Stability Analysis of the Stochastic SIR Model with Discrete Delay

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Abstract

The purpose of this paper is to propose the stochastic infectious model with time delay and to study the stability of the disease-free steady state. In the prevalence of infectious diseases, environmental change and individual difference cause some kinds of random fluctuations in the infection recovery rate, immune effect, etc. Hence, the stochastic infectious model plays an important role in the analysis of the infection disease. Moreover, in the vector-borne diseases such as malaria and dengue fever, there exists time delay caused by an incubation period in the virus development in the vectors (mosquitoes) on the transmission of disease. Taking these facts into consideration, we propose a stochastic SIR (susceptible-infected-recovered) model with time delay. We analyze stability of the disease-free steady state, and study the influence of time delay and the random noise on the stability by numerical simulations.

1 Introduction

In February 2016, the World Health Organization (WHO) declared a state of emergency in response to the ongoing outbreak of Zika fever. Thus, including hepatitis C, HIV/AIDS and malaria, the various kinds of infectious diseases still remain as a threat to humankind. There has been growing interest in controlling infectious diseases to public health. Hence, the infectious disease prevention and the control is one of the important social issues [1]. Because of the difficulty of an experiment on a human body for infectious disease, mathematical models have become important tools in analyzing the spread and control of infectious diseases [2]-[8]. Up until now a variety of infectious models have been proposed, and many studies have been performed using the proposed models [2-9].

In the vector-borne infectious diseases such as malaria and Dengue fever, there exists time delay caused by an incubation period in the virus development in the vectors (mosquitoes) on the transmission of disease. Moreover, noting that in the real spread of the infectious disease, changes in the environment and the weather cause some kinds of random fluctuations in the infection, the recovery rates and others, we propose the stochastic infectious model with time delay.

In Section 2, one of the conventional infectious models, SIR (Susceptible-Infected-Recovered) model with time delay, is firstly explained, and then for more practical analysis of the spread of infectious diseases, we propose the stochastic SIR model with time delay. In the stochastic model, the randomly fluctuating recovery rate is considered because of the individual difference of the recovery rate. In Section 3, we summarize some definitions of the stability of the stochastic system with delay. Since the stability of the disease-free steady state is related to whether or not the infectious disease spreads, in Section 4, we study the stability of the disease-free steady state using the stochastic Lyapunov function. We derive the sufficient condition for the disease-free steady state to be stable. It is known that the multiplicative noise changes the bifurcation point [10]. Hence, calculating the maximum Lyapunov exponent of the considered system, we consider the influence of the random noise on the stability. In Section 5, the effect of the random noise and time delay of infection on the stability are studied through the numerical simulations.

2 Stochastic SIR Model with Discrete Time Delay

Letting the population densities of the susceptible, the infected and the recovered at time \( t \) be \( S(t) \), \( I(t) \) and \( R(t) \), we consider the interaction between each population as shown in Fig. 1. Figure 1 implies the infectious model (SIR model) with discrete time delay \( h \) below:

\[
\dot{S}(t) = \mu - (\mu + u)S(t) - \beta S(t)I(t - h), \quad (1)
\]
\[
\dot{I}(t) = \beta S(t)I(t - h) - (\mu + \gamma)I(t), \quad (2)
\]
\[
R(t) = \gamma I(t) - \mu R(t) + uS(t), \quad (3)
\]

where \( u \in (0, 1] \) denotes the percentage of susceptible individuals being vaccinated per unit of time, \( \beta, \mu, \gamma \) are the infection, the death (or birth) and the recovery rates, and the positive constant \( h \) is time delay of the infection.
Since the densities $S(t)$, $I(t)$ and $R(t)$ are the ratio of each population to the total population, the following relation holds:

$$N(t) \equiv S(t) + I(t) + R(t) = 1.$$  

Moreover, it should be noted that $N(t) = 1$ is a stable steady state of (1) to (3).

