Local stochastic stability of SIRS models without Lyapunov functions

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Abstract

Most of stability analysis for stochastic epidemiological models involve Lyapunov functions. This work shows how sufficient conditions for the local stochastic asymptotic stability of a nonlinear system can be derived from the stability analysis of an ordinary linear system. In the particular stochastic SIR/SIRS models proposed here to illustrate the technique, the stability study of the obtained ordinary systems reduces to calculate the spectrum of the governing matrix.

Keywords: Stochastic differential systems, linear test system, mean square stability, stochastic stability, mathematical epidemiology, SIRS model, basic reproductive number

1. Introduction

Mathematical models for simulating the spread of biological agents are a topical issue due to the pandemic situation that we are experiencing worldwide. Modern Mathematical Epidemiology is based on the work of Kermack and McKendrick [16] and, consequently, the great majority of models appeared in the scientific literature are compartmental and deterministic. Moreover, the dynamics is usually based on ordinary differential equations whose variables stand for the total population amount or density belonging to each of the compartments [12]. Although this deterministic approach allows obtaining good simulations when population is large, it is not entirely suitable when the dynamics of real biological agents is studied; note that if it were possible to restart a real epidemic process, it is not reasonable to expect that under the same initial conditions the same individuals would be infected at exactly the same instant of time). Consequently, stochasticity is an inherent characteristic of these processes that plays a determining role

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when any of the following situations occurs: when the number of infectious individuals is small, when control measures are successfully implemented, at the initial stage or the trough phase of the epidemic outbreak. In this cases, and for the sake of reality, it is critical to consider randomness and to introduce it into the mathematical model.

Obviously, this scenario cannot be modelled by means of "traditional" ordinary differential equations. To overcome such drawback, stochastic differential equations (SDE for short) can be employed in the case of global models. In addition, other stochastic techniques such as discrete Markov chains or Bayesian networks have been used for computational models following the individual-based paradigm [19].

Stochastic differential equations have been considered to model and simulate different types of phenomena in Physics, Biology, Finance, Epidemiology, etc., see [18] and the references therein. More recent examples can be seen in [34], where the uncertainties in electrical power systems are modeled by means of SDEs; in [8], where a new modeling approach based on Ito stochastic differential equations for the neutron count distribution in a subcritical core is proposed; in [22], where the price dynamics of financial markets is studied using SDEs; or in [27], where the dynamics of a breast tumor is modeled by means of Ito SDEs. Models to simulate pathogen propagation based on SDEs have been proposed and analyzed, see, e.g., [21, 28, 35]. As in the case of deterministic models based on ordinary differential equations (ODEs), a qualitative study of the stochastic system must accompany the description of the model.

Most of stability analysis for SDEs models are based on the derivation of sufficient conditions for stability obtained from the construction of Lyapunov functions [7, 14, 20, 23, 32] and, with the thresholds involved, the consequent definition of the stochastic version of the basic reproductive number. In some cases it can be difficult to find suitable Lyapunov functions; in general, there are no systematic methods for constructing them. Our main goal is to overcome this drawback presenting a systematic way to obtain sufficient conditions for the stochastic stability of the equilibrium solution based on the mean-square stability of its linearized problem. For this linear problem, necessary and sufficient conditions can be obtained from the stability analysis of an ordinary differential system.

The rest of the paper is organized as follows: In Section 2 the background of the stability of stochastic differential equations is presented; the mathematical description of both, SIR and SIRS models, is introduced in Section 3; in Section 4 the stability of the stochastic SIR model is tackled. Some illustrative numerical simulations are presented in Section 5. Finally the conclusions are presented in Section 6.

2. Fundamentals of SDEs stability

For our purposes we consider a filtered probability space $(\Omega, \mathcal{F}, \mathcal{F}_t, P)$, an m-dimensional Wiener process $\{W_t = (W_t^1, \ldots, W_t^m)\}_{t\geq 0}$ and an autonomous d-dimensional stochastic differential equation (SDE)

$$dX_t = f(X_t)dt + \sum_{k=1}^{m} g_k(X_t)dW_t^k, \qquad t_0 \leq t, \tag{1}$$

where the functions f,$g_1, \ldots, g_m$ are assumed to be defined and measurable in $\mathbb{R}^d$ and to satisfy a Lipschitz condition of the form

$$|f(x) - f(y)| + \sum_{k^1}^{m} |g_k(x) - g_k(y)| \leq K|x - y| \tag{2}$$

for all $x, y \in \mathbb{R}^d$. This assumption ensures the existence of a unique solution of the (1) with the initial condition $X_{t_0} = c$ if c is $\mathcal{F}_{t_0}$-measurable, see, e.g. Arnold [3]). From now on, we suppose that $c \in \mathbb{R}^d$ and the unique solution, starting at time t_0 at c will be denoted $X_t(c)$.

Suppose now that

$$f(0) = 0, \quad g_k(0) = 0, \qquad k = 1, \ldots, m; \tag{3}$$

in this case the equilibrium position $X_t \equiv 0$ is the unique solution of (1) with initial condition $c = 0$.

2.1. Stochastic stability

Definition 1. *The equilibrium position of* (1) *is said to be stochastically stable (or stable in probability) if, for every* $\epsilon > 0$*,*

$$\lim_{c\to 0} P\left\{\sup_{t\geq t_0} |X_t(c)| \geq \epsilon\right\} = 0.$$

Definition 2. *The equilibrium position is said to be stochastically asymptotically stable (or asymptotically stable in probability) if it is stochastically stable and*

$$\lim_{c\to 0} P\left\{\lim_{t\to\infty} X_t(c) = 0\right\} = 1.$$

Under suitable conditions, stochastic stability behaviour of a stochastic differential equation can be derived, see e.g. theorems 7.1 and 7.2 in Khasminskii [17], from the study of its linearized equation

$$dX_t = A\, X_t dt + \sum_{k=1}^{m} B_k\, X_t\, dW_t^k \tag{4}$$

with coefficients given by the constant matrices

$$A = \frac{\partial f}{\partial x}(0), \quad B_k = \frac{\partial g_k}{\partial x}(0), \quad K = 1, \ldots, m. \tag{5}$$

In particular, we state:

Theorem 1. *Consider the SDE* (1) *whose coefficients* $f = f(x)$ *and* $g_k = g_k(x)$ *are* $\mathbb{R}^d$*-valued functions that satisfy the Lipschitz condition* (2) *together with* (3)*. If, in addition, they are differentiable functions with continuous partial derivatives at* $x = 0$*, then the stochastic asymptotic stability of the equilibrium position of the linear SDE* (4)-(5) *implies the stochastic asymptotic stability of the equilibrium position of the original equation* (1)*.*

2.2. Mean square stability

For any vector or matrix Z, let us denote by $Z^\top$ its transpose. Under the Lipschitz assumption for the coeffcients of the SDE (1) together with the initial constant condition, existence of the moments of the solution is assured, see [3]; in particular, the mean $m_t = \mathrm{E}[X_t]$, the second moment $P(t) = \mathrm{E}[X_t X_t^\top]$, or $\mathrm{E}|X_t|^p$ for any $p > 0$ exist.

