

## DYNAMIC ANALYSIS OF A FRACTIONAL SVIR SYSTEM MODELING AN INFECTIOUS DISEASE \*

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**Abstract.** Infectious diseases that spread by microorganisms, viruses and bacteria that can be transmitted very quickly from person to person and have negative effect on public health should be treated as soon as possible. In order to eliminate structures that are harmful to the body, or strengthen the immune system, which is the totality of cells, structures and processes, individuals are vaccinated and that way the disease is suppressed. In this manner, infectious diseases are prevented from significantly threatening public health. This paper presents the vaccination effect on an infectious disease modeled by a fractional order SVIR system. The model discussed is defined in terms of the Caputo derivative. The existence and uniqueness of the solutions of the system are handled and stability analysis at the equilibrium numerical solutions of the system are obtained by the Adams Bashforth Moulton method. The results obtained are interpreted according to the reproduction number  $R^c$ , which is a threshold parameter for the disease to become epidemic or not. For this purpose, the dependencies of numerical solutions on problem parameters are displayed graphically by Matlab software.

**Key words:** Fractional SVIR system, Caputo derivate, numerical solution, infectious disease.

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## 1. Introduction

Diseases such as smallpox, rubella, mumps and influenza are transmitted from person to person through microorganisms, bacteria, viruses and helminths. These diseases may spread differently through in the population, for example, a very large and short-term outbreak is called an epidemic. On the other hand, if the disease persists in population, it is called endemic. The spread of the disease depends not only on the route of transmission, the causative agent of infection, the latent period, but also on social, cultural, economic and demographic characteristics. Therefore, people have been fighting with epidemic diseases for many years because these diseases have negative effects on society, and other aspects of social life, e.g. tourism. Both individuals and society are affected by these diseases; therefore, it is very important to take precautions against them. Good nutrition and cleanliness are some of the ways to prevent infectious diseases. However, as seen from the COVID-19 epidemic, when these measures are not sufficient, the most effective way to prevent them is vaccination. Vaccination strengthens the immune system and helps it fight against diseases. When the immune system encounters unknown substances, it attacks them, and the substances are not stored in the body. Thus, even if an individual gets the disease, owing to the vaccine, the immune system can develop a stronger defense against the disease, and its lethality is reduced. That is why the immune system is strengthened through vaccination. As a result of vaccination, the occupancy rate in hospitals and therefore the financial burden of health care systems in treatment processes is reduced. Mathematical modelling of diseases and treatment, solution techniques to reduce the effects of the outbreak of infectious diseases, as well as clinical research to find different ways of prevention have increased over years.

In addition to clinical research, mathematical modeling is very important in determining the course of diseases and treatment methods [1, 2, 3, 4, 5]. Mathematical epidemiology, which is the intersection of medical sciences and mathematics, deals with these issues. McKendrick and Kermack [5] are the founders of the mathematical epidemic model for the spread of influenza, known as the SIR model. The mathematical models of measles, mumps, chickenpox, which are epidemic diseases, are described using the classical equations of London and Yorke [6]. Anderson and May [7] studied the mathematical models of infectious diseases transmitted by viruses, bacteria and helminths. Hethcote [8] made research on the measles and rubella models and three types of infectious disease models, while [9] investigated SIS, SIR models with and without vital dynamics. Alexander et al. [10] investigated the mathematical model of the influenza vaccine. The inoculated SIS model (SVIS) and the inoculated SIR model were studied by Shim [11] and by D'Onofrio [12], respectively. Khan et al. [13] proposed the mathematical model that includes the incidence rate and vaccination, whereas the SVIR model described the effects of two-stage vaccination (vaccination of new-borns, migrants and susceptible populations). Wang et al. [14] discussed the effects of the age of susceptibility, the age of vaccination and the age of relapse (for removed individuals) on the spread of the infectious disease. Parsamanesh and Farnoosh [15] analysed the SIS epidemic model, which includes the vaccination schedule.

