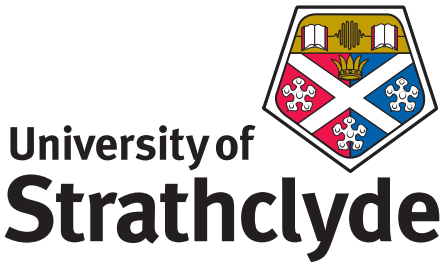

Epidemics under Environmental Noise by Stochastic SIS Model

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Siyang Cai

Department of Mathematics and Statistics

University of Strathclyde

Glasgow, UK

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Abstract

In this research, three different stochastic SIS models are concerned with different environmental noises. We firstly introduce two perturbations in the classical deterministic susceptible-infected-susceptible (SIS) epidemic model. Gray *et al.* [1] in 2011 used a perturbation on β in SIS model. Based on their previous work, we consider another perturbation on the parameter $\mu + \gamma$ and formulate the original model as a stochastic differential equation (SDE) with two independent Brownian motions for the number of infected population. We then prove that our model has a unique and bounded global solution $I(t)$. Also we establish conditions for extinction and persistence of the infected population $I(t)$. Under the conditions of persistence, we show that there is a unique stationary distribution and derive its mean and variance. Computer simulations illustrate our results and provide evidence to back up our theory. This work is published in JMAA [2].

We then introduce the second model replacing two independent Brownian motions in the first model by two correlated Brownian motions. We consider the two same perturbations in the deterministic SIS model and formulate the original model as a stochastic differential equation (SDE) with two correlated Brownian motions for the number of infected population, based on previous work from Gray *et al.* in 2011 and Hening's work [3] in 2017. Conditions for the solution to become extinct and persistent are then stated, followed by computer simulations to illustrate the results. Compared to the formal model, the conditions of extinction are extended after correlation between two white noises is considered. However, we are not able to compute the mean and variance of the stationary distribution. Note that this section has also been published as an article in Nonlinear Dynamics in 2019 [4].

Moreover, we combined the first model with [5] to add telegraph noise by using Markovian switching to generate the third model. Similarly, conditions for extinction and persistence are then given and proved, followed by explanation on the stationary distribution. Computer simulations are clearly illustrated with different sets of parameters, which support our theorems in this chapter. Compared to two previous models, conditions are given based on the overall behaviour of the solution but not separately specified in every state of the Markov chain.

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Notations

positive	:	> 0.
nonpositive	:	≤ 0.
negative	:	< 0.
nonnegative	:	≥ 0.
a.s.	:	almost surely, or P-almost surely, or with probability 1.
$A := B$	:	A is defined by B or A is denoted by B.
$\emptyset$	:	the empty set.
$\mathbf{1}_A$	:	the indicator function of A, i.e. $\mathbf{1}_A(x)$=1 if $x \in A$ or otherwise 0.
A^c	:	the complement of A in Ω.
$A \subset B$	:	$A \cap B^c = \emptyset$.
$A \subset B$ a.s.	:	$P(A \cap B^c) = 0$.
$A \setminus B$	:	the set that contains all the elements of A that are not in B.
$a \wedge b$	:	the minimum of a and b.
$a \vee b$	:	the maximum of a and b.
$f : A \to B$	:	the mapping f from A to B.
R_0^D	:	the basic reproduction number of the deterministic SIS model.
$V(x)$	:	the Lyapunov functions.
V_x	:	$= (V_{x_1}, ..., V_{x_d}) = (\frac{\partial V}{\partial x_1}, ..., \frac{\partial V}{\partial x_d})$.
V_{xx}	:	$= (\frac{\partial^2 V}{\partial x_i \partial x_j})_{d \times d}$.
$LV(x)$	:	the Itô operator on Lyapunov functions.
R_+	:	the set of all nonnegative real numbers.
R^d	:	the d-dimensional Euclidean space.
R_+^d	:	$=\{x \in R^d : x_i > 0, 1 \leq i \leq d\}$.
$\mathcal{B}^d$	:	the Borel-σ-algebra.
A^T	:	the transpose of a vector or matrix A.
trace A	:	the trace of a square matrix $A = (a_{ij})_{d \times d}$.
diag A	:	the diagonal of a square matrix diag $A_{d \times d} = (a_{11}, a_{22}, ..., a_{dd})$.
$A > 0$	:	all the elements of the vector A are positive.
$\mathcal{L}^p([a,b]; R^d)$	:	the family of R^d-valued $\mathcal{F}_t$-adapted process $\{f(t)\}_{a \leq t \leq b}$ such that $\int_a^b \lvert f(t)\rvert^p \mathrm{d}t < \infty$ a.s.
$\mathcal{L}^p(R_+; R^d)$	:	the family of R^d-valued $\mathcal{F}_t$-adapted process $\{f(t)\}_{t \geq 0}$

such that $\{f(t)\}_{0\le t\le T} \in \mathcal{L}^p([0,T];R^d)$ for every $T>0$.

$\mathcal{M}^p([a,b];R^d)$: the family of processes $\{f(t)\}_{a\le t\le b}$ in $\mathcal{L}^p([a,b];R^d)$ such that $\mathbb{E}\int_a^b |f(t)|^p \mathrm{d}t < \infty$.

$\mathcal{M}^p(R_+;R^d)$: the family of process $\{f(t)\}_{a\le t\le b}$ such that for every $T>0$ $\{f(t)\}_{0\le t\le T} \in \mathcal{M}^p([0,T];R^d)$.

$C^{2,1}(D^d\times R_+;R)$: the family of all real-valued functions $V(x,t)$ defined on $D\times R_+$ which are continuously twice differentiable in $x\in D$, and once differentiable in $t\in R_+$.

Chapter 1

Introduction

In this research, we are going to introduce the establishment of three different stochastic epidemic SIS models. As epidemics have already been common issues for centuries, it is necessary and important to introduce some typical deadly diseases in the history initially in this section. Then previous research on deterministic epidemic models will be explained. Combined with a brief definition of environmental noises, we will finally introduce stochastic epidemic models in previous work with different noises.

1.1 Infectious Disease in Human Society

Epidemics of infectious diseases have already become a great threat to human beings. A disease that we do not know well about can be dangerous and fatal with its high contagious rate and lack of professional medical control. In history, there are many examples of diseases that become serious issues to the human society. For instance, the Black Death [6, 7, 8] killed approximately $25\% - 50\%$ of Europe's population between 1347 and 1350. It was the first epidemic of the second plague pandemic which kept recurring for around 500 years. In 1870 to 1940, Russia suffered from typhus [9, 10, 11], an acute febrile disease that was spread by the clothes louse, which people of all age were subjected to. Typhus can be recognized

and traced back to as early as 1084 in Spain. It was carried and spread by armies into civilian populations through survivors and refugees [12]. The mortality rate can vary between 5% to 40% or even higher. About 2 to 3 million lives were taken from 1918 to 1922. Small outbreaks emerged in Russia in 1997 even after the disease was controlled in 1940, which caused by the relapse of those who were primarily infected. Although in the 21st century, technology has developed fast enough to support medical research, we still do not fully find cures to diseases like HIV (human immunodeficiency virus) [13, 14], which attacks and weakens the immune system by depleting CD4+ T-cells. According to the reports of the World Health Organization [15], there were approximately 37.9 million people living with HIV in 2018, with about 1.7 million new infections globally. Moreover, Jones *et al.* [16] indicate the emerging infectious diseases are increasing globally based on their database, which have caused a significant impact on global health and economy. Antimicrobial drug resistance is one of the reasons for why it is not easy to remove diseases.

Consequently, the indispensability of studying diseases is obvious. Although eradication of epidemics is not easy and even impractical in some cases, people never stop to find different types of methods to control the diseases [17]. Therefore, it is the most important to know how diseases transmit among populations. Gottfried [18] suggests that there are four concerns on transmission of diseases. The first concern is the environment where the diseases develop. Temperature, landscape and climate are possible contributions to the outbreak. The second consideration is the causation of the disease. The natural ecology defines that all epidemics are caused by parasites which are related to some larger organisms. For example, *Rickettsia prowazekii* [19] is observed in the lice which causes typhus. The third factor is the toxicity of disease. Non-lethal infections are usually mildly deleterious, while lethal diseases have periodical behaviour to kill large numbers of a population during an outbreak. And the final concern about epidemics is the way of transmission, for instance through venereal contact.

Such complex phenomena can usually be described by mathematical modelling in order to reflect the four factors of diseases [20]. Okubo [21] regards mathematical epidemic models as crucial and necessary factors in quantitative analysis and

prediction of the dynamic behaviour of diseases [22]. The methods are basically the same as those used in other fields such as biophysics and molecular biology, while the aim of building mathematical treatment is to develop a decision-making model to help analyse the results of choosing different strategies to control the diseases [23], which directly determines the selection of an immunization programme, the allocation of limited resources and the optimal control eradication techniques. There are mainly two different kinds of mathematical epidemic models: deterministic epidemic models and stochastic epidemic models. A deterministic model has certain parameters that only take the given value, while a stochastic model can be derived from deterministic model, with some parameters replaced by random variables to further explain the population dynamics [24] in the behaviour of disease. There are also other ways to involve stochasticity in the deterministic epidemic models, such as introducing demographic stochasticity. Now we are going to firstly introduce some classical examples of deterministic epidemic models, followed by how to establish stochastic epidemic models by considering environmental noises.

1.2 Deterministic Epidemic Models

Epidemics can be modelled by deterministic epidemic models, or compartmental models. Such a model usually divides the whole population suffering from a typical disease into different subgroups which represent different stages of infection. For example, a Susceptible-Infected-Removed (SIR) model is a very simple deterministic epidemic model with three different groups: "Susceptible" population are those individuals that are very likely to be infected by the disease; "Infected" individuals have been infected by disease and "Removed" group are those who have already been infected and recovered from the disease, granting permanent immunity. If we denote $S(t)$, $I(t)$ and $R(t)$ to be the three groups of populations at time t, this SIR model can be expressed in the following form:

$$\begin{cases} \frac{\mathrm{d}S(t)}{\mathrm{d}t} = \mu N - \mu S(t) - \beta I(t)S(t), \\ \frac{\mathrm{d}I(t)}{\mathrm{d}t} = \beta I(t)S(t) - \gamma I(t) - \mu I(t), \\ \frac{\mathrm{d}R(t)}{\mathrm{d}t} = \gamma I(t) - \mu R(t), \end{cases} \tag{1.1}$$

with $S(t) + I(t) + R(t) = N$ for all $t \geq 0$, where N is the total population size with a per capita death rate μ. This model was firstly defined by Kermack and McKendrick in 1927 and hence it is also called the Kermack-McKendrick model [25]. The following assumptions are made in this model [26]:

- Firstly, βN is the number of individuals that an other average member in the population has enough contact with to transmit disease in a unit of time. Thus, β can be regarded as the disease transmission coefficient, representing how fast the disease is developing.
- Secondly, γ is the number of individuals who get cured and leave the infected population. Note that those individuals who have been cured would never come back to the susceptible group. This indicates the permanent immunity of the recovery. Consequently, γ is usually considered as the rate at which infected individuals become cured.
- Also, there is no entry into or departure from the whole population. The birth rate and the death rate are the same.

As a result, it is clear from the SIR model (1.1) assumptions and definitions that the disease is transmitting to develop with some certain speed, while measures are taken at the same time to eliminate the threat with another rate, which means there must be a threshold to determine the outbreak or extinction of the disease. This is decided by the basic reproduction number [27] of an epidemic model, which is usually denoted by R_0^D [28]. In the SIR model, $R_0^D = \frac{\beta N}{\mu+\gamma}$ and when $R_0^D < 1$, the disease can never cause a proper epidemic outbreak and will die out in finite time. On the contrary, if $R_0^D > 1$, the disease will keep persisting in the model [29]. Obviously, the basic reproduction number is extremely important in the study of epidemic models.

However, the SIR model is very restrictive. It is only suitable for disease like chickenpox, measles and mumps etc [30, 31, 32, 33], in which those individuals who have recovered from infections are no longer considered to be susceptible. Clearly, the SIR model only represents a small amount of diseases, while we still need an epidemic model that can describe diseases without protective immunity. A

susceptible-infected-susceptible (SIS) model would be more reasonable and practical in such cases, that individuals who are cured from the disease will be immediately included in the susceptible group. This model is defined as follows:

$$\begin{cases} \frac{\mathrm{d}S(t)}{\mathrm{d}t} = \mu N - \beta S(t)I(t) + \gamma I(t) - \mu S(t), \\ \frac{\mathrm{d}I(t)}{\mathrm{d}t} = \beta S(t)I(t) - (\mu + \gamma)I(t). \end{cases} \tag{1.2}$$

$S(t)$ and $I(t)$ are susceptible and infected population, where $S + I = N$ for all $t \geq 0$ and N is the total size of population. Similarly, μ is the per capita death rate, γ is the rate at which infected individuals become cured and β is the disease transmission coefficient.

There are many diseases that are possible to be explained by the SIS model. As a result, SIS epidemic models are studied in many excellent work. For example, Hethcote and Yorke [34] found the basic reproduction number $R_0^D = \frac{\beta N}{\mu+\gamma}$ of the deterministic SIS model. When $R_0^D \leq 1$, the number of infected individuals tends to zero if the time $t \to \infty$, which indicates the eradication of the disease. Otherwise, when $R_0^D > 1$, the number of infected individuals tends to a positive number $N(1 - \frac{1}{R_0^D})$ when $t \to \infty$, which means the disease will maintain in the population system without an outbreak. Based on these results, a stochastic SIS model without demography (the birth and death rate in SIS model) is then defined to describe gonorrhea [35], which is a sexually transmitted disease. Their assumption indicates that the population is homogeneous because there is no entry and leave in the total population size. Hence it is suitable for homosexual population to explain the transmission. However, other researches on gonorrhea such as [36, 37, 38] focus on non-homogenous population which is more practical and reasonable.

There is also a special example of using an SIS model to explain transmission of disease. Luo and Tay [39] explained the spread of computer virus in a computer network by using the SIS epidemic model combined with Graph Theory. Their research target is to find the infection source in the network and this can be formulated as a maximum likelihood (ML) estimation problem by treating the only infected node s^* at the beginning and time t as parameters to be estimated. This model is not only suitable to figure out the threat in cyber security [40, 41], but can also be used to describe the spread of ideas and opinion in social media

such as Twitter [42].

Clearly, deterministic SIS and SIR models are very easy to analyse, while susceptible, infected and recovered stages are not all the stages for some diseases that have complicated biological mechanics. Hence sometimes $M(t)$ and $E(t)$ populations are introduced into the SIS epidemic models to fully complete the process of transmission [43]. Here $M(t)$ are those individuals who have passive immunity at time t which is usually provided by maternal antibodies. Once those individuals lose the immunity, they immediately become susceptible to the disease. Another subgroup is $E(t)$, which are those individuals who have been exposed to the disease but are not yet infectious at time t. This population represents the incubation stage of the disease. It is suitable for modelling a disease with long incubation period, such as HIV, to consider an exposed stage in the epidemic model [43, 44]. Moreover, some epidemic models are not simply derived from SIR or SIS models. For instance, Greenhalgh and Hay [45] established a mathematical model for the spread of HIV and AIDS amongst people who inject drugs. This model is based on a model created by Kaplan [46]. Greenhalgh and Hay made assumptions to improve the model, which is consequently more practical and realistic.

From previous work of deterministic epidemic models, it is obvious that those mathematical models focus on using parameters to measure the extent of disease toxicity and effectiveness of corresponding actions. Different equations are used to represent different stages during the spread of disease. However, the deterministic epidemic models are not able to reflect the influence of environment, while environment is one of the four crucial factors of studying disease behaviour. As environment has potentially great impact in ecology, biology and epidemiology research [47, 48, 49], we are going to introduce environmental noises in the following section. Then we will show some examples of stochastic epidemic models, derived from deterministic models with different types of environmental noises.

1.3 Environmental Noises

Population systems are significantly influenced by the random variation from the environment [50]. Changes of environments usually have corresponding effects in the transmission of disease. For example, a global temperature increase of $2-3^{\circ}C$ increases the number of people who catch malaria by approximately $3-5\%$ according to the research from WHO [51]. Moreover, Patz *et al.* [52] mention that the alternations in global climate and the changes in the geographic environment have negative effects in natural ecosystems, which increase the chance of exposure to parasite disease. The disturbance in the environment can be described as environmental noises and environmental noise can be found in all levels of biology, from molecular, sub-cell processes to the dynamics of immunity system in the human body and the whole population [53].

There are two different types of environmental noises that are usually considered in endemic problems. White noise is a very common noise, which is often described as a random signal with constant intensity at different frequency. It is widely used in fields such as physics, mathematics, biology and telecommunications. A sample of finite time white noise is a random variable with a normal distribution of zero mean and finite variance. This makes white noises easy to analyse. White noise can be regarded as the time derivative of a Brownian motion or Wiener process [54], which is independent at different times and could cause large fluctuation in the model. Although all Brownian motion sample paths are nowhere differentiable, the Itô stochastic integration theory with reference to Brownian motion is well-built by K. Itô [55]. A white noise is generally introduced in epidemic models by considering a perturbation [56] on a certain parameter, which makes this parameter no longer a certain value, but a random variable with normal distribution.

Telegraph noise is another type of noise that has completely different mathematical mechanics. Telegraph noise is also named as burst noise. In physics, burst noise is a type of electronic noise which can be found in semiconductors and ultra-thin gate oxide films [57]. It consists of instant transitions between two or more different levels of voltages. The switching time is random. Clearly, telegraph

noise is also a stochastic process and is commonly modelled by using continuous-time or discrete-time Markov chain [58]. A Markov chain is a stochastic model which is named after the Russian mathematician Andrey Markov [59]. There are many examples of using Markov chains in mathematical biology to emphasize the switching in the model, especially in population dynamics. In epidemic models, telegraph noise is also introduced to indicate that the transmission of disease is influenced by the switch among different environments or regimes [60]. For instance, the Leslie matrix [61] is an application of a Markov chain in modelling of population growth. There are also many other useful and crucial properties of a Markov chain, which we will be discussing in the next chapter.

It is obvious that in some cases, multiple noises can be considered in the epidemic models. For example, a finite number of independent Brownian motions can be used to introduce the disturbance of multiple independent, or correlated white noises. Moreover, regime switching [62] is a more general case which includes both white noises and telegraph noise in an epidemic model. In stochastic modelling, this is simply completed by considering a finite-dimensional Brownian motion and a Markov chain in the deterministic model. Note that usually the Markov chain and Brownian motion are assumed to be independent.

As we have given a brief introduction to the environmental noises in epidemic model, we are going to present examples of stochastic epidemic models. White noise, telegraph noise and regime switching cases will be explained in detail, followed by some well-known results in previous research.

1.4 Stochastic Epidemic Models

In this section, we will give some examples on how to establish stochastic epidemic models by introducing noises in deterministic epidemic models. Firstly, let us have a look at the white noise cases in stochastic epidemic models. Recall the SIR model

(1.1):

$$\begin{cases} \frac{\mathrm{d}S(t)}{\mathrm{d}t} = \mu N - \mu S(t) - \beta I(t)S(t), \\ \frac{\mathrm{d}I(t)}{\mathrm{d}t} = \beta I(t)S(t) - \gamma I(t) - \mu I(t), \\ \frac{\mathrm{d}R(t)}{\mathrm{d}t} = \gamma I(t) - \mu R(t). \end{cases}$$

Tornatore *et al.* [63] analysed the stability of a stochastic SIR model with white noise by replacing β with a perturbation of β : $\tilde{\beta}\mathrm{d}t = \beta\mathrm{d}t + \sigma\mathrm{d}B(t)$. Here $B(t)$ is a one-dimensional Brownian motion. Thus the SIR model becomes a stochastic SIR model with Brownian motion:

$$\begin{cases} \mathrm{d}S(t) = (\mu N - \mu S(t) - \beta I(t)S(t))\mathrm{d}t - \beta I(t)S(t)\mathrm{d}B(t), \\ \mathrm{d}I(t) = (\beta I(t)S(t) - \gamma I(t) - \mu I(t))\mathrm{d}t + \beta I(t)S(t)\mathrm{d}B(t), \\ \mathrm{d}R(t) = (\gamma I(t) - \mu R(t))\mathrm{d}t. \end{cases}$$

Results show that if $0 < \beta N < (\mu+\gamma-\frac{\sigma^2}{2})\wedge 2\mu$ then the disease-free equilibrium $E_0 = (N, 0, 0)$ is stable. This means the system will be free from infection and the disease will die out. Also, their computer simulations suggest that if $(\mu + \gamma - \frac{\sigma^2}{2}) \wedge 2\mu < \beta N < \mu + \gamma + \frac{\sigma^2}{2}$, the disease will also die out. Ji *et al.* [64] studied this model in further. They stated that such a stability is exponentially mean-square stable. They also pointed out that when $\beta N > \mu + \gamma$, then the solution of the stochastic SIR model will oscillate around a positive level, which indicate that the disease will not die out but prevail in the population. This will happen when the white noise is considered small enough. They then expanded the results to a multi-group stochastic epidemic model [65]. However, according to Ji *et al.*, they wish to find a threshold in stochastic SIR mode which is similar to the basic reproduction number R_0^D. Hence in 2014, Ji and Jiang [64] reviewed the stochastic SIR model and defined the stochastic reproduction number as

$$R_0^S = R_0^D - \frac{\sigma^2 N^2}{2(\mu+\gamma)} = \frac{\beta N}{\mu+\gamma} - \frac{\sigma^2 N^2}{2(\mu+\gamma)}.$$

Clearly, the stochastic reproduction number is smaller than the basic reproduction number in the deterministic SIR model, which to some extent proves that white noises have positive impact on the epidemic models. Based on these research, various of stochastic versions of (1.1) [66, 67] are further discussed.

Similarly, based on the research of Tornatore *et al.* [63] and a generalized study of stochastic SIRS (susceptible-infected-removed-susceptible) model by Lu [68], Gray *et al.* [1] used the same perturbation in a deterministic SIS model to establish a stochastic SIS model:

$$\mathrm{d}I(t) = [\beta(N - I(t))I(t) - (\mu + \gamma)I(t)]\mathrm{d}t + \sigma I(t)(N - I(t))\mathrm{d}B(t).$$

They fully explained the perturbation in both a biological way and a mathematical way to prove that introducing perturbation in a deterministic SIS model is biologically reasonable. Then they analysed the long-term behaviour of the solution. The stochastic reproduction number in their model is not changed $R_0^S = \frac{\beta N}{\mu+\gamma} - \frac{\sigma^2 N^2}{2(\mu+\gamma)}$ and it is still the threshold between extinction and persistence of the disease. They firstly pointed out that when $R_0^S < 1$ and with either $\sigma^2 \leq \frac{\beta}{N}$, or $\sigma^2 > \frac{\beta}{N} \vee \frac{\beta^2}{2(\mu+\gamma)}$, the disease will die out with probability one. Moreover, the computer simulation results illustrated that when $R_0^S < 1$ and $\frac{\beta}{N} < \sigma^2 \leq \frac{\beta^2}{2(\mu+\gamma)}$, the system will also be disease-free, though they do not have a proof. On the other hand, when $R_0^S > 1$, the disease will prevail in the system around a positive level with probability 1. Also, they proved that there is a unique stationary distribution under this circumstance. Explicit expressions of mean and variance are then derived. This is a very interesting result. It indicates that the introduction of white noise in the deterministic model expands the threshold of extinction. For those parameters that will not result in disease-free equilibrium in the deterministic model, it becomes possible in the stochastic model. Obviously, this is another example of the introduction of white noise, which changes the original basic reproduction number in the deterministic model into a stochastic reproduction number, and potentially stabilizes an otherwise unstable system [1]. Now we want to show some telegraph noise cases in stochastic epidemic models.

Based on Takeuchi's research in 2006 [69] of introducing telegraph noise in Lotka-Volterra model, Gray *et al.* [5] used a two-state Markov chain to represent the switching between two environments in deterministic SIS model. Hence they replaced the white noise epidemic SIS model (1.3) with a telegraph noise epidemic SIS model:

$$\frac{\mathrm{d}I(t)}{\mathrm{d}t} = I(t)[\alpha_{r(t)} - \beta_{r(t)}I(t)], \tag{1.3}$$

where $r(t)$ is a Markov chain with state space $\mathbb{S} = \{1, 2\}$ and generator

$$\Gamma = \begin{pmatrix} -v_{12} & v_{12} \\ v_{21} & -v_{21} \end{pmatrix},$$

and $(\pi_1, \pi_2) = (\frac{v_{21}}{v_{12}+v_{21}}, \frac{v_{12}}{v_{12}+v_{21}})$ is the stationary distribution of the Markov chain. Note that in order to make the model easy to analyse, they let $\alpha_i = \beta_i N - \mu_i - \gamma_i$, $i \in \mathbb{S} = \{1, 2\}$, which takes different values in two states with reference to different environments. They firstly defined the stochastic reproduction number R_0^S. However, this stochastic reproduction number is very complex which they do not pursue. As a result, they defined a threshold to analyse extinction and persistence:

$$T_0^S = \frac{\pi_1\beta_1 N + \pi_2\beta_2 N}{\pi_1(\mu_1 + \gamma_1) + \pi_2(\mu_2 + \gamma_2)}.$$

And it is easy to see

- $T_0^S < 1$ if and only if $\pi_1\alpha_1 + \pi_2\alpha_2 < 0$;
- $T_0^S = 1$ if and only if $\pi_1\alpha_1 + \pi_2\alpha_2 = 0$;
- $T_0^S > 1$ if and only if $\pi_1\alpha_1 + \pi_2\alpha_2 > 0$,

which is simple to view. They then pointed out that when $T_0^S < 1$, the disease will die out in probability one; when $T_0^S > 1$, the disease will prevail in the system around a positive level almost surely. These results clearly indicate that the introduction of telegraph noise also expands the condition of disease-free equilibrium. Model (1.3) is actually two different deterministic SIS models connected by Markov switching, which has the basic reproduction number $R_{0_i}^D = \frac{\beta_i N}{\mu_i+\gamma_i}$ for all state i. In deterministic SIS models, we strictly need every $R_{0_i}^D \leq 1$ to cause the elimination of disease in every state. However, conditions in Gray *et al.*'s work only need $\pi_1\alpha_1 + \pi_2\alpha_2 < 0$. This means, after introducing telegraph noise in a deterministic SIS model, we do not need all states go to extinction to have a disease-free equilibrium. In some states, the disease can persist around a certain positive levels, while the average-type condition is still satisfied, which results in the elimination of the disease from a global point of view.

Furthermore, based on [5], Greenhalgh *et al.* [60] extended the results in a stochastic SIRS model. The same threshold is defined and used to determine the extinction and persistence of the solution. Similar results are obtained to prove that telegraph noise is a potentially positive factor to extinction of the disease.

Now we want to state some examples of stochastic epidemic models with regime switching. In these models, both white noise and telegraph noise are introduced, while noises are usually assumed to be independent. For example, Luo and Mao [70] introduced white noise and telegraph noise in the Lotka-Volterra model. However, they studied the ultimate boundedness of the solution, while Li *et al.* [71] analysed the Lyapunov function in the stochastic Lotka-Volterra model, which is developed by Khasminskii [72], in order to explain stochastic permanence. They clearly pointed out that permanence in the overall behaviour does not need permanence in every state. In some states, the solution can even become extinct. Based on [70, 71], Liu *et al.* [73] focused on the ergodic property, recurrence and the stationary distribution of the solution in a Lotka–Volterra system with pollination mutualism [74, 75, 76, 77]. Computer simulation illustrates the fluctuation in the solution, while the integral average converges to a fixed point, which supports the recurrence and a stationary distribution in their theory.

Besides, in some stochastic epidemic research, there are other properties in the population system that are analysed mathematically. For instance, a stochastic population system with delay [78] is often used to describe the time delay of a certain event. Population dynamics in the forest can be a very good example. A cut forest may take more than 20 years to reach the maturity of harvesting. Hence in such cases, time delay must be built in the stochastic model. In epidemic problems, time delay usually occurs when a recovered individual will not be infectious until a period of time after becoming infected [79, 80]. Moreover, in some research, a different noise, Lévy noise [81, 82], is used to emphasize jumps of sudden events such as earthquake and hurricane in the environment [83, 84]. It can be mathematically described by using Lévy process, which is a more general process than Brownian motion. It is not only used in epidemic models, but widely applied in stochastic financial modelling such as [85, 86]. Another approach in epidemic models is to mathematically disturb the transmission of disease with medical strategy.

Vaccination is one of the best methods to prevent an outbreak of disease and it is commonly introduced in the study of epidemic models. Li and Ma [87] used $V(t)$ to represent those individuals that are immune to an infection at time t as the result of vaccination in a stochastic SIS model. They also clearly stated in the assumptions of the model that the proportion of the vaccinated individuals is a constant q, with a probability function $Q(t)$ as the probability of vaccinated individuals that still carry the immunity before returning to the susceptible group at time t. Zhao and Jiang [88] also studied the threshold R_0^S of this model in 2014, while Lin and Jiang [89] focused on finding the stationary distribution of this stochastic SIS model with vaccination.

Here we complete a brief introduction to this research and clearly, the abundant previous work has built a firm foundation in studying stochastic epidemic models. Now let us summarize some common interests in research of stochastic epidemic models, which are also consequently our research targets. We want to know:

- if there exists a unique solution of the given model. This is the initial requirement of studying a stochastic differential equation, which is also reasonable in epidemic study;
- if the solution is always positive, or non-negative. Clearly, solutions that will go below zero are meaningless in representing the number of populations;
- if the solution is bounded. We usually want our solution to be bounded within the number of total population size. Obviously, solutions that will exceed N are also meaningless. As we usually assume that there is no entry in the epidemic model, this is will cause contradiction;
- under what circumstances, the solution will persist, or become extinct. This is of great importance in studying stochastic epidemic models. We aim to find a threshold, or stochastic reproduction number R_0^S of our models to determine the extinction and persistence of the disease;
- if there is a stationary distribution of the stochastic model. This is usually examined when the disease persists.

Obviously, stochastic epidemic models are not fully explored. For instance, in the study of a stochastic SIS model such as [1], only β is considered to be disturbed by white noise and then perturbed. However, in real cases, it is more likely that all parameters are influenced. Moreover, Gray *et al.* emphasized the importance of using perturbation on β to study the transmission behaviour of the diseases, while $\mu + \gamma$ is the recovery rate and death rate in the system, which can represent the removal of diseases in the total population. Gou and Jin [90] mentioned that a smaller recovery rate in the population system keeps infected individuals longer in that state after they catch the disease, which potentially undermines the resistance to epidemics in the whole population. Hence it is meaningful to study the noise disturbance in $\mu + \gamma$. However, there is no work focusing on introducing a perturbation on the parameter $\mu + \gamma$. Thus, this research aims to fill the gap in stochastic SIS epidemic modelling, which will start from introducing another white noise on the parameter $\mu + \gamma$ based on [1] in SIS model. Moreover, the new perturbation on $\mu + \gamma$ has a square-root diffusion coefficient related to number of susceptible individuals. This is caused by our assumption of estimation, which is inspired by the square root process [91]. And this will be our first model in Chapter 3, which can be regarded as a generalization of Gray *et al.*'s work [1] and it has been published in JMAA (Journal of Mathematical Analysis and Applications) in 2019. In this chapter, we will firstly prove the uniqueness and boundedness of the solution, followed by giving conditions of extinction and persistence. Also, a unique stationary distribution is stated with its mean and variance. Examples are performed using computer software to illustrate our results.

In Chapter 4 and Chapter 5, we will discuss this model further in two different directions. In Chapter 4, we will consider the correlation between two white noises, which is a more generalized case in real epidemic problems but there is little previous work discussing the correlation in SIS model. It is also a published work in Nonlinear Dynamics in 2019. However, from the results we can confirm that introducing correlation does have a positive impact such as expanding the conditions of extinction, while on the other hand, we fail to derive the explicit expression of mean and variance of the stationary distribution. In Chapter 5, we will combine [5] with our first model to formulate a stochastic SIS model with regime switching to discuss the impact of telegraph noise. A finite state Markov

chain is considered to represent the switching among different environments in our model. Results in this chapter are very different, which focuses on the overall behaviour of the solution instead of solutions in different separate states.

Before we introduce and establish our models, we want to firstly walk through the basic stochastic theory, including definitions of probability space, Itô integrals, stochastic process and stochastic differential equations.

Chapter 2

Stochastic Theory

In order to make this research self-contained, we shall generally review the basic knowledge of probability theory and stochastic process, following by the definition of Brownian motions and Markov chains. Important properties are introduced and explained. Then we proceed to state the well-known Itô's formula, as well as a generalised Itô's formula. Stochastic differential equations are then defined, followed by a well-known theorem on stationary distribution.

2.1 Basic Notations of Probability Theory

Firstly, if we assume the possible outcomes, in other words, a set of elementary event as Ω, then the set of only observable or interesting events $\mathcal{F} \subset \Omega$ should have the following properties:

- $\emptyset \in \mathcal{F}$, where $\emptyset$ is the empty set.
- $A \in \mathcal{F} \Rightarrow A^C \in \mathcal{F}$, where $A^C = \Omega - A$ is the complement of A in Ω.
- $\{A_i\}_{i \geq 1} \subset \mathcal{F} \Rightarrow \cup_{i=1}^{\infty} A_i \subset \mathcal{F}$.

A family of $\mathcal{F}$ with these three conditions is called a *σ-algebra*. The pair of $(\Omega, \mathcal{F})$ is called a *measurable space*, and the elements of $\mathcal{F}$ are called *$\mathcal{F}$-measurable sets*

instead of events. If $\mathcal{C}$ is a family of subsets of Ω, then there exists a smallest *σ-algebra* $\sigma(\mathcal{C})$ on Ω which contains $\mathcal{C}$. This $\sigma(\mathcal{C})$ is called the *σ-algebra generated by* $\mathcal{C}$. If $\Omega = R^d$ and $\mathcal{C}$ is the family of all open sets in R^d, then $\mathcal{B} = \sigma(\mathcal{C})$ is called the *Borel σ-algebra* and the elements of $\mathcal{B}^d$ are called the *Borel sets*.

A real-valued function $X : \Omega \to R$ is said to be *$\mathcal{F}$-measurable* if

$$\{\omega : X(\omega) \leq a\} \in \mathcal{F} \text{ for all } a \in R.$$

The function X is also called a real-valued *$\mathcal{F}$-measurable random variable*. An R^d-valued function $X(\omega) = (X_1(\omega), ..., X_d(\omega))$ is said to be *$\mathcal{F}$-measurable* if all the elements X_i are *$\mathcal{F}$-measurable*. Similarly, a $d \times m$-matrix-valued function $X(\omega) = (X_{ij}(\omega))_{d\times m}$ is said to be *$\mathcal{F}$-measurable* if all the elements X_{ij} are *$\mathcal{F}$-measurable*.

A *probability measure* P on a measurable space $(\Omega, \mathcal{F})$ is a function $P : \mathcal{F} \to [0, 1]$ such that

- $P(\Omega)$=1;
- for any disjoint sequence $\{A_i\}_{i\geq 1} \subset \mathcal{F}$ $(A_i \cap A_j = \emptyset \text{ if } i \neq j)$

$$P(\bigcup_{i=1}^{\infty} A_i) = \sum_{i=1}^{\infty} P(A_i).$$

The triple $(\Omega, \mathcal{F}, P)$ is called a *probability space*. If X is a real-valued random variable and is *integrable* with respect to the probability measure P, then the integral

$$\mathbb{E}(X) = \int_{\Omega} X(\omega) \mathrm{d}P(\omega),$$

is called the *expectation* of X. Also,

$$\mathbb{V}\mathrm{ar}(X) = \mathbb{E}(X - \mathbb{E}(X))^2,$$

is called the *variance* of X. And $\mathbb{E}|X|^p$ is called the pth moment of X. Also if X is an R^d-valued random variable, then X induces a probability measure μ_X on the Borel measurable space $(R^d, \mathcal{B}^d)$, which is defined by

$$\mu_X(B) = P\{\omega : X(\omega) \in B\} \text{ for } B \in \mathcal{B}^d,$$

and μ_X is called the distribution of X. The expectation of X can be written as

$$\mathbb{E}(X) = \int_{R^d} x \mathrm{d}\mu_X(x).$$

More generally, if $g : R^d \to R^m$ is Borel measurable, we then have the *transformation formula*

$$\mathbb{E}\, g(X) = \int_{R^d} g(x) \mathrm{d}\mu_X(x).$$

Now we want to introduce almost surely convergence as this will be crucial to our following research. If X and X_k, $k \geq 1$ are R^d-valued random variables, then if there exists a P-null set $\Omega_0 \in \mathcal{F}$ such that for every $\omega \notin \Omega_0$, the sequence $\{X_k(\omega)\}$ converges to $X(\omega)$ in the usual sense in R^d, then $\{X_k\}$ is said to converge to X *almost surely* or *with probability 1*, and we write $\lim_{k\to\infty} X_k = X$ a.s.

2.2 Stochastic Processes

Let $(\Omega, \mathcal{F}, P)$ be a probability space. A *filtration* $\{\mathcal{F}_t\}_{t\geq 0}$ is an increasing sequence of sub-σ-algebras of $\mathcal{F}$, where $\mathcal{F}_t \subset \mathcal{F}_s \subset \mathcal{F}$ for all $0 \leq t < s < \infty$. Then the filtration is said to be *right continuous* if $\mathcal{F}_t = \bigcap_{s>t} \mathcal{F}_s$ for all $t > 0$. When the probability space is complete, the filtration is said to satisfy the *usual conditions* if it is right continuous and $\mathcal{F}_0$ contains all P-null sets.

Throughout this thesis, unless otherwise specified, we let $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geqslant 0}, P)$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ satisfying the usual conditions. We also define $\mathcal{F}_\infty = \sigma(\bigcup_{t\geq 0} \mathcal{F}_t)$, i.e. the σ-algebra generated by $\bigcup_{t\geq 0} \mathcal{F}_t$. Now let us define the stochastic process.