Definition 3. *The equilibrium position is said to have a stable second moment if for every* $\epsilon > 0$ *there exists a* $\delta > 0$ *such that for all* $|c| \leq \delta$

$$\sup_{t_0 \leq t \leq \infty} |E[X_t(c) X_t^\top(c)]| \leq \epsilon.$$

If in addition

$$\lim_{t \to \infty} |E[X_t(c) X_t^\top(c)]| = 0$$

for all c *in a neighborhood of* $x = 0$*, the equilibrium position is said to have an asymptotical stable second moment.*

Definition 4. *The equilibrium position is said to be mean-square stable if for every* $\epsilon > 0$ *there exists a* $\delta > 0$ *such that for all* $|c| \leq \delta$

$$\sup_{t_0 \leq t \leq \infty} E|X_t(c)|^2 = 0.$$

If in addition for all c *in a neighborhood of* $x = 0$

$$\lim_{t \to \infty} E|X_t(c)|^2 = 0,$$

the equilibrium position is said to be mean-square asymptotically stable.

It is easy to prove, see [3], that (asymptotical) stability in mean square is equivalent to (asymptotical) stability of the second moment. Although in general the second moment of the solution of a general SDE does not satisfy a simple equation, for the linear case it is known, see Theorem 8.5.5 in [3], the form of the (ordinary) matrix differential equation that it fulfills. In particular, for the linear SDE (4) where A, B_k are $d \times d$ matrices with constant entries, the second moment $P(t) = E[X_t\, X_t^\top] = (p_{ij}(t))$ of the solution satisfies the equation

$$\frac{dP(t)}{dt} = AP(t) + P(t)A^\top + \sum_{k=1}^{m} B_k P(t) B_k^\top . \tag{6}$$

Since $P(t)$ is symmetric, (6) reduces to a linear system of $d(d+1)/2$ differential equations of the form

$$\frac{dY}{dt} = \mathcal{M}\, Y\,, \tag{7}$$

where the components of the vector Y are $p_{ij} = E[X_t^i\, X_t^j]$, $i, j = 1, \ldots, d, i \le j$. Then the asymptotic mean-square stability of the equilibrium position of (4), identical to the asymptotic stability of the second moment $P(t)$), is equivalent to the asymptotical stability of the equilibrium position $Y \equiv 0$ of the linear system (7). Since this condition reduces to the requirement that all the eigenvalues of $\mathcal{M}$ lie in the left half-plane, we conclude that the linear test system (4) is asymptotically MS-stable if and only if the spectral abscissa of the auxiliary matrix $\mathcal{M}$ is negative, i.e.

$$\nu(\mathcal{M}) = \max\{\Re(\lambda) : \lambda \in \sigma(\mathcal{M})\} < 0,$$

where $\sigma(\mathcal{M})$ denotes the spectrum of the matrix $\mathcal{M}$.

2.3. MS-stability vs stochastic stability

For linear SDEs with constant coefficients, Gikhman [9] proved, see also [3, 17], that MS-stability implies stochastic asymptotical stability:

Theorem 2. *The solution $X_t = 0$ of the stochastic linear system with constant coefficients* (4)*, having asymptotic stability of its second-order moments, is stochastically asymptotically stable.*

Combining this fact with the above results of this section, we summarize how to study stochastic asymptotical stability of the (general) SDE (1) by means of the stability analysis of an ordinary linear system: For a SDE (1) fulfilling the conditions of Theorem 1, consider its linearized SDE system (4) with coefficients (5) and the auxiliary ordinary system (7) fulfilled by the second moment of this linear SDE

system: Asymptotical stability of this ordinary system implies asymptotical mean square stability (and then asymptotical stochastic) of the linear SDE, which in turn implies asymptotical stochastic stability of the original SDE.

3. The SIR/SIRS compartmental model

3.1. The (classic) deterministic approach

As it was mentioned, the mathematical model proposed by Kermack and McKendrick can be considered as the cornerstone of the modern Mathematical Epidemiology. In this compartmental model the population is divided into three classes: susceptible (S), infected (I), and recovered (R) individuals. In the simplest case the system of ordinary differential equations that governs the dynamics of the SIR/SIRS model is given by:

$$\begin{aligned} \frac{dS(t)}{dt} &= -\delta\, I(t)S(t) + \gamma\, R(t) \\ \frac{dI(t)}{dt} &= \delta\, I(t)S(t) - \lambda\, I(t) \\ \frac{dR(t)}{dt} &= \lambda\, I(t) - \gamma\, R(t) \end{aligned} \tag{8}$$

where $0 < \delta \leq 1$ represents the probability that a relationship with an infectious leads to contagion, $0 \leq \gamma \leq 1$ is the loss-of-immunity rate (that is, the rate at which recovered individuals return to susceptible compartment), and $0 < \lambda \leq 1$ stands for the recovery rate (the rate at which infected individuals recover from the disease). If $\gamma = 0$ there is no flow from the recovered compartment to the susceptible compartment and the classic SIR model is obtained: This model captures the dynamics of diseases that confers permanent immunity, such as measles or rubella, see e.g. [11], as well as a variety of epidemics, see [26]. Recent applications of this model to COVID-19 can be found in [4], [6], [29] or [30]. The basic SIRS model ($\gamma > 0$) is derived when immunity is temporary and after a certain period of time the recovered individual becomes susceptible again. This model might be appropriate for diseases as tetanus, influenza or cholera, see e.g. [10].

Notice that in the previous models the total population $N(t) = S(t) + I(t) + R(t)$ remains constant through time. This tenet can be relaxed and system (8) can be extended taking into account demography (births, natural deaths, and migrations) and mortality caused by the disease, obtaining the general system

$$\begin{aligned}
\frac{dS(t)}{dt} &= b - \delta I(t)S(t) + \gamma R(t) - \mu S(t) \\
\frac{dI(t)}{dt} &= \delta I(t)S(t) - \lambda I(t) - \mu I(t) - \alpha I(t) \\
\frac{dR(t)}{dt} &= \lambda I(t) - \gamma R(t) - \mu R(t)
\end{aligned} \tag{9}$$

where $0 < \mu \leq 1$ is the natural death rate, $0 < \alpha \leq 1$ is the disease-caused death rate, and the constant rate b is the influx into susceptible compartment, for example, due to migration. This model was proposed in [1] to study transmitted diseases of mice, see also [25]; for a survey of recent applications, see [30].

