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The impact of a power law-induced memory effect on the SARS-CoV-2 transmission

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A R T I C L E   I N F O

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A B S T R A C T

It is well established that COVID-19 incidence data follows some power law growth pattern. Therefore, it is natural to believe that the COVID-19 transmission process follows some power law. However, we found no existing model on COVID-19 with a power law effect only in the disease transmission process. Inevitably, it is not clear how this power law effect in disease transmission can influence multiple COVID-19 waves in a location. In this context, we developed a completely new COVID-19 model where a force of infection function in disease transmission follows some power law. Furthermore, different realistic epidemiological scenarios like imperfect social distancing among home-quarantined individuals, disease awareness, vaccination, treatment, and possible reinfection of the recovered population are also considered in the model. Applying some recent techniques, we showed that the proposed system converted to a COVID-19 model with fractional order disease transmission, where order of the fractional derivative ($\alpha$) in the force of infection function represents the memory effect in disease transmission. We studied some mathematical properties of this newly formulated model and determined the basic reproduction number ($R_0$). Furthermore, we estimated several epidemiological parameters of the newly developed fractional order model (including memory index $\alpha$) by fitting the model to the daily reported COVID-19 cases from Russia, South Africa, UK, and USA, respectively, for the time period March 01, 2020, till December 01, 2021. Variance-based Sobol’s global sensitivity analysis technique is used to measure the effect of different important model parameters (including $\alpha$) on the number of COVID-19 waves in a location ($W_C$). Our findings suggest that $\alpha$ along with the average transmission rate of the undetected (symptomatic and asymptomatic) cases in the community ($\beta_1$) are mainly influencing multiple COVID-19 waves in those four locations. Numerically, we identified the regions in the parameter space of $\alpha$ and $\beta_1$ for which multiple COVID-19 waves are occurring in those four locations. Furthermore, our findings suggested that increasing memory effect in disease transmission ($\alpha \rightarrow 0$) may decrease the possibility of multiple COVID-19 waves and as well as reduce the severity of disease transmission in those four locations. Based on all the results, we try to identify a few non-pharmaceutical control strategies that may reduce the risk of further SARS-CoV-2 waves in Russia, South Africa, UK, and USA, respectively.

1. Introduction

The novel Corona-virus known as COVID-19 caused by severe acute respiratory syndrome corona-virus 2 (SARS-CoV-2) has led to a severe loss of mankind worldwide and created an unprecedented challenge to public health, food security and world trade [1]. As of 16th January 2022, a total of 324 million confirmed cases and 5.5 million deaths notified worldwide [2]. Some of the most affected countries due to the current COVID-19 outbreak are the USA, UK, India, Brazil, Russia, etc. [2]. In all of these locations, more than one COVID-19 waves were observed [2] and almost in all of these countries, the later waves were found to be more severe (in terms of COVID-19 transmission) than the first wave [3–6]. Several factors includes the human social behavior, infection prevention policies, transmission variability, loss of natural immunity, vaccine efficacy, etc. may influence the number of COVID-19 waves in these locations [7]. However, among these factors isolating the key epidemiological parameter/parameters which mainly influencing number of epidemic waves in a location is a challenging task.

Recently, many studies suggest that COVID-19 incidence growth exhibits some power law [8–11]. This power law growth pattern in COVID-19 cases is due to the implementation of some containment measures like lockdown, social distancing, vaccination, etc. which drastically alter the
transmission dynamics of the outbreak [8–11]. Therefore, it is natural to believe that there is a significant relationship between the power law, and the COVID-19 transmission process [12–14]. Furthermore, many real life systems follow some power law which can describe an underlying regularity in the properties of a system [15,16]. Recently, several studies established that fractional derivatives and integrals are convolutions with a power law [17–19]. Using this approach few fractional order compartmental model with power law disease transmission is already developed to study the dynamics on different diseases [20–22]. However, in the context of COVID-19, we found no mathematical model with the effect of power law only in disease transmission process. Thus it is utmost important to develop a mathematical model that should address gaps between the data and the current knowledge about the process of COVID-19 transmission.

Understanding the spread dynamics of COVID-19 and predicting the pandemic height, stabilization time of the ongoing peak, and the forthcoming wave of the epidemic are the main objectives behind modeling COVID-19 disease transmission. In this context several mathematical studies can be found in disease literature [23–32]. Sardar et al. [23] developed a compartmental model on COVID-19 to analyze the effect of lockdown in different Indian states. They found that lockdown will be effective in those locations where higher percentage of symptomatic COVID-19 infection is found in the population. Sardar and Rana [24] constructed a new COVID-19 to analyze the risk of disease transmission occurring from hospitals and quarantined centers. Analyzing data from six states and overall India, the authors argued that a large outbreak may trigger by the hospital based transmission. Özkoş et al. [25] developed a fractional order model on COVID-19 to access the dynamics of Omicron variant and its relationship with heart attack in COVID-19 positive patients. Analyzing real COVID-19 data from the United Kingdom, the authors found that the rate of heart attack has an increasing relationship with the Omicron cases in the community. Recently, Özkoş and Yavuz [26] developed a fractional order co-infection model to study the effect of COVID-19 on diabetes patients in Turkey. By fitting daily COVID-19 cases from this country, the authors estimate the basic reproduction number (\(R_0\)) and perform its sensitivity analysis in terms of memory effect (order of the fractional order derivative). Authors also studied some dynamical properties of this newly formulated fractional order model. Ikrım et al. [27] restructured a SIVR (susceptible, Infected, Vaccinated, and Recovered) stochastic epidemic model to study the effect of white noise in COVID-19 dynamics. They derive the stochastic basic reproduction number (\(R_b\)), and studied some properties of this stochastic model. Finally, they showed that a sufficient large white noise can play a key role in the extinction of COVID-19. Recently, Naik et al. [28] proposed and analyzed a fractional order epidemic model on COVID-19 with two differential operators namely, the Caputo operator and the Atangana–Baleanu–Caputo operator. Using data from Pakistan, authors studied the impact of alternative drugs on COVID-19. They estimate the basic reproduction number and provide a long term forecast of the COVID-19 cases in Pakistan. Yavuz et al. [29] developed a new COVID-19 model to study the effect of vaccine campaign. They studied some dynamical properties of the model in terms of the basic reproduction number (\(R_b\)). Jentsch et al. [30] developed a coupled COVID-19 transmission model to determine how social and epidemiological dynamics interact with one another. They found that effective vaccination strategy that reduce the mortality depends on the time course of the pandemic in the population. Musa et al. [31] developed a mathematical model on COVID-19 to study the effect of awareness and different hospitalization scenarios. Analyzing COVID-19 data from Nigeria they found that increasing awareness on maintaining social distancing in a population along with different non-pharmaceutical interventions can effectively reduce the burden of the disease in the population. Moore et al. [32] constructed an age-structured model to study the effect of two dose COVID-19 vaccination. They found that relaxation in non-pharmaceutical interventions can have some serious negative effects in terms of controlling COVID-19 transmission.

All of these studies, authors used either integer order ODE systems [23,24,29–32] or they considered power law effect in the population (fractional rate of change of population) [25–28] to develop a model on COVID-19. It is well established that the dynamics of a population are mainly driven by the birth and death process, and these processes are mostly Markovian and do not follow a power law [33,34]. Therefore, these models may not be suitable to capture the power law effect in COVID-19 transmission [20,35–38]. To the best of our knowledge, there is no mathematical modeling study found in the literature that considered the power law effect only in COVID-19 transmission process. Consequently, it is unclear how this power law exponent (order of the R–L fractional derivative in the force of infection) in disease transmission can influence multiple COVID-19 waves in a location. Finding some suitable answer to these questions motivate us to undertake this work.

The main objective of this manuscript is to develop a new COVID-19 model with a power law effect on the disease transmission process. We use some recent techniques [20,21] to establish a relationship between the Riemann–Liouville fractional derivative that appeared in the force of infection function of the newly developed COVID-19 model to the power law age of infection function. In this new COVID-19 model, the R–L fractional derivative (represents the power law effect) appears in the force of infection function in disease transmission. Imperfect social distancing, awareness, vaccination, treatment, and possible re-infection scenario of the recovered cases are also considered in the model. The newly developed model is calibrated to the daily reported COVID-19 cases from Russia, South Africa (SA), United Kingdom (UK), and United States of America (USA) for the time period March 01, 2020, till December 01, 2021. We estimated several key epidemiological parameters of our model using the COVID-19 data obtained from the mentioned four locations. In terms of model fitting, we compare the newly formulated fractional order COVID-19 model with the corresponding integer order ODE model by determining Akaike weights [39] of the two models. Another aim of this work is to determine the parameter/parameters (including order of the R–L fractional derivative) that are most influential on the number of COVID-19 waves (\(W_C\)) in a location. To achieve this, we first develop an algorithm to determine the number of waves in a location using the newly formulated fractional order model. Finally using this algorithm, a global Sobol sensitivity analysis [40] is performed to determine the correlation between various model parameters with the number of COVID-19 waves (\(W_C\)) in a location.

The rest of the paper is arranged as follows in Section 2, we formulate a new COVID-19 mathematical model with general time dependent infection and showed that when the time varying age of infection function follows a power law the developed model can be converted to a COVID-19 model with fractional order transmission. Some basic mathematical properties of the newly developed fractional order model are derived in Section 3. Furthermore, in Section 3, we also provided different mathematical and statistical techniques used in this study. All results and their epidemiological justifications are discussed in Section 4. The paper ends with a brief conclusion (see Section 5) about the different findings of this paper. In Section 5, we also provided an outline of some possible non-pharmaceutical control strategies to reduce the risk of future COVID-19 waves in the four locations namely Russia, South Africa, UK, and USA, respectively.

2. Formulation of fractional order COVID-19 model

The fractional order COVID-19 model is developed based on the concept described in [20,21]. We subdivided the total human population at time \(t\), \(N(t)\), into six mutually exclusive and exhaustive sub-populations namely susceptible (\(S\)), exposed (\(E\)), home quarantined (\(L\)), undetected SARS-CoV-2 infected (\(U\)), confirmed SARS-CoV-2 infected (\(H\)), and recovered (\(R\)), respectively. Symptomatic and asymptomatic COVID-19 infected population in the community together are considered as the undetected compartment (\(U\)). It is already established that awareness campaigns
such as social media & TV advertisements, Govt. camps, etc. plays an important role in reducing the severity of COVID-19 transmission in the community [31,41,42]. Thus in addition to the human population, we also considered the awareness density compartment ($M$).

Following [20,21], we considered general time-dependent infectivity in the force of infection. Following [24], we assume that a small fraction $\rho$ of the susceptible population may contact the confirmed SARS-CoV-2 cases in hospitals and quarantined centers. Thus, a susceptible population ($S$) grows due to a constant recruitment rate $H$, a fraction $\theta_1$ of the recovered population who lose natural immunity at a rate $\omega_1$, and loss of awareness (social distancing behavior) at a rate $\omega$. This class decreased due to natural death at a rate $\mu$, new infection occurring from contacting either an undetected infected in the community ($U$) or a confirmed SARS-CoV-2 infected person ($H$), those susceptible who become aware of the infection at a rate $\psi$, those susceptible who are advised to stay home during lockdown at a rate $l$, and those susceptible who received vaccination at a rate $p$ with vaccine efficacy $\zeta$.

