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Ali Akgü'l

Department of Computer Science and Mathematics, Lebanese American University, Beirut, Lebanon \ IRO, Research Center, University of Halabja, Halabja, 46018, Iraq \ Department of Mathematics, Art and Science Faculty, Siirt University, 56100 Siirt, Turkey \ Department of Mathematics, Mathematics Research Center, Near East University, Near East Boulevard, PC: 99138, Nicosia /Mersin 10 Turkey,
aliakgul00727@gmail.com

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Analysis of COVID-19 Disease Transmission with the Impact of Vaccine

Ali Akgül1,2,3,4

1Department of Computer Science and Mathematics, Lebanese American University, Beirut, Lebanon
2IRO, Research Center, University of Halabja, Halabja, 46018, Iraq
3Department of Mathematics, Art and Science Faculty, Siirt University, 56100 Siirt, Turkey
4Department of Mathematics, Mathematics Research Center, Near East University, Near East Boulevard, PC: 99138, Nicosia /Mersin 10 Turkey

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Abstract: In this work, we incorporate the effects of the vaccine into the construction of the Covid-19 disease model. We discuss the many kinds of equilibria in detail. This is where the stability analysis is shown. Additionally, we fractionalize the model using the Mittag-Leffler kernel. We obtain the model’s solutions by an extremely effective numerical technique. We provide a few computer simulations to show that the proposed method works.

Keywords: Covid-19 model; numerical method; existence of solution; stability analysis; fractal-fractional model; computational analysis.

1 Introduction

The 2019 Coronavirus Disease (also known as COVID-19) was first identified in December 2019 in the Chinese city of Wuhan. It is an acute respiratory syndrome coronavirus carried on by the SARS-CoV-2 virus. All the medical problems start as the cause of a pneumonia of unknown backgrounds [7, 17, 20]. The World Health Organization (WHO) proclaimed COVID-19 an international pandemic on March 11, 2020, as a result of the rapid spread of SARS-CoV-2 worldwide [20, 4, 26]. Due to the COVID-19 virus’s rapid spread, significant global countermeasures have been put in place, including the quarantining of people who are thought to be at risk for the virus, isolating and quarantining confirmed cases, using face masks in public, monitoring contacts, establishing social distance, closing schools and universities, and working remotely from home or in institutions without scholars. Besides these steps, since the beginning of the pandemic state, Governments of countries affected by the disease have been developing and implementing some programmes to come back to “normality”. One of this programmes was the occurrence of certain vaccines regarding COVID-19 [3, 37]. Ones of the well-known vaccines utilized in immunization are: Pfizer, Moderna, AstraZeneca, and Janssen. For Pfizer, Moderna, and AstraZeneca, the interval between two immunization doses is three weeks, four weeks, and twelve weeks, respectively. For Pfizer and Moderna and AstraZeneca vaccinations, efficacy is attained seven days following the dose recall, and after 14 days. After 14 days, the Janssen single-dose vaccination reaches its advertised effectiveness. The effectiveness of vaccination depends on the population’s overall immunity to this pathogen, which reduces the number of susceptible hosts and lessens the impact of infected individuals [25, 27]. It has been suggested that fractional models are crucial for systems with genetic traits. As a result, there are numerous research that deal with fractional analysis. Ghanbari et al. [14] have discussed some new edge detecting methods about the fractional derivatives. Baleanu et al. [5, 6] have investigated the variable-order fractional difference equations. Singh et al. [31] have discussed the methods of mathematical modelling. Shiri et al. [29] have searched the system of the fractional differential algebraic equations with applications. For pandemic planning and informing mass vaccination methods, it is crucial to understand the impact of vaccine on transmission. Numerous studies have demonstrated how vaccination affects COVID-19 infection and hospitalization rates outside of
clinical trial settings [10,9,34,30,2,28,15,38,22,16,18,19]. Some models concerning the vaccination with one or two doses and certain properties of them were discussed in [8,11,23,24,33,35,36]. For more details see [39,40,41,42,43,44,45]. In this study, we add one parameter to the model to examine the effect of immunization on the risk of COVID-19 infection. We evaluate the COVID-19 illness model proposed for investigation with fractal-fractional derivatives because fractional operators with various memory is related to the different type of relaxation process of the non-local dynamical systems. The use of the fractal-fractional operators method to note crossover behavior is well recognized. Our manuscript is structured as follows: Section 1 presents the key definitions and theorems that are relevant to our work. In Sections 2 and 3, we build the model, and Section 3 is where we analyze it. In this section, we highlight the effectiveness of the remedies. In this section, we also present the roundedness. In Section 4, we go over the existence and classification of equilibriums. In this area, we also provide the local stability of the disease-free. In Section 5, we present the backward bifurcation. In Section 6, we go over how the suggested model can be used with the Mittag-Leffler kernel. In this section, we also show examples of the numerical simulations. In the final portion, Section 6, we provide the conclusions. Further, let us give some important definitions useful in our study.

