

MATHEMATICAL ANALYSIS OF AN “SIR” EPIDEMIC MODEL IN A CONTINUOUS REACTOR - DETERMINISTIC AND PROBABILISTIC APPROACHES

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ABSTRACT. In this paper, a mathematical dynamical system involving both deterministic (with or without delay) and stochastic “SIR” epidemic model with nonlinear incidence rate in a continuous reactor is considered. A profound qualitative analysis is given. It is proved that, for both deterministic models, if $\mathcal{R}_d > 1$, then the endemic equilibrium is globally asymptotically stable. However, if $\mathcal{R}_d \leq 1$, then the disease-free equilibrium is globally asymptotically stable. Concerning the stochastic model, the Feller’s test combined with the canonical probability method were used in order to conclude on the long-time dynamics of the stochastic model. The results improve and extend the results obtained for the deterministic model in its both forms. It is proved that if $\mathcal{R}_s > 1$, the disease is stochastically permanent with full probability. However, if $\mathcal{R}_s \leq 1$, then the disease dies out with full probability. Finally, some numerical tests are done in order to validate the obtained results.

1. Introduction

Epidemiological models capable to characterize and quantify the risks of epidemics as well as to optimize the means of controlling them. A founding approach in the 1920s was that of compartmental models, which divide the population into epidemiological classes such as individuals susceptible to infection (S), those who are infectious (I), and those who have acquired immunity as a result of healing (R). Since then, this approach has been used to model many diseases, and continues to be an active research topic.

In the same context, there is a long history of using chemostats to study bacterial and yeast evolution and of attempts to model the population dynamics of the predator-prey interaction of phages and bacteria. The study in [4]

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addresses the connection between evolution and population density in experimental microbial systems, specifically of bacterial viruses (phages) in chemostat populations. In [1], the authors tested the competition between the latent bacteriophage λ and its virulent mutant $\lambda cI857$ throughout experimental epidemics taking place in continuous cultures of *Escherichia coli*. They show that the observed transient selection for virulence and horizontal transmission can be fully explained within the framework of evolutionary epidemiology theory.

Various models have been developed which highlight (in particular) the crucial role played by the parameter \mathcal{R} , describing the average number of new infections due to a sick individual. As one can imagine, if this number is less than 1, then the epidemic will tend to go out, whereas it will be able to persist even to extend to the whole population if \mathcal{R} is great than 1.

Time delays (caused by a variety of factors) are usually used to model the fact that an individual may not be infectious until some time after becoming infected [11,12]. Moreover, it can sometimes be relevant to make a model probabilistic. Indeed, the most of works done previously was based on the assumption of a large population. When this is not the case, interactions between individuals are no longer uniform but have intrinsic randomness [14,17]. That's why, in this work, we will expose both deterministic (with or without time delay) and probabilistic version of the "SIR" epidemic model in a chemostat with nonlinear incidence rate.

This paper is organized as follows. In Section 2, a mathematical dynamical system involving deterministic "SIR" epidemic model with nonlinear incidence rate in a continuous reactor is considered. Two cases are studied, with or without time delay. A profound qualitative analysis is given. The analysis of the local and global stability of equilibrium points is carried out. It is proved that, for the deterministic model, if the reproduction number $\mathcal{R}_d > 1$, then the disease-persistence (endemic) equilibrium is globally asymptotically stable. However, if $\mathcal{R}_d \leq 1$, then the disease-free equilibrium is globally asymptotically stable. In Section 3, a stochastic dynamical system involving an "SIR" epidemic model with nonlinear incidence rate in a chemostat is considered. The Feller's test was combined with the canonical probability method in order to conclude on the long-time dynamics of the stochastic model. The results improve and extend the results obtained for the deterministic model in its both forms. It is proved that if the reproduction number $\mathcal{R}_s > 1$, the disease is stochastically permanent with full probability. However, if $\mathcal{R}_s \leq 1$, then the disease dies out with full probability. Finally, in Section 4, some numerical tests are done in order to validate the obtained results.

2. Deterministic mathematical models

In spite of their simplicity, compartmental models play a crucial role in epidemiology. Their study makes it possible to learn a great deal about the basic behaviours of the epidemiological systems and helps reasoning when one has to

deal with more complex models. Consider, here, the interaction between susceptible and infected individuals in a chemostat. Only susceptible individuals are introduced into the reactor with a constant rate D and an input individual number S_{in} (Fig. 1). We neglect all individuals natural mortality other than one caused by the disease concerned by this study and we take into account the dilution rate only. DS_{in} describes the rate of recruitment of susceptible (as input). γ is the rate at which infectious individuals are recovered. $(D + \gamma)^{-1}$ describes the average infection period.

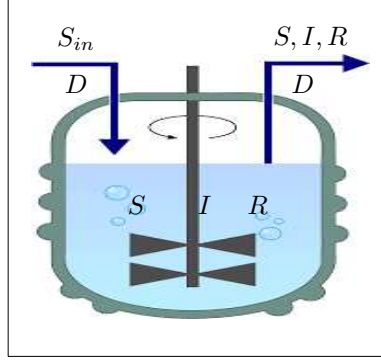


FIGURE 1. Epidemiological model in a continuous reactor

2.1. Deterministic non-delayed “SIR” mathematical model.

The deterministic non-delayed “SIR” mathematical model is given by the following three-dimensional dynamical system of ODEs:

$$(2.1) \quad \begin{cases} \dot{S} = D S_{in} - DS - \mu(I)S, \\ \dot{I} = \mu(I)S - (D + \gamma)I, \\ \dot{R} = \gamma I - DR, \end{cases}$$

with positive initial condition $(S_0, I_0, R_0) \in \mathbb{R}_+^3$. μ represents the saturated incidence rate and it is assumed to satisfy the following Assumption.

Assumption 1. μ is non-negative, $C^1(\mathbb{R}_+)$, increasing ($\mu'(I) > 0$) and concave ($\mu''(I) < 0$) function with $\mu(0) = 0$.

From the concavity assumption on μ , one can easily prove that

$$(2.2) \quad \mu'(I)I < \mu(I) \leq \mu'(0)I, \quad \forall I > 0.$$

Since the compartment R doesn't affect equations of S and I compartments, it is sufficient to consider only system (2.3).

$$(2.3) \quad \begin{cases} \dot{S} = D S_{in} - DS - \mu(I)S, \\ \dot{I} = \mu(I)S - (D + \gamma)I. \end{cases}$$

The closed non-negative cone \mathbb{R}_+^2 is positively invariant [5,6,8,9] by the system (2.3). More precisely,

Proposition 2.1.

- (1) For all initial condition (S_0, I_0) in \mathbb{R}_+^2 , the solution of (2.3) is bounded and has positive components and thus is defined for all $t > 0$.
- (2) System (2.3) admits a positive invariant attractor set of all solution given by $\Omega_1 = \{(S, I) \in \mathbb{R}_+^2 / S + I \leq S_{in}\}$.

Proof. (1) The positivity of the solution is proved by the fact that:

$$\text{Since } S = 0 \text{ then } \dot{S} = DS_{in} > 0 \text{ and if } I = 0, \text{ then } \dot{I} = 0.$$

Next we have to prove the boundedness of solutions of (2.3). By adding both equations of (2.3), one obtains, for $T = S + I - S_{in}$, a single equation for the total number of individuals:

$$\dot{T} = \dot{S} + \dot{I} = D(S_{in} - S - I) - \gamma I = -DT - \gamma I \leq -DT.$$

Then by applying the theory of differential inequalities [2], we obtain

$$(2.4) \quad S + I \leq S_{in} + (S_0 + I_0 - S_{in})e^{-Dt}.$$

Since both terms of the sum are positive, then the solution of (2.3) is bounded.

(2) The invariance of the attractor Ω_1 is simply deduced from inequality (2.4). \square

Given a disease, a fundamental question is whether it can spread in the population. This amounts to calculating the average number of individuals that an infectious individual can infect, as long as it is contagious. This number is called the basic reproduction number [7, 20]. In our case, the reproduction number for the deterministic model (2.3), denoted by \mathcal{R}_d , is given by:

$$(2.5) \quad \mathcal{R}_d = \frac{\mu'(0)S_{in}}{D + \gamma}.$$

Let us establish the equilibrium points of the system (2.3). Define $E^* = (S^*, I^*)$ as an endemic equilibrium where $S^* > 0$ and $I^* > 0$ satisfying

$$(2.6) \quad \begin{cases} DS_{in} = DS^* + \mu(I^*)S^*, \\ \mu(I^*)S^* = (D + \gamma)I^*. \end{cases}$$

Regarding the characteristic equations and characteristic roots of the proposed model (2.3), it is easy to prove the following proposition.

