Stability Analysis of Fractional SIR Model Related to Delay in State and Control Variables

Sanaa L. Khalaf*, Hadeer S. Flayyih

Department of Mathematics, College of Science, Basrah University, Basrah, Iraq

*Corresponding author, E-mail: **sanaasanaa1978@yahoo.com**

Doi:10.29072/basjs.202123

Abstract

The study of a nonlinear mathematical fractional SIR (Susceptible - Infected - Recovered) epidemiological model related to the delay in state and control variables in terms of time is the focus of this paper. The existence of a bounded solution for the fractional SIR epidemic model has been demonstrated, and it is unique. A new set of infection-free equilibrium points has been discovered, and their local stability has been investigated. In addition, using the next-generation matrix method, the basic reproductive number R_0 was calculated.

Article inf.

Received:
31/5/2021
Accepted
7/8/2021
Published
31/8/2021

keywords:

Fractional SIR, Caputo fractional derivative, Local stability, Reproductive number.

1. Introduction

Infectious disease epidemiology is the study and forecasting of the occurrence, spread, and control of infectious diseases in populations. It deals with identifying the factors that effect to disease spread, and then improving the quality of care and health services by providing appropriate strategies for prevention, treatment, and preparation in order to increase the efficiency and efficacy of health services [1, 2]. However, developing and analyzing mathematical models is an important approach in biological mathematics for investigating development rules and determining effective control strategies. The classical susceptible-infectious-recovered (SIR) model, which was inspired by the important papers of Ross [3] in 1916 and Ross and Hudson [4, 5] in 1917, as well as the major contributions of Kermack and McKendrick [6, 7, 8] in 1927-1933, describes the transmission of infectious diseases between susceptible and infective individuals and serves as the basis for almost all subsequent epidemic modeling. Since then, epidemic models have been widely developed in a variety of situations, we mention to the monographs Bailey [9], Thieme [10], Muench [11], Busenberg and Cooke [12], Murray [13] for more information on these issues.

To focus on the dynamical behavior of infectious diseases, studies always ignore demography, such as the death and birth processes, as well as the immigration/emigration process. In this case, the epidemic model will be based on three main rules: contacts between individuals, transmission per contact, and infection development at the individual level [1, 11]. Because an infection does not develop instantly, the third rule can be described by introducing delay between the pathogen's transmission and the moment at which an exposed individual becomes able of transmitting the infection. This delay can be defined using either an extra exposed class (when the period of delay follows an exponential law), resulting in SEIR models, or an age of infection, resulting in age-structured models; for more information on these topics, see [14, 15, 16]. In 2020 [17], M. A. Khan et al. demonstrated the dynamics of a fractional SIR model with a generalized incidence rate using two differential derivatives, the Caputo and the Atangana-Baleanu.

Wang (2006) [18] suggested a model of an epidemic with a restricted treatment budget to better understand the impact of treatment capacity. Driving the basic reproduction number R_0 is not enough to eliminate the disease, as has been shown. In 2008 [19], a disease model with a

saturated occurrence rate and saturated treatment function was investigated, and it was found to be more accurate than the linear form model in describing the real system. In (2009) [20], a SIR epidemic model with nonlinear incidence rate and time delay was investigated. Meanwhile, the endemic equilibrium point's global asymptotic stability has been discussed. More recent research in (2010) [21] has demonstrated the global asymptotic stability by using fewer conditions than (2009) [20]. However, it's worth noting that most previously published works' mathematical models are primarily based on ordinary differential equations (ODEs). Engineering [22, 23, 24, 25] and biology [26, 27, 28] are two fields where fractional calculus has recently become popular. Fractional models of rabies and predator-prey have been investigated in (2007) [29], with the result that fractional differential equations (FDEs) are at least as more robust than their ODEs equivalents. Fractional calculus, as is well recognized, is naturally linked to many adaptive systems of memory and genetic properties that are found in biology. As a result, when utilizing FDEs to describe biological models, fractional derivatives can be fully utilized. Several fractional-order biological models have been proposed recently [30, 31]. The most common method for obtaining the stability conditions for an ODE's equilibrium point is Lyapunov's direct method, which is well known. The authors of [32, 33] developed the fractional Lyapunov direct method for FDEs by introducing the principle of Mittag–Leffler stability. Delavari et al., (2012) [34] suggested that FDEs have global uniform asymptotic stability as an extension.

