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Dynamics of an epidemic model for COVID-19 with Hattaf fractal-fractional operator and study of existence of solutions by means of fixed point theory



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Abstract

Coronavirus disease 2019 (COVID-19) is an infectious disease caused by a new virus called severe acute respiratory syndrome coronavirus 2 (SARS-COV-2). To describe the spread of this infectious disease, we propose a mathematical model including some important aspects, such as the carrier and memory effects as well as the nonlinearity of incidence function. The memory effect is described by the Hattaf fractal-fractional derivative. Sufficient conditions for the existence and uniqueness of solutions are established by means of Krasnoselskii's fixed point theorem and Banach contraction. Furthermore, our results show that the proposed fractal-fractional model has one stable disease-free equilibrium when the basic reproduction number satisfies $\Re_0 \leq 1$ and a unique stable endemic equilibrium when $\Re_0 > 1$. In addition, numerical simulations for different values of fractal and fractional orders are carried out to illustrate the theoretical results.

Keywords: COVID-19, SARS-CoV-2, Krasnoselskii's fixed point theorem, Hattaf fractal-fractional derivative, numerical simulations.

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1. Introduction

Since the first case registered in December 2019 in China, the new infection coronavirus disease 2019 (COVID-19) continue to emerge in the world attaining over 773 million people, and over 6.99 million deaths [29]. In this situation, the world face a sensational loss of human life worldwide and an extraordinary challenge, food systems, the work universe and how we live, relate, and speak with others has been modified for all time, due to the dynamics of COVID-19, including the mortality, contagion factors, which present an extraordinary speed with time to the global health. With these challenges, this infectious disease was declared a global pandemic by the World Health Organization (WHO) on March 11, 2020.

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Email address: elmamouni89.hamza@gmail.com (Hamza EL Mamouni) doi: 10.22436/jmcs.036.04.02 Received: 2024-01-14 Revised: 2024-04-14 Accepted: 2024-07-08 Most individuals infected with COVID-19 experience mild, moderate, or severe symptoms. Common symptoms include muscle aches, severe fatigue or tiredness, headache, new and persistent cough, shortness of breath, hoarse voice, loss or change of sense of taste or smell, etc. Severe symptoms include dyspnea and chest pains [25, 29]. Also, critical pathological manifestations of the disease in the infected population with comorbidities include acute respiratory distress syndrome and respiratory failure [30]. To explore the origin of this infectious disease, the transmission dynamics, and comprehend its profound impact on economies, social structures and public health various factors have been investigated, such as the infectiousness of the disease, the number of susceptible and infected individuals and the rate at which people recover or die. For this reason to compile a comprehensive dataset, many incidence data was collected from divers countries [11, 19, 20].

Over the last three decades, fractal and fractional calculus have developed a new approach to describing real-world phenomena within mathematical concepts. The powerful tools of this theory have given rise to new concepts of differentiation and integration. The pioneering work in this field was initiated by Antangana [5] in 2017, who presented operators based on generalized Mittag-Leffler functions and employed the Hausdorff fractal derivative. Recent contributions to this field have been generalized by Hattaf [15] in 2023, who investigated new differential and integral operators, incorporating a wide range of definitions. The results obtained for the Hattaf fractal-fractional derivative cover many special cases, for instance, the generalized Hattaf fractional (GHF) derivative [13], the Caputo-Fabrizio (CF) fractional derivative [9], the Atangana-Baleanu (AB) fractional derivative [6], and the weighted AB fractional derivative [4].

In the literature, there are numerous applications of fractal and fractional calculus. For instance, Ali et al. [3] developed a fractal-fractional model for COVID-19 with case study of Wuhan. In [1], the authors studied a fractal-fractional modified predator-prey mathematical model with immigrations. In [2], the authors proposed a fractional model for COVID-19 with the effect of asymptomatic and symptomatic transmission by using the caputo fractional model. Paul et al. [27] investigated a fractional order SEIQRD epidemic model in order to study the COVID-19 transmission dynamic in Italy. In 2023, the authors in [26] established a model of prey-predator dynamics by describing the fuzzy fractional diabetes in Caputo's sense. In [23], Mahata et al. studied an SEIRV of COVID-19 epidemic model with optimal control in the context of Caputo fractional sense. In [24], the authors treated the stability analysis and Hopf bifurcation of a fractional order SEIRV epidemic model with a single delay incorporated in the infectious population accounting for the time period required by the said population to recover. The study of [28] explored the dynamical behavior of a fractional order SIR model in the Caputo derivative approach [8].

On the other hand, fixed point theory plays a crucial role in pure and applied mathematics. It provides powerful tools for proving existence theorems and uniqueness of solutions for various models arising from the fields of science and engineering. More recently, Lasfar et al. [22] proposed a new fractional business cycle model and they established the existence of the model solutions by means of fixed point theory. Based on Schauder fixed point theorem and the construction of a pair of upper and lower solutions for an epidemic model introduced in [12], the existence of traveling wave solution of that connects the disease-free equilibrium and the endemic equilibrium has been established. In this study framework, we will use Krasnoselskii's fixed point theorem [7, 21] and Banach's contraction to prove the existence and uniqueness of solutions for a fractal-fractional model describing the dynamics of COVID-19 with carrier and general incidence functions for asymptomatic and symptomatic transmission.

The rest of this paper is outlined as follows. Section 2 is devoted to some interesting preliminary findings essential for the development of this work and the formulation of the Hattaf fractal-fractional COVID-19 model. Section 3 focuses on the existence and uniqueness of solutions for our formulated model. Section 4 analyzes the stability of equilibria. Section 5 deals with an application and some numerical simulations to illustrate the analytical results. Finally, Section 6 ends the paper with a conclusion and some perspectives.

2. Preliminary and fractal-fractional model formulation

In this section, we give the necessary definitions and results that are needed for the proof of the main results, and also present a fractal-fractional model formulation for COVID-19 based on the Hattaf fractal-fractional derivative [15].

Definition 2.1 ([15]). Let \mathcal{I} be an open interval of \mathbb{R} . The Hattaf fractal derivative of a function f(t) with respect to a fractal measure $g(\eta, t)$ is given by

$$\frac{d_g}{dt^{\eta}}f(t) = \lim_{\tau \to t} \frac{f(t) - f(\tau)}{g(\eta, t) - g(\eta, \tau)}, \ \eta > 0.$$
(2.1)

If $\frac{d_g}{dt^{\eta}}f(t)$ exists for all $t \in J$, then f is fractal differentiable on the interval J with order η . We notice that when $g(\eta, t) = t^{\eta}$, we get the Hausdorff fractal derivative [10]. In addition, if $g(\eta, t) = h(t)$ with h'(t) > 0 and f(t) is differentiable, then we obtain the general derivative proposed by Yang [31] and (2.1) becomes

$$\frac{d_g}{dt^\eta}f(t) = \frac{1}{h'(t)}\frac{df(t)}{dt}$$

Next, we recall the definition of the Hattaf fractal-fractional derivative with non-singular kernel in the sense of Caputo.