Finally, the system (9) can be generalized replacing δ by $\beta\frac{k(N)}{N}$ where $k(N)$ represents the contact rate (total number of adequate contacts that an individual has with the entire population per unit of time), and β is the probability that an adequate contact leads to an infection, see [5] or [15]. So, the proposed model is given by

$$\begin{aligned}
\frac{dS(t)}{dt} &= b - \beta\frac{k(N)}{N}S(t)I(t) - \mu S(t) + \gamma R(t) \\
\frac{dI(t)}{dt} &= \beta\frac{k(N)}{N}S(t)I(t) - (\lambda + \mu + \alpha)I(t) \\
\frac{dR(t)}{dt} &= \lambda I(t) - (\mu + \gamma)R(t)\,.
\end{aligned} \tag{10}$$

The term $\beta\frac{k(N)}{N}S(t)I(t)$ is called incidence (new infections per unit of time) and strongly depends on the contact rate $k(N)$. The dynamics of this general model is graphically illustrated by means of the flow diagram shown in Figure 1.

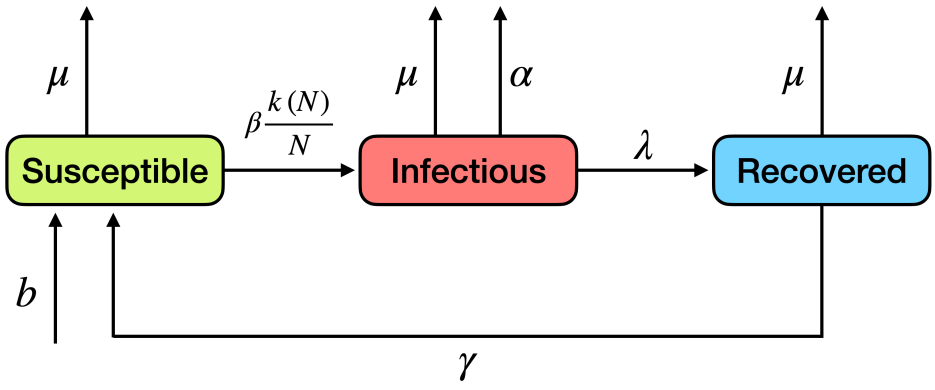

Figure 1: Flow diagram representing the dynamics of SIR and SIRS models.

The population $N(t)$ is variable, and for the function $k = k(N)$ the following assumptions are made [15] in order to consider saturation incidences:

$$k(N) > 0, \quad k'(N) \geq 0, \quad \left(\frac{k(N)}{N}\right)' \leq 0,$$

where the prime denotes the derivative with respect to N. Notice that the class of functions $k(N) = sN^r$, with $0 \leq r \leq 1$, $s \in \mathbb{Z}^+$, fulfills the above conditions. And the particular cases $r = 1$, i.e. $k(N) = sN$, and $r = 0$, i.e. $k(N) = s$, correspond to the bilinear and standard incidence SIRS models with immigration, respectively, see e.g. [25, 33]. For other applications of this model, see [5].

A simple calculus shows that the basic reproductive number associated to this model is

$$R_0 := \frac{\beta k\left(\frac{b}{\mu}\right)}{\alpha + \lambda + \mu}. \tag{11}$$

As is well known, this is the most important threshold epidemiological coefficient; its value -greater or less than 1- determines the behavior of the epidemic process, that is, the stability of the equilibrium points.

The system (10) has a unique disease free equilibrium $p_0 = (b/\mu, 0, 0)$. The linearized system centered at p_0 is given by

$$dX(t) = \begin{pmatrix} -\mu & -\beta\, k\left(\frac{b}{\mu}\right) & \gamma \\ 0 & \beta\, k\left(\frac{b}{\mu}\right) - \lambda - \alpha - \mu & 0 \\ 0 & \lambda & -\gamma - \mu \end{pmatrix} X(t)\, dt$$

with $X(t) = (S(t) - b/\mu, I(t), R(t))$. Since the eigenvalues of the matrix are $-\mu, -\mu - \gamma, \beta\, k\left(\frac{b}{\mu}\right) - (\alpha + \lambda + \mu)$, the local asymptotical stability condition can be written $\beta\, k\left(\frac{b}{\mu}\right) < \alpha + \lambda + \mu$ or, in terms of the reproductive number, as $R_0 < 1$. Notice that this condition does not depend on γ. Moreover, in [15] it is shown that the disease-free equilibrium p_0 is globally asymptotically stable for $R_0 \leq 1$ and unstable for $R_0 > 1$. It is also shown that if $R_0 > 1$ the system (10) has a unique endemic equilibrium $p_1 = (S^*, I^*, R^*)$ with

$$S^* = \frac{N^*}{k(N^*)} \frac{\alpha + \lambda + \mu}{\beta}, \quad I^* = \frac{\gamma + \mu}{\alpha(\gamma + \mu) + \mu(\gamma + \lambda + \mu)} (b - \mu S^*), \quad R^* = \frac{\lambda}{\gamma + \mu} I^*, \tag{12}$$

and N^* is the unique positive solution of the equation

$$\frac{k(N)}{\alpha + \lambda + \mu} \left(1 - \left(1 + \frac{\lambda}{\mu + \gamma}\right) \frac{b - \mu N}{\alpha N}\right) - 1 = 0.$$

In addition, it is shown that p_1 is locally asymptotically stable if $R_0 > 1$.