**Definition 1.** Let \((a,b)\) be an open interval with order \(\eta\) and the function \(f(u)\) be continuous and fractal differentiable. Following that, the generalized Mittag-Leffler type kernel-containing fractal-fractional derivative of \(f\) in the Riemann-Liouville sense with order \(\alpha\) is given as follows by\([1]\):

\[
D_{a}^{\sigma,a,\eta}f(u) = \frac{AB(\sigma)}{1-\sigma} \frac{d}{du} \int_{a}^{u} f(s) \left( \frac{1-s}{1-\sigma} \right)^{\sigma} ds, \quad 0 < \sigma, \eta \leq 1,
\]

where, \(AB(\sigma) = 1 - \sigma + \frac{\sigma}{1-\sigma}\).

**Definition 2.** Given a function \(f\) that is continuous in an open interval, say \((a,b)\), the fractal-fractional integral of order \(\sigma\) that corresponds to the fractal fractional derivative with a kernel of the Mittag-Leffler type is given by \([1]\):

\[
I_{a}^{\sigma,a,\eta}f(u) = \frac{\sigma \eta}{AB(\sigma)} \int_{0}^{u} s^{\sigma-1} f(s) (u-s)^{\eta-1} ds + \frac{\eta(1-\sigma)u^{\eta-1}}{AB(\sigma)} f(u).
\]

2 The dimensional model

We give our model with quarantine segment \([13]\). We will use the following notations concerning the infected populations:

- \(S\) - Susceptible: People who are at risk even though they have not yet contracted the disease because they lack the required antibodies to fight it.
- \(E\) - Exposed: Individuals who are already infected but not yet affected to prevent the infection from spreading. The latent period is the name given to this state, which has a short lifespan that varies based on the sickness.
- \(I\) - Infectious: people who become infected and can spread the virus.
- \(R\) - Recovered: The patients who were successful in beating their sickness. The body creates immunity as an adaptive reaction to the infectious pathogen to stop a healed patient from getting smarter.

The population is considered homogenous, its spatial distribution is uniform, and all susceptible individuals are considered to be equally likely to be infected. This hypothesis make the problem much easier. The mathematical representation for our model with quarantine segment is given by the next system of differential equations:

\[
\frac{dS}{du} = (1-q)A - (\alpha I + \rho + v)S,
\]

\[
\frac{dE}{du} = \alpha SL - (\theta + \rho)E,
\]

\[
\frac{dI}{du} = \theta E - (\mu + \delta + \rho)I,
\]

\[
\frac{dR}{du} = qA + \delta I + vS - \rho R,
\]

where \(v\) is the vaccinated parameter.
3 Model Analysis

3.1 Positivity of solutions

**Theorem 1.** Assume that $S_0, E_0, I_0, R_0 \geq 0$. The solution of Eqs. (3-6) with $(S(0), E(0), I(0), R(0)) = (S_0, E_0, I_0, R_0)$ is non-negative, $S(u), E(u), I(u), R(u) \geq 0$, for $u > 0$.