Noting that model parameters such as the infection and recovery rates in the infectious model contains some kinds of random fluctuations caused by the environmental change and the individual difference, we especially consider the recovery rate with a random fluctuation. Modeling such a randomness by the white Gaussian noise $\eta(t)$, we replace the recovery rate $\gamma$ in (2) and (3) in such a way that

$$\gamma(t) \longrightarrow \gamma(t) + \varepsilon \eta(t),$$  

where $\varepsilon$ is constant.

In addition, using the relation $\eta(t)dt = dw(t)$ between $\eta(t)$ and the Wiener process $w(t)$, the system (1) to (3) is rewritten by the following stochastic SIR model with discrete time delay $h$:

$$dS(t) = \{\mu - (\mu + u)S(t) - \beta S(t)I(t - h)\}dt,$$

$$dI(t) = \{\beta S(t)I(t - h) - (\mu + \gamma)I(t)\}dt - \varepsilon I(t)dw(t),$$

$$dR(t) = \{\gamma I(t) - \mu R(t) + uS(t)\}dt + \varepsilon I(t)dw(t).$$

Since the relation (4) holds in (6) to (8), we consider only (6) and (7) hereafter.

The initial conditions of (6) and (7) are given by

$$S(0) = S_0 > 0, \quad I(s) = I_0 > 0, \quad (-h \leq s \leq 0).$$

### 3 Stability of the Stochastic Time Delay System

In this section, we review the concept of the stochastic stability of the system with time delay. Letting $h$ be positive constant which denotes time delay, we define $x_t$ by

$$x_t(s) = x(t + s), \quad (-h \leq s \leq 0).$$

Then, the stochastic system with time delay is generally described in such a way that

$$dx(t) = f(t, x_t)dt + g(t, x_t)dw(t).$$

The initial condition of (11) is given by

$$x(s) = \varphi(s), \quad -h \leq s \leq 0.$$  

In (11), if $f(t, x_t) = ax(t)(1 - x(-h))$, $g(t, x_t) = x_t(0)$, then (11) yields the stochastic logistic equation with time delay:

$$dx(t) = ax(t)(1 - x(t - h))dt + x(t)dw(t).$$

Assuming $f(t, 0) = g(t, 0) = 0$ and $x_t$ is $n$-dimension in (11), we define the concept of the stochastic stability of zero solution (steady state).

**Definition 1** *Mean Square Stable:* The zero solution of (11) is called mean square stable if for any $\varepsilon > 0$, there exists a $\delta(\varepsilon) > 0$ such that

$$||\varphi||_1 < \delta(\varepsilon) \rightarrow E[|x(t; \varphi)|^2] < \varepsilon, \quad \forall t \geq 0,$$

where $|\cdot|$ is the Euclidean norm, $x(t; \varphi)$ is the solution of (11) with the initial value $\varphi$ and $|| \cdot ||_1$ is defined by

$$||\varphi||_1^2 = \sup_{-h \leq s \leq 0} E[|\varphi(s)|^2].$$

**Definition 2** *Asymptotically Mean Square Stable:* The zero solution of (11) is called asymptotically mean square stable if it is mean square stable and there exists $\delta > 0$ such that

$$||\varphi||_1 < \delta \rightarrow \lim_{t \rightarrow \infty} E[|x(t; \varphi)|^2] = 0.$$  

**Definition 3** *Stable in Probability:* The zero solution of (11) is called stable in probability if for any $\varepsilon_1 > 0, \varepsilon_2 > 0$, there exists $\delta(\varepsilon) \equiv \delta(\varepsilon_1, \varepsilon_2)$ such that

$$P(||\varphi|| < \delta(\varepsilon)) = 1 \rightarrow P(\sup_{t \geq 0} |x(t; \varphi)| > \varepsilon_1) < \varepsilon_2,$$

where $|| \cdot ||$ is given by

$$||x|| = \sup_{-h \leq s \leq 0} |x(s)|.$$  

For simplicity of descriptions, we hereinafter referred to $x(t; \varphi)$ as $x(t)$ unless it causes confusion.

