^p I_v(t)^p) \leq I_h(0)^p I_v(0)^p\, e^{p(\sum_{i=1}^m \pi_i\, F(i) + \frac{\epsilon}{p})t}\, \mathbb{E}(e^{pM_t}),$$

where $M(t) = \int_0^t \sigma_{I_h}^{r(s)} dB_{I_h}(s) + \sigma_{I_v}^{r(s)} dB_{I_v}(s)$ is a real - valued martingale with $M(0) = 0$. The quadratic variation of $pM(t)$ is given by

$$\langle pM, pM\rangle_t = p^2 \int_0^t (\sigma_{I_h}^{r(s)})^2 ds + (\sigma_{I_v}^{r(s)})^2 ds \leq 2p^2 \int_0^t \sigma_{r(s)}^2 ds.$$

Again by the ergodic property of Markov chain for any $\tilde{\epsilon} > 0$,

$$\int_0^t \sigma_{r(s)}^2 ds \leq (\sum_{i=1}^{m} \pi_i\sigma_i^2 + \frac{\tilde{\epsilon}}{p^2})t,$$

thus

$$\langle pM, pM\rangle_t \leq (2p^2 \sum_{i=1}^{m} \pi_i\sigma_i^2 + 2\tilde{\epsilon})t < \infty.$$

Hence by Girsanov's theorem it follows,

$$\mathbb{E}(e^{pM_t}) = \mathbb{E}(e^{\frac{1}{2}\langle pM_t, pM_t\rangle}) \leq e^{(p^2\sum_{i=1}^m \pi_i\sigma_i^2+\tilde{\epsilon})t}.$$

Now

$$\mathbb{E}(I_h(t)^p\, I_v(t)^p) \leq I_h(0)^p I_v(0)^p\, e^{(p\sum_{i=1}^m \pi_i\, F(i)+\epsilon)t+(p^2\sum_{i=1}^m \pi_i\sigma_i^2+\tilde{\epsilon})t},$$

and thus,

$$\ln I_h(t)^p + \ln I_v(t)^p \leq \ln I_h(0) + \ln I_v(0) + p(\sum_{i=1}^m \pi_i\, F(i)+\epsilon)t + (p^2\sum_{i=1}^m \pi_i\sigma_i^2+\tilde{\epsilon})t.$$

This implies

$$\limsup_{t\to\infty} \frac{1}{t}(\ln I_h(t)^p + \ln I_v(t)^p) \leq p(\sum_{i=1}^m \pi_i\, F(i)+\epsilon) + (p^2\sum_{i=1}^m \pi_i\sigma_i^2+\tilde{\epsilon})$$

Letting ϵ and $\tilde{\epsilon}$ go to zero, we have

$$\limsup_{t\to\infty} \frac{1}{t}\mathbb{E}(\ln I_h(t)^p + \ln I_v(t)^p) \leq \sum_{i=1}^m \pi_i(pF(i)+p^2\sigma_i^2)$$

□

4.4 Stochastic asymptotic stability

In this section we continue to study the long term asymptotic behaviour of the solution of system (4.1)–(4.2). Among the different stability concepts for a stochastic differential equation, we consider stochastic asymptotic stability.

Definition 4.4.1. *Consider a nonlinear stochastic differential equation with Markovian switching*

$$dx(t) = f(x(t),t,r(t))dt + g(x(t),t,r(t))dB(t),\ t \geq t_0 \tag{4.7}$$

The trivial solution $x(t) = 0$ *of equation* (4.7) *is said to be:*

1. stochastically stable *or* stable in probability *if for any* $\epsilon \in (0, 1)$, $\rho > 0$ *and* $t_0 \geq 0$ *there exists* $\delta = \delta(\epsilon, \rho, t_0) > 0$ *such that*

$$\mathbb{P}\{|x(t; t_0, x_0, i)| < \rho \, for \; all \, t \geq t_0\} \geq 1 - \epsilon$$

   for any $|x_0| < \delta$.

2. stochastically asymptotically stable *or* asymptotically stable in probability *if it is stochastically stable and, moreover, for any* $\epsilon \in (0, 1)$, $t_0 \geq 0$ *there exists* $\delta = \delta(\epsilon, \rho, t_0) > 0$ *such that*

$$\mathbb{P}\{\lim_{t\to\infty}(x(t; t_0, x_0))\} \geq 1 - \epsilon$$

   for any $|x_0| < \delta$.

3. stochastically asymptotically stable in large *if it is stochastically stable and, moreover,*

$$\mathbb{P}\{\lim_{t\to\infty}(x(t; t_0, x_0, i))\} = 1, \;\; \forall (t_0, x_0, i) \in \mathbb{R}_+ \times \mathbb{R}^n \times \mathcal{M}.$$

Please refer to [38] for the detail. To prove the stochastic asymptotical stability of system (4.1)–(4.2), we use the following theorem [38].

Theorem 4.4. Assume that there are functions $V \in C^{2,1}(\mathbb{R}^n \times \mathbb{R}_+ \times \mathcal{M}; \mathbb{R}_+)$, $\mu_1, \mu_2 \in \mathcal{K}_\infty$ and $\mu_3 \in \mathcal{K}$ such that

$$\mu_1(|x|) \leq V(x, t, i) \leq \mu_2(|x|), \text{and } \mathcal{L}V(x, t, i) \leq -\mu_3(|x|)$$

for all $(x, t, i) \in \mathbb{R}^n \times \mathbb{R}_+ \times \mathcal{M}$. Then the trivial solution of equation (4.7) is stochastically asymptotically stable in large.