Proposition 2.2. (1) If $\mathcal{R}_d \leq 1$, then the system (2.3) admits a disease-free equilibrium $\bar{E} = (S_{in}, 0)$ as the unique equilibrium.

- (2) If $\mathcal{R}_d > 1$, then the system (2.3) admits only two equilibrium: a unique disease-free equilibrium $\bar{E} = (S_{in}, 0)$ and a unique disease-persistence (endemic) equilibrium $E^* = (S^*, I^*)$.

The value of \mathcal{R}_d has a great importance in determining whether there exists an endemic equilibrium or not (as in [3], Theorem 2.3).

Theorem 2.3. (1) *If $\mathcal{R}_d < 1$, then the disease-free equilibrium \bar{E} is locally asymptotically stable.*

(2) *If $\mathcal{R}_d > 1$, then the disease-free equilibrium \bar{E} is unstable and the disease-persistence equilibrium E^* is locally asymptotically stable.*

Proof. The Jacobian matrix at a point (S, I) is given by:

$$J = \begin{pmatrix} -D - \mu(I) & -\mu'(I)S \\ \mu(I) & \mu'(I)S - (D + \gamma) \end{pmatrix}.$$

The Jacobian matrix evaluated at \bar{E} is then given by:

$$\bar{J} = \begin{pmatrix} -D & -\mu'(0)S_{in} \\ 0 & \mu'(0)S_{in} - (D + \gamma) \end{pmatrix} = \begin{pmatrix} -D & -\mu'(0)S_{in} \\ 0 & (D + \gamma)(\mathcal{R}_d - 1) \end{pmatrix}.$$

\bar{J} admits two eigenvalues given by $\lambda_1 = -D < 0$ and $\lambda_2 = (D + \gamma)(\mathcal{R}_d - 1)$. It follows that

- If $\mathcal{R}_d < 1$, then $\lambda_2 < 0$ and \bar{E} is then locally asymptotically stable,
- If $\mathcal{R}_d > 1$, then $\lambda_2 > 0$ and \bar{E} is unstable.

The Jacobian matrix evaluated at E^* is then given by:

$$J^* = \begin{pmatrix} -D - \mu(I^*) & -\mu'(I^*)S^* \\ \mu(I^*) & \mu'(I^*)S^* - (D + \gamma) \end{pmatrix}.$$

The associated characteristic polynomial to J^* is given by

$$P(\lambda) = \lambda^2 + A_1\lambda + A_0,$$

where A_0 and A_1 are given by

$$\begin{cases} A_0 = (D + \gamma)(D + \mu(I^*)) - D\mu'(I^*)S^* \\ \quad = (D + \gamma)\mu(I^*) + \frac{(D + \gamma)}{\mu(I^*)}(\mu(I^*) - \mu'(I^*)I^*), \\ A_1 = 2D + \gamma + \mu(I^*) - \mu'(I^*)S^* \\ \quad = D + \mu(I^*) + \frac{(D + \gamma)}{\mu(I^*)}(\mu(I^*) - \mu'(I^*)I^*). \end{cases}$$

From (2.2), it follows that $A_0 > 0$ and $A_1 > 0$ and thus using Routh-Hurwitz criterion, both eigenvalues have negative real parts. Then if $\mathcal{R}_d > 1$, E^* exists and it is always locally asymptotically stable. This completes the proof. \square

Lemma 2.4. *System (2.3) has no periodic orbits nor polycycles inside Ω_1 .*

Proof. Denote the right-hand side of the system (2.3) by

$$f(S, I) = (f_1(S, I), f_2(S, I))^T$$

and construct the Dulac function $\Upsilon(S, I) = \frac{1}{S\mu(I)}$. Then we obtain

$$\operatorname{div}(\Upsilon f(S, I)) = \frac{\partial(\Upsilon f_1)}{\partial S} + \frac{\partial(\Upsilon f_2)}{\partial I} = -\frac{DS_{in}}{S^2\mu(I)} - (D + \gamma)\frac{\mu(I) - \mu'(I)I}{S\mu^2(I)}.$$

$\operatorname{div}(\Upsilon f(S, I)) < 0$ since μ satisfies (2.2). Then the system (2.3) has no periodic orbits nor polycycles inside Ω_1 . \square

The global stability of the disease-free equilibrium \bar{E} and the disease-persistence equilibrium E^* are given in the following theorem.

Theorem 2.5. (1) *If $\mathcal{R}_d \leq 1$, then the disease-free equilibrium \bar{E} is globally asymptotically stable.*
 (2) *If $\mathcal{R}_d > 1$, then the disease-persistence (endemic) equilibrium E^* is globally asymptotically stable.*

Proof. By Lemma 2.4, the system (2.3) has no periodic orbits nor polycycles inside Ω_1 . Regarding the nature of trivial equilibria, the Poincaré-Bendixon Theorem [16, 19] allows to conclude (for other applications, see [5, 8, 9]). \square

2.2. Deterministic delayed ‘‘SIR’’ mathematical model.

In many situations an individual may not be infectious until some time after becoming infected. Since the system (2.3) does not take proper account of the delays, it can be tempting to simply introduce a time delay in one or more terms of the right-hand side of these equations. Here, we consider the deterministic delayed ‘‘SIR’’ mathematical model given, for $\tau > 0$, by the following dynamical system of ODEs:

$$(2.7) \quad \begin{cases} \dot{S}(t) = D S_{in} - DS(t) - \mu(I(t - \tau))S(t), \\ \dot{I}(t) = \mu(I(t - \tau))S(t) - (D + \gamma)I(t) \end{cases}$$

with positive initial conditions given as functions

$$(2.8) \quad S(\eta) = \theta_1(\eta), I(\eta) = \theta_2(\eta), \eta \in [-\tau, 0],$$

where θ_1 and θ_2 are positive continuous functions defined on $[-\tau, 0]$.

System (2.7) always admits a disease-free equilibrium $\bar{E} = (S_{in}, 0)$ and a unique endemic equilibrium $E^* = (S^*, I^*)$ satisfying

$$(2.9) \quad \begin{cases} DS_{in} = DS^* + \mu(I^*)S^*, \\ \mu(I^*)S^* = (D + \gamma)I^*. \end{cases}$$

As in Section 2, it is easy to see that:

Proposition 2.6.

- (1) *For all initial condition in \mathbb{R}_+^2 , the solution of the system (2.7) is bounded and has positive components and thus is defined for all $t > 0$.*
- (2) *System (2.7) admits Ω_1 as a positive invariant attractor set of all solution.*

Proof. The prove is similar to the one of Proposition 2.1 and it is omitted here. \square

The basic reproduction number for the delayed deterministic model (2.7) is the same as the one given in expression (2.5)

$$\mathcal{R}_d = \frac{\mu'(0)S_{in}}{D + \gamma}.$$

The value of \mathcal{R}_d always determines which equilibrium is stable (as in Theorem 2.3).

Theorem 2.7. (1) *If $\mathcal{R}_d < 1$, then the disease-free equilibrium \bar{E} is locally asymptotically stable.*
 (2) *If $\mathcal{R}_d > 1$, then the disease-persistence equilibrium E^* is locally asymptotically stable.*

Proof. The Jacobian matrix of the linearised model of (2.7) at a point (S, I) is given by:

$$J = \begin{pmatrix} -D - \mu(I) & -\mu'(I)Se^{-\lambda\tau} \\ \mu(I) & \mu'(I)Se^{-\lambda\tau} - (D + \gamma) \end{pmatrix}.$$

The characteristic equation of the linearised model of (2.7) is given by:

$$(2.10) \quad P(\lambda) = \begin{vmatrix} -D - \mu(I) - \lambda & -\mu'(I)Se^{-\lambda\tau} \\ \mu(I) & \mu'(I)Se^{-\lambda\tau} - (D + \gamma) - \lambda \end{vmatrix}.$$

The associated characteristic equation (2.10) evaluated at \bar{E} is then given by:

$$\bar{P}(\lambda) = (\lambda + D)\left(\lambda + D + \gamma - S_{in}\mu'(0)e^{-\lambda\tau}\right).$$

For $\lambda = 0$, $\bar{P}(0) = D(D + \gamma - S_{in}\mu'(0)) = D(D + \gamma)(1 - \mathcal{R}_d)$ therefore 0 can't be a root of \bar{P} for $\mathcal{R}_d < 1$.