Definition 2.2 ([15]). Let $\alpha \in [0, 1)$, $\beta, \gamma, \eta > 0$, and f be a differentiable in the interval (a, b) and fractal differentiable on (a, b) with order $0 < \eta \leq 1$. The generalized Hattaf fractal-fractional derivative of f(t) of order α in the sense of Caputo with respect to the weight function w(t) is defined as follows:

$${}^{FFC}D_{\alpha,t,w}^{\alpha,\beta,\gamma,\eta}f(t) = \frac{N(\alpha)}{1-\alpha}\frac{1}{w(t)}\int_{a}^{t} E_{\beta}[-\mu_{\alpha}(t-\tau)^{\gamma}]\frac{d_{g}}{d\tau^{\eta}}(wf)(\tau)d\tau,$$

where $w \in C^1(a, b)$, w, w' > 0 on [a,b], $N(\alpha)$ is a normalization function obeying N(0) = N(1) = 1, $\mu_{\alpha} = \frac{\alpha}{1-\alpha}$ and $E_{\beta}(t) = \sum_{k=0}^{+\infty} \frac{t^k}{\Gamma(\beta k+1)}$ is the Mittag-Leffler function of parameter β .

Definition 2.2 covers many special cases. In the fact, when $g(t, \eta) = t^{\eta}$, w(t) = 1, and $\beta = \gamma = 1$, we obtain the fractal-fractional derivative with exponential decay kernel [5] given by

$${}^{FFC}D_{\alpha,t,1}^{\alpha,1,1,\eta}f(t) = \frac{N(\alpha)}{1-\alpha} \int_{\alpha}^{t} exp[-\mu_{\alpha}(t-\tau)] \frac{d_{g}}{d\tau^{\eta}} f(\tau) d\tau,$$

where $\frac{d_g}{dt^{\eta}}f(t) = \lim_{\tau \to t} \frac{f(t)-f(\tau)}{t^{\eta}-\tau^{\eta}}$. When $g(t,\eta) = t^{\eta}$, w(t) = 1, and $\beta = 1$, $\gamma = 2$, we also obtain the fractal-fractional derivative with exponential decay kernel [5] given by

$${}^{FFC}D_{\alpha,t,1}^{\alpha,1,1,\eta}f(t) = \frac{N(\alpha)}{1-\alpha} \int_{\alpha}^{t} exp[-\mu_{\alpha}(t-\tau)^{2}] \frac{d_{g}}{d\tau^{\eta}} f(\tau) d\tau$$

When $g(t,\eta) = t^{\eta}$, w(t) = 1, $N(\alpha) = 1 - \alpha + \frac{\alpha}{\Gamma(\alpha)}$, $\beta = \gamma = \alpha$, we obtain the fractal-fractional derivative with generalized Mittag-Leffler kernel [5] given by

$${}^{FFC}D_{\alpha,\tau,1}^{\alpha,\alpha,\alpha,\eta}f(t) = \frac{N(\alpha)}{1-\alpha}\int_{a}^{t} E_{\alpha}[-\mu_{\alpha}(t-\tau)^{\alpha}]\frac{d_{g}}{d\tau^{\eta}}f(\tau)d\tau.$$

When $g(t, \eta) = t$, we obtain the generalized Hattaf fractional (GHF) derivative [13] given by

$$^{C}D_{\alpha,t,w}^{\alpha,\beta,\gamma}f(t) = \frac{N(\alpha)}{1-\alpha}\frac{1}{w(t)}\int_{a}^{t}E_{\beta}[-\mu_{\alpha}(t-\tau)^{\gamma}]\frac{d}{d\tau}(fw)(\tau)d\tau.$$

The next definition recalls the Hattaf fractal-fractional derivative in Riemann-Liouville sense.

$${}^{FFR}D_{\alpha,t,w}^{\alpha,\beta,\gamma,\eta}f(t) = \frac{N(\alpha)}{1-\alpha}\frac{1}{w(t)}\frac{d_g}{dt^{\eta}}\int_a^t E_{\beta}[-\mu_{\alpha}(t-\tau)^{\gamma}]f(\tau)w(\tau)d\tau.$$

Theorem 2.4 ([15]). If $\frac{\partial g(\eta,t)}{\partial t}$ exists and not zero, then

$${}^{FFR}D_{0,t,w}^{\alpha,\beta,\gamma,\eta}f(t) = \left(\frac{\partial g(\eta,t)}{\partial t}\right)^{-1} {}^{R}D_{0,t,w}^{\alpha,\beta,\gamma}f(t),$$

where ${}^{R}D_{0,t,w}^{\alpha,\beta,\gamma}$ is the GHF derivative in Riemann-Liouville sense ([13]) of the function f(t) with respect to the weight function w(t).

For the existence and uniqueness of solution of our fractal-fractional differential model for COVID-19, we need the following result.

Lemma 2.5 ([7, 21, Krasnoselskii's fixed point theorem]). *Let* E *be a nonempty closed convex subset of a Banach space* $(\mathcal{B}, \|.\|)$. *Suppose that* F₁ *and* F₂ *map* E *into* \mathcal{B} *such that*

- (i) $F_1\phi_1 + F_2\phi_2 \in E$, for all $\phi_1, \phi_2 \in E$;
- (ii) F_1 is a contraction with constant k < 1;
- (iii) F_2 is continuous and $F_2(E)$ is contained in a compact subset of \mathcal{B} .

Then $F_1 + F_2$ has a fixed point $\phi \in E$.

Based on [18], we propose the following fractal-fractional differential model for COVID-19:

$$\begin{cases} {}^{\text{FFR}} D_{0,t,w}^{\alpha,\beta,\beta,\eta} x_1(t) = \mathcal{A} - \nu x_1 - \zeta(x_1, x_2) x_2 - \xi(x_1, x_3) x_3, \\ {}^{\text{FFR}} D_{0,t,w}^{\alpha,\beta,\beta,\eta} x_2(t) = \zeta(x_1, x_2) x_2 + \xi(x_1, x_3) x_3 - (\nu + d + \rho + r_1) x_2, \\ {}^{\text{FFR}} D_{0,t,w}^{\alpha,\beta,\beta,\eta} x_3(t) = \rho x_2 - (\nu + d + r_2) x_3, \\ {}^{\text{FFR}} D_{0,t,w}^{\alpha,\beta,\beta,\eta} x_4(t) = r_1 x_2 + r_2 x_3 - \nu x_4, \end{cases}$$

$$(2.2)$$

where $x_1(t)$, $x_2(t)$, $x_3(t)$, and $x_4(t)$ represent the susceptible, carrier (asymptomatic infected), infected, and recovered individuals at time t, respectively. A represents the recruitment rate of susceptible population. The natural death rate in all classes is denoted by v, while d is the death rate due to COVID-19. The rate of transfer from the asymptomatic to symptomatic is denoted by ρ . The parameters r_1 and r_2 are recovery rates of the asymptomatic and symptomatic individuals, respectively. The term $\zeta(x_1, x_2)x_2$ represents the effective contact with carrier, and $\xi(x_1, x_3)x_3$ denotes the effective contact with infected individuals. So, the term $\zeta(x_1, x_2)x_2 + \xi(x_1, x_3)x_3$ is the total asymptomatic infection rate of susceptible individuals. Further, the flowchart of the proposed model (2.2) is illustrated in Figure 1.

