

# A case study of 2019-nCOV cases in Argentina with the real data based on daily cases from March 03, 2020 to March 29, 2021 using classical and fractional derivatives

Pushpendra Kumar<sup>a</sup>, Vedat Suat Erturk<sup>b</sup>, Marina Murillo-Arcila<sup>c,\*</sup>, Ramashis Banerjee<sup>d</sup>,  
A. Manickam<sup>e</sup>

<sup>a</sup>*Department of Mathematics and Statistics, School of Basic and Applied Sciences, Central University of Punjab, Bathinda, Punjab-151001, India*

<sup>b</sup>*Department of Mathematics, Faculty of Arts and Sciences, Ondokuz Mayıs University, Atakum-55200, Samsun, Turkey*

<sup>c</sup>*Instituto Universitario de Matemática Pura y Aplicada, Universitat Politècnica de València, 46022 Valencia, Spain*

<sup>d</sup>*Department of Electrical Engineering, National Institute of Technology, Silchar, India*

<sup>e</sup>*School of Advanced Sciences & Languages, Department of Mathematics, VIT Bhopal University, Kottri Kalan (Village) -466 114, Sehore (District), Madhya Pradesh, India*

---

## Abstract

In this study, our aim is to explore the dynamics of COVID-19 or 2019-nCOV in Argentina considering the parameter values based on the real data of this virus from *March 03, 2020* to *March 29, 2021* which is a data range of more than one complete year. We propose a Atangana-Baleanu type fractional order model and simulate it by using Predictor-Corrector (P-C) method. First we introduce the biological nature of this virus in theoretical way and then formulate a mathematical model to define its dynamics. We use a well-known effective optimization scheme based on renowned trust-region-reflective (TRR) method to perform the model calibration. We have plotted the real cases of COVID-19 and compared our integer-order model with the simulated data along with the calculation of basic reproductive number. Concerning fractional order simulations, first we prove the existence and uniqueness of solution and then write the solution along with the stability of the given P-C method. **Number of graphs at various fractional-order values are simulated to predict the future dynamics of the virus in Argentina which is the main contribution of this paper.**

*Keywords:* COVID-19, Argentina, Mathematical models, TRR algorithm, Atangana-Baleanu non-classical derivative

*2000 MSC:* 34D20, 26A33, 47H10, 92D30

---

## 1. Introduction

2 Coronavirus disease 2019 (COVID-19) is considered as most dangerous epidemic disease  
3 of this decade, which appeared for the first time in Wuhan (China) in the last month of 2019.  
4 It has been observed that coronavirus disease 2019 (COVID-19) is a vital health concern as

---

\*Corresponding author

*Email addresses:* kumarsaraswatpk@gmail.com (Pushpendra Kumar), vserturk@omu.edu.tr (Vedat Suat Erturk), mamuar1@upv.es (Marina Murillo-Arcila), ramashisbanerjee@gmail.com (Ramashis Banerjee), manickammaths2011@gmail.com (A. Manickam)

5 it can be fatal specially in old-aged people. SARS-CoV-2 virus causes COVID-19 disease.  
6 Researchers and medical practitioners have much information about the death due to the  
7 clinical disease but they do not know much about its pathobiology. It has been seen that the  
8 characteristics of cellular answers to COVID-19 are not clearly known and understood, but  
9 based on the previous studies on SARS-CoV, a predictable sequence of the events can be  
10 hypothesized. According to the cells that SARS-CoV infects, COVID-19 can be separated  
11 into three periods that coincide with the clinical parts of the disease [1].

12 The structure of SARS-CoV-2 needs to be understood. It has been found that SARS-  
13 CoV-2 is part of the family of beta-coronavirus, some of which caused other two epidemics  
14 named as MERS-CoV and SARS-CoV as it can be found in reference [2]. In [3] Hui et al.  
15 analysed the structure of such coronaviruses. According to the World Health Organisation  
16 (WHO), the rate of death due to SARS-CoV-2 is lower than the one due to SARS-CoV  
17 though the rate of transmission is higher in case of SARS-CoV-2 as compared to SARS-  
18 CoV. The RNA structure of different coronaviruses has been stated in [4]. It has been  
19 observed that SARS-CoV-2 has distinctive spike proteins and presents a specific peptide,  
20 namely PrrA, which allows to divide the spike protein using cellular proteases enzyme in  
21 order to disseminate from the virus to the cell of the host easily [5, 6]. So, the rate of  
22 transmission of SARS CoV-2 is much higher and this is the prime cause of its spreading  
23 throughout the globe in a very short period of time.

24 It has been observed as soon as SARS-CoV-2 makes its entrance into the epithelial cell  
25 of alveoli of the respiratory tract of human being, the virus activates the immune response  
26 of human being due to the fast multiplication rate of SARS-CoV-2 cells. After that, the pul-  
27 monary tissue from the respiratory tract of human being is damaged causing the stimulation  
28 of more and more white blood cells. This process is known as cytokine release syndrome [7]  
29 which can cause a multiple organ failure [8].

30 There are number of stages included in the infection of COVID-19. The initial stage is  
31 asymptomatic and covers 1 to 2 days after getting infected. It has been noticed that after  
32 inhaling the virus it attaches to epithelial cells from the nasal cavity of human being and  
33 the virus starts the process of replication [9]. In this initial stage, infected people are able to  
34 spread the infection. During the second stage the virus is able to spread and move quickly  
35 down to the respiratory tract along with the conducting passage of air [10]. It has been  
36 observed that for 80% of the infected individuals the COVID-19 illness will be lenient and  
37 it is restricted to upper as well as conducting passage of air. The third stage of the disease  
38 involves hypoxia and ground glass infiltrates along with the growth of acute respiratory  
39 distress syndrome (ARDS). It has been seen that 20% of the infected individuals will move  
40 to third stage of the disease and they will develop pulmonary invasions which will lead to  
41 severe disease. From the initial estimation the rate of severity is about 2% but it varies with  
42 age [1]. In this stage SARS-Cov-2 virus is able to reach the units of the lung which are  
43 responsible for exchange of gas and infect the type II cells of alveoli. SARS-CoV is able to  
44 spread within type II cells and huge viral particles are liberated [11]. Old-aged people are at  
45 high risk since they have low immune response which can lead to severe consequences [12].

46 Mathematical models are best way to describe the dynamics of the disease. Many mathe-  
47 matical models have been provided to illustrate the behaviour of COVID-19 by using integer  
48 and fractional-order derivatives [13, 14, 15, 16, 17, 18]. A fractional-order COVID-19 model  
49 with delay has been proposed in [19]. *Atangana et al.* in [20] have discussed a mathematical  
50 model of COVID-19 with deterministic and stochastic approaches. Authors in [21, 22, 23]  
51 have also simulated the dynamics of 2019-nCoV by using effective mathematical models. A

52 study on fractional-order model of HIV is given in [24]. Authors in [25] proposed a research  
53 on the dynamics of a population model. In [26], a COVID-19 model along with necessary  
54 awareness programs has been explored. The roles of isolation and quarantine measures for  
55 COVID-19 outbreaks are given in [27]. Authors in [28] have solved a model of huanglongbing  
56 transmission with an effective numerical algorithm. A psychological model in fractional-order  
57 sense has introduced in [29]. *Kumar et al.* in [30] have described a time-delay fractional-order  
58 mathematical model of oncolytic virotherapy. Many other epidemics have been described by  
59 using fractional mathematical models [31, 32]. Recently, authors in [33] studied the structure  
60 of rabies, and canine distemper virus epidemics by using generalised Caputo non-classical  
61 derivative. Two fractional-order mathematical models for describing mosaic disease have  
62 been given in [34]. A environmental study in fractional order sense is given in [35].

63 This paper is formulated to trace the dynamics of COVID-19 cases in Argentina by using  
64 Atangana-Baleanu type fractional mathematical model. The study is framed as follows:  
65 In section 2, we describe the integer-order model dynamics. In section 3, we plot the real-  
66 data cases of COVID-19 in Argentina and calculate the parameter values for our simulations.  
67 Section 4 is organized in number of sub-sections where we simulate the fractional-order model  
68 and perform all theoretical and graphical simulations. Finally in section 5, we provide our  
69 conclusions about the study.

## 70 2. Model description

In this paper, we consider a compartmental mathematical model [36]. Natural births and death rates have not been considered in the model as those have no impact on the short-term outbreaks of COVID-19. The mentioned model focuses on two distinct groups of susceptible individuals: susceptible individuals given by  $S(t)$  and confined individuals who follow lock-down or confinement intervention partially as the confinement is not perfect. We symbolize the class of confined individuals as  $C(t)$ . The rest of the population is compartmentalized as follows: exposed individuals  $E(t)$  at time  $t$ , asymptomatic (having no clinical symptoms or mild symptoms) individuals  $A(t)$ , quarantined symptomatic infectious individuals  $Q(t)$ , hospitalized or isolated individuals  $H(t)$  and recovered individuals  $R(t)$ .

In the given model,  $p$  is the transmission rate from confined susceptible humans to rejoined unconfined susceptible humans,  $q\gamma$  is the exposure rate of the asymptomatic humans,  $r_4\sigma_2$  is the progression rate from severely infected to hospitalization and quarantine rate is given by  $(1-q)\gamma$ .  $r_1\sigma_1$  denotes the transmission rate of the asymptomatic humans to become severely infectious.  $r_2\sigma_1$  shows the transmission rate of confinement or hospitalization to isolation of the asymptomatic group. The natural recovery of soft symptomatic humans is represented by the rate  $(1-r_1-r_2)\sigma_1$ , whereas  $(1-r_3-r_4)\sigma_2$  is the recovery rate for  $Q$  classes. In the model,  $\delta_h$ ,  $\delta_q$ , and  $\delta_a$  are stood for the COVID-19 death rates in the given model classes, respectively. The structure of the non-linear model based on the given parameters explanation

Parameter	Description
$\beta$	Contact rate
$\eta$	Relative transmissibility of quarantined infected carrier
$p$	Rate of transition from C(t) to S(t)
$c$	Confinement rate
$\epsilon$	Confinement efficacy
$\gamma$	Rate of transition from E(t) to Q(t)
$q$	Rate of exposed individuals becoming quarantined
$\sigma_1$	Rate of transition from A(t) to Q(t)
$\sigma_2$	Transition rate from Q(t) to A(t)
$\sigma_3$	Rate of transition from hospitalised to recovered group
$r_1$	Fraction of A(t) becoming quarantined humans
$r_2$	Rate of unquarantined infected humans going to be hospitalised
$r_3$	Rate of quarantined infected humans moving to unquarantined infected humans
$r_4$	Rate of quarantined infected humans going to be hospitalised
$\delta_a$	Rate of 2019-nCOV deaths in unquarantined infected humans
$\delta_q$	Rate of 2019-nCOV deaths in quarantined infected humans
$\delta_h$	Rate of 2019-nCOV deaths in hospitalised

Table 1: Model parameters and their description

is provided as follows:

$$\left\{ \begin{array}{l}
 \frac{dS}{dt}(t) = pC(t) - cS(t) - \left[ \frac{\beta(A(t) + \eta Q(t))}{N(t)} \right] S(t), \\
 \frac{dC}{dt}(t) = cS(t) - pC(t) - (1 - \epsilon) \left[ \frac{\beta(A(t) + \eta Q(t))}{N(t)} \right] C(t), \\
 \frac{dE}{dt}(t) = \left[ \frac{\beta(A(t) + \eta Q(t))}{N(t)} \right] [S(t) + (1 - \epsilon)C(t)] - \gamma E(t), \\
 \frac{dA}{dt}(t) = q\gamma E(t) + r_3\sigma_2 Q(t) - (\sigma_1 + \delta_a)A(t), \\
 \frac{dQ}{dt}(t) = (1 - q)\gamma E(t) + r_1\sigma_1 A(t) - (\sigma_2 + \delta_q)Q(t) \\
 \frac{dH}{dt}(t) = r_2\sigma_1 A(t) + r_4\sigma_2 Q(t) - (\sigma_3 + \delta_h)H(t) \\
 \frac{dR}{dt}(t) = (1 - r_1 - r_2)\sigma_1 A(t) + (1 - r_3 - r_4)\sigma_2 Q(t) + \sigma_3 H(t)
 \end{array} \right. \quad (1)$$

The parameters are given in Table 1. A brief discussion on the model characteristics like boundedness and positivity of solutions is given in reference [36]. Authors in [36] calculated the greatest disease-free equilibrium point which is defined by

$$x_0 = (S_0, C_0, 0, 0, 0, 0, 0)' = \left( \frac{pN_0}{p+c}, \frac{cN_0}{p+c}, 0, 0, 0, 0, 0 \right)'.$$