3.2. The stochastic version

The deterministic approach to the problem of modeling epidemic propagation, shown in subsection 3.1 by means of the SIR/SIRS compartmental model, is the classic one. However, due to the inherent randomness of epidemiological processes it seems more appropriate (or, at least, reasonable) to resort to stochastic techniques. The incidence is the key term in (10) and, specifically, the nature of the infection rate β is highly random. As a consequence it is of interest to propose compartmental models whose dynamics is governed by stochastic differential equations where the parameter β is perturbed by the addition of a white noise ξ_t with intensity σ resulting in $\beta+\sigma\xi_t$. Then the deterministic SIR/SIRS model becomes to the stochastic (in the Itô sense) SIR/SIRS model:

$$\begin{aligned} dS_t &= \left(-\beta\frac{k(N)}{N}S_tI_t - \mu S_t + \gamma R_t + b\right)dt - \sigma\frac{k(N)}{N}S_tI_t\,dW_t \\ dI_t &= \left(\beta\frac{k(N)}{N}S_tI_t - (\lambda+\mu+\alpha)I_t\right)dt + \sigma\frac{k(N)}{N}S_tI_t\,dW_t \\ dR_t &= (\lambda I_t - (\mu+\gamma)R_t)\,dt \end{aligned} \tag{13}$$

The stochastic system (13) is a slight generalization of some models presented in the literature: in [14] and [32] the SIR model ($\gamma = 0$) is considered, with particular value $b = 0$ in the second text; in [23] the case $b = \mu$, $\alpha = 0$ is studied.

The SDE (13) has a unique equilibrium $p_0 = (b/\mu, 0, 0)$. Using the notions of stochastic stability and their relations introduced in Section 2 we shall analyze the stability behaviour of this stochastic disease-free equilibrium.

4. Stochastic Stability of the stochastic SIR model

Linearizing (10) around the equilibrium p_0 and using the new variable $X_t = (X_t^1, X_t^2, X_t^3) = (S_t - b/\mu, I_t, R_t)$, we obtain the linear SDE

$$dX_t = \begin{pmatrix} -\mu & -\beta\,k\left(\frac{b}{\mu}\right) & \gamma \\ 0 & \beta\,k\left(\frac{b}{\mu}\right) - \lambda - \alpha - \mu & 0 \\ 0 & \lambda & -\gamma-\mu \end{pmatrix} X_t\,dt + \begin{pmatrix} 0 & -\sigma\,k\left(\frac{b}{\mu}\right) & 0 \\ 0 & \sigma\,k\left(\frac{b}{\mu}\right) & 0 \\ 0 & 0 & 0 \end{pmatrix} X_t\,dW_t. \tag{14}$$

From here, taking $Y = (p_{11}(t), p_{22}(t), p_{33}(t), p_{12}(t), p_{13}(t), p_{23}(t))^\top$, with $p_{ij}(t) = \mathrm{E}[X_t^i X_t^j]$, the corresponding linear system (6) reduces to the ordinary differential equation (7) with

$$\mathcal{M} = \begin{pmatrix} -2\mu & \sigma^2\overline{k}^2 & 0 & -2\beta\,\overline{k} & 2\gamma & 0 \\ 0 & \sigma^2\overline{k}^2 + 2\beta\,\overline{k} - 2(\alpha+\lambda+\mu) & 0 & 0 & 0 & 0 \\ 0 & 0 & -2(\gamma+\mu) & 0 & 0 & 2\lambda \\ 0 & -\sigma^2\overline{k}^2 - \beta\,\overline{k} & 0 & \beta\,\overline{k} - (\alpha+\lambda+2\mu) & 0 & \gamma \\ 0 & 0 & \gamma & \lambda & -\gamma-2\mu & -\beta\,\overline{k} \\ 0 & \lambda & 0 & 0 & 0 & \beta\,\overline{k} - (\alpha+\gamma+\lambda+2\mu) \end{pmatrix}$$

where $\overline{k}$ stands for $k\left(\frac{b}{\mu}\right)$.

Theorem 3. *The trivial solution of the linear SDE* (14) *is asymptotically mean-square stable if and only if*

$$\frac{\beta\,k\left(\frac{b}{\mu}\right) + \frac{1}{2}\sigma^2\left(k\left(\frac{b}{\mu}\right)\right)^2}{\alpha+\lambda+\mu} < 1 \tag{15}$$

holds.

Proof. The eigenvalues of $\mathcal{M}$ are (the real values) $\lambda_1 = -2\mu$, $\lambda_2 = -2(\gamma+\mu)$, $\lambda_3 = -\gamma - 2\mu$, $\lambda_4 = \beta k\left(\frac{b}{\mu}\right) - (\alpha+\lambda+2\mu)$, $\lambda_5 = \beta k\left(\frac{b}{\mu}\right) - (\alpha+\lambda+2\mu+\gamma)$ and $\lambda_6 = \sigma^2\left(k\left(\frac{b}{\mu}\right)\right)^2 + 2\beta\,k\left(\frac{b}{\mu}\right) - 2(\alpha+\lambda+\mu)$. Since the parameters μ, λ, β, b are positive and $\gamma, \alpha \geq 0$, it is obvious that condition

$$\sigma^2\left(k(b/\mu)\right)^2 + 2\beta\,k(b/\mu) - 2(\alpha+\lambda+\mu) < 0 \tag{16}$$

s equivalent to $\lambda_i < 0$, $i = 1, \ldots, 6$. □

Equation (15) can be rewritten as $R_0^{MSL} < 1$, where

$$R_0^{MSL} := \frac{\beta\,k\left(\frac{b}{\mu}\right) + \frac{1}{2}\sigma^2\left(k\left(\frac{b}{\mu}\right)\right)^2}{\alpha+\lambda+\mu}\,. \tag{17}$$

This leads to a epidemiological threshold coefficient, named *MS basic reproductive number* and conclude that the linearized system is MS-stable if and only if

$$R_0^{MSL} = R_0\left(1 + \frac{\sigma^2}{2\beta}k\left(\frac{b}{\mu}\right)\right) < 1. \tag{18}$$

Finally, from the above results we can conclude:

Theorem 4. *If condition* (15) *holds, then the disease free equilibrium* $p_0 = \left(\frac{b}{\mu}, 0, 0\right)$ *of the SIR/SIRS model* (10) *is stochastically asymptotically stable.*

PROOF. If condition (15) holds, the linear system (14) is asymptotically MS-stable and then, by Theorem 2, stochastically asymptotically stable. From here, Theorem 1 gives that the trivial solution of (10) is stochastically asymptotically stable □

Remarks. Notice that

1. Condition (15) does not depend on γ.
2. For SIR/SIRS models with bilinear incidence and $s = 1$, i.e. $k(N) = N$, condition (15) becomes

$$\frac{\beta \frac{b}{\mu} + \frac{1}{2}\sigma^2 \left(\frac{b}{\mu}\right)^2}{\alpha + \lambda + \mu} < 1.$$

   (a) This condition coincides with the sufficient condition given for the SIR model in [14]. If, in addition, $\mu = b$, this condition improves the one stated in [32].

   (b) When $\mu = b$ and $\alpha = 0$, the condition reduces to

$$\frac{\beta + \frac{1}{2}\sigma^2}{\lambda + \mu} < 1,$$

   which coincides with the sufficient condition for stochastic asymptotical stability of the trivial solution given for the SIRS model in [23].

