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Dynamics of a Generalized Fractional Epidemic Model of COVID-19 with Carrier Effect

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Abstract: Currently, coronavirus disease 2019 (COVID-19) continues to cause several new cases and deaths, and further new variants of the virus responsible this disease appear. The present paper proposes a new mathematical model to understand the mechanisms of the spread of COVID-19 and better describe its dynamics. The modes of infection spread of COVID-19 via asymptomatic and symptomatic individuals are modeled by two general nonlinear incidence functions in order to include several types of incidence rates existing in the literature. When a disease outbreak within a community, individuals acquire information about this disease. Therefore, the proposed model take into account the memory effect on the outbreaks of COVID-19. This effect is modeled by a fractional order derivative in Caputo sense. The mathematical analysis of the proposed model is rigorously investigated, including the computation of the basic reproduction number $\mathcal{R}_0$ and the stability of equilibria.

Keywords: COVID-19, SARS-CoV-2, incidence rate, basic reproduction number, fractional differential equations, stability analysis.

1. INTRODUCTION

Coronavirus disease 2019 (COVID-19) is a infectious disease caused by a new coronavirus called severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). World Health Organization (WHO) first learned of this new virus on 31 December 2019, following a report of a cluster of cases of viral pneumonia detected in Wuhan City, Hubei Province of China. Due to rapid worldwide outbreak of COVID-19, the WHO declared it a pandemic on 11 March 2020. Over 2.7 million new cases were reported on 7 March 2021 with 116 166 652 cumulative cases, 60 323 new deaths and 2 582 528 cumulative deaths [18]. Since the appearance of the first case in Morocco on 2 March 2020 in the city of Casablanca, the disease has caused 8 716 deaths on 12 March 2021 according to new statistics given by Moroccan Ministry of Health (MMH) [13]. However, a total of 13 671 deaths was confirmed in Iraq on the same day [19]. The outbreak COVID-19 impacted nearly every side of society worldwide. Therefore, the pandemic has required a substantial response by public health authorities at every level and considered aformidable global public health challenge.

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Despite the prevention measures, such as hand washing, social distancing, quarantine of suspected cases, isolation of confirmed cases, the closure of schools and non-essential businesses and services, and also the start of vaccination in some countries, COVID-19 continues to cause new cases and deaths, and new variants of SARS-CoV-2 appear.

In fact, the transmission or outbreaks of the COVID-19 consists of direct and indirect contacts between infective and susceptible individuals, it seems that the several mathematical models well described the COVID-19. However, attention must be paid to the unique traits of COVID-19. These traits are (a) the presence of individuals who exhibit infectious symptoms even during the incubation period, such as such as High body temperature, body aches and pains, queasiness, or diarrhea typically (2 - 14) days after infective to the virus, (b) There are asymptomatic individuals called a carrier, who can move freely and transmit infection, which makes controlling the situation complicated such that, approximately half of the COVID-19 patients $(40 - 50\%)$ in one study did not show any symptoms from above in (a). For example, there are several reports that have investigated the possibility of asymptomatic carriers of COVID-19. A report of Japanese travelers from Wuhan, China approximates the proportion of carriers of disease but asymptomatic at 30 %. In addition, studies of every person in an isolated Italian village revealed that 50-75 % of people were asymptomatic. A report of a nursing home in the USA found 30 % of patients were asymptomatic on the day of testing for COVID-19, with 4 % remaining asymptomatic upon follow-up a week later. A report in hospitalized patients in Beijing found that 5 % of patients testing positive for COVID-19 had asymptomatic infections see [3–5, 10, 15].

Recently, many mathematical models have been developed to describe the spread of infectious diseases, such as hepatitis B, HIV transmission and Ebola virus disease. For more information, we refer the reader to the recent book published by Hattaf and Dutta about this subject in [7]. For COVID-19, Moussaoui and Zerga [14] proposed a mathematical model that takes into account strategies against COVID-19 such as wearing masks and respecting safety distances. A model for the dynamics of COVID-19 with quarantine strategy and media coverage effect was presented by Mohsen et al. in [12], while a mathematical model of COVID-19 pandemic involving the infective immigrants was also studied in [11]. A fractional order model with bilinear incidence rate was introduced in [2] to study transmission dynamics of COVID-19 in Japan 2020. Modeling of the dynamics of COVID-19 inside the human body was presented in [9].

The main objective of this study is to develop a new mathematical model in order to better describe the dynamics of the COVID-19 in human population by taking into account the effects of memory and carrier, and also others aspects such as the non-linearity of the incidence function, the death rate due to COVID-19 and the recovery rates of the asymptomatic and symptomatic individuals. To do this, the structure of this work is outlined as follows. The next section is devoted to the formulation of model, the existence of equilibria and the computation of the basic reproduction number $\mathcal{R}_0$. In Section 3, we use mathematical analysis to establish global stability results for the proposed model. Some applications of our main results are presented in Section 4. Eventually, the conclusion is given in Section 5.

2. MODEL FORMULATION AND EQUILIBRIA

In this section, we first construct a mathematical model that takes into account the effects of memory and carrier. Then we divide the total population into four classes $\mathcal{S}(t)$, $\mathcal{C}(t)$, $\mathcal{I}(t)$ and $\mathcal{R}(t)$ that represent susceptible, carrier (asymptomatic infected individuals), infected and recovered individuals at time t, respectively. The dynamics

of the four classes is governed by the following nonlinear system of FDEs:

$$\begin{cases} D^{\alpha}\mathcal{S}(t) &= \mathcal{A} - \nu\mathcal{S} - \Phi(\mathcal{S},\mathcal{C})\mathcal{C} - \Psi(\mathcal{S},\mathcal{I})\mathcal{I}, \\ D^{\alpha}\mathcal{C}(t) &= \Phi(\mathcal{S},\mathcal{C})\mathcal{C} + \Psi(\mathcal{S},\mathcal{I})\mathcal{I} - (\nu + d + \gamma + r_1)\mathcal{C}, \\ D^{\alpha}\mathcal{I}(t) &= \gamma\mathcal{C} - (\nu + d + r_2)\mathcal{I}, \\ D^{\alpha}\mathcal{R}(t) &= r_1\mathcal{C} + r_2\mathcal{I} - \nu\mathcal{R}, \end{cases} \tag{2.1}$$