Theorem 4.5. For each $i \in \mathcal{M}$, assume that

$$(\mu_1 + \phi) - \frac{\beta_1^i b_1}{\mu_1} - \frac{\beta_2^i b_1}{2\mu_1} - \frac{\beta^i b_2}{\mu_2} - \frac{{\sigma_{I_h}^i}^2}{2} > 0 \text{ and } \mu_2 - \frac{\beta_2^i b_1}{\mu_1} - \frac{\beta^i b_2}{2\mu_2} - \frac{{\sigma_{I_v}^i}^2}{2} > 0.$$

Then the trivial solution $x(t) = 0$ of system (4.1)–(4.2) is stochastically asymptotically stable in the large.

Proof. For any $i \in \mathcal{M}$, let $V(I_h, I_v, i) = I_h^2 + I_v^2$. Then we have

$$\begin{aligned}
\mathcal{L}V &= 2I_h\left(\frac{\beta_1^r b_1}{\mu_1} I_h - \beta_1^r I_h^2 + \frac{\beta_2^r b_1}{\mu_1} I_v - \beta_2^r I_h I_v - (\mu_1 + \phi) I_h\right) + (\sigma_{I_h}^r)^2 I_h^2 \\
&\quad + 2I_v\left(\frac{\beta^r b_2}{\mu_2} I_h - \beta^r I_h I_v - \mu_2 I_v\right) + (\sigma_{I_v}^r)^2 I_v^2 \\
&= I_h^2\left(2\frac{\beta_1^r b_1}{\mu_1} - 2(\mu_1 + \phi) + (\sigma_{I_h}^r)^2\right) + I_v^2\left(-2\mu_2 + (\sigma_{I_v}^r)^2\right) - 2\beta_1^r I_h^3 \\
&\quad + I_h I_v\left(\frac{2\beta_2^r b_1}{\mu_1} - \frac{2\beta^r b_2}{\mu_2} - 2\beta_2^r I_h^2 I_v - 2\beta^r I_h I_v^2\right) \\
&\leq -I_h^2\left(2(\mu_1 + \phi) - \frac{2\beta_1^r b_1}{\mu_1} - {\sigma_{I_h}^r}^2 - \frac{\beta_2^r b_1}{\mu_1} - \frac{\beta^r b_2}{\mu_2}\right) - I_v^2\left(2\mu_2 - {\sigma_{I_v}^r}^2 - \frac{\beta_2^r b_1}{\mu_1} - \frac{\beta^r b_2}{\mu_2}\right) \\
&\leq -\lambda(I_h^2 + I_v^2) = -\lambda|x|^2
\end{aligned}$$

where $\lambda = \min\{2(\mu_1 + \phi) - \frac{2\beta_1^r b_1}{\mu_1} - {\sigma_{I_h}^r}^2 - \frac{\beta_2^r b_1}{\mu_1} - \frac{\beta^r b_2}{\mu_2},\ 2\mu_2 - {\sigma_{I_v}^r}^2 - \frac{\beta_2^r b_1}{\mu_1} - \frac{\beta^r b_2}{\mu_2}\} > 0$.

Let $\mu_1(|x|) = \frac{1}{2}|x|^2$, $\mu_2(|x|) = |x|^2$ and $\mu_3(|x|) = \lambda|x|^2$. Then clearly, $\mu_1(|x|) \leq V(x, t, i) \leq \mu_2(|x|)$ and $\mathcal{L}V \leq -\mu_3(|x|)$. Also $\mu_1, \mu_2 \in \mathcal{K}_\infty$ and $\mu_3 \in \mathcal{K}$. The conclusion follows from theorem 4.4. □

4.5 Stationary distribution

In this section, we prove that system (4.1)–(4.2) has a unique stationary distribution. For that purpose, we use stochastic Lyapunov function to show the ergodicity and positive recurrence of the system. Then, the conclusion of existence and uniqueness of the stationary distribution follows from the following lemma [62].

Lemma 4.5.1. *Assume the following conditions are satisfied;*

1. *for any $i \neq j$, $q_{ij} > 0$,*

2. *for any $i \in \mathcal{M}$, the diffusion matrix $D(x,i) = g(x,r)g(x,r)^T$ is symmetric and*

$$\lambda|\xi|^2 \leq \langle D(x,i)\,\xi, \xi\rangle \leq \lambda^{-1}|\xi|^2 \text{ for all } x,\, \xi \in \mathbb{R}^n \text{ and } \lambda \in (0,1],$$

3. *there exists a nonempty open set $\mathcal{D}$ with compact closure, such that for each $i \in \mathcal{M}$, there exists a nonnegative function $V(\cdot\,,i) : \mathcal{D}^c \to \mathbb{R}$ such that $V(\cdot\,,i)$ is twice continuously differentiable and for some $\alpha > 0$, $\mathcal{L}V(x,i) \leq -\alpha$, $(x,i) \in \mathcal{D}^c \times \mathcal{M}$,*

then $(x(t), r(t))$ of system (4.7) is ergodic and positive recurrent. That is, there exists a unique stationary distribution $\zeta(\cdot\,,\cdot)$ such that for any Borel measurable function $h : \mathbb{R}^n \times \mathcal{M} \to \mathbb{R}$ satisfying

$$\sum_{i\in\mathcal{M}} \int_{R^n} |h(x,i)|\zeta(dx,i) < \infty,$$

we have

$$\mathbb{P}\left(\lim_{t\to\infty} \int_0^t h(x(s), r(s))\, ds = \sum_{i=1}^m \int_{R^n} h(x,i)\zeta(x,i)dx\right) = 1.$$

