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A fractional-order multi-vaccination model for COVID-19 with non-singular kernel

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Abstract This work examines the impact of multiple vaccination strategies on the dynamics of COVID-19 in a population using the Atangana-Baleanu derivative. The existence and uniqueness of solution of the model is proven using Banach’s fixed point theorem. Local and global asymptotic stability of the equilibria of the model is also proven (under some conditions). Conditions for the existence of a unique or multiple equilibria are also derived and the model is shown to undergo backward bifurcation under certain scenarios. Using available data for the Pfizer, Moderna and Janssen vaccination programme for the city of Texas, United States of America from March 13, 2021 to June 29, 2021, the model is fitted using the three data sets. The three vaccination rates \( m_1 \), \( m_2 \) and \( m_3 \) corresponding to each vaccine as well as the effective contact rate for COVID-19 transmission, \( b_1 \), are estimated. Simulations of the model under different vaccination strategies are carried out. The results show that the three vaccination strategies not only cause significant reduction in the new asymptomatic and vaccinated symptomatic cases but also cause great decrease in the total number of vaccinated symptomatic individuals with severe COVID-19 illness.

1. Introduction

The new Coronavirus Disease 2019 (COVID-19), discovered first in Wuhan in December 2019 has spread rapidly across countries of the world like wildfire. As of 3rd August, 2021, the cumulative global cases stand at 198,993,405 with 4,237,546 deaths [1]. As of the same date, only 4,181,520,964 COVID-19 vaccines have been administered worldwide [1]. COVID-19 common symptoms include: cold-like illness, fever, fatigue, muscle pains, loss of taste or smell, shortness of breath, dry cough and sore throat. Recently, various strains of COVID-19 have emerged, including the “highly transmissible” and more deadly SARS-CoV-2 Delta variant, also known...
as lineage B.1.617.2 [2]. The World Health Organization (WHO) has described this variant as a variant of serious concern. The variant has been detected in more than 80 countries and is proposed to spread to more countries. The variant has also been associated with a surge in cases in countries where it is the dominant strain in circulation [2]. In addition to the various non-pharmaceutical control strategies against COVID-19, some of the vaccines that have gained emergency use authorization (EUA) by the United States Centers for Disease Control and Prevention (CDC) are: the Pfizer-BioNtech with 95% efficacy against symptomatic COVID-19, the Moderna vaccine with 94.5% efficacy and the Janssen vaccine, developed by Johnson & Johnson with 67% efficacy, and many others [3,4]. The vaccines have proven to be effective against SARS-CoV-2 infections, including asymptomatic infections and symptomatic cases, severe COVID-19 illness and deaths [4].

Recent trends in epidemiological research revealed that applying non-integer order differential equations is vital in obtaining good results for dynamical systems. The classical mathematical models of the integer-order derivatives have been greatly employed in studying infectious diseases [10–15, 16–19,46,27,28,23–25]. For instance, Oname et al. [9] considered and analyzed an integer-order model for the dynamics of Human papillomavirus and Chlamydia trachomatis using optimal control. Also, the authors in [14] considered an integer mathematical model for the dynamics of COVID-19 in Lagos, Nigeria. They examined the impact of various non-pharmaceutical interventions on the control of the disease. In [46], the authors considered a mathematical model for the co-dynamics of COVID-19 and Dengue in Brazil, with optimal control and cost-effectiveness analysis. Furthermore, Bonyah et al. [18] considered and analyzed a mathematical model for the co-infection of dengue fever and Zika virus. These models have major flaws in many cases. The reason is that the classical differential operator is based on the concept of rates of change, and that change is determined using just two points, which is a major limitation. These limitations have created a big vacuum for other methodologies to come up, such as fractional differential operators which involve both non-local and non-singular kernel and uses the power law function as its kernel.

Fractional derivatives have been used extensively in the literature to capture the effect of memory on the system dynamics [29,30]. In epidemiological modelling, fractional derivatives and fractional integrals are very significant, because they provide an excellent instrument for the description of memory and hereditary properties of various materials and processes [36]. This is in fact the fundamental merit of fractional calculus in comparison with integer order systems, in which such effects are abandoned. It is obvious to notice the advantages of fractional derivatives and integrals in the modelling biological, medical, mechanical and electrical properties of real life issues and in investigating the dynamics of infectious diseases [31–35,37,38] and the references therein. Particularly, Khan and Atangana [32] formulated a fractional order model for the dynamics of COVID-19 population dynamics. The model was fitted to the reported cases in Wuhan, China. The basic reproduction of the model was estimated from the fitting, and they showed that decreasing the fractional order value leads to decrease in infection cases. The author in [31] considered a delayed SIR epidemic model with Mittag-Leffler fractional derivative. He provided conditions for the existence and uniqueness of solution of the model. The author also proved the global asymptotic stability of the disease-free and the endemic equilibria using the Lyapunov direct method. Nwajeri and Oname [37] in their paper, considered a fractional order model for Human papillomavirus (HPV) and Chlamydia trachomatis (CT) co-infection in the presence of screening and treatment. They showed that for lower fractional order values, the CT only treatment strategy has more positive population level impact on the number of infected individuals with HPV.

The concept of non-singular kernel fractional derivatives and integrals such as Atangana-Baleanu and Caputo-Fabrizio have recently shown to be more productive in description of dynamic models compared to the well-known singular kernel fractional derivatives like Caputo, Riemann-Liouville, Grunwald-Letnikov etc. These two fractional derivatives are widely applicable in the solution of real-world problem: in physics, biology, chemistry, medical, mechanics, sciences, and engineering, see in [20–22,26,39,40,43,45]. Fractional differential operators with singular kernel (Riemann-Liouville derivative) use exponential decay law as kernel while fractional differential operators with non-local and non-singular kernel (Atangana-Baleanu derivative) use the generalized Mittag-Leffler function as kernel [8]. The non-singular kernel is easier to use in theoretical analysis, numerical calculations and best describe the real world problem in mathematical models. This is one of the reasons we deem non-singular kernel fractional derivatives fit and riveting for this particular subject matter.

In our study, we present a mathematical model that investigates the impact of multiple COVID-19 vaccinations on the dynamics of the disease in a population. The model is novel and has not been considered before. We analyze the model using the Atangana-Baleanu derivative, which is the most preferred fractional derivative in modeling biological processes, as it captures the effect of memory due to its non-local and non-singular kernel. The rest of the paper is organized as follows: Section 2 presents the preliminaries, where brief discussion of fractional calculus is given. In Section 3 we discuss the formulation of the model, non-negativity of the solution model, basic reproduction number and the local asymptotic stability of the disease-free equilibrium. The existence and uniqueness of the solution of the model is presented in Section 4. The numerical scheme for the solution of the model is given in Section 5. In Section 6, simulations of the model including discussions take the center stage, while Section 7 gives the conclusion.

2. Preliminaries

This section presents brief fundamentals of fractional calculus and some theorems applied in the paper. The Atangana-Baleanu fractional derivatives and integrals which uses non-local and non-singular is stated. This fractional derivatives best describes the component of our model in comparison to other fractional derivatives like Caputo and Grunwald-Letnikov. It is also easier to compute and gives more room to the analysis of the model.

Definition 2.1 [29]. Let $f \in H^p(a_1, a_2), a_2 > a_1, \vartheta \in [0, 1]$, then the Atangana-Baleanu (AB) fractional derivative of a function $f(t)$ of order $\vartheta \in \mathbb{R}^+$ is defined by
\[
\frac{a}{\Gamma(1 - \theta)} D_0^\theta f(t) = \mathcal{B}(\vartheta) \frac{1}{1 - \vartheta} \int_{a_1}^{t} f(\epsilon) E_{\vartheta} \left[-\vartheta (t - \epsilon)^{\vartheta - 1}\right] \, d\epsilon,
\]
where \(\mathcal{B}(\vartheta)\) denotes a normalization function satisfying \(\mathcal{B}(0) = \mathcal{B}(1) = 1\).