3. For SIR/SIRS models with standard incidence, i.e. $k(N) = s \in \mathbb{Z}^+$, condition (15) becomes

$$\frac{\beta s + \frac{1}{2}\sigma^2 s^2}{\alpha + \lambda + \mu} < 1.$$

5. Numerical simulations

We carry out two groups of numerical experiments to confirm the theoretical results presented above. The first one (experiments 1-4) refers to stochastic stability of the equilibrium of equation (13), whereas the second one (experiments 5-6) is concerned with the mean-square stability of system (14). Some parameters were fixed for all experiments:

$$k(N) = N,\ b = 0.5,\ \mu = 0.5,\ \gamma = 0.4,\ \lambda = 0.3,\ \alpha = 0.2. \tag{19}$$

Notice that since $\alpha + \lambda + \mu = 1$ and $b = \mu$, then $R_0 = \beta$ and $R_0^{MSL} = \beta + \sigma^2/2$.

For each experiment of the first group, a pair of values of β, σ is selected and equation (13) with initial value $(S_0, I_0, R_0) = (0.9, 0.5, 0.3)$, is solved numerically in the interval $[0, 50]$ using the Euler-Maruyama method with step-size $\Delta = 1/10$. We repeat the integration to obtain 10 trajectories of the exact solution. On the other hand for each experiment we solve the deterministic system (10) with the same parameters and initial value $(S(0), I(0), R(0)) = (0.8, 0.7, 0.2)$. Then the components S, I, R of the 10 trajectories of the solution of the stochastic problem and the solution of the deterministic problem are represented together for the sake of comparison (Figures 2-5). The deterministic solution acts as a reference of the stochastic solution with initial value $(S_0, I_0, R_0) = (0.8, 0.7, 0.2)$ that can be compared visually with the 10 trajectories of stochastic solution with initial value $(S_0, I_0, R_0) = (0.9, 0.5, 0.3)$.

The second group (experiments 5-6) is devoted to illustrate the necessary an sufficient condition for MS-stability of the linear system (14). To approximate the second moment of the solution of the linear SDE a batch of 1000 simulations are carried out. Each realization is obtained integrating (14) with the stochastic trapezoidal method along the interval $[0, 50]$ with step-size $\Delta = 1/4$ and initial value $(S_0, I_0, R_0) = (0.8, 0.7, 0.6)$. The stochastic trapezoidal method is the particular member of the family of stochastic θ-methods [13] obtained with $\theta = 1/2$. The numerical solutions obtained integrating with this semi-implicit method has shown special behavior in the replication of MS-stability properties of the exact solution, see e.g. [13, 31]. Then for each component U (X, Y, or Z) the values

$$\mathrm{E}\left[U_n^2\right] = \frac{1}{1000}\sum_{j=1}^{1000}(u_n^j)^2$$

were calculated as an approximation of $\mathrm{E}[U_t^2]$.

Experiment 1. We take $\beta = 0.8$ and $\sigma = 0.2$. Then $R_0^{MSL} = 0.84 < 1$ and from Theorem 4 the equilibrium is stochastically stable (Figure 2). In agreement with Theorem 4 the figure shows that (15) is a sufficient condition for the stochastic asymptotic stability of the disease free equilibrium point of equation (13).

Experiment 2. We take $\beta = 0.8$ and $\sigma = 1$. Then $R_0^{MSL} = 1.31$; therefore the sufficient condition of Theorem 4 does not hold. Figure 3 shows stochastic stability around the equilibrium point $(1, 0, 0)$; the graphic then suggests that (15) is not a necessary condition for stochastic asymptotic stability. This result is in agreement with the assertion made in [32], where it is said that $\beta - \sigma^2/2 < \alpha + \lambda + \mu$ is the sufficient condition guested from numerical experiments.

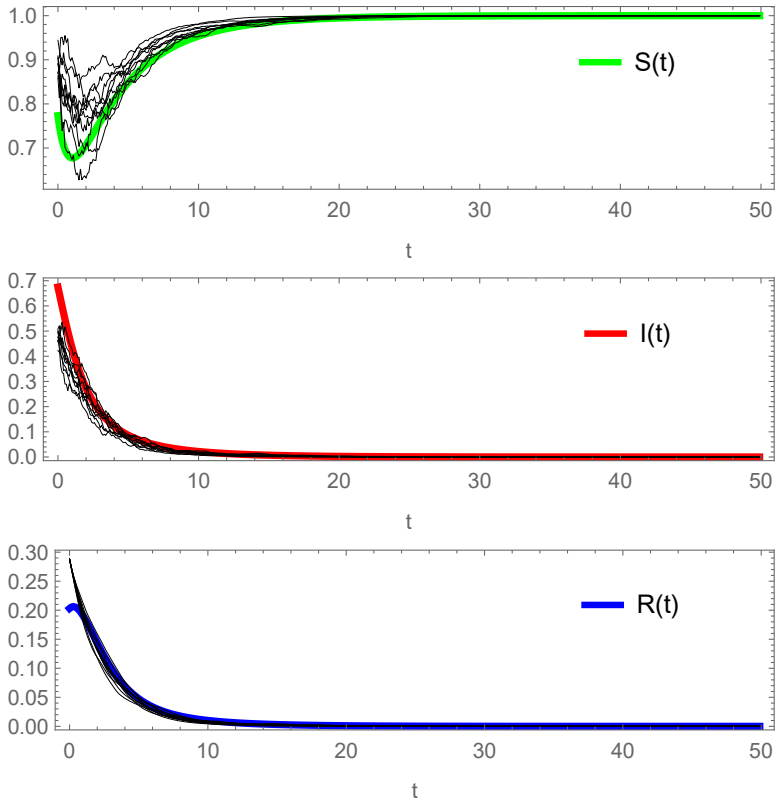

Figure 2: Components of the solution of the deterministic system (10) with initial value $(0.8, 0.7, 0.2)$ (colored thick line) together with 10 trajectories of the solution of the stochastic system (13) with initial value $(0.9, 0.5, 0.3)$ (black thin lines), in both cases with the parameters (19) together with $\beta = 0.8$ and $\sigma = 0.2$

Experiment 3. We take $\beta = 1.2$ and $\sigma = 1$. As in the second experiment, the guested experimental sufficient condition for stability $\beta - \sigma^2/2 < 1 = \alpha + \lambda + \mu$ holds; in agreement with it, Figure 4 shows stochastic stability around the equilibrium point $(1, 0, 0)$. But notice that the situation differs from Experiment 2. In this case $R_0 > 1$ and the general theory ensures for equation (10) that its endemic equilibrium $p_1 = (\frac{5}{6}, \frac{5}{42}, \frac{5}{136}) \approx (0.82, 0.12, 0.04)$ is stable, whereas its disease-free equilibrium is unstable. So, $(1, 0, 0)$ is an unstable equilibrium point of the deterministic system (10) that becomes a stable equilibrium of the corresponding stochastic system (13). This example illustrates the fact reported in the literature, see e.g. [2, 24], of how the perturbation of a deterministic unstable system by a noise can result in a stable SDE.