**Proof.** We have form equation (Eq. 3) that,

$$\frac{dS(u)}{du} = (1-q)A - S(u)y(u),$$

where

$$y(u) = (aI + \rho + v).$$

This give by integrating equation (7),

$$s(u) = \left( \int_{0}^{u} \rho e^{k \gamma(t) \alpha} d\tau + S_0 \right) e^{-k \gamma(u) \alpha} > 0.$$

Therefore, $S(u)$ is positive for all $u$. From equation (4), we acquire

$$\frac{dE(u)}{du} \geq -(\theta + \rho)E(u),$$

or equivalently,

$$-\frac{de(u)}{du} \leq (\theta + \rho)E(u),$$

Using the Grönwall’s lemma for Eq. (10) with initial state $E_0$ we obtain the following,

$$-e(u) \leq -E_0 e^{-\int_{0}^{u} (\theta + \rho) d\tau} < 0$$

This prove that $E(u) > 0$. Then, we have $I(u), R(u) > 0$. Since the model’s solutions are always positive, we draw this conclusion.

3.2 Boundedness

**Theorem 2.** The compact set $\Psi$ described as:

$$\Psi = \{(S(u), E(u), I(u), R(u)) \in \mathbb{R}^4 : S(u) + E(u) + I(u) + R(u) \leq \frac{A}{\rho} \},$$

where $(S(u), E(u), I(u), R(u))$ are the solutions of the model (Eqs. 3-6), by $(S_0, E_0, I_0, R_0)$, and it is a positively invariant region.

**Proof.** Denote by $L(u) = S(u) + E(u) + I(u) + R(u)$ then we have

$$\frac{dL(u)}{du} = -L(u)\rho - \mu \cdot i + A,$$

Therefore, $L(u) \leq \left( L(0) \exp(-\rho u) + \frac{A}{\rho} (1 - \exp(-\rho u)) \right)$, where $L(0)$ is the initial condition of $L(u)$. Therefore $0 < L(u) < \frac{A}{\rho}$ as $u$ goes to $+\infty$ and $\frac{dL(u)}{du} < 0$ for $L > \frac{A}{\rho}$. This presents that $\Psi$ is positively invariant.
4 Existence and classification of equilibria

The disease-free equilibrium of model is given by

\[ K_0(S, E, I, R) = \left( \frac{A(1-q)}{\rho + v}, 0, \frac{A(\rho q + v)}{\rho + v} \right) \]  

(12)

The other steady state solution called by endemic equilibrium \((K_1)\) is obtained,

\[ S = (\mu + \delta + \rho)(\rho + \theta), \]  

(13)

\[ E = \frac{(1-q)A}{\rho + \theta} - \frac{(\mu + \delta + \rho)v}{\rho + \theta} \]  

(14)

\[ I = \frac{(\theta(1-q)A)}{\rho + \theta} - \frac{v}{\alpha} \]  

(15)

\[ R = \frac{((\rho^2 + (\theta + \delta + \mu)\rho + \theta \mu)q + \theta \delta)}{(\mu + \delta + \rho)(\rho + \theta)} - \frac{\delta}{\alpha} \]  

(16)

Then, we get:

\[ R_0 = \frac{\alpha A(1-q)}{\rho + v} \frac{A}{\mu + \delta + \rho}(\mu + \delta + \rho) \]  

(17)

4.1 Local stability of the disease-free solution

The Jacobian matrix of Eqs.(3-6) at the disease-free solution \(K_0\) is presented as:

\[ J(K_0) = \begin{bmatrix} -\rho - v & 0 & \frac{\alpha A(q-1)}{\rho + v} & 0 \\ 0 & -\rho - \theta & -\frac{\alpha A(q-1)}{\rho + v} & 0 \\ 0 & \theta & -\mu - \delta - \rho & 0 \\ v & 0 & \delta & -\rho \end{bmatrix} \]  

(18)

Its eigenvalues are:

\[ \lambda_i = \begin{bmatrix} -\rho \\ -[\rho + v] \\ \lambda_3 \\ \lambda_4 \end{bmatrix} \]  

