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Fractional order epidemiological model of SARS-CoV-2 dynamism involving Alzheimer's disease

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ABSTRACT

In this paper, we study a Caputo–Fabrizio fractional order epidemiological model for the transmission dynamism of the severe acute respiratory syndrome coronavirus 2 pandemic and its relationship with Alzheimer's disease. Alzheimer's disease is incorporated into the model by evaluating its relevance to the quarantine strategy. We use functional techniques to demonstrate the proposed model stability under the Ulam–Hyres condition. The Adams–Bashforth method is used to determine the numerical solution for our proposed model. According to our numerical results, we notice that an increase in the quarantine parameter has minimal effect on the Alzheimer's disease compartment.

1. Introduction

No doubt, the merciless outbreaks of infectious disease pandemics have impacted humanity's history. Entire nations and civilizations have gone off the face of the earth throughout history. Beginning with biblical pharaonic pandemics that suddenly hit Ancient Egypt around 1715 BC, the mid - sixteenth century "cocoliztli" epidemics killed some 13 million people, completely destroying the Mesoamerican native population, as well as the Black Death pandemic that erupted in Europe in 1348 BC, killed over 25 million people in just five years [1]. The pandemic influenza virus of 1918–1919 spread across all part of the world, killing an estimated 40 million people worldwide. AIDS, measles, malaria, and tuberculosis have all been re-emerging epidemics in recent decades, killing millions of infected individuals each year. According to a report on the global AIDS epidemic released by the World Health Organization (WHO) and the United Nations AIDS Programme (UNAIDS), an estimated 37.7 million individuals, including 1.7 million children, were living with HIV at the end of 2020 [2]. SARS-COV-2 pandemic has stabilized world progress and put the globe into silent mode, in the twenty-first century, where rapid technological and theoretical advancements have substantially strengthened our armament in prevention and control of epidemics. The SARS-COV-2 virus causes Coronavirus (COVID-19), a recent infectious disease. The emergence of minor to severe respiratory difficulties is one of the most well-known symptoms of this condition, with the patient recovering without therapy in some circumstances. This virus causes symptoms such as fever, cough, loss of taste. The infected individuals sometimes show less common symptoms such as sore throat, headache, rash, among others. COVID-19's age-related transmissibility has become a public health concern in this context. Adults over the age of 60 have had the most serious health problems, with deadly effects for those over the age of 80. This is attributable to the high prevalence of underlying health issues in older people [3–5]. Coronavirus, like all viruses, evolves throughout time. The majority of the changes have no effect on the virus's properties. Some of these changes, however, may have an effect on the virus's features, such as how easily it spreads, the severity and scope of the sickness it causes, or the efficacy of vaccines, therapeutic drugs, and diagnostic tests. The authors in [6] considered COVID-19 model where a brief details of infection were discussed. They showed that the main source of infection was the seafood industry. In [7], they also analyzed the impact of non-pharmaceutical interventions on the dynamics COVID-19 involving optimal control strategy. The authors in [8] modeled a new COVID-19 model incorporated quarantine and isolation. They discussed relevant mathematical results.

Quarantine is one of the most effective strategies to limit the transmission route of this disease, because the symptoms of the sickness may not emerge on the infected individual at all times, allowing the virus to spread more swiftly. According to mental health experts, the global SARS-COV-2 virus pandemic quarantine implemented on millions of people is neither simple nor overstated since it is a one-of-a-kind and unprecedented action that restricts day-to-day movement of people. Many people suffer from psychological and neurological problems as a result of this predicament, especially those who do not deal with it

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positively. Recently, there has been a worldwide concerned of the rising tide of the memory difficulties among all population. According to previous studies, depression in the old age has been connected to cognitive impairment and the risk of Alzheimer's disease [9–12]. Alzheimer's disease is a brain disorder that causes memory and cognitive function to deteriorate over time. Alzheimer's disease is a neuro degenerative disorder having higher fatality in the elderly due to delayed treatment caused by a lower detection rate. The lockdown has affected people's memories not only the elderly population but all ages. Although there are presently no medical treatments for this disease, numerous medications promise to delay or drastically diminish the symptoms and harmful consequences it has on the patient.

Many medical research [13,14] have mentioned the impact of the SARS-CoV-2 pandemic on Alzheimer's disease and increased morbidity and death in infected patients with recent Neuroinflammation. SARS-CoV-2 has accelerated the progression of brain inflammatory neurodegeneration, and elderly people are more prone to poor consequences following SARS-CoV-2 infection due to the immune response and excessive inflammation. It has been observed that societies or communities found themselves in an isolated routine during the SARS-CoV-2 pandemic are already experiencing memory difficulties. Furthermore, some studies have found that communities that were isolated during the SARS-CoV-2 pandemic are at an increased risk for Alzheimer's Disease, and that this severity may create an exceptionally high-risk profile for certain demographics like Asian Americans, African Americans, and Hispanic Americans [15].

When dealing with an epidemic, it is important to anticipate future, and understand how to control the epidemic's progress. Hence, numerous academicians from several fields have contributed to the analysis, creation of models, and study of significant COVID-19 prevention strategies. In the past, mathematical models were employed to simulate SARS-CoV-2 epidemics (see for example [16–19]). Even while results from the use of equations of the integer-order have been somewhat successful, results from the use of equations of the fractional-order are still preferable in terms of how closely they relate to reality when they depict real phenomena. Fractional calculus has grown in popularity and importance as a result of its proved applications in system biology and several sectors of science [20]. In fractional calculus, all non-negative order derivatives and integrals are allowed. Fractional derivatives have the advantage of not being a local attribute [21]. Not surprisingly, fractional-order models are commonly used in epidemiology to understand the complexity of infectious diseases. In the realm of mathematical biology, the Caputo–Fabrizio (CF) fractional-order operator has been used over Atangana–Baleanu, beta derivatives, and a few more to design numerous epidemiological models such as dengue fever, smoking, tuberculosis, measles, Ebola, and other diseases, as shown in [22–30]. For instance, in [31], the authors discussed a novel model of human liver where CF fractional operator were used and concluded that when the operator reduce from 1, the rate of infection reduces. In [32], the authors also considered existence theory and numerical solutions to smoking model under CF fractional operator. Rajagopal and his co-authors proposed a fractional-order SEIRD model for the spread of COVID-19 and compared it with the real data and integer-order cases [33]. Ahmed and his co-authors proposed a five-term dynamical system to understand the trade-off between the lockdown and the spread of the virus [34].

The authors of [35] investigated Hepatitis E disease model under CF derivative. They concluded that CF operator is a powerful mathematical tool to study biological models more comprehensively and also a fastest convergent tool. In [36] the authors investigated epidemiological model for epidemic childhood diseases. In their paper, they gave technical comparison of the Caputo and CF fractional derivative results. The authors in [37] used fractional operators to study dynamics of Chagas-HIV epidemic model. In [38], mathematical model for pneumococcal pneumonia infection have been investigated via CF fractional operator. In [39], the author studied fractional order alcoholism model via CF operator. Recently, this SARS-CoV-2 pandemic has received many mathematical models using CF fractional derivative. Not long ago, the authors in [40] proposed mathematical model for SARS-CoV-2 pandemic via Caputo, CF and ABC fractional derivatives and studied the dynamics of the pandemic in the Pakistan with actual data. In [41], using CF fractional operator, mathematical model of SARS-CoV-2 pandemic in India was proposed. They discussed interesting interventions for the spread of SARS-CoV-2 pandemic. The authors in [42] applied variable CF fractional order to investigate COVID-19 model and discussed some interested result for the proposed model. More articles have outlined the significant impact of CF fractional differential and integral operator in studying the complex dynamics of non-infectious and infectious disease (see for example [43–47]).

The aforementioned literature on the importance of CF fractional derivative on dynamical systems, we formulate a novel model for SARS-CoV-2 pandemic incorporating Alzheimer's disease. The memory and heredity features are aim of dealing with fractional-order systems in our model, which gives us a more realistic method to biological systems, that help in deal with complicated behavioral patterns of biological systems. The memory function allows fractional order models to incorporate more knowledge from the past, allowing for more accurate prediction and translation. The major goal of this research is to determine the scope of the SARS-CoV-2 epidemic, to forecast what might happen in the future and how to prevent the disease from spreading, and to determine the influence of COVID-19 quarantine on Alzheimer's disease. Motivated greatly by these and considering the aforementioned papers, there is no single mathematical formulation for SARS-CoV-2 pandemic incorporating Alzheimer's disease, and this research reveal some critical qualitative information about the disease's course.