A family $\{X_t\}_{t\in I}$ of R^d-valued random variables is called a *stochastic process* with *parameter set* I and *state space* R^d. The parameter set I is usually defined as $R_+ = [0, \infty)$. If $\{X_t\}_{t\geq 0}$ is an R^d-valued stochastic process, it is *continuous* if for almost all $\omega \in \Omega$, $X_t(\omega)$ is continuous on $t \geq 0$. Also it is said to be *integrable* if for every $t \geq 0$, X_t is and integrable random variable. It is said to be $\{\mathcal{F}_t\}$-adapted if for every t, X_t is $\mathcal{F}$-measurable.

A *stopping time* is usually used in studying stochastic processes. A random variable $\tau : \Omega \to [0, \infty]$ (it may take the value ∞) is called an $\{\mathcal{F}_t\}$-stopping time if $\{\omega : \tau(\omega) < t\} \in \mathcal{F}_t$ for any $t \geq 0$.

Now we want to introduce a special stochastic process. An R^d-valued $\{\mathcal{F}_t\}$-adapted integrable process $\{M_t\}_{t\geq 0}$ is called a *martingale with respect to* $\{\mathcal{F}_t\}$ or simply, martingale, if

$$\mathbb{E}\,(M_t|\mathcal{F}_s) = M_s \quad \text{ a.s. for all } 0 \leq s < t < \infty.$$

Here we want to specific some properties of martingale. Firstly we introduce the Doob martingale stopping theorem.

Theorem 2.2.1 (Doob martingale stopping theorem)**.** *Let $\{M_t\}_{t\geq 0}$ be an R^d-valued martingale with respect to $\{\mathcal{F}_t\}$, and let θ, ρ be two finite stopping times. Then*

$$\mathbb{E}\,(M_\theta|\mathcal{F}_\rho) = M_{\theta\wedge\rho} \quad \text{ a.s.}$$

In particular, if τ is a stopping time, then

$$\mathbb{E}\,(M_{\tau\wedge s}|\mathcal{F}_s) = M_{\tau\wedge s} \quad \text{ a.s.}$$

for all $0 \leq s < t < \infty$.

A stochastic process $\{X_t\}_{t\geq 0}$ is called *square-integrable* if $\mathbb{E}\,|X_t|^2 < \infty$ for every $t \geq 0$. If $M = \{M_t\}_{t\geq 0}$ is a real-valued square-integrable continuous martingale, then there exists a unique continuous integrable adapted increasing process denoted by $\{\langle M, M\rangle_t\}$ such that $\{M_t^2 - \langle M, M\rangle_t\}$ is a continuous martingale vanishing at $t = 0$. $\{\langle M, M\rangle_t\}$ is called the *quadratic variation* of M. Particularly, if τ is a finite stopping time, we have

$$\mathbb{E}\,M_\tau^2 = \mathbb{E}\,\{\langle M, M\rangle_\tau\}.$$

Furthermore, A right continuous adapted process $\{M_t\}_{t\geq 0}$ is called a *local martingale* if there exists a non-decreasing sequence $\{\tau_k\}_{k\geq 1}$ of stopping times with $\tau_k \uparrow \infty$ a.s. such that every $\{M_{\tau\wedge t} - M_0\}_{t\geq 0}$ is a martingale. From the Doob martingale stooping theorem, it is easy to see that every martingale is a local martingale but the converse is not true. Now based on these definitions we give the strong law of large numbers for martingale.

Theorem 2.2.2 (Strong Law of large numbers)**.** *Let $M = \{M_t\}_{t\geq 0}$ be a real-valued continuous local martingale vanishing at $t = 0$. Then*

$$\lim_{t\to\infty}\{\langle M, M\rangle_t\} = \infty \quad a.s. \quad \Rightarrow \lim_{t\to\infty} \frac{M_t}{\{\langle M, M\rangle_t\}} = 0 \quad a.s.$$

Also,

$$\limsup_{t\to\infty} \frac{\{\langle M, M\rangle_t\}}{t} < \infty \quad a.s. \qquad \Rightarrow \lim_{t\to\infty} \frac{M_t}{t} = 0 \quad a.s.$$

2.3 Brownian Motions

In this section we firstly give the definition of a Brownian motion. The term Brownian motion is used to describe the random motion of particles suspended in a fluid (a liquid or a gas) resulting from their collision with the fast-moving molecules in the fluid. This was initially observed by Robert Brown in 1827 [92, 93]. It is not only used in mathematical problems, but also widely used in physics, botany and biology.

Now let $(\Omega, \mathcal{F}, P)$ be the probability space with the filtration $\{\mathcal{F}_t\}_{t\geq 0}$. A standard one-dimensional Brownian motion is a real-valued continuous $\{\mathcal{F}_t\}$-adapted process $\{B_t\}_{t\geq 0}$ with the following properties:

- $B_0 = 0$ a.s.;
- for $0 \leq< s < t < \infty$, the increment $B_t - B_s$ is normally distributed with mean 0 and variance $t - s$;
- for $0 \leq< s < t < \infty$, the increment $B_t - B_s$ is independent of $\mathcal{F}_s$.

Also, if $\{B_t\}_{t\geq 0}$ is a Brownian motion and $0 \leq t_0 < t_1 < ... < t_k < \infty$, then the increments $B_{t_i} - B_{t_{i-1}}$, $1 \leq i \leq k$ are independent and we say that the Brownian motions has *independent increments*. Clearly, the distribution of $B_{t_i} - B_{t_{i-1}}$ only relies on $t_i - t_{i-1}$ and we say that the Brownian motion has stationary increments.

Although sometimes the probability space does not need to be complete, we still assume our Brownian motions in this paper are defined on a complete prob-

ability space $(\Omega, \mathcal{F}, P)$ with the filtration $\{\mathcal{F}_t\}_{t\geq 0}$ satisfying usual conditions. The following list states some of the most important properties of the Brownian motion:

- $\{B_t\}$ is a continuous square-integrable martingale and its quadratic variation $\{\langle B, B\rangle_t\} = t$ for all $t \geq 0$;
- From the strong law of large number, we have
$$\lim_{t\to\infty} \frac{B_t}{t} = 0;$$
- For almost every $\omega \in \Omega$, the Brownian motion sample path $B(\omega)$ is nowhere differentiable;
- For almost every $\omega \in \Omega$, the Brownian motion sample path $B(\omega)$ is locally Hölder continuous [94] with exponent δ if $\delta \in (0, \frac{1}{2})$. However, for almost every $\omega \in \Omega$, the Brownian motion sample path $B(\omega)$ is nowhere Hölder continuous with exponent $\delta > \frac{1}{2}$;
- $\{-B_t\}$ is a Brownian motion with respect to the filtration $\{\mathcal{F}_t\}$;
- Let $c > 0$. Define
$$X_t = \frac{B_{ct}}{\sqrt{c}} \quad \text{for } t \geq 0.$$
Then $\{X_t\}$ is a Brownian motion with respect to filtration $\{\mathcal{F}_{ct}\}$.

Similarly, we can define a d-dimensional Brownian motion $\{B_t = (B_t^1, B_t^2, ..., B_t^d)\}_{t\geq 0}$ if every $\{B_t^i\}$ is a one-dimensional Brownian motion, and $\{B_t^i\}$, ... , $\{B_t^d\}$ are independent.

2.4 Stochastic Integral

In this section we will introduce the establishment of the Itô stochastic integral:

$$\int_0^t f(s)dB(s).$$

This integral was firstly defined by K. Itô in 1949 [55]. We will introduce the definition first, followed by some properties of stochastic integral.

A real-valued stochastic process $g = \{g(t)\}_{a\le t\le b}$ is called a simple process if there exists a partition $a = t_0 < t_1 < t_2 < ... < t_k = b$ of $[a, b]$, and bounded random variables ξ_i, $0 \le i \le k-1$ such that ξ_i is $\{\mathcal{F}_{t_i}\}$-measurable and

$$g(t) = \xi_0 \mathbf{1}_{[t_0,t_1]}(t) + \sum_{i=1}^{k-1} \xi_i \mathbf{1}_{(t_i,t_{i+1}]}(t).$$

Denote by $\mathcal{M}_0([a, b]; R)$ the family of all such processes and clearly $\mathcal{M}_0([a, b]; R) \subset \mathcal{M}^2([a, b]; R)$. Now we give the definition of the Itô integral for such simple processes.

For a simple process g in $\mathcal{M}_0([a, b]; R)$, define

$$\int_a^b g(t)\mathrm{d}B_t = \sum_{i=0}^{k-1} \xi_i (B_{t_{i+1}} - B_{t_i}),$$

and call it the stochastic integral of g with respect to the Brownian motion $\{B_t\}$ or the Itô integral. Using the approximation result, this can be extended from only simple process to processes in $\mathcal{M}^2([a, b]; R)$, which leads to the following definition.

Let $f \in \mathcal{M}^2([a, b]; R)$. The Itô integral of f with respect to $\{B_t\}$ is defined by

$$\int_a^b f(t)\mathrm{d}B_t = \lim_{n\to\infty} \int_a^b g_n(t)\mathrm{d}B_t \quad \text{in } L^2(\Omega; R),$$

where $\{g_n\}$ is a sequence of simple process such that

$$\lim_{n\to\infty} \mathbb{E} \int_a^b |f(t) - g_n(t)|^2 \mathrm{d}t = 0.$$

The stochastic integral has some important properties which we will use in later sections. Hence we summarize it here. If f, $g \in \mathcal{M}^2([a, b]; R)$, then

- $\int_a^b f(t)\mathrm{d}B_t$ is $\mathcal{F}_b$-measurable;
- $\mathbb{E} \int_a^b f(t)\mathrm{d}B_t = 0$;
- $\mathbb{E}\,|\int_a^b f(t)\mathrm{d}B_t|^2 = \mathbb{E} \int_a^b |f(t)|^2\mathrm{d}t$;
- $\int_a^b [\alpha f(t) + \beta g(t)]\mathrm{d}B_t = \alpha \int_a^b f(t)\mathrm{d}B_t + \beta \int_a^b g(t)\mathrm{d}B_t$.

2.5 Markov Process

In this section we are going to introduce some basic knowledge about a Markov process. A d-dimensional $\mathcal{F}_t$-adapted process $\{X_t\}_{t\geq 0}$ is called a *Markov process* if the following *Markov property* is satisfied: for all $0 \leq s \leq t < \infty$ and $A \in \mathcal{B}(R^d)$,

$$P(X(t) \in A|\mathcal{F}_s) = P(X(t) \in A|X(s)),$$

which is also equivalent to the following definition: for any bounded Borel measurable function: $\varphi : R^d \to R$ and $0 \leq s \leq t < \infty$,

$$\mathbb{E}\left(\varphi(X(t))|\mathcal{F}_s\right) = \mathbb{E}\left(\varphi(X(t))|X(s)\right).$$

The *transition probability* of the Markov process is a function $P(s, x; t, A)$, defined on $0 \leq s \leq t < \infty$, $x \in R^d$ and $A \in \mathcal{B}(R^d)$, which has the following properties:

- For every $0 \leq s \leq t < \infty$ and $A \in \mathcal{B}(R^d)$,

$$P(s, X(s); t, A) = P(X(t) \in A|X(s));$$

- $P(s, x; t, \cdot)$ is a probability measurable on $B(R^d)$ for every $0 \leq s \leq t < \infty$ and $x \in R^d$;

- $P(s, \cdot; t, A)$ is a Borel measurable for every $0 \leq s \leq t < \infty$ and $A \in \mathcal{B}(R^d)$;

- The Kolmogorov-Chapman equation

$$P(s, x; t, A) = \int_{R^d} P(u, y; t, A)P(s, x; u, \mathrm{d}y),$$

  holds for any $0 \leq s \leq u \leq t < \infty$, $x \in R^d$ and $A \in \mathcal{B}(R^d)$.

A stochastic process $X = \{X_t\}_{t\geq 0}$ which is defined on a probability space $(\Omega, \mathcal{F}, P)$ with values in a countable set Ξ (state space), is called a *continuous-time Markov Chain* if for any finite set $0 \leq t_1 < t_2 < ... < t_n < t_{n+1}$ of times, and corresponding set $i_1, i_2, ..., i_{n-1}, i, j$ of states in Ξ, such that $P\{X(t_n) = i, X(t_{n-1}) = i-1, ..., X(t_1) = i_1\} > 0$, we have

$$P\{X(t_{n_1}) = j|X(t_n) = i, X(t_{n-1}) = i-1, ..., X(t_1) = i_1\}$$

$$= P\{X(t_{n_1}) = j | X(t_n) = i\}.$$

If for all t and s such that $0 \leq s \leq t < \infty$ and all $i,\ j \in \Xi$ the conditional probability $P\{X(t) = j | X(s) = i\}$ depends only on $t - s$, then we say that the process $X = \{X_t\}_{t \geq 0}$ is *homogeneous*. In this case, we must have $P\{X(t) = j | X(s) = i\} = P\{X(t-s) = j | X(0) = i\}$, and the function

$$P_{ij}(t) := P\{X(t) = j | X(s) = i\},\ i, j \in \Xi, t \geq 0,$$

is called the *transition probability* of the process. We also define $\Gamma = (\nu_{ij})_{i,j \in \Xi}$ to be the *generator* of the Markov chain. If the state space is finite we can write it as $\mathbb{S} = \{1, 2, ..., N\}$, then the process is called a continuous-time finite Markov chain. We will discuss Markov chain switching in the following chapters, so in this research we will assume that all Markov chains are finite and all states are stable. For such a Markov chain, almost every sample path is right continuous step function. Moreover, if $P(t) = (P_{ij}(t))_{N \times N}$ is the transition probability matrix, and $\Gamma = (\nu_{ij})_{N \times N}$ is the generator of a finite N-state Markov chain [95], then

$$P(t) = e^{-t\Gamma}.$$

Furthermore, the generator $\Gamma = (\nu_{ij})_{N \times N}$ can be represented as a stochastic integral with respect to a Poisson random measure. Let Δ_{ij} be consecutive, left closed, right open intervals of the real line each having length ν_{ij} such that

$$\begin{aligned}
\Delta_{12} &= [0, \nu_{12}), \\
\Delta_{13} &= [\nu_{12}, \nu_{12} + \nu_{13}), \\
&\vdots \\
\Delta_{1N} &= \left[\sum_{j=2}^{N-1} \nu_{1j}, \sum_{j=2}^{N} \nu_{1j} \right), \\
\Delta_{21} &= \left[\sum_{j=2}^{N} \nu_{1j}, \sum_{j=2}^{N} \nu_{1j} + \nu_{21} \right), \\
\Delta_{23} &= \left[\sum_{j=2}^{N} \nu_{1j} + \nu_{21}, \sum_{j=2}^{N} \nu_{1j} + \nu_{21} + \nu_{23} \right), \\
&\vdots
\end{aligned}$$

$$\Delta_{2N} = \left[\sum_{j=2}^{N} \nu_{1j} + \sum_{j=1, j\neq 2}^{N-1} \nu_{2j}, \sum_{j=2}^{N} \nu_{1j} + \sum_{j=1, j\neq 2}^{N} \nu_{2j} \right),$$
$$\vdots$$

and so on. Now we define a function $h : \mathbb{S} \times R \to R$ by

$$h(i, y) = \begin{cases} j - i, & \text{if } y \in \Delta_{ij}, \\ 0, & \text{otherwise.} \end{cases}$$

Then

$$\mathrm{d}X(t) = \int_R h(X(t-), y)\gamma(\mathrm{d}t, \mathrm{d}y),$$

with initial condition $X(0) = i_0$, where $\gamma(\mathrm{d}t, \mathrm{d}y)$ is a Poisson random measure with density $\mathrm{d}t \times \mu(\mathrm{d}y)$, in which μ is the Lebesgue measure on R.

There are some very important definitions of properties in a Markov chain. For a Markov chain $X(t)$ with state space $\mathbb{S}$, a state $i \in \mathbb{S}$ is said to communicate with another state $j \in \mathbb{S}$ if i and j can switch to each other. A communicating class is a maximal set of states $S' \subset \mathbb{S}$ such that every pair of states in S' communicates with each other. A Markov chain is also said to be *irreducible* if the state space is a single communicating class, or in other words, it is irreducible if possible if any state in the state space can switch to any state.

Another crucial property of Markov chain is its recurrence. A state $i \in \mathbb{S}$ of Markov chain is said to be *transient* if there is a non-zero probability for starting from state i to never return to itself. Otherwise state i is said to be recurrent, which indicates that the mean recurrence time for state i is finite. If we define $\tau_i := \inf\{t > T_1 : X(t) = i\}$, where T_1 is the first jump time away from state i, then if

$$\mathbb{E}\,\tau_i < \infty,$$

we say that state i is positive recurrent.

The stationary distribution of a Markov chain is usually focused on in many different cases. If we denote $\pi = (\pi_1, ..., \pi_n)$ as the stationary distribution of the Markov chain $X(t)$, with finite state space $\mathbb{S}(t) = \{1, 2, ..., N\}$ and transition matrix $P(t) = (P_{ij}(t))_{N\times N}$ as well as the generator $\Gamma = (\nu_{ij})_{N\times N}$, then the stationary

distribution is defined as

$$\pi = \pi P(t) \quad \text{for all } t \geq 0,$$

or

$$\pi\Gamma = 0.$$

Based on the definition of irreducible, recurrence and stationary distribution, we now introduce the ergodicity of a Markov chain. The Markov chain $X(t)$ is said to be *ergodic* if it is irreducible and positive recurrent, and for any function $f : \mathbb{S} \to R$, we have

$$P\left\{\frac{1}{t}f(X(s))\mathrm{d}s \underset{t\to\infty}{\longrightarrow} \sum_{j\in\mathbb{S}} \pi_j f(j)\right\} = 1.$$

And this is the *ergodic property* [95]. Now we have fully introduced Brownian motions and Markov chains. In the next section, we are going to focus on building the Itô formula. A multi-dimensional Itô formula will be derived firstly, followed by a generalised Itô formula suitable for Markovian switching cases.

2.6 Itô's Formula

Based on the Itô integral definition in the last section, we are going to establish Itô's formula in this section. Firstly, we will derive the one-dimensional Itô's formula. Let $\{B_t\}_{t\geq 0}$ be a one-dimensional Brownian motion defined on the complete probability space $(\Omega, \mathcal{F}, P)$ adapted to the filtration $\{\mathcal{F}_t\}_{t\geq 0}$. Let $\mathcal{L}^1(R_+; R^d)$ denote the family of all R^d-valued measurable $\{\mathcal{F}_t\}$-adapted processes $f = \{f(t)\}_{t\geq 0}$ such that

$$\int_0^T |f(t)|\mathrm{d}t < \infty \qquad \text{a.s. for every } T > 0.$$

Moreover, a one-dimensional Itô process is a continuous adapted process $x(t)$ on $t \geq 0$ of the form

$$x(t) = x(0) + \int_0^t f(s)\mathrm{d}s + \int_0^t g(s)\mathrm{d}B_s,$$

where $f \in \mathcal{L}^1(R_+; R)$ and $g \in \mathcal{L}^2(R_+; R)$. We shall say that $x(t)$ has stochastic differential $\mathrm{d}x(t)$ on $t \geq 0$ given by

$$\mathrm{d}x(t) = f(t)\mathrm{d}t + g(t)\mathrm{d}B_t.$$

Now let $C^{2,1}(R^d \times R_+; R)$ denote the family of all real valued functions $V(x,t)$ defined on $R^d \times R_+$ such that they are continuously twice differentiable in x and once in t. If $V \in C^{2,1}(R^d \times R_+; R)$, we set

$$V_t = \frac{\partial V}{\partial t},\ V_x = \left(\frac{\partial V}{\partial x_1}, ..., \frac{\partial V}{\partial x_d}\right),\ V_{xx} = \left(\frac{\partial^2 V}{\partial x_i \partial x_j}\right)_{d\times d}.$$

Clearly when $V \in C^{2,1}(R \times R_+; R)$, we have $V_x = \frac{\partial V}{\partial x}$ and $V_{xx} = \frac{\partial^2 V}{\partial x^2}$, which is the one-dimensional case. Then $V(x(t), t)$ is again an Itô process with the stochastic differential given by

$$\begin{aligned}\mathrm{d}V(x(t),t) = [V_t(x(t),t) + V_x(x(t),t)f(t) + \frac{1}{2}V_{xx}(x(t),t)g^2(t)]\mathrm{d}t \\ + V_x(x(t),t)g(t)\mathrm{d}B_t.\end{aligned}$$

And this is the one-dimensional Itô's formula. However this is not enough for multi-dimensional problem. Thus we are going to expand the space and state the multi-dimensional Itô's formula. Hence now if we assume $f \in \mathcal{L}^1(R_+; R^d)$ and $g \in \mathcal{L}^1(R_+; R^{d\times m})$. Let $V \in C^{2,1}(R^d \times R_+; R)$ and $V(x(t), t)$ is also an Itô process with the stochastic differential given by

$$\begin{aligned}\mathrm{d}V(x(t),t) = [V_t(x(t),t) + V_x(x(t),t)f(t) + \frac{1}{2}trace(g^T(t)V_{xx}(x(t),t)g(t))]\mathrm{d}t \\ + V_x(x(t),t)g(t)\mathrm{d}B_t.\end{aligned}$$

Furthermore, if we let $r(t)$, $t \geq 0$, be a right-continuous N-state Markov chain on the probability space. $r(t)$ only takes value in a finite state space $\mathbb{S} = \{1, 2, ..., N\}$, with generator $\Gamma = (\nu_{ij})_{N\times N}$ defined as

$$\mathbb{P}\{r(t+\delta) = j \mid r(t) = i\} = \begin{cases}\nu_{ij}\delta + o(\delta), \text{ if } i \neq j, \\ 1 + \nu_{ij}\delta + o(\delta), \text{ if } i = j,\end{cases}$$

where $\delta > 0$ and $\nu_{ij} \geq 0$ is the transition rate from state i to j for $i \neq j$. Note that $\nu_{ii} = -\sum_{1\leq j\leq M,\ j\neq i}\nu_{ij}$ and we assume that the Markov chain $r(\cdot)$

is independent of the Brownian motion $B(\cdot)$. Now we would like to know how a function $V : R^d \times R_+ \times \mathbb{S} \to R$ will map the pair process $(x(t), r(t))$ into another process $V(x(t), t, r(t))$. So if $f \in \mathcal{L}^1(R_+; R^d)$, $g \in \mathcal{L}^1(R_+; R^{d\times m})$ and $V \in C^{2,1}(R^d \times R_+ \times \mathbb{S}; R)$, we have for any $t \geq 0$

$$\begin{aligned} V(x(t), t, r(t)) = V(x(0), 0, r(0)) &+ \int_0^t LV(x(s), s, r(s))\mathrm{d}s \\ &+ \int_0^t V_x(x(s), s, r(s))g(x(s), s, r(s))\mathrm{d}B(s) \\ &+ \int_0^t \int_R (V(x(s), s, i_0 + h(r(s), l)) - V(x(s), s, r(s)))\mu(\mathrm{d}s, \mathrm{d}l), \end{aligned}$$

with

$$\begin{aligned} LV(x, t, i) = V_t(x, t, i) + V_x(x, t, i)f(t) &+ \frac{1}{2}trace(g^T(t)V_{xx}(x, t, i)g(t)) \\ &+ \sum_{j=1}^{N} \nu_{ij}V(x, t, j), \end{aligned}$$

where the function h is defined in last section and $\mu(\mathrm{d}s, \mathrm{d}l) = \gamma(\mathrm{d}s, \mathrm{d}l) - \mu(\mathrm{d}l)\mathrm{d}s$ is a martingale measure while μ and γ have also been defined in last section. And this is the generalised Itô's formula which is suitable for an Itô process with Markovian switching. If we have two bounded stopping time τ_1 and τ_2 such that $0 \leq \tau_1 < \tau_2$ a.s., then for bounded $V(x(t), t, r(t))$ and $LV(x(t), t, r(t))$ etc on $t \in [\tau_1, \tau_2]$, we have

$$\begin{aligned} \mathbb{E}\, V(x(t), t, r(t)) = \mathbb{E}\, V(x(\tau_1)\tau_1, r(\tau_1)) &\\ + \mathbb{E} \int_{\tau_1}^{\tau_2} V(x(s), s, r(s))\mathrm{d}s.& \end{aligned}$$

2.7 Stochastic Differential Equation

Firstly we will give the definition of stochastic differential equation without Markovian switching. Let $(\Omega, \mathcal{F}, P)$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ satisfying the usual conditions. Let $B(t) = (B_1(t), ..., B_m(t))$, $t \geq 0$ be an m-dimensional Brownian motion defined on the space. Let $0 \leq t_0 < T \leq \infty$. Let x_0 be an $\mathcal{F}_t$-measurable R^d-valued random variable such that $\mathbb{E}\,|x_0|^2 < \infty$. Let

$f : R^d \times [t_0, T] \to R^d$, and $g : R^d \times [t_0, T] \to R^{d\times m}$ be both Borel measurable. Consider the d-dimensional stochastic differential equation of Itô type

$$\mathrm{d}x(t) = f(x(t), t)\mathrm{d}t + g(x(t), t)\mathrm{d}B(t), \tag{2.1}$$

on $t \in [t_0, T]$, with initial value $x(t_0) = x_0$. This SDE is equivalent to the following stochastic integral equation:

$$x(t) = x_0 + \int_{t_0}^{t} f(x(s), s)\mathrm{d}s + \int_{t_0}^{t} g(x(s), s)\mathrm{d}B(s), \quad t \in [t_0, T]. \tag{2.2}$$

Then we need to give the definition of the solution. An R^d-valued stochastic process $\{x(t)\}_{0\le t\le T}$ is called the solution of (2.1) if:

- $\{x(t)\}$ is continuous and $\mathcal{F}_t$-adapted;
- $\{f(x(t), t)\} \in \mathcal{L}^1([t_0, T]; R^d)$ and $\{g(x(t), t)\} \in \mathcal{L}^2([t_0, T]; R^{d\times m})$;
- equation (2.2) holds for every $t \in [t_0, T]$ with probability 1.

Also, a solution $\{x(t)\}$ is said to be *unique* if any other solution $\{\bar{x}(t)\}$ is indistinguishable from $\{x(t)\}$, that is

$$P\{x(t) = \bar{x}(t) \text{ for all } t_0 \le t \le T\} = 1.$$

However, it is not ensured to have a solution for any stochastic differential equation. Hence now we give the conditions to guarantee the existence and uniqueness of the solution to equation (2.1). Assume that there exists two positive constants K and $\bar{K}$ such that

- (Lipschitz condition) for all $x, y \in R$ and $t \in [t_0, T]$

$$|f(x,t) - f(y,t)|^2 \bigvee |g(x,t) - g(y,t)|^2 \le \bar{K}|x - y|^2;$$

- (Linear growth condition) for all $x, y \in R^d \times [t_0, T]$

$$|f(x,t)|^2 \bigvee |g(x,t)|^2 \le K(1 + |x|^2).$$

Then there exists an unique solution $x(t)$ to equation (2.1) and the solution belongs to $\mathcal{M}^2([t_0, T]; R^d)$. However the Lipschitz condition is too restrictive. It requires f and g changing less faster than a linear function of x in $[t_0, T]$. Clearly there are too many functions that do not meet this requirement. As a result, a local Lipschitz condition is used to replace the Lipschitz condition.

- (local Lipschitz condition) For every integer $n \geq 1$, there exits a positive constant $\bar{K}_n$ such that for all $x, y \in R$ and $t \in [t_0, T]$ with $|x| \vee |y| \leq n$,

$$|f(x,t) - f(y,t)|^2 \bigvee |g(x,t) - g(y,t)|^2 \leq \bar{K}_n |x - y|^2;$$

Moreover, the linear growth condition can also be replaced by a monotone condition:

- (monotone condition) There exits a positive constant K such that for all $(x,t) \in R^d \times [t_0, T]$, such that

$$x^T f(x,t) + \frac{1}{2}|g(x,t)|^2 \leq K(1 + |x|^2);$$

There are also more general conditions for existence and uniqueness of the solution such as Khasminskii's condition [72]. However, the four conditions we have introduced should be enough for the research in following sections. Now we are going to focus on the definition of SDEs with Markovian switching.

2.8 SDEs with Markovian Switching

Now based on the last section, we can now establish conditions for the existence of a solution for the stochastic differential equation with Markovian switching. First of all, let us recall $r(t)$, $t \geq t_0$ to be a right-continuous Markov chain on the probability space in a finite state space $\mathbb{S} = \{1, 2, ..., N\}$ with the generator $\Gamma = (\nu_{ij})_{N \times N}$ defined as

$$\mathbb{P}\{r(t+\delta) = j \mid r(t) = i\} = \begin{cases} \nu_{ij}\delta + o(\delta), \text{ if } i \neq j, \\ 1 + \nu_{ij}\delta + o(\delta), \text{ if } i = j, \end{cases}$$

where $\delta > 0$ and $\nu_{ij} \geq 0$ is the transition rate from state i to j for $i \neq j$. Note that $\nu_{ii} = -\sum_{1 \leq j \leq M,\ j \neq i} \nu_{ij}$ and we assume that the Markov chain $r(\cdot)$ is independent of the Brownian motion $B(\cdot)$. Then a stochastic differential equation with Markovian switching should have the form

$$\mathrm{d}x(t) = f(x(t), t, r(t))\mathrm{d}t + g(x(t), t, r(t))\mathrm{d}B(t), \quad t_0 \leq t \leq T, \tag{2.3}$$

with initial value $x(t_0) = x_0 \in L^2_{\mathcal{F}_{t_0}}(\Omega; R^d)$ and $r(t_0) = r_0$, where r_0 is an $\mathbb{S}$-valued $\mathcal{F}_{t_0}$-measurable random variable and we should now let $f : R^d \times [t_0, T] \times \mathbb{S} \to R^d$, and $g : R^d \times [t_0, T] \times \mathbb{S} \to R^{d \times m}$. Similarly, an R^d-valued stochastic process $\{x(t)\}_{0 \leq t \leq T}$ is called the solution of (2.3) if:

- $\{x(t)\}$ is continuous and $\mathcal{F}_t$-adapted;
- $\{f(x(t), t, r(t))\} \in \mathcal{L}^1([t_0, T]; R^d)$ and $\{g(x(t), t, r(t))\} \in \mathcal{L}^2([t_0, T]; R^{d \times m})$;
- equation

$$x(t) = x_0 + \int_{t_0}^{t} f(x(s), s, r(s))\mathrm{d}s + \int_{t_0}^{t} g(x(s), s, r(s))\mathrm{d}B(s), \tag{2.4}$$

  holds for every $t \in [t_0, T]$ with probability 1.

Also, a solution $\{x(t)\}$ is said to be *unique* if any other solution $\{\bar{x}(t)\}$ is indistinguishable from $\{x(t)\}$.

Conditions for a unique solution can also be expanded. Assume that there exists two positive constants K and $\bar{K}$ such that

- (Lipschitz condition) for all $x, y \in R$, $t \in [t_0, T]$ and $i \in \mathbb{S}$,

$$|f(x, t, i) - f(y, t, i)|^2 \bigvee |g(x, t, i) - g(y, t, i)|^2 \leq \bar{K}|x - y|^2;$$

- (Linear growth condition) for all $x, y, i \in R^d \times [t_0, T] \times \mathbb{S}$,

$$|f(x, t, i)|^2 \bigvee |g(x, t, i)|^2 \leq K(1 + |x|^2).$$

Then there exists an unique solution $x(t)$ to equation (2.3) and the solution belongs to $\mathcal{M}^2([t_0, T]; R^d)$. This Lipschitz condition can also be substituted by the local Lipschitz condition

- (local Lipschitz condition) For every integer $n \geq 1$, there exits a positive constant K_n such that for all $i \in \mathbb{S}$, $x, y \in R$ and $t \in [t_0, T]$ with $|x| \vee |y| \leq n$,

$$|f(x,t,i) - f(y,t,i)|^2 \bigvee |g(x,t,i) - g(y,t,i)|^2 \leq K_n |x-y|^2.$$

Similarly, we can give a more general monotone condition rather than the linear growth condition. In some situations, the monotone condition can be satisfied while the linear growth condition is not. Clearly when the linear growth condition holds then the monotone condition is satisfied. However the converse is not true.

- (monotone condition) There exits a positive constant K such that for all $(x,t,i) \in R^d \times [t_0, T] \times \mathbb{S}$, such that

$$x^T f(x,t,i) + \frac{1}{2}|g(x,t,i)|^2 \leq K(1+|x|^2).$$

Now we have established conditions for the existence of a solution of both types of stochastic differential equations. In the study of SDEs, the long-term behaviour plays an very important role. In the next section we will introduce a theorem on existence and uniqueness of a stationary distribution of the solution.

2.9 Stationary Distribution

In this section we are going to introduce a well-known result from Khasinskii about how to find the stationary distribution of a SDE. Firstly we define the concept of "stationary". A stochastic process $\{X(t)\} = X(t,\omega)$, $(-\infty < t < \infty)$ with values in R^d is said to be stationary if for every finite sequence of number $t_1, ..., t_n$, the joint distribution of the random variable $X(t_1+h), ..., X(t_n+h)$ is independent of h. In other words, the joint probability distribution does not change when shifted in time.

In the study of SDEs, the concept of a stationary distribution is a crucial and necessary part. Now we initially let $X(t)$ be a regular time-homogeneous Markov

process in R^d described by the SDE in the following form

$$\mathrm{d}X(t) = b(X)\mathrm{d}t + \sum_{r=1}^{k} \sigma_r(X)\mathrm{d}B_r(t).$$

Then a *diffusion matrix* is defined by

$$A(x) = (a_{ij}(x)), \quad a_{ij} = \sum_{r=1}^{k} \sigma_r^i(x)\sigma_r^j(x).$$

Khaminskii [72] then gives two conditions for the existence and uniqueness of a stationary distribution of the process $X(t)$. So if there exists an open domain $U \subset R^d$ with regular boundary, such that

- In the domain U and some neighbourhood thereof, the smallest eigenvalue of the diffusion matrix $A(x)$ is bounded away from zero;
- If $x \in R^d \setminus U$, the mean time τ at which a path issuing from x reaches the set U is finite, and $\sup_{x \in K} \mathbb{E}(\tau) < \infty$ for every compact subset $K \subset R^d$,

then $X(t)$ has a unique stationary distribution μ. If $f(x)$ is a integrable function with respect to μ, then

$$P\Big\{\frac{1}{T}\int_0^T f(X(t))\mathrm{d}t \underset{T\to\infty}{\longrightarrow} \int_{R^d} f(y)\mu(\mathrm{d}y)\Big\}.$$

Here we have finished introducing basic stochastic theory that will be used later. Now in the following three chapters, we are going to establish three different stochastic SIS models. In each chapter, a general introduction of the model will be explained. Some previous works will be stated to clearly provide the motivation and inspiration of our research. Then we will show some properties of the model, including the conditions of extinction and persistence. Also, computer simulation will be illustrated to back up our theory.

Chapter 3

SIS Epidemic Model with two Independent Brownian Motions

3.1 Introduction

Research on epidemics modelled by introducing deterministic compartmental models make great contribution to understanding the behaviour of epidemics and helping control of deadly diseases [96, 97]. For example, Capasso [97] introduces the Kermack-Mckendrick model to describe diseases that offer permanent immunity after an individual catching the diseases for a period of time. However, some diseases such as sexually transmitted and bacterial disease do not have permanent immunity. Susceptible individuals will catch the disease at some time to become infected, while after a short period of time infected individuals will become susceptible again. The Susceptible-infected-susceptible (SIS) model is a very simple but also commonly used model to describe such epidemic problems [34]. $S(t)$ and $I(t)$ are used to represent the numbers of susceptible and infected populations at time t. The deterministic model is

$$\begin{cases} \frac{\mathrm{d}S(t)}{\mathrm{d}t} = \mu N - \beta S(t)I(t) + \gamma I(t) - \mu S(t), \\ \frac{\mathrm{d}I(t)}{\mathrm{d}t} = \beta S(t)I(t) - (\mu + \gamma)I(t). \end{cases} \tag{3.1}$$

with initial values $S_0 + I_0 = N$ and here N is the total size of population. μ is the per capita death rate and γ is the rate at which infected individuals become cured. β is the disease transmission coefficient. With the condition $S + I = N$, we can rewrite the original two ODEs (3.1) into

$$\mathrm{d}I(t) = [\beta(N - I(t))I(t) - (\mu + \gamma)I(t)]\mathrm{d}t. \tag{3.2}$$

Deterministic models are not enough to describe problems in the real world because parameters are easily influenced by all kinds of circumstances with uncertainty. Thus stochastic models with different environmental noises are more appropriate in epidemic problems. There are many examples studying the behaviour of both deterministic [97, 98] and stochastic [99, 100, 101, 102] SIS epidemic models. Different medical means on controlling the disease are also mathematically applied in SIS model such as [103, 104, 105].

Gray *et al.* [1] firstly consider the perturbation on β in the deterministic SIS model to discuss the disturbance of white noise. They initially analyse (3.2) in a small time interval $[t, t + \mathrm{d}t)$ with the d notation for small change in any quantity. Hence we have $\mathrm{d}I(t) = I(t + \mathrm{d}t) - I(t)$ in (3.2). Then the disease transmission coefficient β can be regarded as the rate at which each infected individual make contacts with other individuals and the total number of new infections in the small time interval is $\beta I(t)S(t)\mathrm{d}t$ and also, a single infected individual makes $\beta\mathrm{d}t$ potentially infectious contacts with other individuals in the small time interval. Consequently, when some stochastic environmental factor is introduced on each individual in the population, they replace β by a random variable $\tilde{\beta}$

$$\tilde{\beta}\mathrm{d}t = \beta\mathrm{d}t + \sigma_1\mathrm{d}B_1(t). \tag{3.3}$$

Here $\mathrm{d}B_1(t) = B_1(t + \mathrm{d}t) - B_1(t)$ is the increment of a standard Brownian motion. Hence the potentially infectious contacts made by a single infected individual with another individual in the population in the small time interval $[t, t + \mathrm{d}t)$ are normally distributed with mean $\beta\mathrm{d}t$ and variance $\sigma_1^2\mathrm{d}t$. Also, Zhao *et al.* [106] use the same perturbation in a SIS model with vaccination and then find the conditions for the disease to become extinct and persist. There are also many other

contributions on different epidemic models using multiple environmental noises [100, 102, 107, 108].