Using the procedure given in [37], the matrices  $F$  and  $V$  for calculating the basic reproductive number are given by:

$$F = \begin{pmatrix} 0 & \beta \frac{S_0 + (1-\epsilon)C_0}{N_0} & \eta \beta \frac{S_0 + (1-\epsilon)C_0}{N_0} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad (2)$$

$$V = \begin{pmatrix} \gamma & 0 & 0 \\ -q\gamma & (\sigma_1 + \delta_a) & -r_3\sigma_2 \\ -(1-q)\gamma & -r_1\sigma_1 & (\sigma_2 + \delta_q) \end{pmatrix}.$$

As a consequence, they obtained the basic reproductive number as the spectral-radius of the generation matrix,  $FV^{-1}$ :

$$\mathcal{R}_c = \rho(FV^{-1}) = \beta \frac{S_0 + (1-\epsilon)C_0}{N_0} \frac{(r_1\sigma_1\eta + (\sigma_2 + \delta_q))q + ((\sigma_1 + \delta_a)\eta + \sigma_2r_3)(1-q)}{(\sigma_1 + \delta_a)(\sigma_2 + \delta_q) - r_1r_3\sigma_1\sigma_2}. \quad (3)$$

71 Here  $\rho(\cdot)$  denotes the spectral-radius operator.

72 **Lemma 1.** *If  $\mathcal{R}_c < 1$ , the epidemic-free equilibrium  $x_0$  is locally asymptotically stable and*  
 73 *unstable if  $\mathcal{R}_c > 1$ .*

*Proof.* First, we observe that the last two equations of (1) are not coupled to the rest of equations of the model. As the total population size,  $N_0$ , is constant, we get  $S + C = N_0 - (E + A + Q + H + R)$ . Then the local stability of the given system (1) can be analysed via remaining model of variables  $(E, A, Q)$ . As a consequence, we establish that the Jacobian matrix related to these variables is written by

$$\mathcal{J} = \begin{pmatrix} -\gamma & \beta \frac{(S_0 + (1-\epsilon)C_0)}{N_0} & \beta \eta \frac{(S_0 + (1-\epsilon)C_0)}{N_0} \\ q\gamma & -(\sigma_1 + \delta_a) & r_3\sigma_2 \\ (1-q)\gamma & r_1\sigma_1 & -(\sigma_2 + \delta_q) \end{pmatrix}.$$

The roots of the next characteristic polynomial correspond to the eigenvalues of  $\mathcal{J}$ :

$$P_{\mathcal{J}}(x) = x^3 + a_2x^2 + a_1x + a_0,$$

74 where,  $a_2 = \gamma + k_2 + k_1$ ,  $a_2 = (k_1k_2 - \sigma_1\sigma_2r_1r_3) \left[ \frac{N_0k_2 + N_0k_1}{N_0(k_1k_2 - \sigma_1\sigma_2r_1r_3)} \gamma + 1 - \gamma \frac{(\eta(1-q) + q)}{q(k_2 + r_1\sigma_1\eta) + (1-q)(\eta k_1 + r_3\sigma_2)} \mathcal{R}_c \right]$   
 75 and  $a_0 = \gamma(k_1k_2 - r_1r_3\sigma_1\sigma_2)(1 - \mathcal{R}_c)$ . Note that  $a_2$  is always positive.  $a_0$  and  $a_1$  are positive  
 76 as long as  $\mathcal{R}_c < 1$ . Therefore, all eigenvalues of  $\mathcal{J}$  have negative real parts. Consequently,  
 77 the epidemic-free equilibrium  $x_0$ , is locally asymptotically stable if  $\mathcal{R}_c < 1$ .  $\square$

Excluding confinement measures, i.e. if  $\epsilon = 0$ ,  $\mathcal{R}_c$  is convergent to the basic reproductive number,  $\mathcal{R}_0$ , written by

$$\mathcal{R}_0 = \beta \frac{(r_1\sigma_1\eta + (\sigma_2 + \delta_q))q + ((\sigma_1 + \delta_a)\eta + \sigma_2r_3)(1-q)}{(\sigma_1 + \delta_a)(\sigma_2 + \delta_q) - r_1r_3\sigma_1\sigma_2}. \quad (4)$$

Using (3), it follows that

$$\mathcal{R}_c = \mathcal{R}_0 \left( \frac{S_0 + (1-\epsilon)C_0}{N_0} \right) = \mathcal{R}_0 \left( \frac{p + (1-\epsilon)c}{p + c} \right). \quad (5)$$

Parameters	Probable range	base value	TRR output	Reference
$\beta$	0.5-1.5	0.5	1.2757	Fitted
$\sigma_1$	0.001-0.1	0.03	0.01	Fitted
$\sigma_2$	0.1-0.9	0.3	0.3488	Fitted
$\sigma_3$	0.1-0.9	0.3	0.6917	Fitted
$r_1$	0.1-0.9	0.5	0.302	Estimated
$r_2$	0.1-0.9	0.5	0.302	Estimated
$r_3$	0.1-0.9	0.5	0.2227	Estimated
$r_4$	0.1-0.9	0.5	0.3172	Estimated
$\delta_a$	0.001-0.1	0.01	0.1	Fitted
$\delta_q$	0.001-0.1	0.01	0.09	Fitted
$\delta_h$	0.001-0.1	0.01	0.0998	Fitted
$p$	0.0005-0.1	0.05	0.00051	Estimated
$\eta$	0.4-0.6	0.5	0.5077	Fitted
$c$	0.001-0.1	0.005	0.0155	Estimated
$\gamma$	1/14-1/3	1/5.1	0.1673	Fitted
$\epsilon$	0.1-0.99	0.7	0.8417	Estimated
$q$	0.001-0.5	0.2	0.1181	Estimated

Table 2: Model parameters calibration by using the mentioned scheme

### 78 3. Model calibration and forecasting

A recently proposed optimization scheme, which is an evolution of Levenberg-Marquardt one [36, 38] and depends on trust-region-reflective (TRR) scheme, is employed to perform the model (1) calibration. This robust optimization method can be applied for simulating non-linear least-squares problems. The implementation of the scheme is done with the help of `lsqcurvefit` function, which is included in Optimization Toolbox from MATLAB. Possible parameter values are calculated by this function. The real data of daily collected cases in Argentina are collected from the trusted data website which can be verified in [39]. We used 7-day running average of the given reported COVID-19 cases analysed in the calibration of model because of changeable structure of real data as it can be seen in Figure 2. The daily testing in Argentina is really inconsistent as in all over the nations. By the early updates, the total population size of Argentina is around 45,481,402 [39]. As initial population amount, we get  $S(0) = 45481396$ ,  $C(0) = 0$ ,  $E(0) = 0$ ,  $A(0) = 0$ ,  $H(0) = 0$ ,  $R(0) = 0$  and  $Q(0) = 1$ . The numerical solutions are achieved solving the following optimization equation:

$$\min_{\psi} \| (NPC_{predict}(t), NRC_{predict}(t)) - (NPC_{data}, NRC_{data}) \| \quad (6)$$

79 where  $\psi = \{\beta, \sigma_1, \sigma_2, \sigma_3, r_1, r_2, r_3, r_4, \delta_a, \delta_q, \delta_h, \eta, p, c, \epsilon, \gamma, q\}$  is shown in Table 2.

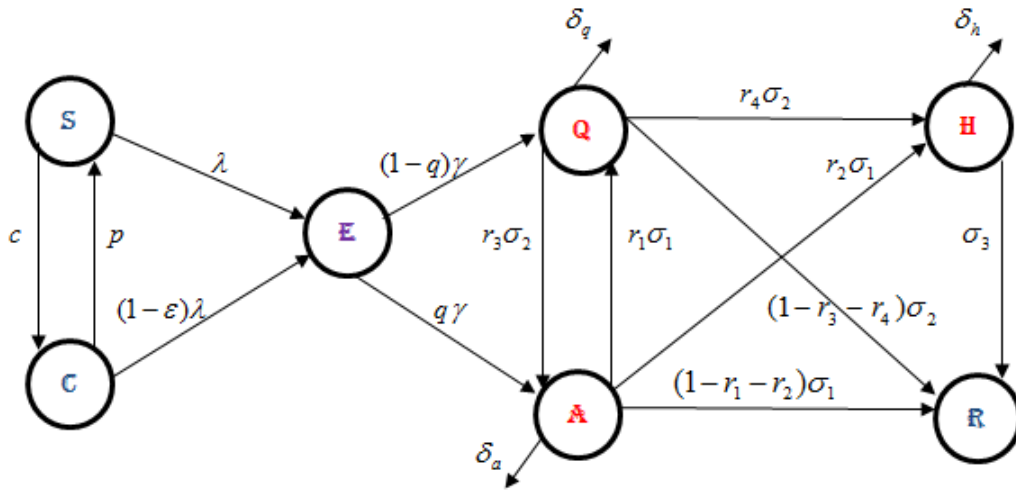


Figure 1: Model frame

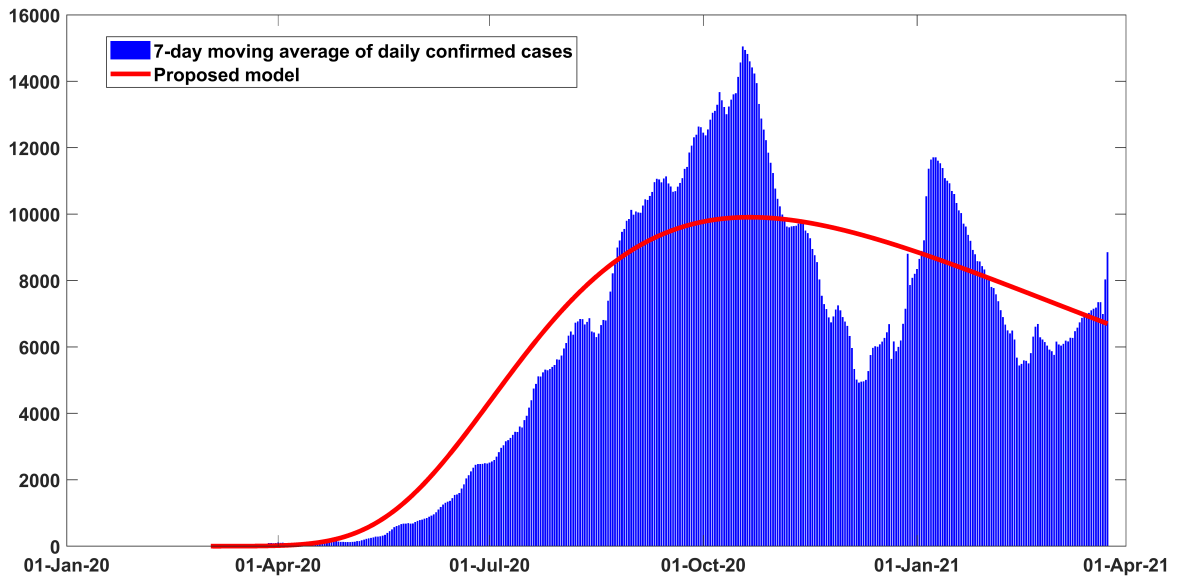


Figure 2: Output of the model performance fitting for daily cases of infection in Argentina from March 03, 2020 to March 29, 2021.

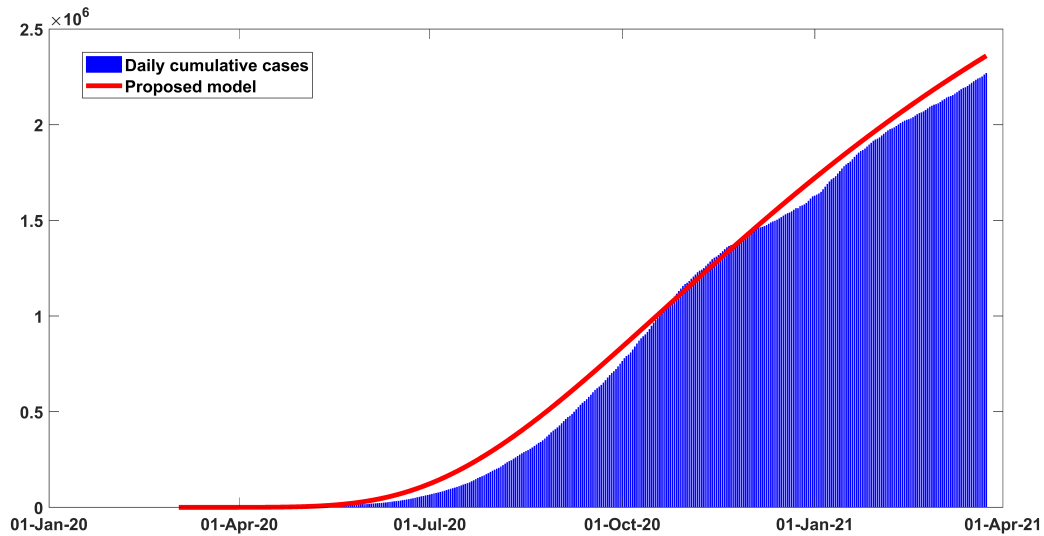


Figure 3: Output of the model performance fitting for cumulative cases of infection in Argentina from March 03, 2020 to March 29, 2021.

