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A fractional order model for Dual Variants of COVID-19 and HIV co-infection via Atangana-Baleanu derivative

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Abstract In this paper, a new mathematical model for dual variants of COVID-19 and HIV co-infection is presented and analyzed. The existence and uniqueness of the solution of the proposed model have been established using the well known Banach fixed point theorem. The model is solved semi-analytically using the Laplace Adomian decomposition Method. The impact of the Atangana-Baleanu fractional derivative on the dynamics of the proposed model is studied. The work also highlights the impact of COVID-19 vaccination on the dynamics of the co-infection of both diseases. The model is fitted to real COVID-19 data from Botswana. The impact of COVID-19 variants on HIV prevalence using simulations is also assessed. Simulation for the class of individuals co-infected with HIV and the wild or Delta COVID-19 variant reveals a significant decrease, as vaccination rate is increased. The impact of fractional order on different epidemiological classes is also studied. Drawing the plot of total infected population with the wild and Delta COVID-19 variants, at different vaccination rates, it is concluded that, as vaccination rate is increased, there is a significant reduction in population infected with the wild and delta COVID-19 variants. The plot of class of individuals co-infected with HIV and the wild or Delta COVID-19 variant is more interesting; as vaccination rate is increased, the co-infected populations experience a significant decrease. Thus, stepping up vaccination against the different variants of COVID-19 could reduce co-infection cases largely, among people already infected with HIV.

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1. Introduction

SARS-CoV-2 co-infections with other microorganisms such as influenza virus, Mycoplasma pneumoniae, cytomegalovirus, Legionella, HIV, and even co-infection with multiple respiratory viruses have been investigated in the literature [1]. Qin et al. [2] observed that the functioning of an immune system is greatly reduced due to co-current infections with both HIV and SARS-COV-2. Suwanwongse and Shabarek [3] studied SARS-CoV-2 infections in patients with Human immune deficiency virus (HIV) and found that CD+ T-cell greatly suffers dysfunction in co-infected patients. The World Health Organization (WHO) has also confirmed individuals co-infected with SARS-CoV-2 and HIV are most likely to suffer from severe illness leading to death [1]. Furthermore, a great increase in cytokine production has been noticed in persons co-infected with HIV and SARS-CoV-2 infection which could lead to increased viral load and subsequent immune suppression [1]. “The HIV pandemic is most severe in Southern Africa. Over 10% of all people infected with HIV/AIDS reside within the region. Adult HIV prevalence exceeds 15% in Eswatini, Botswana, Lesotho and South Africa, while an additional six countries report adult HIV prevalence of at least 10% [4]. It has been observed that the COVID-19 pandemic took the spotlight from HIV cases in Africa [5]”. However, we have the co-existence these two deadly viruses with us. There is, therefore, an urgent need to sensitize governments and agencies, that the surveillance network built for HIV long before now can equally be used for the COVID-19 response so as to combat these deadly menaces.

Studies carried out on the epidemiology of diseases applying classical integer order derivatives have their limitations as they could hardly capture the memory effect. Note that the memory effect means the future state of an operator of a given function depends on the current state and the historical behavior of the state of a dynamic system [10]. Knowing the memory of the past events, the spread of the disease in the future could be controlled easily. The incorporation of this effect is a motivation behind the development of other robust methodologies, such as fractional differential operators which incorporate non-local and non-singular kernels and exponential kernels. It is worth mentioning that the fractional order derivatives and integrals are playing a vital role in epidemiological modeling and other real world problems [14-23] as they capture the memory effect and nonlocal properties [6,10].

Fractional differential operators which depend on a power-law kernel were first presented by Riemann-Liouville and Caputo. But, these operators contain singular kernels which constitute limitations to their use in modeling biological and other physical phenomenon [7]. In order to overcome these limitations, Caputo and Fabrizio (CF) [8] and Atangana and Baleanu (AB) [6] came up with more improved operators which involve the exponential kernel and the generalized Mittag-Leffler function, respectively. Nonetheless, there has been some arguments against the Caputo-Fabrizio derivative that “the kernel is not local; the associated integral is not a fractional operator but just the average of the function and its integral and merely a filter, thereby giving more preference to the usage of Atangana-Baleanu derivative in modelling complex real life phenomena” [6].

Omate et al. [11] studied a co-infection model for COVID-19 and tuberculosis using the Atangana-Baleanu derivative. They showed the effect of COVID-19 re-infection on the dynamics of the co-infection of both diseases and established the conditions for co-existence and elimination of the two diseases. Authors in [12] considered a co-infection model for COVID-19 and diabetes via the fractional derivative with Mittag-Leffler law (AB derivative) and showed that, mass COVID-19 vaccination was necessary to contain the spread of COVID-19 and diabetes co-infections in Indonesia. In [10], author considered an SIR model with delay using the AB derivative. The model was solved using the Homotopy Analysis method. The impacts of the fractional derivative on the dynamics of the states of model were presented via simulations. Jagdev et al., [13] considered a dynamical COVID-19 model and analyzed with the AB derivative. They used the q-homotopy analysis Sumudu transform method (q-HASTM) for solving the model.

In this work, we list our contribution as follows:

i. A new mathematical model for the co-dynamics of different variants of COVID-19 and HIV is considered and studied using the Atangana-Baleanu derivative.

ii. The existence and uniqueness of the solution of the proposed model employing the Banach fixed point theorem has been presented.

iii. The model is solved semi-analytically using the Laplace Adomian decomposition Method.

iv. The impact of the Atangana-Baleanu fractional derivative on the dynamics of the proposed model has been presented.

v. The impact of COVID-19 vaccination on the dynamics of co-infection of COVID-19 and HIV is highlighted.

vi. The impact of COVID-19 variants on HIV prevalence using simulations is also assessed.

1.1. Preliminaries

In this section, we recall some basic concepts from fractional calculus and some known theorems needed in the sequel.

Definition 1.1. The Atangana-Baleanu fractional derivative for a given function of order \( \theta \) in Caputo sense is defined by

\[
a_{\beta}^{\alpha} D_{\beta}^{\alpha} f(t) = \frac{\mathcal{K}(\theta)}{(1-\theta)} \int_{a}^{t} \frac{df(\tau)}{d\tau} E_0[-\theta(t-\tau)^{\theta}] d\tau,
\]

where \( \mathcal{K}(\theta) = (1 - \theta) + \frac{\theta}{\Gamma(\theta)} \), denotes a normalization function satisfying \( \mathcal{K}(0) = \mathcal{K}(1) = 1 \), and \( E_{\theta}(\cdot) \) is the Mittag-Leffler function, defined by,

\[
E_{\theta}(x) = \sum_{k=0}^{\infty} \frac{x^{\theta k}}{\Gamma(\theta k + 1)}, \quad \beta > 0.
\]

Definition 1.2 [6]. Let \( f \in H^{1}(a_{1}, a_{2}), a_{2} > a_{1}, \theta \in [0, 1], \) then the Atangana-Baleanu integral (in Caputo sense) of a function \( f(t) \) of order \( \theta \) is defined by

\[
a_{a_{1}}^{a_{2}} \int_{a_{1}}^{a_{2}} t^{\theta \alpha - 1} f(t) \mathcal{K}(\theta) \frac{\mathcal{K}(\theta)}{(1-\theta)} \int_{a_{1}}^{t} f(\tau)(t-\tau)^{\theta - 1} d\tau.
\]
Definition 1.3 [6]. The Laplace transform of the Atangana-Baleanu fractional derivative of order $\theta$ in Caputo sense is given by

$$\mathcal{L}^{ABC}_0 (t^\theta f(t)) = \mathcal{H}(t) \frac{d^\theta \mathcal{L}[f(t)]}{d\theta^t} - \frac{\theta f(0)}{\theta(1 - \theta)}.$$

where $\mathcal{L}$ is the Laplace transform operator.