Now based on the previous work of Gray *et al.* [1], we now consider another perturbation on $(\mu + \gamma)$ with (3.3) existing in traditional SIS model. Within the same small time interval $[t, t + \mathrm{d}t)$, we regard $(\mu + \gamma)I(t)\mathrm{d}t$ as the total number of infected individuals becoming cured or pass away in this time interval. In other words, this is the total reduction of infections. Hence each single individual contributes $(\mu + \gamma)\mathrm{d}t$ in the reduction of infections in the small time interval $[t, t + \mathrm{d}t)$.
Then we introduce stochasticity on $(\mu + \gamma)$. $(\mu + \gamma)$ is replaced by a random variable $(\tilde{\mu} + \tilde{\gamma})$

$$(\tilde{\mu} + \tilde{\gamma})\mathrm{d}t = (\mu + \gamma)\mathrm{d}t + \sigma_2\sqrt{N - I(t)}\mathrm{d}B_2(t). \tag{3.4}$$

Here we do not simply set $(\tilde{\mu} + \tilde{\gamma})\mathrm{d}t = (\mu + \gamma)\mathrm{d}t + \sigma_2\mathrm{d}B_2(t)$ to be the second perturbation. When susceptible population $S(t) = N - I(t)$ is large, which means there are few infected individuals, the error of estimating μ and γ will be large. Thus we suppose that the variance of estimating $\mu + \gamma$ is proportional to the number of susceptible population. As a result, the reduction of infections caused by medical care and death of a single infected individual in the small time interval $[t, t+\mathrm{d}t)$ is normally distributed with mean $(\mu+\gamma)\mathrm{d}t$ and variance ${\sigma_2}^2(N-I(t))\mathrm{d}t$. This is also a biologically reasonable model because the variance trends to 0 when $\mathrm{d}t$ goes to 0.

Such a diffusion coefficient in square root form is widely used in financial stochastic differential equations such as the Square Root Process. Mao [91] indicates that the Square Root Process may be more appropriate if the asset price volatility does not increase dramatically when $S(t)$ increases ($S(t)$ greater than 1), because the variance of error term is proportional to $S(t)$. Meanwhile, in epidemic modelling, Liang *et al.* introduce demographic stochasticity [102] in the deterministic SIS model based on Allen's work [101]. The diffusion coefficient of their SDE SIS model is $\sqrt{\beta I(t)(N - I(t)) + (\mu + \gamma)}$ which is very similar to ours. However, to the best of our knowledge, there is not enough work on incorporating white noise with square-root diffusion into the epidemic models. As a result, in

this chapter we aim to fill the gap.

As a result, we assume that two Brownian motions $B_1(t)$ and $B_2(t)$ are independent. We then substitute two perturbations in our SIS ODE (3.2). We have

$$\begin{aligned} \mathrm{d}I(t) = [\beta(N - I(t))I(t) - (\mu + \gamma)I(t)]\mathrm{d}t + \sigma_1 I(t)(N - I(t))\mathrm{d}B_1(t) \\ - \sigma_2 I(t)\sqrt{N - I(t)}\mathrm{d}B_2(t), \end{aligned} \tag{3.5}$$

with initial value $I(0) = I_0 \in (0, N)$. In the following sections we will concentrate on giving some properties of the solution $I(t)$ of this SDE. This chapter is a published work in February 2019 on Journal of Mathematical Analysis and Applications [2].

3.2 Unique and Bounded Solution

In order for the model to make sense, we need to prove that the solution of our SDE has a unique global solution which remain within $(0, N)$, with the initial value $I_0 \in (0, N)$.

Theorem 3.2.1. *If $\mu + \gamma \geq \frac{1}{2}\sigma_2^2 N$, then for any given initial value $I(0) = I_0 \in (0, N)$, the SDE has a unique global positive solution $I(t) \in (0, N)$ for all $t \geq 0$ with probability one, namely,*

$$\mathbb{P}\{I(t) \in (0, N),\ \forall t \geq 0\} = 1.$$

Proof. The coefficients of our SDE (3.5) are locally Lipschitz continuous and for any given initial value, there is a unique maximal local solution $I(t)$ on $t \in [0, \tau_e)$, where τ_e is the explosion time [91]. Let $k_0 \geq 0$ be sufficiently large to satisfy $\frac{1}{k_0} < I_0 < N - \frac{1}{k_0}$. For each integer $k \geq k_0$, define the stopping time

$$\tau_k = \inf\{t \in [0, \tau_e)\ : I(t) \notin (1/k, N - 1/k)\}.$$

In this chapter, we set $\inf\emptyset = \infty$. Obviously, τ_k is increasing when $k \to \infty$. And we set $\tau_\infty = \lim_{k\to\infty} \tau_k$. It is clear that $\tau_\infty \leq \tau_e$ almost surely. So if we can

show that $\tau_\infty = \infty$ a.s., then $\tau_e = \infty$ a.s. and $I(t) \in (0, N)$ a.s. for all $t \geq 0$. Here we assume $\tau_\infty = \infty$ a.s. is not true. Then we can find a pair of constants $T > 0$ and $\epsilon \in (0, 1)$ such that

$$\mathbb{P}\{\tau_\infty \leq T\} > \epsilon.$$

So we can find an integer $k_1 \geq k_0$ large enough, such that

$$\mathbb{P}\{\tau_k \leq T\} \geq \epsilon, \quad \forall k \geq k_1. \tag{3.6}$$

Define a function $V : (0, N) \to \mathbb{R}_+$ by

$$V(x) = -\log x - \log(N - x) + \log \frac{N^2}{4},$$

and

$$V_x = -\frac{1}{x} + \frac{1}{N - x},\ V_{xx} = \frac{1}{x^2} + \frac{1}{(N - x)^2}.$$

Let $f(t) = \beta(N - I(t))I(t) - (\mu + \gamma)I(t)$, $g(t) = (\sigma_1 I(t)(N - I(t)), -\sigma_2\sqrt{N - I(t)}I(t))$ and $\mathrm{d}B(t) = (\mathrm{d}B_1(t), \mathrm{d}B_2(t))$. By Itô's formula [91], we have, for any $t \in [0, T]$ and $k \geq k_1$

$$\mathbb{E}V(I(t \wedge \tau_k)) = V(I_0) + \mathbb{E}\int_0^{t\wedge\tau_k} LV(I(s))\mathrm{d}s + \mathbb{E}\int_0^{t\wedge\tau_k} V_x g(s)\mathrm{d}B(s), \tag{3.7}$$

where $\mathbb{E}\int_0^{t\wedge\tau_k} V_x g(s)\mathrm{d}B(s) = 0$. Also it is easy to show that

$$\begin{aligned} LV(x) &= -\beta(N - x) + (\mu + \gamma) + \beta x - (\mu + \gamma)\frac{x}{N - x} \\ &\quad + \frac{1}{2}(\sigma_1^2(N - x)^2 + \sigma_1^2 x^2 + \sigma_2^2(N - x) + \sigma_2^2\frac{x^2}{N - x}) \\ &\leq -\beta(N - x) + (\mu + \gamma) + \beta x + \frac{1}{2}[\sigma_1^2(N - x)^2 + \sigma_1^2 x^2 + \sigma_2^2(N - x)] \\ &\leq C, \end{aligned} \tag{3.8}$$

where C is a constant when $\mu + \gamma \geq \frac{1}{2}\sigma_2^2 N$ and $x \in (0, N)$. Then we have

$$\begin{aligned} \mathbb{E}V(I(t \wedge \tau_k)) &\leq V(I_0) + \mathbb{E}\int_0^{t\wedge\tau_k} C\mathrm{d}s \\ &\leq V(I_0) + Ct, \end{aligned} \tag{3.9}$$

which yields that

$$\mathbb{E}V(I(T \wedge \tau_k)) \leq V(I_0) + CT. \tag{3.10}$$

Set $\Omega_k = \{\tau_k \leq T\}$ for $k \geq k_1$ and we have $\mathbb{P}(\Omega_k) \geq \epsilon$. For every $\omega \in \Omega_k$, $I(\tau_k, \omega)$ equals either $1/k$ or $N - 1/k$ and we have

$$V(I(\tau_k, \omega)) = \log \frac{N^2}{4(N - 1/k)1/k}.$$

Hence

$$\begin{aligned} \infty > V(I_0) + CT &\geq \mathbb{E}[\mathbb{I}_{\Omega_k}(\omega) V(I(\tau_k, \omega))] \\ &\geq \mathbb{P}(\Omega_k) \log \frac{N^2}{4(N - 1/k)1/k} \\ &\geq \epsilon \log \frac{N^2}{4(N - 1/k)1/k}. \end{aligned}$$

Letting $k \to \infty$ will lead to the contradiction

$$\infty > V(I_0) + CT = \infty.$$

□

So the assumption is wrong and we must have $\tau_\infty = \infty$ almost surely, whence the proof is now completed. However, the condition for our model to have bounded positive solution $\mu + \gamma \geq \frac{1}{2}\sigma_2^2 N$ might be confusing to readers. There are two different ways to understand this condition. In [1] there is no constraint on σ_1 but after adding second perturbation on $\mu + \gamma$, the square root term will trend to infinity very fast when $I(t) \to N$. So there must be a condition on σ_2 to neutralize it. Also, by the classical Feller test in Mao's book [91] on Mean Reverting Square Root Process, there is a very similar result on constraining the coefficient before square root term in order to make the solution always positive.

3.3 Extinction

In this section, we will discuss the conditions for the disease to die out in our SDE model (3.5). Here we give the conditions for the solution $I(t)$ of our SDE becoming extinct.

Theorem 3.3.1. *Given that* $R_0^S := R_0^D - \frac{\sigma_1^2 N^2 + \sigma_2^2 N}{2(\mu+\gamma)} = \frac{\beta N}{\mu+\gamma} - \frac{\sigma_1^2 N^2 + \sigma_2^2 N}{2(\mu+\gamma)} < 1$, *then for any given initial value* $I(0) = I_0 \in (0, N)$, *the solution of SDE* (3.5) *obeys*

$$\limsup_{t\to\infty} \frac{1}{t} \log I(t) < 0 \ a.s.. \tag{3.11}$$

if one of the following three conditions is satisfied

- $\sigma_1^2 N + \frac{1}{2}\sigma_2^2 \le \beta$ or,
- $\frac{1}{2}\sigma_2^2 \ge \beta$ or,
- $(\beta - \sigma_1\sqrt{2(\mu+\gamma)}) \vee (\beta - \sigma_1^2 N) < \frac{1}{2}\sigma_2^2 < \beta$.

Namely, $I(t)$ *will trend to zero exponentially a.s. And the disease will die out with probability one.*

Proof. Here we use Itô's formula

$$\frac{\log I(t)}{t} = \frac{\log I_0}{t} + \frac{1}{t}\int_0^t L\tilde{V}(I(s))\mathrm{d}s + \frac{1}{t}\int_0^t \sigma_1(N - I(s))\mathrm{d}B_1(s) - \frac{1}{t}\int_0^t \sigma_2\sqrt{(N - I(s))}\mathrm{d}B_2(s), \tag{3.12}$$

with $\tilde{V} = \log x$ and $L\tilde{V}$ is defined by

$$L\tilde{V}(x) = \beta(N - x) - (\mu + \gamma) - \frac{1}{2}[\sigma_1^2(N - x)^2 + \sigma_2^2(N - x)], x \in (0, N). \tag{3.13}$$

According to the large number theorem for martingales [91], we must have

$$\limsup_{t\to\infty} \frac{1}{t}\left\{\int_0^t \sigma_1(N - I(s))\mathrm{d}B_1(s) - \int_0^t \sigma_2\sqrt{(N - I(s))}\mathrm{d}B_2(s)\right\} = 0. \tag{3.14}$$

So if we can prove $L\tilde{V} \le \tilde{C} < 0$, then $\limsup_{t\to\infty} \frac{1}{t}\log I(t) < 0$ a.s. ($\tilde{C}$ is a constant).We first examine $L\tilde{V}$ at 0 and N. $L\tilde{V}(N) = -(\mu + \gamma) < 0$ and $L\tilde{V}(0) = \beta N - (\mu + \gamma) - \frac{1}{2}(\sigma_1^2 N^2 + \sigma_2^2 N)$ so we must have firstly

$$L\tilde{V}(0) < 0, \text{which is ensured by} R_0^S < 1. \tag{3.15}$$

$L\tilde{V}(x)$ has the maximal value when

$$x = \hat{x} = \frac{-\beta + \sigma_1^2 N + \frac{1}{2}\sigma_2^2}{\sigma_1^2} = N + \frac{\frac{1}{2}\sigma_2^2 - \beta}{\sigma_1^2}, \tag{3.16}$$

and

$$L\tilde{V}(\hat{x}) = \frac{1}{2}\frac{(\beta - \frac{1}{2}\sigma_2^2)^2}{\sigma_1^2} - (\mu + \gamma), \tag{3.17}$$

is the maximal value of $L\tilde{V}$ when $x \in \mathbb{R}$.

So we need to discuss the following three different cases:

Case 1. $\hat{x} \leq 0$

With $L\tilde{V} < 0$ at 0 and N, if we have $\hat{x} \leq 0$ then $L\tilde{V} < 0$ for all $x \in (0, N)$. Consequently, $L\tilde{V} < 0$ for all $x \in (0, N)$ if

$$\sigma_1^2 N + \frac{1}{2}\sigma_2^2 \leq \beta. \tag{3.18}$$

Case 2. $\hat{x} \geq N$

This is similar to **Case 1**. $L\tilde{V} < 0$ for all $x \in (0, N)$.So we must have

$$\frac{1}{2}\sigma_2^2 \geq \beta. \tag{3.19}$$

Case 3. $\hat{x} \in (0, N)$

In this case we need to make sure the maximal value $L\tilde{V}(\hat{x}) < 0$. So we have

$$L\tilde{V}(\hat{x}) = \frac{1}{2}\frac{(\beta - \frac{1}{2}\sigma_2^2)^2}{\sigma_1^2} - (\mu + \gamma) < 0. \tag{3.20}$$

Also,

$$\frac{1}{2}\sigma_2^2 < \beta, \tag{3.21}$$

and

$$\sigma_1^2 N + \frac{1}{2}\sigma_2^2 > \beta, \tag{3.22}$$

is required for $\hat{x}$ within $(0, N)$. Rearrange and we therefore have the result.

$$(\beta - \sigma_1\sqrt{2(\mu + \gamma)}) \vee (\beta - \sigma_1^2 N) < \frac{1}{2}\sigma_2^2 < \beta. \tag{3.23}$$

Hence when any of the three cases is satisfied, we must have $L\tilde{V} \leq \tilde{C} < 0$ ($\tilde{C}$ is a constant). It then follows that

$$\limsup_{t\to\infty} \frac{\log I(t)}{t} \leq \limsup_{t\to\infty} \frac{\log I_0}{t} + \limsup_{t\to\infty} \frac{1}{t}\tilde{C}t + 0 < 0 \quad a.s..$$

Therefore we now have obtained the proof. □

The stochastic reproduction number R_0^S that we define in this section is obviously smaller than the $R_0^S = \frac{\beta N}{\mu+\gamma} - \frac{\sigma_1^2 N^2}{2(\mu+\gamma)}$ in [1]. To some extent, with the introduction of a new perturbation on $\mu + \gamma$, we can possibly conclude in extinction for situations that may not happen in Gray *et al.*'s model. Moreover, though the second perturbation is not linear, we still have the Itô operator $L\tilde{V}$ to be a quadratic function, which is simple to discuss. This indicate that after introducing a white noise with nonlinear property, it does not weaken our result but instead generalized it.

3.4 Persistence

In this section we want to discuss the conditions for disease to persist in our model. However, there are many definitions of persistence in stochastic dynamic problems [1, 5, 91, 99, 100, 107, 109]. For example, in Mao's book [91] he gives a very general definition of persistence, which only needs the disease to never become extinction with probability 1, such that

$$\liminf_{t\to\infty} I(t) > 0.$$

Gray *et al.* [1] have showed the persistence of their model as oscillations around a positive level. This is a very strong result in epidemic problem. As our work is an extension of [1], we give the following theorem

Theorem 3.4.1. *If* $R_0^S = R_0^D - \frac{\sigma_1^2 N^2 + \sigma_2^2 N}{2(\mu+\gamma)} = \frac{\beta N}{\mu+\gamma} - \frac{\sigma_1^2 N^2 + \sigma_2^2 N}{2(\mu+\gamma)} > 1$*, then for any given initial value* $I(0) = I_0 \in (0, N)$*, the solution of* (3.5) *follows*

$$\limsup_{t\to\infty} I(t) \geq \xi \text{ and } \liminf_{t\to\infty} I(t) \leq \xi \quad a.s., \tag{3.24}$$

where

$$\xi = \frac{-\beta + \sigma_1^2 N + \frac{1}{2}\sigma_2^2 + \sqrt{\beta^2 - \sigma_2^2\beta - 2\sigma_1^2(\mu+\gamma) + \frac{1}{4}\sigma_2^4}}{\sigma_1^2}, \tag{3.25}$$

which is the only positive root of $L\tilde{V} = 0$ *in* $(0, N)$*.* $I(t)$ *will be above or below the level* ξ *infinitely often with probability one.*

Proof. When $R_0^S > 1$, recall (3.13) that

$$L\tilde{V}(x) = \beta(N-x) - (\mu+\gamma) - \frac{1}{2}[\sigma_1^2(N-x)^2 + \sigma_2^2(N-x)], x \in (0,N),$$

and we have $L\tilde{V}(0) > 0$, $L\tilde{V}(N) = -(\mu+\gamma) < 0$ and $\xi > \hat{x} = \frac{-\beta+\sigma_1^2 N+\frac{1}{2}\sigma_2^2}{\sigma_1^2}$. So $L\tilde{V}(x)$ is strictly increasing in $(0, 0\vee\hat{x})$ and strictly decreasing in $(0\vee\hat{x}, N)$.

Here we recall (3.12)

$$\frac{\log I(t)}{t} = \frac{\log I_0}{t} + \frac{1}{t}\int_0^t L\tilde{V}(I(s))\mathrm{d}s + \frac{1}{t}\int_0^t \sigma_1(N-I(s))\mathrm{d}B_1(s) - \frac{1}{t}\int_0^t \sigma_2\sqrt{(N-I(s))}\mathrm{d}B_2(s).$$

By the large number theorem for martingales [91], there is an $\Omega_2 \subset \Omega$ with $\mathbb{P}\{\Omega_2\} = 1$ such that for every $\omega \in \Omega_2$

$$\lim_{t\to\infty}\frac{1}{t}\left\{\int_0^t \sigma_1(N-I(s))\mathrm{d}B_1(s) - \int_0^t \sigma_2\sqrt{(N-I(s))}\mathrm{d}B_2(s)\right\} = 0. \tag{3.26}$$

Now we assume that $\limsup_{t\to\infty} I(t) \geq \xi$ *a.s.* is not true. Then there must be a small $\epsilon \in (0,1)$ such that

$$\mathbb{P}\left\{\limsup_{t\to\infty} I(t) \leq \xi - 2\epsilon\right\} > \epsilon. \tag{3.27}$$

Let $\Omega_1 = \{\limsup_{t\to\infty} I(t) \leq \xi - 2\epsilon\}$, then for every $\omega \in \Omega_1$, there exists $T = T(\omega)$ large enough, such that

$$I(t,\omega) \leq \xi - 2\epsilon + \epsilon = \xi - \epsilon, \text{ when } t \geq T(\omega), \tag{3.28}$$

which means when $t \geq T(\omega)$, $L\tilde{V}(I(t,\omega)) \geq L\tilde{V}(\xi-\epsilon)$. So we have for any fixed $\omega \in \Omega_1 \cap \Omega_2$ and $t \geq T(\omega)$

$$\begin{aligned}\liminf_{t\to\infty}\frac{1}{t}\log I(t,\omega) &\geq 0 + \lim_{t\to\infty}\frac{1}{t}\int_0^{T(\omega)} L\tilde{V}(I(s,\omega))\mathrm{d}s + \lim_{t\to\infty}\frac{1}{t}L\tilde{V}(\xi-\epsilon)(t-T(\omega)) \\ &\geq L\tilde{V}(\xi-\epsilon) > 0,\end{aligned}$$

which yields

$$\lim_{t\to\infty} I(t,\omega) = \infty, \tag{3.29}$$

and this contradicts with $\omega \in \Omega_1$. So we must have $\limsup_{t\to\infty} I(t) \geq \xi$ almost surely.

Similarly, if we assume that $\liminf_{t\to\infty} I(t) \leq \xi$ *a.s.* is not true. Then there must be a small $\delta \in (0,1)$ such that

$$\mathbb{P}\left\{\liminf_{t\to\infty} I(t) \geq \xi + 2\delta\right\} > \delta. \tag{3.30}$$

Let $\Omega_3 = \{\liminf_{t\to\infty} I(t) \geq \xi + 2\delta\}$, then for every $\omega \in \Omega_3$, there exists $T' = T'(\omega)$ large enough, such that

$$I(t,\omega) \geq \xi + 2\delta - \delta = \xi + \delta, \text{ when } t \geq T'(\omega). \tag{3.31}$$

Now we fix any $\omega \in \Omega_3 \cap \Omega_2$ and $t \geq T'(\omega)$ in (3.12) and we have

$$\begin{aligned}\limsup_{t\to\infty} \frac{1}{t}\log I(t,\omega) &\leq 0 + \lim_{t\to\infty}\frac{1}{t}\int_0^{T'(\omega)} L\tilde{V}(I(s,\omega))\mathrm{d}s + \lim_{t\to\infty}\frac{1}{t}L\tilde{V}(\xi+\delta)(t-T'(\omega)) \\ &\leq L\tilde{V}(\xi+\delta) < 0,\end{aligned}$$

which yields

$$\lim_{t\to\infty} I(t,\omega) = 0, \tag{3.32}$$

and this contradicts $\omega \in \Omega_3$. So we must have $\liminf_{t\to\infty} I(t) \leq \xi$ almost surely. □

In the next section we will identify the stationary distribution of the solution under the condition of persistence.

3.5 Stationary Distribution

In this section we will prove that there exists a unique stationary distribution of our SDE model (3.5) when the solution persists and oscillates around the positive level ξ. So here we give the first theorem of this section.

Theorem 3.5.1. *If $R_0^S > 1$, then our SDE model (3.5) has a unique stationary distribution*

In order to complete our proof, we need to initially use a well-known result from Khaminskii's book as a lemma [72].

Lemma 3.5.2. *The SDE model has a unique stationary distribution if there is a strictly proper subinterval (a, b) of $(0, N)$ such that $\mathbb{E}(\tau) < \infty$ for all $I_0 \in (0, a] \cup [b, N)$, where*

$$\tau = \inf\{t \geq 0 : I(t) \in (a, b)\}, \tag{3.33}$$

also,

$$\sup_{I_0 \in [\bar{a}, \bar{b}]} \mathbb{E}(\tau) < \infty, \tag{3.34}$$

for every interval $[\bar{a}, \bar{b}] \subset (0, N)$

There is also another condition requiring the sum of square of the diffusion coefficients of the SDE to be bounded away from zero for all $I \in (a, b)$. As this is obvious for (3.5), we do not need to point it out here. Hence here we will prove **Theorem 3.5.1** using **Lemma 3.5.2**.

Proof. Firstly we need to fix any (a, b) such that,

$$0 < a < \xi < b < N. \tag{3.35}$$

Recall $L\tilde{V}$ in last section, we can see that

$$L\tilde{V}(x) \geq L\tilde{V}(0) \wedge L\tilde{V}(a), \text{if } 0 < x \leq a, \tag{3.36}$$

$$L\tilde{V}(x) \leq L\tilde{V}(b), \text{if } b \leq x < N. \tag{3.37}$$

Also, recall (3.12)

$$\log I(t) = \log I_0 + \int_0^t L\tilde{V}(I(s))\mathrm{d}s + \int_0^t \sigma_1(N - I(s))\mathrm{d}B_1(s) - \int_0^t \sigma_2\sqrt{(N - I(s))}\mathrm{d}B_2(s),$$

and define

$$\tau = \inf\{t \geq 0 : I(t) \in (a, b)\}. \tag{3.38}$$

Step 1. Firstly we need to discuss one possible situation when the initial value starts in $(0, a]$. Hence for all $t \geq 0$ and any $I_0 \in (0, a]$, from (3.36), we have

$$\begin{aligned}\mathbb{E}\log I(t\wedge\tau) &= \mathbb{E}\log I_0 + \mathbb{E}\int_0^{t\wedge\tau} L\tilde{V}(I(s))\mathrm{d}s + 0\\ &\geq \log I_0 + \mathbb{E}(L\tilde{V}(0)\wedge L\tilde{V}(a))(t\wedge\tau). \end{aligned} \tag{3.39}$$

From the definition of τ, we know that

$$\log a \geq \mathbb{E}\log I(t\wedge\tau) \text{ when } I_0 \in (0, a]. \tag{3.40}$$

Rearrange we have

$$\mathbb{E}(t\wedge\tau) \leq \frac{\log\left(\frac{a}{I_0}\right)}{L\tilde{V}(0)\wedge L\tilde{V}(a)},$$

when $t \to \infty$

$$\mathbb{E}(\tau) \leq \frac{\log\left(\frac{a}{I_0}\right)}{L\tilde{V}(0)\wedge L\tilde{V}(a)} < \infty, \quad \forall I_0 \in (0, a]. \tag{3.41}$$

This means the solution will proceed into (a, b) in finite time.

Step 2. Now similarly, we need to discuss the other situation when $I_0 \in [b, N)$. For all $t \geq 0$ and any $I_0 \in [b, N)$, from (3.37), we have

$$\begin{aligned}\mathbb{E}\log I(t\wedge\tau) &= \mathbb{E}\log I_0 + \mathbb{E}\int_0^{t\wedge\tau} L\tilde{V}(I(s))\mathrm{d}s + 0\\ &\leq \log I_0 + \mathbb{E}\left[(L\tilde{V}(b))(t\wedge\tau)\right]. \end{aligned} \tag{3.42}$$

From the definition of τ, we know that

$$\log b \leq \mathbb{E}\log I(t\wedge\tau) \text{ when } I_0 \in [b, N). \tag{3.43}$$

Rearrange we have

$$\log b \leq \log I_0 + L\tilde{V}(b)\mathbb{E}(t\wedge\tau),$$

$$\mathbb{E}(t\wedge\tau) \leq \frac{\log\left(\frac{I_0}{b}\right)}{\mid L\tilde{V}(b)\mid},$$

when $t \to \infty$

$$\mathbb{E}(\tau) \leq \frac{\log\left(\frac{I_0}{b}\right)}{\mid L\tilde{V}(b)\mid} < \infty, \quad \forall I_0 \in [b, N). \tag{3.44}$$

Hence the solution will proceed into (a, b) as well. Combine the results from both **Step 1** and **Step 2**, we complete the proof. □

Now we know the existence of a unique stationary distribution. Consequently, we will derive the mean and variance of this stationary distribution. It is necessary to state that the probability density function of the stationary distribution can be identified by Kolmogorov-Chapman equation [99, 110]. However it is very complicated and unnecessary. Hence here we follow Khasminskii's work to derive the moments of the stationary distribution.

Theorem 3.5.3. *If $R_0^S > 1$ and denote m and v as the mean and variance of the stationary distribution of SDE model* (3.5)*. Then we have*

$$m = \frac{2\beta(R_0^S - 1)(\mu + \gamma)}{2\beta^2 - \sigma_1^2(\beta N + \mu + \gamma) - \sigma_2^2\beta}, \tag{3.45}$$

and

$$v = \frac{\beta N - \mu - \gamma}{\beta} m - m^2. \tag{3.46}$$

Proof. For any $I_0 \in (0, N)$, we firstly recall (3.5) in the integral form

$$I(t) = I_0 + \int_0^t [\beta(N - I(s))I(s) - (\mu + \gamma)I(s)]\mathrm{d}s + \int_0^t \sigma_1 I(s)(N - I(s))\mathrm{d}B_1(s) \\ \int_0^t -\sigma_2 I(s)\sqrt{N - I(s)}\mathrm{d}B_2(s). \tag{3.47}$$

Dividing both sides by t and when $t \to \infty$, applying the ergodic property of the stationary distribution [72] and also the large number theorem of martingales, we have the result that

$$0 = (\beta N - \mu - \gamma)m - \beta m_2, \tag{3.48}$$

where m, m_2 are the mean and second moment of the stationary distribution. Also,we need to consider (3.12) as well

$$\frac{\log I(t)}{t} = \frac{\log I_0}{t} + \frac{1}{t}\int_0^t L\tilde{V}(I(s))\mathrm{d}s + \frac{1}{t}\int_0^t \sigma_1(N - I(s))\mathrm{d}B_1(s) \\ - \frac{1}{t}\int_0^t \sigma_2\sqrt{(N - I(s))}\mathrm{d}B_2(s),$$

When $t \to \infty$, we have

$$\frac{1}{2}\sigma_1^2 m_2 - \left(\sigma_1^2 N + \frac{1}{2}\sigma_2^2 - \beta\right) m = \beta N - \mu - \gamma - \frac{1}{2}\sigma_1^2 N^2 - \frac{1}{2}\sigma_2^2 N.$$

Note that $\beta N - \mu - \gamma - \frac{1}{2}\sigma_1^2 N^2 - \frac{1}{2}\sigma_2^2 N = (R_0^S - 1)(\mu + \gamma)$. Rewrite this

$$\frac{1}{2}\sigma_1^2 m_2 - \left(\sigma_1^2 N + \frac{1}{2}\sigma_2^2 - \beta\right) m = (R_0^S - 1)(\mu + \gamma).$$

Rearrange and we have

$$m = \frac{2\beta(R_0^S - 1)(\mu + \gamma)}{2\beta^2 - \sigma_1^2(\beta N + \mu + \gamma) - \sigma_2^2\beta}. \tag{3.49}$$

Also,

$$v = m_2 - m^2 = \frac{\beta N - \mu - \gamma}{\beta} m - m^2. \tag{3.50}$$

□

Here we complete our proof of the stationary distribution. Now in the next section, we will illustrate our theory in R to further explain our results.

3.6 Simulation

In this section, we will give some simulation examples in R to back up our theory. Firstly, we will examine three different conditions in **Theorem 3.3.1**, where the solution trends to be extinction. We shall firstly assume that there is a certain kind of disease which currently prevails in a population. The unit of time is one day and the population sizes are measured in units of 1 million, unless otherwise stated. Consequently, we use a similar data set as in [1].

$$N = 100,\ \beta = 0.4,\ \mu + \gamma = 45,\ \sigma_1 = 0.03.$$

In order to find the value of σ_2, we initially need the model to make sense, so we have

$$\sigma_2 \leq 2(\mu + \gamma)/N = 0.9, \tag{3.51}$$

and also if there is extinction in our model, we need

$$R_0^S < 1, \text{which results in } \sigma_2 \geq 0. \tag{3.52}$$

Using these parameters in the other three conditions, we have the corresponding σ_2 to satisfy the three conditions in extinction.

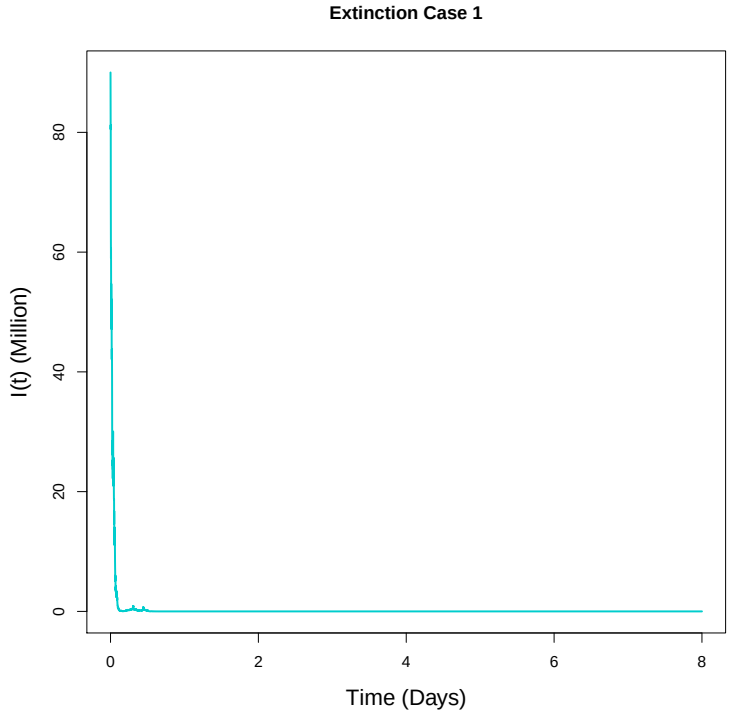
- Condition 1: $\sigma_2 \leq 0.78740078$ or,
- Condition 2: $\sigma_2 \geq 0.8944272$ or,
- Condition 3: $0.78740078 < \sigma_2 < 0.8944271$.

A wide range of parameters and initial values have been randomly chosen, which satisfy those conditions to simulate the solutions. Results from those simulations match with the theoretical results. Hence here we only choose one set of parameter for each different conditions (0.3, 0.9 and 0.82 respectively) and plot our model by using the Euler-Maruyama (EM) Method [91, 99] in R, with step size $\Delta = 0.001$ and one large and one small initial values. The computer simulations are presented in **Figures 3.1, 3.2 and 3.3**. Clearly, our results in this section are illustrated and supported by the simulations. With the values of parameters, the disease will die out.

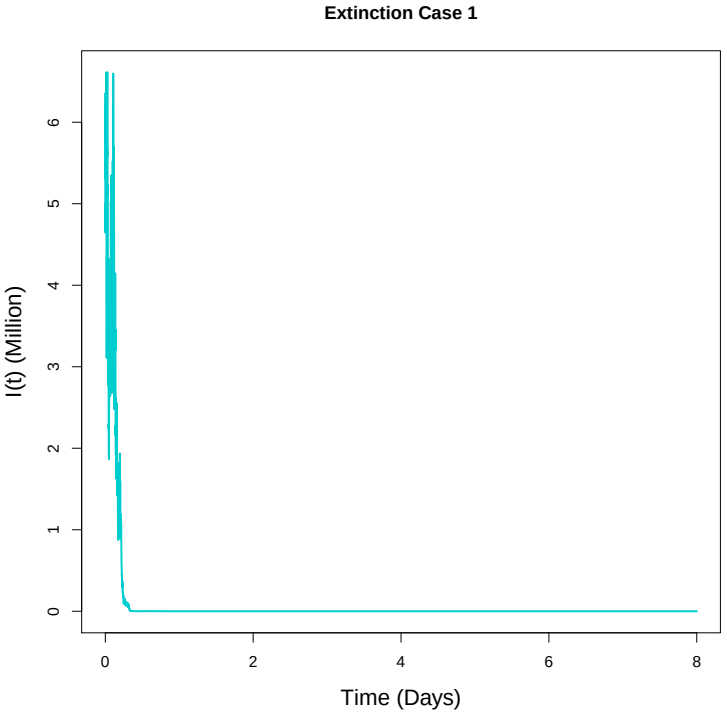
In order to illustrate **Theorem 3.4.1**, we choose the following parameter values to meet the persistence condition.

$$N = 100,\ \beta = 0.5,\ \mu + \gamma = 45,\ \sigma_1 = 0.03.$$

With $R_0^S > 1$, we have $\sigma_2 < 0.1$. Hence here we choose $\sigma_2 = 0.02$ and the level is $\xi = 1.1698004$. Similarly, as the level ξ is very close to zero, we use both large and small initial values and plot the level ξ in the simulation plots to illustrate the results to avoid loss of generality. From **Figure 3.4**, it is clear that the number of infectious population will fluctuate around the level ξ, which has been marked by red lines in **Figure 3.4**. Thus the disease will not die out or explode, which means the disease will persist. It is also needs to be pointed out that solution in **Figure 3.4** can go between 0 and 1. Although we are taking population units to be one million, it will be biologically meaningless if we change the unit to be one person.

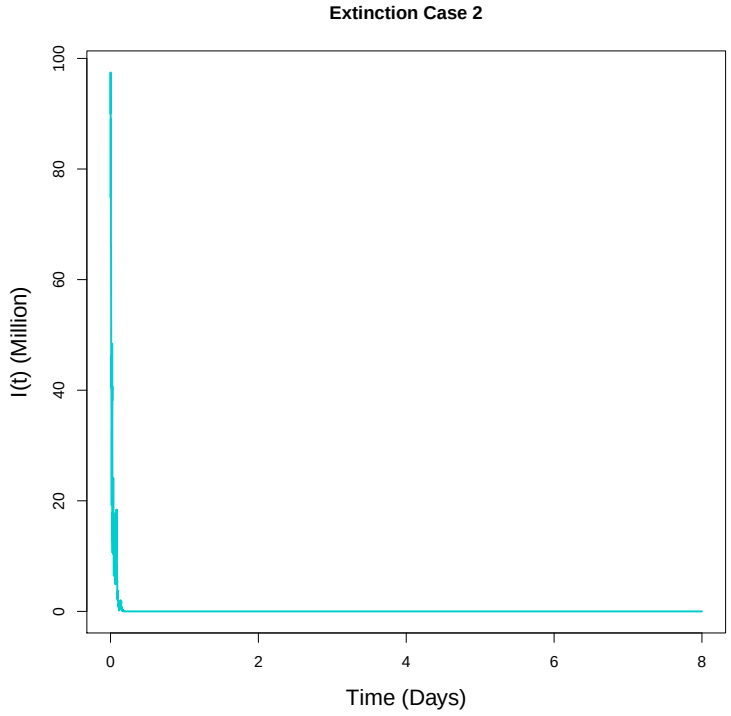


(a) $I(0) = 90$.

