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Caputo fractional-order SEIRP model for COVID-19 Pandemic

Saheed O. Akindeinde\textsuperscript{a,b,}\textsuperscript{*}, Eric Okyere\textsuperscript{c}, Adebayo O. Adewumi\textsuperscript{a}, Ramoshewu S. Lebelo\textsuperscript{d}, Olanrewaju O. Fabelurin\textsuperscript{a}, Stephen E. Moore\textsuperscript{e}

\textsuperscript{a} Department of Mathematics, Obafemi Awolowo University, 220005 Ile-Ife, Nigeria
\textsuperscript{b} Department of Mathematics and Statistical Sciences, Botswana International University of Science and Technology (BIUST), Private Bag 016, Palapye, Botswana
\textsuperscript{c} Department of Mathematics and Statistics, University of Energy and Natural Resources, Ghana
\textsuperscript{d} Faculty of Human Sciences, Vaal University of Technology, Vanderbijlpark, South Africa
\textsuperscript{e} Department of Mathematics, University of Cape Coast, Cape Coast, Ghana

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Abstract We propose a Caputo-based fractional compartmental model for the dynamics of the novel COVID-19 pandemic. The newly proposed nonlinear fractional order model is an extension of a recently formulated integer-order COVID-19 mathematical model. Using basic concepts such as continuity and Banach fixed-point theorem, existence and uniqueness of the solution to the proposed model were shown. Furthermore, we analyze the stability of the model in the context of Ulam-Hyers and generalized Ulam-Hyers stability criteria. The concept of next-generation matrix was used to compute the basic reproduction number $R_0$, a number that determines the spread or otherwise of the disease into the general population. We also investigated the local asymptotic stability for the derived disease-free equilibrium point. Numerical simulation of the constructed epidemic model was carried out using the fractional Adam-Bashforth-Moulton method to validate the obtained theoretical results.

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1. Introduction

In the first quarter of 2020, the World Health Organization (WHO) declared COVID-19 as a pandemic that had affected several countries in all continents, see [1]. The novel coronavirus has several mutations that have occurred in many countries with varied symptoms. Some of the most common symptoms are fever, dry cough, and tiredness whiles other less common symptoms include body aches and pains, sore throat,
functions that are of Volterra–type and investigated uniform extended and studied the cholera epidemic mathematical equations to confirm the obtained theoretical results. Javidi et al. presented mathematical model and also presented numerical simulations to establish the existence and uniqueness of solutions for the differential order derivative. The author in [77] used Lyapunov functions to analytically solve their proposed model and compared it with the initial 67 days reported data on confirmed infected and death cases in Wuhan city. In recent studies, the authors in [96] proposed a Caputo-type nonlinear COVID-19 epidemiological model to explore the significance of lockdown dynamics in controlling the spread of the infectious disease. They further presented the uniqueness and existence of solutions for the mathematical model under lockdown using Banach and Schauder fixed theorems. In [97], a susceptible-exposed-infected-isolated-recovered compartmental modeling framework with constant total population dynamics is used to construct a new SEIQR Caputo fractional order COVID-19 mathematical model. Owusu-Mensah and co-authors [98], have also studied a new COVID-19 epidemic model using Caputo fractional order differential operators to describe the transmission dynamics of Rubella epidemic in Pakistan.

Due to the powerful nature of fractional order differential equations and their derivative operators used in constructing realistic mathematical representations of real-world problems in science, finance, and engineering [82–88], some recent studies have considered the mathematical modeling of this deadly COVID-19 pandemic using some of these useful derivative operators. Naik et al. [89] provided a detailed qualitative analysis as well as parameter estimation and numerical simulations for a nonlinear COVID-19 epidemiological model constructed by Caputo and Atangana-Baleanu fractional derivative operators. In another study [90], the authors formulated an autonomous nonlinear Atangana-Baleanu fractional order differential equation model to study the COVID-19 epidemic in Nigeria. Baleanu et al. [91] in their new study considered an extended version of the integer-order epidemic model proposed and analyzed by Chen et al. [92] to include Caputo-Fabrizio derivative. With the use of fixed point theory, they proved that the nonlinear Caputo-Fabrizio fractional order COVID-19 model has a unique solution. They also applied the homotopy analysis transform method and generated the approximate solution for the model problem in convergent series. A Caputo fractional order deterministic epidemic model for COVID-19 infection is developed and studied in [93]. They used the well-known Banach contraction mapping principle to establish the existence and uniqueness of the solution for the mathematical model. In a new mathematical modeling study [94], the authors proposed a SEIQRDP fractional order deterministic model characterized by Caputo derivative to examine the novel coronavirus epidemic. A SEIPAHRF Caputo fractional order compartmental model is proposed to analyze COVID-19 transmission dynamics in Wuhan [95]. They numerically solved their proposed model and compared it with the initial 67 days reported data on confirmed infected and death cases in Wuhan city. In recent studies, the authors in [96] proposed a Caputo-type nonlinear COVID-19 epidemiological model to explore the significance of lockdown dynamics in controlling the spread of the infectious disease. They further presented the uniqueness and existence of solutions for the mathematical model under lockdown using Banach and Schauder fixed theorems. In [97], a susceptible-exposed-infected-isolated-recovered compartmental modeling framework with constant total population dynamics is used to construct a new SEIQR Caputo fractional order COVID-19 mathematical model. Owusu-Mensah and co-authors [98], have also studied a new COVID-19 epidemic model using Caputo fractional order differential operators to describe the transmission dynamics of Rubella epidemic in Pakistan.
derivative in fractional calculus. The well-known and powerful generalized form of Adams Bashforth–Moulton iterative scheme was applied to numerically solve their formulated nonlinear fractional order differential equation model. In another study, the authors in [99] used an extended version of the classical SEIR epidemic model to construct a data-driven Caputo-based COVID-19 fractional order mathematical model.

Our motivation for this present study is based on the aforementioned literature on the application of fractional order differential equations in modelling nonlinear systems related to real-world problems and more especially infectious diseases’ transmission dynamics. Recent research works and studies in literature extensively demonstrate or reveals that mathematical modelling of nonlinear systems with fractional order differential operators gives more realistic results than the classical integer-order based models, see, e.g., [81,89,100–102]. We therefore apply the well-known and reliable Caputo derivative operator in fractional calculus to extend an existing COVID-posedness of the integer-order model in [103], and obtain dis-in Section 3 both mathematical and epidemiological well-posedness of the integer-order model proposed in [103] characterized by the classical integer order derivative. The goal of the present paper is two folds, first, we want to establish both the mathematical and epidemiological well-posedness of the integer-order model proposed in [103] and employ an approximate analytical technique to obtain long-term dynamics of the disease. Second, we modify and extend the existing epidemic model using dimensionally consistent Caputo derivative operator which has been extensively demonstrated in the literature to be one of the useful and powerful derivative operators to describe more efficiently memory effect dynamics that exist in real-world phenomena. It is important to mention that, our study is further motivated by the recent research works conducted by the authors in [104–106].