(19)

with

\[ \lambda_3 = \frac{1}{2(\rho + v)} \left[ - (\rho + v)(\theta + \delta + 2 \rho + \mu) + \sqrt{Y} \right], \]

\[ \lambda_4 = \frac{1}{2(\rho + v)} \left[ - (\rho + v)(\theta + \delta + 2 \rho + \mu) - \sqrt{Y} \right] < 0, \]

\[ Y = 4(\rho + \theta)(\mu + \delta + \rho)(\rho + v)^2 R_0 + (\rho + v)^2(\theta - \delta - \mu)^2 > 0. \]

Thus, we reach \(\lambda_3 = 0\) when \(R_0 = 1\), \(\lambda_3 < 0\) for \(R_0 < 1\) and \(\lambda_3 > 0\) when \(R_0 > 1\).

**Lemma 1.** The disease-free solution \(K_0\) is locally asymptotically stable if \(R_0 < 1\) and unstable if \(R_0 > 1\).

**Theorem 3.** \(K_0\) is globally asymptotically stable when \(A \alpha \theta < \rho (\mu + \delta + \rho)(\rho + \theta)\).
We assume Theorem 4. The Jacobian matrix of $\alpha \theta$ is 
$J = 
\begin{bmatrix}
-\rho - v & 0 & -\frac{(\mu + \delta + \rho)(\rho + \theta)}{\theta} & 0 \\
0 & -\rho - \theta & -\frac{(\mu + \delta + \rho)(\rho + \theta)}{\theta} & 0 \\
0 & \theta & -\delta - \rho - \mu & 0 \\
v & 0 & \delta & -\rho 
\end{bmatrix}
$ (26)

The right eigenvector of $J$ for the zero eigenvalue is 
$(w_1, w_2, w_3, w_4) = 
\begin{bmatrix}
\rho(\mu + \delta + \rho)(\rho + \theta) \\
0 & \theta & \rho v \rho v - \theta \rho v - \rho v \rho v \rho v \rho v \rho v \\
0 & 0 & \theta & \rho v \rho v - \theta \rho v - \rho v \rho v \rho v \rho v \\
0 & 0 & 0 & \rho \rho v \rho v \theta
\end{bmatrix}
$ (27)

The left eigenvector is $(v_1, v_2, v_3, v_4) = (0, \frac{\theta}{\rho + \theta}, 1, 0)$. Next, applying same methodology as in [21] we derive the expressions of the two variables $a$ and $b$. The parameter $a$ is described as:

$$a = \sum_{k,j=1}^{4} v_k w_i w_j \frac{\partial^2 f_i}{\partial x_k \partial x_j}$$
which is equivalent (since $v_1 = v_4 = 0$) to:

$$a = v_2 w_1 w_3 \frac{\partial^2 f_2}{\partial x_1 \partial x_3} + v_2 w_3 w_1 \frac{\partial^2 f_2}{\partial x_3 \partial x_1}. \quad (28)$$

Substituting for the different derivatives gives:

$$a = -2 \frac{\theta^2 (\mu + \delta + \rho) \rho^2 (\rho + v) \alpha}{((-(\mu - \rho) v + \delta \rho) \theta - \rho v (\mu + \delta + \rho))^2}.$$

We have next

$$b = \sum_{k,i=1}^4 v_3 w_i \frac{\partial^2 f_k}{\partial x_i \partial \alpha}, \quad (29)$$

which is also reduced to

$$b = v_2 w_3 \frac{\partial^2 f_2}{\partial x_3 \partial \alpha} = \frac{\theta^2 \rho A (1 - q)}{(\rho + \theta) (-(\mu - \rho) v + \delta \rho) \theta - \rho v (\mu + \delta + \rho)}. \quad (30)$$

Therefore, we conclude that $b$ is always positive when

$$v < \frac{\theta \delta \rho}{(\mu + \rho) \theta + \rho (\mu + \delta + \rho)} \quad (31)$$

Thus, the backward bifurcation is not exist since $a < 0$. The proof is completed.

Next result assure the asymptotically stability of the equilibrium point of the model.

