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Numerical solution and stability analysis of a nonlinear vaccination model with historical effects

Davood Rostamy* and Ehsan Mottaghi†‡

Abstract

In this paper, we extend the classical vaccination epidemic model from a deterministic framework to a model with historical effects by formulating it as a system of fractional-order differential equations (FDEs). The basic reproduction number R_0 of the resulting fractional model is computed and it is shown that if R_0 is less than one, the disease-free equilibrium is locally asymptotically stable. Particularly, we analytically calculate a certain threshold-value for R_0 and present the existence conditions of endemic equilibrium. By using stability analysis, we prove stability and α-stability of the endemic equilibrium points. The proposed model is applied on *Pertussis* disease and the fractional nonlinear system of the model is solved by applying multi-step generalized differential transform method (MSGDTM). Our results show that historical effects play an important role on the disease spreading.

Keywords: epidemic model, fractional-order differential equations, equilibrium points, stability, differential transform method.

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*Department of Mathematics, Imam Khomeini International University, P.O. Box 34149-16818, Qazvin, Iran, Email: rostamy@khayam.ut.ac.ir.

†Department of Mathematics, Imam Khomeini International University, P.O. Box 34149-16818, Qazvin, Iran, Email: ehsanmottaghi@ipm.ir

‡Corresponding Author.

1. Introduction

In the epidemiology, mathematical models of infectious diseases are commonly used to imply more realistic aspects of diseases spreading [19]. Many of current models are an extension of the classical susceptible-infected-recovered (SIR) model of Kermack and McKendrick [20]. During recent years, attentions of researchers have been given to vaccination and prevention policies [31, 21, 36, 4, 12]. The extension of SIR model that includes vaccination is called SIRV. Different approaches are used to implement the SIRV model. For example, Arino et al. applied ordinary differential equations [6] and Allen et al. implemented the model based on stochastic differential equations [3]. In mentioned approaches, the state of system at each time does not depend on the previous history of system. Therefore, these approaches do not incorporate the historical effects and lead to systems with no memory. However, many new works have been devoted to study of spreading processes with memory [35, 33, 7]. Studying previous epidemics and the way we have dealt with them can reveal new insights to present and future epidemics. For example, human experiences from *Plague* and *Influenza* epidemics in the past helped to contain the recent *Ebola* or *Influenza* outbreaks more effectively. Hence, the better idea is developing new models with past memory and historical effects.

An appropriate candidate to employ past effects could be fractional calculus [22]. Fractional calculus, the generalized form of ordinary differentiation and integration to non-integer order [25] has unique features such as nonlocality and memory, making it highly applicable in many fields of science and engineering [34, 32, 9, 28]. There are different definitions of the fractional derivative. Among them, Riemann-Liouville and Caputo fractional derivative have been used more than others. Comparing these two fractional derivatives, one easily arrives at the fact that Caputo derivative of a constant is equal to zero, which is not the case for the Riemann-Liouville derivative [30]. The main concern of the paper thus focuses on the Caputo derivative of order $\alpha > 0$, which is rather applicable in real application [15, 2]. Fractional calculus has previously been used in epidemiological studies [11, 17, 10, 5]. Previous works rarely discussed about influences of memory on spreading diseases in a epidemic model which includes vaccination strategies.

In this work, we extend the classical SIRV epidemic model from a deterministic framework to a model with historical effects. To consider long-time historical effects, we choose a power-law function as kernel with respect to time such that distant past events have far less effect on present, compared to near past events. This kernel guarantees existence of scaling feature as it is an intrinsic nature of most phenomena [23, 29]. Applying fractional calculus produces a system of fractional-order nonlinear differential equations (FDEs). We investigate the influences of memory on diseases outbreak in the model. We also solve the nonlinear fractional system by using the multi-step generalized differential transform method (MSGDTM) which is a modified version of GDTM [27]. It is shown that the approximate solutions obtained by MSGDTM algorithm are more accurate than GDTM during a longer time [26, 14].

The rest of the paper is organized as follows. In the next section, by using fractional calculus operators, the classical SIRV epidemic model is transformed from a deterministic framework to a model with historical effects. In Section 3, we first evaluate the disease-free equilibrium point of our fractional model and show that if the basic reproduction number R_0 is less than one, this equilibrium point is locally asymptotically stable. Then, it is shown that the fractional model may has multiple endemic equilibrium points. In such case, we exactly obtain a certain threshold-value of R_0 denoted by R_C. According to parameters R_0 and R_C, we express existence conditions of endemic equilibrium points and some stability results are proved. Specially, it is proved that the stability conditions of the endemic equilibrium points depend on the value of α. Section 4 describes the multi-step

generalized differential transform method (MSGDTM) for solving the fractional nonlinear system obtained in Section 2. In Section 5, we solve the proposed fractional SIRV model for parameters values of *Pertussis* disease. Then, the numerical results are presented to demonstrate the influences of memory on the disease spreading. Finally, conclusions are given in Section 6.

2. SIRV model with historical effects

The standard SIRV model is described by a system of ordinary fractional differential equations given by

$$\begin{cases} S'(t) = (1-\eta)b - bS(t) - \beta I(t) S(t) - \phi S(t) + \theta V(t) + \vartheta R(t), \\ I'(t) = \beta I(t) S(t) + \sigma\beta I(t) V(t) - (b+\mu) I(t), \\ R'(t) = \mu I(t) - (\vartheta + b) R(t), \\ V'(t) = \eta b + \phi S(t) - (b+\theta) V(t) - \sigma\beta I(t) V(t), \end{cases} \tag{2.1}$$

with the following non-negative initial conditions:

$$S(0) = S_0, \quad I(0) = I_0, \quad R(0) = R_0, \quad V(0) = V_0, \tag{2.2}$$

in which $I(t)$, $S(t)$, $R(t)$ and $V(t)$ denote the number of infected, susceptible, recovered and vaccinated individuals in the population, respectively. Also, the population size is constant and it is assumed $I(t) + S(t) + R(t) + V(t) = 1$ at any time t. In this model, it is assumed that death and birth occur with the same constant rate $b > 0$. Newborns are vaccinated with the rate $\eta \in [0, 1]$ at birth. Parameter β is the transmission rate between infected and susceptible individuals. Also, the factor $1 - \sigma$ is the effect of vaccine, which means that if $\sigma = 0$, the vaccine is complete and if $0 < \sigma < 1$, the vaccine is incomplete. Moreover, ϕ is the vaccination rate. The vaccine does not provide lifelong protection and its protection is reduced with the rate θ. Infected individuals are recovered with the rate $\mu > 0$ and have temporary immunity. They leave this immunity with the rate ϑ.