The paper is organized as follows. Following the essential preliminaries on fractional calculus in Section 2, we examine in Section 3 both mathematical and epidemiological well-posedness of the integer-order model in [103], and obtain disease dynamics using the approximate analytical technique proposed in [107,108]. Section 4 of the paper is devoted to the formulation, well-posedness, local and global stability analysis of the fractional order model. Therein, using basic concepts such as continuity and Banach fixed-point theorem, the existence and uniqueness of the solution to the proposed model were shown. Furthermore, we discussed stability analysis of the model in the context of Ulam-Hyers and generalized Ulam-Hyers stability criteria. Also, the concept of next-generation matrix was used to compute the basic reproduction number $R_0$, a number that determines the spread or otherwise of the disease into the general population. We conclude the paper with numerical simulation of the fractional order disease model using fractional Adam-Bashforth–Moulton; and discussion of results.

2. Preliminaries on fractional calculus

In this section, we introduce well-known definitions and Lemma in fractional calculus that are relevant to the current article. The interested reader can see the monograph [109] and the article [110] for the proofs and further references.

Definition 2.1 [109]. The fractional order integral of the function $g \in L^1([0,b],\mathbb{R}^+)$ of order $v \in \mathbb{R}^+$ is defined in the sense of Riemann-Liouville as

$$^C\mathcal{I}_t^v g(t) = \frac{1}{\Gamma(v)} \int_0^t (t-\tau)^{v-1} g(\tau) d\tau, \quad t > 0,$$

where $[0,b] \subset \mathbb{R}^+$ and $\Gamma$ is the Euler gamma function defined by $\Gamma(v) = \int_0^\infty t^{v-1} e^{-t} dt$, $v > 0$.

Definition 2.2 [109]. For a function $g \in C^n([0,b])$, the Caputo fractional order derivative of order $v$ is defined by

$$D^v g(t) = \frac{1}{\Gamma(n-v)} \int_0^t (t-\tau)^{n-v-1} g^{(n)}(\tau) d\tau, \quad t > 0,$$

where $n = \lfloor v \rfloor + 1$ and $\lfloor v \rfloor$ denotes the smallest integer that is less or equal to $v$.

Lemma 2.3 [109]. Let $v \geq 0$ and $n = [v] + 1$. Then

$$^C\mathcal{I}_t^v (D^v g(t)) = g(t) - \frac{v}{k!} \sum_{k=0}^{n-1} \frac{g^{(k)}(0)}{\Gamma(k+1)} t^k.$$

In particular, if $0 < v \leq 1$, then

$$^C\mathcal{I}_t^v (D^v g(t)) = g(t) - g_0.$$  

Lemma 2.4 [110] Generalized Mean Value Theorem. For $0 < v \leq 1$, let $g(t) \in C([a,b])$ and $D^v g(t) \in (a,b)$. Then it holds

$$g(t) = g(a) + \frac{1}{\Gamma(v)} D^v g(\eta)(t-a)^v, \quad 0 \leq \eta \leq t, \quad \forall t \in (a,b).$$

3. Model framework: Integer order model

It is well known that the ravaging Covid-19 virus is transmitted through human-to-human interactions and transmission through the environment. When an infected person sneezes or coughs, the virus is released into the immediate environment which, experts believe, stays viable for as long as five days. We, therefore, consider two interacting populations of humans and pathogens denoted by $N(t)$ and $P(t)$ respectively. At any time $t$, the total population $N(t)$ is assumed to comprise the susceptible population $S(t)$, the exposed $E(t)$, the asymptomatic infected $I_A(t)$, the symptomatic infected $I_S(t)$ and the recovered population $R(t)$. Thus, $N(t) = S(t) + I_A(t) + I_S(t) + R(t)$. These interactions are depicted in Fig. 1 below as reported in [103]. In the figure, $b$ denotes the rate at which the human population is born into susceptible class $S(t)$. The terms $\frac{\beta S}{N(t)}$ and $\frac{\beta I_A(t)}{N(t)}$ represent rate at which the susceptible get infected by pathogens and through interactions with infectious asymptomatic $I_A(t)$ and infectious symptomatic $I_S(t)$. The denominators in the above terms factor in the adherence to recent experts’ advice on social distancing and the use of face masks which minimizes contact with infectious individuals, and transmission through the environment.
Based on the above description, the authors in [103] proposed the following integer-order differential equation to model the dynamics of the transmission of COVID-19 taking into account environment and social distancing, namely

\[
\begin{align*}
S(t) &= b - \frac{\beta S(t)P(t)}{1 + \alpha P(t)} + \psi E - \mu S, \\
E(t) &= \frac{\beta S(t)P(t)}{1 + \alpha P(t)} - \psi E - \mu E - \omega E, \\
I_A(t) &= (1 - \delta)\omega E - (\mu + \sigma)I_A - \gamma s I_A, \\
I_S(t) &= \delta \omega E - (\mu + \sigma)I_S - \gamma s I_S, \\
P(t) &= \eta_A I_A + \eta_S I_S - \mu P,
\end{align*}
\]

with initial conditions \( S(0) = S_0 > 0, E(0) = E_0 > 0, I_A(0) = I_{A0} > 0, I_S(0) = I_{S0} > 0, R(0) = R_0 > 0 \). Therein, the authors derived the disease-free equilibrium point and basic reproduction number of the model, and performed numerical simulation. However, the analysis in article [103] did not examine the essential well-posedness of the proposed model. Here in the present paper, for completeness, we begin our exposition by establishing qualitative properties of the integer order model which ensures that the model is both mathematically and epidemiological well-posed i.e. existence, uniqueness, positivity and boundedness of the solutions to the model. Furthermore, establishing the aforementioned properties allows us to extend the study in [103] to fractional order model, which accommodate the memory effect typically associated with epidemic disease models. In addition, we shall obtain approximate analytical solutions of the model via a multistage method proposed in [107,108].

### 3.1. Well-posedness

For the fact that (2) models human populations, all the model parameters are assumed non-negative, see Table 1 for values and description of the model parameters. It then remains to show that unique solution exists for the model and that the state variables remain bounded and non-negative for all time \( t > 0 \).

**Theorem 3.1 (Existence and uniqueness of solution).** Let \( t_f \in \mathbb{R}^+ \). The dynamical system (2) admits a unique solution on interval \((0,t_f)\) for initial conditions satisfying \( S(0) > 0, E(0) > 0, I_A(0) > 0, I_S(0) > 0, R(0) > 0, P(0) > 0 \).

