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Numerical simulation of a Caputo fractional 
epidemic model for the novel coronavirus with the 
impact of environmental transmission

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Abstract The coronavirus infectious disease (COVID-19) is a novel respiratory disease reported in 2019 in China. The infection is very destructive to human lives and caused millions of deaths. Various approaches have been made recently to understand the complex dynamics of COVID-19. The mathematical modeling approach is one of the considerable tools to study the disease spreading pattern. In this article, we develop a fractional order epidemic model for COVID-19 in the sense of Caputo operator. The model is based on the effective contacts among the population and environmental impact to analyze the disease dynamics. The fractional models are comparatively better in understanding the disease outbreak and providing deeper insights into the infectious disease dynamics. We first consider the classical integer model studied in recent literature and then we generalize it by introducing the Caputo fractional derivative. Furthermore, we explore some fundamental mathematical analysis of the fractional model, including the basic reproductive number $R_0$ and equilibrium stability utilizing the Routh–Hurwitz and the Lyapunov function approaches. Besides theoretical analysis, we also focused on the numerical solution. To simulate the model, we use the well-known generalized Adams–Bashforth Moulton Scheme. Finally, the influence of some of the model essential parameters on the dynamics of the disease is demonstrated graphically.

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1. Introduction

In December 2019, a severe respiratory illness was emerged among the people of Wuhan city, having 11 million human population located in central China. The infection was caused due to a novel coronavirus, which was found in a single
individual suffering from disease and identified in many others patients. Initially, it was noticed that the disease is a zoonosis, which means infectious disease transfer from animals to humans. The primary sources of the pandemic were observed in the Huanan Seafood Market and the Seafood wholesale market in Wuhan because 55 percent of the first 425 confirmed humans. The primary sources of the pandemic were observed which means infectious disease transfer from animals to patients. Initially, it was noticed that the disease is a zoonosis, individual suffering from disease and identified in many others.

The uncertain origin of the infection imposed challenges to disease control. Zhou et al. [2] observed that wild animals such as bats, civets and minks contribute to spreading the infection. On the other hand, the clinical observation indicated that the incubation period of this disease is from 2 to 14 days. There may be no symptoms in this incubation period in the infected individual. Such patients who are not familiar with their infection are also responsible for transmitting the disease [3]. Moreover, the available vaccines are in experimental stage for this virus as it is the new one. To control the disease, the only way in the current situation is the strict implementation of non-pharmaceutical interventions.

Many researchers studied different mathematical models based on the possible COVID-19 transmissions. The models were mostly constructed with the help of integer and fractional order derivatives. A basic epidemic model describing the dynamics of COVID-19 is studied by Wu et al. [4]. An epidemic model investigating the role of environmental transmission on the infection incidence is studied in [5]. The fractional COVID-19 epidemic models based on fuzzy set theory are developed in [6,7]. Beigi et al. [8] studied an extended version of SEIR mathematical model by incorporating a vaccine compartment in order to analyze the consequences of vaccine on the disease incidence. Ahmad et al. [9] investigated the SEIR time-fractional model using Atangana-Baleanu operator to model the transmission of Coronavirus in Pakistan. Moreover, a new fractional model with Caputo and Atangana-Baleanu-Caputo operators describing the disease dynamics was analyzed by Naik et al. [10]. To demonstrate the long term-time prediction of COVID-19 outbreak, two fractional operators based on non-singular kernels was considered in [11]. The dynamical role of quarantine and latency period on the disease incidence is studied in [12]. Mathematical analysis of an epidemic model, which describes the outbreak of COVID-19 infection diseases is carried out in [13]. The authors developed a new technique named as $q$-Homotopy analysis transform method for the solution of the proposed system of nonlinear differential equations. Rezapour et al. [14] formulated an epidemic model for the transmission of COVID-19 by introducing Caputo fractional differential operator. The proposed model is treated numerically with a fractional Euler scheme. Based on real data, they provide the numerical simulation to predict the transmission of COVID-19 in Iran and in the world.