### 4 Stability Analysis of the Disease-free Steady State

In this section, we consider the stability of the disease-free steady state of the stochastic SIR model with time delay (6) and (7). The disease-free steady state means the equilibrium solution with $I(t) \equiv 0$ in (6) and (7). If the disease-free steady state is stable, even if the infectious disease breaks, prevalence of disease has eventually ended. Hence, the stability analysis of the disease-free steady state is very important in the epidemiology. Moreover, using the stability condition of the disease-free steady state, we are able to know the necessary vaccination rate to control prevalence of infectious disease.

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[Diagram of interaction between each population shown.]
In (22) and (23), we study the stability of the linearized equations (22) and (23). Before considering the stability of (22) and (23), we have the following results.

**Theorem 1** Under the following condition, the origin of (22) and (23) is asymptotically mean square stable.

\[ 0 < \beta < \beta \equiv \frac{1}{S_f} \min \left\{ 2(\mu + u), \mu + \gamma - \frac{1}{2} \varepsilon^2 \right\}. \]  

(26)

The outline of the proof is as follows. (The detailed proof of Theorem 1 is described in Appendix A.)

First, we introduce the function \( V_1(t, x_t) \) such that

\[ V_1(t, x_t) = x_t^2 + A x_t^2 + B \int_{t-h}^{t} x_s(s)^2 \, ds, \]

(27)

where \( A \) and \( B \) are given by

\[ A = \frac{2 \beta S_f}{2(\mu + \gamma - \beta S_f) - \varepsilon^2}, \quad B = (1 + A) \beta S_f. \]  

(28)

By proving that the function \( V_1(t, x_t) \) is a stochastic Lyapunov function and there exists the positive constant \( k \) such that

\[ \frac{\partial V_1(t, x_t)}{\partial t} + \mathcal{L}_1 V_1(t, x_t) \leq -k |x(t)|^2, \]

(29)

the proof of Theorem 1 is performed.

In (29), the operator \( \mathcal{L}_1(\cdot) \) is a generating operator of (24) and (25) given by

\[ \mathcal{L}_1(\cdot) = \left\{ \frac{\partial (\cdot)}{\partial x} \right\} f + \frac{1}{2} \text{tr} \left\{ \left( \frac{\partial}{\partial x} \left( \frac{\partial (\cdot)}{\partial x} \right) g g^T \right) \right\}, \]

(30)

where \( f = [f_1 \ f_2]' \), \( g = [g_1 \ g_2]' \) and \( f_i, g_i (i = 1, 2) \) are defined by

\[ f_1 = -(\mu + u)x_1(t) - \beta S_f x_2(t - h), \]

(31)

\[ f_2 = \beta S_f x_2(t - h) - (\mu + \gamma)x_2(t), \]

(32)

and where \([\cdot]'\) denotes the transpose of \([\cdot]\).

For the stability of (22) and (23), we have the following results.

**Theorem 2** Under the same condition as Theorem 1, the origin of (22) and (23) is stable in probability.

The detailed proof of Theorem 2 is described in Appendix B. The proof is performed by the following procedure.

First, for any \( \delta > 0 \), considering \( x_t \) with

\[ P\{ \sup_{-h \leq s \leq 0} |x_s(s)| < \delta \} = 1, \]

(34)

we define \( V_2(t, x_t) \) as

\[ V_2(t, x_t) = x_t^2(t) + A x_t^2(t) + C \int_{t-h}^{t} x_s(s)^2 \, ds, \]

(35)

where \( A \) is defined by (28) and \( C \) is given by

\[ C = (1 + A) \beta S_f + \beta \delta. \]

(36)

By proving that the function \( V_2(t, x_t) \) is the stochastic Lyapunov function, i.e., which satisfies

\[ \frac{\partial V_2(t, x_t)}{\partial t} + \mathcal{L}_2 V_2(t, x_t) \leq 0, \]

(37)

the proof of Theorem 2 is performed.