Theorem 1.1 [9]. Let $(X, ||.||)$ be a Banach space and $T : X \to X$ a contraction on $X$, that is, there exists a constant $a \in [0, 1)$ such that $||T(x) - T(y)|| \leq a||x - y||$, for all $x, y \in X$. Then

i. $T$ has fixed point $x^* \in X$, that is, $Tx^* = x^*$.

ii. A sequence $\{x_n\}_{n=0}^{\infty}$ given by $x_{n+1} = Tx_n$, for $n = 0, 1, 2, 3, \ldots$, converges to $x^*$.

2. The model

At any given time $t$, the total population $N(t)$ is divided into several mutually exclusive epidemiological states: susceptible $S(t)$, individuals vaccinated against COVID-19 $V(t)$, infectious individuals with the wild COVID-19 variant $C(t)$, infectious individuals with the Delta COVID-19 variant $D(t)$, individuals who have recovered from COVID-19 $R(t)$, individuals with HIV infection $V(t)$, individuals co-infected with the wild COVID-19 variant and HIV $V_{CW}(t)$, and individuals co-infected with the Delta COVID-19 variant and HIV infection $V_{CD}(t)$. The population of unvaccinated susceptibles is increased due to recruitment at the rate $\psi \Lambda$, where $\psi$ is the fraction of newly recruited unvaccinated individuals, and $\Lambda$ is the total recruitment rate. Individuals in this epidemiological state can suffer infection with the wild and delta COVID-19 variants at the rates $\beta_1[C(t) + x_W V_{CW}(t)]$ and $\beta_2[D(t) + x_D V_{CD}(t)]$, respectively. Those in this class can also acquire HIV infection at the rate $\beta_3[V(t) + V_{CW}(t) + V_{CD}(t)]$. Individuals in this class are vaccinated at the rate $\mu$. Natural death is assumed for individuals in this class, just as in other epidemiological states, at the rate $\eta$. The class of vaccinated individuals $V(t)$ is increased due to recruitment at the rate $(1 - \psi) \Lambda$. Individuals in this epidemiological state can suffer infection with the wild and delta COVID-19 variants at the reduced rates $(1 - \theta)\beta_1[C(t) + x_W V_{CW}(t)]$ and $(1 - \zeta)\beta_2[D(t) + x_D V_{CD}(t)]$, respectively, where $\theta$ and $\zeta$ are the efficacies of the vaccine against the wild and delta variants. Those in this class can also acquire HIV infection at the rate $\beta_3[V(t) + V_{CW}(t) + V_{CD}(t)]$. Also, individuals infected with the wild or delta COVID-19 variant can suffer additional infection with HIV at the rates $\zeta_1[C(t) + x_W V_{CW}(t)]$ and $\zeta_2[D(t) + x_D V_{CD}(t)]$, where $\zeta$ is the modification parameter accounting for susceptibility to COVID-19 by HIV infected individuals. We have also assumed recovery only from COVID-19, since no cure yet for HIV. Also co-infected individuals can transmit either COVID-19 or HIV, and not mixed infections concurrently. The model is given by the following system of non-linear ordinary differential equations (model parameters are explained in Table 1):

| Parameter | Description | Value | Reference |
|-----------|-------------|-------|-----------|
| $\psi$    | Fraction of newly recruited unvaccinated COVID-19 | 0.5   | Assumed   |
| $\Lambda$ | Recruitment rate | $\frac{1.480.571}{365 \text{ per day}}$ | [29] |
| $\mu$    | COVID-19 vaccination rate | 0.442 | [30] |
| $\beta_1$ | Wild COVID-19 contact rate | $2.2003 \times 10^{-7}$ | Fitted |
| $\beta_2$ | Delta COVID-19 contact rate | $6.5 \times 10^{-7}$ | Fitted |
| $\beta_3$ | HIV contact rate | $0.015$ | [33] |
| $\theta$ | COVID-19 vaccine efficacy against the wild variant | 0.90  | [25] |
| $\zeta$  | COVID-19 vaccine efficacy against the Delta variant | 0.60  | [25] |
| $x_W, x_D$ | Modification parameter for increased transmissibility of COVID-19 among co-infected individuals due to increased viral load | 1.0  | Assumed |
| $\phi_1$ | COVID-19 recovery rates for individuals in compartments $C(t)$ | 0.002 | Fitted |
| $\phi_2$ | COVID-19 recovery rates for individuals in compartments $D(t)$ | 0.007 | Fitted |
| $\phi_3, \phi_4$ | COVID-19 recovery rates for individuals in compartments $V_{CW}$ and $V_{CD}$, respectively | $1/\phi$ | Assumed |
| $\eta$ | Natural death rate | $\frac{0.2425}{365 \text{ per day}}$ | [29] |
| $\delta_W$ | Wild COVID-19 induced death rate | 0.0017 | Fitted |
| $\delta_D$ | Delta COVID-19 induced death rate | 0.0075 | Fitted |
| $\delta_V$ | HIV induced death rate | $\frac{0.2425}{365 \text{ per day}}$ | [33] |
| $\delta_W, \delta_D$ | Disease induced death rates for co-infected individuals in $V_{CW}$ and $V_{CD}$ compartments, respectively | 0.0015 | Assumed |
| $\zeta$ | Modification parameter for increased susceptibility to COVID-19 by HIV/AIDS infected individuals | 1.2  | [3] |
Theorem 2.1. The closed set
\[ \mathcal{S} = \{ (\mathcal{S}, \mathcal{V}, \mathcal{W}, \mathcal{D}, \mathcal{R}, \mathcal{F}, \mathcal{F}_C, \mathcal{F}_{CD}) \in \mathbb{R}^8_+ : 0 \leq \mathcal{S} + \mathcal{V} + \mathcal{W} + \mathcal{D} + \mathcal{R} + \mathcal{F} + \mathcal{F}_C + \mathcal{F}_{CD} \leq \Lambda / \eta \}, \]

is positively invariant with respect to the model (1).

Proof. Adding all the equations of the model (1), gives

\[ F = \begin{pmatrix}
\beta_1 [\mathcal{S} + (1-\vartheta)\mathcal{V}] \\
0 & \beta_2 [\mathcal{S} + (1-\zeta)\mathcal{V}] & 0 & 0 & z_w \beta_1 [\mathcal{S} + (1-\vartheta)\mathcal{V}] & 0 & 0 \\
0 & 0 & \beta_3 [\mathcal{S} + (1-\zeta)\mathcal{V}] & 0 & z_d \beta_2 [\mathcal{S} + (1-\zeta)\mathcal{V}] & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0
\end{pmatrix}, \]

which can be re-written as:

\[ F_{\mathcal{S}}^0 \mathcal{S} + F_{\mathcal{V}}^0 \mathcal{V} + F_{\mathcal{W}}^0 \mathcal{W} + F_{\mathcal{D}}^0 \mathcal{D} + F_{\mathcal{R}}^0 \mathcal{R} + F_{\mathcal{F}}^0 \mathcal{F} + F_{\mathcal{F}_C}^0 \mathcal{F}_C + F_{\mathcal{F}_{CD}}^0 \mathcal{F}_{CD} \leq \Lambda - \eta N. \]

Applying the Laplace transform on the above inequality we have

\[ N(t) \leq \frac{\mathcal{S}^0(0)}{\mathcal{S}(0) + (1-\vartheta)\eta} N(0) + \frac{(1-\theta)\Lambda}{\mathcal{S}(0) + (1-\theta)\eta}, \]

\[ E_{\mathcal{S}}^0 \left( -\frac{\theta\eta}{\mathcal{S}(0) + (1-\theta)\eta} \mathcal{S} \right) + \frac{\theta\Lambda}{\mathcal{S}(0) + (1-\theta)\eta} E_{\mathcal{S}}^{\theta+1} \left( -\frac{\theta\eta}{\mathcal{S}(0) + (1-\theta)\eta} \mathcal{S} \right), \]

The Mittag-Leffler function \( E_\eta ax \) is asymptotic in nature [6]. Thus, we have that \( N(t) \leq \frac{\Lambda}{\eta} \) as \( t \to \infty \). As a result, the system (1) has the solution in \( \mathcal{S} \). Thus, the given system is positively invariant. □