**Theorem 5.** $K_1$ is asymptotically stable when $R_0 > 1$.

**Proof:** The components of the endemic equilibrium point can be rewritten, as follows,

$$S = \frac{(1 - q) A}{R_0 (\rho + v)}$$
$$E = \frac{(1 - q) (R_0 - 1) A}{R_0 (\rho + \theta)}$$

The Jacobian matrix at the endemic equilibrium point is,

$$J = \begin{bmatrix}
-\mathcal{R}_0 (\rho + v) & 0 & -(\mu + \delta + \rho) (\rho + \theta) \\
\rho + v (\mathcal{R}_0 - 1) - \rho - \theta & \frac{(-\mu + \delta + \rho) (\rho + \theta)}{\theta} & 0 \\
0 & \theta & -\mu - \delta - \rho \\
v & 0 & \delta - \rho
\end{bmatrix} \quad (32)$$

The eigenvalues are the solutions of the characteristic polynomial

$$[H + \rho] [H^1 + (\mathcal{R}_0 (\rho + v) + \theta + \delta + 2 \rho + \mu) H^2 + (\rho + v) (\theta + \delta + 2 \rho + \mu) \mathcal{R}_0 H + (\rho + v) (\mu + \delta + \rho) (\rho + \theta) (\mathcal{R}_0 - 1)] = 0. \quad (33)$$

One of the eigenvalues of equation (33) is $H = -\rho$. Instead of calculating the other three eigenvalues of Equation 33, which is a difficult task, the Routh-Hurwitz stability criterion applied to the characteristic equation of the third order remaining submatrix, where

$$a_0 = 1,$$
$$a_1 = (\mathcal{R}_0 (\rho + v) + \theta + \delta + 2 \rho + \mu),$$
$$a_2 = (\rho + v) (\theta + \delta + 2 \rho + \mu) \mathcal{R}_0,$$
$$a_3 = (\rho + v) (\mu + \delta + \rho) (\rho + \theta) (\mathcal{R}_0 - 1).$$
It is obvious that $a_1, a_2$ and $a_3$ is positive when $\mathcal{R}_0 > 1$. For the other condition, we have

$$a_1a_2 - a_3 = (\rho + v)^2(\theta + \delta + 2\rho + \mu)\mathcal{R}_0^2 + (\rho + v)(\theta^2 + (\delta + 3\rho + \mu)\theta + 3\rho^2 + (3\delta + 3\mu)\rho + (\delta + \mu)^2)\mathcal{R}_0 + (\rho + v)(\mu + \delta + \rho)(\rho + \theta) > 0.$$ 

which always holds. Therefore, all solutions to Equation 33 have real portions that are negative. As a result, the model’s equilibrium point is only locally asymptotically stable when $\mathcal{R}_0 > 1$.

6 Application of the Model with Mittag-Leffler Kernel

We consider next, the following derivatives.

$$\begin{align*}
\frac{\alpha}{0} FFM D^\sigma_u S(u) &= (1 - q)A - (\alpha I + \rho + v)S, \\
\frac{\alpha}{0} FFM D^\sigma_u E(u) &= \alpha SI - (\theta + \rho)E, \\
\frac{\alpha}{0} FFM D^\sigma_u I(u) &= \theta E - (\mu + \delta + \rho)I, \\
\frac{\alpha}{0} FFM D^\sigma_u R(u) &= qA + \delta I + vS - \rho R.
\end{align*}$$

Then, we obtain

$$\begin{align*}
\frac{\alpha}{0} AB D^\sigma_u S(u) &= \eta u^{n-1}(1 - q)A - (\alpha I + \rho + v)S, \\
\frac{\alpha}{0} AB D^\sigma_u E(u) &= \eta u^{n-1}(\alpha SI - (\theta + \rho)E), \\
\frac{\alpha}{0} AB D^\sigma_u I(u) &= \eta u^{n-1}(\theta E - (\mu + \delta + \rho)I), \\
\frac{\alpha}{0} AB D^\sigma_u R(u) &= \eta u^{n-1}(qA + \delta I + vS - \rho R).
\end{align*}$$