**Proof.** By defining \( y(t) = (S(t), E(t), I_A(t), I_S(t), R(t), P(t))^T \), then (2) can be written as \( y'(t) = F(y(t)) = (f_1, f_2, f_3, f_4, f_5, f_6)^T \) where the \( f_i \) are functions of \( y(t) \). By assumption the initial condition \( y(0) = (S(0), E(0), I_A(0), I_S(0), R(0), P(0))^T \) \( > 0 \). Thus computing and examining the entries of the Jacobian \( J(F(y)) \), e.g.,

\[
\begin{align*}
J_{11} &= \frac{\partial f_1}{\partial S} = \frac{\beta P}{1 + \alpha P} - \frac{\beta P I_A + I_S}{1 + \alpha P}, \\
J_{12} &= \frac{\partial f_1}{\partial E} = \psi, \\
J_{13} &= \frac{\partial f_1}{\partial I_A} = -\frac{\beta S}{(1 + \alpha P)} = J_{14} = \frac{\partial f_1}{\partial I_S}, \\
J_{15} &= \frac{\partial f_1}{\partial R} = 0, \\
J_{16} &= \frac{\partial f_1}{\partial P} = -\frac{\beta P}{(1 + \alpha P)}, \quad \text{etc}
\end{align*}
\]
Table 1 Model parameters and their descriptions [103].

| Parameter description                                      | Symbol | Value                  |
|-----------------------------------------------------------|--------|------------------------|
| Birth rate of the human population                        | b      | 0.00018 days⁻¹         |
| Natural human death rate                                   | μ      | 4.563 × 10⁻⁵ days⁻¹    |
| Human life expectancy                                      | 1/μ    | 60 years               |
| Natural death rate of pathogens in the environment        | μₚ     | 0.1724 days⁻¹          |
| Proportion of interaction with an infectious environment   | z₁     | 1.00                   |
| Proportion of interaction with an infectious individual    | z₂     | 0.10                   |
| Rate of transmission from S to E due to contact with P     | β₁     | 0.00414                |
| Rate of transmission from S to E due to contact with I₄ and/or I₅ | β₂     | 0.0115                 |
| Proportion of symptomatic infectious people                | δ      | 0.7                    |
| Progression rate from E back to S due to robust immune system | ψ      | 0.0051                 |
| Progression rate from E to either I₄ or I₅                 | ω      | 0.09                   |
| Death rate due to the coronavirus                          | σ      | 0.0018                 |
| Rate of recovery of the symptomatic population            | γₛ     | 0.05 days⁻¹            |
| Rate of recovery of the asymptomatic human population     | γ₄     | 0.0714 days⁻¹          |
| Rate of virus spread to environment by symptomatic infectious individuals | ηₛ   | 0.1 days⁻¹             |
| Rate of virus spread to environment by asymptomatic infectious individuals | η₄   | 0.1 days⁻¹             |

reveal that both the right hand side of (2) namely F and its Jacobian are continuous for t > 0. Thus, F satisfies a Lipschitz condition on Rₖ³. The existence and uniqueness of solution for some time interval (0, t₁) follows from Picard-Lindelof Theorem.

Theorem 3.2 (Positivity of solution). The state variables S(t), E(t), I₄(t), I₅(t), R(t), P(t) of (2) with non-negative initial data remains non-negative for all t > 0.

Proof. Let S(0) = S₀ > 0. It follows from the first equation of (2) that

\[
\frac{dS}{dt} \geq \left( \frac{β₁ P}{1 + z₁ P} + \frac{β₂ (I₄ + I₅)}{1 + z₂ (I₄ + I₅) + μ} \right) S,
\]

and upon integration, one obtains

\[
S(t) \geq S₀ e^{-(μ + δ) t} > 0,
\]

for all t > 0 where

\[
δ = \int₀^t \left( \frac{β₁ P(τ)}{1 + z₁ P(τ)} + \frac{β₂ (I₄(τ) + I₅(τ))}{1 + z₂ (I₄(τ) + I₅(τ))} \right) dτ.
\]

Similar arguments yield

\[
E(t) \geq E(0)e^{-(μ + δ) t} > 0,
\]

\[
I₄(t) \geq I₄(0)e^{-(μ + δ) t} > 0,
\]

\[
I₅(t) \geq I₅(0)e^{-(μ + δ) t} > 0,
\]

\[
R(t) \geq R(0)e^{−μt} > 0,
\]

\[
P(t) \geq P(0)e^{−δt} > 0.
\]

and as a consequence, non-negativity of the remaining state variables are obtained. □

Theorem 3.3 (Boundedness of solution). The integer order model (2) has solutions bounded within invariant region given by

\[
Ω = \left\{ (S, E, I₄, I₅, R, P) \in \mathbb{R}^6 : 0 \leq N(t) \leq \frac{b}{μ}, 0 \leq P(t) \leq \frac{ηb}{μμₚ} \right\},
\]

where η = η₄ + η₅.

Proof. Using the fact that N(t) = S(t) + E(t) + I₄(t) + I₅(t) + R(t), it follows from (2) that

\[
N'(t) = b - (μ + σ)(I₄ + I₅) - μR - S - E,
\]

\[
= b - μN - σ(I₄ + I₅),
\]

\[
\leq b - μN.
\]

Thus considering the initial valued problem N'(t) = b − μN, N(0) = N₀, and invoking comparison theorem [111], it follows that

\[
N(t) \leq N₀e^{−μt} + \frac{b}{μ}(1 - e^{−μt}),
\]

and consequently

\[
\limsup_{t \to \infty} N(t) \leq \frac{b}{μ}.
\]

For the pathogen population, using the fact that I₄ + I₅ ≤ N ≤ b/μ, the last equation of (2) yields

\[
P'(t) = η₄ I₄ + η₅ I₅ - μₚ P,
\]

\[
\leq η(I₄ + I₅) - μₚ P, \quad η = η₄ + η₅,
\]

\[
\leq η \frac{b}{μ} - μₚ P,
\]

and consequently P(t) ≤ η₀ e⁻μₚt + \frac{b}{μₚ} (1 − e⁻μₚt). Thus

\[
\limsup_{t \to \infty} P(t) \leq \frac{b}{μₚ}. \quad □
\]