80 In Figures 2 and 3 it is shown that our model fits really well to the given real-data  
 81 of Argentina. The estimated value of the basic reproductive number  $\mathcal{R}_0$  is about  $\sim 1.41$   
 82 (95%  $CI : 1.2 - 1.6$ ) as of March 10, 2021. This value could change (increase or decrease)  
 83 in future cause of the new wave of Covid-19 which will totally depend on our health care  
 84 measures. The disease could fade out at the end of December 2021. All necessary parameter  
 85 values aspects applied to justify this scenario are mentioned in Table 2.

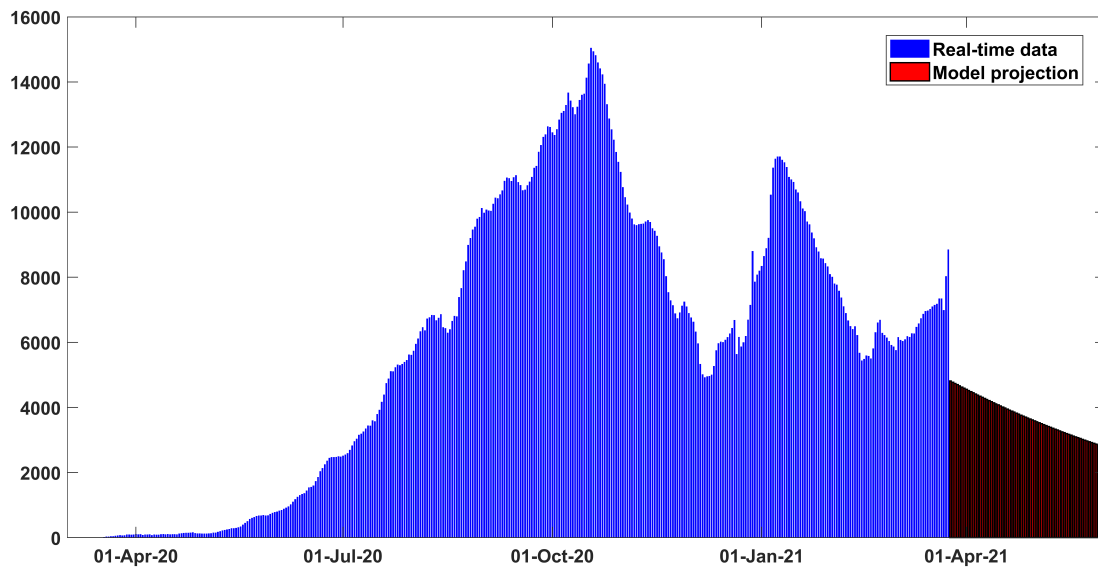


Figure 4: New daily reported cases projected and calibrated for Argentina from early March 2020 to late May 2021.

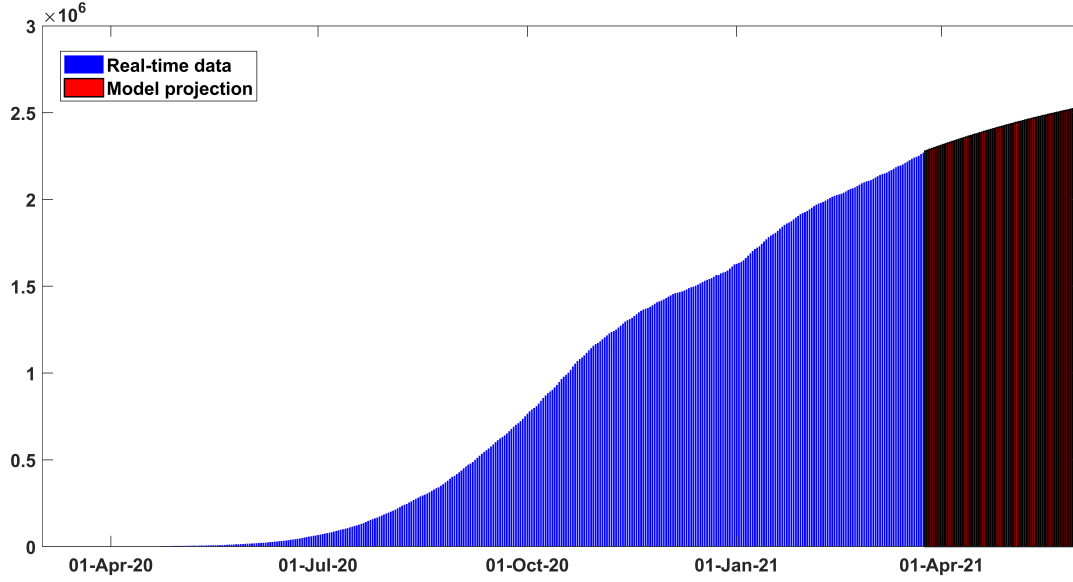


Figure 5: Cumulative infected cases fitted and projected for Argentina from early March to 2020 late May 2021.

#### 86 4. Atangana-Baleanu fractional order model

87 From the above integer-order simulations, we can see that the given model is working  
 88 good to project real-data for future. However, the integer-order model is not giving us much  
 89 varieties in our predictions. In such cases, fractional order derivatives always provide us a  
 90 chance to obtain better predictions under the given real data range. Motivated by this fact,  
 91 we replace the above classical model into Atangana-Baleanu type fractional order model  
 92 which is defined under the Mittag-Leffler kernel.

##### 93 4.1. Preliminaries

94 Some important definitions are recalled here.

**Definition 1.** [40] Given  $\mathcal{S} \in \mathcal{H}^1(u, v)$ , where  $v > u$  and  $0 \leq \Phi \leq 1$ , the non-classical type Atangana-Baleanu (AB) derivative is stated as:

$${}_{u}^{ABC}D_t^{\Phi}(\mathcal{S}(t)) = \frac{ABC[\Phi]}{1-\Phi} \int_u^t \mathcal{S}'(\eta) E_{\Phi} \left[ \Phi \frac{(t-\eta)^{\Phi}}{\Phi-1} \right] d\eta. \quad (7)$$

95 where  $ABC[\Phi]$  verifying  $ABC[0] = ABC[1] = 1$  designates the normalization function and  
 96  $E_{\Phi}(\cdot)$  is the Mittag-Leffler function with one-parameter.

**Definition 2.** [40] The non-classical type AB integral for normalization function  $ABC[\Phi]$  is set as

$${}_{u}^{ABC}I_t^{\Phi}(\mathcal{S}(t)) = \frac{1-\Phi}{ABC[\Phi]} \mathcal{S}(t) + \frac{\Phi}{\Gamma(\Phi)ABC[\Phi]} \int_u^t \mathcal{S}(\eta) (t-\eta)^{\Phi-1} d\eta. \quad (8)$$

97 **Lemma 2.** ([40]) *The solution of the given system for  $0 < \Phi < 1$*

$$\begin{aligned} {}^{ABC}D_0^\Phi x(t) &= z(t), \quad t \in [0, T], \\ x(0) &= x_0, \end{aligned}$$

*is stated as*

$$x(t) = x_0 + \frac{(1 - \Phi)}{ABC(\Phi)} z(t) + \frac{\Phi}{ABC(\Phi)\Gamma(r)} \int_0^t (t - \omega)^{\Phi-1} z(\omega) d\omega.$$

**Lemma 3.** [41] *If  $0 < \Phi < 1$  and  $a_1$  is a non-negative integer, then there exist constants  $C_{\Phi,1} > 0$  and  $C_{\Phi,2} > 0$  only dependent on  $\Phi$ , such that*

$$(a_1 + 1)^\Phi - a_1^\Phi \leq C_{\Phi,1}(a_1 + 1)^{\Phi-1},$$

*and*

$$(a_1 + 2)^{\Phi+1} - 2(a_1 + 1)^{\Phi+1} + a_1^{\Phi+1} \leq C_{\Phi,2}(a_1 + 1)^{\Phi-1}.$$

**Lemma 4.** [41] *Let us suppose  $\nu_{p,n} = (n - p)^{\Phi-1}$  ( $p = 1, 2, \dots, n - 1$ ) and  $\nu_{p,n} = 0$  for  $p \geq n$ ,  $\Phi, M, h, T > 0$ ,  $a_1 h \leq T$  and  $a_1$  is a positive integer. Let  $\sum_{p=a_1}^{p=n} \nu_{p,n} |e_p| = 0$  for  $k > n \geq 1$ . If*

$$|e_n| \leq Mh^\Phi \sum_{p=1}^{n-1} \nu_{p,n} |e_p| + |\eta_0|, \quad n = 1, 2, \dots, a_1,$$

*then*

$$|e_{a_1}| \leq C|\eta_0|, \quad a_1 = 1, 2, \dots$$

98 *where  $C$  is a positive constant independent of  $a_1$  and  $h$ .*

#### 99 4.2. Unique solution existence for fractional order model

100 Let consider the Banach space  $\mathbf{Z} = \mathbf{X} \times \mathbf{X} \times \mathbf{X} \times \mathbf{X} \times \mathbf{X} \times \mathbf{X} \times \mathbf{X}$ , where  $\mathbf{X} = C[0, T]$   
 101 endowed with the norm-function  $\|A\| = \|(S, C, E, A, Q, H, R)\| = \max_{t \in [0, T]} [|S(t)| + |C(t)| +$   
 102  $|E(t)| + |A(t)| + |Q(t)| + |H(t)| + |R(t)|]$ .

103 **Theorem 1.** [42] *Consider  $\mathbf{B}$  to be a convex subset of  $\mathbf{Z}$  and let  $\mathbf{F}, \mathbf{G}$  depict couple operators*  
 104 *satisfying*

- 105 1.  $\mathbf{F}u + \mathbf{G}u \in \mathbf{B} \quad \forall u \in \mathbf{B}$ ;
- 106 2.  $\mathbf{F}$  is a contraction;
- 107 3.  $\mathbf{G}$  is compact and continuous.