In (37), the operator \( \mathcal{L}_2(\cdot) \) is defined by replacing \( f = [f_1 \ f_2]' \) in (31) and (32) by

\[ f_1 = -(\mu + u)x_1(t) - \beta (x_1(t) + S_f)x_2(t - h), \]

(38)

\[ f_2 = \beta (x_1(t) + S_f)x_2(t - h) - (\mu + \gamma)x_2(t), \]

(39)

the function \( g \) in \( \mathcal{L}_2(\cdot) \) is given by (33).

From Theorems 1 and 2, if we choose the vaccination rate \( u \) so as to satisfy the condition (26), the disease-free steady state becomes stable, so that the prevalence of the infectious disease is controlled.

In [10], by considering the change of the shape of the stationary probability density function(spdf), it is shown that the multiplicative noise changes the bifurcation point. The change of the shape of the spdf is related to the P-(phenomenological) bifurcations [11]. In this paper, since the P-bifurcation approach has a possibility of missing the bifurcation branch in the bifurcation diagram, we consider the D(dynamical)- bifurcation [11] of the stochastic infectious model with time delay. The D-bifurcation is defined using the invariant measure and it has a strong relation with the Lyapunov exponent. Hence, we calculate the maximum Lyapunov exponent \( \ell \) of the system (22) and (23) under
the values of parameters such as \( u = 0.01, \mu = 0.1, \gamma = 0.08, h = 5 \), and the result is shown in Fig. 2. Figure 2 depicts the dependency of the maximum Lyapunov exponent on the noise strength \( \varepsilon \) and the infection rate \( \beta \). Since the D-bifurcation point is characterized by the value of the bifurcation parameter at which the maximum Lyapunov exponent becomes zero, Fig. 2 shows that the bifurcation point shifts to the right with increasing of the strength of the noise. This fact means that the region of the infection rate \( \beta \) in which the disease-free steady state is stable expands with the increase of the strength \( \varepsilon \) of the noise. Therefore, there exists the region of the infection rate in which it is unstable under the no noise, whereas it becomes stable under the noise. In other words, the random noise has an effect of the stabilization of the system.

Remark 1: The sufficient condition (26) of Theorems 1 and 2 means that the stability region of the infection rate decreases with the increase of the strength \( \varepsilon \) of the noise. Although the condition (26) does not coincide with the result of Fig. 2, it is caused by the fact that (26) is the sufficient condition of the stability.

Fig. 2: Dependency of the maximum Lyapunov exponent on the noise strength \( \varepsilon \) and the infection rate \( \beta \)

Choosing the same parameter values as Fig. 2, setting the infection rate as \( \beta = 0.2 \), we performed simulations under the no noise and noise with the strength \( \varepsilon = 0.1 \). Since it follows from the magnified figure (Fig. 3) of Fig. 2 that \( \beta(0) < 0.2 < \beta(0.1) \), the disease-free steady state with the infection rate \( \beta = 0.2 \) is unstable under the no noise but stable under the noise. The results of simulations are shown in Figs. 4 and 5. Figures 4 and 5 denote the time evolutions of susceptible \( S(t) \) and infective \( I(t) \) under the noise and the no noise. Figures 4 and 5 establish the validity of Fig. 2, i.e., the disease-free steady state is stable under the noise from Fig. 4, whereas it is unstable under the no noise from Fig. 5. In Fig. 5, \( (S(t), I(t)) \) converges to the neighborhood of the deterministic endemic steady state \( (S_e, I_e) = (\mu, \gamma) / \beta, (\beta \mu - (\mu + u)(\mu + \gamma)) / (\beta(\mu + \gamma)) \approx (0.9, 0.006) \). The endemic steady state means the steady state that infection is maintained in the population.