3.2. Approximate analytical solution of the integer-order model

Here, we shall adopt the multistage technique proposed in [107, 108] to compute approximate analytical solution to the integer-order model (2). For that purpose, let us define

\[
U = \frac{1}{1 + μₚ} \text{ and } W = \frac{1}{1 + μₚ (μₚ + μₜ)}
\]

so that (2) is transformed to an equivalent polynomial system


\[ S'(t) = b - \beta_1 S(t) P(t) - \beta_2 S(t) I_4(t) + I_4(t) W(t) + \psi E - \mu S, \]

\[ E'(t) = \beta_1 S(t) P(t) + \beta_2 S(t) I_4(t) - I_4(t) W(t) - \psi E - \mu E - \omega E, \]

\[ I_4'(t) = (1 - \delta) \omega E - (\mu + \sigma) I_4(t) - \gamma_4 I_4, \]

\[ I_5'(t) = \delta \omega E - (\mu + \sigma) I_5(t) - \gamma_5 I_5, \]

\[ R'(t) = \gamma_5 I_5 + \gamma_4 I_4 - \mu R, \]

\[ P'(t) = \eta_4 I_4 + \eta_5 I_5 - \mu P, \]

\[ U'(t) = -\rho \frac{E(t) S(t)}{S(t) + \rho} (\eta_4 I_4 + \eta_5 I_5 - \mu P), \]

\[ W'(t) = -\rho \frac{E(t) S(t)}{S(t) + \rho} (\eta_4 I_4 + \eta_5 I_5 - \mu P), \]

which is now amenable to the proposed technique. By writing \( S(t) = \sum_{n=0}^{\infty} S_n t^n \), it follows from the above polynomial system that the coefficients \( S_n \) are obtained recursively through:

\[(n + 1) S_n = b - \beta_1 (S(t) P(t) + \beta_2 (S(t) I_4(t) W(t) + \psi E - \mu S), S_0 = S(0).\]

In a similar manner, approximate analytical solutions of the remaining compartments are computed. Such series solution often have small interval of convergence. Therefore to improve convergence, we choose a safe step length \( h \), (see [107,108]) and compute a piecewise continuous approximate solution which is convergent in the entire integration interval. The dynamics of the disease progression for each sub-population are displayed in Figs. 2a–3b and 4a, b below, confirming the results in [103].

4. Fractional order model

Fractional derivatives are generally believed to model disease epidemics more realistically because of their capability to capture the memory effect often associated with the human body’s response to diseases. Here, we propose a fractional order variant of (2) given by

\[ D^\alpha_t S(t) = b - \beta_1 S(t) P(t) - \beta_2 (S(t) I_4(t) + I_4(t) W(t) + \psi E - \mu S, \]

\[ D^\alpha_t E(t) = \beta_1 S(t) P(t) + \beta_2 S(t) I_4(t) - I_4(t) W(t) - \psi E - \mu E - \omega E, \]

\[ D^\alpha_t I_4(t) = (1 - \delta) \omega E - (\mu + \sigma) I_4(t) - \gamma_4 I_4, \]

\[ D^\alpha_t I_5(t) = \delta \omega E - (\mu + \sigma) I_5(t) - \gamma_5 I_5, \]

\[ D^\alpha_t R(t) = \gamma_5 I_5 + \gamma_4 I_4 - \mu R, \]

\[ D^\alpha_t P(t) = \eta_4 I_4 + \eta_5 I_5 - \mu P, \]

subject to initial conditions \( S(0) = S_0 > 0, E(0) = E_0 > 0, I_4(0) = I_{40} > 0, I_5(0) = I_{50} > 0, R(0) = R_0 > 0 \). In the above, operator \( D^\alpha \) denotes Caputo fractional derivative of order \( 0 < \alpha \leq 1 \). Noting that all the model parameters except \( \alpha_1, \alpha_2 \) and \( \delta \) have dimensions \( 1/t \), we have raised these parameters to power of \( \alpha \) for dimensional consistency emphasized by [59].

4.1. Qualitative properties of solution

In this section, we examine the mathematical and biological well-posedness of the fractional order model. In essence, we prove that solution of the fractional model is bounded and remains positive as long as a positive initial condition is given. Furthermore, we prove the existence and uniqueness of the solution to the modified model.

Let \( X(t) = (S(t), E(t), I_4(t), I_5(t), R(t), P(t))^T \) and \( \mathbf{X}(t, X(t)) = (\phi_i)^T, i = 1, 2, \ldots, 6 \) where

\[ \phi_1 = b - \beta_1 S(t) P(t) - \beta_2 (S(t) I_4(t) + I_4(t) W(t) + \psi E - \mu S, \]

\[ \phi_2 = \beta_1 S(t) P(t) + \beta_2 S(t) I_4(t) - I_4(t) W(t) - \psi E - \mu E - \omega E, \]

\[ \phi_3 = (1 - \delta) \omega E - (\mu + \sigma) I_4(t) - \gamma_4 I_4, \]

\[ \phi_4 = \delta \omega E - (\mu + \sigma) I_5(t) - \gamma_5 I_5, \]

\[ \phi_5 = \gamma_5 I_5 + \gamma_4 I_4 - \mu R, \]

\[ \phi_6 = \eta_4 I_4 + \eta_5 I_5 - \mu P. \]

![Fig. 2](image-url) Solution paths for \( S(t), E(t) \) for the integer-order model.
Then the dynamical system (5) can be written as
\[ D_a^t X(t) = \mathcal{N}(t, X(t)), \quad X(0) = X_0 \geq 0, \quad t \in [0, b], \quad 0 < a \leq 1. \]

(6)

In the above, the condition \( X(0) \geq 0 \) is to be interpreted component-wise. Problem (6), which is equivalent to fractional differential Eq. (5), in turn has integral representation
\[ X(t) = X_0 + \frac{1}{\Gamma(a)} \int_0^t (t-\tau)^{a-1} \mathcal{N}(\tau, X(\tau)) d\tau. \]

(7)

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(7)

Next, we shall analyse model (5) through the integral representation above. For that purpose, let \( \mathcal{E} = C([0, b]; \mathbb{R}) \) denote the Banach space of all continuous functions from \([0, b]\) to \(\mathbb{R}\) endowed with the norm
\[ \|X\|_{\mathcal{E}} = \sup_{t \in [0,b]} |X(t)|. \]

where
\[ |X(t)| = |S(t)| + |E(t)| + |I_A(t)| + |I_S(t)| + |R(t)| + |P(t)|. \]

Note that \( S, E, I_A, I_S, R, P \) all belong to \( C([0, b]; \mathbb{R}) \). Furthermore, we define the operator \( P : \mathcal{E} \to \mathcal{E} \) by
\[(PX)(t) = X_0 + \frac{1}{\Gamma(2)} \int_0^t (t-z)^{t-1} \cdot \mathcal{K}(\tau, X(\tau)) d\tau.\]  
(8)

Note that operator \(P\) is well-defined due to the obvious continuity of \(\mathcal{K}\).

**4.1.1. Positivity and boundedness of solution**

For the fractional-order model to be biologically well-posed, its solution is expected to be positive and bounded at all times. These properties are established in the sequel.

**Theorem 4.1.** Let \(X(t) = (S(t), E(t), I_s(t), I_a(t), R(t), P(t))^T\). Then for \(X(0) > 0\), the solution \(X(t)\) of (5) is bounded, and remains positive for \(t \geq 0\).