Liu et al. [16] investigated the classical model of infection under the effect of vaccination. On the other hand, the course of many infectious diseases is directly dependent on the hereditary characteristics of the individual. In addition, the symptoms of chronic infectious disease in the individual are always seen in comparison to the previous experience. This reveals that in addition to being hereditary, infectious diseases also show a memory behavior. For example, while examining clinical findings of upper respiratory tract infections, which are frequently seen in individuals, it has been noticed that people usually have a genetic predisposition. In addition, the disease manifests itself with similar symptoms each time, such that the patient can often get a diagnosis. As a result, the inherited and memory features of infectious diseases suggest that modeling these diseases with fractional derivatives would be more realistic. Fractional operators can accurately give the hereditary and memory effects to the model due to their integral description with their singular/non-singular kernel functions. With this motivation, we aim to generalize the model [16] using the Caputo fractional derivative as one of the most commonly used fractional derivatives which requires integer-order initial conditions. The main purpose of this study is to investigate the existence and uniqueness of the fractional SVIR system as well as to investigate its numerical solutions. For this purpose, we have used the Grünwald-Letnikov (GL) approximation while obtaining the numerical results by considering the relationship between the Caputo and the GL definitions [33, 40, 41, 42].

The current article is divided into six parts. The description of the fractional sequential nonlinear infectious disease system is presented in section 2. In section 3, some theoretical aspects and a stability analysis of a nonlinear model for the basis of equilibrium points are given. In the section 4, we study the existence and uniqueness of the solutions for the fractional SVIR model. In section 5, numerical solutions are held by GL approximation and some simulations are given to validate the results. Finally, we conclude the results in section 6.

## 2. The fractional SVIR (epidemic) model with vaccination effect

The non-linear system consists of  $S$ ,  $V$ ,  $I$ ,  $R$  representing the fractions of susceptible (not sick, but will be sick), vaccination intensity, infected (having sick microorganisms and infecting others) and recovered (cannot infect others) individuals, respectively. Model parameters are given below:

- $\theta$  is the ratio of susceptible individuals eliminated by vaccination,
- $a$  is the recruitment and natural mortality rate of the population,
- $b$  is the rate of transmission in contact with the infected individual and the susceptible individual,
- $\xi$  is the rate of transmission in contact with an infected individual and a susceptible person after vaccination,
- $\beta$  is the average rate for susceptible individuals who have acquired immunity and transferred convalescent individuals,

- $\eta$  is the recovery rate of infected individuals where all parameters are positive.

The prescribed model is presented as follows [16]:

$$(2.1) \quad \begin{aligned} \frac{dS}{dt} &= a - S(a + bI + \theta), \\ \frac{dV}{dt} &= \theta S - V(\xi I + \beta + a), \\ \frac{dI}{dt} &= I(bS + \xi V - \eta - a), \\ \frac{dR}{dt} &= \beta V + \eta I - aR \end{aligned}$$

When  $\theta = 0$  means that there is no vaccine effect on the model, in which case the model is reduced to a classical SIR model discussed in [16]. In the literature, nonlinear integer-order differential equation systems have been studied in many fields such as physics, engineering, and health sciences. However, in recent years, researches on fractional-order nonlinear models with different kernel structures have been quite remarkable when it comes to modeling [21, 22, 23, 27, 29].

The most frequently used definitions in fractional-order models are Caputo [17, 18, 19, 20], Caputo-Fabrizio (CF) [30], and Atangana-Baleanu (AB) [24, 25, 26, 28] fractional derivatives. This is due to the fact that Riemann-Liouville, Caputo, Riesz, etc. have explained real-life problems more precisely using different types of fractional derivatives, such as [31, 32]. While analyzing the numerical solutions of fractional-order models, it can be easily seen that the change in the compartments slows down or accelerates according to the change in fractional order. This is an expected result for fractional-order models, because fractional derivatives overcome the weakness of integer derivatives in modeling slow and fast propagation processes.

We should now reconsider the basic fractional derivative definitions used in this study:

**Definition 2.1.** [33] Let  $g(t)$  be a function for fractional order  $\alpha$  ( $n - 1 \leq \alpha < n$ ).

$${}_0D_t^\alpha g(t) = \frac{1}{\Gamma(n - \alpha)} \left( \frac{d}{d\tau} \right)^n \int_0^t (t - \tau)^{n - \alpha - 1} g(\tau) d\tau$$

is the Riemann-Liouville fractional derivative.

**Definition 2.2.** [33] Let  $g(t)$  be a time dependent function and  $\alpha$  ( $n - 1 \leq \alpha < n$ ).

$${}_0^C D_t^\alpha g(t) = \frac{1}{\Gamma(n - \alpha)} \int_0^t (t - \tau)^{n - \alpha - 1} \left( \frac{d}{d\tau} \right)^n g(\tau) d\tau$$

is the Caputo fractional derivative.