**Definition 2.2** [29]. Let \(f \in H^1(a, a_2), a_2 > a_1, \vartheta \in [0, 1]\), then the Atangana-Baleanu (AB) fractional integral of a function \(f(t)\) of order \(\vartheta \in \mathbb{R}^+\) is defined by

\[
\frac{a}{\Gamma(1 - \theta)} D_0^\theta f(t) = \frac{1 - \vartheta}{\mathcal{B}(\vartheta)} f(t) + \frac{\vartheta}{\mathcal{B}(\vartheta) \Gamma(\vartheta)} \int_{a_1}^{t} f(\epsilon) (t - \epsilon)^{\vartheta - 1} \, d\epsilon
\]

**Lemma 2.1** [29]. For \(f \in H^1(a, a_2)\), the following Newton-Leibniz formula is satisfied,

\[
\frac{a}{\Gamma(1 - \theta)} D_0^\theta f(t) = f(t) - f(a)
\]

**Theorem 2.2** [29]. The following result holds for a function \(f \in C[a, \beta]\)

\[
\left\| \frac{a}{\Gamma(1 - \theta)} D_0^\theta f(t) \right\| < \mathcal{B}(\vartheta) \frac{1}{1 - \vartheta} \| f(t) \|, \quad \text{where} \quad \| f(t) \| = \max_{\alpha < \vartheta < \beta} |f(t)|
\]

The Lipschitz condition is fulfilled by ABC derivatives expressed as follows:

\[
\left\| \frac{a}{\Gamma(1 - \theta)} D_0^\theta f_1(t) - \frac{a}{\Gamma(1 - \theta)} D_0^\theta f_2(t) \right\| < \delta \| f_1(t) - f_2(t) \|
\]

**Theorem 2.3** [29]. The unique solution of the differential equation with fractional order \(\vartheta \in (0, 1]\) given by

\[
\frac{a}{\Gamma(1 - \theta)} D_0^\theta f(t) = r(t)
\]

is of the form

\[
f(t) = \frac{1 - \vartheta}{\mathcal{B}(\vartheta)} r(t) + \frac{\vartheta}{\mathcal{B}(\vartheta) \Gamma(\vartheta)} \int_{a_1}^{t} r(\epsilon) (t - \epsilon)^{\vartheta - 1} \, d\epsilon
\]

**Lemma 2.4** [31]. Suppose that \(w \in \mathbb{R}^m\) is a vector of differentiable function such that for any \(t > 0\)

\[
\frac{a}{\Gamma(1 - \theta)} D_0^\theta \left(\left[w - w^*\right]^T Q \left[w - w^*\right] \right) \leq \left[w - w^*\right]^T Q \frac{a}{\Gamma(1 - \theta)} D_0^\theta \left[w - w^*\right], \quad \vartheta \in (0, 1),
\]

where \(Q \in \mathbb{R}^{m \times m}\) is a constant, square matrix and positive definite.

3. Model formulation

The total human population at time \(t\), denoted by \(N_h(t)\), is split into a mutually exclusive sub-populations of unvaccinated susceptible individuals \((S(t))\), individuals vaccinated with the Pfizer vaccine \((V_1(t))\), individuals vaccinated with the Moderna vaccine \((V_2(t))\), asymptomatic infectious individuals \((A(t))\), vaccinated asymptomatic infectious individuals \((\bar{A}(t))\), unvaccinated symptomatic infectious individuals \((I_s(t))\), vaccinated symptomatic infectious individuals \((\bar{I}_s(t))\), symptomatic infectious individuals with severe illness \((I_v(t))\) and recovered individuals \((R(t))\). Thus, \(N_h(t) = S + V_1 + V_2 + V_3 + A + \bar{A} + I_s + \bar{I}_s + I_v + R\). We assume that individuals with severe COVID-19 illness are completely isolated and do not come in contact with the general population.

The population of unvaccinated susceptible individuals, \(S\), is generated by the recruitment of individuals at a rate \(\Lambda\). This population is decreased upon infection with COVID-19, following effective contact with infected individuals at the rate:

\[
\lambda = \frac{\beta (\Lambda + I_s + \eta_s I_v)}{N_h}.
\]

The modification parameter \(0 < \vartheta < 1\), accounts for reduced transmission probability of asymptomatic infectious individuals relative to symptomatic infectious. Also, the parameter \(\eta_s\) accounts for the infectiousness of vaccinated infectious relative to unvaccinated infectious. \(\kappa\) is a modification parameter which accounts for infectiousness of dually infected individuals due to TB. This population is further reduced by natural death at a rate \(\mu\). All the compartments of the model also suffer natural death at this rate. In (1), \(\beta\) is the effective contact rate for the COVID-19 transmission.

The model for COVID-19 transmission dynamics in a population is given by the following system of non-linear fractional differential equations of order \(\vartheta \in (0, 1]\), with Table 1 describing the associated parameters in the model.

\[
\begin{align*}
\frac{dS}{dt} &= -\beta S I_v - \gamma_s S + \gamma_v I_v, \\
\frac{dV_1}{dt} &= \beta S I_v - \gamma_v V_1, \\
\frac{dV_2}{dt} &= \gamma_v V_1 - \gamma_v V_2, \\
\frac{dV_3}{dt} &= \mu V_3 - \mu V_3, \\
\frac{dA}{dt} &= \gamma_v I_v - \gamma_v A, \\
\frac{d\bar{A}}{dt} &= \mu A - \mu A, \\
\frac{dI_s}{dt} &= \gamma_s S - \gamma_s I_s, \\
\frac{d\bar{I}_s}{dt} &= \gamma_s I_s - \gamma_v I_v, \\
\frac{dI_v}{dt} &= \gamma_s A - \gamma_v I_v, \\
\frac{dR}{dt} &= \gamma_v I_v - \gamma_v R.
\end{align*}
\]

3.1. Fundamentals of the model

The boundedness and positivity of the solutions which shows that system (2) is both mathematically and biologically well-posed is presented.

3.1.1. Non-negativity of the solution

**Theorem 3.1.** The closed set

\[
\mathcal{D} = \left\{ (S, V_1, V_2, \bar{V}_1, A, \bar{A}, I_s, I_v, R) \in \mathbb{R}^+ : S + V_1 + V_2 + A + \bar{A} + I_s + I_v + R < \frac{\Lambda}{\mu} \right\}
\]

is positively invariant with respect to the model (2).

**Proof.** Adding all the equations of the system (2) gives

\[
\frac{dN_h}{dt} = \Lambda - \mu N_h(t) - [d_a I_s + d_u I_v + d_o I_v].
\]

From (3), we have that

\[
\Lambda - (\mu + 3\delta) N_h \leq \frac{dN_h}{dt} < \Lambda - \mu N_h,
\]

where \(\delta = \min\{d_a, d_u, d_o\}\).
Table 1 Description of variables and parameters in the model (2).

| Variable | Interpretation |
|----------|----------------|
| $S$      | Unvaccinated Susceptible individuals |
| $V_1$    | Vaccinated with vaccine 1 (Pfizer) |
| $V_2$    | Vaccinated with vaccine 2 (Moderna) |
| $V_3$    | Vaccinated with vaccine 3 (Oxford Johnson & Johnson) |
| $A$      | Asymptomatic individuals (vaccinated and unvaccinated) |
| $I_s$    | Unvaccinated symptomatic individuals |
| $I_v$    | Vaccinated symptomatic individuals |
| $I_i$    | Symptomatic (vaccinated and unvaccinated) individuals with severe illness and hospitalized (under complete isolation) |
| $R$      | Recovered humans |

| Parameter | Interpretation | Baseline value | Reference |
|-----------|----------------|----------------|-----------|
| $\mu$     | Natural death rate rate | 0.0016708 $\text{day}^{-1}$ | [41] |
| $\Lambda$ | Recruitment rate | $\frac{17.63}{280000} \text{day}^{-1}$ | [41] |
| $\beta$   | Effective transmission rate | 0.00016708 Fitted | |
| $\zeta_1$ | Pfizer vaccine efficacy | 0.95 | [6] |
| $\zeta_2$ | Moderna vaccine efficacy | 0.945 | [5] |
| $\zeta_3$ | Janssen vaccine efficacy | 0.67 | [7] |
| $v_1$     | Pfizer vaccination rate | 0.0059 $\text{day}^{-1}$ | Fitted |
| $v_2$     | Moderna vaccination rate | 0.0042 $\text{day}^{-1}$ | Fitted |
| $v_3$     | Janssen vaccination rate | 0.00055 $\text{day}^{-1}$ | Fitted |
| $\rho$    | Fraction of unvaccinated susceptibles who progress to asymptomatic stage | 0.5 | Assumed |
| $\theta$  | Modification parameter for reduced transmissibility of asymptomatic individuals | 0.7 | [14] |
| $f_1$     | Fraction of vaccinated susceptibles who progress to asymptomatic stage | 0.5 | Assumed |
| $\gamma_s, \gamma_v, \gamma_i$ | Recovery rates for individuals in the $A, I_s, I_v, I_i$ classes, respectively | 0.13978 $\text{day}^{-1}$ | [14] |
| $\alpha$  | Rate of development of severe COVID-19 illness | 0.3 | Assumed |
| $\phi$    | Efficacy of the vaccines against severe COVID-19 illness | 0.8 | Inferred from [4] |
| $d_{ac}, d_{av}, d_{vi}$ | Disease induced death rates for individuals in the $I_s, I_v, I_i$ classes, respectively | 0.015 | [14] |