We assume that a fraction $\theta$ of the home-quarantined population ($L$) maintained proper social distancing measures issued by the government and policymakers. Thus, the remaining $(1-\theta)$ of the home-quarantined population are mixing with the community and therefore, may get an infection in contact with the community undetected cases ($U$). In general, the number of awareness campaign and the COVID-19 cases proportionally increases at the beginning of the epidemic. However, after the disease become endemic in the population, the density of the awareness campaign approach to a constant value. Hence, we considered a saturating type awareness response function $\psi \frac{M}{M+1} \frac{M}{M+1}$, where $\psi$ is the awareness response intensity of a susceptible population. We also assume that an individual in the home-quarantined compartment will eventually become less aware after a time period $(\frac{1}{\omega})$, and move again to the susceptible compartment. Furthermore, it is more likely that an infinitesimal percentage of the home quarantined population may be exposed to transmission occurring from confirmed cases ($H$). For simplicity, we assumed this percentage to be negligible. Therefore, the home-quarantined population increased due to the inflow of aware individuals (either from the influence of media or from the implementation of lockdown by the government) at rates $\psi$ and $l$ respectively, and a fraction $(1-\theta)$ of the recovered population who lose their natural immunity at a rate $\omega_2$. This population decreased due to natural death at a rate $\mu$, vaccination at a rate $p$, loss of awareness at a rate $\omega_2$ and due to contact with the community infected at a rate $\beta_1$.

The exposed population ($E$) increased due to the inflow of newly SARS-CoV-2 infected coming from susceptible ($S$) and home-quarantined ($L$) compartments, respectively. This population decreased due to natural death at a rate $\mu$ and those individuals who become infectious after the incubation period $(\frac{1}{\gamma})$.

Undetected infected population ($U$) increased due to the inflow of infectious coming from the exposed compartment at a rate $\gamma$. This population decreased due to the natural recovery at a rate $\gamma_1$, the natural death at a rate $\mu_2$, and those individuals who tested COVID-19 positive at a rate $\gamma_2$. Although, it is possible that the undetected cases can die due to SARS-CoV-2 infection in the community, however, those cases-related deaths in the community will be counted as confirmed SARS-CoV-2 deaths. Due to this reason, in our model, SARS-CoV-2-related deaths are considered only for the confirmed infected population ($H$) at a rate $\delta$.

The confirmed SARS-CoV-2 infected population ($H$) increased due to the inflow of undetected infected individuals who tested positive at a rate $\gamma_2$. We assume that a fraction $(\phi)$ of the total confirmed COVID-19 infected population required hospitalization and therefore, they received treatment with efficacy $\chi$ and the remaining fraction $(1-\phi)$ of the population does not require treatment, and therefore, they recover naturally at a rate $\gamma_3$. Thus, the confirmed COVID-19 population ($H$) decreased due to natural death at a rate $\mu$, natural & treatment-induced recovery, and SARS-CoV-2 related death at a rate $\delta$.

COVID-19 cured individuals from undetected ($U$) and confirmed ($H$) infected compartments moved to recovered class ($R$). This population also increases due to the inflow of vaccinated individuals from the susceptible and the home quarantined population. Numerous studies indicate the possibility of reinfection of the recovered COVID-19 cases [43,44] and therefore, we assumed a fraction $(\theta)$ of the recovered population ($R$) move to susceptible ($S$) compartment after the period $(\frac{1}{\omega_2})$ of natural immunity. The remaining fraction $(1-\theta)$ of the recovered population become more aware and moved to the home quarantined compartment after the period $(\frac{1}{\omega_2})$ of natural immunity.

Awareness density ($M$) increases in proportion to the number of confirmed COVID-19 cases ($H$) in the population. Due to the fixed budget of the media campaign, we assumed saturating type growth function $\frac{\phi M}{M+1}$, where $\eta$ is the awareness growth rate. We assume awareness density ($M$) degrades at a rate $d$.

Therefore, based on the above assumptions, we have the following system of differential equations that represent COVID-19 transmission dynamics with general time-dependent infectivity:

$$
\frac{dS(t)}{dt} = H + \omega L(t) - \psi S(t) \frac{M}{M+1} - \rho_p S(t) - \frac{\beta_1}{\theta} S(t) \Phi_1(t, 0) \left[ \int_0^t \frac{\kappa(t-i)U(i)}{\Phi_1(i, 0)} di \right] \\
- \frac{\beta_2 \rho}{N - \theta L} S(t) \Phi_2(t, 0) \left[ \int_0^t \frac{\kappa(t-i)H(i)}{\Phi_2(i, 0)} di \right] - (\mu + \lambda) S(t) + \omega_1 \psi L(t),
$$

$$
\frac{dL(t)}{dt} = \lambda S(t) + \psi S(t) \frac{M}{M+1} - \rho_p L(t) - \frac{\beta_1 (1-\theta)}{\theta} L(t) \Phi_1(t, 0) \left[ \int_0^t \frac{\kappa(t-i)U(i)}{\Phi_1(i, 0)} di \right] \\
- (\mu + \omega)L(t) + \omega_1 (1-\theta) R(t),
$$

$$
\frac{dE(t)}{dt} = \frac{\beta_1}{\theta} S(t) \Phi_1(t, 0) \left[ \int_0^t \frac{\kappa(t-i)U(i)}{\Phi_1(i, 0)} di \right] + \frac{\beta_2}{\theta} L(t) \Phi_1(t, 0) \left[ \int_0^t \frac{\kappa(t-i)H(i)}{\Phi_1(i, 0)} di \right] \\
+ \frac{\beta_1 (1-\theta)}{\theta} L(t) \Phi_1(t, 0) \left[ \int_0^t \frac{\kappa(t-i)U(i)}{\Phi_1(i, 0)} di \right] - (\sigma + \mu) E(t),
$$

$$
\frac{dU(t)}{dt} = \sigma E(t) - (\gamma_1 + \gamma_2 + \mu) U(t),
$$

$$
\frac{dH(t)}{dt} = \gamma_2 U(t) - [1 - \omega_2 \gamma_1 + \omega_2 \gamma_2] H(t) - (\mu + \delta) H(t),
$$

$$
\frac{dR(t)}{dt} = \gamma_1 U(t) + \rho_p (S(t) + L(t)) + [(1-\omega_2) \gamma_1 + \omega_2 \gamma_2] H(t) - (\mu + \omega_2) R(t),
$$

(2.1)
time-dependent infection function. The covid19 disease transmission model (2.1) can be converted to incorporate a power law form by assuming the disease spreading process carries certain information of its previous stage. Following [20–22], the functions \( \Phi_1(t, \dot{i}) \) and \( \Phi_2(t, \dot{i}) \) have the following form:

\[
\Phi_1(t, \dot{i}) = e^{-\left(\gamma_1 + \gamma_2 + \mu \alpha \dot{i} \dot{i} \right)}\]

\[
\Phi_2(t, \dot{i}) = e^{-\left[1^{1+\beta_1} + \gamma_2 x + 2 \mu \right] \alpha \dot{i} \dot{i}}\]

\[
2.1. \text{Derivation of a power law form COVID-19 transmission model}
\]

Many recent studies suggest that COVID-19 incidence data exhibit some power law [8–11]. Therefore, it is natural to assume that COVID-19 disease transmission process follows a power law [8–10, 10–12] i.e. disease spreading process carries certain information of its previous stage of interaction. The covid19 disease transmission model (2.1) can be converted to incorporate a power law form infection rate by assuming the time-dependent infection function \( \rho_i(t) \) in (2.2) as follows:

\[
\rho_i(t) = \frac{e^{\alpha t}}{T(a)}, \quad 0 < a < 1.
\]

Using Eq. (2.2) we have:

\[
L[k(t); s] = s^{1-a}.
\]

Applying the convolution theorem in Laplace transform, we have:

\[
\int_0^t \frac{k(t-\dot{i})U(\dot{i})}{\Phi_1(\dot{i}, 0)} d\dot{i} = L^{-1}\left[ s^{1-a} L\left( \frac{U(t)}{\Phi_1(t, 0)} \right) \right],
\]

\[
\int_0^t \frac{k(t-\dot{i})H(\dot{i})}{\Phi_2(\dot{i}, 0)} d\dot{i} = L^{-1}\left[ s^{1-a} L\left( \frac{H(t)}{\Phi_2(t, 0)} \right) \right].
\]

The Riemann–Liouville (R-L) fractional derivative of order \( n \), with \( 0 \leq n < 1 \), is defined as follows [45]:

\[
vD^\alpha f(t) = \frac{1}{\Gamma(1-n)} \frac{d}{dt} \int_0^t (t-\tau)^{n-a} f(\tau) d\tau.
\]

From Eq. (2.7), taking Laplace transform with respect to \( s \) and using some properties of Laplace transform [45], we have:

\[
L \left[ vD^\alpha f(t); s \right] = s^\alpha F(s) - v \theta^{1-a} f(0+).
\]

\[
(2.8)
\]

where, \( L \left[ f(t); s \right] = F(s) \).

Following [21], and using Eqs. (2.6) & (2.8), we have:

\[
vD_1^{1-a} \left[ \frac{U(t)}{\Phi_1(t, 0)} \right] = \int_0^t \frac{k(t-\dot{i})U(\dot{i})}{\Phi_1(\dot{i}, 0)} d\dot{i},
\]

\[
vD_1^{1-a} \left[ \frac{H(t)}{\Phi_2(t, 0)} \right] = \int_0^t \frac{k(t-\dot{i})H(\dot{i})}{\Phi_2(\dot{i}, 0)} d\dot{i},
\]

\[
vD_1^{1-a} \left[ \frac{U(t)}{\Phi_1(t, 0)} \right] = \int_0^t \frac{k(t-\dot{i})U(\dot{i})}{\Phi_1(\dot{i}, 0)} d\dot{i}.
\]

Thus using results in (2.9), the system (2.1), become:

\[
\frac{dM(t)}{dt} = \delta M(t) \left[ \frac{\eta H(t)}{1 + H(t)} - dM(t) \right]
\]

\[
\frac{dM(t)}{dt} = \beta_1 S(t) M(t) - \beta_2 \frac{S(t) M(t)}{1 + M(t)} - \beta_3 P S(t) - \frac{\beta_4}{N - \theta L} S(t) \Phi_1(t, 0) vD_1^{1-a} \left[ \frac{U(t)}{\Phi_1(t, 0)} \right]
\]

\[
\frac{dH(t)}{dt} = \beta_1 S(t) M(t) - \beta_2 \frac{S(t) M(t)}{1 + M(t)} - \beta_3 P S(t) - \frac{\beta_4}{N - \theta L} S(t) \Phi_1(t, 0) vD_1^{1-a} \left[ \frac{U(t)}{\Phi_1(t, 0)} \right]
\]

\[
\frac{dL(t)}{dt} = IS(t) + \psi S(t) M(t) - \beta_2 \frac{S(t) M(t)}{1 + M(t)} - \beta_3 P S(t) - \frac{\beta_4}{N - \theta L} S(t) \Phi_1(t, 0) vD_1^{1-a} \left[ \frac{U(t)}{\Phi_1(t, 0)} \right]
\]

\[
\frac{dE(t)}{dt} = \beta_1 S(t) M(t) - \beta_2 \frac{S(t) M(t)}{1 + M(t)} - \beta_3 P S(t) - \frac{\beta_4}{N - \theta L} S(t) \Phi_1(t, 0) vD_1^{1-a} \left[ \frac{U(t)}{\Phi_1(t, 0)} \right]
\]

\[
\frac{dH(t)}{dt} = \alpha E(t) - (\mu + \gamma_1 + \gamma_2) U(t),
\]

\[
\frac{dH(t)}{dt} = \gamma_2 U(t) - [1 - v)(\gamma_1 + \gamma_2) H(t) - (\mu + \delta) H(t),
\]

\[
\frac{dH(t)}{dt} = \gamma_2 U(t) - [1 - v)(\gamma_1 + \gamma_2) H(t) - (\mu + \delta) H(t),
\]

\[
\frac{dH(t)}{dt} = \gamma_2 U(t) - [1 - v)(\gamma_1 + \gamma_2) H(t) - (\mu + \delta) H(t),
\]
where, $\Phi_1(t,0)$ and $\Phi_2(t,0)$ are provided in Eq. (2.3).