108 *Then  $\mathbf{F}u + \mathbf{G}u = u$  possess at least one solution.*

Now the generalization of the classical model (1) in Atangana-Baleanu fractional derivative sense reads as follows:

$$\left\{ \begin{aligned} {}^{ABC}D_t^\Phi S(t) &= pC(t) - cS(t) - \left[ \frac{\beta(A(t) + \eta Q(t))}{N(t)} \right] S(t), \\ {}^{ABC}D_t^\Phi C(t) &= cS(t) - pC(t) - (1 - \epsilon) \left[ \frac{\beta(A(t) + \eta Q(t))}{N(t)} \right] C(t), \\ {}^{ABC}D_t^\Phi E(t) &= \left[ \frac{\beta(A(t) + \eta Q(t))}{N(t)} \right] [S(t) + (1 - \epsilon)C(t)] - \gamma E(t), \\ {}^{ABC}D_t^\Phi A(t) &= q\gamma E(t) + r_3\sigma_2 Q(t) - (\sigma_1 + \delta_a)A(t), \\ {}^{ABC}D_t^\Phi Q(t) &= (1 - q)\gamma E(t) + r_1\sigma_1 A(t) - (\sigma_2 + \delta_q)Q(t), \\ {}^{ABC}D_t^\Phi H(t) &= r_2\sigma_1 A(t) + r_4\sigma_2 Q(t) - (\sigma_3 + \delta_h)H(t), \\ {}^{ABC}D_t^\Phi R(t) &= (1 - r_1 - r_2)\sigma_1 A(t) + (1 - r_3 - r_4)\sigma_2 Q(t) + \sigma_3 H(t), \end{aligned} \right. \quad (9)$$

where  ${}^{ABC}D_t^\Phi$  is the Atangana-Baleanu operator of fractional order  $\Phi$ . Now we rewrite the right-hand side of model (9) as follows:

$$\begin{cases} f_1(t, S, \dots, R) = pC(t) - cS(t) - \left[ \frac{\beta(A(t) + \eta Q(t))}{N(t)} \right] S(t), \\ f_2(t, S, \dots, R) = cS(t) - pC(t) - (1 - \epsilon) \left[ \frac{\beta(A(t) + \eta Q(t))}{N(t)} \right] C(t), \\ f_3(t, S, \dots, R) = \left[ \frac{\beta(A(t) + \eta Q(t))}{N(t)} \right] [S(t) + (1 - \epsilon)C(t)] - \gamma E(t), \\ f_4(t, S, \dots, R) = q\gamma E(t) + r_3\sigma_2 Q(t) - (\sigma_1 + \delta_a)A(t), \\ f_5(t, S, \dots, R) = (1 - q)\gamma E(t) + r_1\sigma_1 A(t) - (\sigma_2 + \delta_q)Q(t), \\ f_6(t, S, \dots, R) = r_2\sigma_1 A(t) + r_4\sigma_2 Q(t) - (\sigma_3 + \delta_h)H(t), \\ f_7(t, S, \dots, R) = (1 - r_1 - r_2)\sigma_1 A(t) + (1 - r_3 - r_4)\sigma_2 Q(t) + \sigma_3 H(t). \end{cases} \quad (10)$$

109 By using (10), we have

$$\begin{aligned} {}^{ABC}D_t^\Phi \mathcal{A}(t) &= \Phi(t, \mathcal{A}(t)), \quad t \in [0, \tau], \quad 0 < \Phi \leq 1, \\ \mathcal{A}(0) &= \mathcal{A}_0. \end{aligned} \quad (11)$$

According to Lemma 2, (11) yields to

$$\mathcal{A}(t) = \mathcal{A}_0(t) + \left[ \Phi(t, \mathcal{A}(t)) - \Phi_0(t) \right] \frac{(1 - \Phi)}{\text{ABC}(\Phi)} + \frac{\Phi}{\Gamma(\Phi)\text{ABC}(\Phi)} \int_0^t (t - \omega)^{\Phi-1} \Phi(\omega, \mathcal{A}(\omega)) d\omega, \quad (12)$$

where

$$\mathcal{A}(t) = \begin{cases} S(t) \\ C(t) \\ E(t) \\ A(t) \\ Q(t) \\ H(t) \\ R(t) \end{cases}, \quad \mathcal{A}_0(t) = \begin{cases} S_0 \\ C_0 \\ E_0 \\ A_0 \\ Q_0 \\ H_0 \\ R_0 \end{cases}, \quad \Phi(t, \mathcal{A}(t)) = \begin{cases} f_1(t, S, \dots, R) \\ f_2(t, S, \dots, R) \\ f_3(t, S, \dots, R) \\ f_4(t, S, \dots, R) \\ f_5(t, S, \dots, R) \\ f_6(t, S, \dots, R) \\ f_7(t, S, \dots, R) \end{cases}, \quad \Phi_0(t) = \begin{cases} f_1(0, S_0, \dots, R_0) \\ f_2(0, S_0, \dots, R_0) \\ f_3(0, S_0, \dots, R_0) \\ f_4(0, S_0, \dots, R_0) \\ f_5(0, S_0, \dots, R_0) \\ f_6(0, S_0, \dots, R_0) \\ f_7(0, S_0, \dots, R_0) \end{cases} \quad (13)$$

110 Applying (12) and (13), the two operators  $\mathbf{F}$ ,  $\mathbf{G}$  are defined as:

$$\begin{aligned} \mathbf{F}(\mathcal{A}) &= \mathcal{A}_0(t) + \left[ \Phi(t, \mathcal{A}(t)) - \Phi_0(t) \right] \frac{(1 - \Phi)}{\text{ABC}(\Phi)}, \\ \mathbf{G}(\mathcal{A}) &= \frac{\Phi}{\Gamma(\Phi)\text{ABC}(\Phi)} \int_0^t (t - \omega)^{\Phi-1} \Phi(\omega, \mathcal{A}(\omega)) d\omega. \end{aligned} \quad (14)$$

111 We now state some basic axioms and Lipschitzian hypothesis for later showing the existence  
112 and uniqueness of solution:

**(H1)** There are  $C_\Phi, D_\Phi > 0$  such that

$$|\Phi(t, \mathcal{A}(t))| \leq C_\Phi |\mathcal{A}| + D_\Phi.$$

(H2) There is  $L_\Phi > 0$  such that  $\forall \mathcal{A}, \bar{\mathcal{A}} \in \mathbf{Z}$  it follows

$$|\Phi(t, \mathcal{A}) - \Phi(t, \bar{\mathcal{A}})| \leq L_\Phi \|\mathcal{A} - \bar{\mathcal{A}}\|.$$

113 **Theorem 2.** Under the hypothesis (H1, H2), the equation (12) possess at least one solu-  
 114 tion which implies that fractional model (9) possess the equal number of solutions only if  
 115  $\frac{(1-\Phi)}{\text{ABC}(\Phi)} L_\Phi < 1$ .

116 *Proof.* The proof is given in two parts as it can be seen below:

117 **Step I:** Consider  $\bar{\mathcal{A}} \in \mathbf{B}$ , where  $\mathbf{B} = \{\mathcal{A} \in \mathbf{Z} : \|\mathcal{A}\| \leq \rho, \rho > 0\}$  is closed and convex. The  
 118 operator  $\mathbf{F}$  provided in (14) gives

$$\begin{aligned} \|\mathbf{F}(\mathcal{A}) - \mathbf{F}(\bar{\mathcal{A}})\| &= \frac{(1-\Phi)}{\text{ABC}(\Phi)} \max_{t \in [0, \tau]} \left| \Phi(t, \mathcal{A}(t)) - \Phi(t, \bar{\mathcal{A}}(t)) \right|, \\ &\leq \frac{(1-\Phi)}{\text{ABC}(\Phi)} L_\Phi \|\mathcal{A} - \bar{\mathcal{A}}\|. \end{aligned} \quad (15)$$

119 Thus,  $\mathbf{F}$  is a contraction.

120 **Step-II:** We want  $\mathbf{G}$  to be relatively compact. Clearly, it is sufficient if  $\mathbf{G}$  is equicontinuous,  
 121 and bounded. Indeed,  $\mathbf{G}$  is continuous as  $\Phi$  is continuous and for all  $\mathcal{A} \in \mathbf{B}$ , one has

$$\begin{aligned} \|\mathbf{G}(\mathcal{A})\| &= \max_{t \in [0, \tau]} \left\| \frac{\Phi}{\Gamma(\Phi)\text{ABC}(\Phi)} \int_0^t (t-\omega)^{\Phi-1} \Phi(\omega, \mathcal{A}(\omega)) d\omega \right\|, \\ &\leq \frac{\Phi}{\Gamma(\Phi)\text{ABC}(\Phi)} \int_0^\tau (\tau-\omega)^{\Phi-1} |\Phi(\omega, \mathcal{A}(\omega))| d\omega, \\ &\leq \frac{\tau^\Phi}{\text{ABC}(\Phi)\Gamma(\Phi)} [C_\Phi \rho + D_\Phi]. \end{aligned} \quad (16)$$

122 Hence (16) shows the boundedness of  $\mathbf{G}$ . For equicontinuity we assume  $t_1 > t_2 \in [0, \tau]$ , so  
 123 that

$$\begin{aligned} |\mathbf{G}(\mathcal{A}(t_1)) - \mathbf{G}(\mathcal{A}(t_2))| &= \frac{\Phi}{\text{ABC}(\Phi)\Gamma(\Phi)} \left| \int_0^{t_1} (t_1-\omega)^{\Phi-1} \Phi(\omega, \mathcal{A}(\omega)) d\omega - \int_0^{t_2} (t_2-\omega)^{\Phi-1} \Phi(\omega, \mathcal{A}(\omega)) d\omega \right|, \\ &\leq \frac{[C_\Phi \rho + D_\Phi]}{\text{ABC}(\Phi)\Gamma(\Phi)} [t_1^\Phi - t_2^\Phi]. \end{aligned} \quad (17)$$

Right-hand side in (17) goes to zero as  $t_1 \rightarrow t_2$  and then

$$|\mathbf{G}(\mathcal{A}(t_1)) - \mathbf{G}(\mathcal{A}(t_2))| \rightarrow 0, \text{ as } t_1 \rightarrow t_2$$

124 since  $\mathbf{G}$  is continuous. Because of the boundedness and continuity of  $\mathbf{G}$ , then  $\mathbf{G}$  is uniformly  
 125 continuous and bounded. According to Arzelá-Ascoli theorem,  $\mathbf{G}$  is thus relatively compact  
 126 and therefore entirely continuous. Consequently, the integral equation (12) and also the  
 127 method have at least one solution.  $\square$

128 We now proceed to show the uniqueness of the solution.

129 **Theorem 3.** Assuming (H2), the equation (12) has a unique solution and this implies that  
 130 model (9) has a unique solution only if  $\left[ \frac{(1-\Phi)L_\Phi}{\text{ABC}(\Phi)} + \frac{\tau^\Phi L_\Phi}{\text{ABC}(\Phi)\Gamma(\Phi)} \right] < 1$ .

131 *Proof.* Consider  $\mathbf{T} : \mathbf{Z} \rightarrow \mathbf{Z}$  defined as

$$\mathbf{T}\mathcal{A}(t) = \mathcal{A}_0(t) + \left[ \Phi(t, \mathcal{A}(t)) - \Phi_0(t) \right] \frac{(1 - \Phi)}{\text{ABC}(\Phi)} + \frac{\Phi}{\text{ABC}(\Phi)\Gamma(\Phi)} \int_0^t (t - \omega)^{\Phi-1} \Phi(\omega, \mathcal{A}(\omega)) d\omega, \quad t \in [0, \tau]. \quad (18)$$

132 Given  $\mathcal{A}, \bar{\mathcal{A}} \in \mathbf{Z}$ , then one can take

$$\begin{aligned} \|\mathbf{T}\mathcal{A} - \mathbf{T}\bar{\mathcal{A}}\| &\leq \frac{(1 - \Phi)}{\text{ABC}(\Phi)} \max_{t \in [0, \tau]} \left| \Phi(t, \mathcal{A}(t)) - \Phi(t, \bar{\mathcal{A}}(t)) \right|, \\ &+ \frac{\Phi}{\Gamma(\Phi)\text{ABC}(\Phi)} \max_{t \in [0, \tau]} \left| \int_0^t (t - \omega)^{\Phi-1} \Phi(\omega, \mathcal{A}(\omega)) d\omega - \int_0^t (t - \omega)^{\Phi-1} \Phi(\omega, \bar{\mathcal{A}}(\omega)) d\omega \right|, \\ &\leq \Xi \|\mathcal{A} - \bar{\mathcal{A}}\|, \end{aligned} \quad (19)$$

133 where

$$\Xi = \left[ \frac{(1 - \Phi)L_\Phi}{\text{ABC}(\Phi)} + \frac{\tau^\Phi L_\Phi}{\Gamma(\Phi)\text{ABC}(\Phi)} \right]. \quad (20)$$

134 Thus,  $\mathbf{T}$  is a contraction. Therefore, the Eq. (12) possess a unique solution and so does  
135 model (9).