Applying the Laplace transform on the above inequality we have

$$N_h(t) \leq \left( \frac{\beta(t)}{(1 - \sigma) \mu} N_h(0) + \frac{(1 - \sigma) A}{(1 - \sigma) \mu} E_{\alpha,1} \left( -\frac{\mu}{(1 - \sigma) \mu} t^\alpha \right) \right) + \frac{\beta \alpha}{(1 - \sigma) \mu} E_{\alpha,0,1} \left( -\frac{\mu}{(1 - \sigma) \mu} t^\alpha \right) \quad \text{(4)}$$

The Mittag-Leffler function $E_{\alpha,1}$ is asymptotic in nature [29]. Thus, we have that $N_h(t) \leq \frac{c}{\sigma}$ as $t \to \infty$. As a result, the system (2) has the solution in $\mathcal{D}$. Thus, the given system is positively invariant. □

3.2. Basic reproduction number of the co-infection model (2)

The COVID-19 model (2) has a DFE, obtained by setting the right-hand sides of the equations in the model (2) to zero, given by

$$\mathcal{A}_0 = \begin{pmatrix} S' & V_1' & V_2' & V_3' & A' & I_s' & I_v' & I_i' & R' \end{pmatrix}^T = \begin{pmatrix} \frac{\beta \rho^S}{N_h} & \frac{\beta \rho^S}{N_h} & \frac{\beta \rho^S}{N_h} & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

The linear stability of the disease free equilibrium, $\xi_0$, can be established using the next generation operator method on the system (2). Using the notation in [42], the matrix $F$ (of new infections) and the matrix $V$ (of the transfer of individuals between compartments) are respectively, given by

$$F = \begin{pmatrix} \frac{\beta \rho^S}{N_h} & \frac{\beta \rho^S}{N_h} & \frac{\beta \rho^S}{N_h} & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

with

$$\mathcal{A} = pS' + f_1(1 - \zeta_1)V_1' + f_2(1 - \zeta_2)V_2' + f_3(1 - \zeta_3)V_3'$$

$$\mathcal{A}^{-1} = (1 - f_1)(1 - \zeta_1)V_1' + (1 - f_2)(1 - \zeta_2)V_2' + (1 - f_3)(1 - \zeta_3)V_3'$$

Hence, it follows from [42] that the basic reproduction number of the model (2), denoted by $R_0$, is given by

$$R_0 = \beta \left( \frac{(1 - p) \mathcal{A}_1 \mathcal{A}_2 \mathcal{A}_3 \mathcal{A}_4 \mathcal{A}_5 \mathcal{A}_6 \mathcal{A}_7 \mathcal{A}_8 \mathcal{A}_9 \mathcal{A}_{10} \mathcal{A}_{11} \mathcal{A}_{12}}{\mu (v_1 + v_2 + v_3) \mathcal{A}_1 \mathcal{A}_2 \mathcal{A}_3} \right)$$

with

$$\mathcal{A}_1 = (1 - f_1)(1 - \zeta_1)v_1 + (1 - f_2)(1 - \zeta_2)v_2 + (1 - f_3)(1 - \zeta_3)v_3$$

$$\mathcal{A}_2 = \rho \mu + f_1(1 - \zeta_1)v_1 + f_2(1 - \zeta_2)v_2 + f_3(1 - \zeta_3)v_3$$
3.3. Local asymptotic stability of the disease-free equilibrium

**Theorem 3.2.** The DFE, $N_0$, of the model (2) is locally asymptotically stable (LAS) if $R_e < 1$, and unstable if $R_e > 1$.

**Proof.** The local stability of the model (2) is analysed by the Jacobian matrix of the system (2) evaluated at the COVID-19-free equilibrium $N_0$, given by:

$$J(x_0) = \begin{pmatrix}
-v_1 - p & 0 & 0 & 0 & 0 \\
0 & -p & 0 & 0 & 0 \\
0 & 0 & -p & 0 & 0 \\
0 & 0 & 0 & -p & 0 \\
0 & 0 & 0 & 0 & -p
\end{pmatrix}$$

The characteristic equation of the above matrix, for the case when $\omega = 1$, is given by:

$$(\lambda + \mu)^2(\lambda + \mu)(\lambda + v_1 + v_2 + v_3 + \mu)$$

$$+ (\mu + 1)(\mu + 1)(\mu + 1)(\mu + 1)(\mu + 1) S + \eta \theta \mu \cdot \mu$$

$$- (\lambda + \mu)^2(\lambda + \mu)(\lambda + v_1 + v_2 + v_3 + \mu)$$

$$+ (\lambda + v_1 + v_2 + v_3 + \mu)$$

$$= 0.$$ (6)

The eigenvalues are given by:

$$\lambda_1 = -\mu, \quad \lambda_2 = -\mu, \quad \lambda_3 = -\mu, \quad \lambda_4 = -\mu, \quad \lambda_5 = -\mu,$$

and the solutions of the characteristic polynomial

$$\lambda^3 + \beta_{11}\lambda^2 + \beta_{22}\lambda + \beta_{33} = 0,$$ (7)

where

$$\beta_{11} = (\lambda + v_1 + v_2 + v_3 + \mu)$$

$$\beta_{22} = (\lambda + v_1 + v_2 + v_3 + \mu)$$

$$+ (\lambda + v_1 + v_2 + v_3 + \mu)$$

$$\beta_{33} = (\lambda + v_1 + v_2 + v_3 + \mu)$$

Applying the Routh-Hurwitz criterion, the Eq. (7) will have roots with negative real parts if and only if $\beta_{11} > 0$, $\beta_{22} > 0$, and $\beta_{33} > 0$ (if $R_e < 1$). Thus, the DFE, $f(V_0)$ is locally asymptotically stable if $R_e < 1$. □

Since $\text{Im} (\lambda_k) = 0$ for $k = 0, 1, 2, 3, \ldots, 9$, $\arg(\lambda_k) = \pi > 2\pi$ for $0 < \alpha < 1$.

The epidemiological implication of Theorem 3.2 is that COVID-19 can be eliminated from the population when $R_e < 1$ and if the initial sizes of the population of the model are in the region of attraction of the DFE.

4. Existence and uniqueness of the solution

4.1. Existence of solution

In this case, we consider the shortened expression for our model (2) so as to establish the existence and uniqueness of the solution.

$$\begin{cases}
\frac{dR}{dt} G(t) = \mathcal{G}(t, G(t)), \\
G(0) = G_0,
\end{cases}$$ (8)

where the vector $G = (S, V_1, V_2, V_3, A, I_1, I, R)$ denotes the model compartments and $\mathcal{G} = (g_1, g_2, g_3, g_4, g_5, g_6, g_7, g_8, g_9)$ denotes the continuous vector function such that

$$g_1(t, S(t)) = \sum_{i=1}^{5} \frac{a_i(t)}{S(t)} S(t)$$

$$g_2(t, V_1(t)) = \sum_{i=1}^{5} \frac{b_i(t)}{V_1(t)} V_1(t)$$

$$g_3(t, V_2(t)) = \sum_{i=1}^{5} \frac{c_i(t)}{V_2(t)} V_2(t)$$

$$g_4(t, V_3(t)) = \sum_{i=1}^{5} \frac{d_i(t)}{V_3(t)} V_3(t)$$

$$g_5(t, \cdot) = \sum_{i=1}^{5} \frac{e_i(t)}{A(t)} A(t)$$

$$g_6(t, \cdot) = \sum_{i=1}^{5} \frac{f_i(t)}{I(t)} I(t)$$

$$g_7(t, \cdot) = \sum_{i=1}^{5} \frac{g_i(t)}{R(t)} R(t)$$

$$g_8(t, \cdot) = \sum_{i=1}^{5} \frac{h_i(t)}{I(t)} I(t)$$

$$g_9(t, \cdot) = \sum_{i=1}^{5} \frac{i_i(t)}{I(t)} I(t).$$

Application of ABC integral into the fractional systems yields the following Volterra integral equations

$$S(t) = S(0) + \int_0^t \frac{dS(t)}{dt} \mathcal{G}(t, S(t)) dt + \int_0^t \int_0^{\tau} \mathcal{G}(t, S(t)) d\tau \, dt,$$

$$V_1(t) = V_1(0) + \int_0^t \frac{dV_1(t)}{dt} \mathcal{G}(t, V_1(t)) dt + \int_0^t \int_0^{\tau} \mathcal{G}(t, V_1(t)) d\tau \, dt,$$

$$V_2(t) = V_2(0) + \int_0^t \frac{dV_2(t)}{dt} \mathcal{G}(t, V_2(t)) dt + \int_0^t \int_0^{\tau} \mathcal{G}(t, V_2(t)) d\tau \, dt,$$

$$V_3(t) = V_3(0) + \int_0^t \frac{dV_3(t)}{dt} \mathcal{G}(t, V_3(t)) dt + \int_0^t \int_0^{\tau} \mathcal{G}(t, V_3(t)) d\tau \, dt,$$

$$A(t) = A(0) + \int_0^t \frac{dA(t)}{dt} \mathcal{G}(t, A(t)) dt + \int_0^t \int_0^{\tau} \mathcal{G}(t, A(t)) d\tau \, dt,$$

$$I(t) = I(0) + \int_0^t \frac{dI(t)}{dt} \mathcal{G}(t, I(t)) dt + \int_0^t \int_0^{\tau} \mathcal{G}(t, I(t)) d\tau \, dt,$$

$$R(t) = R(0) + \int_0^t \frac{dR(t)}{dt} \mathcal{G}(t, R(t)) dt + \int_0^t \int_0^{\tau} \mathcal{G}(t, R(t)) d\tau \, dt.$$