The following is the layout of this paper: in section 2, we gave some preliminary definitions of divertive and integral in the fractional concept. Then, in order to discuss the stability analysis, some useful theorems and lemma were added. Section 3 depicts the Caputo fractional SIR model with delay in state and control variables. We confirmed that the model's solutions were unique and non-negative, and looked into their stability in section 4. Finally, came to a conclusion in section 5.

2. Some Fractional Calculus Concepts

The definitions of fractional derivative and fractional integral are presented in this section With some key properties and theorems.

Definition 1 [35] The left and right Riemann-Liouville fractional (R-LF) integrals of order α are given by:

$$ {}_aJ_t^{\alpha} z(t) = \frac{1}{\Gamma(\alpha)} \int_a^t (t-\xi)^{\alpha-1} z(\xi) d\xi \tag{1} $$

$$ {}_tJ_b^{\alpha} z(t) = \frac{1}{\Gamma(\alpha)} \int_t^b (\xi-t)^{\alpha-1} z(\xi) d\xi . \tag{2} $$

Also, the right (left) Riemann-Liouville fractional (R-LF) derivative and the right (left) Caputo fractional (CF) derivative of order α are defined as follows:

$$ {}_tD_b^{\alpha} z(t) = \frac{1}{\Gamma(m-\alpha)} (-1)^m \frac{d^m}{dt^m} \int_t^b (\xi-t)^{m-\alpha-1} z(\xi) d\xi \tag{3} $$

$$ {}_aD_t^{\alpha} z(t) = \frac{1}{\Gamma(m-\alpha)} \frac{d^m}{dt^m} \int_a^t (t-\xi)^{m-\alpha-1} z(\xi) d\xi \tag{4} $$

$$ {}_t^C D_b^{\alpha} z(t) = \frac{(-1)^m}{\Gamma(m-\alpha)} \int_t^b (\xi-t)^{m-\alpha-1} z^{(m)}(\xi) d\xi \tag{5} $$

$$ {}_a^C D_t^{\alpha} z(t) = \frac{1}{\Gamma(m-\alpha)} \int_a^t (t-\xi)^{m-\alpha-1} z^{(m)}(\xi) d\xi . \tag{6} $$

Where $\alpha \in (m-1, m)$, $m \in \mathbb{N}$.

Properties [36]

1- Let $z_1, z_2 : [a,b] \to \mathbb{R}$. Then there are $\beta_1, \beta_2 \in \mathbb{R}$ such that

$$ {}_a^C D_t^{\alpha} (\beta_1 z_1(t) + \beta_2 z_2(t)) = \beta_1 {}_a^C D_t^{\alpha} z_1(t) + \beta_2 {}_a^C D_t^{\alpha} z_2(t) \tag{7} $$

2- Let K is constant. Then

$$ {}_a^C D_t^{\alpha} K = 0 \tag{8} $$

Remark [37] R-LF derivative and CF derivative have the following relationship:

$$ {}_aD_t^{\alpha} z(t) = {}_a^C D_t^{\alpha} z(t) + \sum_{i=0}^{m-1} \frac{z^{(i)}(a)}{\Gamma(\mathrm{i}+1-\alpha)} (t-a)^{i-\alpha} \tag{9} $$

$$ {}_tD_b^{\alpha} z(t) = {}_t^C D_b^{\alpha} z(t) + \sum_{i=0}^{m-1} \frac{z^{(i)}(\mathrm{b})}{\Gamma(\mathrm{i}+1-\alpha)} (t-b)^{i-\alpha} \tag{10} $$

Consider the following generalized Caputo fractional differential equation [38]:

$$ {}_a^C D_t^{\alpha} z(t) = g(t, z(t)), \qquad 0 < \alpha \le 1 \tag{11} $$

Subject to

$$ z(t_0) = z_0 \tag{12} $$

The CF differential equation (11) has equilibrium point $z^*(t)$ if and only if $g(t, z^*(\mathrm{t})) = 0$.