### 136 4.3. Derivation of Solution

137 To date, number of numerical approximation algorithms have been proposed to solve the  
138 various kind of models describing the real world problems. When we apply such algorithms  
139 the analysis of convergence and stability are two important aspects of method effectiveness.  
140 For solving our model, we are going to use Predictor-Corrector method which has been  
141 defined probably for all fractional-order operators. Here we implement this algorithm in  
142 the sense of Atangana-Baleanu operator for writing the solution of the proposed COVID-19  
143 model. For more information about this technique see reference [43]. We first recall Eq. (11)  
144 and consider

$$\begin{aligned} {}^{ABC}\mathbf{D}_t^\Phi \mathcal{A}(t) &= \Phi(t, \mathcal{A}(t)), \quad t \in [0, \tau], \quad 0 < \Phi \leq 1, \\ \mathcal{A}(0) &= \mathcal{A}_0. \end{aligned} \quad (21)$$

The fractional Volterra integral equation is stated as

$$\mathcal{A}_{i+1} = \mathcal{A}_0 + (1 - \Phi)\Phi(t_{i+1}, \mathcal{A}_{i+1}) + \frac{\Phi}{\Gamma(\Phi)} \int_0^{t_{i+1}} (t_{i+1} - s)^{\Phi-1} \Phi(s, \mathcal{A}(s)) ds. \quad (22)$$

According to the method proposed in [43] for  $\Phi \in [0, 1]$ ,  $0 \leq t \leq T$  and considering  $h = T/N$   
and  $t_n = nh$ , for  $n = 0, 1, 2, \dots, N \in \mathbb{Z}^+$ , the Corrector formula for the given system is

$$\mathcal{A}_{i+1} = \mathcal{A}_0 + \frac{\Phi h^\zeta}{\Gamma(\Phi + 2)} \left( a_{i+1, i+1} \Phi(t_{i+1}, \mathcal{A}_{i+1}^P) + \sum_{j=0}^i a_{i+1, j} \Phi(t_j, \mathcal{A}_j) \right) \quad (23)$$

145 where

$$a_{i+1, j} = \begin{cases} i^{\Phi+1} - (i - \Phi)(i + 1)^\Phi & \text{if } j = 0, \\ (i - j + 2)^{\Phi+1} + (i - j)^{\Phi+1} - 2(i - j + 1)^{\Phi+1} & \text{if } 1 \leq j \leq i, \\ 1, & j = i + 1. \end{cases} \quad (24)$$

and

$$a_{i+1,i+1} = 1 + \frac{(1 - \Phi)\Gamma(\Phi + 2)}{\Phi h^\Phi}.$$

The predictor formula is attained as

$$\mathcal{A}_{i+1}^P = \mathcal{A}_0 + \frac{h^\Phi}{\Gamma(\Phi)} \sum_{j=0}^i b_{i+1,j} \Phi(t_j, \mathcal{A}_j), \quad (25)$$

146 where

$$b_{i+1,j} = \begin{cases} -(i-j)^\Phi + (i-j+1)^\Phi, & j = 0, \dots, i-1, \\ 1 + \frac{(1-\Phi)\Gamma(\Phi)}{h^\Phi}, & j = i. \end{cases} \quad (26)$$

Hence the corrector formulae for the given model (9) are stated as

$$\begin{aligned}
S_{i+1} &= S_0 + \frac{\Phi h^\Phi}{\Gamma(\Phi + 2)} \left( (a_{i+1,i+1})f_1(t_{i+1}, S_{i+1}^P, C_{i+1}^P, E_{i+1}^P, A_{i+1}^P, Q_{i+1}^P, H_{i+1}^P, R_{i+1}^P) \right. \\
&\quad \left. + \sum_{j=0}^i (a_{i+1,j})f_1(t_i, S_j, C_j, E_j, A_j, Q_j, H_j, R_j) \right), \\
C_{i+1} &= C_0 + \frac{\Phi h^\Phi}{\Gamma(\Phi + 2)} \left( (a_{i+1,i+1})f_2(t_{i+1}, S_{i+1}^P, C_{i+1}^P, E_{i+1}^P, A_{i+1}^P, Q_{i+1}^P, H_{i+1}^P, R_{i+1}^P) \right. \\
&\quad \left. + \sum_{j=0}^i (a_{i+1,j})f_2(t_i, S_j, C_j, E_j, A_j, Q_j, H_j, R_j) \right), \\
E_{i+1} &= E_0 + \frac{\Phi h^\Phi}{\Gamma(\Phi + 2)} \left( (a_{i+1,i+1})f_3(t_{i+1}, S_{i+1}^P, C_{i+1}^P, E_{i+1}^P, A_{i+1}^P, Q_{i+1}^P, H_{i+1}^P, R_{i+1}^P) \right. \\
&\quad \left. + \sum_{j=0}^i (a_{i+1,j})f_3(t_i, S_j, C_j, E_j, A_j, Q_j, H_j, R_j) \right), \\
A_{i+1} &= A_0 + \frac{\Phi h^\Phi}{\Gamma(\Phi + 2)} \left( (a_{i+1,i+1})f_4(t_{i+1}, S_{i+1}^P, C_{i+1}^P, E_{i+1}^P, A_{i+1}^P, Q_{i+1}^P, H_{i+1}^P, R_{i+1}^P) \right. \\
&\quad \left. + \sum_{j=0}^i (a_{i+1,j})f_4(t_i, S_j, C_j, E_j, A_j, Q_j, H_j, R_j) \right), \\
Q_{i+1} &= Q_0 + \frac{\Phi h^\Phi}{\Gamma(\Phi + 2)} \left( (a_{i+1,i+1})f_5(t_{i+1}, S_{i+1}^P, C_{i+1}^P, E_{i+1}^P, A_{i+1}^P, Q_{i+1}^P, H_{i+1}^P, R_{i+1}^P) \right. \\
&\quad \left. + \sum_{j=0}^i (a_{i+1,j})f_5(t_i, S_j, C_j, E_j, A_j, Q_j, H_j, R_j) \right), \\
H_{i+1} &= H_0 + \frac{\Phi h^\Phi}{\Gamma(\Phi + 2)} \left( (a_{i+1,i+1})f_6(t_{i+1}, S_{i+1}^P, C_{i+1}^P, E_{i+1}^P, A_{i+1}^P, Q_{i+1}^P, H_{i+1}^P, R_{i+1}^P) \right. \\
&\quad \left. + \sum_{j=0}^i (a_{i+1,j})f_6(t_i, S_j, C_j, E_j, A_j, Q_j, H_j, R_j) \right), \\
R_{i+1} &= R_0 + \frac{\Phi h^\Phi}{\Gamma(\Phi + 2)} \left( (a_{i+1,i+1})f_7(t_{i+1}, S_{i+1}^P, C_{i+1}^P, E_{i+1}^P, A_{i+1}^P, Q_{i+1}^P, H_{i+1}^P, R_{i+1}^P) \right. \\
&\quad \left. + \sum_{j=0}^i (a_{i+1,j})f_7(t_i, S_j, C_j, E_j, A_j, Q_j, H_j, R_j) \right),
\end{aligned} \tag{27}$$

where

$$\begin{aligned}
S_{i+1}^P &= S_0 + \frac{h^\Phi}{\Gamma(\Phi)} \sum_{j=0}^i b_{i+1,j} f_1(t_j, S_j, C_j, E_j, A_j, Q_j, H_j, R_j), \\
C_{i+1}^P &= C_0 + \frac{h^\Phi}{\Gamma(\Phi)} \sum_{j=0}^i b_{i+1,j} f_2(t_j, S_j, C_j, E_j, A_j, Q_j, H_j, R_j), \\
E_{i+1}^P &= E_0 + \frac{h^\Phi}{\Gamma(\Phi)} \sum_{j=0}^i b_{i+1,j} f_3(t_j, S_j, C_j, E_j, A_j, Q_j, H_j, R_j), \\
A_{i+1}^P &= A_0 + \frac{h^\Phi}{\Gamma(\Phi)} \sum_{j=0}^i b_{i+1,j} f_4(t_j, S_j, C_j, E_j, A_j, Q_j, H_j, R_j), \\
Q_{i+1}^P &= Q_0 + \frac{h^\Phi}{\Gamma(\Phi)} \sum_{j=0}^i b_{i+1,j} f_5(t_j, S_j, C_j, E_j, A_j, Q_j, H_j, R_j), \\
H_{i+1}^P &= H_0 + \frac{h^\Phi}{\Gamma(\Phi)} \sum_{j=0}^i b_{i+1,j} f_6(t_j, S_j, C_j, E_j, A_j, Q_j, H_j, R_j), \\
R_{i+1}^P &= R_0 + \frac{h^\Phi}{\Gamma(\Phi)} \sum_{j=0}^i b_{i+1,j} f_7(t_j, S_j, C_j, E_j, A_j, Q_j, H_j, R_j).
\end{aligned} \tag{28}$$

147 *4.3.1. Stability of the method*

148 **Theorem 4.** *The given algorithm (27)-(28) is conditionally stable.*

Proof. Let  $\tilde{\mathcal{A}}_0, \tilde{\mathcal{A}}_j$  ( $j = 0, \dots, i+1$ ) and  $\tilde{\mathcal{A}}_{i+1}^P$  ( $i = 0, \dots, N-1$ ) be perturbations of  $\mathcal{A}_0, \mathcal{A}_j$  and  $\mathcal{A}_{i+1}^P$ , respectively. Then, the perturbation equations derived by using Eqs. (27) and (28) are given below

$$\tilde{\mathcal{A}}_{i+1}^P = \tilde{\mathcal{A}}_0 + \frac{h^\Phi}{\Gamma(\Phi)} \sum_{j=0}^i b_{i+1,j} (\Phi(t_j, \mathcal{A}_j + \tilde{\mathcal{A}}_j) - \Phi(t_j, \mathcal{A}_j)), \tag{29}$$

$$\begin{aligned}
\tilde{\mathcal{A}}_{i+1} &= \tilde{\mathcal{A}}_0 + \frac{\Phi h^\Phi}{\Gamma(\Phi+2)} \left( a_{i+1,i+1} (\Phi(t_{i+1}, \mathcal{A}_{i+1}^P + \tilde{\mathcal{A}}_{i+1}^P) - \Phi(t_{i+1}, \mathcal{A}_{i+1}^P)) \right. \\
&\quad \left. + \sum_{j=0}^i a_{i+1,j} (\Phi(t_j, \mathcal{A}_j + \tilde{\mathcal{A}}_j) - \Phi(t_j, \mathcal{A}_j)) \right).
\end{aligned} \tag{30}$$

According to the Lipschitz condition, we get

$$|\tilde{\mathcal{A}}_{i+1}| \leq \Phi_0 + \frac{\Phi h^\Phi M}{\Gamma(\Phi+2)} \left( a_{i+1,i+1} |\tilde{\mathcal{A}}_{i+1}^P| + \sum_{j=1}^i a_{j,i+1} |\tilde{\mathcal{A}}_j| \right), \tag{31}$$

where  $\Phi_0 = \max_{0 \leq i \leq N} \{ |\tilde{\mathcal{A}}_0| + \frac{\Phi h^\Phi M a_{i,0}}{\Gamma(\Phi+2)} |\tilde{\mathcal{A}}_0| \}$ . Also, from Eq.(3.18) in [41] we write

$$|\tilde{\mathcal{A}}_{i+1}^P| \leq \eta_0 + \frac{h^\Phi M}{\Gamma(\Phi)} \sum_{j=1}^i b_{j,i+1} |\tilde{\mathcal{A}}_j|, \tag{32}$$

where  $\eta_0 = \max_{0 \leq i \leq N} \{ |\tilde{\mathcal{A}}_0| + \frac{h^\Phi M b_{n,0}}{\Gamma(\Phi)} |\tilde{\mathcal{A}}_0| \}$ . Substituting  $|\tilde{\mathcal{A}}_{i+1}^P|$  from Eq. (32) into Eq. (31) reads as follows

$$\begin{aligned} |\tilde{\mathcal{A}}_{i+1}| &\leq \gamma_0 + \frac{\Phi h^\Phi M}{\Gamma(\Phi + 2)} \sum_{j=1}^i \left( a_{i+1,j} + \frac{h^\Phi M a_{i+1,i+1} b_{i+1,j}}{\Gamma(\Phi)} \right) |\tilde{\mathcal{A}}_j| \\ &\leq \gamma_0 + \frac{\Phi h^\Phi M C_{\Phi,2}}{\Gamma(\Phi + 2)} \sum_{j=1}^i (i+1-j)^{\Phi-1} |\tilde{\mathcal{A}}_j|, \end{aligned} \quad (33)$$

149 where  $\gamma_0 = \max \{ \Phi_0 + \frac{\Phi h^\Phi M a_{i+1,i+1}}{\Gamma(\Phi + 2)} \eta_0 \}$ .  $C_{\Phi,2} > 0$  is constant and only depends on  $\Phi$  (see  
150 Lemma 3) and  $h$  is surmised to be small enough. From Lemma 4 we get  $|\tilde{\mathcal{A}}_{i+1}| \leq C\gamma_0$ , which  
151 finishes the requirement.  $\square$