2.2. Basic reproduction number of the co-infection model (1)

The model (1) has a DFE given by

\[ \mathcal{J}_0 = \left( \mathcal{S}^0, \mathcal{V}^0, \mathcal{W}^0, \mathcal{D}^0, \mathcal{R}^0, \mathcal{F}^0, \mathcal{F}_C^0, \mathcal{F}_{CD}^0 \right) = \left( \frac{\alpha_{\mathcal{S}}}{\vartheta + \mu + \alpha_{\mathcal{V}}}, 0, 0, 0, 0, 0, 0, 0 \right). \]

The basic reproduction number of the model (1) is calculated using the same approach in [24]. The transfer matrices are

\[ V = \begin{pmatrix}
\Omega_1 & 0 & 0 & 0 & 0 \\
0 & \Omega_2 & 0 & 0 & 0 \\
0 & 0 & -\phi_3 & -\phi_4 & 0 \\
0 & 0 & 0 & \Omega_4 & 0 \\
0 & 0 & 0 & 0 & \Omega_5
\end{pmatrix}, \]

where, the inverse of \( V \) is given by

\[ V^{-1} = \begin{pmatrix}
\frac{1}{\Omega_1} & 0 & 0 & 0 & 0 \\
0 & \frac{1}{\Omega_2} & 0 & 0 & 0 \\
0 & 0 & \frac{1}{\phi_3} & \frac{1}{\phi_4} & 0 \\
0 & 0 & 0 & \frac{1}{\Omega_4} & 0 \\
0 & 0 & 0 & 0 & \frac{1}{\Omega_5}
\end{pmatrix}, \]
\( \Omega = \phi_1 + \eta + \delta_W, \Omega_2 = \phi_2 + \eta + \delta_D, \Omega_3 = \eta + \delta_W, \Omega_4 = \phi_3 + \eta + \delta_V + \delta_D. \)

The model basic reproduction number is given by \( R_0 = \max\{R_{0W}, R_{0D}, R_{0V}\} \) where \( R_{0W} \) and \( R_{0D} \) and \( R_{0V} \) are, the wild and delta SARS-CoV-2 and HIV associated reproduction numbers, respectively, given by
\[
R_{0W} = \frac{\beta_1 N (1-\vartheta)(\eta + \mu)}{\eta (\eta + \delta_W)}, \quad R_{0D} = \frac{\beta_2 N (1-\zeta)(\eta + \mu)}{\eta (\eta + \delta_D)}, \quad R_{0V} = \frac{\beta_3 N}{\eta (\eta + \delta_V)}.
\]

2.3. Local asymptotic stability of the disease free equilibrium (DFE) of the co-infection model.

**Theorem 2.2.** The DFE, \( \mathcal{X}_0 \), of the model (1) is locally asymptotically stable (LAS) if \( R_0 < 1 \), and unstable if \( R_0 > 1 \).

**Proof.** The local stability of the model (1) is analyzed by the Jacobian matrix of the system (1) evaluated at the COVID-19-free equilibrium, \( \mathcal{X}_0 \), given by:
\[
\begin{pmatrix}
-(\mu + \eta) & 0 & -\beta_1 S & -\beta_2 S \\
\mu & -\eta & -\beta_1 (1-\vartheta) & -\beta_2 (1-\zeta)
\end{pmatrix}
\]
\[
\begin{pmatrix}
\begin{pmatrix}
0 & 0 & \beta_1 N (1-\vartheta) + (1-\vartheta) S - \Omega_1 \\
0 & 0 & \beta_2 N (1-\zeta) + (1-\zeta) V - \Omega_2 \\
0 & 0 & \phi_1 - \Omega_3 \\
0 & 0 & \phi_2 - \Omega_4 \\
0 & 0 & \phi_3 + \frac{\phi_4 N}{\gamma} - \Omega_5
\end{pmatrix}
\end{pmatrix}
\]

The characteristic equation of the above matrix is given by
\[
(\lambda + \eta)^2 (\lambda + \eta + \mu)(\lambda + \Omega_1)(\lambda + \Omega_2) - \lambda A_{\beta_1} = 0.
\]

The eigenvalues are given by:
\[
\lambda_1 = -\eta \ (\text{with multiplicity of two}), \quad \lambda_2 = -\eta - \mu, \quad \lambda_3 = -\Omega_4, \quad \lambda_4 = -\Omega_5,
\]
and the roots of the equations below:
\[
\lambda + \Omega_1 (1 - R_{0W}) = 0, \quad \lambda + \Omega_2 (1 - R_{0D}) = 0, \quad \lambda + \Omega_3 (1 - R_{0V}) = 0,
\]

Applying the Routh Hurwitz criterion, the three equations in (10) will have roots with negative real parts if and only if \( R_{0W} < 1, R_{0D} < 1, \) and \( R_{0V} < 1 \) respectively. Thus, the DFE, \( \mathcal{X}_0 \) is locally asymptotically stable if \( R_0 = \max\{R_{0W}, R_{0D}, R_{0V}\} < 1 \). The epidemiological implication of Theorem 2.2 is that the diseases can be eliminated from the population when \( R_0 < 1 \) and if the initial sizes of the population of the model are in the region of attraction of the DFE. □

2.4. Existence of solution

Let us re-write model (1) in the form:
\[
\begin{cases}
\frac{dX}{dt} = \xi(t, Z(t)) \\
Z(0) = Z_0,
\end{cases}
\]

where the vector \( Z(t) = (S(t), V(t), W(t), D(t), R(t), W(t), V(t), F(t), C(t)) \in \mathbb{R}^8 \), for \( t \in [0, T_{max}] \), represents the compartmental states of the model and \( \xi(t) \) denotes a continuous vector defined by the right hand sides of (1).

\[
\begin{pmatrix}
0 & -\beta_1 S & -\beta_2 S & 0 & 0 & 0 & 0 & 0 \\
-\eta & 0 & -\beta_1 (1-\vartheta) & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & -\beta_2 (1-\zeta) & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & -\eta & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & -\eta & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & -\eta \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & -\eta \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & -\eta
\end{pmatrix}
\]

**Theorem 2.3.** The functions \( \xi(t) \), for \( n = 1, 2, 3, 4, 5, 6, 7, 8 \) satisfy the Lipschitz condition for the second argument for:
\[
\sup_{0 \leq r \leq 1} \|S\| \leq c_1, \quad \sup_{0 \leq r \leq 1} \|V\| \leq c_2, \quad \sup_{0 \leq r \leq 1} \|W\| \leq c_3, \quad \sup_{0 \leq r \leq 1} \|D\| \leq c_4, \quad \sup_{0 \leq r \leq 1} \|R(t)\| \leq c_5, \quad \sup_{0 \leq r \leq 1} \|F(t)\| \leq c_6, \quad \sup_{0 \leq r \leq 1} \|C(t)\| \leq c_7, \quad c_1 > 0, j = 1, 2, \ldots, 8.
\]

**Proof.** Consider \( \xi(t, \mathcal{F}(t)) \), where \( \mathcal{F} \) is another solution, say,
Take $\zeta_2(t, \mathcal{G}_V(t))$, where $\mathcal{G}_V$ is another solution, say,

$$
\|\zeta_2(\mathcal{G}_V) - \zeta_2(\mathcal{G}_V)\| \leq \| (1 - \psi) \mathcal{G} + \mu \mathcal{F} - ((1 - \psi) \beta_1(\mathcal{F}_W + z_W \mathcal{F}_C) + (1 - \zeta) \beta_2(\mathcal{F}_D + z_D \mathcal{F}_C) + \eta + \beta_3(\mathcal{F}_W + z_W \mathcal{F}_C + z_D \mathcal{F}_C)) \| \mathcal{G}_V \\
- (1 - \psi) \mathcal{G} - \mu \mathcal{F} - ((1 - \psi) \beta_1(\mathcal{F}_W + z_W \mathcal{F}_C) + (1 - \zeta) \beta_2(\mathcal{F}_D + z_D \mathcal{F}_C) + \eta + \beta_3(\mathcal{F}_W + z_W \mathcal{F}_C + z_D \mathcal{F}_C)) \| \mathcal{G}_V \\
\leq \| (1 - \psi) \beta_1(\mathcal{F}_W + z_W \mathcal{F}_C) + (1 - \zeta) \beta_2(\mathcal{F}_D + z_D \mathcal{F}_C) + \eta + \beta_3(\mathcal{F}_W + z_W \mathcal{F}_C + z_D \mathcal{F}_C)) \| \| \mathcal{G}_V - \mathcal{G}_V \|
$$