Next, we show that the characteristic equation has no eigenvalues with non-negative real parts. Let $\lambda = a + ib$. Then

$$Re(\lambda + D + \gamma - S_{in}\mu'(0)e^{-\lambda\tau}) = a + D + \gamma - S_{in}\mu'(0)e^{-a\tau} \cos(\tau b) = 0.$$

One deduces that

$$a + D + \gamma = S_{in}\mu'(0)e^{-a\tau} \cos(\tau b)$$

or also

$$1 + \frac{a}{D + \gamma} = \mathcal{R}_d e^{-a\tau} \cos(\tau b).$$

By way of contradiction, assume that $a > 0$ then

$$\left|1 + \frac{a}{D + \gamma}\right| > 1 \text{ and } \left|\mathcal{R}_d e^{-a\tau} \cos(\tau b)\right| < 1,$$

which is impossible. Therefore, for $\mathcal{R}_d < 1$, all roots of \bar{P} must have negative real parts ($a < 0$), and hence the disease-free equilibrium \bar{E} is locally asymptotically stable for all $\tau > 0$.

If $\mathcal{R}_d > 1$, the associated characteristic polynomial (2.10) evaluated at E^* is then given by:

$$\begin{aligned} P^*(\lambda) &= (\lambda + D + \mu(I^*)) (\lambda + D + \gamma - \mu'(I^*)S^*e^{-\lambda\tau}) \\ &\quad + \mu(I^*)\mu'(I^*)S^*e^{-\lambda\tau} \\ &= (\lambda + D + \mu(I^*)) (\lambda + D + \gamma) - (\lambda + D + \mu(I^*))\mu'(I^*)S^*e^{-\lambda\tau} \\ &\quad + \mu(I^*)\mu'(I^*)S^*e^{-\lambda\tau} \\ &= (\lambda + D + \mu(I^*)) (\lambda + D + \gamma) - (\lambda + D)\mu'(I^*)S^*e^{-\lambda\tau}. \end{aligned}$$

Assume that $P^*(\lambda) = 0$ then

$$(\lambda + D + \mu(I^*)) (\lambda + D + \gamma) = (\lambda + D)\mu'(I^*)S^*e^{-\lambda\tau}$$

therefore $(1 + \frac{\mu(I^*)}{\lambda + D})(\lambda + D + \gamma) = \mu'(I^*)S^*e^{-\lambda\tau}$.

For $\tau > 0$ we have $P^*(0) = (D + \gamma)(D + \mu(I^*)) - DS^*\mu'(I^*)$.

From the second equation of (2.9), we have

$$P^*(0) = (D + \gamma)(D + \mu(I^*)) - D\frac{\mu'(I^*)(D + \gamma)I^*}{\mu(I^*)}.$$

Using (2.2), we obtain

$$P^*(0) > (D + \gamma)(D + \mu(I^*)) - D(D + \gamma) = (D + \gamma)\mu(I^*) > 0$$

then $\lambda = 0$ can't be a root of P^* .

Next, we show that the characteristic equation has no eigenvalues with non-negative real parts. By way of contradiction, assume that there is one eigenvalue λ with nonnegative real part. Then

$$(2.11) \quad \left| \left(1 + \frac{\mu(I^*)}{\lambda + D}\right)(\lambda + D + \gamma) \right| = \left| \mu'(I^*)S^*e^{-\lambda\tau} \right|.$$

Note that $\left|1 + \frac{\mu(I^*)}{\lambda + D}\right| > 1$ and $|\lambda + D + \gamma| > D + \gamma$. Therefore

$$\left| \left(1 + \frac{\mu(I^*)}{\lambda + D}\right)(\lambda + D + \gamma) \right| > D + \gamma.$$

Now by considering the right hand side of (2.11), and since $\tau, \operatorname{Re}(\lambda) > 0$ one has $\left| \mu'(I^*)S^*e^{-\lambda\tau} \right| < \mu'(I^*)S^*$. From the second equation of (2.9), we have

$$\left| \mu'(I^*)S^*e^{-\lambda\tau} \right| < \frac{\mu'(I^*)(D + \gamma)I^*}{\mu(I^*)}.$$

By using (2.2), we obtain $\left| \mu'(I^*)S^*e^{-\lambda\tau} \right| < D + \gamma$. This leads to a contradiction. Therefore, all roots of P^* must have negative real parts, and hence the disease-free equilibrium E^* is locally asymptotically stable for all $\tau > 0$. This completes the proof. \square

The global stability of the disease-free equilibrium \bar{E} and the disease-persistence equilibrium E^* are given in the following theorem.

- Theorem 2.8.** (1) *If $\mathcal{R}_d \leq 1$, then the disease-free equilibrium \bar{E} is globally asymptotically stable.*
 (2) *If $\mathcal{R}_d > 1$, then the disease-persistence equilibrium E^* is globally asymptotically stable.*

Proof. Let (S, I) to be a solution of the system (2.7) and define the Lyapunov function

$$V_1(t) = S(t) + I(t) - S^* \ln\left(\frac{S}{S^*}\right) - \int_{I^*}^{I(t)} \frac{\mu(I^*)}{\mu(\eta)} d\eta \\ + (D + \gamma)I^* \int_0^\tau \left[\frac{\mu(I(t-\theta))}{\mu(I^*)} - 1 - \ln\left(\frac{\mu(I(t-\theta))}{\mu(I^*)}\right) \right] d\theta.$$

The equilibrium E^* is the only internal stationary point and minimum point of $V_1(t)$, and $V_1(t) \rightarrow +\infty$ at the boundary of the positive quadrant. Consequently, E^* is the global minimum point, and the function is bounded from below. Next we denote $I(t - \tau)$ by I_τ , the derivative of $V_1(t)$ along solution of the system (2.7) is given by

$$\dot{V}_1 = \left(1 - \frac{S^*}{S}\right)\dot{S} + \left(1 - \frac{\mu(I^*)}{\mu(I)}\right)\dot{I} + (D + \gamma)I^* \int_0^\tau \frac{d}{dt} \left[\frac{\mu(I(t-\theta))}{\mu(I^*)} - 1 - \ln\left(\frac{\mu(I(t-\theta))}{\mu(I^*)}\right) \right] d\theta \\ = \left(1 - \frac{S^*}{S}\right)(D S_{in} - DS - \mu(I(t-\tau))S) + \left(1 - \frac{\mu(I^*)}{\mu(I)}\right)(\mu(I(t-\tau))S - (D + \gamma)I) - (D + \gamma)I^* \int_0^\tau \frac{d}{d\theta} \left[\frac{\mu(I(t-\theta))}{\mu(I^*)} - 1 - \ln\left(\frac{\mu(I(t-\theta))}{\mu(I^*)}\right) \right] d\theta \\ = \left(1 - \frac{S^*}{S}\right)(DS^* + \mu(I^*)S^* - DS - \mu(I_\tau)S) + \left(1 - \frac{\mu(I^*)}{\mu(I)}\right) \times (\mu(I_\tau)S - (D + \gamma)I) - (D + \gamma)I^* \left[\frac{\mu(I(t-\theta))}{\mu(I^*)} - 1 - \ln\left(\frac{\mu(I(t-\theta))}{\mu(I^*)}\right) \right]_0^\tau \\ = \frac{S - S^*}{S(t)}(D(S^* - S) + \mu(I^*)S^* - \mu(I_\tau)S) + \frac{\mu(I) - \mu(I^*)}{\mu(I)} \times (\mu(I_\tau)S - (D + \gamma)I) - (D + \gamma)I^* \left[\frac{\mu(I_\tau)}{\mu(I^*)} - \frac{\mu(I)}{\mu(I^*)} - \ln\left(\frac{\mu(I_\tau)}{\mu(I)}\right) \right] \\ = -D \frac{(S - S^*)^2}{S} + \frac{S - S^*}{S}(\mu(I^*)S^* - \mu(I_\tau)S) + \frac{\mu(I) - \mu(I^*)}{\mu(I)}$$

$$\times \left(\mu(I_\tau)S - (D + \gamma)I \right) - (D + \gamma)I^* \left[\frac{\mu(I_\tau)}{\mu(I^*)} - \frac{\mu(I)}{\mu(I^*)} - \ln \left(\frac{\mu(I_\tau)}{\mu(I)} \right) \right].$$