**Theorem 4.1.** The continuous function $\mathcal{G}_k$, for $k = 1, 2, \ldots, 9$, satisfies the Lipschitz condition for the second argument if and only if;

$$\sup_{0 < \alpha < \beta \leq 1} \| \mathcal{G}_k(\alpha) - \mathcal{G}_k(\beta) \| < \phi.$$ (9)

**Proof of Theorem 4.1** Let $C([0, T]) : \mathbb{R} \rightarrow \mathbb{R}$ be the Banach space of all continuous functions from $[0, T] \rightarrow \mathbb{R}$ so that using the basic fixed point theorem, we show that the function $\mathcal{G}_k, k = 1, 2, \ldots, 9$, is Lipschitz continuous with Lipschitz constants. Let $\overline{S}$ be the second solution and using triangle inequality endowed with Chebyshev norm we have the following relationships

$$\| \mathcal{G}_k(S) - \mathcal{G}_k(S) \| < \left\| A - \left( v_1 + v_2 + v_3 + \frac{a_1(t)}{S(t)} + \mu \right) S \right\|,$$

$$\| \right\| A - \left( v_1 + v_2 + v_3 + \frac{a_1(t)}{S(t)} + \mu \right) S \|.$$

$$\| v_1 + v_2 + v_3 + \mu \| \| \mathcal{G}_k(S) - \mathcal{G}_k(S) \|.$$ (11)

where $L_{g_1} = v_1 + v_2 + v_3 + \mu$ and $g_1$ be the second solution and using triangle inequality we have
\[ \|G_2(V_1) - G_2(F_1)\| \leq \|v_1 S - \left(1 - \mu \right) + \frac{\beta_0 V_1}{\mu} + \mu F_1 \| + \left(1 - \mu \right) + \frac{\beta_0 V_1}{\mu} + \mu F_1 \| \]
\[ = L_{g_1} \| V_1 - F_1 \|, \]
where \( L_{g_1} = 1 - \xi_1 + \mu + \beta_0 q_1 + q_2 + \eta_q_3 \).

Similarly, for \( G_2(t, V_2(t)) \),

\[ \|G_2(V_2) - G_2(F_2)\| \leq \|v_2 S - \left(1 - \mu \right) + \frac{\beta_0 V_2}{\mu} + \mu F_2 \| + \left(1 - \mu \right) + \frac{\beta_0 V_2}{\mu} + \mu F_2 \| \]
\[ = L_{g_2} \| V_2 - F_2 \|, \]
where \( L_{g_2} = 1 - \xi_2 + \mu + \beta_0 q_1 + q_2 + \eta_q_3 \).

For \( G_2(t, A_1(t)) \),

\[ \|G_2(A_1) - G_2(A_0)\| \leq \left(1 - p \right) \frac{\beta_0 (I + \eta_q_1)}{N_3} + \left(1 - p \right) \frac{\beta_0 (I + \eta_q_1)}{N_3} \]
\[ = L_{g_3} \| A_1 - A_0 \|, \]
where \( L_{g_3} = (p_1 + f_1 (1 - \xi_1) q_1 + f_2 (1 - \xi_2) q_2 + \beta_0 f_3 (1 - \xi_3) q_3 + \eta_q) \beta_0 \eta_q \).

For \( G_2(t, I_1) \),

\[ \|G_2(I_1) - G_2(I_0)\| \leq \left(1 - p \right) \frac{\beta_0 (I + \eta_q_1)}{N_3} + \left(1 - p \right) \frac{\beta_0 (I + \eta_q_1)}{N_3} \]
\[ + \left(1 - p \right) \frac{\beta_0 (I + \eta_q_1)}{N_3} \]
\[ = L_{g_4} \| I_1 - I_0 \|, \]
where \( L_{g_4} = \gamma_{I_0} + d_{I_0} + \mu + (p - 1) q_{I_0} \).

For \( G_2(t, R_1) \),

\[ \|G_2(R_1) - G_2(R_0)\| \leq \left(1 - p \right) \frac{\beta_0 (I + \eta_q_1)}{N_3} + \left(1 - p \right) \frac{\beta_0 (I + \eta_q_1)}{N_3} \]
\[ = L_{g_5} \| R_1 - R_0 \|, \]
where \( L_{g_5} = \gamma_{R_0} + d_{R_0} + \mu \).

Thus with the agreement in (11)-(20), \( G_{g_k}(t, R_1(t), I_1(t), \ldots, I_9(t), R_0(t), A_0(t), A_1(t), I_0(t), R_0(t)) \) satisfies the Lipschitz condition in its second argument with the Lipschitz constant

\[ L_{g_5} = \left(1 - p \right) \frac{\beta_0 (I + \eta_q_1)}{N_3} \]

Consider the following Neumann series,

\[ S_e(t) = S(0) + \int_0^t \frac{1}{(t - \tau)^{1-p}} G_{g_5}(c, S_{e_1}(\tau)) d\tau + \int_0^t \frac{1}{(t - \tau)^{1-p}} G_{g_5}(c, S_{e_0}(\tau)) d\tau \]

It follows that the Neumann series is convergent by considering,

\[ |S_{e_1}(t) - S_{e_1}(0)| < \frac{\beta_0 (I + \eta_q_1)}{N_3} \left| \frac{1}{(t - \tau)^{1-p}} G_{g_5}(c, S_{e_1}(\tau)) \right| \]

Applying similar process yields

\[ |V_{\text{max}}(t) - V_{\text{max}}(0)| < \frac{\beta_0 (I + \eta_q_1)}{N_3} \left| \frac{1}{(t - \tau)^{1-p}} G_{g_5}(c, S_{\text{max}}(\tau)) \right| \]

It follows that
satisfying the integral Eq. (10), then endowed by Chebychev norm

Evidently, the right hand sides of inequalities in (22) tends to zero as $n \to \infty$ uniformly on $[0, T]$. By taking limits in (21), we see that $S, V_1, V_2, V_3, A, I_0, I_1, I_2, R$ satisfies the original integral (10), thereby proving the existence of the continuous functions.

4.2. Uniqueness of solution

**Theorem 4.2.** The fractional model has a unique solution if

$$L_{S_0} < \left( \frac{(1-\theta)}{\mathcal{A}(\theta)} + \frac{T_{\text{max}}}{\mathcal{B}(\theta) \Gamma(\theta)} \right)^{-1} \cdot $$

(23)

**Proof.** To demonstrate the uniqueness of the solution, we assume that $S, V_1, V_2, V_3, A, I_0, I_1, I_2, R$ is another solution satisfying the integral Eq. (10), then endowed by Chebychev norm we get

$$||S(t) - S(t)|| \leq \left( \frac{1}{\mathcal{A}(\theta)} ||S(t) - S(t)|| + \frac{T_{\text{max}}}{\mathcal{B}(\theta) \Gamma(\theta)} \right) L_{S_0} - 1 \cdot $$

(24)

Applying the same process yields

$$ \left( \frac{(1-\theta)}{\mathcal{A}(\theta)} + \frac{T_{\text{max}}}{\mathcal{B}(\theta) \Gamma(\theta)} \right) L_{S_0} - 1 \cdot $$

(25)

Substituting the expressions in (26) into the force of infection at steady state

$$ \lambda^* = \frac{\beta_0 (0.4 \lambda^* + \lambda^*_n + \eta_3 \lambda^*_n)}{N_b^*}, $$

(27)

and simplifying, we have

$$ \epsilon_1 \lambda^{*4} + \epsilon_2 \lambda^{*3} + \epsilon_3 \lambda^{*2} + \epsilon_4 \lambda^* + \epsilon_5 = 0 $$

(28)

with

$$ \epsilon_1 = \gamma_{1} \lambda^{*4} + \gamma_{2} \lambda^{*3} + \gamma_{3} \lambda^{*2} + \gamma_{4} \lambda^* + \gamma_{5} = 0 $$