For simplicity, we define

$$\begin{align*}
K(u, S, E, I, R) &= \eta u^{n-1}(1 - q)A - (\alpha I + \rho + v)S, \\
L(u, S, E, I, R) &= \eta u^{n-1}(\alpha SI - (\theta + \rho)E), \\
M(u, S, E, I, R) &= \eta u^{n-1}(\theta E - (\mu + \delta + \rho)I), \\
N(u, S, E, I, R) &= \eta u^{n-1}(qA + \delta I + vS - \rho R).
\end{align*}$$

Then, we get

$$\begin{align*}
\frac{\alpha}{0} AB D^\sigma_u S(u) &= K(u, S, E, I, R), \\
\frac{\alpha}{0} AB D^\sigma_u E(u) &= L(u, S, E, I, R), \\
\frac{\alpha}{0} AB D^\sigma_u I(u) &= M(u, S, E, I, R), \\
\frac{\alpha}{0} AB D^\sigma_u R(u) &= N(u, S, E, I, R).
\end{align*}$$

Applying the AB integral yields,

$$\begin{align*}
S(u) &= S(0) - \frac{1 - \sigma}{\alpha/0} AB[\sigma] K(u, S, E, I, R) + \frac{\sigma}{\alpha/0} AB[\sigma] \int_0^u (u - p)^{\alpha - 1} K(p, S, E, I, R) dp, \\
E(u) &= E(0) - \frac{1 - \sigma}{\alpha/0} AB[\sigma] L(u, S, E, I, R) + \frac{\sigma}{\alpha/0} AB[\sigma] \int_0^u (u - p)^{\alpha - 1} L(p, S, E, I, R) dp, \\
I(u) &= I(0) - \frac{1 - \sigma}{\alpha/0} AB[\sigma] M(u, S, E, I, R) + \frac{\sigma}{\alpha/0} AB[\sigma] \int_0^u (u - p)^{\alpha - 1} M(p, S, E, I, R) dp, \\
R(u) &= R(0) - \frac{1 - \sigma}{\alpha/0} AB[\sigma] N(u, S, E, I, R) + \frac{\sigma}{\alpha/0} AB[\sigma] \int_0^u (u - p)^{\alpha - 1} N(p, S, E, I, R) dp.
\end{align*}$$
We discretize these equations at $u_{m+1}$ as:

\[
S^{m+1} = S^0 + \frac{1 - \sigma}{AB(\sigma)} \sum_{i=0}^{m} \left[ \frac{h^\sigma K(u_{i-1}, S^m, E^m, R^m)}{\Gamma(\sigma + 2)} \right] \left[ (m + 1 - i)^\sigma (m - i + 2 + \sigma) - (m - i)^\sigma (m - i + 2 + 2\sigma) \right] \\
+ \frac{\sigma}{AB(\sigma) \Gamma(\sigma)} \int_{0}^{u_{m+1}} (u_{m+1} - p)^{\sigma-1} K(p, S, E, R) dp,
\]

\[
E^{m+1} = E^0 + \frac{1 - \sigma}{AB(\sigma)} \sum_{i=0}^{m} \left[ \frac{h^\sigma L(u_{i-1}, S^m, E^m, R^m)}{\Gamma(\sigma + 2)} \right] \left[ (m + 1 - i)^\sigma (m - i + 2 + \sigma) - (m - i)^\sigma (m - i + 2 + 2\sigma) \right] \\
+ \frac{\sigma}{AB(\sigma) \Gamma(\sigma)} \int_{0}^{u_{m+1}} (u_{m+1} - p)^{\sigma-1} L(p, S, E, R) dp,
\]

\[
I^{m+1} = I^0 + \frac{1 - \sigma}{AB(\sigma)} \sum_{i=0}^{m} \left[ \frac{h^\sigma M(u_{i-1}, S^m, E^m, R^m)}{\Gamma(\sigma + 2)} \right] \left[ (m + 1 - i)^\sigma (m - i + 2 + \sigma) - (m - i)^\sigma (m - i + 2 + 2\sigma) \right] \\
+ \frac{\sigma}{AB(\sigma) \Gamma(\sigma)} \int_{0}^{u_{m+1}} (u_{m+1} - p)^{\sigma-1} M(p, S, E, R) dp.
\]

