ISSN 1392-5113 Nonlinear Analysis: Modelling and Control, 2017, Vol. 22, No. 1, 64–83
http://dx.doi.org/10.15388/NA.2017.1.5

Stability analyses of deterministic and stochastic SEIRI epidemic models with nonlinear incidence rates and distributed delay

Hong Zhang[a,1], **Juan Xia**[a], **Paul Georgescu**[b,2]

[a]Department of Financial Mathematics, Jiangsu University,
Zhenjiang, 212013, China
hongzhang@ujs.edu.cn; 1165469734@qq.com

[b]Department of Mathematics, Technical University of Iaşi,
Bd. Copou 11, 700506 Iaşi, Romania
v.p.georgescu@gmail.com

Received: June 18, 2015 / **Revised:** December 17, 2015 / **Published online:** December 2, 2016

Abstract. In this paper, deterministic and stochastic SEIRI epidemic models featuring a distributed latent period and general, unspecified nonlinear incidence and growth rates for the susceptible class are proposed and investigated from a stability viewpoint. By applying Lyapunov–LaSalle invariance principle, we first obtain sufficient conditions for the global stability of equilibria of the deterministic model. On the basis of this result, we subsequently derive sufficient conditions for asymptotic stability of the stochastic model. Finally, numerical simulations are given to illustrate the previously obtained theoretical framework.

Keywords: compartmental model, delay differential system, nonlinear incidence, disease relapse, global stability of equilibria, Lyapunov functional.

1 Introduction

In many traditional disease propagation models descending from the celebrated ones proposed by Kermack and McKendrick, use is made of three compartments, or classes, namely the class of susceptible individuals (S), the class of infective individuals (I) and the class of recovered individuals (R). For diseases such as influenza, measles or tuberculosis, in which subjects may become infected after an adequate contact with an infective individual without actually being infective during a certain initial period of

[1]The author was supported by the National Natural Science Foundation of China, grant ID 11201187, the Scientific Research Foundation for the Returned Overseas Chinese Scholars and the China Scholarship Council.
[2]The author was supported by a grant of the Romanian National Authority for Scientific Research, CNCS-UEFISCDI, project No. PN-II-ID-PCE-2011-3-0557.

latency, one needs to keep track of the class of exposed (or latent) individuals (E) as well. Assuming that the latent period is exponentially distributed, the subsequent model is represented by a system of ODEs. If, however, the latent period has a general distribution, one obtains a delayed integro-differential system [2, 11].

The process of disease transmission is often described by mass action or standard incidence rates, which rely on simplifying assumptions such as homogeneous mixing, constant contact rates or constant probability of transmitting infection per contact. However, it is often meaningful to consider the effects of behavioral adaptation of individuals to minimize the risk of infection, of the prevention policies and of the saturating contact rates, which altogether lead to a possibly bi-nonlinear incidence rate [5, 10].

For diseases such as herpes, tuberculosis or malaria, recovered individuals may experience a relapse of the disease due to an incomplete treatment or to the reactivation of a latent infection and then reenter the class of infectives. It has been observed in [3] that an individual who is infected with herpes passes throughout successive episodes of latency and relapse all his life. Also, the chance of a relapse happening may be influenced by the presence of concurrent diseases, being observed in [13] that patients with concurrent diabetes suffer worse treatment outcomes and a higher relapse rate during the treatment of tuberculosis. It may even be the case that relapses are more severe than the initial infection. This happens with the varicella-zoster virus (VZV), which is the etiologic agent of both varicella (chicken pox; primary infection) and herpes zoster (shingles; reactivation of latent infection). Even though varicella is extremely contagious, it is usually benign, while the neurologic complications of herpes zoster, including chronic encephalitis and contralateral hemiparesis, are serious, even potentially lethal, in both immunocompetent and immunocompromised individuals. See, for instance, [7]. In this regard, disease propagation models with relapse are investigated from a stability viewpoint in [4, 6]. It is also easily conceivable that the environmental perturbations, coupled with uncertainty factors, may sometimes drastically limit the usefulness of deterministic epidemic models, being necessary to consider the effect of stochastic perturbations, as done, for instance, in [16].

In this paper, we introduce a deterministic SEIRI epidemic model with abstract nonlinear incidence and a general distributed time delay, in which individuals may experience disease relapse, that is, the return of signs and symptoms of a disease after a remission [4]. We express the stability of the disease-free equilibrium and of the endemic equilibrium in terms of a threshold parameter, which governs not only the stability of the equilibria, but also the very existence of the endemic equilibrium. To this purpose, we employ Lyapunov functionals constructed ad hoc. We then discuss the influence of stochastic perturbations of white noise type upon the stability of the system, also by Lyapunov's second method.

The paper is organized as follows. In the next section, we propose our deterministic SEIRI epidemic model, prove its well-posedness from a biological viewpoint by establishing the boundedness and positivity of trajectories, and then discuss the existence of the equilibria together with their stability. In Section 3, we consider a version of our model, which accounts for the effect of stochastic perturbations and prove the respective stochastic asymptotic stability of the disease-free equilibrium and the endemic equilibrium under additional constraints. Finally, we complement our results in Section 4 by means of providing numerical simulations and indicating several concluding remarks.

2 The deterministic SEIRI epidemic model

2.1 The model and its relevance

Let us denote by $\mathbf{P}(t)$ the probability that an individual remains in the exposed class t time units after entering without taking death into account. Let us also suppose that $\mathbf{P}$ satisfies the following biologically motivated assumptions:

(P) $\mathbf{P} : [0,\infty) \to [0,1]$ is nonincreasing and piecewise continuous, possibly with finitely many jumps, and satisfying $\mathbf{P}(0+) = 1$, $\lim_{t\to\infty} \mathbf{P}(t) = 0$ and $0 < \int_0^\infty \mathbf{P}(u)\,\mathrm{d}u < \infty$.

Assuming that the force of infection is expressed by the standard incidence, van den Driessche et al. introduced and discussed in [14] the following SEIRI disease propagation model with relapse:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= \mu - \mu S(t) - \beta S(t) I(t)\\
\frac{\mathrm{d}E}{\mathrm{d}t} &= \beta S(t) I(t) - \mu E(t) + \beta \int_0^t S(\xi) I(\xi) \mathrm{e}^{-\mu(t-\xi)}\,\mathrm{d}_t \mathbf{P}(t-\xi)\,\mathrm{d}\xi,\\
\frac{\mathrm{d}I}{\mathrm{d}t} &= -\beta \int_0^t S(\xi) I(\xi) \mathrm{e}^{-\mu(t-\xi)}\,\mathrm{d}_t \mathbf{P}(t-\xi)\,\mathrm{d}\xi + \delta R(t) - (\mu+\gamma) I(t),\\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \gamma I(t) - (\mu+\delta) R(t),
\end{aligned} \tag{1}$$

In the above model (1), S, E, I and R represent the (rescaled) sizes of the susceptible, exposed, infectious and recovered populations, respectively, μ is the birth (and death) rate, β is the transmission coefficient, γ is the recovery rate of infective individuals and δ is the relapse rate of recovered individuals, which subsequently return to the infective compartment, the integrals being considered in the sense of Riemann–Stieltjes.

