Stability Analysis of the Stochastic Delayed Infectious Model with Vaccination*

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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 rate, immune effect, etc. Hence, the stochastic infectious model plays an important role in the analysis of the infectious 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 on the transmission of disease. Taking these facts into consideration, we propose a stochastic susceptible-infected-recovered model with time delay. We analyze stability of the disease-free steady state using the stochastic Lyapunov function, 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. Infectious diseases still remain as a threat to humankind. Hence, the infectious disease prevention and the control is one of the important social issues [1–3]. 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 [4–10]. In [4,5], taking a vaccination rate as a bifurcation parameter, they show that a backward bifurcation is possible under the inadequate vaccination rate. In [6], the infectious model with the nonlinear incident rate is proposed and the backward bifurcation is studied. In [7–9], choosing the vaccination rate as the control input, the optimal control problems for the deterministic infectious models are considered using the maximum principle. Moreover, the stochastic optimal control problem for the infectious model is studied using the stochastic maximum principle in [10].

In the vector-borne infectious diseases such as malaria [11] and deng fever, there exists time delay caused by an incubation period in the virus development in the vectors (mosquitoes) on the transmission of disease. Moreover, in the real spread of the infectious disease, changes in the environment and the weather cause some kinds of random fluctuations in the infection and the recovery rates. Taking these facts into consideration, we propose the stochastic infectious model with time delay.

In Section 2, one of the conventional infectious models, the SIR(susceptible-infected-recovered) model with time delay, is 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 applied in consideration of the individual difference of the recovery rate. In Section 3, we summarize some definitions of the stability of the stochastic system with time 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. After analyzing the stability of the disease-free steady state of the deterministic system, we consider the stability of the disease-free steady state of the stochastic delayed system using the stochastic Lyapunov function. We derive the sufficient condition for the stochastic disease-free steady state to be stable.

Since it is known that the multiplicative noise has a capacity to change the bifurcation point [12], by calculating the maximum Lyapunov exponent of the stochastic delayed SIR 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 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. Fig. 1 yields the infectious model (SIR model) with time delay $h$ below:

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

where $u \in [0,1]$ denotes the vaccination rate, $\beta, \mu, \gamma$ are the infection rate, the death (or birth), the recovery rates, and the positive constant $h$ is time delay of the infection.

![Fig. 1 Interaction between each population](image)

Setting the total density as $N(t) = S(t) + I(t) + R(t)$, it follows from (1) to (3) that the following relation holds under $N(0) = 1$:

\[ N(t) = 1. \quad (4) \]

In general, model parameters in the infectious model contain some kinds of random fluctuations caused by the environmental change and the individual difference. In this paper, we especially consider the effect of the random fluctuation in the infected density $I(t)$ on the behavior of susceptible population density $S(t)$. Hence, we introduce the random fluctuation in (2). The noises in the birth (or death), the infection and the recovery rates, $\mu, \beta$ and $\gamma$ induce the random fluctuation in (2). Because, $\mu$ and $\beta$ are contained in (1) and (2), the random fluctuations of $\mu$ and $\beta$ directly induce the random fluctuations in both of the susceptible and the infected densities. Since (1) to (3) denote the model of the infectious disease without reinfection such as mumps, the recovered density $R(t)$ has no effect on the other population densities. In other words, (1) and (2) are independent of (3). Then, we consider the random fluctuation in the recovery rate $\gamma$. 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

\[ \gamma(t) \longrightarrow \gamma(t) + c\eta(t), \quad (5) \]

where $c$ 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 time delay $h$:

\[ \begin{align*}
\dot{S}(t) & = -\mu S(t)S - \beta S(t)I(t-h), \\
\dot{I}(t) & = \beta S(t)I(t-h) - (\mu + \gamma)I(t), \\
\dot{R}(t) & = \gamma I(t) - \mu R(t) + uS(t),
\end{align*} \]

Equation (4) holds in (6)~(8), because it follows from (6)~(8) that $N(t) = 0$, and we have $N(t) = 1$ under $N(0) = 1$.