The ordinary differential equations describe a epidemic process with no memory and historical effects. In order to consider the historical effects, we first rewrite system (2.1) in terms of time dependent integrations as follows:

$$\begin{cases} S'(t) = \int_0^t \kappa(t-\xi)\big[(1-\eta)b - bS(\xi) - \beta I(\xi) S(\xi) - \phi S(\xi) + \theta V(\xi) + \vartheta R(\xi)\big] d\xi, \\ I'(t) = \int_0^t \kappa(t-\xi)\big[\beta I(\xi) S(\xi) + \sigma\beta I(\xi) V(\xi) - (b+\mu) I(\xi)\big] d\xi, \\ R'(t) = \int_0^t \kappa(t-\xi)\big[\mu I(\xi) - (\vartheta + b) R(\xi)\big] d\xi, \\ V'(t) = \int_0^t \kappa(t-\xi)\big[\eta b + \phi S(\xi) - (b+\theta) V(\xi) - \sigma\beta I(\xi) V(\xi)\big] d\xi, \end{cases} \tag{2.3}$$

in which $\kappa(t-\xi)$ is assumed as kernel with respect to time and is equal to Dirac delta function $\delta(t-\xi)$. Dirac delta function can be replaced with any arbitrary function which leads to a type of time correlations. To consider long-time historical effects, we choose a power-law function as kernel such that distant past events have far less effect on present, compared to near past events. We define the power-law kernel by

$$\kappa(t-\xi) = \frac{1}{\Gamma(\alpha-1)} (t-\xi)^{\alpha-2}, \tag{2.4}$$

which $\alpha \in (0, 1]$ and $\Gamma(.)$ is the Gamma function. According to this definition, the strength of historical effects is increased by reducing the value of α. By substituting this

new power-law kernel in the system (2.2), we obtain

$$
\begin{cases}
S'(t) = \frac{1}{\Gamma(\alpha-1)} \int_0^t (t-\xi)^{\alpha-2} \big[(1-\eta)b - bS(\xi) - \beta I(\xi) S(\xi) - \phi S(\xi) \\
\qquad\qquad + \theta V(\xi) + \vartheta R(\xi)\big] d\xi, \\
I'(t) = \frac{1}{\Gamma(\alpha-1)} \int_0^t (t-\xi)^{\alpha-2} \big[\beta I(\xi) S(\xi) + \sigma\beta I(\xi) V(\xi) - (b+\mu) I(\xi)\big] d\xi, \\
R'(t) = \frac{1}{\Gamma(\alpha-1)} \int_0^t (t-\xi)^{\alpha-2} \big[\mu I(\xi) - (\vartheta+b) R(\xi)\big] d\xi, \\
V'(t) = \frac{1}{\Gamma(\alpha-1)} \int_0^t (t-\xi)^{\alpha-2} \big[\eta b + \phi S(\xi) - (b+\theta) V(\xi) - \sigma\beta I(t) V(t)\big] d\xi.
\end{cases} \tag{2.5}
$$

Using fractional calculus [30, 25], for a function $g : [a,b] \to \mathbb{R}$, the left fractional integral of order $\alpha > 0$ is defined by

$$
{}_a I_t^{\alpha} g(t) = \frac{1}{\Gamma(\alpha)} \int_a^t (t-x)^{\alpha-1} g(x) dx. \tag{2.6}
$$

So, it is obvious that the right hand side of the above system is a fractional integral of order $(\alpha-1)$ on the interval $[0,t]$. For a continuous function $g(t)$ on the $[a,b]$ interval, left Caputo derivative of order $\alpha > 0$ is defined as follows [8, 16]:

$$
{}_{t_0}^{c} D_t^{\alpha} g(t) = {}_a I_t^{n-\alpha} D^n g(t), \tag{2.7}
$$

where n is an integer number satisfying $\alpha \in (n-1, n)$ and $D = \frac{d}{dt}$. By applying the fractional Caputo derivative of order $(\alpha-1)$ on both side of the preceding system, we obtain the following system

$$
\begin{cases}
{}_0^c D_t^{\alpha} S(t) = (1-\eta)b - bS(t) - \beta I(t) S(t) - \phi S(t) + \theta V(t) + \vartheta R(t), \\
{}_0^c D_t^{\alpha} I(t) = \beta I(t) S(t) + \sigma\beta I(t) V(t) - (b+\mu) I(t), \\
{}_0^c D_t^{\alpha} R(t) = \mu I(t) - (\vartheta+b) R(t), \\
{}_0^c D_t^{\alpha} V(t) = \eta b + \phi S(t) - (b+\theta) V(t) - \sigma\beta I(t) V(t).
\end{cases} \tag{2.8}
$$

In this representation, the governing equations of the system (2.8) have fractional derivatives of order $\alpha \in (0,1]$ in the sense of Caputo which guarantee the presence of memory. The strength of memory effects can be controlled by α. As α approaches to 1, the influences of memory are decreased and the system (2.8) tends to a system with no memory, *i.e.* standard SIRV model.

Similar to [11], it is easy to show that the initial value problem (2.8)-(2.2) is well posed, namely solutions remain non-negative for non-negative initial conditions. As the population is constant, we have $V(t) = 1 - S(t) - I(t) - R(t)$ at any time t. Therefore, we can consider the following system instead of (2.8)

$$
\begin{cases}
{}_0^c D_t^{\alpha} S(t) = f_1(S(t), I(t), R(t)) \\
\qquad\qquad = b + \theta - \eta b - (b + \phi + \theta) S(t) - \beta I(t) S(t) - \theta I(t) + (\vartheta - \theta) R(t), \\
{}_0^c D_t^{\alpha} I(t) = f_2(S(t), I(t), R(t)) \\
\qquad\qquad = (\sigma\beta - b - \mu) I(t) + (\beta - \sigma\beta) I(t) S(t) - \sigma\beta I(t) R(t) - \sigma\beta I^2(t), \\
{}_0^c D_t^{\alpha} R(t) = f_3(S(t), I(t), R(t)) \\
\qquad\qquad = \mu I(t) - (\vartheta + b) R(t).
\end{cases} \tag{2.9}
$$

In the next section, we investigate the influences of memory on diseases outbreak in the obtained fractional SIRV model.