where the susceptible individuals are recruited at a rate $\mathcal{A}$ and become infected either by effective contact with carrier at rate $\Phi(\mathcal{S},\mathcal{C})\mathcal{C}$ or by effective contact with infected individuals at rate $\psi(\mathcal{S},\mathcal{I})\mathcal{I}$. So, the term $\Phi(\mathcal{S},\mathcal{C})\mathcal{C} + \psi(\mathcal{S},\mathcal{I})\mathcal{I}$ denotes the total asymptomatic infection rate of susceptible individuals. The natural death rate in all classes is denoted by ν, while d is the death rate due to COVID-19. The rate of transfer from the asymptomatic to symptomatic is denoted by γ. The parameters r_1 and r_2 are recovery rates of the asymptomatic and symptomatic individuals, respectively. Finally, D^{α} denotes the Caputo fractional derivative with $\alpha \in (0, 1]$ that describes the memory effect. A schematic diagram of model (2.1) is shown in Figure 1.

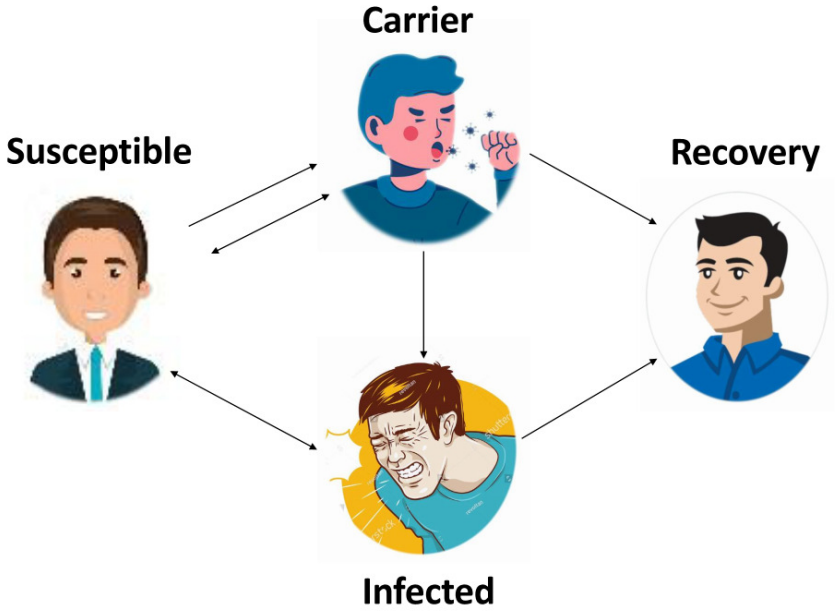

Fig. 2.1. The diagram of model (2.1).

Obviously, the first three equations of (2.1) do not depend on the variable $\mathcal{R}$, model (2.1) can be rewrite by following format

$$\begin{cases} D^{\alpha}\mathcal{S}(t) &= \mathcal{A} - \nu\mathcal{S} - \Phi(\mathcal{S},\mathcal{C})\mathcal{C} - \Psi(\mathcal{S},\mathcal{I})\mathcal{I}, \\ D^{\alpha}\mathcal{C}(t) &= \Phi(\mathcal{S},\mathcal{C})\mathcal{C} + \Psi(\mathcal{S},\mathcal{I})\mathcal{I} - d_1\mathcal{C}, \\ D^{\alpha}\mathcal{I}(t) &= \gamma\mathcal{C} - d_2\mathcal{I}, \end{cases} \tag{2.2}$$

where $d_1 = \nu + d + \gamma + r_1$ and $d_2 = \nu + d + r_2$. Furthermore and according to [8], we assume that the infection rate is the general incidences Φ and Ψ are continuously differentiable in the interior of $\mathbb{R}^2_+$ and satisfy the two following conditions:

$$(H_1)\ \ \Phi(0,\mathcal{C}) = 0,\ \frac{\partial\Phi}{\partial\mathcal{S}}(\mathcal{S},\mathcal{C}) > 0,\ \frac{\partial\Phi}{\partial\mathcal{C}}(\mathcal{S},\mathcal{C}) \leq 0 \text{ for all } \mathcal{S},\mathcal{C} \geq 0.$$

(H_2) $\Psi(0,\mathcal{I})=0$, $\dfrac{\partial\Psi}{\partial\mathcal{S}}(\mathcal{S},\mathcal{I})>0$, $\dfrac{\partial\Psi}{\partial\mathcal{I}}(\mathcal{S},\mathcal{I})\leq 0$ for all $\mathcal{S},\mathcal{I}\geq 0$.

Next, we study the existence of equilibria. Clearly, $\mathcal{E}_f(\mathcal{S}^0,0,0)$ is a disease-free equilibrium of (2.2), where $\mathcal{S}^0=\frac{\mathcal{A}}{\nu}$. Hence, the basic reproduction number of (2.2) can be calculated as follows

$$\mathcal{R}_0=\frac{d_2\Phi(\mathcal{S}^0,0)+\gamma g(\mathcal{S}^0,0)}{d_1d_2}, \tag{2.3}$$

which can be rewritten as $\mathcal{R}_{01}+\mathcal{R}_{02}$, where

- $\mathcal{R}_{01}=\dfrac{\Phi(\mathcal{S}^0,0)}{d_1}$ is the basic reproduction number for asymptomatic mode of transmission.
- $\mathcal{R}_{02}=\dfrac{\gamma\Psi(\mathcal{S}^0,0)}{d_1d_2}$ is the basic reproduction number for symptomatic mode of transmission.