The structure for this paper is as follows: In Section 2, the definitions and some significant properties of the CF fractional operator are given. In Section 3, we described the model in classical and CF fractional order perspective. In Section 4, we determined the classical analysis of our proposed model, thus, positivity, boundedness and invariant region. In Section 5, we discussed the existence and uniqueness results using Banach and Krasnoselskii's type fixed point theorem. In Section 6, we examined the proposed model stability under HU stability type. In Section 7, by employing Lagrange interpolation, we analyze the numerical solutions of the proposed model. In Section 8, we used the numerical results in Section 6 and run the numerical simulation. Finally, Section 9 sum-up our results and suggestions are given for future studies.

2. Preliminaries

In this section, we recall some critical concepts, lemmas, and definitions to study our proposed model.

Definition 2.1 ([32,40]). Let $Y \in H^1(a,b), b > a$, and $\mu_* \in (0,1)$. Then the CF derivative can be defined as

$$ {}^{CF}_{a}D^{\mu_*}_{\sigma} Y(\sigma) = \frac{G(\mu_*)}{1-\mu_*} \int_a^{\sigma} Y'(x) exp\left[-\mu_* \frac{\sigma - s}{1-\mu_*}\right] ds.$$

Here, $G(\mu_*)$ is a normalization function, where $G(0) = G(1) = 1$. The fractional integral of CF is defined by:

$$ I^{\mu_*}_{\sigma} Y(\mu_*) = \frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)} Y(\sigma) + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)} \int_0^{\sigma} Y(s) ds, \sigma \geq 0.$$

Lemma 2.2 ([41,42,48]). *Suppose that $y(\sigma) \in L_p[0,\eta]$, then the solution of fractional differential equation*

$$\begin{cases} {}^{CF}D^{\mu_*}_{\sigma} Y(\sigma) = y(\sigma), \sigma \in [0,\eta], \\ Y(0) = Y_0, \end{cases}$$

is given by

$$ Y(\sigma) = Y_0 + \frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)} Y(\sigma) + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)} \int_0^{\sigma} Y(s) ds, \sigma \geq 0.$$

Table 1
Interpretation of parameters in the model.

Parameter	Interpretation
Λ	Human recruitment rate
α	Rate of restriction Λ
μ_1	Natural death rate
μ_2	Covid-19 induce death rate
μ_3	The death rate from Alzheimer's disease
η	Modification parameter for the increase of infectivity of S_2
γ_1	The rate of been quarantined.
γ_2	The probability of developing Alzheimer's disease of susceptible individuals
ϕ	The rate of movement from S_1 to S_2
δ_1	The rate of effective screening for the individuals infected without symptoms of SARS-CoV-2
δ_2	The rate of effective screening for the individuals infected with SARS-CoV-2
ψ	Progression rate of the Covid-19
ε	Proportion of newly infected humans moving to I_S
σ_1	The rate recovery from I_A
σ_2	The rate recovery from I_S
θ_1	The rate of quarantine I_A
θ_2	The rate of quarantine I_S
ξ_1	The rate of infected with Alzheimer's disease in I_A class
ξ_2	The rate of infected with Alzheimer's disease in I_S class
ν	The rate of developing Alzheimer's disease after recovery
m	The probability of developing Alzheimer's disease due to quarantine
τ	Relative infectiousness factor for asymptomatic humans

Lemma 2.3 ([48]). *From Krasnoselskii's fixed point theorem if we assume that $\boldsymbol{B}^* \subset \boldsymbol{M}^*$, be a closed convex non-empty subset of $\boldsymbol{B}^*$ and $\exists$ two operator Ω_1^* and Ω_2^* then we will have the following*

(i) $\Omega_1^*\Theta + \Omega_2^* Y \in \boldsymbol{B}^*,\ \forall Y \in \boldsymbol{B}^*$;

(ii) Ω_1^* *is contraction and* Ω_2^* *continuous and compact. Then there exist at least one solution* $Y \in \boldsymbol{B}^*$ *such that*

$$\Omega_1^* Y + \Omega_2^* Y = Y.$$

3. Model formulation

We consider the entire human population N at time t thus, $N(t)$, where we subdivided into mutually exclusive compartments, which are susceptible humans with moderate risk of Covid-19 and Alzheimer's disease infection $S_1(t)$, thus young population (<60 years old), susceptible humans with high risk of Covid-19 and Alzheimer's disease infection $S_2(t)$, thus aged population (≥60 years old), exposed population $E(t)$, asymptomatically infected population $I_A(t)$, symptomatically infected population $I_S(t)$, individuals in isolation centers $Q(t)$, recovered population $R(t)$ and individuals who have had a Alzheimer's disease $A_D(t)$. Thus, $N(t)$, is given by $N(t) = S_1(t) + S_2(t) + E(t) + I_D(t) + I_S(t) + Q(t) + R(t) + A_D(t)$. Table 1 gives detail interpretation of parameters in the model, while we assumed the state variables and parameters to be all positive. Hence, our proposed model is described by the following system of differential equations;

$$\begin{cases} \frac{dS_1(t)}{dt} = \alpha\Lambda - \left(\mu_1 + \gamma_1 + \gamma_2\right)S_1 - \lambda S_1 - \phi S_1, \\ \frac{dS_2(t)}{dt} = (1-\alpha)\Lambda + \phi S_1 - \eta\lambda S_2 - \left(\mu_1 + \gamma_1 + \gamma_2\right)S_2, \\ \frac{dE(t)}{dt} = \lambda(S_1 + \eta S_2) - (\psi + \mu_1 + \delta_1 + \delta_2)E, \\ \frac{dI_A(t)}{dt} = \varepsilon\psi E + \delta_1 E - (\mu_1 + \sigma_1 + \theta_1 + \xi_1)I_A, \\ \frac{dI_S(t)}{dt} = (1-\varepsilon)\psi E + \delta_2 E - (\mu_1 + \mu_2 + \sigma_2 + \theta_2 + \xi_2)I_S, \\ \frac{dQ(t)}{dt} = \gamma_1(S_1 + S_2) + \theta_1 I_A + \theta_2 I_S - (m + \mu_1 + \mu_2)Q, \\ \frac{dR(t)}{dt} = \sigma_1 I_A + \sigma_2 I_S - (\nu + \mu_1)R, \\ \frac{dA_D(t)}{dt} = \gamma_2(S_1 + S_2) + \nu R + \xi_1 I_A + \xi_2 I_S + mQ - (\mu_1 + \mu_3)A_D, \end{cases} \tag{1}$$

where $t > 0$ with the initial conditions $S_1(0) = S_{1(0)} \geq 0$, $S_2(0) = S_{2(0)} \geq 0$, $E(0) = E_{(0)} \geq 0$, $I_A(0) = I_{A(0)} \geq 0$, $I_S(0) = I_{S(0)} \geq 0$, $Q(0) = Q_{(0)} \geq 0$, $R(0) = R_{(0)} \geq 0$, $A_D(0) = A_{D(0)} \geq 0$. Where the force of infection of the model (1) above is given by $\lambda = \frac{\tau I_A + I_S}{N}$. The aforementioned differential operators cannot describe the nonlocal dynamics, due to the involvement of singular kernel. To overcome these complications a new class of fractional operator has been introduced in [49]. Therefore, for better understanding of the SARS-CoV-2 model, it is required to reformulate the SARS-CoV-2 model above to the fractional-order model. According to the explanation of time-dependent kernel defined by the power law correlation function, presented in [50], our considered Caputo–Fabrizio fractional order derivative model for SARS-CoV-2 incorporate Alzheimer's disease is defined as follows;