3. Asymptotic stability of equilibrium points

It is obvious that if $E = (S_e, I_e, R_e)$ is an equilibrium point of the system (2.9) then $E = (S_e, I_e, R_e, V_e)$ is an equilibrium point of the system (2.8) where $V_e = 1 - S_e - I_e - R_e$.

Therefore, we investigate existence and stability conditions of equilibrium points of the system (2.9) in this section. The equilibrium points of the system (2.9) satisfy in the following system

$$\begin{cases} {}_0^cD_t^\alpha S(t) = 0, \\ {}_0^cD_t^\alpha I(t) = 0, \\ {}_0^cD_t^\alpha R(t) = 0. \end{cases} \tag{3.1}$$

So, the system (2.9) always has the following unique disease-free equilibrium

$$\widetilde{E} = (\widetilde{S}, \widetilde{I}, \widetilde{R}) = \left(\frac{b+\theta-\eta b}{b+\theta+\phi}, 0, 0 \right). \tag{3.2}$$

According to the system (3.1), the endemic equilibrium points are obtained by solving the quadratic equation $P(I) = AI^2 + BI + C = 0$, where

$$\begin{aligned} A &= -\frac{\sigma\beta^2(b+\vartheta+\mu)}{(b+\vartheta)}, \\ B &= \sigma\beta^2 - \sigma\beta b - \sigma\beta\phi - \sigma\beta\mu - \beta b - \beta\theta - \frac{\beta\mu}{b+\vartheta}(b+\theta+\sigma\phi), \\ C &= \beta(b+\sigma\phi+\theta+\sigma\eta b-\eta b) - (b+\mu)(b+\phi+\theta). \end{aligned} \tag{3.3}$$

It can be shown that if I^* be a positive real root of the above quadratic equation then $E^* = (S^*, I^*, R^*)$ is the endemic equilibrium point of the system (2.9) where

$$\begin{aligned} S^* &= \frac{(b+\vartheta)(b+\mu-\sigma\beta) + \sigma\beta I^*(b+\vartheta+\mu)}{(b+\vartheta)\beta(1-\sigma)}, \\ R^* &= \frac{\mu I^*}{b+\vartheta}. \end{aligned} \tag{3.4}$$

According to the system (3.1), if $E^* = (S^*, I^*, R^*)$ be an endemic equilibrium point then

$$(\sigma\beta - b - \mu) + (\beta - \sigma\beta)S^*(t) - \sigma\beta R^*(t) - \sigma\beta I^*(t) = 0.$$

Therefore, we have

$$\sigma\beta(1 - S^*(t) - I^*(t) - R^*(t)) + \beta S^*(t) = b + \mu.$$

On the other hand, the population is constant and $V^*(t) = 1 - S^*(t) - I^*(t) - R^*(t)$. Hence, we obtain $\sigma V^*(t) + S^*(t) = \frac{b+\mu}{\beta} < 1$. Thus if $\beta \leq b + \mu$, then the system (2.9) has no endemic equilibrium point.

3.1. Theorem. *Let*

$$R_0 = \frac{\beta}{b+\mu}\left(\frac{b+\theta+\sigma\phi-\eta b(1-\sigma)}{b+\theta+\phi} \right), \tag{3.5}$$

be the basic reproduction number. Then, the disease-free equilibrium $\widetilde{E}$ of the system (2.9) is locally asymptotically stable if $R_0 < 1$.

Proof. We perturb the disease-free equilibrium $\widetilde{E} = (\widetilde{S}, \widetilde{I}, \widetilde{R})$ by adding positive terms $\varepsilon_1(t)$, $\varepsilon_2(t)$ and $\varepsilon_3(t)$, respectively, that is

$$S(t) = \widetilde{S} + \varepsilon_1(t), \quad I(t) = \widetilde{I} + \varepsilon_2(t) \quad \textit{and} \quad R(t) = \widetilde{R} + \varepsilon_3(t).$$

According to the systems (2.9) and (3.1), we obtain

$$\begin{cases} {}_0^cD_t^\alpha \varepsilon_1(t) = f_1\left(\widetilde{S}+\varepsilon_1(t), \widetilde{I}+\varepsilon_2(t), \widetilde{R}+\varepsilon_3(t)\right), \\ {}_0^cD_t^\alpha \varepsilon_2(t) = f_2\left(\widetilde{S}+\varepsilon_1(t), \widetilde{I}+\varepsilon_2(t), \widetilde{R}+\varepsilon_3(t)\right), \\ {}_0^cD_t^\alpha \varepsilon_3(t) = f_3\left(\widetilde{S}+\varepsilon_1(t), \widetilde{I}+\varepsilon_2(t), \widetilde{R}+\varepsilon_3(t)\right). \end{cases}$$

For $i = 1, 2, 3$, we have

$$f_i\left(\widetilde{S} + \varepsilon_1(t), \widetilde{I} + \varepsilon_2(t), \widetilde{R} + \varepsilon_3(t)\right) \simeq f_i(\widetilde{S}, \widetilde{I}, \widetilde{R}) + \frac{\partial f_i}{\partial S}(\widetilde{S}, \widetilde{I}, \widetilde{R})\ \ \varepsilon_1(t) + \frac{\partial f_i}{\partial I}(\widetilde{S}, \widetilde{I}, \widetilde{R})\ \ \varepsilon_2(t) + \frac{\partial f_i}{\partial R}(\widetilde{S}, \widetilde{I}, \widetilde{R})\ \ \varepsilon_3(t).$$

By replacing $f_i(\widetilde{S}, \widetilde{I}, \widetilde{R}) = 0$, for $i = 1, 2, 3$, we attain the following linearized system

$$_0^c D_t^\alpha \varepsilon = J\varepsilon,$$

with initial values

$$\varepsilon_1(0) = S_0 - \widetilde{S}, \quad \varepsilon_2(0) = I_0 - \widetilde{I} \quad and \quad \varepsilon_3(0) = R_0 - \widetilde{R},$$

where $\varepsilon = (\varepsilon_1(t), \varepsilon_2(t), \varepsilon_3(t))^T$ and J is the Jacobian matrix evaluated at the point $\widetilde{E}$. We have $B^{-1}\, J\, B = C$ where C is a diagonal matrix of J given by

$$C = \begin{bmatrix} \lambda_1 & 0 & 0 \\ 0 & \lambda_2 & 0 \\ 0 & 0 & \lambda_3 \end{bmatrix},$$

where λ_1, λ_2 and λ_3 are the eigenvalues of J and B is the eigenvectors of J. By considering $J = BCB^{-1}$, we obtain the linear fractional-order system

$$_0^c D_t^\alpha \zeta = C\zeta,$$

where $\zeta = (\zeta_1(t), \zeta_2(t), \zeta_3(t))^T = B^{-1}\varepsilon$. The solutions of the above system are given by Mittag-Leffler functions

$$\zeta_i(t) = \sum_{n=0}^{\infty} \frac{t^{n\alpha} {\lambda_i}^n}{\Gamma(n\alpha + 1)} \zeta_i(0) = E_\alpha(\lambda_i t^\alpha)\zeta_i(0), \qquad i = 1, 2, 3.$$