Here,  $\Gamma(\cdot)$  represents Euler’s gamma function. If the initial conditions are homogeneous, these two fractional derivative definitions are equal.

Infectious diseases, which have also been discussed in the current study, are one of the subjects in which fractional derivatives are effectively used in the modeling [34, 35, 36]. In this study, we generalize the model in [16] with the Caputo derivative as follows:

$$\begin{aligned}
 (2.2) \quad D_t^\alpha S &= a^\alpha - S(a^\alpha + b^\alpha I + \theta^\alpha), \\
 D_t^\alpha V &= \theta^\alpha S - V(\xi^\alpha I + \beta^\alpha + a^\alpha), \\
 D_t^\alpha I &= I(b^\alpha S + \xi^\alpha V - \eta^\alpha - a^\alpha), \\
 D_t^\alpha R &= \beta^\alpha V + \eta^\alpha I - a^\alpha R.
 \end{aligned}$$

For this purpose, we make the parameters with alpha-order appropriately without ignoring the meaningfulness [43].

### 3. Equilibrium points and stability analysis

Disease free equilibrium points and endemic equilibrium points are calculated in this section. First, the system given by (2.2) is assumed time-independent

$$\begin{aligned}
 (3.1) \quad a^\alpha - S(a^\alpha + b^\alpha I + \theta^\alpha) &= 0, \\
 \theta^\alpha S - V(\xi^\alpha I + \beta^\alpha + a^\alpha) &= 0, \\
 I(b^\alpha S + \xi^\alpha V - \eta^\alpha - a^\alpha) &= 0, \\
 \beta^\alpha V + \eta^\alpha I - a^\alpha R &= 0
 \end{aligned}$$

and  $I = 0$  in Eq. (3.1), we get

$$\begin{aligned}
 S_0 &= \frac{a^\alpha}{a^\alpha + \theta^\alpha}, \quad V_0 = \frac{\theta^\alpha a^\alpha}{(a^\alpha + \theta^\alpha)(a^\alpha + \beta^\alpha)}, \\
 I_0 &= 0, \quad R_0 = \frac{\theta^\alpha \beta^\alpha}{(a^\alpha + \theta^\alpha)(a^\alpha + \beta^\alpha)}.
 \end{aligned}$$

Then the disease-free equilibrium point  $E_0 = (S_0, V_0, I_0, R_0)$  can be written as follows:

$$\left( \frac{a^\alpha}{a^\alpha + \theta^\alpha}, \frac{\theta^\alpha a^\alpha}{(a^\alpha + \theta^\alpha)(a^\alpha + \beta^\alpha)}, 0, \frac{\theta^\alpha \beta^\alpha}{(a^\alpha + \theta^\alpha)(a^\alpha + \beta^\alpha)} \right).$$

The reproduction number denotes the rate of new infectious individuals after contacting infectious and susceptible individuals with each other. This number is a biological threshold parameter for cases where a disease turns into endemic, epidemic, and pandemic [16]. The reproduction number ( $R^C$ ) for the fractional-order system (2.2) is

$$R^C = \frac{b^\alpha a^\alpha}{(a^\alpha + \theta^\alpha)(a^\alpha + \eta^\alpha)} + \frac{\xi^\alpha \theta^\alpha a^\alpha}{(a^\alpha + \theta^\alpha)(a^\alpha + \beta^\alpha)(a^\alpha + \eta^\alpha)}.$$

The equilibrium points of the system can also be written clearly depending on the reproduction number. Thus, the stability of the system at each equilibrium point can be interpreted depending on the comparison of  $(R^C)$  with the 1 threshold value.