Following [19,46,47], we defined the tempered fractional integral and derivative as follows:

$$
\mathcal{T}_0^\mu f(t) = \frac{1}{\Gamma(a)} \int_0^t (t-s)^{\alpha-1} e^{-\beta(t-s)} f(s) \, ds,
$$

$$
\mathcal{T}_0^{\mu,\beta} f(t) = \left( \frac{d}{dt} + \gamma \right)^{\alpha-1} \left[ \mathcal{T}_0^\mu f(t) \right],
$$

(2.11)

where, $n := [\text{Re}(\alpha)] + 1$ with $\text{Re}(\alpha) > 0$ and $\text{Re}(\beta) \geq 0$.

Following [46,47], the R-L fractional integral and derivative can be written in terms of the tempered fractional integral and derivative as follows:

$$
\mathcal{T}_0^{\mu,\beta} f(t) = e^{-\beta t} \mathcal{T}_0^{\mu} [e^{\beta t} f(t)],
$$

$$
\mathcal{T}_0^{\mu,\beta} f(t) = e^{-\beta t} \mathcal{T}_0^{\mu} [e^{\beta t} f(t)].
$$

(2.12)

Using the relation in (2.12), the system (2.10) becomes:

$$
dS(t) = \begin{array}{c}
H + \omega L(t) - \psi S(t) \left( \frac{1}{1 + M(t)} - \frac{\beta_1^0}{N - \theta L} S(t) \right) \mathcal{T}_0^{1} \left[ e^{\beta_1 t} \right] U(t) - \frac{\beta_1^0}{N - \theta L} S(t) \mathcal{T}_0^{1} \left[ e^{\beta_1 t} \right] H(t) \\
- \left( \mu + \lambda \right) S(t) + \omega_0 \theta R(t),
\end{array}
$$

$$
dL(t) = \begin{array}{c}
\psi S(t) \left( \frac{1}{1 + M(t)} - \frac{\beta_1^0}{N - \theta L} L(t) \right) \mathcal{T}_0^{1} \left[ e^{\beta_1 t} \right] U(t) - \frac{\beta_1^0}{N - \theta L} L(t) \mathcal{T}_0^{1} \left[ e^{\beta_1 t} \right] H(t) \\
- \left( \mu + \lambda \right) L(t) + \omega_1 (1 - \theta_1) R(t),
\end{array}
$$

$$
dE(t) = \begin{array}{c}
\frac{\beta_1^0}{N - \theta L} S(t) \mathcal{T}_0^{1} \left[ e^{\beta_1 t} \right] U(t) + \frac{\beta_1^0}{N - \theta L} L(t) \mathcal{T}_0^{1} \left[ e^{\beta_1 t} \right] H(t) \\
\frac{\beta_1^0}{N - \theta L} \left( \sigma + \mu \right) E(t),
\end{array}
$$

(2.13)

$$
dU(t) = \sigma E(t) - a_1 U(t),
$$

$$
dH(t) = \gamma_2 U(t) - [(1 - \nu) \gamma_3 + \nu \gamma_3] H(t) - (\mu + \delta) H(t),
$$

$$
dR(t) = \gamma_1 U(t) + p \xi (S(t) + L(t)) + [(1 - \nu) \gamma_3 + \nu \gamma_3] H(t) - (\mu + \omega_2) R(t),
$$

$$
dM(t) = \frac{\eta H(t)}{1 + H(t)} - d M(t),
$$

where, $a_1 = \gamma_1 + \gamma_2 + \mu$, and $a_2 = \mu + (1 - \nu) \gamma_3 + \nu \gamma_3 + \delta$.

Using the results in [46,47], we have:

$$
\mathcal{T}_0^{\mu,\beta} f(t) = \sum_{m=0}^{\infty} \frac{(-\beta t)^m}{m!} \int_0^t (t-s)^{\alpha-1} e^{-\beta(t-s)} f(s) \, ds,
$$

$$
\mathcal{T}_0^{\mu,\beta} f(t) = e^{-\beta t} \mathcal{T}_0^{\mu} [e^{\beta t} f(t)].
$$

(2.14)

Using the Theorem (2.8) in [46], the series in the right hand side of (2.14) is convergent. Therefore, we have the following relation:

$$
\mathcal{T}_0^{\mu,\beta} f(t) = \sum_{m=0}^{\infty} \frac{(-\beta t)^m}{m!} \int_0^t (t-s)^{\alpha-1} e^{-\beta(t-s)} f(s) \, ds,
$$

$$
= \int_0^t \sum_{m=0}^{\infty} \frac{(-\beta t)^m}{m!} (t-s)^{\alpha-1} e^{-\beta(t-s)} f(s) \, ds,
$$

$$
= \frac{1}{F(a-1)} \int_0^t e^{-\beta(t-s)} (t-s)^{\alpha-1} f(s) \, ds.
$$

(2.15)
Using the relation (2.15) Eq. (2.13) becomes,

\[
\begin{align*}
\frac{dS(t)}{dt} &= H + \omega L(t) - \psi S(t)M(t) - \beta S(t) - \frac{\beta \rho}{N - \theta L} S(t) \int_0^t e^{-\alpha(t-s)}(t-s)^{\nu-2}U(s) ds \\
&\quad - \frac{\beta \rho}{N - \theta L} S(t) \int_0^t e^{-\alpha(t-s)}(t-s)^{\nu-2}H(s) ds - (\mu + \lambda) S(t) + \omega \theta R(t), \\
\frac{dL(t)}{dt} &= L(t) + \psi S(t)M(t) - \beta L(t) - \frac{\beta \rho}{N - \theta L} L(t) \int_0^t e^{-\alpha(t-s)}(t-s)^{\nu-2}U(s) ds \\
&\quad - (\mu + \omega L(t) + \omega \theta R(t)), \\
\frac{dE(t)}{dt} &= \psi S(t)M(t) - \beta E(t) - \frac{\beta \rho}{N - \theta L} E(t) \int_0^t e^{-\alpha(t-s)}(t-s)^{\nu-2}U(s) ds \\
&\quad - (\mu + \alpha) E(t), \\
\frac{dR(t)}{dt} &= \psi S(t)M(t) - \beta R(t) - \frac{\beta \rho}{N - \theta L} R(t) \int_0^t e^{-\alpha(t-s)}(t-s)^{\nu-2}U(s) ds \\
&\quad - (\mu + \omega R(t)), \\
\frac{dM(t)}{dt} &= \frac{\delta M(t)}{\tau M(t)} - dM(t).
\end{align*}
\]

(2.16)

2.2. Corresponding integer order (ODE) COVID-19 model

If we choose \( \rho_1 = 1 \), then from Eq. (2.2), we have, \( \kappa(t) = \delta(t) \) (Dirac-delta), and Eq. (2.1) becomes:

\[
\begin{align*}
\frac{dS(t)}{dt} &= H + \omega L(t) - \psi S(t)M(t) - \beta S(t) - \frac{\beta \rho}{N - \theta L} S(t)U(t) - \frac{\beta \rho}{N - \theta L} S(t)H(t) \\
&\quad - (\mu + \lambda) S(t) + \omega \theta R(t), \\
\frac{dL(t)}{dt} &= L(t) + \psi S(t)M(t) - \beta L(t) - \frac{\beta \rho}{N - \theta L} L(t)U(t) - (\mu + \omega L(t) + \omega \theta R(t)), \\
\frac{dE(t)}{dt} &= \beta S(t)U(t) + \frac{\beta \rho}{N - \theta L} E(t)H(t) + \frac{\beta \rho}{N - \theta L} L(t)U(t) - (\sigma + \mu) E(t), \\
\frac{dU(t)}{dt} &= \sigma E(t) - (\gamma_1 + \gamma_2 + \mu) U(t), \\
\frac{dH(t)}{dt} &= \gamma_2 U(t) - \left( [1 - \nu] \gamma_3 + \nu \gamma_5 \chi \right) H(t) - (\mu + \delta) H(t), \\
\frac{dR(t)}{dt} &= \gamma_1 U(t) + \rho S(t) + L(t) + \left( [1 - \nu] \gamma_3 + \nu \gamma_5 \chi \right) H(t) - (\mu + \omega R(t)), \\
\frac{dM(t)}{dt} &= \frac{\delta M(t)}{\tau M(t)} - dM(t).
\end{align*}
\]

(2.17)

For biological feasibility, we assumed that all parameters and initial conditions of the models (2.10), and (2.17) are non-negative. Epidemiological information of the model (2.10) parameters are provided in Table 1. A flow diagram of the Model (2.10) is provided in Fig. 1.

3. Materials and methods

3.1. Some mathematical properties of the model (2.13) in terms of a threshold quantity \( R_0 \)

In Propositions 1 and 2 (see Appendix A), we have established that every forward solution of the newly developed fractional order COVID-19 system (2.13) is always non-negative and bounded if it starts from a non-negative initial condition. Thus, solution of the system (2.13) is biologically feasible. We established that the system (2.13) has an unique disease-free equilibrium \( (\Psi_0) \), which is globally asymptotically stable.
when a threshold quantity $R_0 < 1$ otherwise it is an unstable equilibrium (see Proposition 3 Appendix A). This threshold quantity $R_0$ defined as follows (see Appendix A):

$$R_0 = \frac{\beta_1 \gamma_1}{(\mu + \sigma)(\mu + \gamma_1 + \gamma_2)^{\alpha}} = \frac{\beta_2 \rho \sigma \gamma_2 (\mu + \omega + \phi_c)}{(\mu + \sigma)\left[\mu + (1 - \nu)\gamma_3 + \nu\gamma_3 + \delta\right]^{\alpha} (\mu + \gamma_1 + \gamma_2)(\mu + \omega + \phi_c + (1 - \theta)\delta)}.$$  

Threshold quantity $R_0$ defined in (3.1) is similar to the concept of the basic reproduction number in epidemiology, which indicates the disease potential in a population consists of only susceptible [62]. However, $R_0$ in Eq. (3.1) depends on the power law coefficient (see equation (2.4)) $\alpha$ $(0 < \alpha < 1)$. Furthermore, the first term on the right-hand side of the expression (3.1), indicates disease potential occurring from a community infection, and the second term measures the transmission occurring from confirmed cases in hospitals and quarantined centers. We denote these sub-reproduction numbers as $R_c$ (the community reproduction number), and $R_H$ (the hospital reproduction number) and they are defined as follows:

$$R_c = \frac{\beta_2 \rho \sigma \gamma_2 (\mu + \omega + \phi_c)}{(\mu + \sigma)\left[\mu + (1 - \nu)\gamma_3 + \nu\gamma_3 + \delta\right]^{\alpha} (\mu + \gamma_1 + \gamma_2)(\mu + \omega + \phi_c + (1 - \theta)\delta)}.$$  

$$R_H = \frac{\beta_1 \gamma_1}{(\mu + \sigma)(\mu + \gamma_1 + \gamma_2)^{\alpha}}.$$  

### 3.2. COVID-19 data source

To estimate several important epidemiological model (2.10) parameters (see Table 1), we use daily COVID-19 reported cases and deaths for the period March 01, 2020, till December 01, 2021, from Russia, South Africa (SA), United Kingdom (UK), and United States of America (USA), respectively. Daily reported cases and death data at the initial phase of the epidemic are noisy. For smoothness purposes, we use seven day moving average of the daily COVID-19 cases and deaths for model (2.10) fitting. Daily COVID-19 reported cases, deaths, and demographic data of the mentioned countries were collected from [2].