Since the first three equations of (2.2) do not depend on the last variable x_4 and based on Theorem 2.4, system (2.2) can be rewritten as

$$\begin{cases} {}^{C}D_{0,t,w}^{\alpha,\beta,\beta}x_{1}(t) = \frac{\partial g(\eta,t)}{\partial t} \left(\mathcal{A} - \nu x_{1} - \zeta(x_{1},x_{2})x_{2} - \xi(x_{1},x_{3})x_{3} \right), \\ {}^{C}D_{0,t,w}^{\alpha,\beta,\beta}x_{2}(t) = \frac{\partial g(\eta,t)}{\partial t} \left(\zeta(x_{1},x_{2})x_{2} + \xi(x_{1},x_{3})x_{3} - (\nu + d + \rho + r_{1})x_{2} \right), \\ {}^{C}D_{0,t,w}^{\alpha,\beta,\beta}x_{3}(t) = \frac{\partial g(\eta,t)}{\partial t} \left(\rho x_{2} - (\nu + d + r_{2})x_{3} \right), \end{cases}$$
(2.3)

with initial conditions $x_i(0) = x_{i,0}$ for i = 1, 2, 3.



Figure 1: The flowchart of model (2.2).

3. Existence and uniqueness of solutions

In this section, we study the existence and uniqueness of solutions of model (2.3). System (2.3) can be written in the following form:

$$\begin{cases} {}^{C}D_{0,t,w}^{\alpha,\beta,\beta}X(t) = \frac{\partial g(\eta,t)}{\partial t}\Phi(t,X(t)), \ t \in [0,T], \\ X(0) = X_{0}, \end{cases}$$
(3.1)

where
$$X(t) = (x_1(t), x_2(t), x_3(t))^T$$
, $X_0 = (x_1(0), x_2(0), x_3(0))^T$, and $\Phi(t, X(t)) = (\Phi_1(t, X(t)), \Phi_2(t, X(t)))$,
 $\Phi_3(t, X(t)))^T$ with

$$\begin{split} & \Phi_1(t, X(t)) = \mathcal{A} - \nu x_1 - \zeta(x_1, x_2) x_2 - \xi(x_1, x_3) x_3, \\ & \Phi_2(t, X(t)) = \zeta(x_1, x_2) x_2 + \xi(x_1, x_3) x_3 - (\nu + d + \gamma + r_1) x_2, \\ & \langle \Phi_3(t, X(t)) = \rho x_2 - (\nu + d + r_2) x_3. \end{split}$$

Applying the GHF integral in both sides of (3.1), we get

$$X(t) = \frac{w(0)X_0}{w(t)} + \frac{1-\alpha}{N(\alpha)} \frac{\partial g(\eta, t)}{\partial t} \Phi(t, X(t)) + \frac{\alpha}{N(\alpha)\Gamma(\beta)w(t)} \int_0^t (t-\tau)^{\beta-1} \frac{\partial g(\eta, \tau)}{\partial \tau} w(\tau) \Phi(\tau, X(\tau)) d\tau.$$
(3.2)

Let $\mathcal{B} = C([0,T], \mathbb{R}^3)$ be the Banach space of continuous functions from [0,T] to \mathbb{R}^3 defined with the norm

$$\|\varphi(t)\| = \sup_{t\in[0,T]} |\varphi(t)|.$$

Furthermore, we consider the following hypotheses.

- (\mathfrak{H}_1) There exist positive constants ϕ , ψ and $\varepsilon \in [0,1)$ such that $\Phi(\mathfrak{t}, X(\mathfrak{t})) \leqslant \phi \|X\|^{\varepsilon} + \psi$.
- (\mathcal{H}_2) There exists a positive constant $M_1 > 0$ for all X, \tilde{X} , such that $|\Phi(t, X(t)) \Phi(t, \tilde{X}(t))| \leq M_1 ||X \tilde{X}||$.
- (\mathfrak{H}_3) There exists a positive constant $M_2 > 0$ such that for all $\eta > 0$ and $t \in [0, T]$ we have

$$\left|\frac{\partial g(\eta, t)}{\partial t}\right| \leqslant M_2$$

In addition, we define the following operator $\mathcal{F}: C([0,T],\mathbb{R}^3) \longrightarrow C([0,T],\mathbb{R}^3)$ such that

$$\mathcal{F}X(t) = \mathcal{F}_1X(t) + \mathcal{F}_2X(t),$$

where

$$\begin{cases} \mathcal{F}_1 X(t) = \frac{w(0)X_0}{w(t)} + \frac{1-\alpha}{N(\alpha)} \frac{\partial g(\eta, t)}{\partial t} \Phi(t, X(t)), \\ \mathcal{F}_2 X(t) = \frac{\alpha}{N(\alpha)\Gamma(\beta)w(t)} \int_0^t (t-\tau)^{\beta-1} \frac{\partial g(\eta, \tau)}{\partial \tau} w(\tau) \Phi(\tau, X(\tau)) d\tau. \end{cases}$$

Consequently, (3.2) can be written as

$$\begin{aligned} \mathcal{F}X(t) &= \frac{w(0)X_0}{w(t)} + \frac{1-\alpha}{N(\alpha)} \frac{\partial g(\eta, t)}{\partial t} \Phi(t, X(t)) \\ &+ \frac{\alpha}{N(\alpha)\Gamma(\beta)w(t)} \int_0^t (t-\tau)^{\beta-1} \frac{\partial g(\eta, \tau)}{\partial \tau} w(\tau) \Phi(\tau, X(\tau)) d\tau. \end{aligned}$$

Theorem 3.1. Suppose that (\mathfrak{H}_1) , (\mathfrak{H}_2) , and (\mathfrak{H}_3) hold such that $\frac{M_1M_2(1-\alpha)}{N(\alpha)} < 1$. Then model (2.3) has at least one solution.

Proof. First, we prove that \mathcal{F}_1 is a contraction. Consider

$$\mathsf{E} = \{ \mathsf{X} \in \mathcal{B} : \|\mathsf{X}\| \leqslant \mathcal{L}, \mathcal{L} > 0 \},$$

which is closed and convex set. For all $X, \tilde{X} \in E$, we have

$$|\mathfrak{F}_1 X(t) - \mathfrak{F}_1 \tilde{X}(t)| \leq \frac{M_1 M_2 (1-\alpha)}{N(\alpha)} \|X - \tilde{X}\|.$$

Then

$$\|\mathcal{F}_1 X - \mathcal{F}_1 \tilde{X}\| \leq \frac{M_1 M_2 (1-\alpha)}{N(\alpha)} \|X - \tilde{X}\|.$$

Since $\frac{M_1M_2(1-\alpha)}{N(\alpha)} < 1$, we deduce that \mathcal{F}_1 is a contraction.