The initial conditions of (6)~(8) are given by

\[ \begin{align*}
S(0) & = S_0 > 0, \quad I(s) = I_0 > 0, \quad (-h \leq s \leq 0), \\
R(0) & = 1 - S_0 - I_0 > 0.
\end{align*} \]

It should be noted that the solutions of (6)~(8) are nonnegative because noises in (7) and (8) are multiplicative noise and the deterministic solutions of (1)~(3) are nonnegative [12].

Equations (6) and (7) are independent of (8) and $R(t)$ is determined by (4), hence we consider only (6) and (7) hereafter.

3. Stability of the Stochastic Delayed System

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

\[ x_i(s) = x(t+s), \quad (-h \leq s \leq 0). \quad (10) \]

Then, the stochastic delayed system is generally described by the functional differential equation:

\[ dx(t) = f(t, x_i)dt + g(t, x_i)dw(t). \quad (11) \]

The initial condition of (11) is given by

\[ x(s) = \varphi(s), \quad -h \leq s \leq 0. \quad (12) \]

Noting that (11) is the functional equation of $x_i(\cdot)$, if setting as $f(t, x_i) = ax_i(0)(1-x_i(-h))$, $g(t, x_i)(\cdot) = x_i(0)$ in (11), then (11) yields the stochastic logistic equation with time delay $h$ such that

\[ dx(t) = ax(t)(1-x(t-h))dt + x(t)dw(t). \quad (13) \]

Assuming $f(t,0) = g(t,0) = 0$ and $x_i$ is $n$-dimension in (11), we define 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

\[ ||\varphi|| < \delta(\varepsilon) \rightarrow E[|x(t;\varphi)|^2] < \varepsilon, \quad \forall t \geq 0, \quad (14) \]

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
\[
||\varphi||_1^2 = \sup_{-h \leq s \leq 0} E\{||\varphi(s)||^2\}. \tag{15}
\]

**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
\[
||\varphi||_1 < \delta \rightarrow \lim_{t \to \infty} E\{|x(t;\varphi)|^2\} = 0. \tag{16}
\]

**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
\[
P\{||\varphi|| < \delta(\varepsilon)\} = 1 \rightarrow P\{\sup_{t \geq 0} |x(t;\varphi)| > \varepsilon_1\} < \varepsilon_2, \tag{17}
\]
where \( ||\cdot|| \) is defined by
\[
||x|| = \sup_{-h \leq s \leq 0} |x(s)|. \tag{18}
\]
For simplicity of descriptions, we hereafter 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 infectious model with time delay. The disease-free steady state means the equilibrium solution with zero infected individuals 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.

Denoting the disease-free steady state of (6) and (7) as \((S_f, I_f)\), we have
\[
(S_f, I_f) = \left( \frac{\mu}{\mu + u}, 0 \right). \tag{19}
\]
In order to make the origin the steady state, we introduce the change of variable such that
\[
x_1(t) = S(t) - S_f, \quad x_2(t) = I(t). \tag{20}
\]
Then, we have
\[
dx_1 = \{- (\mu + u)x_1(t) - \beta(x_1(t) + S_f)x_2(t - h)\}dt, \tag{21}
\]
\[
dx_2 = \{\beta(x_1(t) + S_f)x_2(t - h) - (\mu + \gamma)x_2(t)\}dt - ex_2(t)dw(t). \tag{22}
\]
The linear parts of (21) and (22) are given by
\[
dx_1 = \{- (\mu + u)x_1(t) - \beta S_f x_2(t - h)\}dt, \tag{23}
\]
\[
dx_2 = \{\beta S_f x_2(t - h) - (\mu + \gamma)x_2(t)\}dt - ex_2(t)dw(t). \tag{24}
\]

### 4.1 Stability of the Deterministic Linear Delayed System

Since the behaviors of (21) and (22) are bound up with the stability of the corresponding deterministic linear system, setting as \( e = 0 \) in (24), we first consider the stability of the deterministic linear system below:
\[
\dot{x}_1(t) = -(\mu + u)x_1(t) - \beta S_f x_2(t - h), \tag{25}
\]
\[
\dot{x}_2(t) = \beta S_f x_2(t - h) - (\mu + \gamma)x_2(t). \tag{26}
\]

The characteristic equation of (25) and (26) becomes
\[
\lambda - \beta S_f e^{-\lambda h} + \mu + \gamma = 0. \tag{27}
\]

Choosing the infection rate \( \beta \) as the bifurcation parameter and setting \( \beta \equiv (\mu + \gamma)/S_f \), we study the property of the solution \( \lambda \equiv \lambda(\beta) \) of (28).