Kumar et al. [15] studies a fractional epidemic model for the dynamics of corona-virus outbreak. The authors treated the model numerically with both Adams type predictor-corrector and Hermite wavelets schemes. An epidemic model for the dynamics of COVID-19 infectious disease was proposed by Ali et al. [16] as well. For approximate solution, the Laplace Adomian decomposition technique was used. Furthermore, they discuss the local and global asymptomatic stability. Farman et al. [17] studied the fractional SEIR epidemic model for measles disease. The Laplace Adomian Decomposition method was used to obtain an approximate solution. Kumar et al. [18] provided the numerical solution of the SIR system of differential equation using the Bernstein Wavelet. Finally, they compared it with the Adams–Bashforth predictor–corrector scheme in [19]. Ali et al. [20] discussed the dynamic of the COVID-19 model of fractional order and investigate the stability analysis using the Lyapunov function. A fractional-order model for COVID-19 model is suggest by Oud et al. [21], considering the impact of quarantine, isolation and environment. The motive of the conducted study is to explore the spreading pattern of the pandemic and spot the consequences of quarantine and isolation on the dynamics. Abudo et al. [22] focused on the numerical solution of a nonlinear system of fractional differential equations that describe the dynamics of destructive Coronavirus. The numerical procedure used for the solution is the fractional Adams–Bashforth method. Abdulwassaa et al. [23] developed a fractional model for novel coronavirus. The study’s purpose was to identify hereafter in the behaviour of the confirmed infected cases. The fractional model describing the epidemic of rotavirus in the Atangana-Baleanu sense is proposed by Ahmad et al. [24]. For analysis, the Adams–Bashforth procedure for the fractional case was utilized. They also provided the Ulam-Hyers stability for the fractional system. Shah et al. [25] formulated the mathematical model of COVID-19 for the qualitative analysis. The model was based on infectious and healthy individuals. They proved the uniqueness and existence of the proposed model. Moreover, the Ulam types stability was presented by using nonlinear analysis. Some other recent relevant literature can be found in [26–28].

This study presents the dynamics of novel COVID-19 via a fractional modeling approach. The phenomena are modeled in the form of the system of fractional-order differential equation using Caputo fractional derivative. Parallel with the numerical simulation, detailed theoretical analysis is accomplished of the fractional case. Finally, we provide the numerical simulation of the problem using the Generalized Adams–Bashforth Moulton method and sketch out the impact of some critical parameters of the proposed model on the dynamics of Coronavirus. The rest of the paper is composed as follows. The basic concepts of the fractional derivative are presented in Section 2. Section 3 depicts the mathematical formulation of the COVID-19 epidemic model of fractional order. The detailed theoretical analysis is illustrated in Section 4, and includes the existence, uniqueness and stability analysis. In Section 5 we discuss the impact of some important model parameters on the basic reproductive number. Section 6 describes the numerical procedure to obtain the approximate solution of the model and provides some profound insights about the effect of various model parameters with the help of graphs. In Section 7, we conclude the work with some helpful suggestions about the epidemic of Coronavirus.
Numerical simulation of a Caputo fractional epidemic model for the novel coronavirus with the impact

2. Preliminaries

Definition 2.1. Let \( f(\tau) \in C^k \), then for the fractional derivative with order \( \alpha \), in the Caputo sense is defined in [29] as,

\[
C^\alpha D^k f(\tau) = \frac{1}{\Gamma(k - \alpha)} \int_0^\tau (\tau - \eta)^{k-\alpha} f^{(k)}(\eta) \, d\eta,
\]

where \( k = \lfloor \alpha \rfloor + 1 \) with \( \lfloor \alpha \rfloor \) is the integer part of real number \( \alpha \). Evidently, \( C^\alpha D^k f(\tau) \rightarrow f(\tau) \) as \( \alpha \rightarrow 1 \).

Definition 2.2. For a function \( f : R^+ \rightarrow R \), the fractional integral of order \( \alpha > 0 \) is stated as,

\[
F(f(\tau)) = \frac{1}{\Gamma(k - \alpha)} \int_0^\tau (\tau - \eta)^{k-\alpha} f(\eta) \, d\eta,
\]

where \( \tau > 0 \) and \( 0 < \alpha < 1 \).

Definition 2.3. [30] A constant point \( u^* \) is said to be the equilibrium point of the Caputo system

\[
C^\alpha D^\alpha u(\tau) = F(\tau, u(\tau)), \quad \alpha \in (0, 1),
\]

if and only if \( F(\tau, u^*) = 0 \).

3. Mathematical formulation

We consider that the pandemic of coronavirus among the human population is categorized as: the population that is at risk of getting infected is termed as susceptible \( (S) \); those who have no symptoms yet but can infect others are termed as exposed \( (E) \); those having fully developed disease symptoms are placed in the infected category \( (I) \); and those who have recovered from the disease are placed in the recovered population \( (R) \). Finally, the symbol \( C_i \) is used to describe the viral load in the environment. The classical, or integer order, model for the transmission of the COVID-19 studied in [5] can be stated as follows:

\[
\begin{align*}
\frac{dS}{dt} &= -\beta SE - \beta_1 SI - \beta_{C,0} SC_i - \mu S, \\
\frac{dE}{dt} &= \beta SE + \beta_1 SI + \beta_{C,0} SC_i - (\beta + \mu)E, \\
\frac{dI}{dt} &= \beta E - (\omega + \gamma + \mu)I, \\
\frac{dR}{dt} &= \gamma I - \mu R, \\
\frac{dC_i}{dt} &= \zeta_1 E + \zeta_2 I - \sigma C_i,
\end{align*}
\]

subjected to the initial conditions: \( S(0) = S_0 \geq 0, E(0) = E_0 \geq 0, I(0) = I_0 \geq 0, R(0) = R_0 \geq 0, C_i(0) = C_{i0} \geq 0 \).

