Mathematical analysis of a fractional-order "SIR" epidemic model with a general nonlinear saturated incidence rate in a chemostat

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Abstract

In the present work, a fractional-order differential equation based on the Susceptible-Infected-Recovered (SIR) model with nonlinear incidence rate in a continuous reactor is proposed. A profound qualitative analysis is given. The analysis of the local and global stability of equilibrium points is carried out. It is proved that if the basic reproduction number $\mathcal{R} > 1$ then the disease-persistence (endemic) equilibrium is globally asymptotically stable. However, if $\mathcal{R} \leq 1$, then the disease-free equilibrium is globally asymptotically stable. Finally, some numerical tests are done in order to validate the obtained results.

1 Introduction

The first epidemiological models appeared at the beginning of the 20th century [3, 18]. It was in 1927 that Kermack and McKendrick proposed the first comprehensive model for modeling an epidemic. The main idea comes from the fact that in discrete time, the number of new infections is proportional to the product of the number of infected and susceptible.

The spread of an infectious agent in a population is a dynamic phenomenon: the numbers of susceptible and infected individuals evolve over time, depending on the contacts in which the agent moves from an infected individual to a healthy individual not immune, infecting it in turn. Such a phenomenon can be studied by modeling it with differential equations and by determining its behavior through the numerical resolution of these equations.

The qualitative study of epidemiological models such as the "SIR" model is and has been a field of intense and varied research [2, 3, 7, 16, 19, 20, 22, 24, 28, 37, 38]. An excellent review of the

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literature, but not very recent, was made by Hethcote [18].

In this paper, I revisit the classical "SIR" epidemic model in a chemostat but with a general nonlinear saturated incidence rate and by considering the fractional-order time derivative instead of the classical ordinary differential equations.

The chemostat is an experimental device used to analyze the growth of populations of microorganisms (Fig. 1). It was introduced simultaneously by A. Novick and L. Szilard [29] in the 1936s and by J. Monod [27] in the 1950s. The mathematical growth of a species of bacteria in the chemostat is due to C. Spicer [34]. From this date there are many articles relating to the competition of several species.

In a chemostat, an epidemic model can also be understood as a competition model where various pathogen strains compete for the the same susceptible host as only resource [26, 32]. Such models predict the strain with the largest basic reproduction number to be the winner. In [32], it is proved that this prediction amount to the same if the per capita functional responses of infective individuals to the density of susceptible are proportional to each other but that they are different if the functional responses are non-proportional.

The important aspect in the model that considered by many researchers to interpret the dynamical behaviour of the infectious disease is the susceptible-infected-recovered model (SIR) introduced by Kermack and McKendrick in 1927 [21]. The considered population here is subdivided into three
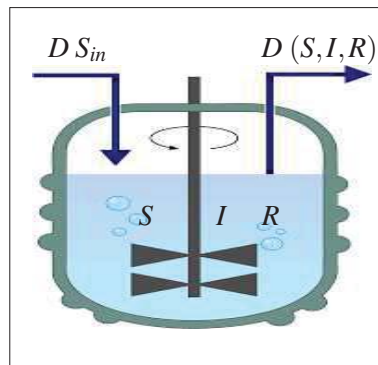

Fig. 1: "SIR" epidemic model in a continuous reactor

subgroups of individuals. Each group has different epidemiological significance: the compartments of Susceptible, the compartment of Infected and the compartment of Recovered, which are respectively represented by the following letters S, I and R. The model developed here has then three components, S, I and R known as 'SIR' model of infectious disease transmission in a chemostat. I neglect all individuals natural mortality other than one caused by the disease concerned by this study and I take into account the dilution rate (D) only. Only susceptible individuals are introduced into the reactor with a constant rate D and an input individual number S_{in} (Fig. 1). Note that S_{in} can be seen as the new cases infected per unit of time by one infective individual. DS_{in} describes the rate of recruitment of susceptible (as input), this includes newborns who are born susceptible in the type of infection considered. γ is the rate at which infectious agents recover their health. $(D + \gamma)^{-1}$ describes the average infection period.

The epidemic is spread by contacts between infected individuals and susceptible individuals. The number of these contacts is proportional to S and I, respective populations of susceptible and infected individuals. The patients recover on average after a time $1/\gamma$, they are then immune and can no longer infect other people or be re-infected.