Then, we obtain

\[
S^{m+1} = S^0 + \frac{1 - \sigma}{AB(\sigma)} \sum_{i=0}^{m} \left[ \frac{h^\sigma K(u_{i-1}, S^m, E^m, R^m)}{\Gamma(\sigma + 2)} \right] \left[ (m + 1 - i)^\sigma (m - i + 2 + \sigma) - (m - i)^\sigma (m - i + 2 + 2\sigma) \right] \\
+ \frac{\sigma}{AB(\sigma) \Gamma(\sigma)} \int_{0}^{u_{m+1}} (u_{m+1} - p)^{\sigma-1} K(p, S, E, R) dp,
\]

\[
E^{m+1} = E^0 + \frac{1 - \sigma}{AB(\sigma)} \sum_{i=0}^{m} \left[ \frac{h^\sigma L(u_{i-1}, S^m, E^m, R^m)}{\Gamma(\sigma + 2)} \right] \left[ (m + 1 - i)^\sigma (m - i + 2 + \sigma) - (m - i)^\sigma (m - i + 2 + 2\sigma) \right] \\
+ \frac{\sigma}{AB(\sigma) \Gamma(\sigma)} \int_{0}^{u_{m+1}} (u_{m+1} - p)^{\sigma-1} L(p, S, E, R) dp,
\]

\[
I^{m+1} = I^0 + \frac{1 - \sigma}{AB(\sigma)} \sum_{i=0}^{m} \left[ \frac{h^\sigma M(u_{i-1}, S^m, E^m, R^m)}{\Gamma(\sigma + 2)} \right] \left[ (m + 1 - i)^\sigma (m - i + 2 + \sigma) - (m - i)^\sigma (m - i + 2 + 2\sigma) \right] \\
+ \frac{\sigma}{AB(\sigma) \Gamma(\sigma)} \int_{0}^{u_{m+1}} (u_{m+1} - p)^{\sigma-1} M(p, S, E, R) dp.
\]
Table 1: Values of the parameters used for numerical simulations.

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<th>Values</th>
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$$R^m+1\sigma = R^0 + \frac{1 - \sigma}{AB(\sigma)}N(u_{m+1}, S^m, E^m, I^m, \lambda^m) + \frac{\sigma}{AB(\sigma)} \sum_{i=0}^{m} \left[ h^\sigma N(u_i, S^m, E^m, I^m, \lambda^m) \frac{\Gamma(\sigma+2)}{\Gamma(\sigma+2)} \right] \left[ (m+1-i)^\sigma (m-i+2+\sigma) 
- (m-i)^\sigma (m-i+2+2\sigma) \right]$$

by the method using [32]. Let us now list the values of the parameters utilized in numerical simulations in the table below. Next, we will give some numerical simulations by Figures 1-3. We used initial conditions as: $S(0) = 37.538; E(0) = 13.923; I(0) = 23.191; R(0) = 13.213$. We used the values of the parameters as given in Table 1. In Figure 1, we consider the fractal dimension $\eta = 1$. We show the simulations for different values of fractional order $\sigma$, as follows. Figure 2, we consider the fractal dimension $\eta = 0.9$. In Figure 3, we consider the fractal dimension $\eta = 0.8$. Studying all these figures, we can remark the effects of the fractional order and the fractal dimension.
Fig. 1: Numerical simulations for different values of fractional order and fractal dimension $\eta = 1$.

Fig. 2: Numerical simulations for different values of fractional order and fractal dimension $\eta = 0.9$. 
Conclusions

In this work, fractal-fractional modeling was used to account for the Covid-19 disease model. We used a very effective technique to the model to get new results. In this study, we also examined mathematical properties such the presence and classification of the model’s equilibria. We also discussed stability analysis, and to show the usefulness of the method, we presented some numerical simulations for different values of the fractal dimension.

References