Model (1), in its high degree of generality, encompasses several particular cases, which are important on their own. It has been observed in [14] that particularizing $\mathbf{P}(t) = \mathrm{e}^{-\epsilon t}$ leads to an ODE system, while particularizing $\mathbf{P}(t)$ as a step function,

$$\mathbf{P}(t) = \begin{cases} 1, & t \in [0,\tau],\\ 0, & t > \tau, \end{cases}$$

leads to the following delayed system:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= \mu - \mu S(t) - \beta S(t) I(t),\\
\frac{\mathrm{d}E}{\mathrm{d}t} &= \beta S(t) I(t) - \beta \mathrm{e}^{-b\tau} S(t-\tau) I(t-\tau) - \mu E(t),
\end{aligned} \tag{2a}$$

$$\begin{aligned}\frac{\mathrm{d}I}{\mathrm{d}t} &= \beta \mathrm{e}^{-b\tau} S(t-\tau) I(t-\tau) + \delta R(t) - (\mu+\gamma) I(t),\\ \frac{\mathrm{d}R}{\mathrm{d}t} &= \gamma I(t) - (\mu+\delta) R(t),\end{aligned} \tag{2b}$$

whose local stability was then discussed. In [15], Xu discussed a version of (2) with ratio-dependent incidence, replacing the bilinear incidence rate considered in [14], and derived conditions for the global stability of the endemic equilibrium and of the disease-free equilibrium in terms of a threshold parameter via appropriately constructed Lyapunov functionals.

In what follows, motivated by the approach and the results of van den Driessche et al. [14] and Xu [15], and also by the global stability analysis for a related SIRI model without delay performed in [6], we shall consider first the following general deterministic SEIRI model with an abstract nonlinear incidence rate, a distributed latent period and the relapse of recovered individuals:

$$\begin{aligned}\frac{\mathrm{d}S}{\mathrm{d}t} &= n\big(S(t)\big) - f\big(S(t), I(t)\big),\\ \frac{\mathrm{d}E}{\mathrm{d}t} &= f\big(S(t), I(t)\big) - \mu E(t) - \int_0^h Q(\xi) \mathrm{e}^{-\mu\xi} f\big(S(t-\xi), I(t-\xi)\big)\,\mathrm{d}\xi,\\ \frac{\mathrm{d}I}{\mathrm{d}t} &= \int_0^h Q(\xi) \mathrm{e}^{-\mu\xi} f\big(S(t-\xi), I(t-\xi)\big)\,\mathrm{d}\xi - (\mu+\gamma+\alpha) I(t) + \delta R(t),\\ \frac{\mathrm{d}R}{\mathrm{d}t} &= \gamma I(t) - (\mu+\delta) R(t).\end{aligned} \tag{3}$$

In the above model (3), $n(S)$ is the influx of healthy individuals into the susceptible class, while μ and α are natural death rate and the disease-induced death rate, respectively. Also, γ is the recovery rate of the infected population, δ is the relapse rate of recovered individuals, which then became infectious again. The incidence function $f = f(S, I)$ is given in an abstract form, encompassing several classical situations, as it shall be seen below, h is the maximal length of the latent period, that is, the maximal time after which infected individuals become infectious and $Q = -\mathbf{P}'$ assuming implicitly that $\mathbf{P}$ is piecewise C^1.

Throughout this paper, we shall employ the following assumptions (i)–(v), except for the case in which (i) will be substituted by (i′), which shall be explicitly indicated whenever necessary:

(i) n is a continuous function on $\mathbb{R}^+$ such that there exists a $S_0 > 0$ satisfying $n(S_0) = 0$ and $(n(S) - n(S_0))(S - S_0) < 0$ for $0 \leqslant S \neq S_0$.

(ii) f is a locally Lipschitz continuous function on $\mathbb{R}^+ \times \mathbb{R}^+$ satisfying $f(S, 0) = f(0, I) = 0$.

(iii) f is a strictly increasing function of S for fixed I and a strictly increasing function of I for fixed S.

(iv) $\Phi(S,I) \doteq f(S,I)/I$ is a bounded, strictly decreasing function of I for fixed S.
(v) $\kappa(S) \doteq \lim_{I\to 0^+} \Phi(S,I)$ is a continuous and increasing function of S.

When necessary, assumption (i) may be substituted by the following one:

(i′) n is a strictly decreasing continuous function on $\mathbb{R}^+$ such that there exists a $S_0 > 0$ such that $n(S_0) = 0$ and $\lim_{S\to\infty} n(S) = -\infty$,

situation that will be explicitly indicated if it occurs. Let us note that, by (iv) and (v),

$$\Phi(S,I) \leqslant \kappa(S), \quad f(S,I) \leqslant \kappa(S)I \quad \text{for all } I > 0. \tag{4}$$

Essentially, assumption (i) states that n is positive at small densities (below S_0) and negative at higher densities (above S_0). Since the term $-f(S(t),I(t))$, which accompanies $n(S)$ in the right-hand side of the first equation in (3), has negative sign, this assumption is meant to limit the growth of the susceptible class, keeping its size under a maximal value S_0, and is satisfied by both the usual linear and logistic growth rates. Assumption $(i)'$ is somewhat more specialized, excluding the logistic growth rate because of its lack of monotonicity, being used to derive the uniqueness of the endemic equilibrium. The meaning of (ii) is that if there are no susceptibles or no infectives, then obviously there is no disease transmission, while (iii) expresses the fact that increasing the size of the susceptible or of the infective class increases the chance for the occurrence of new infections if the size of the other class is kept constant. Assumption (iv) describes the saturation of the infection rate as the size of the infective class grows larger, while (v) describes the fact that a small number of infectives introduced in a totally susceptible population will produce a certain amount of new infections, which is larger if the initial pool of susceptibles is larger.

Remark 1. Examples of functions n modelling the growth of the susceptible class, which satisfy assumption (i) are the linear growth $n(S) = \Lambda - \mu S$, where Λ is a positive constant, and the combination of the linear growth and the logistic growth $n(S) = \alpha(\Lambda - \mu S) + \zeta(rS(1 - S/K))$, in which r is the intrinsic growth rate, K is the carrying capacity, α and ζ are positive constants, although only the first example satisfies assumption (i′) as well without additional conditions.

Remark 2. In model (3), the death phenomena are assumed to follow a linear pattern, highlighted through the use of the terms $-\mu E(t)$, $-(\mu + \alpha)I(t)$ and $-\mu R(t)$ to represent death-related losses from the exposed, infective and recovered class, respectively. However, the recruitment of new susceptibles, may follow a pattern, which is essentially nonlinear. This happens since the recruitment of new susceptibles is subject to a host of external factors beyond biology (behavioral, as quantified by individual risk-taking or risk-avoidance strategies, informational, as represented by the media coverage of the disease, which may in turn have behavioral influence, medical, as given by the availability of vaccines, which may act to decrease the pool of new susceptibles, and not only).

Remark 3. Examples of incidence functions f satisfying assumptions (ii)–(v) include the bilinear incidence, the saturated incidence $\beta SI/(d + S + I)$, the modified saturated

incidence $\beta SI/(1+\alpha_1 S+\alpha_2 I)$, the standard incidence, and other common incidence functions such as $\beta SI/((S+A_S)(I+A_I))$, in which A_S and A_I are positive constants, and $\beta S^p I^q$ ($p>0$, $0<q<1$).