This paper is organized as follows. In section 2, a fractional-order mathematical dynamical

system involving deterministic "SIR" epidemic model with nonlinear incidence rate in a continuous reactor is considered. The analysis of the local and global stability of equilibrium points is carried out. It is proved that, for the deterministic model, if $\mathscr{R} > 1$, then the disease-persistence (endemic) equilibrium is globally asymptotically stable. However, if $\mathscr{R} \leq 1$, then the disease-free equilibrium is globally asymptotically stable. Finally, in section 4, some numerical tests are done in order to validate the obtained results.

2   Mathematical model and properties

Notions of non-integer differentiation and integration are an effective tool for characterizing the behavior of a large category of infinite dimensional dynamical systems. The applications are numerous, whether in electricity, heat, chemistry or signal processing. Fractional calculus is a domain of mathematics whose purpose is to extend the definitions of traditional derivatives to non-integer orders. The fractional derivative represents the generalization to non-integer orders of the derivative [5], just like the real exponent power function which corresponds to the "extension" of the full exponent power function. Several definitions have been proposed for the non-integer derivation. It should be noted, however, that these definitions do not always lead to identical results but are globally equivalent for a large number of functions. In this paper, the Caputo derivative approach will be used due to its application advantages. The most important advantage is that the initial conditions for fractional order is the same as that of integer order, avoiding solvability issues.
I first give some definitions that I use later in this paper.

For an arbitrary function $f(t)$, the definition of the Caputo fractional derivative is defined as follows:

$$D_C^\alpha f(t) = J^{n-\alpha}[f^{(n)}(t)] = \frac{1}{\Gamma(n-\alpha)} \int_0^t (t-s)^{n-\alpha-1} f^{(n)}(s) ds \tag{2.1}$$

where n is the first integer which is greater than α.

The Laplace transform of the Caputo fractional derivative is given by

$$\mathscr{L}(D_C^\alpha f(t)) = \lambda^\alpha F(s) - \sum_{k=0}^{n-1} f^{(k)}(0)\lambda^{\alpha-k-1}. \tag{2.2}$$

The Mittag-Leffler function is defined by the following infinite power series:

$$E_{\alpha,\beta}(z) = \sum_{k=0}^{+\infty} \frac{z^k}{\Gamma(\alpha k + \beta)}. \tag{2.3}$$

The Laplace transform of the Mittag-Leffler function is given by

$$\mathscr{L}[t^{\beta-1} E_{\alpha,\beta}(\pm \alpha t^\alpha)] = \frac{s^{\alpha-\beta}}{s^\alpha \mp \alpha}. \tag{2.4}$$

Let $\alpha, \beta > 0$ and $z \in \mathbb{Z}$, and the Mittag-Leffler functions satisfy the equality given by [4, Theorem 4.2]

$$E_{\alpha,\beta}(z) = z E_{\alpha,\alpha+\beta}(z) + \frac{1}{\Gamma(\beta)}. \tag{2.5}$$

D^α denotes the Caputo fractional derivative of order $0 < \alpha \leq 1$ defined for an arbitrary function $f(t)$ by [30] as follows:

$$D^\alpha f(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t (t-x)^{-\alpha} f'(x) dx.$$

The considered mathematical model is given by the following three-dimensional dynamical system of Fractional Differential Equations (FDEs):

$$\begin{cases} D^{\alpha}S(t) &= D\,S_{in} - DS(t) - \mu(I(t))S(t), \\[2mm] D^{\alpha}I(t) &= \mu(I(t))S(t) - (D+\gamma)I(t) \\[2mm] D^{\alpha}R(t) &= \gamma I(t) - DR(t) \end{cases} \tag{2.6}$$

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 bounded concave function such that $\mu(0) = 0$, $\mu'(I) > 0$ and $\mu''(I) < 0$.

Remark 1.   • The saturated incidence rate satisfies $\mu(I) \leq \mu'(0)I, \forall I \geq 0$ and $\mu(I) > \mu'(I)I, \forall I > 0$.

   • The classical Monod function can be used to express transmission rate of infection from infected individuals to susceptible ones.

$$\mu(I) = \frac{\bar{\mu}I}{k+I} \tag{2.7}$$

$\bar{\mu}$ represents the transmission rate of the disease. k is the Monod constant which is equal to the number of infected individuals when the saturated incidence rate is $\dfrac{\bar{u}}{2}$.