Theorem 1 [39] If all of the eigenvalues of the Jacobian matrix of system (11) satisfy the following condition at the equilibrium point, the Caputo fractional dynamic system (11) is locally asymptotically stable.

$$ |\arg(\lambda)| > \frac{\alpha\pi}{2} \tag{13} $$

Definition 2 [40] Suppose that $\mathrm{H}(s)$ is the Laplace transform of the function $\mathrm{H}(t)$. Then $\mathrm{H}(s)$ of the Caputo derivative is get as follow

$$ L\{{}_a^C D_t^{\alpha} \mathrm{H}(t), s\} = s^{\alpha} \mathrm{H}(s) - \sum_{i=0}^{m-1} s^{\alpha-i-1} \mathrm{H}^{(i)}(0), \quad \alpha \in (m-1, m),\ m \in \mathbb{N} \tag{14} $$

Definition 3 [40] The function $E_{\mathrm{r,n}}(t)$ for $t \in \mathbb{R}$ is defined by

$$ E_{\mathrm{r,n}}(t) = \sum_{i=0}^{\infty} \frac{t^i}{\Gamma(\mathrm{r}\, i + n)}, \qquad r, \mathrm{n} > 0 \tag{15} $$

Where $E_{\mathrm{r,n}}(t)$ is called the generalized Mittag-Leffler function and satisfies

$$ 1- \quad E_{\mathrm{r,n}}(t) = t\, E_{\mathrm{r,r}+n}(t) + \frac{1}{\Gamma(\mathrm{n})}, \qquad r, \mathrm{n} > 0 \tag{16} $$

$$2- \quad L\{t^{n-1}E_{r,n}(\pm\beta t^r)\} = \frac{s^{r-n}}{s^r \mp \beta} \tag{17}$$

Where L is the Laplace transform of $E_{r,n}(t)$.

3. Mathematical Fractional SIR Model

In this model, the inhabitance is divided into three parts of the disease: (S) represents people exposed to the disease; (I) represents people who carry the disease and they are able to transmit it to others; (R) represents people recovering from illness. One can contract the disease by coming into contact with people who have it, and once do, he will be immune to it for the rest of your life.

The next set of fractional differential equations with non-negative initial conditions describes the dynamics of people.

$$\begin{aligned} {}_0^C D_t^\alpha S(t) &= \Lambda - \beta S(t)\frac{I(t)}{\bar{N}(t)} - dS(t) \\ {}_0^C D_t^\alpha I(t) &= \beta S(t)\frac{I(t)}{\bar{N}(t)} - (\gamma + d + \varepsilon)I(t) \\ {}_0^C D_t^\alpha R(t) &= \gamma I(t) - dR(t) \end{aligned} \tag{18}$$

Such that

$$S(0) = S_0, I(0) = I_0, R(0) = R_0 \tag{19}$$

and $\bar{N}(t) = S(t) + I(t)$ I symbolize the universal people number at t time. Where the movement between the dissimilar states are qualified by the parameters found in the Table 1:

Table 1: Represent parameters for SIR fractional model

Parameter	Description
Λ	The recruitment people rate of susceptible.
β	The rate efficient connect.
d	The rate normal mortality.
γ	The recovery rate.
ε	The death rate resulting from disease.

The aim of this study is to reduce the number of vulnerable and injured persons to a minimum and to maximize the number of patients who get remedies. After being exposed to injury, we include a control that shows the percentage of people who are vaccinated per time unit (see model 18). To obtain a more realistic model, bearing in mind that the movement of people exposed to infection who have been vaccinated from the susceptibility class to the recovered class is subject to delay. Therefore, we will enter the time delay in the model as follows: at time t only a percentage of exposed peoples that have been taken the vaccine τ time unit ago, that is, at time $t-\tau$, are subtracted from the susceptible and added to the recovered group. The nonlinear fractional equations give model with time delay in state and control variables as follows:

$$\begin{aligned} {}_0^C D_t^\alpha S(t) &= \Lambda - \beta S(t)\frac{I(t)}{\bar{N}(t)} - dS(t) - u(t-\tau)S(t-\tau) \\ {}_0^C D_t^\alpha I(t) &= \beta S(t)\frac{I(t)}{\bar{N}(t)} - (\gamma + d + \varepsilon)I(t) \\ {}_0^C D_t^\alpha R(t) &= \gamma I(t) - dR(t) + u(t-\tau)S(t-\tau) \end{aligned} \tag{20}$$

For biological causes, we suppose, $S(\varphi), I(\varphi), R(\varphi)$ are non-negative continuous functions and $u(\varphi)=0$ for $\varphi \in [-\tau, 0]$. The control u is presumed to be integrable, $0 \le u \le b < 1$ and b is a given constant.