Fig. 3: Magnified figure of Fig. 2 in the range of the infection rate \( 0.18 \leq \beta \leq 0.23 \)

Fig. 4: Time evolutions of susceptible \( S(t) \) and infective \( I(t) \) under the noise

5 Simulations

In this section, we study the influence of the random noise and the time delay on the stability of the disease-free steady state by the numerical simulations.

5.1 The Influence of the Noise on the Stability

We denote the infection rate \( \beta \) at which the maximum Lyapunov exponent \( \ell \) becomes zero as \( \beta(a) \) under the noise strength \( \varepsilon = a \), i.e., \( \ell(\beta(a)) = 0 \). Then, it follows from Fig. 2 that \( \beta(a) \) is a monotonically increasing function of \( a \). In the infection rate \( \beta \) within the region \( \beta(0) < \beta < \beta(a) \), the disease-free steady state is unstable under the no noise, however, it becomes stable under the noise with the strength \( \varepsilon = a \).
5.2 The Influence of Time Delay on the Stability

Setting the infection rate $\beta = 0.22$, the noise strength $\varepsilon = 0.02$ and the other parameter values are the same in Section 5.1, we study the influence of time delay $h$ on the stability of the disease-free steady state under $h = 0$ (no delay), 5, 10, 15. In these parameter values, the disease-free steady state is unstable because the Lyapunov exponent $\ell$ is positive as estimated from Figs. 2 and 3. The results of simulations are shown in Fig. 6. Figure 6 trends that the infective $I(t)$ converges to the neighborhood of the deterministic endemic steady state $I_e = 0.056$ independently of time delay $h$. Hence, Fig. 6 means that time delay has little influence on the stability.

In order to see the difference of the behavior of infective in detail, we show the magnified figure of Fig. 6 in the time interval $[0, 100]$ in Fig. 7.

In the early stage of the time evolution of the infective $I(t)$, although there exists a little effect of time delay on the behavior, the effect of time delay eventually disappears as shown in Fig. 6. Although the result is omitted, in the case where the disease-free steady state is stable, regardless of time delay, the infective $I(t)$ converges to zero. Hence, the effect of time delay eventually disappears as well as the case of the unstable case.

6 Conclusions

In this paper, we have proposed the stochastic infectious model with time delay and studied the stability of the disease-free steady state and the influence of the random noise and the time delay on the stability by the numerical simulations. We derived the sufficient condition for the disease-free steady state to be stable using the stochastic Lyapunov function. By calculating the Lyapunov exponent, we have shown that the random noise has an effect to stabilize the disease-free steady state. In other words, there exists the region of the infection rate in which the disease-free steady is unstable under the no noise but stable under the noise. This fact is confirmed by the numerical simulations. From the viewpoint of the bifurcation theory, we are able to say that the random noise considered in this paper shifts the deterministic bifurcation point in the direction of stabilization. In [10], it is shown that the multiplicative noise changes the deterministic bifurcation point by calculating the extremal point of the stationary probability density function. This approach is called the bifurcation analysis based on the P-bifurcation, however, this approach has the disadvantage that some bifurcation branch may be missed in the bifurcation diagram. In order to overcome this drawback, we employed the D-bifurcation approach which
is related to the Lyapunov exponent. By calculating the Lyapunov exponent, we have shown that the deterministic bifurcation point moves to the right with increasing of the intensity of the noise.

From the results of the numerical simulations, it turns out that time delay of the infection has little influence on the stability. As shown in Fig. 7, the decreasing of the intensity of the noise. increasing of the noise.

From Theorems 1 and 2, we are able to know the vaccination rate $u$ necessary to inhibit to become epidemic. That is, if we choose the vaccination rate $u$ so as to satisfy the condition (26), the disease-free steady state becomes stable, so that the prevalence of the infectious disease is controlled. In the no noise case, we estimate the vaccination rate $u$ necessary to inhibit to become epidemic by taking the noise strength $\varepsilon = 0$ in (26). However, since (26) is the sufficient condition for the disease-free steady state to be stable, it should be noted that there is a possibility to exist the smaller vaccination rate $u$ than one deduced from (26).