$$\begin{cases} {}^{CF}D_t^{\mu_*} S_1(t) = \alpha\Lambda - \left(\mu_1 + \gamma_1 + \gamma_2\right)S_1 - \lambda S_1 - \phi S_1, \\ {}^{CF}D_t^{\mu_*} S_2(t) = (1-\alpha)\Lambda + \phi S_1 - \eta\lambda S_2 - \left(\mu_1 + \gamma_1 + \gamma_2\right)S_2, \\ {}^{CF}D_t^{\mu_*} E(t) = \lambda(S_1 + \eta S_2) - (\psi + \mu_1 + \delta_1 + \delta_2)E, \\ {}^{CF}D_t^{\mu_*} I_A(t) = \varepsilon\psi E + \delta_1 E - (\mu_1 + \sigma_1 + \theta_1 + \xi_1)I_A, \\ {}^{CF}D_t^{\mu_*} I_S(t) = (1-\varepsilon)\psi E + \delta_2 E - (\mu_1 + \mu_2 + \sigma_2 + \theta_2 + \xi_2)I_S, \\ {}^{CF}D_t^{\mu_*} Q(t) = \gamma_1(S_1 + S_2) + \theta_1 I_A + \theta_2 I_S - (m + \mu_1 + \mu_2)Q, \\ {}^{CF}D_t^{\mu_*} R(t) = \sigma_1 I_A + \sigma_2 I_S - (\nu + \mu_1)R, \\ {}^{CF}D_t^{\mu_*} A_D(t) = \gamma_2(S_1 + S_2) + \nu R + \xi_1 I_A + \xi_2 I_S + mQ - (\mu_1 + \mu_3)A_D, \end{cases} \tag{2}$$

where $t > 0$ with the initial conditions $S_1(0) = S_{1(0)}$, $S_2(0) = S_{2(0)}$, $E(0) = E_{(0)}$, $I_A(0) = I_{A(0)}$, $I_S(0) = I_{S(0)}$, $Q(0) = Q_{(0)}$, $R(0) = R_{(0)}$, $A_D(0) = A_{D(0)}$ subject to $\min(S_1, S_2, E, I_A, I_S, Q, R, A_D) \geq 0$, and we assume that dimension of both model sides are the same.

4. Basic dynamics of the model

We examine the dynamics of the positivity, boundedness, and invariance of the proposed model's solutions in this section. In an epidemics model, it is important to evaluate population survival and the expansion that is naturally constrained by scarce resources. As a result, we demonstrate the following theorems.

Theorem 1. *Let* $S_1(0) \geq 0$, $S_2(0) \geq 0$, $E(0) \geq 0$, $I_A(0) \geq 0$, $I_S(0) \geq 0$, $Q(0) \geq 0$, $R(0) \geq 0$, $A_D(0) \geq 0$, *such that the solution set*

$$\Psi = \{S_1(t),\ S_2(t),\ E(t),\ I_A(t),\ I_S(t),\ Q(t),\ R(t),\ A_D(t)\} \in R_+^8,$$

of the proposed model (1) are positive for all $t > 0$. *Furthermore*

$$\limsup_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu}.$$

Proof. Let $t_1 = \sup\{S_1(0) \geq 0,\ S_2(0) \geq 0,\ E(0) \geq 0,\ I_A(0) \geq 0,\ I_S(0) \geq 0,\ Q(0) \geq 0,\ R(0) \geq 0,\ A_D(0) \geq 0\} \in [0, t]$. Let us now consider the model's initial dynamical Eq. (2) where $t_1 > 0$. Keep in mind that the starting values are higher than zero, then,

$$\frac{dS_1}{dt} = \alpha\Lambda - (\mu_1 + \gamma_1 + \gamma_2)S_1 - \lambda S_1 - \phi S_1.$$

For simplicity, we let $M_1^* = [\mu_1 + \gamma_1 + \gamma_2 + \lambda + \phi]$. Thus,

$$\frac{dP_1}{dt} + M_1^* S_1(t) = \alpha\Lambda.$$

This lead to

$$\frac{d}{dt}\big(S_1(t)exp\big(\int_0^{t_1} M_1^*(s)ds\big)\big) = (\alpha\Lambda)exp\big(\int_0^{t_1} M_1^*(s)ds\big).$$

So, we get

$$S_1(t) = S_1(0)exp\big(-\int_0^{t_1} M_1^*(s)ds\big)\Big[\int_0^{t_1}(\alpha\Lambda)exp\big(\int_0^{t_1} M_1^*(s)ds\big)\Big] + exp\big(-\int_0^{t_1} M_1^*(s)ds\big)\Big[\int_0^{t_1}(\alpha\Lambda)exp\big(\int_0^{s} M_1^*(s)ds\big)\Big].$$

Therefore, we proved that $S_1(0) > 0$ for all $t > 0$. Similarly, we can prove that $S_2(0) \geq 0,\ E(0) \geq 0,\ I_A(0) \geq 0,\ I_S(0) \geq 0,\ Q(0) \geq 0,\ R(0) \geq 0,\ A_D(0) \geq 0$. for all $t > 0$.

Now, we get the following expression for boundedness $\frac{dN}{dt} = \Lambda - \mu N$, such that $\Lambda - \mu N \leq \frac{dN}{dt} \leq \Lambda - \mu N$, and hence, $\liminf_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu} \leq \limsup_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu}$.

Considering invariant, we let $\Phi = \Omega \subset R_+ \times R_+$ where

$$\Psi = \{(S_1(t),\ S_2(t),\ E(t),\ I_A(t),\ I_S(t),\ Q(t),\ R(t),\ A_D(t)) \in R_+^8 : N(t) \leq \frac{\Lambda}{\mu}\}.$$

Now, for positively invariant of R_+^8,

$$\frac{dN}{dt} = \Lambda - \mu N,$$

solving this equality we get as follows

$$N(t) \leq N(0)e^{-\mu(t)} + \frac{\Lambda}{\mu}\big(1 - e^{-\mu}(t)\big).$$

Therefore,

$$\limsup_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu}.$$

As a result, the SARS-CoV-2 transmission model that includes Alzheimer's disease is known to be mathematically well-posed and fall within the realm of possibility Φ.

Theorem 2. *The solution of (2) along with initial conditions is positively invariant and bounded in* R_+^7.

Proof. Using the results in [51] and taking the account of the initial values given, from model (2) we obtain

$$\begin{cases} {}^{CF}D_t^{\mu_*} S_1(t)|_{S_1(0)} = a\Lambda \geq 0, \\ {}^{CF}D_t^{\mu_*} S_2(t)|_{S_2(0)} = (1-a)\Lambda + \phi S_1 \geq 0, \\ {}^{CF}D_t^{\mu_*} E(t)|_{E(0)} = \lambda(S_1 + \eta S_2) \geq 0, \\ {}^{CF}D_t^{\mu_*} I_A(t)|_{I_A(0)} = \varepsilon\psi E + \delta_1 E \geq 0, \\ {}^{CF}D_t^{\mu_*} I_S(t)|_{I_S(0)} = (1-\varepsilon)\psi E + \delta_2 E \geq 0, \\ {}^{CF}D_t^{\mu_*} Q(t)|_{Q(0)} = \gamma_1(S_1 + S_2) + \theta_1 I_A + \theta_2 I_S \geq 0, \\ {}^{CF}D_t^{\mu_*} R(t)|_{R(0)} = \sigma_1 I_A + \sigma_2 I_S \geq 0, \\ {}^{CF}D_t^{\mu_*} I_S(t)|_{I_S(0)} = \gamma_2(S_1 + S_2) + \nu R + \xi_1 I_A + \xi_2 I_S + mQ \geq 0. \end{cases} \tag{3}$$

Suppose $S_{1(0)}, S_{2(0)}, E_{(0)}, I_{A(0)}, I_{S(0)}, Q_{(0)}, R_0, A_{D(0)} \in R_+^8$, for all $t > 0$, then from (3) the solution of the model 2 cannot escape from the hyperplanes $S_1 = 0,\ S_2 = 0, E = 0,\ I_A = 0,\ I_S = 0,\ Q = 0,\ R = 0,\ A_D = 0$. This conclude that the $\in R_+^8$, is a positive invariant set.