According to results of Matignon [24], if $|\arg\ \lambda_i| > \alpha\frac{\pi}{2}$ then $\zeta_i(t)$ is decreasing and therefore $\varepsilon_i(t)$ is also decreasing, for each $i = 1, 2, 3$. Thus, the disease-free equilibrium point $\widetilde{E}$ is asymptotically stable if all the eigenvalues λ_i of the Jacobian matrix J evaluated at $\widetilde{E}$ satisfy the following condition:

$$|\arg\ \lambda_i| > \alpha\frac{\pi}{2}. \tag{3.6}$$

The Jacobian matrix of the system (2.9) evaluated at $\widetilde{E}$ is as follows:

$$J(\widetilde{E}) = \begin{bmatrix} -b - \phi - \theta & \frac{-\beta(1-\eta)b - \beta\theta}{b+\phi+\theta} - \theta & -\theta + \vartheta \\ 0 & \beta\,(1-\sigma)\,\frac{b(1-\eta)+\theta}{b+\phi+\theta} + \sigma\beta - b - \mu & 0 \\ 0 & \mu & -b - \vartheta \end{bmatrix}.$$

It is enough to show that all eigenvalues of $J(\widetilde{E})$, have negative real parts. The eigenvalues of $J(\widetilde{E})$ are

$$-(b + \vartheta)\,, -(b + \theta + \phi)\,, \frac{\beta\,(b + \theta + \sigma\phi + \eta b\sigma - \eta b) - (b + \mu)\,(b + \theta + \phi)}{b + \theta + \phi}.$$

It can be shown that if $R_0 < 1$, then

$$\frac{(b + \theta + \sigma\phi + \eta b\sigma - \eta b)}{b + \theta + \phi} < \frac{(b + \mu)}{\beta},$$

and all the eigenvalues of $J(\widetilde{E})$ have negative real parts. □

We can show that $C = (b+\mu)(b+\theta+\phi)(R_0-1)$. Therefore, the following equation can be considered instead of the equation $P(I)=0$,

$$Q(I) := \frac{-AI^2}{(b+\mu)(b+\theta+\phi)} + \frac{-BI}{(b+\mu)(b+\theta+\phi)} + 1 = R_0. \tag{3.7}$$

When $\beta > b+\mu$, if the quadratic equation $Q(I)=R_0$ has no, one or two positive real root, then the system (2.9) has no, one or two endemic equilibrium points. In the next theorem, we exactly obtain a minimum of R_0 and prove that it can be considered as a certain threshold-value of R_0.

3.2. Theorem. *Let*

$$R_C = 1 - \frac{\left((b+\vartheta)(\sigma(b+\phi+\mu)-\sigma\beta+b+\theta)+\mu(b+\theta+\sigma\phi)\right)^2}{4\sigma(b+\vartheta)(b+\mu)(b+\vartheta+\mu)(b+\theta+\phi)}$$

be the minimum value of the curve $Q(I)$ and $\beta > b+\mu$, then

1. *If $R_0 > 1$ or $R_0 = 1$, $B > 0$, then the system (2.9) has the unique endemic equilibrium point E_u^*.*
2. *If $R_C = R_0 < 1$ and $B > 0$, then the system (2.9) has the unique endemic equilibrium point E_c^*.*
3. *If $R_C < R_0 < 1$ and $B > 0$, then the system (2.9) has two endemic equilibrium points E_1^*, E_2^*.*
4. *If $R_0 < R_C$, then there is no endemic equilibrium point.*

Proof. Since $A < 0$, the curve $Q(I)$ has a minimum value. By direct calculation, it is obtained that this minimum value occurs at point (I_C, R_C) where

$$I_C = \frac{(b+\vartheta)(\sigma\beta-b-\theta-\sigma(b+\phi+\mu))-\mu(b+\theta+\sigma\phi)}{2\sigma\beta(b+\vartheta+\mu)},$$

$$R_C = 1 - \frac{\left((b+\vartheta)(\sigma(b+\phi+\mu)-\sigma\beta+b+\theta)+\mu(b+\theta+\sigma\phi)\right)^2}{4\sigma(b+\vartheta)(b+\mu)(b+\vartheta+\mu)(b+\theta+\phi)}.$$

As mentioned above, the y-intercept of curve $y = Q(I)$ is 1. Therefore, there are the following cases:

1. If $R_0 > 1$, then the equation (3.7) has two real roots and one of them is non-negative and greater than I_C. If $R_0 = 1$, then the equation (3.7) has a non-zero real root such that it is non-negative and greater than I_C when $B > 0$. So, the system (2.9) has the unique endemic equilibrium point E_u^* such that $I_u^* > I_C$.
2. If $R_C = R_0 < 1$, then the equation (3.7) has a repeated real root which is non-negative when $B > 0$. Thus, the system (2.9) has the unique endemic equilibrium point E_c^* such that $I_c^* = I_C$.
3. If $R_C < R_0 < 1$, then the equation (3.7) has two real roots I_1^* and I_2^*. If $B > 0$, then these roots are non-negative. Thus, the system (2.9) has two endemic equilibrium points E_1^* and E_2^* such that $I_1^* < I_C < I_2^*$.

□

In the following theorem, it is shown that the stability of endemic equilibrium points introduced in Theorem 3.2 is related to value of α.