Theorem 2 The region $\Psi = \{(S, I, R) \in \mathbf{R}^{+3} : S \ge 0, I \ge 0, R \ge 0\}$ for system (20) is uniformly bounded.

Proof: From (20) the total population satisfies

$${}_0^C D_t^\alpha \bar{N}(t) = \Lambda - d\bar{N}(t) - \varepsilon I(t) \tag{21}$$

Where $\bar{N}(t) = S(t) + I(t) + R(t)$.

$${}_0^C D_t^\alpha \bar{N}(t) \le \Lambda - d\bar{N}(t) \tag{22}$$

We can get Eq. (22) by applying the Laplace transform.

$$s^\alpha L\{\bar{N}(t)\} - s^{\alpha-1}\bar{N}(0) \le \frac{\Lambda}{s} - d\, L\{\bar{N}(t)\} \tag{23}$$

$$(s^\alpha + d)\,\mathrm{L}\{\bar{N}(t)\} \le \frac{\Lambda}{s} + s^{\alpha-1}\bar{N}(0) \tag{24}$$

$$L\{\bar{N}(t)\} \le \frac{s^{-1}}{s^{\alpha}+d}\Lambda + \frac{s^{\alpha-1}}{s^{\alpha}+d}\bar{N}(0) \tag{25}$$

Then can deduce from Eqs. (17) and (16) that if $(S,I,R) \in \Psi$, then

$$\bar{N}(t) \le \Lambda t^{\alpha} \mathrm{E}_{\alpha,\alpha+1}(-dt^{\alpha}) + \mathrm{E}_{\alpha,1}(-dt^{\alpha})\bar{N}(0) \tag{26}$$

$$\le \frac{\Lambda}{d}[dt^{\alpha}\mathrm{E}_{\alpha,\alpha+1}(-dt^{\alpha}) + \mathrm{E}_{\alpha,1}(-dt^{\alpha})] \tag{27}$$

$$\le \frac{\Lambda}{d}[dt^{\alpha}\mathrm{E}_{\alpha,\alpha+1}(-dt^{\alpha}) - dt^{\alpha}\mathrm{E}_{\alpha,\alpha+1}(-dt^{\alpha}) + \frac{1}{\Gamma(1)}] \tag{28}$$

$$\le \frac{\Lambda}{d} \tag{29}$$

Solutions in the system (20) are restricted as follows

$$\Psi = \{(S,I,R) \in \mathbf{R}^{+3} : \bar{N}(t) \le \frac{\Lambda}{d}\} \tag{30}$$

Theorem 3 There is a unique solution for (20) satisfying $S \ge 0, I \ge 0$ and $R \ge 0$ for $t \ge 0$.

Proof Rewrite (20) in the following form:

$${}_{0}^{C}D_{t}^{\alpha}Y(t) = AY(t) + G(Y(t), Y(t-\tau)) = F(Y(t), Y(t-\tau)) \tag{31}$$

Where

$$Y(t) = [S(t) \quad I(t) \quad R(t)]^{T}, \tag{32}$$

$$A = \begin{pmatrix} -d & 0 & 0 \\ 0 & -\gamma - d - \varepsilon & 0 \\ 0 & \gamma & -d \end{pmatrix}, \tag{33}$$

$$G(\mathrm{Y}, \mathrm{Y}_{\tau}) = [\Lambda - \beta S(t)\frac{I(t)}{\bar{N}(t)} \quad \beta S(t)\frac{I(t)}{\bar{N}(t)} \quad u(t-\tau)S(t-\tau)]^{T} \tag{34}$$

