Journal of Basics and Applied Sciences Research (JOBASR)
ISSN (print): 3026-9091, ISSN (online): 1597-9962
Volume 3(4) July 2025
DOI: https://dx.doi.org/10.4314/jobasr.v3i4.6

Analysis of Existence and Uniqueness of Solutions of Fractional-Order Mathematical Model of HIV/AIDS

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Keywords:
Mathematical modelling,
Atangana-Baleanu-Caputo derivative,
Data fitting,
Fractional Order,
TB model,
Existence & uniqueness,
Sensitivity analysis

ABSTRACT
In this study, the Atangana-Baleanu-Caputo (ABC) fractional derivative with the Mittag-Leffler kernel is applied to analyze the transmission dynamics of an HIV/AIDS model. The Picard-Lindelöf method is applied to establish the existence and uniqueness of the model's solution. The findings showed that early detection, timely treatment, awareness campaigns, and stigma reduction play a crucial role in curbing the spread of HIV/AIDS within the population. Furthermore, the MATLAB *fmincon* algorithm is employed to simulate the model, providing realistic insights into the disease progression under different scenario. The simulation results show that effective treatment of infected individuals, along with reduced contact rate through safe sex practices, can significantly decrease HIV/AIDS transmission. The incorporation of fractional calculus enhances the model's accuracy, as non-integer order derivatives better capture memory effects compared to traditional models.

INTRODUCTION

The Human Immunodeficiency Virus (HIV) is a type of retrovirus that compromises the immune system by specifically attacking CD4+ T lymphocytes, ultimately resulting in a gradual decline in immune function (UNAIDS, 2024). Without appropriate treatment, HIV progressively impairs the body's capacity to resist infections and diseases, culminating in Acquired Immuno deficiency Syndrome (AIDS)—the most advanced phase of HIV infection (World Health Organization [WHO], 2024). During this phase, individuals face a heightened risk of opportunistic infections, including tuberculosis (TB) and various cancers, which significantly increase the risk of death (Centers for Disease Control and Prevention [CDC], 2024). The development and widespread use of antiretroviral therapy (ART) have led to substantial reductions in HIV-related mortality and extended the life expectancy of people living with the virus (National Institutes of Health [NIH], 2024). Nevertheless, despite medical progress, HIV/AIDS continues to pose a significant global health burden, affecting millions across the world. Sub-Saharan Africa remains the epicenter of the epidemic, with the region accounting for approximately two-thirds of global HIV cases (WHO, 2024).

In Nigeria, an estimated 1.4% of adults aged 15–49 are infected, and about 1.9 million individuals are currently living with HIV (National Agency for the Control of AIDS [NACA], 2024). Alarming figures from 2023 show that approximately 26,000 children between ages 0–14 contracted HIV, and 15,000 children in that same age bracket died due to AIDS-related complications (Vanguard News, 2024). These statistics underscore the critical importance of maintaining robust prevention, early detection, and treatment efforts to curb the epidemic.

Mathematical modeling has become instrumental in understanding how HIV spreads and in evaluating the effectiveness of various control measures. These models offer valuable perspectives on how elements such as sexual practices, access to treatment, and public health initiatives influence transmission dynamics ("Agbata et al., 2024"; Huang et al., 2023). For instance, Hamou et al. (2024) employed fractional calculus in modeling to incorporate memory-dependent dynamics, enhancing the precision of epidemic forecasts. Similarly, Meetei et al. (2024) used actual epidemiological data to support a compartmental modeling framework, emphasizing the importance of data-informed models for public health decision-making.

