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Commun. Math. Biol. Neurosci. 2022, 2022:96

<https://doi.org/10.28919/cmbn/7650>

ISSN: 2052-2541

VACCINATION STRATEGY AND PSYCHOLOGICAL EFFECTS IN A FRACTIONAL ORDER EPIDEMIC MODEL

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Abstract. Various approaches have been made recently to understand the complex dynamics of many epidemic diseases like COVID-19. The mathematical modeling approach is one of the considerable tools to study the disease spreading pattern. In this paper we study a fractional order SIR epidemic model with nonmonotone incidence rate and vaccination involving a Caputo type fractional derivative. Existence and uniqueness results for the problem are established which means that our model is biologically and mathematically well posed. We Firstly give some preliminaries results. Then we calculate the equilibria and investigate their global stability. Finally, we present some numerical simulations to support our analytical findings.

Keywords: SIR epidemic model; nonmonotone incidence rate; Caputo fractional derivative; stability; Vaccination.

2010 AMS Subject Classification: 91D10, 93A30.

1. INTRODUCTION

Epidemiology is an interdisciplinary branch of science, it uses mathematics concepts, such as statistics, to study biological phenomena, including the epidemics. It is an ancient science, which has developed singularly with the great epidemics such as the Spanish flu, then, with the possibilities of numerical simulation offered by computer advances.

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Received August 5, 2022

Modeling an epidemic consists of creating its mathematical representation. Different conceptual frameworks exist for such representations, but, once the framework is fixed, the modeller must determine the values of the parameters that will best correspond to the epidemic modeled to the actual epidemic for the chosen representation. The modeling therefore assumes back and forth between mathematical calculations and data from the field (epidemiology or medical research). Several models have been used in this direction to study epidemic phenomena like simple SIR models. Compartmental epidemiological models derive from the work of Kermack and McKendrick on the plague epidemic in India, themselves derived from the so-called "predatory prey" model, put into equation by Lotka and Volterra in the 20th century. They are used to describe the temporal evolution of a disease within a population. At first glance, this method divides individuals into three categories, representing their state of illness and called compartments. In the so-called SIR models, there are the compartments: S, in which the healthy individuals susceptible to infection are found (likely). I, for infected and infectious individuals. R, for individuals removed from the model (removed), not likely to be infected, because cured and immune or deceased [1]. In this paper we use fractional order differential equations that have been attracted considerable interest in many fields such as physics, bioengineering, finance and biology (see for instance [2, 3, 4, 5]). Due to the memory property of fractional derivative, modeling by fractional differential equations have gained much importance to describe complex phenomena with memory which is the case in many biological systems (see [5, 6, 7, 8, 9]).

Population dynamics of infectious diseases, via fractional order derivative models, has been paid increasing attention in the past few years see for instance [5, 10, 11, 12]. In [5], the authors studied the memory effect on epidemic evolution using the Caputo fractional derivative. Dos Santos et al. [10] investigated the global stability of fractional SIR epidemic model. In [11], Mouaouine et al. presented and analyzed a fractional epidemic model with nonlinear incidence rate. Many other works involving fractional order derivative are mainly concerned with SIR-type models (see for instance [7, 8, 13, 14, 15]). In this paper, we consider a fractional vaccinated epidemic model with non-monotonic incidence function given by

$$(1.1) \quad \begin{cases} D^\alpha S = \Lambda - \beta G(I)S - (\mu + \nu)S, \\ D^\alpha I = \beta G(I)S - (\mu + d + r)I, \\ D^\alpha R = \nu S + rI - \mu R. \end{cases}$$

The population is divided into three compartments, depending on the epidemiological status of individuals: numbers of susceptible ($S(t)$), infectious ($I(t)$) and recovered ($R(t)$) at time t . Λ is the recruitment rate, μ is the natural death rate, while d is the death rate due to disease and r is the recovery rate of the infectious individuals. To introduce the role of intervention strategies into the fractional order SIR system, the incidence rate is modeled by a general non-monotonic incidence function introduced by Cai et al. [16]. According to the same authors, $G(I) = \frac{I}{f(I)}$, where $0 \leq \frac{1}{f(I)} < \infty$ represent the intervention strategies (see [18]) for the reduction of valid contact coefficient β , $f'(I) > 0$, and there is a $\delta > 0$ such that $\left(\frac{I}{f(I)}\right)' > 0$ for $0 < I \leq \delta$ and $\left(\frac{I}{f(I)}\right)' < 0$ for $I \geq \delta$. If $G(I)$ is a non-monotonic function, it means that $G(I)$ increases when the infected population is small and decreasing when the number of infected individuals becomes important, this can be used to explain psychological effect: the infection force can decrease when the number of infected individuals is increasing, which leads to a reduction of the number of contacts per unit of time, (for more details see [16, 17, 18, 19]).