- **Case A** \( \beta = \hat{\beta} \): In this case, since (28) has always the zero solution \( \lambda = 0 \), the origin of (25) and (26) never become asymptotically stable. We study the stability in more detail. First, assuming that \( \lambda = p + iq \) \((p > 0)\) is the solution of (28), we have
\[
p - \beta S_f e^{-\lambda h} \cos(qh) + \mu + \gamma = 0, \tag{29}
\]
\[
q + \beta S_f e^{-\lambda h} \sin(qh) = 0. \tag{30}
\]
Noting that \( p, h > 0 \), from (29) and (30), we have
\[
(p + \mu + \gamma)^2 + q^2 = (\beta S_f)^2 e^{-2ph} < (\beta S_f)^2. \tag{31}
\]

Noting \( \beta = \hat{\beta} \) and using \( \beta S_f = \hat{\beta} S_f = \mu + \gamma \) in (31), we obtain
\[
p^2 + 2p(\mu + \gamma) + q^2 < 0. \tag{32}
\]

Since \( \mu + \gamma > 0 \), (32) contradicts \( p > 0 \). Hence, we have \( p \leq 0 \). As mentioned above, since (28) has always the zero solution \( \lambda = 0 \) in the case of \( \beta = \hat{\beta} \), the origin never becomes asymptotically stable. Therefore, we conclude that \( p = 0 \) and the origin is stable.

- **Case B** \( \beta \neq \hat{\beta} \): In the case where the stability changes, since the eigenvalue comes across the imaginary axis with change of the bifurcation parameter, we consider whether or not (28) has pure imaginary solution. Setting as \( \lambda = iq \) \((q > 0)\), i.e., \( p = 0 \) in (29) to (30), we have
\[
-\beta S_f \cos(qh) + \mu + \gamma = 0, \tag{33}
\]
\[
q + \beta S_f \sin(qh) = 0. \tag{34}
\]

Equations (33) and (34) yield
\[ q^2 = (\beta S_f)^2 - (\mu + \gamma)^2 = S_f^2(b^2 - \beta^2). \]

Regarding (35), we study the stability in two cases such as \( \beta > \hat{\beta} \) and \( \beta < \hat{\beta} \).

(B-1) \( \beta > \hat{\beta} \): In this case, from (35), there exists a pure imaginary solution of (28). Since it follows from (28) that

\[ \frac{d\lambda}{dz} = \frac{\lambda + \mu + \gamma}{\beta(1 + h(\mu + \gamma + \lambda))}, \]

we have

\[ \text{sgn}\left\{ \frac{dR(\lambda)}{d\beta}\bigg|_{\lambda=i\gamma} \right\} = \text{sgn}\left\{ \Re\left( \frac{d\lambda}{d\beta}\bigg|_{\lambda=i\gamma} \right) \right\} \]

\[ = \text{sgn}\left\{ \frac{(\mu + \gamma)(1 + (\mu + \gamma)h) + q^2h}{\beta(1 + (\mu + \gamma)h)^2 + (qh)^2} \right\} = 1, \quad (37) \]

where \( \text{sgn}(x) \) is the signum function of \( x \) and \( \Re(\lambda) \) denotes the real part of \( \lambda \).

Equation (37) yields the following relation:

\[ \frac{dR(\lambda)}{d\beta}\bigg|_{\lambda=i\gamma} > 0. \quad (38) \]

Hence, the eigenvalue comes across from left to right the imaginary axis at \( i\gamma \) with increasing of \( \beta \). Considering the result of (Case A) and (38), we have

\[ \Re(\lambda(\hat{\beta})) = 0. \quad (39) \]

It follows from (38) and (39) that in the case of \( \beta > \hat{\beta} \), the origin is unstable.