The parameter \( \Delta \) denotes the recruitment rate, and \( \mu \) is the human host population natural death rate. \( \beta^{-1} \) is the period of incubation, \( \omega \) shows the death rate due to infection, and \( \gamma \) is the disease recovery rate. \( \zeta_1 \) and \( \zeta_2 \) denote the contributing rate to virus reservoir in the environment due exposed and infected population respectively. The parameter \( \sigma \) is the rate of removal of virus from the environment or surfaces. On the other hand, \( \beta_{E} \) is the rate of transmission between the susceptible and exposed population, while the \( \beta_1 \) is the transmission rate between the susceptible and infected population and finally, \( \beta_{C,0} \) is the rate of transmission of infection from the environment to human.

3.1. Fractional extension of the model

Most natural phenomena including epidemiological dynamics involve time memory effect and are valuable to demonstrate the facts about nature related processes having non-local dynamics. Models with fractional derivatives handle these issues in better way because non-integral order derivatives contain time-dependent kernels. Many fractional derivatives can be found in the literature but the most common is Caputo fractional derivative. The key advantage of using Caputo fractional derivative is it takes the same form of initial conditions as in the case of classical derivatives, which means it does not require the fractional initial values. Motivated by these useful facts we reformulate COVID-19 model (1) in fractional form by adopting Caputo fractional time derivative. Now By introducing the time-dependent kernel we define the power-law correlation in the following

\[
\kappa(t - \tau) = \frac{1}{\Gamma(\alpha - 1)}(t - \tau)^{\alpha - 2},
\]

and we can express system (1) in terms of the integral as:

\[
\begin{align*}
\frac{dS}{dt} &= \int_0^t \kappa(t - \tau) \left[ A - \beta SE - \beta_1 SI - \beta_{C,0} SC_i - \mu S \right] \, d\tau,
\frac{dE}{dt} &= \int_0^t \kappa(t - \tau) \left[ \beta SE + \beta_1 SI + \beta_{C,0} SC_i - (\beta + \mu)E \right] \, d\tau,
\frac{dI}{dt} &= \int_0^t \kappa(t - \tau) \left[ \beta E - (\omega + \gamma + \mu)I \right] \, d\tau,
\frac{dR}{dt} &= \int_0^t \kappa(t - \tau) \left[ \gamma I - \mu R \right] \, d\tau,
\frac{dC_i}{dt} &= \int_0^t \kappa(t - \tau) \left[ \zeta_1 E + \zeta_2 I - \sigma C_i \right] \, d\tau.
\end{align*}
\]

Next, apply the Caputo type derivative having order \( \alpha - 1 \) and substituting (2) in (5) yields to,

\[
\begin{align*}
C^{\alpha-1} \frac{dS}{dt} &= C^{\alpha-1} I^{(\alpha-1)} \left[ A - \beta SE - \beta_1 SI - \beta_{C,0} SC_i - \mu S \right], \\
C^{\alpha-1} \frac{dE}{dt} &= C^{\alpha-1} I^{(\alpha-1)} \left[ \beta SE + \beta_1 SI + \beta_{C,0} SC_i - (\beta + \mu)E \right], \\
C^{\alpha-1} \frac{dI}{dt} &= C^{\alpha-1} I^{(\alpha-1)} \left[ \beta E - (\omega + \gamma + \mu)I \right], \\
C^{\alpha-1} \frac{dR}{dt} &= C^{\alpha-1} I^{(\alpha-1)} \left[ \gamma I - \mu R \right], \\
C^{\alpha-1} \frac{dC_i}{dt} &= C^{\alpha-1} I^{(\alpha-1)} \left[ \zeta_1 E + \zeta_2 I - \sigma C_i \right].
\end{align*}
\]

The operators \( C^{\alpha-1} I^{(\alpha-1)} \) and \( I^{(\alpha-1)} \) are inverse to each other and leads to,

\[
\begin{align*}
C^\alpha S &= A - \beta SE - \beta_1 SI - \beta_{C,0} SC_i - \mu S, \\
C^\alpha E &= \beta SE + \beta_1 SI + \beta_{C,0} SC_i - (\beta + \mu)E, \\
C^\alpha I &= \beta E - (\omega + \gamma + \mu)I, \\
C^\alpha R &= \gamma I - \mu R, \\
C^\alpha C_i &= \zeta_1 E + \zeta_2 I - \sigma C_i.
\end{align*}
\]
4. The analysis of fractional model