Secondly, we prove that \mathcal{F}_2 is compact. We have

$$\begin{split} \|\mathcal{F}_{2}X\| &\leq \max_{\mathbf{t}\in[0,T]} \bigg| \frac{\alpha}{\mathsf{N}(\alpha)\Gamma(\beta)w(\mathbf{t})} \int_{0}^{\mathbf{t}} (\mathbf{t}-\tau)^{\beta-1} \frac{\partial g(\eta,\tau)}{\partial \tau} w(\tau) \Phi(\tau,X(\tau)) d\tau \\ &\leq \frac{\alpha M_{2}T^{\beta}}{\mathsf{N}(\alpha)\Gamma(\beta+1)} \bigg(\varphi \|X\|^{\epsilon} + \psi \bigg). \end{split}$$

Hence, for all $X \in E$, \mathfrak{F}_2 is bounded. For equicontinuity, let $t_1, t_2 \in [0, T]$ such that $t_2 < t_1$, then

$$\begin{aligned} |\mathcal{F}_{2}X(t_{1}) - \mathcal{F}_{2}X(t_{2})| &= \frac{\alpha}{\mathsf{N}(\alpha)\Gamma(\beta)} \bigg| \int_{0}^{t_{1}} (t_{1} - \tau)^{\beta - 1} \frac{\partial g(\eta, \tau)}{\partial \tau} \frac{w(\tau)}{w(t_{1})} \Phi(\tau, X(\tau)) d\tau \\ &- \int_{0}^{t_{2}} (t_{2} - \tau)^{\beta - 1} \frac{\partial g(\eta, \tau)}{\partial \tau} \frac{w(\tau)}{w(t_{2})} \Phi(\tau, X(\tau)) d\tau \bigg|. \end{aligned}$$

Therefore, $\lim_{t_2 \to t_1} |\mathcal{F}_2 Y(t_1) - \mathcal{F}_2 Y(t_2)| = 0$. Thus, \mathcal{F}_2 is equicontinuous. By Arzela-Ascoli theorem, we deduce that \mathcal{F}_2 is compact. Furthermore \mathcal{F}_2 is continuous since X is continuous. Finally to verify the item (i) of Lemma 2.5, we notice that \mathcal{F}_1 is a contraction, then

$$\|\mathbf{X}\| \leqslant \|(\mathbf{I} - \mathcal{F}_1)\mathbf{X}\|,$$

and since $F_2(E)$ is contained in a compact subset of \mathcal{B} we deduce by appropriate construction of the subset E that for fixed Y in E the contraction $X \to \mathcal{F}_1X + \mathcal{F}_2Y$ has a fixed point in E, then $X \in E$. Therefore, according to Lemma 2.5, we conclude that model (2.3) has at least one solution.

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Theorem 3.2. If $\left(\frac{1-\alpha}{N(\alpha)} + \frac{\alpha T^{\beta}}{N(\alpha)\Gamma(\beta+1)}\right) M_1 M_2 < 1$, then model (2.3) has a unique solution. *Proof.* Let $X, \tilde{X} \in C([0, T], \mathbb{R}^3)$. We have

$$\begin{split} \|\mathcal{F}X - \mathcal{F}\tilde{X}\| &\leqslant \|\mathcal{F}_{1}X - \mathcal{F}_{1}\tilde{X}\| + \|\mathcal{F}_{2}X - \mathcal{F}_{2}\tilde{X}\| \\ &\leqslant \frac{(1-\alpha)M_{2}}{\mathsf{N}(\alpha)} \max_{\mathsf{t}\in[0,\mathsf{T}]} \left| \Phi(\mathsf{t},X(\mathsf{t})) - \Phi(\mathsf{t},\tilde{X}(\mathsf{t})) \right| + \frac{\alpha M_{2}}{\mathsf{N}(\alpha)\Gamma(\beta)} \\ &\times \max_{\mathsf{t}\in[0,\mathsf{T}]} \left| \int_{0}^{\mathsf{t}} (\mathsf{t}-\tau)^{\beta-1} \frac{w(\tau)}{w(\mathsf{t})} \Phi(\tau,X(\tau)) d\tau - \int_{0}^{\mathsf{t}} (\mathsf{t}-\tau)^{\beta-1} \frac{w(\tau)}{w(\mathsf{t})} \Phi(\tau,\tilde{X}(\tau)) d\tau \right| \\ &\leqslant \left(\frac{1-\alpha}{\mathsf{N}(\alpha)} + \frac{\alpha \mathsf{T}^{\beta}}{\mathsf{N}(\alpha)\Gamma(\beta+1)} \right) \mathsf{M}_{1}\mathsf{M}_{2} \|X - \tilde{X}\|. \end{split}$$

Since $\left(\frac{1-\alpha}{N(\alpha)} + \frac{\alpha T^{\beta}}{N(\alpha)\Gamma(\beta+1)}\right) M_1 M_2 < 1$, we deduce that \mathcal{F} is a contraction. Consequently, the fractal-fractional differential COVID-19 model (2.3) has a unique solution.

4. Equilibria and their stability

In this section, we first study the existence of equilibria of system (2.3) for the case w(t) = 1 and we assume that $\frac{\partial g(\eta, t)}{\partial t}$ exists and not zero. As in [17], we assume that the general incidence functions ζ and ξ are continuously differentiable and satisfy the following hypotheses:

 $\begin{array}{ll} (\mathfrak{H}_4) \ \zeta(0,x_2)=0, \ \frac{\partial \zeta}{\partial x_1}(x_1,x_2)>0, \ \frac{\partial \zeta}{\partial x_2}(x_1,x_2)\leqslant 0, \ \text{for all} \ x_1,x_2\geqslant 0; \\ (\mathfrak{H}_5) \ \xi(0,x_3)=0, \ \frac{\partial \xi}{\partial x_1}(x_1,x_3)>0, \ \frac{\partial \xi}{\partial x_3}(x_1,x_3)\leqslant 0, \ \text{for all} \ x_1,x_3\geqslant 0. \end{array}$

We can verify that $\mathcal{E}(x_1^0, 0, 0)$, where $x_1^0 = \frac{\mathcal{A}}{\nu}$, is a disease-free equilibrium of (2.3). According to [18], system (2.3) has the basic reproduction number as follows:

$$\mathcal{R}_0 = \frac{d_2 \zeta(x_1^0, 0) + \rho \xi(x_1^0, 0)}{d_1 d_2},$$

where $d_1 = \nu + d + \rho + r_1$ and $d_2 = \nu + d + r_2$. The second equilibrium of system (2.3) satisfies the following system of equations

$$\mathcal{A} - \nu x_1 - \zeta(x_1, x_2) x_2 - \xi(x_1, x_3) x_3 = 0, \quad \zeta(x_1, x_2) x_2 + \xi(x_1, x_3) x_3 - d_1 x_2 = 0, \quad \rho x_2 - d_2 x_3 = 0$$