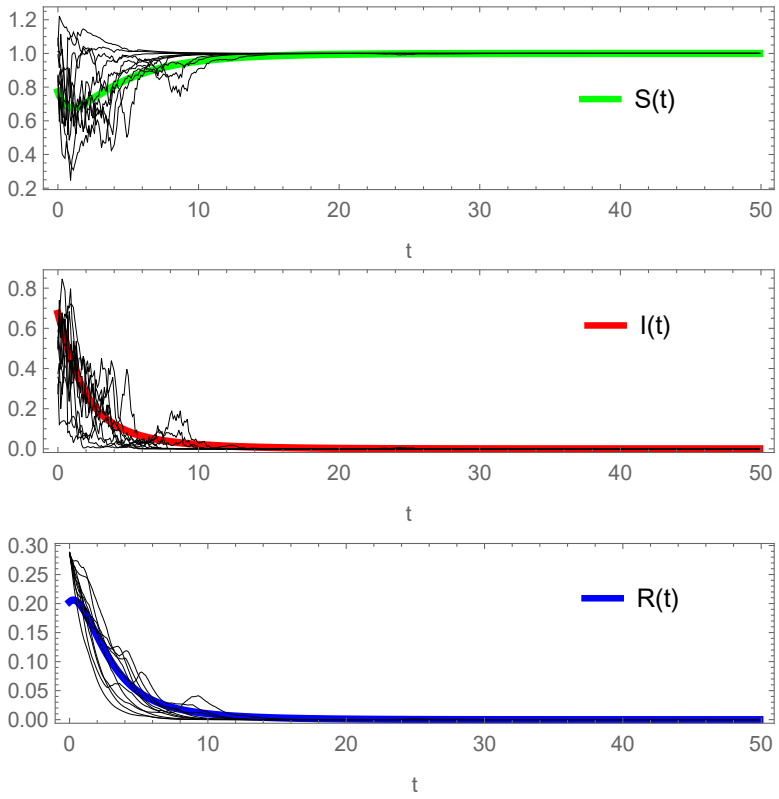

Figure 3: Components of the solution of the deterministic system (10) with initial value $(0.8, 0.7, 0.2)$ (colored thick line) together with 10 trajectories of the solution of the stochastic system (13) with initial value $(0.9, 0.5, 0.3)$ (black thin lines), in both cases with the parameters (19) together with $\beta = 0.8$ and $\sigma = 1$

Experiment 4. We take $\beta = 2.3$ and $\sigma = 1$. In this case $R_0 > 1$, $R_0^{MSL} > 1$ and the experimental sufficient condition $\beta - \sigma^2/2 > 1$ holds. The endemic equilibrium $p_1 = (\frac{10}{23}, \frac{15}{46}, \frac{5}{46}) \approx (0.43, 0.33, 0.11)$ of the deterministic system (10) is stable. Figure 5 shows instability of the equilibrium $(1, 0, 0)$ for both deterministic (10) and stochastic (13) systems.

160 *Experiment 5.* As in Experiment 1, we set $\beta = 0.8$ and $\sigma = 0.2$. In this case $R_0^{MSL} = 0.84 < 1$. The results are shown in the left plot of Figure 6, where the approximate values of $\mathrm{E}[X_t^2]$, $\mathrm{E}[Y_t^2]$ and $\mathrm{E}[Z_t^2]$ are plotted versus t. In agreement with Theorem 3 the figure shows that the trivial solution of the linear SDE (14) is mean-square stable.

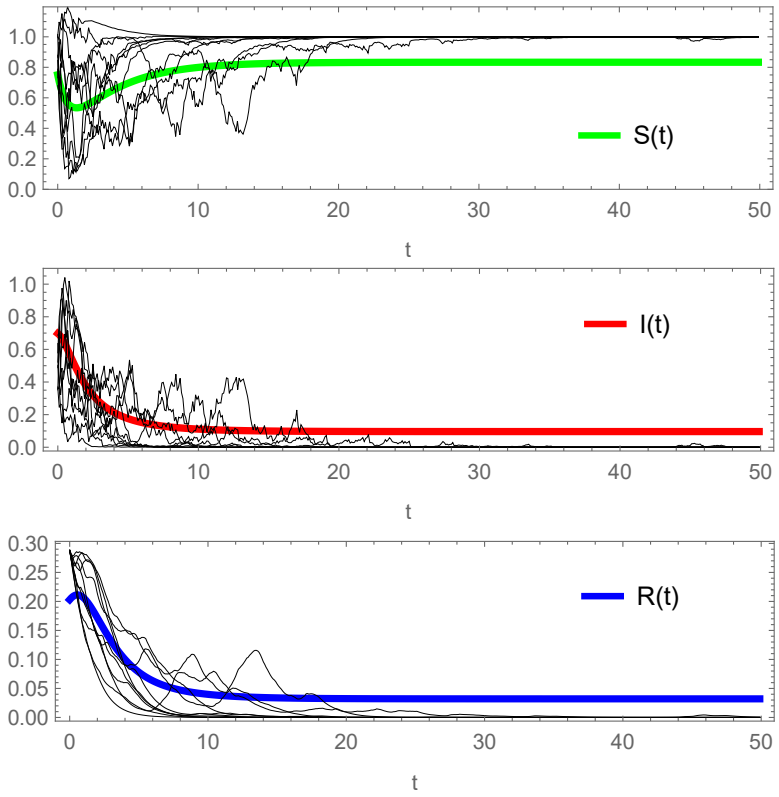

Figure 4: Components of the solution of the deterministic system (10) with initial value $(0.8, 0.7, 0.2)$ (colored thick line) together with 10 trajectories of the solution of the stochastic system (13) with initial value $(0.9, 0.5, 0.3)$ (black thin lines), in both cases with the parameters (19) together with $\beta = 1.2$ and $\sigma = 1$

Experiment 6. As in Experiment 2, we set $\beta = 0.8$ and $\sigma = 1$. In this case $R_0^{MSL} = 1.3 > 1$. The results are shown in the right plot of Figure 6. In agreement with Theorem 3 the figure shows that the trivial solution of the linear SDE (14) is mean-square unstable.