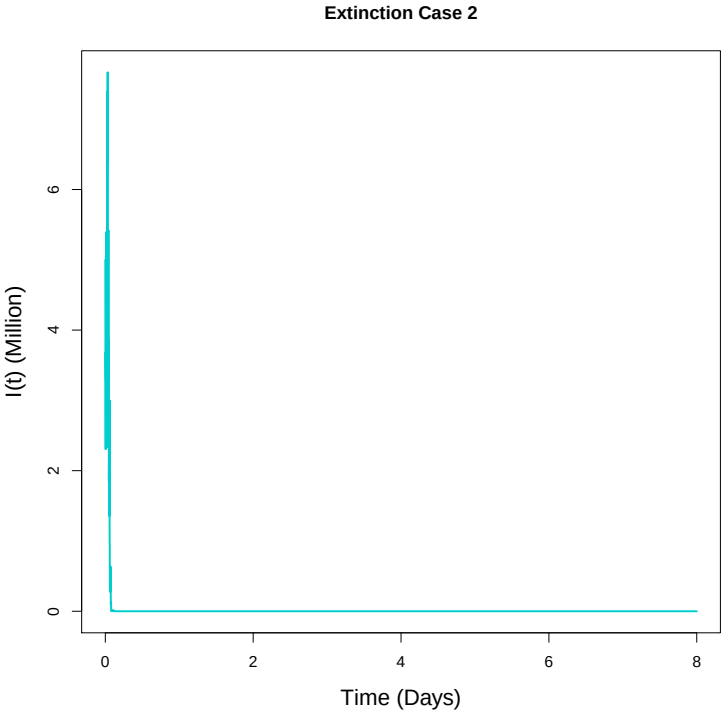


(b) $I(0) = 5$.

Figure 3.1: Extinction with Condition 1.

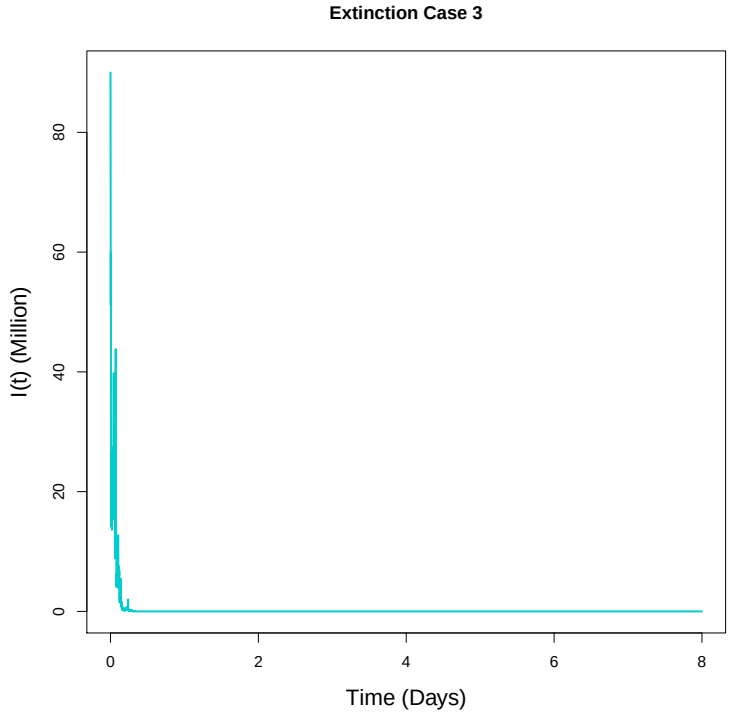


(a) $I(0) = 90$.

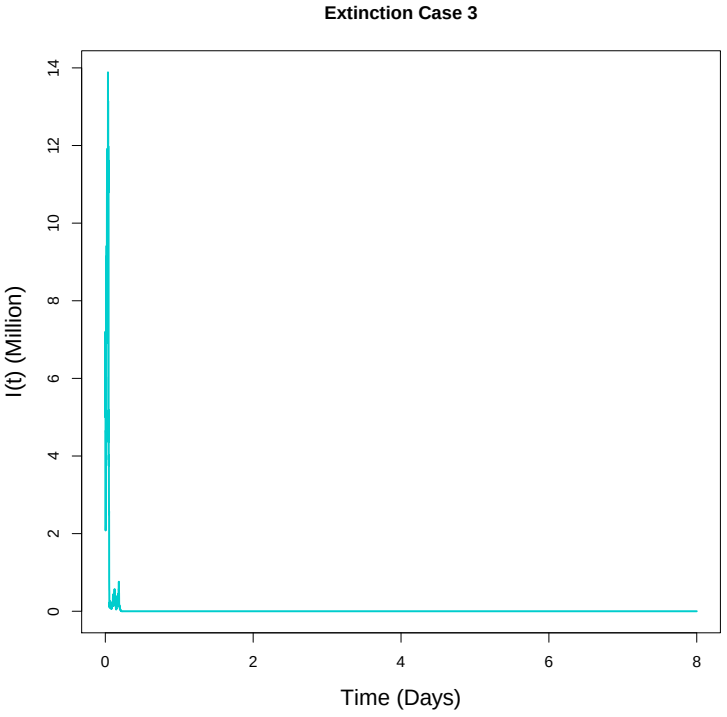


(b) $I(0) = 5$.

Figure 3.2: Extinction with Condition 2.

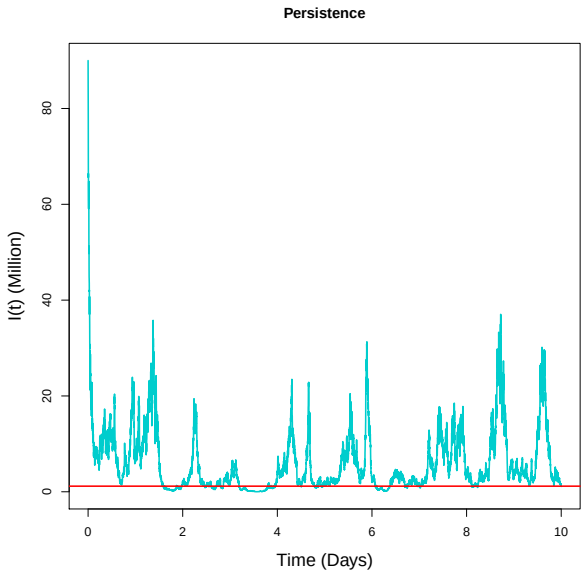


(a) $I(0) = 90$.

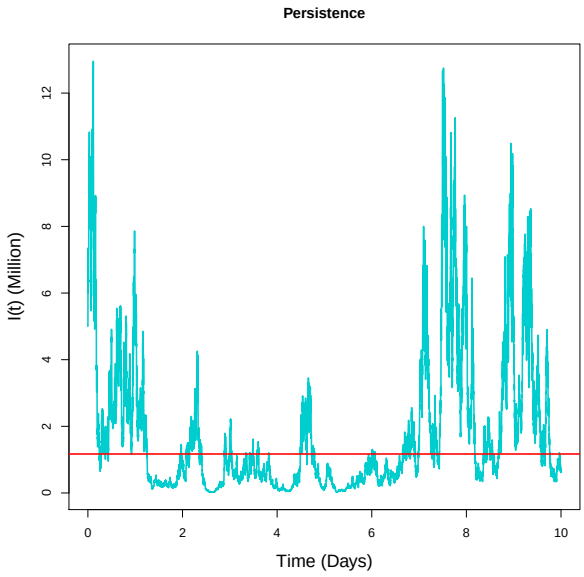


(b) $I(0) = 5$.

Figure 3.3: Extinction with Condition 3.

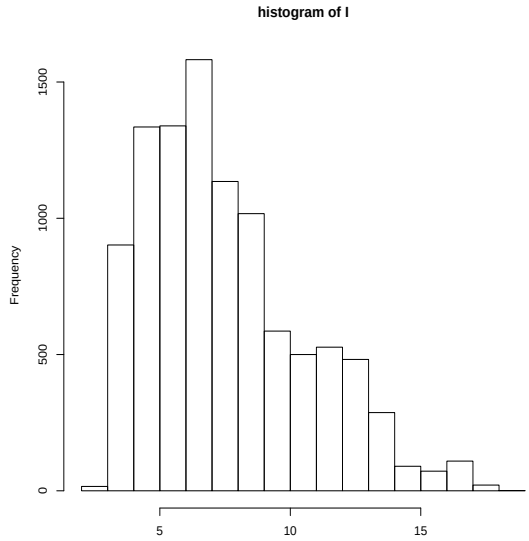


(a) $I(0) = 90$.

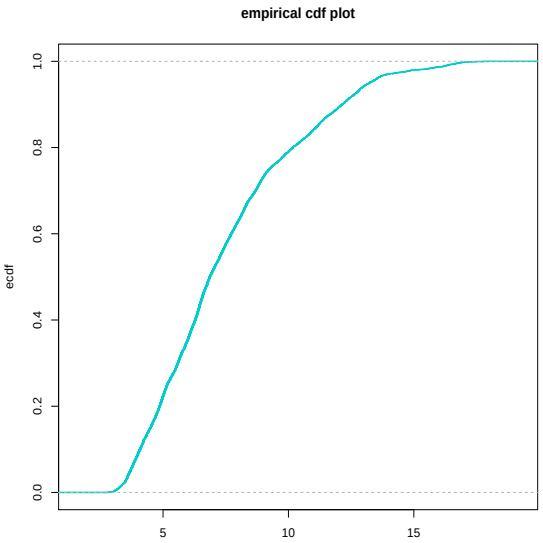


(b) $I(0) = 5$.

Figure 3.4: Persistence.



(a) $I(t) : \sigma_1 = 0.02, \sigma_2 = 0.01$.



(b) ecdf.

Figure 3.5: Stationary Distribution.

In order to generate a stationary distribution, we firstly choose the following data set

$$N = 100,\ \beta = 0.5,\ \mu + \gamma = 45,\ \sigma_1 = 0.02,\ \sigma_2 = 0.01.$$

$R_0^S = 1.06656 > 1$ so the disease will persist and there is a unique stationary distribution of our model according to **Theorem 3.5.1**. And for these parameters, the mean and variance of the stationary distribution of our model (from **Theorem 3.5.3**) are

$$m = 6.483386,\ v = 22.799567.$$

In order to reach the stationary distribution in our simulation, we set a long run of 200,000 iterations with step size $\Delta = 0.001$ and then store the last 10,000 iterations to show the recurrent of our solution. The results from simulations show that

$$m = 6.479609,\ v = 22.7355167.$$

Figure 3.5 also displays the path of $I(t)$ and the empirical cumulative distribution functions for the last 10,000 samples of the simulation.

3.7 Summary

In this chapter, based on Gray *et al.*'s work in 2011 [1], we reasonably replace $\mu+\gamma$ with a random variable to introduce another white noise in the SIS model. Compared to [1], our perturbation is in a different form. It has a square-root diffusion coefficient related to the susceptible population. Introducing this perturbation turns the original model into a nonlinear stochastic SIS model. However, our results indicate that adding this noise still has positive impact. For example, the stochastic reproduction number R_0^S is expanded. Also, it is interesting that although we introduce a nonlinear noise in our model, using the same $\tilde{V} = \log x$ as in [1], we have the new $L\tilde{V}$ of our model (3.5) still quadratic and easy to analyse. Hence, results are consequently generalized from [1].

Here we complete the study of our first model (3.5). In our assumptions, we initially assume that two Brownian motions are independent. However, if we construct two noises by using real data to simulate two Brownian motions, they are highly likely to be correlated. Also, studying correlated noises in the SIS model is not common but reasonable and necessary. Hence inspired by a presentation from

Professor Alexandru Hening on correlated Lotka-Volterra food chain model [3], we are going to consider the two white noises to be correlated in the next chapter.

Chapter 4

SIS Epidemic Model with two Correlated Brownian Motions

4.1 Introduction

In **Chapter 3**, we introduced a new perturbation (3.4) into Gray's model in 2011[1]

$$(\tilde{\mu}+\tilde{\gamma})\mathrm{d}t = (\mu+\gamma)\mathrm{d}t + \sigma_2\sqrt{N-I(t)}\mathrm{d}B_4(t),$$

and we obtained our model (3.5)

$$\begin{aligned}\mathrm{d}I(t) = [\beta(N-I(t))I(t) - (\mu+\gamma)I(t)]\mathrm{d}t + \sigma_1 I(t)(N-I(t))\mathrm{d}B_3(t)\\ - \sigma_2 I(t)\sqrt{N-I(t)}\mathrm{d}B_4(t),\end{aligned}$$

with initial value $I(0) = I_0 \in (0, N)$, and we assumed that B_3 and B_4 are two independent Brownian Motions.

However, it is interesting to consider if there is a relationship between these two perturbations. And if we use the same data in the real world to construct these two Brownian motions, they are very likely to be correlated [111]. And there is a previous work focusing on correlation of Brownian motions in dynamic systems. Hu *et al.* [108] consider two correlated stochastic disturbances in the form of Gaussian white noise in an epidemic deterministic model constructed by Roberts

and Jowett [18]. Also, Hening and Nguyen [3] construct a stochastic Lotka-Volterra food chain system by introducing a number n of correlated Brownian motions into the deterministic food chain model, where n is the total species number in the food chain. They use a coefficient matrix to convert the vector of correlated Brownian motions to a vector of independent standard Brownian motions. Inspired by Emmerich [111], Hu *et al.* [108] and Hening [3], we are going to replace B_3 and B_4 by two correlated Brownian motions to introduce correlation of noises into the SIS epidemic model. Considering two correlated Brownian motions, one with linear diffusion coefficient and the other with Hölder continuous diffusion coefficient, is clearly different from other work on stochastic SIS models. Though Hölder continuous diffusion coefficients and correlations of white noises are often involved in stochastic financial and biological models [91], there is no related work based on the deterministic SIS model. As a result, in this chapter we aim to fill this gap.

We now consider B_3 and B_4 in our model (3.5) to be correlated. Replace B_3 and B_4 by correlated Brownian motions E_1 and E_2.

$$\begin{aligned} \mathrm{d}I(t) = [\beta(N - I(t))I(t) - (\mu + \gamma)I(t)]\mathrm{d}t + \sigma_1 I(t)(N - I(t))\mathrm{d}E_1(t) \\ - \sigma_2 I(t)\sqrt{N - I(t)}\mathrm{d}E_2(t). \end{aligned} \tag{4.1}$$

Note that E_1 and E_2 can be written as

$$(E_1, E_2)^T = A(B_1, B_2)^T,$$

where (B_1, B_2) is a vector of independent Brownian motions and A is the coefficient matrix where

$$A = \begin{bmatrix} a_1 & 0 \\ a_2 & a_3 \end{bmatrix}, \ a_1, a_2, a_3 \text{ are constants.}$$

Note that we do not set the coefficient matrix to be $A = \begin{bmatrix} a_1 & a_4 \\ a_2 & a_3 \end{bmatrix}$, where a_4 is also a constant. From the knowledge of Linear Algebra we can always find an appropriate pair of independent Brownian motions (B_1, B_2), which makes $a_4 = 0$ in order to eliminate one parameter. Hence we have

$$\mathrm{d}E_1(t) = a_1\mathrm{d}B_1(t), \ \mathrm{d}E_2(t) = a_2\mathrm{d}B_1(t) + a_3B_2(t). \tag{4.2}$$

Also, define the correlation of E_1 and E_2

$$\begin{aligned}\rho &= \frac{\mathrm{Cov}(E_1(t), E_2(t))}{\sqrt{\mathbb{V}\mathrm{ar}(E_1(t))\mathbb{V}\mathrm{ar}(E_2(t))}} \\ &= \frac{a_1 a_2}{\mid a_1 \mid \sqrt{a_2^2 + a_3^2}}\end{aligned} \tag{4.3}$$

with

$$0 < |\rho| < 1.$$

Note that when $\rho = 0$, E_1 and E_2 are independent Brownian motions.

Now substituting (4.2) into (4.1), we have

$$\begin{aligned}\mathrm{d}I(t) = &[\beta(N - I(t))I(t) - (\mu + \gamma)I(t)]\mathrm{d}t \\ &+ [a_1\sigma_1 I(t)(N - I(t)) - a_2\sigma_2 I(t)\sqrt{N - I(t)}]\mathrm{d}B_1(t) \\ &- a_3\sigma_2 I(t)\sqrt{N - I(t)}\mathrm{d}B_2(t),\end{aligned} \tag{4.4}$$

with initial value $I(0) = I_0 \in (0, N)$ and this is our new model. In the following sections, we will focus on the long-time properties of the solution to model (4.4). This chapter is also a published work in June 2019 in Nonlinear Dynamics [4].

4.2 Unique and Bounded Solution

We firstly want to know if the solution of our model (4.4) has a unique solution. Also, we need this solution to be positive and bounded within $(0, N)$ because it is meaningless for the number of the infected population to exceed the the number of whole population. So here we give **Theorem 4.2.1**.

Theorem 4.2.1. *If $\mu + \gamma \geq \frac{1}{2}(a_2^2 + a_3^2)\sigma_2^2 N$ and $a_1 a_2 > 0$, then for any given initial value $I(0) = I_0 \in (0, N)$, the SDE (4.4) has a unique global positive solution $I(t) \in (0, N)$ for all $t \geq 0$ with probability one, namely,*

$$\mathbb{P}\{I(t) \in (0, N),\ \forall t \geq 0\} = 1.$$

Proof. By the local Lipschitz condition, there must be an unique solution for our SDE (4.4) for any given initial value. So there is a unique maximal local solution

$I(t)$ on $t \in [0, \tau_e)$, where τ_e is the explosion time [91]. Let $k_0 \geq 0$ be sufficiently large to satisfy $\frac{1}{k_0} < I_0 < N - \frac{1}{k_0}$. For each integer $k \geq k_0$, define the stopping time

$$\tau_k = \inf\{t \in [0, \tau_e) \ : I(t) \notin (1/k, N - 1/k)\}.$$

Set $\inf\emptyset = \infty$. Clearly, τ_k is increasing when $k \to \infty$. And we set $\tau_\infty = \lim_{k\to\infty} \tau_k$. It is obvious that $\tau_\infty \leq \tau_e$ almost surely. So if we can show that $\tau_\infty = \infty$ a.s., then $\tau_e = \infty$ a.s. and $I(t) \in (0, N)$ a.s. for all $t \geq 0$.

Assume that $\tau_\infty = \infty$ is not true. Then we can find a pair of constants $T > 0$ and $\epsilon \in (0, 1)$ such that

$$\mathbb{P}\{\tau_\infty \leq T\} > \epsilon.$$

So we can find an integer $k_1 \geq k_0$ large enough, such that

$$\mathbb{P}\{\tau_k \leq T\} \geq \epsilon \quad \forall k \geq k_1. \tag{4.5}$$

Define a function $V : (0, N) \to \mathbb{R}_+$ by

$$V(x) = -\log x - \log(N - x) + \log\frac{N^2}{4},$$

and

$$V_x = -\frac{1}{x} + \frac{1}{N - x},\ V_{xx} = \frac{1}{x^2} + \frac{1}{(N - x)^2}.$$

Let $f(t) = \beta(N - I(t))I(t) - (\mu + \gamma)I(t)$, $g(t) = (a_1\sigma_1 I(t)(N - I(t)) - a_2\sigma_2\sqrt{N - I(t)}I(t), -a_3\sigma_2 I(t)\sqrt{N - I(t)})$ and $\mathrm{d}B(t) = (\mathrm{d}B_1(t), \mathrm{d}B_2(t))$. By Itô's formula [91], we have, for any $t \in [0, T]$ and any k

$$\mathbb{E}V(I(t \wedge \tau_k)) = V(I_0) + \mathbb{E}\int_0^{t\wedge\tau_k} LV(I(s))\mathrm{d}s + \mathbb{E}\int_0^{t\wedge\tau_k} V_x g(s)\mathrm{d}B(s), \tag{4.6}$$

where $\mathbb{E}\int_0^{t\wedge\tau_k} V_x g(s)\mathrm{d}B(s) = 0$. Also it is easy to show that

$$\begin{aligned}
LV(x) &= -\beta(N - x) + (\mu + \gamma) + \beta x - (\mu + \gamma)\frac{x}{N - x} \\
&\quad + \frac{1}{2}\left[\frac{1}{x^2} + \frac{1}{(N - x)^2}\right][a_1^2\sigma_1^2 x^2(N - x)^2 \\
&\quad + (a_2^2 + a_3^2)\sigma_2^2 x^2(N - x) - 2a_1a_2\sigma_1\sigma_2 x^2(N - x)^{\frac{3}{2}}] \\
&\leq -\beta(N - x) + (\mu + \gamma) + \beta x + \frac{1}{2}a_1^2\sigma_1^2(N - x)^2 + \frac{1}{2}a_1^2\sigma_1^2 x^2 + \frac{\mu + \gamma}{N}\sigma_2^2(N - x)
\end{aligned}$$

$$\leq C,$$

where C is a constant when $\mu + \gamma \geq \frac{1}{2}(a_2^2 + a_3^2)\sigma_2^2 N$, $a_1 a_2 > 0$ and $x \in (0, N)$.

Then we have

$$\begin{aligned} \mathbb{E}V(I(t \wedge \tau_k)) &\leq V(I_0) + \mathbb{E}\int_0^{t\wedge\tau_k} C\mathrm{d}s \\ &\leq V(I_0) + Ct, \end{aligned} \tag{4.7}$$

which yields that

$$\mathbb{E}V(I(T \wedge \tau_k)) \leq V(I_0) + CT. \tag{4.8}$$

Set $\Omega_k = \{\tau_k \leq T\}$ for $k \geq k_1$ and we have $\mathbb{P}(\Omega_k) \geq \epsilon$. For every $\omega \in \Omega_k$, $I(\tau_k, \omega)$ equals either $1/k$ or $N - 1/k$ and we have

$$V(I(\tau_k, \omega)) = \log \frac{N^2}{4(N - 1/k)1/k}.$$

Hence

$$\begin{aligned} \infty > V(I_0) + CT &\geq \mathbb{E}[\mathbb{I}_{\Omega_k}(\omega)V(I(\tau_k, \omega))] \\ &= \mathbb{P}(\Omega_k) \log \frac{N^2}{4(N - 1/k)1/k} \\ &\geq \epsilon \log \frac{N^2}{4(N - 1/k)1/k}, \end{aligned}$$

and letting $k \to \infty$ will lead to the contradiction

$$\infty > V(I_0) + CT = \infty.$$

□

So the assumption is not reasonable and we must have $\tau_\infty = \infty$ almost surely, whence the proof is now complete. Compared to the result from Gray *et al.* [1], the condition is now related to $(a_2^2 + a_3^2)$. The square root terms are the reasons for us to give this condition as when $N - I(t) \to 0$, $\sqrt{N - I(t)}$ changes rapidly. This can also be an explanation to the readers that the condition is dependent on a_2 and a_3.

4.3 Extinction

The previous section has already provided us with enough evidence that our model has a unique positive bounded solution. However we do not know under what circumstances the disease will die out or persist and this is of great importance in study of epidemic models. In this section, we will discuss the conditions for the disease to become extinction in our SDE model (4.4). Here we give **Theorem 4.3.1** and we will discuss persistence in the next section.

Theorem 4.3.1. *Given that the stochastic reproduction number of our model* $R_0^S := \frac{\beta N}{\mu+\gamma} - \frac{a_1^2\sigma_1^2N^2+(a_2^2+a_3^2)\sigma_2^2N-2a_1a_2\sigma_1\sigma_2N^{\frac{3}{2}}}{2(\mu+\gamma)} < 1$*, then for any given initial value* $I(0) = I_0 \in (0, N)$*, the solution of the SDE obeys*

$$\limsup_{t\to\infty} \frac{1}{t}\log I(t) < 0 \ a.s. \tag{4.9}$$

if one of the following conditions is satisfied

- $\frac{1}{2}(a_2^2+a_3^2)\sigma_2^2 \geq \beta$ *and* $a_1a_2 < 0$*;*
- $\frac{1}{2}(a_2^2+a_3^2)\sigma_2^2 \geq \beta + \frac{3}{2}a_1a_2\sigma_1\sigma_2\sqrt{N} - a_1^2\sigma_1^2N$ *and* $3a_2\sigma_2 \geq 4\sqrt{N}a_1\sigma_1$*;*
- $\frac{1}{2}(a_2^2+a_3^2)\sigma_2^2 < \beta \wedge (\beta + \frac{3}{2}a_1a_2\sigma_1\sigma_2\sqrt{N} - a_1^2\sigma_1^2N)$*;*
- $\frac{1}{2}(a_2^2+a_3^2)\sigma_2^2 \geq \beta + \frac{9}{16}a_2^2\sigma_2^2$.

namely, I(t) will almost surely trend to zero exponentially. And the disease will die out with probability one.

Proof. Here we use the Itô formula with $\tilde{V} = \log x$,

$$\frac{\log I(t)}{t} = \frac{\log I_0}{t} + \frac{1}{t}\int_0^t L\tilde{V}(I(s))\mathrm{d}s + \frac{1}{t}\int_0^t \frac{1}{I(s)}g(I(s))\mathrm{d}B(s), \tag{4.10}$$

and according to the large number theorem for martingales [91], we must have

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t \frac{1}{I(s)}g(I(s))\mathrm{d}B(s) = 0.$$

So if we want to prove $\limsup_{t\to\infty} \frac{1}{t}\log I(t) < 0$ almost surely, we need to find the conditions for $L\tilde{V}(x)$ to be strictly negative in $(0, N)$. $L\tilde{V}$ is defined by

$$\begin{aligned} L\tilde{V} &= \frac{1}{x}[\beta(N-x) - (\mu+\gamma)]x \\ &\quad - \frac{1}{2x^2}[a_1^2\sigma_1^2x^2(N-x)^2 + (a_2^2+a_3^2)\sigma_2^2x^2(N-x) - 2a_1a_2\sigma_1\sigma_2x^2(N-x)^{\frac{3}{2}}] \\ &= \beta(N-x) - (\mu+\gamma) - \frac{1}{2}a_1^2\sigma_1^2(N-x)^2 - \frac{1}{2}(a_2^2+a_3^2)\sigma_2^2(N-x) \\ &\quad + a_1a_2\sigma_1\sigma_2(N-x)^{\frac{3}{2}}. \end{aligned} \tag{4.11}$$

And it is clear that

$$L\tilde{V}(N) = -(\mu+\gamma) < 0,$$

and

$$L\tilde{V}(0) < 0,$$

is ensured by $R_0^S < 1$. However we do not know the behaviour of $L\tilde{V}$ in $(0, N)$ and it is no longer quadratic as (3.13) was, which makes it not easy to analyse. As a result, we derive the first derivative of $L\tilde{V}$.

$$\frac{\mathrm{d}L\tilde{V}}{\mathrm{d}x} = -\beta + a_1^2\sigma_1^2(N-x) + \frac{1}{2}(a_2^2+a_3^2)\sigma_2^2 - \frac{3}{2}a_1a_2\sigma_1\sigma_2\sqrt{N-x}. \tag{4.12}$$

This is a quadratic function of $z = \sqrt{N-x}$. So by assuming $D(z) = \frac{\mathrm{d}L\tilde{V}}{\mathrm{d}x}$, we have

$$D(z) = a_1^2\sigma_1^2z^2 - \frac{3}{2}a_1a_2\sigma_1\sigma_2z + \frac{1}{2}(a_2^2+a_3^2)\sigma_2^2 - \beta, \tag{4.13}$$

where $z \in (0, \sqrt{N})$. The axis of symmetry of (4.13) is $\hat{z} = \frac{3a_2\sigma_2}{4a_1\sigma_1}$.

Here we are going to discuss different cases for (4.13).

Case 1. If $\frac{1}{2}(a_2^2+a_3^2)\sigma_2^2 \geq \beta$ and $a_1a_2 < 0$ ($\hat{z} < 0$).

From the behaviour of the quadratic function (4.13), we know that the value of this function will be always positive in $(0, \sqrt{N})$. This means $L\tilde{V}$ increases when x increases. As $L\tilde{V}(N) < 0$, we have $L\tilde{V} \leq L\tilde{V}(N) < 0$. This leads to extinction. Although this condition contradicts with the condition to have a bounded solution, we still want to specific this result. Although we do not obtain a proof with $a_1a_2 < 0$ in the last section, it is still possible that the under some

condition with $a_1 a_2 < 0$, the solution of (4.4) is unique and bounded.

Case 2. If $\frac{1}{2}(a_2^2 + a_3^2)\sigma_2^2 \geq \beta$, $D(\sqrt{N}) \geq 0$ and $\hat{z} = \frac{3a_2\sigma_2}{4a_1\sigma_1} \geq \sqrt{N}$.

In this case, the value of $D(z)$ is always positive within $z \in (0, \sqrt{N})$, which leads to the similar result as in **Case 1**. So we have

$$\frac{1}{2}(a_2^2 + a_3^2)\sigma_2^2 \geq \beta + \frac{3}{2}a_1 a_2 \sigma_1 \sigma_2 \sqrt{N} - a_1^2 \sigma_1^2 N,$$

with $\hat{z} > \sqrt{N}$.

Case 3. If $\frac{1}{2}(a_2^2 + a_3^2)\sigma_2^2 < \beta$ and $D(\sqrt{N}) < 0$.

This condition makes sure that the value of $D(z)$ is strictly negative in $(0, \sqrt{N})$, which indicates that $L\tilde{V}$ decreases when x increases. With $L\tilde{V}(N) < 0$ and $L\tilde{V}(0) < 0$, this case results in extinction and we have

$$\frac{1}{2}(a_2^2 + a_3^2)\sigma_2^2 < \beta \wedge \left(\beta + \frac{3}{2}a_1 a_2 \sigma_1 \sigma_2 \sqrt{N} - a_1^2 \sigma_1^2 N\right).$$

Case 4. If $\Delta = \frac{9}{4}(a_1 a_2 \sigma_1 \sigma_2)^2 - 4a_1^2\sigma_1^2[\frac{1}{2}(a_2^2 + a_3^2)\sigma_2^2 - \beta] \leq 0$.

We have $\frac{1}{2}(a_2^2 + a_3^2)\sigma_2^2 \geq \beta + \frac{9}{16}a_2^2\sigma_2^2$. In this case $D(z)$ will be positive in $(0, \sqrt{N})$ so $L\tilde{V}$ increases when x increases. Similarly extinction still maintains in this case. □

In the deterministic SIS model, we have the result that if $R_0^D < 1$, the disease will die out. However from our results in this section, we can see that our stochastic reproduction number R_0^S is always less than the deterministic reproduction number $R_0^D = \frac{\beta N}{\mu+\gamma}$, which indicates that the noise and correlation in our model help expand the conditions of extinction. For those parameters that will not cause the dying out of disease in the deterministic SIS model, extinction will become possible if we consider the second perturbation (3.4) along with the correlation. On the other hand, the $L\tilde{V}$ is not linear after introducing the correlation, which causes the loss in the results. For example, from the behaviour of $D(z)$, we know that it is possible

that $L\tilde{V}$ can reach a negative maximum for an unknown $\hat{x} \in (0, N)$, which clearly leads to extinction. However, under this circumstance we are not able to derive the maximum to give a proper condition due to the nonlinear Itô operator.

4.4 Persistence

In this section, we firstly define persistence in this paper as there are many definitions in stochastic dynamic models to define persistence [1, 5, 91, 99, 100, 107, 109, 112]. However, our model (4.4) is based on [1]. As a result, we want to give a similar definition of persistence in our model (4.4). So here we give **Theorem 4.4.1** to give a condition for the solution of (4.4) oscillating around a positive level.

Theorem 4.4.1. *If $R_0^S > 1$, then for any given initial value $I(0) = I_0 \in (0, N)$, the solution of (4.4) follows*

$$\limsup_{t\to\infty} I(t) \geq \xi \text{ and } \liminf_{t\to\infty} I(t) \leq \xi \quad a.s. \tag{4.14}$$

ξ is the only positive root of $L\tilde{V} = 0$ in $(0, N)$. I(t) will be above or below the level ξ infinitely often with probability one.

Proof. When $R_0^S > 1$, recall (4.11) that if $\tilde{V} = \log x$

$$L\tilde{V} = \beta(N-x)-(\mu+\gamma)-\frac{1}{2}a_1^2\sigma_1^2(N-x)^2-\frac{1}{2}(a_2^2+a_3^2)\sigma_2^2(N-x)+a_1a_2\sigma_1\sigma_2(N-x)^{\frac{3}{2}}.$$

We have $L\tilde{V}(0) > 0$ which is guaranteed by $R_0^S > 1$, and $L\tilde{V}(N) = -(\mu+\gamma) < 0$. As $L\tilde{V}(x)$ is a continuous function in $(0, N)$, there must be a positive root of $L\tilde{V}(x) = 0$ in $(0, N)$. Moreover, as $L\tilde{V}(0) > 0$ and $L\tilde{V}(N) < 0$, there must be a subinterval of $(0, \sqrt{N})$ for z, where $D(z) < 0$. Consequently, as $D(z)$ is a quadratic function, there are only four possible situations as follows:

- $D(z) < 0$ which means that $L\tilde{V}(x)$ strictly decreases;

- $D(z)$ is initially positive then negative, which means that $\frac{\mathrm{d}L\tilde{V}}{\mathrm{d}x}$ is firstly negative then positive and $L\tilde{V}(x)$ firstly decreases to a negative minimum then increases to $L\tilde{V}(N) < 0$;
- $D(z)$ is initially negative then positive, which means that $\frac{\mathrm{d}L\tilde{V}}{\mathrm{d}x}$ is firstly positive then negative and $L\tilde{V}(x)$ firstly increases to a positive maximum and then decrease to $L\tilde{V}(N) < 0$;
- $D(z)$ is initially positive then negative, and finally positive, which means that $L\tilde{V}(x)$ increases to a positive maximum then decreases to a negative minimum, and finally increases to $L\tilde{V}(N) < 0$.

In all of those four cases, $L\tilde{V}(x) = 0$ will only have one unique positive root ξ in $(0, N)$. Hence $L\tilde{V}(x)$ will only have one unique positive root in $(0, N)$ when $R_0^S > 1$.

Here we recall (4.10)

$$\frac{\log I(t)}{t} = \frac{\log I_0}{t} + \frac{1}{t}\int_0^t L\tilde{V}(I(s))\mathrm{d}s + \frac{1}{t}\int_0^t \frac{1}{I(s)} g(I(s))\mathrm{d}B(s).$$

According to the large number theorem for martingales [91], there is an $\Omega_2 \subset \Omega$ with $\mathbb{P}\{\Omega_2\} = 1$ such that for every $\omega \in \Omega_2$

$$\lim_{t\to\infty} \frac{1}{t}\int_0^t \frac{1}{I(s)} g(I(s))\mathrm{d}B(s) = 0.$$

Now we assume that $\limsup_{t\to\infty} I(t) \geq \xi$ *a.s.* is not true. Then there must be a small $\epsilon \in (0, 1)$ such that

$$\mathbb{P}\{\limsup_{t\to\infty} I(t) \leq \xi - 2\epsilon\} > \epsilon. \tag{4.15}$$

Let $\Omega_1 = \{\limsup_{t\to\infty} I(t) \leq \xi - 2\epsilon\}$, then for every $\omega \in \Omega_1$, there exists $T = T(\omega)$ large enough, such that

$$I(t, \omega) \leq \xi - 2\epsilon + \epsilon = \xi - \epsilon, \text{ when } t \geq T(\omega),$$

which means when $t \geq T(\omega)$, $L\tilde{V}(I(t, \omega)) \geq L\tilde{V}(\xi - \epsilon)$. Then we have for any fixed $\omega \in \Omega_1 \cap \Omega_2$ and $t \geq T(\omega)$

$$\liminf_{t\to\infty} \frac{1}{t}\log I(t, \omega) \geq 0 + \lim_{t\to\infty} \frac{1}{t}\int_0^{T(\omega)} L\tilde{V}(I(s, \omega))\mathrm{d}s + \lim_{t\to\infty} \frac{1}{t} L\tilde{V}(\xi - \epsilon)(t - T(\omega))$$

$$\geq L\tilde{V}(\xi - \epsilon) > 0,$$

which yields

$$\lim_{t\to\infty} I(t,\omega) = \infty. \tag{4.16}$$

and this contradicts with $\omega \in \Omega_1$. So we must have $\limsup_{t\to\infty} I(t) \geq \xi$ almost surely.

Similarly, if we assume that $\liminf_{t\to\infty} I(t) \leq \xi$ *a.s.* is not true. Then there must be a small $\delta \in (0,1)$ such that

$$\mathbb{P}\{\liminf_{t\to\infty} I(t) \geq \xi + 2\delta\} > \delta. \tag{4.17}$$

Let $\Omega_3 = \{\liminf_{t\to\infty} I(t) \geq \xi + 2\delta\}$, then for every $\omega \in \Omega_3$, there exists $T' = T'(\omega)$ large enough, such that

$$I(t,\omega) \geq \xi + 2\delta - \delta = \xi + \delta, \text{ when } t \geq T'(\omega).$$

Now we fix any $\omega \in \Omega_3 \cap \Omega_2$ and when $t \geq T'(\omega)$, $L\tilde{V}(I(t,\omega)) \leq L\tilde{V}(\xi+\delta)$ and so we have

$$\begin{aligned}\limsup_{t\to\infty} \frac{1}{t}\log I(t,\omega) &\leq 0 + \lim_{t\to\infty}\frac{1}{t}\int_0^{T'(\omega)} L\tilde{V}(I(s,\omega))\mathrm{d}s + \lim_{t\to\infty}\frac{1}{t}L\tilde{V}(\xi+\delta)(t - T'(\omega)) \\ &\leq L\tilde{V}(\xi+\delta) < 0,\end{aligned}$$

which yields

$$\lim_{t\to\infty} I(t,\omega) = 0. \tag{4.18}$$

and this contradicts $\omega \in \Omega_3$. So we must have $\liminf_{t\to\infty} I(t) \leq \xi$ almost surely. Hence the proof is complete. □

Under the assumption of correlation between two white noises, this result is not weakened. We still can find a positive level, where the solution will oscillate around in long-term behaviour under the condition of persistence. However, we are not able to give the explicit form of this level ξ in this section because $L\tilde{V}$ is not linear. Although we cannot solve $L\tilde{V} = 0$ in this section, we will use computer simulation to help illustrate **Theorem 4.4.1** later in the simulation section.