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 rate [37], and is denoted $\mathscr{R}$. It is considered in a population where all individuals are healthy, except the infectious individual introduced. If $\mathscr{R} < 1$, then an individual infects on average less than one, which means that the disease will disappear from the population eventually. In contrast, if $\mathscr{R} > 1$, then the disease can spread in the population. Determining $\mathscr{R}$ according to the parameters of the model thus makes it possible to calculate the conditions under which the disease is spreading.
In our case, the basic reproduction number for the system (2.6), denoted by $\mathscr{R}$, is given by:

$$\mathscr{R} = \frac{\mu'(0)S_{in}}{(D+\gamma)} \tag{2.8}$$

$\mathbb{R}^3_+$, the closed non-negative cone in $\mathbb{R}^3$, is positively invariant ([8, 9, 10, 11, 12, 13, 14, 31]) for the system (2.6). More precisely,

Proposition 1.

1. For all initial condition (S_0, I_0, R_0) in $\mathbb{R}^3_+$, the solution of system (2.6) is bounded and has positive components and thus is defined for all $t > 0$.

2. System (2.6) admits a positive invariant attractor set of all solution given by $\Omega = \{(S, I, R) \in \mathbb{R}^3_+ \,/\, S + I + R \leq S_{in}\}$.

Proof.   1. The positivity of the solution is proved by the fact that :
   Since $S = 0$ then $D^{\alpha}S = DS_{in} > 0$, if $I = 0$ then $D^{\alpha}I = 0$, and if $R = 0$ then $D^{\alpha}R = \gamma I > 0$.
   Next I have to prove the boundedness of solutions of (2.6). By adding three equations of system (2.6), one obtains, for $T = S + I + R - S_{in}$, a single equation for the total number of individuals :

$$D^{\alpha}T(t) = D^{\alpha}S(t) + D^{\alpha}I(t) + D^{\alpha}R(t) = D(S_{in} - S - I - R) = -DT.$$

I solve the above equation by applying the Laplace transform (2.2) , I obtain

$$\lambda^{\alpha}\mathscr{L}(T(t)) - \lambda^{\alpha-1}T(0) = -D\mathscr{L}(T(t))$$

that can be written as below using the Laplace transform properties (2.4) and equality (2.5),

$$(\lambda^{\alpha} + D)\mathscr{L}(T(t)) = \lambda^{\alpha-1}T(0).$$

Then

$$\begin{aligned}\mathscr{L}(T(t)) &= \frac{\lambda^{\alpha-1}}{(\lambda^{\alpha}+D)}T(0)\\ &\leq t^{\alpha-1}E_{\alpha,\alpha}(-Dt^{\alpha})T(0)\end{aligned}$$

where $0 < \alpha \leq 1$ and $E_{a,b}(z)$ is the two parameter Mittag-Leffler function with parameter a and b [4]. Since Mittag-Leffler function is an entire function, thus $E_{\alpha,\alpha}(-Dt^{\alpha})$ is bounded for all $t > 0$. Therefore I have

$$\lim_{t \mapsto +\infty} T(t) \leq 0 \tag{2.9}$$

Thus, closed set Ω is positively invariant and attracting to the system (2.6).

Since all terms of the sum are positive, then the solution of system (2.6) is bounded.

2. The invariance of the attractor Ω is simply deduced from inequality (2.9) .

$\square$

Remark 2. Since the compartment R doesn't affect equations of S and I compartments, it is sufficient to consider only both first equations of system (2.6).

$$\begin{cases} D^{\alpha}S(t) &= D\,S_{in} - DS(t) - \mu(I(t))S(t), \\ D^{\alpha}I(t) &= \mu(I(t))S(t) - (D+\gamma)I(t) \end{cases} \tag{2.10}$$

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

Define $E^* = (S^*, I^*)$ as an endemic equilibrium of system (2.10) where $S^* > 0$ and $I^* > 0$ satisfying

$$\begin{cases} DS_{in} = DS^* + \mu(I^*)S^*, \\ \mu(I^*)S^* = (D+\gamma)I^*. \end{cases} \tag{2.11}$$

Regarding the characteristic equations and characteristic roots of the proposed model (2.10), it is easy to prove the following proposition.

Proposition 2.
- If $\mathscr{R} \leq 1$ then system (2.10) admits a disease-free equilibrium $\bar{E} = (S_{in}, 0)$ as the unique equilibrium.

- If $\mathscr{R} > 1$ then system (2.10) 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 $\mathscr{R}$ has a great importance in determining whether there exists an endemic equilibrium or not (as in [2], Theorem 2.3).

Theorem 1.
- If $\mathscr{R} < 1$, then the disease-free equilibrium $\bar{E}$ is locally asymptotically stable.