The function $G(\mathrm{Y}, \mathrm{Y}_{\tau})$ on the right-hand side of Eq. (31) satisfies

$$\left|G(Y_1(t), Y_1(t-\tau)) - G(Y_2(t), Y_2(t-\tau))\right| \le L_1 \left|Y_1(t) - Y_2(t)\right| + L_2 \left|Y_1(t-\tau), Y_2(t-\tau)\right| \tag{35}$$

Where L_1 , $L_2 \ge 0$, independent of the $S(t), I(t)$ and $R(t)$

Also

$$|Y_1(t)-Y_2(t)|=|S_1(t)-S_2(t)|+|I_1(t)-I_2(t)|+|R_1(t)-R_2(t)| \tag{36}$$

$$\begin{aligned}|Y_1(t-\tau)-Y_2(t-\tau)|=&|S_1(t-\tau)-S_2(t-\tau)|+|I_1(t-\tau)-I_2(t-\tau)|\\&+|R_1(t-\tau)-R_2(t-\tau)|\end{aligned} \tag{37}$$

Then from Eq. (31) we get

$$\begin{aligned}|F(Y_1(t),Y_1(t-\tau))-F(Y_2(t),Y_2(t-\tau))|&=|AY_1(t)+G(Y_1(t),Y_1(t-\tau))-AY_2(t)-G(Y_2(t),Y_2(t-\tau))|\\&=|A(Y_1(t)-Y_2(t))+G(Y_1(t),Y_1(t-\tau))-G(Y_2(t),Y_2(t-\tau))|\\&\le|A(Y_1(t)-Y_2(t))|+|G(Y_1(t),Y_1(t-\tau))-G(Y_2(t),Y_2(t-\tau))|\end{aligned} \tag{38}$$

From Eq. (35) we get

$$\begin{aligned}|F(Y_1(t),Y_1(t-\tau))-F(Y_2(t),Y_2(t-\tau))|&\le\|A\||Y_1(t)-Y_2(t)|+L_1|Y_1(t)-Y_2(t)|+L_2|Y_1(t-\tau),Y_2(t-\tau)|\\&=(\|A\|+L_1)|Y_1(t)-Y_2(t)|+L_2|Y_1(t-\tau),Y_2(t-\tau)|\end{aligned} \tag{39}$$

therefore, we get

$$|F(Y_1(t),Y_1(t-\tau))-F(Y_2(t),Y_2(t-\tau))|\le L(|Y_1(t)-Y_2(t)|+|Y_1(t-\tau),Y_2(t-\tau)|) \tag{40}$$

where

$$L=\max(\|A\|+L_1;L_2)<\infty \tag{41}$$

Since the function F is uniformly Lipschitz continuous. Then from the control definition $u(t)$ and the constraint on $S\ge 0, I\ge 0$ and $R\ge 0$, notice that a solution of the Eq. (31) exists [41].

4. Analysis of the Fractional SIR model's Stability

The equilibrium points (S^*,I^*,R^*) of model (20) can get by solving the next equations

$$\begin{aligned}&\Lambda-\beta S(t)\frac{I(t)}{\bar{N}(t)}-dS(t)-u(t-\tau)S(t-\tau)=0\\&\beta S(t)\frac{I(t)}{\bar{N}(t)}-(\gamma+d+\varepsilon)I(t)=0\\&\gamma I(t)-dR(t)+u(t-\tau)S(t-\tau)=0\end{aligned} \tag{42}$$

We note that the model (20) has a disease-free equilibrium point $E_0 = (S_0, I_0, R_0) = (\frac{\Lambda}{d}, 0, 0)$, which represents the removal of disease**.**

4.1 The basic reproductive number R_0

In this section, we will introduce the base reproduction number using the next generation matrix method to investigate the local stability of disease-free homeostasis [35].

Now, calculate the base reproduction number R_0, where R_0 is the eigenvalue of the matrix $G = FV^{-1}$, where F indicates new infections, while V indicates the transmission of infection from one place to another. Both are calculated in an equilibrium-free equilibrium state and are thus derived as follows.