Since the two first equations in system (1.1) are independent of the third equation, we can reduce this system to the following:

$$(1.2) \quad \begin{cases} D^\alpha S = \Lambda - \beta G(I)S - (\mu + \nu)S, \\ D^\alpha I = \beta G(I)S - (\mu + d + r)I. \end{cases}$$

This study is outlined as follows. In the next section, we present some preliminaries results. We show that our model is biologically and mathematically well posed. In Section 3, using the fractional Lyapunov method, we study the global stability of the proposed model. In the last part we present some numerical simulations examples to support our theoretical results.

2. PRELIMINARIES AND EQUILIBRIA

There are many definitions of fractional derivative. Each of them has own physical interpretation. In this section, we will only introduce the Caputo definition that we will need in the rest of this paper.

Definition 2.1. *The fractional integral of order $\alpha > 0$ of a function $f : \mathbb{R}_+ \rightarrow \mathbb{R}$ is defined as follows:*

$$I^\alpha f(t) = \frac{1}{(\alpha)} \int_0^t (t-x)^{\alpha-1} f(x) dx,$$

Where $(.)$ is the Gamma function.

Definition 2.2. *The Caputo fractional derivative of order $\alpha > 0$ of a classe C^n function $f : \mathbb{R}_+ \rightarrow \mathbb{R}$ is given by*

$$(2.1) \quad D_t^\alpha f(t) = I^{n-\alpha} D_t^n f(t),$$

where $\alpha \in (n-1, n)$, $n \in \mathbb{N}^*$. In particular, when $\alpha \in (0, 1)$, we have

$$(2.2) \quad D_t^\alpha f(t) = \frac{1}{(1-\alpha)} \int_0^t \frac{f'(x)}{(t-x)^\alpha} dx.$$

Definition 2.3. *Let $\alpha > 0$. The function E_α defined by*

$$E_\alpha(z) = \sum_{j=0}^{\infty} \frac{z^j}{(\alpha j + 1)}$$

is called the Mittag-Leffler function of parameter α .

Let $X(t) = \begin{pmatrix} S(t) \\ I(t) \end{pmatrix}$, then we can reformulate the system (1.2) as follows:

$$(2.3) \quad D^\alpha X(t) = F(X(t)),$$

where

$$F(X(t)) = \begin{pmatrix} \Lambda - \beta G(I)S(t) - (\mu + \nu)S(t) \\ \beta G(I)S(t) - (\mu + d + r)I(t) \end{pmatrix}.$$

For biological reasons, we assume that :

$$(2.4) \quad S(0) \geq 0, I(0) \geq 0.$$

In order to establish the global existence of solutions for system (1.2) with initial conditions (2.4), we need the following lemma.

Lemma 2.1. *Assume that the vector function $F : \mathbb{R}^2 \rightarrow \mathbb{R}^2$ satisfies the following conditions:*

- (1) $F(X)$ and $\frac{\partial F}{\partial X}$ are continuous.
- (2) $\|F(X)\| \leq \omega + \lambda \|X\| \forall X \in \mathbb{R}^2$, where ω and λ are two positive constants.

Then system (1.2) has a unique solution.

The proof of this lemma follows immediately from [22]

Theorem 2.1. *For any initial conditions satisfying (2.4), then system (1.2) has a unique solution on $[0, \infty)$, and this solution remains non-negative and bounded for all $t \geq 0$. In addition, we have*

$$N(t) \leq N(0) + \frac{\Lambda}{\mu},$$

where $N(t) = S(t) + I(t)$.

Proof. Let

$$\varepsilon = \begin{pmatrix} \Lambda \\ 0 \end{pmatrix}, A = \begin{pmatrix} -(\mu + \nu) & 0 \\ 0 & -(\mu + d + r) \end{pmatrix}, B = \begin{pmatrix} -\beta \\ \beta \end{pmatrix}$$

then system can be written as follows:

$$(2.5) \quad F(X) = \varepsilon + AX + B \frac{IS}{f(I)}.$$

Then

$$\|F(X)\| \leq \|\varepsilon\| + \|B\| \left\| \frac{IS}{f(I)} \right\| + \|A\| \|X\|.$$

It follows by Lemma 2.1 that the system has a unique solution. Next, we establish the non-negativity of this solution. Since

$$D^\alpha S_{t=0} = \Lambda \geq 0,$$

$$D^\alpha I_{t=0} = 0.$$

Then we deduce that the solution of system (1.2) remains non-negative for all $t \geq 0$.