The initial conditions are assumed to be of the form

$$S(\theta)=\varphi_1(\theta),\quad E(\theta)=\varphi_2(\theta),\quad I(\theta)=\varphi_3(\theta),\quad R(\theta)=\varphi_4(\theta),\quad \theta\in[-h,0],$$

where $\varphi_1,\varphi_2,\varphi_3,\varphi_4\in \mathrm{C}([-h,0],\mathbb{R}^+)$, the Banach space of continuous functions mapping the interval $[-h,0]$ into $\mathbb{R}^+$, endowed with the sup-norm, such that $\varphi_1(0)>0$, $\varphi_3(0)>0$. The existence and uniqueness of the solutions of systems (3) then follow from standard results in the theory of delay differential equations (see, for instance, [9]).

2.2 The well-posedness

A first step towards establishing the adequacy of model (3) is to prove that its trajectories are bounded and positivity-preserving.

Lemma 1. *The solutions of system* (3) *are ultimately uniformly bounded, a feasible region being*

$$\Omega=\left\{(S,E,I,R)\;\middle|\; 0\leqslant S\leqslant S_0,\ 0\leqslant S+E+I+R\leqslant\frac{2\overline{n}}{\overline{d}}\right\},$$

where

$$\overline{n}=\sup_{0\leqslant S\leqslant S_0} n(S)\quad \textit{and}\quad \overline{d}=\min\left\{\mu,\frac{\overline{n}}{S_0}\right\}.$$

Proof. By (i), it is easy to see that $S(t)\in[0,S_0]$ for all $t\geqslant 0$ provided that $S(0)\in[0,S_0]$. Further, summing up all equations in (3) gives

$$\frac{\mathrm{d}}{\mathrm{d}t}(S+E+I+R)\leqslant 2\overline{n}-\overline{d}(S+E+I+R).$$

Hence,

$$\limsup_{t\to\infty}(S+E+I+R)\leqslant\frac{2\overline{n}}{\overline{d}},$$

which completes the proof. □

To advance our well-posedness discussion, we observe that the first, third and fourth equations of system (3) do not refer to the class E, which means that we could simplify system (3) to the following lower-dimensional one:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t}&=n(S)-f(S,I),\\
\frac{\mathrm{d}I}{\mathrm{d}t}&=\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}f\big(S(t-\xi),I(t-\xi)\big)\,\mathrm{d}\xi-(\mu+\gamma+\alpha)I+\delta R,\\
\frac{\mathrm{d}R}{\mathrm{d}t}&=\gamma I-(\mu+\delta)R.
\end{aligned}\tag{5}$$

However, note that it is not possible to prove the boundedness of the solutions of (5) directly due to the absence of the balancing negative integral term. This is why we had to consider the dynamics of the "complete" system (3) first. By an argument similar to the one employed in [14, Lemma 2.1] (see also [8]), one obtains the following biological well-posedness result.

Lemma 2. *The solutions of system* (5) *are nonnegative.*

2.3 The existence of the equilibria

In what follows, we shall establish the global dynamics of system (5), assuming that (i′) holds instead of (i), as (i) may not be enough to ensure the uniqueness of the endemic equilibrium.

System (5) always has a disease-free equilibrium $E_0 = (S_0, 0, 0)$. To discuss the existence of a positive equilibrium (and subsequently its stability), let us define the basic reproduction number of (5) by

$$\mathcal{R}_0 \doteq \frac{(\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi)\kappa(S_0)}{\mu+\gamma+\alpha-\frac{\delta\gamma}{\mu+\delta}}.$$

Theorem 1. *If* $\mathcal{R}_0 > 1$*, then system* (5) *admits a unique endemic equilibrium* $E^* = (S^*, I^*, R^*)$.

Proof. For the existence of the endemic equilibrium E^* to hold, one sees that the following equilibrium conditions need to be satisfied:

$$n(S^*) = \frac{(\mu+\gamma+\alpha-\frac{\delta\gamma}{\mu+\delta})I^*}{\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi}, \qquad \frac{f(S^*,I^*)}{I^*} - \frac{\mu+\gamma+\alpha-\frac{\delta\gamma}{\mu+\delta}}{\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi} = 0.$$

Note that these conditions can be restated as

$$n(S^*) = \frac{\kappa(S_0)}{\mathcal{R}_0}I^*, \qquad \Phi(S^*,I^*) - \frac{\kappa(S_0)}{\mathcal{R}_0} = 0,$$

the first one being equivalent to

$$S^* = n^{-1}\left(\frac{\kappa(S_0)}{\mathcal{R}_0}I^*\right),$$

where n^{-1} is the inverse function of n. To determine I^* via a continuity argument and having the previous equalities as motivations, we define

$$F(I) \doteq \Phi\left(n^{-1}\left(\frac{\kappa(S_0)}{\mathcal{R}_0}I\right), I\right) - \frac{\kappa(S_0)}{\mathcal{R}_0}. \tag{6}$$

It follows from assumptions (i′) and (iv) that F is strictly monotone decreasing on $[0, n(0)\mathcal{R}_0/\kappa(S_0)]$. Also, from assumption (ii),

$$F\left(\frac{n(0)\mathcal{R}_0}{\kappa(S_0)}\right) = \Phi\left(n^{-1}\big(n(0)\big), \frac{n(0)\mathcal{R}_0}{\kappa(S_0)}\right) - \frac{\kappa(S_0)}{\mathcal{R}_0} < 0. \tag{7}$$

Let now $S_1 \in (0, S_0)$ be arbitrary. Since

$$\lim_{I\to 0+} n^{-1}\left(\frac{\kappa(S_0)}{\mathcal{R}_0} I\right) = n^{-1}(0) = S_0,$$

it follows that there is $\varepsilon > 0$ such that

$$n^{-1}\left(\frac{\kappa(S_0)}{\mathcal{R}_0} I\right) > S_1 \quad \text{for } I \in (0, \varepsilon).$$

By assumption (iii), it follows that

$$F(I) > \Phi(S_1, I) - \frac{\kappa(S_0)}{\mathcal{R}_0} \quad \text{for } I \in (0, \varepsilon),$$

which implies that

$$\lim_{I\to 0^+} F(I) \geqslant \lim_{I\to 0^+} \Phi(S_1, I) - \frac{\kappa(S_0)}{\mathcal{R}_0} = \kappa(S_1) - \frac{\kappa(S_0)}{\mathcal{R}_0}.$$

Since $S_1 \in (0, S_0)$ was arbitrary, it follows from assumption (v) that

$$\lim_{I\to 0^+} F(I) \geqslant \kappa(S_0) - \frac{\kappa(S_0)}{\mathcal{R}_0} > 0. \tag{8}$$

Although the converse inequality is not essential to our argument, it also follows from (4) and (6) that

$$F(I) \leqslant \kappa\left(n^{-1}\left(\frac{\kappa(S_0)}{\mathcal{R}_0} I\right)\right) - \frac{\kappa(S_0)}{\mathcal{R}_0},$$

which leads to

$$\lim_{I\to 0^+} F(I) \leqslant \kappa(S_0) - \frac{\kappa(S_0)}{\mathcal{R}_0}. \tag{9}$$

From (8) and (9), it then follows that

$$\lim_{I\to 0^+} F(I) = \kappa(S_0) - \frac{\kappa(S_0)}{\mathcal{R}_0} > 0. \tag{10}$$

From (7) and (10) (or (8)), together with the continuity and strict monotonicity of F, there exists a unique $I^* \in (0, n(0)\mathcal{R}_0/\kappa(S_0))$ such that $F(I^*) = 0$, the corresponding S^* and I^* being uniquely defined by

$$S^* = n^{-1}\left(\frac{\kappa(S_0)}{\mathcal{R}_0} I^*\right), \quad R^* = \frac{\gamma I^*}{\mu + \delta}.$$

This concludes the existence and uniqueness of the endemic equilibrium E^*. □

2.4 The stability of the equilibria

After having determined the existence of the equilibria for system (5), we now turn our attention to the study of their stability. We start with the stability of E_0, which will be proved via the use of the Lyapunov–LaSalle invariance principle.