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A Proof of Theorem 1

It follows from [7],[12] that if (26) holds and there exist positive constants $k_i$, $i = 0, 1, 2$ such that

\begin{align}
(a) \quad k_0E\{|x(t)|^2\} &\leq E\{V(t, x_t)\} \leq k_1||x_1||^2_i, \\
(b) \quad \frac{\partial V(t, x_t)}{\partial t} + LV(t, x_t) &\leq -k_2|x(t)|^2,
\end{align}

then the zero solution of (24) and (25) is asymptotically mean square stable.

Using (10) and (15), since it is easily shown that the relation (40) holds, it suffices for the proof of Theorem 1 to prove (41).

From (24), (25), (27) and (30), we have

\[
\frac{\partial V(t, x_t)}{\partial t} + L V(t, x_t) = B x_2^2 - B x_2^2 (t-h)^2 \\
+ 2x_1 (\{ (\mu + u) x_1 - \beta S f x_2 (t-h) \} \\
+ 2A x_2 \{ \beta S f x_2(t-h) - (\mu + \gamma) x_2 \} + A e^2 x_2^2 \\
- \{ 2(\mu + u) - \beta S f \} x_2^2 - \{ 2A (\mu + \gamma) - A \beta S_f \} \\
- A e^2 - B \} x_2^2 + \{(1+A)\beta S_f - B\} x_2^2 (t-h)^2.
\]

(Using (28), we have)

\[
= -\{ 2(\mu + u) - \beta S_f \} x_2^2 - \beta S_f x_2^2 \\
\leq -\alpha|\alpha(t)|^2,
\]

where $\alpha = \min\{2(\mu + u) - \beta S_f, \beta S_f\}$.
B Proof of Theorem 2

It suffices for the proof of Theorem 2 to prove (37). It follows from (28), (35) and (36) that

\[
\frac{\partial V_2(t,x_1)}{\partial t} + \mathcal{L}_2 V_2(t,x_1) = Cx_2^2 - Cx_2(t-h)^2
\]

\[+2x_1\{(\mu + u)x_1 - \beta x_1 S_f x_2(t-h)\}
+2Ax_2\{\beta x_1 S_f x_2(t-h) - (\mu + \gamma)x_2\} + A\varepsilon x_2^2
\] (42)

Noting that from (34), the following relations hold

\[
2x_1(x_1+S_f)x_2(t-h) \leq (\delta+S_f)x_1^2 + (\delta+S_f)x_2(t-h)^2
\] (43)

\[
2x_2(x_1+S_f)x_2(t-h) \leq \delta x_1^2 + (\delta+S_f)x_2^2 + S_f x_2(t-h)^2,
\] (44)

(The R.H.S. of (42)) \[\leq -\{2(\mu + u) - \beta(\delta + S_f) - A\beta\}x_1^2
\]

- \{2A(\mu + \gamma) - A\varepsilon^2 - C - A\beta(\delta + S_f)\}x_2^2

+ \{\beta(\delta + S_f) + A\beta S_f - C\}x_2(t-h)^2

(Using (36), we have)

\[= -2(\mu + u) - \beta S_f - \beta\delta(1 + A)\}x_1^2
\]

- \{(2\mu + 2\gamma - \varepsilon^2 - 2\beta S_f)A - \beta(\delta + S_f) - \beta\delta A\}x_2^2

(Using (28), we have)

\[= -2(\mu + u) - \beta S_f - \beta\delta(1 + A)\}x_1^2
\]

- \{\beta S_f - \delta\beta(1 + A)\}x_2.

Hence, for a sufficiently small \(\delta > 0\), it follows from (26) that the relation (37) holds.