The second equilibrium point of (2.2) satisfies the following system of equations

$$\begin{aligned}\mathcal{A}-\nu\mathcal{S}-\Phi(\mathcal{S},\mathcal{C})\mathcal{C}-\Psi(\mathcal{S},\mathcal{I})\mathcal{I} &= 0, &(2.4)\\ \Phi(\mathcal{S},\mathcal{C})\mathcal{C}+\Psi(\mathcal{S},\mathcal{I})\mathcal{I}-d_1\mathcal{C} &= 0, &(2.5)\\ \gamma\mathcal{C}-d_2\mathcal{I} &= 0. &(2.6)\end{aligned}$$

Hence, $\mathcal{C}=\dfrac{\mathcal{A}-\nu\mathcal{S}}{d_1}$, $\mathcal{I}=\dfrac{\gamma\mathcal{C}}{d_2}=\dfrac{\gamma(\mathcal{A}-\nu\mathcal{S})}{d_1d_2}$ and

$$d_2\Phi(\mathcal{S},\frac{\mathcal{A}-\nu\mathcal{S}}{d_1})+\gamma\Psi(\mathcal{S},\frac{\gamma(\mathcal{A}-\nu\mathcal{S})}{d_1d_2})=d_1d_2.$$

Since $\mathcal{C}=\dfrac{\mathcal{A}-\nu\mathcal{S}}{d_1}\geq 0$, we have $\mathcal{S}\leq\dfrac{\mathcal{A}}{\nu}$. Then there is no epidemiological equilibrium when $\mathcal{S}>\dfrac{\mathcal{A}}{\nu}$. The function h can be define on the closed interval $[0,\frac{\mathcal{A}}{\nu}]$ by

$$h(\mathcal{S}) \;=\; d_2\Phi(\mathcal{S},\frac{\mathcal{A}-\nu\mathcal{S}}{d_1})+\gamma\Psi(\mathcal{S},\frac{\gamma(\mathcal{A}-\nu\mathcal{S})}{d_1d_2})-d_1d_2.$$

We have $h(0)=-d_1d_2<0$, $h(\dfrac{\mathcal{A}}{\nu})=d_1d_2(\mathcal{R}_0-1)$ and

$$h'(\mathcal{S})=d_2\left(\frac{\partial\Phi}{\partial\mathcal{S}}-\frac{\nu}{d_1}\frac{\partial\Phi}{\partial\mathcal{C}}\right)+\gamma\left(\frac{\partial\Psi}{\partial\mathcal{S}}-\frac{\gamma\nu}{d_1d_2}\frac{\partial\Psi}{\partial\mathcal{I}}\right)>0.$$

Therefore, the equation $h(\mathcal{S})=0$ has a unique root $\mathcal{S}^*\in(0,\dfrac{\mathcal{A}}{\nu})$ when $\mathcal{R}_0>1$.

This implies that our model has a unique endemic equilibrium $\mathcal{E}^*(\mathcal{S}^*,\mathcal{C}^*,\mathcal{I}^*)$ under the condition $\mathcal{R}_0>1$.

Summarizing all the above cases in the following result.

Theorem 2.1:
Let $\mathcal{R}_0$ be defined by (2.3).

(i) If $\mathcal{R}_0 \leq 1$, then system (2.2) has only disease-free equilibrium of the form $\mathcal{E}_f(\mathcal{S}^0, 0, 0)$, where $\mathcal{S}^0 = \frac{\mathcal{A}}{\nu}$.

(ii) If $\mathcal{R}_0 > 1$, then system (2.2) has a unique endemic equilibrium $\mathcal{E}^*(\mathcal{S}^*, \mathcal{C}^*, \mathcal{I}^*)$, where $\mathcal{S}^* \in (0, \frac{\mathcal{A}}{\nu})$, $\mathcal{C}^* = \dfrac{\mathcal{A} - \nu\mathcal{S}^*}{d_1}$ and $\mathcal{I}^* = \dfrac{\gamma(\mathcal{A} - \nu\mathcal{S}^*)}{d_1 d_2}$. Also, the disease-free equilibrium exists.

3. GLOBAL DYNAMICS

In this section, we analyze the stability of the disease-free equilibrium $\mathcal{E}_f$ and the endemic equilibrium $\mathcal{E}^*$.

Theorem 3.1:
The disease-free equilibrium E_f is globally asymptotically stable if $\mathcal{R}_0 \leq 1$ and unstable if $\mathcal{R}_0 > 1$.

Proof
We define the following Lyapunov functional

$$\mathcal{F}(t) = \mathcal{C} + \frac{\Psi(\mathcal{S}^0, 0)}{d_2}\mathcal{I}.$$

Then

$$\begin{aligned} D^\alpha \mathcal{F}(t) &= D^\alpha \mathcal{C} + \frac{\Psi(\mathcal{S}^0, 0)}{d_2} D^\alpha \mathcal{I} \\ &= \Phi(\mathcal{S}, \mathcal{C})\mathcal{C} + \Psi(\mathcal{S}, \mathcal{I})\mathcal{I} - d_1 C + \frac{\Psi(\mathcal{S}^0, 0)}{d_2}(\gamma\mathcal{C} - d_2\mathcal{I}). \end{aligned}$$

Using $\mathcal{A} = \nu\mathcal{S}^0$, we get

$$D^\alpha \mathcal{F}(t) = d_1\left(\frac{d_2\Phi(\mathcal{S}, \mathcal{I}) + \gamma\Psi(\mathcal{S}^0, 0)}{d_1 d_2} - 1\right)\mathcal{C} + \Big(\Psi(\mathcal{S}, \mathcal{I}) - \Psi(\mathcal{S}^0, 0)\Big)\mathcal{I}.$$

From the first equation of (2.2), we have

$$D^\alpha \mathcal{S} \leq \mathcal{A} - \nu\mathcal{S},$$

which leads to

$$\mathcal{S}(t) \leq \mathcal{S}(0)E_\alpha(-\nu t^\alpha) + \frac{\mathcal{A}}{\nu}\left[1 - E_\alpha(-\nu t^\alpha)\right],$$

where $E_\alpha(z) = \displaystyle\sum_{k=0}^{\infty} \frac{z^\alpha}{\Gamma(\alpha k + 1)}$ denotes the Mittag-Leffler function of one parameter α.