Infectious compartments are found in the system (20) as follows.

$$\begin{aligned} {}_0^C D_t^\alpha S(t) &= \Lambda - \beta S(t)\frac{I(t)}{\bar{N}(t)} - dS(t) - u(t-\tau)S(t-\tau) \\ {}_0^C D_t^\alpha I(t) &= \beta S(t)\frac{I(t)}{\bar{N}(t)} - (\gamma + d + \varepsilon)I(t) \end{aligned} \tag{43}$$

Let $\Omega = [I, S]^T$. Then the system (43) can be written as

$${}_0^C D_t^\alpha \Omega = \tilde{F}(\Omega) - \tilde{V}(\Omega) \tag{44}$$

where

$$\tilde{F}(\Omega) = \begin{bmatrix} \beta S(t)\frac{I(t)}{\bar{N}(t)} \\ 0 \end{bmatrix} \tag{45}$$

and

$$\tilde{V}(\Omega) = \begin{bmatrix} (\gamma + d + \varepsilon)I(t) \\ -\Lambda + \beta S(t)\frac{I(t)}{\bar{N}(t)} + dS(t) + u(t-\tau)S(t-\tau) \end{bmatrix} \tag{46}$$

At the disease-free equilibrium point E_0, we obtain the matrices F and V as follows:

$$F = \left[\frac{\partial \tilde{F}_r(E_0)}{\partial y_p} \right] = \begin{bmatrix} 0 & \beta \\ 0 & 0 \end{bmatrix} \tag{47}$$

and

$$V = \left[\frac{\partial \tilde{V}_r(E_0)}{\partial y_p} \right] = \begin{bmatrix} 0 & \gamma + d + \varepsilon \\ d + \chi_{[0,T_f - \tau]} u(t) & \beta \end{bmatrix} \tag{48}$$

where $1 \le r$, $p \le 2$

where $$X_{[0,T_f-\tau]}(t) = \begin{cases} 1, & t \in [0, T_f - \tau] \\ 0, & otherwise \end{cases}$$

Matrix V is inversed as follows:

$$V^{-1} = \begin{bmatrix} -\dfrac{\beta}{(\gamma + d + \varepsilon)(d + \chi_{[0,T_f-\tau]} u(t))} & \dfrac{1}{d + \chi_{[0,T_f-\tau]} u(t)} \\ \dfrac{1}{\gamma + d + \varepsilon} & 0 \end{bmatrix} \tag{49}$$

Now, by multiplying matrices (47) and (49) we will get

$$G = \begin{bmatrix} \dfrac{\beta}{\gamma + d + \varepsilon} & 0 \\ 0 & 0 \end{bmatrix} \tag{50}$$

The eigenvalues from the matrix (50) are

$$\lambda = \begin{bmatrix} 0 \\ \dfrac{\beta}{\gamma + d + \varepsilon} \end{bmatrix} \tag{51}$$

It is from eigenvalues the R_0 is essentially known as

$$R_0 = \frac{\beta}{\gamma + d + \varepsilon} \tag{52}$$

Note that when $R_0 < 1$, then disease ends in the population and infection disappears. Also, when $R_0 > 1$ then disease will persist in the population.

4.2 Local stability of the infection-free equilibrium

Theorem 4 In the proposed fractional-order SIR epidemic model, the free equilibrium of disease E_0 is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 \geq 1$.

Proof: From the Caputo fractional system (20), we can get the Jacobian matrix as follows:

$$\tilde{J} = \begin{bmatrix} -d - \chi_{[0,T_f-\tau]}\, u(t) & -\beta & 0 \\ 0 & \beta - \gamma - d - \varepsilon & 0 \\ \chi_{[0,T_f-\tau]}\, u(t) & \gamma & -d \end{bmatrix} \tag{53}$$

We now use Eq. (53) to extract the Jacobian matrix's eigenvalues

$$\lambda_1 = -d \tag{54}$$

$$\lambda_2 = -d - \chi_{[0,T_f-\tau]}\, u(t) \tag{55}$$

$$\lambda_3 = \beta - \gamma - d - \varepsilon \tag{56}$$

Note that λ_1 and λ_2 have a real negative part. Now, we will prove that λ_3 has a real negative part.