Then $x_2 = \frac{A - \nu x_1}{d_1}$, $x_3 = \frac{\rho(A - \nu x_1)}{d_1 d_2}$, and

$$d_2\zeta(x_1, \frac{A - \nu x_1}{d_1}) + \rho\xi(x_1, \frac{A - \nu x_1}{d_1 d_2}) = d_1 d_2$$

Since $x_2 = \frac{A - vx_1}{d_1} \ge 0$, we have $x_1 \le \frac{A}{v}$. Therefore, there is no epidemiological equilibrium when $x_1 > \frac{A}{v}$. Consider the function \mathcal{K} defined on the closed interval $[0, \frac{A}{v}]$ by

$$\mathcal{K}(\mathbf{x}_1) = \mathbf{d}_2 \zeta \left(\mathbf{x}_1, \frac{\mathcal{A} - \mathbf{v} \mathbf{x}_1}{\mathbf{d}_1} \right) + \rho \xi \left(\mathbf{x}_1, \frac{\mathcal{A} - \mathbf{v} \mathbf{x}_1}{\mathbf{d}_1 \mathbf{d}_2} \right) - \mathbf{d}_1 \mathbf{d}_2.$$

We have $\mathcal{K}(0) = -d_1 d_2 < 0$, $\mathcal{K}(\frac{A}{v}) = d_1 d_2 (\mathcal{R}_0 - 1)$, and

$$\mathcal{K}'(\mathbf{x}_1) = \mathbf{d}_2\left(\frac{\partial \zeta}{\partial \mathbf{x}_1} - \frac{\mathbf{v}}{\mathbf{d}_1}\frac{\partial \zeta}{\partial \mathbf{x}_2}\right) + \rho\left(\frac{\partial \xi}{\partial \mathbf{x}_1} - \frac{\rho \mathbf{v}}{\mathbf{d}_1 \mathbf{d}_2}\frac{\partial \xi}{\partial \mathbf{x}_3}\right) > 0.$$

Then the equation $\mathcal{K}(x_1) = 0$ has a unique root $x_1^* \in (0, \frac{A}{\nu})$ when $\mathcal{R}_0 > 1$. We conclude that our model has a unique endemic equilibrium when the condition $\mathcal{R}_0 > 1$ holds.

From the above results, we get the following theorem.

Theorem 4.1.

- (i) If $\mathcal{R}_0 \leq 1$, then system (2.3) admits one disease-free equilibrium of the form $\mathcal{E}(x_1^0, 0, 0)$, where $x_1^0 = \frac{A}{\gamma}$.
- (ii) If $\mathcal{R}_0 > 1$, then system (2.3) admits a unique endemic equilibrium $\mathcal{E}^*(\mathbf{x}_1^*, \mathbf{x}_2^*, \mathbf{x}_3^*)$ besides \mathcal{E} , where $\mathbf{x}_1^* \in (0, \frac{A}{\nu})$, $\mathbf{x}_2^* = \frac{A \nu \mathbf{x}_1^*}{d_1}$, and $\mathbf{x}_3^* = \frac{\rho(A \nu \mathbf{x}_1^*)}{d_1 d_2}$.

Next, we investigate the stability of the disease-free equilibrium \mathcal{E} and the endemic equilibrium \mathcal{E}^* of model (2.3). For simplicity, we denote ${}^{C}D_{0,t,w}^{\alpha,\beta,\beta}$ by $\mathcal{D}_{t,w}^{\alpha,\beta}$. For the rest of our study, we suppose w(t) = 1 and $\frac{\partial g(\eta,t)}{\partial t} > 0$ for all $\eta > 0$.

Theorem 4.2. Suppose that hypotheses (\mathfrak{H}_4) - (\mathfrak{H}_5) hold. Then the disease-free equilibrium \mathcal{E} of model (2.3) is stable if $\mathcal{R}_0 \leq 1$.

Proof. Let $\Omega = \{(x_1, x_2, x_3) \in \mathbb{R}^3, x_1 \leq x_1^0\}$ and suppose that $\Re_0 < 1$. Consider $\sigma < \frac{1-\Re_0}{\Re_0}$ and let the following Lyapunov functional

$$V_1(x_1, x_2, x_3) = \sigma(x_1^0 - x_1) + x_2 + (\sigma + 1) \frac{\xi(x_1^0, 0)}{d_2} x_3$$

We have

$$\begin{split} \mathcal{D}_{t,1}^{\alpha,\beta} V_1(x_1,x_2,x_3) &= -\sigma \mathcal{D}_{t,1}^{\alpha,\beta} x_1 + \mathcal{D}_{t,1}^{\alpha,\beta} x_2 + (\sigma+1) \frac{\xi(x_1^0,0)}{d_2} \mathcal{D}_{t,1}^{\alpha,\beta} x_3 \\ &\leqslant d_1 \frac{\partial g(\eta,t)}{\partial t} \left(\frac{(\sigma+1) d_2 \zeta(x_1^0,0) + (\sigma+1) \rho \xi(x_1^0,0)}{d_1 d_2} - 1 \right) x_2 \\ &\quad + \frac{\partial g(\eta,t)}{\partial t} \left(-\sigma(x_1^0 - x_1) + (\sigma+1) \left(\xi(x_1,x_3) - \xi(x_1^0,0) \right) x_3 \right) \\ &= -\frac{\partial g(\eta,t)}{\partial t} \sigma(x_1^0 - x_1) + d_1 \frac{\partial g(\eta,t)}{\partial t} \left((\sigma+1) \mathcal{R}_0 - 1 \right) x_2 \\ &\quad + (\sigma+1) \frac{\partial g(\eta,t)}{\partial t} \left(\xi(x_1,x_3) - \xi(x_1^0,0) \right) x_3. \end{split}$$

Since ζ , ξ are continuously differentiable functions satisfying hypotheses (\mathcal{H}_4)-(\mathcal{H}_5), we have

$$\mathcal{D}_{t,1}^{\alpha,\beta}V_1(x_1,x_2,x_3) \leqslant 0.$$

By applying Theorem 5 of [14], we deduce that the disease-free equilibrium of system (2.3) is stable in Ω when $\mathcal{R}_0 \leq 1$.