Before proving the existence and uniqueness of the stationary distribution, let us substitute $x(t) = \ln(I_h(t))$ and $y(t) = \ln(I_v(t))$, and rewrite system (4.1)–(4.2) as follows:

$$dx(t) = \left(\frac{\beta_1^r b_1}{\mu_1} - \beta_1^r e^x + \frac{\beta_2^r b_1}{\mu_1}\frac{e^y}{e^x} - \beta_2^r e^y - \mu_1 - \phi - \frac{1}{2}\sigma_x^2\right)dt + \sigma_x^r dB_x(t), \tag{4.8}$$

$$dy(t) = \left(\frac{\beta^r b_2}{\mu_2}\frac{e^x}{e^y} - \beta^r e^x - \mu_2 - \frac{1}{2}\sigma_y^2\right)dt + \sigma_y^r dB_y(t). \tag{4.9}$$

Remark 4.5.2. (i) Since the ergodic property and positive recurrence of system (4.1)–(4.2) is equivalent to that of (4.8)–(4.9), we only need to prove for the later system [33, 59].

(ii) To prove the existence of a unique stationary distribution, we impose the following assumptions.

For each $i \in \mathcal{M}$,

1. $\frac{\beta_1^i b_1}{\mu_1} + \frac{\beta^i b_2}{\mu_2} - \mu_1 - \phi > 0$ and $\frac{\beta_2^i b_1}{\mu_1} - \mu_2 > 0$.
2. $\sum_{i=1}^{m} \pi_i R_i > 0$, where $R_i := \frac{\beta_1^i b_1}{\mu_1} - (\mu_1 + \phi + \mu_2 + \frac{1}{2}\sigma_x^2 + \frac{1}{2}\sigma_y^2)$.

Lemma 4.5.3. *Under assumption 1, for $i = 1, 2, \ldots, m$ the system*

$$\begin{aligned} c_1(i)\left(\frac{\beta_1^i b_1}{\mu_1} + \sum_l q_{il} - \mu_1 - \phi\right) + c_2(i)\frac{\beta^i b_2}{\mu_2} &= -2\beta_1^i \\ c_1(i)\frac{\beta_2^i b_1}{\mu_1} + c_2(i)(\sum_l q_{il} - \mu_2) &= -\beta_2^i \end{aligned} \tag{4.10}$$

has a unique solution $(c_1(1), c_1(2), \ldots, c_1(m), c_2(1), c_2(2), \ldots, c_2(m)) \in \mathcal{R}^{2m}$.

Proof. Let

$$A = \begin{bmatrix} \frac{\beta_1^1 b_1}{\mu_1} + q_{11} - \mu_1 - \phi & q_{12} & \ldots & q_{1m} & \frac{\beta^1 b_2}{\mu_2} & 0 & \ldots & 0 \\ q_{21} & \frac{\beta_1^2 b_1}{\mu_1} + q_{22} - \mu_1 - \phi & \ldots & q_{1m} & 0 & \frac{\beta^2 b_2}{\mu_2} & \ldots & 0 \\ \ldots & \ldots & \ldots & \ldots & \ldots & \ldots & \ldots & \ldots \\ q_{m1} & q_{m2} & \ldots & \frac{\beta_1^2 b_1}{\mu_1} + q_{mm} - \mu_1 - \phi & 0 & 0 & \ldots & \frac{\beta^m b_2}{\mu_2} \\ \frac{\beta_2^1 b_1}{\mu_1} & 0 & 0 & \ldots & q_{11} - \mu_2 & q_{12} & \ldots & q_{1m} \\ \frac{\beta_2^2 b_1}{\mu_1} & 0 & 0 & \ldots & q_{21} - \mu_2 & q_{22} & \ldots & q_{2m} \\ \ldots & \ldots & \ldots & \ldots & \ldots & \ldots & \ldots & \ldots \\ 0 & 0 & \ldots & \frac{\beta_2^m b_1}{\mu_1} & q_{m1} & q_{m2} & \ldots & q_{mm} - \mu_2 \end{bmatrix}$$

$$C = (c_1(1), c_1(2), \ldots, c_1(m), c_2(1), c_2(2), \ldots, c_2(m))^T,$$

and

$$B = (-2\beta_1^1, -2\beta_1^2, \ldots, -2\beta_1^m, -\beta_2^1, -\beta_2^2, \ldots, -\beta_2^m)^T.$$

Then system (4.10) can be written as $AC = B$. It is enough to show that all the leading principal minors of A are positive, and the conclusion of the lemma will follow from the fact that A is a nonsingular $M-$matrix [38].

For $1 \leq k \leq m$, the leading principal sub-matrix A_k is given by

$$A_k = \begin{bmatrix} \frac{\beta_1^1 b_1}{\mu_1} + q_{11} - \mu_1 - \phi & q_{12} & \dots & q_{1k} \\ q_{21} & \frac{\beta_1^2 b_1}{\mu_1} + q_{22} - \mu_1 - \phi & \dots & q_{1k} \\ \dots & \dots & \dots & \dots \\ q_{k1} & q_{k2} & \dots & \frac{\beta_1^k b_1}{\mu_1} + q_{kk} - \mu_1 - \phi \end{bmatrix}$$