6. Conclusions

Sufficient conditions for the local stochastic stability of a stochastic SIR/SIRS model are derived using a direct analysis of the mean square stability of the corresponding linearized problem. Although the obtained conditions coincide with other presented in the literature, the procedure to obtain them, avoiding the use of Lyapunov functions, has the advantage of being a systematic way to address the

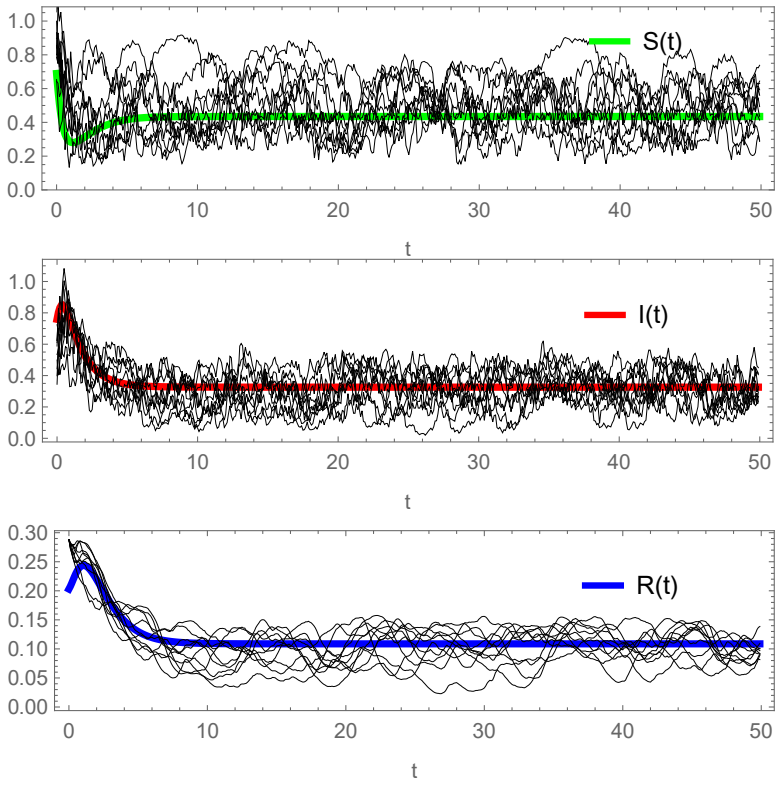

Figure 5: Components of the solution of the deterministic system (10) with initial value $(0.8, 0.7, 0.2)$ (colored thick line) together with 10 trajectories of the solution of the stochastic system (13) with initial value $(0.9, 0.5, 0.3)$ (black thin lines), in both cases with the parameters (19) together with $\beta = 2.3$ and $\sigma = 0.2$

problem: it reduces to the calculus of the spectrum of a matrix, as in the presented model, or, when it is not possible, to the use of Routh-Hurwitz theorem or equivalent results.

While the theoretical sufficient condition $\frac{\beta+\sigma^2/2}{\alpha+\lambda+\mu} < 1$ is confirmed by some numerical example, other experiments suggest that it is not a necessary condition for stochastic asymptotic stability. In fact, the lower value $\frac{\beta-\sigma^2/2}{\alpha+\lambda+\mu}$ seems to be experimentally the threshold for stochastic asymptotic stability. This point deserves a more detailed study in another work.

The stochastic compartmental SIRS model analyzed in this work can deal with the problem of virus spread with certain particular characteristics. Specifically, (1) epidemiological periods are assumed to be extremely short, and thus the influx rate b is supposed constant; and (2) the virulence of the biological agent results in a high mortality rate among infected individuals, and consequently the rate of deaths

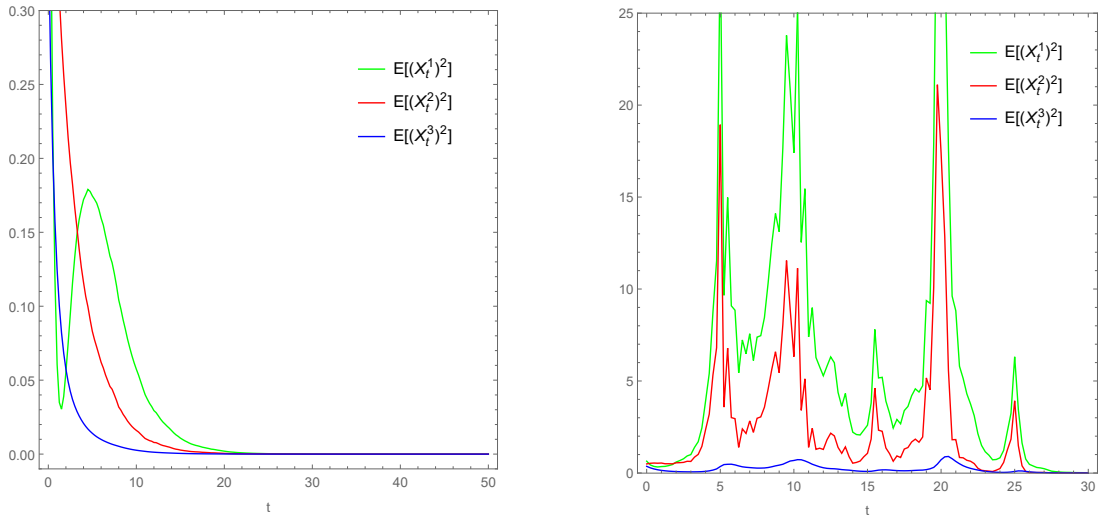

Figure 6: Numerical approximations of the second moments $\mathrm{E}(X_t^1)^2$, $\mathrm{E}(X_t^2)^2$, $\mathrm{E}(X_t^3)^2$ of the solution of the linear system (14) with $\sigma = 0.2$ (left plot), $\sigma = 1$ (right plot) and shared parameters (19) and $\beta = 0.8$ in both cases

is proportional to the infected compartment: $\alpha I(t)$. Moreover, for the sake of simplicity, this is also supposed for natural deaths. Note that this is the situation that occurs, for example, with biological agents as *Vibrio choleare* O1) (cholera), *Vibrio parahaemolyticus*, *Neisseria meningitidis*, *Ebolavirus* (ebola hemorrhagic fever), *SARS-CoV-2* in certain age groups, etc.