4.5 Stationary Distribution

To find a stationary distribution of our SDE model (4.4) is of great important. We can also clearly see the existence of a stationary distribution from simulation results. In order to complete our proof, we need to initially use a well-known result from Khaminskii as a lemma [72].

Lemma 4.5.1. *The SDE model* (4.4) *has a unique stationary distribution if there is a strictly proper subinterval* (a, b) *of* $(0, N)$ *such that* $\mathbb{E}(\tau) < \infty$ *for all* $I_0 \in (0, a] \cup [b, N)$*, where*

$$\tau = \inf\{t \geq 0 : I(t) \in (a, b)\},$$

also,

$$\sup_{I_0 \in [\bar{a}, \bar{b}]} \mathbb{E}(\tau) < \infty,$$

for every interval $[\bar{a}, \bar{b}] \subset (0, N)$

Note that the other condition in Khasminskii's theory, which requires the sum of square of the diffusion coefficients to be bounded away from zero for all $I \in (a, b)$, is obvious for our model (4.4). Hence we do not need to point it out here. Now we give the following **Theorem 4.5.2** and the proof by using **Lemma 4.5.1**.

Theorem 4.5.2. *If* $R_0^S > 1$*, then our SDE model* (4.4) *has a unique stationary distribution.*

Proof. Firstly we need to fix any (a, b) such that,

$$0 < a < \xi < b < N,$$

and recall the discussion of $L\tilde{V}$ in last section, we can see that

$$L\tilde{V}(x) \geq L\tilde{V}(0) \wedge L\tilde{V}(a) > 0, \text{if } 0 < x \leq a, \tag{4.19}$$

$$L\tilde{V}(x) \leq L\tilde{V}(b) \vee L\tilde{V}(N) < 0, \text{if } b \leq x < N. \tag{4.20}$$

Also, recall (4.10)

$$\log I(t) = \log I_0 + \int_0^t L\tilde{V}(I(s))\mathrm{d}s + \int_0^t \frac{1}{I(s)} g(I(s))\mathrm{d}B(s),$$

and define

$$\tau = \inf\{t \geq 0 : I(t) \in (a, b)\}.$$

Step 1. We firstly look into the case when I_0 starts in $(0, a]$. Hence for all $t \geq 0$ and any $I_0 \in (0, a]$, using (4.19) in (4.10), we have

$$\begin{aligned}\mathbb{E}\log I(t \wedge \tau) &= \mathbb{E}\log I_0 + \mathbb{E}\int_0^{t\wedge\tau} L\tilde{V}(I(s))\mathrm{d}s + 0 \\ &\geq \log I_0 + \mathbb{E}(L\tilde{V}(0) \wedge L\tilde{V}(a))(t \wedge \tau).\end{aligned}$$

From the definition of τ, we know that

$$\log a \geq \mathbb{E}\log I(t \wedge \tau) \text{ when } I_0 \in (0, a].$$

Hence we have

$$\mathbb{E}(t \wedge \tau) \leq \frac{\log(\frac{a}{I_0})}{L\tilde{V}(0) \wedge L\tilde{V}(a)},$$

and when $t \to \infty$, we have

$$\mathbb{E}(\tau) \leq \frac{\log(\frac{a}{I_0})}{L\tilde{V}(0) \wedge L\tilde{V}(a)} < \infty, \forall I_0 \in (0, a].$$

Clearly this indicates the solution $I(t)$ will rise into (a, b) in finite time.

Step 2. Now similarly, we assume the solution starts in $[b, N)$. Consequently, for all $t \geq 0$ and any $I_0 \in [b, N)$, using (4.20) in (4.10), we have

$$\begin{aligned}\mathbb{E}\log I(t \wedge \tau) &= \mathbb{E}\log I_0 + \mathbb{E}\int_0^{t\wedge\tau} L\tilde{V}(I(s))\mathrm{d}s + 0 \\ &\leq \log I_0 + \mathbb{E}(L\tilde{V}(b) \vee L\tilde{V}(N))(t \wedge \tau).\end{aligned}$$

From the definition of τ, we know that

$$\log b \leq \mathbb{E}\log I(t \wedge \tau) \text{ when } I_0 \in [b, N).$$

Hence we have

$$\log b \leq \log I_0 + (L\tilde{V}(b) \vee L\tilde{V}(N))\mathbb{E}(t \wedge \tau),$$

rearranging we get

$$\mathbb{E}(t \wedge \tau) \leq -\frac{\log(\frac{b}{I_0})}{|\,(L\tilde{V}(b) \vee L\tilde{V}(N))\,|},$$

and when $t \to \infty$, we conclude

$$\mathbb{E}(\tau) \leq \frac{\log\left(\frac{I_0}{b}\right)}{\mid (\tilde{LV}(b) \vee \tilde{LV}(N)) \mid} < \infty, \quad \forall I_0 \in [b, N).$$

And this means our solution $I(t)$ will go down into (a, b) in finite time. Combining the results from both **Step 1** and **Step 2**, we complete the proof of **Theorem 4.5.2**. □

Now we need to give the mean and variance of the stationary distribution.

Theorem 4.5.3. *If $R_0^S > 1$ and denote m and v as the mean and variance of the stationary distribution of SDE model* (4.4)*. Then we have*

$$\beta v = (\beta N - \mu - \gamma)m - \beta m^2. \tag{4.21}$$

Proof. For any $I_0 \in (0, N)$, we firstly recall (4.4) in the integral form

$$\begin{aligned} I(t) = I_0 &+ \int_0^t [\beta(N - I(s))I(s) - (\mu + \gamma)I(s)]\mathrm{d}s \\ &+ \int_0^t [a_1\sigma_1 I(s)(N - I(s)) - a_2\sigma_2 I(s)\sqrt{N - I(s)}]\mathrm{d}B_1(s) \\ &- \int_0^t a_3\sigma_2 I(s)\sqrt{N - I(s)}\mathrm{d}B_2(s). \end{aligned}$$

Dividing both sides by t and when $t \to \infty$, applying the ergodic property of the stationary distribution [72] and also the large number theorem of martingales, we have the result that

$$0 = (\beta N - \mu - \gamma)m - \beta m_2,$$

where m, m_2 are the mean and second moment of the stationary distribution. So we have

$$0 = (\beta N - \mu - \gamma)m - \beta(v + m^2),$$

then rearrange to have

$$\beta v = (\beta N - \mu - \gamma)m - \beta m^2.$$

□

We have tried to get other equations of higher order moment of $I(t)$ in order to solve m and v but fail to get a result. This is also caused by the nonlinear $\L\tilde{V}$. In **Chapter 3**, we can formulate another equation of m and m_2 but it is clearly not applicable in this chapter. Though we do not have an explicit formula of the mean and the variance of the stationary distribution like [1], simulations can still be effective to prove (4.21).

4.6 Simulation

In this section, we use Euler-Maruyama method [1, 113, 114, 115] implemented in R to simulate the solutions in extinction, persistence and stationary distribution examples. A wide range of parameters and initial values have been randomly chosen to illustrate our theoretical results. Before we start to show the simulation results, we shall initially assume that the unit of time is one day and the population sizes are measured in units of 1 million, which is still the same as those in the previous chapter.

Firstly, for each case in extinction we initially give a complete set of parameters to satisfy not only the extinction conditions, but also $\mu + \gamma \geq \frac{1}{2}(a_2^2 + a_3^2)\sigma_2^2 N$ to make sure the uniqueness and boundedness of solutions. Also, both large and small initial values are used in all 4 cases for better illustration. We then choose the step size $\Delta = 0.001$ and plot the solutions with 5000 iterations.

Case 1.

$$N = 100,\ \beta = 0.4,\ \mu + \gamma = 45,\ \sigma_1 = 0.02,\ \sigma_2 = 0.95,$$

$$a_1 = 2, a_2 = -0.4, a_3 = 0.9, R_0^S < 1, \rho = -0.406138 \in (-1, 0).$$

Case 2.

$$N = 100,\ \beta = 0.4,\ \mu + \gamma = 45,\ \sigma_1 = 0.02,\ \sigma_2 = 0.95,$$

$$a_1 = 1.4, a_2 = 0.4, a_3 = 0.9, R_0^S < 1, \rho = 0.406138 \in (0, 1).$$

Case 3.

$$N = 100,\ \beta = 0.4,\ \mu + \gamma = 45,\ \sigma_1 = 0.02,\ \sigma_2 = 0.05,$$

$$a_1 = 0.8, a_2 = 0.5, a_3 = 1.6, R_0^S < 1.$$

Case 4.

$$N = 100,\ \beta = 0.4,\ \mu + \gamma = 45,\ \sigma_1 = 0.02,\ \sigma_2 = 0.9,$$

$$a_1 = 3, a_2 = 0.3, a_3 = 1, R_0^S < 1.$$

The simulation results (**Figure 4.1, 4.2, 4.3, 4.4**) are clearly supporting our theorem and illustrating the extinction of disease. Note that these conditions are not all the conditions for extinction. We only considered the situation that $D(z)$ is either strictly positive or strictly negative. Otherwise there will be much more complicated cases when $L\tilde{V}$ is not monotonic in $(0, N)$.

Then for the persistence examples, we choose the values of our parameter as following

$$N = 100,\ \beta = 0.5,\ \mu + \gamma = 45,\ \sigma_1 = 0.02,\ \sigma_2 = 0.05.$$

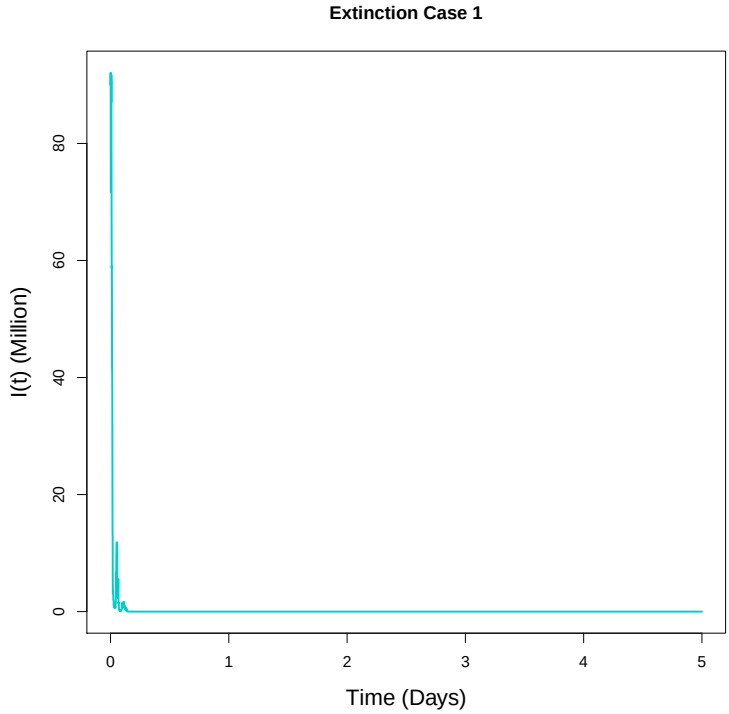
In order to prove the generality of our result, we use two sets of different $\{a_1, a_2, a_3\}$.

$$a_1 = 1, a_2 = 0.7, a_3 = 1.6, \rho_1 = 0.4008 > 0, R_0^S = 1.07375,$$

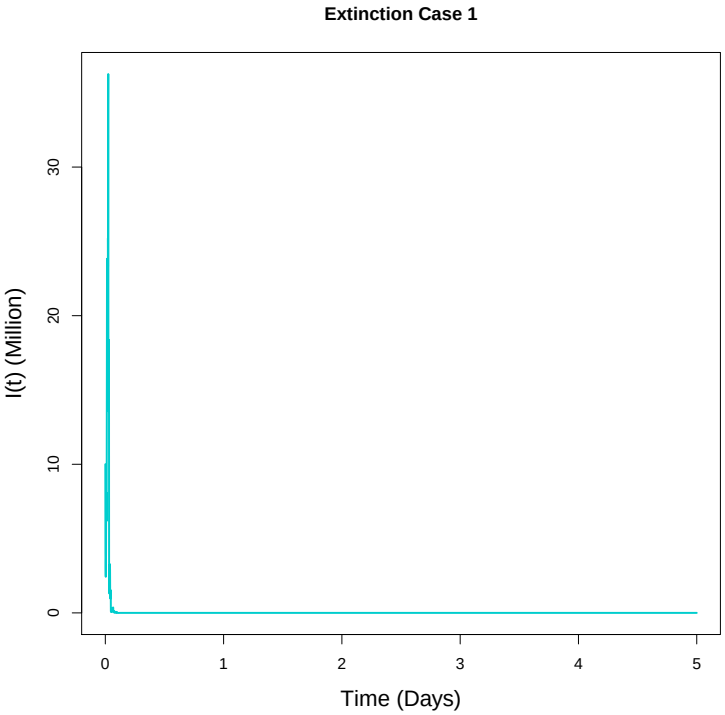
and

$$a_1 = 0.1, a_2 = 0.5, a_3 = 0.8, \rho_2 = 0.53 > 0, R_0^S = 1.1093056.$$

In both cases, we firstly use Newton-Raphson Method [116] in R to find a approximation to the roots ξ of both $L\tilde{V}$, which are 7.092595 and 9.852507 respectively. Then we use Euler-Maruyama method [1, 113] implemented in R to plot the solutions of our SDE with both large and small initial values, following by using red lines to indicate the level ξ. The step size is also 0.001 and 20,000 iterations is used in each example. In the following **Figure 4.5** and **Figure 4.6**, **Theorem 4.4.1.** is clearly illustrated and supported.

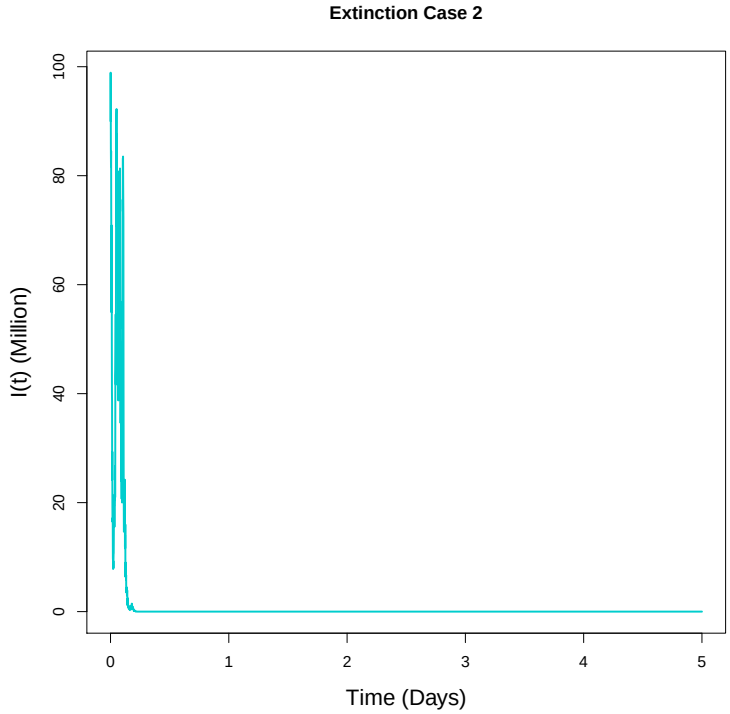


(a) $I(0) = 80$.

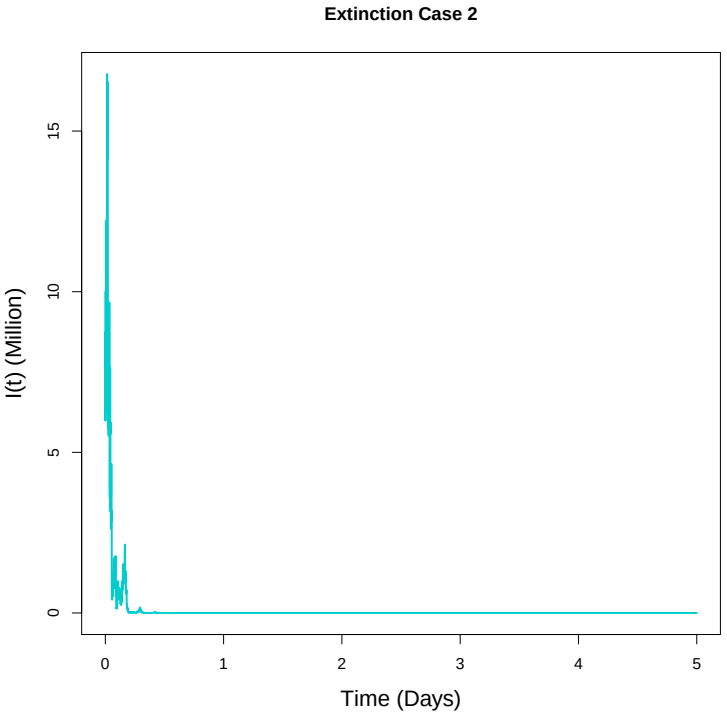


(b) $I(0) = 10$.

Figure 4.1: Extinction Case 1.

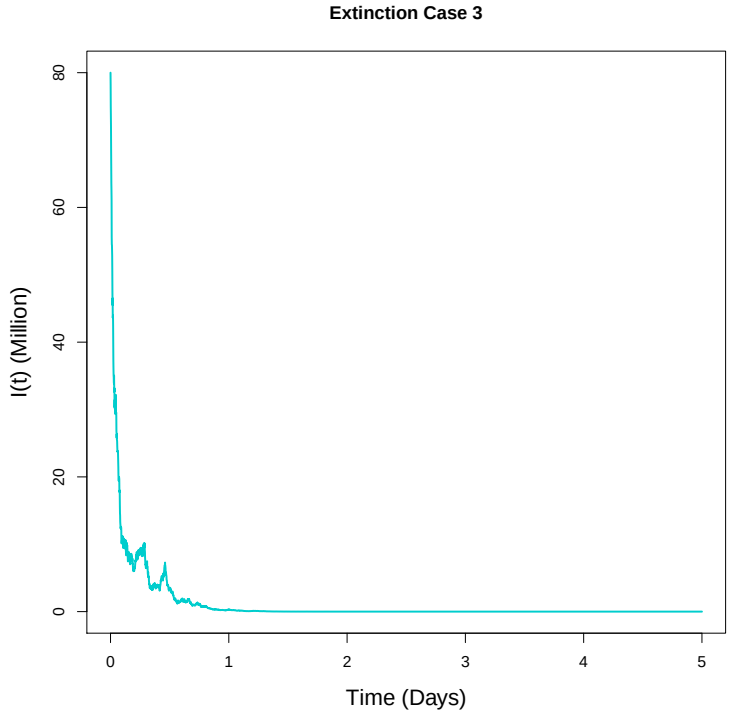


(a) $I(0) = 80$.

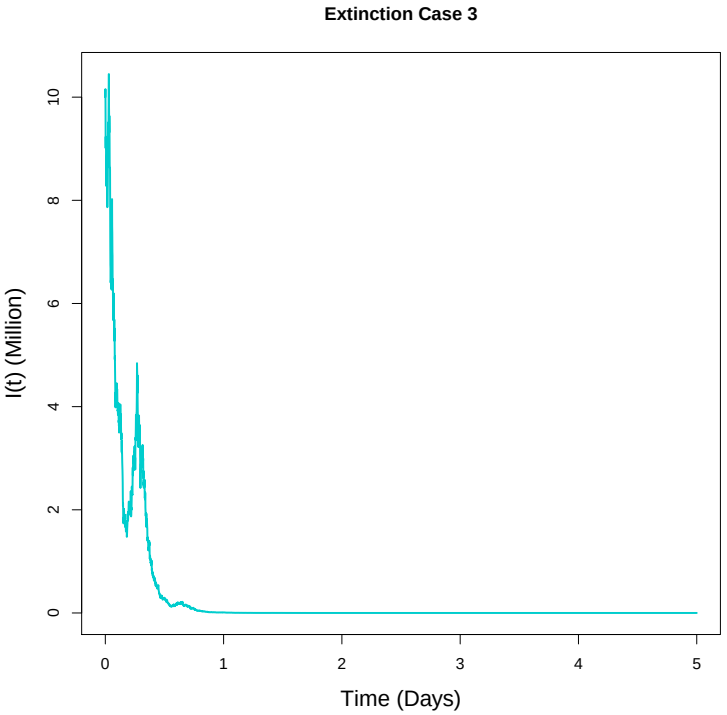


(b) $I(0) = 10$.

Figure 4.2: Extinction Case 2.

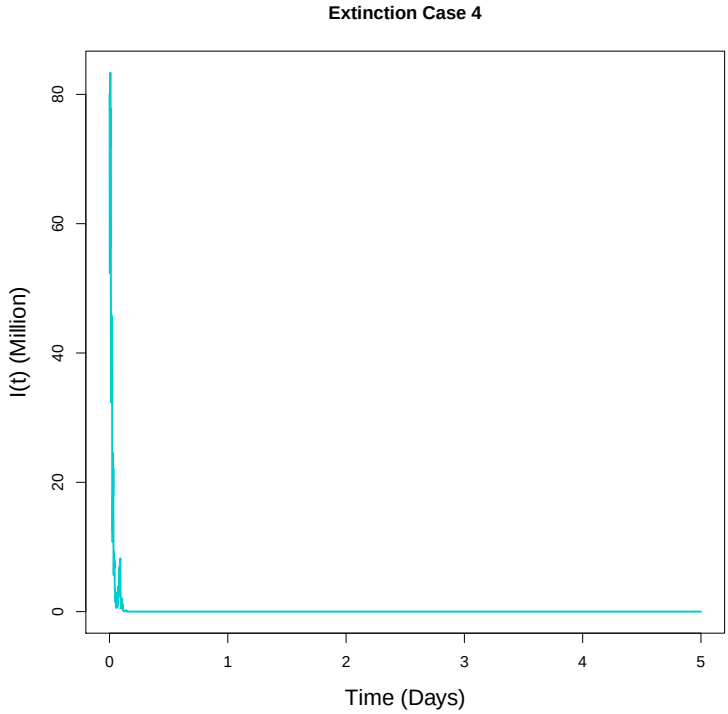


(a) $I(0) = 80$.

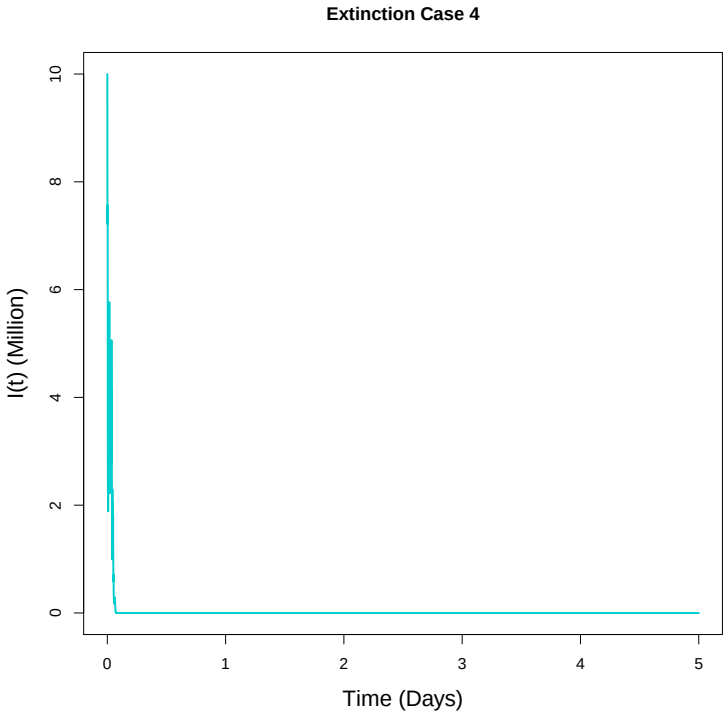


(b) $I(0) = 10$.

Figure 4.3: Extinction Case 3.

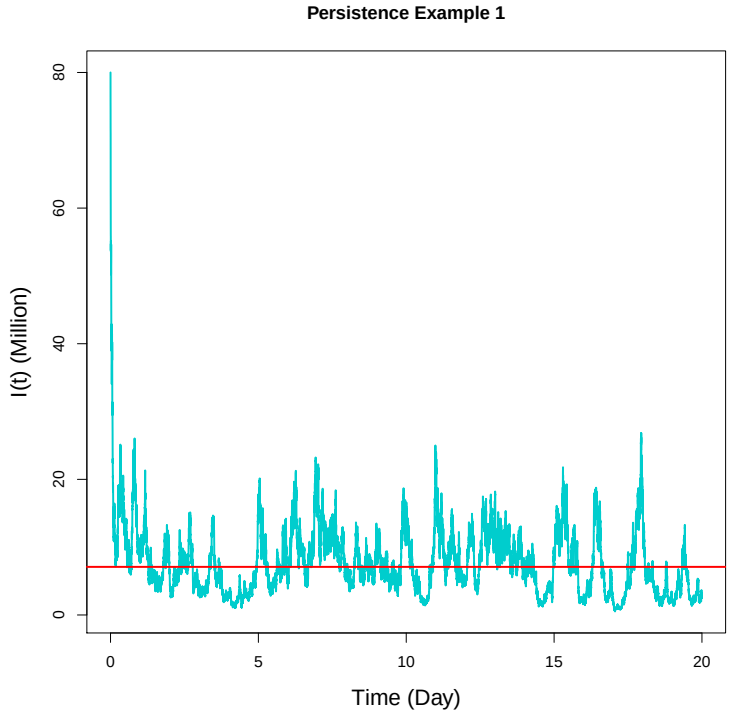


(a) $I(0) = 80$.

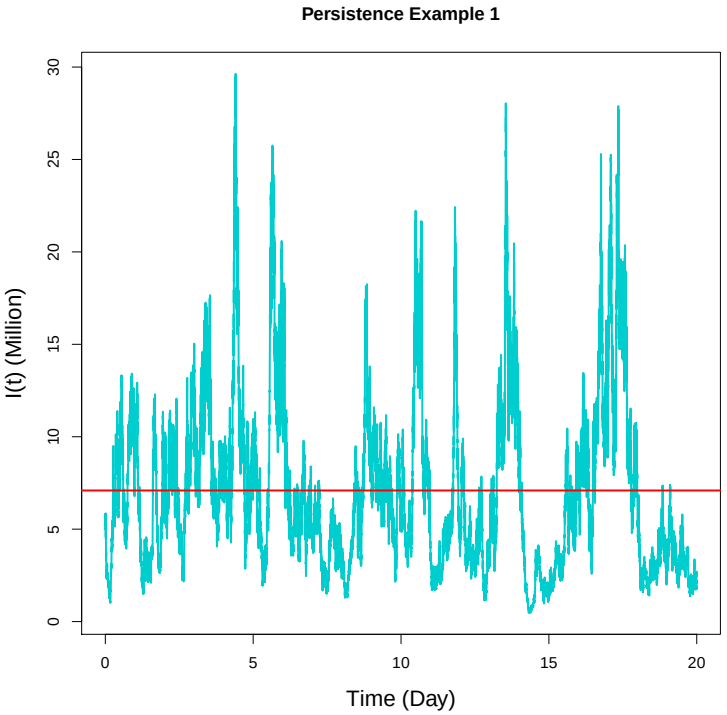


(b) $I(0) = 10$.

Figure 4.4: Extinction Case 4.

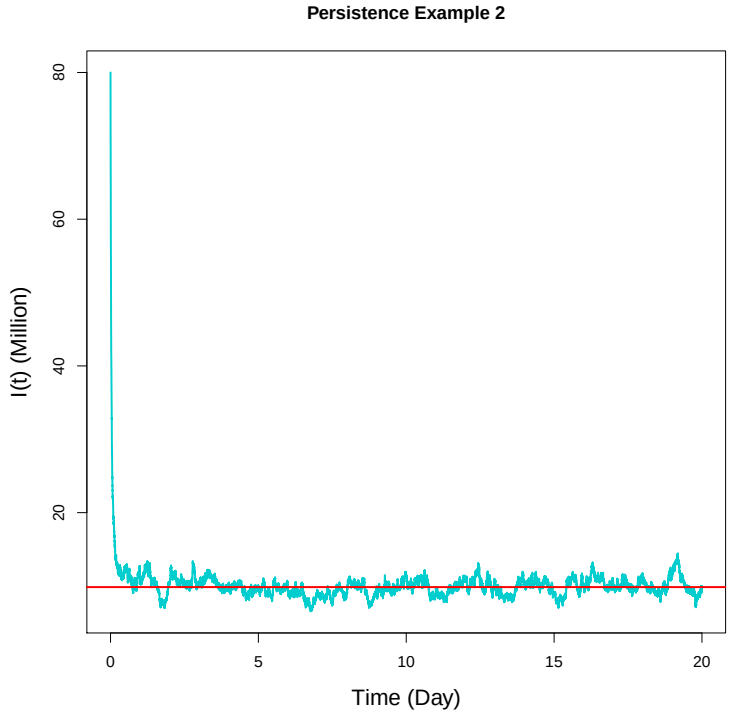


(a) $I(0) = 80$.

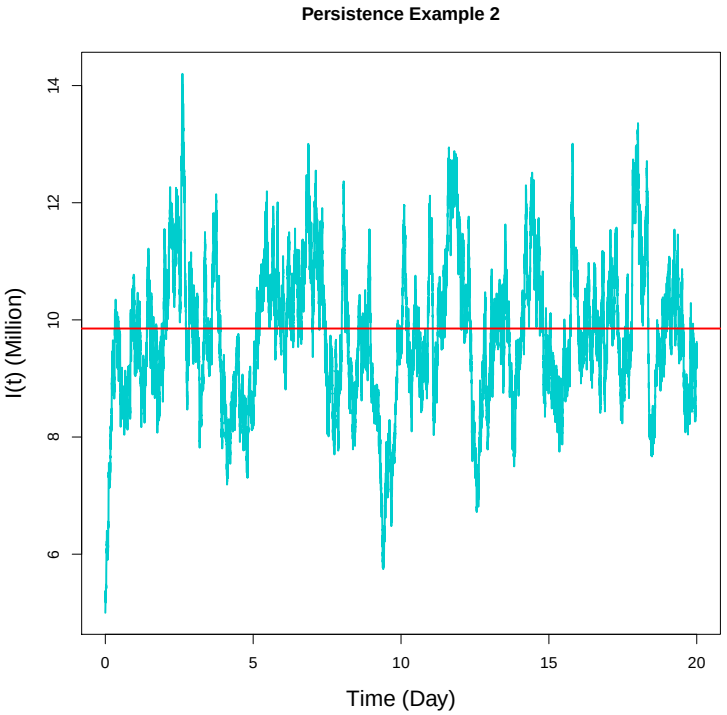


(b) $I(0) = 5$.

Figure 4.5: Persistence Example 1.

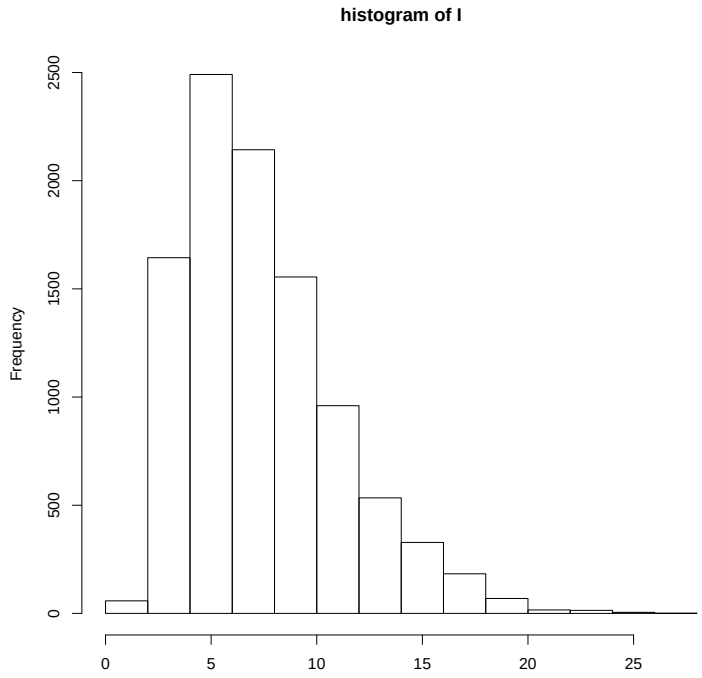


(a) $I(0) = 80$.

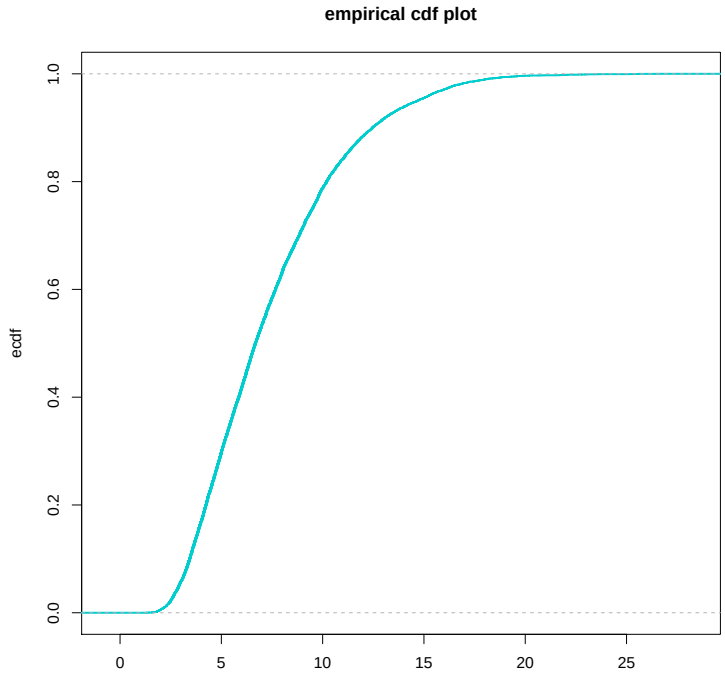


(b) $I(0) = 5$.

Figure 4.6: Persistence Example 2.

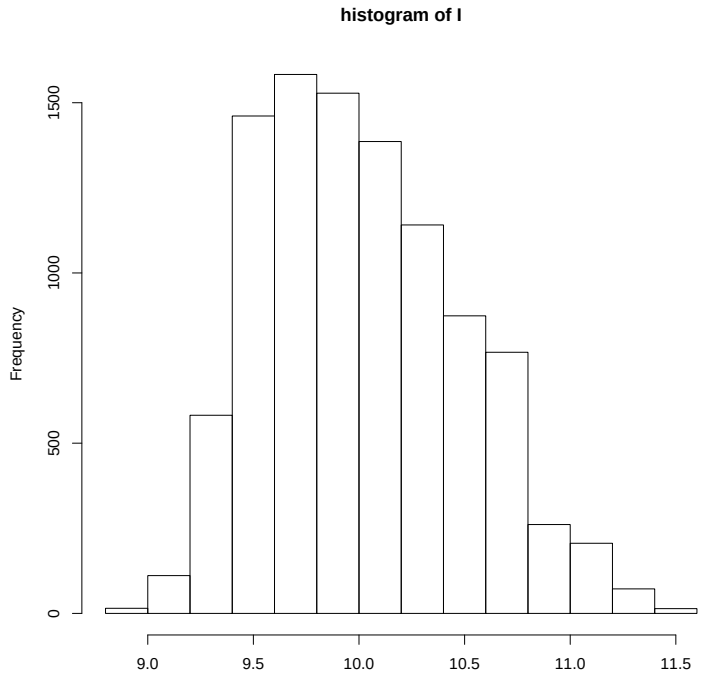


(a) histogram.

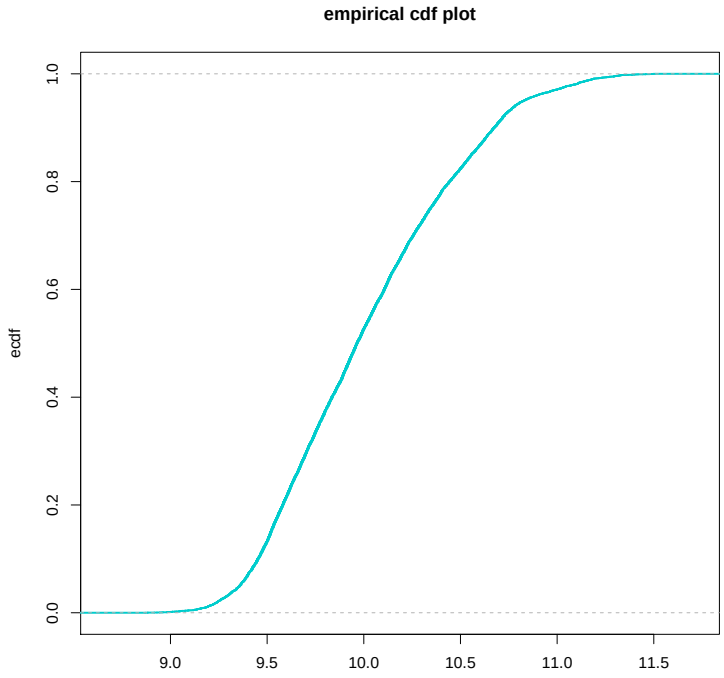


(b) ecdf.

Figure 4.7: Stationary Example 1.



(a) histogram.



(b) ecdf.

Figure 4.8: Stationary Example 2.

To generate stationary distribution examples, we use the following sets of our parameters in the persistence examples

$$N = 100,\ \beta = 0.5,\ \mu + \gamma = 45,\ \sigma_1 = 0.02,\ \sigma_2 = 0.01,$$

which have different values of $\{a_1, a_2, a_3\}$

$$a_1 = 1, a_2 = 0.7, a_3 = 1.6, \rho_1 = 0.4008 > 0, R_0^S = 1.07375,$$

and

$$a_1 = 0.1, a_2 = 0.5, a_3 = 0.8, \rho_2 = 0.53 > 0, R_0^S = 1.1093056.$$

Now we simulate the path of $I(t)$ for a long run of 200,000 iterations with step size 0.001 by using the Euler-Maruyama method. And we only reserve the last 10,000 iterations to illustrate the recurrent solution of our model. We also use the samples to calculate mean and variance of the two stationary distribution to see if they satisfied the equation (4.21). In both cases, the results of left side and right side of the equation (4.21) are 11.7632 and 11.7955, 0.1292906 and 0.1273215 respectively so we can conclude that the mean and variance of the stationary distribution, satisfy equation (4.21). **Figure 4.7 and 4.8** are the histograms and empirical cumulative distribution plots for each case of the last 10,000 iterations.