- If $\mathscr{R} > 1$, 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:

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

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

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

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

As μ is a concave function and $\mu(I^*) > \mu'(I^*)I^*$, it follows that $A_0 > 0$ and $A_1 > 0$ and thus using Routh-Hurwitz criterion, both eigenvalues have negative real parts. Thus, if $\mathscr{R} > 1$, then E^* exists and it is always locally asymptotically stable. This completes the proof. □

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

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

Proof. Let (S, I) to be a solution of the system (2.10) and define the Lyapunov function

$$V_1(t) = S(t) + I(t) - S^*\ln(\frac{S}{S^*}) - \int_{I^*}^{I(t)} \frac{\mu(I^*)}{\mu(\eta)} d\eta$$

The equilibrium E^* is the only internal stationary point and minimum point of $V_1(t)$, and $V_1(t) \mapsto +\infty$ at the boundary of the positive quadrant. Consequently, E^* is the global minimum point, and the

function is bounded from below.

The Caputo fractional derivative of $V_1(t)$ along solution of system (2.10) is given by

$$
\begin{aligned}
D^\alpha V_1(t) &= \left(1 - \frac{S^*}{S}\right)D^\alpha S(t) + \left(1 - \frac{\mu(I^*)}{\mu(I)}\right)D^\alpha I(t) \\[2mm]
&= \left(1 - \frac{S^*}{S}\right)\left(D\,S_{in} - DS - \mu(I)S\right) + \left(1 - \frac{\mu(I^*)}{\mu(I)}\right)\left(\mu(I)S - (D+\gamma)I\right) \\[2mm]
&= \left(1 - \frac{S^*}{S}\right)\left(DS^* + \mu(I^*)S^* - DS - \mu(I)S\right) + \left(1 - \frac{\mu(I^*)}{\mu(I)}\right)\left(\mu(I)S - (D+\gamma)I\right) \\[2mm]
&= \frac{S - S^*}{S}\left(D(S^* - S) + (\mu(I^*)S^* - \mu(I)S)\right) + \frac{\mu(I) - \mu(I^*)}{\mu(I)}\left(\mu(I)S - (D+\gamma)I\right) \\[2mm]
&= -D\frac{(S - S^*)^2}{S} + (D+\gamma)I^*\frac{\mu(I^*) - \mu(I)}{\mu(I)}\left(\frac{I}{I^*} - \frac{\mu(I)}{\mu(I^*)}\right) - (D+\gamma)I^*\left(\frac{S^*}{S} - 1 - \ln(\frac{S^*}{S})\right)
\end{aligned}
$$

On the one hand, μ is concave and then

- $\dfrac{\mu(I)}{\mu(I^*)} \geq \dfrac{I}{I^*}$ for all $0 \leq I \leq I^*$ and

- $\dfrac{\mu(I)}{\mu(I^*)} \leq \dfrac{I}{I^*}$ for all $I \geq I^*$.

Then $\dfrac{\mu(I^*) - \mu(I)}{\mu(I)}\left(\dfrac{I}{I^*} - \dfrac{\mu(I)}{\mu(I^*)}\right) \leq 0$ for all $I \geq 0$. On the other hand, $x - 1 - \ln(x) > 0$ for all $x > 0$ thus

- $\left(\dfrac{S^*}{S} - 1 - \ln(\dfrac{S^*}{S})\right) > 0, \quad \forall S \geq 0$

Since all parameters of the model are non-negative, it follows that $D^\alpha V_1 \leq 0$. Therefore, all the conditions of [23] are satisfied. This proves that E^* is globally asymptotically stable where $\mathscr{R} > 1$.

Let (S,I) to be a solution of the system (2.10) and define the Lyapunov function

$$
V_2(t) = S(t) + I(t) - S_{in}\ln(\frac{S}{S_{in}}),
$$

The equilibrium $\bar{E}$ is the only internal stationary point and minimum point of $V_2(t)$, and $V_2(t) \mapsto +\infty$ at the boundary of the positive quadrant. Consequently, $\bar{E}$ is the global minimum point, and the function is bounded from below.

The Caputo fractional derivative of $V_2(t)$ along solution of system (2.10) is given by

$$
\begin{aligned}
D^\alpha V_2(t) &= \left(1 - \frac{S_{in}}{S}\right)D^\alpha S(t) + D^\alpha I(t) \\[2mm]
&= \left(1 - \frac{S_{in}}{S}\right)\left(D\,S_{in} - DS - \mu(I)S\right) + \mu(I)S - (D+\gamma)I \\[2mm]
&= \frac{S - S_{in}}{S}\left(D\,(S_{in} - S) - \mu(I)S\right) + \mu(I)S - (D+\gamma)I \\[2mm]
&= -\frac{D(S_{in} - S)^2}{S} + (D+\gamma)\left(\frac{S_{in}}{(D+\gamma)}\mu(I) - I\right)
\end{aligned}
$$