Theorem 2. *If $\mathcal{R}_0 \leqslant 1$, then the disease-free equilibrium E_0 is globally asymptotically stable.*

Proof. Let us construct the Lyapunov functional

$$V(t) = \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\left[\int_{S_0}^{S(t-\xi)}\left(1-\frac{\kappa(S_0)}{\kappa(s)}\right)\mathrm{d}s\right]\mathrm{d}\xi + I + \frac{\delta}{\mu+\delta}R$$
$$+ \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\left(\int_{t-\xi}^{t}\frac{\kappa(S_0)}{\kappa\big(S(s)\big)}f\big(S(s),I(s)\big)\,\mathrm{d}s\right)\mathrm{d}\xi.$$

The derivative of V along the solutions of (5) reads then as

$$\frac{\mathrm{d}V(t)}{\mathrm{d}t} = \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\left(1-\frac{\kappa(S_0)}{\kappa\big(S(t-\xi)\big)}\right)\big(n\big(S(t-\xi)\big) - f\big(S(t-\xi),I(t-\xi)\big)\big)\,\mathrm{d}\xi$$
$$+ \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}f\big(S(t-\xi),I(t-\xi)\big)\,\mathrm{d}\xi - (\mu+\alpha+\gamma)I + \frac{\gamma\delta}{\mu+\delta}I$$
$$+ \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\left[\frac{\kappa(S_0)}{\kappa(S(t))}f\big(S(t),I(t)\big) - \frac{\kappa(S_0)}{\kappa(S(t-\xi))}f\big(S(t-\xi),I(t-\xi)\big)\right]\mathrm{d}\xi.$$

By direct computations, one obtains that

$$\frac{\mathrm{d}V(t)}{\mathrm{d}t} = \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}n\big(S(t-\xi)\big)\left(1-\frac{\kappa(S_0)}{\kappa(S(t-\xi))}\right)\mathrm{d}\xi$$
$$- \left(\mu+\gamma+\alpha-\frac{\delta\gamma}{\mu+\delta}\right)I(t)$$
$$+ \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi\,\frac{\kappa(S_0)}{\kappa(S(t))}f\big(S(t),I(t)\big). \tag{11}$$

From the assumptions (i) and (v), it is seen that, for all $t \geqslant 0$,

$$n\big(S(t-\xi)\big)\left(1-\frac{\kappa(S_0)}{\kappa(S(t-\xi))}\right) = \big(n\big(S(t-\xi)\big)-n(S_0)\big)\left(1-\frac{\kappa(S_0)}{\kappa(S(t-\xi))}\right) \leqslant 0. \tag{12}$$

Using (4), it follows that

$$\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\frac{\kappa(S_0)}{\kappa(S(t))}f\big(S(t),I(t)\big)\,\mathrm{d}\xi \leqslant \left(\mu+\gamma+\alpha-\frac{\delta\gamma}{\mu+\delta}\right)I(t)\mathcal{R}_0. \tag{13}$$

From (11), (12) and (13), one then obtains that

$$\frac{\mathrm{d}V(t)}{\mathrm{d}t} \leqslant \left(\mu+\gamma+\alpha-\frac{\delta\gamma}{\mu+\delta}\right)I(t)(\mathcal{R}_0-1) \leqslant 0.$$

Also, one notes that $\{E_0\}$ is the largest invariant set in $\{(S,I,R) \mid \mathrm{d}V(t)/\mathrm{d}t = 0\}$. Finally, by applying Lyapunov–LaSalle invariance principle, we obtain that E_0 is globally asymptotically stable. □

In what follows, we shall observe that the inequality $\mathcal{R}_0 > 1$ ensures not only the existence of the endemic equilibrium E^* (which has already been proved), but also its global stability, using an approach similar to the one employed above.

Theorem 3. *If $\mathcal{R}_0 > 1$, then the endemic equilibrium E^* is globally asymptotically stable.*

Proof. To prove the global stability of the endemic equilibrium E^*, let us use the Lyapunov functional V defined by

$$V(t) = V_1(t) + V_2(t) + V_3(t) + V_4(t)$$

with

$$V_1(t) = \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi\left(S(t)-S^*-\int_{S^*}^{S(t)}\frac{f(S^*,I^*)}{f(s,I^*)}\,\mathrm{d}s\right),$$

$$V_2(t) = I(t)-I^*-I^*\ln\frac{I(t)}{I^*},$$

$$V_3(t) = \frac{\delta}{\mu+\delta}\left(R(t)-R^*-R^*\ln\frac{R(t)}{R^*}\right),$$

$$V_4(t) = \int_0^h\left[\int_{t-\xi}^t Q(\xi)\mathrm{e}^{-\mu\xi}\left(f\big(S(s),I(s)\big)-f(S^*,I^*)-f(S^*,I^*)\ln\frac{f\big(S(s),I(s)\big)}{f(S^*,I^*)}\right)\mathrm{d}s\right]\mathrm{d}\xi.$$

Computing the time derivatives of V_1, V_2, V_3, V_4 along the solutions of (5) yields

$$\begin{aligned}
\frac{\mathrm{d}V_1(t)}{\mathrm{d}t} &= \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi\left(1-\frac{f(S^*,I^*)}{f(S(t),I^*)}\right)\big(n\big(S(t)\big)-f\big(S(t),I(t)\big)\big),\\
\frac{\mathrm{d}V_2(t)}{\mathrm{d}t} &= \left(1-\frac{I^*}{I(t)}\right)\left(\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}f\big(S(t-\xi),I(t-\xi)\big)\,\mathrm{d}\xi\right.\\
&\quad\left.+\delta R(t)-(\mu+\alpha+\gamma)I(t)\right),\\
\frac{\mathrm{d}V_3(t)}{\mathrm{d}t} &= \frac{\delta}{\mu+\delta}\left(1-\frac{R^*}{R(t)}\right)\big(\gamma I(t)-(\mu+\delta)R(t)\big),\\
\frac{\mathrm{d}V_4(t)}{\mathrm{d}t} &= \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\left[f\big(S(t),I(t)\big)-f\big(S(t-\xi),I(t-\xi)\big)\right.\\
&\quad\left.+f(S^*,I^*)\ln\frac{f\big(S(t-\xi),I(t-\xi)\big)}{f(S(t),I(t))}\right]\mathrm{d}\xi.
\end{aligned}$$

By using the above expressions of the derivatives, cancelling similar terms and rearranging the remaining ones, it follows that

$$\begin{aligned}
\frac{\mathrm{d}V(t)}{\mathrm{d}t} &= \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi\left(1-\frac{f(S^*,I^*)}{f(S(t),I^*)}\right)n\big(S(t)\big)\\
&\quad+\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi\,\frac{f(S^*,I^*)}{f(S(t),I^*)}f\big(S(t),I(t)\big)+\delta R^*\\
&\quad-\frac{I^*}{I(t)}\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}f\big(S(t-\xi),I(t-\xi)\big)\,\mathrm{d}\xi-\frac{\delta\gamma}{\mu+\delta}\frac{R^*}{R(t)}I(t)\\
&\quad-\left[(\mu+\alpha+\gamma)-\frac{\delta\gamma}{\mu+\delta}\right]I(t)-\frac{I^*}{I(t)}\delta R(t)+(\mu+\alpha+\gamma)I^*\\
&\quad+\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}f(S^*,I^*)\ln\frac{f(S(t-\xi),I(t-\xi))}{f(S(t),I(t))}\,\mathrm{d}\xi. \qquad (14)
\end{aligned}$$