4.7 Summary

In this chapter, we generalize our model (3.5) in **Chapter 3** by replacing two independent Brownian motions with two correlated Brownian motions, which is inspired by Hening's work [3]. The reason of introducing correlation between noises is obvious: it is more suitable for real problems and complicated cases in epidemics. Results also confirm that we have stochastic reproduction number $R_0^S = \frac{\beta N}{\mu+\gamma} - \frac{a_1^2\sigma_1^2N^2+(a_2^2+a_3^2)\sigma_2^2N-2\rho\sigma_1\sigma_2N^{\frac{3}{2}}}{2(\mu+\gamma)}$ strictly smaller than the basic reproduction number, which is generalized from model (3.5). We also keep the strong persistence result and the unique stationary distribution in the new model (4.4), which indicates that involving correlation between the noises in the model does not change these results but expands them.

However, on the other hand, we lose the definitions of some features in the new model (4.4). For instance, in the extinction section, we know that there is one possible case that $L\tilde{V}$ has non-boundary negative maximum, which will result in the eradication of disease. However we are not able to give conditions depending on such a circumstance; in the persistence section, although we know that the solution will fluctuate around a positive level, indicating the persistence of the disease, we do not know exactly what this positive level is; in the section on the stationary distribution, despite the fact that we prove the existence of a unique stationary distribution when the disease persists in the population system, we only deduce one equation for the mean and variance. These are caused by the nonlinear $L\tilde{V} = \beta(N-x) - (\mu+\gamma) - \frac{1}{2}a_1^2\sigma_1^2(N-x)^2 - \frac{1}{2}(a_2^2+a_3^2)\sigma_2^2(N-x) + \rho\sigma_1\sigma_2(N-x)^{\frac{3}{2}}$ with $\tilde{V} = \log x$, or in other words, we are not able to find a proper Lyapunov function $V(I(t), t)$, such that the Itô operator is easily analysed.

Consequently, we still consider the model (4.4) in **Chapter 4** as a generalization of model (3.5) in **Chapter 3**, which can represent a more complicated epidemic problem in the real world. However, we would consider them separately because the results of model (3.5) are more complete.

Here we have completed our work in **Chapter 4**. In the next section, we will review our model (3.5) in a completely different direction, which is based on a previous work from Gray *et al.* on the stochastic SIS model with telegraph noise. A finite-state Markov chain will be initially explained, followed by analysis on the solution and simulation as well. Then we will compare the results of this new model in **Chapter 5** with our previous outcomes.

Chapter 5

SIS Epidemic Model with Regime Switching

5.1 Introduction

In the last two chapters, we have discussed the deterministic SIS model (3.2)

$$\mathrm{d}I(t) = [\beta(N - I(t))I(t) - (\mu + \gamma)I(t)]\mathrm{d}t,$$

with two perturbations (3.3)

$$\tilde{\beta}\mathrm{d}t = \beta\mathrm{d}t + \sigma_1\mathrm{d}B_1(t),$$

and (3.4)

$$(\tilde{\mu} + \tilde{\gamma})\mathrm{d}t = (\mu + \gamma)\mathrm{d}t + \sigma_2\sqrt{N - I(t)}\mathrm{d}B_2(t),$$

and we then have our SIS SDE model with two Brownian motions (3.5).

$$\begin{aligned}\mathrm{d}I(t) = [\beta(N - I(t))I(t) - (\mu + \gamma)I(t)]\mathrm{d}t + \sigma_1 I(t)(N - I(t))\mathrm{d}B_1(t)\\ - \sigma_2 I(t)\sqrt{N - I(t)}\mathrm{d}B_2(t).\end{aligned}$$

In our first model (3.5), B_1 and B_2 were considered initially as independent Brownian motions. And then we defined correlations between B_1 and B_2 to involve correlation between white noises.

However, not only white noise but also colour noise is used in classical deterministic epidemic models to describe different influence of environmental noise on population systems. For example, telegraph noise is a typical colour noise that has been studied widely in epidemic models. Telegraph noise can be illustrated as switching among different regimes, which can represent important information in the model such as change of seasons in a year, or different weathers [117]. If assuming that the future switching is only based on current state and the waiting time for the next switching has exponential distribution, we can use a a finite-state Markov chain to describe such behaviour.

There is much previous research based on using Markovian switching in stochastic epidemic models to study the effect of telegraph noise. For instance, Greenhalgh *et al.* [60] introduce telegraph noise in SIRS model by using a two-state Markov chain to study the asymptotic behaviour of the solution. Mao [118] studies the stationary distribution of SDE Lotka-Volterra systems under telegraph noise. Also, Zhang *et al.* [119] consider a regime switching which includes both white noise and telegraph noise in an SIS model under vaccination. Furthermore, Liu and Wang [120] extend the classical theory of SDEs with single Markovian switching by introducing a finite-state multi-Markovian switching.

Note that based on [5, 121], Gray *et al.* also construct a stochastic SIS model with two-state Markov chain.

$$\frac{I(t)}{\mathrm{d}t} = I(t)[\alpha_{r(t)} - \beta_{r(t)}I(t)], \tag{5.1}$$

where $\alpha_i = \beta_i N - \mu_i - \gamma_i$, i is the Markov chain state. Hence in this section, we are going to consider a finite-state Markov chain in our model (3.5) to involve the effect of telegraph noise in our model.

Now firstly we need to define the Markov chain. Let $r(t)$, $t \geq 0$, be a right-continuous M-state Markov chain on the probability space. $r(t)$ only takes value in a finite state space $\mathbb{S} = \{1, 2, ..., M\}$, with generator $\Gamma = (\nu_{ij})_{M\times M}$ defined as

$$\mathbb{P}\{r(t+\delta) = j \mid r(t) = i\} = \begin{cases} \nu_{ij}\delta + o(\delta), \text{ if } i \neq j, \\ 1 + \nu_{ij}\delta + o(\delta), \text{ if } i = j, \end{cases} \tag{5.2}$$

where $\delta > 0$ and $\nu_{ij} \geq 0$ is the transition rate from state i to j for $i \neq j$. Note that

$\nu_{ii} = -\Sigma_{1\leq j\leq M,\ j\neq i}\nu_{ij}$. And Almost every sample path of $r(\cdot)$ is a right-continuous step function with a finite number of sample jumps in any finite subinterval of $\mathbb{R} = [0, \infty)$ [95]. To be specific, there is a sequence $\{\tau_k\}_{k\geq 0}$ of finite-valued $\mathcal{F}_t$-stopping times such that $0 = \tau_0 < \tau_1 < ... < \tau_k \to \infty$ a.s. and

$$r(t) = \sum_{k=0}^{\infty} r(\tau_k)\mathbf{1}_{[\tau_k,\tau_{k+1})}(t), \tag{5.3}$$

where $\mathbf{1}_A$ denotes the indicator function of set A. Also, we define $\Pi = (\pi_1, \pi_2, ..., \pi_M)$ to be the unique stationary distribution of this Markov chain and $\sum_1^M \pi_i = 1$. Now we suppose that in the SIS epidemic model (3.5) the parameters $\mu_i,\ \beta_i,\ \gamma_i,\ \sigma_{1,i},\ \sigma_{2,i}$ are all positive numbers ($i \in \mathbb{S}$). Then we have our previous stochastic SIS SDE model (3.5) with Markovian switching given by

$$\begin{aligned} \mathrm{d}I(t) = [\beta_{r(t)}(N - I(t))I(t) - (\mu_{r(t)} + \gamma_{r(t)})I(t)]\mathrm{d}t + \sigma_{1,r(t)}I(t)(N - I(t))\mathrm{d}B_1(t) \\ - \sigma_{2,r(t)}I(t)\sqrt{N - I(t)}\mathrm{d}B_2(t), \end{aligned} \tag{5.4}$$

with $I_0 \in (0, N)$ and $r(0) = r_0 \in \mathbb{S}$. Also, B_1 and B_2 are independent Brownian motions.

5.2 Unique and Bounded Solution

In order for the model to make sense, we need to prove that the solution of our SDE has a unique global solution which remains within $(0, N)$, with the initial value $I_0 \in (0, N)$, $r(0) = r_0 \in \mathbb{S}$.

Theorem 5.2.1. *If* $\min\left\{\frac{2(\mu_i+\gamma_i)}{\sigma_{2,i}^2}\right\} \geq N$, *for all* $i \in \mathbb{S}$, *then for any given initial value* $I(0) = I_0 \in (0, N)$ *and* $r(0) = r_0 \in \mathbb{S}$, *the SDE has a unique global positive solution* $I(t) \in (0, N)$ *for all* $t \geq 0$ *with probability one, namely,*

$$\mathbb{P}\{I(t) \in (0, N),\ \forall t \geq 0\} = 1$$

Proof. It is obvious that for any $i \in \mathbb{S}$, the corresponding coefficients of our SDE (5.4) are locally Lipschitz continuous. And for any k and τ_k defined as (5.3),

our solution of the equation (5.4) is uniquely determined on $t \in [\tau_k, \tau_{k+1})$, with $r(\tau_k) = i_k \in \mathbb{S}$. As a result, we see that the equation (5.4) has a unique solution on $t \in \mathbb{R}_+$.

So now for any given initial value, there is a unique maximal local solution $I(t)$ on $t \in [0, \tau_e)$, where τ_e is the explosion time [99]. Let $k_0 \geq 0$ be sufficiently large to satisfy $\frac{1}{k_0} < I_0 < N - \frac{1}{k_0}$. For each integer $k \geq k_0$, define the stopping time

$$\tau_k = \inf\{t \in [0, \tau_e) \ : I(t) \notin (1/k, N - 1/k)\}$$

In this chapter, we set $\inf\emptyset = \infty$. Obviously, τ_k is increasing when $k \to \infty$. And we set $\tau_\infty = \lim_{k\to\infty} \tau_k$. It is clear that $\tau_\infty \leq \tau_e$ almost sure. So if we can show that $\tau_\infty = \infty$ a.s., then $\tau_e = \infty$ a.s. and $I(t) \in (0, N)$ a.s. for all $t \geq 0$.

Here we assume $\tau_\infty = \infty$ a.s. is not true. Then we can find a pair of constants $T > 0$ and $\epsilon \in (0, 1)$ such that

$$\mathbb{P}\{\tau_\infty \leq T\} > \epsilon.$$

So we can find an integer $k_1 \geq k_0$ large enough, such that

$$\mathbb{P}\{\tau_k \leq T\} \geq \epsilon \quad \forall k \geq k_1. \tag{5.5}$$

Define a function $V : (0, N) \to \mathbb{R}_+$ by

$$V(x) = -\log x - \log(N - x) + \log \frac{N^2}{4},$$

which is independent of the Markov chain $r(t)$, and

$$V_x = -\frac{1}{x} + \frac{1}{N - x}, \ V_{xx} = \frac{1}{x^2} + \frac{1}{(N - x)^2}.$$

We let $f(t) = \beta_{r(t)}(N - I(t))I(t) - (\mu_{r(t)} + \gamma_{r(t)})I(t)$, $g(t) = (\sigma_{1,r(t)}I(t)(N - I(t)), -\sigma_{2,r(t)}\sqrt{N - I(t)}I(t))$ and $\mathrm{d}B(t) = (\mathrm{d}B_1(t), \mathrm{d}B_2(t))$. Then by Itô's formula [99], we have, for any $t \in [0, T]$ and $k \geq k_1$

$$\mathbb{E}V(I(t \wedge \tau_k)) = V(I_0) + \mathbb{E}\int_0^{t\wedge\tau_k} LV(I(s))\mathrm{d}s + \mathbb{E}\int_0^{t\wedge\tau_k} V_x g(s)\mathrm{d}B(s), \tag{5.6}$$

where $\mathbb{E}\int_0^{t\wedge\tau_k} V_x g(s)\mathrm{d}B(s) = 0$. Also it is easy to show that

$$LV(x, i) = -\beta_i(N - x) + (\mu_i + \gamma_i) + \beta_i x - (\mu_i + \gamma_i)\frac{x}{N - x}$$

$$
\begin{aligned}
&+\frac{1}{2}\left(\sigma_{1,i}^2(N-x)^2+\sigma_{1,i}^2x^2+\sigma_{2,i}^2(N-x)+\sigma_{2,i}^2\frac{x^2}{N-x}\right)\\
&\leq -\beta_i(N-x)+(\mu_i+\gamma_i)+\beta_i x\\
&+\frac{1}{2}[\sigma_{1,i}^2(N-x)^2+\sigma_{1,i}^2x^2+\sigma_{2,i}^2(N-x)]\\
&\leq C, \qquad (5.7)
\end{aligned}
$$

where C is a constant when $\mu_i+\gamma_i \geq \frac{1}{2}\sigma_{2,i}^2 N$ for all $i \in \mathbb{S}$ and $x \in (0,N)$. Hence when $\min\{\frac{2(\mu_i+\gamma_i)}{\sigma_{2,i}^2}\} \geq N$, we have

$$
\begin{aligned}
\mathbb{E}V(I(t\wedge\tau_k)) &\leq V(I_0)+\mathbb{E}\int_0^{t\wedge\tau_k} C\mathrm{d}s\\
&\leq V(I_0)+Ct, \qquad (5.8)
\end{aligned}
$$

which yields that

$$
\mathbb{E}V(I(T\wedge\tau_k)) \leq V(I_0)+CT. \qquad (5.9)
$$

Now set $\Omega_k=\{\tau_k \leq T\}$ for $\forall k \geq k_1$ and we have $\mathbb{P}(\Omega_k) \geq \epsilon$. For every $\omega \in \Omega_k$, $I(\tau_k,\omega)$ equals either $1/k$ or $N-1/k$ and we have

$$
V(I(\tau_k,\omega)) = \log\frac{N^2}{4(N-1/k)1/k}.
$$

Hence

$$
\begin{aligned}
\infty > V(I_0)+CT &\geq \mathbb{E}[\mathbf{1}_{\Omega_k}(\omega)V(I(\tau_k,\omega))]\\
&\geq \mathbb{P}(\Omega_k)\log\frac{N^2}{4(N-1/k)1/k}\\
&= \epsilon\log\frac{N^2}{4(N-1/k)1/k}.
\end{aligned}
$$

Let $k\to\infty$ will lead to the contradiction

$$
\infty > V(I_0)+CT = \infty.
$$

So the assumption is wrong and we must have $\tau_\infty=\infty$ almost surely, whence the proof is now complete. □

The result is very similar to **Theorem 3.2.1** as we are not able to find a better substitution at this moment. However, in the following sections we will manage to

give conditions by using the ergodic theory of the Markov chain [95, 122]. Those results will be stated in an average-type form which combined the parameters in each state with its corresponding Markov chain stationary distribution π_i, $i \in \mathbb{S}$. This will let us no longer examine the solution state by state but as a whole.

5.3 Extinction

In the study of stochastic epidemic models, the extinction of disease is usually one of the most crucial issues. So similarly, in this section, we will firstly give an almost sure extinction condition for the disease to die out.

Theorem 5.3.1. *Given that* $R_0^S = \frac{\sum \pi_i \beta_i N}{\sum \pi_i (\mu_i + \gamma_i)} - \frac{\sum \pi_i (\sigma_{1,i}^2 N^2 + \sigma_{2,i}^2 N)}{2 \sum \pi_i (\mu_i + \gamma_i)} < 1$, *then for any given initial value* $I(0) = I_0 \in (0, N)$ *and* $r(0) = r_0 \in \mathbb{S}$, *the solution of 5.4 obeys*

$$\limsup_{t \to \infty} \frac{1}{t} \log I(t) < 0 \ a.s.,$$

if we have

$$\sum \pi_i \left[\frac{1}{2} \frac{(\beta_i - \frac{1}{2}\sigma_{2,i}^2)^2}{\sigma_{1,i}^2} - (\mu_i + \gamma_i) \right] < 0.$$

Proof. Define a function by

$$\tilde{V}(x, i) = \log x, x \in (0, N),$$

and we have

$$\tilde{V}_x(x, i) = \frac{1}{x}, \tilde{V}_{xx}(x, i) = -\frac{1}{x^2},$$

which are independent of the Markov chain state i. So by Itô's formula, we then obtain

$$\frac{\log I(t)}{t} = \frac{\log I_0}{t} + \frac{1}{t} \int_0^t L\tilde{V}(I(s)) \mathrm{d}s + \frac{1}{t} \int_0^t \sigma_{1,i}(N - I(s)) \mathrm{d}B_1(s) - \frac{1}{t} \int_0^t \sigma_{2,i} \sqrt{(N - I(s))} \mathrm{d}B_2(s), \quad (5.10)$$

where $L\tilde{V}$ is defined by

$$L\tilde{V}(x, i) = \beta_i(N - x) - (\mu_i + \gamma_i) - \frac{1}{2}[\sigma_{1,i}^2(N - x)^2 + \sigma_{2,i}^2(N - x)], x \in (0, N). \quad (5.11)$$

When $t \to \infty$ and according to the large number theorem for martingales [91, 99] and the ergodic theory of Markov chain, from the given conditions, we must have

$$\begin{aligned}\limsup_{t\to\infty} \frac{1}{t}\log I(t) &= \limsup_{t\to\infty} \frac{1}{t}\int_0^t L\tilde{V}(I(s))\mathrm{d}s \\ &\leq \lim_{t\to\infty}\int_0^t \left[\frac{1}{2}\frac{(\beta_i - \frac{1}{2}\sigma_{2,i}^2)^2}{\sigma_{1,i}^2} - (\mu_i+\gamma_i)\right]\mathrm{d}s \\ &= \sum \pi_i \left[\frac{1}{2}\frac{(\beta_i - \frac{1}{2}\sigma_{2,i}^2)^2}{\sigma_{1,i}^2} - (\mu_i+\gamma_i)\right] < 0,\end{aligned}$$

as required. □

From the conditions $\sum \pi_i \left[\frac{1}{2}\frac{(\beta_i-\frac{1}{2}\sigma_{2,i}^2)^2}{\sigma_{1,i}^2} - (\mu_i+\gamma_i)\right] < 0$, we can see that our extinction in the solution of (5.4) does not require every state to be extinction. In some states i, we can have $\frac{1}{2}\frac{(\beta_i-\frac{1}{2}\sigma_{2,i}^2)^2}{\sigma_{1,i}^2} - (\mu_i+\gamma_i) > 0$ which may result in persistence during those typical states according to **Theorem 3.3.1**. As long as the average-type condition is satisfied, extinction can be approached.

5.4 Persistence

In this section, we are going to give conditions for persistence. Though there are many different definitions in persistence, we want to find a condition of oscillating around a positive level like **Theorem 3.4.1**. Hence we give the following theorem.

Theorem 5.4.1. *If* $R_0^S = \frac{\sum \pi_i\beta_i N}{\sum \pi_i(\mu_i+\gamma_i)} - \frac{\sum \pi_i(\sigma_{1,i}^2N^2+\sigma_{2,i}^2N)}{2\sum \pi_i(\mu_i+\gamma_i)} > 1$, *then for any given initial value* $I(0) = I_0 \in (0, N)$ *and* $r(0) = r_0 \in \mathbb{S}$, *the solution of (5.4) follows*

$$\limsup_{t\to\infty} I(t) \geq \xi \text{ and } \liminf_{t\to\infty} I(t) \leq \xi \quad a.s., \tag{5.12}$$

where ξ *is the only positive root of* $\mathcal{K}(x) = 0$ *in* $x \in (0, N)$

$$\begin{aligned}\mathcal{K}(x) = &\sum \pi_i \left[\beta_i N - (\mu_i+\gamma_i) - \frac{1}{2}\sigma_{1,i}^2N^2 - \frac{1}{2}\sigma_{2,i}^2N\right] \\ &+ \sum \pi_i(\sigma_{1,i}^2N + \frac{1}{2}\sigma_{2,i}^2 - \beta_i)x - \frac{1}{2}\sum \pi_i\sigma_{1,i}^2x^2.\end{aligned} \tag{5.13}$$

In other words, $I(t)$ *will be above or below the level* ξ *infinitely often with probability one.*

Proof. From $R_0^S > 1$, we have $\mathcal{K}(0) = \sum \pi_i[\beta_i N - (\mu_i + \gamma_i) - \frac{1}{2}\sigma_{1,i}^2 N^2 - \frac{1}{2}\sigma_{2,i}^2 N] > 0$ and $\mathcal{K}(N) = -\sum \pi_i(\mu_i + \gamma_i) < 0$. So as a quadratic function, $\mathcal{K}(x)$ must have only one positive root in $(0, N)$. To begin the proof, we firstly assume that $\limsup_{t\to\infty} I(t) \geq \xi$ a.s. were not true. Then we can find a small $\epsilon > 0$ for $\mathbb{P}(\Omega_1) > \epsilon$ where

$$\Omega_1 = \{\omega \in \Omega : \limsup_{t\to\infty} I(t) < \xi - 2\epsilon\}.$$

Also by the ergodic theory [95, 99], we have $\mathbb{P}(\Omega_2) = 1$, where for any $\omega \in \Omega_2$, such that

$$\lim_{t\to\infty} \frac{1}{t}\int_0^t L\tilde{V}(\xi - \epsilon)\mathrm{d}s = \mathcal{K}(\xi - \epsilon) > \mathcal{K}(\xi) = 0.$$

So for any $\omega \in \Omega_1 \cap \Omega_2$, there is a positive $T = T(\omega)$, such that $\forall t \geq T$

$$I(t) \leq \xi - \epsilon.$$

Then we must have

$$\begin{aligned}\liminf_{t\to\infty} \frac{1}{t}\log I(t,\omega)\mathrm{d}s &\geq \liminf_{t\to\infty} \frac{1}{t}\log(I_0)\mathrm{d}s + \liminf_{t\to\infty} \frac{1}{t}\int_0^T L\tilde{V}(I(s,\omega))\mathrm{d}s + \mathcal{K}(\xi - \epsilon) \\ &> 0.\end{aligned}$$

This implies that

$$\lim_{t\to\infty} I(t,\omega) \to \infty, \tag{5.14}$$

which contradicts our previous assumption. Therefore $\limsup_{t\to\infty} I(t) \geq \xi$ a.s. must hold.

Similarly if we assume that $\liminf_{t\to\infty} I(t) \leq \xi$ a.s. were not true, then we can find a small $\delta > 0$ for $\mathbb{P}(\Omega_3) > \delta$ where

$$\Omega_3 = \{\omega \in \Omega : \liminf_{t\to\infty} I(t) > \xi + 2\delta\}.$$

Also we have

$$\lim_{t\to\infty} \frac{1}{t}\int_0^t L\tilde{V}(\xi + \delta)\mathrm{d}s = \mathcal{K}(\xi + \delta) < \mathcal{K}(\xi) = 0,$$

for any $\omega \in \Omega_3 \cap \Omega_2$. Hence there is a positive $T = T(\omega)$, such that $\forall t \geq T$

$$I(t) \geq \xi + \delta.$$

Then we have that

$$\limsup_{t\to\infty}\frac{1}{t}\log I(t,\omega)\mathrm{d}s \le \limsup_{t\to\infty}\frac{1}{t}\log(I_0)\mathrm{d}s + \limsup_{t\to\infty}\frac{1}{t}\int_0^T L\tilde{V}(I(s,\omega))\mathrm{d}s + \mathcal{K}(\xi+\delta)$$
$$< 0.$$

This implies that

$$\lim_{t\to\infty} I(t,\omega) \to 0, \tag{5.15}$$

which contradicts our previous assumption again. Therefore $\liminf_{t\to\infty} I(t) \le \xi$ a.s. must hold. □

Similarly we can see from the result that $R_0^S = \frac{\sum \pi_i\beta_i N}{\sum \pi_i(\mu_i+\gamma_i)} - \frac{\sum \pi_i(\sigma_{1,i}^2N^2+\sigma_{2,i}^2N)}{2\sum \pi_i(\mu_i+\gamma_i)} > 1$, we have $\sum \pi_i[\beta_i - (\mu_i+\gamma_i) - \frac{1}{2}(\sigma_{1,i}^2N^2+\sigma_{2,i}^2N)] > 0$. Hence by **Theorem 3.4.1**, not all states need to be persistent. For some states i, we can have $\beta_i - (\mu_i+\gamma_i) - \frac{1}{2}(\sigma_{1,i}^2N^2+\sigma_{2,i}^2N) < 0$ which possibly results in extinction. However the solution can still persist from the point of view of the overall behaviour.

5.5 Stationary Distribution

There are many different methods to prove the stationary distribution in a stochastic model with regime switching. For example, Zhu and Yin [123] use Lyapunov functions to develop necessary conditions for positive recurrence [124] and ergodicity in a hybrid system. Based on their results, Liu *et al.* [73] prove that their stochastic Lotka-Volterra model has a unique stationary distribution by proving the positive recurrence and ergodic property of the solution. However, these results have strong connections to Khasminskii's theory in stationary distribution. Hence in this section, we firstly recall Khasminskii's theory [72] as a lemma.

Lemma 5.5.1. *The SDE model* (5.4) *has a unique stationary distribution if there is a strictly proper subinterval* (a,b) *of* $(0,N)$ *such that* $\mathbb{E}(\tau) < \infty$ *for all* $I_0 \in (0,a]\cup[b,N)$*, where*

$$\tau = \inf\{t \ge 0 : I(t) \in (a,b)\}.$$

Also,

$$\sup_{I_0\in[\bar{a},\bar{b}]} \mathbb{E}(\tau) < \infty,$$

for every interval $[\bar{a},\bar{b}] \subset (0,N)$.

Note that the other condition in Khasminskii's theory is clearly satisfied in our model (5.4). So now we give the following **Theorem 5.5.2** and the proof by using **Lemma 5.5.1**.

Theorem 5.5.2. *When* $R_0^S > 1$*, our SDE model* (4.4) *has a unique stationary distribution if there exists a vector* $C = (C_1, C_2, ..., C_M), C_i \in \mathbb{R}$*, such that*

$$\text{diag}(A) + \Gamma C^T > 0 \text{ for all } i \in \mathbb{S}. \tag{5.16}$$

Note that $A = \begin{bmatrix} \alpha_1 & & \\ & \ddots & \\ & & \alpha_M \end{bmatrix}$*, where* $\alpha_i = \beta_i N - (\mu_i + \gamma_i) - \frac{1}{2}(\sigma_{1,i}^2 N^2 + \sigma_{2,i}^2 N)$.
Also, $\Gamma = (\nu_{ij})_{M\times M}$ *is the generator of the* M*-state Markov chain.*

Proof. **Step 1.** Firstly we examine a Lyapunov function $V_1(x,i) = \log x$ with initial value $I_0 \in [b,N)$ and $r(0) = r_0$. Recall (3.13) with Markov switching states

$$LV_1(x,i) = \beta_i(N-x) - (\mu_i+\gamma_i) - \frac{1}{2}[\sigma_{1,i}^2(N-x)^2 + \sigma_{2,i}^2(N-x)], x \in (0,N). \tag{5.17}$$

And it is obvious that $LV_1(N,i) = -(\mu_i+\gamma_i) < 0$. So there must exist a constant b near N, such that for any $x \in [b,N)$ and $i \in \mathbb{S}$

$$LV_1(x,i) \le -q \quad \text{(where } q \text{ is a positive constant)}. \tag{5.18}$$

Consequently, for all $t \ge 0$ and any $I_0 \in [b,N)$, we then have

$$\begin{aligned} \log b \le \mathbb{E}\log I(t\wedge\tau) &= \mathbb{E}\log I_0 + \mathbb{E}\int_0^{t\wedge\tau} LV_1(I(s),r(s))\mathrm{d}s + 0 \\ &\le \log I_0 - q\mathbb{E}(t\wedge\tau). \end{aligned}$$

Rearrange and we have

$$\mathbb{E}(t\wedge\tau) \le \frac{\log\frac{I_0}{b}}{q}. \tag{5.19}$$

Let $t \to \infty$, we have

$$\mathbb{E}(\tau) \leq \frac{\log \frac{I_0}{b}}{q} < \infty, \forall I_0 \in [b, N). \tag{5.20}$$

Here we complete **Step 1**, which clearly indicates that we can find a positive b near the boundary N, such that the solution will proceed into $(0, b)$ in finite time with the initial value $I_0 \in [b, N)$.

Step 2. Secondly, we choose a Lyapunov function as $V_2(x, i) = \log(e^{C_i}x) = C_i + \log x$, where $C_i \in \mathbb{R}$ are constants. We want to find a positive a near 0, such that the expected time for the solution, starting in $(0, a]$, to proceed into (a, N) is finite. As this Lyapunov function contains parameter switching with Markov chain states, the previous Itô formula no longer works. Instead, a generalised Itô formula is suitable here [73, 99, 125, 126]. This formula was proved by Skorokhod [127]. Using the generalised Itô formula, we have

$$\begin{aligned} LV_2(x,i) =& \beta_i(N-x) - (\mu_i + \gamma_i) - \frac{1}{2}[\sigma_{1,i}^2(N-x)^2 + \sigma_{2,i}^2(N-x)] \\ &+ \sum_{j=1}^{M} \nu_{ij}(C_j + \log x). \end{aligned} \tag{5.21}$$

Clearly, $\sum_{j=1}^{M} \nu_{ij} \log x = 0$. So we have

$$LV_2(x,i) = \beta_i(N-x) - (\mu_i + \gamma_i) - \frac{1}{2}[\sigma_{1,i}^2(N-x)^2 + \sigma_{2,i}^2(N-x)] + \sum_{j=1}^{M} \nu_{ij}C_j. \tag{5.22}$$

And $LV_2(0,i) = \beta_i N - (\mu_i + \gamma_i) - \frac{1}{2}(\sigma_{1,i}^2 N^2 + \sigma_{2,i}^2 N) + \Sigma_{j=1}^{M} \nu_{ij}C_j > 0$ is ensured for all $i \in \mathbb{S}$ by the given condition (5.16). So there must exist a constant a near 0, such that for all $x \in (0, a]$ and $i \in \mathbb{S}$

$$LV_2(x,i) \geq p \text{ (where } p \text{ is a positive constant)}. \tag{5.23}$$

Consequently, for all $t \geq 0$ and any $I_0 \in (0, a]$ and $r(0) = r_0$, we then have

$$\begin{aligned} \max_{i \in \mathbb{S}}\{C_i\} + \log a \geq \mathbb{E}\log(e^{C_{r(t\wedge\tau)}} I(t \wedge \tau)) &= C_{r_0} + \mathbb{E}\log I_0 + \mathbb{E}\int_0^{t\wedge\tau} LV_2(I(s))\mathrm{d}s + 0 \\ &\geq \min_{i \in \mathbb{S}}\{C_i\} + \log I_0 + p\mathbb{E}(t \wedge \tau). \end{aligned}$$

Rearrange and let $t \to \infty$, we have

$$\mathbb{E}(\tau) \leq \frac{\log \frac{a}{I_0} + \max_{i,j\in\mathbb{S}}\{C_i - C_j\}}{p} < \infty, \forall I_0 \in (0, a]. \tag{5.24}$$

This indicate that we can find a positive a near 0, such that the solution will rise into (a, N) in finite time. Combine the results from both **Step 1** and **Step 2** and (a, b) is the open set we need to find in **Lemma 5.5.1**. Hence we complete the proof. □

However, in this section, we do not intend to derive the mean and variance of this stationary distribution. Now in model (5.4), all parameters have been replaced by random variables. Thus during the deduction of mean and variance, after applying the Itô formula to $I(t)$ and dividing both sides by t with $t \to \infty$, terms such as $\lim_{t\to\infty} \int_0^t \beta_i I(s)\mathrm{d}s$ are now related to the joint distributions of random variables and $I(t)$, which are very hard to compute. Hence we stop here by only providing the proof of unique stationary distribution. We will give a further discussion in the simulation section, by examining the integral average of the solution $\frac{1}{t}\int_0^t I(s)\mathrm{d}s$.

5.6 Simulation

In this section, we also use Euler-Maruyama method [1, 113] implemented in R to simulate the solutions in extinction, persistence and stationary distribution examples. Parameters with random values are chosen to combine with different initial values, which generate simulations that support our theoretical proof in the previous sections. As a result, we will only give some of the examples in this section. Before we start, we shall again assume that the unit of time is one day and the population sizes are measured in units of 1 million. So firstly to simulate an extinction solution, we assume a simple Markov chain generator

$$\nu_{12} = 1, \nu_{21} = 2.$$

So we have stationary distribution of this Markov chain

$$\pi_1 = \frac{2}{3}, \pi_2 = \frac{1}{3}.$$

For both states we have $N = 100$ fixed. For the first example, parameters are defined as

$$\beta_1 = 0.4, \mu_1 + \gamma_1 = 45, \sigma_{1,1} = 0.03, \sigma_{2,1} = 0.6;$$

$$\beta_2 = 0.3, \mu_2 + \gamma_2 = 25, \sigma_{1,2} = 0.04, \sigma_{2,2} = 0.1.$$

For both states we also have

$$\frac{1}{2}\frac{(\beta_1 - \frac{1}{2}\sigma_{2,1}^2)^2}{\sigma_{1,1}^2} - (\mu_1 + \gamma_1) = -18.1;$$

$$\frac{1}{2}\frac{(\beta_2 - \frac{1}{2}\sigma_{2,2}^2)^2}{\sigma_{1,2}^2} - (\mu_2 + \gamma_2) = 2.2.$$

Clearly by **Theorem 3.3.1** and **Theorem 3.4.1**, the disease will die out in state 1 but persist in state 2. However from the average-type result in **Theorem 5.3.1** we have

$$\Sigma\pi_i \left[\frac{1}{2}\frac{(\beta_i - \frac{1}{2}\sigma_{2,i}^2)^2}{\sigma_{1,i}^2} - (\mu_i + \gamma_i)\right] = -11.3 < 0,$$

which indicates extinction in our solution. Also in the second example, parameters are defined as

$$\beta_1 = 0.4, \mu_1 + \gamma_1 = 45, \sigma_{1,1} = 0.03, \sigma_{2,1} = 0.6;$$

$$\beta_2 = 0.5, \mu_2 + \gamma_2 = 65, \sigma_{1,2} = 0.04, \sigma_{2,2} = 0.4.$$

For both states we also have

$$\frac{1}{2}\frac{(\beta_1 - \frac{1}{2}\sigma_{2,1}^2)^2}{\sigma_{1,1}^2} - (\mu_1 + \gamma_1) = -18.1;$$

$$\frac{1}{2}\frac{(\beta_2 - \frac{1}{2}\sigma_{2,2}^2)^2}{\sigma_{1,2}^2} - (\mu_2 + \gamma_2) = -9.88.$$

Clearly by **Theorem 3.3.1**, the disease will die out in both state 1 and state 2 and from the average-type result in **Theorem 5.3.1** we have

$$\sum \pi_i \left[\frac{1}{2}\frac{(\beta_i - \frac{1}{2}\sigma_{2,i}^2)^2}{\sigma_{1,i}^2} - (\mu_i + \gamma_i)\right] = -15.36 < 0,$$

which indicates extinction in our solution.

Now by using the Euler-Maruyama Method in R and assuming the step size is 0.001 and $r_0 = 1$, we can see the results in **Figure 5.1**, **Figure 5.2**, **Figure 5.3**

and **Figure 5.4** clearly show that the solutions tend to 0 after 5 days, with both large and small initial values. We can also see in **Figure 5.1** and **Figure 5.2** that there are some decreasing and increasing behaviours early in the plots, indicating the Markovian switching between extinction state to non-extinction state with Brownian motions. The corresponding Markov chains $r(t)$ for all cases are also illustrated.

Similarly, for persistence examples, we want to simulate a solution of (5.4) with a simple two-state Markov chain. Firstly we still fix $N = 100$. Then we want to make the solution persist in only one of the states according to **Theorem 3.4.1** but by the average-type result from **Theorem 5.4.1**, it still have persistence in the whole behaviour. Hence we assume the parameters in the first example as

$$\beta_1 = 0.4, \mu_1 + \gamma_1 = 45, \sigma_{1,1} = 0.03, \sigma_{2,1} = 0.01.$$

So in state 1 we have

$$\beta_1 N - (\mu_1 + \gamma_1) - \frac{1}{2}(\sigma_{1,1}^2 N^2 + \sigma_{2,1}^2 N) = -9.5 < 0,$$

which means $R_{0_1}^S < 1$ so by **Theorem 3.3.1** disease in state 1 will die out. Also parameters in state 2 are

$$\beta_2 = 0.6, \mu_2 + \gamma_2 = 35, \sigma_{1,2} = 0.04, \sigma_{2,2} = 0.1.$$

Then in state 2 we have

$$\beta_2 N - (\mu_2 + \gamma_2) - \frac{1}{2}(\sigma_{1,2}^2 N^2 + \sigma_{2,2}^2 N) = 16.5 > 0.$$

which means $R_{0_2}^S > 1$ so by **Theorem 3.4.1** disease in state 2 will persist. Now we define the Markov chain generator

$$\nu_{12} = 3, \nu_{21} = 4.$$

So we have stationary distribution of this Markov chain

$$\pi_1 = \frac{4}{7}, \pi_2 = \frac{3}{7}.$$

And we can see that

$$\pi_1(\beta_1 N - (\mu_1+\gamma_1) - \frac{1}{2}(\sigma_{1,1}^2 N^2 + \sigma_{2,1}^2 N)) + \pi_2(\beta_2 N - (\mu_2+\gamma_2) - \frac{1}{2}(\sigma_{1,2}^2 N^2 + \sigma_{2,2}^2 N)) > 0,$$

which means $R_0^S = \frac{\sum \pi_i \beta_i N}{\sum \pi_i(\mu_i+\gamma_i)} - \frac{\sum \pi_i(\sigma_{1,i}^2 N^2+\sigma_{2,i}^2 N)}{2\sum \pi_i(\mu_i+\gamma_i)} > 1$ so our solution will be oscillating around a positive level $\xi = 4.478064$ infinitely often by **Theorem 5.4.1**.