(13)

where $\varphi_{\zeta_2} = (1 - \psi) \beta_1[\mathcal{F}_W + z_W \mathcal{F}_C] + (1 - \zeta) \beta_2[\mathcal{F}_D + z_D \mathcal{F}_C] + \eta + \beta_3[\mathcal{F}_W + z_W \mathcal{F}_C + z_D \mathcal{F}_C]

Consider $\zeta_3(t, \mathcal{G}_W(t))$, where $\mathcal{G}_W$ is another solution, say,

$$
\| \zeta_3(\mathcal{G}_W) - \zeta_3(\mathcal{G}_W) \| \leq \| \beta_1[\mathcal{F}_W + z_W \mathcal{F}_C] + (1 - \psi) \mathcal{F}_V - ((1 - \psi) \beta_1(\mathcal{F}_V + z_W \mathcal{F}_C + z_D \mathcal{F}_C) + \eta + \delta_W) \| \mathcal{G}_W \\
- \beta_1(\mathcal{F}_W + z_W \mathcal{F}_C) + (1 - \psi) \mathcal{F}_V - ((1 - \psi) \beta_1(\mathcal{F}_V + z_W \mathcal{F}_C + z_D \mathcal{F}_C) + \eta + \delta_W) \| \mathcal{G}_W \\
\leq \| \beta_1[\mathcal{F}_V + (1 - \psi) \mathcal{F}_V + (1 - \psi) \beta_1(\mathcal{F}_V + z_W \mathcal{F}_C + z_D \mathcal{F}_C) + \eta + \delta_W) \| \| \mathcal{G}_W - \mathcal{G}_W \|
$$

(14)

where $\varphi_{\zeta_3} = \beta_1[\mathcal{F}_V + (1 - \psi) \mathcal{F}_V] + (1 - \psi) \beta_1[\mathcal{F}_V + z_W \mathcal{F}_C + z_D \mathcal{F}_C] + \eta + \delta_W)

Consider $\zeta_4(t, \mathcal{G}_D(t))$, where $\mathcal{G}_D$ is another solution, say,

$$
\| \zeta_4(\mathcal{G}_D) - \zeta_4(\mathcal{G}_D) \| \leq \| \beta_2[\mathcal{F}_D + z_D \mathcal{F}_C] + (1 - \zeta) \mathcal{F}_V - ((1 - \zeta) \beta_2(\mathcal{F}_D + z_D \mathcal{F}_C) + \eta + \delta_D) \| \mathcal{G}_D \\
- \beta_2(\mathcal{F}_D + z_D \mathcal{F}_C) + (1 - \zeta) \mathcal{F}_V - ((1 - \zeta) \beta_2(\mathcal{F}_D + z_D \mathcal{F}_C) + \eta + \delta_D) \| \mathcal{G}_D \\
\leq \| \beta_2[\mathcal{F}_V + (1 - \zeta) \mathcal{F}_V + (1 - \zeta) \beta_2(\mathcal{F}_D + z_D \mathcal{F}_C) + \eta + \delta_D) \| \| \mathcal{G}_D - \mathcal{G}_D \|
$$

(15)

where $\varphi_{\zeta_4} = \beta_2[\mathcal{F}_V + (1 - \zeta) \mathcal{F}_V] + (1 - \zeta) \beta_2[\mathcal{F}_D + z_D \mathcal{F}_C] + \eta + \delta_D)

Following similar manner, and applying triangle inequality we have:

$$
\| \zeta_2(\mathcal{G}_V) - \zeta_2(\mathcal{G}_V) \| \leq \varphi_{\zeta_2} \| \mathcal{G}_V - \mathcal{G}_V \|, \| \zeta_3(\mathcal{G}_W) - \zeta_3(\mathcal{G}_W) \| \leq \varphi_{\zeta_3} \| \mathcal{G}_W - \mathcal{G}_W \|, \| \zeta_4(\mathcal{G}_D) - \zeta_4(\mathcal{G}_D) \| \leq \varphi_{\zeta_4} \| \mathcal{G}_D - \mathcal{G}_D \|
$$

(16)

where $\varphi_{\zeta_2} = (1 - \psi) \beta_1[\mathcal{F}_W + z_W \mathcal{F}_C] + (1 - \zeta) \beta_2[\mathcal{F}_D + z_D \mathcal{F}_C] + \eta + \beta_3[\mathcal{F}_W + z_W \mathcal{F}_C + z_D \mathcal{F}_C],

\varphi_{\zeta_3} = \beta_1[\mathcal{F}_V + (1 - \psi) \mathcal{F}_V] + (1 - \psi) \beta_1[\mathcal{F}_V + z_W \mathcal{F}_C + z_D \mathcal{F}_C] + \eta + \delta_W),

\varphi_{\zeta_4} = \beta_2[\mathcal{F}_V + (1 - \zeta) \mathcal{F}_V] + (1 - \zeta) \beta_2[\mathcal{F}_D + z_D \mathcal{F}_C] + \eta + \delta_D),

\varphi_{\zeta_5} = (\eta + \beta_3[\mathcal{F}_W + z_W \mathcal{F}_C + z_D \mathcal{F}_C],

\varphi_{\zeta_6} = \beta_3[\mathcal{F}_W + z_W \mathcal{F}_C] + \beta_3[\mathcal{F}_W + z_W \mathcal{F}_C] + \beta_3[\mathcal{F}_W + z_W \mathcal{F}_C] + \delta \psi + \delta_W),

\varphi_{\zeta_7} = \beta_3[\mathcal{F}_W + z_W \mathcal{F}_C] + \beta_3[\mathcal{F}_W + z_W \mathcal{F}_C] + \beta_3[\mathcal{F}_W + z_W \mathcal{F}_C] + \delta \psi + \delta_W).
Thus, \( \xi_\eta \), fulfills the Lipschitz condition, for 
\( \eta = 1, 2, 3, 4, 5, 6, 7, 8 \). \( \square \)

**Theorem 2.4.** There exists a unique solution in \( C([0, \mathcal{F}_{\text{max}}], \mathbb{R}^8) \) to the initial value problem (11) provided that
\[
\left( \frac{1 - \theta}{\mathcal{K}(\theta)} + \frac{\theta}{\mathcal{K}(\theta) \Gamma(\theta + 1)} \right) \mathcal{F}_{\text{max}} < 1. \tag{17}
\]

**Proof.** Applying the Atangana-Baleanu fractional integral on the both sides of (11), we have
\[
Z(t) = Z_0 + \frac{1 - \theta}{\mathcal{K}(\theta)} \xi(t, Z(t)) + \frac{\theta}{\mathcal{K}(\theta) \Gamma(\theta)} \int_0^t (t - \tau)^{\theta - 1} \xi(\tau, Z(\tau)) d\tau. \tag{18}
\]

Let \( \mathcal{H} = (0, \mathcal{F}_{\text{max}}) \).