3.3. Theorem. *Suppose E_u^*, E_c^*, E_1^* and E_2^* be endemic equilibrium points introduced in Theorem 3.2, then:*

1. *Endemic equilibrium points E_c^* and E_1^* are unstable.*
2. *If $\alpha \le \frac{2}{3}$, endemic equilibrium points E_u^* and E_2^* are locally asymptotically α-stable.*
3. *If $\alpha > \frac{2}{3}$ and $\vartheta \ge \theta$, E_u^* and E_2^* are locally asymptotically stable.*

Proof. The Jacobian matrix of the system (2.9) evaluated at the endemic equilibrium point E^* is given by

$$J(E^*) = \begin{bmatrix} -b-\phi-\theta-\beta I^* & -\beta S^*-\theta & -\theta+\vartheta \\ \beta I^*(1-\sigma) & -\sigma\beta I^* & -\sigma\beta I^* \\ 0 & \mu & -b-\vartheta \end{bmatrix}.$$

According to the proof of Theorem 3.1, if all eigenvalues of the Jacobian matrix $J(E^*)$ satisfy the condition (3.6), then the endemic equilibrium point E^* is asymptotically stable. The characteristic equation of this matrix is $P(\lambda) = \lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0$ where

$$\begin{aligned} a_2 &= 2b+\vartheta+\theta+\phi+\beta I^*(1+\sigma), \\ a_1 &= (b+\vartheta)(b+\phi+\theta)+\beta I^*(2b\sigma+\sigma\mu+\sigma\phi+\sigma\vartheta+b+\vartheta+\theta) \\ &\quad +\beta^2 I^*(\sigma I^*+(1-\sigma)S^*), \\ a_0 &= \beta I^*[\mu(\theta-\vartheta)+\sigma\mu(b+\vartheta+\phi)+(b+\vartheta)(\theta+\sigma b+\sigma\phi)] \\ &\quad +\beta^2 I^*[(1-\sigma)(b+\vartheta)S^*+\sigma I^*(\mu+b+\vartheta)] \\ &= 2\sigma\beta^2(b+\vartheta+\mu)I^*(I^*-I_C). \end{aligned} \tag{3.8}$$

For every endemic equilibrium point E^*, it is obvious that $a_1, a_2 > 0$. According to the proof of Theorem 3.2, for the endemic equilibrium point E_c^*, we have $I_c^* = I_C$ and $a_0 = 0$. So, the endemic equilibrium point E_c^* is unstable. Similarly, for the endemic equilibrium point E_1^*, we have $I_1^* < I_C$, then $a_0 < 0$. From Descartes' rule of signs, it is clear that the equation $P(\lambda) = 0$ has at least one positive real root. Therefore, the endemic equilibrium point E_1^* is unstable. For the endemic equilibrium points E_u^* and E_2^*, we have $I_u^* > I_C$ and $I_2^* > I_C$, respectively. So we obtain $a_0 > 0$. From Descartes' rule of signs, the equation $P(\lambda) = 0$ has three negative real roots or one negative real root and two complex roots. If the equation $P(\lambda) = 0$ has three negative real roots, then E_u^* and E_2^* are stable. We now assume the equation $P(\lambda) = 0$ has one negative real root $\lambda_1 = -z$ and two complex roots $\lambda_{2,3} = x \pm iy$, then

$$P(\lambda) = \lambda^3 + \lambda^2(-2x+z) + \lambda(x^2+y^2-2xz) + z(x^2+y^2).$$

Therefore, we have

$$a_2 = -2x+z,\ a_1 = x^2+y^2-2xz,\ a_0 = z(x^2+y^2).$$

If $a_2a_1 - a_0 > 0$, then $-2x\left[(x-z)^2+y^2\right] > 0$. So, if $a_2a_1 - a_0 > 0$, then λ_2 and λ_3 must have negative real parts. It can be shown that if $\vartheta \geq \theta$, then $a_2a_1 - a_0 > 0$ and the roots of equation $P(\lambda) = 0$ have negative real parts. Furthermore, we have $a_2, a_1 \geq 0$, so $x^2+y^2 \geq 2xz$ and $z \geq 2x$. Thus we obtain

$$x^2\left(1+\frac{x^2}{y^2}\right) \geq 2xz \geq 4x^2.$$

The above equation show that $\sec^2(Arg\lambda_{2,3}) \geq 4$ and $\frac{\pi}{3} \leq Arg(\lambda_{2,3}) \leq \frac{2\pi}{3}$. So, if $\alpha \leq \frac{2}{3}$, then $|Arg\lambda_{2,3}| > \alpha\frac{\pi}{2}$ and E_u^*, E_2^* are stable. □

Theorem 3.3 shows that the stability of the endemic equilibrium points can be controlled by modifying the order of the fractional derivatives. In the fact, the fractional SIRV model can be achieved the steady state meanwhile standard SIRV model is unstable. Hence, considering historical effects increase the stability region of the SIRV model.

4. MSGDTM algorithm to solve the nonlinear fractional system (2.9) with initial values (2.2)

In recent years, Odibat et al. introduced a new method, called the generalized differential transform method (GDTM), for solving linear and nonlinear fractional-order differential equations [27]. Although the GDTM is used to provide approximate solutions for a wide class of nonlinear problems in terms of convergent series, it has some drawbacks. The series solution always converges in a very small region and it has been shown that the obtained approximate solution is valid only for a short time in some problems [13, 26]. Here, we solve the nonlinear fractional system (2.9) with initial values (2.2) by applying the multi-step generalized differential transform method (MSGDTM) which is a modified version of GDTM. It is shown that the approximate solutions obtained by MSGDTM algorithm are more accurate than GDTM during a longer time [26, 14]. Also, it is shown that MSDTM has a significant performance compared with the Runge-Kutta method when the order of the derivative is one [1].

Assume that the interval $[0,T]$ is divided into M subintervals $[t_{m-1},t_m]$ by using the nodes $t_m = mh$ where $h = T/M$ and $m = 1,2,...,M$. The main ideas of the MSGDTM are as follows:

First, we apply the GDTM to the initial value problem (2.9)-(2.2) over the interval $[0,t_1]$. The Kth-order approximate solutions can be expressed by the following finite series

$$\begin{cases} S_1(t) = \sum\limits_{n=0}^{K} \overline{S}_1(n) t^{\alpha n}, \\ I_1(t) = \sum\limits_{n=0}^{K} \overline{I}_1(n) t^{\alpha n}, \\ R_1(t) = \sum\limits_{n=0}^{K} \overline{R}_1(n) t^{\alpha n}, \end{cases} \tag{4.1}$$

in which $\overline{S}_1(n)$, $\overline{I}_1(n)$ and $\overline{R}_1(n)$ are the differential transforms for $S(t)$, $I(t)$ and $R(t)$ over the interval $[0,t_1]$, respectively. These transforms satisfy the following recurrence relations