**Proof.** We start by establishing positivity of solution. Following [112], consider the trajectory of solution along the \(S\)-axis where \(E(0) = I_a(0) = I_s(0) = R(0) = P(0)\), and \(S(0) = S_0 > 0\). Then \(D^\alpha_S(t) > b^\alpha - \mu^S\). \(S(0) = S_0\) whose solution is given by \(S(t) = S_0 e^{-\mu^S t} + \int_0^t (1 - e^{-\mu^S t}) dt > 0\). Since \(E(0) > 0\), \(I_a(0) > 0\), \(I_s(0) > 0\), \(R(0) > 0\), \(P(0) > 0\), similar arguments yield

\[E(t) = E(0) e^{-\left(\psi^E + \mu^E + \sigma^E\right) t},\]
\[I_a(t) = I_a(0) e^{-\left(\psi^I_a + \sigma^I_a + \gamma^I_a\right) t},\]
\[I_s(t) = I_s(0) e^{-\left(\psi^I_s + \mu^I_s + \gamma^I_s\right) t},\]
\[R(t) = R(0) e^{-\left(\mu^R + \gamma^R\right) t},\]
\[P(t) = P(0) e^{-\left(\mu^P + \gamma^P\right) t} > 0,\]

showing non-negative invariance of the axes.

Now, since the solution to the model (5) is positive in the \(E - I_a - I_s - R - P\) plane, let \(t^* > 0\) such that \(S(t^*) = 0\), \(E(t^*) > 0\), \(I_a(t^*) > 0\), \(I_s(t^*) > 0\), \(R(t^*) > 0\), \(P(t^*) > 0\), and \(S(t^*) < S(t^*)\). On this plane,

\[D^\alpha_S(t)|_{t=t^*} = b^s > 0.\]  
(9)

By Caputo fractional mean value theorem (see **Lemma 2.4.**), it holds \(S(t^*) - S(t^*) = \frac{1}{\Gamma(\alpha)} \int_{t^*}^t (t)\). Therefore, using (9), we obtain \(\hat{\mathcal{S}}(t^*) > S(t^*)\), contradicting our earlier assumption for \(t^*\). Thus, any solution \(S(t)\) is non-negative for all \(t \geq 0\). The remaining variables can be treated similarly. Hence, solution \(X(t)\) remains positive for all \(t \geq 0\).

Finally, for boundedness, proceeding as in the integer order case (see the proof of **Theorem 3.3.**), one obtains \(N(t) \leq N_0 E_0 (\mu^E t^2) + \frac{C^E}{\Gamma(1)} (1 - E_0 (\mu^E t^2))\) and consequently \(\lim_{t \to \infty} N(t) < \frac{\mu^E}{\Gamma(2)}\). Similarly, \(\lim_{t \to \infty} P(t) < \frac{\mu^P}{\Gamma(2)}\).

**4.1.2. Existence of unique and uniformly stable solution**

Here, we establish existence, uniqueness and uniform stability of solutions to 4.5. The following preliminary result is in order.

**Lemma 4.2.** Let \(\bar{X} = (\bar{S}, \bar{E}, \bar{I}_s, \bar{I}_a, \bar{R}, \bar{P})^T\). The function \(\mathcal{K} = (\phi_i)^T\) defined above satisfies

\[\|\mathcal{K}(t, X(t)) - \mathcal{K}(t, \bar{X}(t))\|_d \leq L_{\mathcal{K}}\|X - \bar{X}\|_d,\]

for some \(L_{\mathcal{K}} > 0\).

**Proof.** From the first component of \(\mathcal{K}\), we observe that

\[|\phi_1(t, X(t)) - \phi_1(t, \bar{X}(t))| = \left| \beta_1^1 \left\{ \left( \frac{S(t)P(t)}{S(t)+P(t)} - \frac{S(0)P(0)}{S(0)+P(0)} \right) - \frac{S(t)P(t)}{S(t)+P(t)} \right\} \right| + \psi^E |E(t) - E(t)| - \mu^E |S(t) - S(t)|,\]

\[\leq \left| \beta_1^1 \left\{ \left( \frac{S(t)P(t)}{S(t)+P(t)} - \frac{S(0)P(0)}{S(0)+P(0)} \right) - \frac{S(t)P(t)}{S(t)+P(t)} \right\} \right| + \psi^E |E(t) - E(t)| + \mu^E |S(t) - S(t)|.\]

However,

\[f_1(t) = |P(t)| + \frac{1}{|S(t)|} f_2(t) + |S(t)|,\]

\[f_2(t) = |S(t)| - \bar{S}(t) + f_3(t) |P(t) - \bar{P}(t)|,\]

where

\[f_1(t) = |P(t)| + \frac{1}{|S(t)|} f_2(t) + |S(t)|,\]

\[f_2(t) = |S(t)| - \bar{S}(t) + f_3(t) |P(t) - \bar{P}(t)|,\]

\[f_3(t) = |P(t)| + \frac{1}{|S(t)|} f_2(t) + |S(t)|,\]

Similarly, we obtain

\[g_1(t) = |I_a(t)| + 2 \bar{a} |I_a(t) - \bar{I}_a(t)| + 2 \bar{a} |I_s(t) - \bar{I}_s(t)| + g_2(t)\]

\[g_2(t) = |I_s(t)| + 2 \bar{a} |I_s(t) - \bar{I}_s(t)| + g_3(t),\]

\[g_3(t) = |I_a(t)| + 2 \bar{a} |I_a(t) - \bar{I}_a(t)| + 2 \bar{a} |I_s(t) - \bar{I}_s(t)| + g_4(t)\]

\[g_4(t) = |I_s(t)| + 2 \bar{a} |I_s(t) - \bar{I}_s(t)|,\]

Thus, altogether we have

\[|\psi^E |E(t) - E(t)| - \mu^E |S(t) - S(t)|,\]

\[= M_1(t) |E(t) - E(t)| + \mu^E |S(t) - S(t)|>,\]

\[\leq M_1(t) |E(t) - E(t)| + \mu^E |S(t) - S(t)| > 0,\]

where

\[L_1 = \mu^E + \psi^E + \alpha^E\]

\[+ \max_{a \in [0,\bar{a}]} \left\{ b_1^E |f_1(t) + b_2^E |f_2(t) + b_3^E |g_1(t) + b_4^E |g_2(t) + b_5^E |g_3(t) + b_6^E |g_4(t)\right\}.\]

In a similar manner, one obtains

\[|\phi_2(t, X(t)) - \phi_2(t, \bar{X}(t))| \leq L_2 |S(t) - \bar{S}(t)| + |E(t) - E(t)| + |I_a(t) - \bar{I}_a(t)| + |I_s(t) - \bar{I}_s(t)| + |P(t) - \bar{P}(t)|.,\]

where

\[L_2 = \mu^E + \psi^E + \alpha^E\]

\[+ \max_{a \in [0,\bar{a}]} \left\{ b_1^E |f_1(t) + b_2^E |f_2(t) + b_3^E |g_1(t) + b_4^E |g_2(t) + b_5^E |g_3(t) + b_6^E |g_4(t)\right\}.\]

For the remaining components of \(\mathcal{K}\), it holds

\[|\phi_3(t, X(t)) - \phi_3(t, \bar{X}(t))| \leq L_3 |E(t) - E(t)| + |I_a(t) - \bar{I}_a(t)| + |I_s(t) - \bar{I}_s(t)| + |\bar{R}(t) - \bar{R}(t)|.,\]

where

\[L_3 = |1 - \alpha^\sigma + \mu^\sigma + \sigma^\sigma + \gamma^\sigma + \mu^\gamma + \mu^\gamma|\]

\[L_3 = |1 - \alpha^\sigma + \mu^\sigma + \sigma^\sigma + \gamma^\sigma + \mu^\gamma + \mu^\gamma|\]

Consequently,
Theorem 3.2. Formally Lyapunov stability of solution follows from [114, Theorem 3.1].