### Table 1

Description of the models (2.10) and (2.17) parameters and their biologically feasible ranges.

| Parameter | Biological meaning | Range of values | Reference |
|-----------|--------------------|-----------------|-----------|
| $N$       | Total Population   | Varies over different region | [48]       |
| $\Pi = \mu \times N$ | Average recruitment rate of human population in a country/region | Varies over different region | [48]       |
| $a$       | Order of the fractional derivative (power law exponent) | $(0 - 1)$ | Estimated |
| $1/\mu$   | Average life expectancy of human at birth in a country/region | Varies over different region | [48]       |
| $\beta_1$ | Average rate of transmission from undetected SARS-CoV-2 cases | $(0 - 10)$ day$^{-1}$ | [49]       |
| $\beta_2$ | Average rate of transmission from confirmed SARS-CoV-2 cases | $(0 - 10)$ day$^{-1}$ | [49,50]    |
| $t$       | Average lockdown rate | $(0 - 1)$ day$^{-1}$ | [23]       |
| $z$       | Lockdown period | Varies over different country/region | [51]       |
| $\frac{1}{z}$ | Incubation period of COVID-19 | 5.1 days | [52]       |
| $\frac{1}{\tau_1}$ | Infectious period of the undetected cases | $(15.1 - 21)$ days | [53]       |
| $\tau_2$ | COVID-19 testing rate | $(0 - 1)$ day$^{-1}$ | [23]       |
| $\frac{1}{\tau_3}$ | Infectious period of the confirmed cases | $(15.1 - 20)$ days | [53]       |
| $\delta$ | Average case fatality rate | Varies over different country/region | Estimated$^a$ |
| $\rho$    | Fraction of the susceptible population exposed to confirmed SARS-CoV-2 cases | $(0 - 0.1)$ | [54,55]    |
| $\frac{1}{\tau_4}$ | Period of natural immunity in COVID-19 | $(1 - 8)$ months | [43]       |
| $\theta_2$ | Fraction of the recovered populations return to the susceptible state due to loss of immunity | $(0 - 1)$ | Estimated |
| $\zeta$   | Vaccine efficacy | $(0.704)$ | [56]       |
| $\chi$    | Treatment efficacy | $(0 - 2)$ | Assumed |
| $\psi$    | Awareness response rate of susceptible population | $(0 - 1)$ day$^{-1}$ | [57,58]    |
| $\eta$    | Awareness growth rate | $(0 - 1)$ day$^{-1}$ | [57,58]    |
| $\delta$  | Awareness degradation rate | $(0 - 1)$ day$^{-1}$ | [57,58]    |
| $\rho$    | Vaccination rate | $(0 - 1)$ day$^{-1}$ | [59]       |
| $\nu$     | Fraction of confirmed COVID-19 cases that received treatment | 0.1 | [60]       |
| $\theta$  | Fraction of the home quarantined population maintaining the proper social distancing measures | $(0 - 1)$ | [61]       |

$^a$Average case fatality rate $\delta = \frac{T_D}{T_H}$. $T_D$ = Total number of death, $T_H$ = Total number of cases, and $N_i$ = Total number of datapoint.
3.3. Estimation procedure

Epidemiological important parameters of the model (2.10) that are estimated from the data from the mentioned four locations are order of the fractional order derivative (\(\alpha\)), average transmission rate from undetected SARS-CoV-2 cases (\(\beta_1\)), average transmission rate from confirmed SARS-CoV-2 cases (\(\beta_2\)), fraction of susceptible population that are exposed to confirmed SARS-CoV-2 cases (\(\rho\)), infectious period of the undetected cases (\(\gamma_1\)), COVID-19 testing rate (\(\gamma_2\)), infectious period of confirmed cases (\(\gamma_3\)), average lockdown rate (\(\lambda\)), fraction of home quarantined population who maintained proper social distancing (\(\eta\)), awareness response rate of susceptible population (\(\omega\)), treatment efficacy (\(\chi\)), awareness growth rate (\(\lambda_4\)), awareness degradation rate (\(\lambda_5\)), average transmission rate from confirmed cases (\(\gamma\)), fraction of recover populations who become susceptible after loss of natural immunity (\(\lambda_3\)), fraction of home quarantined population (\(\lambda_2\)), and vaccination rate (\(\rho\)), respectively. We estimated these parameters within their biological feasible ranges provided in Table 1. Except for the order of the fractional order derivative (\(\alpha\)), the remaining parameters are also estimated for the corresponding ODE COVID-19 model (2.17). From the models (2.10) and (2.17), daily COVID-19 reported cases (\(C_j\)) during the \(j\)th time interval \([t_j, t_j + \Delta t_j]\) is:

\[
C_j(\hat{\theta}) = \gamma_2 \int_{t_j}^{t_j + \Delta t_j} U(t_j, \hat{\theta}) \, dt_j,
\]

(3.4)

where, \(\Delta t_j\) is the time step length and \(\hat{\theta}\) is the set of unknown parameters of the models (2.10) & (2.17) that are estimated. Let, \(N\) observation from the data and from the models (2.10) & (2.17) are \(\{A_1, A_2, \ldots, A_N\}\) and \(\{C_1(\hat{\theta}), C_2(\hat{\theta}), \ldots, C_N(\hat{\theta})\}\), respectively. Therefore, we constructed the sum of squares function as follows:

\[
SS(\hat{\theta}) = \sum_{i=1}^{K} [A_i - C_i(\hat{\theta})]^2,
\]

(3.5)

MATLAB-based nonlinear least-square solver “lsqnonlin” is used to fit simulated and observed daily COVID-19 reported cases for these four countries during the mentioned time duration. Delayed Rejection Adaptive Metropolis–Hastings algorithm [63] is used to derive the 95% confidence region around these parameter estimates. The detail estimation procedure is provided in [20]. For numerical solution of the fractional order COVID-19 model (2.10), we developed a nonstandard finite difference scheme based on [64] (see Appendix C).

3.4. Selecting best model among the models (2.10) and (2.17)

There are several statistical methods (e.g. Adjusted \(R^2\), Likelihood ratio test, Akaike Information Criterion, etc.) to compare multiple models in terms of their fitting and complexity [39]. Among these techniques, two criteria widely used in ecology and epidemiology, namely, Akaike Information Criterion (AIC) [65–67] and Bayesian Information Criterion (BIC) [68]. In this paper, we used Akaike Information Criterion (AIC) to determine the best model (in terms of fitting and complexity) among the newly formulated fractional order model (2.10) and corresponding integer order ODE model (2.17). Using derivation in [39,63,69], we have following formula for AIC:

\[
AIC = SS(\hat{\theta}) + 2k.
\]

(3.6)
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Fig. 2. In Fig. 2 A, B and C (first column), we plotted daily confirmed cases \( C(t) \) derived formed the model (2.10) which represent 1st, 2nd and 3rd wave, respectively. In Fig. 2 A1, B1 and C1, we plotted \( \frac{dC}{dt} \) and the rectangle shows the number of sign changes in the time series of \( \frac{dC}{dt} \).

where, \( SS(\theta) \) is the sum of square error defined in (3.5) and \( k \) is the number of estimated parameters of the model (2.10). Following [39,69], we derived Akaike weights \( (W_i) \) of the models (2.10) and (2.17), respectively. Akaike weight \( (W_i) \) lies between 0 and 1, and can be considered as the probability that the model \( i \) is the best model for the empirical data from a group of models [39,66,67].

3.5. An algorithm for counting number COVID-19 waves in a region

One of the main aims of this study is to determine some epidemiological parameters of model (2.10) that are most influential in creating multiple COVID-19 waves in a location. To achieve this goal, we first have to count the number of COVID-19 waves \( (W_C) \) from the model (2.10) solution (daily confirmed COVID-19 cases). We considered daily confirmed cases, \( C(t) \), as a function of time, then determined the number of COVID-19 waves \( (W_C) \) in a location by counting sign change in \( \frac{dC}{dt} \). The Algorithm is provided below:

1. If sign change of \( \frac{dC}{dt} =1 \), then exactly one COVID-19 wave occur during this period. Therefore \( W_C = 1 \) (see Figs. 2-A and 2-A1).
2. If \( 2 \leq \text{sign change of } \frac{dC}{dt} <4 \), then exactly two COVID-19 waves observe during this period. Therefore, \( W_C = 2 \) (see Figs. 2-B and 2-B1).
3. If \( 4 \leq \text{sign change of } \frac{dC}{dt} <6 \), then exactly three COVID-19 waves occur during this period. Therefore, \( W_C = 3 \) (see Figs. 2-C and 2-C1).
4. If \( 6 \leq \text{sign change of } \frac{dC}{dt} <8 \), then fourth COVID-19 waves observe during this period. Therefore, \( W_C = 4 \).
5. If \( 8 \leq \text{sign change of } \frac{dC}{dt} \), then we have fifth or more COVID-19 waves observe during this period. Therefore, \( W_C \geq 5 \).

There may be some initial oscillation in the daily confirmed cases \( (C(t)) \) obtained from the model (2.10) simulation. To remove this initial fluctuation, we neglect the first 20 time points of the daily confirmed cases \( (C(t)) \).