Suppose that $\lambda_3 < 0$, we get

$$\beta - \gamma - d - \varepsilon < 0$$

$$\beta < \gamma + d + \varepsilon$$

$$\frac{\beta}{\gamma + d + \varepsilon} < 1 \tag{57}$$

Since $R_0 = \frac{\beta}{\gamma + d + \varepsilon}$, then from Eq. (57) we have $R_0 < 1$. Then, in the absence of infection, E_0 is locally asymptotically stable.

5. Conclusions

To investigate time delays in state and control variables, we used a Caputo fractional SIR epidemiological model. Then shown that the model's solutions are both bounded and positive. In the local condition that was checked in the stable model, also obtained the infection-free equilibrium point and the model was locally stable. The results of infection-free equilibrium of this model are locally asymptotically stable, depending on the reproduction number.

References

[1] R. R. Brookman, Infectious disease: Acquired immunodeficiency syndrome, Journal of Adolescent Health Care, **9** (1988) 246-247.

[2] F. Cobo, Epidemiology of infectious diseases in immigrants, in Imported Infectious Diseases, Elsevier, 2014.

[3] R. Ross, An application of the theory of probabilities to the study of a priori pathometry. Part I," Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character, **92 (**1916**)** 204-230.

[4] R. Ross, H. P. Hudson, An application of the theory of probabilities to the study of a priori pathometry. part II, Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character, **93** (1917) 212-225.

[5] R. Ross, H. P. Hudson, An application of the theory of probabilities to the study of a priori pathometry. part III, Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character, **93** (1917) 225-240.

[6] W. O. Kermack, A. G. McKendrick, A contribution to the mathematical theory of epidemics, Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character, **115** (1927) 700-721.

[7] W. O. Kermack, A. G. McKendrick, Contributions to the mathematical theory of epidemics. II. the problem of endemicity, Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character, **138** (1932) 55-83.

[8] W. O. Kermack, A. G. McKendrick, Contributions to the mathematical theory of epidemics. III. further studies of the problem of endemicity, Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character, **141 (**1933**)**, 94-122.

[9] N. T. Bailey, The mathematical theory of epidemics, tech. rep., 1957.

[10] K. J. Sharkey, Deterministic epidemiological models at the individual level," J. Math. Bio., **57** (2008) 311-331.

[11] H. Muench, Catalytic models in epidemiology. Harvard University Press, 2013.

[12] S. Busenberg, K. Cooke, Vertically transmitted diseases: models and dynamics, **23**. Springer Science & Business Media, 2012.

[13] J. D. Murray, Mathematical biology: I. An introduction, **17**. Springer Science & Business Media, 2007.

[14] H. R. Thieme, C. Castillo-Chavez, How may infection-age-dependent infectivity affect the dynamics of HIV/AIDS?, SIAM J. Appl. Math., **53** (1993) 1447-1479.

[15] G. F. Webb, Theory of nonlinear age-dependent population dynamics. CRC Press, 1985.

[16] H. R. Thieme, Mathematics in population biology, Princeton University Press, 2018.

[17] M. A. Khan, M. Ismail, S. Ullah, M. Farhan, Fractional order SIR model with generalized incidence rate, AIMS Math., **5** (2020) 1856-1880.

[18] W. Wang, Backward bifurcation of an epidemic model with treatment, Math. Bioscie., **201**(2006) 58-71.

[19] X. Zhang, X. Liu, Backward bifurcation of an epidemic model with saturated treatment function, J Math. Analy Appl., **348** (2008) 433-443.

[20] R. Xu, Z. Ma, Global stability of a SIR epidemic model with nonlinear incidence rate and time delay, Nonlinear Analysis: Real World Applic.,**10** (2009) 3175-3189.

[21] C. C. McCluskey, Global stability for an SIR epidemic model with delay and nonlinear incidence, Nonlinear Analysis: Real World Applic., **11** (2010) 3106-3109.

[22] Y. Fan, X. Huang, Z. Wang, Y. Li, Nonlinear dynamics and chaos in a simplified memristor-based fractional-order neural network with discontinuous memductance function, Nonlinear Dyn., **93** (2018) 611-627.

[23] Y. Fan, X. Huang, Z. Wang, Y. Li, Global dissipativity and quasi-synchronization of asynchronous updating fractional-order memristor-based neural networks via interval matrix method, J Frank Inst., **355** (2018) 5998-6025.