For $\Re_0 > 1$, we suppose following inequalities for the incidence functions ζ and ξ satisfy, for all $x_1, x_2, x_3 > 0$,

$$\left(1 - \frac{\zeta(x_1, x_2)}{\zeta(x_1, x_2^*)}\right) \left(\frac{\zeta(x_1, x_2^*)}{\zeta(x_1, x_2)} - \frac{x_2}{x_2^*}\right) \leqslant 0, \quad \left(1 - \frac{\zeta(x_1^*, x_2^*)\xi(x_1, x_3)}{\zeta(x_1, x_2^*)\xi(x_1^*, x_3^*)}\right) \left(\frac{\zeta(x_1, x_2^*)\xi(x_1^*, x_3^*)}{\zeta(x_1^*, x_2^*)\xi(x_1, x_3)} - \frac{x_3}{x_3^*}\right) \leqslant 0.$$
 (\mathcal{H}_6)

Applying (ii) of Theorem 2 in [14], we get the following result.

Lemma 4.3. Let $u(t) \in \mathbb{R}$ be a continuously differentiable function. For any constant u^* and for any time $t \ge a$, we define $K(t) = \int_{u^*}^{u(t)} \frac{\zeta(x_1^*, x_2^*)}{\zeta(x, x_2^*)} dx$. Then we have

$$\mathcal{D}_{t,1}^{\alpha,\beta}\mathsf{K}(t) \geq \frac{\zeta(x_1^*, x_2^*)}{\zeta(u, x_2^*)} \mathcal{D}_{t,1}^{\alpha,\beta} \mathfrak{u}(t).$$

By [14, Corollary 2], we get the following.

Lemma 4.4. Let $u(t) \in \mathbb{R}^+$ be a continuously differentiable function and $u^* > 0$. Then, for any time $t \ge a$, we have

$$\mathcal{D}_{t,1}^{\alpha,\beta}\left[u(t)-u^*-u^*\ln\frac{u(t)}{u^*}\right] \leqslant \left(1-\frac{u^*}{u(t)}\right)\mathcal{D}_{t,1}^{\alpha,\beta}u(t)$$

Theorem 4.5. Assume that $\Re_0 > 1$ and (\Re_4) - (\Re_6) hold. Then the endemic equilibrium \mathcal{E}^* of model (2.3) is stable. *Proof.* Consider a Lyapunov functional defined by

$$V_{2}(x_{1}, x_{2}, x_{3}) = x_{1} - x_{1}^{*} - \int_{x_{1}^{*}}^{x_{1}} \frac{\zeta(x_{1}^{*}, x_{2}^{*})}{\zeta(x, x_{2}^{*})} dx + x_{2}^{*} \mathcal{P}\left(\frac{x_{2}}{x_{2}^{*}}\right) + \frac{\xi(x_{1}^{*}, x_{3}^{*})}{d_{2}} x_{3}^{*} \mathcal{P}\left(\frac{x_{3}}{x_{3}^{*}}\right),$$

where $\mathcal{P}(x) = x - 1 - \ln x$ for x > 0. By Lemmas 4.3 and 4.4, we obtain

$$\mathcal{D}_{t,w}^{\alpha,\beta} V_2(x_1, x_2, x_3) \leqslant \left(1 - \frac{\zeta(x_1^*, x_2^*)}{\zeta(x_1, x_2^*)}\right) \mathcal{D}_{t,w}^{\alpha,\beta} x_1(t) + \left(1 - \frac{x_2^*}{x_2}\right) \mathcal{D}_{t,w}^{\alpha,\beta} x_2(t) + \frac{\xi(x_1^*, x_3^*)}{d_2} \left(1 - \frac{x_3^*}{x_3}\right) \mathcal{D}_{t,w}^{\alpha,\beta} x_3(t).$$

Then

$$\begin{split} \left(\frac{\partial g(\eta, t)}{\partial t}\right)^{-1} \mathcal{D}_{t,w}^{\alpha,\beta} \mathsf{V}_2(x_1, x_2, x_3) \leqslant \left(1 - \frac{\zeta(x_1^*, x_2^*)}{\xi(x_1, x_2^*)}\right) \left(\mathcal{A} - \mathsf{v} x_1 - \zeta(x_1, x_2) x_2 - \xi(x_1, x_3) x_3\right) + \left(1 - \frac{x_2^*}{x_2}\right) \\ \times \left(\zeta(x_1, x_2) x_2 + \xi(x_1, x_3) x_3 - d_1 x_2\right) + \frac{\xi(x_1^*, x_3^*)}{d_2} \left(1 - \frac{x_3^*}{x_3}\right) \left(\rho x_2 - d_2 x_3\right). \end{split}$$

Since $\mathcal{A} = \nu x_1^* + \zeta(x_1^*, x_2^*) x_2^* + \xi(x_1^*, x_3^*) x_3^*$, $\zeta(x_1^*, x_2^*) x_2^* + \xi(x_1^*, x_3^*) x_3^* = d_1 x_2^*$ and $\rho x_2^* = d_2 x_3^*$, we get by simple calculus that

$$\begin{split} \left(\frac{\partial g(\eta,t)}{\partial t}\right)^{-1} \mathcal{D}_{t,w}^{\alpha,\beta} V_2(x_1,x_2,x_3) \\ &\leqslant v x_1^* \left(1-\frac{x_1}{x_1^*}\right) \left(1-\frac{\zeta(x_1^*,x_2^*)}{\zeta(x_1,x_2^*)}\right) + \zeta(x_1^*,x_2^*) x_2^* \left(-1-\frac{x_2}{x_2^*}+\frac{\zeta(x_1,x_2^*)}{\zeta(x_1,x_2)}+\frac{\zeta(x_1,x_2)x_2}{\zeta(x_1,x_2)x_2^*}\right) \\ &+ \xi(x_1^*,x_3^*) x_3^* \left(-1-\frac{x_3}{x_3^*}+\frac{\zeta(x_1,x_2^*)\xi(x_1^*,x_3^*)}{\zeta(x_1^*,x_2^*)\xi(x_1,x_3)}+\frac{\zeta(x_1^*,x_2^*)\xi(x_1,x_3)x_3}{\zeta(x_1,x_2^*)\xi(x_1^*,x_3^*)x_3^*}\right) \\ &+ \zeta(x_1^*,x_2^*) x_2^* \left(3-\frac{\zeta(x_1,x_2^*)}{\zeta(x_1,x_2)}-\frac{\zeta(x_1^*,x_2^*)}{\zeta(x_1,x_2^*)}-\frac{\zeta(x_1,x_2)}{\zeta(x_1^*,x_2^*)}\right) \\ &+ \xi(x_1^*,x_3^*) x_3^* \left(4-\frac{\zeta(x_1^*,x_2^*)}{\zeta(x_1,x_2^*)}-\frac{\zeta(x_1,x_2^*)\xi(x_1^*,x_3^*)}{\zeta(x_1^*,x_2^*)\xi(x_1,x_3)}-\frac{\xi(x_1,x_3)x_3x_2^*}{\xi(x_1^*,x_3^*)x_3^*x_2}-\frac{x_2x_3^*}{x_2^*x_3}\right). \end{split}$$

