The SIS Epidemic Model with Markovian Switching

A. Gray, D. Greenhalgh, X. Mao, J. Pan

Department of Mathematics and Statistics, University of Strathclyde, Glasgow G1 1XH, U.K.

Abstract

Population systems are often subject to environmental noise. Motivated by Takeuchi et al. [36], we will discuss in this paper the effect of telegraph noise on the well-known SIS epidemic model. We establish the explicit solution of the stochastic SIS epidemic model, which is useful in performing computer simulations. We also establish the conditions for extinction and persistence for the stochastic SIS epidemic model and compare these with the corresponding conditions for the deterministic SIS epidemic model. We first prove these results for a two-state Markov chain and then generalise them to a finite state space Markov chain. Computer simulations based on the explicit solution and the Euler–Maruyama scheme are performed to illustrate our theory. We include a more realistic example using appropriate parameter values for the spread of *Streptococcus pneumoniae* in children.

Key words: SIS epidemic model, Markov chain, extinction, persistence, Streptococcus pneumoniae.

1 Introduction

Population systems are often subject to environmental noise and there are various types of environmental noise, e.g. white or colour noise (see e.g. [11, 15, 25, 26, 28, 34, 36]). It is therefore critical to discover whether the presence of such noise affects population systems significantly.

For example, consider a predator-prey Lotka-Volterra model

$$\begin{cases} \dot{x}_1(t) = x_1(t)(a_1 - b_1 x_2(t)), \\ \dot{x}_2(t) = x_2(t)(-c_1 + d_1 x_1(t)), \end{cases}$$
(1.1)

where a_1, b_1, c_1 and d_1 are positive numbers. It is well known that the population develops periodically if there is no influence of environmental noise (see e.g. [14, 35]). However, if the factor of environmental noise is taken into account, the system will change significantly. Consider a simple colour noise, say telegraph noise. Telegraph noise can be illustrated as a switching between two or more regimes of environment, which differ by factors such as nutrition or rainfall (see e.g. [11, 34]). The switching is memoryless and the waiting time for the next switch has an exponential distribution. We can hence model the regime switching by a finite-state Markov chain. To make it simple, assume that there are only two regimes and the system obeys equation (1.1) when it is in regime 1, while it obeys another predator-prey Lotka-Volterra model

$$\begin{cases} \dot{x}_1(t) = x_1(t)(a_2 - b_2 x_2(t)), \\ \dot{x}_2(t) = x_2(t)(-c_2 + d_2 x_1(t)) \end{cases}$$
(1.2)

in regime 2. The switching between these two regimes is governed by a Markov chain r(t) on the state space $\mathbb{S} = \{1, 2\}$. The population system under regime switching can therefore be described by the stochastic model

$$\begin{cases} \dot{x}_1(t) = x_1(t)(a_{r(t)} - b_{r(t)}x_2(t)), \\ \dot{x}_2(t) = x_2(t)(-c_{r(t)} + d_{r(t)}x_1(t)). \end{cases}$$
(1.3)

This system is operated as follows: If r(0) = 1, the system obeys equation (1.1) till time τ_1 when the Markov chain jumps to state 2 from state 1; the system will then obey equation (1.2) from time τ_1 till time τ_2 when the Markov chain jumps to state 1 from state 2. The system will continue to switch as long as the Markov chain jumps. If r(0) = 2, the system will switch similarly. In other words, equation (1.3) can be regarded as equations (1.1) and (1.2) combined, switching from one to the other according to the law of the Markov chain. Equations (1.1) and (1.2) are hence called the subsystems of equation (1.3).

Clearly, equations (1.1) and (1.2) have their unique positive equilibrium states as $(p_1, q_1) = (c_1/d_1, a_1/b_1)$ and $(p_2, q_2) = (c_2/d_2, a_2/b_2)$, respectively. Recently, Takeuchi et al. [36] revealed a very interesting and surprising result: If the two equilibrium states of the subsystems are different, then all positive trajectories of equation (1.3) always exit from any compact set of \mathbb{R}^2_+ with probability 1; on the other hand, if the two equilibrium states coincide, then the trajectory either leaves from any compact set of \mathbb{R}^2_+ or converges to the equilibrium state. In practice, two equilibrium states are usually different, whence Takeuchi et al. [36] show that equation (1.3) is neither permanent nor dissipative. This is an important result as it reveals the significant effect of environmental noise on the population system: both subsystems (1.1) and (1.2) develop periodically, but switching between them makes them become neither permanent nor dissipative.

Markovian environments are also very popular in many other fields of biology. As examples Padilla and Adolph [32] present a mathematical model for predicting the expected fitness of phenotypically plastic organisms experiencing a variable environment and discuss the importance of time delays in this model, and Anderson [1] discusses the optimal exploitation strategies for an animal population in a Markovian environment. Additionally Peccoud and Ycart [33] propose a Markovian model for the gene induction process, and Caswell and Cohen [8] discuss the effects of the spectra of the environmental variation in the coexistence of metapopulations.

Motivated by Takeuchi et al. [36], we will discuss the effect of telegraph noise on the well-known SIS epidemic model [17, 18]. The SIS epidemic model is one of the simplest epidemic models and is often used in the literature to model diseases for which there is no immunity. Examples of such diseases include gonorrhea [18], pneumococcus [21, 23] and tuberculosis. Continuous time Markov chain and stochastic differential equation SIS epidemic models are discussed by Brauer et al. [6] in their textbook. Ianelli, Milner and Pugliese [20] study age-structured epidemic models, as do Feng, Huang and Castillo-Chavez [13]. Neal [30, 31] studies

deterministic and stochastic SIS epidemic models. Li, Ma and Zhu [22] analyse backward bifurcation in an SIS epidemic model with vaccination and Van den Driessche and Watmough [37] study backward bifurcation in an SIS epidemic model with hysteresis. More recently, Andersson and Lindenstrand [3] analyse an open population stochastic SIS epidemic model where both infectious and susceptible individuals reproduce and die. Gray et al. [16] establish the stochastic SIS model by parameter perturbation. There are many other examples of SIS epidemic models in the literature. Also, other two similar models for diseases with permanent immunity and diseases with a latent period before becoming infectious, the SIR (Susceptible-Infectious-Recovered) and the SEIR (Susceptible-Exposed-Infectious-Recovered) model respectively are studied by Yang et al. [39] and stochastic perturbations are introduced in these two models. Liu and Stechlinski [24] analyse the stochastic SIR model with contact rate being modelled by a switching parameter. Bhattacharyya and Mukhopadhyay [5] study the SI (Susceptible-Infected) model for prey disease with prey harvesting and predator switching. Artelejo, Economou and Lopez-Herrero [4] propose some efficient methods to obtain the distribution of the number of recovered individuals and discuss its relationship with the final epidemic size in the SIS and SIR stochastic epidemic models.

The classical deterministic SIS epidemic model is described by the following 2-dimensional ODE

$$\begin{cases} \frac{dS(t)}{dt} = \mu N - \beta S(t)I(t) + \gamma I(t) - \mu S(t), \\ \frac{dI(t)}{dt} = \beta S(t)I(t) - (\mu + \gamma)I(t), \end{cases}$$
(1.4)

subject to S(t) + I(t) = N, along with the initial values $S(0) = S_0 > 0$ and $I(0) = I_0 > 0$, where I(t) and S(t) are respectively the number of infectious and susceptible individuals at time t in a population of size N, and μ and γ^{-1} are the average death rate and the average infectious period respectively. β is the disease transmission coefficient, so that $\beta = \lambda/N$, where λ is the disease contact rate of an infective individual. λ is the per day average number of contacts which if made with a susceptible individual would result in the susceptible individual becoming infected.

It is easy to see that I(t) obeys the scalar Lotka–Volterra model

$$\frac{dI(t)}{dt} = I(t)[\beta N - \mu - \gamma - \beta I(t)], \qquad (1.5)$$

which has the explicit solution

$$I(t) = \begin{cases} \left[e^{-(\beta N - \mu - \gamma)t} \left(\frac{1}{I_0} - \frac{\beta}{\beta N - \mu - \gamma} \right) + \frac{\beta}{\beta N - \mu - \gamma} \right]^{-1}, & \text{if } \beta N - \mu - \gamma \neq 0, \\ \left[\frac{1}{I_0} + \beta t \right]^{-1}, & \text{if } \beta N - \mu - \gamma = 0. \end{cases}$$
(1.6)

Defining the basic reproduction number for the deterministic SIS model

$$R_0^D = \frac{\beta N}{\mu + \gamma},\tag{1.7}$$

we can conclude (see e.g. [35]):

• If $R_0^D \leq 1$, $\lim_{t\to\infty} I(t) = 0$.

• If $R_0^D > 1$, $\lim_{t\to\infty} I(t) = \frac{\beta N - \mu - \gamma}{\beta}$. In this case, I(t) will monotonically decrease or increase to $\frac{\beta N - \mu - \gamma}{\beta}$ if $I(0) > \frac{\beta N - \mu - \gamma}{\beta}$ or $< \frac{\beta N - \mu - \gamma}{\beta}$, respectively, while $I(t) \equiv \frac{\beta N - \mu - \gamma}{\beta}$ if $I(0) = \frac{\beta N - \mu - \gamma}{\beta}$.

Taking into account the environmental noise, the system parameters μ , β and γ may experience abrupt changes. In the same fashion as in Takeuchi et al. [36], we may model these abrupt changes by a Markov chain. As a result, the classical SIS model (1.4) evolves to a stochastic SIS model with Markovian switching of the form

$$\begin{cases} \frac{dS(t)}{dt} = \mu_{r(t)}N - \beta_{r(t)}S(t)I(t) + \gamma_{r(t)}I(t) - \mu_{r(t)}S(t), \\ \frac{dI(t)}{dt} = \beta_{r(t)}S(t)I(t) - (\mu_{r(t)} + \gamma_{r(t)})I(t), \end{cases}$$
(1.8)

where r(t) is a Markov chain with a finite state space. The main aim of this paper is to discuss the effect of the noise in the form of Markov switching. We will not only show the explicit solution but will also investigate the asymptotic properties, including extinction and persistence.

To make our theory more understandable, we will begin with the special case where the Markov chain has only 2 states, as in Takeuchi et al. [36]. We will then generalise our theory to the general case where the Markov chain has a finite number of states, M.