If  $I \neq 0$  or  $I > 0$  is considered in Eq. (3.1), we get

$$(3.2) \quad \begin{aligned} S_* &= \frac{a^\alpha}{a^\alpha + b^\alpha I_* + \theta^\alpha}, \\ V_* &= \frac{\theta^\alpha a^\alpha}{(a^\alpha + b^\alpha I_* + \theta^\alpha)(\xi^\alpha I_* + \beta^\alpha + a^\alpha)}, \\ R_* &= \frac{\theta^\alpha \beta^\alpha}{(a^\alpha + b^\alpha I_* + \theta^\alpha)(\xi^\alpha I_* + \beta^\alpha + a^\alpha)} + \frac{\eta^\alpha I_*}{a^\alpha}, \end{aligned}$$

and

$$(3.3) \quad b^\alpha S_* + \xi^\alpha V_* - \eta^\alpha - a^\alpha = 0.$$

While substituting Eq. (3.2) into Eq. (3.3), we obtain,

$$\begin{aligned} &\xi^\alpha b^\alpha (a^\alpha + \eta^\alpha) I_*^2 + ((a^\alpha + \eta^\alpha)((a^\alpha + \theta^\alpha) \xi^\alpha + (a^\alpha + \beta^\alpha) b^\alpha) - \xi^\alpha b^\alpha a^\alpha) I_* \\ &+ (a^\alpha + \eta^\alpha)(a^\alpha + \beta^\alpha)(a^\alpha + \theta^\alpha) \left( 1 - \frac{b^\alpha a^\alpha (a^\alpha + \beta^\alpha) - \theta^\alpha \xi^\alpha a^\alpha}{(a^\alpha + \theta^\alpha)(a^\alpha + \beta^\alpha)(a^\alpha + \eta^\alpha)} \right) = 0. \end{aligned}$$

$I_*$  is the positive root of

$$X_1 I_*^2 + X_2 I_* + X_3 (1 - R^C)$$

where

$$\begin{aligned} X_1 &= \xi^\alpha b^\alpha (a^\alpha + \eta^\alpha) \\ X_2 &= (a^\alpha + \eta^\alpha)((a^\alpha + \theta^\alpha) \xi^\alpha + (a^\alpha + \beta^\alpha) b^\alpha) - \xi^\alpha b^\alpha a^\alpha \\ X_3 &= (a^\alpha + \eta^\alpha)(a^\alpha + \beta^\alpha)(a^\alpha + \theta^\alpha). \end{aligned}$$

Since the problem parameters are positive we reach  $X_1 > 0$ ,  $X_3 > 0$  and  $R^C > 1$ . Thus, the endemic equilibrium point  $E_* = (S_*, V_*, I_*, R_*)$  is obtained

$$\left( \frac{a^\alpha}{a^\alpha + b^\alpha I_* + \theta^\alpha}, \frac{\theta^\alpha a^\alpha}{(a^\alpha + b^\alpha I_* + \theta^\alpha)(\xi^\alpha I_* + \beta^\alpha + a^\alpha)}, \right. \\ \left. I_*, \frac{\theta^\alpha \beta^\alpha}{(a^\alpha + b^\alpha I_* + \theta^\alpha)(\xi^\alpha I_* + \beta^\alpha + a^\alpha)} + \frac{\eta^\alpha I_*}{a^\alpha} \right).$$

**Lemma 3.1.** *The system is locally asymptotically stable if  $R^C < 1$  and unstable if  $R^C > 1$  at  $E_0$ .*

*Proof.* We compute the Jacobian matrix of the system (2.2).

$$J(S, V, I, R) = \begin{pmatrix} -(a^\alpha + \theta^\alpha + b^\alpha I) & 0 & -b^\alpha S & 0 \\ \theta^\alpha & -(a^\alpha + \beta^\alpha + \xi^\alpha I) & -\xi^\alpha V & 0 \\ b^\alpha I & \xi^\alpha I & b^\alpha S + \xi^\alpha V - a^\alpha - \eta^\alpha & 0 \\ 0 & \beta^\alpha & \eta^\alpha & -a^\alpha \end{pmatrix}.$$

Hence, we obtain

$$J(E_0) = \begin{pmatrix} -(a^\alpha + \theta^\alpha) & 0 & -b^\alpha S_0 & 0 \\ \theta^\alpha & -(a^\alpha + \beta^\alpha) & -\xi^\alpha V_0 & 0 \\ 0 & 0 & b^\alpha S_0 + \xi^\alpha V_0 - a^\alpha - \eta^\alpha & 0 \\ 0 & \beta^\alpha & \eta^\alpha & -a^\alpha \end{pmatrix}.$$