How to cite this article: Ezugorie I.G., Ahman, Q.O., Agbata, B.C., Atabo, V.O., Senewo, E.O, and Micheal B.D. (2025). Analysis of Existence and Uniqueness of Solutions of Fractional-Order Mathematical Model of HIV/AIDS. *Journal of Basics and Applied Sciences Research*, *3*(4), 40-51. https://dx.doi.org/10.4314/jobasr.v3i4.6

Their work also explored HIV transmission among men who have sex with men (MSM), revealing how interpersonal relationships affect infection rates. These modeling approaches not only deepen scientific comprehension of HIV spread but also guide policymakers in developing effective prevention and treatment programs. Prevention strategies remain a cornerstone in combating the HIV/AIDS epidemic. Biomedical innovations such as pre-exposure prophylaxis (PrEP) and long-acting injectable antiretrovirals have significantly mitigated transmission risks (WHO, 2024). Tools like the dapivirine vaginal ring and injectable cabotegravir have provided high-risk populations—especially adolescent girls and women in sub-Saharan Africa—with new prevention options (UNAIDS, 2024). Furthermore, mathematical models have been employed to assess the potential benefits of scaling up these preventive interventions. For example, a study featured in the *Journal of Inequalities and Applications* utilized fractal-fractional derivatives in modeling HIV transmission, illustrating how various strategies can substantially reduce new infections. Alongside these biomedical advances, behavioral approaches—such as consistent condom usage, voluntary medical male circumcision, and harm reduction efforts for people who inject drugs—remain vital in limiting the virus's spread (CDC, 2024).

Despite these achievements, significant obstacles continue to hamper global progress in addressing HIV/AIDS. Social stigma, discrimination, and economic hardships remain key barriers to accessing prevention, testing, and treatment services, particularly in low- and middle-income countries (UNAIDS, 2024). In response, Nigeria has intensified its campaign to eliminate mother-to-child HIV transmission and expand pediatric treatment programs (NACA, 2024). The government has also created national and regional task forces aimed at accelerating PMTCT (Prevention of Mother-to-Child Transmission) services and ensuring that no child is born with HIV (Vanguard News, 2024). These initiatives are aligned with the broader international objective of eradicating AIDS as a public health threat by the year 2030 (UNAIDS, 2024).

Table 1. Description of Variables and Model parameters.

Variables	**Interpretation**
S	Susceptible humans
E	Exposed humans
I	Diagnosed HIV infected class
A	Diagnosed HIV/AIDS infected class
T	HIV/AIDS infected patients under treatment
Parameter	**Description**
Λ	Recruitment Rate
β	Contact rate between uninfected population and the infected individuals
μ	Natural death rate
σ	Disease induced death rate
θ	Progression rate from E to I
ω	Modification parameter rate accounts for reduced rate of infection
ϕ_1	Progression rate from I to A
ϕ_2	Progression rate from T to I
ε_1	Treatment rate of AIDS infected humans
ε_2	Progression rate from T back to A due

The Atangana–Baleanu–Caputo (ABC) fractional derivative is a contemporary advancement in mathematical analysis that extends beyond traditional calculus by incorporating memory and hereditary characteristics into differential equations. Unlike conventional derivatives, it utilizes a non-local and non-singular Mittag-Leffler kernel, enabling the modeling of systems in which historical states influence present and future behavior (Atangana and Baleanu, 2016). This makes the ABC derivative particularly well-suited for complex systems such as those encountered in epidemiology, where temporal factors play a critical role in understanding disease dynamics. When applied to HIV/AIDS modeling, the ABC fractional derivative presents notable benefits by accounting for the time-dependent and nonlinear interactions associated with the transmission and treatment of the virus. Ullah et al. (2019) employed this derivative in constructing a model for HIV-1 infection, showing that it yields a more precise depiction of disease progression compared to classical approaches. By integrating memory effects, the model provides deeper insights into how historical infection patterns and treatment responses shape the current and future trajectory of the disease, which is essential for formulating targeted interventions.

In addition, the ABC derivative has been effectively used in the study of co-infections, including simultaneous infections with HIV and COVID-19. For example, Owolabi et al. (2022) created a fractional-order model based on the ABC derivative to explore the interaction between various COVID-19 variants and HIV.