$$A_{k+m} = \begin{bmatrix} \frac{\beta_1^1 b_1}{\mu_1} + q_{11} - \mu_1 - \phi & q_{12} & \dots & q_{1m} & \frac{\beta^1 b_2}{\mu_2} & 0 & \dots & 0 \\ q_{21} & \frac{\beta_1^2 b_1}{\mu_1} + q_{22} - \mu_1 - \phi & \dots & q_{1m} & 0 & \frac{\beta^2 b_2}{\mu_2} & \dots & 0 \\ \dots & \dots & \dots & \dots & \dots & \dots & \dots & \dots \\ q_{m1} & q_{m2} & \dots & \frac{\beta_1^m b_1}{\mu_1} + q_{mm} - \mu_1 - \phi & 0 & 0 & \dots & \frac{\beta^k b_2}{\mu_2} \\ \frac{\beta_2^1 b_1}{\mu_1} & 0 & 0 & \dots & q_{11} - \mu_2 & q_{12} & \dots & q_{1k} \\ 0 & \frac{\beta_2^2 b_1}{\mu_1} & 0 & \dots & q_{21} & q_{22} - \mu_2 & \dots & q_{2k} \\ \dots & \dots & \dots & \dots & \dots & \dots & \dots & \dots \\ 0 & 0 & \dots & \frac{\beta_2^k b_1}{\mu_1} & q_{k1} & q_{k2} & \dots & \mu_2 q_{kk} \end{bmatrix}$$

For A_k the sum of the i^{th} row for $1 \leq i \leq k$ is given by

$$\begin{aligned} \sum_{j=1}^{k} a_{ij} &= \frac{\beta_1^i b_1}{\mu_1} + \sum_{j=1}^{k} q_{ij} - \mu_1 - \phi, \quad \text{since } \sum_{j=1}^{m} q_{ij} = 0 \\ &= \frac{\beta_1^i b_1}{\mu_1} + \sum_{j=k+1}^{m} q_{ij} - \mu_1 - \phi > 0. \end{aligned}$$

This follows from the first assumption and $q_{ij} > 0$ for $i \neq j$.

Similarly, the sum of the i^{th} row of the matrix A_{k+m} is given as follows.

If $1 \leq i \leq m$, then

$$\begin{aligned} \sum_{j=1}^{k+m} a_{ij} &= \frac{\beta_1^i b_1}{\mu_1} + \sum_{j=1}^{m} q_{ij} + \frac{\beta^i b_2}{\mu_2} - \mu_1 - \phi, \quad \text{since } \sum_{j=1}^{m} q_{ij} = 0 \\ &= \frac{\beta_1^i b_1}{\mu_1} + \frac{\beta^i b_2}{\mu_2} - \mu_1 - \phi > 0. \end{aligned}$$

and if $m \leq i \leq m+k$, then

$$\sum_{j=1}^{k+m} a_{ij} = \frac{\beta_2^i b_1}{\mu_1} + \sum_{j=1}^{k} q_{ij} - \mu_2, \quad \text{since } \sum_{j=1}^{m} q_{ij} = 0$$
$$= \frac{\beta_2^i b_1}{\mu_1} + \sum_{j=k+1}^{m} q_{ij} - \mu_2 > 0.$$

Again this follows from the first assumption and $q_{ij} > 0$ for $i \neq j$. □

Theorem 4.6. If assumptions 1 and 2 hold, then for any initial value $(x(0), y(0), r(0))$ system (4.8)–(4.9) has a unique stationary distribution.

Proof. The first condition of lemma 4.5.1 follows from the fact that the transition rate is positive. That is for $i \neq j$, $q_{ij} > 0$.

Also the diffusion matrix of system (4.8)–(4.9) is given by

$$D(x, y, i) = diag((\sigma_x^i)^2, (\sigma_y^i)^2).$$

Thus, the second condition of lemma 4.5.1 follows since $D(x, y, i)$ is positive semi-definite.

Finally, to show the third condition of lemma 4.5.1, let $\theta \in (0, 1)$ and define $V_1(x, y) = \frac{1}{\theta+1}(e^x + e^y)^{\theta+1}$ and $V_2(x, y, i) = c_1(i)e^x + c_2(i)e^y - x - y - \zeta_i$. Then we have

$$\begin{aligned}
\mathcal{L}V_1 &= (e^x + e^y)^\theta \left(\frac{\beta_1^r b_1}{\mu_1} e^x - \beta_1^r e^{2x} + \frac{\beta_2^r b_1}{\mu_1} e^y - \beta_2^r e^{x+y} - (\mu_1 + \phi)e^x + \frac{\beta^r b_2}{\mu_2} e^x - \beta_1^r e^{x+y} - \mu_2 e^y \right) \\
&\quad + \frac{1}{2}\theta(e^x + e^y)^{\theta-1}(e^{2x}\sigma_x^2 + e^{2y}\sigma_y^2) \\
&\leq -\left(\mu_1 + \phi - \frac{\beta_1^r b_1}{\mu_1} - \frac{\beta^r b_2}{\mu_2} - \frac{\theta}{2}\sigma_x^2 \right) e^{(\theta+1)x} - \left(\mu_2 - \frac{\beta_2^r b_1}{\mu_1} - \frac{\theta}{2}\sigma_y^2 \right) e^{(\theta+1)y} \\
&\leq -\left(\mu_1 + \phi - \frac{\tilde{\beta}_1 b_1}{\mu_1} - \frac{\tilde{\beta} b_2}{\mu_2} - \frac{\theta}{2}\tilde{\sigma}_x^2 \right) e^{(\theta+1)x} - (\mu_2 - \frac{\tilde{\beta}_2 b_1}{\mu_1} - \frac{\theta}{2}\tilde{\sigma}_y^2) e^{(\theta+1)y},
\end{aligned}$$

where $\tilde{\lambda} = \max_{i \in \mathcal{M}} \lambda^i$.