Then $\limsup_{t\to\infty} \mathcal{S}(t) \leq \dfrac{\mathcal{A}}{\nu}$, which involves that all omega limit points verify $\mathcal{S}(t) \leq \mathcal{S}^0$. Thus, it suffices to consider solutions for which $\mathcal{S}(t) \leq \mathcal{S}^0$. According to the explicit expression of $\mathcal{R}_0$ given in (2.3) and (H_1)-(H_2), we obtain

$$D^\alpha \mathcal{F}(t) \leq d_1\big(\mathcal{R}_0 - 1\big)\mathcal{C}.$$

Then $D^\alpha \mathcal{F}(t) \leq 0$ when $\mathcal{R}_0 \leq 1$. Furthermore, $D^\alpha \mathcal{F}(t) = 0$ if and only if $\mathcal{S} = \mathcal{S}^0$, $\mathcal{C} = 0$ and $\mathcal{I} = 0$. Therefore, $\{\mathcal{E}_f\}$ is the largest invariant set in $\{(\mathcal{S}, \mathcal{C}, \mathcal{I}) \mid D^\alpha \mathcal{F}(t) = 0\}$. It

follows from the LaSalle's invariance principale [16] that $\mathcal{E}_f$ is globally asymptotically stable when $\mathcal{R}_0 \leq 1$.

On the other hand, the characteristic equation of model (2.2) at $\mathcal{E}_f$ is given by

$$(\nu + \lambda)(\alpha + \nu + \lambda)\Theta(\lambda) = 0, \tag{3.7}$$

where

$$\Theta(\lambda) = \lambda^2 + \big(d_1 + d_1 - \Phi(\mathcal{S}^0, 0)\big)\lambda + d_1 d_2(1 - \mathcal{R}_0).$$

We have $\lim\limits_{\lambda \to +\infty} \Theta(\lambda) = +\infty$ and $\Theta(0) = d_1 d_2(1 - \mathcal{R}_0) < 0$ when $\mathcal{R}_0 > 1$. Then we have the eigenvalue $\lambda_0 \in (0, +\infty)$ such that $\Theta(\lambda_0) = 0$, which implies that the equation (3.7) at $\mathcal{E}_f$ has at least one positive root if $\mathcal{R}_0 > 1$. Therefore, E_f is saddle (unstable) whenever $\mathcal{R}_0 > 1$. □

In the following, we assume that $\mathcal{R}_0 > 1$ and the incidence functions Φ and Ψ satisfy, for all $\mathcal{S}, \mathcal{C}, \mathcal{I} > 0$, the following assumption

$$\begin{array}{l} \left(1 - \dfrac{\Phi(\mathcal{S}, \mathcal{C})}{\Phi(\mathcal{S}, \mathcal{C}^*)}\right)\left(\dfrac{\Phi(\mathcal{S}, \mathcal{C}^*)}{\Phi(\mathcal{S}, \mathcal{C})} - \dfrac{\mathcal{C}}{\mathcal{C}^*}\right) \leq 0, \\ \left(1 - \dfrac{\Phi(\mathcal{S}^*, \mathcal{C}^*)\Psi(\mathcal{S}, \mathcal{I})}{\Phi(\mathcal{S}, \mathcal{C}^*)\Psi(\mathcal{S}^*, \mathcal{I}^*)}\right)\left(\dfrac{\Phi(\mathcal{S}, \mathcal{C}^*)\Psi(\mathcal{S}^*, \mathcal{I}^*)}{\Phi(\mathcal{S}^*, \mathcal{C}^*)\Psi(\mathcal{S}, \mathcal{I})} - \dfrac{\mathcal{I}}{\mathcal{I}^*}\right) \leq 0. \end{array} \tag{H_4}$$

To establish the global dynamics of system (2.2) when $\mathcal{R}_0 > 1$, we need the following Lemma.

Lemma 3.1:
Let k be a continuous function and u be a continuously differentiable function. For any constant c and $\alpha \in (0, 1]$, the Caputo fractional derivative of the function K defined by

$$K(t) = \int_c^{u(t)} k(x)dx, \tag{3.8}$$

satisfies the following property

$$D^\alpha K(t) = k(u(t))D^\alpha u(t) - \frac{t^{-\alpha}v(0)}{\Gamma(1-\alpha)} - \frac{\alpha}{\Gamma(1-\alpha)}\int_0^t (t-\tau)^{-\alpha-1}v(\tau)d\tau, \tag{3.9}$$

where

$$v(\tau) = k(u(t))\big(u(t) - u(\tau)\big) + \int_{u(t)}^{u(\tau)} k(x)dx.$$

In particular, we have $D^\alpha K(t) = k(u(t))D^\alpha u(t)$ when $\alpha = 1$. Moreover,

(i) *If k is a increasing function, then*

$$D^\alpha K(u(t)) \leq k(u(t))D^\alpha u(t). \tag{3.10}$$

(ii) *If k is a decreasing function, then*

$$D^\alpha K(u(t)) \geq k(u(t))D^\alpha u(t). \tag{3.11}$$

Proof
By using the definition of the Caputo fractional derivative, we have

$$D^\alpha K(t) - k(u(t))D^\alpha u(t) = \frac{1}{\Gamma(1-\alpha)}\int_0^t (t-\tau)^{-\alpha}u'(\tau)\big(k(u(\tau)) - k(u(t))\big)d\tau.$$

Clearly, $v'(\tau) = u'(\tau)\big(k(u(\tau)) - k(u(t))\big)$ and $v(t) = 0$. Integrating by parts the last integral, we get

$$D^\alpha K(t) - k(u(t))D^\alpha u(t) = \frac{1}{\Gamma(1-\alpha)}\big[(t-\tau)^{-\alpha}v(\tau)\big]_{\tau=0}^{\tau=t} - \frac{\alpha}{\Gamma(1-\alpha)}\int_0^t (t-\tau)^{-\alpha-1}v(\tau)d\tau.$$

From L'Hopital's rule, we have

$$\begin{aligned}\lim_{\tau\to t}(t-\tau)^{-\alpha}v(\tau) &= \lim_{\tau\to t}\frac{v(\tau)}{(t-\tau)^\alpha}\\ &= \lim_{\tau\to t}\frac{u'(\tau)\big(k(u(\tau)) - k(u(t))\big)}{-\alpha(t-\tau)^{\alpha-1}}\\ &= \lim_{\tau\to t} -\frac{1}{\alpha}(t-\tau)^{1-\alpha}u'(\tau)\big(k(u(\tau)) - k(u(t))\big) = 0.\end{aligned}$$