(B-2) \( \beta < \hat{\beta} \): In this case, the pure imaginary solution does not exist from (35). Hence, eigenvalue does not across the imaginary axis even if the bifurcation parameter \( \beta \) changes, so that the stability of the origin also does not change.

From (29), we have

\[ p = \beta S_f e^{-ph} \cos(qh) - (\mu + \gamma). \quad (40) \]

We assume that \( p \geq 0 \), then noting \( h > 0 \) and \( \beta < \hat{\beta} \), (40) yields

\[ \text{(The R.H.S. of (40))} \leq S_f(\beta - \hat{\beta}) < 0. \quad (41) \]

The relation (41) contradicts the assumption \( p \geq 0 \), so that we have \( p < 0 \). Hence, in the case of \( \beta < \hat{\beta} \), the origin is asymptotically stable.

Consequently, we have the result below:

[Lemma 1] Denoting \( \hat{\beta} \equiv (\mu + \gamma)/S_f \), the stability of the deterministic linear delayed system (25) and (26) is summarized below:

(a) If \( \beta = \hat{\beta} \), the origin is stable,
(b) If \( \beta < \hat{\beta} \), the origin is asymptotically stable,
(c) If \( \beta > \hat{\beta} \), the origin is unstable.

In this way, \( \hat{\beta} \) is the bifurcation point of the deterministic linear delayed system (25) and (26).

After we study the stability of the linearized stochastic delayed system (23) and (24), we consider the stability of the stochastic delayed system of (21) and (22). We have the following result of the stability of (23) and (24).

[Theorem 1] Under the following condition, the origin of (23) and (24) is asymptotically mean square stable.

\[ 0 < \beta < \beta^* \equiv \frac{1}{S_f} \min\left\{ 2(\mu + u), \mu + \gamma - \frac{1}{2} e^2 \right\}. \quad (42) \]

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

First, we introduce the function \( V_1(t, x_1) \) such that

\[ V_1(t, x_1) = x_1^2 + Ax_2^2 + B \int_{t-h}^t x_2(s)^2 ds, \quad (43) \]

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

\[ A = \frac{2\beta S_f}{2(\mu + \gamma - \beta S_f)} - e^2, \quad B = (1 + A)\beta S_f. \quad (44) \]

It should be noted that the constant \( A \) is positive from the condition (42).

By proving that the function \( V_1(t, x_1) \) is a stochastic Lyapunov function and there exists the positive constant \( k \) such that

\[ \frac{\partial V_1(t, x_1)}{\partial t} + L_1 V_1(t, x_1) \leq -k|x(t)|^2, \quad (45) \]

the proof of Theorem 1 is performed.

In (45), the operator \( L_1(\cdot) \) is a generating operator of (23) and (24) given by

\[ L_1(\cdot) = \left\{ \frac{\partial(\cdot)}{\partial x} \right\} f + \frac{1}{2} \text{tr} \left\{ \left( \frac{\partial}{\partial x} \left( \frac{\partial(\cdot)}{\partial x} \right) gg' \right) \right\}. \quad (46) \]

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

\[ f_1 = -(\mu + u) x_1(t) - \beta S_f x_2(t - h), \quad (47) \]
\[ f_2 = \beta S_f x_2(t - h) - (\mu + \gamma) x_2(t), \quad (48) \]
\[ g_1 = 0, \quad g_2 = -e x_2(t), \quad (49) \]

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

( Remark 1) Noting that \( S_f \) defined by (19) is decreasing function of the vaccination rate \( u \), the condition (42) implies that the stable region of the infection rate \( \beta \) increases with \( u \). Hence, in the case of the disease-free steady state is unstable under the no vaccination, there is a possibility of stabilizing the disease-free steady state by vaccination. Moreover, we can estimate the necessary vaccination rate to control the infectious disease spreading by (42).

( Remark 2) Using the deterministic bifurcation point \( \hat{\beta} \) in Lemma 1, we can rewrite \( \beta^* \) in (42) as

\[ \beta^* \equiv \min \left\{ 2(\mu + u)\hat{\beta}/(\mu + \gamma), \hat{\beta} - e^2/(2S_f) \right\}. \quad (50) \]
Equations (42) and (50) show the relation of the stable region of the infection rate $\beta$ between deterministic and stochastic systems.