Finally, we prove the boundedness of solution. From system (1.2), and by adding all the equations, we obtain

$$(2.6) \quad \begin{aligned} D^\alpha N(t) &= \Lambda - (\mu + \nu)S(t) - (\mu + d + r)I(t) \\ &\leq \Lambda - \mu N(t). \end{aligned}$$

Solving (2.6), we get

$$D^\alpha N(t) \leq \left(-\frac{\Lambda}{\mu} + N(0) \right) E_\alpha(-\mu t^\alpha) + \frac{\Lambda}{\mu},$$

Since $0 \leq E_\alpha(-\mu t^\alpha) \leq 1$, we get

$$N(t) \leq N(0) + \frac{\Lambda}{\mu}.$$

This completes the proof. □

We define the basic reproduction number R_0 [23] of our model by

$$R_0 = \frac{\beta \Lambda}{(\mu + \nu)(\mu + d + r)f(0)}.$$

First, we discuss the existence of equilibria for system (1.2). It is easy to see that system (1.2) has always a disease-free equilibrium $E_0(S_0, 0)$ where $S_0 = \frac{\Lambda}{\mu}$. We assume that $R_0 > 1$ and suppose that $E^*(S^*, I^*)$ is an endemic equilibrium, we show the existence and the uniqueness of E^* , such that $S^* > 0, I^* > 0$ and

$$(2.7) \quad \begin{cases} \Lambda - \beta \frac{I^* S^*}{f(I^*)} - (\mu + \nu)S^* = 0, \\ \beta \frac{I^* S^*}{f(I^*)} - (\mu + d + r)I^* = 0. \end{cases}$$

It follows that

$$(2.8) \quad S^* = \frac{\mu + d + r}{\beta} f(I^*)$$

and I^* determined by

$$(2.9) \quad \frac{\beta}{(\mu + \nu)(\mu + d + r)} = \frac{\beta}{(\mu + \nu)} I^* + f(I^*).$$

Substituting (2.8) into the first equation of system (2.7), we obtain

$$\Lambda - \frac{(\mu + \nu)(\mu + d + r)}{\beta} f(I^*) - (\mu + d + r)I^* = 0.$$

Let H be the function defined as

$$H(x) = \Lambda - \frac{(\mu + \nu)(\mu + d + r)}{\beta} f(x) - (\mu + d + r)x.$$

We prove that the equation $H(x) = 0$ has a unique solution. We have

$$H'(x) = -\frac{(\mu + \nu)(\mu + d + r)}{\beta} f'(x) - (\mu + d + r),$$

we can see that $H' < 0$, then H is a decreasing function. On the other hand, we have $\lim_{x \rightarrow \infty} H(x) = -\infty$ and

$$\begin{aligned} H(0) &= \Lambda - \frac{(\mu + \nu)(\mu + d + r)}{\beta} f(0) \\ &= \frac{(\mu + \nu)(\mu + d + r)}{\beta} (R_0 - 1). \end{aligned}$$

Since $R_0 > 1$ we have $H(0) > 0$. Therefore, by means of the intermediate value theorem and since H is decreasing, there is a unique solution of the equation $H(x) = 0$. It follows that system (1.2) has unique endemic equilibrium $E^* = (S^*, I^*)$.

From the discussion above we get the following result:

Theorem 2.2. (1) *The system (1.2) has a unique disease-free equilibrium E_0 if $R_0 \leq 1$.*

(2) *If $R_0 > 1$, the disease-free equilibrium is still present and system (1.2) has a unique endemic equilibrium $E^*(S^*, I^*)$.*

3. GLOBAL STABILITY OF THE EQUILIBRIA

In this section, we investigate the global stability of the disease-free equilibrium E_0 and the endemic equilibrium E^* .