This section presents the theoretical analysis of the proposed fractional COVID-19 model (5). We provide the basic reproductive number $\mathcal{R}_0$ using next generation matrix techniques and present the stability of the disease-free and the endemic equilibrium points. Finally, for the local and global stability, we established suitable criteria. The details are presented in the subsequent sections. The disease free equilibrium (DFE) exist if there is no infection in the compartments i.e. $I = 0$. To evaluate the equilibrium point we substitute $D^*_F(t) = 0$, $D^*_I(t) = 0$, $D^*_R(t) = 0$, $D^*_C(t) = 0$. Thus we obtain

$$P_0 = (S_0, E_0, I_0, R_0, C_0) = \left( \frac{A}{\mu}, 0, 0, 0, 0 \right).$$

The next generation matrix techniques is used to compute the basic reproductive number $\mathcal{R}_0$ for the model given by (5). The new infection occurs in exposed compartment $E$ is $\beta_{EB}SE + \beta_{EB}SI + \beta_{EB}SC$, and there is no new infection in $I$ and $C$ compartment. On other hand, the transition between the compartment are $(\beta + \mu)E$, $-\beta E + \alpha_1 I$, and $-\xi_1 E - \xi_2 I + \sigma C$. Therefore, $F = \left[ \beta_{EB}SE + \beta_{EB}SI + \beta_{EB}SC \right]$, and $V = \left[ \beta + \mu \right]$.

The characteristic polynomial is $P(\xi) = (\xi + \mu)(\xi^3 + a_2\xi^2 + a_1\xi + a_0)$.

The endemic equilibrium point is locally asymptotically stable if $\mathcal{R}_0 < 1$. Moreover, it is to ensure that the quantity $a_1 a_2 - a_0$ is positive. Thus the DFE point is locally asymptotically stable.

4.2. Endemic equilibrium point

The endemic equilibrium $EE$ shown by $P_{EE} = (S_*, E_*, I_*, R_*, C_*)$ exists with the infection in the compartments i.e. $I \neq 0$, for evaluation substitute $D^*_F(t) = 0$, $D^*_I(t) = 0$, $D^*_R(t) = 0$, $D^*_C(t) = 0$ in (5) we have,

$$A - SL - \mu S = 0,$$

$$SL + (\beta + \mu) E = 0,$$

$$\beta E - (\alpha + \gamma + \mu) I = 0, \quad (10)$$

$$\gamma I - \mu R = 0,$$

$$\xi_1 E + \xi_2 I - \sigma C = 0,$$

where $\lambda = \beta_{EB}E + \beta R I + \beta_{C,0}C.$

$$\mathcal{R}_0 < 1 \text{ implies } a_0, a_1, a_2 \text{ are positive.}$$

Thus

$$\mathcal{R}_0 > 1 \implies \text{unstable for } \mathcal{R}_0 > 1.$$

4.1. Local stability

Theorem 4.1. The disease free equilibrium point is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable for $\mathcal{R}_0 > 1$.

Proof. The Jacobian of the system (5) at the DFE point $P_0 = (\frac{A}{\mu}, 0, 0, 0, 0)$ is,

$$J(P_0) = \begin{pmatrix}
-\mu & -\beta_{EB}S_0 & -\beta_{EB}S_0 & 0 & -\beta_{C,0}S_0 \\
0 & -\beta_{EB}S_0 - \beta - \mu & -\beta_{EB}S_0 & 0 & -\beta_{C,0}S_0 \\
0 & 0 & -\alpha_1 & 0 & 0 \\
0 & 0 & 0 & -\gamma & -\mu \\
\xi_1 & \xi_2 & 0 & 0 & -\sigma
\end{pmatrix}.$$

The characteristic polynomial is

$$P(\xi) = (\xi + \mu)(\xi^3 + a_2\xi^2 + a_1\xi + a_0),$$

where

$$a_2 = \sigma + \alpha + (\beta + \mu)(1 - a_1),$$

$$a_1 = \sigma(\beta + \mu)(1 - a_1) + \alpha(\beta + \mu)(1 - a_3) + \sigma o_1 + \xi_1 S_0(\xi_1 + \sigma C_0) - \omega S_0 R_0,$$

$$a_0 = \frac{\omega S_0 R_0 (\omega S_0 R_0 - \omega S_0 R_0 + \omega S_0 R_0) o_1 (\xi_1 + \sigma C_0)}{\omega S_0 R_0 (\omega S_0 R_0 - \omega S_0 R_0 + \omega S_0 R_0)}.$$
The endemic equilibrium point is locally asymptotically stable if $R_0 > 1$ and unstable for $R_0 < 1$.