5. Existence and uniqueness results for the SARS-CoV-2 transmission model incorporate Alzheimer's disease

It is important to ask weather the dynamical problem we proposed exist or not. This is the basic question and will answered by the theory of fixed points. Regarding to the aforesaid need as the integral is differentiable, we can rewrite the right sides of model (2) as follows

$$\begin{cases} \aleph_1(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)) \\ = \alpha\Lambda - (\mu_1 + \gamma_1 + \gamma_2)S_1 - \lambda S_1 - \phi S_1, \\ \aleph_2(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)) \\ = (1-\alpha)\Lambda + \phi S_1 - \eta\lambda S_2 - (\mu_1 + \gamma_1 + \gamma_2)S_2, \\ \aleph_3(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)) \\ = \lambda(S_1 + \eta S_2) - (\psi + \mu_1 + \delta_1 + \delta_2)E \\ \aleph_4(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)) \\ = \varepsilon\psi E + \delta_1 E - (\mu_1 + \sigma_1 + \theta_1 + \xi_1)I_A, \\ \aleph_5(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)) \\ = (1-\varepsilon)\psi E + \delta_2 E - (\mu_1 + \mu_2 + \sigma_2 + \theta_2 + \xi_2)I_S, \\ \aleph_6(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)) \\ = \gamma_1(S_1 + S_2) + \theta_1 I_A + \theta_2 I_S - (m + \mu_1 + \mu_2)Q, \\ \aleph_7(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)) \\ = \sigma_1 I_A + \sigma_2 I_S - (\nu + \mu_1)R, \\ \aleph_8(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)) \\ = \gamma_2(S_1 + S_2) + \nu R + \xi_1 I_A + mQ \\ + \xi_2 I_S - (\mu_1 + \mu_3)A_D. \end{cases} \tag{4}$$

From (4), the developed model (2) can be written in the form;

$$\begin{cases} {}^{CF}D_t^{\mu_*}\aleph(t) = \Phi(t, \aleph(t)),\ t \in [0, \eta],\ 0 < \mu_* \leq 1, \\ \aleph(0) = \aleph_0, \end{cases} \tag{5}$$

$$\aleph(t) = \begin{cases} S_1(t), \\ S_2(t), \\ E(t), \\ I_A(t)(t), \\ I_D(t), \\ Q(t), \\ R(t), \\ A_D(t), \end{cases} \quad \aleph_0 = \begin{cases} S_1(0), \\ S_2(0), \\ E(0), \\ I_A(0)(t), \\ I_D(0), \\ Q(0), \\ R(0), \\ A_D(0), \end{cases} \tag{6}$$

therefore,

$$\Phi(t, \aleph(t)) = \begin{cases} \aleph_1(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)), \\ \aleph_2(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)), \\ \aleph_3(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)), \\ \aleph_4(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)), \\ \aleph_5(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)), \\ \aleph_6(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)), \\ \aleph_7(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)), \\ \aleph_8(t, S_1(t), S_2(t), E(t), I_A(t), I_S(t), Q(t), R(t), A_D(t)). \end{cases} \tag{7}$$

With the help of Lemma 6.4, system (5) yields,

$$\begin{cases} \aleph(t) = \aleph_0(t) + \frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)}\Phi(t, \aleph(t)) + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)} \\ \quad \times \int_0^t \Phi(s, \aleph(s))ds. \end{cases} \tag{8}$$

Further, let say $\boldsymbol{B}_* = C([0,\eta])$ is the Banach space, supposing that the following assumptions hold

(H_1) There exist a nonnegative constant Q_*, W_*, and $k_* \in [0,1)$ such that

$$\Phi(t,\aleph(t)) \le Y_*|\aleph|^{k_*} + Z_*.$$

(H_2) There exist a nonnegative constant $\mathbf{C}_\rho > 0$ for all $\aleph, \tilde{\aleph} \in \boldsymbol{B}_*$ then

$$|\Phi(t,\aleph(t)) - \Phi(t,\tilde{\aleph}(t))| \le \mathbf{C}_\rho[|\aleph - \tilde{\aleph}|].$$

Also, let us define operator $\mathbf{T}_p : \boldsymbol{B}_* \to \boldsymbol{B}_*$ such that

$$\mathbf{T}_p\aleph(t) = M_1^*\aleph(t) + M_2^*\aleph(t),$$

basically, we can see that

$$\begin{cases} M_1^*\aleph(t) = \aleph_0(t) + + \dfrac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)}\Phi(t,\aleph(t)), \\ M_2^*\aleph(t) = \dfrac{2\mu_*}{(2-\mu_*)G(\mu_*)}\displaystyle\int_0^t \Phi(s,\aleph(s))ds. \end{cases} \tag{9}$$

From this knowledge (8) can be written as

$$\begin{cases} \mathbf{T}_p\aleph(t) = \aleph_0(t) + \dfrac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)}\Phi(t,\aleph(t)) + \dfrac{2\mu_*}{(2-\mu_*)G(\mu_*)} \\ \qquad \times \displaystyle\int_0^t \Phi(s,\aleph(s))ds. \end{cases} \tag{10}$$

Theorem 3. *Suppose that (H_1) and (H_2) hold, such that, $\dfrac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)}\mathbf{C}_\rho < 1$, then the SARS-CoV-2 transmission model incorporate Alzheimer's disease has at least one solution.*

Proof. For simplicity, we divide the proof into two steps.

Step 1. We proof that operator M_1^* is contraction. Then, let $\tilde{\aleph} \in \Pi$, where $\Pi = \{\aleph \in \mathbf{W}_* : \ \|\aleph\| \le \phi, \ \phi > 0\}$ is a close convex set, thus

$$\begin{aligned} |M_1^*\aleph(t) - M_2^*\aleph(t)| &= \frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)} \\ &\quad \times \max_{\mu_*\in[0,\eta]} |\Phi(t,\aleph(t)) - \Phi(\sigma,\tilde{\aleph}(t))|, \\ &\le \frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)}\mathbf{C}_\rho\|\aleph - \tilde{\aleph}\|. \end{aligned} \tag{11}$$

Thus,

$$\|M_1^*\aleph(t) - M\Omega_2^*\aleph(t)\| \le \frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)}\mathbf{C}_\rho\|\aleph - \tilde{\aleph}\|.$$

Hence M_1^* is contraction since $\dfrac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)}\mathbf{C}_\rho < 1$.

Step 2. We proof that M_2^* is compact and also continuous, for all $\aleph \in \Pi$, then M_2^* will be continuous as $\aleph$ is continuous, thus

$$\begin{aligned} \|M_2^*(\aleph)\| &= \max_{t\in[0,\eta]} |\frac{2\mu_*}{(2-\mu_*)G(\mu_*)}\int_0^t \Phi(s,\aleph(s))ds|, \\ &\le \frac{2\mu_*}{(2-\mu_*)G(\mu_*)}\eta\int_0^t |\Phi(s,\aleph(s))|ds. \\ &\le \frac{2\mu_*}{(2-\mu_*)G(\mu_*)}\eta[Y_*|\aleph|^{k_*} + Z_*]. \end{aligned} \tag{12}$$

Hence M_2^* is boundedness. For equicontinuous, let $t_1, t_2 \in [0,\eta]$, such that

$$\begin{aligned} |(M_2^*\aleph)(t_1) - (M_2^*\aleph)(t_2)| &= \frac{2\mu_*}{(2-\mu_*)G(\mu_*)} \max_{t\in[0,\eta]} \Big| \int_0^{t_1} \Phi(s,\aleph(s))ds \\ &\quad - \int_0^{t_2} \Phi(s,\aleph(s))ds\Big| \\ &\le \frac{2\mu_*}{(2-\mu_*)G(\mu_*)}[Y_*|\aleph|^{k_*} + Z_*]|t_1 - t_2|. \end{aligned} \tag{13}$$

As $t_1 \to t_2$, then $|(M_2^*\aleph)(t_1) - (M_2^*\aleph)(t_2)| \to 0$ which make operator M_2^* an equicontinuous and compact by Arzela–Ascoli theorem. Therefore by Lemma 2.3 the existence for the SARS-CoV-2 transmission model incorporate Alzheimer's disease has at least one solution. □

Theorem 4. *Suppose that $\exists$ a nonnegative integer $\Lambda_\rho > 0$ such that*

$$\Lambda_\rho = \left[\frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)}\mathbf{L}_\rho + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)}\eta\mathbf{L}_\rho\right] < 1, \tag{14}$$

then operator $\mathbf{T}_p$ has a unique fixed point.