3.6. Determining correlation between model (2.10) parameters and the number of COVID-19 waves in a location

We identified nine key parameters of the model (2.10) that may influence the number of COVID-19 waves \( (W_C) \) in the four locations Russia, South Africa (SA), United Kingdom (UK), and USA, respectively. Parameters are provided below:

(i) Order of the fractional derivative \( (\alpha) \).
(ii) Average rate of transmission form the undetected COVID-19 cases \( (\beta_1) \).
(iii) Average rate of transmission form the confirmed COVID-19 cases \( (\beta_2) \).
(iv) Fraction of the susceptible population exposed to confirmed COVID-19 cases \( (\rho) \).
(v) Average COVID-19 testing rate \( (\gamma_2) \).
(vi) Fraction of the home-quarantined population maintaining the proper social distancing measures \( (\theta) \).
(vii) Average awareness response rate of the susceptible population \( (\psi) \).
(viii) Period of natural immunity \( (\frac{1}{\omega}) \).
(ix) Fraction of the recovered individuals return to the susceptible state due to loss of immunity \( (\theta_1) \).
A scatter plot between each of these parameters with \( W_C \) suggests a nonlinear and non-monotone relationship. Thus to determine the individual and the collective effect of these parameters on \( W_C \), we required a variance-based global sensitivity analysis method [70–72]. The main advantage of applying a variance-based method over a regression-based technique in determining global sensitivity analysis of model parameters is that they do not make any prior assumptions about the linearity or the monotonicity of the input–output relationship between model parameters, and the responses [40,70,71]. In this work, we applied Sobol’s method of global sensitivity analysis [40,72,73] to compute the first order effect (\( S_{\theta_j} \)) and total order effect (\( S_{\theta_j} \)) of the mentioned nine parameters of the model (2.10) on the response \( W_C \). Based on the results in [40,73], we have the following results regarding \( S_{\theta_j} \) and \( S_{\theta_k} \):

1. \( 0 \leq S_{\theta_j} \leq S_{\theta_k} \leq 1 \), where \( j = 1, 2, \ldots, 9 \).
2. \( S_{\theta_j} = S_{\theta_k} = 0 \) means that \( W_C \) does not depend on the parameter \( \theta_k \).
3. \( S_{\theta_j} = S_{\theta_k} = 1 \) implies that \( W_C \) depends only on the parameter \( \theta_k \).

Nine parameters mentioned above are sampled within their corresponding ranges (see Table 1) using the Latin hypercube sampling technique [74]. Remaining model (2.10) parameters are fixed either to their estimated values (see Table 2) or to their epidemiological fixed values (see Table 1).

### 4. Result and discussion

Fractional order COVID-19 model (2.10) and the corresponding ODE model (2.17) fitting to the daily COVID-19 cases from Russia, South Africa, UK, and USA, respectively for the time-period March 01, 2020, till December 01, 2021, is provided in Fig. 3. Biological interpretation of the order of the fractional derivative \( (\alpha) \) indicates an index of memory [38,75,76]. \( \alpha \rightarrow 1 \) implies a system does not carry any information about its previous states and as \( \alpha \) close to zero, a system carries more information about its previous states [38,75,76]. In our developed COVID-19 model (2.10), fractional derivative used only in disease transmission and the estimated value of \( (\alpha) \) and its 95% CI indicates (see Table 2) a sufficient amount of information effect in disease transmission in all of the four locations namely Russia, South Africa, UK, and USA, respectively. In most of the locations (except South Africa), the average community transmission rate (\( \beta_1 \)) is found to be lesser than the average transmission that occurred from confirmed cases (\( \beta_1 \)) [see Table 2]. Furthermore, estimates of the fraction of susceptible populations that are exposed to confirmed SARS-CoV-2 cases (\( \rho \)) in South Africa, UK, and Russia, respectively indicate a small percentage of transmission occurring from confirmed COVID-19 cases in comparison to the transmission occurring from the undetected cases in the community (see Table 2). However, in USA, the estimate of \( \rho \) suggests that 48% to 49% of COVID-19 transmission originates from the confirmed COVID-19 cases in hospitals and quarantined centers (see Table 2). The estimate of the fraction of home quarantined population who maintained proper social distancing (\( \theta \)) indicates in UK, Russia, and South Africa, an
COVID-19 may become endemic (90 days to contacting undetected COVID-19 cases from the community (see Table 2). Estimated reinfection period \((\tau)\) derived from the newly developed fractional order model (2.10) suggests that in each of the countries, almost negligible amount of home-quarantined populations are susceptible to community infection (see Table 2) during the lockdown. However, in South America and UK, this behavior of home quarantined individuals is due to forced lockdown imposed by the government, not due to awareness as the estimate of the awareness response intensity \((\psi)\) is found to be lower for these two countries (see Table 2). Furthermore, in USA, our results suggest that 30% to 38% of the home-quarantined population do not maintain proper social distancing and therefore, are prone to infection by contacting undetected COVID-19 cases from the community (see Table 2). Estimated reinfection period \((\tau)\) are found to be shortest (around 60 days to 90 days) in Russia and longest in UK (around 109 days to 180 days) (see Table 2). Trends in the estimates of \(\theta\) (see Tables 1 and 2) in all four locations suggest that after recovery individuals tend to maintain proper social distancing measures. Similar to the fractional order model (2.10), Table 3 provides the estimated parameter values for the COVID-19 ODE model (2.17).

The estimated threshold quantity \(R_0\) derived from the newly developed fractional order model (2.10) suggests that in each of the countries, COVID-19 may become endemic \((R_0 \approx 1)\) (see Table 4). Furthermore, the estimate of \(R_C\) and \(R_H\) (see Materials and Methods section for details) indicate that majority of the COVID-19 transmission occurs in these four countries via the community undetected cases \((R_C \gg R_H)\) (see Table 4).

Comparison of the newly developed fractional order COVID-19 model (2.10) with the corresponding ODE model (2.17) based on their AIC values (model fitting) along with other multi-model inference quantities [39,66] suggest that fractional order COVID-19 model (2.10) is a better model compare to the corresponding ODE model (2.17) in capturing trend of the COVID-19 data from UK and South Africa (see Table 5). However, COVID-19 ODE model (2.17) found out to be the best model in case of data from Russia and USA (see Table 5). Results follows from Eq. (2.4) suggest that when age of infection function follows a power law then the general time dependent force of infection function in the COVID-19 system (2.1) converted to a fractional order force of infection function and consequently, the system (2.1) become the COVID-19 system (2.10) with fractional order disease transmission. Thus, in those regions where COVID-19 transmission does not follow a power law, the newly formulated COVID-19 fractional order model (2.10) may not fit well with the data from these regions. Thus, there may be a possibility that COVID-19 transmission in Russia and USA may not be following a power law effect in disease transmission.
Fig. 3. Two COVID-19 models [Eqs. (2.10) and (2.17)] fitting to the daily COVID-19 cases from Russia, South Africa, UK, and USA, respectively, for the time period 1st March, 2020 to 1st December 2021. In the first column, represents fitting of the fractional order COVID-19 model and the second column represents, corresponding ODE model (2.17) fitting. Red lines are represented by daily notified cases from the data and black lines are the corresponding models (2.10) & (2.17) solution. Shaded region is the 95% confidence region, respectively. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 5
Different multi-model inference quantities corresponding to the fractional order COVID-19 model (2.10) and integer order (ODE) COVID-19 model (2.17), respectively.

| Country  | Russia          | South Africa | UK           | USA            |
|----------|-----------------|---------------|--------------|----------------|
| AIC      | Fractional order COVID-19 Model (2.10) | 2.5048 E+10   | 1.0583 E+10  | 3.8556 E+10    | 3.4914 E+11   |
|          | Corresponding ODE Model (2.17)         | 9.4349 E+09   | 1.1372 E+10  | 5.9853 E+10    | 3.0977 E+11   |
| A_1      | Fractional order COVID-19 Model (2.10) | 1.5613 E+10   | 0             | 0              | 3.9370 E+10   |
|          | Corresponding ODE Model (2.17)         | 0             | 7.8900 E+8   | 2.1303 E+10    | 0             |
| E/R      | Fractional order COVID-19 Model (2.10) | $\infty$      | 1             | 1              | $\infty$      |
|          | Corresponding ODE Model (2.17)         | 1             | $\infty$     | $\infty$      | 1             |
| W_C     | Fractional order COVID-19 Model (2.10) | 0             | 1             | 1              | 0             |
|          | Corresponding ODE Model (2.17)         | 1             | 0             | 0              | 1             |

The estimated first order ($S_{th}$) and total order ($S_{th})$ Sobol’s sensitivity indices [40,72,73] of the nine key parameters (see Section 3) of the fractional order COVID-19 model (2.10) on the response $W_C$ (the number of COVID-19 waves in a location) for the four regions indicate that order of the fractional derivative $\alpha$ (power law coefficient in the age of infection function) and the average transmission rate of undetected COVID-19 cases in the community ($\beta_1$) are mainly correlated to the formation of multiple COVID-19 waves in those four mentioned locations (see Fig. 4). Furthermore, it is found to have the most elevated effect on the number of COVID-19 waves ($W_C$) in Russia and South Africa compared to the UK and USA (see Fig. 4). Furthermore, a heat map of $W_C$ for the four locations by varying $\alpha$ and $\beta_1$ to their biologically feasible ranges identifies the values of these two parameters for which one or more COVID-19 waves occur in these four countries (see Fig. 5). The power law exponent $a$ in Eq. (2.4) represents the order of the R-L fractional order derivative in the COVID-19 model (2.10). Following [75], $a$ represents an index of memory effect in the disease transmission process. Further exploring the memory effect in disease transmission, we drew a scatter plot of $W_C$ by varying $a$ (see Fig. 6) (Fixing other parameters to their estimated mean or biological values, see Tables 1 and 2). We found that the number of waves ($W_C$) increases as $a$ increases (0 → 1) up to a threshold value ($a_r$) in each of the four mentioned locations. This threshold value ($a_r$) varies over the four regions (see Fig. 6). Thus, we may conclude that increasing the memory effect in the COVID-19 transmission process ($a \rightarrow 0$) will decrease the number of COVID-19 waves in a location. To further strengthen this fact, we perform a global sensitivity analysis of $a$ and $\beta_1$ on two responses namely, the total number of COVID-19 cases during the period March 01, 2020, till December 01, 2021 ($C_T$) and the basic
Fig. 4. 1st order \( (S_{\theta i}) \) and the total order \( (S_{T\theta i}) \) Sobol’s sensitivity indices of the nine key epidemiological parameters of the COVID-19 models (2.10) and (2.17), respectively, with the response the number of COVID-19 waves \( (W_C) \) for Russia, South Africa, UK, and USA. Epidemiological information of this nine key parameters are provided in Table 1. In the first column represents Sobol’s sensitivity indices \( (S_{\theta i}) \) for the fractional order COVID-19 model (2.10) and in the second column, we plotted Sobol’s sensitivity indices for the integer order (ODE) COVID-19 model (2.17).

Fig. 5. Heat-map of the number of COVID-19 waves \( (W_C) \) in Russia, South Africa, UK, and USA, respectively, by varying two most influential parameters \( \alpha \) and \( \beta_1 \) of the fractional order COVID-19 model (2.10). Color bar represents the number of waves \( (W_C) \). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

reproduction number \( (R_0) \), respectively (see Fig. 7). A non-linear and monotone relationship observed between the two parameters \( (\alpha \) and \( \beta_1 \)) with the mentioned two responses \( (C_T \) and \( R_0) \), respectively. Therefore, global sensitivity analysis of \( \alpha \) and \( \beta_1 \) on two responses \( (C_T \) and \( R_0) \) are performed by computing the partial rank correlation coefficients (PRCC) [74] (see Fig. 7). To determine the PRCC [74] for each of the mentioned parameters on the responses \( C_T \) and \( R_0) \), we have drawn 500 samples of \( \alpha \) and \( \beta_1 \) from their biologically feasible ranges (see Table 1) using Latin Hypercube Sampling (LHS) technique [74]. Other parameters used during computing PRCC are taken from Tables 1 and 2. We found that both \( \alpha \) and \( \beta_1 \) have positive correlation with the responses total number of COVID-19 cases during the period March 01, 2020, till December 01, 2021 \( (C_T) \)
14

and the basic reproduction number \(R_0\) [see Fig. 7]. Moreover, the memory in the disease transmission process \(\alpha\) has a higher positive correlation in comparison to \(\beta_1\) with the mentioned two responses \((C_T \text{ and } R_C)\) for all four locations Russia, South Africa, UK, and USA, respectively. Therefore, increasing the memory effect \(\alpha \rightarrow 0\) may reduce the number of COVID-19 cases in a location. Thus, to prevent the risk of larger future COVID-19 waves in the mentioned four locations, policymakers may focus on increasing memory effect (reducing \(\alpha\)) in disease transmission (see Figs. 6, and 7).