Their research demonstrated the method's effectiveness in capturing the intricate dynamics of co-infection, offering valuable guidance for public health policy and epidemic control (Atangana and Baleanu, 2019). Overall, these applications highlight the ABC fractional derivative's potential to enhance the accuracy and applicability of epidemiological models, thereby supporting more informed and effective disease control measures.

Preliminaries

Definition 2.1. On the interval $\chi \in [0, 1]$, by taking the function $f \in H^1(a,b), b > a,$ so that ABC derivative is given by

$$ {}^{ABC}_{a}D^{\chi}_{\xi} f(\xi) = \frac{M(\xi)}{1-\chi}\int_a^{\chi} f'(\theta)E_{\chi}\left[-\chi\frac{(\xi-\theta)^{\chi}}{1-\chi}\right]d\theta,$$

with $M(0) = M(1) = 1$, where $M(\chi)$ is a normalization function (Atangana & Baleanu, 2016).

Definition 2.2. (Atangana and Baleanu, 2016) On the interval $\chi \in [0, 1]$,by taking the function $f \in H^1(a,b), b > a,$ which is not differentiable, hence the Atangana-Baleanu fractional derivative in Riemann-Liouville sense is defined as

$$ {}^{ABR}_{a}D^{\chi}_{\xi} f(\xi) = \frac{M(\xi)}{1-\chi}\frac{d}{d\xi}\int_a^{\chi} f(\theta)E_{\chi}\left[-\chi\frac{(\xi-\theta)^{\chi}}{1-\chi}\right]d\theta,$$

Definition 2.3. The ABC fractional derivative for the fractional integral of order χ is given by

$$ {}^{AB}_{a}I^{\chi}_{\xi} f(\xi) = \frac{1-\chi}{\mathcal{A}(\chi)} f(t) + \frac{\chi}{\mathrm{A}(\chi)\Gamma(\chi)}\int_a^{\chi} f(\mathrm{y})(\xi-y)^{\chi-1}dy,$$

If $\chi = 0$ and $\chi = 1$, the initial function and ordinary integral are obtained, below.

In the next sections, we will investigate the Laplace transform operators and applied fundamental theorems associated with these derivatives (Atangana & Baleanu, 2016). The connection between these operators and the Laplace Transform will be established.

$$\mathcal{L}\left\{ {}^{ABR}_{0}D^{\chi}_{\xi}\left[f(\xi)\right]\right\}(l) = \frac{L(\chi)}{1-\chi}\frac{l^{\chi}\mathcal{L}\left\{f(\xi)\right\}(l)}{l^{\chi}+\frac{\chi}{1-\chi}}$$

and

$$\mathcal{L}\left\{ {}^{ABC}_{0}D^{\chi}_{\xi}\left[f(\xi)\right]\right\}(l) = \frac{\mathrm{M}(\chi)}{1-\chi}\frac{l^{\chi}\mathcal{L}\left\{f(\xi)\right\}(l) - l^{\chi-1}f(0)}{l^{\chi}+\frac{\chi}{1-\chi}}$$

Theorem 2.1. (Atangana and Baleanu, 2019). Consider the close interval [a, b] and use f to represent a continuous function defined on it. We establish the following inequality, which is true for any point lying in [a, b]:

$$\left\| {}^{ABR}_{0}D^{\chi}_{\xi}\left[f(\xi)\right]\right\| < \frac{\mathrm{M}(\chi)}{1-\chi}\left\|f(\theta)\right\|,$$

Where $\left\|f(\theta)\right\| = \max_{a\le\xi\le b}\left|f(\theta)\right|$.