Here we have used the first equation of (2.9). In the next we use the second equation of (2.9) to obtain:

$$\begin{aligned} \dot{V}_1 &= -D \frac{(S - S^*)^2}{S} + (D + \gamma)I^* \left(1 - \frac{S^*}{S} \right) - \mu(I_\tau)(S - S^*) + \frac{\mu(I) - \mu(I^*)}{\mu(I)} \\ &\quad \times \left(\mu(I_\tau)S - (D + \gamma)I \right) \\ &\quad - (D + \gamma)I^* \left[\frac{\mu(I_\tau)}{\mu(I^*)} - \frac{\mu(I)}{\mu(I^*)} - \ln \left(\frac{S\mu(I_\tau)}{S^*\mu(I)} \right) - \ln \left(\frac{S^*}{S} \right) \right] \\ &= -D \frac{(S - S^*)^2}{S} - (D + \gamma)I^* \left(\frac{S^*}{S} - 1 - \ln \left(\frac{S^*}{S} \right) \right) - \mu(I_\tau)(S - S^*) \\ &\quad + \frac{\mu(I) - \mu(I^*)}{\mu(I)} \left(\mu(I_\tau)S - (D + \gamma)I \right) \\ &\quad - (D + \gamma)I^* \left[\frac{\mu(I_\tau)}{\mu(I^*)} - \frac{\mu(I)}{\mu(I^*)} - \ln \left(\frac{S\mu(I_\tau)}{S^*\mu(I)} \right) \right]. \end{aligned}$$

We compute the term containing $\mu(I_\tau)$ separately

$$-\mu(I_\tau) \left(S - S^* - S \left(1 - \frac{\mu(I^*)}{\mu(I)} \right) + (D + \gamma) \frac{I^*}{\mu(I^*)} \right) = -\mu(I_\tau)(D + \gamma)I^* \frac{S}{S^*\mu(I)}$$

then we get

$$\begin{aligned} \dot{V}_1 &= -D \frac{(S - S^*)^2}{S} - (D + \gamma)I^* \left(\frac{S^*}{S} - 1 - \ln \left(\frac{S^*}{S} \right) \right) - (D + \gamma)I^* \frac{S\mu(I_\tau)}{S^*\mu(I)} \\ &\quad - (D + \gamma)I \left(1 - \frac{\mu(I^*)}{\mu(I)} \right) - (D + \gamma)I^* \left[-\frac{\mu(I)}{\mu(I^*)} - \ln \left(\frac{S\mu(I_\tau)}{S^*\mu(I)} \right) \right] \\ &= -D \frac{(S - S^*)^2}{S} - (D + \gamma)I^* \left(\frac{S^*}{S} - 1 - \ln \left(\frac{S^*}{S} \right) \right) - (D + \gamma)I^* \\ &\quad \times \left[\frac{S\mu(I_\tau)}{S^*\mu(I)} - 1 - \ln \left(\frac{S\mu(I_\tau)}{S^*\mu(I)} \right) \right] - (D + \gamma)I^* \left(\frac{I}{I^*} \left(1 - \frac{\mu(I^*)}{\mu(I)} \right) + 1 - \frac{\mu(I)}{\mu(I^*)} \right) \\ &= -D \frac{(S - S^*)^2}{S} - (D + \gamma)I^* \left(\frac{S^*}{S} - 1 - \ln \left(\frac{S^*}{S} \right) \right) - (D + \gamma)I^* \\ &\quad \times \left[\frac{S\mu(I_\tau)}{S^*\mu(I)} - 1 - \ln \left(\frac{S\mu(I_\tau)}{S^*\mu(I)} \right) \right] + (D + \gamma)I^* \frac{\mu(I^*) - \mu(I)}{\mu(I)} \left(\frac{I}{I^*} - \frac{\mu(I)}{\mu(I^*)} \right). \end{aligned}$$

Using (2.2), $I \mapsto \frac{\mu(I)}{I}$ is a decreasing function and then $\frac{\mu(I)}{\mu(I^*)} \geq \frac{I}{I^*}$ for all $0 \leq I \leq I^*$ and $\frac{\mu(I)}{\mu(I^*)} \leq \frac{I}{I^*}$ for all $I \geq I^*$. Then $\frac{\mu(I^*) - \mu(I)}{\mu(I)} \left(\frac{I}{I^*} - \frac{\mu(I)}{\mu(I^*)} \right) \leq 0$ for all $I \geq 0$.

Moreover, $x - 1 - \ln(x) > 0$ for all $x > 0$ thus $\frac{S^*}{S} - 1 - \ln(\frac{S^*}{S}) > 0, \forall S > 0$ and $\frac{S\mu(I_\tau)}{S^*\mu(I)} - 1 - \ln(\frac{S\mu(I_\tau)}{S^*\mu(I)}) > 0, \forall I_\tau > 0, S > 0$. Since all parameters of the model are non-negative, it follows that $\dot{V}_1 \leq 0$. Therefore, all the conditions of [12, Corollary 5.2, p. 30] are satisfied. This proves that E^* is globally asymptotically stable for all $\tau > 0$ where $\mathcal{R}_d > 1$.

Let (S, I) to be a solution of the system (2.7) and define the Lyapunov function

$$V_2(t) = S(t) + I(t) - S_{in} \ln\left(\frac{S}{S_{in}}\right) + S_{in} \int_0^\tau \mu(I(t-\theta))d\theta.$$

Since the equilibrium \bar{E} is the minimum point of $V_2(t)$. Consequently, \bar{E} is the global minimum point. The derivative of $V_2(t)$ along solution of the system (2.7) is given by

$$\begin{aligned} \dot{V}_2 &= \left(1 - \frac{S_{in}}{S}\right)\dot{S} + \dot{I} + S_{in} \int_0^\tau \frac{d}{dt}\mu(I(t-\theta))d\theta \\ &= \left(1 - \frac{S_{in}}{S}\right)\left(D S_{in} - DS - \mu(I_\tau)S\right) + \mu(I_\tau)S - (D + \gamma)I \\ &\quad - S_{in} \int_0^\tau \frac{d}{d\theta}\mu(I(t-\theta))d\theta \\ &= \frac{S - S_{in}}{S}\left(D(S_{in} - S) - \mu(I_\tau)S\right) + \mu(I_\tau)S - (D + \gamma)I - S_{in}\mu(I_\tau) \\ &\quad + S_{in}\mu(I) \\ &= -\frac{D(S_{in} - S)^2}{S} + (D + \gamma)\left(\frac{S_{in}}{D + \gamma}\mu(I) - I\right). \end{aligned}$$

From (2.2), we have

$$\begin{aligned} \dot{V}_2(t) &\leq -\frac{D(S_{in} - S)^2}{S} + (D + \gamma)\left(\frac{S_{in}}{D + \gamma}\mu'(0) - 1\right)I \\ &= -\frac{D(S_{in} - S)^2}{S} + (D + \gamma)(\mathcal{R}_d - 1)I. \end{aligned}$$

Since all parameters of the model are non-negative and $\mathcal{R}_d < 1$, it follows that $\dot{V}_2 \leq 0$. Therefore, again, all the conditions of [12, Corollary 5.2, p. 30] are satisfied. This proves that $\{\bar{E}\}$ is globally asymptotically stable for any $\tau > 0$ when $\mathcal{R}_d < 1$.

Now assume that $\mathcal{R}_d = 1$. Therefor $\dot{V}_2 = 0$ means that $S = S_{in}$ and the largest compact invariant set in $\{(S, I) \in \Omega : \dot{V}_2 = 0\}$ is the singleton $\{\bar{E}\}$. Therefore, by the Lasalle’s invariance principle (see, for instance, [12, Theorem 5.3, p. 30]), $\{\bar{E}\}$ is globally asymptotically stable (for other applications, see [5, 8, 9]). \square

3. Stochastic “SIR” epidemic mathematical model and results

The epidemiological models usually studied are deterministic models. However, while they may be more difficult to study and less accurate, probabilistic models are a natural way of modelling the evolution of an epidemic: each individual has a certain probability of being infected with the disease. An important part of the study of these stochastic problems will be to determine if, when the size of the population increases, they converge towards a deterministic problem. In this section, we will introduce a stochastic model of spread of an epidemic. We will also merge susceptible compartment with the recovered one. The proposed stochastic mathematical model is then given by

$$(3.1) \quad \begin{cases} dS = [D(S_{in} - S) + \gamma I - \mu(I)S] dt - \sigma\mu(I)S dW, \\ dI = [\mu(I)S - (D + \gamma)I] dt + \sigma\mu(I)S dW \end{cases}$$

with initial condition $(S_0, I_0) \in \mathbb{R}_+^2$ and $\sigma > 0$.