We have

$$3 - \frac{\zeta(x_1, x_2^*)}{\zeta(x_1, x_2)} - \frac{\zeta(x_1^*, x_2^*)}{\zeta(x_1, x_2^*)} - \frac{\zeta(x_1, x_2)}{\zeta(x_1^*, x_2^*)} \leqslant 0,$$

and

$$4 - \frac{\zeta(x_1^*, x_2^*)}{\zeta(x_1, x_2^*)} - \frac{\zeta(x_1, x_2^*)\xi(x_1^*, x_3^*)}{\zeta(x_1^*, x_2^*)\xi(x_1, x_3)} - \frac{\xi(x_1, x_3)x_3x_2^*}{\xi(x_1^*, x_3^*)x_3^*x_2} - \frac{x_2x_3^*}{x_2^*x_3} \leqslant 0.$$

By (\mathcal{H}_4) , we get

$$\left(1-\frac{x_1}{x_1^*}\right)\left(1-\frac{\zeta(x_1^*,x_2^*)}{\zeta(x_1,x_2^*)}\right)\leqslant 0.$$

On the other hand, inequality (\mathcal{H}_6) leads to

$$-1 - \frac{x_2}{x_2^*} + \frac{\zeta(x_1, x_2^*)}{\zeta(x_1, x_2)} + \frac{\zeta(x_1, x_2)x_2}{\zeta(x_1, x_2^*)x_2^*} = \left(1 - \frac{\zeta(x_1, x_2^*)}{\zeta(x_1, x_2^*)}\right) \left(\frac{\zeta(x_1, x_2^*)}{\zeta(x_1, x_2)} - \frac{x_2}{x_2^*}\right) \leqslant 0,$$

and

$$\begin{split} &-1 - \frac{x_3}{x_3^*} + \frac{\zeta(x_1, x_2^*)\xi(x_1^*, x_3^*)}{\zeta(x_1^*, x_2^*)\xi(x_1, x_3)} + \frac{\zeta(x_1^*, x_2^*)\xi(x_1, x_3)x_3}{\zeta(x_1, x_2^*)\xi(x_1^*, x_3^*)x_3^*} \\ &= \left(1 - \frac{\zeta(x_1, x_2^*)\xi(x_1^*, x_3^*)}{\zeta(x_1^*, x_2^*)\xi(x_1, x_3)}\right) \left(\frac{\zeta(x_1, x_2^*)\xi(x_1^*, x_3^*)}{\zeta(x_1^*, x_2^*)\xi(x_1, x_3)} - \frac{x_3}{x_3^*}\right) \leqslant 0. \end{split}$$

Hence, $\left(\frac{\partial g(\eta,t)}{\partial t}\right)^{-1} \mathcal{D}_{t,w}^{\alpha,\beta} V_2(x_1,x_2,x_3) \leqslant 0$. Thus, $\mathcal{D}_{t,w}^{\alpha,\beta} V_2(x_1,x_2,x_3) \leqslant 0$. It follows from [14, Theorem 5] that the endemic equilibrium \mathcal{E}^* of system (2.3) is stable when $\mathcal{R}_0 > 1$.

5. Application and numerical simulations

As an application of our theoretical results, we choose

$$\begin{cases} \mathcal{D}_{t,1}^{\alpha,\beta} x_1(t) = \frac{\partial g(\eta,t)}{\partial t} \left(\mathcal{A} - \nu x_1 - \frac{\kappa_1 x_1 x_2}{1 + \epsilon_1 x_2} - \frac{\kappa_2 x_1 x_3}{1 + \epsilon_2 x_3} \right), \\ \mathcal{D}_{t,1}^{\alpha,\beta} x_2(t) = \frac{\partial g(\eta,t)}{\partial t} \left(\frac{\kappa_1 x_1 x_2}{1 + \epsilon_1 x_2} + \frac{\kappa_2 x_1 x_3}{1 + \epsilon_2 x_3} - d_1 x_2 \right), \\ \mathcal{D}_{t,1}^{\alpha,\beta} x_3(t) = \frac{\partial g(\eta,t)}{\partial t} \left(\rho x_2 - d_2 x_3 \right). \end{cases}$$
(5.1)

Model (5.1) is a special case of system (2.3) with κ_1 and κ_2 represent the infection rates of carrier and infected individuals, respectively. Also ϵ_1 and ϵ_2 are the saturation rates. Here, we have $\zeta(x_1, x_2) = \frac{\kappa_1 x_1}{1 + \epsilon_1 x_2}$ and $\xi(x_1, x_3) = \frac{\kappa_2 x_1}{1 + \epsilon_2 x_3}$. In this case, the basic reproduction number \mathcal{R}_0 of (5.1) is given by

$$\Re_0 = \frac{\mathcal{A}(\mathbf{d}_2 \kappa_1 + \rho \kappa_2)}{\nu \mathbf{d}_1 \mathbf{d}_2}$$

Obviously, ζ and ξ satisfy the conditions (\mathcal{H}_4), (\mathcal{H}_5), and (\mathcal{H}_6). By applying Theorems 4.2 and 4.5, we get the following corollary.

Corollary 5.1.

- (i) When $\mathcal{R}_0 \leq 1$, the disease-free equilibrium \mathcal{E} of model (5.1) is stable.
- (ii) When $\Re_0 > 1$, the equilibrium \mathcal{E} becomes unstable and the endemic equilibrium \mathcal{E}^* of model (5.1) is stable.

By applying the GHF integral in both sides of equations of model (5.1), we obtain

$$\begin{split} x_1(t) &= x_1(0) + \frac{1-\alpha}{N(\alpha)} \frac{\partial g(\eta, t)}{\partial t} G_1(t, x_1(t)) + \frac{\alpha}{N(\alpha)\Gamma(\beta)} \int_0^t (t-\tau)^{\beta-1} \frac{\partial g(\eta, \tau)}{\partial \tau} G_1(\tau, x_1(\tau)) d\tau, \\ x_2(t) &= x_2(0) + \frac{1-\alpha}{N(\alpha)} \frac{\partial g(\eta, t)}{\partial t} G_2(t, x_2(t)) + \frac{\alpha}{N(\alpha)\Gamma(\beta)} \int_0^t (t-\tau)^{\beta-1} \frac{\partial g(\eta, \tau)}{\partial \tau} G_2(\tau, x_2(\tau)) d\tau, \\ x_3(t) &= x_3(0) + \frac{1-\alpha}{N(\alpha)} \frac{\partial g(\eta, t)}{\partial t} G_3(t, x_3(t)) + \frac{\alpha}{N(\alpha)\Gamma(\beta)} \int_0^t (t-\tau)^{\beta-1} \frac{\partial g(\eta, \tau)}{\partial \tau} G_3(\tau, x_3(\tau)) d\tau, \end{split}$$

where

$$\begin{split} & \mathsf{G}_{1}\left(t,x_{1}(t)\right)=\mathcal{A}-\nu x_{1}-\frac{\kappa_{1}x_{1}x_{2}}{1+\varepsilon_{1}x_{2}}-\frac{\kappa_{2}x_{1}x_{3}}{1+\varepsilon_{2}x_{3}},\\ & \mathsf{G}_{2}\left(t,x_{2}(t)\right)=\frac{\kappa_{1}x_{1}x_{2}}{1+\varepsilon_{1}x_{2}}+\frac{\kappa_{2}x_{1}x_{3}}{1+\varepsilon_{2}x_{3}}-d_{1}x_{2},\\ & \mathsf{G}_{3}\left(t,x_{3}(t)\right)=\rho x_{2}-d_{2}x_{3}. \end{split}$$