Hence,

$$D^\alpha K(t) = k(u(t))D^\alpha u(t) - \frac{t^{-\alpha}v(0)}{\Gamma(1-\alpha)} - \frac{\alpha}{\Gamma(1-\alpha)}\int_0^t (t-\tau)^{-\alpha-1}v(\tau)d\tau.$$

Now let us consider the following function

$$\psi_{c,k}(\tau) = k(c)\big(c-\tau\big) + \int_c^\tau k(x)dx.$$

It is obvious that $\psi'_{c,k}(\tau) = k(\tau) - k(c)$. Assume that k is a increasing function. In this case, the function $\psi_{c,k}(\tau)$ is decreasing on the interval $(-\infty, c]$ and increasing on $[c, +\infty)$ with $\psi_{c,k}(c) = 0$. Then $\psi_{c,k}(\tau)$ has the global minimum at $\tau = c$. Thus,

$$\psi_{c,k}(\tau) \geq 0, \text{ for all } (c,\tau) \in \mathbb{R}^2.$$

Since $v(\tau) = \psi_{u(t),k}(u(\tau))$, we deduce that $v(\tau) \geq 0$ and then (i). By using the same technique, we can easily prove (ii). By applying of the fundamental theorem of analysis, we obtain

$$D^\alpha K(t) = k(u(t))D^\alpha u(t), \text{ when } \alpha = 1. \tag{3.12}$$

This completes the proof. □

Remark 3.1:
Lemma 3.1 extends the lemma presented in [1] that allows to find Lyapunov candidate functions for demonstrating the stability of many fractional order systems, and also the elementary lemma given in [17] that estimates fractional derivatives of Volterra-type Lyapunov functions in the sense Caputo for any monotone function. In fact, we have

$$\frac{1}{2}D^\alpha u^2(t) = D^\alpha \int_c^{u(t)} x dx \leq u(t)D^\alpha u(t), \tag{3.13}$$

and

$$D^\alpha\big[u(t) - u^* - u^*\ln\frac{u(t)}{u^*}\big] = D^\alpha u(t) - u^* D^\alpha \int_{u^*}^{u(t)} \frac{1}{x}dx \leq \left(1 - \frac{u^*}{u(t)}\right)D^\alpha u(t). \tag{3.14}$$

Theorem 3.2:
Assume that $\mathcal{R}_0 > 1$ and (H_4) holds. Then the endemic equilibrium $\mathcal{E}^*$ is globally asymptotically stable.

Proof
We define a Lyapunov functional as follows

$$\mathcal{G}(t) = \mathcal{S} - \mathcal{S}^* - \int_{\mathcal{S}^*}^{\mathcal{S}} \frac{\Phi(\mathcal{S}^*, \mathcal{C}^*)}{\Phi(X, \mathcal{C}^*)} dX + \mathcal{C}^* \chi\left(\frac{\mathcal{C}}{\mathcal{C}^*}\right) + \frac{\Psi(\mathcal{S}^*, \mathcal{I}^*)}{d_2} \mathcal{I}^* \chi\left(\frac{\mathcal{I}}{\mathcal{I}^*}\right), \tag{3.15}$$

where $\chi(\hat{x}) = \hat{x} - 1 - \ln \hat{x}$ for $\hat{x} > 0$.

Since $S \mapsto \frac{1}{\Phi(X, \mathcal{C}^*)}$ is a decreasing function, it follows from (ii) of Lemma 3.1 and (3.14) that

$$\begin{aligned} D^\alpha \mathcal{G}(t) \leq & \left(1 - \frac{\Phi(\mathcal{S}^*, \mathcal{C}^*)}{\Phi(\mathcal{S}, \mathcal{C}^*)}\right) D^\alpha \mathcal{S}(t) + \left(1 - \frac{\mathcal{C}^*}{\mathcal{C}}\right) D^\alpha \mathcal{C}(t) \\ & + \frac{\Psi(\mathcal{S}^*, \mathcal{I}^*)}{d_2} \left(1 - \frac{\mathcal{I}^*}{\mathcal{I}}\right) D^\alpha \mathcal{I}(t) \\ = & \left(1 - \frac{\Phi(\mathcal{S}^*, \mathcal{C}^*)}{\Phi(\mathcal{S}, \mathcal{C}^*)}\right) \left(\mathcal{A} - \nu \mathcal{S} - \Phi(\mathcal{S}, \mathcal{C}) \mathcal{C} - \Psi(\mathcal{S}, \mathcal{I}) \mathcal{I}\right) \\ & + \left(1 - \frac{\mathcal{C}^*}{\mathcal{C}}\right) \left(\Phi(\mathcal{S}, \mathcal{C}) \mathcal{C} + \Psi(\mathcal{S}, \mathcal{I}) \mathcal{I} - d_1 \mathcal{C}\right) \\ & + \frac{\Psi(\mathcal{S}^*, \mathcal{I}^*)}{d_2} \left(1 - \frac{\mathcal{I}^*}{\mathcal{I}}\right) \left(\gamma \mathcal{C} - d_2 \mathcal{I}\right). \end{aligned}$$

Since $\mathcal{A} = \nu \mathcal{S}^* + \Phi(\mathcal{S}^*, \mathcal{C}^*) \mathcal{C}^* + \Psi(\mathcal{S}^*, \mathcal{I}^*) \mathcal{I}^*$, $\Phi(\mathcal{S}^*, \mathcal{C}^*) \mathcal{C}^* + \Psi(\mathcal{S}^*, \mathcal{I}^*) \mathcal{I}^* = d_1 \mathcal{C}^*$ and $\gamma \mathcal{C}^* = d_2 \mathcal{I}^*$, we have