2 SIS Model with Markovian Switching

Throughout this paper, unless otherwise specified, we let $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq 0}, \mathbb{P})$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ satisfying the usual conditions (i.e. it is increasing and right continuous while \mathcal{F}_0 contains all \mathbb{P} -null sets). Let $r(t), t \geq 0$, be a right-continuous Markov chain on the probability space taking values in the state space $\mathbb{S} = \{1, 2\}$ with the generator

$$\Gamma = \begin{pmatrix} -\nu_{12} & \nu_{12} \\ \\ \nu_{21} & -\nu_{21} \end{pmatrix}.$$

Here $\nu_{12} > 0$ is the transition rate from state 1 to 2, while $\nu_{21} > 0$ is the transition rate from state 2 to 1, that is

$$\mathbb{P}\{r(t+\delta) = 2|r(t) = 1\} = \nu_{12}\delta + o(\delta) \text{ and } \mathbb{P}\{r(t+\delta) = 1|r(t) = 2\} = \nu_{21}\delta + o(\delta),$$

where $\delta > 0$. It is well known (see e.g. [2]) that 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 $\mathbb{R}_+ := [0, \infty)$. More precisely, there is a sequence $\{\tau_k\}_{k\geq 0}$ of finite-valued \mathcal{F}_t -stopping times such that $0 = \tau_0 < \tau_1 < \cdots < \tau_k \to \infty$ almost surely and

$$r(t) = \sum_{k=0}^{\infty} r(\tau_k) I_{[\tau_k, \tau_{k+1})}(t), \qquad (2.1)$$

where throughout this paper I_A denotes the indicator function of set A. Moreover, given that $r(\tau_k) = 1$, the random variable $\tau_{k+1} - \tau_k$ follows the exponential distribution with parameter ν_{12} , namely

$$\mathbb{P}(\tau_{k+1} - \tau_k \ge T | r(\tau_k) = 1) = e^{-\nu_{12}T}, \quad \forall T \ge 0,$$

while given that $r(\tau_k) = 2$, $\tau_{k+1} - \tau_k$ follows the exponential distribution with parameter ν_{21} , namely

$$\mathbb{P}(\tau_{k+1} - \tau_k \ge T | r(\tau_k) = 2) = e^{-\nu_{21}T}, \quad \forall T \ge 0.$$

The sample paths of the Markov chain can therefore be simulated easily using these exponential distributions (we will illustrate this in Section 6 below). Furthermore, this Markov chain has a unique stationary distribution $\Pi = (\pi_1, \pi_2)$ given by

$$\pi_1 = \frac{\nu_{21}}{\nu_{12} + \nu_{21}}, \quad \pi_2 = \frac{\nu_{12}}{\nu_{12} + \nu_{21}}.$$
(2.2)

After recalling these fundamental concepts of the Markov chain, let us return to the stochastic SIS epidemic model (1.8). We assume that the system parameters β_i, μ_i, γ_i $(i \in \mathbb{S})$ are all positive numbers. Given that I(t) + S(t) = N, we see that I(t), the number of infectious individuals, obeys the stochastic Lotka–Volterra model with Markovian switching given by

$$\frac{dI(t)}{dt} = I(t)[\alpha_{r(t)} - \beta_{r(t)}I(t)], \qquad (2.3)$$

where

$$\alpha_i := \beta_i N - \mu_i - \gamma_i, \quad i \in \mathbb{S}.$$
(2.4)

It is sufficient to study equation (2.3) in order to understand the full dynamics of the stochastic SIS epidemic model (1.8), hence we will concentrate on this equation only in the remainder of this paper. We will refer to equations (1.8) or (2.3) in the rest of the paper as 'the stochastic SIS model' or 'the stochastic SIS epidemic model', although other stochastic versions of the SIS model exist, e.g. a simple Markovian model describing only demographic stochasticity. The following theorem shows that this equation has an explicit solution for any given initial value in (0, N).

Theorem 2.1 For any given initial value $I(0) = I_0 \in (0, N)$, there is a unique solution I(t) on $t \in \mathbb{R}_+$ to equation (2.3) such that

$$\mathbb{P}(I(t) \in (0, N) \text{ for all } t \ge 0) = 1.$$

Moreover, the solution has the explicit form

$$I(t) = \frac{\exp\left(\int_0^t \alpha_{r(s)} ds\right)}{\frac{1}{I_0} + \int_0^t \exp\left(\int_0^s \alpha_{r(u)} du\right) \beta_{r(s)} ds}.$$
(2.5)

Proof. Fix any sample path of the Markov chain. Without loss of generality we may assume that this sample path has its initial value r(0) = 1, as the proof is the same if r(0) = 2. We first observe from (2.1) that r(t) = 1 for $t \in [\tau_0, \tau_1)$. Hence equation (2.3) becomes

$$\frac{dI(t)}{dt} = I(t)[\alpha_1 - \beta_1 I(t)]$$

on $t \in [\tau_0, \tau_1)$. But this equation has a unique solution on the entire set of $t \in \mathbb{R}_+$ and the solution will remain within (0, N). Hence the solution of equation (2.3), I(t), is uniquely determined on $t \in [\tau_0, \tau_1)$ and, by continuity, for $t = \tau_1$ as well. Obtaining $I(\tau_1) \in (0, N)$, we further consider equation (2.3) for $t \in [\tau_1, \tau_2)$, which has the form

$$\frac{dI(t)}{dt} = I(t)[\alpha_2 - \beta_2 I(t)].$$

This equation has a unique solution on $t \ge \tau_1$ and the solution will remain within (0, N). Hence the solution of equation (2.3), I(t), is uniquely determined on $t \in [\tau_1, \tau_2)$ and, by continuity, for $t = \tau_2$ as well. Repeating this procedure, we see that equation (2.3) has a unique solution I(t) on $t \in \mathbb{R}_+$ and the solution remains within (0, N) with probability one.

After showing $I(t) \in (0, N)$, we may define

$$y(t) = \frac{1}{I(t)}, \quad t \ge 0,$$

in order to obtain the explicit solution. Compute

$$\frac{dy(t)}{dt} = -\frac{1}{I(t)^2} \frac{dI(t)}{dt}$$
$$= -\frac{1}{I(t)^2} I(t) (\alpha_{r(t)} - \beta_{r(t)} I(t))$$
$$= \beta_{r(t)} - \frac{\alpha_{r(t)}}{I(t)}$$
$$= \beta_{r(t)} - \alpha_{r(t)} y(t).$$

By the well-known variation-of-constants formula (see e.g. [28, p.96]), we have

$$y(t) = \Phi(t) \Big(y(0) + \int_0^t \Phi^{-1}(s) \beta_{r(s)} \, ds \Big),$$

where $\Phi(t) = e^{-\int_0^t \alpha_{r(s)} ds}$. This yields the desired explicit solution (2.5) immediately. \Box

3 The Basic Reproduction Number

Naturally we wish to examine the behaviour of the stochastic SIS epidemic model (2.3) and we may ask what is the corresponding basic reproduction number R_0^S ? Recall that the basic reproduction number is the expected number of secondary cases caused by a single newly-infected case entering the disease-free population at equilibrium [10].

In our case the disease-free equilibrium (DFE) is S = N, I = 0. The individuals can be divided into two types, those who arrive when r(t) = 1, and those that arrive when r(t) = 2. Suppose that a newly infected individual enters the DFE when the Markov chain is in state 1. Then the next events that can happen are that the individual dies at rate μ_1 , recovers at rate γ_1 or the Markov chain switches at rate ν_{12} . Hence the expected time until the first event is

$$\frac{1}{\mu_1 + \gamma_1 + \nu_{12}}$$

During this time each of the N susceptibles at the DFE is infected at rate β_1 . So the total expected number of people infected in this time interval is

$$\frac{\beta_1 N}{\mu_1 + \gamma_1 + \nu_{12}}.$$
(3.1)

If the first event is either that the infected individual dies or recovers, no more people will be infected before the first switch. If the first event is that the Markov chain switches the expected number of people infected before the first switch is given by (3.1). Hence whatever happens the expected number of people infected before the first switch is given by (3.1).

The expected number of individuals infected between the first and second switches is

$$\frac{\nu_{12}}{\mu_1 + \gamma_1 + \nu_{12}} \frac{\beta_2 N}{\mu_2 + \gamma_2 + \nu_{21}}$$

and between the second and third switches

$$\frac{\nu_{21}}{\mu_2 + \gamma_2 + \nu_{21}} \frac{\nu_{12}}{\mu_1 + \gamma_1 + \nu_{12}} \frac{\beta_1 N}{\mu_1 + \gamma_1 + \nu_{12}} = p \frac{\beta_1 N}{\mu_1 + \gamma_1 + \nu_{12}}$$

where $p = \frac{\nu_{12}\nu_{21}}{(\mu_1 + \gamma_1 + \nu_{12})(\mu_2 + \gamma_2 + \nu_{21})}$.

Hence this individual infects in total

$$m_{11} = \frac{\beta_1 N}{\mu_1 + \gamma_1 + \nu_{12}} (1 + p + p^2 + \dots) = \frac{\beta_1 N}{\mu_1 + \gamma_1 + \nu_{12}} \frac{1}{1 - p}$$

individuals while the Markov chain is in state 1 and

$$m_{12} = \frac{\nu_{12}}{\mu_1 + \gamma_1 + \nu_{12}} \frac{\beta_2 N}{\mu_2 + \gamma_2 + \nu_{21}} \frac{1}{1 - p}$$

individuals while the Markov chain is in state 2.

Similarly we can derive the expected number of individuals infected by a single newly infected individual entering the DFE when the Markov chain is in state 2. We deduce that the next generation matrix giving the expected number of secondary cases caused by a single newly infected individual entering the DFE is

$$\begin{pmatrix} m_{11} & m_{12} \\ m_{21} & m_{22} \end{pmatrix} = \frac{1}{1-p} \begin{pmatrix} a_1 & p_1 a_2 \\ p_2 a_1 & a_2 \end{pmatrix},$$

where $a_1 = \frac{\beta_1 N}{\mu_1 + \gamma_1 + \nu_{12}}, \ a_2 = \frac{\beta_2 N}{\mu_2 + \gamma_2 + \nu_{21}}, \ p_1 = \frac{\nu_{12}}{\mu_1 + \gamma_1 + \nu_{12}}$ and

 $p_2 = \frac{\nu_{21}}{\mu_2 + \gamma_2 + \nu_{21}}.$

The basic reproduction number for the stochastic epidemic model is the largest eigenvalue of this matrix

$$R_0^S = \frac{a_1 + a_2 + \sqrt{(a_1 + a_2)^2 - 4a_1a_2(1 - p)}}{2(1 - p)}.$$
(3.2)

However we do not pursue this further here.

4 Extinction

In the study of the SIS epidemic model, extinction is one of the important issues. In this section we will discuss this issue. Recall that for the deterministic SIS epidemic model (1.5), the basic reproduction number R_0^D was also the threshold between disease extinction and persistence, with extinction for $R_0^D \leq 1$ and persistence for $R_0^D > 1$. In the stochastic model, there are different types of extinction and persistence, for example almost sure extinction, extinction in mean square and extinction in probability. In the rest of the paper we examine a threshold

$$T_0^S = \frac{\pi_1 \beta_1 N + \pi_2 \beta_2 N}{\pi_1 (\mu_1 + \gamma_1) + \pi_2 (\mu_2 + \gamma_2)}$$
(4.1)

for almost sure extinction or persistence of our stochastic epidemic model. However this threshold is different to R_0^S which might be more relevant to other types of extinction or persistence.

We will see later that the stochastic SIS model (2.3) will become extinct (meaning that $\lim_{t\to\infty} I(t) = 0$) with probability one if $T_0^S < 1$. Before we state this result, let us state a proposition which gives an equivalent condition for $T_0^S < 1$ in terms of the system parameters α_i and the stationary distribution of the Markov chain.

Proposition 4.1 We have the following alternative condition on the value of T_0^S :

- $T_0^S < 1$ 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 $\pi_1 \alpha_1 + \pi_2 \alpha_2 > 0$.

The proof of this proposition is straightforward, so is omitted. We can now state our theory on extinction.

Theorem 4.2 If $T_0^S < 1$, then, for any given initial value $I_0 \in (0, N)$, the solution of the stochastic SIS epidemic model (2.3) obeys

$$\limsup_{t \to \infty} \frac{1}{t} \log(I(t)) \le \alpha_1 \pi_1 + \alpha_2 \pi_2 \quad a.s.$$

$$(4.2)$$

By Proposition 4.1, we hence conclude that I(t) tends to zero exponentially almost surely. In other words, the disease dies out with probability one.