Proof. Let $\aleph, \tilde{\aleph} \in \boldsymbol{W}_*$, then we say

$$\begin{aligned} \|\mathbf{T}_p\aleph - \mathbf{T}_p\tilde{\aleph}\| &\le \|M_1^*\aleph - M_1^*\tilde{\aleph}\| + \|M_2^*\aleph - \Omega_2^*\tilde{\aleph}\|, \\ &\le \frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)} \max_{t\in[0,\eta]} \Big|\Phi(t,\aleph(t)) - \Phi(t,\tilde{\aleph}(t))| \\ &\quad + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)} \\ &\quad \times \max_{t\in[0,\eta]} \Big| \int_0^t \Phi(s,\aleph(s))ds - \int_0^t \Phi(s,\tilde{\aleph}(s))ds\Big| \\ &\le \big[\frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)}\mathbf{C}_\rho + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)}\eta\mathbf{C}_\rho\big]\|\aleph - \tilde{\aleph}\|, \\ &= \Lambda_\rho\|\aleph - \tilde{\aleph}\|. \end{aligned} \tag{15}$$

Hence, by Banach contraction principle, $\mathbf{T}_p$ has a unique fixed point. Consequently, the SARS-CoV-2 transmission model incorporate Alzheimer's disease has unique solution. □

6. Hyers–Ulam (HU) stability results for the SARS-CoV-2 transmission model incorporate Alzheimer's disease

Stability is one of crucial component of differential equation. There has been many stability-type concepts that study dynamical systems, the HU stability-type concept has recently used for many epidemiological models, due to the approximation properties in the solutions which reduce the burden of getting exact solutions, for example (see [52]) and references therein. To assess and analyze the SARS-CoV-2 transmission model incorporating Alzheimer's disease, we apply the concept of HU stability-type to get approximate solution for our proposed model.

Definition 6.1. The proposed model is HU stable if for $\delta > 0$ and letting $\aleph \in \boldsymbol{W}_*$ be any solution of below inequality

$${}^{CF}D_t^{\mu_*}\aleph(t) - \Phi(t,\aleph(t)) \le \delta, \ \forall t \in [0,\eta]; \tag{16}$$

and with a unique solution $\tilde{\aleph}$ of problem (16) with a positive constant $C_q > 0$, such that,

$$\|\aleph - \tilde{\aleph}\| \le C_q\delta, \ \forall t \in [0,\eta]. \tag{17}$$

Definition 6.2. Given a function $\phi \in C(R,R)$, such that $\phi(0) = 0$ for any solution $\aleph$ of (16) and $\tilde{\aleph}$ be unique solution of (16), then

$$\|\aleph - \tilde{\aleph}\| \le \phi(\delta), \tag{18}$$

then the system (16) is generalized HU stable.

Remark 6.3. Suppose $\chi(t) \in C([0,\eta],R)$, we say $\aleph \in \boldsymbol{W}_*$ satisfies inequality (18) suppose that,

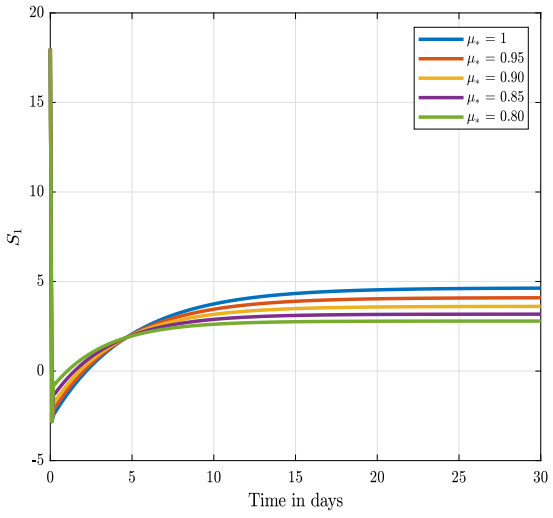
(i) $|\chi(t)| \le \delta$, for all $t \in [0,\eta]$,

(ii) ${}^{CF}D_t^{\mu_*}\aleph(t) = \Phi(t,\aleph(t)) + \chi(t)$, $\forall t \in [0,\eta]$.

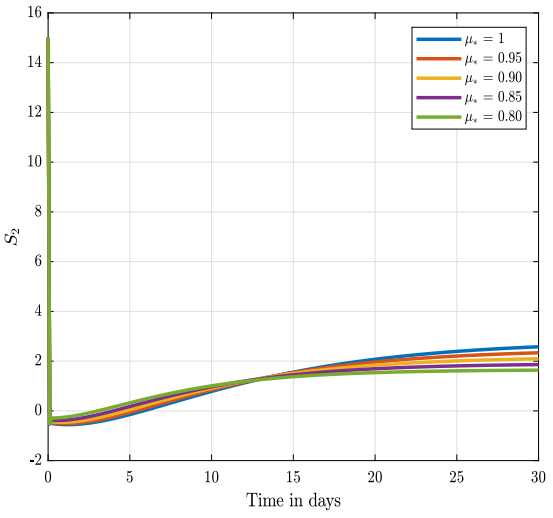
Now, we consider the resulting perturbation equation of system (16) as follows;

$$\begin{cases} {}^{CF}D_t^{\mu_*}\aleph(t) = \Phi(t,\aleph(t)) + \chi(t), \\ \aleph(0) = \aleph_0. \end{cases} \tag{19}$$

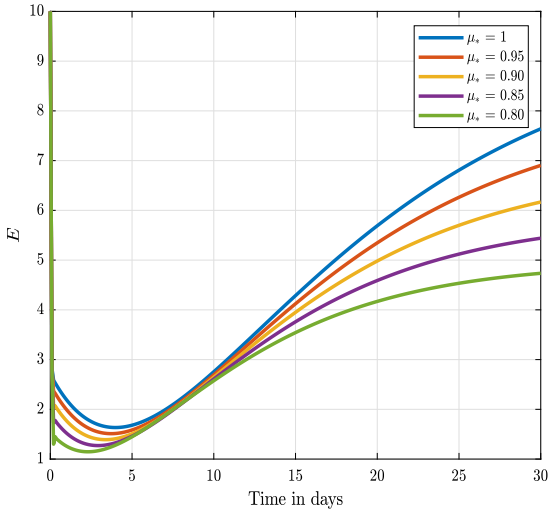
The below Lemma is necessary for our proves.



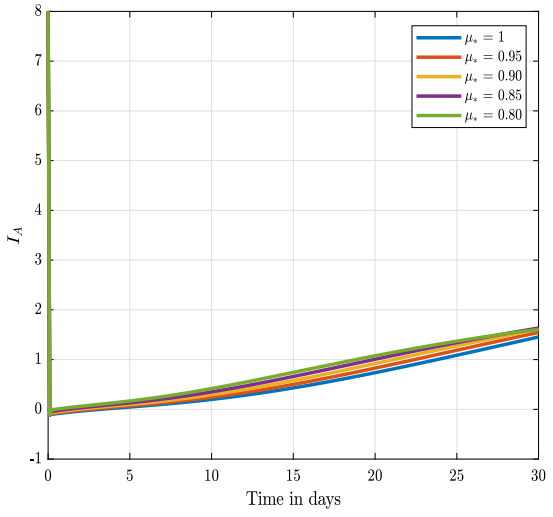
(a) Dynamics of Susceptible (S_1) Class



(b) Dynamics of Susceptible (S_2) Class



(c) Dynamics of Exposed (E) Class



(d) Dynamics of Asymptomatic Infected (I_A) class

Fig. 1. Numerical trajectory of the CF-fractional order derivative, μ_* of the model (2).

Table 2
Parameter values.