$$\begin{aligned} D^\alpha \mathcal{G}(t) \leq & \nu \mathcal{S}^* \left(1 - \frac{\mathcal{S}}{\mathcal{S}^*}\right) \left(1 - \frac{\Phi(\mathcal{S}^*, \mathcal{C}^*)}{\Phi(\mathcal{S}, \mathcal{C}^*)}\right) \\ & + \Phi(\mathcal{S}^*, \mathcal{C}^*) \mathcal{C}^* \left(2 - \frac{\Phi(\mathcal{S}^*, \mathcal{C}^*)}{\Phi(\mathcal{S}, \mathcal{C}^*)} + \frac{\Phi(\mathcal{S}, \mathcal{C}) \mathcal{C}}{\Phi(\mathcal{S}, \mathcal{C}^*) \mathcal{C}^*} - \frac{\mathcal{C}}{\mathcal{C}^*}\right) \\ & + \Psi(\mathcal{S}^*, \mathcal{I}^*) \mathcal{I}^* \left(3 - \frac{\Phi(\mathcal{S}^*, \mathcal{C}^*)}{\Phi(\mathcal{S}, \mathcal{C}^*)} + \frac{\Phi(\mathcal{S}^*, \mathcal{C}^*) \Psi(\mathcal{S}, \mathcal{I}) \mathcal{I}}{\Phi(\mathcal{S}, \mathcal{C}^*) \Psi(\mathcal{S}^*, \mathcal{I}^*) \mathcal{I}^*} - \frac{\mathcal{I}}{\mathcal{I}^*} \right. \\ & \left. - \frac{\Psi(\mathcal{S}, \mathcal{I}) \mathcal{I} \mathcal{C}^*}{\Psi(\mathcal{S}^*, \mathcal{I}^*) \mathcal{I}^* \mathcal{C}} - \frac{\mathcal{I}^* \mathcal{C}}{\mathcal{I} \mathcal{C}^*}\right). \end{aligned}$$

Hence,

$$\begin{aligned} D^{\alpha}\mathcal{G}(t) \leq\ & \nu\mathcal{S}^*\left(1-\frac{\mathcal{S}}{\mathcal{S}^*}\right)\left(1-\frac{\Phi(\mathcal{S}^*,\mathcal{C}^*)}{\Phi(\mathcal{S},\mathcal{C}^*)}\right) \\ & +\Phi(\mathcal{S}^*,\mathcal{C}^*)\mathcal{C}^*\left(-1-\frac{\mathcal{C}}{\mathcal{C}^*}+\frac{\Phi(\mathcal{S},\mathcal{C}^*)}{\Phi(\mathcal{S},\mathcal{C})}+\frac{\Phi(\mathcal{S},\mathcal{C})\mathcal{C}}{\Phi(\mathcal{S},\mathcal{C}^*)\mathcal{C}^*}\right) \\ & +\Psi(\mathcal{S}^*,\mathcal{I}^*)\mathcal{I}^*\left(-1-\frac{\mathcal{I}}{\mathcal{I}^*}+\frac{\Phi(\mathcal{S},\mathcal{C}^*)\Psi(\mathcal{S}^*,\mathcal{I}^*)}{\Phi(\mathcal{S}^*,\mathcal{C}^*)\Psi(\mathcal{S},\mathcal{I})}+\frac{\Phi(\mathcal{S}^*,\mathcal{C}^*)\Psi(\mathcal{S},\mathcal{I})\mathcal{I}}{\Phi(\mathcal{S},\mathcal{C}^*)\Psi(\mathcal{S}^*,\mathcal{I}^*)\mathcal{I}^*}\right) \\ & +\Phi(\mathcal{S}^*,\mathcal{C}^*)\mathcal{C}^*\left(3-\frac{\Phi(\mathcal{S},\mathcal{C}^*)}{\Phi(\mathcal{S},\mathcal{C})}-\frac{\Phi(\mathcal{S}^*,\mathcal{C}^*)}{\Phi(\mathcal{S},\mathcal{C}^*)}-\frac{\Phi(\mathcal{S},\mathcal{C})}{\Phi(\mathcal{S}^*,\mathcal{C}^*)}\right) \\ & +\Psi(\mathcal{S}^*,\mathcal{I}^*)\mathcal{I}^*\left(4-\frac{\Phi(\mathcal{S}^*,\mathcal{C}^*)}{\Phi(\mathcal{S},\mathcal{C}^*)}-\frac{\Phi(\mathcal{S},\mathcal{C}^*)\Psi(\mathcal{S}^*,\mathcal{I}^*)}{\Phi(\mathcal{S}^*,\mathcal{C}^*)\Psi(\mathcal{S},\mathcal{I})}-\frac{\Psi(\mathcal{S},\mathcal{I})\mathcal{I}\mathcal{C}^*}{\Psi(\mathcal{S}^*,\mathcal{I}^*)\mathcal{I}^*\mathcal{C}}-\frac{\mathcal{C}\mathcal{I}^*}{\mathcal{C}^*\mathcal{I}}\right). \end{aligned}$$

From above inequality, we get that

$$3-\frac{\Phi(\mathcal{S},\mathcal{C}^*)}{\Phi(\mathcal{S},\mathcal{C})}-\frac{\Phi(\mathcal{S}^*,\mathcal{C}^*)}{\Phi(\mathcal{S},\mathcal{C}^*)}-\frac{\Phi(\mathcal{S},\mathcal{C})}{\Phi(\mathcal{S}^*,\mathcal{C}^*)}\leq 0,$$

and

$$4-\frac{\Phi(\mathcal{S}^*,\mathcal{C}^*)}{\Phi(\mathcal{S},\mathcal{C}^*)}-\frac{\Phi(\mathcal{S},\mathcal{C}^*)\Psi(\mathcal{S}^*,\mathcal{I}^*)}{\Phi(\mathcal{S}^*,\mathcal{C}^*)\Psi(\mathcal{S},\mathcal{I})}-\frac{\Psi(\mathcal{S},\mathcal{I})\mathcal{I}\mathcal{C}^*}{\Psi(\mathcal{S}^*,\mathcal{I}^*)\mathcal{I}^*\mathcal{C}}-\frac{\mathcal{C}\mathcal{I}^*}{\mathcal{C}^*\mathcal{I}}\leq 0.$$

The hypothesis (H_1) leads to

$$\left(1-\frac{\mathcal{S}}{\mathcal{S}^*}\right)\left(1-\frac{\Phi(\mathcal{S}^*,\mathcal{C}^*)}{\Phi(\mathcal{S},\mathcal{C}^*)}\right)\leq 0.$$