Proof. It is easy to see that

$$\frac{d\log(I(t))}{dt} = \alpha_{r(t)} - \beta_{r(t)}I(t).$$
(4.3)

This implies that, for any t > 0,

$$\frac{\log(I(t))}{t} \le \frac{\log(I(0))}{t} + \frac{1}{t} \int_0^t \alpha_{r(s)} ds,$$

since $\beta_{r(t)} > 0$ and $I(t) \in (0, N)$. Letting $t \to \infty$ we hence obtain

$$\limsup_{t \to \infty} \frac{1}{t} \log(I(t)) \le \limsup_{t \to \infty} \frac{1}{t} \int_0^t \alpha_{r(s)} ds$$

However, by the ergodic theory of the Markov chain (see e.g. [2]) we have

$$\lim_{t \to \infty} \frac{1}{t} \int_0^t \alpha_{r(s)} ds = \alpha_1 \pi_1 + \alpha_2 \pi_2 \quad a.s$$

We therefore must have

$$\limsup_{t \to \infty} \frac{1}{t} \log(I(t)) \le \alpha_1 \pi_1 + \alpha_2 \pi_2 \quad a.s.,$$

as required. \Box

Let us now make a few comments. First of all, let us recall that the stochastic SIS model (2.3) can be regarded as the result of the following two subsystems

$$\frac{dI(t)}{dt} = I(t)[\alpha_1 - \beta_1 I(t)] \tag{4.4}$$

and

$$\frac{dI(t)}{dt} = I(t)[\alpha_2 - \beta_2 I(t)], \qquad (4.5)$$

switching from one to the other according to the law of the Markov chain. If both $\alpha_1 < 0$ and $\alpha_2 < 0$, then the corresponding R_0^D values for both subsystems (4.4) and (4.5) are less than 1, whence both subsystems become extinct. In this case, T_0^S for the stochastic SIS model (2.3) is less than 1, hence it will become extinct, and of course this is not surprising. However, if only one of α_1 and α_2 is negative, say $\alpha_1 < 0$ and $\alpha_2 > 0$, for example, one subsystem (4.4) becomes extinct but the other (4.5) is persistent. However, if the rate of the Markov chain switching from state 2 to 1 is relatively faster than that from 1 to 2, so that $\alpha_1\pi_1 + \alpha_2\pi_2 < 0$, then the overall system (2.3) will become extinct. This reveals the important role of the Markov chain in the extinction.

We next recall that in the deterministic SIS model (1.5) the disease will always go extinct even if $R_0^D = 1$. The reader may ask what happens to the stochastic SIS model (2.3) if the corresponding $T_0^S = 1$? Although we have a strong feeling that the disease will always become extinct, we have not been able to prove it so far. In Section 6.3 we show some simulations to illustrate this case.

5 Persistence

Let us now turn to the case when $T_0^S > 1$. The following theorem shows that the disease will be persistent in this case, meaning that $\lim_{t\to\infty} I(t) > 0$.

Theorem 5.1 If $T_0^S > 1$, then, for any given initial value $I_0 \in (0, N)$, the solution of the stochastic SIS model (2.3) has the properties that

$$\liminf_{t \to \infty} I(t) \le \frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} \quad a.s.$$
(5.1)

and

$$\limsup_{t \to \infty} I(t) \ge \frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} \quad a.s.$$
(5.2)

In other words, the disease will reach the neighbourhood of the level $\frac{\pi_1\alpha_1 + \pi_2\alpha_2}{\pi_1\beta_1 + \pi_2\beta_2}$ infinitely many times with probability one.

Proof. Let us first prove assertion (5.1). If this were not true, then we can find an $\varepsilon > 0$ sufficiently small for $\mathbb{P}(\Omega_1) > 0$ where

$$\Omega_1 = \left\{ \omega \in \Omega : \liminf_{t \to \infty} I(t) > \frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} + \varepsilon \right\}.$$
(5.3)

On the other hand, by the ergodic theory of the Markov chain, we have that $\mathbb{P}(\Omega_2) = 1$, where for any $\omega \in \Omega_2$,

$$\lim_{t \to \infty} \frac{1}{t} \int_0^t \left(\alpha_{r(s)} - \beta_{r(s)} \left[\frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} + \varepsilon \right] \right) ds = \pi_1 \left(\alpha_1 - \beta_1 \left[\frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} + \varepsilon \right] \right) + \pi_2 \left(\alpha_2 - \beta_2 \left[\frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} + \varepsilon \right] \right) = -(\pi_1 \beta_1 + \pi_2 \beta_2) \varepsilon.$$
(5.4)

Now consider any $\omega \in \Omega_1 \cap \Omega_2$. Then there is a positive number $T = T(\omega)$ such that

$$I(t) \ge \frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} + \varepsilon \quad \forall t \ge T.$$

It then follows from (4.3) that

$$\log(I(t)) \le \log(I_0) + \int_0^T (\alpha_{r(s)} - \beta_{r(s)}I(s))ds + \int_T^t \left(\alpha_{r(s)} - \beta_{r(s)} \left[\frac{\pi_1\alpha_1 + \pi_2\alpha_2}{\pi_1\beta_1 + \pi_2\beta_2} + \varepsilon\right]\right)ds$$

for all $t \geq T$. Dividing both sides by t and then letting $t \to \infty$, we obtain that

$$\limsup_{t \to \infty} \frac{1}{t} \log(I(t)) \le -(\pi_1 \beta_1 + \pi_2 \beta_2)\varepsilon,$$

where (5.4) has been used. This implies that

$$\lim_{t \to \infty} I(t) = 0.$$

But this contradicts (5.3). The required assertion (5.1) must therefore hold.

The procedure to prove assertion (5.2) is very similar. In fact if (5.2) were not true, we can then find an $\varepsilon > 0$ sufficiently small for $\mathbb{P}(\Omega_3) > 0$, where

$$\Omega_3 = \left\{ \omega \in \Omega : \limsup_{t \to \infty} I(t) < \frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} - \varepsilon \right\}.$$
(5.5)

By the ergodic theory we also have that $\mathbb{P}(\Omega_4) = 1$, where for any $\omega \in \Omega_4$,

$$\lim_{t \to \infty} \frac{1}{t} \int_0^t \left(\alpha_{r(s)} - \beta_{r(s)} \left[\frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} - \varepsilon \right] \right) ds = \pi_1 \left(\alpha_1 - \beta_1 \left[\frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} - \varepsilon \right] \right) + \pi_2 \left(\alpha_2 - \beta_2 \left[\frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} - \varepsilon \right] \right) = (\pi_1 \beta_1 + \pi_2 \beta_2) \varepsilon.$$
(5.6)

If we consider any $\omega \in \Omega_3 \cap \Omega_4$, there is a positive number $T = T(\omega)$ such that

$$I(t) \le \frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} - \varepsilon \quad \forall t \ge T$$

From (4.3) we have that

$$\log(I(t)) \ge \log(I_0) + \int_0^T (\alpha_{r(s)} - \beta_{r(s)}I(s))ds + \int_T^t \left(\alpha_{r(s)} - \beta_{r(s)} \left[\frac{\pi_1\alpha_1 + \pi_2\alpha_2}{\pi_1\beta_1 + \pi_2\beta_2} - \varepsilon\right]\right)ds$$

for all $t \ge T$. Dividing both sides by t and then letting $t \to \infty$ while using (5.6) as well, we obtain that

$$\liminf_{t \to \infty} \frac{1}{t} \log(I(t)) \ge (\pi_1 \beta_1 + \pi_2 \beta_2)\varepsilon.$$

This implies that

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

which contradicts (5.5). Therefore assertion (5.2) must hold. \Box

To reveal more properties of the stochastic SIS model, we observe from Proposition 4.1 that $T_0^S > 1$ is equivalent to the condition that $\pi_1 \alpha_1 + \pi_2 \alpha_2 > 0$. This may be divided into two cases: (a) both α_1 and α_2 are positive; and (b) only one of α_1 and α_2 is positive. Without loss of generality, we may assume that $0 < \alpha_1/\beta_1 = \alpha_2/\beta_2$ or $0 < \alpha_1/\beta_1 < \alpha_2/\beta_2$ in Case (a), while $\alpha_1/\beta_1 \le 0 < \alpha_2/\beta_2$ in Case (b). So there are three different cases to be considered under condition $T_0^S > 1$. Let us present a lemma in order to show another new result.

Lemma 5.2 The following statements hold with probability one:

(i) If
$$0 < \alpha_1/\beta_1 = \alpha_2/\beta_2$$
, then $I(t) = \alpha_1/\beta_1$ for all $t > 0$ when $I_0 = \alpha_1/\beta_1$.

- (ii) If $0 < \alpha_1/\beta_1 < \alpha_2/\beta_2$, then $I(t) \in (\alpha_1/\beta_1, \alpha_2/\beta_2)$ for all t > 0 whenever $I_0 \in (\alpha_1/\beta_1, \alpha_2/\beta_2)$.
- (iii) If $\alpha_1/\beta_1 \leq 0 < \alpha_2/\beta_2$, then $I(t) \in (0, \alpha_2/\beta_2)$ for all t > 0 whenever $I_0 \in (0, \alpha_2/\beta_2)$.

Proof. Case (i) is obvious. To prove Case (ii), we may assume, without loss of generality, that r(0) = 1. Recalling (2.1) and the properties of the deterministic SIS model (1.5) which we stated in Section 1, we see that I(t) will monotonically decrease during the time interval $[\tau_0, \tau_1]$ but never reach α_1/β_1 , whence $I(t) \in (\alpha_1/\beta_1, \alpha_2/\beta_2)$. At time τ_1 , the Markov chain switches to state 2 and will not jump to state 1 until time τ_2 . During this time interval $[\tau_1, \tau_2]$, I(t) will monotonically increase but never reach α_2/β_2 , whence $I(t) \in (\alpha_1/\beta_1, \alpha_2/\beta_2)$ again. Repeating this argument, we see that I(t) will remain within $(\alpha_1/\beta_1, \alpha_2/\beta_2)$ forever. Similarly, we can show Case (iii). \Box

In the following study we will use the Markov property of the solutions (see e.g. [27, 29]). For this purpose, let us denote by \mathbb{P}_{I_0,r_0} the conditional probability measure generated by the pair of processes (I(t), r(t)) given the initial condition $(I(0), r(0)) = (I_0, r_0) \in (0, N) \times \mathbb{S}$.

Theorem 5.3 Assume that $T_0^S > 1$ and let $I_0 \in (0, N)$ be arbitrary. The following statements hold with probability one:

- (i) If $0 < \alpha_1/\beta_1 = \alpha_2/\beta_2$, then $\lim_{t\to\infty} I(t) = \alpha_1/\beta_1$.
- (ii) If $0 < \alpha_1/\beta_1 < \alpha_2/\beta_2$, then

$$\frac{\alpha_1}{\beta_1} \leq \liminf_{t \to \infty} I(t) \leq \limsup_{t \to \infty} I(t) \leq \frac{\alpha_2}{\beta_2}$$

(iii) If $\alpha_1/\beta_1 \leq 0 < \alpha_2/\beta_2$, then

$$0 \le \liminf_{t \to \infty} I(t) \le \limsup_{t \to \infty} I(t) \le \frac{\alpha_2}{\beta_2}.$$

Proof. Case (i). If $I_0 = \alpha_1/\beta_1$, then $I(t) = \alpha_1/\beta_1$ for all $t \ge 0$, whence the assertion holds. If $I_0 < \alpha_1/\beta_1$, it is easy to see that I(t) increases monotonically on $t \ge 0$, hence $\lim_{t\to\infty} I(t)$ exists. By Theorem 5.1, we therefore have

$$\lim_{t \to \infty} I(t) = \frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} \quad a.s.$$

But, given $\alpha_1/\beta_1 = \alpha_2/\beta_2$, we compute

$$\frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} = \frac{\pi_1 \alpha_1 + \pi_2 \alpha_1 \beta_2 / \beta_1}{\pi_1 \beta_1 + \pi_2 \beta_2} = \frac{\alpha_1}{\beta_1}.$$

We therefore have $\lim_{t\to\infty} I(t) = \alpha_1/\beta_1$ a.s. Similarly, we can show this for $I_0 > \alpha_1/\beta_1$.