Parameter	Value	Parameter	Value
Λ	0.99	α	0.98
μ_1	0.000001	μ_2	0.00019
μ_3	0.0039	η	0.82
γ_1	0.008	γ_2	0.010
δ_1	0.0002	δ_2	0.01
θ_1	0.0056	θ_2	0.0076
ξ_1	0.01	ξ_2	0.053
ν	0.00002	τ	0.0003
m	0.009	ψ	0.08
ε	0.2	ϕ	0.07
σ_1	0.019	σ_2	0.0231

Lemma 6.4. *From Eq. (30), we say the following result hold. Thus,*

$$|\aleph(t) - \mathbf{T}_p \Phi(t, \aleph(t))| \leq \Big[\frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)} + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)}\eta\Big]\delta.$$

Proof. Consider Lemma 6.4, relatively, solution for Eq. (19) is given as;

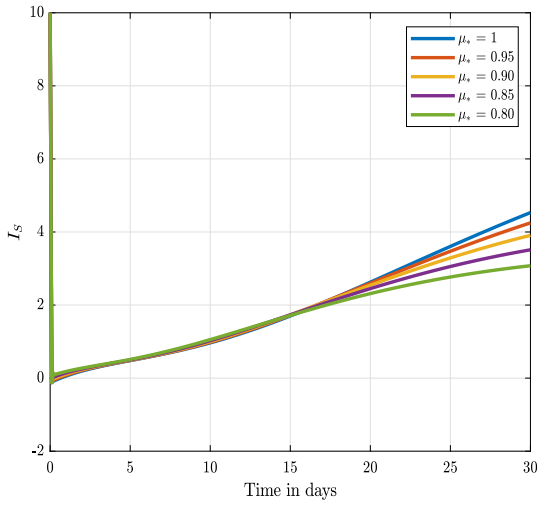
$$\aleph(t) = \aleph_0 + {}^{CF}I_t^{\mu_*}\Phi(t, \aleph(t)) + {}^{CF}I_t^{\mu_*}\chi(t).$$

Now, with the help of (10), we deduce that

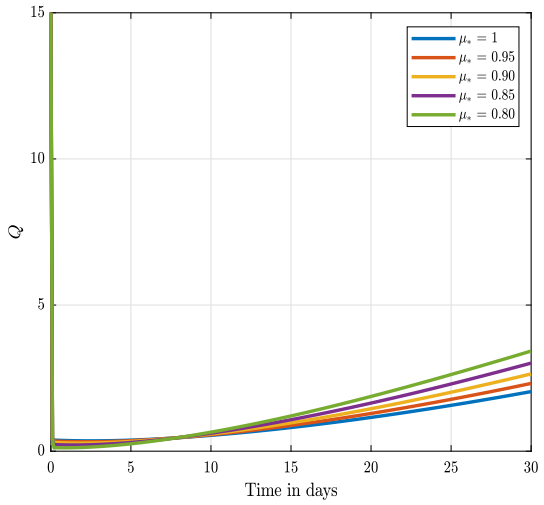
$$\begin{aligned}|\aleph(\sigma) - \mathbf{T}_p\Phi(t, \aleph(t))| &\leq \Big[\frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)}|\chi(\sigma)| + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)} \\ &\quad \times \eta\int_0^t |\chi(\sigma)|ds\Big] \\ &\leq \Big[\frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)}\mathbf{C}_\rho + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)}\eta\mathbf{C}_\rho\Big]\delta. \quad \square\end{aligned} \tag{20}$$

Theorem 5. *Suppose that the system (16) is HU stable, if there exist*

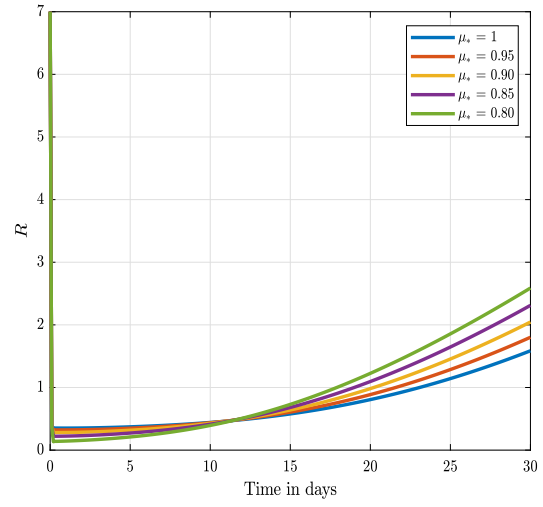
$$\Big[\frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)} + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)}\eta\Big] < 1.$$



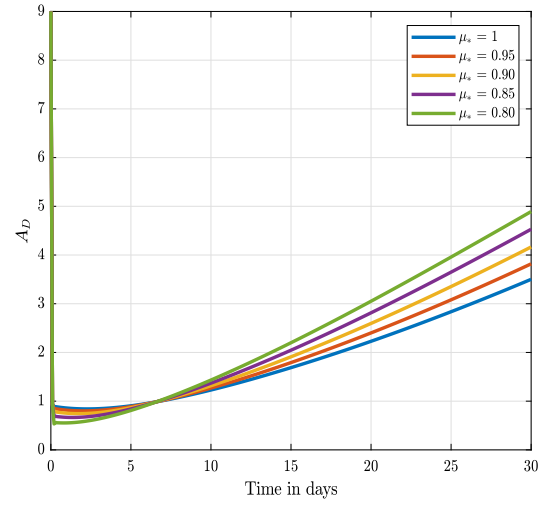
(a) Dynamics of Symptomatic Infected (I_S) class



(b) Dynamics of Quarantine (Q) Class



(c) Dynamics of Recovery (R) Class



(d) Dynamics of class infected with Alzheimer's disease

Fig. 2. Numerical trajectory of the CF-fractional order derivative, μ_* of the model (2).

Proof. With the help from Lemma 6.4, let $\aleph \in \boldsymbol{W}_*$ be any solution and $\tilde{\aleph} \in \boldsymbol{W}_*$ be unique solution for considered problem (17), then

$$\begin{aligned}
|\aleph(t) - \tilde{\aleph}(t)| &= |\aleph(t) - \mathbf{T}_p\tilde{\aleph}(t)| \\
&\leq |\aleph(t) - \mathbf{T}_p\aleph(t)| + |\mathbf{T}_p\aleph(t) - \mathbf{T}_p\tilde{\aleph}(t)| \\
&\leq \Big[\frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)} + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)}\eta\Big]\delta \\
&+ \Big[\frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)}\mathbf{C}_\rho + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)}\eta\mathbf{C}_\rho\Big]\|\aleph - \tilde{\aleph}\| \qquad (21) \\
&\leq \frac{\big[\frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)} + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)}\eta\big]}{\big[\frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)}\mathbf{C}_\rho + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)}\eta\mathbf{C}_\rho\big]}\delta.
\end{aligned}$$

Thus,

$$\|\aleph(t) - \tilde{\aleph}(t)\| \leq \frac{\big[\frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)} + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)}\eta\big]}{\big[\frac{2(1-\mu_*)}{2(1-\mu_*)G(\mu_*)}\mathbf{C}_\rho + \frac{2\mu_*}{(2-\mu_*)G(\mu_*)}\eta\mathbf{C}_\rho\big]}\delta.$$

Hence, we conclude that, the SARS-CoV-2 transmission model incorporate Alzheimer's disease has at least one solution. Consequently, it is generalized HU stable. □

7. Numerical scheme

In this section, we present the numerical results for SARS-CoV-2 transmission model incorporate Alzheimer's disease base on the Lagrange interpolation. The Cauchy problem of the CF fractional derivative can be given as;

$$^{CF}D_t^{\mu_*}\aleph(t) = \Phi(t, \aleph(t)). \qquad (22)$$

On the other hand, we can be expressed (22) as

$$\aleph(t) = \aleph_0(t) + \frac{(1-\mu_*)}{G(\mu_*)}\Phi(t, \aleph(t)) + \frac{\mu_*}{G(\mu_*)} \times \int_0^t \Phi(s, \aleph(s))ds. \qquad (23)$$

Taking (23) at the point $t_{z_*+1} = (z_* + 1)h$ and $t_{z_*} = z_*h$, $z_* = 0, 1, 2, 3, \ldots$, with h being the time step, we have