Theorem 3.1. *If $R_0 \leq 1$, the disease-free equilibrium E_0 is globally asymptotically stable.*

Proof. We consider the following Lyapunov function:

$$\Phi_0(t) = \left(S - S_0 - S_0 \ln \frac{S}{S_0} \right) + I.$$

We calculate the fractional time derivation of Φ_0 along the solution of system (1.2). Then we get

$$D^\alpha \Phi_0(t) \leq \left(1 - \frac{S_0}{S} \right) D^\alpha S + D^\alpha I,$$

Using the fact that $\Lambda = (\mu + \nu)S_0$, we obtain

$$\begin{aligned} D^\alpha \Phi_0(t) &\leq -\frac{\mu + \nu}{S}(S - S_0)^2 - \frac{\beta(S - S_0)}{f(I)}I + \beta \frac{SI}{f(I)} - (\mu + d + r)I \\ &= -\frac{\mu + \nu}{S}(S - S_0)^2 + \beta \frac{S_0 I}{f(I)} - (\mu + d + r)I. \end{aligned}$$

Using the fact that $f(I)$ is an increasing function, then $\frac{1}{f(I)} \leq \frac{1}{f(0)}$. It follow that

$$\begin{aligned} D^\alpha \Phi_0(t) &\leq -\frac{\mu + \nu}{S}(S - S_0)^2 + \beta \frac{S_0 I}{f(0)} - (\mu + d + r)I \\ &= -\frac{\mu + \nu}{S}(S - S_0)^2 + (\mu + d + r)(R_0 - 1)I. \end{aligned}$$

Since $R_0 \leq 1$, then $D^\alpha \Phi_0(t) \leq 0$. Furthermore $D^\alpha \Phi_0(t) = 0$ holds, if and only if $S = S_0$ and $I = 0$. Consequently, the largest invariant set of $\{(S, I) \in \mathbb{R}_+^2 : D^\alpha \Phi_0(t) = 0\}$ is the singleton $\{E_0\}$. By fractional LaSalle's invariance principle [6, 24], E_0 is globally asymptotically stable. \square

Theorem 3.2. *The endemic equilibrium E^* is globally asymptotically stable whenever $R_0 > 1$.*

Proof. We consider the following Lyapunov function:

$$\Phi_1(t) = \frac{1}{2}(S - S^* + I - I^*)^2 + a \left(I - I^* - I^* \ln \frac{I}{I^*} \right),$$

where a is a positive constant to be determined later. We calculate the fractional time derivation of Φ_1 along the solution of system (1.2). Then we obtain

$$D^\alpha \Phi_1(t) \leq (D^\alpha S + D^\alpha I)(S - S^* + I - I^*) + a \left(1 - \frac{I^*}{I} \right) D^\alpha I,$$

hence

$$\begin{aligned} D^\alpha \Phi_1(t) &\leq (S - S^* + I - I^*)(\Lambda - (\mu + \nu)S - (\mu + d + r)I) \\ &\quad + a \left(1 - \frac{I^*}{I} \right) \left(\beta \frac{SI}{f(I)} - (\mu + d + r)I \right). \end{aligned}$$

Using the fact that

$$\begin{aligned} \Lambda &= (\mu + \nu)S^* + (\mu + d + r)I^*, \\ \mu + d + r &= \frac{\beta S^*}{f(I^*)}, \end{aligned} \tag{3.1}$$

we obtain

$$\begin{aligned}
D^\alpha \Phi_1(t) &\leq (S - S^* + I - I^*)((\mu + \nu)(S^* - S) + (\mu + d + r)(I^* - I)) \\
&\quad + \frac{a\beta}{f(I^*)}(I - I^*)(S - S^*) - a\beta(I - I^*) \left(\frac{1}{f(I^*)} - \frac{1}{f(I)} \right) S \\
&\leq -(\mu + \nu)(S - S^*)^2 - (\mu + d + r)(I - I^*)^2 \\
&\quad + \left[a \frac{\beta}{f(I^*)} - (2\mu + d + \nu + r) \right] (S - S^*)(I - I^*) \\
&\quad - a\beta(I - I^*) \left(\frac{1}{f(I^*)} - \frac{1}{f(I)} \right) S.
\end{aligned}$$

Choose $a = \frac{(2\mu + d + r + \nu)}{\beta} f(I^*)$, since $f(I)$ is an increasing function we have $(I - I^*) \left(\frac{1}{f(I^*)} - \frac{1}{f(I)} \right) \geq 0$. Then we get

$$\begin{aligned}
D^\alpha \Phi_1(t) &\leq -(\mu + \nu)(S - S^*)^2 - (\mu + d + r)(I - I^*)^2 \\
&\quad - a\beta(I - I^*) \left(\frac{1}{f(I^*)} - \frac{1}{f(I)} \right) S.
\end{aligned}$$