5. Conclusion

By analyzing COVID-19 incidence data from different countries around the globe, several studies concluded that at the beginning of the epidemic, data trends followed some exponential growth, and as the epidemic progress over the years, data trended to exhibit a power law growth pattern [8–11,13,77–79]. In general, power law growth in COVID-19 cases is directly related to control measures, in the sense that the less strict the control, the smaller the power law exponent, and hence the slower the disease progresses to its end [8,13,77–79]. Thus, it is reasonable to assume that the COVID-19 transmission process follows some power law [79]. Recently, several studies established that fractional derivatives (Riemann–Liouville and Caputo) and integrals are convoluted with a power law [17–19,80,81], where the order of the fractional derivative \(\alpha\) represents the power law exponent [21]. Recently, Du et al. [75] proved that the order of the fractional derivative \(\alpha\) in a dynamical system can be used as an index of memory, where, \(\alpha \rightarrow 0\) implies the increasing memory effect in the system. Thus, using a fractional derivative to build up a COVID-19 model may address gaps between data and the existing knowledge on the COVID-19 transmission process. However, in all of the existing fractional order models on COVID-19 [25–28,82–85], instead of taking fractional derivatives in the force of infection in disease transmission, authors used fractional derivatives only in the population (left-hand side of the system replacing ordinary derivatives with some fractional order derivative), which in general a Markovian process [20,21,35,36,38]. Consequently, these models may not be appropriate to study the power law/memory effect in the transmission process of COVID-19.

In this context, we developed a new COVID-19 mathematical model (2.1) with a general time-dependent age of infection function in disease transmission. Using few recent techniques [20–22,38], we showed that when the age of infection function in disease transmission follows a power law, then the system (2.1) transformed to a COVID-19 model (2.10) with a fractional order force of infection function in disease transmission. Therefore, in this newly developed COVID-19 model (2.10), we considered the power law/memory effect exclusively in disease transmission process. Furthermore, several realistic epidemiological assumptions like imperfect social distancing behavior of the home-quarantined individuals, media awareness campaign, vaccination, treatment, and reinfection scenario of the recovered population also considered in this newly developed fractional order COVID-19 system (2.10). We explored several important mathematical properties like the biological feasibility of the solution (positive invariance), existence and global stability of the disease-free equilibrium \((P_0)\), etc., of the newly developed COVID-19 model (2.10) [see Appendix A]. We determine three fundamental threshold quantities \((R_0, R_C, \text{ and } R_H)\) of the model (2.10), and all of these threshold quantities depend on the memory index \(\alpha\) (power law exponent). Several parameters of the newly formulated fractional order COVID-19 model (2.10) are estimated (see Table 2) by fitting the model (2.10) to the daily COVID-19 reported cases from Russia, South Africa, UK, and USA, respectively, from the time-period March 01, 2020, till December 01, 2021. Furthermore, using variance-based Sobol’s global sensitivity analysis method, we investigated the impact of various parameters of the fractional order COVID-19 model (2.10) on the number of COVID-19 waves \((W_C)\) in those four regions (see Fig. 4). We found that the memory index in the disease force of infection \(\alpha\) [power law exponent in the age of infection function] and average rate of transmission from the undetected cases \(\beta_1\) are most influential in generating multiple COVID-19 waves in all of the four locations (see Fig. 4). Furthermore, a heat map of the number of COVID-19 waves (see Fig. 5) identified the regions in the biologically feasible parameter...
quarantined population. However, it is more likely all individuals (susceptible, exposed, and infected) may maintain social distancing during the
susceptible individuals are home-quarantined due to awareness or lockdown. Thus, we neglect the possibility of cross-infection within the home-
lower the severity of infection in Russia, South Africa, UK, and USA, respectively.
strategy to control the values \( \alpha \) may focus on restricting
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\( \alpha \) may focus on restricting with some recent results of the power law effect on COVID-19 incidence growth pattern \([8,13,77–79]\). Therefore, government and policymakers
lockdown, social distancing, use of face-mask, therefore, using these measures may control future waves in these locations. These findings agree
mentioned four locations. As power law/memory effect in disease transmission is directly related to some forced COVID-19 control measures like
transmission (\(2.10\)) has a very high positive correlation (\(>0.9\)) with both the responses \(C_T\) and \(R_0\), respectively (see Fig. 7). Thus, we can conclude
that increasing the memory effect in COVID-19 transmission reduces the number of waves as well as decrease the severity of the outbreak in those
mentioned four locations. As power law/memory effect in disease transmission is directly related to some forced COVID-19 control measures like
lockdown, social distancing, use of face-mask, therefore, using these measures may control future waves in these locations. These findings agree
with some recent results of the power law effect on COVID-19 incidence growth pattern \([8,13,77–79]\). Therefore, government and policymakers
may focus on restricting \(\alpha\) and \(\beta_1\) to restrict the forthcoming waves in these four locations (see Figs. 5-to-7). Following, we discussed a possible
strategy to control the values \(\alpha\) and \(\beta_1\):

- It is well established that the order of the fractional derivative (\(\alpha\)) can be used as an index of memory \([75,86–88]\). For the newly developed
COVID-19 system (\(2.10\)), the order of the fractional derivative \(\alpha\) lies in \(0 < \alpha < 1\). Therefore, if \(\alpha \rightarrow 0\) then it signifies the system (\(2.10\)) tends
to become a Markovian system (integer order ODE system) i.e. it does not carry any information about the previous transmission states, and
if \(\alpha \rightarrow 0\), then becomes an ideal system that contains all information about its previous states of transmission. As COVID-19 transmission
occurred by an interaction between susceptible and infected individuals, therefore, in the fractional order COVID-19 system (\(2.10\)), memory
in the transmission interprets as the information carries among individuals (susceptible and infected) as the epidemic progress in the
population. Several non-pharmaceutical control strategies like social distancing, use of a face mask, use of sanitizer, awareness, etc. may
increase the information effect (reducing \(\alpha\)) and therefore, severity of the infection and as well as the number of waves will be reduced (see
Figs. 6, and 7).
- The average transmission rate (\(\beta_1\)) can be controllable by applying various non-pharmaceutical control strategies like social distancing, use
of a face-mask, use of sanitizer, vaccination, awareness, etc.

We sincerely hope that applying the above strategies in controlling \(\alpha\) and \(\beta_1\), may reduce the risk of further COVID-19 waves and as well as
lower the severity of infection in Russia, South Africa, UK, and USA, respectively.
The current study has some boundaries and may be extended from diverse perspectives. In the COVID-19 model (\(2.10\)), we assumed that only
the susceptible individuals are home-quarantined due to awareness or lockdown. Thus, we neglect the possibility of cross-infection within the home-
quarantined population. However, it is more likely all individuals (susceptible, exposed, and infected) may maintain social distancing during the

![Fig. 7. Global sensitivity analysis of two parameters namely, \(\alpha\) and \(\beta_1\) on the responses 1. Total number of COVID-19 cases and 2. The basic reproduction number (\(R_0\)) for the locations Russia, South Africa, UK, and USA, respectively. Total number of COVID-19 cases are measured during the period 1st March, 2020 to 1st December, 2021 for each of the mentioned four locations. Effect of Uncertainty of these two parameters on the two mentioned responses are measured using the Partial Rank Correlation Coefficients (PRCC). 500 samples for each parameters were drawn using the Latin hypercube sampling technique (LHS) from their respective ranges provided in Table 1. Other parameter values used during computing the two mentioned responses are fixed to their estimated mean values (see Table 2).](image-url)
epidemic, and there is a high probability of cross-infection among these home-quarantined populations. Moreover, in our newly formed COVID-19 model (2.10), we assumed power law effect only in the disease transmission process. However, some recent studies found that the COVID-19 death process also follows some power law effect [77]. Furthermore, for the fractional order COVID-19 system (2.10), we are unable to show analytically the existence and stability of the endemic-equilibrium point/points. Consequently, the possibility of the occurrence of the backward bifurcation or any other bi-stability among the equilibrium points is not analyzed. We leave these biologically and mathematically challenging problems for our future endeavors.

CRediT authorship contribution statement

Tahajuddin Sk: Data curation, Formal analysis, Investigation, Methodology, Software, Validation, Writing – original draft. Santosh Biswas: Writing – original draft, Writing – review & editing. Tridip Sardar: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Software, Supervision, Validation, Writing – original draft, Writing – review & editing.

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.

Data availability

Data references have already been incorporated into the manuscript. The information is freely accessible.

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Appendix A

Lemma A.1 (Lemma 2 in [89]). Suppose \( \Omega \subset \mathbb{R} \times C^n \) is open, \( f_i \in C(\Omega, \mathbb{R}), i = 1, 2, 3, \ldots, n \). If \( f_i|_{x(t)=0,x \in \mathbb{C}^n} \geq 0 \), \( X_i = [x_1(t), x_2(t), \ldots, x_n(t)]^T, i = 1, 2, 3, \ldots, n \), then \( C^n_{\geq 0} = \{ \phi = (\phi_1, \ldots, \phi_n) : \phi \in C([-\tau, 0], \mathbb{C}^n) \} \), is the invariant domain of the following equations

\[
\frac{dx_i(t)}{dt} = f_i(t, X_i), \quad i \geq \sigma, \quad i = 1, 2, 3, \ldots, n.
\]

where, \( \mathbb{R}^n_{\geq 0} = \{(x_1, \ldots, x_n) : x_i \geq 0, \quad i = 1, \ldots, n\} \).

Proposition 1. \( \mathbb{R}^n_{\geq 0} \) is an invariant domain for the SARS-CoV-2 system (2.13).