Proof. Let $X_t : [0,b] \times \Gamma \rightarrow \mathbb{R}^b$ be clearly continuous on its domain. Thus, existence of solution to (5) follows from [113, Theorem 3.1].

For uniqueness, we shall use Banach contraction mapping principle on operator $P$ defined in (8) above. For that purpose, we show that $P$ is both a self map and a contraction. Firstly, by definition, $\sup_{x\in[0,b]} \left\|X(t,0)\right\| = b^\prime$. Let us now define $\kappa > \|X_0 + \Omega X\|_{\infty}$ and a closed convex set $B_\kappa = \{X \in \mathcal{E} : \|X\|_\kappa \leq \kappa\}$. Thus for self map property, it suffices to show that $PB_\kappa \subseteq B_\kappa$. So, let $X \in B_\kappa$, then

$$\|PX\|_\kappa \leq \sup_{n \in [0,b]} \left\{ \left| \int_{t_n}^{t_{n+1}} \left( \mathcal{F}(t, X(t)) - \mathcal{F}(t, X(t)) \right) dt \right| \right\}$$

Let us now define $\mathcal{F}(t, X(t)) = \mathcal{F}(t, 0)$, where $\mathcal{F}(t, X(t)) = \mathcal{F}(t, X(t))$ and $\mathcal{F}(t, 0) = \mathcal{F}(t, 0)$. Consequently, the disease-free equilibrium point $X(t)$ is solution of (5). Uniqueness of solution follows from [114, Theorem 3.2].

4.2. Equilibria and Basic reproduction number of the fractional order model

By setting the left hand side of (5) to zero, one obtains the equilibrium points. Disease-free equilibrium points are those where $I_4 = I_5 = 0$ and $P = 0$. These immediately imply $E = 0$ and $R = 0$. Thus, we obtain $b^\prime - \mu^\prime S^\ast = 0$ or $S^\ast = b^\prime/\mu^\prime$. Consequently, the disease-free equilibrium point $S^\ast = b^\prime/\mu^\prime$. The basic reproduction number is the spectral radius of matrix $K = F \Gamma^{-1}$, the total production of new infections over the course of outbreak. Using symbolic computation Maple17 software, we compute $K = \begin{pmatrix} \frac{\rho_1^\prime + \rho_2^\prime}{\rho_2^\prime} + \frac{\rho_3^\prime (1 - \delta) \alpha^\prime}{\rho_2^\prime} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \end{pmatrix}$

As suggested by [115], the above matrix can be further reduced to a $2 \times 2$ matrix through a certain transformation matrix $E$, while still preserving the dominant eigenvalue. It is immediate
from matrix $F$ that such a transformation matrix is
$$
E = \begin{pmatrix}
1 & 0 \\
0 & 0 \\
0 & 0 \\
0 & 1
\end{pmatrix}.
$$
Let us now define the reduced matrix $K_S$ by
$$
K_S = E^TKE = \begin{pmatrix}
\frac{\partial \phi^0 \phi^0}{\partial \xi_1^0} + \frac{\partial \phi^0 (1-\delta) \phi^0}{\partial \xi_1^0} + \frac{\partial \phi^0 \phi^0}{\partial \xi_3^0} \\
\frac{\partial \phi^0 \phi^0}{\partial \xi_2^0} + \frac{\partial \phi^0 (1-\delta) \phi^0}{\partial \xi_2^0} + \frac{\partial \phi^0 \phi^0}{\partial \xi_3^0} \\
0
\end{pmatrix}.
$$
Consequently, it follows that
$$
\rho(K) = \rho(K_S) = \frac{1}{2} \left( \text{trace}(K_S) + \sqrt{\text{trace}(K_S)^2 - 4 \det(K_S)} \right).
$$
Hence,
$$
R_0 = \frac{\beta^0 b^0 \phi^0}{\mu^0 c_1^0} + \frac{\beta^0 b^0 (1-\delta) \phi^0}{\mu^0 c_1^0} + \sqrt{\left( \frac{\partial \phi^0 \phi^0}{\partial \xi_1^0} + \frac{\partial \phi^0 (1-\delta) \phi^0}{\partial \xi_1^0} + \frac{\partial \phi^0 \phi^0}{\partial \xi_3^0} \right)^2 + \left( \frac{\partial \phi^0 \phi^0}{\partial \xi_2^0} + \frac{\partial \phi^0 (1-\delta) \phi^0}{\partial \xi_2^0} + \frac{\partial \phi^0 \phi^0}{\partial \xi_3^0} \right)^2 + \frac{\partial \phi^0 \phi^0}{\partial \xi_1^0} + \frac{\partial \phi^0 (1-\delta) \phi^0}{\partial \xi_1^0} + \frac{\partial \phi^0 \phi^0}{\partial \xi_3^0}}.
$$

### 4.2.1. Local stability analysis

Here, we establish local stability of the disease-free equilibrium point $E_0$ of (5) by examining the nature of the eigenvalues of the linearization matrix of (5), namely the Jacobian $J(E_0)$.

**Theorem 4.4.** The disease-free equilibrium point $E_0$ of model (5) is locally asymptotically stable if all the eigenvalues $\lambda_i$, $i \in \{1, 2, 3, 4, 5, 6\}$ of $J(E_0)$ satisfy $|\text{arg}(\lambda_i)| > \frac{\pi}{2}$.