The eigenvalue  $\lambda_1 = -a^\alpha$  is computed easily and is real and negative. The other eigenvalues are  $\lambda_2 = -(a^\alpha + \theta^\alpha) < 0$ ,  $\lambda_3 = -(a^\alpha + \beta^\alpha) < 0$  and  $\lambda_4 = b^\alpha S_0 + \xi^\alpha V_0 - a^\alpha - \eta^\alpha = (a^\alpha + \eta^\alpha)(R^C - 1)$  are obtained. If  $R^C < 1$ , the eigenvalue  $\lambda_4$  is negative. So the all eigenvalues of  $J(E_0)$  is negative and the disease free equilibrium  $E_0$  is locally asymptotically stable. If  $R^C > 1$ , the eigenvalue  $\lambda_4$  is positive and  $E_0$  is unstable.  $\square$

**Lemma 3.2.** *The system is locally asymptotically stable if  $R^C > 1$  at endemic equilibrium point  $E_*$ .*

*Proof.* The Jacobian matrix of the system (2.2) for  $E_*$  is

$$J(E_*) = \begin{pmatrix} -(a^\alpha + \theta^\alpha + b^\alpha I_*) & 0 & -b^\alpha S_* & 0 \\ \theta^\alpha & -(a^\alpha + \beta^\alpha + \xi^\alpha I_*) & -\xi^\alpha V_* & 0 \\ b^\alpha I_* & \xi^\alpha I_* & (b^\alpha S_* + \xi^\alpha V_* - a^\alpha - \eta^\alpha) & 0 \\ 0 & \beta^\alpha & \eta^\alpha & -a^\alpha \end{pmatrix}.$$

The characteristic equation of  $J(E_*)$  is  $(a^\alpha + \lambda)(\lambda^3 + p_1\lambda^2 + p_2\lambda + p_3) = 0$  where

$$\begin{aligned} p_1 &= \frac{a^\alpha}{S_*} + \frac{\theta^\alpha S_*}{V_*} > 0, \\ p_2 &= \frac{\theta^\alpha S_*}{V_*} + \xi^{2\alpha} V_* I_* + b^{2\alpha} S_* I_* > 0, \\ p_3 &= (\theta\xi b)^\alpha S_* I_* + \frac{\theta^\alpha b^{2\alpha} S_*^2 I_*}{V_*} + \frac{a^\alpha \xi^{2\alpha} V_* I_*}{S_*} > 0, \end{aligned}$$

and hence,

$$\begin{aligned} p_1 p_2 - p_3 &= \frac{a^\alpha}{S_*} \left( \frac{a^\alpha \theta^\alpha}{V_*} + b^\alpha S_* I_* \right) + \frac{\theta^\alpha S_*}{V_*} \left( \frac{a^\alpha \theta^\alpha}{V_*} + \xi^\alpha V_* I_* \right) - (\theta\xi b)^\alpha S_* I_* \\ &= \frac{a^{2\alpha} \theta^\alpha}{S_* V_*} (a^\alpha + \theta^\alpha + b^\alpha I_*) b^{2\alpha} S_* I_* + \frac{a^\alpha \theta^{2\alpha} S_*}{V_*^2} + \theta^\alpha S_* I_* (b^\alpha - \xi^\alpha)^2 \\ &\quad + (\theta\xi b)^\alpha S_* I_* > 0. \end{aligned}$$

So, we get  $p_1 > 0$ ,  $p_3 > 0$  and  $p_1 p_2 > p_3$  for  $R^C > 1$ . By the Routh-Hurwitz criterion in [37], all the eigenvalues of the above characteristic equations have a negative real part. So, the equilibrium point  $E_*$  is locally asymptotically stable.  $\square$

#### 4. The existence and uniqueness of the system solution

**Lemma 4.1.** *Assume that  $g(t) \in C[a, b]$  and  $D_t^\alpha g(t) \in C[a, b]$  for  $\alpha \in (0, 1]$ , then we get*

$$g(t) = g(a) + \frac{1}{\Gamma(\alpha)} D_t^\alpha g(\tau) (t-a)^\alpha, \quad a < \tau < t, \quad \forall t \in (a, b]$$

and it is called *Generalized Mean Value Theorem [38]*.