Note that Assumption 1 ensure that

$$\mu(I) \le \mu'(0)I, \quad \forall I > 0.$$

Then

$$D^\alpha V_2(t) \le -\frac{D(S_{in}-S)^2}{S} + (D+\gamma)\Big(\frac{S_{in}}{(D+\gamma)}\mu'(0)-1\Big)I = -\frac{D(S_{in}-S)^2}{S} + (D+\gamma)(\mathscr{R}-1)I.$$

Since all parameters of the model are non-negative and $\mathscr{R} < 1$, it follows that $D^\alpha V_2 \le 0$. Therefore, again, using [23], $\{\bar{E}\}$ is globally asymptotically stable for $\mathscr{R} < 1$.

Now, if $\mathscr{R} = 1$, then $D^\alpha V_2 = 0$ if and only if $S = S_{in}$ and the largest compact invariant set in $\{(S,I) \in \Omega : D^\alpha V_2 = 0\}$ is the singleton $\{\bar{E}\}$. Therefore, by the LaSalle's invariance principle (see, for instance, [23]), $\{\bar{E}\}$ is globally asymptotically stable (for other applications, see [8, 9, 10, 13, 31]). □

3   Numerical Simulations

The system (2.6) has the following form

$$D_C^\alpha y(t) = f(t,y(t)), \quad y(0) = y_0 \tag{3.1}$$

There are several analytical and numerical methods have been proposed to solve such systems (3.1). Diethelm and Freed [6] proposed the well-known algorithm called FracPECE, using the classical predict, evaluate, correct, evaluate (PECE) type approach, but modified in order to solve fractional-order derivative equations [15]. This approach combines fractional Adams-Bashforth-Moulton methods.

Suppose that the time interval $[0,T]$ is discretized uniformly into N sub-intervals; define $t_j = j\,dt, n = 0,1,\cdots,N$, where $dt = T/N$ is the time step. Let y_j be the exact value of a function $y(t)$ at time step t_j.
Firstly, I calculate the predictor y_{n+1}^P according to

$$y_{n+1}^P = y_0 + \frac{1}{\Gamma(\alpha)} \sum_{j=0}^{n} b_{j,n+1} f(t_j,y_j) \tag{3.2}$$

where

$$b_{j,n+1} = \frac{dt^\alpha}{\alpha}\Big((n+1-j)^\alpha - (n-j)^\alpha\Big). \tag{3.3}$$

Then I evaluate $f(t_{n+1},y_{n+1}^P)$, use this to determine the corrector y_{n+1} by means of equation

$$y_{n+1} = y_0 + \frac{1}{\Gamma(\alpha)}\Big(\sum_{j=0}^{n} a_{j,n+1} f(t_j,y_j) + a_{n+1,n+1} f(t_{n+1},y_{n+1}^P)\Big) \tag{3.4}$$

where

$$a_{j,n+1} = \frac{dt^\alpha}{\alpha(\alpha+1)}\Big((n+2-j)^{\alpha+1} - 2(n+1-j)^{\alpha+1} + (n-j)^{\alpha+1}\Big). \tag{3.5}$$

Finally I evaluate $f(t_{n+1},y_{n+1})$ which is then used in the next integration step.

I performed numerical simulations for system (2.6) using FracPECE algorithm. I use classical Monod functions to express transmission rate of infection from infected individuals to susceptible ones $\mu(I) = \frac{\bar{\mu}I}{(k+I)}$ with $\bar{\mu}, k > 0$ which satisfies Assumption 1.

Two cases were considered. The first case performs the global stability of the disease-persistence equilibrium. The second case performs the global stability of the disease-free equilibrium. In a first case, the parameters are chosen such $S_{in} = 10, D = 1, \gamma = 2, k = 3, \bar{\mu} = 5$ and $\mathscr{R} = 5.5556 > 1$. The solution of system (2.6) converge asymptotically to E^* (Fig. 2 (a)). This performs the global stability of the disease-persistence equilibrium $E^* = (S^*, I^*, R^*)$ when $\mathscr{R} > 1$. Note that $S^* + I^* + R^* = S_{in}$. In a second case, the parameters are chosen such $\alpha = 0.8, S_{in} = 1, D = 1, \gamma = 2, k = 3, \bar{\mu} = 5$ and $\mathscr{R} = 0.5556 < 1$. The solution of system (2.6) converge asymptotically to $\bar{E} = (S_{in}, 0, 0) = (1, 0, 0)$ (Fig. 3 (a)). This performs the global stability of the disease-free equilibrium $\bar{E} = (S_{in}, 0, 0)$ when $\mathscr{R} \leq 1$.