Also

$$\begin{aligned}
\mathcal{L}V_2 &= c_1(i)\left(\frac{\beta_1^i b_1}{\mu_1}e^x - \beta_1^i e^{2x} + \frac{\beta_2^i b_1}{\mu_1}e^y - \beta_2^i e^{x+y} - (\mu_1+\phi)e^x\right) + \sum_l c_1(i)q_{il}e^x \\
&+ c_2(i)\left(\frac{\beta^i b_2}{\mu_2}e^x - \beta_1^i e^{x+y} - \mu_2 e^y\right) + \sum_l c_2(i)q_{il}e^y - \frac{\beta_1^i b_1}{\mu_1} + \beta_1^i e^x - \frac{\beta_2^i b_1}{\mu_1}\frac{e^y}{e^x} \\
&+ \beta_2^i e^y + \mu_1 + \phi + \frac{1}{2}\sigma_x^2 - \frac{\beta^i b_2}{\mu_2}\frac{e^x}{e^y} + \beta_1^i e^x + \mu_2 + \frac{1}{2}\sigma_y^2 - \sum_l q_{il}\zeta_l \\
&\leq e^x\left(c_1(i)(\frac{\beta_1^i b_1}{\mu_1} - \mu_1 - \phi + \sum_l q_{il}) + c_2(i)\frac{\beta^i b_2}{\mu_2} + 2\beta_1^i\right) \\
&+ e^y\left(c_1(i)\frac{\beta_2^i b_1}{\mu_1} + c_2(i)(\sum_l q_{il} - \mu_2) + \beta_2^i\right) + (-\frac{\beta_1^i b_1}{\mu_1} + \mu_1 + \phi + \mu_2 + \frac{1}{2}\sigma_x^2 + \frac{1}{2}\sigma_y^2) - \sum_l q_{il}\zeta_l \\
&= -\frac{\beta_1^i b_1}{\mu_1} + \mu_1 + \phi + \mu_2 + \frac{1}{2}\sigma_x^2 + \frac{1}{2}\sigma_y^2 - \sum_l q_{il}\zeta_l \\
&\leq -R_i - \sum_l q_{il}\zeta_l
\end{aligned}$$

Note that the last expression follows from lemma 4.5.3. Since we assume that the system can switch from one regime to any other, the generator matrix Q is irreducible. Thus for $R = (R_1, R_2, \ldots, R_m)^T$, there exist $\zeta = (\zeta_1, \zeta_2, \ldots, \zeta_m)^T$ such that

$$Q\zeta = (\sum_{i=1}^m \pi_i R_i)\vec{1} - R.$$

This is equivalent to

$$-R_i - \sum_{j=1}^m q_{ij}\zeta_j = -\sum_{i=1}^m \pi_i R_i$$

and therefore we conclude that

$$\mathcal{L}V_2 \leq -\sum_{i=1}^m \pi_i R_i.$$

Finally, define $V = V_1 + V_2$. Then,

$$\mathcal{L}V \leq -(\mu_1 + \phi - \frac{\tilde{\beta}_1 b_1}{\mu_1} - \frac{\tilde{\beta} b_2}{\mu_2} - \frac{\theta}{2}\tilde{\sigma}_x^2)e^{(\theta+1)x} - (\mu_2 - \frac{\tilde{\beta}_2 b_1}{\mu_1} - \frac{\theta}{2}\tilde{\sigma}_y^2)e^{(\theta+1)y} - \sum_{i=1}^m \pi_i R_i.$$

Now, as $x, y \to \infty$, $\mathcal{L}V \leq -\infty$ and as $x, y \to -\infty$, $\mathcal{L}V \leq -\sum_{k=1}^{m} \pi_k R_k$

Thus by taking $\kappa > 0$, sufficiently large and letting $\mathcal{D} = (-\kappa, \kappa) \times (-\kappa, \kappa)$ we have for any $(x, y, i) \in \mathcal{D}^c \times \mathcal{M}$, it follows that there exists $\alpha > 0$ such that $\mathcal{L}V \leq -\alpha$. □

4.6 Numerical simulations and examples.

In this section, we conduct numerical simulations to illustrate some of the theoretical results. For this purpose, we adopt the following parameter values. Let $r(t), t \geq 0$ be a right-continuous Markov chain taking values in $\mathcal{M} = \{1, 2, 3\}$, and it is generated by

$$Q = \begin{bmatrix} -3 & 2 & 1 \\ 1 & -2 & 1 \\ 1 & 1 & -2 \end{bmatrix}.$$

Thus the stationary distribution is given by $\pi = (1/4,\ 5/12,\ 1/3)$. To demonstrate corollary (4.3.1) we chose the following values,

$$b_1 = 10,\ b_2 = 0.1,\ \mu_1 = 0.83,\ \phi = 0.35,\ \mu_2 = 0.1,\ \beta^1 = 0.002,\ \beta^2 = 0.001,\ \beta^3 = 0.003$$

$$\beta_1^1 = 0.005,\ \beta_1^2 = 0.004,\ \beta_1^3 = 0.006,\ \beta_2^1 = 0.00003,\ \beta_2^2 = 0.000025,\ \beta_2^3 = 0.00004.$$

For each $i \in \mathcal{M}$ we take the following noise intensities

$$\sigma_{S_h}^i = \sigma_{I_h}^i = \sigma_{S_v}^i = \sigma_{I_v}^i = 0.2.$$

Thus using equation (4.5) we get

$$\sum_{i=1}^{3} \pi_i F(i) = -1.1373 < 0.$$

By corollary (4.3.1) the disease-free equilibrium $E_0 = (12.0482, 0, 1, 0)$ is almost surely exponentially stable, as shown in figure (4.1) below. Now we keep the noise intensities the same