A nonlinear incidence rate plays an important role in the evolution of infectious diseases, because epidemic models described by nonlinear incidence rates may be more suitable and realistic, which also exhibit much richer dynamics. The standard incidence rate $\bar{\mu}I$ was proposed and used in many references, for example, [13, 15, 17, 22]. The classical Monod nonlinear saturated incidence rate $\frac{\bar{\mu}I}{k+I}$ was also used in some works, see for example, [14, 21].

In this work, the Monod function will be used to express transmission rate of infection from infected individuals to susceptible ones.

Assumption 2. The saturated incidence rate μ is given by

$$(3.2) \quad \mu(I) = \frac{\bar{\mu}I}{k+I}.$$

Where $\bar{\mu}$ is the maximum transmission rate of infection and k is the Michaelis-Menten (or half-saturation) constant.

Note that $W(\cdot)$ is a stochastic process known as the standard Wiener process having the following properties:

- $W(0) = 0$.
- $W(\cdot)$ is continuous with probability 1.
- The process $\{W(t)\}_{t \geq 0}$ has stationary, independent increments.
- The increment $W(t+s) - W(s)$ has the normal $(0, t)$ distribution.
- W has an intensity $\frac{\sigma^2 \bar{\mu}^2}{k^2}$.

The positivity and boundedness of the solution of the system (3.1) is given hereafter.

Proposition 3.1.

- (1) For all initial condition in \mathbb{R}_+^2 , the solution of the system (3.1) is bounded and has positive components and thus is defined for all $t > 0$.

- (2) *System (3.1) admits $\Omega_2 = \{(S, I) \in \mathbb{R}_+^2 / S + I = S_{in}\}$ as a positive invariant attractor set of all solution.*

Proof. The proof is similar to Proposition 2.1 and then it is omitted here. \square

The basic reproduction number for the stochastic model (3.1) is denoted by \mathcal{R}_s and it is given by:

$$(3.3) \quad \mathcal{R}_s = \frac{\bar{\mu}S_{in}}{k(D + \gamma)} - \frac{\sigma^2 S_{in}^2}{2(D + \gamma)}.$$

The solutions of the system (3.1) are exponentially convergent towards the set Ω_2 and we are interested in the asymptotic behaviour of these solutions. It is enough to restrict the study of the asymptotic behaviour of the system (3.1) to Ω_2 . In fact the asymptotic behaviour of the solutions of the restriction of (3.1) on Ω_2 will be informative for the complete system and is given by:

$$(3.4) \quad dI = \left[\frac{\bar{\mu}I}{k + I}(S_{in} - I) - (D + \gamma)I \right] dt + \sigma \frac{\bar{\mu}I}{k + I}(S_{in} - I) dW,$$

where the initial condition $I_0 \in (0, S_{in})$.

Assumption 3.

$$(3.5) \quad dX(t) = \phi(X(t))dt + \psi(X(t))dW(t),$$

such that (i) $\forall X \in Y, \psi^2(X) > 0$ and (ii) $\forall X \in Y, \exists \varepsilon > 0, \int_{X-\varepsilon}^{X+\varepsilon} \frac{1+|\phi(r)|}{\psi^2(r)} dr < +\infty$ where $X(0) \in \mathbb{R}_+, Y = (\tau, \eta)$ and $-\infty \leq \tau < \eta \leq +\infty$.

Lemma 3.2. *If Assumption 3 is satisfied, then let $X(t)$ be a non-explosive solution of (3.5) in $Y = (\tau, \eta)$. Then for all constant $c \in Y$, the scale function is given by*

$$\zeta(x) = \int_c^x \exp\left(-2 \int_c^u \frac{\phi(r)}{\psi^2(r)} dr\right) du.$$

It follows that if $\zeta(\tau^+) > -\infty, \zeta(\tau^-) = +\infty$, then

$$\mathbb{P}\left(\lim_{t \rightarrow +\infty} X(t) = \tau\right) = \mathbb{P}\left(\sup_{t \geq 0} X(t) < \eta\right) = 1.$$

Lemma 3.3. *Suppose that (3.5) admits, for $\psi(X(\cdot)) \equiv 1$, a non-explosive solution in finite time which is unique in the sense of probability law. Consider the two functions $\alpha(x)$ and $\beta(x)$ given by*

$$\alpha(x) = \int_0^x \exp\left(2 \int_0^u \phi(r) dr\right) du, \quad \beta(x) = \int_0^x \exp\left(-2 \int_0^u \phi(r) dr\right) du$$

with $\alpha(+\infty) < +\infty, \alpha(-\infty) = -\infty, \beta(+\infty) = +\infty, \beta(-\infty) = -\infty$. Then

$$\forall \Gamma \in \mathbb{R}, \quad \mathbb{P}\left(\lim_{t \rightarrow +\infty} X(t) < \Gamma\right) = 1,$$

which means that $X(t) \rightarrow -\infty$ in probability meaning.

Proof. See references [10] and [18] for the proofs of both Lemma 3.2 and Lemma 3.3. \square

Hereafter, we give one main result in the following theorem.

Theorem 3.4. *Let $I(t)$ to be the solution of (3.4) such that the initial condition $I_0 \in (0, S_{in})$. Then, if $\mathcal{R}_s < 1$ therefore*

$$\mathbb{P}\left(\lim_{t \rightarrow +\infty} I(t) = 0\right) = 1.$$

Proof. Using Lemma 3.2 and (3.4) and by considering the functions

$$\phi(I) = \left[\frac{\bar{\mu}I}{k+I}(S_{in} - I) - (D + \gamma)I \right] \quad \text{and} \quad \psi(I) = \sigma \frac{\bar{\mu}I}{k+I}(S_{in} - I),$$

one can easily obtain

$$\begin{aligned} 2 \int_c^u \frac{\phi(r)}{\psi^2(r)} dr &= \frac{2}{\sigma^2 \bar{\mu}^2} \left[\int_c^u \frac{kS_{in}\bar{\mu} + \bar{\mu}S_{in} + (S_{in}^2 - k^2)(D + \gamma)}{S_{in}^2(S_{in} - r)} dr \right. \\ &\quad \left. + \int_c^u \frac{k\bar{\mu}S_{in} - k^2(D + \gamma)}{S_{in}^2 r} dr - \int_c^u \frac{(k + S_{in})^2(D + \gamma)}{S_{in}(S_{in} - r)^2} dr \right] \\ &= \frac{-2}{\sigma^2 \bar{\mu}^2} \left[\frac{kS_{in}\bar{\mu} + \bar{\mu}S_{in} + (S_{in}^2 - k^2)(D + \gamma)}{S_{in}^2} \ln(S_{in} - u) \right. \\ &\quad \left. - \frac{k\bar{\mu}S_{in} - k^2(D + \gamma)}{S_{in}^2} \ln(u) + \frac{(k + S_{in})^2(D + \gamma)}{S_{in}(S_{in} - u)} \right] + C, \end{aligned}$$

where C is a constant.

An integral calculation permits to obtain

$$\begin{aligned} \zeta(I) &= \exp(-C) \int_c^I \exp\left(\frac{2}{\sigma^2 \bar{\mu}^2} \left[\frac{kS_{in}\bar{\mu} + \bar{\mu}S_{in} + (S_{in}^2 - k^2)(D + \gamma)}{S_{in}^2} \ln(S_{in} - u) \right. \right. \\ &\quad \left. \left. - \frac{k\bar{\mu}S_{in} - k^2(D + \gamma)}{S_{in}^2} \ln(u) + \frac{(k + S_{in})^2(D + \gamma)}{S_{in}(S_{in} - u)} \right] \right) du \\ &= \exp(-C) \int_c^I \left[u^{-\frac{2}{\sigma^2 \bar{\mu}^2} \frac{k\bar{\mu}S_{in} - k^2(D + \gamma)}{S_{in}^2}} \right. \\ &\quad \times (S_{in} - u)^{\frac{2}{\sigma^2 \bar{\mu}^2} \frac{kS_{in}\bar{\mu} + \bar{\mu}S_{in} + (S_{in}^2 - k^2)(D + \gamma)}{S_{in}^2}} \\ &\quad \left. \times \exp\left(\frac{2}{\sigma^2 \bar{\mu}^2} \frac{(k + S_{in})^2(D + \gamma)}{S_{in}(S_{in} - u)} \right) \right] du. \end{aligned}$$