By (H_4), we get

$$-1-\frac{\mathcal{C}}{\mathcal{C}^*}+\frac{\Phi(\mathcal{S},\mathcal{C}^*)}{\Phi(\mathcal{S},\mathcal{C})}+\frac{\Phi(\mathcal{S},\mathcal{C})\mathcal{C}}{\Phi(\mathcal{S},\mathcal{C}^*)\mathcal{C}^*}=\left(1-\frac{\Phi(\mathcal{S},\mathcal{C})}{\Phi(\mathcal{S},\mathcal{C}^*)}\right)\left(\frac{\Phi(\mathcal{S},\mathcal{C}^*)}{\Phi(\mathcal{S},\mathcal{C})}-\frac{\mathcal{C}}{\mathcal{C}^*}\right)\leq 0$$

and

$$\begin{aligned} & -1-\frac{\mathcal{I}}{\mathcal{I}^*}+\frac{\Phi(\mathcal{S},\mathcal{C}^*)\Psi(\mathcal{S}^*,\mathcal{I}^*)}{\Phi(\mathcal{S}^*,\mathcal{C}^*)\Psi(\mathcal{S},\mathcal{I})}+\frac{\Phi(\mathcal{S}^*,\mathcal{C}^*)\Psi(\mathcal{S},\mathcal{I})\mathcal{I}}{\Phi(\mathcal{S},\mathcal{C}^*)\Psi(\mathcal{S}^*,\mathcal{I}^*)\mathcal{I}^*} \\ =\ & \left(1-\frac{\Phi(\mathcal{S}^*,\mathcal{C}^*)\Psi(\mathcal{S},\mathcal{I})}{\Phi(\mathcal{S},\mathcal{C}^*)\Psi(\mathcal{S}^*,\mathcal{I}^*)}\right)\left(\frac{\Phi(\mathcal{S},\mathcal{C}^*)\Psi(\mathcal{S}^*,\mathcal{I}^*)}{\Phi(\mathcal{S}^*,\mathcal{C}^*)\Psi(\mathcal{S},\mathcal{I})}-\frac{\mathcal{I}}{\mathcal{I}^*}\right)\leq 0. \end{aligned}$$

Therefore, $D^{\alpha}\mathcal{G}(t)\leq 0$ with equality holds if and only if $\mathcal{S}=\mathcal{S}^*$, $\mathcal{C}=\mathcal{C}^*$ and $\mathcal{I}=\mathcal{I}^*$. From LaSalle's invariance principle, we conclude that $\mathcal{E}^*$ is globally asymptotically stable. □

4. APPLICATIONS AND NUMERICAL SIMULATIONS

In this section, we first apply our above analytical results to the following fractional COVID-19 model

$$\begin{cases} D^{\alpha}\mathcal{S}(t) &= \mathcal{A}-\nu\mathcal{S}-\dfrac{\beta_1\mathcal{S}\mathcal{C}}{1+\epsilon_1\mathcal{C}}-\dfrac{\beta_2\mathcal{S}\mathcal{I}}{1+\epsilon_2\mathcal{I}}, \\ D^{\alpha}\mathcal{C}(t) &= \dfrac{\beta_1\mathcal{S}\mathcal{C}}{1+\epsilon_1\mathcal{C}}+\dfrac{\beta_2\mathcal{S}\mathcal{I}}{1+\epsilon_2\mathcal{I}}-d_1\mathcal{C}, \\ D^{\alpha}\mathcal{I}(t) &= \gamma\mathcal{C}-d_2\mathcal{I}. \end{cases} \tag{4.16}$$

where β_1 and β_2 are the infection rates caused by carrier and infected individuals, respectively. The nonnegative constants ϵ_1 and ϵ_2 are the saturation rates.

Model (4.16) is a special case of system (2.2) with $\Phi(\mathcal{S},\mathcal{C}) = \dfrac{\beta_1\mathcal{S}}{1+\epsilon_1\mathcal{C}}$ and $\Psi(\mathcal{S},\mathcal{I}) = \dfrac{\beta_2\mathcal{S}}{1+\epsilon_2\mathcal{I}}$. It easy that see the basic reproduction number $\mathcal{R}_0$ is given by

$$\mathcal{R}_0 = \frac{\mathcal{A}(d_2\beta_1 + \gamma\beta_2)}{\nu d_1 d_2}. \tag{4.17}$$

Evidently, the infected rates Φ and Ψ satisfy the conditions $(H_1) - (H_3)$. According to Theorems 3.1 and 3.2, we get the following result.

Corollary 4.1:

(i) *When $\mathcal{R}_0 \leq 1$, the disease-free equilibrium $\mathcal{E}_f$ of model (4.16) is globally asymptotically stable.*

(ii) *When $\mathcal{R}_0 > 1$, the equilibrium $\mathcal{E}_f$ gets unstable and the endemic equilibrium $\mathcal{E}^*$ of model (4.16) is globally asymptotically stable.*

The second application of our results concerns a fractional model proposed in 2020 by Das and Samanta [2] to study the transmission dynamics of COVID-19 in Japan. This recent fractional model is a particular case of system (4.16), it suffices to take $\epsilon_1 = \epsilon_2 = 0$ and $r_1 = r_2 = d = 0$. In [2], the authors just discussed the local stability of the equilibria and calculated epidemic peak in Japan scenario. They proved that the disease-free equilibrium is locally asymptotically stable if $\mathcal{R}_0 < 1$ and the endemic equilibrium is locally asymptotically stable under $\mathcal{R}_0 > 1$ and others three conditions (see, Theorem 2 [2]). By applying Corollary 4.1, we deduce that when $\mathcal{R}_0 \leq 1$, the disease-free equilibrium of model [2] is globally asymptotically stable. However, this equilibrium becomes unstable and the endemic equilibrium is globally asymptotically stable under only $\mathcal{R}_0 > 1$. This improve the asymptotic stability results presented recently in [2].

For numerical simulations, we take the some parameters of model (4.16) accordingly to the World Health Organization reports as well as adopted by many references as stated in Table 4.1 below.