Let us denote

$$\begin{aligned}
T &= -\left[(\mu+\alpha+\gamma)-\frac{\delta}{\mu+\delta}\right]I(t)-\frac{I^*}{I(t)}\delta R(t)\\
&\quad+(\mu+\alpha+\gamma)I^*-\frac{\delta\gamma}{\mu+\delta}\frac{R^*}{R(t)}I(t)+\delta R^*.
\end{aligned}$$

Using the equilibrium relations

$$(\mu+\alpha+\gamma)I^* - \frac{\delta}{\mu+\delta}\gamma I^* = f(S^*,I^*)\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi,$$
$$\gamma I^* = (\mu+\delta)R^*,$$

it follows that

$$T = \left(1-\frac{I(t)}{I^*}\right)f(S^*,I^*)\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi - \delta R^*(t)\left(\sqrt{\frac{I^*}{I(t)}\frac{R(t)}{R^*}} - \sqrt{\frac{R^*}{R(t)}\frac{I(t)}{I^*}}\right)^2. \tag{15}$$

Consequently, using (14), (15) and the equilibrium relation $n(S^*) = f(S^*,I^*)$, it is seen that

$$\begin{aligned}\frac{\mathrm{d}V(t)}{\mathrm{d}t} &= \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi\left(1-\frac{f(S^*,I^*)}{f(S(t),I^*)}\right)\big(n(S(t)-n(S^*)\big)\\ &\quad + f(S^*,I^*)\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi\left(\frac{f(S(t),I(t))}{f(S(t),I^*)} - \frac{I(t)}{I^*} + \frac{I(t)f(S(t),I^*)}{I^*f(S(t),I(t))} - 1\right)\\ &\quad - f(S^*,I^*)\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi\left(\frac{I(t)f(S(t),I^*)}{I^*f(S(t),I(t))} - 1\right)\\ &\quad - f(S^*,I^*)\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi\left(\frac{f(S^*,I^*)}{f(S(t),I^*)} - 1\right)\\ &\quad - f(S^*,I^*)\left[\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\left(\frac{I^*f\big(S(t-\xi),I(t-\xi)\big)}{I(t)f(S^*,I^*)} - 1\right.\right.\\ &\quad \left.\left. + \ln\frac{f(S(t-\xi),I(t-\xi))}{f(S(t),I(t))}\right)\mathrm{d}\xi\right] - \delta R^*(t)\left(\sqrt{\frac{I^*}{I(t)}\frac{R(t)}{R^*}} - \sqrt{\frac{R^*}{R(t)}\frac{I(t)}{I^*}}\right)^2\\ &= T_1+T_2+T_3+T_4+T_5+T_6.\end{aligned} \tag{16}$$

To estimate $\mathrm{d}V(t)/\mathrm{d}t$, we shall employ the inequality

$$1 - x + \ln x \leqslant 0 \quad \text{for all } x > 0 \tag{17}$$

with equality if and only if $x = 1$. Since

$$\ln \frac{f\big(S(t-\xi), I(t-\xi)\big)}{f(S(t), I(t))} = \ln \frac{I(t) f(S(t), I^*)}{I^* f(S(t), I(t))} + \ln \frac{f(S^*, I^*)}{f(S(t), I^*)} + \ln \frac{I^* f\big(S(t-\xi), I(t-\xi)\big)}{I(t) f(S^*, I^*)},$$

it is seen that

$$\begin{aligned} &T_3 + T_4 + T_5 \\ &\quad = -f(S^*, I^*) \int_0^h Q(\xi) \mathrm{e}^{-\mu\xi} \,\mathrm{d}\xi \left[\frac{I(t) f(S(t), I^*)}{I^* f(S(t), I(t))} - 1 + \ln \frac{I(t) f(S(t), I^*)}{I^* f(S(t), I(t))} \right] \\ &\qquad - f(S^*, I^*) \int_0^h Q(\xi) \mathrm{e}^{-\mu\xi} \,\mathrm{d}\xi \left[\frac{f(S^*, I^*)}{f(S(t), I^*)} - 1 + \ln \frac{f(S^*, I^*)}{f(S(t), I^*)} \right] \\ &\qquad - f(S^*, I^*) \Bigg[\int_0^h Q(\xi) \mathrm{e}^{-\mu\xi} \bigg(\frac{I^* f(S(t-\xi), I(t-\xi))}{I(t) f(S^*, I^*)} - 1 \\ &\qquad + \ln \frac{I^* f(S(t-\xi), I(t-\xi))}{I(t) f(S^*, I^*)} \bigg) \,\mathrm{d}\xi \Bigg]. \end{aligned} \tag{18}$$

Also, a rearrangement of terms yields

$$T_2 = -f(S^*, I^*) \int_0^h Q(\xi) \mathrm{e}^{-\mu\xi} \,\mathrm{d}\xi \left(1 - \frac{f(S(t), I(t))}{f(S(t), I^*)} \right) \left(1 - \frac{\Phi(S(t), I^*)}{\Phi(S(t), I(t))} \right). \tag{19}$$

From (16), (18) and (19), one obtains that

$$\begin{aligned} \frac{\mathrm{d}V(t)}{\mathrm{d}t} &= \int_0^h Q(\xi) \mathrm{e}^{-\mu\xi} \,\mathrm{d}\xi \left(1 - \frac{f(S^*, I^*)}{f(S(t), I^*)} \right) \big(n\big(S(t) - n(S^*)\big) \\ &\quad - f(S^*, I^*) \int_0^h Q(\xi) \mathrm{e}^{-\mu\xi} \,\mathrm{d}\xi \left(1 - \frac{f(S(t), I(t))}{f(S(t), I^*)} \right) \left(1 - \frac{\Phi(S(t), I^*)}{\Phi(S(t), I(t))} \right) \\ &\quad - f(S^*, I^*) \int_0^h Q(\xi) \mathrm{e}^{-\mu\xi} \,\mathrm{d}\xi \left[\frac{I(t) f(S(t), I^*)}{I^* f(S(t), I(t))} - 1 + \ln \frac{I(t) f(S(t), I^*)}{I^* f(S(t), I(t))} \right] \end{aligned}$$

$$
\begin{aligned}
&- f(S^*, I^*) \int_0^h Q(\xi) \mathrm{e}^{-\mu\xi} \,\mathrm{d}\xi \left[\frac{f(S^*, I^*)}{f(S(t), I^*)} - 1 + \ln \frac{f(S^*, I^*)}{f(S(t), I^*)} \right] \\
&- f(S^*, I^*) \left[\int_0^h Q(\xi) \mathrm{e}^{-\mu\xi} \left(\frac{I^* f\big(S(t-\xi), I(t-\xi)\big)}{I(t) f(S^*, I^*)} \right.\right. \\
&\left.\left. - \ln \frac{I^* f(S(t-\xi), I(t-\xi))}{I(t) f(S^*, I^*)} \right) \mathrm{d}\xi \right] - \delta R^*(t) \left(\sqrt{\frac{I^*}{I(t)} \frac{R(t)}{R^*}} - \sqrt{\frac{R^*}{R(t)} \frac{I(t)}{I^*}} \right)^2.
\end{aligned}
$$