Now define the operator \( \mathcal{G} : C(\mathcal{H}, \mathbb{R}^8) \rightarrow C(\mathcal{H}, \mathbb{R}^8) \) by:
\[
\mathcal{G}[Z](t) = V(t) \tag{19}
\]

where,
\[
V(t) = Z_0 + \frac{1 - \theta}{\mathcal{K}(\theta)} \xi(t, V(t)) + \frac{\theta}{\mathcal{K}(\theta) \Gamma(\theta)} \int_0^t (t - \tau)^{\theta - 1} \xi(\tau, V(\tau)) d\tau.
\]

The supremum norm on \( C(\mathcal{H}, \mathbb{R}^8) \), \( \| V \|_\mathcal{H} \), is given by:
\[
\| V \|_\mathcal{H} = \sup_{t \in \mathcal{H}} \| V(t) \|, \quad \forall \ V \in C(\mathcal{H}, \mathbb{R}^8).
\]

Clearly, \( C(\mathcal{H}, \mathbb{R}^8) \) equipped with \( \| \cdot \|_\mathcal{H} \) is a Banach space.

Suppose, \( Y \) is the fixed point of the operator \( \mathcal{G} : C(\mathcal{H}, \mathbb{R}^8) \rightarrow C(\mathcal{H}, \mathbb{R}^8) \), then \( Y \) becomes a solution of (11) and we have,
\[
\mathcal{G}[Y](t) = Y(t),
\]

where,
\[
Y(t) = Z_0 + \frac{1 - \theta}{\mathcal{K}(\theta)} \xi(t, Y(t)) + \frac{\theta}{\mathcal{K}(\theta) \Gamma(\theta)} \int_0^t (t - \tau)^{\theta - 1} \xi(\tau, Y(\tau)) d\tau.
\]

Applying eq (19), we have that,
\[
\| \mathcal{G}[Y] - \mathcal{G}[Y](t) \|_\mathcal{H} = \left\| Z_0 + \frac{1 - \theta}{\mathcal{K}(\theta)} \xi(t, V(t)) + \frac{\theta}{\mathcal{K}(\theta) \Gamma(\theta)} \int_0^t (t - \tau)^{\theta - 1} \xi(\tau, V(\tau)) d\tau - \left[ Z_0 + \frac{1 - \theta}{\mathcal{K}(\theta)} \xi(t, Y(t)) + \frac{\theta}{\mathcal{K}(\theta) \Gamma(\theta)} \int_0^t (t - \tau)^{\theta - 1} \xi(\tau, Y(\tau)) d\tau \right] \right\|
\]
\[
\leq \left\| \frac{1 - \theta}{\mathcal{K}(\theta)} \left( \xi(t, V(t)) - \xi(t, Y(t)) \right) + \frac{\theta}{\mathcal{K}(\theta) \Gamma(\theta)} \int_0^t (t - \tau)^{\theta - 1} \left( \xi(\tau, V(\tau)) - \xi(\tau, Y(\tau)) \right) d\tau \right\|.
\]

Since the operator \( \xi \) satisfies the Lipschitz condition, we have
\[
\| \mathcal{G}[V] - \mathcal{G}[Y](t) \|_\mathcal{H} \leq \frac{1 - \theta}{\mathcal{K}(\theta)} \| V(t) - Y(t) \| + \frac{\theta}{\mathcal{K}(\theta) \Gamma(\theta)} \int_0^t (t - \tau)^{\theta - 1} \| V(\tau) - Y(\tau) \| d\tau,
\]
\[
\leq \frac{1 - \theta}{\mathcal{K}(\theta)} \| V(t) - Y(t) \| + \frac{\theta}{\mathcal{K}(\theta) \Gamma(\theta)} \left( \int_0^t (t - \tau)^{\theta - 1} \| V(\tau) - Y(\tau) \| d\tau \right)^{\| V \|_\mathcal{H}},
\]
\[
\leq \frac{(1 - \theta)\mathcal{F}_{\text{max}}}{\mathcal{K}(\theta) \Gamma(\theta + 1)} \| V - Y \|_\mathcal{H}, \tag{21}
\]

where \( \mathcal{F}_{\text{max}} \) is the value of the integral \( \int_0^t (t - \tau)^{\theta - 1} d\tau \), evaluated at the final time, \( \mathcal{F}_{\text{max}} \).

Thus if the condition (17) holds then
\[
\| \mathcal{G}[V] - \mathcal{G}[Y] \| \leq \left( \frac{1 - \theta}{\mathcal{K}(\theta)} + \frac{\theta}{\mathcal{K}(\theta) \Gamma(\theta + 1)} \right) \| V - Y \| \text{ and the operator } \mathcal{G} \text{ becomes a contraction. Therefore } \mathcal{G} \text{ has a unique fixed point which is a solution to the initial value problem (11) and hence a solution to the system (1)}. \quad \square
\]

### 3. Numerical scheme

#### 3.1. Basic idea of Laplace Adomian Decomposition Method for FDEs

Consider the following system of fractional differential equations [26,27]
\[
\begin{align*}
\frac{d^\alpha y_i(t)}{d\tau^\alpha} &= L(y_1, y_2, y_3, \ldots, y_n) + N(y_1, y_2, y_3, \ldots, y_n), \quad t \geq 0, \ i = 1, 2, 3, \ldots, n. \tag{22}
\end{align*}
\]

with initial conditions, \( y_i(0) = y_{i0} \).

The fractional derivative in (22) is expressed in the Atangana Baleanu derivative in Caputo sense. The Linear and nonlinear terms are represented by \( L \) and \( N \), respectively. Applying the Laplace transform to both sides of the system (22), we have
\[
\mathcal{L}\left\{ \frac{d^\alpha y_i(t)}{d\tau^\alpha} \right\} = \mathcal{L}\left\{ L(y_1, y_2, y_3, \ldots, y_n) + N(y_1, y_2, y_3, \ldots, y_n) \right\} \tag{23}
\]

From the definition of Laplace transform for the Atangana-Baleanu derivative, we have that,
\[
\mathcal{L}\left\{ \frac{d^\alpha y_i(t)}{d\tau^\alpha} \right\}(s) = \mathcal{K}(\theta) s^\alpha \mathcal{L}\{ y_i(t) \}(s) - s^{\alpha - 1} y_{i0},
\]

so that
\[
\mathcal{K}(\theta) s^\alpha \mathcal{L}\{ y_i(t) \}(s) - s^{\alpha - 1} y_{i0} = \mathcal{L}\{ L(y_1, y_2, y_3, \ldots, y_n) + N(y_1, y_2, y_3, \ldots, y_n) \}
\]

\[
\mathcal{L}\{ L(y_1, y_2, y_3, \ldots, y_n) + N(y_1, y_2, y_3, \ldots, y_n) \} \tag{24}
\]

\]
which can be written as
\[
\mathcal{L} \{ y_i(t) \} = \frac{y_i(0)}{s} + \frac{s^\theta (1-\theta)}{s^\theta \mathcal{K}(\theta)} + N(y_1, y_2, y_3, \ldots, y_n)
\]

According to the Adomian decomposition method, the solution will be in the following series type

\[
y_i(t) = \sum_{j=0}^{\infty} y_{ij}(t), i = 1, 2, 3, \ldots, n.
\]

By the Adomian Decomposition Method, the nonlinear terms \( N(y_1, y_2, y_3, \ldots, y_n) \) can be decomposed as \( \sum_{j=0}^{\infty} A_{ij} \), where \( i = 1, 2, 3, \ldots, n \), and the polynomial \( A_{ij} \) is defined thus,

\[
A_{ij} = \sum_{j=0}^{\infty} A_{ij} = \frac{1}{j!} \left. \frac{d^j}{dt^j} \left[ \sum_{n=0}^{\infty} \int_{0}^{t} y_{ij}(\tau) \, d\tau \right] \right|_{t=0}
\]

Applying Eqs. (24)–(27) in system (22), we have

\[
\mathcal{L} \{ \sum_{j=0}^{\infty} y_{ij}(t) \} = \frac{y_i(0)}{s} + \frac{s^\theta (1-\theta)}{s^\theta \mathcal{K}(\theta)} + \mathcal{L} \{ L(y_{1j}, y_{2j}, y_{3j}, \ldots, y_{nj}) + A_0 \}
\]