Therefore, $D^\alpha \Phi_1(t) \leq 0$. Hence, the largest invariant set of $\{(S, I) \in \mathbb{R}_+^2 : D^\alpha \Phi_1(t) = 0\}$ is the singleton $\{E^*\}$. By fractional LaSalle's invariance principle [6, 24], E^* is globally asymptotically stable. \square

4. MAIN RESULTS: NUMERICAL SIMULATIONS

In this section, we illustrate our mathematical findings by numerical simulations, for this purpose we consider the non-monotonic incidence function presented by Xiao and Ruan in [17] as follow:

$$(4.1) \quad G(I) = \frac{I}{f(I)} = \frac{I}{1 + mI^2}.$$

Then the fractional order epidemic system becomes:

$$(4.2) \quad \begin{cases} D^\alpha S(t) = \Lambda - \beta \frac{I(t)S(t)}{1 + mI(t)^2} - (\mu + \nu)S(t), \\ D^\alpha I(t) = \beta \frac{I(t)S(t)}{1 + mI(t)^2} - (\mu + d + r)I(t). \end{cases}$$

It is easy to see that

$$R_0 = \frac{\beta\Lambda}{(\mu + \nu)(\mu + d + r)}.$$

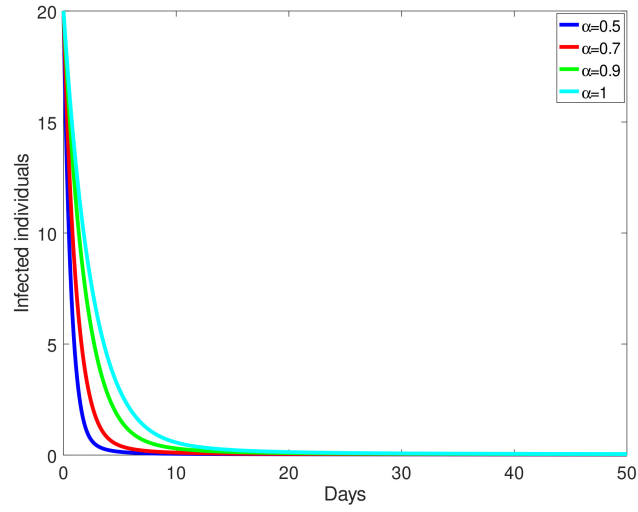


FIGURE 1. Stability of the infected population when $R_0 = 0.98$.

In order to solve system (4.2), we apply the algorithm presented in [25] and we will use the parameter values from the Table 1.

Parameters	Fig. 1	Fig. 2	Fig. 3	Fig. 4
Λ	30	30	17	17
μ	0.08	0.08	0.08	0.08
ν	0.001	0.001	0.001	0.001
d	0.1	0.1	0.1	0.1
r	0.2	0.2	0.2	0.2
β	0.001	0.001	0.003	0.003
m	2	2	2	2

TABLE 1. The taken parameters for the different results of global stability.

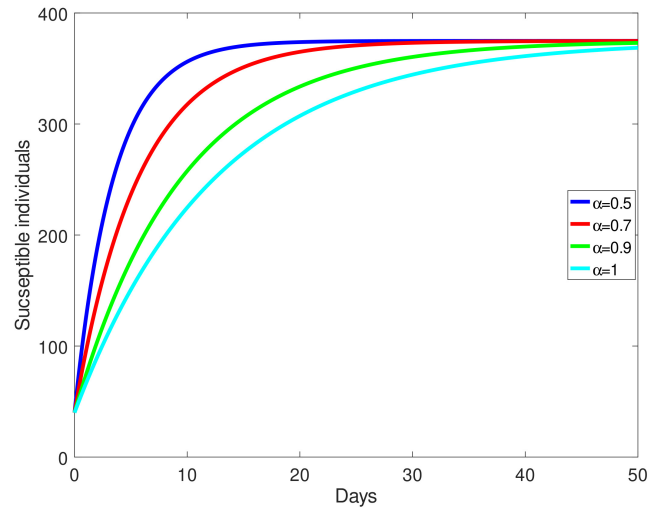


FIGURE 2. Stability of the susceptible population when $R_0 = 0.98$.