Since f is strictly increasing and n is strictly decreasing, it is seen that

$$
\left(1 - \frac{f(S^*, I^*)}{f(S, I^*)} \right) \big(n(S) - n(S^*) \big) \leqslant 0
$$

with equality if and only if $S = S^*$. Also, since Φ is strictly decreasing,

$$
-f(S^*, I^*) \left(1 - \frac{f(S, I)}{f(S, I^*)} \right) \left(1 - \frac{\Phi(S, I^*)}{\Phi(S, I)} \right) \leqslant 0
$$

with equality if and only if $I = I^*$. In addition, it follows from (17) that $\mathrm{d}V(t)/\mathrm{d}t \leqslant 0$ for all $t \geqslant 0$, and the equality holds only at the the endemic equilibrium E^*. By Lyapunov–LaSalle invariance principle, E^* is globally asymptotically stable, which completes the proof. □

3 A stochastic SIRI epidemic model

3.1 The model

We assume that system (5) is subject to stochastic perturbations of white noise type, which are directly proportional to the distances between $S(t)$, $I(t)$ and $R(t)$ and their respective equilibrium values $\hat{S}$, $\hat{I}$ and $\hat{R}$. Here $\hat{E} = (\hat{S}, \hat{I}, \hat{R})$ represents a generic equilibrium of (5), that is, $\hat{E}$ may be either the endemic equilibrium E^* or the disease-free equilibrium E_0.

After the introduction of white noise perturbations, system (5) takes the following stochastic form:

$$
\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= n(S) - f(S, I) + \sigma_1 (S - \hat{S}) \frac{\mathrm{d}B_1}{\mathrm{d}t}, \\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \int_0^h Q(\xi) \mathrm{e}^{-\mu\xi} f\big(S(t-\xi), I(t-\xi)\big) \,\mathrm{d}\xi - (\mu + \gamma + \alpha) I \\
&\quad + \delta R + \sigma_2 (I - \hat{I}) \frac{\mathrm{d}B_2}{\mathrm{d}t}, \\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \gamma I - (\mu + \delta) R + \sigma_3 (R - \hat{R}) \frac{\mathrm{d}B_3}{\mathrm{d}t},
\end{aligned}
\tag{20}
$$

where B_1, B_2 and B_3 are independent standard Brownian motions defined on a complete probability space $(\Omega, \mathcal{F}, \mathbf{P})$ and σ_i^2 represent the respective intensities of B_i, $i = 1, 2, 3$. It is easy to see that the equilibrium $\hat{E}$ of the deterministic system (5) is still an equilibrium for the stochastic system (20). We shall now study the stability of the disease-free equilibrium E_0 and of the endemic equilibrium E^* of (20), respectively, again by constructing appropriate Lyapunov functionals.

3.2 A stochastic stability analysis

Let us consider the nth-dimensional stochastic functional differential equation

$$\mathrm{d}\mathcal{X} = \mathfrak{f}(\mathcal{X}_t, t)\,\mathrm{d}t + \mathfrak{g}(\mathcal{X}_t, t)\,\mathrm{d}\mathcal{B}(t) \tag{21}$$

with initial condition $\mathcal{X}(t_0) = \mathcal{X}_0 \in \mathrm{C}^+([-h, 0], \mathbb{R}^n)$, the space of continuous functions from $[-h, 0]$ to $\mathbb{R}^n$ with norm $\|\psi\| = \sup_{\theta\in[-h,0]} |\psi(\theta)|$.

Suppose that (21) admits the trivial solution. Also, let $\mathrm{C}^{2,1}(\mathbb{R}^n \times [t_0, \infty); \mathbb{R}^+)$ be the family of all nonnegative functions $V(\mathcal{X}, t)$ defined on $\mathbb{R}^n \times [t_0, \infty)$, which are continuously differentiable, twice in $\mathcal{X}$ and once in t. Define the differential operator L associated with (21) by

$$L = \frac{\partial}{\partial t} + \sum_{i=1}^{n} \mathfrak{f}_i\big(\mathcal{X}(t-\xi), t\big)\frac{\partial}{\partial \mathcal{X}_i} + \frac{1}{2}\sum_{i,j=1}^{n} \big[\mathfrak{g}^{\mathrm{T}}\big(\mathcal{X}(t-\xi), t\big)\mathfrak{g}\big(\mathcal{X}(t-\xi), t\big)\big]\frac{\partial^2}{\partial \mathcal{X}_i \partial \mathcal{X}_j}.$$

Definition 1.

(i) The trivial solution of (21) is said to be stochastically stable or stable in probability if for every pair of $\epsilon \in (0, 1)$ and $r > 0$, there exists a $\hat{\delta} = \hat{\delta}(\epsilon, r, t_0)$ such that $\mathbf{P}\{|\mathcal{X}(t; t_0, \mathcal{X}_0)| < r \text{ for all } t \geqslant t_0\} \geqslant 1 - \epsilon$ whenever $|\mathcal{X}_0| < \hat{\delta}$. Otherwise, the trivial solution of it is said to be stochastically unstable.

(ii) The trivial solution of (21) is said to be stochastically asymptotically stable if it is stochastically stable and, moreover, for every $\epsilon \in (0, 1)$, there exists a $\hat{\delta} = \hat{\delta}(\epsilon, t_0)$ such that $\mathbf{P}\{\lim_{t\to\infty} \mathcal{X}(t; t_0, \mathcal{X}_0) = 0\} \geqslant 1 - \epsilon$ whenever $|\mathcal{X}_0| < \hat{\delta}$.

Definition 2. A continuous non-negative function $V(\mathcal{X}, t)$ is said to be decrescent if, for some $\nu \in \vartheta$, $V(\mathcal{X}, t) \leqslant \nu(|\mathcal{X}|)$ for all $\mathrm{C}^+([-h, 0], \mathbb{R}^n) \times [t_0, \infty)$, where ϑ denotes the family of all continuous nondecreasing functions ν: $\mathbb{R}^+ \to \mathbb{R}^+$ such that $\nu(0) = 0$ and $\nu(s) > 0$ if $s > 0$.