By injecting $I = S_{in}^-$ and $\rho = \frac{1}{(S_{in}-u)}$ in the expression of $\zeta(I)$, we obtain

$$\begin{aligned} \zeta(S_{in}^-) &\geq \exp(-C) \times u \frac{2k^2(D+\gamma)}{\sigma^2\bar{\mu}^2 S_{in}^2} - \frac{2k}{S_{in} \sigma^2\bar{\mu} S_{in}} \\ &\times \int_{1/(S_{in}-c)}^{+\infty} \left[\rho \frac{2}{\sigma^2\bar{\mu}^2} \frac{kS_{in}\bar{\mu} + \bar{\mu}S_{in} + (S_{in}^2 - k^2)(D+\gamma)}{S_{in}^2} - 2 \right. \\ &\times \left. \exp\left(2 \frac{(k+S_{in})^2(D+\gamma)}{\sigma^2\bar{\mu}^2 S_{in}} \rho\right) \right] d\rho \\ &= +\infty. \end{aligned}$$

Note that if $\mathcal{R}_s < 1$ therefore $\frac{2}{\sigma^2\bar{\mu}^2} \frac{k\bar{\mu}S_{in} - k^2(D+\gamma)}{S_{in}^2} < 1$. Let $I = 0^+$, then we obtain

$$\begin{aligned} -\zeta(0^+) &\leq \exp(-C) \times S_{in} \frac{2k\bar{\mu} + \bar{\mu} + S_{in}(D+\gamma)}{\sigma^2\bar{\mu}^2 S_{in}} \times (S_{in}-u) \frac{2k^2(D+\gamma)}{\sigma^2\bar{\mu}^2 S_{in}^2} \\ &\times \exp\left(\frac{2}{\sigma^2\bar{\mu}^2} \frac{(k+S_{in})^2(D+\gamma)}{S_{in}(S_{in}-u)}\right) \int_0^c u \frac{2}{\sigma^2\bar{\mu}^2} \frac{k\bar{\mu}S_{in} - k^2(D+\gamma)}{S_{in}^2} du \\ &\leq \exp(-C) \times S_{in} \frac{2k\bar{\mu} + \bar{\mu} + S_{in}(D+\gamma)}{\sigma^2\bar{\mu}^2 S_{in}} \times (S_{in}-u) \frac{2k^2(D+\gamma)}{\sigma^2\bar{\mu}^2 S_{in}^2} \\ &\times \exp\left(\frac{2}{\sigma^2\bar{\mu}^2} \frac{(k+S_{in})^2(D+\gamma)}{S_{in}(S_{in}-u)}\right) \\ &\times \frac{1}{1 - \frac{2}{\sigma^2\bar{\mu}^2} \frac{k\bar{\mu}S_{in} - k^2(D+\gamma)}{S_{in}^2}} \times c \frac{1 - \frac{2}{\sigma^2\bar{\mu}^2} \frac{k\bar{\mu}S_{in} - k^2(D+\gamma)}{S_{in}^2}}{1 - \frac{2}{\sigma^2\bar{\mu}^2} \frac{k\bar{\mu}S_{in} - k^2(D+\gamma)}{S_{in}^2}} \\ &< +\infty, \end{aligned}$$

which means that $\zeta(0^+) > -\infty$. Then the use of Lemma 3.2 completes the proof. \square

Theorem 3.5. *Let $I(t)$ to be the solution of (3.4) such that the initial condition $I_0 \in (0, S_{in})$. Then if $\mathcal{R}_s = 1$ therefore $\lim_{t \rightarrow +\infty} I(t) = 0$ in probability meaning.*

Proof. Consider the function

$$V = f(I) = \frac{k}{\sigma\bar{\mu}S_{in}} \left[\ln(I) - \frac{(k+S_{in})}{k} \ln(S_{in}-I) \right].$$

Now, using the Itô's formula, we obtain $dV = \phi(V)dt + dW$ where

$$\begin{aligned} \phi(V) = & \frac{1}{\sigma\bar{\mu}} \left(\bar{\mu} - \frac{(D+\gamma)(k+f^{-1}(V))}{S_{in}-f^{-1}(V)} \right. \\ & \left. - \sigma^2\bar{\mu}^2 \frac{k(S_{in}-f^{-1}(V))^2 - (k+S_{in})(f^{-1}(V))^2}{2DS_{in}(k+f^{-1}(V))^2} \right) \end{aligned}$$

with $I = f^{-1}(V)$ since $\mathcal{R}_s = 1$. Let $r = f(\chi)$, one can easily obtain

$$\begin{aligned} 2 \int_0^u \phi(r)dr &= \frac{2k^2}{\sigma^2\bar{\mu}^2} \int_{f^{-1}(0)}^{f^{-1}(u)} \left(\frac{\bar{\mu}}{k} - \frac{(D+\gamma)(k+\chi)}{k(S_{in}-\chi)} \right. \\ & \quad \left. - \frac{\sigma^2\bar{\mu}^2}{k^2} \frac{D(S_{in}-\chi)^2 - (D+DS_{in}/k)\chi^2}{2DS_{in}(1+\chi/k)^2} \right) \frac{(k+\chi)}{k(S_{in}-\chi)\chi} d\chi \\ &= -\frac{2k^2}{\sigma^2\bar{\mu}^2} \left[\ln(f^{-1}(u)) \left(\frac{D+\gamma}{S_{in}^2} + \frac{\sigma\bar{\mu}}{2k} - \frac{\bar{\mu}}{kS_{in}} \right) \right. \\ & \quad \left. + \ln(S_{in}-f^{-1}(u)) \left(\bar{\mu} \frac{k+S_{in}}{k^2S_{in}} - (D+\gamma) \frac{k^2-S_{in}^2}{k^2S_{in}} + \frac{\sigma\bar{\mu}}{2k} \right) \right. \\ & \quad \left. \times \frac{(D+\gamma)}{k^2S_{in}^2} \frac{(k+S_{in})^2}{(S_{in}-f^{-1}(u))} - \frac{\sigma\bar{\mu}}{2k} \ln\left(1 + \frac{f^{-1}(u)}{k}\right) \right] + C, \end{aligned}$$

where C is a constant. By injecting $u = f(\vartheta)$ in the above equality, we obtain

$$\begin{aligned} \alpha(I) = & \frac{k \exp(C)}{\sigma\bar{\mu}} \int_{f^{-1}(0)}^{f^{-1}(I)} \left[\frac{\left(\frac{k+\vartheta}{k}\right) \frac{\sigma\bar{\mu}+k}{\sigma\bar{\mu}}}{\vartheta \frac{2k^2}{\sigma^2\bar{\mu}^2} \left(\frac{D+\gamma}{S_{in}^2} + \frac{\sigma\bar{\mu}}{2k} - \frac{\bar{\mu}}{kS_{in}} \right) + 1} \right. \\ & \left. \times \frac{\exp\left(\frac{(D+\gamma)(k+S_{in})^2}{k^2(S_{in}-\vartheta)}\right)}{\frac{2k^2}{(S_{in}-\vartheta)\sigma^2\bar{\mu}^2} \left(\bar{\mu} \frac{k+S_{in}}{k^2S_{in}} - (D+\gamma) \frac{k^2-S_{in}^2}{k^2S_{in}} + \frac{\sigma\bar{\mu}}{2k} \right) + 1} \right] d\vartheta. \end{aligned}$$

Note that $f^{-1}(0) \in (0, S_{in})$, $\lim_{I \rightarrow +\infty} f^{-1}(I) = S_{in}$, $\lim_{I \rightarrow -\infty} f^{-1}(I) = 0^+$. By Lemma 3.3, one deduces that

$$\begin{aligned} \alpha(+\infty) \leq & \frac{k \exp(C)}{\sigma\bar{\mu}} \left(\frac{k+S_{in}}{k} \right) \frac{\sigma\bar{\mu}+k}{\sigma\bar{\mu}} \\ & \times \int_{f^{-1}(0)}^{S_{in}} \left[\frac{1}{\vartheta \frac{2k^2}{\sigma^2\bar{\mu}^2} \left(\frac{D+\gamma}{S_{in}^2} + \frac{\sigma\bar{\mu}}{2k} - \frac{\bar{\mu}}{kS_{in}} \right) + 1} \right] \end{aligned}$$

$$\begin{aligned}
 & \times \frac{\exp\left(-\frac{2k^2}{\sigma^2\bar{\mu}^2} \times \frac{(D+\gamma)}{k^2} \times \frac{(k+S_{in})^2}{S_{in}(S_{in}-\vartheta)}\right)}{(S_{in}-\vartheta)\sigma^2\bar{\mu}^2\left(\bar{\mu}\frac{k+S_{in}}{k^2S_{in}} - (D+\gamma)\frac{k^2-S_{in}^2}{k^2S_{in}} + \frac{\sigma\bar{\mu}}{2k}\right) + 1} \Big] d\vartheta \\
 & < +\infty
 \end{aligned}$$