#### 152 4.4. Graphical justifications

153 Now we plot the fractional-order solution by considering the parameter values simulated  
154 in Table 2. This time we used *Mathematica* software. In figure 6, we plotted the susceptible  
155 population  $S(t)$  and in figure 7, the plot of confined individuals  $C(t)$  is given. We can  
156 observe that when time increases then the population of susceptible individuals is decreasing  
157 which is a good indication for Argentina. In figure 8, we plotted the nature of exposed  
158 individuals  $E(t)$ . We notice that when the fractional order values decreases then the peak  
159 of  $E(t)$  shifts to the next month. At fractional-order  $\Phi = 1$ , peak occurs at near around  
160 1 July 2020 and when we shifted to lower values of fractional order then we receive the peak  
161 near around 31 July 2020, at  $\Phi = 0.95$ , near around 29 Sep 2020, at  $\Phi = 0.85$ , and near  
162 around 28 Dec 2020 when  $\Phi = 0.75$ . This shows how the fractional-order derivatives give us  
163 more varieties to predict the real data structure for future. Similarly, in figure 9, we plotted  
164 the nature of asymptomatic individuals  $A(t)$  where we can see that at fractional-order  $\Phi = 1$ ,  
165 peak occurs at near around 31 July 2020 and when we shift to lower values of fractional order  
166 then we receive the peak near around 15 Aug 2020 at  $\Phi = 0.95$ , near around 15 Oct 2020 at  
167  $\Phi = 0.85$ , and near around 27 Jan 2021 when  $\Phi = 0.75$ . In this series, figure 10 plotted the  
168 nature of quarantined individuals  $Q(t)$  where different peaks can be seen. Here at fractional-  
169 order  $\Phi = 1$ , peak occurs at near around 15 July 2020 and when we shifted to lower values of  
170 fractional order then we receive the peak near around 31 July 2020 at  $\Phi = 0.95$ , near around  
171 29 Sep 2020 at  $\Phi = 0.85$ , and near around 28 Dec 2020 at  $\Phi = 0.75$ . Figure 11 described the  
172 behaviour of hospitalized individuals  $H(t)$  for the given time range. Here at fractional-order  
173  $\Phi = 1$ , peak occurs at near around 15 July 2020 and when we shifted to lower values of  
174 fractional order then we receive the peak near around 31 July 2020 at  $\Phi = 0.95$ , near around  
175 29 Sep 2020 at  $\Phi = 0.85$ , and near around 30 Dec 2020 at  $\Phi = 0.75$ . At the last, figure 12  
176 shows the nature of recovered individuals  $R(t)$  versus given time range.