**Lemma 4.2.** [38] *Assume that  $g(t) \in C[a, b]$  and  $D_t^\alpha g(t) \in C[a, b]$  for  $\alpha \in (0, 1]$ .  $g(t)$  is non-increasing on  $[a, b]$  if  $D_t^\alpha g(t) \leq 0$ , for each  $t \in [a, b]$ .  $g(t)$  is non-decreasing on  $[a, b]$  if  $D_t^\alpha g(t) \geq 0$ , for each  $t \in [a, b]$ .*

**Theorem 4.1.** [39] *The fractional order SVIR system (2.2) has a unique solution in  $R_+^4$ , and the closed set  $\Omega = \{(S, V, I, R) \in R_+^4 : S + V + I + R = 1\}$  is positively invariant with respect to model (2.2).*

*Proof.* By considering Theorem 3.1 and Remark 3.2 in [39], we must show  $\|f(t, g)\| \leq \omega + \lambda \|g(t)\|$  for the initial value problems as follow:

$$\begin{cases} D^\alpha g(t) = f(t, g(t)) \\ g(t_0) = g_0, \quad \alpha \in (0, 1] \end{cases}$$

Assume that  $g_1(t) = S(t)$ ,  $g_2(t) = V(t)$ ,  $g_3(t) = I(t)$ ,  $g_4(t) = R(t)$ ,  $g_1(0) = S(0)$ ,

$$g_2(0) = V(0), \quad g_3(0) = I(0), \quad g_4(0) = R(0), \quad g(t) = \begin{pmatrix} g_1(t) \\ g_2(t) \\ g_3(t) \\ g_4(t) \end{pmatrix}, \quad a = \begin{pmatrix} a^\alpha \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

$$B_1 = \begin{pmatrix} -(a^\alpha + \theta^\alpha) & 0 & 0 & 0 \\ \theta^\alpha & -(\beta^\alpha + a^\alpha) & 0 & 0 \\ 0 & 0 & -(\eta^\alpha + a^\alpha) & 0 \\ 0 & \beta^\alpha & \eta^\alpha & -a^\alpha \end{pmatrix},$$

$$B_2 = \begin{pmatrix} -b^\alpha & 0 & 0 & 0 \\ 0 & \xi^\alpha & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad B_3 = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & b^\alpha & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad B_4 = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & \xi^\alpha & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}.$$

When the matrix representations are substituted in system (2.2),

$$\begin{aligned} D^\alpha g(t) &= B_1 g(t) + g_3(t) B_2 g(t) + g_1(t) B_3 g(t) \\ &\quad + g_2(t) B_4 g(t) + a. \end{aligned}$$

Then, we can write

$$f(t, g(t)) = B_1g(t) + g_3(t)B_2g(t) + g_1(t)B_3g(t) + g_2(t)B_4g(t) + a,$$

and

$$\begin{aligned} \|f(t, g(t))\| &= \|B_1g(t) + g_3(t)B_2g(t) + g_1(t)B_3g(t) \\ &\quad + g_2(t)B_4g(t) + a\| \\ &\leq \|a\| + \|B_1\| \|g(t)\| + \|B_2\| \|g(t)\| \\ &\quad + \|B_3\| \|g(t)\| + \|B_4\| \|g(t)\| \\ &= \omega + (\|B_1\| + \|B_2\| + \|B_3\| \\ &\quad + \|B_4\|) \|g(t)\| \\ &= \omega + \lambda \|g(t)\|. \end{aligned}$$

Hence, system (2.2) has a unique solution.

Now, we show that the solution of the model (2.2) is positive. From the model (2.2), get:

$$\begin{aligned} D_t^\alpha S|_{S=0} &= a^\alpha \geq 0, \\ D_t^\alpha E|_{V=0} &= \theta^\alpha S \geq 0, \\ D_t^\alpha I|_{I=0} &= 0, \\ D_t^\alpha K|_{R=0} &= \beta^\alpha V + \xi^\alpha I \geq 0 \end{aligned}$$

and with Lemma 4, we get  $S(t), V(t), I(t), R(t) \geq 0$  for any  $t \geq 0$ .

All equations in system (2.2) are added side by side and we obtain

$$(4.1) \quad D_t^\alpha T(t) = D_t^\alpha S(t) + D_t^\alpha V(t) + D_t^\alpha I(t) + D_t^\alpha R(t),$$

and then

$$(4.2) \quad D_t^\alpha T(t) = a^\alpha - a^\alpha T(t).$$

Solving the fractional differential equation (4.2) by Laplace transform gives, we obtain  $0 \leq T(t) \leq (1 - E_\alpha(-a^\alpha t^\alpha)) + T(0)(E_\alpha(-a^\alpha t^\alpha))$  for  $t \rightarrow \infty$ , then we have  $0 \leq T(t) \leq 1$ .