**Proof.** The Jacobian of (5) evaluated at the disease-free equilibrium $E_0$ is given by

$$
J(E_0) = \begin{pmatrix}
-\mu^0 & \phi^0 & -k_2 & -k_2 & 0 & -k_1 \\
0 & \phi^0 & \mu^0 + \sigma^0 & -k_2 & k_2 & 0 \\
0 & 0 & (1-\delta) \mu^0 & -k_2 & -\phi^0 & -k_2 \\
0 & 0 & -\phi^0 & \phi^0 + \sigma^0 + \gamma^0 & 0 & 0 \\
0 & 0 & 0 & \phi^0 & \phi^0 + \sigma^0 + \gamma^0 & -\mu^0 \\
0 & 0 & 0 & 0 & \phi^0 & -\phi^0
\end{pmatrix}
$$

where
$$
k_1 = \frac{\beta^0 b^0}{\mu^0}, \quad k_2 = \frac{\beta^0 b^0}{\mu^0}.
$$

The characteristic equation of $J(E_0)$ is computed directly as
$$
(\lambda + \mu^0)^3 (\lambda + B_1 \lambda^2 + B_2 \lambda^2 + B_3 \lambda + B_4) = 0.
$$
where
$$
B_1 = c_1^0 + c_2^0 + c_3^0 + \mu^0,
$$
$$
B_2 = (c_1^0 + c_3^0) \mu^0 + c_3^0 c_2^0 + c_2^0 c_3^0 - \omega^0 k_2,
$$
$$
B_3 = \mu^0 (c_1^0 c_2^0 + c_2^0 c_3^0 + c_3^0 c_3^0) + c_3^0 c_1^0 c_2^0 + k_2 \omega^0 ((\delta - 1) \eta_2^0 - \eta_3^0 \delta)
$$
$$
- \omega^0 k_2 (\mu^0 + (1 - \delta) c_2 + \delta c_3),
$$
$$
B_4 = \mu^0 \phi^0 c_2^0 c_3^0 + \omega^0 k_2 \phi^0 (\delta - 1) c_2 + \delta c_3
$$
$$
+ \omega^0 k_1 ((\delta - 1) c_2 \eta_3^0 - \eta_2^0 \delta c_3).
$$

The characteristic equation is (5), $P(\lambda) = \lambda^3 + B_1 \lambda^2 + B_2 \lambda^2 + B_3 \lambda + B_4 = 0$.

Routh-Hurwitz conditions are known to be necessary and sufficient for the roots of this equation to satisfy $|\text{arg}(\lambda_i)| > \frac{\pi}{2}$.

However, to simplify notation in the sequel, let us define the discriminant (or resultant) of $P(\lambda)$ as

$$
D(P) = \begin{vmatrix}
1 & B_1 & B_2 & B_3 & B_4 & 0 & 0 \\
0 & 1 & B_1 & B_2 & B_3 & B_4 & 0 \\
0 & 0 & 1 & B_1 & B_2 & B_3 & B_4 \\
0 & 0 & 0 & 1 & B_1 & B_2 & B_3 \\
0 & 4 & 3B_1 & 2B_2 & B_3 & 0 & 0 \\
0 & 0 & 4 & 3B_1 & 2B_2 & B_3 & 0 \\
0 & 0 & 0 & 0 & 4 & 3B_1 & 2B_2 & B_3
\end{vmatrix}.
$$

Using the results in [87,116], we have the following

i. If

$$
\Delta_1 = B_1, \quad \Delta_2 = \begin{vmatrix}
B_1 & 1 & 0 \\
B_3 & B_2 & B_4 \\
0 & B_4 & B_3
\end{vmatrix}, \quad \Delta_3 = \begin{vmatrix}
0 & B_1 & 0 \\
B_3 & B_2 & B_4 \\
B_4 & B_3 & B_1
\end{vmatrix},
$$

then for $\alpha = 1$, the necessary and sufficient condition for the equilibrium point $E_0$ to be locally asymptotically stable are

$$
\Delta_1 > 0, \Delta_2 > 0, \Delta_3 = 0, B_4 > 0,
$$

and the above conditions are sufficient for $E_0$ to be locally asymptotically stable for all $\alpha \in [0, 1)$.

ii. If $D(P) > 0, B_1 > 0, B_2 < 0$ and $\alpha > \frac{1}{2}$, then the equilibrium point $E_0$ is unstable.

iii. If $D(P) < 0, B_1 > 0, B_2 > 0, B_3 > 0, B_4 > 0$ and $\alpha < \frac{1}{2}$, then the equilibrium point $E_0$ is locally asymptotically stable. Furthermore, if $D(P) < 0, B_1 < 0, B_2 > 0, B_3 < 0, B_4 > 0$, then the equilibrium point $E_0$ is unstable.

iv. If $D(P) < 0, B_1 > 0, B_2 > 0, B_3 > 0, B_4 > 0$ and $B_2 = \frac{B_3}{B_4}$, then the equilibrium point $E_0$ is locally asymptotically stable, for all $\alpha \in (0, 1)$.

v. $B_4 > 0$ is the necessary condition for the equilibrium point $E_0$ to be locally asymptotically stable. \(\square\)

### 4.2.2. Global stability

We establish the global stability of the fractional model (6) in the sense of Ulam-Hyers [117]. The Ulam-Hyers stability has been an active research area since it was first introduced by Ulam in 1940 at Winsconsin University and a follow-up work by Rassias in the years between 1982 and 1998. Recently the authors in [20] established Ulam-Hyers stability of a nonlinear fractional model of Covid-19 pandemic.

For clarity of the discussion that follows, let us introduce the inequality given by

$$
|D(P)X(t) - \mathcal{M}(t, X(t))| \leq \epsilon, \quad t \in [0, b].
$$

We say a function $X \in \mathcal{E}$ is a solution of (10) if and only if there exists $h \in \mathcal{E}$ satisfying

$$
P(\lambda) = \lambda^3 + B_1 \lambda^2 + B_2 \lambda^2 + B_3 \lambda + B_4 = 0.
$$
Fig. 5  Population dynamics of the fractional model for COVID-19 epidemic for $x \in \{1, 0.9, 0.8, 0.7\}$ labelled respectively a-d.
exists a continuous function \(\phi_{\mathcal{K}} : \mathbb{R}^+ \to \mathbb{R}^+\) with \(\phi_{\mathcal{K}}(0) = 0\), such that, for each solution \(\tilde{X} \in \mathcal{E}\) of (10), there exists a solution \(X \in \mathcal{E}\) of (6) with

\[
\| \tilde{X}(t) - X(i) \|_\delta \leq \phi_{\mathcal{K}} \epsilon, \quad t \in [0, b].
\]

**Theorem 4.7.** Let the hypothesis and result of Lemma 4.2 hold, \(\Omega = \frac{\mu^*}{\Gamma(\alpha + 1)}\) and \(1 - \Omega L_{\mathcal{K}} > 0\). Then, the fractional order model (6) (and equivalently (5)) is Ulam-Hyers stable and consequently generalized Ulam-Hyers stable.