For the stability of (21) and (22), we have the following results.

[Theorem 2] Under the same condition as Theorem 1, the origin of (21) and (22) is stable in probability.

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

First, for any $\delta > 0$, considering $x_t$ satisfies

$$P\left\{ \sup_{-h \leq s \leq 0} |x_t(s)| < \delta \right\} = 1,$$

we define $V(t,x_t)$ as

$$V_2(t,x_t) = x_1^2(t) + Ax_2^2(t) + C \int_{t-h}^{t} x_2(s)^2 ds,$$

where $A$ is defined by (44) and $C$ is given by

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

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

$$\frac{\partial V_2(t,x_t)}{\partial t} + L_2 V_2(t,x_t) \leq 0,$$

the proof of Theorem 2 is performed.

In (54), the operator $L_2(\cdot)$ is defined by replacing $f = [f_1, f_2]^T$ in (47) and (48) by

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

$$f_2 = \beta(x_1(t) + S_f)x_2(t-h) - (\mu + \gamma)x_2(t).$$

the function $g$ in $L_2(\cdot)$ is given by (49).

From Theorems 1 and 2, if we choose the vaccination rate $u$ so as to satisfy the condition (42), the disease-free steady state becomes stable, so that the prevalence of the infectious disease is controlled.

In [13,14] by considering the change of the shape of the stationary probability density function (pdf), it is shown that the multiplicative noise changes the bifurcation point. The change of the shape of the pdf is related to the P-(phenomenological) bifurcations [14]. In this paper, since the P-bifurcation approach has a possibility of missing the bifurcation branch in the bifurcation diagram, so we consider the D(dynamical)-bifurcation [14] 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 (21) and (22) 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 $\ell$ on the noise strength $\epsilon$ 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 $\epsilon$ 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 3 The sufficient condition (42) of Theorems 1 and 2 means that the stability region of the infection rate decreases with the increase of the strength $\epsilon$ of the noise. Although the condition (42) does not coincide with the result of Fig. 2, it is caused by the fact that (42) is one of the sufficient condition of the stability. The construction of the Lyapunov function with the stability condition that the stability region of the infection rate increases with the increase of the strength $\epsilon$ of the noise is an issue in the future.

Whereas Fig. 2 numerically shows the region of the infection rate $\beta$ for the disease-free steady state to be stable (or unstable), (42) is useful and meaningful in the sense that the stable region of $\beta$ is theoretically described.

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 Noise Effect on the Stability

Before considering the influence of the noise on the stability, we consider the validity of Theorems 1 and 2. Choosing the same values of parameters as Fig. 2, i.e., $u = 0.01, \mu = 0.1, \gamma = 0.08, h = 5$, and setting the noise coefficient $\epsilon$ as $\epsilon = 0.1$, we have $\beta^* = 0.1925$ in (42). Hence, under the infection rate $\beta = 0.19 < \beta^*$,
the disease-free steady state is stable. The simulation result under $\beta = 0.19$ is shown in Fig. 3, which gives the validity of Theorems 1 and 2 because Fig. 3 means that the disease-free steady state is stable under (42).

![Fig. 3 Time evolutions of susceptible $S(t)$ and infected $I(t)$ under the noise](image)

Under the no vaccination (i.e., $u = 0$), and if all the other parameters are the same as in Fig. 3, we have $\beta^* = 0.1750 < \beta = 0.19$. The simulation result is shown in Fig. 4. In Fig. 4, the infected $I(t)$ converges to the neighborhood of the deterministic endemic steady state $(S_e, I_e) = ((\mu + \gamma)/\beta, (\beta \mu - \mu (\mu + \gamma))/(\beta (\mu + \gamma))) \cong (0.95, 0.03)$, hence the disease-free steady state is unstable under no vaccination. The endemic steady state means the steady state that infection is maintained in the population. Hence, the deterministic endemic steady state is given by the steady solution of (1) and (2) such that $0 < I_e \leq 1$. It should be noted that the deterministic endemic steady state under no vaccination is stable under no time delay. However, even if there exists time delay, we conjecture that the deterministic endemic steady state under no vaccination is stable because time delay has little effect on the stability from the consideration in the next section. The stability analysis of the endemic steady state is a problem in the future.