**Proof.** The Jacobian of the system (5) at the endemic equilibrium point (EEP) is,

$$J(P_e) = \begin{pmatrix} -\mu - \beta_{EB} S_0 & -\beta_{RS} S_0 & 0 & -\beta_{C,0} S_0 \\ \beta_{EB} S_0 - \beta - \mu & \beta_{RS} S_0 & 0 & 0 \\ 0 & 0 & -\omega_1 & 0 \\ 0 & 0 & \gamma & -\mu \\ 0 & \xi_1 & \xi_2 & 0 & -\sigma \end{pmatrix}.$$  

The characteristic polynomial is

$$P(\lambda) = (\lambda + \mu)(\lambda^2 + \xi \lambda^2 + b_2 \lambda + b_1 \lambda + b_0),$$

where,

$$b_3 = \sigma \omega_1 + \frac{R_0}{S_0} + (\beta + \mu) \left(1 - \frac{R_1}{R_0}\right),$$

$$b_2 = (\beta + \mu + \omega_1) \left(\sigma - (\sigma + \mu) \frac{R_1}{R_0}\right) + \omega_1 (\beta + \mu) \left(1 - \frac{R_2}{R_0}\right) + \frac{R_0}{S_0} \beta_{C,0},$$

$$b_1 = \frac{R_0}{S_0} \left(\frac{c \beta_0 + (\sigma + \omega_1) \beta_{EB}}{\beta_{C,0}}\right),$$

$$b_0 = \mu \omega_1 \sigma (\beta + \mu) (R_0 - 1) > 0 \iff R_0 > 1.$$  

The positivity of the coefficients $b_0, b_1, b_2$ and $b_3$ is claim by $R_0 > 1$. This meets the Routh-Hurwitz stability condition $b_3 b_2 b_1 > b_1 b_2^2 + b_2 b_1 b_0$. Hence the endemic equilibrium point EE is locally asymptotically stable.

### 4.3. Global stability

To check the global asymptotical stability, of the disease free equilibria of the system represent by (5) we use the technique of the Lyapunov function. The subsequent theorem yield to the desired result.

**Theorem 4.3.** The DEF of the model (5) is global asymptotically stable if $R_0 < 1$.

**Proof.** Assume the Lyapunov function of the form

$$L(t) = g_1 E(t) + g_2 I(t) + g_3 V(t).$$

(14)

The unknown coefficients, $g_i > 0$ for $i = 1, 2, 3$ are constants and need to be determined. The utilization of Caputo derivative of $L(t)$ leads to:

$$D_t^\alpha L(t) = g_1 D_t^\alpha E(t) + g_2 D_t^\alpha I(t) + g_3 D_t^\alpha V(t).$$

$$= g_1 S_0 \beta_{EB} E + \beta_{RS} I + \beta_{C,0} C_i - (\beta + \mu) E + g_2 (\beta E - (\omega + \gamma + \mu) I) + g_3 (\xi_1 E + \xi_2 I - \sigma C_i).$$

As $S \leq S_0$ then

$$D_t^\alpha L(t) \leq g_1 S_0 \beta_{EB} E + \beta_{RS} I + \beta_{C,0} C_i - (\beta + \mu) E + g_2 (\beta E - (\omega + \gamma + \mu) I) + g_3 (\xi_1 E + \xi_2 I - \sigma C_i).$$

by choosing $g_1 = \sigma$, $g_2 = \frac{\sigma \beta_{EB} + \xi \beta_{RS} + \xi \beta_{C,0}}{\beta}$ and $g_3 = S_0 \beta_{C,0}$ we have $D_t^\alpha L(t) \leq \sigma (\beta + \mu) (R_0 - 1) E$.

Therefore $D_t^\alpha L(t) \leq 0$ as $R_0 \leq 1$ and $D_t^\alpha L(t) = 0 \iff E = I = C_i = 0$. It follows from System (5) that $R \to 0$ and $S \to \frac{C}{S}$. Hence $(S, E, I, R, C_i) \to \left(\frac{C}{S}, 0, 0, 0, 0\right)$ whereas $t \to \infty$. Thus, utilizing the Lyapunov stability facts, developed in [30] for the fractional case, the solution of the fractional COVID-19 model (5) tends to $P_0$ as $t \to \infty$. Hence, it follows that the DEF of the model (5) is globally asymptotically stable.

### 5. Interpretation of $R_0$ versus model parameters

This section presents the impact of some necessary parameters of COVID-19 model (5) on the basic reproductive number $R_0$ with the associated contour plots. Fig. 1 presents the variation of $R_0$ with respect to the effective contact rates $\beta_{EB}$ and $\beta_{RS}$. It is observed that $R_0$ has a smaller value with a decrease in both $\beta_{EB}$ and $\beta_{RS}$ than 1. Likewise, $R_0$ is reduced to a value less than 1 with a decrease in the exposed individuals contact rate $\beta_{EB}$ and the environmental transmission rate $\beta_{C,0}$. This effect is
shown in Fig. 2. To reduce the secondary infection number, we need to restrict $\beta_{E0}$, because the asymptomatic individuals invisibly contributed to the infection due to the long incubation duration of the disease. Fig. 3 illustrates the impact of

![Fig. 2](image1)
Fig. 2 The influence of transmission rate due to exposed class $\beta_{E0}$ and environmental transmission rate $\beta_{E_C0}$ on $R_0$.