Matching the terms on both sides of (28), we obtain

\[
\mathcal{L} \{ y_{0m}(t) \} = \frac{y_{0m}(0)}{s}, \quad \mathcal{L} \{ y_{10}(t) \} = \frac{s^\theta (1-\theta)+\theta}{s^\theta \mathcal{K}(\theta)} \mathcal{L} \{ L(y_{10}, y_{20}, y_{30}, \ldots, y_{nm}) + A_0 \}
\]

\[
\mathcal{L} \{ y_{i+1}(t) \} = \frac{s^\theta (1-\theta)+\theta}{s^\theta \mathcal{K}(\theta)} \mathcal{L} \{ L(y_{ij}, y_{2j}, y_{3j}, \ldots, y_{nj}) + A_0 \}
\]
Applying the inverse Laplace transform, we obtain

\[ y_0(t) = \mathcal{L}^{-1}\left[ \frac{\omega_0}{s} \right], \]

\[ \ldots \]

\[ y_{(n)}(t) = \mathcal{L}^{-1}\left\{ \frac{1}{s} \left( \mathcal{L}^{-1}\left[ \mathcal{L}(y_{(n)}(t), y_{(n-1)}(t), \ldots, y_0(t), A_{(n)}) \right] + A_{(n)} \right) \right\} \]

and so on. Hence we obtain the required solution

\[ y_0(y_1, y_2, y_3, \ldots, y_n) = y_0(t) + y_1(t) + y_2(t) + \ldots \]

(31)

which can be written as

\[ \mathcal{L}^{(s)}(f(t)) = \frac{\omega_0}{s} + \frac{\omega_1}{s^2} + \ldots \]

\[ \mathcal{L}^{(s)}(g(t)) = \frac{\omega_0}{s} + \frac{\omega_1}{s^2} + \ldots \]

\[ \mathcal{L}^{(s)}(h(t)) = \frac{\omega_0}{s} + \frac{\omega_1}{s^2} + \ldots \]

\[ \mathcal{L}^{(s)}(i(t)) = \frac{\omega_0}{s} + \frac{\omega_1}{s^2} + \ldots \]

\[ \mathcal{L}^{(s)}(j(t)) = \frac{\omega_0}{s} + \frac{\omega_1}{s^2} + \ldots \]

(32)

According to the Adomian decomposition method, the solution will be in the following series type

\[ \mathcal{S}(t) = \sum_{n=0}^{\infty} \mathcal{S}_n(t), \quad \mathcal{V}_1(t) = \sum_{n=0}^{\infty} \mathcal{V}_{1n}(t), \quad \mathcal{W}_n(t) = \sum_{n=0}^{\infty} \mathcal{W}_{nn}(t), \quad \mathcal{D}_n(t) = \sum_{n=0}^{\infty} \mathcal{D}_{nn}(t), \]

(34)

The nonlinear terms in the model (1) can be decomposed as

\[ \mathcal{S}(t) = \sum_{n=0}^{\infty} \mathcal{S}_n(t), \quad \mathcal{V}_1(t) = \sum_{n=0}^{\infty} \mathcal{V}_{1n}(t), \quad \mathcal{W}_n(t) = \sum_{n=0}^{\infty} \mathcal{W}_{nn}(t), \quad \mathcal{D}_n(t) = \sum_{n=0}^{\infty} \mathcal{D}_{nn}(t) \]
Applying the inverse Laplace transform, we obtain

\[
\sum_{n=0}^{\infty} \mathcal{A}_n(\mathcal{V}_w, \mathcal{F}) = \mathcal{V}_w(t) \mathcal{F}(t), \quad \sum_{n=0}^{\infty} \mathcal{A}_n(\mathcal{V}_{cw}, \mathcal{F}) = \mathcal{V}_{cw}(t) \mathcal{F}(t), \quad \sum_{n=0}^{\infty} \mathcal{A}_n(\mathcal{V}_d, \mathcal{F}) = \mathcal{V}_d(t) \mathcal{F}(t),
\]

where the polynomial \(A_n(x, y)\) is defined as

\[
A_n(x, y) = \sum_{j=0}^{n} \frac{d^n}{dx^n} \left[ \sum_{j=0}^{n} \sum_{l=0}^{j} \hat{A}_{j,l}(t) \hat{A}_{j,l}(t) \right]_{j=0}^{n}
\]

(35)

Particularly, we have that

\[
\mathcal{A}_{10}(\mathcal{V}_w, \mathcal{F}) = \mathcal{V}_w(0) \mathcal{F}(0), \quad \mathcal{A}_{11}(\mathcal{V}_w, \mathcal{F}) = \mathcal{V}_w(1) \mathcal{F}(1) + \mathcal{V}_w(0) \mathcal{F}(1), \quad \mathcal{A}_{12}(\mathcal{V}_w, \mathcal{F}) = \mathcal{V}_w(2) \mathcal{F}(0) + \mathcal{V}_w(1) \mathcal{F}(1) + \mathcal{V}_w(0) \mathcal{F}(2),
\]

\[
\mathcal{A}_{13}(\mathcal{V}_w, \mathcal{F}) = \mathcal{V}_w(3) \mathcal{F}(0) + \mathcal{V}_w(2) \mathcal{F}(1) + \mathcal{V}_w(1) \mathcal{F}(2) + \mathcal{V}_w(0) \mathcal{F}(3),
\]

\[
\mathcal{A}_{14}(\mathcal{V}_w, \mathcal{F}) = \mathcal{V}_w(4) \mathcal{F}(0) + \mathcal{V}_w(3) \mathcal{F}(1) + \mathcal{V}_w(2) \mathcal{F}(2) + \mathcal{V}_w(1) \mathcal{F}(3) + \mathcal{V}_w(0) \mathcal{F}(4)
\]

(36)

Applying the inverse Laplace transform, we obtain

\[
\mathcal{V}_w(t) = \mathcal{F}(0), \quad \mathcal{V}_w(t) = \mathcal{V}_w(0), \quad \mathcal{V}_w(t) = \mathcal{V}_w(0), \quad \mathcal{V}_w(t) = \mathcal{V}_w(0), \quad \mathcal{V}_w(t) = \mathcal{V}_w(0), \quad \mathcal{V}_w(t) = \mathcal{V}_w(0), \quad \mathcal{V}_w(t) = \mathcal{V}_w(0)
\]

\[
\mathcal{V}_{cw}(t) = \mathcal{V}_{cw}(0), \quad \mathcal{V}_{cw}(t) = \mathcal{V}_{cw}(0)
\]

\[
\mathcal{F}(t) = \left[ \frac{1}{\eta \beta} \left( \frac{1}{\eta \beta} + \frac{\alpha}{\eta \beta} \right) \right]^{1/2} \left[ \frac{1}{\eta \beta} \left( \frac{1}{\eta \beta} + \frac{\alpha}{\eta \beta} \right) \right]^{1/2}
\]

\[
\mathcal{G}_1(t) = \left[ \frac{1}{\eta \beta} \left( \frac{1}{\eta \beta} + \frac{\alpha}{\eta \beta} \right) \right]^{1/2} \left[ \frac{1}{\eta \beta} \left( \frac{1}{\eta \beta} + \frac{\alpha}{\eta \beta} \right) \right]^{1/2}
\]