(a) $(S(t), I(t), R(t))$ behavior.

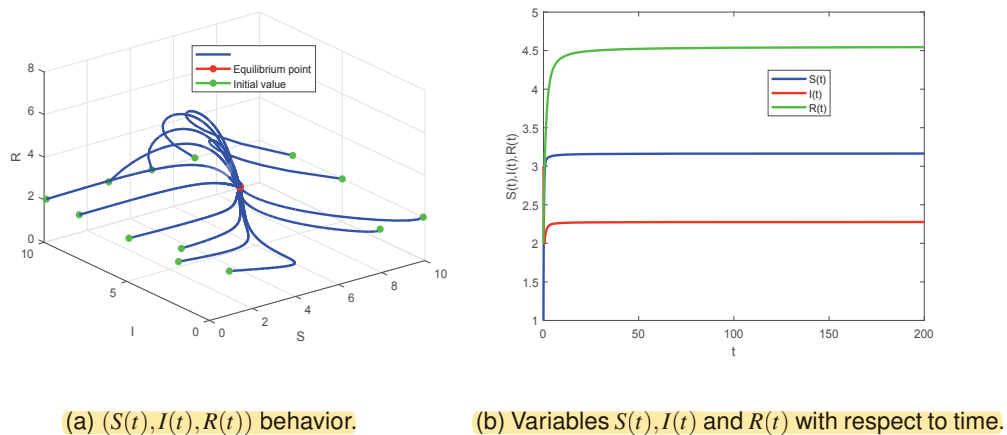(b) Variables $S(t), I(t)$ and $R(t)$ with respect to time.

Fig. 2: Parameters are fixed to $\alpha = 0.8, S_{in} = 10, D = 1, \gamma = 2, k = 3$ and $\bar{\mu} = 5$, thus $\mathscr{R} = 5.5556 > 1$ and the solution of system (2.6) converge asymptotically to E^*.

(a) $(S(t), I(t), R(t))$ behavior.

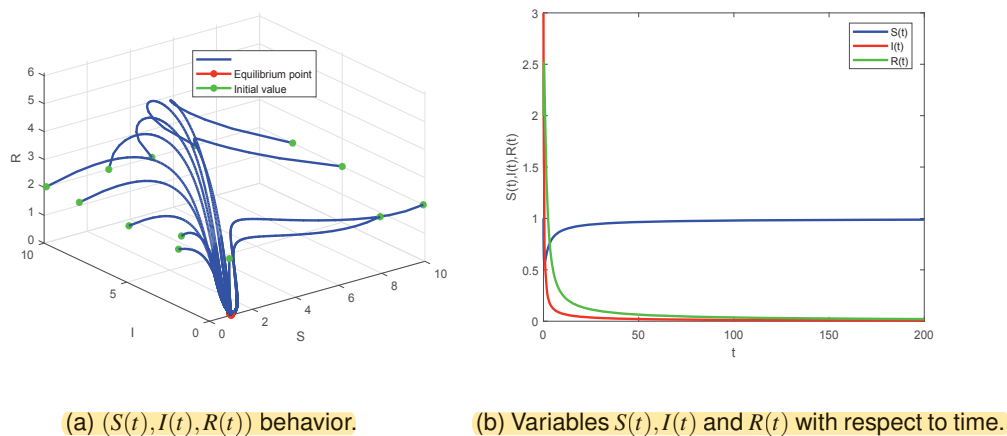(b) Variables $S(t), I(t)$ and $R(t)$ with respect to time.

Fig. 3: Parameters are fixed to $\alpha = 0.8, S_{in} = 1, D = 1, \gamma = 2, k = 3$ and $\bar{\mu} = 5$, thus $\mathscr{R} = 0.5556 < 1$ and the solution of system (2.6) converge asymptotically to $\bar{E}$.

4 Conclusion

A Fractional-order mathematical three-dimensional dynamical system involving a deterministic "SIR" epidemic model with a general nonlinear saturated incidence rate in a reactor is proposed. A profound qualitative analysis is given for each form. The analysis of the local and global stability of equilibrium points is carried out. It is proved that if $\mathscr{R} > 1$ then the disease-persistence (endemic) equilibrium is globally asymptotically stable. However, if $\mathscr{R} \leq 1$, then the disease-free equilibrium is globally asymptotically stable. I consider the optimal control problem relative to this epidemic model by minimizing the infected and susceptible populations and maximizing the recovered populations. Numerical test were used to validate the obtained results.