Since the solution set of the model given in  $\Omega$  with the initial conditions remain in  $\Omega$ , the region  $\Omega$  is positively invariant for model (2.2).  $\square$

### 5. Numerical simulations via Grünwald-Letnikov method

Analytical solutions of nonlinear fractional differential equation systems are often impossible to find. The GL approach that we apply in this study is one of them

[40, 41, 42]. The GL definition, which is equivalent to the RL definition, is based on the finite-difference scheme and is defined as follows:

$$(5.1) \quad D^\alpha g(t) \approx h^{-\alpha} \sum_{k=0}^{\lceil t/h \rceil} (-1)^k \binom{\alpha}{k} g(t - kh),$$

where  $h$  is step size and  $\lceil t \rceil$  denotes the integer part of  $t$ , and for  $0 < \alpha < 1$ ,

$${}_0^C D_t^\alpha g(t) = D^\alpha g(t) - \frac{t^{-\alpha}}{\gamma(1-\alpha)} g_0.$$

For numerical algorithm, the discretized form of  $D^\alpha g(t)$  is used as  $\sum_{k=0}^{\lceil t_n/h \rceil} c_k^\alpha g(t_{n-k})$  where  $t_n = nh$ , and  $c_k^\alpha$  are the GL coefficients given as follows:

$$c_k^{(\alpha)} = h^{-\alpha} (-1)^k \binom{\alpha}{k}, \quad k = 0, 1, 2, \dots$$

We can evaluate these coefficients as follow:

$$(5.2) \quad c_0^{(\alpha)} = h^{-\alpha}, \quad c_k^{(\alpha)} = \left(1 - \frac{\alpha+1}{k}\right) c_{k-1}^{(\alpha)}, \quad k = 1, 2, 3, \dots$$

First, in Figures (5.1), we evaluate the dependence of the numerical solutions of the system on the fractional parameter when the disease is in the endemic state, i.e.  $R^c < 1$ . We use the parameters while obtaining the numerical simulations  $\theta = 10$ ,  $a = 1$ ,  $b = 10$ ,  $\xi = 2$ ,  $\beta = 8$ ,  $\eta = 4$  and initial values  $S_0 = 2$ ,  $V_0 = 1$ ,  $I_0 = 1$ ,  $R_0 = 0$  given in [16]. In the model discussed, susceptible individuals are vaccinated. The decrease in the number of susceptible individuals is a natural consequence of this assumption. It is possible that the number of vaccinated individuals may decrease as seen in Figure (5.1), as vaccinated individuals interact with infected individuals at a rate of  $\xi$ . Moreover, as the  $\alpha$  value decreases, the number of vaccinated individuals seems to decrease more rapidly. We can interpret this as follows: If the infectious disease represented by the model regresses faster thanks to vaccination, this situation should be modeled with the system of the order of  $\alpha = 0.7$ . This means that vaccination is highly effective in controlling the disease modeled with  $\alpha = 0.7$ . Infected individuals in Figure (5.1) also supports this result. As seen in this figure, in the case of  $\alpha = 0.98$ , despite the vaccination policy, the number of infected individuals reaches the maximum value exceeding two times, while the maximum value reached at  $\alpha = 0.7$  is much smaller. The response of the system for different values of  $\alpha$  represents the response of infectious diseases of different strength to vaccination. When  $\alpha = 0.7$ , the number of infected individuals is low compared to other conditions, and as a natural result, the number of individuals recovering is low. In Figures (5.2), we aim to examine the response of the model, assuming parameters  $\theta = 10$ ,  $a = 1$ ,  $b = 20$ ,  $\xi = 15$ ,  $\beta = 2$  and  $\eta = 1$  in case of pandemic given in [16], i.e.  $R^C < 1$ . Initial conditions are assumed