Case (ii). If $I_0 \in (\alpha_1/\beta_1, \alpha_2/\beta_2)$, then the assertion follows from Lemma 5.2 directly. Let us now assume that $I_0 \ge \alpha_2/\beta_2$. Given $0 < \alpha_1/\beta_1 < \alpha_2/\beta_2$, it is easy to show that

$$\frac{\alpha_1}{\beta_1} < \frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} < \frac{\alpha_2}{\beta_2}.$$

Consider a number

$$\kappa \in \left(\frac{\pi_1\alpha_1 + \pi_2\alpha_2}{\pi_1\beta_1 + \pi_2\beta_2}, \frac{\alpha_2}{\beta_2}\right),$$

and define the stopping time

$$\rho_{\kappa} = \inf\{t \ge 0 : I(t) \le \kappa\},\$$

where throughout this paper we set $\inf \emptyset = \infty$ (in which \emptyset denotes the empty set as usual). By Theorem 5.1 we have

$$\mathbb{P}(\rho_k < \infty) = 1$$

while by the continuity of I(t) we have $I(\rho_{\kappa}) = \kappa$. Set

$$\bar{\Omega} = \left\{ \alpha_1 / \beta_1 \le \liminf_{t \to \infty} I(t) \le \limsup_{t \to \infty} I(t) \le \alpha_2 / \beta_2 \right\}$$

and denote its indicator function by $\mathbb{I}_{\bar{\Omega}}.$ By the strong Markov property, we compute

$$\mathbb{P}(\bar{\Omega}) = \mathbb{E}(\mathbb{I}_{\bar{\Omega}}) = \mathbb{E}(\mathbb{E}(\mathbb{I}_{\bar{\Omega}} | \mathcal{F}_{\rho_{\kappa}})) = \mathbb{E}(\mathbb{E}(\mathbb{I}_{\bar{\Omega}} | I(\rho_{\kappa}), r(\rho_{\kappa}))) = \mathbb{E}(\mathbb{P}_{I(\rho_{\kappa}), r(\rho_{\kappa})}(\bar{\Omega})) = \mathbb{E}(\mathbb{P}_{\kappa, r(\rho_{\kappa})}(\bar{\Omega}))$$

But, by Lemma 5.2, $\mathbb{P}_{\kappa,r(\rho_{\kappa})}(\bar{\Omega}) = 1$ and hence we have $\mathbb{P}(\bar{\Omega}) = 1$ as required. Similarly, we can show that $\mathbb{P}(\bar{\Omega}) = 1$ for $I_0 \leq \alpha_1/\beta_1$.

Case (iii). It is obvious that $0 \leq \liminf_{t\to 0} I(t)$, while the assertion that $\liminf_{t\to 0} I(t) \leq \alpha_2/\beta_2$ can be proved in the same way as Case (ii) was proved. The proof is therefore complete. \Box

Under the condition $T_0^S > 1$, the theorem above shows precisely that I(t) will tend to α_1/β_1 with probability one if $\alpha_1/\beta_1 = \alpha_2/\beta_2$. However, it is quite rare to have $\alpha_1/\beta_1 = \alpha_2/\beta_2$ in practice. It is therefore more useful to study the case when, say, $\alpha_1/\beta_1 < \alpha_2/\beta_2$ in a bit more detail. In the proof above, we have in fact shown a slightly stronger result than Theorem 5.3 states, namely we have shown that

$$\mathbb{P}(I(t) \in (0 \lor (\alpha_1/\beta_1), \ \alpha_2/\beta_2) \text{ for all } t \ge \rho_{\kappa}) = 1,$$
(5.7)

where we use the notation $a \lor b = \max(a, b)$. It would be interesting to find out how I(t) will vary within the interval $(0 \lor (\alpha_1/\beta_1), \alpha_2/\beta_2)$ in the long term. The following theorem shows that I(t) can take any value up to the boundaries of the interval infinitely many times (though never reach them) with positive probability.

Theorem 5.4 Assume that $T_0^S > 1$ and $0 < \frac{\alpha_1}{\beta_1} < \frac{\alpha_2}{\beta_2}$, and let $I_0 \in (0, N)$ be arbitrary. Then for any $\varepsilon > 0$, sufficiently small for

$$\frac{\alpha_1}{\beta_1} + \varepsilon < \frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} < \frac{\alpha_2}{\beta_2} - \varepsilon,$$

the solution of the stochastic SIS epidemic model (2.3) has the properties that

$$\mathbb{P}\Big(\liminf_{t\to\infty} I(t) < \frac{\alpha_1}{\beta_1} + \varepsilon\Big) \ge e^{-\nu_{12}T_1(\varepsilon)},\tag{5.8}$$

and

$$\mathbb{P}\Big(\limsup_{t \to \infty} I(t) > \frac{\alpha_2}{\beta_2} - \varepsilon\Big) \ge e^{-\nu_{21}T_2(\varepsilon)},\tag{5.9}$$

where $T_1(\varepsilon) > 0$ and $T_2(\varepsilon) > 0$ are defined by

$$T_1(\varepsilon) = \frac{1}{\alpha_1} \left(\log\left(\frac{\beta_1}{\alpha_1} - \frac{\beta_2}{\alpha_2}\right) + \log\left(\frac{\alpha_1}{\beta_1} + \varepsilon\right) - \log\left(\frac{\varepsilon\beta_1}{\alpha_1}\right) \right)$$
(5.10)

and

$$T_2(\varepsilon) = \frac{1}{\alpha_2} \left(\log\left(\frac{\beta_1}{\alpha_1} - \frac{\beta_2}{\alpha_2}\right) + \log\left(\frac{\alpha_2}{\beta_2} - \varepsilon\right) - \log\left(\frac{\varepsilon\beta_2}{\alpha_2}\right) \right).$$
(5.11)

Proof. Let T > 0 be arbitrary. Define the stopping time

$$\sigma_1 = \inf\{t \ge T : I(t) \in (\alpha_1/\beta_1 + \varepsilon, \alpha_2/\beta_2 - \varepsilon)\}$$

By Theorem 5.1, we have $\mathbb{P}(\sigma_1 < \infty) = 1$, while we see from the proof of Theorem 5.3 that

$$\mathbb{P}(I(t) \in (\alpha_1/\beta_1, \alpha_2/\beta_2) \text{ for all } t \ge \sigma_1) = 1.$$
(5.12)

To prove assertion (5.8), we define another stopping time

$$\sigma_2 = \inf\{t \ge \sigma_1 : r(t) = 1\}.$$

Clearly, $\mathbb{P}(\sigma_2 < \infty) = 1$ and by the right-continuity of the Markov chain, $r(\sigma_2) = 1$. By the memoryless property of an exponential distribution, the probability that the Markov chain will not jump to state 2 before $\sigma_2 + T_1(\varepsilon)$ is

$$\mathbb{P}(\Omega_1) = e^{-\nu_{12}T_1(\varepsilon)},\tag{5.13}$$

where $\Omega_1 = \{r(\sigma_2 + t) = 1 \text{ for all } t \in [0, T_1(\varepsilon)]\}$. Now, consider any $\omega \in \Omega_1$ and consider I(t) on $t \in [\sigma_2, \sigma_2 + T_1(\varepsilon)]$. Note that it obeys the differential equation

$$\frac{dI(t)}{dt} = I(t)(\alpha_1 - \beta_1 I(t)),$$

with initial value $I(\sigma_2) \in (\alpha_1/\beta_1, \alpha_2/\beta_2)$. By the explicit solution of this equation (see Section 1), we have

$$I(\sigma_2 + T_1(\varepsilon)) = \left[e^{-\alpha_1 T_1(\varepsilon)} \left(\frac{1}{I(\sigma_2)} - \frac{\beta_1}{\alpha_1}\right) + \frac{\beta_1}{\alpha_1}\right]^{-1}$$

On the other hand, by (5.10), we have

$$\left[e^{-\alpha_1 T_1(\varepsilon)} \left(\frac{\beta_2}{\alpha_2} - \frac{\beta_1}{\alpha_1}\right) + \frac{\beta_1}{\alpha_1}\right]^{-1} = \frac{\alpha_1}{\beta_1} + \varepsilon_2$$

Since $I(\sigma_2) < \alpha_2/\beta_2$, we must therefore have

$$I(\sigma_2 + T_1(\varepsilon)) < \frac{\alpha_1}{\beta_1} + \varepsilon.$$

Consequently

$$\mathbb{P}\Big(\inf_{T \le t < \infty} I(t) < \frac{\alpha_1}{\beta_1} + \varepsilon\Big) \ge \mathbb{P}(\Omega_1) = e^{-\nu_{12}T_1(\varepsilon)}.$$
(5.14)

Noting that

$$\left(\liminf_{t\to\infty} I(t) < \frac{\alpha_1}{\beta_1} + \varepsilon\right) = \bigcap_{0< T<\infty} \left(\inf_{T\le t<\infty} I(t) < \frac{\alpha_1}{\beta_1} + \varepsilon\right),$$

we can let $T \to \infty$ in (5.14) to obtain assertion (5.8). Similarly, we can prove the other assertion (5.9). \Box

Theorem 5.5 Assume that $T_0^S > 1$ (namely $\pi_1 \alpha_1 + \pi_2 \alpha_2 > 0$) and $\frac{\alpha_1}{\beta_1} \le 0 < \frac{\alpha_2}{\beta_2}$. Let $I_0 \in (0, N)$ be arbitrary. Then for any $\varepsilon > 0$, sufficiently small for

$$\varepsilon < \frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} < \frac{\alpha_2}{\beta_2} - \varepsilon,$$

the solution of the stochastic SIS model (2.3) has the properties that

$$\mathbb{P}\Big(\liminf_{t \to \infty} I(t) < \varepsilon\Big) \ge e^{-\nu_{12}T_3(\varepsilon)},\tag{5.15}$$

and

$$\mathbb{P}\Big(\limsup_{t \to \infty} I(t) > \frac{\alpha_2}{\beta_2} - \varepsilon\Big) \ge e^{-\nu_{21}T_4(\varepsilon)},\tag{5.16}$$

where $T_3(\varepsilon) > 0$ and $T_4(\varepsilon) > 0$ are defined by

$$T_3(\varepsilon) = \frac{1}{\alpha_1} \left(\log\left(\frac{\beta_2}{\alpha_2} - \frac{\beta_1}{\alpha_1}\right) + \log\left(\varepsilon\frac{\alpha_1}{\beta_1}\right) - \log\left(\frac{\alpha_1}{\beta_1} - \varepsilon\right) \right)$$
(5.17)

and

$$T_4(\varepsilon) = \frac{1}{\alpha_2} \left(\log\left(\frac{2}{\varepsilon} - \frac{\beta_2}{\alpha_2}\right) + \log\left(\frac{\alpha_2}{\beta_2} - \varepsilon\right) - \log\left(\varepsilon\frac{\beta_2}{\alpha_2}\right) \right).$$
(5.18)

Proof. Let T > 0 be arbitrary. Define the stopping time

$$\sigma_3 = \inf\{t \ge T : I(t) \in (\varepsilon, \alpha_2/\beta_2 - \varepsilon)\}$$

By Theorem 5.1, we have $\mathbb{P}(\sigma_3 < \infty) = 1$, while we see from the proof of Theorem 5.3 that

$$\mathbb{P}(I(t) \in (0, \alpha_2/\beta_2) \text{ for all } t \ge \sigma_3) = 1.$$
(5.19)