**Proof.** Let \(X\) be a unique solution of (6) guaranteed by Theorem 4.3. \(\tilde{X}\) satisfies (10). Then recalling the expressions (7), (11), we have for \(\epsilon > 0, t \in [0, b]\) that

\[
\| \tilde{X} - X \|_\delta = \sup_{i \in [0, b]} | \tilde{X}(i) - X(i) |,
\]

\[
\leq \sup_{i \in [0, b]} \left| \tilde{X}(t) - X_0 - \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha-1} \mathcal{K}(\tau, \tilde{X}(\tau)) \, d\tau \right|,
\]

\[
\leq \sup_{i \in [0, b]} \left| \tilde{X}(t) - X_0 - \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha-1} \mathcal{K}(\tau, \tilde{X}(\tau)) \, d\tau \right|,
\]

\[
+ \sup_{i \in [0, b]} \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha-1} | \mathcal{K}(\tau, \tilde{X}(\tau)) - \mathcal{K}(\tau, X(\tau)) | \, d\tau,
\]

\[
\leq \Omega \epsilon + \frac{L_{\mathcal{K}}}{\Gamma(\alpha)} \sup_{i \in [0, b]} \int_0^t (t - \tau)^{\alpha-1} | \tilde{X}(\tau) - X(\tau) | \, d\tau,
\]

\[
\leq \Omega \epsilon + \Omega L_{\mathcal{K}} \| \tilde{X} - X \|_\delta,
\]

from which we obtain \(\| \tilde{X} - X \|_\delta \leq C_{\mathcal{K}} \epsilon\) where \(C_{\mathcal{K}} = \frac{\Omega}{1 - \Omega L_{\mathcal{K}}}\). \(\square\)
Fig. 7 Effect of new rate of infections through the environment ($x_1$) and through contact with infected individuals ($x_2$) on population dynamics.
5. Numerical simulation and discussion

This section provides some illustrative numerical simulations to explain the dynamical behavior of the Caputo fractional order deterministic nonlinear COVID-19 mathematical model. The numerical solution of a nonlinear mathematical model using the appropriate iterative scheme is very important in mathematical modeling. For this purpose, we have used the Adams-type predictor-corrector iterative scheme constructed in [118,119] to solve fractional order differential equations. The method relies on the equivalent integral formulation of our fractional model (5) written in the form (6). Consider a uniform discretization of [0, b] given by \( t_n = nh, n = 0, 1, 2, \ldots, N \) where 0 < h = b/n denote the step size. Now, given any approximation \( X_h(t_i) \approx X(t_i) \), we obtain the next approximation \( X_h(t_{i+1}) \) using the Adams-type predictor-corrector iterative scheme as follows;

**Predictor:**

\[
X\big(h, (t_{i+1}) = \sum_{k=0}^{n} \frac{x_{k}}{k!} t_{i} + \frac{1}{\Gamma(2)} \sum_{j=0}^{n} b_{i+1} \Phi(t_j, X_h(t_j))
\]

**Corrector:**

\[
X\big(h, (t_{i+1}) = \sum_{k=0}^{n} \frac{x_{k}}{k!} t_{i} + \frac{h^2}{\Gamma(3)} \sum_{j=0}^{n} a_{i+1} \Phi(t_j, X_h(t_j)),
\]

with

\[
 a_{i+1} = \begin{cases} 
 \frac{n+1 - (n - x)(n + 1)^2}{(n - i + 2)^2 + (n - j)^2}, & \text{if } i = 0, \\
 2(n - i + 1)^2, & \text{if } 0 < i < n, \\
 1, & \text{if } i = n + 1,
\end{cases}
\]

and

\[
b_{i+1} = \frac{h^2}{\Gamma(3)} [n - i + 1]^2 - (n - i)^2.
\]

For varying values of the derivative order \( \alpha \), the dynamics of the fractional order model are presented in Figs. 5 below. We have used the model parameter in Table 1 as reported in [103] and initial data S(0) = 93000, E(0) = 1000, I_a(0) = 50, I_e(0) = 50, R(0) = 0, P(0) = 500. For \( \alpha = 1 \), coinciding with the integer-order model, the obtained dynamics is consistent with those reported in Section 3 and [103]. In Figs. 5, effect of the fractional order \( \alpha \) on disease progression are demonstrated. Furthermore, the basic reproduction number of the disease-free equilibrium point \( \Sigma = (\bar{S}, \bar{E}, 0, 0, 0, 0) = (1.9861, 0.0, 0.0, 0.0, 0.0) \) for \( \alpha = 0.5 \) was computed as \( R_0 = 0.87831 < 1 \), showing the fulfillment of the necessary and sufficient conditions for local asymptotic stability of the disease-free equilibrium. We also found out that for fractional orders considered namely \( \alpha \in \{0.9, 0.8, 0.7\} \), and with new infection rates \( z_1 = z_2 = 0.1 \), the corresponding computed \( R_0 \in [0.90850, 0.82590] \) showing that the COVID-19 pandemic is controllable and will effectively die out as long as there is compliance with social distancing/lockdown regulations, and if infectious and infected individuals are appropriately quarantined, thereby preventing contamination of the environment through virus shedding (Fig. 6).

Recall that the constants \( z_1 \) and \( z_2 \) represent the proportion of interaction with the infectious environment and infectious individuals respectively. Consider a scenario of a contaminated environment \( z_1 = 0.05 \) where there is higher chances of contracting COVID-19 through the environment than through infectious individuals \( I_a \) or \( I_e \). In this situation, a rapid decline in the susceptible population is noticeable in Fig. 7a during the first ten days. This is expected as more people get exposed through the virus-laden environment, explaining the rapid rise in the exposed population and the infected population within the same time period, Fig. 7b, c and d. In comparison with other scenarios, namely \( z_2 = 0.05, z_3 = 0.1 \) where there is a higher risk of contracting COVID-19 virus through contacts with infectious individuals than through the environment; and \( z_3 = 0.1, z_4 = 0.1 \) depicting generally low infection rate from both sources, the number of exposed, asymptomatic infectious and symptomatic infectious populations peaked in the former.

6. Conclusion

We have proposed a fractional order epidemic model for COVID-19 disease dynamics based on the integer-order model of [103]. This study presented:

- Qualitative analysis of both integer order and fractional order models. For the integer order, we used a multistage technique to derive approximate analytical solution via a polynomial system.
- Also, we established the well-posedness of the fractional model via the Banach fixed point theorem. We proved the local asymptotic stability of the fractional model by using the concept of next-generation matrix for computing basic reproduction number \( R_0 \).
- Furthermore, we proved the global stability of the model in the sense of Ulam-Hyers stability criteria.
- Finally, numerical simulations confirmed the established properties of the proposed model, and more importantly elucidated the need for compliance with regulations on basic preventive measures such as social distancing, quarantining of infected and infected individuals, and frequent hand washing to rid the population of the deadly virus.

Indeed, the analysis of the proposed model is far from being complete. Therefore, future research efforts in this direction will consider the following improvements of the present work:

- extension of the model to capture stochastic dynamics;
- estimation of model parameters, including the order of the fractional derivative, based on the vast available data on COVID-19; and
- investigation of the effectiveness of various available COVID-19 vaccines using optimal control formulation.
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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