(29)

Evidently using (23), the inequalities in (24) and (25) is true for all $||S(t) - S(t)|| = 0$, $||V_1(t) - V_1(t)|| = 0$, $||V_2(t) - V_2(t)|| = 0$, $||V_3(t) - V_3(t)|| = 0$, $||A(t) - A(t)|| = 0$, $||I_0(t) - I_0(t)|| = 0$, $||I_1(t) - I_1(t)|| = 0$, $||I_2(t) - I_2(t)|| = 0$, $||R(t) - R(t)|| = 0$. Hence $S = S, V_1 = V_1, V_2 = V_2, V_3 = V_3, A = A, I_0 = I_0, I_1 = I_1, I_2 = I_2, R = R$ everywhere and therefore there is just one continuous solution for the fractional model. $\square$

4.3. Existence of endemic equilibrium of the model

The endemic equilibrium of the fractional order model are obtained by setting the right hand sides of the model Eqs. (2) to zero, that is,

$$ ^{\alpha_0}D^t_0 d^* S = 0, \quad ^{\alpha_0}D^t_0 d^* V_1 = 0, \quad ^{\alpha_0}D^t_0 d^* V_2 = 0, \quad ^{\alpha_0}D^t_0 d^* V_3 = 0, \quad ^{\alpha_0}D^t_0 d^* A = 0, \quad ^{\alpha_0}D^t_0 d^* I_0 = 0, \quad ^{\alpha_0}D^t_0 d^* I_1 = 0, \quad ^{\alpha_0}D^t_0 d^* I_2 = 0, \quad ^{\alpha_0}D^t_0 d^* R = 0. $$

The endemic equilibrium (EE) of the model (2) is given by

$$ S^* = \frac{\Lambda}{\gamma_1 + \gamma_2 + \gamma_3 + \mu}, $$

$$ V_1^* = \frac{(\gamma_1 + \gamma_2 + \gamma_3 + \mu) \left[1 - \xi_1 \lambda^* + \mu \right]}{\gamma_1 \gamma_2 \gamma_3 \lambda^*}, $$

$$ V_2^* = \frac{(\gamma_1 + \gamma_2 + \gamma_3 + \mu) \left[1 - \xi_2 \lambda^* + \mu \right]}{\gamma_1 \gamma_2 \gamma_3 \lambda^*}, $$

$$ V_3^* = \frac{(\gamma_1 + \gamma_2 + \gamma_3 + \mu) \left[1 - \xi_3 \lambda^* + \mu \right]}{\gamma_1 \gamma_2 \gamma_3 \lambda^*}, $$

$$ A^* = \frac{(\gamma_1 + \gamma_2 + \gamma_3 + \mu) \left[1 - \xi_4 \lambda^* + \mu \right]}{\gamma_1 \gamma_2 \gamma_3 \lambda^*}, $$

$$ I_0^* = \frac{(\gamma_1 + \gamma_2 + \gamma_3 + \mu) \left[1 - \xi_5 \lambda^* + \mu \right]}{\gamma_1 \gamma_2 \gamma_3 \lambda^*}, $$

$$ I_1^* = \frac{(\gamma_1 + \gamma_2 + \gamma_3 + \mu) \left[1 - \xi_6 \lambda^* + \mu \right]}{\gamma_1 \gamma_2 \gamma_3 \lambda^*}, $$

$$ I_2^* = \frac{(\gamma_1 + \gamma_2 + \gamma_3 + \mu) \left[1 - \xi_7 \lambda^* + \mu \right]}{\gamma_1 \gamma_2 \gamma_3 \lambda^*}, $$

$$ R^* = \frac{(\gamma_1 + \gamma_2 + \gamma_3 + \mu) \left[1 - \xi_8 \lambda^* + \mu \right]}{\gamma_1 \gamma_2 \gamma_3 \lambda^*}. $$

(26)
where
\[
\mathbf{K}_1 = \gamma_a + \mu, \quad \mathbf{K}_2 = \gamma_d + d_w + \alpha + \mu, \\
\mathbf{K}_3 = \gamma_d + d_w + (1 - \phi)\alpha + \mu, \quad \mathbf{K}_4 = \gamma_d + d_w + \mu, \\
\psi_1 = (1 - \xi_1), \quad \psi_2 = (1 - \xi_2), \quad \psi_3 = (1 - \xi_3), \\
\mathbf{H}_1 = \mu(1 - \psi_1)\eta_1[0, \phi, \mathbf{K}_2, \mathbf{K}_3 + (1 - f_1)\eta_1, \mathbf{K}_4, \mathbf{K}_2], \\
\mathbf{H}_2 = \mu(1 - \psi_2)\eta_2[0, \phi, \mathbf{K}_2, \mathbf{K}_3 + (1 - f_2)\eta_1, \mathbf{K}_4, \mathbf{K}_2], \\
\mathbf{H}_3 = \mu(1 - \psi_3)\eta_3[0, \phi, \mathbf{K}_2, \mathbf{K}_3 + (1 - f_3)\eta_1, \mathbf{K}_4, \mathbf{K}_2].
\]

The components of the EEP are obtained upon solving for \( \lambda^* \) from the polynomial (28), and substituting the positive values of \( \lambda^* \) into the expressions in (26). Moreover, it follows from (29) that the coefficient \( \mathbf{E}_1 \) is always positive, and \( \mathbf{E}_3 \) is positive (negative) if \( \mathbf{R}_e \) is less (greater) than unity. The following results can be established.

**Theorem 4.3.** The model (2) has:

(i) four or two endemic equilibria if \( \mathbf{E}_2 > 0, \mathbf{E}_3 < 0, \mathbf{E}_4 > 0 \) and \( \mathbf{R}_e < 1 \),

(ii) two endemic equilibria if \( \mathbf{E}_2 > 0, \mathbf{E}_3 > 0, \mathbf{E}_4 < 0 \) and \( \mathbf{R}_e < 1 \),

(iii) no endemic equilibrium otherwise, if \( \mathbf{R}_e < 1 \).

The first two items of Theorem 4.3 (i) - (ii) suggest the possibility of backward bifurcation in the model (2) when \( \mathbf{R}_e < 1 \). A backward bifurcation diagram for the model is presented in Fig. 1, in the presence of imperfect COVID-19 vaccines. It is worthy of note to show that, setting the parameters \( \zeta_1 = \zeta_2 = \zeta_3 = 1 \), reduces the quartic (28) to:

\[
\mathbf{K}_1, \mathbf{K}_2, \mathbf{K}_3, \mu \lambda^* + \mu^3(1 - \mathbf{R}_e) = 0,
\]

resulting in no sign changes in the polynomial equation for \( \mathbf{R}_e < 1 \). However, for \( \mathbf{R}_e > 1 \) a unique endemic equilibrium exists.

4.4. Global asymptotic stability analysis of the disease-free equilibrium

We shall consider a special case of the model (2) with perfect vaccines (ruling out the occurrence of backward bifurcation) given by

\[
\begin{align*}
\frac{dS}{dt} &= \lambda - v_1S - v_2S - v_3S - \frac{\beta(I + L)}{N_0}S - \mu S, \\
\frac{dV_1}{dt} &= v_1S - \mu V_1, \\
\frac{dV_2}{dt} &= v_2S - \mu V_2, \\
\frac{dV_3}{dt} &= v_3S - \mu V_3, \\
\frac{dI}{dt} &= p(\beta I/L) - (\gamma_a + \mu)I, \\
\frac{dL}{dt} &= (1 - p)(\beta I/L) - (\gamma_a + d_w + \alpha + \mu)L, \\
\frac{dR}{dt} &= r_0\alpha + \gamma_dL + \gamma_dI - \mu R.
\end{align*}
\]

Following the approach in [42], the basic reproduction number of the reduced model, with perfect vaccine (\( \zeta_1 = \zeta_2 = \zeta_3 = 1 \)) is given by

\[
\mathbf{R}_e = \mathbf{R}_e|_{\zeta_1=\zeta_2=\zeta_3=1} = \frac{\beta((1 - p)\mathbf{K}_1 + p\mathbf{K}_2\theta)}{\mathbf{K}_1 \mathbf{K}_2}.
\]

Let us consider the following Lyapunov function for the purpose of investigating the stability of the disease free equilibrium for the sub model

\[
\mathcal{L}_1 = \frac{\theta}{\mathbf{K}_1}A + \frac{1}{\mathbf{K}_1}I_w + \frac{1}{\mathbf{K}_2}I_u
\]

where,

\[
\mathcal{L}_1' = \frac{\theta}{\mathbf{K}_1}D_I A + \frac{1}{\mathbf{K}_2}D_I I_u(t)
\]

Substituting the right hand sides of (30) into the above, we have

\[
\mathcal{L}_1' = \frac{\theta}{\mathbf{K}_1} \left( \frac{p(\beta(I + L))}{N_0} - (\gamma_a + \mu)A \right)
\]

which can be simplified into:

\[
\mathcal{L}_1' = \frac{\theta}{\mathbf{K}_1} \left( \frac{p(\beta(I + L))}{N_0} - (\gamma_a + \mu)A \right) + \frac{1}{\mathbf{K}_2} \left( \frac{(1 - p)(\beta(I + L))}{N_0} - (\gamma_a + d_w + \alpha + \mu)L \right)
\]

Simplifying further, we have that

\[
\mathcal{L}_1' \leq (\theta + I_u)(\mathbf{R}_e - 1)
\]

Clearly \( \mathbf{R}_e < 1 \) is satisfied since the above inequality with positive parameters shows that \( D_1' \mathcal{L}_1' \) is non-positive. Thus we can now say that the disease free equilibrium is globally asymptotically stable if \( \mathbf{R}_e < 1 \) and unstable if \( \mathbf{R}_e > 1 \).