To prove assertion (5.15), we define another stopping time

$$\sigma_4 = \inf\{t \ge \sigma_3 : r(t) = 1\}.$$

Clearly, $\mathbb{P}(\sigma_4 < \infty) = 1$ and by the right-continuity of the Markov chain, $r(\sigma_4) = 1$. By the memoryless property of an exponential distribution, the probability that the Markov chain will not jump to state 2 before $\sigma_4 + T_3(\varepsilon)$ is

$$\mathbb{P}(\Omega_2) = e^{-\nu_{12}T_3(\varepsilon)},\tag{5.20}$$

where $\Omega_2 = \{r(\sigma_4 + t) = 1 \text{ for all } t \in [0, T_3(\varepsilon)]\}$. Now, consider any $\omega \in \Omega_2$ and consider I(t) on $t \in [\sigma_4, \sigma_4 + T_3(\varepsilon)]$. Note that it obeys the differential equation

$$\frac{dI(t)}{dt} = I(t)(\alpha_1 - \beta_1 I(t)),$$

with initial value $I(\sigma_4) \in (0, \alpha_2/\beta_2)$. By the explicit solution of this equation (see Section 1), we have

$$I(\sigma_4 + T_3(\varepsilon)) = \left[e^{-\alpha_1 T_3(\varepsilon)} \left(\frac{1}{I(\sigma_4)} - \frac{\beta_1}{\alpha_1}\right) + \frac{\beta_1}{\alpha_1}\right]^{-1}$$

On the other hand, by (5.17), we have

$$\left[e^{-\alpha_1 T_3(\varepsilon)} \left(\frac{\beta_2}{\alpha_2} - \frac{\beta_1}{\alpha_1}\right) + \frac{\beta_1}{\alpha_1}\right]^{-1} = \varepsilon.$$

Since $I(\sigma_4) < \alpha_2/\beta_2$, we must therefore have

$$I(\sigma_4 + T_1(\varepsilon)) < \varepsilon.$$

Consequently

$$\mathbb{P}\Big(\inf_{T \le t < \infty} I(t) < \varepsilon\Big) \ge \mathbb{P}(\Omega_2) = e^{-\nu_{12}T_3(\varepsilon)}.$$
(5.21)

Noting that

$$\Big(\liminf_{t\to\infty} I(t)<\varepsilon\Big)=\bigcap_{0< T<\infty}\Big(\inf_{T\leq t<\infty} I(t)<\varepsilon\Big),$$

we can let $T \to \infty$ in (5.21) to obtain assertion (5.15).

To prove the other assertion (5.16) we define the stopping time

$$\sigma_5 = \inf\{t \ge T : r(t) = 2\},$$

where T > 0 is arbitrary. Clearly $\mathbb{P}(\sigma_5 < \infty) = 1$. We define another stopping time

$$\sigma_6 = \inf\left\{t \ge \sigma_5 : r(t) = 2, I(t) \ge \frac{1}{2} \left(\frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2}\right)\right\}$$

Suppose that $I(t) < \frac{1}{2} \left(\frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} \right)$ when $t = \sigma_5$, I(t) will eventually increase across this level by Theorem 5.1. Note that I(t) increases monotonically when r(t) = 2 whilst it decreases monotonically when r(t) = 1. If r(t) switches back to state 1 before I(t) increases over this level and starts decreasing, since the $\limsup_{t\to\infty} I(t) \ge \frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2}$, I(t) will increase across this level later on i.e. r(t) = 2 when $I(t) = \frac{1}{2} \left(\frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} \right)$. Therefore we have $\mathbb{P}(\sigma_6 < \infty) = 1$. And by the right-continuity of the Markov chain, $r(\sigma_6) = 2$. By the memoryless property of an exponential distribution, the probability that the Markov chain will not jump to state 1 before $\sigma_6 + T_4(\varepsilon)$ is

$$\mathbb{P}(\Omega_3) = e^{-\nu_{21}T_4(\varepsilon)},\tag{5.22}$$

where $\Omega_3 = \{r(\sigma_6 + t) = 2 \text{ for all } t \in [0, T_4(\varepsilon)]\}$. Now, consider any $\omega \in \Omega_3$ and consider I(t) on $t \in [\sigma_6, \sigma_6 + T_4(\varepsilon)]$. Note that it obeys the differential equation

$$\frac{dI(t)}{dt} = I(t)(\alpha_2 - \beta_2 I(t)),$$

with initial value $I(\sigma_6) \geq \frac{1}{2} \left(\frac{\pi_1 \alpha_1 + \pi_2 \alpha_2}{\pi_1 \beta_1 + \pi_2 \beta_2} \right) > \frac{\varepsilon}{2}$. By the explicit solution of this equation (see Section 1), we have

$$I(\sigma_6 + T_4(\varepsilon)) = \left[e^{-\alpha_2 T_4(\varepsilon)} \left(\frac{1}{I(\sigma_6)} - \frac{\beta_2}{\alpha_2}\right) + \frac{\beta_2}{\alpha_2}\right]^{-1}.$$

On the other hand, by (5.18), we have

$$\left[e^{-\alpha_2 T_4(\varepsilon)} \left(\frac{2}{\varepsilon} - \frac{\beta_2}{\alpha_2}\right) + \frac{\beta_2}{\alpha_2}\right]^{-1} = \frac{\alpha_2}{\beta_2} - \varepsilon.$$

Since $I(\sigma_6) > \frac{\varepsilon}{2}$, we must therefore have

$$I(\sigma_6 + T_4(\varepsilon)) > \frac{\alpha_2}{\beta_2} - \varepsilon.$$

Consequently

$$\mathbb{P}\Big(\sup_{T \le t < \infty} I(t) > \frac{\alpha_2}{\beta_2} - \varepsilon\Big) \ge \mathbb{P}(\Omega_3) = e^{-\nu_{21}T_4(\varepsilon)}.$$
(5.23)

Noting that

$$\Big(\limsup_{t\to\infty} I(t) > \frac{\alpha_2}{\beta_2} - \varepsilon\Big) = \bigcap_{0< T<\infty} \Big(\sup_{T\le t<\infty} I(t) > \frac{\alpha_2}{\beta_2} - \varepsilon\Big),$$

we can let $T \to \infty$ in (5.23) to obtain assertion (5.16). \Box

Define

$$R_{01}^D = \frac{\beta_1 N}{\mu_1 + \gamma_1}$$
 and $R_{02}^D = \frac{\beta_2 N}{\mu_2 + \gamma_2}$.

Note that if $\alpha_j > 0$ then $R_{0j}^D > 1$ for j = 1, 2 and

$$\frac{\alpha_j}{\beta_j} = N\left(1 - \frac{1}{R_{0j}^D}\right)$$

is the endemic level of disease after a long time in the SIS model (1.4) with $\beta = \beta_j$, $\mu = \mu_j$ and $\gamma = \gamma_j$. If $\alpha_1 \leq 0$ then $R_{01}^D \leq 1$ and disease eventually dies out in the corresponding SIS model. So in general in the first model the disease prevalence eventually approaches $0 \vee (\alpha_1/\beta_1)$ and in the second model the disease prevalence eventually approaches α_2/β_2 . These are the two levels between which the disease oscillates in the Markov chain switching model.

6 Simulations

In this section we shall assume that all parameters are given in appropriate units.

6.1 Extinction case

Example 6.1.1 Assume that the system parameters are given by

$$\mu_1 = 0.45, \mu_2 = 0.05, \gamma_1 = 0.35, \gamma_2 = 0.15, \beta_1 = 0.001, \beta_2 = 0.004, N = 100,$$

 $\nu_{12} = 0.6, \text{ and } \nu_{21} = 0.9.$

So $\alpha_1 = -0.7$, $\alpha_2 = 0.2$, $\pi_1 = 0.6$, and $\pi_2 = 0.4$ (see Section 2 for definitions).

Noting that

$$\alpha_1 \pi_1 + \alpha_2 \pi_2 = -0.34,$$

we can therefore conclude, by Theorem 4.2, that for any given initial value $I(0) = I_0 \in (0, N)$, the solution of (2.3) obeys

$$\limsup_{t \to \infty} \frac{1}{t} \log(I(t)) \le -0.34 < 0 \quad a.s.$$

That is, I(t) will tend to zero exponentially with probability one.

The computer simulation in Figure 1(a) supports this result clearly, illustrating extinction of the disease. Furthermore, $\alpha_1 < 0$ while $\alpha_2 > 0$ in this case, which means that one subsystem dies out while the other subsystem is persistent. Figure 1(a) shows some decreasing then increasing behaviour early on, but the general trend tends to zero, illustrating extinction for the system as a whole. The Euler-Maruyama (EM) method [28, 29] is also applied to approximate the solution I(t). The two lines are very close to each other, showing that the EM method gives a very good approximation to the true solution in this case.

Example 6.1.2 Assume that the system parameters are given by

$$\mu_1 = 0.45, \mu_2 = 0.05, \gamma_1 = 0.35, \gamma_2 = 0.15, \beta_1 = 0.006, \beta_2 = 0.0015, N = 100$$

$$\nu_{12} = 0.6$$
, and $\nu_{21} = 0.9$.

So $\alpha_1 = -0.2$, $\alpha_2 = -0.05$, $\pi_1 = 0.6$, and $\pi_2 = 0.4$ (see Section 2 for definitions).

Noting that

$$\alpha_1 \pi_1 + \alpha_2 \pi_2 = -0.14,$$

we can therefore conclude, by Theorem 4.2, that for any given initial value $I(0) = I_0 \in (0, N)$, the solution of (2.3) obeys

$$\limsup_{t \to \infty} \frac{1}{t} \log(I(t)) \le -0.14 < 0 \quad a.s.$$

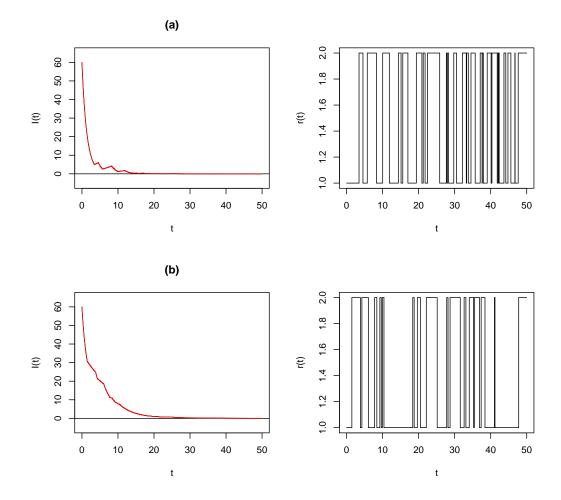


Figure 1: Computer simulation of I(t) and its corresponding Markov chain r(t), using the parameter values in Example 6.1.1 for (a) and in Example 6.1.2 for (b), I(0) = 60 for both cases, and the exponential distribution for the switching times of r(t), with r(0) = 1. The black line is for I(t) using formula (2.5) and the red line is for the EM method. (The two lines are very close to each other, so we hardly see the black line in the plot.)

That is, I(t) will tend to zero exponentially with probability one. The computer simulation in Figure 1(b) supports this result clearly, illustrating extinction of the disease. Both α_1 and α_2 are less than zero in this case, which means that both subsystems die out. Figure 1(b) shows a trend of decreasing all the time but at different speeds, which reveals that property. As before, the EM method gives a good approximation in this case as well.

6.2 Persistence case

Example 6.2.1 Assume that the system parameters are given by

$$\mu_1 = 0.45, \mu_2 = 0.05, \gamma_1 = 0.35, \gamma_2 = 0.15, \beta_1 = 0.01, \beta_2 = 0.012, N = 100, \beta_2 = 0.012, \beta$$

 $\nu_{12} = 0.6$, and $\nu_{21} = 0.9$.