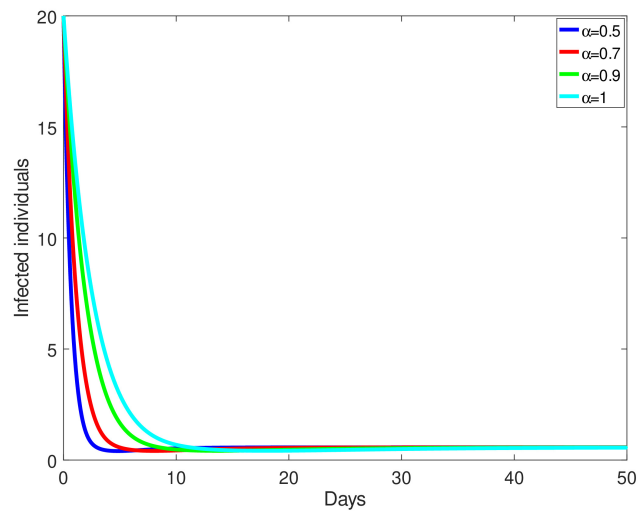


FIGURE 3. Stability of the infected population when $R_0 = 1.67$.

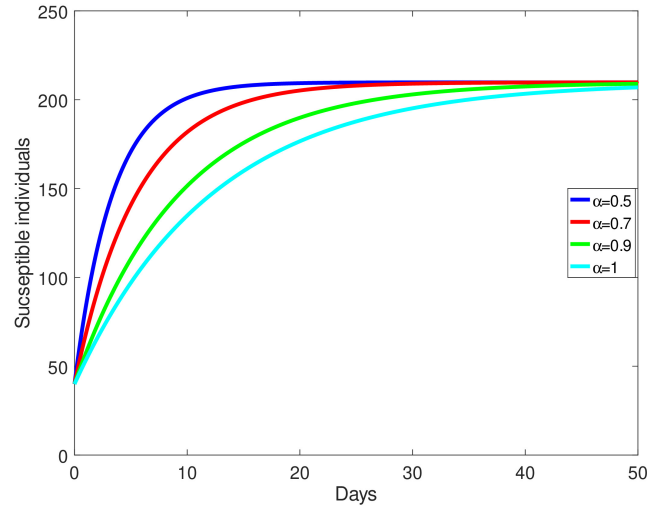


FIGURE 4. Stability of the susceptible population when $R_0 = 1.67$.

Figures 1 and 2 show the evolution of the number of infected and susceptible individuals during the period of observation. The used parameters in these figures are given in Table 1; for these parameters, we have $R_0 = 0.98 < 1$. It can be seen that the four curves corresponding to different values of α converge toward the disease-free equilibrium E_0 , this indicates that the disease-free equilibrium E_0 is globally asymptotically stable which is consistent of the theoretical results.

Figures 3,4 depict the dynamics of infected and susceptible population during the period of observation. In these figures, the basic reproduction number R_0 is greater than unity, it can be seen that the four curves corresponding to different values of α converge toward the endemic equilibrium E^* , this indicates that the disease-free equilibrium E_0 is globally asymptotically stable which support the theoretical results.

Finally, we can clearly observe that the different values of α affect the time to reach the steady states, but have no effect on the stability of disease-free equilibrium and the endemic equilibrium. In addition, the system converges rapidly to its equilibrium states when the value of α is very small.

5. FIELDS OF APPLICATION OF FRACTIONAL MODELS

The coronavirus infectious disease (COVID-19) is a novel respiratory disease reported in 2019 in China. The infection is very destructive to human lives and caused millions of deaths, this is why several fractional epidemic models have been applied in this direction. Note that many developments or adaptations of these classic models have been introduced very recently, during the current pandemic Covid 19. The primary objective of these models is to predict the course of the disease in a specific context, with measures aimed at limiting the spread of the disease.

In [26] they study the effectiveness of the modelling approach on the pandemic due to the spreading of the novel COVID-19 disease and develop a susceptible-infected-removed (SIR) model that provides a theoretical framework to investigate its spread within a community. This work shows the importance of modelling the spread of COVID-19 by the SIR model that they propose here, as it can help to assess the impact of the disease by offering valuable predictions.

6. CONCLUSION

In this paper, a fractional vaccinated SIR epidemic model with the Caputo fractional derivative and general non-monotone incidence rate is presented and analyzed. We first established the global existence, positivity and boundedness of solutions as this is essential in any population dynamics models. Using Lyapunov method and fractional La-Salle invariance principle for fractional differential equations, we proved the global stability of both equilibria. Numerical simulations are carried to support the theoretical results. The results show that different values of α affect the time to reach the steady states, but have no effect on the stability of disease-free equilibrium and the endemic equilibrium, and the smaller the value of the fractional order α the faster the system converges to its equilibrium states.

In the near future, it will be interesting to explore more mathematical tools to reflect the reality, for example using fractional stochastic differential equations to discuss the qualitative behavior of the model.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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