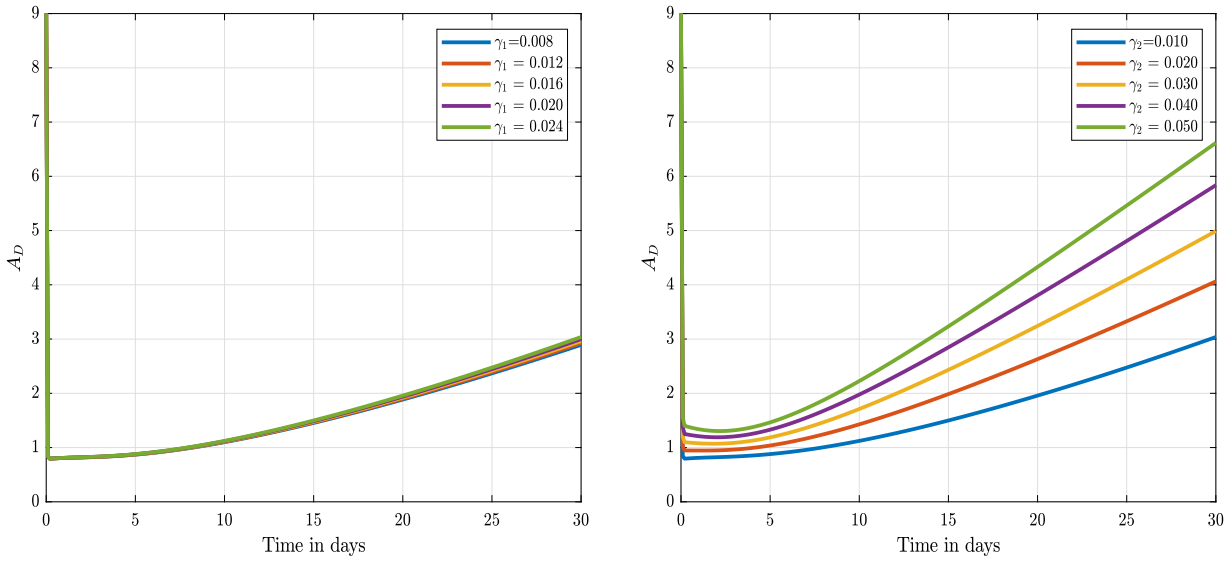


Fig. 3. Dynamics of individuals who have had a Alzheimer's disease over time under different γ_1 (left) and γ_2 (right).

$$\aleph(t_{z_*+1}) = \aleph(0) + \frac{(1-\mu_*)}{G(\mu_*)}\Phi(t_{z_*}, \aleph(t_{z_*})) + \frac{\mu_*}{G(\mu_*)} \times \int_{t_{z_*}}^{t_{z_*+1}} \Phi(s, \aleph(s))ds, \tag{24}$$

$$\aleph(t_{z_*}) = \aleph(0) + \frac{(1-\mu_*)}{G(\mu_*)}\Phi(t_{z_*-1}, \aleph(t_{z_*-1})) + \frac{\mu_*}{G(\mu_*)} \times \int_{t_{z_*}}^{t_{z_*+1}} \Phi(s, \aleph(s))ds. \tag{25}$$

Taking (24) and (25) results in

$$\aleph(t_{z_*+1}) - \aleph(t_{z_*}) = \frac{(1-\mu_*)}{G(\mu_*)}\big(\Phi(t_{z_*}, \aleph(t_{z_*})) - \Phi(t_{z_*-1}, \aleph(t_{z_*-1}))\big) + \frac{\mu_*}{G(\mu_*)} \times \int_{t_{z_*}}^{t_{z_*+1}} \Phi(s, \aleph(s))ds. \tag{26}$$

Eq. (26) in two-step Lagrange polynomial gives

$$\begin{aligned}\aleph(t_{z_*+1}) - \aleph(t_{z_*}) = {} & \frac{(1-\mu_*)}{G(\mu_*)}\big(\Phi(t_{z_*}, \aleph(t_{z_*})) - \Phi(t_{z_*-1}, \aleph(t_{z_*-1}))\big) \\ & + \frac{\mu_*}{G(\mu_*)} \times \int_{t_{z_*}}^{t_{z_*+1}} \Big[\frac{\Phi(t_{z_*}, \aleph(t_{z_*}))}{h}(s - t_{z_*-1}) \\ & - \frac{\Phi(t_{z_*-1}, \aleph(t_{z_*-1}))}{h}(s - t_{z_*}) \Big]\, ds.\end{aligned} \tag{27}$$

The above Eq. (27) leads to

$$\begin{aligned}\aleph(t_{z_*+1}) - \aleph(t_{z_*}) = {} & \frac{(1-\mu_*)}{G(\mu_*)}\big(\Phi(t_{z_*}, \aleph(t_{z_*})) - \Phi(t_{z_*-1}, \aleph(t_{z_*-1}))\big) \\ & + \frac{\mu_*}{G(\mu_*)} \times \Big[\frac{\Phi(t_{z_*}, \aleph(t_{z_*}))}{h} \int_{t_{z_*}}^{t_{z_*+1}} (s - t_{z_*-1})ds \\ & - \frac{\Phi(t_{z_*-1}, \aleph(t_{z_*-1}))}{h} \int_{t_{z_*}}^{t_{z_*+1}} (s - t_{z_*})ds \Big]\,.\end{aligned} \tag{28}$$

Solving the integrals in Eq. (28) yields

$$\begin{aligned}\int_{t_{z_*}}^{t_{z_*+1}} (s - t_{z_*-1})ds &= \frac{3}{2}h^2, \\ \int_{t_{z_*}}^{t_{z_*+1}} (s - t_{z_*})ds &= \frac{1}{2}h^2.\end{aligned} \tag{29}$$

Substituting Eq. (29) into Eq. (28), then the generalize numerical scheme of CF is as follows;

$$\begin{aligned}\aleph_{z_*+1} = {} & \aleph_{z_*} + \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{3h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*}, \aleph_{z_*}) \\ & - \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*-1}, \aleph_{z_*-1}).\end{aligned} \tag{30}$$

Thus, in terms of our CF-fractional Listeriosis model we get;

$$\begin{aligned}S_{1_{z_*+1}} = {} & S_{1_{z_*}} + \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{3h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*}, S_{1_{z_*}}) \\ & - \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*-1}, S_{1_{z_*-1}}).\end{aligned} \tag{31}$$

$$\begin{aligned}S_{2_{z_*+1}} = {} & S_{2_{z_*}} + \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{3h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*}, S_{2_{z_*}}) \\ & - \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*-1}, S_{2_{z_*-1}}).\end{aligned} \tag{32}$$

$$\begin{aligned}E_{z_*+1} = {} & E_{z_*} + \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{3h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*}, E_{z_*}) \\ & - \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*-1}, E_{z_*-1}).\end{aligned} \tag{33}$$

$$\begin{aligned}I_{A_{z_*+1}} = {} & I_{A_{z_*}} + \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{3h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*}, I_{A_{z_*}}) \\ & - \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*-1}, I_{A_{z_*-1}}).\end{aligned} \tag{34}$$

$$\begin{aligned}I_{S_{z_*+1}} = {} & I_{S_{z_*}} + \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{3h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*}, I_{S_{z_*}}) \\ & - \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*-1}, I_{S_{z_*-1}}).\end{aligned} \tag{35}$$

$$\begin{aligned}Q_{z_*+1} = {} & Q_{z_*} + \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{3h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*}, Q_{z_*}) \\ & - \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*-1}, Q_{z_*-1}).\end{aligned} \tag{36}$$

$$\begin{aligned}R_{z_*+1} = {} & R_{z_*} + \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{3h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*}, R_{z_*}) \\ & - \Big[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{h\mu_*}{2G(\mu_*)}\Big]\Phi(t_{z_*-1}, R_{z_*-1}).\end{aligned} \tag{37}$$

$$A_{D_{z_*+1}} = A_{D_{z_*}} + \left[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{3h\mu_*}{2G(\mu_*)}\right]\Phi(t_{z_*}, A_{D_{z_*}}) - \left[\frac{(1-\mu_*)}{G(\mu_*)} + \frac{h\mu_*}{2G(\mu_*)}\right]\Phi(t_{z_*-1}, A_{D_{z_*-1}}). \tag{38}$$