So $\alpha_1 = 0.2$, $\alpha_2 = 1$, $\pi_1 = 0.6$, and $\pi_2 = 0.4$.

Noting that

$$\alpha_1 \pi_1 + \alpha_2 \pi_2 = 0.52,$$

we can therefore conclude, by Theorem 5.3, that for any given initial value $I(0) = I_0 \in (0, N)$, the solution of (2.3) obeys

$$\frac{\alpha_1}{\beta_1} = 20 \le \liminf_{t \to \infty} I(t) \le \limsup_{t \to \infty} I(t) \le 83.33 = \frac{\alpha_2}{\beta_2}.$$

That is, I(t) will eventually enter the region (20, 83.33) if I(0) is not in this region, and will be attracted in this region once it has entered. Also, by Theorem 5.4, I(t) can take any value up to the boundaries of (20, 83.33) but never reach them.

The computer simulations in Figure 2(a), (b) and (c), using different initial values I(0), support these results clearly. As before, the EM method gives a good approximation of the true solution.

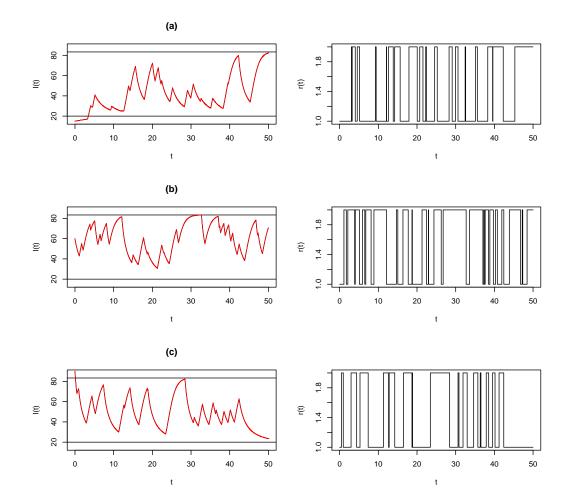


Figure 2: Computer simulation of I(t) and its corresponding Markov chain r(t), using the parameter values in Example 6.2.1, with I(0) = 15 for (a), I(0) = 60 for (b) and I(0) = 90 for (c), and the exponential distribution for the switching times of r(t), with r(0) = 1. The black line is for I(t) using formula (2.5) and the red line for the EM method. (The two lines are very close to each other, so we hardly see the black line in the plot.) The horizontal lines in the plot of I(t) indicate levels $\frac{\alpha_1}{\beta_1}$ and $\frac{\alpha_2}{\beta_2}$.

Example 6.2.2 Assume that the system parameters are given by

$$\mu_1 = 0.45, \ \mu_2 = 0.05, \gamma_1 = 0.35, \gamma_2 = 0.15, \beta_1 = 0.004, \beta_2 = 0.012, N = 100.004$$

$$\nu_{12} = 0.6$$
, and $\nu_{21} = 0.9$.

So $\alpha_1 = -0.4$, $\alpha_2 = 1$, $\pi_1 = 0.6$, and $\pi_2 = 0.4$.

Noting that

$$\alpha_1 \pi_1 + \alpha_2 \pi_2 = 0.16,$$

we can therefore conclude, by Theorem 5.3, that for any given initial value $I(0) = I_0 \in (0, N)$, the solution of (2.3) obeys

$$0 \le \liminf_{t \to \infty} I(t) \le \limsup_{t \to \infty} I(t) \le 83.33 = \frac{\alpha_2}{\beta_2}.$$

That is, I(t) will eventually enter the region (0, 83.33) if I(0) is not in this region, and will be attracted in this region once it has entered. Also, by Theorem 5.5, I(t) can take any value up to the boundaries of (0, 83.33) but never reach them.

The computer simulations in Figure 3 support this result clearly.

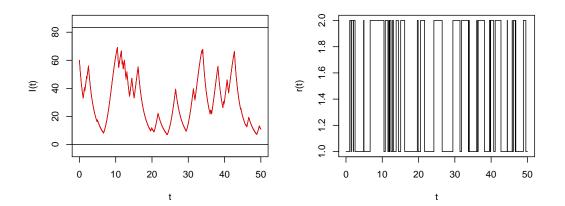


Figure 3: Computer simulation of I(t) using the parameter values in Example 6.2.2 and its corresponding Markov chain r(t), using formula (2.5) (black line) and the EM method (red line) for I(t), with I(0) = 60, and the exponential distribution for the switching times of r(t), with r(0) = 1. (The two lines are very close to each other, so we hardly see the black line in the plot.) The horizontal lines in the plot of I(t) indicate the levels 0 and $\frac{\alpha_2}{\beta_2}$.

6.3 $T_0^S = 1$ Case

Example 6.3.1 Assume that the system parameters are given by

$$\mu_1 = 0.45, \mu_2 = 0.05, \gamma_1 = 0.35, \gamma_2 = 0.15, \beta_1 = 0.006, \beta_2 = 0.005, N = 100,$$

 $\nu_{12} = 0.6, \text{ and } \nu_{21} = 0.9.$

So $\alpha_1 = -0.2$, $\alpha_2 = 0.3$, $\pi_1 = 0.6$, and $\pi_2 = 0.4$.

Note that

$$\alpha_1 \pi_1 + \alpha_2 \pi_2 = 0$$

in this case, which is equivalent to $T_0^S = 1$. As mentioned in Section 4, we have not been able to prove the behaviour of I(t) in this case. However, the simulation results in Figure 4 confirm our suspicion that the disease will always become extinct.

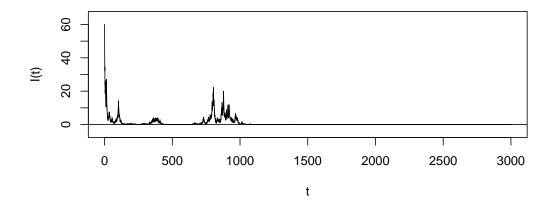


Figure 4: Computer simulation of I(t) using the parameter values in Example 6.3.1, using formula (2.5) for I(t), with I(0) = 60, and the exponential distribution for the switching times of r(t), with r(0) = 1.

7 Generalisation

We have discussed the simplest case where the Markov chain has only two states, in the previous sections. Now we are going to generalise the results to the case where the Markov chain r(t) has finite state space $\mathbb{S} = \{1, 2, ..., M\}$. The generator for r(t) is defined as

$$\Gamma = (\nu_{ij})_{M \times M},$$

where $\nu_{ii} = -\sum_{1 \le j \le M, j \ne i} \nu_{ij}$, and $\nu_{ij} > 0$ $(i \ne j)$ is the transition rate from state i to j, that is

$$\mathbb{P}\{r(t+\delta) = j | r(t) = i\} = \nu_{ij}\delta + o(\delta),$$

where $\delta > 0$. As before, there is a sequence $\{\tau_k\}_{k\geq 0}$ of finite-valued \mathcal{F}_t -stopping times such that $0 = \tau_0 < \tau_1 < \cdots < \tau_k \to \infty$ almost surely and

$$r(t) = \sum_{k=0}^{\infty} r(\tau_k) I_{[\tau_k, \tau_{k+1})}(t).$$

Moreover, given that $r(\tau_k) = i$, the random variable $\tau_{k+1} - \tau_k$ follows the exponential distribution with parameter $-\nu_{ii}$, namely

$$\mathbb{P}(\tau_{k+1} = j | \tau_k = i) = \frac{\nu_{ij}}{-\nu_{ii}}, \quad j \neq i, \quad \mathbb{P}(\tau_{k+1} - \tau_k \ge T | r(\tau_k) = i) = e^{\nu_{ii}T}, \quad \forall T \ge 0.$$

Furthermore, the unique stationary distribution of this Markov chain $\Pi = (\pi_1, \pi_2, ..., \pi_M)$ satisfies

$$\begin{cases} \Pi\Gamma = 0\\ \sum_{i=1}^{M} \pi_i = 1 \end{cases}$$

Following a similar procedure we still can show that for any given initial value $I(0) = I_0 \in (0, N)$, there is a unique solution I(t) on $t \in \mathbb{R}_+$ to equation (2.3) such that

$$\mathbb{P}(I(t) \in (0, N) \text{ for all } t \ge 0) = 1,$$

and the solution still has the form (2.5).

In the general finite state space Markov chain case it is possible to derive an explicit expression for the basic reproduction number R_0^S in the stochastic Markov switching model analogous to (3.2) expressed as the largest eigenvalue of a positive matrix. We define T_0^S for the general case as

$$T_0^S = \frac{\sum_{k=1}^M \pi_k \beta_k N}{\sum_{k=1}^M \pi_k (\mu_k + \gamma_k)}.$$

Similarly to Proposition 4.1, we have the following alternative conditions on the value of T_0^S :

Proposition 7.1 We have the following alternative condition on the value of T_0^S :

- $T_0^S < 1$ if and only if $\sum_{k=1}^M \pi_k \alpha_k < 0$;
- $T_0^S = 1$ if and only if $\sum_{k=1}^M \pi_k \alpha_k = 0$;
- $T_0^S > 1$ if and only if $\sum_{k=1}^M \pi_k \alpha_k > 0$.

If $T_0^S < 1$, similarly to Theorem 4.2, we can show:

Theorem 7.2 For any given initial value $I_0 \in (0, N)$, the solution of the stochastic SIS model (2.3) obeys

$$\limsup_{t \to \infty} \frac{1}{t} \log(I(t)) \le \sum_{k=1}^{M} \pi_k \alpha_k \quad a.s.$$

By the more general condition stated above, we hence conclude that I(t) tends to zero exponentially almost surely. This means that the disease dies out with probability one.

For the case that $T_0^S > 1$, Theorem 5.1 can be generalised as follows:

Theorem 7.3 If $T_0^S > 1$, for any given initial value $I_0 \in (0, N)$, the solution of the stochastic SIS model (2.3) has the properties that

$$\liminf_{t \to \infty} I(t) \le \frac{\sum_{k=1}^{M} \pi_k \alpha_k}{\sum_{k=1}^{M} \pi_k \beta_k} \quad a.s.$$

and

$$\limsup_{t \to \infty} I(t) \ge \frac{\sum_{k=1}^{M} \pi_k \alpha_k}{\sum_{k=1}^{M} \pi_k \beta_k} \quad a.s.,$$

which means the disease will reach the neighbourhood of the level $\frac{\sum_{k=1}^{M} \pi_k \alpha_k}{\sum_{k=1}^{M} \pi_k \beta_k}$ infinitely many times with probability one. This shows that the disease will be persistent in this case.

Lemma 5.2 can be generalised as follows:

Lemma 7.4 Without loss of generality we assume that $\alpha_1/\beta_1 \leq \alpha_2/\beta_2 \leq ... \leq \alpha_M/\beta_M$ and the following statements hold with probability one:

- (i) If $0 < \alpha_1/\beta_1 = \alpha_2/\beta_2 = ... = \alpha_M/\beta_M$, then $I(t) = \alpha_1/\beta_1$ for all t > 0 when $I_0 = \alpha_1/\beta_1$.
- (ii) If $0 < \alpha_1/\beta_1 \le \alpha_2/\beta_2 \le \dots \le \alpha_M/\beta_M$, then $I(t) \in (\alpha_1/\beta_1, \alpha_M/\beta_M)$ for all t > 0 whenever $I_0 \in (\alpha_1/\beta_1, \alpha_M/\beta_M)$.
- (iii) If $\alpha_j/\beta_j \leq 0$ (for some $j \in (1, M 1)$) and $\alpha_1/\beta_1 \leq \alpha_2/\beta_2 \leq ... \leq \alpha_M/\beta_M$ then $I(t) \in (0, \alpha_M/\beta_M)$ for all t > 0 whenever $I_0 \in (0, \alpha_M/\beta_M)$.