5. Numerical scheme for the solution of the model

In this section, we derive the numerical method for the stated fractional differential system (2). We shall adopt the scheme given in [29] in order to approximate the Atangana-Baleanu fractional Integral. The stability and the convergence analysis of the numerical method has been discussed extensively in [8]. Applying the fundamental theorem of fractional calculus on (8), we have

\[
G(t) = G_0 + \frac{1 - \vartheta}{\mathbf{A}(\vartheta)} G(t, G(t)) + \frac{\vartheta}{\mathbf{A}(\vartheta)} G(t, G(t)) + \tilde{G}(t, \tau, G(t))
\]

\[
\times \int_0^t (t - \tau)^{\vartheta - 1}G(t, G(t))d\tau.
\]

At \( t = t_{n+1} = (n + 1)h \), where \( h = t_{n+1} - t_n \) is the time space, the above equation discretizes to

\[
G(t_{n+1}) = G(t_n) + \frac{1 - \vartheta}{\mathbf{A}(\vartheta)} G(t_n, G(t_n)) + \frac{\vartheta}{\mathbf{A}(\vartheta)} G(t_n, G(t_n)) + \tilde{G}(t, \tau, G(t))d\tau.
\]

Applying Lagrange two-points interpolation polynomial into (35), the numerical scheme for the general fractional system reduces to

\[
G(t_{n+1}) = G(t_n) + \frac{1 - \vartheta}{\mathbf{A}(\vartheta)} G(t_n, G(t_n)) + \frac{\vartheta}{\mathbf{A}(\vartheta)} \sum_{t_{n+1} - \tau_0}^{t_{n+1}} \left[ \beta'(\bar{t}), \tilde{G}(\bar{t}, G(t)) \right]
\]

\[
- \left( \frac{(n - \tau_0)^{\vartheta - 1}(n - \tau_0 + 1)^{\vartheta - 1}}{(n - \tau_0 + 2)^{\vartheta - 1}} \right) G(t_{n+1} + \xi_0, G(t_{n+1} + \xi_0)) + \tilde{G}(t, \tau, G(t))d\tau.
\]

\[
- \left( \frac{(n - \tau_0 + 1)^{\vartheta - 1}(n - \tau_0 + 2)^{\vartheta - 1}}{(n - \tau_0 + 2)^{\vartheta - 1}} \right) G(t_{n+1} + \xi_1, G(t_{n+1} + \xi_1)) + \tilde{G}(t, \tau, G(t))d\tau.
\]

\[
- \left( \frac{(n - \tau_0 + 2)^{\vartheta - 1}(n - \tau_0 + 3)^{\vartheta - 1}}{(n - \tau_0 + 3)^{\vartheta - 1}} \right) G(t_{n+1} + \xi_2, G(t_{n+1} + \xi_2)) + \tilde{G}(t, \tau, G(t))d\tau.
\]

\[
- \left( \frac{(n - \tau_0 + 3)^{\vartheta - 1}(n - \tau_0 + 4)^{\vartheta - 1}}{(n - \tau_0 + 4)^{\vartheta - 1}} \right) G(t_{n+1} + \xi_3, G(t_{n+1} + \xi_3)) + \tilde{G}(t, \tau, G(t))d\tau.
\]
Adopting the numerical scheme \((36)\) into the fractional system \((2)\) yields the following numerical solution:

\[
S(t_{n+1}) = S(t_n) + \left(1 - \frac{\mu}{\Delta t}\right) S(t_n, S(t_n)) + \frac{\mu}{\Delta t} \sum_{\epsilon = 0}^{\infty} \left[ \frac{\mu S(t_n, S(t_n))}{\Gamma(\vartheta + 2)} \left( (\varpi - \epsilon + 1)^\vartheta (\varpi - \epsilon + \vartheta + 2) - (\varpi - \epsilon)^\vartheta (\varpi - \epsilon + 2\vartheta + 2) \right) \right].
\]

\[
V_1(t_{n+1}) = V_1(t_n) + \left(1 - \frac{\mu}{\Delta t}\right) V_1(t_n, V_1(t_n)) + \frac{\mu}{\Delta t} \sum_{\epsilon = 0}^{\infty} \left[ \frac{\mu V_1(t_n, V_1(t_n))}{\Gamma(\vartheta + 2)} \left( (\varpi - \epsilon + 1)^\vartheta (\varpi - \epsilon + \vartheta + 2) - (\varpi - \epsilon)^\vartheta (\varpi - \epsilon + 2\vartheta + 2) \right) \right].
\]

\[
A(t_{n+1}) = A(t_n) + \left(1 - \frac{\mu}{\Delta t}\right) A(t_n, A(t_n)) + \frac{\mu}{\Delta t} \sum_{\epsilon = 0}^{\infty} \left[ \frac{\mu A(t_n, A(t_n))}{\Gamma(\vartheta + 2)} \left( (\varpi - \epsilon + 1)^\vartheta (\varpi - \epsilon + \vartheta + 2) - (\varpi - \epsilon)^\vartheta (\varpi - \epsilon + 2\vartheta + 2) \right) \right].
\]

6. Numerical simulations and discussions

6.1. Baseline values of the parameters and model Fitting

We estimate the COVID-19 vaccination rate and contact rate for the city of Texas, USA. Other COVID-19 related parameters are obtained from the literature as shown in Table 1. The population of Texas is estimated at 29,200,000 [41]. Life expectancy in the United states is estimated at 75 years [41]. Hence, the recruitment rate \(\Lambda\) is set at \(\frac{29,200,000}{365} \approx 80000\) per day, and the natural death rate \(\mu\) set at \(\frac{1}{75,365}\) per day. The total unvaccinated susceptible population is set at \(S(0) = 24,000,000\). As at 13th March 2021, the cumulative number of individuals vaccinated with the Pfizer, Moderna and Janssen vaccines in the city of Texas is estimated at 4,115,127; 4,016,005 and 129,859, respectively [44]. Hence we set \(V_1(0) = 4,115,127\), \(V_2(0) = 4,016,005\) and \(V_3 = 129,859\). The total active COVID-19 cases in the city of Texas as at 13th March, 2021 is estimated
at 92,485. Hence we set \( A(0) = 50,000, I_s(0) = 17,000, I_t(0) = 15,000, I_v(0) = 10,000 \) and \( R(0) = 5000 \).

The model fitting was performed using \texttt{fmincon} function in the Optimization Toolbox of MATLAB [47]. The stability of the numerical method has been well established [47]. Using the data sets for those vaccinated with the Pfizer vaccine, Moderna vaccine and Janssen vaccine in the city of Texas, USA, from March 13, 2021 to June 29, 2021 [44], we estimated the three vaccination rates \( m_1, m_2, m_3 \) and the contact rate \( b \) as follows:

- \( m_1, \text{Pfizer vaccination rate} = 0.0059 \) day\(^{-1}\)
- \( m_2, \text{Moderna vaccination rate} = 0.0042 \) day\(^{-1}\)
- \( m_3, \text{Janssen vaccination rate} = 0.00053 \) day\(^{-1}\)

The effective contact rate for COVID-19 transmission is estimated at 0.00016708 day\(^{-1}\), so that the basic reproduction number is \( R_0 = 1.1946 \).