Lemma 3. *If there exists a positive-definite decrescent function $V(\mathcal{X}, t) \in \mathrm{C}^{2,1}(\mathbb{R}^n \times [t_0, \infty); \mathbb{R}^+)$ such that $LV(\mathcal{X}, t)$ is negative-definite, then the trivial solution of* (21) *is stochastically asymptotically stable.*

Let us now consider the stability of $\hat{E}$. By applying the variable change

$$x = S - \hat{S}, \qquad y = I - \hat{I}, \qquad z = R - \hat{R},$$

system (20) can be restated as

$$\begin{aligned}
\frac{\mathrm{d}x}{\mathrm{d}t} &= n(x+\hat{S}) - f(x+\hat{S}, y+\hat{I}) + \sigma_1 x \frac{\mathrm{d}B_1}{\mathrm{d}t}, \\
\frac{\mathrm{d}y}{\mathrm{d}t} &= \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi} f\big(x(t-\xi)+\hat{S}, y(t-\xi)+\hat{I}\big)\,\mathrm{d}\xi \\
&\quad - (\mu+\gamma+\alpha)(y+\hat{I}) + \delta(z+\hat{R}) + \sigma_2 y \frac{\mathrm{d}B_2}{\mathrm{d}t}, \\
\frac{\mathrm{d}z}{\mathrm{d}t} &= \gamma y - (\mu+\delta)z + \sigma_3 z \frac{\mathrm{d}B_3}{\mathrm{d}t},
\end{aligned} \tag{22}$$

for which the null solution is now of interest. For the sake of notational simplicity, let us denote

$$f_{\hat{S}} = \frac{\partial f}{\partial S}(\hat{S}, \hat{I}), \qquad \overline{Q} = \int_0^h Q(\xi)\,\mathrm{d}\xi,$$

$$f_{\hat{I}} = \frac{\partial f}{\partial I}(\hat{S}, \hat{I}), \qquad \overline{Q}_\mu = \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\,\mathrm{d}\xi.$$

Consider the following linearized version of (22):

$$\begin{aligned}
\frac{\mathrm{d}x}{\mathrm{d}t} &= n'(\hat{S})x - f_{\hat{S}}x - f_{\hat{I}}y + \sigma_1 x \frac{\mathrm{d}B_1}{\mathrm{d}t}, \\
\frac{\mathrm{d}y}{\mathrm{d}t} &= \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\big(f_{\hat{S}}x(t-\xi) + f_{\hat{I}}y(t-\xi)\big)\,\mathrm{d}\xi \\
&\quad - (\mu+\gamma+\alpha)y + \delta z + \sigma_2 y \frac{\mathrm{d}B_2}{\mathrm{d}t}, \\
\frac{\mathrm{d}z}{\mathrm{d}t} &= \gamma y - (\mu+\delta)z + \sigma_3 z \frac{\mathrm{d}B_3}{\mathrm{d}t},
\end{aligned} \tag{23}$$

Theorem 4. *Let n is differentiable and the following conditions hold*:

(i) $\sigma_1^2 < f_{\hat{S}} - 2n'(\hat{S}) - f_{\hat{I}}$;
(ii) $\sigma_2^2 < 2(\mu+\alpha) + \gamma - \delta - (f_{\hat{S}} + f_{\hat{I}})\overline{Q}_\mu\overline{Q} - 2f_{\hat{I}}$;
(iii) $\sigma_3^2 < 2\mu + \delta - \gamma$;

then the null solution of (23) *is stochastically asymptotically stable.*

Proof. Let us define

$$U = U_1 + U_2 + U_3 \quad \text{with} \quad U_1 = x^2,\ U_2 = y^2,\ U_3 = z^2.$$

It follows that

$$LU_1 = 2x\big[n'(\hat{S})x - f_{\hat{S}}x - f_{\hat{I}}y\big] + \sigma_1^2 x^2, \qquad LU_3 = 2z\big[\gamma y - (\mu+\delta)z\big] + \sigma_3^2 z^2,$$
$$LU_2 = 2y\left[\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}\big(f_{\hat{S}}x(t-\xi) + f_{\hat{I}}y(t-\xi)\big)\,\mathrm{d}\xi - (\mu+\gamma+\alpha)y + \delta z\right] + \sigma_2^2 y^2.$$

Consequently, one has

$$\begin{aligned} LU = {} & x^2\big[2(n'(\hat{S}) - f_{\hat{S}}) + \sigma_1^2\big] + y^2\big[-2(\mu+\gamma+\alpha) + \sigma_2^2\big] \\ & + z^2\big[-2(\mu+\delta) + \sigma_3^2\big] - 2f_{\hat{I}}xy + 2(\delta+\gamma)yz \\ & + 2\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}x(t-\xi)\,\mathrm{d}\xi \cdot f_{\hat{S}}y + 2\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}y(t-\xi)\,\mathrm{d}\xi \cdot f_{\hat{I}}y. \end{aligned}$$

Note also that

$$2\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}x(t-\xi)\,\mathrm{d}\xi \cdot f_{\hat{S}}y \leqslant k_1 y^2 + \frac{1}{k_1}f_{\hat{S}}^2\left(\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}x(t-\xi)\,\mathrm{d}\xi\right)^2$$
$$2\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}y(t-\xi)\,\mathrm{d}\xi \cdot f_{\hat{I}}y \leqslant k_2 y^2 + \frac{1}{k_2}f_{\hat{I}}^2\left(\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}y(t-\xi)\,\mathrm{d}\xi\right)^2,$$

where k_1, $k_2 > 0$ are to be chosen later on. To further estimate the integral terms, one obtains using the integral version of the Cauchy–Schwarz inequality that

$$\left(\int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}x(t-\xi)\,\mathrm{d}\xi\right)^2 \leqslant \overline{Q}_\mu \int_0^h Q(\xi)\mathrm{e}^{-\mu\xi}x^2(t-\xi)\,\mathrm{d}\xi,$$

a similar inequality holding with y in place of x. Let us now define

$$W = U + U_4,$$
$$U_4 = \frac{1}{k_1}f_{\hat{S}}^2\overline{Q}_\mu \int_0^h Q(\xi)\int_{t-\xi}^t x^2(\tau)\,\mathrm{d}\tau\,\mathrm{d}\xi + \frac{1}{k_2}f_{\hat{I}}^2\overline{Q}_\mu \int_0^h Q(\xi)\int_{t-\xi}^t y^2(\tau)\,\mathrm{d}\tau\,\mathrm{d}\xi.$$

Then

$$\begin{aligned} LW \leqslant {} & x^2\left[2(n'(\hat{S}) - f_{\hat{S}}) + \sigma_1^2 + \frac{1}{k_1}f_{\hat{S}}^2\overline{Q}_\mu\overline{Q} + f_{\hat{I}}\right] \\ & + y^2\left[-2(\mu+\gamma+\alpha) + \sigma_2^2 + k_1 + k_2 + \frac{1}{k_2}f_{\hat{I}}^2\overline{Q}_\mu\overline{Q} + f_{\hat{I}} + (\delta+\gamma)\right] \\ & + z^2\big[-2(\mu+\delta) + \sigma_3^2 + (\delta+\gamma)\big]. \end{aligned}$$

Choose $k_1 = f_{\hat{S}}\overline{Q}_\mu\overline{Q}$, $k_2 = f_{\hat{I}}\overline{Q}_\mu\overline{Q}$. Then

$$\begin{aligned} LW \leqslant{}& x^2\big[2(n'(\hat{S}) - f_{\hat{S}}) + \sigma_1^2 + (f_{\hat{S}} + f_{\hat{I}})\big] \\ &+ y^2\big[-2\mu - \gamma - 2\alpha + \delta + \sigma_2^2 + 2f_{\hat{I}} + (f_{\hat{S}} + f_{\hat{I}})\overline{Q}_\mu\overline{Q}\big] \\ &+ z^2\big[-2\mu - \delta + \sigma_3^2 + \gamma\big]. \end{aligned}$$

It now follows from conditions (i)–(iii) that LW is negative definite, which implies that $\hat{E}$ is stochastically asymptotically stable. □