and

$$\begin{aligned}
 \alpha(-\infty) & \leq -\frac{k \exp(C)}{\sigma\bar{\mu}} \frac{1}{(f^{-1}(0))\sigma^2\bar{\mu}^2\left(\frac{D+\gamma}{S_{in}^2} + \frac{\sigma\bar{\mu}}{2k} - \frac{\bar{\mu}}{kS_{in}}\right) + 1} \\
 & \times \frac{\exp\left(-\frac{2k^2}{\sigma^2\bar{\mu}^2} \times \frac{(D+\gamma)}{k^2} \times \frac{(k+S_{in})^2}{S_{in}(S_{in}-f^{-1}(0))}\right)}{S_{in}\sigma^2\bar{\mu}^2\left(\bar{\mu}\frac{k+S_{in}}{k^2S_{in}} - (D+\gamma)\frac{k^2-S_{in}^2}{k^2S_{in}} + \frac{\sigma\bar{\mu}}{2k}\right) + 1} \int_{0^+}^{f^{-1}(0)} \frac{1}{\vartheta} d\vartheta \\
 & = -\infty.
 \end{aligned}$$

Similarly, one can also prove that $\beta(+\infty) = +\infty$ and $\beta(-\infty) = -\infty$. The proof is then complete. \square

Before giving a second main result for this section, we first recall a useful definition.

Definition. System (3.4) is said to be stochastically permanent, if for any $\varepsilon \in (0, 1)$, there exist positive constants $\delta_1 = \delta_1(\varepsilon)$ and $\delta_2 = \delta_2(\varepsilon)$ such that

$$\liminf_{t \rightarrow +\infty} \mathbb{P}\{I(t) \leq \delta_1\} \geq 1 - \varepsilon, \quad \liminf_{t \rightarrow +\infty} \mathbb{P}\{I(t) \geq \delta_2\} \geq 1 - \varepsilon,$$

where $I(t)$ is an arbitrary solution of (3.4) for any initial value $I_0 \in \mathbb{R}_+$.

Theorem 3.6. *Let $I(t)$ to be the solution of (3.4) such that the initial condition $I_0 \in (0, S_{in})$. Then, if $\mathcal{R}_s > 1$ therefore the equation (3.4) is stochastically permanent.*

Proof. Let $I(t)$ be the solution of (3.4) then according to the Itô's formula, one obtains

$$\begin{aligned}
 (3.6) \quad d(I^{-\eta}) & = -\eta I^{-\eta} \left[\frac{\bar{\mu}(S_{in}-I)}{k+I} - (D+\gamma) - \frac{\sigma^2\bar{\mu}^2(\eta+1)(S_{in}-I)^2}{2(k+I)^2} \right] dt \\
 & \quad - \frac{\sigma\bar{\mu}\eta I^{-\eta}(S_{in}-I)}{k+I} dW \\
 & = -\eta I^{-\eta} \left[\frac{\bar{\mu}S_{in}}{k} - D - \gamma - \frac{\sigma^2\bar{\mu}^2(\eta+1)S_{in}^2}{2k^2} \right] dt + G(t)dt \\
 & \quad - \frac{\sigma\bar{\mu}\eta I^{-\eta}(S_{in}-I)}{k+I} dW,
 \end{aligned}$$

where η is a constant such that $0 < \eta < 1$ and

$$\begin{aligned} G(t) &= \eta I^{-\eta} \left[\frac{\bar{\mu} S_{in}}{k} - \frac{\bar{\mu}(S_{in} - I)}{k + I} - \frac{\sigma^2 \bar{\mu}^2 (\eta + 1) S_{in}^2}{2k^2} \right. \\ &\quad \left. + \frac{\sigma^2 \bar{\mu}^2 (\eta + 1) (S_{in} - I)^2}{2(k + I)^2} \right] \\ &\leq \frac{\bar{\mu} \eta (S_{in} + k) S_{in}^{1-\eta}}{k^2}. \end{aligned}$$

Let

$$H = \eta \left[\frac{\bar{\mu} S_{in}}{k} - D - \gamma - \frac{\sigma^2 \bar{\mu}^2 (\eta + 1) S_{in}^2}{2k^2} \right].$$

Suppose that η is chosen to be small enough such that $H > 0$. Now multiply both sides of expression (3.6) by e^H then integrate on $(0, t)$ to obtain

$$I^{-\eta}(t) = e^{-Ht} I^{-\eta}(0) + \int_0^t G(r) e^{-H(t-r)} dr - \int_0^t \frac{\sigma \bar{\mu} \eta I^{-\eta}(r) (S_{in} - I(r))}{k + I(r)} dW(r)$$

then taking the expectation yields

$$\mathbb{E}[I^{-\eta}(t)] = e^{-Ht} I^{-\eta}(0) + \mathbb{E} \int_0^t G(r) e^{-H(t-r)} dr \leq I^{-\eta}(0) + \frac{\bar{\mu} \eta (S_{in} + k) S_{in}^{1-\eta}}{k^2 H}.$$

As $I(t) \in (0, S_{in}]$, then using the Chebyshev inequality, one obtains

$$\begin{aligned} \mathbb{P}\{S_{in} \geq I(t) \geq \delta_2\} &= \mathbb{P}\{I(t) \geq \delta_2\} = 1 - \mathbb{P}\{I^{-\eta}(t) \leq \delta_2^{-\eta}\} \\ &\geq 1 - \delta_2^\eta \mathbb{E}[I^{-\eta}(t)] \geq 1 - \delta_2^\eta \left[I^{-\eta}(0) + \frac{\bar{\mu} \eta (S_{in} + k) S_{in}^{1-\eta}}{k^2 H} \right]. \end{aligned}$$

Simply taken δ_2 small enough such that $\delta_2^\eta \left[I^{-\eta}(0) + \frac{\bar{\mu} \eta (S_{in} + k) S_{in}^{1-\eta}}{k^2 H} \right] < \varepsilon$, one obtains then $\liminf_{t \rightarrow +\infty} \mathbb{P}\{I(t) \geq \delta_2\} \geq 1 - \varepsilon$. Similarly, it can be proved that $\liminf_{t \rightarrow +\infty} \mathbb{P}\{I(t) \leq \delta_1\} \geq 1 - \varepsilon$. \square

4. Numerical simulations

We illustrated numerical simulations for systems (2.3)-(2.7)-(3.1). For each system, two cases were considered. The first case confirms the global stability of the disease-persistence equilibrium. The second case illustrates the global stability of the disease-free equilibrium.

4.1. Deterministic ‘‘SIR’’ epidemic model

In a first case, the parameters of system (2.3) are chosen such $S_{in} = 20000$, $D = 50$, $\gamma = 10$, $k = 300$, $\bar{\mu} = 40$, $\tau = 0.04$ and then $\mathcal{R}_d = 444.4 > 1$.

In Figure 2, we can see that the solutions of system (2.3) converge asymptotically to E^* . This validates the global stability of the disease-persistence equilibrium $E^* = (S^*, I^*)$ when $\mathcal{R}_d > 1$. Note that $S^* + I^* \leq S_{in}$.

In a second case, the parameters are chosen such $S_{in} = 2000$, $D = 500$, $\gamma = 10$, $k = 300$, $\bar{\mu} = 40$, $\tau = 0.04$ and then $\mathcal{R}_d = 0.52 < 1$.

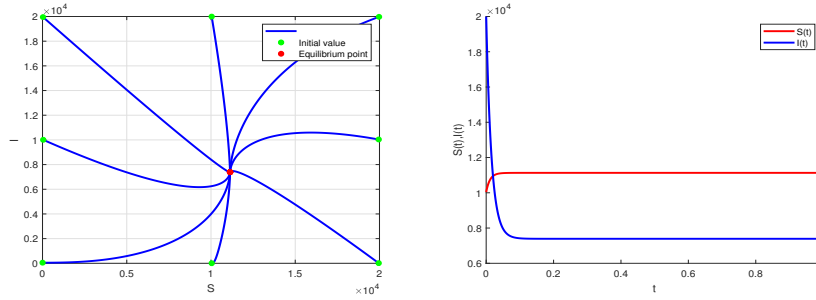


FIGURE 2. (S, I) behaviours for system (2.3) with $\mathcal{R}_d = 444.4$.