Theorem 5.3 can be generalised as follows:

Theorem 7.5 Assume that $T_0^S > 1$ and let $I_0 \in (0, N)$ be arbitrary. The following statements hold with probability one:

- (i) If $0 < \alpha_1/\beta_1 = \alpha_2/\beta_2 = \dots = \alpha_M/\beta_M$, then $\lim_{t\to\infty} I(t) = \alpha_1/\beta_1$.
- (ii) If $0 < \alpha_1/\beta_1 \le \alpha_2/\beta_2 \le \dots \le \alpha_M/\beta_M$, then

$$\frac{\alpha_1}{\beta_1} \le \liminf_{t \to \infty} I(t) \le \limsup_{t \to \infty} I(t) \le \frac{\alpha_M}{\beta_M}$$

(iii) If $\alpha_j/\beta_j \leq 0$ (for some $j \in (1, M - 1)$) and $\alpha_1/\beta_1 \leq \alpha_2/\beta_2 \leq ... \leq \alpha_M/\beta_M$, then

$$0 \le \liminf_{t \to \infty} I(t) \le \limsup_{t \to \infty} I(t) \le \frac{\alpha_M}{\beta_M}$$

These stronger results indicate that I(t) will enter the region $(0 \vee (\alpha_1/\beta_1), \alpha_M/\beta_M)$ in finite time and with probability one will stay in this region once it is entered.

Theorem 5.4 can be generalised as follows:

Theorem 7.6 Assume that $T_0^S > 1$ and $0 < \alpha_1/\beta_1 \le \alpha_2/\beta_2 \le ... \le \alpha_M/\beta_M$, and let $I_0 \in (0, N)$ be arbitrary. Then for any $\varepsilon > 0$, sufficiently small for

$$\frac{\alpha_1}{\beta_1} + \varepsilon < \frac{\sum_{k=1}^M \pi_k \alpha_k}{\sum_{k=1}^M \pi_k \beta_k} < \frac{\alpha_M}{\beta_M} - \varepsilon,$$

the solution of the stochastic SIS model (2.3) has the properties that

$$\mathbb{P}\Big(\liminf_{t\to\infty} I(t) < \frac{\alpha_1}{\beta_1} + \varepsilon\Big) \ge e^{\nu_{11}T_1(\varepsilon)},$$

and

$$\mathbb{P}\Big(\limsup_{t\to\infty} I(t) > \frac{\alpha_M}{\beta_M} - \varepsilon\Big) \ge e^{\nu_{MM}T_2(\varepsilon)},$$

where $T_1(\varepsilon) > 0$ and $T_2(\varepsilon) > 0$ are defined by

$$T_1(\varepsilon) = \frac{1}{\alpha_1} \left(\log\left(\frac{\beta_1}{\alpha_1} - \frac{\beta_M}{\alpha_M}\right) + \log\left(\frac{\alpha_1}{\beta_1} + \varepsilon\right) - \log\left(\varepsilon\frac{\beta_1}{\alpha_1}\right) \right)$$
(7.1)

and

$$T_2(\varepsilon) = \frac{1}{\alpha_M} \left(\log\left(\frac{\beta_1}{\alpha_1} - \frac{\beta_M}{\alpha_M}\right) + \log\left(\frac{\alpha_M}{\beta_M} - \varepsilon\right) - \log\left(\varepsilon\frac{\beta_M}{\alpha_M}\right) \right).$$
(7.2)

Also, Theorem 5.5 can be generalised as follows:

Theorem 7.7 Assume that $T_0^S > 1$, that is $\sum_{k=1}^M \pi_k \alpha_k > 0$, and $\alpha_j / \beta_j \leq 0$ (for some $j \in (1, M - 1)$). Let $I_0 \in (0, N)$ be arbitrary. Then for any $\varepsilon > 0$, sufficiently small for

$$\varepsilon < \frac{\sum_{k=1}^{M} \pi_k \alpha_k}{\sum_{k=1}^{M} \pi_k \beta_k} < \frac{\alpha_M}{\beta_M} - \varepsilon.$$

the solution of the stochastic SIS model (2.3) has the properties that

$$\mathbb{P}\Big(\liminf_{t\to\infty}I(t)<\varepsilon\Big)\geq e^{\nu_{11}T_3(\varepsilon)},$$

and

$$\mathbb{P}\Big(\limsup_{t\to\infty} I(t) > \frac{\alpha_M}{\beta_M} - \varepsilon\Big) \ge e^{\nu_{MM}T_4(\varepsilon)}.$$

Here $T_3(\varepsilon) > 0$ and $T_4(\varepsilon) > 0$ are defined by

$$T_3(\varepsilon) = \frac{1}{\alpha_1} \left(\log\left(\frac{\beta_M}{\alpha_M} - \frac{\beta_1}{\alpha_1}\right) + \log\left(\varepsilon\frac{\alpha_1}{\beta_1}\right) - \log\left(\frac{\alpha_1}{\beta_1} - \varepsilon\right) \right)$$
(7.3)

and

$$T_4(\varepsilon) = \frac{1}{\alpha_M} \left(\log\left(\frac{2}{\varepsilon} - \frac{\beta_M}{\alpha_M}\right) + \log\left(\frac{\alpha_M}{\beta_M} - \varepsilon\right) - \log\left(\varepsilon\frac{\beta_M}{\alpha_M}\right) \right).$$
(7.4)

Theorem 7.6 and Theorem 7.7 show that I(t) will take any value arbitrarily close to the boundaries $(0 \vee (\alpha_1/\beta_1), \alpha_M/\beta_M)$ but never reach them.

The proofs are all very similar to the simple case, so they are omitted here.

To prove (7.4) analogously to the simple case we define the stopping times

$$\sigma_5 = \inf\{t \ge T : r(t) = M\}$$

where T > 0 is arbitrary and

$$\sigma_6 = \inf\left\{t \ge \sigma_5 : r(t) = M, \ I(t) \ge \frac{1}{2} \left(\frac{\sum_{k=1}^M \pi_k \alpha_k}{\sum_{k=1}^M \pi_k \beta_k}\right)\right\}.$$

By Theorem 7.3 if I(t) ever goes beneath $\frac{1}{2} \frac{\sum_{k=1}^{M} \pi_k \alpha_k}{\sum_{k=1}^{M} \pi_k \beta_k}$ it will eventually increase above this level. Hence I(t) is above this level when the Markov chain switches state infinitely often. Each time that this happens it is either initially in state M, or switches to state M with probability at least

$$q = \min_{n \in [1,2,\dots M-1]} \frac{\nu_{nM}}{-\nu_{nn}} > 0$$

Therefore each time after σ_5 that I(t) reaches the level $\frac{1}{2} \frac{\sum_{k=1}^{M} \pi_k \alpha_k}{\sum_{k=1}^{M} \pi_k \beta_k}$ we will have a value of $t \ge \sigma_5$ with r(t) = Mand I(t) above the level $\frac{1}{2} \frac{\sum_{k=1}^{M} \pi_k \alpha_k}{\sum_{k=1}^{M} \pi_k \beta_k}$ with probability at least q. So considering the first X times after σ_5 that I(t) reaches this level

$$P(\sigma_6 < \infty) \ge 1 - (1 - q)^X.$$

Letting $X \to \infty$ we deduce that $P(\sigma_6 < \infty) = 1$. The proof proceeds as in the simple case.

8 A Slightly More Realistic Example

As a slightly more realistic example to illustrate the two state case, we consider *Streptococcus pneumoniae* (*S. pneumoniae*) amongst children under 2 years in Scotland. This may display a phenomenon called capsular switching, such that when an individual is co-infected with two strains (or serotypes) of pneumococcus, the outer polysaccharide capsule that surrounds the genetic pneumococcal material may switch, thus giving serotypes with possibly different infectivities and infectious periods [7, 9]. In reality the situation is very complicated, with many pneumococcal serotypes and sequence types (sequence types are ways of coding the genetic material). This is thought to be due to genetic transfer of material between the two serotypes.

Example 8.1 We illustrate our model by applying it with suitable parameter values to two strains of pneumococcus with switching between them, although the real situation is much more complicated than the model allows. The parameter values used are taken from Lamb, Greenhalgh and Robertson [21] as follows, where N is the number of children under 2 years old in Scotland:

$$\begin{split} N &= 150000, \ \gamma_1 = \gamma_2 = 1/(7.1 \text{ wk}) = 0.1408/\text{wk} = 0.02011/\text{day} \ [38], \\ \mu &= 1/(104 \text{ wk}) = 9.615 \times 10^{-3}/\text{wk} = 1.3736 \times 10^{-3}/\text{day}, \\ \beta_1 &= 1.5041 \times 10^{-6}/\text{wk} = 2.1486 \times 10^{-7}/\text{day} \text{ corresponding to } R_{01}^D = 1.5 \ [12], \\ \beta_2 &= 2.0055 \times 10^{-6}/\text{wk} = 2.8650 \times 10^{-7}/\text{day} \text{ corresponding to } R_{02}^D = 2 \ [40]. \end{split}$$

As further support that these values for R_0 are reasonable Hoti et al. [19] give $R_0^D = 1.4$ for the spread of S. Pneumoniae in day-care cohorts in Finland.

So $\alpha_1 = 0.0107454/\text{day}$ and $\alpha_2 = 0.0214914/\text{day}$. We set

$$\nu_{12} = 0.06$$
/day and $\nu_{21} = 0.09$ /day.

So $\pi_1 = 0.6$, and $\pi_2 = 0.4$.

From these values, T_0^S is about 1.7 in this case. Noting that

$$\alpha_1 \pi_1 + \alpha_2 \pi_2 = 0.0150438 > 0,$$

we can therefore conclude, by Theorem 5.3, that for any given initial value $I(0) = I_0 \in (0, N)$, the solution of (2.3) obeys

$$\frac{\alpha_1}{\beta_1} = 50011.17 \le \liminf_{t \to \infty} I(t) \le \limsup_{t \to \infty} I(t) \le 75013.61 = \frac{\alpha_2}{\beta_2}.$$

That is, I(t) will eventually enter the region (50011.17, 75013.61) if I(0) is not in this region, and will be attracted in this region once it has entered. The computer simulations in Figure 5 support this result clearly.

We vary the values for the transition rates ν_{12} and ν_{21} . Figure 6 shows how the different values of the transition rates affect the behaviour of I(t). We notice that it takes longer to switch between the two states when the transition rates are small, so I(t) is more likely to approach the boundaries.

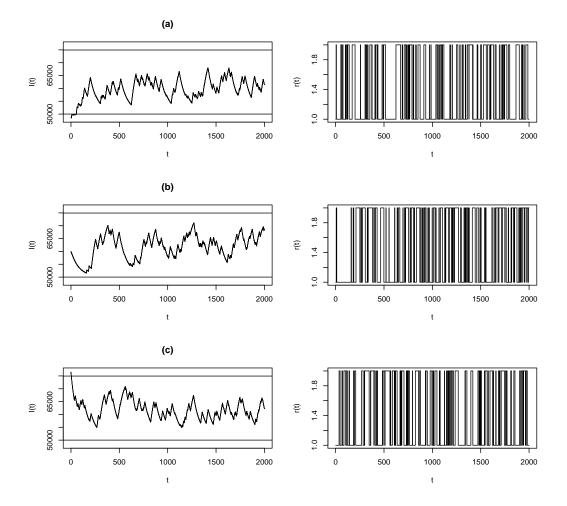


Figure 5: Computer simulation of I(t) using the parameter values in Example 8.1 and its corresponding Markov chain r(t), using formula (2.5) for I(t), with I(0) = 48500 for (a), I(0) = 60000 for (b) and I(0) = 76500 for (c), and the exponential distribution for the switching times of r(t), with r(0) = 1. The horizontal lines in the plot of I(t) indicate the levels $\frac{\alpha_1}{\beta_1}$ and $\frac{\alpha_2}{\beta_2}$.