![Fig. 3](image2)
Fig. 3 The influence of exposed transmission rate $\beta_{E0}$ and rate of contribution to Coronavirus $\zeta_1$ on $R_0$.

![Fig. 4](image3)
Fig. 4 Influence of exposed transmission rate $\beta_{E0}$ and natural death rate $\mu$ on $R_0$. 

![Fig. 5](image4)
exposed individuals contact $\beta_{E0}$ and their contribution rate $\zeta_1$ to the virus reservoir. It is observed that $R_0$ increases linearly. The effect of potential contact $\beta_{E0}$ and the natural death rate $\mu$, on the basic reproductive number $R_0$ is depicted in Fig. 4. It shows that $R_0$ increases with an increase in $\beta_{E0}$ and decreases significantly with increase in $\mu$. In Conclusion, the greater the effective contact, the greater will be $R_0$ and having a reverse effect with $\mu$.

6. Numerical method and simulation

6.1. Numerical scheme

This section deals with the numerical solution of the Caputo fractional order COVID-19 epidemic model (5). To find the approximate solution we utilized the generalized Adams– Bashforth-Moulton method. This scheme is briefly described as follows:

Initially, we summarized the problem in the following nonlinear differential equation of fractional order

$$D_t^\alpha y(t) = f(t, y(t)), \text{ for the case } \tau \in [0, T],$$

$$y^{(k)}(0) = y^{(k)}_0, \text{ whenever } k = 0, \ldots, m - 1 \text{ and } m = [\alpha].$$

Eq. (16) can be described in Volter integral form as

$$y(t) = \sum_{k=0}^{n-1} \int_0^t \frac{h^k}{\Gamma(k+1)} \left[ \int_0^\tau (\tau - \eta)^{k-1} f(\eta, y(\eta)) d\eta \right] d\tau + \sum_{k=n}^{m} \frac{h^k}{\Gamma(k+1)} \left[ \int_0^\tau (\tau - \eta)^{k-1} f(\eta, y(\eta)) d\eta \right] d\tau,$$

Next, by putting $h = \frac{T}{N}$, $y_n = nh$, where we have $n = 0, \ldots, N \in Z^+$. In a result of mentioned setting, Eq. (17) leads to the following iterative formulae

$$y(t_{n+1}) = \sum_{k=0}^{n-1} \frac{h^k}{\Gamma(k+1)} \left[ \int_0^\tau \left( \frac{h}{\Gamma(\alpha+1)} \sum_{j=0}^{n-1} f(y(t_j)) + \frac{h^k}{\Gamma(k+1)} \sum_{j=n}^{m} f(y(t_j)) \right) d\tau \right],$$

$$y(t_{n+1}) = \sum_{k=0}^{m} \frac{h^k}{\Gamma(k+1)} \left[ \int_0^\tau (\tau - \eta)^{k-1} f(\eta, y(\eta)) d\eta \right],$$

and the predicted value $y^p(t_{n+1})$ is given by,

$$y^p(t_{n+1}) = \sum_{k=0}^{m} \frac{h^k}{\Gamma(k+1)} \left[ \int_0^\tau (\tau - \eta)^{k-1} f(\eta, y(\eta)) d\eta \right],$$

where,

$$a_{j,n+1} = \begin{cases} h^{n+1} \frac{h}{\Gamma(\alpha+1)} \sum_{j=0}^{n-1} f(y(t_j)), & \text{if } j = 0 \\ \frac{(n-j)^{n+1}}{\Gamma(n+1)} + \frac{(n-j+2)^{n+1}}{\Gamma(n+1)} - 2 \times \frac{(n-j+1)^{n+1}}{\Gamma(n+1)}, & \text{if } 0 \leq j \leq n \\ 1, & \text{if } j = n+1 \end{cases},$$

and

$$b_{j,n+1} = \frac{(n-j+1)^{n+1} - (n-j)^{n+1}}{\Gamma(n+1)} \text{ if } 0 \leq j \leq n.$$ 