Fig. 3 and 4 suggest that Theorems 1 and 2 are useful in the sense that we can estimate the necessary vaccination rate to control the infectious disease spreading.

Next, we consider the effect of the noise on the stability by simulations. We denote the infection rate $\beta$ at which the maximum Lyapunov exponent $\ell$ becomes zero as $\beta(a)$ under the noise strength $\epsilon = a$, 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 $\epsilon = a$.

Setting the same values of parameters as Fig. 2 and choosing the infection rate as $\beta = 0.2$, we performed simulations under the no noise and noise with the strength $\epsilon = 0.1$. Since it follows from the magnified figure (Fig. 5) 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.

We confirm this stabilizing effect of the random noise by numerical simulations. The results of simulations are shown in Figs. 6 and 7. Fig. 6 denotes that the disease-free steady state is stable under the no noise because the infected $I(t)$ converges to zero. In Fig. 7, in order to see in detail the behavior of the infected $I(t)$, we show the time evolution of $I(t)$ only under two different initial conditions of $I(t)$. Fig. 7 means that the disease-free steady state is unstable under the no noise because the infected $I(t)$ converges to the deterministic endemic steady state $I_e \cong 0.006$ independently of the initial condition.

![Fig. 4 Time evolutions of susceptible $S(t)$ and infected $I(t)$ under no vaccination](image)

![Fig. 5 Magnified figure of Fig. 2 in the range of the infection rate $0.18 \leq \beta \leq 0.23$](image)
5.2 The Influence of Time Delay on the Stability

Setting the noise strength $\epsilon = 0.02$, the infection rate $\beta = 0.22$ and the other parameter values are the same as 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 stability of the disease-free steady state is not guaranteed from Theorems 1 and 2, and it is estimated to be unstable from Fig. 5. The results of simulations are shown in Fig. 8. Fig. 8 trends that the infected $I(t)$ converges to the neighborhood of the deterministic endemic steady state $I_e = 0.056$ independently of time delay $h$. Hence, Fig. 8 means that time delay has little influence on the stability.

In order to see the difference of the behavior of infected in detail, we show the magnified figure of Fig. 8 in the time interval $[0, 100]$ in Fig. 9. In the early stage of the time evolution of the infected $I(t)$, although there exists a little effect of time delay on the behavior, the effect of time delay eventually disappears as shown in Fig. 8. Although the result is omitted, in the case where the disease-free steady state is stable, regardless of time delay, the infected $I(t)$ converges to zero. Hence, the effect of time delay eventually disappears as well as the case of the unstable case.

It follows from Lemma 1 that time delay $h$ has no relation with the stability of the deterministic system (25) and (26). Moreover, Theorem 1 and 2 also mean that time delay is independent of the stability of the stochastic system (21) and (22). However, since Theorems 1 and 2 give the one of sufficient condition for the stochastic system to be stable, there is possibility that time delay has reference to stability. Then, by numerical simulations, we have shown that time delay has no relation with stability.

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 time delay on the stability by the numerical simulations. We derived the sufficient condition for the disease-free steady state to be sta-
ble using the 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 can conclude that the random noise considered in this paper shifts the deterministic bifurcation point in the direction of stabilization. In [13], 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 P-bifurcation analysis, 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. Although there exits some effect of time delay on the behavior of infected in the early stage of infection, infected eventually converges to the neighborhood of the deterministic endemic steady state independently of time delay.

From Theorems 1 and 2, we can 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 (42), the disease-free steady state becomes stable, so that the prevalence of the infectious disease is controlled.

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Appendix

Appendix 1. Proof of Theorem 1

It follows from [11,15] that if there exist positive constants $k_0, k_1$ and $k$ such that

\[
(a) \quad k_0 E\{|x(t)|^2\} \leq E\{V_1(t,x_1)\} \leq k_1 |x_1|^2 , \quad (A1)
\]
\[
(b) \quad \frac{\partial V_1(t,x_1)}{\partial t} + L_1 V_1(t,x_1) \leq -k |x(t)|^2 , \quad (A2)
\]

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

Since (10) and (15) easily yield the relation (A1), it suffices for the proof of Theorem 1 to prove (A2).