References

[1] Birkhoff, G., Rota, G.C., (1962). Ordinary Differential Equations. Ginn Boston.

[2] Bonzi, B., Fall, A., Iggidr, A. and Sallet, G., (2011). Stability of Differential Susceptibility and Infectivity Epidemic Models. J. Math. Biol., 62, 39-64. https://doi.org/10.1007/s00285-010-0327-y.

[3] Diekmann, O., Heesterbeek, J., (2000). Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis, and Interpretation. Wiley, Hoboken.

[4] Diethelm K, (2004). The analysis of fractional differential equations: an application-oriented exposition using operators of caputo type. Springer.

[5] Diethelm, K., Ford N.J., (2002). Analysis of fractional differential equations. J. Math. Anal. Appl., 260(2), 229-48. https://doi.org/10.1006/jmaa.2000.7194.

[6] Diethelm, K., Freed, A.D., (1998). The fracPECE subroutine for the numerical solution of differential equations of fractional order. Forschung Wiss Rechnen 57-71.

[7] El Hajji, M., (2018). Boundedness and asymptotic stability of nonlinear Volterra integro-differential equations using Lyapunov functional. J. King Saud Univ. Sci., 31(1). https://doi.org/10.1016/j.jksus.2018.11.012.

[8] El Hajji, M., (2018). How can inter-specific interferences explain coexistence or confirm the competitive exclusion principle in a chemostat. Int. J. Biomath., 11(8), 1850111 (20 pages). https://doi.org/10.1142/S1793524518501115.

[9] El Hajji, M., Chorfi N., Jleli, M., (2017). Mathematical modelling and analysis for a three-tiered microbial food web in a chemostat. Electron. J. Diff. Eqns., 2017(255), 1-13. https://ejde.math.txstate.edu/Volumes/2017/255/elhajji.pdf.

[10] El Hajji, M., Chorfi N., Jleli, M., (2015). Mathematical model for a membrane bioreactor process, Electron. J. Diff. Eqns., 2015(315), 1-7. https://ejde.math.txstate.edu/Volumes/2015/315/elhajji.pdf.

[11] El Hajji, M., Rapaport, A., (2010). Design of a cascade observer for a model of bacterial batch culture with nutrient recycling. IFAC Proceedings Volumes. 43(6), 203-208. https://doi.org/10.3182/20100707-3-BE-2012.0038.

[12] El Hajji, M., Mazenc, F. Harmand, J., (2010). A mathematical study of a syntrophic relationship of a model of anaerobic digestion process. Math. Biosci. Eng. 7(3), 641-656. https://doi.org/10.3934/mbe.2010.7.641

[13] El Hajji, M., Rapaport, A., (2009). Practical coexistence of two species in the chemostat - A slow-fast characterization. Math. Biosci. 218(1), 33-39. https://doi.org/10.1016/j.mbs.2008.12.003.

[14] El Hajji, M. Harmand, J. Chaker, H. Lobry, C., (2009). Association between competition and obligate mutualism in a chemostat. J. Biol. Dynamics, 3(6), 635-647. https://doi.org/10.1080/17513750902915978.

[15] Garrappa, R., (2015). Trapezoidal methods for fractional differential equations: theoretical and computational aspects. Math. Comput. Simul., 11, 96-112. https://doi.org/10.1016/j.matcom.2013.09.012.

[16] Gumel, A., McCluskey, C., Watmough, J., (2006). An Sveir Model for Assessing Potential Impact of an Imperfect Anti-Sars Vaccine. Math. Biosc. Eng., 3, 485-512. https://doi.org/10.3934/mbe.2006.3.485.

[17] Hale, J.K., (1969). Ordinary differential equations, John Wiley & Sons, New York.

[18] Hethcote, H.W., (2000). The Mathematics of Infectious Diseases. SIAM Review, 42, 599-653. https://doi.org/10.1137/S0036144500371907.

[19] Huang, G., Takeuchi, Y., Ma, W., Wei, D., (2010). Global Stability for Delay SIR and SEIR Epidemic Models with Nonlinear Incidence Rate. Bull. Math. Biol., 72(5), 1192-1207. https://doi.org/10.1007/s11538-009-9487-6.

[20] Hu, Z.-X., Ma, W.-B., Ruan, S.-G., (2012). Analysis of SIR epidemic models with nonlinear incidence rate and treatment. Math. Biosci., 238, 12-20. https://doi.org/10.1016/j.mbs.2012.03.010.