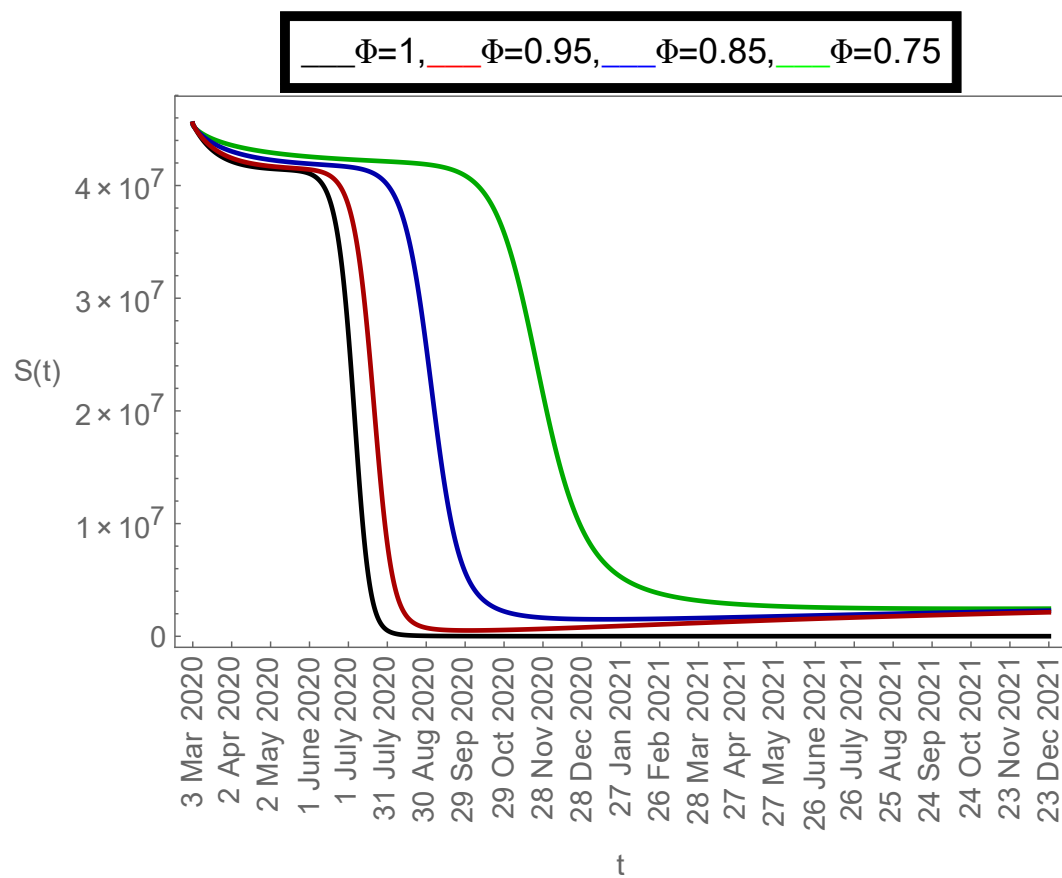


Figure 6: Dynamics of  $S(t)$  population

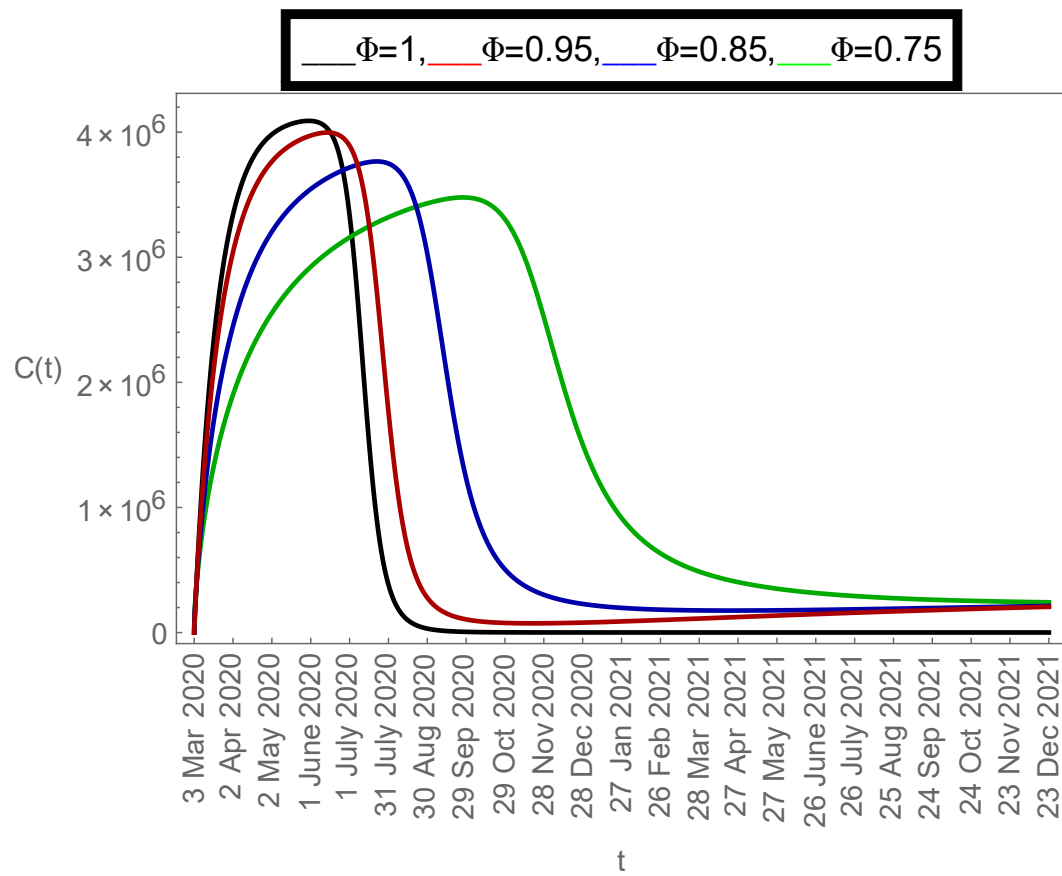


Figure 7: Dynamics of  $C(t)$  population

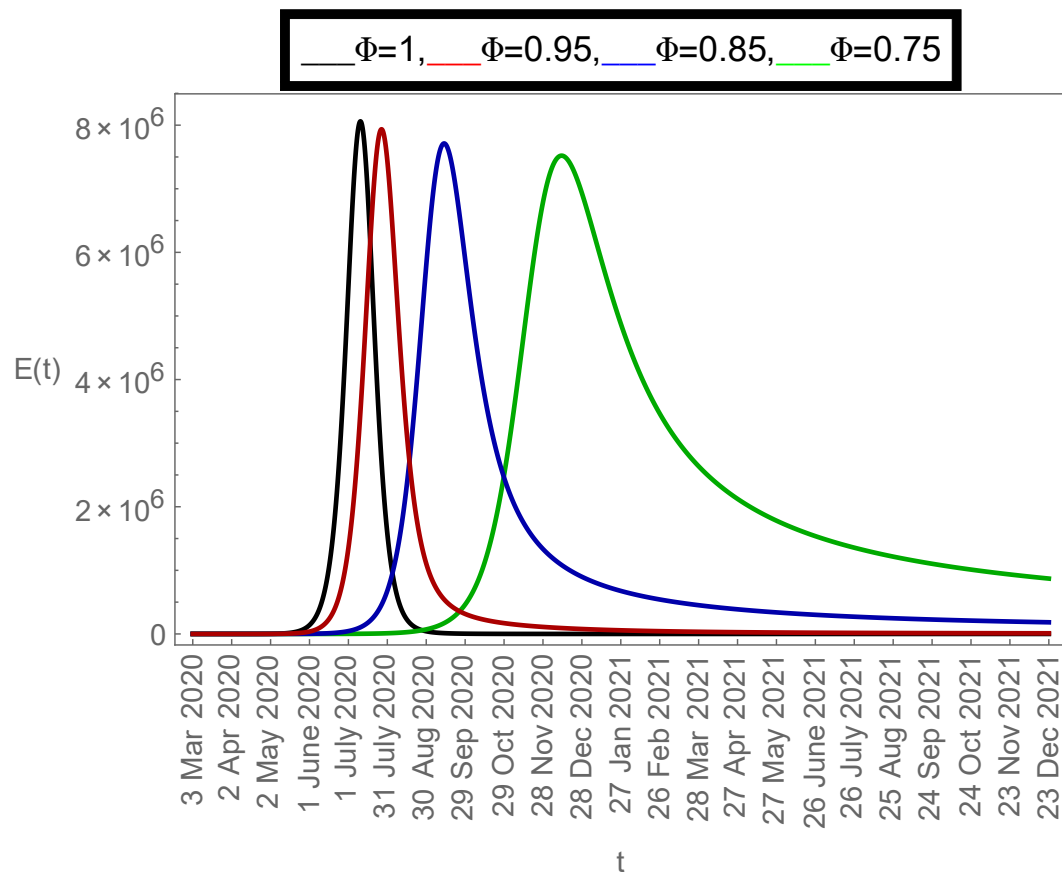


Figure 8: Dynamics of  $E(t)$  population

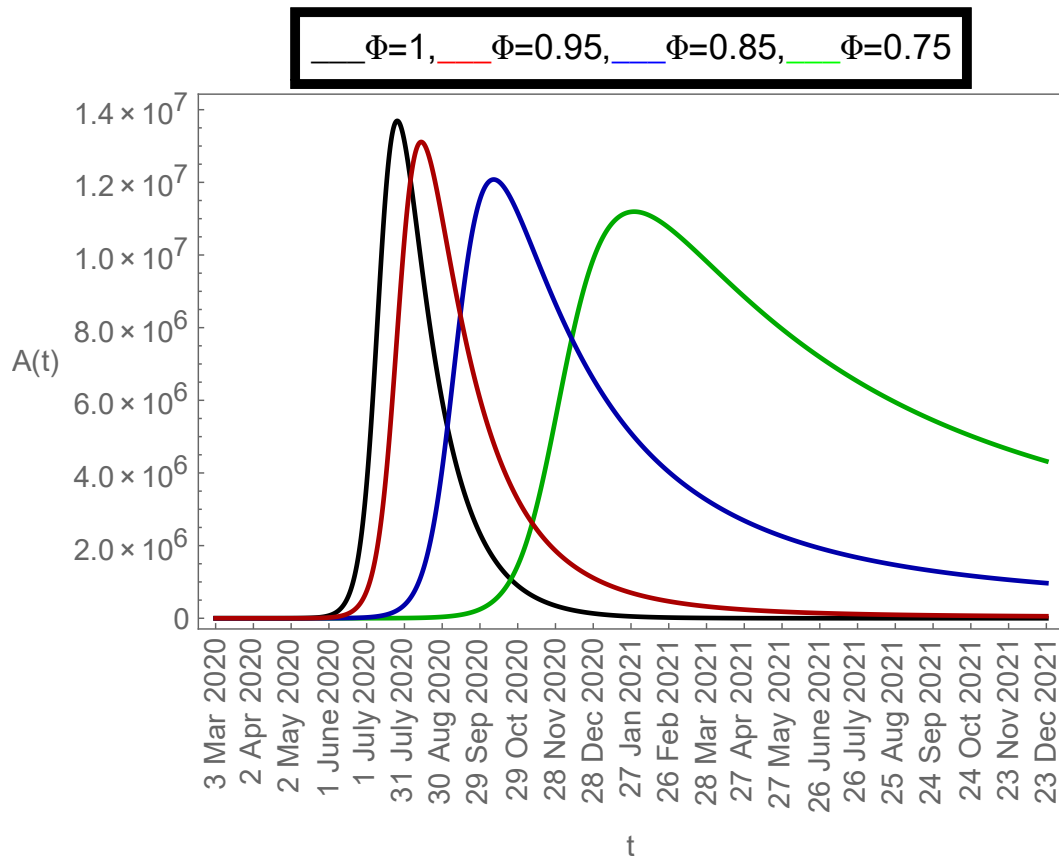


Figure 9: Dynamics of  $A(t)$  population

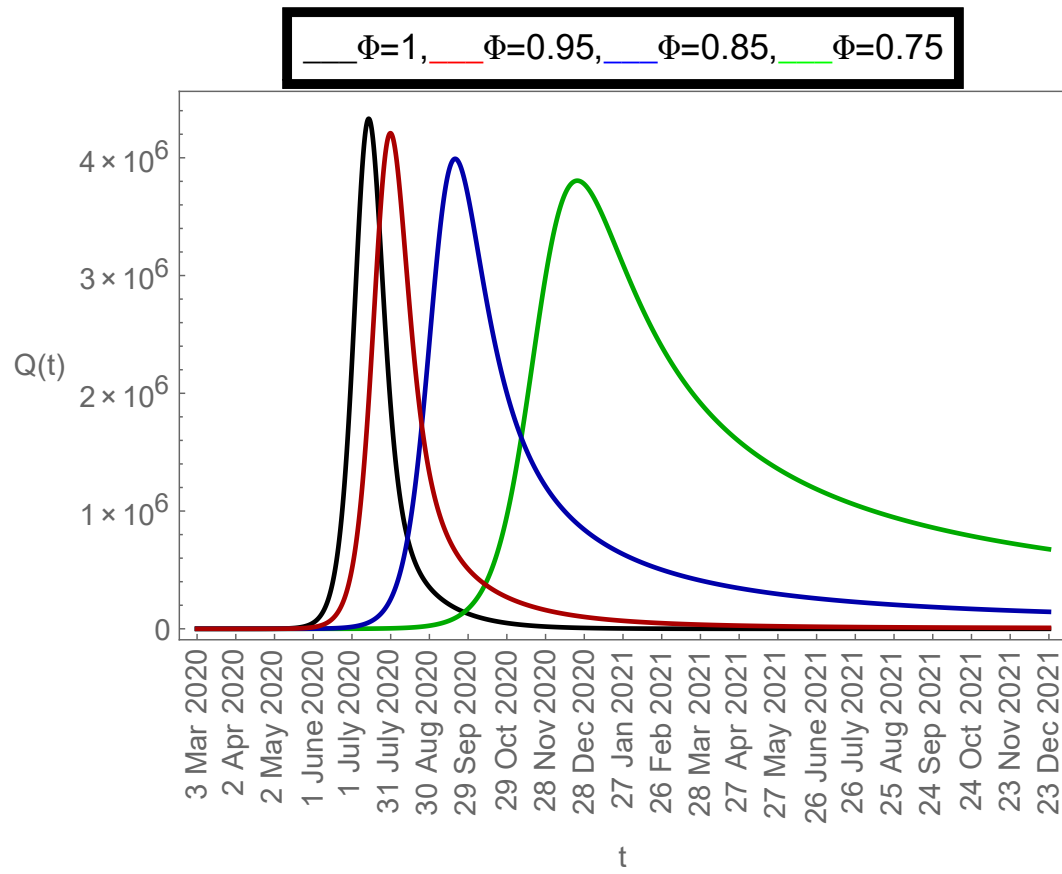


Figure 10: Dynamics of  $Q(t)$  population

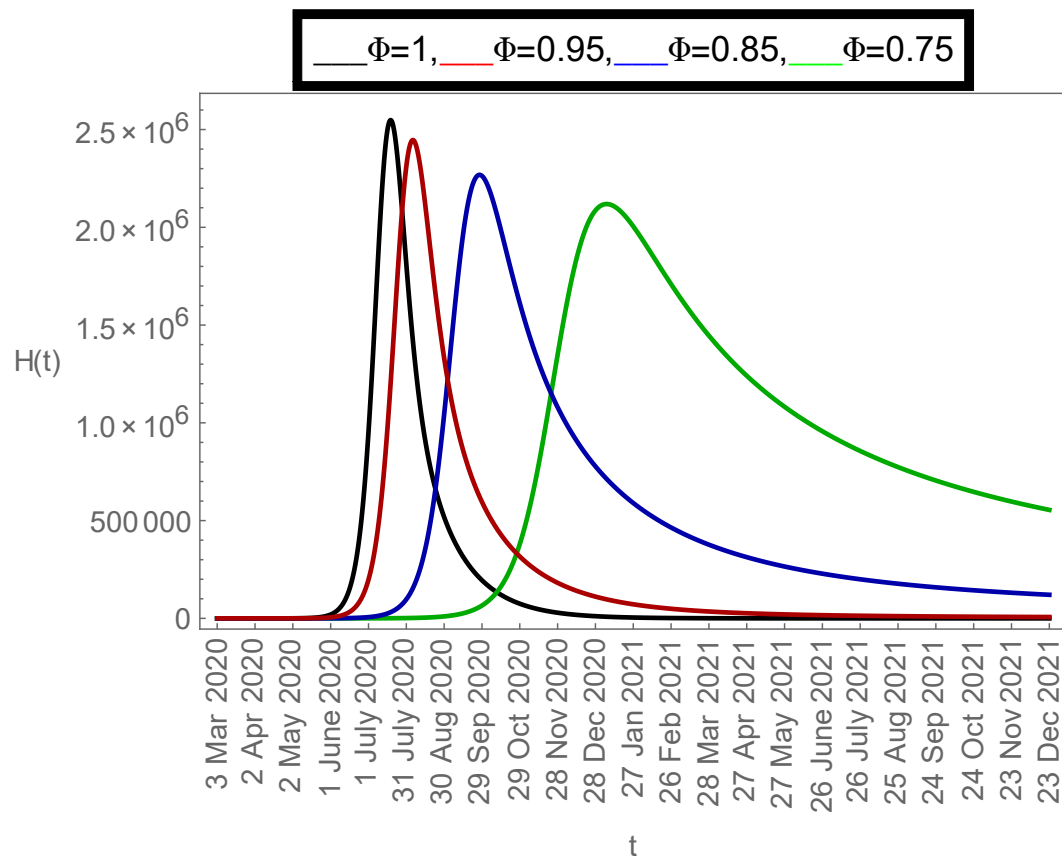


Figure 11: Dynamics of  $H(t)$  population

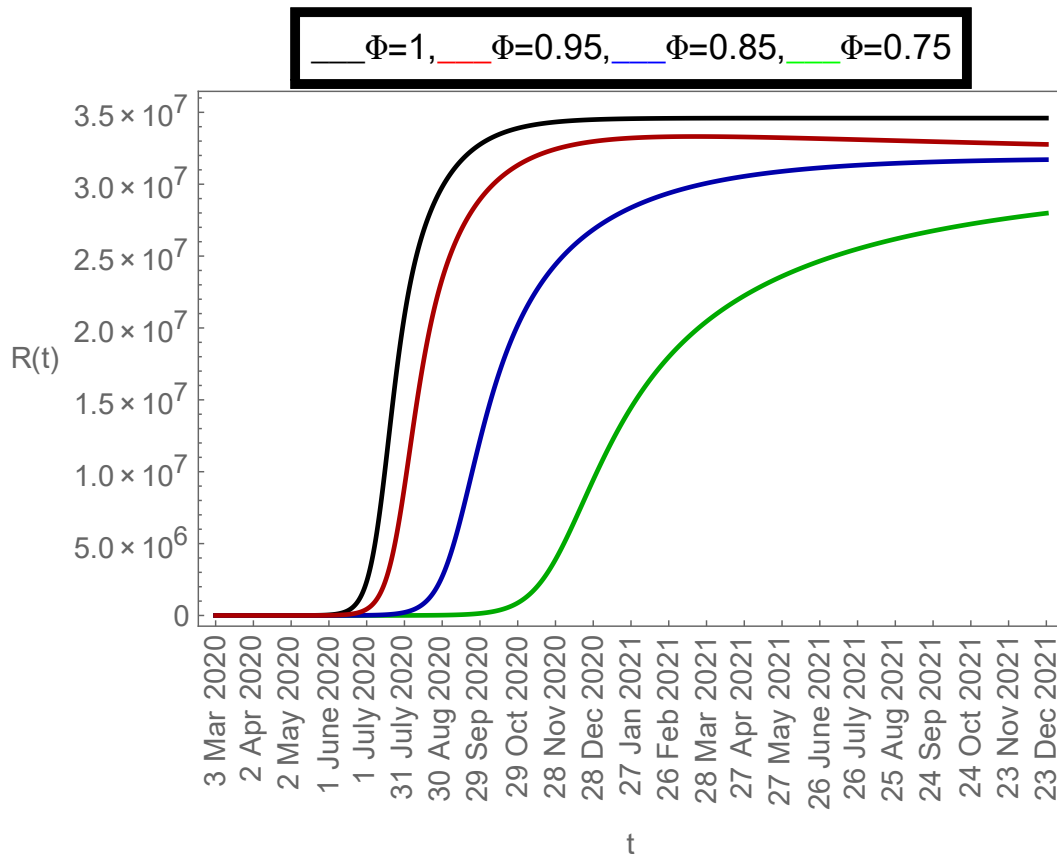


Figure 12: Dynamics of  $R(t)$  population

177 All above given graphs confirm that the COVID-19 disease is under control in Argentina  
 178 at this time stage (based on the given data simulations). However, it is well-known that  
 179 if the population do not follow all health care measures then the situation may change in  
 180 the future. The given fractional-order model under Mittag-Leffler kernel gives very well  
 181 outputs to predict the dynamics of COVID-19 in Argentina. We used different fractional-  
 182 order values for the comparisons because an integer-order model can provide a single peak  
 183 prediction which does not show the varieties in the predictions. Different fractional-order  
 184 values give various predictions which can take place in future more accurately. This is the  
 185 ultimate advantage to use fractional derivatives.

## 186 5. Conclusion

187 In our observations, we have explored the dynamics of most deathly disease of this decade,  
 188 COVID-19 in Argentina by considering parameter values based on real data. Firstly, we  
 189 considered the reported cases of this virus from *March* 03, 2020 to *March* 29, 2021 and  
 190 projected it for the future time period by using our models. We proposed a Atangana-Baleanu  
 191 type fractional order model and solved it employing Predictor-Corrector (P-C) algorithm.  
 192 After analysing biological nature of this virus, we formulated a mathematical structure to  
 193 define its dynamics. We used a well-known effective optimization method based on renowned  
 194 trust-region-reflective (TRR) scheme to perform the model calibration. We plotted the  
 195 real cases of COVID-19 and fitted our integer-order model with the data along with the  
 196 calculation of basic reproductive number. In the simulations of fractional-order analysis,

197 first we proved the existence of unique solution and then wrote the solution of the model  
198 along with the stability of the given P-C method. We performed separate graphs for every  
199 model classes at different fractional-order values to predict the future dynamics of the virus  
200 in Argentina. At the end, we conclude that this virus is under control in Argentina for  
201 future under the given data range and all health care measures. In the future, the given  
202 estimated parameter values and models can be used to provide further predictions on the  
203 transmission of this virus. The proposed mathematical model is effective and trustable for  
204 future uses. **Not only from a mathematical point of view but also from a medical perspective,**  
205 **this research study may become very useful for the medical authorities to simulate the**  
206 **outbreaks of COVID-19 in Argentina for future time period. Still, all the peak predictions**  
207 **which have been simulated in this paper, do not claim with 100% surety that the dynamics**  
208 **of COVID-19 will be as it is. It may differ cause of many health care measures (like, isolation**  
209 **strategies, hospital facilities, and vaccine availability, etc.) which have been mentioned in**  
210 **this study.**

### 211 **Availability of data and materials**

212 All the data is included in the paper.

### 213 **Conflict of interest**

214 This research does not have any conflict of interest. There is funding available to support  
215 this study.

### 216 **Funding**

217 The third author is supported by MICINN and FEDER, Project PID2019-105011GB-I00.

### 218 **Authors contribution statement**

219 **Pushendra Kumar:** Conceptualization, Data curation, Investigation, Methodology,  
220 Formal analysis, Visualization, Resources, Project administration, Writing-original draft.