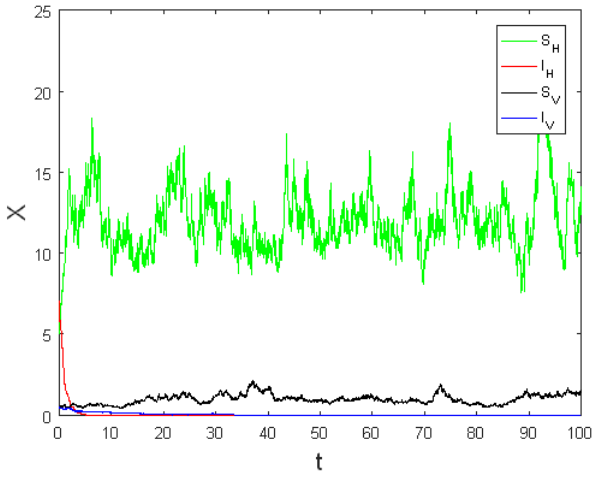

Figure 4.1: Almost surely exponential stability of the disease-free equilibrium.

as in the above example and consider the following values of parameters

$$b_1 = 5,\ b_2 = 4,\ \mu_1 = 0.83,\ \phi = 0.35,\ \mu_2 = 0.3,\ \beta^1 = 0.06,\ \beta^2 = 0.05,\ \beta^3 = 0.055$$

$$\beta_1^1 = 0.5,\ \beta_1^2 = 0.6,\ \beta_1^3 = 0.65,\ \beta_2^1 = 0.3,\ \beta_2^2 = 0.25,\ \beta_2^3 = 0.4.$$

Both assumptions 1 and 2 are satisfied with $\sum_{k=1}^{3} \pi_k R_k = 1.9137$. Thus by theorem 4.6 system (4.1)–(4.2) has a unique stationary distribution and this is verified in the figure 4.3.

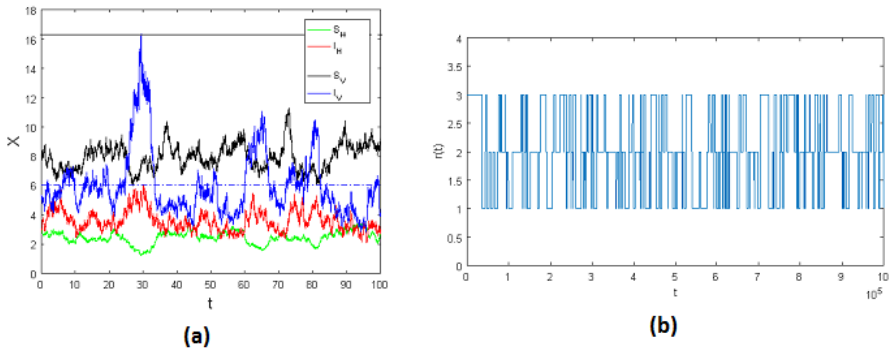

Figure 4.2: Trajectories of solution of system (4.1)–(4.2) and the corresponding path of a single Markov chain $r(t)$ for the parameter values given above.

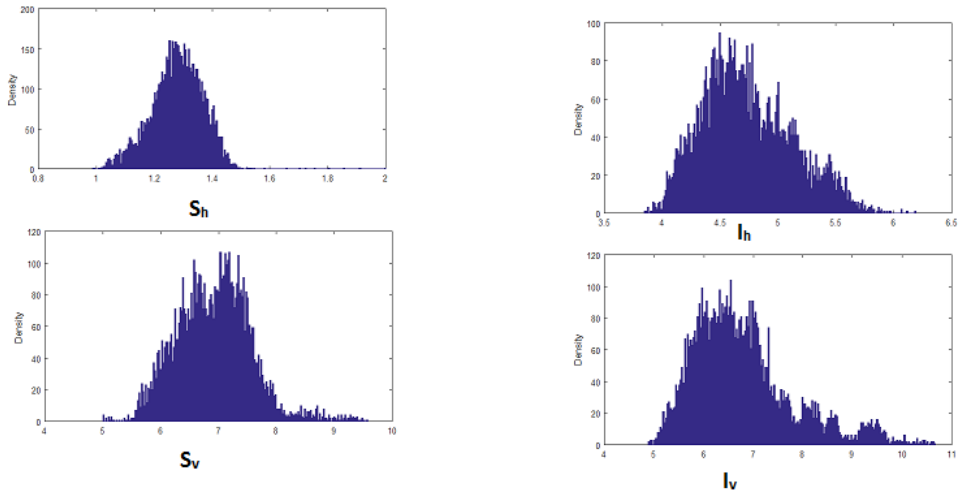

Figure 4.3: Frequency histograms of the solution of system (4.1)–(4.2)

.

Remark 4.6.1. It is important to see that we might have cases in which the infection is persistence in the deterministic case ($\mathcal{R}_0 > 1$), while there is an extinction in the regime-switching stochastic case as shown in figure 4.4 . This is due to a large environmental fluctuation in the infectious group I_h. In order to demonstrate this we consider the following values of parameters.