9 Summary

In this paper, we have introduced telegraph noise to the classical SIS epidemic model and set up the stochastic SIS model. Note that the model assumes that the system switches between the two regimes and the Markov switching is independent of the state of the system. Such an assumption is similar to that made in other papers [11, 24, 34, 36]. For example external factors such as temperature or availability of food could cause the

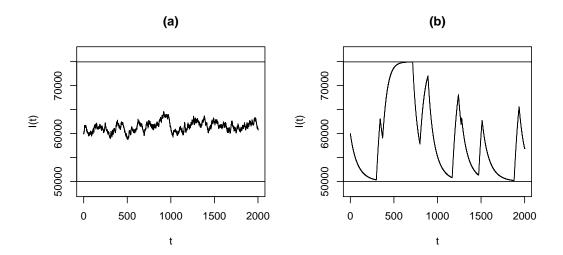


Figure 6: Computer simulation of I(t) using the parameter values in Example 8.1, with $\nu_{12} = 0.6$ /day and $\nu_{21} = 0.9$ /day for (a), and $\nu_{12} = 0.006$ /day and $\nu_{21} = 0.009$ /day for (b), using formula (2.5) for I(t) with I(0) = 60000, and the exponential distribution for the switching times of r(t), with r(0) = 1. The horizontal lines in the plot of I(t) indicate the levels $\frac{\alpha_1}{\beta_1}$ and $\frac{\alpha_2}{\beta_2}$ (which the values of I(t) never quite reach).

disease to spread faster or slower and switch between two or more regimes. In such a situation it is reasonable to assume that the switching parameter does not depend on the state of the system. We have established the explicit solution for the stochastic SIS model and also established conditions for extinction and persistence of the disease. For the stochastic Markov switching model a threshold value T_0^S was defined for almost sure persistence or extinction. We started with the special case in which the Markov chain has only two states and then generalised our theory to the general case where the Markov chain has M states. Theorem 7.2 shows that if $T_0^S < 1$, the disease will die out. Theorem 7.3 shows that if $T_0^S > 1$, then the disease will persist. We also showed Theorem 7.5 that if $T_0^S > 1$ the number of infectious individuals will enter $(0 \lor (\alpha_1/\beta_1), \alpha_M/\beta_M)$ in finite time, and with probability one will stay in the interval once entered, and moreover the number of infectious individuals can take any value up to the boundaries of $(0 \lor (\alpha_1/\beta_1), \alpha_M/\beta_M)$ but never reach them (Theorems 7.6 and 7.7).

For
$$j = 1, 2, ..., M$$
, define $R_{0j}^D = \frac{\beta_j N}{\mu_j + \gamma_j}$. Note that if $\alpha_j > 0$ then $R_{0j}^D > 1$ and
$$\frac{\alpha_j}{\beta_j} = N\left(1 - \frac{1}{R_{0j}^D}\right)$$

is the long-term endemic level of disease in the SIS model (1.4) with $\beta = \beta_j$, $\mu = \mu_j$ and $\gamma = \gamma_j$. If $\alpha_j \leq 0$ then $R_{0j}^D \leq 1$ and disease eventually dies out in the same SIS model. Hence $0 \vee (\alpha_1/\beta_1)$ is the smallest and α_M/β_M is the largest long-term endemic level of disease in each of the *M* separate SIS models between which the Markov chain switches.

We have not been able to prove extinction for the case when $T_0^S = 1$, but the computer simulation shows that the disease would die out after a long period of time, as we suspect. We have illustrated our theoretical results with computer simulations, including an example with realistic parameter values for *S. pneumoniae* amongst young children.

Acknowledgements

The authors would also like to thank the Scottish Government, the British Council Shanghai and the Chinese Scholarship Council for their financial support.

References

- [1] Anderson, D.R., Optimal exploitation strategies for an animal population in a Markovian environment: a theory and an example, *Ecology* 56, (1975), 1281-1297.
- [2] Anderson, W.J., Continuous-Time Markov Chains, Springer-Verlag, Berlin-Heidelberg, 1991.
- [3] Andersson, P. and Lindenstrand, D., A stochastic SIS epidemic with demography: initial stages and time to extinction, J. Math. Biol. 62, (2011), 333-348.
- [4] Artalejo, J.R., Economou, A. and Lopez-Herrero, M.J., On the number of recovered individuals in the SIS and SIR stochastic epidemic models, *Math. Biosci.* 228, (2010), 45-55.
- [5] Bhattacharyya, R. and Mukhopadhyay, B., On an eco-epidemiological model with prey harvesting and predator switching: Local and global perspectives, *Nonlinear Anal.: Real World Appl.* 11, (2010), 3824-3833.
- [6] Brauer, F., Allen, L.J.S., Van den Driessche, P. and Wu, J., Mathematical Epidemiology, Lecture Notes in Mathematics, No. 1945, Mathematical Biosciences Subseries, Springer-Verlag, Berlin-Heidelberg, 2008.
- [7] Brugger, S.D., Hathaway, L.J. and Mühlemann, K., Detection of Streptococcus pneumoniae strain cocolonization in the nasopharynx, J. Clin. Microbiol. 47(6), (2009), 1750-1756.
- [8] Caswell, H. and Cohen, J.E., Red, white and blue: Environmental variance spectra and coexistence in metapopulations, J. Theoret. Biol. 176 (1995), 301-316.
- [9] Coffey, T.J., Enright, M.C., Daniels, M., Morona, J.K., Morona, R., Hryniewicz, W., Paton, J.C. and Spratt, B.G., Recombinational exchanges at the capsular polysaccharide biosynthetic locus lead to frequent serotype changes among natural isolates of *Streptococcus pneumoniae*, Mol. Microbiol. 27, (1998), 73-83.
- [10] Diekmann, O. and Heesterbeek, J.A.P., Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis and Interpretation, John Wiley, Chichester, 2000.

- [11] Du, N.H., Kon, R., Sato, K. and Takeuchi, Y., Dynamical behaviour of Lotka-Volterra competition systems: non autonomous bistable case and the effect of telegraph noise, J. Comput. Appl. Math. 170 (2004), 399–422.
- [12] Farrington, P., What is the reproduction number for pneumococcal infection, and does it matter? in 4th International Symposium on Pneumococci and Pneumococcal Diseases, May 9-13 2004 at Marina Congress Center, Helsinki, Finland, 2004.
- [13] Feng, Z., Huang, W. and Castillo-Chavez, C., Global behaviour of a multi-group SIS epidemic model with age-structure, J. Diff. Eqns. 218(2) (2005), 292-324.
- [14] Gilpin, M.E., Predator-Prey Communities, Princeton University Press, Princeton, 1975.
- [15] Gopalsamy, K., Stability and Oscillations in Delay Differential Equations of Population Dynamics, Kluwer Academic, Dordrecht, 1992.
- [16] Gray, A., Greenhalgh, D., Hu, L., Mao, X. and Pan, J., A stochastic differential equation SIS epidemic model, SIAM J. Appl. Math. 71, (2011), 876-902.
- [17] Hethcote, H.W., Qualitative analyses of communicable disease models, Math. Biosci. 28 (1976), 335–356.
- [18] Hethcote, H.W. and Yorke, J.A., Gonorrhea Transmission Dynamics and Control, Lecture Notes in Biomathematics 56, Springer-Verlag, Berlin-Heidelberg, 1994.
- [19] Hoti, F., Erasto, P., Leino, T. and Auronen, K., 2009, Outbreaks of Streptoccocus Pneumoniae in day care cohorts in Finland - implications for elimination of transmission, BMC Infectious Diseases 9 (2009), 102, doi:10.1186/1471-2334-9-102.
- [20] Iannelli, M., Milner, F. A. and Pugliese, A., Analytical and numerical results for the age-structured SIS epidemic model with mixed inter-intracohort transmission, SIAM J. Math. Anal. 23(3) (1992), 662–688.
- [21] Lamb, K.E., Greenhalgh, D. and Robertson, C., A simple mathematical model for genetic effects in pneumococcal carriage and transmission, J. Comput. Appl. Math. 235(7) (2010), 1812–1818.
- [22] Li, J., Ma, Z. and Zhou, Y., Global analysis of an SIS epidemic model with a simple vaccination and multiple endemic equilibria, Acta Mathematica Scienta 26 (2006), 83–93.
- [23] Lipsitch, M., Vaccination against colonizing bacteria with multiple serotypes, Proc. Nat. Acad. Sci. 94 (1997), 6571-6576.
- [24] Liu, X. and Stechlinski, P., Pulse and constant control schemes for epidemic models with seasonality, Nonlinear Anal.: Real World Appl. 12, (2011), 931-946.
- [25] Mao, X., Stability of Stochastic Differential Equations with Respect to Semimartingales, Longman Scientific and Technical, London, 1991.

- [26] Mao, X., Exponential Stability of Stochastic Differential Equations, Marcel Dekker, New York, 1994.
- [27] Mao, X., Stability of stochastic differential equations with Markovian switching, Stoc. Proc. Appl. 79 (1999), 45–67.
- [28] Mao, X., Stochastic Differential Equations and Applications, Horwood Publishing, 2nd Edition, Chichester, 2007.
- [29] Mao, X. and Yuan, C., Stochastic Differential Equations with Markovian Switching, Imperial College Press, London, 2006.
- [30] Neal, P., Stochastic and deterministic analysis of SIS household epidemics, Adv. Appl. Prob. 38(4) (2006), 943-968.
- [31] Neal, P., The SIS great circle epidemic model, J. Appl. Prob. 45(2) (2008), 513-530.
- [32] Padilla, D.K. and Adolph, S.C., Plastic inducible morphologies are not always adaptive: the importance of time delays in a stochastic environment, *Evol. Ecol.* 10 (1996), 105-117.
- [33] Peccoud, J. and Ycart, B, Markovian modeling of gene-product synthesis, *Theoret. Pop. Biol.* 48(2) (1995), 222-234.
- [34] Slatkin, M., The dynamics of a population in a Markovian environment, *Ecology* 59 (1978), 249–256.
- [35] Takeuchi, Y., Global Dynamical Properties of Lotka-Volterra Systems, World Scientific Publishing Company, Singapore, 1996.
- [36] Takeuchi, Y., Du, N.H., Hieu, N.T. and Sato, K., Evolution of predator-prey systems described by a Lotka-Volterra equation under random environment, J. Math. Anal. Appl. 323 (2006), 938–957.
- [37] Van den Driessche, P. and Watmough, J. A simple SIS epidemic model with backward bifurcation, J. Math. Biol. 40 (2000), 525-540.
- [38] Weir, A., Modelling the impact of vaccination and competition on pneumococcal carriage and disease in Scotland. Unpublished Ph.D. Thesis, University of Strathclyde, Glasgow, Scotland, 2009.
- [39] Yang, Q., Jiang, D., Shi, N. and Ji, C., The ergodicity and extinction of stochastically perturbed SIR and SEIR epidemic models with saturated incidence, J. Math. Anal. Appl. 388, (2012), 248-271.
- [40] Zhang, Q., Arnaoutakis, K., Murdoch, C., Lakshman, R., Race, G., Burkinshaw, R. and Finn, A., Mucosal immune responses to capsular pneumococcal polysaccharides in immunized preschool children and controls with similar nasal pneumococcal colonization rates, *Pediatr. Infect. Dis. J.* 23 (2004), 307-313.