(36)
\[ V_{CW1}(t) = \left[ \beta_1 [Cw_0 + 2w V_{CW0}] V_{j0} + \beta_1 [V_{j0} + 2w V_{CW0} + 2d V_{CDB0}] Cw_0 - (\phi_3 + \eta + \delta v + \delta h_2) V_{CW0} \right] \left[ \frac{1}{\frac{\mathcal{F}(\theta)}{\mathcal{F}_{(\theta)}}} \right] (1 - \theta + \frac{\theta^p}{\Gamma(\theta)}) \]

\[ V_{CDB1}(t) = \left[ \beta_1 [C_{DB0} + 2p V_{CD0}] V_{j0} + \beta_1 [V_{j0} + 2p V_{CD0} + 2p V_{CDB0}] C_{DB0} - (\phi_4 + \eta + \delta v + \delta h_2) V_{CD0} \right] \left[ \frac{1}{\frac{\mathcal{F}(\theta)}{\mathcal{F}_{(\theta)}}} \right] (1 - \theta + \frac{\theta^p}{\Gamma(\theta)}) \]

\[ S_{2}(t) = \left[ \psi A - (\beta_1 [Cw_0 + 2w V_{CW0}] V_{j0} + \beta_1 [V_{j0} + 2w V_{CW0} + 2d V_{CDB0}] Cw_0 - (\phi_4 + \eta + \delta v + \delta h_2) V_{CW0} \right] \left[ \frac{1}{\frac{\mathcal{F}(\theta)}{\mathcal{F}_{(\theta)}}} \right] (1 - \theta + \frac{\theta^p}{\Gamma(\theta)}) \]

\[ V_{CD2}(t) = \left[ \beta_1 [Cw_0 + 2w V_{CW0}] V_{j0} + \beta_1 [V_{j0} + 2w V_{CW0} + 2d V_{CDB0}] Cw_0 - (\phi_4 + \eta + \delta v + \delta h_2) V_{CW0} \right] \left[ \frac{1}{\frac{\mathcal{F}(\theta)}{\mathcal{F}_{(\theta)}}} \right] (1 - \theta + \frac{\theta^p}{\Gamma(\theta)}) \]

\[ V_{CD2}(t) = \left[ \beta_1 [Cw_0 + 2w V_{CW0}] V_{j0} + \beta_1 [V_{j0} + 2w V_{CW0} + 2d V_{CDB0}] Cw_0 - (\phi_4 + \eta + \delta v + \delta h_2) V_{CW0} \right] \left[ \frac{1}{\frac{\mathcal{F}(\theta)}{\mathcal{F}_{(\theta)}}} \right] (1 - \theta + \frac{\theta^p}{\Gamma(\theta)}) \]

and so on. Thus, we obtain the required solution

\[ S(t) = S_0(t) + S_1(t) + S_2(t) + \ldots, I(t) = I_{10}(t) + I_{11}(t) + I_{12}(t) + \ldots, W(t) = W_{10}(t) + W_{11}(t) + W_{12}(t) + \ldots, \]

\[ D(t) = D_{10}(t) + D_{11}(t) + D_{12}(t) + \ldots, H(t) = H_{10}(t) + H_{11}(t) + H_{12}(t) + \ldots, \]

\[ C(t) = C_{CW1}(t) + C_{CW2}(t) + \ldots, D(t) = D_{CD1}(t) + D_{CD2}(t) + \ldots \]

(73)

4. Numerical Simulations

4.1. Uncertainty and Sensitivity Analysis

As a result of uncertainties that may arise in parameter estimation, global sensitivity analysis is carried out in this section, following the approach in [28]. We perform a Latin Hypercube Sampling (LHS) on the parameters in the model. For the sensitivity analysis, a Partial Rank Correlation Coefficient (PRCC) was calculated between values of the parameters in the response function and the values of the response function derived from the sensitivity analysis. A total of 1,000 simulations (of the model (1)) per LHS run were carried out. As shown in Fig. 1a, when the wild SARS-CoV-2 variant associated reproduction number \( R_{0w} \) is used as a response function, the effective contact rate for wild SARS-CoV-2 variant transmission, \( \beta_1 \), positively correlated and the vaccine efficacy against the wild SARS-CoV-2 variant \( \phi_1 \), negatively correlated, as well as the COVID-19 recovery rate \( \phi_1 \), negatively correlated dominate the disease dynamics. Therefore, to effectively, reduce the spread of the wild SARS-CoV-2 variant in the population, efforts must geared towards reducing the disease spread and also increase effective vaccine administration to susceptible individuals. Similar conclusions can be made when the delta SARS-CoV-2 variant associated reproduction number, \( R_{0d} \) is used as input. This can be noticed in Figs. 1b, where also the vaccine efficiency against the wild SARS-CoV-2 variant, the effective contact rate for delta variant transmission and the recovery rate dominate the dynamics of the disease. Using the HIV associated reproduction number \( R_{0v} \) as a response function, the dominant parameter is the effective contact rate for HIV transmission, \( \beta_1 \). This can be observed in Fig. 1c. When the class of individuals co-infected with the wild SARS-CoV-2 variant and HIV is used as a response function, as depicted in Fig. 1d, the dominant parameters are: the effective contact rates for wild SARS-CoV-2 and HIV transmissions, respectively. Similar conclusion is reached when the class of individuals co-infected with the delta SARS-CoV-2 variant and HIV is used as a response function, as shown in Fig. 1e. This shows that to avert high incidence of co-infection cases in the population, efforts must be enhanced to also reduce single infection cases.
Fig. 2 Model fitting.

Fig. 3 Solution profiles for some epidemiological states using integer and fractional order derivatives. In Figs. 4a-d, \( \mathcal{X}(\theta) = 1; \beta_1 = 0.000003; \beta_2 = 0.000003; \beta_3 = 0.0000014 \). In Figs. 4e-f, \( \mathcal{X}(\theta) = 1; \beta_1 = 0.0003; \beta_2 = 0.0003; \beta_3 = 0.0005 \). Other parameters are given in Table 1.
4.2. Data fitting and parameter estimation

The model fitting was performed using the fmincon function in the Optimization Toolbox of MATLAB. The routine was used to minimize the sum of squared differences between each observed cumulative case data point and the corresponding case point obtained from the model (1). We implement our model fitting for an epidemic period in Botswana starting from December 27, 2020 to February 16, 2022 (the period when both variants were in circulation). The complete COVID-19 data used for fitting can be found in [31]. Fig. 2 shows that our model behaves very well with the dataset. We estimated the COVID-19 related parameters as follows:

\[ b_1 = \frac{2.0003}{10^{-7}}, \quad b_2 = 6.5 \times 10^{-7}, \quad \phi_1 = 0.0020, \quad \phi_2 = 0.0070, \quad \delta_W = 0.0017, \quad \delta_D = 0.0075, \]

so that the associated reproduction numbers are \( R_{0W} = 4.7668, R_{0D} = 4.5817 \). It is also imperative to state here, that COVID-19 daily cumulative data are more readily available than the HIV daily cumulative data. As such, we obtain the HIV related parameters from relevant literatures, as shown in Table 1. Using these parameters, the estimated HIV associated basic reproduction number is \( R_{0V} = 2.5817 \). Our simulations in the subsequent subsection show some correlation with the cumulative number of individuals infected with HIV in Botswana, which is estimated around \( 3.7 \times 10^7 \) [32].

The fmincon’s optimization routine syntax:

\[
x = \text{fmincon}(\text{@modelfun}, x0, A, b, Aeq, beq, lb, ub, \text{nonlcon}, \text{options}),
\]

starts at \( x_0 \) (the initial guesses) and finds an optimum \( x \) to the function described in \( \text{@modelfun} \) that fits the model to a given data set, subject to the nonlinear inequalities \( c(x) \) or equalities \( ceq(x) \) defined in nonlcon, and also subject to the linear inequalities \( A \cdot x \leq b \) and linear equalities \( Aeq \cdot x = beq \), defined in \( A, b, Aeq, beq \), respectively. \( x_0 \) can be a scalar, vector, or matrix. \( lb \) and \( ub \) are the bounds on the parameters to be estimated. The optimization parameters and error tolerance are specified in \( \text{options} \).