$$\begin{cases} \overline{S}_1(n+1) = \dfrac{\Gamma(\alpha n+1)}{\Gamma(\alpha(n+1)+1)} \Big[(b+\theta-\eta b)\delta(n) - (b+\phi+\theta)\overline{S}(n) \\ \qquad\qquad - \beta \sum\limits_{l=0}^{n} \overline{I}(l)\overline{S}(n-l) - \theta \overline{I}(n) + (\vartheta-\theta)\overline{R}(n) \Big], \\ \overline{I}_1(n+1) = \dfrac{\Gamma(\alpha n+1)}{\Gamma(\alpha(n+1)+1)} \Big[(\sigma\beta - b - \mu)\overline{I}(n) + (\beta-\sigma\beta) \sum\limits_{l=0}^{n} \overline{S}(l)\overline{I}(n-l) \\ \qquad\qquad - \sigma\beta \sum\limits_{l=0}^{n} \overline{R}(l)\overline{I}(n-l) - \sigma\beta \sum\limits_{l=0}^{n} \overline{I}(l)\overline{I}(n-l) \Big], \\ \overline{R}_1(n+1) = \frac{\Gamma(\alpha n+1)}{\Gamma(\alpha(n+1)+1)} \Big[\mu \overline{I}(n) - (\vartheta+b)\overline{R}(n) \Big], \end{cases} \tag{4.2}$$

where $\delta(n) = 1$ when $n = 0$ and equals 0 otherwise. The differential transforms of the initial values are given by $\overline{S}_1(0) = S_0$, $\overline{I}_1(0) = I_0$ and $\overline{R}_1(0) = R_0$.

For $m \geq 2$, we now apply the GDTM to the initial value problem (2.9)-(2.2) over the interval $[t_{m-1},t_m]$. We repeat the process and generate a sequence of approximate solutions $S_m(t)$, $I_m(t)$ and $R_m(t)$ for $m = 2,3,...,M$. Finally the MSGDTM yields the

following solution:

$$S(t) = \begin{cases} S_1(t) = \sum_{n=0}^{K} \overline{S}_1(n) t^{\alpha n}, & t \in [0, t_1] \\ S_2(t) = \sum_{n=0}^{K} \overline{S}_2(n)(t-t_1)^{\alpha n}, & t \in [t_1, t_2] \\ \vdots \\ S_M(t) = \sum_{n=0}^{K} \overline{S}_M(n)(t-t_{M-1})^{\alpha n}, & t \in [t_{M-1}, t_M], \end{cases} \tag{4.3}$$

$$I(t) = \begin{cases} I_1(t) = \sum_{n=0}^{K} \overline{I}_1(n) t^{\alpha n}, & t \in [0, t_1] \\ I_2(t) = \sum_{n=0}^{K} \overline{I}_2(n)(t-t_1)^{\alpha n}, & t \in [t_1, t_2] \\ \vdots \\ I_M(t) = \sum_{n=0}^{K} \overline{I}_M(n)(t-t_{M-1})^{\alpha n}, & t \in [t_{M-1}, t_M], \end{cases} \tag{4.4}$$

$$R(t) = \begin{cases} R_1(t) = \sum_{n=0}^{K} \overline{R}_1(n) t^{\alpha n}, & t \in [0, t_1] \\ R_2(t) = \sum_{n=0}^{K} \overline{R}_2(n)(t-t_1)^{\alpha n}, & t \in [t_1, t_2] \\ \vdots \\ R_M(t) = \sum_{n=0}^{K} \overline{R}_M(n)(t-t_{M-1})^{\alpha n}, & t \in [t_{M-1}, t_M], \end{cases} \tag{4.5}$$

where $\overline{S}_m(n)$, $\overline{I}_m(n)$, and $\overline{R}_m(n)$ for $m = 1, 2, ..., M$ satisfy the recurrence relations in the system (4.2) with initial conditions $\overline{S}_m(0) = S_m(t_{m-1}) = S_{m-1}(t_{m-1})$, $\overline{I}_m(0) = I_m(t_{m-1}) = I_{m-1}(t_{m-1})$, and $\overline{R}_m(0) = R_m(t_{m-1}) = R_{m-1}(t_{m-1})$.

5. Numerical results

Parameters List for pertussis disease

Parameter	Value/days	Meaning
b	$1/(75 \times 365)$	Average lifetime 75 years
η	0.9	Ratio of vaccinated newborns
θ	$1/(5 \times 365)$	Average vaccine waning time
β	0.4	Transmission rate
μ	1/21	Average infectious period 21 days
ϕ	0.05	Vaccination rate
ϑ	1/31	Average immunity period 31 days
σ	0 to 0.2	Effective rate of vaccine is between 0.8 to 1

Table 1. Parameter values obtained from an study on *pertussis disease*.

To illustrate the numerical results, we use the parameter values obtained from an study on *pertussis disease* [18]. These value are shown in Table 1. In Table 2, equilibrium points of the system (2.9) are computed using values of Table 1 for $\sigma = 0.15$, $\sigma = 0.1$ and $\sigma = 0.05$. It can be seen that if $\sigma = 0.15$, the value of R_0 is greater than 1 and the model has the unique endemic equilibrium point E_u^*. On the other hand if $\sigma = 0.1$, the value of R_0 is between 1 and R_C and the model have two endemic equilibrium points E_1^* and

E_2^*. Moreover, if $\sigma = 0.05$, the value of R_0 is less than 1 and the model has the disease-free equilibrium point $\widetilde{E}$. These results are consistent with Theorem 3.2 meaning that the values of R_0 and R_C play an important role on the stability analysis of equilibrium points. Table 2 shows that by increasing the effect of vaccine (by decreasing parameter σ) the *pertussis disease* can be contained.

Numerical results for existence of equilibrium points

σ	R_0	R_C	B	Equilibrium points
0.15	1.336832	0.698558	0.013138	$E_u^* = (0.0822845, 0.2715756, 0.4004438, 0.2456951)$
0.1	0.921731	0.807790	0.008566	$E_1^* = (0.0281086, 0.0248889, 0.0366992, 0.9103032)$ $E_2^* = (0.0739100, 0.1914716, 0.2823288, 0.4522895)$
0.05	0.506629	0.916437	0.003993	$\widetilde{E} = (0.0109045, 0, 0, 0.9890955)$

Table 2. Show equilibrium points of the system 2.9 using values of Table 1 for $\sigma = 0.15$, $\sigma = 0.1$ and $\sigma = 0.05$.