Setting as $x_i(t) = x_i, (i = 1,2)$, $\bar{x}_2 = x_2(t-h)$, from (23), (24) and (43), we have

\[
\frac{\partial V_1(t,x_1)}{\partial t} + L_1 V_1(t,x_1) = B x_2^2 - B \bar{x}_2^2 + 2x_1 \{ -(\mu + u)x_1 \}
\]
\[
- \beta S_f \bar{x}_2 + 2Ax_2 \{ \beta S_f \bar{x}_2 - (\mu + \gamma)x_2 \} + A e^2 x_2^2
\]
\[
\leq -2(\mu + u) - \beta S_f x_2^2 - 2A(\mu + \gamma) - \beta S_f
\]
\[
- A e^2 - B \} x_2^2 + \{ (1 + A)\beta S_f - B \} x_2^2
\]

(Using (44), we have)

\[
= -2(\mu + u) - \beta S_f x_2^2 - \beta S_f x_2^2 \leq -k |x(t)|^2 ,
\]

where $k = \min \{ 2(\mu + u) - \beta S_f, \beta S_f \} > 0$. (from (42))

Appendix 2. Proof of Theorem 2

If (54) and the relation below hold, the zero solution of (21) and (22) is stable in probability [11,15]:
(c) \( k_0 |x(t)|^2 \leq V_2(t,x_t) \leq k_1 |x_t|^2 \). \hspace{1cm} (A3)

Since (A3) is easily derived from (10) and (18), it suffices for the proof of Theorem 2 to prove (54). It follows from (21), (22), (52) that

\[
\begin{align*}
\frac{\partial V_2(t,x_t)}{\partial t} + L_2 V_2(t,x_t) & = C x_1^2 - C x_2^2 \\
& + 2x_1 \{ -(\mu + u)x_1 - \beta(x_1 + S_f)\bar{x}_2 \} \\
& + 2Ax_2 \{ \beta(x_1 + S_f)\bar{x}_2 - (\mu + \gamma)x_2 \} + Ac^2 \bar{x}_2^2 \hspace{1cm} (A4)
\end{align*}
\]

Noting that from (51), the following relations hold

\[
\begin{align*}
2x_1(x_1 + S_f)\bar{x}_2 & \leq (\delta + S_f)x_1^2 + (\delta + S_f)\bar{x}_2^2 \hspace{1cm} (A5) \\
2x_2(x_1 + S_f)\bar{x}_2 & \leq \delta x_1^2 + (\delta + S_f)x_2^2 + S_f \bar{x}_2^2, \hspace{1cm} (A6)
\end{align*}
\]

(The R.H.S. of (A3)) \( \leq \{ 2(\mu + u) - \beta(\delta + S_f) \\
- A\beta S_f \} x_1^2 - \{ 2A(\mu + \gamma) - Ac^2 - C \\
- A\beta(\delta + S_f) \} \bar{x}_2^2 + \{ (1 + A)\beta S_f + \beta\delta - C \} \bar{x}_2^2
\]

(Using (53), we have)

\[
\begin{align*}
& = - \{ 2(\mu + u) - \beta S_f - \beta(1 + A) \} x_1^2 \\
& - \{ (2\mu + 2\gamma - \epsilon^2 - 2\beta S_f)A - \beta(\delta + S_f) - \beta\delta A \} \bar{x}_2^2
\end{align*}
\]

(Noting (2\(\mu + 2\gamma - \epsilon^2 - 2\beta S_f \)A = 2\(\beta S_f \) from (44), we have)

\[
\begin{align*}
& = - \{ 2(\mu + u) - \beta S_f - \beta(1 + A) \} x_1^2 \\
& - \beta(\epsilon^2 - \delta(1 + A)) \bar{x}_2^2. \hspace{1cm} (A7)
\end{align*}
\]

Hence, for a sufficiently small \( \delta > 0 \), we have (54).

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