We use the numerical method based on the Lagrange polynomial interpolation cited in [16] to approximate the solution of model (5.1). By interpolating at point $t_n = n\Delta t$, we obtain

$$\begin{split} x_{1,n+1} &= x_{1,0} + \frac{1-\alpha}{N(\alpha)} G_1(t_n, x_{1,n}) + \frac{\alpha(\Delta t)^{\beta}}{N(\alpha)\Gamma(\beta+2)} \\ &\times \sum_{k=0}^n \frac{\partial g(\eta, t_k)}{\partial t_k} G_1(t_k, x_{1,k}) \mathcal{A}_{n,k,\beta} + G_1(t_{k-1}, x_{1,k-1}) \mathcal{B}_{n,k,\beta}, \\ x_{2,n+1} &= x_{2,0} + \frac{1-\alpha}{N(\alpha)} G_2(t_n, x_{1,n}) + \frac{\alpha(\Delta t)^{\beta}}{N(\alpha)\Gamma(\beta+2)} \\ &\times \sum_{k=0}^n \frac{\partial g(\eta, t_k)}{\partial t_k} G_2(t_k, x_{2,k}) \mathcal{A}_{n,k,\beta} + G_2(t_{k-1}, x_{2,k-1}) \mathcal{B}_{n,k,\beta}, \\ x_{3,n+1} &= x_{3,0} + \frac{1-\alpha}{N(\alpha)} G_3(t_n, x_{3,n}) + \frac{\alpha(\Delta t)^{\beta}}{N(\alpha)\Gamma(\beta+2)} \\ &\times \sum_{k=0}^n \frac{\partial g(\eta, t_k)}{\partial t_k} G_3(t_k, x_{3,k}) \mathcal{A}_{n,k,\beta} + G_3(t_{k-1}, x_{3,k-1}) \mathcal{B}_{n,k,\beta}, \end{split}$$

where

$$\begin{split} \mathcal{A}_{\mathbf{n},\mathbf{k},\boldsymbol{\beta}} &= (\mathbf{n}-\mathbf{k}+1)^{\beta}(\mathbf{n}-\mathbf{k}+2+\boldsymbol{\beta}) - (\mathbf{n}-\mathbf{k})^{\beta}(\mathbf{n}-\mathbf{k}+2+2\boldsymbol{\beta}),\\ \mathcal{B}_{\mathbf{n},\mathbf{k},\boldsymbol{\beta}} &= (\mathbf{n}-\mathbf{k})^{\beta}(\mathbf{n}-\mathbf{k}+1+\boldsymbol{\beta}) - (\mathbf{n}-\mathbf{k}+1)^{\beta+1}. \end{split}$$

For numerical simulations, let $N(\alpha) = 1 - \alpha + \frac{\alpha}{\Gamma(\alpha)}$. Furthermore, we take $g(t, \eta) = \frac{t^{2-\eta}}{2-\eta}$. The valuers of the others parameters are given in Table 1, which are taken from [18].

Parameters	Definition	Value
A	Recruitment rate	50
κ ₁	Transmission contact rate between x_1 and x_3	$1.2 imes 10^{-5}$
κ2	Transmission contact rate between x_1 and x_3	Varied
ρ	Symptoms period	$1/7~{ m day}^{-1}$
ϵ_1	The measure of inhibition effect for carrier	0.04
ϵ_1	The measure of inhibition effect for infected	0.01
ν	Natural death rate	$0.01~{ m day}^{-1}$
r_1	Recovery rate from carrier	$1/21~{ m day}^{-1}$
r_2	Recovery rate from infected	$1/15~{ m day}^{-1}$
d	Death due to disease rate	$0.1~{ m day}^{-1}$

Table 1: Parameter values model (5.1).

Consider $\kappa_2 = 5.5 \times 10^{-5}$, we have $\Re_0 = 0.9532 \leq 1$. Therefore, model (5.1) has a disease-free equilibrium $\mathcal{E}(5000, 0, 0)$. By Corollary 5.1 (i), we know that \mathcal{E} is stable. Figure 2 illustrates this result. For $\kappa_2 = 1.3 \times 10^{-5}$, we have $\Re_0 = 1.9489 > 1$. Thus, model (5.1) has a endemic equilibrium. By Corollary 5.1 (i), we know that \mathcal{E}^* is stable. Figure 4 demonstrates this finding.

We notice that when the parameter α is close to 1, the graph of susceptible, asymptomatic, and infected individuals converges rapidly to the disease-free equilibrium or to the endemic equilibrium. Also, we observe the effect of modifying the fractal order η on the graph of susceptible individuals through Figures 2, 3, 4, and 5. While in Figures 6 and 7, we notice the effect of varying the fractional order β on susceptible and infected individuals.



Figure 2: Dynamics of model (5.1) when $\mathcal{R}_0=0.9532\leqslant 1,$ $\eta=1,$ and $\beta=1.$



Figure 3: Dynamics of model (5.1) when $\mathcal{R}_0=0.9532\leqslant 1, \eta=0.87,$ and $\beta=1.$



Figure 4: Dynamics of model (5.1) when $\mathcal{R}_0=1.9489>1,$ $\eta=1,$ and $\beta=1.$



Figure 6: Dynamics of model (5.1) when $\Re_0 = 0.9532 \leqslant 1$, $\eta = 1$, and $\beta = 0.7$.



Figure 7: Dynamics of model (5.1) when $\Re_0=1.9489>1,$ $\eta=1,$ and $\beta=0.7.$

6. Conclusion

In this work, we have proposed a fractal-fractional epidemic model for COVID-19 with carrier effect. The two modes of transmission via direct contact with asymptomatic and symptomatic individuals have been modeled by two general incidence functions. The existence and uniqueness of solution has been established by Krasnoselskii's fixed point theorem which combines between Banach contraction principal and Schauder fixed point theorem. The stability conditions of the equilibrium points have bee investigated by means of Lyapunov functionals. Finally, we have presented numerical simulations to support our analytical results. We conclude that the dynamics of our formulated model has a significant result by using the Hattaf fractal-fractional derivative, which covers and generalizes various nonlocal operators existing in the literature.

It is known that COVID-19 can be transmitted not only through direct contact with asymptomatic and symptomatic people, but it can also spread through a contaminated environment [18]. In future research, it would be very interesting to extend our model by taking into account the three transmission modes and other factors.

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