According to Theorems 3.1 and 3.3, we expect the equilibrium points E_u^*, E_1^* and $\widetilde{E}$ to be locally asymptotically stable and the equilibrium point E_2^* to be unstable. Here, we apply MSGDTM algorithm to solve the system (2.9) for $\sigma = 0.15$, $\sigma = 0.1$ and $\sigma = 0.05$ using values of Table 1 and the following initial values:

$$S(0) = 0.8, I(0) = 0.2, R(0) = 0, V(0) = 0. \tag{5.1}$$

The numerical results are shown in Figures 1, 2 and 3. These figures show that the system (2.9) achieves in the steady state for all four cases of α. These results confirm the results of Theorems 3.1 and 3.3. As we can see, the numerical solutions depend continuously on the fractional-order derivative α and the model reaches the equilibrium point at a faster rate by reducing α. In other words, the model approaches the steady state at a faster rate when the effect of memory factor is increased.

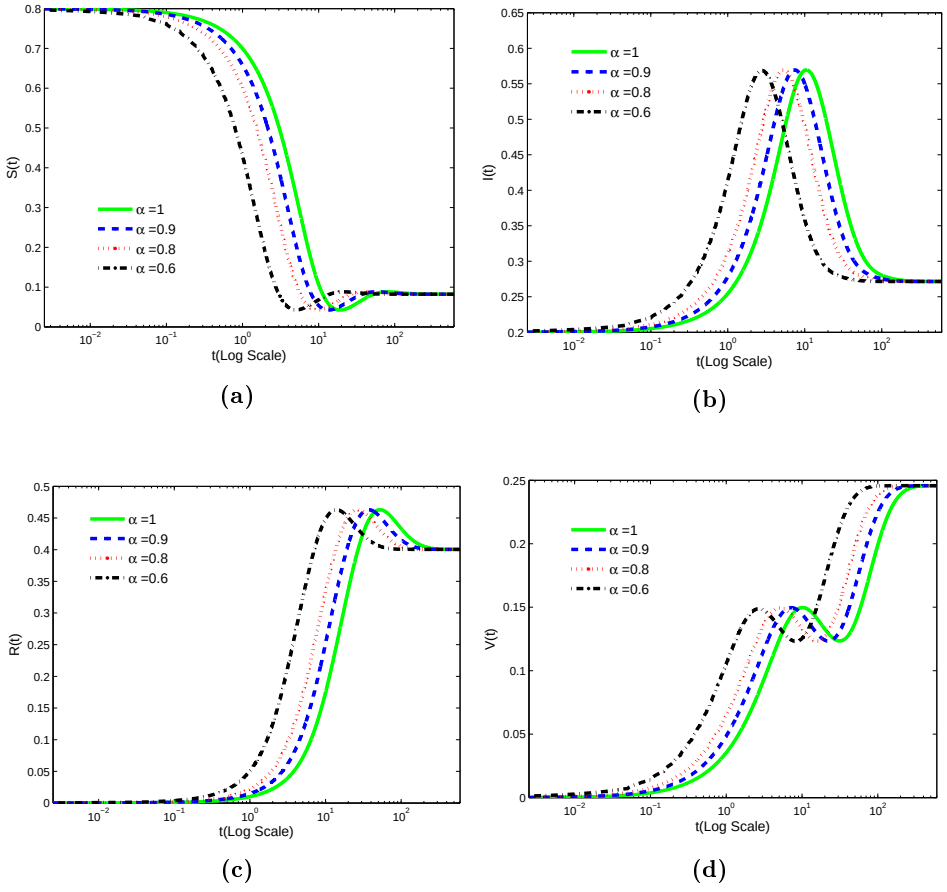

Figure 1. Numerical simulation of the system (2.9) with initial conditions (5.1) for the parameters introduced in table 1, $\sigma = 0.15$ and $\alpha = 0.6, 0.8, 0.9, 1$.

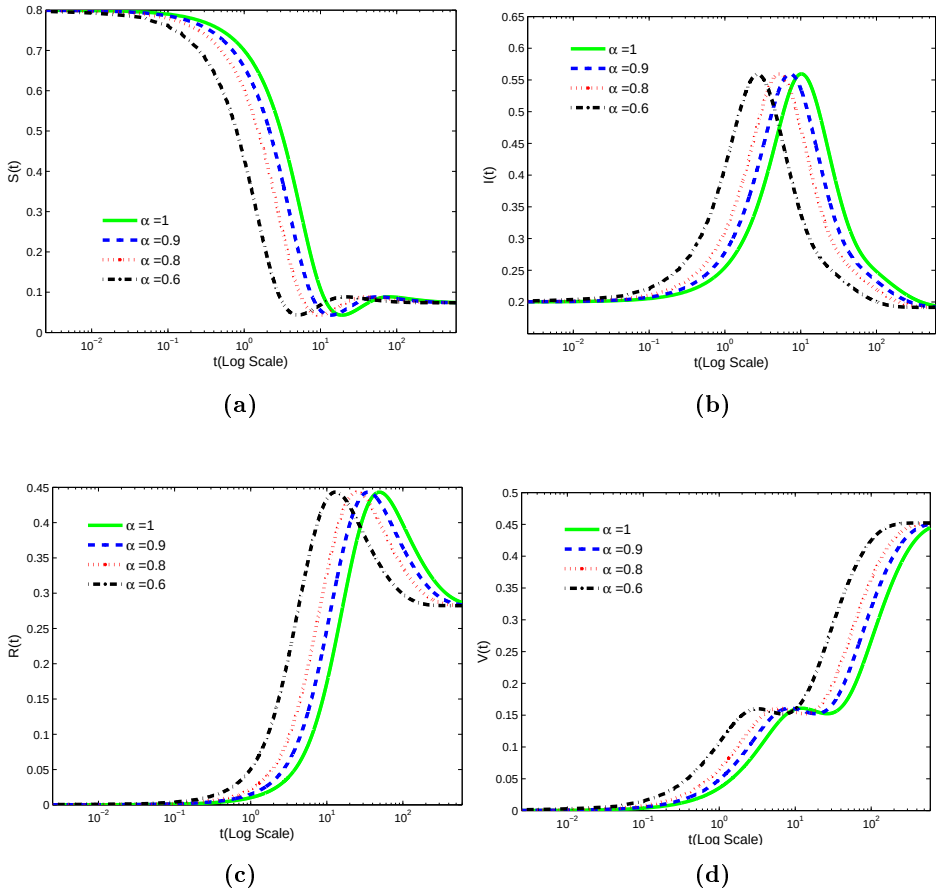

Figure 2. Numerical simulation of the system (2.9) with initial conditions (5.1) for the parameters introduced in table 1, $\sigma = 0.1$ and $\alpha = 0.6, 0.8, 0.9, 1$.