### 6.2. Pfizer only vaccination strategy

Simulations of the vaccination model (2) when the strategy that implements only Pfizer Bio-NTech vaccine \((f_1 \neq 0, r_1 \neq 0, \xi_1 \neq 0, \phi \neq 0)\) is administered, are presented in Figs. 5–7, respectively. It is observed that when this intervention strategy is implemented, for \( \beta = 0.40017414 \), there is a significant reduction in the number of asymptomatic individuals infected with COVID-19 (as shown in Fig. 5, where a total of 681,900 new asymptomatic COVID-19 cases were averted after 120 days of vaccine administration). Also, this strategy also has positive population level impact on the number of vaccinated symptomatic infectious individuals infected with COVID-19 (as depicted by Fig. 6, where a total of 597,400 new infections were averted in the first 100 days of vaccine administration). Moreover, this strategy averts a total of 1,033,300 severe COVID-19 cases among vaccinated symptomatic infectious after 100 days of vaccine administration.
Fig. 3  Fitting the model to cumulative number of individuals vaccinated with the Moderna vaccine in Texas, USA, when the three data sets: Cumulative number of individuals vaccinated with the Pfizer vaccine, Moderna vaccine and Janssen vaccine are used, concurrently.

Fig. 4  Fitting the model to cumulative number of individuals vaccinated with the Janssen vaccine in Texas, USA, when the three data sets: Cumulative number of individuals vaccinated with the Pfizer vaccine, Moderna vaccine and Janssen vaccine are used, concurrently.

Fig. 5  Simulation of the total number of asymptomatic individuals when Pfizer only vaccination strategy is implemented.
6.3. Moderna only vaccination strategy

Simulations of the vaccination model (2) when the strategy that implements only Moderna vaccine \((f_2 \neq 0, v_2 \neq 0, \xi_2 \neq 0, \phi = 0)\) is administered, are presented in Figs. 8–10, respectively. It is observed that when this intervention strategy is implemented, for \(\beta = 0.40017414\), there is a significant reduction in the number of asymptomatic individuals infected with COVID-19 (as presented in Fig. 8, where a total of 661,500 new asymptomatic COVID-19 cases were averted in the first 120 days of implementing this strategy). Also, this strategy averts 592,560 new symptomatic cases among vaccinated individuals infected with COVID-19 (as shown in Fig. 9). In addition, this strategy averts a significant number of severe COVID-19 cases among vaccinated symptomatic infectious (as depicted by Fig. 10, where a total of 985,900 new asymptomatic COVID-19 cases were averted in the first 100 days of implementing this strategy).

6.4. Janssen only vaccination strategy

Simulations of the vaccination model (2) when the strategy that implements only Janssen vaccine \((f_3 \neq 0, v_3 \neq 0, \xi_3 \neq 0, \phi = 0)\) is administered, are presented in Figs. 11–13, respectively. It is observed that when this intervention strategy is implemented, for \(\beta = 0.40017414\), there is a significant reduction in the number of asymptomatic individuals infected with COVID-19 (as depicted by Fig. 11, where about 177,800 new asymptomatic COVID-19 cases were averted after 120 days of implementing this strategy). Also, this strategy also has positive population level impact on the number of vaccinated symptomatic infectious individuals infected with COVID-19 (as shown in Fig. 12, where 472,200 new infections were averted among vaccinated individuals after 100 days of commencing of this strategy). Moreover, this strategy averts a total of 346,100 severe COVID-19 cases among vaccinated symptomatic infectious in the first 100 days of commencing this vaccination programme.
Fig. 8  Simulation of the total number of asymptomatic individuals when Moderna only vaccination strategy is implemented.

Fig. 9  Simulation of the total number of vaccinated symptomatic individuals when Moderna only vaccination strategy is implemented.

Fig. 10  Simulation of the total number of symptomatic individuals with severe COVID-19 illness when Moderna only vaccination strategy is implemented.
6.5. Comparing the impact of the three vaccines

It is observed that the Pfizer vaccine has more positive population level impact compared to the Moderna vaccine. In a similar manner, the Moderna vaccine has a higher positive population level impact in comparison with the Janssen vaccine. It is also observed that when Pfizer and Moderna vaccination controls are applied the plots tend towards the

Fig. 11  Simulation of the total number of asymptomatic individuals when Janssen only vaccination strategy is implemented.

Fig. 12  Simulation of the total number of vaccinated symptomatic individuals when Janssen only vaccination strategy is implemented.

Fig. 13  Simulation of the total number of symptomatic individuals with severe COVID-19 illness when Janssen only vaccination strategy is implemented.

6.5. Comparing the impact of the three vaccines

It is observed that the Pfizer vaccine has more positive population level impact compared to the Moderna vaccine. In a similar manner, the Moderna vaccine has a higher positive population level impact in comparison with the Janssen vaccine. It is also observed that when Pfizer and Moderna vaccination controls are applied the plots tend towards the
disease free equilibrium much faster than when the Janssen vaccination control is implemented. Moreover, we noticed that the three vaccines have different impacts on the dynamics of each infected compartment due to varying vaccine efficacies, and different values of the basic reproduction number.

7. Conclusion

In this work, we have examined the impact of multiple vaccination strategies on the dynamics of COVID-19 in a population using the Atangana-Baleanu Derivative. The existence and uniqueness of solution of the model is proven using Banach’s fixed point theorem. Conditions for the existence of a unique or multiple equilibria are also derived and the model is shown to undergo backward bifurcation under certain scenarios. Local and global asymptotic stability of the equilibria of the model is also proven (under some conditions). The model is fitted using three data sets: The Pfizer, Moderna and Janssen Vaccination data for the city of Texas, United States of America from March 13, 2021 to June 29, 2021. The three vaccination rates \( r_1, r_2 \) and \( r_3 \) corresponding to each vaccine as well as the effective contact rate for COVID-19 transmission, \( \beta \), are estimated. Simulations of the model under different vaccination strategies are carried out. It is observed that the three vaccination strategies not only cause significant reduction in asymptomatic and vaccinated symptomatic new cases but also caused great decrease in the total number of vaccinated symptomatic individuals with severe COVID-19 cases.

The results have shown how the three major vaccines approved for use in the United States can help curtail the spread of COVID-19 infections as well as severe COVID-19 cases. The results of this study are consistent with the recommendations of the World Health Organization (WHO) and the United States Centers for Disease Control and Prevention (CDC), that many of the approved COVID-19 vaccines have “proven to be effective against SARS-CoV-2 infections, including asymptomatic infections and symptomatic cases, severe COVID-19 illness and deaths” [4]. The model can also serve as a template for multiple vaccination study in any country or geographical region. Further studies can discuss the stochastic version of the model. Also, we can consider multiple vaccinations and how these can also help curb the emerging COVID-19 variants of global concern.

Data Availability Statement

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Code Availability

The codes used during the current study are available from the corresponding author on reasonable request.

Authors’ contributions Statement

Andrew Oname: performed the calculations and wrote in part the original draft and edited the final manuscript. Daniel Okuonghae: designed the concept of the paper, supervised the work and edited the final manuscript. Ugochukwu Nwajeri: performed the calculations and wrote in part the original draft. Chibueze Oyengeche: Participated in the investigation, and wrote in part the original draft.