221 **Vedat Suat Erturk:** Supervision, Software, Investigation, Validation, Visualization,  
222 Writing-review & editing.

223 **Marina Murillo-Arcila:** Conceptualization, Investigation, Supervision, Writing-review &  
224 editing.

225 **Ramashis Banerjee** Writing-review & editing.

226 **A. Manickam** Writing-review & editing.

### 227 **Acknowledgements**

228 Not applicable.

## 229 References

- 230 [1] Z. Wu, J. M. McGoogan, Characteristics of and important lessons from the coronavirus  
231 disease 2019 (covid-19) outbreak in china: summary of a report of 72 314 cases from  
232 the chinese center for disease control and prevention, *Jama* 323 (13) (2020) 1239–1242.
- 233 [2] C. Zhang, W. Zheng, X. Huang, E. W. Bell, X. Zhou, Y. Zhang, Protein structure and  
234 sequence reanalysis of 2019-ncov genome refutes snakes as its intermediate host and the  
235 unique similarity between its spike protein insertions and hiv-1, *Journal of proteome  
236 research* 19 (4) (2020) 1351–1360.
- 237 [3] D. S. Hui, E. I. Azhar, T. A. Madani, F. Ntoumi, R. Kock, O. Dar, G. Ippolito, T. D.  
238 Mchugh, Z. A. Memish, C. Drosten, et al., The continuing 2019-ncov epidemic threat  
239 of novel coronaviruses to global healththe latest 2019 novel coronavirus outbreak in  
240 wuhan, china, *International journal of infectious diseases* 91 (2020) 264–266.
- 241 [4] A. A. Elfiky, S. M. Mahdy, W. M. Elshemey, Quantitative structure-activity relationship  
242 and molecular docking revealed a potency of anti-hepatitis c virus drugs against human  
243 corona viruses, *Journal of medical virology* 89 (6) (2017) 1040–1047.
- 244 [5] X. Li, J. Zai, Q. Zhao, Q. Nie, Y. Li, B. T. Foley, A. Chaillon, Evolutionary history,  
245 potential intermediate animal host, and cross-species analyses of sars-cov-2, *Journal of  
246 medical virology* 92 (6) (2020) 602–611.
- 247 [6] D. Wrapp, N. Wang, K. S. Corbett, J. A. Goldsmith, C.-L. Hsieh, O. Abiona, B. S.  
248 Graham, J. S. McLellan, Cryo-em structure of the 2019-ncov spike in the prefusion  
249 conformation, *Science* 367 (6483) (2020) 1260–1263.
- 250 [7] J. Villar, H. Zhang, A. S. Slutsky, Lung repair and regeneration in ards: role of pecam1  
251 and wnt signaling, *Chest* 155 (3) (2019) 587–594.
- 252 [8] H. Wang, S. Ma, The cytokine storm and factors determining the sequence and severity  
253 of organ dysfunction in multiple organ dysfunction syndrome, *The American journal of  
254 emergency medicine* 26 (6) (2008) 711–715.
- 255 [9] Y. Wan, J. Shang, R. Graham, R. S. Baric, F. Li, Receptor recognition by the novel  
256 coronavirus from wuhan: an analysis based on decade-long structural studies of sars  
257 coronavirus, *Journal of virology* 94 (7).
- 258 [10] N. L.-S. Tang, P. K.-S. Chan, C.-K. Wong, K.-F. To, A. K.-L. Wu, Y.-M. Sung, D. S.-C.  
259 Hui, J. J.-Y. Sung, C. W.-K. Lam, Early enhanced expression of interferon-inducible  
260 protein-10 (cxcl-10) and other chemokines predicts adverse outcome in severe acute  
261 respiratory syndrome, *Clinical chemistry* 51 (12) (2005) 2333–2340.
- 262 [11] J. Gu, C. Korteweg, Pathology and pathogenesis of severe acute respiratory syndrome,  
263 *The American journal of pathology* 170 (4) (2007) 1136–1147.
- 264 [12] J. C. Ho, K. N. Chan, W. H. Hu, W. K. Lam, L. Zheng, G. L. Tipoe, J. Sun, R. Leung,  
265 K. W. Tsang, The effect of aging on nasal mucociliary clearance, beat frequency, and  
266 ultrastructure of respiratory cilia, *American journal of respiratory and critical care  
267 medicine* 163 (4) (2001) 983–988.

- 268 [13] P. Kumar, V. S. Erturk, M. Murillo-Arcila, A new fractional mathematical modelling  
269 of covid-19 with the availability of vaccine, *Results in Physics* (2021) 104213.
- 270 [14] P. Kumar, V. S. Erturk, A case study of covid-19 epidemic in india via new gener-  
271 alised caputo type fractional derivatives, *Mathematical Methods in the Applied Sciences*  
272 (2021) 1–14.
- 273 [15] P. Kumar, V. S. Erturk, H. Abboubakar, K. S. Nisar, Prediction studies of the epi-  
274 demic peak of coronavirus disease in brazil via new generalised caputo type fractional  
275 derivatives, *Alexandria Engineering Journal*.
- 276 [16] K. N. Nabi, P. Kumar, V. S. Erturk, Projections and fractional dynamics of covid-19  
277 with optimal control strategies, *Chaos, Solitons & Fractals* (2021) 110689.
- 278 [17] V. S. Erturk, P. Kumar, Solution of a covid-19 model via new generalized caputo-type  
279 fractional derivatives, *Chaos, Solitons & Fractals* (2020) 110280.
- 280 [18] W. Gao, P. Veerasha, H. M. Baskonus, D. Prakasha, P. Kumar, A new study of unre-  
281 ported cases of 2019-ncov epidemic outbreaks, *Chaos, Solitons & Fractals* (2020) 109929.
- 282 [19] P. Kumar, V. Suat Erturk, The analysis of a time delay fractional covid-19 model via  
283 caputo type fractional derivative, *Mathematical Methods in the Applied Sciences* (2020)  
284 1–14.
- 285 [20] A. Atangana, S. İ. Araz, Modeling and forecasting the spread of covid-19 with stochastic  
286 and deterministic approaches: Africa and europe, *Advances in Difference Equations*  
287 2021 (1) (2021) 1–107.
- 288 [21] A. Atangana, Modelling the spread of covid-19 with new fractal-fractional operators:  
289 can the lockdown save mankind before vaccination?, *Chaos, Solitons & Fractals* 136  
290 (2020) 109860.
- 291 [22] A. Atangana, S. İ. Araz, Mathematical model of covid-19 spread in turkey and south  
292 africa: theory, methods, and applications, *Advances in Difference Equations* 2020 (1)  
293 (2020) 1–89.
- 294 [23] A. Atangana, et al., A novel covid-19 model with fractional differential operators with  
295 singular and non-singular kernels: Analysis and numerical scheme based on newton  
296 polynomial, *Alexandria Engineering Journal* 60 (4) (2021) 3781–3806.
- 297 [24] H. Bulut, D. Kumar, J. Singh, R. Swroop, H. M. Baskonus, Analytic study for a frac-  
298 tional model of hiv infection of cd4+ t lymphocyte cells. *math, Nat. Sci* 2 (1) (2018)  
299 33–43.
- 300 [25] Y.-H. Zhoua, Y. Yang, H. Zhang, Stability of non-monotone critical waves in a popu-  
301 lation dynamics model with spatio-temporal delay, *Mathematics in Natural Science* 2  
302 (2018) 8–23.
- 303 [26] S. S. Musa, S. Qureshi, S. Zhao, A. Yusuf, U. T. Mustapha, D. He, Mathematical  
304 modeling of covid-19 epidemic with effect of awareness programs, *Infectious Disease*  
305 *Modelling* 6 (2021) 448–460.

- 306 [27] Z. Memon, S. Qureshi, B. R. Memon, Assessing the role of quarantine and isolation as  
307 control strategies for covid-19 outbreak: a case study, *Chaos, Solitons & Fractals* 144  
308 (2021) 110655.
- 309 [28] P. Kumar, V. Suat Ertürk, K. S. Nisar, Fractional dynamics of huanglongbing trans-  
310 mission within a citrus tree, *Mathematical Methods in the Applied Sciences*.
- 311 [29] P. Kumar, V. S. Erturk, M. Murillo-Arcila, A complex fractional mathematical modeling  
312 for the love story of layla and majnun, *Chaos, Solitons & Fractals* 150 (2021) 111091.
- 313 [30] P. Kumar, V. S. Erturk, A. Yusuf, S. Kumar, Fractional time-delay mathematical mod-  
314 eling of oncolytic virotherapy, *Chaos, Solitons & Fractals* 150 (2021) 111123.
- 315 [31] H. Abboubakar, P. Kumar, V. S. Erturk, A. Kumar, A mathematical study of a tuber-  
316 culosis model with fractional derivatives, *International Journal of Modeling, Simulation,  
317 and Scientific Computing*.
- 318 [32] P. Kumar, N. A. Rangaig, H. Abboubakar, S. Kumar, A malaria model with caputo-  
319 fabrizio and atangana-baleanu derivatives, *International Journal of Modeling, Simula-  
320 tion, and Scientific Computing*.
- 321 [33] P. Kumar, V. S. Erturk, A. Yusuf, K. S. Nisar, S. F. Abdelwahab, A study on canine  
322 distemper virus (cdv) and rabies epidemics in the red fox population via fractional  
323 derivatives, *Results in Physics* (2021) 104281.
- 324 [34] P. Kumar, V. S. Erturk, H. Almusawa, Mathematical structure of mosaic disease using  
325 microbial biostimulants via caputo and atangana-baleanu derivatives, *Results in Physics*  
326 (2021) 104186.
- 327 [35] P. Kumar, V. S. Erturk, Environmental persistence influences infection dynamics for a  
328 butterfly pathogen via new generalised caputo type fractional derivative, *Chaos, Solitons  
329 & Fractals* 144 (2021) 110672.
- 330 [36] K. N. Nabi, H. Abboubakar, P. Kumar, Forecasting of covid-19 pandemic: From integer  
331 derivatives to fractional derivatives, *Chaos, Solitons & Fractals* (2020) 110283.
- 332 [37] P. Van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic  
333 equilibria for compartmental models of disease transmission, *Mathematical biosciences*  
334 180 (1-2) (2002) 29–48.
- 335 [38] K. N. Nabi, Forecasting covid-19 pandemic: A data-driven analysis, *Chaos, Solitons &  
336 Fractals* 139 (2020) 110046.
- 337 [39] Politologue.com, Coronavirus (covid19), [https://coronavirus.politologue.com/  
338 coronavirus-cameroun.CM](https://coronavirus.politologue.com/coronavirus-cameroun.CM) (Accessed 10/07/2020).
- 339 [40] A. Atangana, D. Baleanu, New fractional derivatives with nonlocal and non-singular  
340 kernel: theory and application to heat transfer model, arXiv preprint arXiv:1602.03408.
- 341 [41] C. Li, F. Zeng, The finite difference methods for fractional ordinary differential equa-  
342 tions, *Numerical Functional Analysis and Optimization* 34 (2) (2013) 149–179.

- 343 [42] E. Ahmed, A. El-Sayed, H. El-Saka, G. A. Ashry, On applications of ulam-hyers stability  
344 in biology and economics, arXiv preprint arXiv:1004.1354.
- 345 [43] D. Baleanu, A. Jajarmi, M. Hajipour, On the nonlinear dynamical systems within the  
346 generalized fractional derivatives with mittag-leffler kernel, Nonlinear dynamics 94 (1)  
347 (2018) 397–414.