Fig. 4 Solution profiles for some epidemiological states at different derivative orders. In Figs. 4a-d, \( \mathcal{K}(\theta) = 1; b_1 = 0.000003; b_2 = 0.000003; b_3 = 0.0000014. \) In Figs. 4e-f, \( \mathcal{K}(\theta) = 1; b_1 = 0.0003; b_2 = 0.0003; b_3 = 0.0005. \) Other parameters are given in Table 1.
4.3. Simulations

Due to high prevalence of COVID-19 and HIV in countries in Southern parts of Africa, we have selected a country from the region for demographic data used for our simulations. Particularly, we have chosen Botswana. The sexually active population in Botswana (aged 15–64) is estimated to be 1,480,751 [29]. Also, the life expectancy is 65.24 years [29]. Thus, we set the human natural death rate, \( g = \frac{65.24}{365} \) per day. Since the total population is, \( N(0) = 1,480,751 \), we set the human recruitment rate, \( \Lambda \) to be \( \frac{65,240}{365} \). The initial conditions used for the simulations are assumed as follows: \( S(0) = 800,000; \ C_I(0) = 500,000; \ C_H(0) = 1; \ C_R(0) = 1; \ C_D(0) = 1 \). The parameters estimated from the fitting are presented in Table 1.

4.3.1. Comparing the integer and fractional order derivatives

The simulation of the infected populations, for integer order derivative (when \( h = 1.0 \)) and fractional order derivative (when \( h = 0.90 \)), are presented in Figs. 3a, b, c, d, e and f, respectively. It is observed in these figures, the fractional derivative greatly influenced the dynamics of the diseases, in each epidemiological state over time. The noticed difference when the simulations are carried out via the fractional derivative is due to memory effect which is lacking in the classical integer order operator. The presence of memory effects on past events will affect the spread of the diseases in the future so that they can be controlled easily. Thus, this made the results obtained via the AB derivative better than those obtained from the integer order operator.

4.3.2. Impact of different fractional orders

The simulation of the infected populations, at different fractional order values, when the normalization function, \( \psi(\theta) = 1; \beta_1 = 0.0003; \beta_2 = 0.0003; \beta_3 = 0.0005 \), are presented in Figs. 4a, b, c, d, e and f, respectively. It is observed in these figures that as the fractional order decreases, the pop-
ulations in each epidemiological state decrease as well over time.

4.3.3. Impact of COVID-19 vaccination and parameter accounting for susceptibility to COVID-19 infection

In Figs. 5a and b, we assess the impact of COVID-19 vaccination on the classes of individuals infected with the wild and delta COVID-19 variants. It is observed that, the total infections over time is less when vaccination is implemented, than when it is not implemented. This shows that more efforts should be put in place to ensure mass vaccination of susceptible individuals. This will in turn have a resultant positive impact in reducing co-infection cases with HIV. Simulations of two co-infected classes, \( \mathcal{C} \) and \( \mathcal{D} \), at different values of the modification parameter for increased susceptibility to COVID-19 infection, \( \zeta \), are presented in Figs. 5e and f. It is observed that as susceptibility to COVID-19 infection is decreased among the HIV-infected population, there is a significant decrease in the co-infected cases. This is very interesting and it shows that efforts must be made to ensure we reduce COVID-19 infections in HIV infected individuals. This is necessary so as to reduce worst complications leading to death, as warned by the WHO. More-so, reducing co-infections with COVID-19 among HIV infected persons will also enhance functional immune system, which help the body to fight other opportunistic infections.

5. Conclusion

In this paper, we have presented and analyzed a new mathematical model for dual variants of COVID-19 and HIV co-infection. The existence and uniqueness of the solution of the proposed model have been established via the Banach fixed point theorem. The model was solved semi-analytically using the Laplace Adomian decomposition Method. We presented the impact of the Atangana-Baleanu fractional derivative on the dynamics of the proposed model. The work also highlighted the impact of COVID-19 vaccination on the dynamics of the co-infection of both diseases. We have also assessed the impact of COVID-19 variants on HIV prevalence using simulations.

Sensitivity analysis was carried out on the parameters of the model, using the Latin Hypercube Sampling (LHS). For instance, when the wild (or delta) SARS-CoV-2 variant associated reproduction number \( R_{0w} \) (or \( R_{0d} \)) is used as a response function, the effective contact rate for wild (delta) SARS-CoV-2 variant transmission, \( (\beta_1, \beta_2) \), (positively correlated) and the vaccine efficacy against the wild (delta) SARS-CoV-2 variant \( (\theta_1, \theta_2) \), (negatively correlated), as well as the COVID-19 recovery rate \( (\phi_1, \phi_2) \), (negatively correlated) dominate the disease dynamics. Thus, to effectively reduce the spread of the wild and delta SARS-CoV-2 variants in the population, efforts must geared towards reducing the spread of both variants and also increase effective vaccine administration to susceptible individuals. Also, when the class of individuals co-infected with the delta SARS-CoV-2 variant and HIV is used as a response function, as depicted in Fig. 1d, the dominant parameters include: the effective contact rates for delta SARS-CoV-2 and HIV transmissions, respectively and the vaccine efficacy against the wild SARS-CoV-2 variant. This shows that to avert high incidence of co-infection cases in the population, efforts must be enhanced to also reduce the spread of SARS-CoV-2 and HIV, and also to administer mass effective vaccination coverage.

Other highlights of the simulation results are as follows:

i. The fractional order model was fitted to real data from Botswana

ii. The plot of the total infected population with the wild and Delta COVID-19 variants, at different vaccination rates, depicted by Figs. 5a and b, respectively, show that as vaccination rate is stepped up, there is a significant reduction in the population of those infected with the wild and delta COVID-19 variants. More interestingly, is the plot of class of individuals co-infected with HIV and the wild or Delta COVID-19 variant, as vaccination rate is increased. It is observed that these populations experience a significant decrease, as can be observed in Figs. 5c and d. Thus, stepping up vaccination against the different variants of COVID-19 could greatly reduce co-infection cases, among people already infected with HIV.

iii. It was observed that, the fractional derivative greatly influenced the dynamics of the diseases, in each epidemiological state over time. The noticed difference when the simulations are carried out via the fractional derivative is due to memory effect which is lacking in the classical integer order operator. It is well known that, the presence of memory effects on past events will affect the spread of the diseases in the future so that they can be controlled easily. Thus, this made the results obtained via the AB derivative better than those obtained from the integer order operator.

iv. Simulations also showed that vaccination for COVID-19 could also have a positive population level impact on the classes of individuals co-infected with COVID-19 and HIV.

Our model was proposed based on the focus of different variants of COVID-19 and HIV co-infection only. The co-infection classes considered in the model were those infected with either variant and HIV. We did not investigate the impact of triple co-infection with both variants and HIV. This could be an extension on the model. Also, the emergence of different variants of COVID-19 warrants further studies on their co-infections with other diseases, such as TB, influenza, Malaria and other diseases. We could therefore, consider dual variants of COVID-19 and co-infection with other diseases, apart from HIV. While this is to the best of our knowledge, the first study on dual variants of COVID-19 and HIV, more studies should be devoted to the mathematical (stochastic, agent based modelling, within/intra-host) and epidemiological dynamics of this co-infection. Due to inadequacies and uncertainty of several aspects and characteristics of emerging variants of COVID-19 and HIV, we faced so much difficulty in estimating certain parameters in this study. Some of the parameters difficult to estimate are: the modification parameters accounting for susceptibility of COVID-19 infected individuals to HIV and the modification parameter accounting for susceptibility of HIV infected individuals to different variants of COVID-19. Since mathematical models are to a large extent symbolic representations of biological systems, by construction, they inherit the loss of information which could potentially make the predic-
tion of model outcomes imprecise. Therefore, further studies with more reliable data and detailed information about the co-interactions of different variants of COVID-19 and HIV is viable.

Authors’ contributions Statement:

Andrew Omame: performed the calculations and wrote in part the original draft and edited the final manuscript. Mary Ele Isah: performed the calculations and wrote in part the original draft. Mujahid Abbas: designed the concept of the paper, supervised the work and edited the final manuscript. Abdel-Haleem Abdel-Aty: Participated in the investigation, and wrote in part the original draft. Chibueze Onyenegecha: Participated in the investigation, and wrote in part the original draft.

Declaration of Competing Interest

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

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A fractional order model for Dual Variants of COVID-19 and HIV co-infection

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