By applying the above stated scheme to the system of FDEs given by (4), its discretized form is,

\begin{align*}
S(t_{n+1}) &= S(t_n) + \frac{h^\alpha}{\Gamma(\alpha+1)} \left[ \left( A - \beta_{E0} SE - \beta_{0} SI - \beta_{C0} SC - \mu S \right) \right] \\
&\quad + \sum_{j=0}^{n} a_{j,n+1} \left[ \left( A - \beta_{E0} SE - \beta_{0} SI - \beta_{C0} SC - \mu S \right) \right] \\
E(t_{n+1}) &= E(t_n) + \frac{h^\alpha}{\Gamma(\alpha+1)} \left[ \left( \beta_{E0} SE + \beta_{0} SI + \beta_{C0} SC - (\beta + \mu) E \right) \right] \\
&\quad + \sum_{j=0}^{n} a_{j,n+1} \left[ \left( \beta_{E0} SE + \beta_{0} SI + \beta_{C0} SC - (\beta + \mu) E \right) \right] \\
I(t_{n+1}) &= I(t_n) + \frac{h^\alpha}{\Gamma(\alpha+1)} \left[ \left( \beta_{E0} SE + \beta_{0} SI + \beta_{C0} SC - (\beta + \mu) E \right) \right] \\
&\quad + \sum_{j=0}^{n} a_{j,n+1} \left[ \left( \beta_{E0} SE + \beta_{0} SI + \beta_{C0} SC - (\beta + \mu) E \right) \right],
\end{align*}

where

where

$S'(t_{n+1}) = S(t_0) + \sum_{j=0}^{n} a_{j,n+1} \left[ \left( \beta_{E0} SE + \beta_{0} SI + \beta_{C0} SC - (\beta + \mu) E \right) \right]$

and

$E'(t_{n+1}) = E(t_0) + \sum_{j=0}^{n} a_{j,n+1} \left[ \left( \beta_{E0} SE + \beta_{0} SI + \beta_{C0} SC - (\beta + \mu) E \right) \right]$

and

$I'(t_{n+1}) = I(t_0) + \sum_{j=0}^{n} a_{j,n+1} \left[ \left( \beta_{E0} SE + \beta_{0} SI + \beta_{C0} SC - (\beta + \mu) E \right) \right]$

and

$R'(t_{n+1}) = R(t_0) + \sum_{j=0}^{n} a_{j,n+1} \left[ \left( \beta_{E0} SE + \beta_{0} SI + \beta_{C0} SC - (\beta + \mu) E \right) \right]$

and

$C'(t_{n+1}) = C(t_0) + \sum_{j=0}^{n} a_{j,n+1} \left[ \left( \beta_{E0} SE + \beta_{0} SI + \beta_{C0} SC - (\beta + \mu) E \right) \right]$

and

$\beta_{E0}$ Effective contact rate relative to exposed people

$\beta_{0}$ Effective contact rate relative infected individuals

$\beta_{C0}$ Transmission rate of infection from the environment

$\mu$ Natural death rate

$\alpha$ Incubation rate

$\omega$ Death rate of infection

$\gamma$ Recovery rate from infection

$\zeta_1$ Contribution rate of exposed individuals to Coronavirus

$\zeta_2$ Contribution rate of Infected individuals to Coronavirus

$\sigma$ Removal rate of Coronavirus from the environment


### Table 1: Parameters descriptions and their values in system (1).

| Parameters | Description | Values [per/day]\[5\] |
|------------|-------------|------------------|
| $A$        | Recruitment rate | 271.23 |
| $\beta_{E0}$ | Effective contact rate relative to exposed people | $3.11 \times 10^{-8}$ |
| $\beta_{0}$ | Effective contact rate relative infected individuals | $6.62 \times 10^{-8}$ |
| $\beta_{C0}$ | Transmission rate of infection from the environment | $1.03 \times 10^{-8}$ |
| $\mu$ | Natural death rate | $3.01 \times 10^{-5}$ |
| $\alpha$ | Incubation rate | $\frac{1}{\gamma}$ |
| $\omega$ | Death rate of infection | 0.01 |
| $\gamma$ | Recovery rate from infection | $\frac{1}{\eta}$ |
| $\zeta_1$ | Contribution rate of exposed individuals to Coronavirus | 2.30 |
| $\zeta_2$ | Contribution rate of Infected individuals to Coronavirus | 0 |
| $\sigma$ | Removal rate of Coronavirus from the environment | 1 |
6.2. Result and discussion

This section deals with the numerical simulation of Caputo fractional COVID-19 epidemic model (5), which is carried out for the initial reported information \( S(0) = 8998505, E(0) = 1000, I(0) = 475, R(0) = 10, V(0) = 10000 \). We have chosen the parameter values considered by [5] given in Table 1. The numerical simulation is carried out using generalized Adams–Bashforth Moulton scheme in Python. Initially, the results are plotted for different values of \( \alpha \) in order to observe the behavior of considered population groups based on their disease status. The details are presented in Figs. 5–7. It is noticed that the exposed and infected population increase to maximum and decreases exponentially. According to the biological point of view, this effect is due to the long incubation period of fully developed symptoms. On the other hand, the susceptible population decreases and a significant increase is observed in the recovered population.