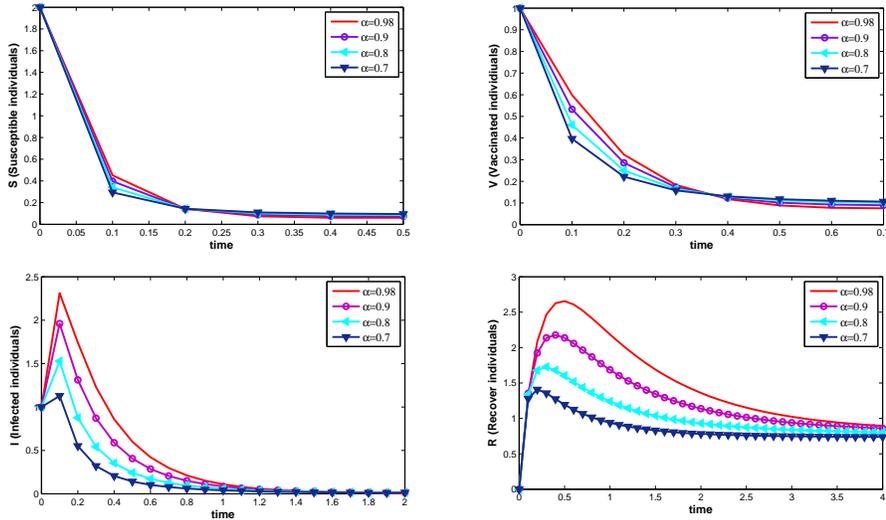


FIG. 5.1: Fractional parameter dependence of the number of susceptible, vaccinated, infected and recovered individuals for the endemic status of the disease.

as  $S_0 = 2, V_0 = 1, I_0 = 1, R_0 = 0$ . As seen in these figures, the maximum value reached by infected individuals increases in case of epidemic despite vaccination. In epidemics, a more aggressive course is observed due to the spread of the disease compared to endemics. In an epidemic situation, the reduction in the number of individuals vaccinated at time  $t = 0.1$  is more successful than endemic case. This means that the vaccination policy implemented is very effective in controlling the epidemic. Once again, desirable results for epidemic disease have been obtained, which is modeled with  $\alpha = 0.7$ , compared to other situations. In case of epidemics, increasing  $\alpha$  values represent the order of infectious disease models that show more aggression despite vaccination.

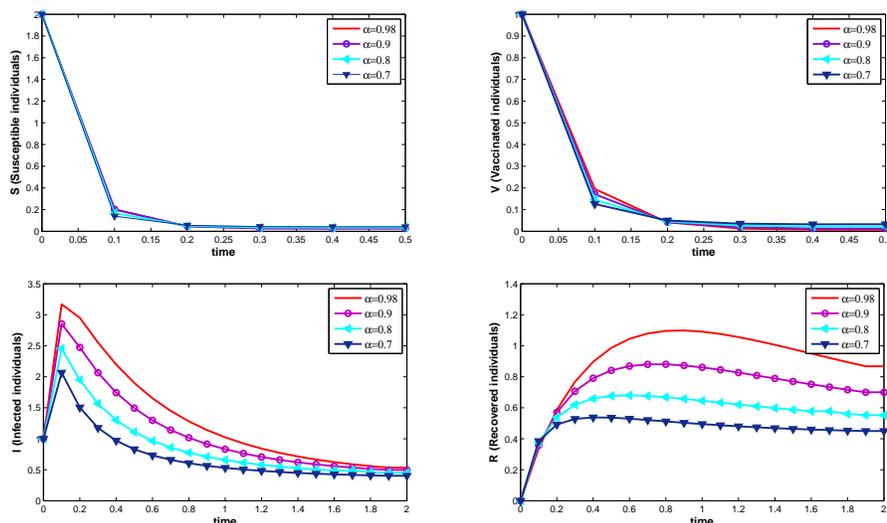


FIG. 5.2: Fractional parameter dependence of the numbers of susceptible and vaccinated individuals for the epidemic status of the disease.

## 6. Concluding remarks

In this study, a SVIR model under the influence of vaccination for an infectious disease has been studied by generalizing it with Caputo fractional derivative. Firstly, the existence and uniqueness of the solutions of the generalized model has been proved. Then, the equilibrium points of the system have been determined and the stability analysis of the system has been made at these points. The GL approach has been applied to the Caputo fractional derivative to obtain the numerical solutions of the generalized system. By using Matlab software, numerical solutions are illustrated via graphs and hence comparative remarks for the effect of fractional parameter have been given in details. The increasing effect of vaccination in both endemic and epidemic situation has been observed at decreasing values of fractional order.

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