[21] Kermack, W.O., McKendrick, A.G., (1927). A contribution to the mathematical theory of epidemics. Proc. R. Soc. A. Math. Phys. Eng. Sci., 115, 700-21. https://doi.org/10.1098/rspa.1927.0118.

[22] Kuang Y., (1993). Delay Differential Equation with Application in Population Dynamics. Academic Press, San Diego.

[23] LaSalle, J.P., (1976). The Stability of Dynamical Systems. 25, SIAM. https://doi.org/10.1137/1.9781611970432.

[24] Li, F., Meng, X.-Z., Cui, Y., (2017). Nonlinear stochastic analysis for a stochastic SIS epidemic model. J. Nonlinear Sci. Appl., 10, 5116-5124. http://dx.doi.org/10.22436/jnsa.010.09.47.

[25] Lin, Y., Xu, C., (2007). Finite difference/spectral approximations for the time-fractional diffusion equation. J. Comput. Phys., 225(2), 1533-1552. https://doi.org/10.1016/j.jcp.2007.02.001.

[26] Martcheva, M., Pilyugin, S. S., Holt, R. D., (2007). Subthreshold and superthreshold coexistence of pathogen variants: The impact of host structure. Math. Biosci., 207, 58-77. https://doi.org/10.1016/j.mbs.2006.09.010.

[27] Monod, J. La technique de culture continue. Ann. Inst. Pasteur, 79(1950), 390-410.

[28] Nkamba, L.N., Ntaganda, J.M., Abboubakar, H. Kamgang, J.C., Castelli, L., (2017). Global Stability of a SVEIR Epidemic Model: Application to Poliomyelitis Transmission Dynamics. Comput. Math. Appl., 5, 98-112. http://dx.doi.org/10.4236/ojmsi.2017.51008.

[29] Novick, A. and Szilard, L., (1936). Experiments with the chemostat on spontaneous mutations of bacteria. Proc. Nat. Acad. Sci., 36, 708-719. https://doi.org/10.1073/pnas.36.12.708.

[30] Pinho, S.T.R., Ferreira, C.P., Esteva, L., Barreto, F.R., Morato, E., Silva, V.C., Teixeira, M.G.L., (2006). Modelling the dynamics of dengue real epidemics. Philos. TransR. Soc. 368, 5679-93. https://doi.org/10.1098/rsta.2010.0278.

[31] Sari, T., El Hajji, M., Harmand, J., (2012). The mathematical analysis of a syntrophic relationship between two microbial species in a chemostat. Math. Biosci. Engng., 9(1), 627-645. https://doi.org/10.3934/mbe.2012.9.627.

[32] Smith H.L., Thieme, H.R., (2013). Chemostat and Epidemics: competition for nutrients/hosts. Math. Biosci. Eng. 10(5&6), 1635-1650. https://doi.org/10.3934/mbe.2013.10.1635.

[33] Smith H.L., Waltman,P., (1995). The theory of the chemostat, Dynamics of microbial competition. Cambridge Studies in Mathematical Biology, Cambridge University Press.

[34] Spicer, C., (1995). The theory of constant growth apparatus. Biometrics.

[35] Sun, X.-P. , Wang, Y.-J., (2008). Stability analysis of a stochastic logistic model with nonlinear diffusion term. Appl. Math. Model., 32, 2067-2075. https://doi.org/10.1016/j.apm.2007.07.012.

[36] Thieme, H.R., (1992). Convergence results and a Poincaré-Bendixson trichotomy for asymptotically autonomous differential equations. J. Math. Biol., 30(7), 755-763. https://doi.org/10.1007/BF00173267.

[37] Van den Driessche, P., Watmough, J., (2002). Reproduction Numbers and Sub-Threshold Endemic Equilibria for Compartmental Models of Disease Transmission. Math. Biosci., 180, 29-48. https://doi.org/10.1016/S0025-5564(02)00108-6.

[38] Wei, H., Jiang, Y., Song, X., Su, G.H., Qiu, S.Z., (2009). Global attractivity and permanence of a SVEIR epidemic model with pulse vaccination and time delay. J. comput. appl. math., 229, 302-312. https://doi.org/10.1016/j.cam.2008.10.046.

[39] Hamdan, N.I., Kilicman, A., (2018). A fractional order SIR epidemic model for dengue transmission. Chaos, Solitons and Fractals, 114, 55-62. https://doi.org/10.1016/j.chaos.2018.06.031.

[40] Mouaouine, A., Boukhouima, A., Hattaf, K., Yousfi, N., (2018). A fractional order SIR epidemic model with nonlinear incidence rate. Advances in Difference Equations. https://doi.org/10.1186/s13662-018-1613-z.