Proof. Therefore, from (2.13), we have:

\[
\begin{align*}
\frac{dS}{dt} \bigg|_{S=0, x \in \mathbb{R}^n_{\geq 0}} & = H + \alpha L + \theta, R > 0, \\
\frac{dL}{dt} \bigg|_{L=0, x \in \mathbb{R}^n_{\geq 0}} & = IS + \psi SM + (1 - \theta) x_0, R \geq 0, \\
\frac{dU}{dt} \bigg|_{U=0, x \in \mathbb{R}^n_{\geq 0}} & = \sigma E \geq 0, \\
\frac{dH}{dt} \bigg|_{H=0, x \in \mathbb{R}^n_{\geq 0}} & = \gamma_1 U \geq 0, \\
\frac{dR}{dt} \bigg|_{R=0, x \in \mathbb{R}^n_{\geq 0}} & = \gamma_1 U + p(1 - \alpha, \alpha S + L + \alpha \gamma_3 + \alpha \gamma_1 X) H \geq 0, \\
\frac{dM}{dt} \bigg|_{M=0, x \in \mathbb{R}^n_{\geq 0}} & = \eta H \geq 0.
\end{align*}
\]

(A.1)

Now we show that \( \frac{dE}{dt} \bigg|_{E=0, x \in \mathbb{R}^n_{\geq 0}} \geq 0 \). Using the result [46] and relation between Riemann–Liouville fractional derivative in [45], and using relation (C.1) and (C.2) (see in Appendix C), we have

\[
\frac{dE}{dt} = \frac{\beta^{\alpha}}{N - \theta L} S(t) \int_0^\infty D^{1-a_1}(t) U(t) + \frac{\beta^{\alpha}}{N - \theta L} S(t) \int_0^\infty D^{1-a_1}(t) H(t) \]

\[
+ \frac{\beta^{\alpha}(1-c) L(t) \int_0^\infty D^{1-a_1}(t) U(t),
\]

\]
Thus, from (A.6), we have

\[ \int_0^t e^{-\alpha(t-s)}(t-s)^{\gamma-1} E(s) ds + \int_0^t e^{-\alpha(t-s)}(t-s)^{\gamma-1} S(s) ds = U(0) \beta_1 e^{-\alpha t} t^{\gamma-1} - \int_0^t \frac{U(0) \beta_1 e^{-\alpha(t-s)} t^{\gamma-1}}{\Gamma(\alpha)(N - \theta L)} S(s) ds + \int_0^t e^{-\alpha(t-s)}(t-s)^{\gamma-1} E(s) ds + \int_0^t e^{-\alpha(t-s)}(t-s)^{\gamma-1} S(s) ds + \int_0^t e^{-\alpha(t-s)}(t-s)^{\gamma-1} U(s) ds + \int_0^t e^{-\alpha(t-s)}(t-s)^{\gamma-1} U(s) ds + \int_0^t e^{-\alpha(t-s)}(t-s)^{\gamma-1} U(s) ds + \int_0^t e^{-\alpha(t-s)}(t-s)^{\gamma-1} U(s) ds. \]

Following Lemma A.1, we have

\[ \eta \gamma \mu + \frac{\nu_1}{\mu} \gamma \mu + \beta_2 e^{\beta_1 t} t^{\gamma-1} - \int_0^t \frac{U(0) \beta_1 e^{-\alpha(t-s)} t^{\gamma-1}}{\Gamma(\alpha)(N - \theta L)} S(s) ds + \int_0^t e^{-\alpha(t-s)}(t-s)^{\gamma-1} E(s) ds + \int_0^t e^{-\alpha(t-s)}(t-s)^{\gamma-1} S(s) ds + \int_0^t e^{-\alpha(t-s)}(t-s)^{\gamma-1} U(s) ds + \int_0^t e^{-\alpha(t-s)}(t-s)^{\gamma-1} U(s) ds + \int_0^t e^{-\alpha(t-s)}(t-s)^{\gamma-1} U(s) ds + \int_0^t e^{-\alpha(t-s)}(t-s)^{\gamma-1} U(s) ds. \]

Thus, following Lemma A.1, \( R_{+0}^1 \) is an invariant region for the system (2.13).

In reality all forward solutions related to a disease process are bounded. Thus, we further restrict our invariant domain \( R_{+0}^7 \) for the model (2.16) to a bounded region define as follows:

\[ D = \left\{ (S, I, E, H, R, M) \in R_{+0}^7 \left| S + L + E + H + R \leq \frac{\Pi}{\mu} M \leq \frac{\Pi}{\mu} \right. \right\}. \]  

Proposition 2. The closed and bounded domain \( D \subset R_{+0}^7 \) is a positively invariant and global attracting set for the system (2.16).

Proof. Adding all equations in the model (2.16), we have:

\[ \frac{dN}{dt} = \Pi - \mu N - \delta H. \]  

As all parameters are non-negative and \( H \in R_{+0}^7 \), therefore, from (A.4), we have:

\[ \frac{dN}{dt} \leq \Pi - \mu N. \]  

From (A.5), it is clear that if \( N(t) < \frac{\Pi}{\mu} \), then \( \frac{dN}{dt} < 0 \). Therefore, following a standard comparison theorem [90], we have:

\[ N(t) \leq \frac{\Pi}{\mu} + \left( N(0) - \frac{\Pi}{\mu} \right) e^{-\mu t}. \]  

Thus, from (A.6), we have \( N(0) \leq \frac{\Pi}{\mu} \Rightarrow N(t) \leq \frac{\Pi}{\mu} \). Therefore, \( D \) is a positively invariant region for the SARS-CoV-2 system (2.16). Moreover, \( N(0) > \frac{\Pi}{\mu} \Rightarrow \lim_{t \to \infty} \sup N(t) \leq \frac{\Pi}{\mu} \), leads to the fact that \( D \subset R_{+0}^6 \) is a global attracting set [91] for the SARS-CoV-2 system (2.16).

Again,

\[ \frac{dM}{dt} = \frac{\eta H(t)}{1 + H(t)} - \frac{dM(t)}{d\mu} \]

\[ \Rightarrow \frac{dM}{dt} + \frac{dM(t)}{d\mu} \leq \eta H(t), \]

\[ \leq \frac{\eta \Pi}{\mu}, \]  

\[ \Rightarrow M \leq \frac{\eta \Pi}{d\mu}. \]

A.1. Disease-free equilibrium

The SARS-CoV-2 system (2.16) has an unique disease-free equilibrium solution \( \Psi_0 \) provided below:

\[ \Psi_0 : (S_*, I_*, E_*, H_*, R_*, M_*) = \left( \frac{\Pi}{(\mu + \omega + \beta_1^* \gamma)}, \frac{\Pi}{(\mu + \omega + \beta_2^* \gamma)}, 0, 0, 0, 0 \right). \]  

A.2. Local stability of \( \Psi_0 \)

The local stability of \( \Psi_0 \) can be derived for the system (2.16)

Proposition 3. The disease-free equilibrium \( \Psi_0 \) of the SARS-CoV-2 model (2.16) is locally asymptotically stable if \( R_0 < 1 \), otherwise it is unstable.

Proof. Integrating the system (2.16), we arrived at the following system of nonlinear integral equations:

\[ S(t) = S(0)e^{-\mu t} + \int_0^t \omega L(\tau)e^{-\mu(\tau-t)} d\tau + \int_0^t \frac{\Pi}{(\mu + \omega + \beta_1^*)} (1 - e^{-\mu \tau}) \]

\[ - \frac{\beta_1^*}{\Gamma(\alpha - 1)} \int_0^t \frac{S(\tau)}{N(\tau - \theta L(\tau))} e^{-\alpha(\tau-s)}(\tau-s)^{\gamma-1} U(s) ds d\tau \]
\[
\begin{align*}
\frac{\partial^2 \rho}{\Gamma(a-1)} \int_0^t S(t) e^{-(\rho^{\mu+\theta}t)} e^{-a_1(t-\gamma_1)} \left( t - \gamma_1 \right)^{\gamma-2} H(s) ds dt, \\
L(t) &= L(0)e^{-(\mu+\theta)\gamma} + \int_0^t L(t) e^{-(\mu+\theta)\gamma} dt \\
&- \frac{\beta^3(1-\theta)}{\Gamma(a-1)} \int_0^t \frac{L(t)}{N(\tau) - \theta N(L(\tau))} e^{-(\mu+\theta)\gamma} \left( t - \gamma_1 \right)^{\gamma-2} U(s) ds dt, \\
E(t) &= E(0)e^{-(\mu+\theta)\gamma} + \frac{\beta_1^3}{\Gamma(a-1)} \int_0^t S(t) e^{-(\mu+\theta)\gamma} \left( t - \gamma_1 \right)^{\gamma-2} U(s) ds dt + \frac{\beta^3(1-\theta)}{\Gamma(a-1)} \int_0^t L(t) e^{-(\mu+\theta)\gamma} \left( t - \gamma_1 \right)^{\gamma-2} U(s) ds dt, \\
H(t) &= H(0)e^{-(\mu+\theta)\gamma} + \int_0^t \gamma_1 U(t) e^{-(\mu+\theta)\gamma} dt \\
R(t) &= R(0)e^{-(\mu+\theta)\gamma} + \int_0^t \gamma_1 U(t) e^{-(\mu+\theta)\gamma} dt + \int_0^t \gamma_1 H(t) e^{-(\mu+\theta)\gamma} dt. \\
\end{align*}
\]

Now from Eq. (A.9)
\[
\begin{align*}
E(t) &= E(0)e^{-(\mu+\theta)\gamma} + \frac{\beta_1^3}{\Gamma(a-1)} A_1 + \frac{\beta^3(1-\theta)}{\Gamma(a-1)} A_2, \\
A_1 &= \int_0^t \frac{S(t)}{N(\tau) - \theta N(L(\tau))} e^{-(\mu+\theta)\gamma} \left( t - \gamma_1 \right)^{\gamma-2} U(s) ds dt, \\
A_2 &= \int_0^t \frac{S(t)}{N(\tau) - \theta N(L(\tau))} e^{-(\mu+\theta)\gamma} \left( t - \gamma_1 \right)^{\gamma-2} U(s) ds dt, \\
A_3 &= \int_0^t \frac{L(t)}{N(\tau) - \theta N(L(\tau))} e^{-(\mu+\theta)\gamma} \left( t - \gamma_1 \right)^{\gamma-2} U(s) ds dt.
\end{align*}
\]

From Eq. (A.9), we have:
\[
\begin{align*}
A_1 &= \int_0^t \frac{S(t)}{N(\tau) - \theta N(L(\tau))} e^{-(\mu+\theta)\gamma} \left( t - \gamma_1 \right)^{\gamma-2} \left[ U(0)e^{-(\mu+\theta)\gamma} + \int_0^t \sigma E(t_1) e^{-(\mu+\theta)\gamma} dt_1 \right] ds dt. \\
&= U(0) \int_0^t \frac{S(t)}{N(\tau) - \theta N(L(\tau))} e^{-(\mu+\theta)\gamma} \left( t - \gamma_1 \right)^{\gamma-2} e^{a_1(t-\gamma_1)} ds dt \\
&+ \int_0^t \frac{S(t)}{N(\tau) - \theta N(L(\tau))} e^{-(\mu+\theta)\gamma} \left( t - \gamma_1 \right)^{\gamma-2} \int_0^t \sigma E(t_1) e^{-(\mu+\theta)\gamma} dt_1 ds dt, \\
A_2 &= \int_0^t \frac{S(t)}{N(\tau) - \theta N(L(\tau))} e^{-(\mu+\theta)\gamma} \left( t - \gamma_1 \right)^{\gamma-2} \left[ H(0)e^{-(\mu+\theta)\gamma} + \int_0^t \gamma_1 U(t) e^{-(\mu+\theta)\gamma} dt + \int_0^t \sigma E(t_1) e^{-(\mu+\theta)\gamma} dt_1 \right] ds dt. \\
&= H(0) \int_0^t \frac{S(t)}{N(\tau) - \theta N(L(\tau))} e^{-(\mu+\theta)\gamma} \left( t - \gamma_1 \right)^{\gamma-2} e^{a_2(t-\gamma_1)} ds dt \\
&+ \gamma_1 U(0) \int_0^t \frac{S(t)}{N(\tau) - \theta N(L(\tau))} e^{-(\mu+\theta)\gamma} \left( t - \gamma_1 \right)^{\gamma-2} \left[ e^{a_2(t_1-\gamma_1)} \int_0^t E(t_1) e^{-(\mu+\theta)\gamma} dt_1 \right] dt, \\
&+ \gamma_2 \sigma U(0) \int_0^t \frac{S(t)}{N(\tau) - \theta N(L(\tau))} e^{-(\mu+\theta)\gamma} \left( t - \gamma_1 \right)^{\gamma-2} \left[ e^{a_2(t_1-\gamma_1)} \int_0^t E(t_1) e^{-(\mu+\theta)\gamma} dt_1 \right] dt_1 ds dt, \\
A_3 &= \int_0^t \frac{L(t)}{N(\tau) - \theta N(L(\tau))} e^{-(\mu+\theta)\gamma} \left( t - \gamma_1 \right)^{\gamma-2} \left[ U(0)e^{-(\mu+\theta)\gamma} + \int_0^t \sigma E(t_1) e^{-(\mu+\theta)\gamma} dt_1 \right] ds dt. \\
\end{align*}
\]
We will now show that \( \lim_{t \to \infty} E(t) = 0 \). Furthermore, let us assume \( \lim_{t \to \infty} \sup E(t) = e_0 \). From definition of lim sup, for any given \( \epsilon > 0 \) there exists \( t_1 \) such that \( E(t) < e_0 + \epsilon \), \( \forall t > t_1 \). We have