[24] Y. Fan, X. Huang, Z. Wang, Y. Li, Quasi-synchronisation of fractional-order memristor-based neural networks with parameter mismatches, IET Cont. Theory Applic., **11** (2017) 2317-2327.

[25] Z. Wang, X. Wang, Y. Li, X. Huang, Stability and hopf bifurcation of fractional-order complex-valued single neuron model with time delay, Int J Bifur Chaos, **27** (2017) 1750209-1750221.

[26] M. Javidi, N. Nyamoradi, Dynamic analysis of a fractional order prey–predator interaction with harvesting, Appl. Math Mod., **37** (2013) 8946-8956.

[27] D. Rostamy, E. Mottaghi, Stability analysis of a fractional-order epidemics model with multiple equilibriums, Adv. Diff. Equ., **170** (2016) 1-12.

[28] A. U. Z. Zafar, K. Rehan, M. Mushtaq, HIV/AIDS epidemic fractional-order model, J Diff. Equ Applic., **23** (2017) 1298-1315.

[29] E. Ahmed, A. El-Sayed, H. El-Saka, Equilibrium points, stability and numerical solutions of fractional-order predator–prey and rabies models, J Math Anal Applic., **325** (2007) 542-553.

[30] E. Ahmed, A. Elgazzar, On fractional order differential equations model for nonlocal epidemics, Phys. A: Stat. Mech. Appl., **379** (2007) 607-614.

[31] Y. Ding, H. Ye, A fractional-order differential equation model of HIV infection of CD4+ T-cells, Math. Comput. Model., **50** (2009) 386-392.

[32] Y. Li, Y. Chen, I. Podlubny, Mittag–leffler stability of fractional order nonlinear dynamic systems, Automatica, **45** (2009) 1965-1969.

[33] Y. Li, Y. Chen, I. Podlubny, Stability of fractional-order nonlinear dynamic systems : Lyapunov direct method and generalized mittag–leffler stability, Comput. Math. with Appl. **59** (2010) 1810-1821.

[34] H. Delavari, D. Baleanu, J. Sadati, Stability analysis of caputo fractional-order nonlinear systems revisited, Nonlinear Dyn., **67** (2011) 2433-2439.

[35] W. Lin, Global existence theory and chaos control of fractional differential equations, J. Math. Anal. Appl. , **332** (2007) 709-726.

[36] P. van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, Math. Biosci., **180** (2002) 2-48.

[37] H. Kheiri, M. Jafari, Optimal control of a fractional-order model for the HIV/AIDS epidemic, Int. J. Biomath., **11** (2018) 1850086-1850108.

[38] C. J. Silva, D. F. Torres, Stability of a fractional HIV/AIDS model, Math. Comput. Simul, **164** (2019) 180-190.

[39] E. Ahmed, A. S. Elgazzar, On fractional order differential equations model for nonlocal epidemics, Phys. A: Stat. Mech. Appl, **379** (2007) 607-614.

[40] I. Ameen, D. Baleanu, H. M. Ali, An efficient algorithm for solving the fractional optimal control of SIRV epidemic model with a combination of vaccination and treatment, Chaos, Solitons & Fractals, **137** (2020) 109892-109918.

[41] G. Birkhoff, G. C. Rota, Ordinary Differential Equations, 4th Edition, John Wiley and Sons, New York, 1989.

سناء لفتة خلف , هدير سمير فليح

قسم الرياضيات ، كلية العلوم ، جامعة البصرة ، البصرة ، العراق

المستخلص

يتمحور هذا البحث حول دراسة نموذج وبائي رياضي غير خطي كسري SIR (حساس ، مصاب ، متعافي) متعلق بالتأخير الزمني في متغيرات الحالة والسيطرة. اثبتنا وجود حل مقيد لنموذج وباء SIR الكسري وهو حل وحيد. تم اكتشاف مجموعة جديدة من نقاط التوازن الخالية من العدوى، ومن ثم التحقيق في استقرارها المحلي. بالإضافة إلى ذلك، باستخدام طريقة مصفوفة الجيل التالي، قمنا بحساب رقم التكاثر الأساسي R_0 .