In order to demonstrate the influences of memory on disease spreading, we compare the approximate solutions evaluated in Figure 2 with the equilibrium point E_2^* of Table 2. Figure 4 reveals the 2-norm error of the approximate solutions evaluated in Figure 2. As we can see, the error decreases when the time is increased for different values of α. Therefor, the approximate solutions evaluated in Figure 2 converge to the equilibrium point E_2^* in all cases. Moreover, the convergence of solutions to E_2^* can be reached faster by reducing α, increasing the strength of historical effects. It should be noted that the step size 0.05 was used in evaluating the approximate solutions in Figures 1-3. Obviously, the efficiency of this approach can be dramatically enhanced by decreasing the step size and computing further terms or components of $S(t), I(t), R(t)$ and $V(t)$.

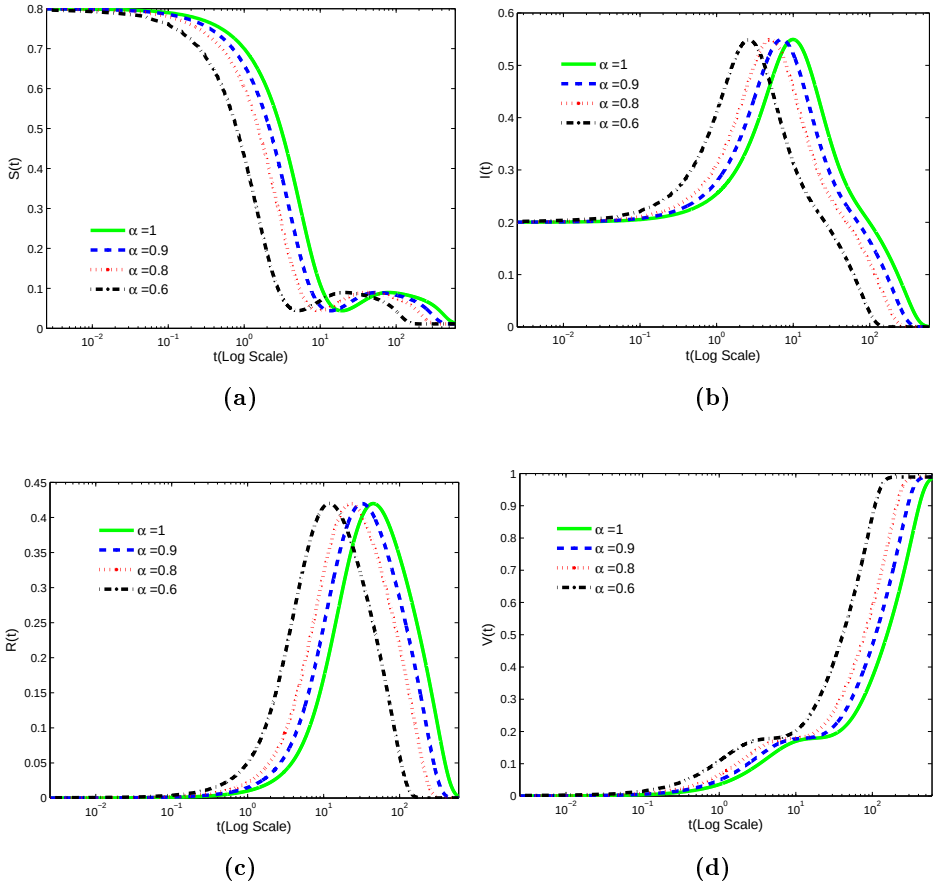

Figure 3. Numerical simulation of the system (2.9) with initial conditions (5.1) for the parameters introduced in table 1, $\sigma = 0.05$ and $\alpha = 0.6, 0.8, 0.9, 1$.

6. Conclusion

In this paper, we generalized the classical SIRV model to a model with historical effects using fractional calculus. We determined the basic reproduction R_0 and proved that if $R_0 < 1$, the disease-free equilibrium is locally asymptotically stable. In standard SIRV model, it is numerically shown that R_0 must be further reduced to be less than a threshold-value in order to ensure that the disease exterminate [6]. But this value has not been obtained exactly. Here, we analytically calculated the threshold-value of R_0, denoted by R_C. Using the values of R_0 and R_C, we expressed the existence conditions of the endemic equilibrium points in Theorem 3.2. Using stability analysis, we proved Theorem 3.3 for the stability and α-stability of the endemic equilibrium points. Theorem 3.3 shows that the stability of the endemic equilibrium points can be controlled by modifying the value of α. In the fact, the fractional SIRV model can be achieved in the steady state by controlling the parameters that affect α. For the parameters values of Table 1 which

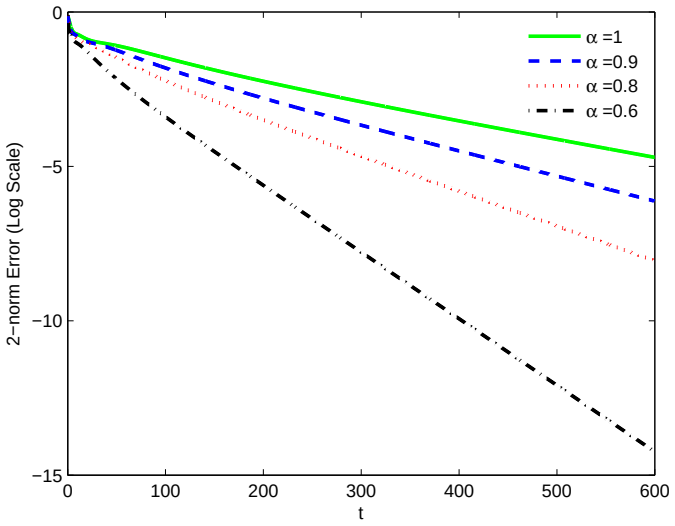

Figure 4. Plot the 2-norm error of the approximate solutions evaluated in Figure 2 for $\alpha = 0.6, 0.8, 0.9, 1$.

are related to *pertussis disease*, we numerically studied results of Theorem 3.2 in Table2. We applied MSGDTM algorithm to the system (2.9) with initial conditions (5.1) using values of Table 1 for $\sigma = 0.05$, $\sigma = 0.1$ and $\sigma = o.15$. The simulated results shown in Figures 1-3 confirm Theorem 3.3. Furthermore, the influences of memory on the disease spreading is illustrated.

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