4 Numerical simulations and concluding remarks

In this section, we first perform a numerical simulation of (5) using MATLAB, the concrete choices of the parameter values being motivated by van den Driessche et al. [14]. The growth rate $n(S)$ and the incidence of infection $f(S, I)$ are particularized in the form

$$n(S) = 0.1 - 0.1S, \qquad f(S, I) = 0.6SI,$$

respectively. It is assumed that the maximal exposed period is 9 months (i.e., $h = 0.75$). Taking $\mu = 0.1$, $\delta = \gamma = 0.5$, $Q(\xi) = 2\mathrm{e}^{-2\xi}$ and $\alpha = 0.4$, we obtain that the disease-free equilibrium $E_0(1, 0, 0)$ of (5) is globally asymptotically stable, since $\mathcal{R}_0 = 0.7767$, while letting $\alpha = 0.2$ and retaining the same values for the other parameters, we obtain $\mathcal{R}_0 = 1.1821$, which implies the the endemic equilibrium $E^* \approx (0.846, 0.03033, 0.0253)$ of (5) is globally asymptotically stable. In addition, to illustrate the respective stochastic asymptotic stability of the disease-free equilibrium E_0 and of the endemic equilibrium E^*, we choose $\delta_1 = 0.4$, $\delta_2 = 0.7$ and $\delta_3 = 0.5$. The numerical simulations in Fig. 1, in which only the behaviour of the susceptible population is plotted, indicate that the S-components

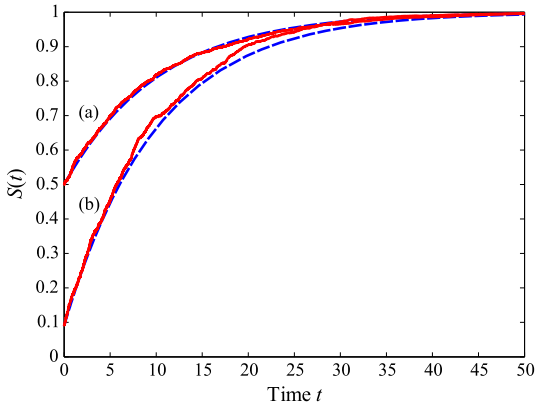

Figure 1. Time series graphs for the susceptible population. The initial conditions (blue dashed curve – the deterministic model; red solid curve – the stochastic model): (a) $\varphi_1(\theta) \equiv 0.5$, $\varphi_3(\theta) \equiv 0.01$, $\varphi_4(\theta) \equiv 0$, $\theta \in [-h, 0]$; (b) $\varphi_1(\theta) \equiv 0.09$, $\varphi_3(\theta) \equiv 0.01$, $\varphi_4(\theta) \equiv 0$, $\theta \in [-h, 0]$. (Online version in color.)

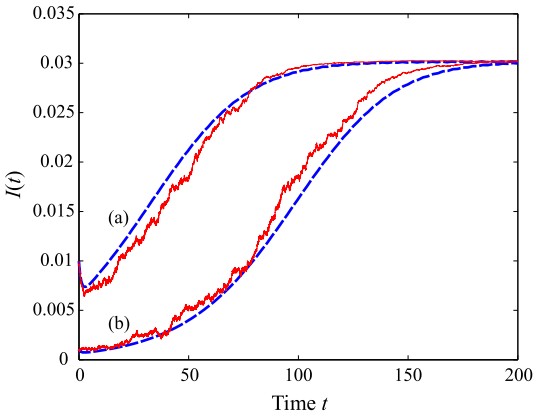

Figure 2. Time series graphs for the infected population. The initial conditions (blue dashed curve – the deterministic model; red solid curve – the stochastic model): (a) $\varphi_1(\theta) \equiv 0.99$, $\varphi_3(\theta) \equiv 0.01$, $\varphi_4(\theta) \equiv 0$, $\theta \in [-h, 0]$; (b) $\varphi_1(\theta) \equiv 0.99$, $\varphi_3(\theta) \equiv 0.001$, $\varphi_4(\theta) \equiv 0$, $\theta \in [-h, 0]$. (Online version in color.)

tend to $S_0 = 1$. Also, the numerical simulations in Fig. 2, in which only the behaviour of the infective population is plotted, indicate that the I-components tend to $I^* \approx 0.03033$.

The above numerical simulations reveal that the solution trajectories for the stochastic model may move around the deterministic steady state whenever the intensity of environmental forces is relatively small. In the near future, we hope to be able to investigate, via a similar approach, abstract multi-group epidemic models, which may help us understand the transmission dynamics of measles [1] and HIV/AIDS [12] and the impact of environmental perturbations upon the parameters characterizing deterministic systems.

References

1. Z. Bai, D. Liu, Modeling seasonal measles transmission in China, *Commun. Nonlinear Sci. Numer. Simul.*, **25**:19–26, 2015.

2. E. Beretta, Y. Takeuchi, Global stability of an SIR epidemic model with time delays, *J. Math. Biol.*, **33**:250–260, 1995.

3. S. Blower, T.C. Porco, G. Darby, Predicting and preventing the emergence of antiviral drug resistance in HSV-2, *Nat. Med.*, **4**:673–678, 1998.

4. C. Vargas de León, On the global stability of infectious diseases models with relapse, *Abstraction & Application*, **9**:50–61, 2013.

5. S. Gao, L. Chen, J.J. Nieto, A. Torres, Analysis of a delayed epidemic model with pulse vaccination and saturation incidence, *Vaccine*, **24**:6037–6045, 2006.

6. P. Georgescu, H. Zhang, A Lyapunov functional for a SIRI model with nonlinear incidence of infection and relapse, *Appl. Math. Comput.*, **219**:8496–8507, 2013.

7. J.W. Gnann Jr., Varicella-zoster virus: Atypical presentations and unusual complications, *J. Infect. Dis.*, **186**(Suppl. 1):91–98, 2002.

8. J. Henderson, R. Luca, Positive solutions for a system of second-order nonlinear multi-point eigenvalue problems, *Appl. Math. Comput.*, **223**:197–208, 2013.
9. Y. Kuang, *Delay Differential Equations with Applications in Population Dynamics*, Academic Press, Boston, 1993.
10. B. Li, S. Yuan, W. Zhang, Analysis on an epidemic model with a ratio-dependent nonlinear incidence rate, *Int. J. Biomath.*, **4**:227–239, 2011.
11. M.Y. Li, Z. Shuai, C. Wang, Global stability of multi-group epidemic models with distributed delays, *J. Math. Anal. Appl.*, **361**:38–47, 2010.
12. A.L. Lloyd, V.A.A. Jansen, Spatiotemporal dynamics of epidemics: Synchrony in metapopulation models, *Math. Biosci.*, **188**:1–16, 2004.
13. A.L. Riza, F. Pearson, C. Ugarte-Gil, B. Alisjahbana, S. van de Vijver, N.M. Panduru, P.C. Hill, R. Ruslami, D. Moore, R. Aarnoutse, J.A Critchley, R. van Crevel, Clinical management of concurrent diabetes and tuberculosis and the implications for patient services, *Lancet Diabetes Endocrinol.*, **2**:740–753, 2014.
14. P. van den Driessche, L. Wang, X. Zou, Modeling diseases with latency and relapse, *Math. Biosci. Eng.*, **4**:205–219, 2007.
15. R. Xu, Global dynamics of an SEIRI epidemiological model with time delay, *Appl. Math. Comput.*, **232**:436–444, 2014.
16. C.J. Yuan, D.Q. Jiang, D. O'Regan, R.P. Agarwal, Stochastically asymptotically stability of the multi-group seir and sir models with random perturbation, *Commun. Nonlinear Sci. Numer. Simul.*, **17**:2501–2516, 2012.