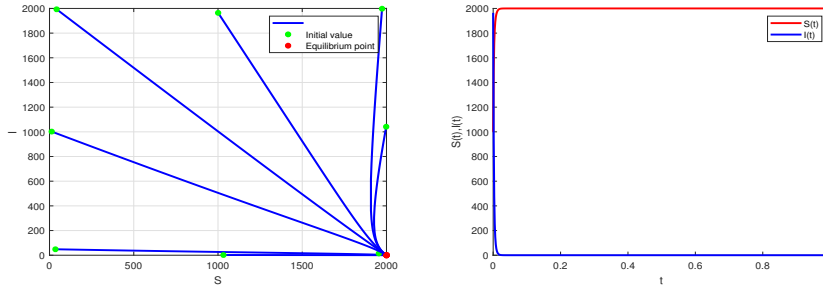


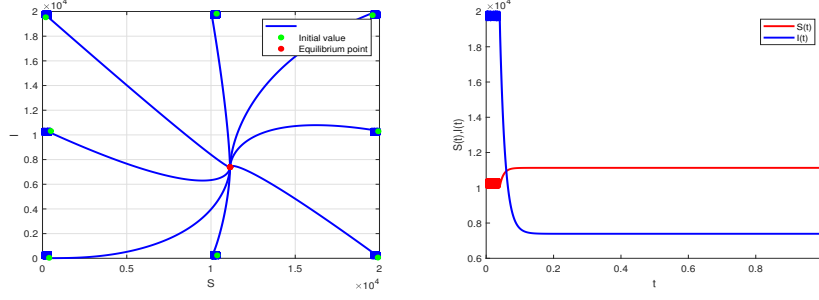
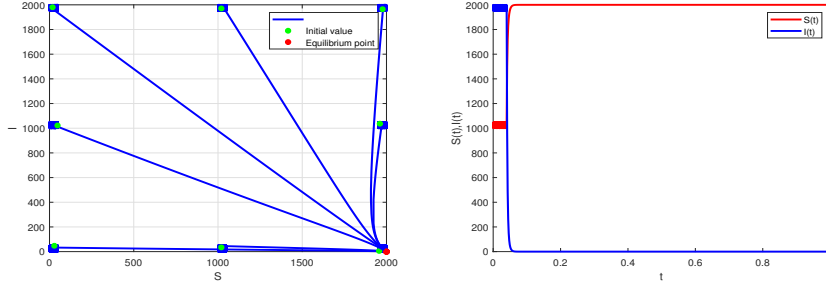
FIGURE 3. (S, I) behaviours for system (2.3) with $\mathcal{R}_d = 0.52$.

In Figure 3, the solutions of system (2.3) converge asymptotically to $\bar{E} = (2000, 0)$. This confirms the global stability of the disease-free equilibrium $\bar{E} = (S_{in}, 0)$ when $\mathcal{R}_d \leq 1$.

4.2. Delayed deterministic “SIR” epidemic model

In a first case, the parameters of system (2.7) are chosen such $S_{in} = 20000$, $D = 50$, $\gamma = 10$, $k = 300$, $\bar{\mu} = 40$, $\tau = 0.04$ and then $\mathcal{R}_d = 444.4 > 1$. The solutions of system (2.7) converge asymptotically to E^* (Figure 4). This confirms the global stability of the disease-persistence equilibrium $E^* = (S^*, I^*)$ when $\mathcal{R}_d > 1$. Note that $S^* + I^* \leq S_{in}$.

In a second case, the parameters are chosen such $S_{in} = 2000$, $D = 500$, $\gamma = 10$, $k = 300$, $\bar{\mu} = 40$, $\tau = 0.04$ and then $\mathcal{R}_d = 0.52 < 1$. The solutions of system (2.7) converge asymptotically to $\bar{E} = (2000, 0)$ (Figure 5). This confirms the global stability of the disease-free equilibrium $\bar{E} = (S_{in}, 0)$ when $\mathcal{R}_d \leq 1$. Initial data on the interval $[-\tau, 0]$ are chosen to be randomly.

FIGURE 4. (S, I) behaviours for system (2.7) with $\mathcal{R}_d = 444.4$.FIGURE 5. (S, I) behaviours for system (2.7) with $\mathcal{R}_d = 0.52$.

4.3. Stochastic “SIR” epidemic model

Consider the following discrimination equations of (3.1) for $n = 1, 2, \dots, M$.

$$\left\{ \begin{array}{l} S(n+1) = S(n) + \left[D(S_{in} - S(n)) + \gamma I(n) - \mu(I(n))S(n) \right] dt \\ \quad - \sigma \mu(I(n))S(n) \rho_n \sqrt{dt} - \frac{\sigma^2}{2} \mu(I(n))S(n) (\rho_n^2 - 1) dt, \\ I(n+1) = I(n) + \left[\mu(I(n))S(n) - (D + \gamma)I(n) \right] dt \\ \quad + \sigma \mu(I(n))S(n) \chi_n \sqrt{dt} + \frac{\sigma^2}{2} \mu(I(n))S(n) (\chi_n^2 - 1) dt, \end{array} \right.$$

where ρ_n, χ_n stand for the Gaussian random variables $N(0, 1)$, dt is the time increment and M is the number of time steps. In a first case, the parameters are chosen such $S_{in} = 5000$, $D = 100$, $\gamma = 2$, $k = 1$, $\bar{\mu} = 30$, $\sigma = 0.0986$ and then $\mathcal{R}_s = 279.4 > 1$. The solutions of system (3.1) converge asymptotically to E^* (Figure 6). This validates the global stability of the disease-persistence equilibrium $E^* = (S^*, I^*)$ when $\mathcal{R}_d > 1$. Note that $S^* + I^* = S_{in} = 5000$.

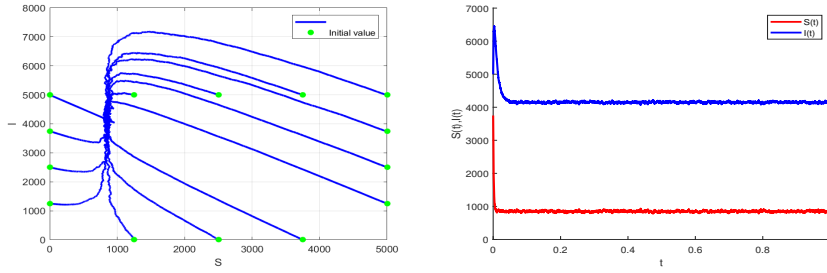


FIGURE 6. (S, I) behaviours for system (3.1) with $\mathcal{R}_s = 279.4$.

In a second case, the parameters are chosen such $S_{in} = 50000$, $D = 500$, $\gamma = 2$, $k = 5000$, $\bar{\mu} = 30$, $\sigma = 0.0002$ and then $\mathcal{R}_s = 0.5 < 1$.

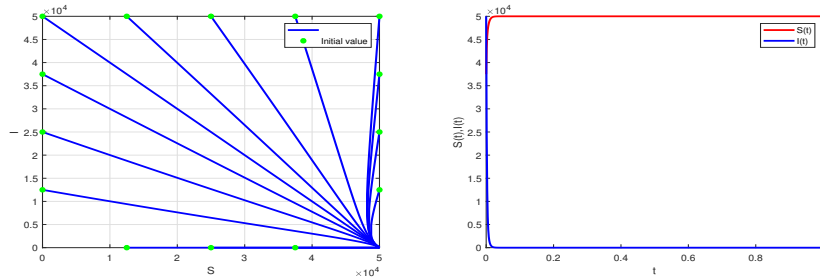


FIGURE 7. (S, I) behaviours for system (3.1) with $\mathcal{R}_s = 0.5$.

The solutions of system (3.1) converge asymptotically to $\bar{E} = (50000, 0)$ (Figure 7). This confirms the global stability of the disease-free equilibrium $\bar{E} = (S_{in}, 0)$ when $\mathcal{R}_s \leq 1$.

5. Conclusion

A mathematical dynamical system involving both deterministic and stochastic “SIR” epidemic model in a continuous reactor is proposed in three different forms; deterministic, delayed and stochastic. It is proved that, for the deterministic model in its both forms, if $\mathcal{R}_d > 1$ then the “endemic” equilibrium is globally asymptotically stable. However, if $\mathcal{R}_d \leq 1$, then the disease-free equilibrium is globally asymptotically stable. For the Stochastic model, Feller’s test and the canonical probability method are applied to study the asymptotic behaviour. The obtained results improve and extend the results obtained for the deterministic model in its both forms.

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