References

[1] E. Dong, H. Du, L. Gardner, Coronavirus COVID-19 global Cases by Johns Hopkins CSSE, Lancet Infect. Dis. (2020) https://gisanddata.maps.arcgis.com/apps/opsdashboard/index.html/bda7594740d40299423467b48e9ecf6 (accessed 3rd August, 2021).
[2] B. Hu, H. Guo, P. Zhou, Z.L. Shi, Characteristics of SARS-CoV-2 and COVID-19, Nat. Rev. Microbiol. 19 (2021) 141–154.
[3] United States Food and Drug Administration, FDA Takes Key Action in Fight Against COVID-19 By Issuing Emergency Use Authorization for First COVID-19 Vaccine, 2020. https://www.fda.gov/news-events/press-announcements/fda-takes-key-action-fight-against-covid-19-issuing-emergency-use-authorization-first-covid-19 (accessed June 17, 2021).
[4] Interim Clinical Considerations for Use of COVID-19 Vaccines Currently Authorized in the United States https://www.cdc.gov/vaccines/covid-19/clinical-considerations/covid-19-vaccines-us.html (accessed July 14, 2021).
[5] United States Food and Drug Administration, FDA Briefing Document Moderna COVID-19 Vaccine, 2020. https://www.fda.gov/media/144434/download (accessed June 17, 2021).
[6] United States Food and Drug Administration, FDA Briefing Document Pfizer-BioNTech COVID-19 Vaccine, 2020. https://www.fda.gov/media/144245/download (accessed June 17, 2021).
[7] United States Food and Drug Administration, FDA Issues Emergency Use Authorization for Third COVID-19 Vaccine, 2021. https://www.fda.gov/news-events/press-announcements/fda-issues-emergency-use-authorization-third-covid-19-vaccine (accessed June 17, 2021).
[8] K.M. Owolabi, A. Atangana, Numerical Methods for Fractional Differentiation, Springer Nature Singapore Ltd., 2019.
[9] A. Oname, C.U. Nnanna, S.C. Inyama, Optimal Control and Cost-Effectiveness Analysis of an HPV-Chlamydia trachomatis co-infection Model, Acta Biotheoretica 2020; https://doi.org/10.1007/s10441-020-09401-z.
[10] A. Khan, H.M. Alshehri, T. Abdeljawad, Q.M. Al-Mdallal, Stability analysis of fractional nabla difference COVID-19 model, Res. Phys. 22 (2021) 103888.
[11] T.N. Sindhu, S. Anum, M.A. Qasem, On the analysis of number of deaths due to Covid-19 outbreak data using a new class of distributions, Res. Phys. 21 (2021) 103747.
[12] A. Shaﬁq, S.A. Lone, T.N. Sindhu, Y. El Khatib, Q.M. Al-Mdallal, T. Muhammad, A new modiﬁed Kies Frchet distribution: Applications of mortality rate of Covid-19, Res. Phys. 28 (2021) 104638.
[13] A. Oname, N. Sene, I. Nometta, C.I. Nwakanma, E.U. Nwafor, N.O. Iheonu, D. Okuonghae, Analysis of COVID-19 and comorbidity co-infection Model, Optim. Contr. Appl. Meth. (2021), https://doi.org/10.1002/oca.2748.
[14] D. Okuonghae, A. Oname, Analysis of a mathematical model for COVID-19 population dynamics in Lagos, Nigeria, Chaos Solitons Fract. 139 (2020) 110032.
[15] A. Oname, D. Okuonghae, U.E. Nwafor, B.U. Odionyenma, A co-infection model for HPV and Syphilis with Optimal Control and Cost-Effectiveness Analysis, Int. J. Biomath. (2021), https://doi.org/10.1142/S1793524521500509.
[16] K.U. Egoue, A. Oname, S.C. Inyama, A co-infection model for Two-Strain Malaria and Cholera with Optimal Control, Int. J. Dyn. Cont. (2021), https://doi.org/10.1007/s40435-020-00748-2.
[17] A. Omame, D. Okuonghae, A co-infection model for oncogenic Human papillomavirus and tuberculosis with optimal control and cost-effectiveness analysis, Optim. Contr. Appl. Meth. (2021), https://doi.org/10.1002/oca.2717.

[18] E. Bonjhay, M.A. Khan, K.O. Okosun, J.F. Gmez-Aguilar, On the co-infection of dengue fever and Zika virus, Optim. Contr. Appl. Meth. 40 (3) (2019) 394-421.

[19] E.O. Alzahrani, W. Ahmad, M.A. Khan, S.J. Malebary, Optimal Control Strategies of Zika Virus Model with Mutant, Comm. Nonl. Sci. Numer. Simul. 93 (2021) 105532, https://doi.org/10.1016/j.cnsns.2020.105532.

[20] S. N dolane, Fractional advection-dispersion equation described by the Caputo left generalized fractional derivative, Palestine, J. Math. 10 (2) (2021) 562–579.

[21] N.H. Sweilam, S.M. Al-Mekhlafi, T. Assiri, et al, Optimal control for cancer treatment mathematical model using Atangana-Baleanu-Caputo fractional derivative, Adv. Differ. Equ. 2020 (2020) 334, https://doi.org/10.1186/s13662-020-02793-9.

[22] N.H. Sweilam, AL-Mekhlafi SM, Baleanu D, A Hybrid Stochastic Fractional Order Coronavirus (2019-nCov) Mathematical Model, Chaos Solitons Fract. 145 (3) (2021) 110762, https://doi.org/10.1016/j.chaos.2021.110762 (in press).

[23] F.A. Rihan, H.J. Alsakaji, Dynamics of a stochastic delay differential model for COVID-19 infection with asymptomatic infected and interacting people: Case study in the UAE, Res. Phys. (2021) 104658.

[24] F.A. Rihan, H.J. Alsakaji, C. Rajivganthi, Stochastic SIRC epidemic model with time-delay for COVID-19, Adv. Differ. Equ. 2020 (2020) 502, https://doi.org/10.1186/s13662-020-02964-8.

[25] A. Omame, S.C. Inyama, Stochastic model and simulation of the prevalence of measles, Int. J. Math. Sci. Eng. 8 (1) (2014) 311–323.

[26] N.H. Sweilama, S.M. Al-Mekhlafi, A. Almutairi, D. Baleanu, A Hybrid Fractional COVID-19 Model with General Population Mask Use: Numerical Treatments, Alexandria Eng. J. 60(30) (2021) 1-14. https://doi.org/10.1016/j.aeje.2021.01.057.

[27] E.C. Chukukere, A. Omame, C.P. Onyenegecha, S.C. Inyama, Mathematical analysis of a model for Chlamydia and Gonorrhea codynamics with optimal control, Res. Phys. 27 (2021) 104566.

[28] A. Omame, D. Okuonghae, R.A. Umana, S.C. Inyama, Analysis of a co-infection model for HPV-TB, Appl. Math. Model. 77 (2020) 881–901.

[29] A. Atangana, D. Baleanu, New fractional derivatives with nonlocal and non-singular kernel: theory and applications to heat transfer model, Therm. Sci. 20 (2) (2016) 763–769.

[30] A. Atangana, A. Secer, A note on fractional order derivatives and table of fractional derivatives of some special functions, ID. 279681, 2013.

[31] N. Sene, SIR epidemic model with Mittag-Leffler fractional derivative, Chaos Solitons Fract. 137 (2020) 109833.

[32] M.A. Khan, A. Atagana, Modelling the dynamics of novel coronavirus (2019-nCOV) with fractional derivative, Alexandria Eng. J. 59 (4) (2020) 2379–2389, https://doi.org/10.1016/j.aej.2020.02.033.

[33] A. Atangana, K.M. Owolabi, New numerical approach for fractional differential equation, Math. Model. Nat. Phenomena 13 (30) (2018) 1.

[34] K.M. Owolabi, Modelling and simulation of a dynamical system with the Atangana-Baleanu fractional derivative, Eur. Phys. J. Plus 133 (2018) 15, https://doi.org/10.1140/epjp/i2018-11863-9.

[35] E. Bonjhay, S.K. Asiedu, Analysis of a Lyapunov fractional-schistosomiasis coinfecion with public health dynamics: Model obtained through Mittag-Leffler function, Discr. Cont. Dyn. Sys.-S 13 (3) (2020) 519–537.

[36] J. Podlubny, Fractional Differential Equations, Maths Sci. Eng., San Diego, 1999.

[37] U.K. Nwajeri, A. Omame, Analysis of a fractional order model for HPV and CT co-infection, Res. Phys. 28 (2021).

[38] A. Omame, M. Abbas, C.P. Onyenegecha, A fractional-order model for COVID-19 and tuberculosis co-infection using Atangana-Baleanu derivative, Chaos Solitons Fract. 151 (1) (2021) 111486.

[39] T. Abdeljawad, F. Madjidi, F. Jarad, N. Sene, On Dynamic Systems in the Frame of Singular Function Dependent Kernel Fractional Derivatives, Math. 7 (2019) 946.

[40] K.A. Abro, J.F. Gomez-Aguilar, A comparison of heat and mass transfer on a Walter’s-B fluid via Caputo-Fabrizio versus Atangana-Baleanu fractional derivatives using the Fox-H function, Euro. Phys. J. Plus 134 (3) (2019) 1–10.

[41] Texas Population, Census Reporter, https://censusreporter.org/profiles/04000US48-texas/ (accessed 26th June, 2021).

[42] P. van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, Math. Biosci. 180 (2002) 29–48.

[43] A. Atangana, J.F. Gomez-Aguilar, Numerical approximation of Riemann-Liouville definition of fractional derivative: From Riemann-Liouville to Atangana-Baleanu, Num. Meth. Part. Diff. Eq. 34 (5) (2017) 1502–1523.

[44] COVID-19 Vaccinations in the US, https://data.cdc.gov/Vaccinations/COVID-19-Vaccinations-in-the-United-States-Jurisdi/unsk-b7fc (accessed 26th June, 2021).

[45] H. Yezep-Martinez, J.F. Gomez-Aguilar, I.O. Sosa, J.M. Reyes, J. Torres-Jimenez, The Feng’s first integral method applied to the nonlinear mKdV space-time fractional partial differential equation, Rev. mex. fis. 62 (4) (2016).

[46] A. Omame, H. Rwizura, M.L. Diagne, et al, COVID-19 and dengue co-infection in Brazil: optimal control and cost-effectiveness analysis, Eur. Phys. J. Plus 136 (2021) 1090, https://doi.org/10.1140/epjp/s13360-021-02030-6.

[47] J. McCall, Genetic algorithms for modelling and optimisation, J. Comput. Appl. Math. 184 (2005) 205–222.