Similarly we also build a model with both states persistent according to **Theorem 3.4.1**. Here we assume parameters as following in the second persistence example.

$$\beta_1 = 0.5, \mu_1 + \gamma_1 = 45, \sigma_{1,1} = 0.02, \sigma_{2,1} = 0.05.$$

So in state 1 we let

$$\beta_1 N - (\mu_1 + \gamma_1) - \frac{1}{2}(\sigma_{1,1}^2 N^2 + \sigma_{2,1}^2 N) = 2.88 > 0,$$

which means $R_{0_1}^S > 1$. And in state 2 we have

$$\beta_2 = 0.6, \mu_2 + \gamma_2 = 35, \sigma_{1,2} = 0.04, \sigma_{2,2} = 0.1.$$

Then in state 2 we have

$$\beta_2 N - (\mu_2 + \gamma_2) - \frac{1}{2}(\sigma_{1,2}^2 N^2 + \sigma_{2,2}^2 N) = 16.5 > 0,$$

which means $R_{0_2}^S > 1$ so by **Theorem 3.4.1** disease in both state 1 and state 2 will persist. With the same Markov chain generator as in the first example

$$\nu_{12} = 3, \nu_{21} = 4.$$

we have

$$\pi_1(\beta_1 N-(\mu_1+\gamma_1)-\frac{1}{2}(\sigma_{1,1}^2 N^2+\sigma_{2,1}^2 N))+\pi_2(\beta_2 N-(\mu_2+\gamma_2)-\frac{1}{2}(\sigma_{1,2}^2 N^2+\sigma_{2,2}^2 N)) > 0,$$

which means $R_0^S = \frac{\sum \pi_i \beta_i N}{\sum \pi_i(\mu_i+\gamma_i)} - \frac{\sum \pi_i(\sigma_{1,i}^2 N^2+\sigma_{2,i}^2 N)}{2\sum \pi_i(\mu_i+\gamma_i)} > 1$ so our solution will be oscillating around a positive level $\xi = 19.05665$ infinitely often by **Theorem 5.4.1**.

Now we use the Euler-Maruyama method [5] in R with step size 0.001 and $r_0 = 1$ to simulate the solution by 50 days. Without the loss of generality, we use both large and small initial values. Clearly the solution is oscillating around the level ξ, which is marked as a red line in **Figure 5.5**, **5.6**, **5.7** and **5.8**. In **Figures 5.5** and **5.6** we can see that during some iterations, the solution tends to zero, and then goes up again to follow the fluctuation. This is clearly caused by the extinction behaviour in state 1.

To generate a stationary distribution we firstly use the same parameters as in the persistence cases. Similarly we fix $N = 100$ and we assume the parameters as follows.

Example 1.

$$\beta_1 = 0.4, \mu_1 + \gamma_1 = 45, \sigma_{1,1} = 0.03, \sigma_{2,1} = 0.01;$$

$$\beta_2 = 0.6, \mu_2 + \gamma_2 = 35, \sigma_{1,2} = 0.04, \sigma_{2,2} = 0.1.$$

Hence we have

$$\alpha_1 = \beta_1 N - (\mu_1 + \gamma_1) - \frac{1}{2}(\sigma_{1,1}^2 N^2 + \sigma_{2,1}^2 N) = -9.5 < 0,$$

and

$$\alpha_2 = \beta_2 N - (\mu_2 + \gamma_2) - \frac{1}{2}(\sigma_{1,2}^2 N^2 + \sigma_{2,2}^2 N) = 16.5 > 0.$$

Example 2.

$$\beta_1 = 0.5, \mu_1 + \gamma_1 = 45, \sigma_{1,1} = 0.02, \sigma_{2,1} = 0.05;$$

$$\beta_2 = 0.6, \mu_2 + \gamma_2 = 35, \sigma_{1,2} = 0.04, \sigma_{2,2} = 0.1.$$

Hence we have

$$\alpha_1 = \beta_1 N - (\mu_1 + \gamma_1) - \frac{1}{2}(\sigma_{1,1}^2 N^2 + \sigma_{2,1}^2 N) = 2.88 > 0,$$

and

$$\alpha_2 = \beta_2 N - (\mu_2 + \gamma_2) - \frac{1}{2}(\sigma_{1,2}^2 N^2 + \sigma_{2,2}^2 N) = 16.5 > 0.$$

With the Markov chain generator,

$$\nu_{12} = 3, \nu_{21} = 4,$$

it is easy to find a pair of C_1 and C_2 to satisfy the condition

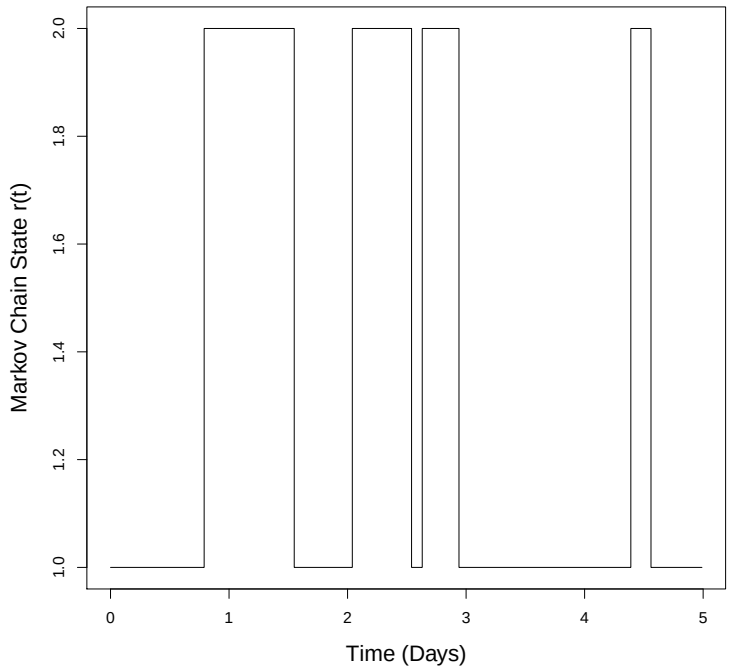
$$\text{diag}(A) + \Gamma C^T > 0.$$

which is required in **Theorem 5.5.2**. For example, $C_1 = 2$ and $C_2 = 6$. So this condition is met and we have a unique stationary distribution. Now by using Euler-Maruyama method in R, we generate a long run of 200,000 iterations with

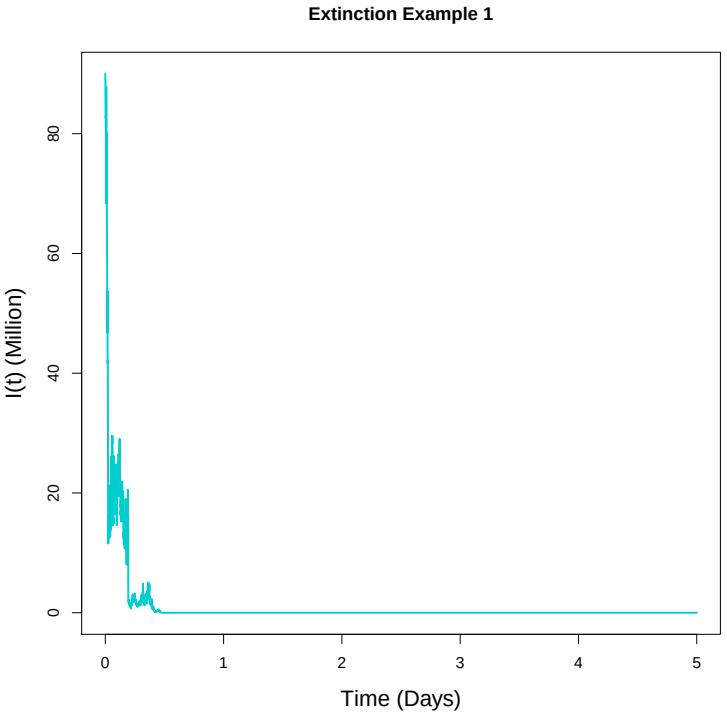
step size $\Delta = 0.001$. And we also plot the integral average of our solution, which is $\frac{1}{T}\int_0^T I(t)\mathrm{d}t$. By Khasminskii's theory [72], this integral will tend to the first moment of our solution if there is a unique stationary distribution. From **Figures 5.9**, **5.10**, **5.11** and **5.12**, we can see our solution fluctuates very intensely, which indicates the recurrence in our model (5.4). Moreover, in **Figures 5.9** and **5.10**, it is clearly illustrated that there are some sequence of iterations where $I(t)$ tends to zero which caused by the extinction in state 1. These results give further explanation to our persistence theory. Also, in each different cases, the integral average of $I(t)$ is also demonstrated, which clearly converges to a fixed positive level, the mean of this stationary distribution. Consequently, the numerical results support our ergodic theorem.

5.7 Summary

In this chapter, we have discussed telegraph noise in an SIS epidemic model based on Gray *et al.*'s work [5]. A finite-state Markov chain is used to describe the switching between different environments in our previous model (3.5), which formulates a stochastic SIS model with two independent Brownian motions and Markovian switching. It is obviously a generalized model of (3.5), which can be applied to more complicated cases in epidemic studies. From our results in each section, we discover very interesting facts that can be related to the disturbance of telegraph noise. From the overall behaviour of the solutions, we can conclude that if we need to eliminate the disease, we do not need to have consistent extinction in every state. In some certain states, disease can even persist in the population. As long as the overall condition in **Theorem 5.3.1** is satisfied, the disease will consequently die out. A similar result is also obtained in the persistence analysis. As long as the condition in **Theorem 5.4.1** is satisfied, the disease will ultimately persist. This is the contribution of considering telegraph noise in our model, which indicates the expansion of extinction conditions. Moreover, while Gray *et al.* did not examine the stationary distribution in their research [5], we regard this as a very important property of the solution and in order to keep up with our previous work in **Chapter 3** and **Chapter 4**, we again prove the existence of a unique

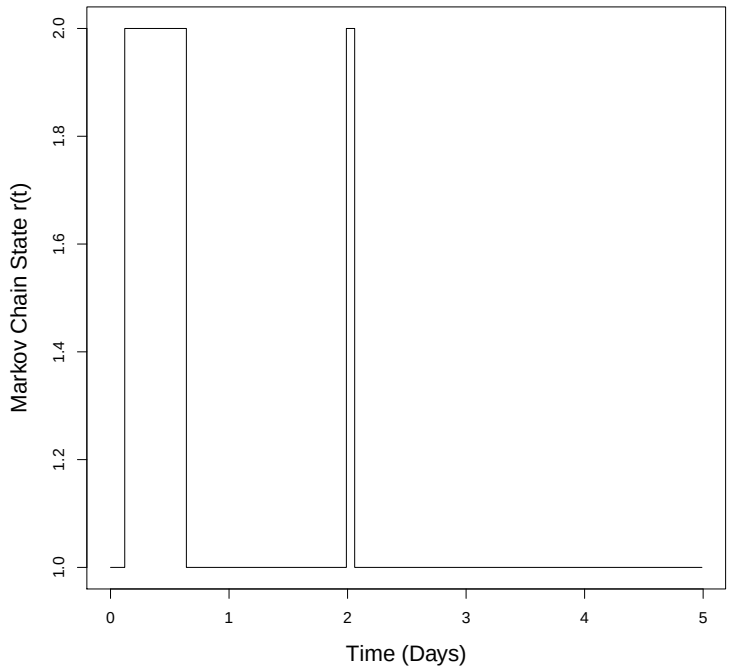


(a) Markov chain.

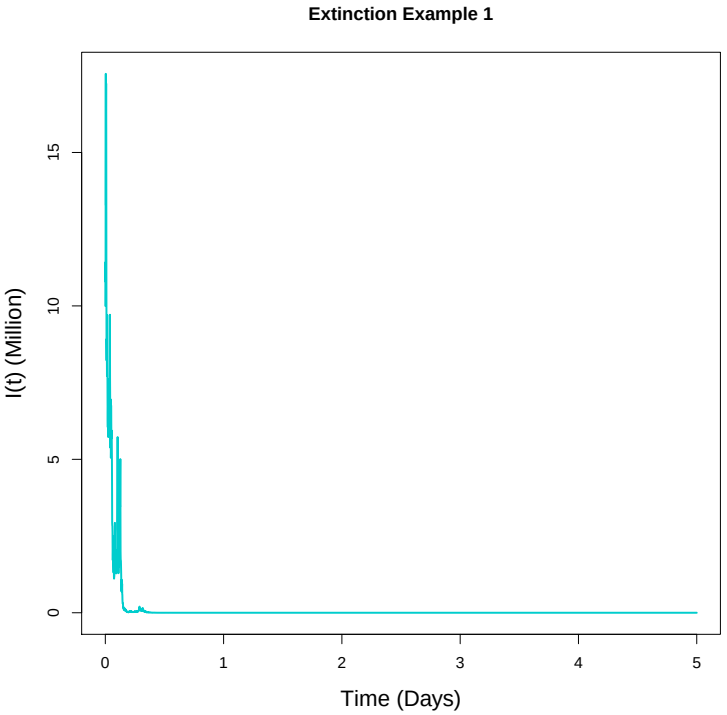


(b) $I(0) = 90$.

Figure 5.1: Extinction Example 1 with $I(0) = 90$.

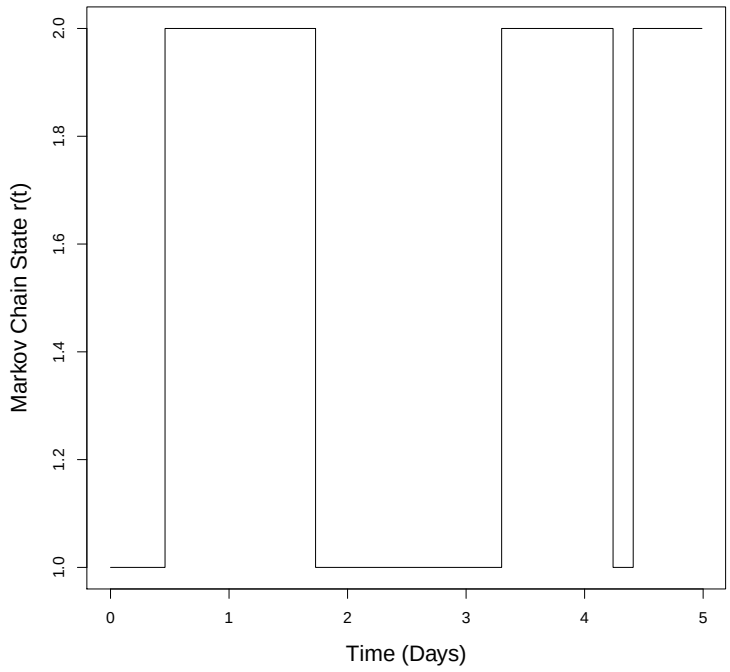


(a) Markov chain.

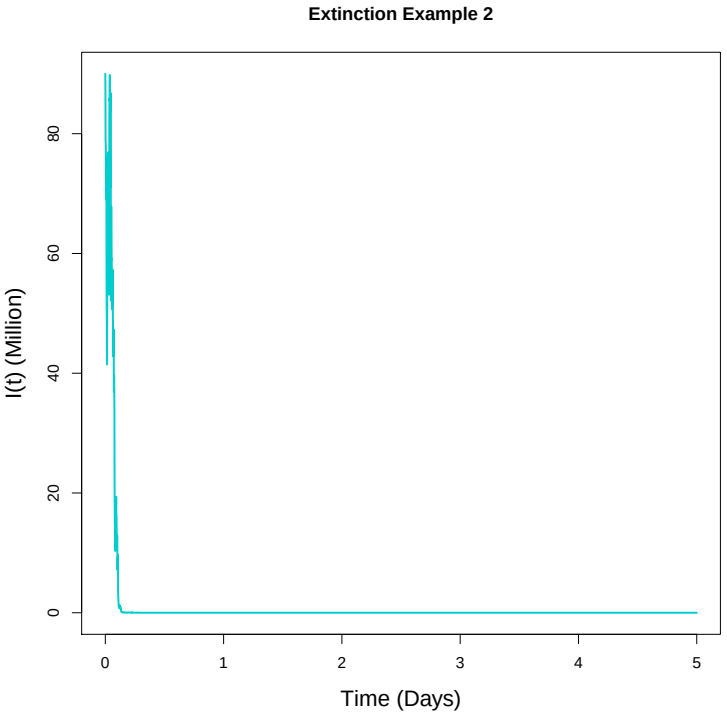


(b) $I(0) = 10$.

Figure 5.2: Extinction Example 1 with $I(0) = 10$.

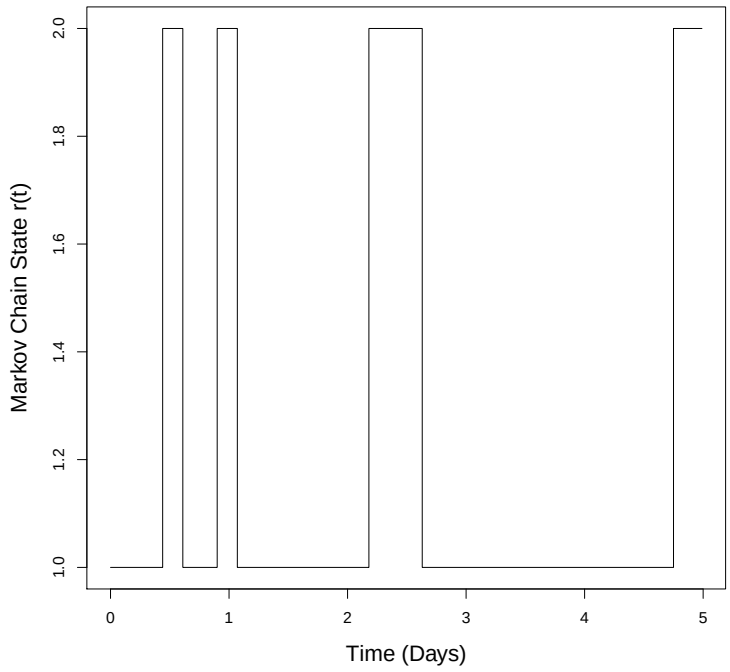


(a) Markov chain.

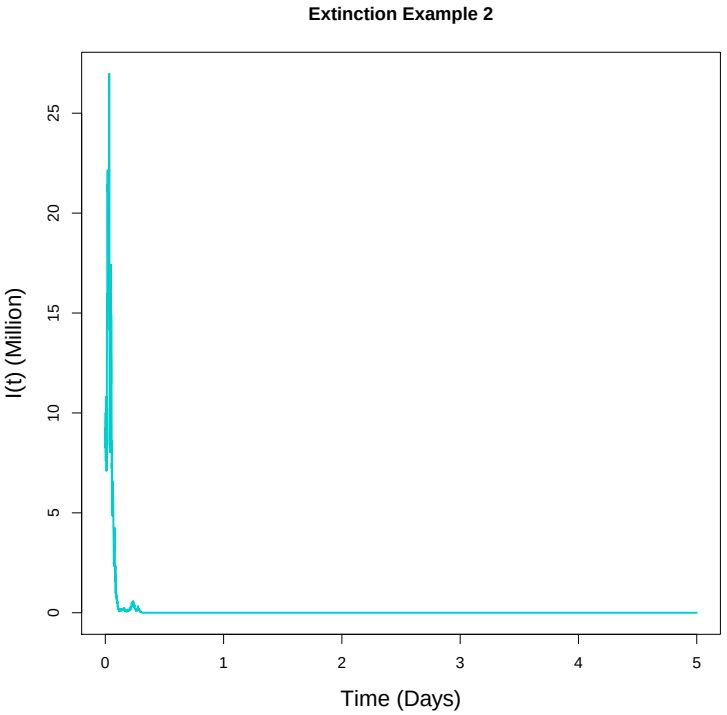


(b) $I(0) = 90$.

Figure 5.3: Extinction Example 2 with $I(0) = 90$.

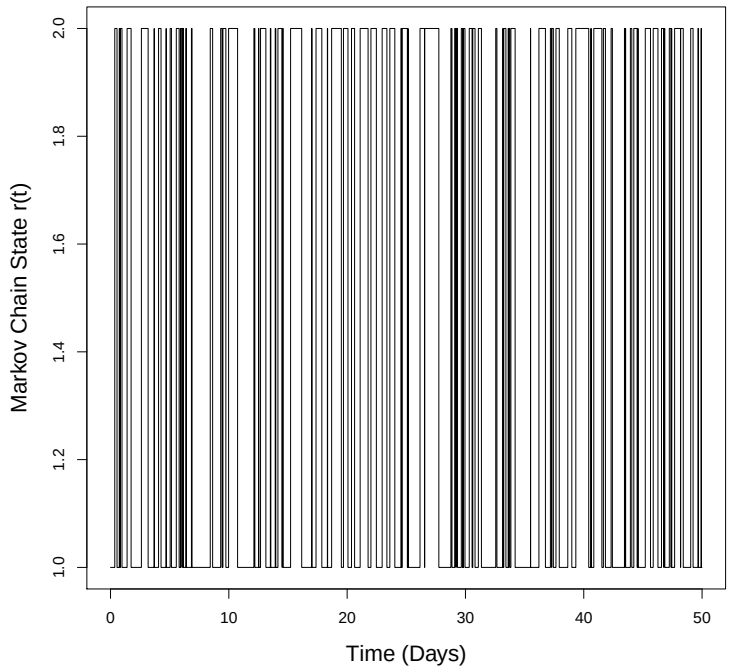


(a) Markov chain.

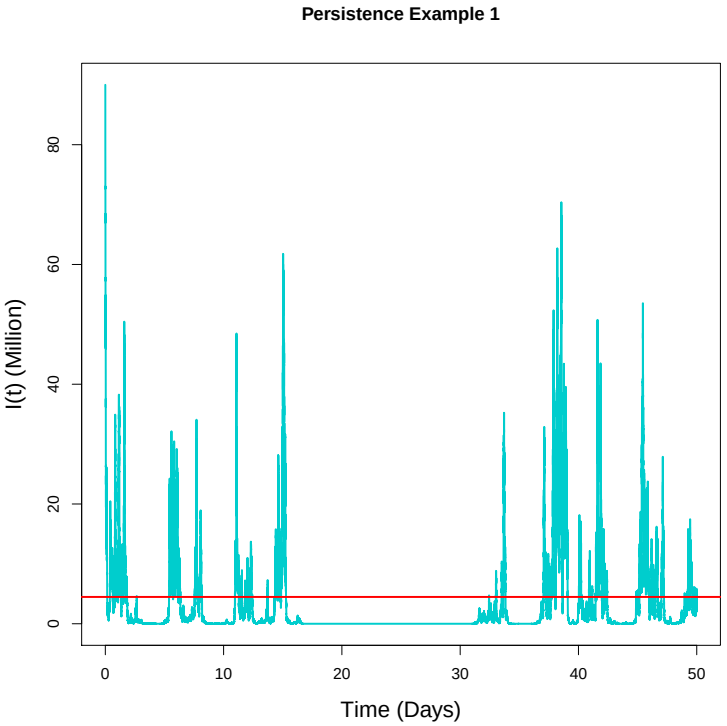


(b) $I(0) = 10$.

Figure 5.4: Extinction Example 2 with $I(0) = 10$.

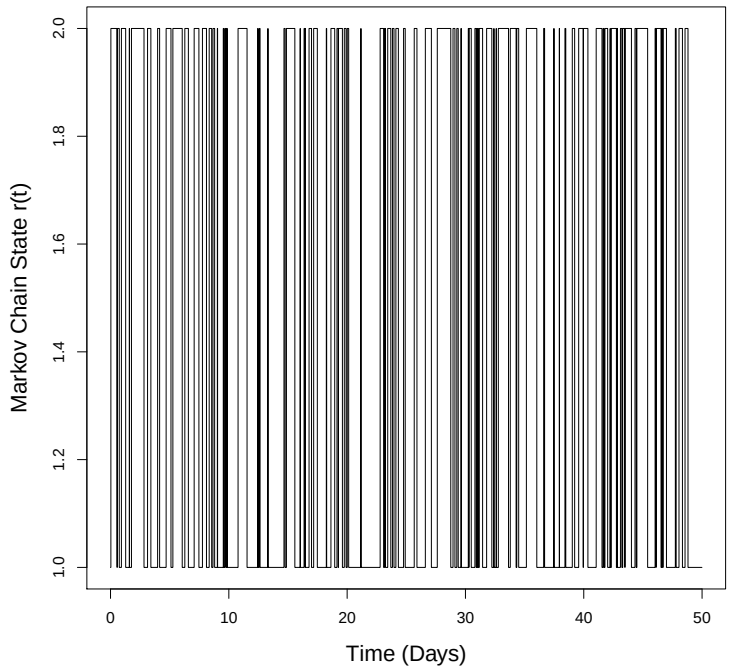


(a) Markov chain.

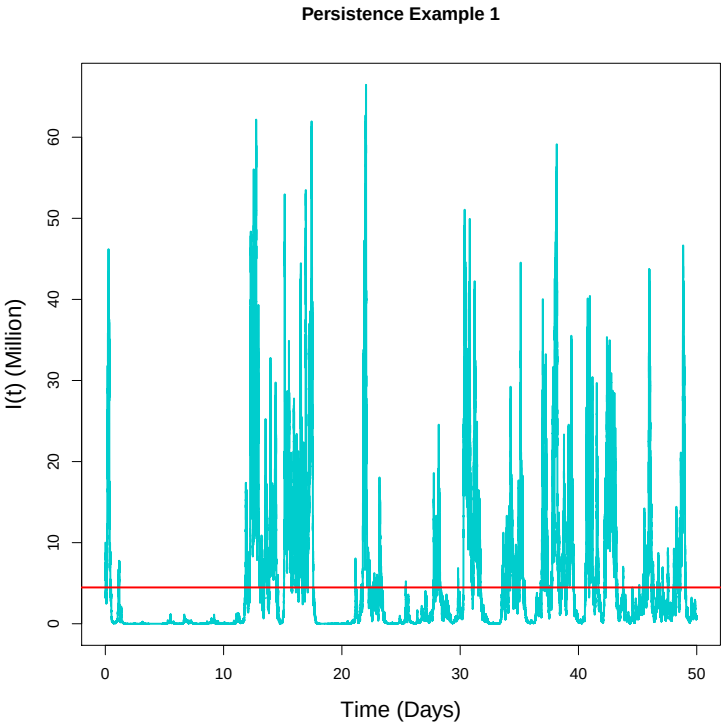


(b) $I(0) = 90$.

Figure 5.5: Persistence Example 1 with $I(0) = 90$.

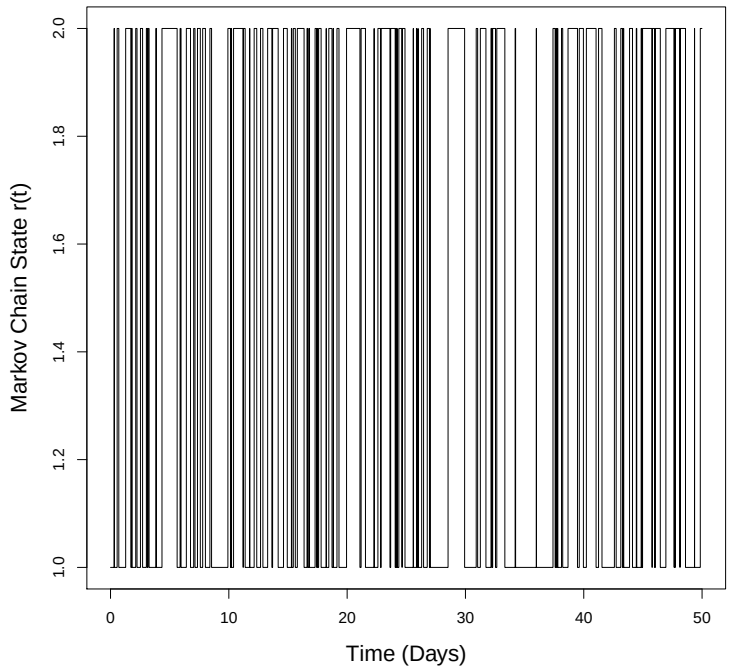


(a) Markov chain.

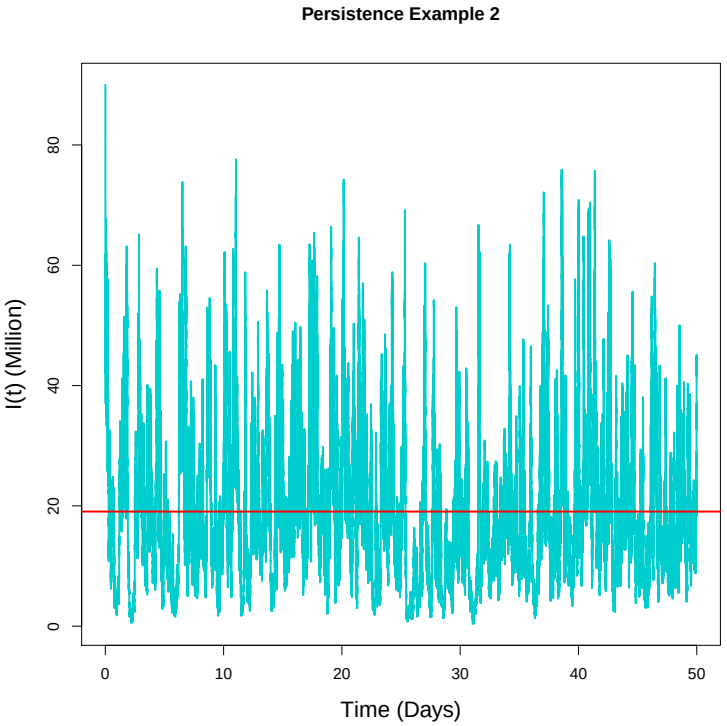


(b) $I(0) = 10$.

Figure 5.6: Persistence Example 1 with $I(0) = 10$.

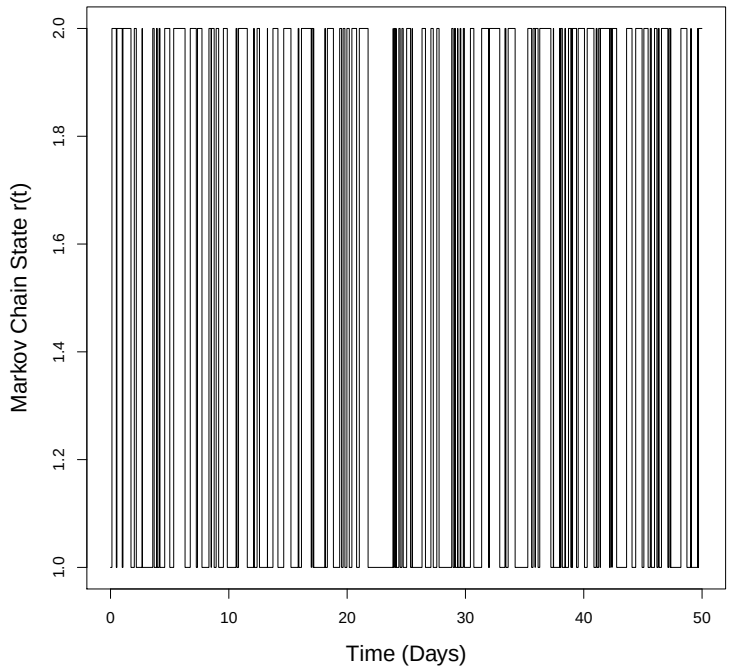


(a) Markov chain.

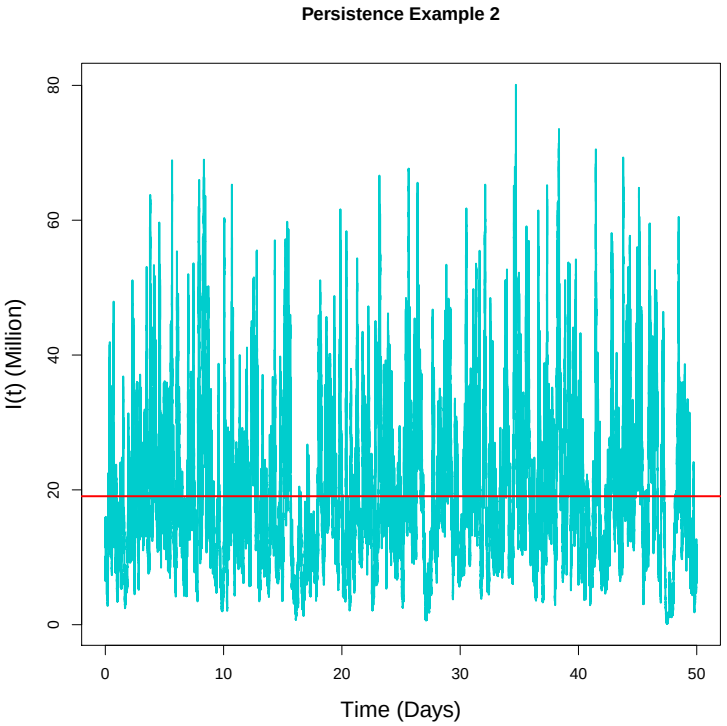


(b) $I(0) = 90$.

Figure 5.7: Persistence Example 2 with $I(0) = 90$.

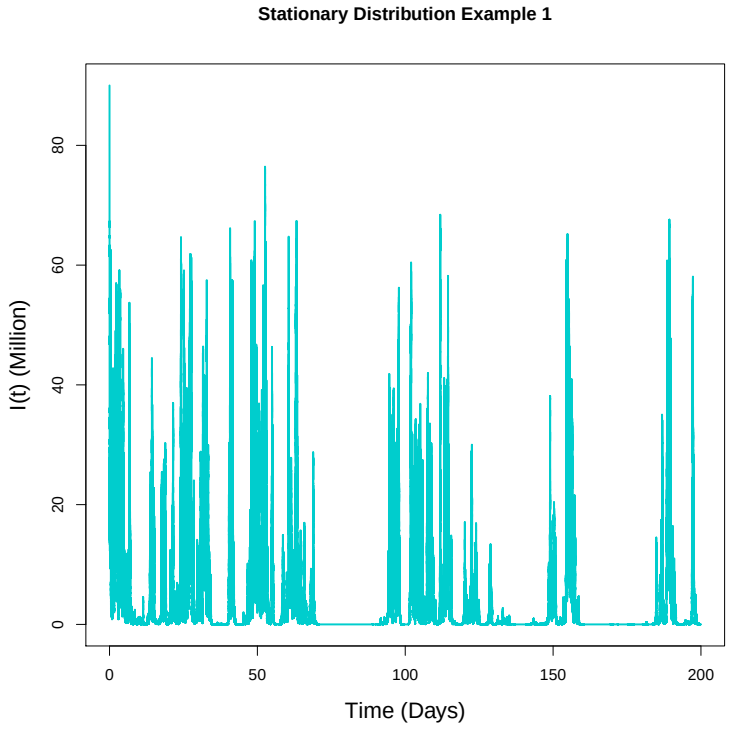


(a) Markov chain.

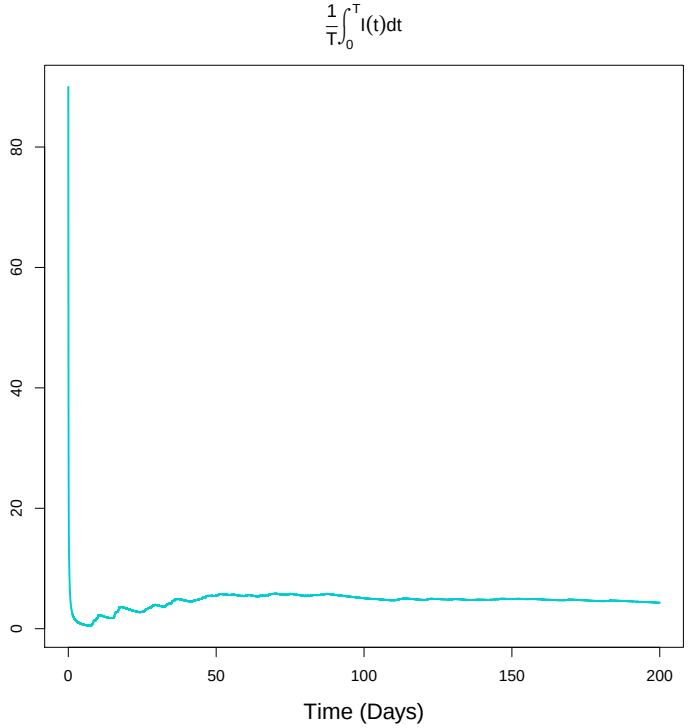


(b) $I(0) = 10$.

Figure 5.8: Persistence Example 2 with $I(0) = 10$.

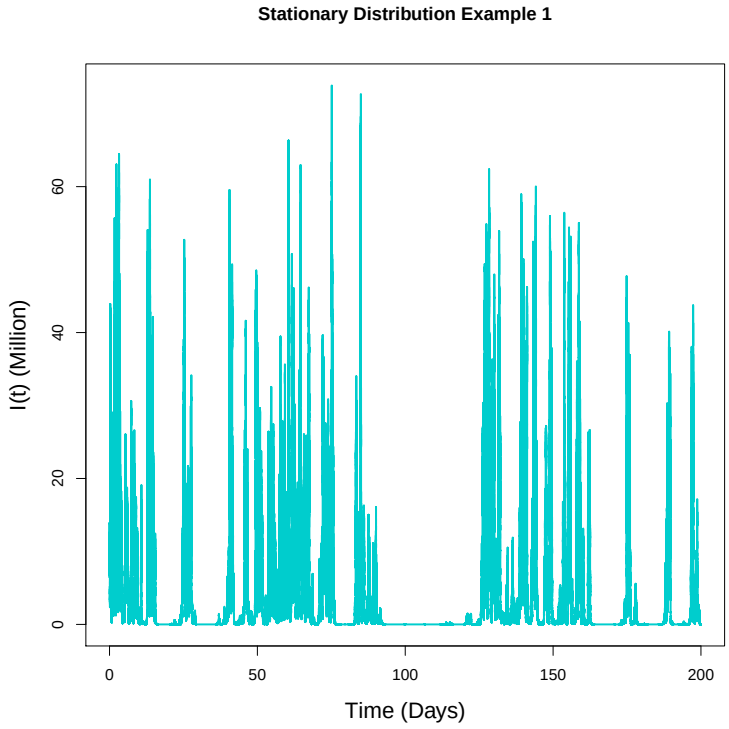


(a) Solution with initial value $I(0) = 90$.