Finally, with the aim to control the epidemic process it is necessary to reduce the numeric value of the MS basic reproductive number R_0^{MSL} as much as possible. In this sense some basic control measures can be easily obtained if only one epidemiological coefficient is changed (and a trivial analysis of the threshold monotonicity is done). For example, for the explicit expression of R_0^{MSL} it is shown that it decreases as β or σ or b decrease, or it also decreases as α or λ or μ increase.

Further work aimed at studying more efficient control strategies involving two or more epidemiological parameters. Furthermore, it is also of great interest to compare and analyze the behavior of solutions of both, deterministic and stochastic models, when they are running with real data.

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References

[1] Anderson, R. M., May, R. M. Population biology of infectious diseases I. Nature 180 (1979) 361-367.

[2] Appleby, J., Mao, X., Rodkina, A. Stabilization and destabilization of nonlinear differential equations by noise. IEEE Trans. Autom. Control 53 (2008) 683-691

[3] Arnold, L. Stochastic differential equations: theory and applications. John Wiley & Sons, New York, 1974.

[4] Barlow, N.S., Weinstein, S.J. Accurate closed-form solution of the SIR epidemic model, Physica D 408 (2020) 132540.

[5] Brauer, F., Castillo-Chavez, C. Mathematical Models in Population Biology and Epidemiology, Springer, New York, 2012.

[6] Cooper, I., Mondal A., Antonopoulos C. A SIR model assumption for the spread of COVID-19 in different communities. Chaos, Solitons and Fractals, 139 (2020) 110057

[7] Dzhalladova, I., Růžičková, M. Růžičková, V., Stability of the zero solution of nonlinear differential equations under the influence of white noise, Advances in Difference Equations, 143 (2015)

[8] Dubi, C., Atar, R. Modeling neutron count distribution in a subcritical core by stochastic differential equations, Ann. Nucl. Energy 111 (2018) 608-615.

[9] Gikhman, I.I. Stability of solutions of stochastic differential equations. In: Limit Theorems Statist. Inference, pp. 14–45. Izdat. “Fan”, Tashkent (1966). English transl.: Selected Transl. Statist. and Probability, vol. 12, pp. 125–154. Am. Math. Soc., Providence, 1973.

[10] Hethcote, H.W. Qualitative analyses of communicable disease models, Math. Biosci. 28 (1976) 335–356.

[11] Hethcote, H.W. Three basic epidemiological models, in Applied Mathematical Ecology, L. Gross, T. G. Hallam, and S. A. Levin, eds., Springer-Verlag, Berlin, 1989, pp. 119–144.

[12] Hethcote, H.W., The Mathematics of Infectious Diseases. SIAM Review 42 (2000) 599-653.

[13] Higham, D.J. Mean-square and asymptotic stability of numerical methods for stochastic ordinary differential equations, SIAM J. Numer. Anal., 38 (2000) 753–769

[14] Ji, C., Jiang, D., Shi, N. The Behavior of an SIR Epidemic Model with Stochastic Perturbation. Stoch. Anal. Appl., 30 (2012) 755–773.

[15] Junjie, C. An SIRS epidemic model, Appl. Math. J. Cinese Univ. Ser. B 19 (2004) 101-108.

[16] Kermack, W. O., and McKendrick, A. G. Contribution to the mathematical theory of epidemics, Proc. R. Soc. Lond. A 115 (1927) 700-721.

[17] Khasminskii, R. Stochastic stability of differential equations, 2nd. ed., Springer, Berlin, 2012.

[18] Kloeden, P.E., Platen, E. Numerical Solution of Stochastic Differential Equations. Springer-Verlag, Berlin (1999). Corrected Third Printing

[19] Kudo T, Kimura T, Inoue Y, Aman H, Hirata K. Stochastic modeling of self-evolving botnets with vulnerability discovery. Comput. Comm. 124 (2018) 101-110.

[20] Lahrouz A., Omari L., Kiouach D. Global analysis of a deterministic and stochastic nonlinear SIRS epidemic model, Nonlinear Analysis: Modelling and Control, 16 (2011) 59-76.

[21] Lahrouz, A., Settati, A., El Fatini, M., Tridane, A. The effect of a generalized nonlinear incidence rate on the stochastic SIS epidemic model, Math. Meth. Appl. Sci. 44 (2021) 1137-1146.

[22] Lima, L.S., Miranda, L.L.B. Price dynamics of the financial markets using the stochastic differential equation for a potential double well, Physica A 490 (2018) 828-833.

[23] Lu, Q. Stability of SIRS system with random perturbations. Physica A 388 (2009) 3677-3686.

[24] Mao, X. Stochastic stabilisation and destabilisation, Systems Control Lett. 23 (1994) 279-290

[25] Mena-Lorca, J., Hethcote, H.W. Dynamic models of infectious diseases as regulators of population sizes, J. Math. Biol. 30 (1992) 693-716.

[26] Murray, J.D. Mathematical Biology: I. An Introduction Springer, 2002

[27] Oroji, A., Omar, M., Yarahmadian, S. An Ito stochastic differential equations model for the dynamics of the MCF-7 breast cancer cell line treated by radiotherapy, J. Theor. Biol. 407 (2016) 128-137.

[28] Phan, T.A., Tian, J.P., Wang, B.X. Dynamics of cholera epidemic models in fluctuating environments, Stoch. Dyn. and Dynamics 21 (2021) Article number 2150011.

[29] Prodanov, D. Analytical parameter estimation of the SIR epidemic model. Applications to the COVID-19 pandemic, Entropy 23 (2021) 59.

[30] Rodrigues, H. S. Application of SIR epidemiological model: new trends, International Journal of Applied Mathematics and Informatics, 10 (2016) 92–97.

[31] Tocino, A., Senosiain, M.J. Mean-square stability analysis of numerical schemes for stochastic differential systems, J. Comput. Appl. Math., 236 (2012) 2660-2672.

[32] Tornatore, E., Buccellato, S.M., Vetro, P. Stability of a stochastic SIR system, Physica A, 354 (2005) 111-126.

[33] Vargas-De-León, C. On the global stability of SIS, SIR and SIRS epidemic models with standard incidence, Chaos, Solitons & Fractals 44 (2011) 1106–1110.

[34] Verdejo, H., Awekin, A., Kliemann, W., Becker, C. Modelling uncertainties in electrical power systems with stochastic differential equations, Int. J. Electr. Power Energy Syst. 113 (2019) 322-332.

[35] Xu, R.M., Guo, R.W. Pontryagin's Maximum Principle for Optimal Control Stochsatic SEIR models, Complexity 2020 (2020) Article number 6479087.