$b_1 = 2,\ b_2 = 1,\ \mu_1 = 0.83,\ \phi = 0.35,\ \mu_2 = 0.3,\ \beta^1 = 0.14,\ \beta^2 = 0.13,\ \beta^3 = 0.15,\ \beta_1^1 = 0.09,$
$\beta_1^2 = 0.08,\ \beta_1^3 = 0.085,\ \beta_2^1 = 0.3,\ \beta_2^2 = 0.25,\ \beta_2^3 = 0.4, \sigma_{I_h}^i = 0.88,\ \sigma_{S_h}^i = \sigma_{S_v}^i = \sigma_{I_v}^i = 0.1.$

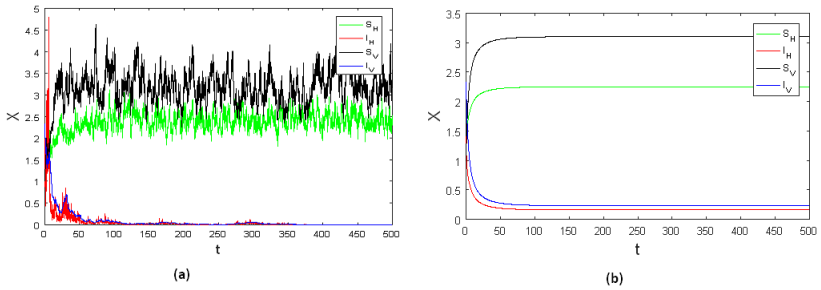

Figure 4.4: Extinction of infection in the regime-switching stochastic case, while existence of endemic equilibrium in the deterministic model.

Chapter 5

Summary

In this dissertation we presented a deterministic and stochastic epidemic model with direct transmission. The deterministic model is a compartmental model, which divides the total host population into susceptible and infected. Similarly, the vector population is divided into susceptible and infected groups. By studying the different ways of transmitting the infection form one compartment to another, we developed a system of nonlinear differential equations that describes the epidemiology of vector-borne disease. We considered the SIS type of structure for the host and the SI type of structure for the vector.

In chapter 2, we presented and analyzed a deterministic vector-host epidemic mode with direct transmission. We first obtained the disease-free equilibrium point E_0 and endemic equilibrium point E_1. Then we calculated the basic reproductive number $\mathcal{R}_0$ using the second generation matrix approach. We proved that if $\mathcal{R}_0 < 1$, the disease-free equilibrium point is both locally and globally asymptotically stable and thus, the infection will be extinct. Similarly if $\mathcal{R}_0 > 1$, the endemic equilibrium point E_1 is both locally and globally asymptotically stable and as a result, the infections will be persistent. Next we provided numerical simulations for different values of parameters to illustrate the analytical results. For this purpose, we took $\mathcal{R}_0 = 0.71$ and $\mathcal{R}_0 = 34.20$. The simulation shows that the infection will die out in the first case, while it persisted in the second case. Finally, using the perturbation of fixed point estimation, we conducted sensitivity analysis of the basic reproductive number to investigate the relative importance of each parameters in relation to it. From this analysis we concluded that, if the values of the parameters are known, we can determine which parameter will have a significant impact on the incidence rate.

In chapter 3 we presented a stochastic vector-host epidemic model with direct transmission, using a nonlinear system of stochastic differential equations. First we derived the stochastic model from the corresponding deterministic model by including environmental fluctuations and studied how these fluctuations affect the epidemiological model presented in chapter 2. For this purpose, we assumed that during the given interval of time, each compartments change according to the deterministic equation described in chapter 2 and by a random amount due to the environmental fluctuations. Under this assumption, we determined the drift and diffusion coefficients of the diffusion process. Then we proved the existence of a unique nonnegative global solution to the system. Next we proved that the solution to the stochastic system is ultimately bounded in probability and stochastically permanent. These properties of the solution indicates how the total population in the model changes in the long run. We also derived the basic reproductive number $\mathcal{R}_0^s$ for the stochastic model, and proved that if $\mathcal{R}_0^s < 1$, then the number of infected hosts and vectors will tend to zero exponentially almost surely. Moreover, we concluded that a very large environmental fluctuation can subdue the number of infected hosts and vectors. By studying the asymptotic stability of the linearized system, we provided conditions for the stability of the nonlinear system. We also studied the necessary conditions to ensure the non-extinction of infection in the model using the idea of persistence in mean. The result indicates that under some assumptions, the solution to the stochastic system will stay away from the disease-free equilibrium point E_0 and oscillate around the endemic equilibrium point E_1. Moreover, we showed that the amplitudes of vibration depends on the values of the noise intensities. Finally, using the Milstein scheme we conducted numerical simulations for the stochastic system. We provided example to show that extinction of infection in the deterministic model implies extinction of infection in the stochastic case, while large environmental fluctuation may suppress infection. We also provided examples and simulations to show how the noise or fluctuation intensities affect the behaviour of the solution.

Finally, in chapter 4 we presented and analyzed a regime switching vector-host epidemic model with direct transmission by including another type of environmental noise. This noise is described as a switching between one or more environments which is memoryless and the waiting time for the next switching follows the exponential distribution. We first proved that for any initial value in $\mathbb{R}^2_+$, the stochastic system has a unique positive global solution and the solution will remain in $\mathbb{R}^2_+$ with probability 1. We then provided conditions for the extinction of infection in the stochastic regime switching model. Particularly, we proved that the disease-free equilibrium point is exponentially stable almost surely. Next we studied the long term asymptotic behaviour of the solution of the stochastic system. By defining an appropriate Lyapunov function, we proved that under some conditions, the trivial solution of the system is stochastically asymptotically stable. We also discussed the existence of a unique stationary distribution. In order to prove this, we used a stochastic Lyapunov function to show that the stochastic system is ergodic and positive recurrent. Finally, we performed numerical simulations to confirm some of the analytical results.

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Appendices

.0.1 Numerical scheme

Given the following $n-$dimensional stochastic differential equation

$$dX_t^i = a^i(t, X_t)\, dt + \sum_{j=1}^{m} b^{i,j}(t, X_t)\, dW_t^j, \quad i = 1, 2, \ldots n, \tag{1}$$

where $X_t = (X_t^1, X_t^2, \ldots, X_t^n)$ and $W_t = (W_t^1, W_t^2, \ldots, W_t^m)$, is an $m-$dimensional Brownian motion such that W_t^i for $i = 1, 2, \ldots, m$ are independent.