\[
A_1 \leq \frac{(\mu + \omega + p \zeta)}{[\mu + \omega + p \zeta + 1(1 - \theta)]} H(0) \int_0^t \int_0^{t_1} e^{-t_1} e^{-2} e^{-2} e^{-2} d\tau d\sigma
\]

\[
+ \frac{(\mu + \omega + p \zeta)}{[\mu + \omega + p \zeta + 1(1 - \theta)]} \Gamma (a - 1) \frac{H(0) e}{(a + \omega + p \zeta + 1(1 - \theta))} + \frac{(\mu + \omega + p \zeta)}{[\mu + \omega + p \zeta + 1(1 - \theta)]} \Gamma (a - 1) \frac{(e_0 + \epsilon)}{\epsilon}
\]

Let us take transformation, \( \tau = t - s = -z_2 \), and \( s - t = z_2 \), we have \( \exists t_2 \) such that \( \lim_{t_\to \infty} \int_0^t e^{-\omega z_2} d\sigma < \epsilon \), \( \forall t > t_2 \). Furthermore, let us assume \( \epsilon > 0 \) and for every \( \epsilon > 0 \). Let us choose any \( t' > t_2 \) we now consider the following integral

\[
A_1 \leq \frac{(\mu + \omega + p \zeta)}{[\mu + \omega + p \zeta + 1(1 - \theta)]} H(0) \int_0^t \int_0^{t_1} e^{-t_1} e^{-2} e^{-2} e^{-2} d\tau d\sigma
\]

\[
+ \frac{(\mu + \omega + p \zeta)}{[\mu + \omega + p \zeta + 1(1 - \theta)]} \Gamma (a - 1) \frac{H(0) e}{(a + \omega + p \zeta + 1(1 - \theta))} + \frac{(\mu + \omega + p \zeta)}{[\mu + \omega + p \zeta + 1(1 - \theta)]} \Gamma (a - 1) \frac{(e_0 + \epsilon)}{\epsilon}
\]

Again from Eq. (A.11), we have:

\[
A_2 \leq \frac{(\mu + \omega + p \zeta)}{[\mu + \omega + p \zeta + 1(1 - \theta)]} H(0) \int_0^t \int_0^{t_1} e^{-t_1} e^{-2} e^{-2} e^{-2} d\tau d\sigma
\]

\[
+ \frac{(\mu + \omega + p \zeta)}{[\mu + \omega + p \zeta + 1(1 - \theta)]} \Gamma (a - 1) \frac{H(0) e}{(a + \omega + p \zeta + 1(1 - \theta))} + \frac{(\mu + \omega + p \zeta)}{[\mu + \omega + p \zeta + 1(1 - \theta)]} \Gamma (a - 1) \frac{(e_0 + \epsilon)}{\epsilon}
\]

Let us choose,

\[
\tau = t - z_1, \ \tau = -s = -z_2, \ \text{and} \ t_1 = z_3, \ \text{and} \ t_1 = z_4, \ \text{we have} \ \exists t_2 \ \text{such that} \ \lim_{t_\to \infty} \int_0^t e^{-\omega z_2} d\sigma < \epsilon, \ \forall t > t_2, \ \exists t_2 \ \text{such that} \ \lim_{t_\to \infty} \int_0^t e^{-\omega z_2} d\sigma < \epsilon, \ \forall t > t_2, \ \text{and for every} \ \epsilon > 0 \ \text{if we choose any} \ t' > t_2, \ \text{we now consider the following integral:}
\]

\[
A_2 \leq \frac{(\mu + \omega + p \zeta)}{[\mu + \omega + p \zeta + 1(1 - \theta)]} H(0) \int_0^t \int_0^{t_1} e^{-t_1} e^{-2} e^{-2} e^{-2} d\tau d\sigma
\]

\[
+ \frac{(\mu + \omega + p \zeta)}{[\mu + \omega + p \zeta + 1(1 - \theta)]} \Gamma (a - 1) \frac{H(0) e}{(a + \omega + p \zeta + 1(1 - \theta))} + \frac{(\mu + \omega + p \zeta)}{[\mu + \omega + p \zeta + 1(1 - \theta)]} \Gamma (a - 1) \frac{(e_0 + \epsilon)}{\epsilon}
\]
\[ A_3 = U(0) \int_0^\tau \frac{L(t)}{N(t) - \theta L(t)} e^{-(\mu + \sigma)(t-\tau)} \int_0^\tau e^{-a_1(t-s)}(\tau - s)^\rho e^{a_1_s}ds d\tau + \sigma \int_0^\tau \frac{L(t)}{N(t) - \theta L(t)} e^{-(\mu + \sigma)(t-\tau)} \int_0^\tau e^{-a_1(t-s)}(\tau - s)^\rho e^{a_1_s}ds d\tau. \] 

Then similarly as above, we have:

\[ A_3 \leq \frac{l \Gamma(a-1)}{[\mu + \omega + p \zeta + l(1-\theta)]} U(0) \int_0^\tau e^{-(\mu + \sigma)(t-\tau)} \int_0^\tau e^{-a_1(t-s)}(\tau - s)^\rho e^{a_1_s}ds d\tau \]

Now using \( A_1, A_2 \) and \( A_3 \) in Eq. (A.10), we have:

\[ E(t) \leq \frac{E(0)e^{-(\mu + \sigma)t} + \beta^2_h}{\Gamma(\alpha - 1)} A_1(t) + \beta^2_p \rho \frac{H(t)}{\alpha - 1} A_2 \frac{1}{\Gamma(\alpha - 1)} A_3(t). \]

where,

\[ K = \left[ E(0) + \frac{\beta^2_h}{\alpha + \sigma} H(0) \right]. \]

We discretize the models (2.10) and (2.17), using a nonstandard finite difference scheme [64]. Following [64], the system (2.10) is discretized below:

Appendix B. Numerical discretization

We discretize the models (2.10) and (2.17), using a nonstandard finite difference scheme [64]. Following [64], the system (2.10) is discretized below:
\[
\begin{align*}
-h(\mu + \sigma) E_p(i + 1), \\
U_j(i + 1) &= U_j(i) + h\sigma E_1(i) - h\sigma U_1(i) + 1, \\
H_j(i + 1) &= H_j(i) + h_2 U_j(i) - h_2 H_j(i) + 1, \\
R_j(i + 1) &= R_j(i) + h_2 U_j(i) + h_2^* [S_j(i) + L_1(i)] + \left[ \gamma_2 + \nu_2(g - 1) \right] H_j(i) - (\mu + \delta + \omega_2) R_j(i) + 1, \\
M_j(i + 1) &= M_j(i) + \frac{h\mu H_j(i)}{1 + H_j(i)} - h d M_j(i) + 1, \tag{B.1}
\end{align*}
\]

\[
\begin{align*}
S_p(i + 1) &= S_p(h + H + h a L_p(i)) - h \mu S_p(i + 1) - \frac{h\mu M_p(i)}{1 + M_p(i)} S_p(i + 1) - \left( \frac{h\mu}{N_p(i)} - \frac{1}{N_p(i)} \right) S_p(i + 1) \\
&= -h \frac{\mu^2}{N_p(i)} S_p(i + 1) - h(\mu + 1) S_p(i + 1) + h|1 - \sigma_\omega| R_p(i), \\
L_p(i + 1) &= L_p(i) + h S_p(i) - h \mu L_p(i + 1) - \left( \frac{h\mu M_p(i)}{1 + M_p(i)} \right) S_p(i + 1) - \frac{h\beta_1 (1 - \theta) U_p(i)}{N_p(i) - \theta L_p(i)} - h(\mu + \omega_1) L_p(i + 1) \\
E_p(i + 1) &= E_p(i) + \frac{h\mu U_p(i)}{N_p(i) - \theta L_p(i)} S_p(i + 1) + \frac{h\beta_1 (1 - \theta U_p(i))}{N_p(i) - \theta L_p(i)} L_p(i + 1) \\
&= -h \frac{\mu^2}{N_p(i)} S_p(i + 1) + \frac{h\beta_1 (1 - \theta U_p(i))}{N_p(i) - \theta L_p(i)} L_p(i + 1) \\
U_j(i + 1) &= U_j(i) + h\sigma E_1(i) - a_2 U_1(i) + 1, \\
H_j(i + 1) &= H_j(i) + h_2 U_j(i) - a_2 H_j(i) + 1, \\
R_j(i + 1) &= R_j(i) + h_2 U_j(i) + h_2^* [S_j(i) + L_1(i)] + \left[ \gamma_2 + \nu_2(g - 1) \right] H_j(i) - (\mu + \delta + \omega_2) R_j(i) + 1, \\
M_j(i + 1) &= M_j(i) + \frac{h\mu H_j(i)}{1 + H_j(i)} - h d M_j(i) + 1. \tag{B.2}
\end{align*}
\]

Appendix C

Using the relation between the Riemann–Liouville fractional derivative and the Caputo derivative [45] and from Eq. (2.10), we have:

\[
\begin{align*}
\mathcal{D}_t^{\alpha}\phi(t) &= \frac{1}{\Gamma(\alpha)} \int_0^t (t-s)^{\alpha-1} \phi^{(\alpha)}(s) ds + \frac{U(0)^{\alpha-1}}{\Gamma(\alpha)}, \\
\mathcal{D}_t^{\alpha}\phi(t) &= \frac{\sigma}{\Gamma(\alpha)} \int_0^t (t-s)^{\alpha-1} \phi^{(\alpha)}(s) ds + \frac{U(0)^{\alpha-1}}{\Gamma(\alpha)}, \tag{C.1}
\end{align*}
\]

\[
\begin{align*}
\mathcal{D}_t^{\alpha}\phi(t) &= \frac{1}{\Gamma(\alpha)} \int_0^t (t-s)^{-1} \phi^{(\alpha)}(s) ds + \frac{U(0)^{\alpha-1}}{\Gamma(\alpha)}, \\
\mathcal{D}_t^{\alpha}\phi(t) &= \frac{\gamma_2}{\Gamma(\alpha)} \int_0^t (t-s)^{\alpha-1} \phi^{(\alpha)}(s) ds + \frac{H(0)^{\alpha-1}}{\Gamma(\alpha)}. \tag{C.2}
\end{align*}
\]

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