Parameter	Definition	Value
A	Recruitment rate	50
β_1	Transmission contact rate between $\mathcal{S}$ and $\mathcal{C}$	1.2×10^{-5}
β_2	Transmission contact rate between $\mathcal{S}$ and $\mathcal{I}$	Varied
γ	Symptoms period	$1/7$ day^{-1}
ϵ_1	The measure of inhibition effect for carrier	0.04
ϵ_2	The measure of inhibition effect for infected	0.01
ν	Natural death rate	0.01 day^{-1}
r_1	Recovery rate from carrier	$1/21$ day^{-1}
r_2	Recovery rate from infected	$1/15$ day^{-1}
d	Death due to disease rate	0.1 day^{-1}

Table 4.1. Parameter values of model (4.16).

For $\beta_2 = 5.5 \times 10^{-5}$, we have $\mathcal{R}_0 = 0.9532 \leq 1$. Then model (4.16) has a disease-free equilibrium $\mathcal{E}_f(5000, 0, 0, 0)$. By Corollary 4.1 (i), we know that $\mathcal{E}_f$ is globally asymptotically stable. Figure 2 demonstrates this result.

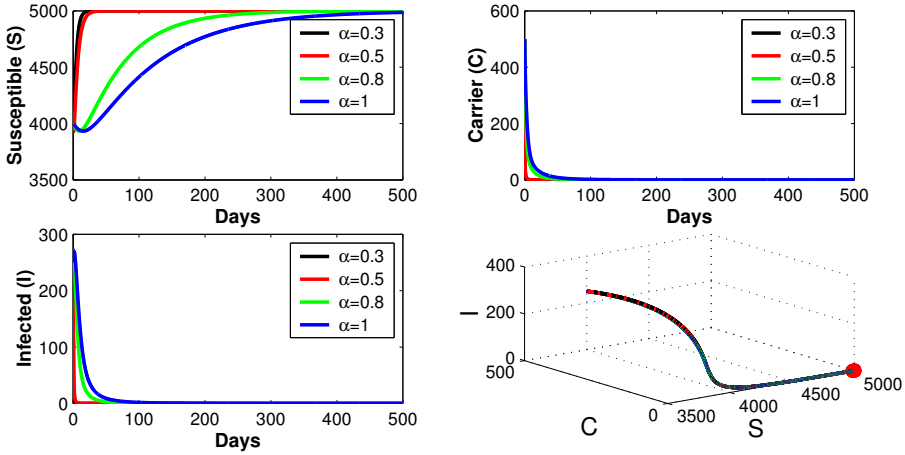

Fig. 4.2. Dynamics of the model (4.16) when $\mathcal{R}_0 = 0.9532 \leq 1$.

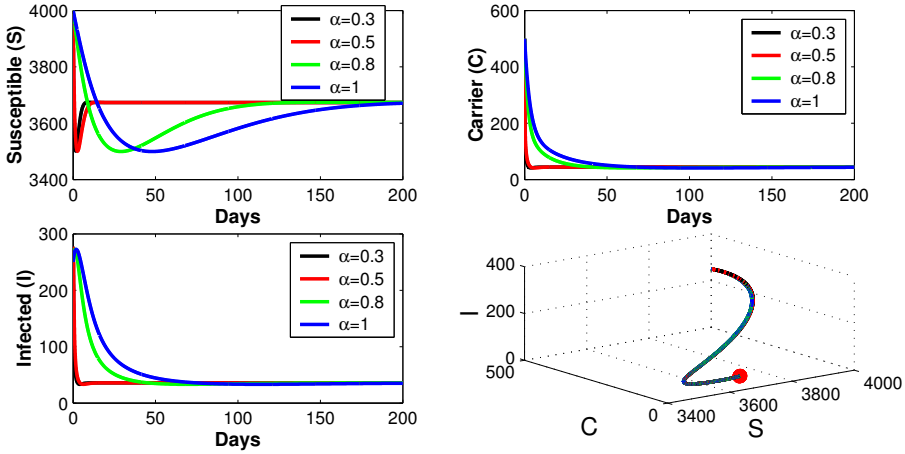

Fig. 4.3. Dynamics of the model (4.16) when $\mathcal{R}_0 = 1.9489 > 1$.

For $\beta_2 = 1.3 \times 10^{-4}$, we have $\mathcal{R}_0 = 1.9489 > 1$. Figure 3 shows that the trajectories of model (4.16) converge to $\mathcal{E}^*(3670.8515, 35.1197, 43.4314)$ from different values of order fractional derivative α. This confirms the global stability result given by Corollary 4.1 (ii).

5. CONCLUSION

In this work, we have presented a new fractional-order model of COVID-19 that takes into modes of infection spread of COVID-19 due to the direct contact between the susceptible individuals, with symptomatic and asymptomatic individuals. The incidence of infection is described by general nonlinear functions. It is proved that the proposed model has two steady states namely disease-free equilibrium and endemic equilibrium. The stability analysis of the model shows that the disease-free equilibrium is globally asymptotically stable if the basic reproduction number $\mathcal{R}_0 \leq 1$, which biologically means that the COVID-19 is cleared. While the endemic equilibrium is globally asymptotically stable when $\mathcal{R}_0 > 1$. In this case, the COVID-19 persists in the world and the infection

becomes similar to many endemic viral diseases to date, such as influenza, AIDS and viral hepatitis. On the other hand, we have established a new generalized lemma to analyze the global stability of the endemic equilibrium. This lemma presents a new property for Caputo fractional derivative when $\alpha \in (0, 1]$ and extends the results presented in [1, 17] for any monotone function. Also, the new generalized lemma can be an important tool to study the global dynamics of many fractional order systems modeling infectious diseases such as COVID-19. Furthermore, the asymptotic stability results of the recent model of COVID-19 in Japan [2] are improved.

Several countries around the world have started vaccination against COVID-19 in order to reduce the prevalence of this dangerous disease. We will extend our model to study the impact of vaccination on the propagation of COVID-19. Also, our model used a fractional derivative with singular kernel. It will be more interesting to extend the model by using a fractional derivative with non-singular kernel as in [6].

DATA AVAILABILITY

The data used to support the findings of this study are available from the corresponding author upon request. The articles used to support the findings of this study are included within the article and are cited at relevant places within the text as references.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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