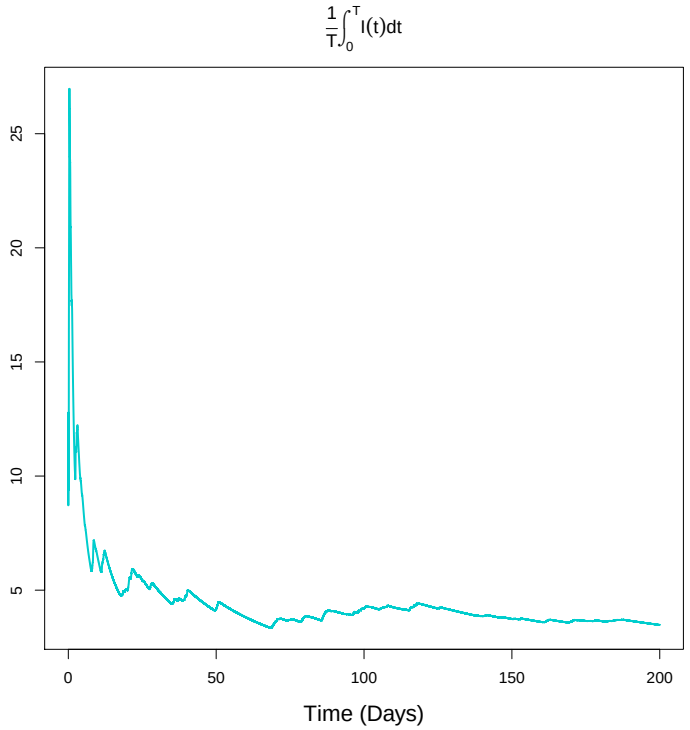


(b) Integral average of the solution.

Figure 5.9: Stationary Distribution Example 1 with $I(0) = 90$.

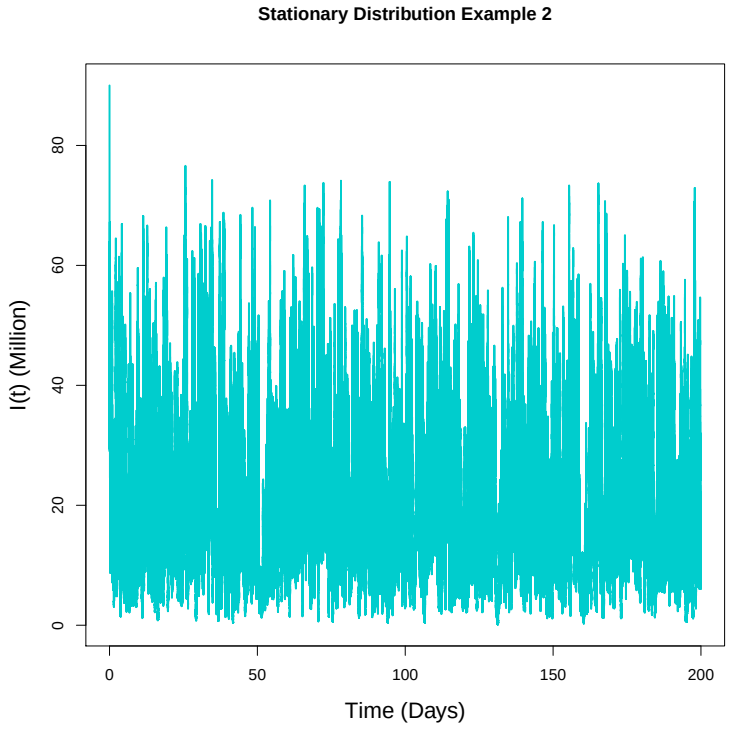


(a) Solution with initial value $I(0) = 10$.

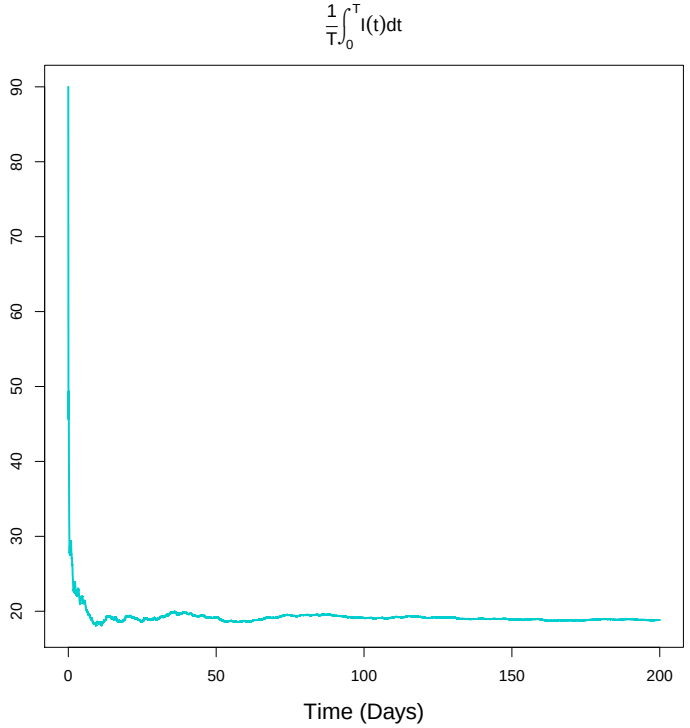


(b) Integral average of the solution.

Figure 5.10: Stationary Distribution Example 1 with $I(0) = 10$.

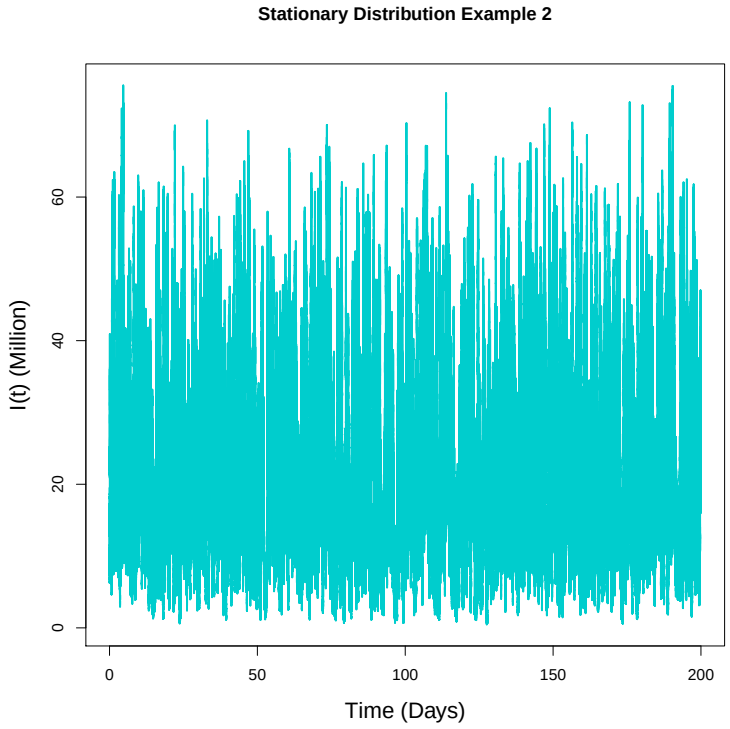


(a) Solution with initial value $I(0) = 90$.

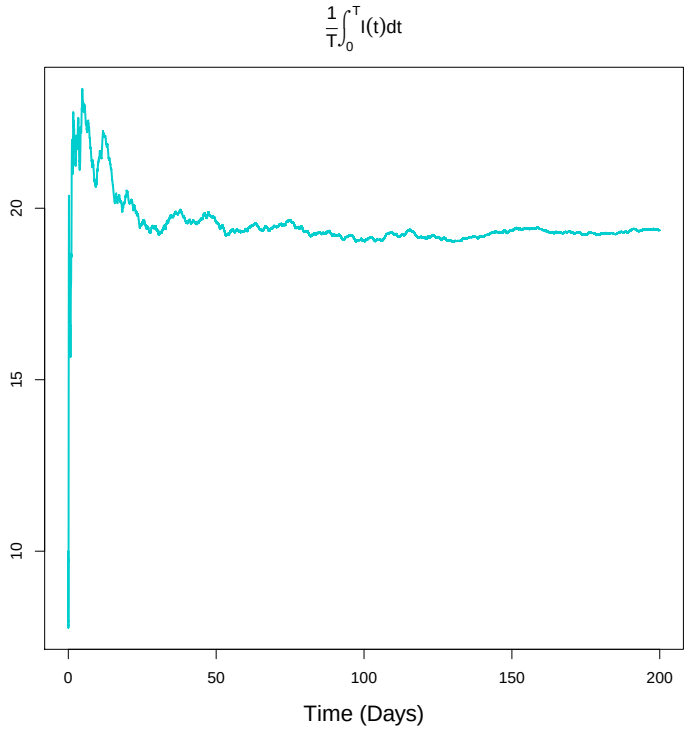


(b) Integral average of the solution.

Figure 5.11: Stationary Distribution Example 2 with $I(0) = 90$.



(a) Solution with initial value $I(0) = 10$.



(b) Integral average of the solution.

Figure 5.12: Stationary Distribution Example 2 with $I(0) = 10$.

stationary distribution by using Khasminskii's theory. A different generalized Itô formula is introduced and applied in this section because we choose a Lyapunov function defined on Markov chain.

There is no doubt that introducing telegraph noise in model (3.5) makes our new model (5.4) more practical and complicated. However, like **Chapter 4**, the results in our model are also weakened and incomplete in some aspects due to the impact of telegraph noise. For example, although the simulation illustrates the integral average as the mean of the stationary distribution, we are not able to compute the explicit expression for the mean and variance; there is also an extra condition in existence of a stationary distribution, $\text{diag}(A) + \Gamma C^T > 0$, which is caused by the use of the generalized Itô formula. Though we have tried many Lyapunov functions, we do not know if there is a better alternative which would not add extra requirement. We also do not know if this condition is always satisfied for all Markov chains so we choose to keep this condition for now; to prove the boundedness of the solution, we still require every state of the solution to be bounded within $(0, N)$ instead of replacing this condition by another one based on the overall behaviour of the solution. If such a condition can be found, then it is possible to conclude that we do not need every state to be bounded to have overall boundedness. This means, in some of the states, solution may proceed beyond N if it is examined individually. However, the disturbance of telegraph noise will always pull the solution back to $(0, N)$ by switching to other bounded states. These are the problems that we are not able to answer now and in this case, it is not biologically realistic in epidemic modelling. However, it would be interesting in stochastic mathematical modelling.

Hence here we have completed the study of our three SIS epidemic models. In the next chapter, we are going to conclude our research and summarize our results.

Chapter 6

Conclusion

In this research we construct the first model (3.5) by introducing another perturbation on $\mu + \gamma$ based on Gray *et al.*'s research[1] with a different form. This SIS SDE model (3.5) with two independent Brownian motions has very similar properties as theirs [1]. We then prove that our model has a unique and positive solution which is bounded in $(0, N)$ with probability 1. Then we define the stochastic reproduction number of our model, which needs a weaker condition for the model to go to extinction compared to the classical deterministic model and the previous model with one perturbation. When $R_0^S < 1$, we find the further three sufficient conditions for the disease to die out. As long as one of these is satisfied, the disease will die out with probability one. When $R_0^S > 1$, we prove that the solution of our model will oscillate around a positive level ξ almost surely. Under this circumstance, we find the unique positive stationary distribution of our SDE model with the expression of mean and variance. Importantly, simulations with different values of parameters are produced to illustrate and support our theoretical results.

Our new perturbation clearly needs σ_2 not too large from **Theorem 3.2.1** to ensure a unique bounded positive solution of (3.5). However, this perturbation extends the requirements for $R_0^S < 1$ compared to the deterministic SIS model and the results in [1]. This means for those parameters that will not cause the disease to die out in the deterministic model as well as Gray's model [1], extinction will become possible if we add the new perturbation. Meanwhile, we find the unique

stationary distribution with no extra conditions, which means that adding our new perturbation in Gray's model [1] will have similar results.

Secondly, we replace independent Brownian motions in our previous model (3.5) by correlated Brownian motions which leads to not only the increasing number of noises compared to Gray *et al.*'s work [1], but also turning the drifting coefficient into a composite term. Then we prove that the stochastic reproduction number R_0^S is the key to define the extinction and persistence of the solution. Similar to our model (3.5), with $R_0^S < 1$ and extra conditions, the disease will die out. When $R_0^S > 1$, we prove that the solution will oscillate around a certain positive level. Moreover, when $R_0^S > 1$, there is a unique stationary distribution of the solution.

Compared to [1], our $L\tilde{V}$ is not quadratic any more, which results in more general and complicated conditions to both extinction and persistence sections. Moreover, compared to our first model [2], in model (4.4) [4] we assume that the Brownian motions are correlated and hence the effects of the correlations on the behaviours of our SIS system are studied. The analytical results including the form of R_0^S and the additional restrictions indicate that the correlations between the Brownian motions do make a significant difference. Also, though we do not know the explicit expression of the persistence level ξ, numerical method are then used to find the exact value under certain circumstances. On the other hand, we have tried to get the explicit expression of the mean and variance by deducing higher moments of $I(t)$ but we are not able to get a better result at this time.

Finally, we review our first model (3.5) and introduce telegraph noise by using Markovian switching. This leads to our third model (5.4), which contains two independent white noises and also a telegraph noise. Model (5.4) can be regarded as combination and extension of both [1] and [5]. Then the new stochastic reproduction number R_0^S is also stated, and we give the extinction and persistence conditions of the solution similarly. We also prove that there is a unique stationary distribution when the solution persists with an extra condition on the Markov chain generator.

Compared to model (3.5), the main difference is that the conditions of extinction and persistence are related to the stationary distribution of the Markovian

switching. These conditions provide requirements on the whole behaviour of the solution instead of every state of it, which reflects the impact from adding telegraph noise in our epidemic SIS system. This means, for example, if extinction is needed in our model, only one state needs to go to extinction if we choose a proper Markov chain. Also, in order to find a stationary distribution, we use a generalised Itô formula [99]. A condition, $\mathrm{diag}(A)+\Gamma C^T > 0$, is stated in the theorem, but we do not know if we can always find a suitable vector C for any Markov generator to satisfy the inequality. Moreover, simulation results are then illustrated, which clearly support our previous theory.

Consequently, from our results in three models, we can conclude that both white noise and telegraph noise have positive impacts on epidemic models. Although there are potential improvements in our models that still yet to be explored, it is no doubt that we have formulated three models to generalize the work from [1, 5] to stabilize those unstable cases in their work, which are also more practical and reasonable in epidemic study.

Bibliography

[1] Alison Gray, David Greenhalgh, Liangjian Hu, Xuerong Mao, and Jiafeng Pan. A stochastic differential equation SIS epidemic model. *SIAM Journal on Applied Mathematics*, 71(3):876–902, 2011.

[2] Siyang Cai, Yongmei Cai, and Xuerong Mao. A stochastic differential equation SIS epidemic model with two independent Brownian motions. *Journal of Mathematical Analysis and Applications*, 474(2):1536–1550, 2019.

[3] Alexandru Hening and Dang H Nguyen. Stochastic Lotka–Volterra food chains. *Journal of Mathematical Biology*, 77(1):135–163, 2018.

[4] Siyang Cai, Yongmei Cai, and Xuerong Mao. A stochastic differential equation SIS epidemic model with two correlated Brownian motions. *Nonlinear Dynamics*, 97(4):2175–2187, 2019.

[5] Alison Gray, David Greenhalgh, Xuerong Mao, and Jiafeng Pan. The SIS epidemic model with Markovian switching. *Journal of Mathematical Analysis and Applications*, 394(2):496–516, 2012.

[6] Philip Ziegler. *The Black Death*. Faber & Faber, 2013.

[7] Robert S Gottfried. *Black Death*. Simon and Schuster, 2010.

[8] Michael Walters Dols. *The Black Death in the Middle East*, volume 5354. Princeton University Press, 2019.

[9] K David Patterson. Typhus and its control in Russia, 1870–1940. *Medical History*, 37(4):361–381, 1993.

[10] Irina Tarasevich, Elena Rydkina, and Didier Raoult. Outbreak of epidemic typhus in Russia. *The Lancet*, 352(9134):1151, 1998.

[11] Yassina Bechah, Christian Capo, Jean-Louis Mege, and Didier Raoult. Epidemic typhus. *The Lancet Infectious Diseases*, 8(7):417–426, 2008.

[12] Elena B Rydkina, Véronique Roux, Eugenia M Gagua, Alexandre B Predtechenski, Irina V Tarasevich, and Didier Raoult. *Bartonella quintana* in body lice collected from homeless persons in Russia. *Emerging Infectious Diseases*, 5(1):176, 1999.

[13] Brigitte Autran, Guislaine Carcelain, Tao Sheng Li, Catherine Blanc, Dominique Mathez, Roland Tubiana, Christine Katlama, Patrice Debre, and Jacques Leibowitch. Positive effects of combined antiretroviral therapy on CD4+ T-cell homeostasis and function in advanced HIV disease. *Science*, 277(5322):112–116, 1997.

[14] Joseph M McCune. The dynamics of CD4+ T-cell depletion in HIV disease. *Nature*, 410(6831):974, 2001.

[15] World Health Organization. Number of people (all ages) living with HIV. `http://apps.who.int/gho/data/node.main.620?lang=en`, 2019. last accessed on 02/11/2019.

[16] Kate E Jones, Nikkita G Patel, Marc A Levy, Adam Storeygard, Deborah Balk, John L Gittleman, and Peter Daszak. Global trends in emerging infectious diseases. *Nature*, 451(7181):990, 2008.

[17] Roy M Anderson and Robert M May. *Infectious diseases of humans: dynamics and control*. Oxford University Press, 1991.

[18] Mick G Roberts and John Jowett. An SEI model with density-dependent demographics and epidemiology. *Mathematical Medicine and Biology: A Journal of the IMA*, 13(4):245–257, 1996.

[19] Sam Robinson, Natalie Leo, Paul Prociv, and Stepehn C Barker. Potential role of head lice, *Pediculus humanus capitis*, as vectors of *Rickettsia prowazekii*. *Parasitology Research*, 90(3):209–211, 2003.

[20] Mart CM de Jong. Mathematical modelling in veterinary epidemiology: why model building is important. *Preventive Veterinary Medicine*, 25(2):183–193, 1995.

[21] Akira Okubo and Simon A Levin. *Diffusion and ecological problems: modern perspectives*, volume 14. Springer Science & Business Media, 2013.

[22] Leonid A Rvachev and Ira M Longini Jr. A mathematical model for the global spread of influenza. *Mathematical Biosciences*, 75(1):3–22, 1985.

[23] Norman TJ Bailey. *The mathematical theory of infectious diseases and its applications*. Charles Griffin & Company Ltd, 1975.

[24] Tomoo Royama. *Analytical population dynamics*, volume 10. Springer Science & Business Media, 2012.

[25] William Ogilvy Kermack and Anderson G McKendrick. A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society of London, Series A*, 115(772):700–721, 1927.

[26] Fred Brauer. The Kermack–McKendrick epidemic model revisited. *Mathematical Biosciences*, 198(2):119–131, 2005.

[27] Fred Brauer, Pauline van den Driessche, and Jianhong Wu. *Mathematical epidemiology*. Springer Berlin, 2008.

[28] Herbert W Hethcote. Three basic epidemiological Models. In Simon A. Levin, Thomas G. Hallam, and Louis J. Gross, editors, *Applied Mathematical Ecology*, pages 119–144. Springer Berlin, 1989.

[29] Linda JS Allen and P Van den Driessche. The basic reproduction number in some discrete-time epidemic models. *Journal of Differential Equations and Applications*, 14(10-11):1127–1147, 2008.

[30] Rand Askalan, Suzanne Laughlin, Supriya Mayank, Anthony Chan, Daune MacGregor, Maureen Andrew, Rosalind Curtis, Brandon Meaney, and Gabrielle deVeber. Chickenpox and stroke in childhood. *Stroke*, 32(6):1257–1262, 2001.

[31] Hal L Smith. Subharmonic bifurcation in an sir epidemic model. *Journal of Mathematical Biology*, 17(2):163–177, 1983.

[32] Centers for Disease Control (CDC et al. Measles prevention. *MMWR supplements*, 38(9):1, 1989.

[33] Anders Hviid, Steven Rubin, and Kathrin Mühlemann. Mumps. *The Lancet*, 371(9616):932–944, 2008.

[34] Herbert W Hethcote and James A Yorke. *Gonorrhea transmission dynamics and control*, volume 56. Springer, 2014.

[35] James A Yorke, Herbert W Hethcote, and Annett Nold. Dynamics and control of the transmission of gonorrhea. *Sexually Transmitted Diseases*, 5(2):51–56, 1978.

[36] Herbert W Hethcote. *Modeling heterogeneous mixing in infectious disease dynamics*. Cambridge: Cambridge University Press, 1996.

[37] Annett Nold. Heterogeneity in disease-transmission modelling. *Mathematical Biosciences*, 52(3-4):227–240, 1980.

[38] Ana Lajmanovich and James A Yorke. A deterministic model for gonorrhea in a nonhomogeneous population. *Mathematical Biosciences*, 28(3-4):221–236, 1976.

[39] Wuqiong Luo and Wee-Peng Tay. Finding an infection source under the sis model. In *2013 IEEE International Conference on Acoustics, Speech and Signal Processing*, pages 2930–2934. IEEE, 2013.

[40] Jin-Cheol Kim, Yong-Keun Lee, Bum-Soon Lim, Sang-Hoon Rhee, and Hyeong-Cheol Yang. Comparison of tensile and knot security properties of surgical sutures. *Journal of Materials Science: Materials in Medicine*, 18(12):2363–2369, 2007.

[41] Wuqiong Luo and Wee-Peng Tay. Identifying multiple infection sources in a network. In *2012 Conference Record of the Forty Sixth Asilomar Conference on Signals, Systems and Computers (ASILOMAR)*, pages 1483–1489. IEEE, 2012.

[42] Meeyoung Cha, Hamed Haddadi, Fabricio Benevenuto, and Krishna P Gummadi. Measuring user influence in twitter: The million follower fallacy. In *Fourth International AAAI Conference on Weblogs and Social Media*, 2010.

[43] Ricardo Almeida, Artur MC Brito da Cruz, Natália Martins, and M Teresa T Monteiro. An epidemiological mseir model described by the caputo fractional derivative. *International Journal of Dynamics and Control*, 7(2):776–784, 2019.

[44] Michael Y Li, John R Graef, Liancheng Wang, and János Karsai. Global dynamics of a SEIR model with varying total population size. *Mathematical Biosciences*, 160(2):191–213, 1999.

[45] David Greenhalgh and Gordon Hay. Mathematical modelling of the spread of HIV/AIDS amongst injecting drug users. *Mathematical Medicine and Biology*, 14(1):11–38, 1997.

[46] Edward H Kaplan. Needles that kill: modeling human immunodeficiency virus transmission via shared drug injection equipment in shooting galleries. *Reviews of Infectious Diseases*, 11(2):289–298, 1989.

[47] Linda Nash. *Inescapable ecologies: A history of environment, disease, and knowledge*. Univ of California Press, 2006.

[48] Austin Bradford Hill. The environment and disease: association or causation? *Proceedings of Royal Society of Medicine*, 58(5):295–300, 1965.

[49] Anthony J McMichael. Population, environment, disease, and survival: past patterns, uncertain futures. *The Lancet*, 359(9312):1145–1148, 2002.

[50] David A Vasseur and Peter Yodzis. The color of environmental noise. *Ecology*, 85(4):1146–1152, 2004.

[51] World Health Organization. Climate change and human health. `https://www.who.int/globalchange/climate/summary/en/index5.html`. last accessed on 02/11/2019.

[52] Jonathan A Patz, Thaddeus K Graczyk, Nina Geller, and Amy Y Vittor. Effects of environmental change on emerging parasitic diseases. *International Journal for Parasitology*, 30(12-13):1395–1405, 2000.

[53] Lev S Tsimring. Noise in biology. *Reports on Progress in Physics*, 77(2):026601, 2014.

[54] Hui-Hsiung Kuo. *White noise distribution theory*. CRC press, 2018.

[55] Kiyosi Itô. Stochastic integration. In Don H Tucker and Hugh B Maynard, editors, *Vector and operator valued measures and applications*, pages 141–148. Elsevier, 1973.

[56] Ali H Nayfeh. *Perturbation methods*. John Wiley & Sons, 2008.

[57] Alok Ranjan, Nagarajan Raghavan, Kalya Shubhakar, Ramesh Thamankar, Joel Molina, Sean J O'Shea, Michael Bosman, and Kin-Leong Pey. CAFM based spectroscopy of stress-induced defects in HfO 2 with experimental evidence of the clustering model and metastable vacancy defect state. In Gaudenzio Menghesso, editor, *2016 IEEE International Reliability Physics Symposium (IRPS)*, pages 7A–4. IEEE, 2016.

[58] Bipin Rajendran and Fabian Pease. Review of noise in semiconductor devices and modeling of noise in surrounding gate MOSFET. *Department of Electrical Engineering, Stanford University*, 2001.

[59] Paul A Gagniuc. *Markov chains: from theory to implementation and experimentation*. John Wiley & Sons, 2017.

[60] David Greenhalgh, Yanfeng Liang, and Xuerong Mao. Modelling the effect of telegraph noise in the SIRS epidemic model using markovian switching. *Physica A: Statistical Mechanics and its Applications*, 462:684–704, 2016.

[61] Manuel O Cáceres and Iris Cáceres-Saez. Random Leslie matrices in population dynamics. *Journal of Mathematical Biology*, 63(3):519–556, 2011.

[62] James D Hamilton. Regime switching models. In Steven N Durlauf and Lawrence E Blume, editors, *Macroeconometrics and time series analysis*, pages 202–209. Springer, 2010.

[63] Elisabetta Tornatore, Stefania Maria Buccellato, and Pasquale Vetro. Stability of a stochastic SIR system. *Physica A: Statistical Mechanics and its Applications*, 354:111–126, 2005.

[64] Chunyan Ji, Daqing Jiang, and Ningzhong Shi. The behavior of an SIR epidemic model with stochastic perturbation. *Stochastic Analysis and Applications*, 30(5):755–773, 2012.

[65] Chunyan Ji, Daqing Jiang, and Ningzhong Shi. Multigroup SIR epidemic model with stochastic perturbation. *Physica A: Statistical Mechanics and its Applications*, 390(10):1747–1762, 2011.

[66] Yuguo Lin, Daqing Jiang, and Peiyan Xia. Long-time behavior of a stochastic SIR model. *Applied Mathematics and Computation*, 236:1–9, 2014.

[67] Daqing Jiang, Jiajia Yu, Chunyan Ji, and Ningzhong Shi. Asymptotic behavior of global positive solution to a stochastic SIR model. *Mathematical and Computer Modelling*, 54(1-2):221–232, 2011.

[68] Qiuying Lu. Stability of SIRS system with random perturbations. *Physica A: Statistical Mechanics and Its Applications*, 388(18):3677–3686, 2009.

[69] Yasuhiro Takeuchi, Nguyen H Du, Nguyen T Hieu, and Kazunori Sato. Evolution of predator–prey systems described by a Lotka–Volterra equation under random environment. *Journal of Mathematical Analysis and Applications*, 323(2):938–957, 2006.

[70] Qi Luo and Xuerong Mao. Stochastic population dynamics under regime switching. *Journal of Mathematical Analysis and applications*, 334(1):69–84, 2007.

[71] Xiaoyue Li, Daqing Jiang, and Xuerong Mao. Population dynamical behavior of Lotka–Volterra system under regime switching. *Journal of Computational and Applied Mathematics*, 232(2):427–448, 2009.

[72] Rafail Khasminskii. *Stochastic stability of differential equations*, volume 66. Springer Science & Business Media, 2011.

[73] Hong Liu, Xiaoxia Li, and Qingshan Yang. The ergodic property and positive recurrence of a multi-group Lotka–Volterra mutualistic system with regime switching. *Systems & Control Letters*, 62(10):805–810, 2013.

[74] Theodore H Fleming and J Nathaniel Holland. The evolution of obligate pollination mutualisms: senita cactus and senita moth. *Oecologia*, 114(3):368–375, 1998.

[75] J Nathaniel Holland and Theodore H Fleming. Geographic and population variation in pollinating seed-consuming interactions between senita cacti (*Lophocereus schottii*) and senita moths (*Upiga virescens*). *Oecologia*, 121(3):405–410, 1999.

[76] J Nathaniel Holland, Donald L DeAngelis, and Judith L Bronstein. Population dynamics and mutualism: functional responses of benefits and costs. *The American Naturalist*, 159(3):231–244, 2002.

[77] Sulin Pang, Feiqi Deng, and Xuerong Mao. Asymptotic properties of stochastic population dynamics. *Dynamics of Continuous Discrete and Impulsive Systems Series A: Mathematical Analysis*, 15(5a):603–620, 2008.

[78] Yang Kuang. *Delay differential equations: with applications in population dynamics*, volume 191. Academic press, 1993.

[79] Connell C McCluskey. Complete global stability for an SIR epidemic model with delay—distributed or discrete. *Nonlinear Analysis: Real World Applications*, 11(1):55–59, 2010.

[80] Xuerong Mao, Chenggui Yuan, and Jiezhong Zou. Stochastic differential delay equations of population dynamics. *Journal of Mathematical Analysis and Applications*, 304(1):296–320, 2005.

[81] Jun Peng and Zaiming Liu. Stability analysis of stochastic reaction-diffusion delayed neural networks with Lévy noise. *Neural Computing and Applications*, 20(4):535–541, 2011.

[82] Andreas E Kyprianou. *Introductory lectures on fluctuations of Lévy processes with applications*. Springer Science & Business Media, 2006.

[83] Meng Liu and Ke Wang. Stochastic Lotka–Volterra systems with Lévy noise. *Journal of Mathematical Analysis and Applications*, 410(2):750–763, 2014.

[84] Jianhai Bao and Chenggui Yuan. Stochastic population dynamics driven by Lévy noise. *Journal of Mathematical Analysis and Applications*, 391(2):363–375, 2012.

[85] Shi-Jie Deng and Wenjiang Jiang. Lévy process-driven mean-reverting electricity price model: the marginal distribution analysis. *Decision Support Systems*, 40(3-4):483–494, 2005.

[86] Martin Baxter. Lévy process dynamic modelling of single-name credits and CDO tranches. *Nomura Fixed Income Quant Group*, 2006.

[87] Li Jianquan and Ma Zhien. Global analysis of SIS epidemic models with variable total population size. *Mathematical and Computer Modelling*, 39(11-12):1231–1242, 2004.

[88] Yanan Zhao and Daqing Jiang. The threshold of a stochastic SIS epidemic model with vaccination. *Applied Mathematics and Computation*, 243:718–727, 2014.

[89] Yuguo Lin, Daqing Jiang, and Shuai Wang. Stationary distribution of a stochastic SIS epidemic model with vaccination. *Physica A: Statistical Mechanics and its Applications*, 394:187–197, 2014.

[90] Wei Gou and Zhen Jin. How heterogeneous susceptibility and recovery rates affect the spread of epidemics on networks. *Infectious Disease Modelling*, 2(3):353–367, 2017.

[91] Xuerong Mao. *Stochastic differential equations and applications*. Elsevier, 2007.

[92] Reuben Hersh and Richard J Griego. Brownian motion and potential theory. *Scientific American*, 220(3):66–77, 1969.

[93] David J Mabberley. Robert Brown of the British Museum: some ramifications. *Archives of Natural History*, 1(1):101–109, 1981.

[94] Renato Fiorenza. *Hölder and locally Hölder Continuous Functions, and Open Sets of Class C^ k, C^{k, lambda}*. Birkhäuser, 2017.

[95] William J Anderson. *Continuous-time Markov chains: An applications-oriented approach*. Springer Science & Business Media, 2012.

[96] Boris Shulgin, Lewi Stone, and Zvia Agur. Pulse vaccination strategy in the SIR epidemic model. *Bulletin of Mathematical Biology*, 60(6):1123–1148, 1998.

[97] Vincenzo Capasso and Gabriella Serio. A generalization of the Kermack-McKendrick deterministic epidemic model. *Mathematical Biosciences*, 42(1-2):43–61, 1978.

[98] Fred Brauer. Mathematical epidemiology: Past, present, and future. *Infectious Disease Modelling*, 2(2):113–127, 2017.

[99] Xuerong Mao and Chenggui Yuan. *Stochastic differential equations with Markovian switching*. Imperial College Press, 2006.

[100] Yanfeng Liang, David Greenhalgh, and Xuerong Mao. A stochastic differential equation model for the spread of HIV amongst people who inject drugs. *Computational and Mathematical Methods in Medicine*, 2016, 2016.

[101] Edward Allen. *Modeling with Itô stochastic differential equations*, volume 22. Springer Science & Business Media, 2007.

[102] David Greenhalgh, Yanfeng Liang, and Xuerong Mao. Demographic stochasticity in the SDE SIS epidemic model. *Discrete and Continuous Dynamical Systems-Series B*, 20(9):2859–2884, 2015.

[103] Boqiang Cao, Meijing Shan, Qimin Zhang, and Weiming Wang. A stochastic SIS epidemic model with vaccination. *Physica A: Statistical Mechanics and its Applications*, 486:127–143, 2017.

[104] Athina Economou, A Gómez-Corral, and M López-García. A stochastic sis epidemic model with heterogeneous contacts. *Physica A: Statistical Mechanics and its Applications*, 421:78–97, 2015.

[105] Andrew L Krause, Lawrence Kurowski, Kamran Yawar, and Robert A Van Gorder. Stochastic epidemic metapopulation models on networks: SIS dynamics and control strategies. *Journal of Theoretical Biology*, 449:35–52, 2018.

[106] Yanan Zhao, Daqing Jiang, and Donal O'Regan. The extinction and persistence of the stochastic SIS epidemic model with vaccination. *Physica A: Statistical Mechanics and Its Applications*, 392(20):4916–4927, 2013.

[107] David Greenhalgh, Yanfeng Liang, and Xuerong Mao. SDE SIS epidemic model with demographic stochasticity and varying population size. *Applied Mathematics and Computation*, 276:218–238, 2016.

[108] Guixin Hu, Meng Liu, and Ke Wang. The asymptotic behaviours of an epidemic model with two correlated stochastic perturbations. *Applied Mathematics and Computation*, 218(21):10520–10532, 2012.

[109] Yongmei Cai and Xuerong Mao. Stochastic prey-predator system with foraging arena scheme. *Applied Mathematical Modelling*, 64:357–371, 2018.

[110] Sheldon M Ross. *Introduction to probability models*. Academic Press, 2014.

[111] Cathrin Van Emmerich. Modelling correlation as a stochastic process. *preprint*, 6(03), 2006.

[112] Yongmei Cai, Siyang Cai, and Xuerong Mao. Analysis of a stochastic predator-prey system with foraging arena scheme. *Stochastics*, 0(0):1–30, 2019.

[113] Desmond J Higham. An algorithmic introduction to numerical simulation of stochastic differential equations. *SIAM Review*, 43(3):525–546, 2001.

[114] Desmond J Higham, Xuerong Mao, and Andrew M Stuart. Strong convergence of Euler-type methods for nonlinear stochastic differential equations. *SIAM Journal on Numerical Analysis*, 40(3):1041–1063, 2002.

[115] Desmond J Higham and Xuerong Mao. Convergence of Monte Carlo simulations involving the mean-reverting square root process. *Journal of Computational Finance*, 8(3):35–61, 2005.

[116] Adi Ben-Israel. A Newton-Raphson method for the solution of systems of equations. *Journal of Mathematical Analysis and Applications*, 15(2):243–252, 1966.

[117] Montgomery Slatkin. The dynamics of a population in a Markovian environment. *Ecology*, 59(2):249–256, 1978.

[118] Xuerong Mao. Stationary distribution of stochastic population systems. *Systems & Control Letters*, 60(6):398–405, 2011.

[119] Xinhong Zhang, Daqing Jiang, Ahmed Alsaedi, and Tasawar Hayat. Stationary distribution of stochastic SIS epidemic model with vaccination under regime switching. *Applied Mathematics Letters*, 59:87–93, 2016.

[120] Meng Liu and Ke Wang. Stochastic differential equations with multi-Markovian switching. *Journal of Applied Mathematics*, 2013, 2013.

[121] Nguyen H Du, Ryusuke Kon, Kazunori Sato, and Yasuhiro Takeuchi. Dynamical behavior of Lotka–Volterra competition systems: non-autonomous bistable case and the effect of telegraph noise. *Journal of Computational and Applied Mathematics*, 170(2):399–422, 2004.

[122] George G Yin and Qing Zhang. *Continuous-time Markov chains and applications: a singular perturbation approach*, volume 37. Springer, 2012.

[123] Chao Zhu and George Yin. Asymptotic properties of hybrid diffusion systems. *SIAM Journal on Control and Optimization*, 46(4):1155–1179, 2007.

[124] James R Norris and James Robert Norris. *Markov chains*. Cambridge University Press, 1998.

[125] Adel Settati and Aadil Lahrouz. Stationary distribution of stochastic population systems under regime switching. *Applied Mathematics and Computation*, 244:235–243, 2014.

[126] Rafail Khasminskii, Chao Zhu, and George G Yin. Stability of regime-switching diffusions. *Stochastic Processes and Their Applications*, 117(8):1037–1051, 2007.

[127] Anatoliĭ Skorokhod. *Asymptotic methods in the theory of stochastic differential equations*, volume 78. American Mathematical Soc., 2009.