In the subsequent section, we analyze the effects of the contact rates of exposed individuals \( \beta_{E_0} \), infected individuals \( \beta_I \), the transmission of infection from environment \( \beta_{E_0} \) and rate of contribution to the coronavirus reservoir \( \zeta_1 \). The impact of these parameters is studied graphically for symptomatic, asymptomatic and recovered individuals. The coronavirus environmental load is also depicted for the mentioned parameters.

Fig. 8 illustrates the influence of effective contact rates \( \beta_{E_0}, \beta_I \), environmental transmission rate \( \beta_{E_0} \) and rate of contribution to coronavirus reservoir \( \zeta_1 \) on the dynamics of exposed individuals. The reduction in the contact rates has a significant decreasing effect on the asymptomatic individuals. The variation of the peak of the curve is observed for 30%, 50% and 70% reduction in the mentioned interaction rates to its estimated baseline value given by [5]. The lowest peak is observed by decreasing the parameters up to 70%. It means that restricting these possible interventions among the

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Fig. 5  Graphical behavior of population in \( S \) and \( R \) compartments for different values of \( \alpha \).

Fig. 6  Graphical behavior of population in \( E \) and \( I \) compartments describing the impact of variation in \( \alpha \).
Fig. 7  The impact of variation in $\alpha$ on the viral load shown in (a) while (b) depicts the dynamics of COVID-19 model.

Fig. 8  The impact of variation in $\beta_E$, $\beta_S$, $\beta_{C,0}$ and $\zeta_1$ on the dynamics exposed individuals.
population will control the infection to some extent from spreading and helps in the elimination of the pandemic.

Fig. 9 interprets the infected individuals response to variation of different infection transmission parameters as mentioned earlier. The behavior is observed for different control measures to analyze its role in disease transmission and control. It is noticed that the infected population decrease by choosing smaller values of each parameter. We decrease 70\% effective contact rate i.e., $\beta_{E0}$ and $\beta_{I0}$ as well as the transmission rate via environment and rate of contribution to coronavirus, in the result the lowest peak is observed. It concludes that by reducing these control measures will possibly minimize or even eliminate the infection. (See Fig. 10).

Fig. 11 clarifies the behavior of coronavirus environmental viral load for different values of the infection transmission parameters and rate of contribution to virus reservoir. The role of this intervention, which possibly contributes to the virus reservoir is observed. A significant reduction is seen in the simulated pandemic curves for smaller values of each parameter. By decreasing the contribution rate to coronavirus $\zeta_1$ by 70\% (i.e. $\zeta_1 = 0.69$) to its estimated value, a faster reduction in the viral load is observed in comparison to other infection transmission parameters as can be seen in (d). It means that smaller the interaction between the population, will decrease the chances of enhancing the contribution rate to coronavirus and will lead to eliminate the disease.
7. Conclusion

In the present work, the generalized Adams–Bashforth Moulton procedure is utilized to solve the Caputo fractional order epidemic model for COVID-19 dynamics. The compartmental models with fractional case are comparatively useful to explore the disease spreading pattern and provide deeper insights into the infectious disease dynamics. We studied the COVID-19 outbreak dynamics using a fractional model with well-known Caputo derivative. The environmental transmission of the disease is taken into account in the proposed epidemic model. Some necessary mathematical analysis of the fractional model including the basic reproductive number $\mathcal{R}_0$, stability analysis of the model equilibria by using the Routh-Hurwitz criteria and the Lyapunov function approach are studied as well. It is noticed that the disease free equilibrium is locally as well as globally asymptotically stable if $\mathcal{R}_0 < 1$, while endemic equilibria is locally asymptotically stable if $\mathcal{R}_0 > 1$. Moreover, the impact of variation of model key parameters on basic reproductive number is depicted graphically. The parameters $\beta_{E,0}, \beta_{I,0}, \beta_{C,0}$ and $\zeta_1$ are found to sensitive and enhance the basic reproductive number $\mathcal{R}_0$ and it results in increasing the infection transmission. By restricting these control measures the infection transmission can be minimized in the population. Compared to the baseline value of each transmission parameter such as the contact rates of exposed individuals $\beta_{E,0}$, infected individuals $\beta_{I,0}$, the transmission of infection from environment $\beta_{C,0}$ and contribution to the corona-virus reservoir $\zeta_1$, we reduce the value up to 70%. It is noticed that reasonable reduction is seen in exposed and infected individuals. Thus, we conclude that by reducing the effective contact rates (by implementing social distancing policy) the infection can be controlled up to some extent. In future, this work can be extended to other fractional operators as well, because dynamics of the analyzed system depends on selected type of fractional operator.
Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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