The Milstein scheme for (1) is given by

$$\begin{aligned} X_{n+1}^i = X_n^i + a^i(t_n, X_n)\Delta_n + \sum_{j=1}^{m} b^{i,j}(t_n, X_n)\Delta W_n^j \\ + \sum_{j_1, j_2=1}^{m} L^{j_1} b^{i,j_2}(t_n, X_n) \int_{t_n}^{t_{n+1}} \int_{t_n}^{t} dW_s^{j_1}\, dW_t^{j_2}, \end{aligned} \tag{2}$$

for $1 \leq i \leq n$. The partial derivative operators L^j for $j = 1, 2, \ldots m$ are given by

$$L^j = \sum_{k=1}^{n} b^{k,j}\, \frac{\partial}{\partial x^k}. \tag{3}$$

Since the diffusion coefficient of the stochastic model (3.5)–(3.8) is a square diagonal noise, the Milstein scheme (2) can be written as follows.

$$\begin{aligned} X^i_{n+1} = X^i_n + a^i(t_n, X_n)\Delta_n + \sum_{j=1}^{m} b^{i\,j}(t_n, X_n)\Delta W^j_n \\ + \frac{1}{2}\sum_{j_1=1}^{m} L^{j_1} b^{i\,j_1}(t_n, X_n)\{(\Delta W^{j_1}_n)^2 - \Delta_n\} \\ + \frac{1}{2}\sum_{\substack{j_1,j_2=1 \\ j_1\neq j_2}}^{m} L^{j_1} b^{i\,j_2}(t_n, X_n)\Delta W^{j_1}_n \Delta W^{j_2}_n, \end{aligned}$$

Applying the above scheme for the stochastic model we get the following discretization. For simplicity denote $X^1 = S_h$, $X^2 = I_h$, $X^3 = S_v$, $X^4 = I_v$.

$$\begin{aligned} X^1_{n+1} &= X^1_n + a^1\Delta_n + b^{11}\Delta W^1_n + \frac{1}{2}(\sigma_{X^1})^2 X^1_n\{(\Delta W^1_n)^2 - \Delta_n\} \\ X^2_{n+1} &= X^2_n + a^2\Delta_n + b^{22}\Delta W^2_n + \frac{1}{2}(\sigma_{X^2})^2 X^2_n\{(\Delta W^2_n)^2 - \Delta_n\} \\ X^3_{n+1} &= X^3_n + a^3\Delta_n + b^{33}\Delta W^3_n + \frac{1}{2}(\sigma_{X^3})^2 X^3_n\{(\Delta W^3_n)^2 - \Delta_n\} \\ X^3_{n+1} &= X^4_n + a^4\Delta_n + b^{44}\Delta W^4_n + \frac{1}{2}(\sigma_{X^4})^2 X^4_n\{(\Delta W^4_n)^2 - \Delta_n\}, \end{aligned}$$

where $(a^1\, a^2\, a^3\, a^4)^T = F(X,t)$ and $(b^{ij})^4_{i,j=1} = G(X,t)$. The noise increments ΔW^i_n are Gaussian random variables $N(0, \Delta_n)$.

.0.2 MATLAB code

```
%MIL Milstein method on 3D finance SDE

% SDE is
% Diseretized Brownian path over [0,1] has delta = 2^(-18).
% Milstein timestep is Delta· sqrt(delta).
% Substeps for double integral are of size delta.
clf
rng default
randn( 'state' ,1)
T = 200; Delta = 2^(-5); delta = Delta^2;
L = T/Delta; K = Delta/delta;
a=0.002; b=0.001; c=0.7; d=0.006; e=0.35; f=0.83; h=50; g=60;
%a=beta_1, b= beta_2, c= mu_2, d=beta, e= phi f= mu_1, g= b_1, h= b_2

sh1=0; sh2=0;
sv1=0; sv2=0;

X1 = zeros(1,L+1); X2 = zeros(1,L+1);
X3 = zeros(1,L+1); X4 = zeros(1,L+1);
% X3 = zeros(1,L+1);
% Y2 = 0;
X1(1) = 20;
X2(1) = 40;
X3(1) = 40;
X4(1) = 15;
% X3(1) = 0.1;

for j = 1:L
% Y1 = 0;
Winc1 = 0; Winc2 = 0;
Winc3 = 0; Winc4 = 0;

a1=g-f*X1(j)-b*X1(j)*X4(j)-a*X1(j)*X2(j)+e*X2(j);
a2=a*X1(j)*X2(j)+b*X1(j)*X4(j)-(f+e)*X2(j);
a3=h-c*X3(j)-d*X2(j)*X3(j);
a4=d*X2(j)*X3(j)-c*X4(j);
b11=sh1*X1(j);  b22=sh2*X2(j);
b33=sv1*X3(j);  b44=sv2*X4(j);

for k = 1:K
    dW1 = sqrt(delta)*randn;
    dW2 = sqrt(delta)*randn;
    dW3 = sqrt(delta)*randn;
    dW4 = sqrt(delta)*randn;
%     Y1 = Y1 + Y2*dW1;
%     Y2 = Y2 + dW2;

    Winc1 = Winc1 + dW1;
    Winc2 = Winc2 + dW2;
    Winc3 = Winc3 + dW3;
    Winc4 = Winc4 + dW4;
```

Figure 1: MATLAB code used for the numerical simulation of the stochastic model.