8. Numerical simulation and discussion

In this section, we provide the numerical solutions to the CF fractional order model for SARS-CoV-2 involving Alzheimer's disease, using the Adams–Bashforth method, taken account of the following initial conditions; $S_1(0) = 18$; $S_2(0) = 15$; $E(0) = 10$; $I_A(0) = 8$; $I_S(0) = 10$; $Q(0) = 15$; $R(0) = 7$; $A_D(0) = 9$, with parameter values in Table 2. Parameters have a crucial influence in disease propagation in numerical solutions, and which fractional order μ_* indicates the best memory effect. As a result, using the parameter values in Table 2, the trajectory of each compartment through time has been simulated for various values of μ_*. Furthermore, graphics for significantly influence parameters have also been obtained. For different values of μ_* with parameter values Figs. 1–4 gives graphical presentation of our proposed SARS-CoV-2 pandemic and Alzheimer's disease. Figs. 1 and 2 subplots, demonstrates the fall and up in different compartment population for the different μ_* values, and it is worth noting that, the forecast is dependent on the value of the fractional order μ_*, implying that the epidemic's evolution and control are linked to memory. For increasing values of μ_*, in Fig. 1(a,b), and (a,c), we observed that susceptible (young age) individuals, susceptible (old age) individuals, symptomatic infected, and exposed individuals all have a declining attitude with no convergency at the end of the simulation time. This behavior in a real-world or biological view point is inevitable as a result of people having no or less knowledge about SARS-CoV-2 associated with Alzheimer's disease and also the emergence and rapid transmission of the SARS-CoV-2 with different variants coming up, especially Omicron variant. In Fig. 2(b) and (d), we observed increasing behavior of the trajectories as the fractional order μ_* reduce from 1. This implies that a high rate of isolation has significant impact on Alzheimer's disease, and hence when there is too much quarantine, the rate of infected person end up with Alzheimer's disease. For different values of γ_1 and γ_2, the time-dependent fluctuation of the Alzheimer's disease class has been explored in Fig. 3. We notice that an increase in γ_2 increases the number of Alzheimer's disease individuals, and an increase in γ_1 has minimal effect on the Alzheimer's disease compartment. We see in Fig. 4 an increase in m increases the number of Alzheimer's disease individuals. Similarly, we see in Fig. 5 an increase in ξ_1 and ξ_2 increases the number of Alzheimer's disease individuals.

9. Conclusion

To assess and analyze the SARS-CoV-2 transmission model incorporating Alzheimer's disease, a mathematical model has been developed and analyzed under CF fractional order derivative. Initial formulation of the model uses a traditional integer order differential system, which is later expanded to include a CF fractional order differential system. We determined the classical analysis of our proposed model, thus, (positivity, boundedness and invariant region). By using the fixed point theorem of Banach and Krasnoselskii's type, the proposed system has been proved to have at least one unique solution. Due to complicated nature of SARS-CoV-2 and its effect associated with Alzheimer's disease, we discussed the proposed model stability under HU stability type to get approximate solution. Numerical analysis and simulations are carried out to check the actual behavior of our proposed model using the Adams–Bashforth technique and the CF fractional order derivative. Base on our numerical findings, if the number of symptomatic and asymptomatic infected individuals in quarantine can be reduced by using techniques such as short-days-quarantine, enforcing nationwide vaccination, rapid antigen testing, improving isolation centers, then it will be possible to control the SARS-CoV-2 and its effect associated with

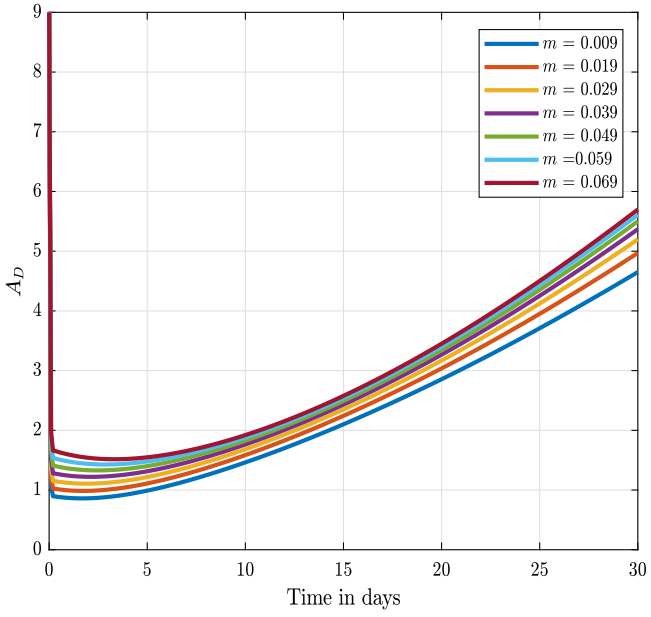


Fig. 4. Numerical trajectory of the Alzheimer's disease over time under different values of m.

Alzheimer's disease. In this regard, this paper gives insight to future research possibilities. In the future, because this work is not supported by real data, alternative types of fractional operators can be considered with real data and also optimal control section and sensitivity analysis are encourage.

CRediT authorship contribution statement

Emmanuel Addai: Conceptualization, Methodology, Formal analysis, Software, Visualization, Writing – original draft, Writing – review & editing. **Lingling Zhang:** Supervision, Methodology, Formal analysis, Visualization, Writing – review & editing. **Ama Kyerewaa Preko:** Formal analysis, Visualization, Writing – review & editing. **Joshua Kiddy K. Asamoah:** Supervision, Methodology, Formal analysis, Software, Visualization, Writing – review & editing.

Declaration of competing interest

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

Data availability

No data was used for the research described in the article.

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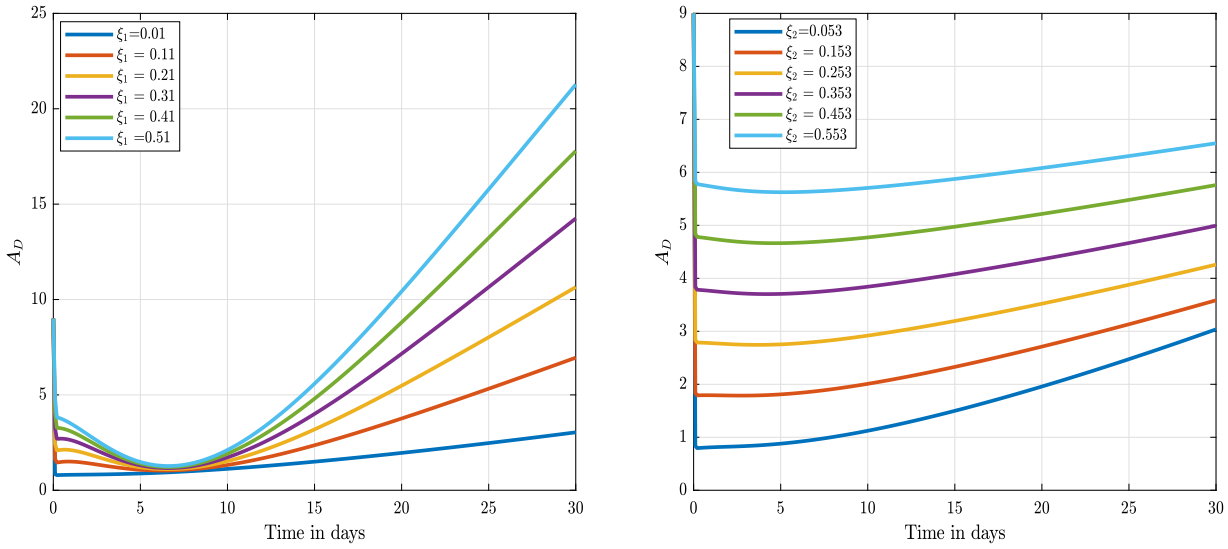

Fig. 5. Dynamics of individuals who have had a Alzheimer's disease over time under different ξ_1 (left) and ξ_2 (right).

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