Theorem 2.2. ((Atangana and Baleanu, 2016)). The *Riemann-Liouville* and Caputo types of Atangana-Baleanu derivative exhibit the lipchitz condition, which is best defined as given below

$$\left\| {}^{ABR}_{0}D^{K}_{\xi}\left[f(\xi)\right] - {}^{ABR}_{0}D^{K}_{\xi}\left[g(\xi)\right]\right\| \le H\left\|f(\xi)-g(\xi)\right\|.$$

and

$$\left\| {}^{ABC}_{0}D^{\chi}_{\xi}\left[f(\xi)\right] - {}^{ABC}_{0}D^{\chi}_{\xi}\left[g(\xi)\right]\right\| = H\left\|f(\xi)-g(\xi)\right\|.$$

MATERIALS AND METHODS

Model Formulation

The human population at time t, denoted by $N(t)$ is sub-divided into five (5) mutually exclusive compartments of Susceptible humans $S(t)$, Exposed humans $E(t)$, Infected humans with HIV $I(t)$, Infected humans with HIV/AIDS $A(t)$ and Individuals on treatment $T(t)$ The total human population is denoted as: $N(t) = S(t) + E(t) + I(t) + A(t) + T(t)$.. The recruitment rate of individuals into the susceptible population is at the rate Λ. λ is the force of infection which reduces the susceptible and increases the exposed human and β denotes the effective contact rate. The populations HIV infected and HIV/AIDS infected human increase by the rates $\theta\omega$ and $\omega(1-\theta)$ respectively. α denotes progression from HIV infected individuals to HIV/AIDS infected individuals and the population of every compartment is decreased by the natural death rate μ. ϕ_1 and ε_2 are the treatment rates of HIV infected individuals and HIV/AIDS infected individuals respectively, ϕ_2 and ε_1 are their various re-infection rates. The HIV/AIDS infected population is further reduced by the disease induced death rate σ.

Model Assumptions

The following mathematical assumptions are used to formulate the model

1. There is re-infection of treated humans from both $I(t)$ *and* $A(t)$.

2. Disease induced death occurs only in the HIV/AIDS compartment
3. The population mixture is homogeneous
4. The transmission of the disease in HIV infected individuals to HIV/AIDS infected individuals is relatively minimal due to effective treatment

Model Equations

From the schematic diagram and model description above, the differential equations modeling the transmission dynamics of HIV/AIDS in the population is given as

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - (\lambda + \mu)S \\
\frac{dE}{dt} &= \lambda S - (\omega + \mu)E \\
\frac{dI}{dt} &= \omega\theta E + \phi_2 T - (\alpha + \phi_1 + \mu)I \\
\frac{dA}{dt} &= \omega(1-\theta)E + \alpha I + \varepsilon_2 T - (\varepsilon_1 + \sigma + \mu)A \\
\frac{dT}{dt} &= \phi_1 I + \varepsilon_1 A - (\varepsilon_2 + \phi_2 + \mu)T
\end{aligned} \tag{1}$$

The force of infection of the HIV/AIDS model in (1) is given as:

$$\lambda = \frac{\beta(I + A)}{N}$$

Given that derivatives of fractional order represent epidemiological patterns better than classical order cases. We therefore modify the TB-model (1) in terms of the ABC derivative given as follows

$$\begin{aligned}
{}^{ABC}_{0}D^{\chi}_{\xi}\, S(\xi) &= \Lambda - (\lambda + \mu)S \\
{}^{ABC}_{0}D^{\chi}_{\xi}\, E(\xi) &= \lambda S - (\omega + \mu)E \\
{}^{ABC}_{0}D^{\chi}_{\xi}\, I(\xi) &= \omega\theta E + \phi_2 T - (\alpha + \phi_1 + \mu)I \\
{}^{ABC}_{0}D^{\chi}_{\xi}\, A(\xi) &= \omega(1-\theta)E + \alpha I + \varepsilon_2 T - (\varepsilon_1 + \sigma + \mu)A \\
{}^{ABC}_{0}D^{\chi}_{\xi}\, T(\xi) &= \phi_1 I + \varepsilon_1 A - (\varepsilon_2 + \phi_2 + \mu)T
\end{aligned} \tag{2}$$

Subject to initial conditions

$$S(0) = S_0, \quad E(0) = E_0, \quad I(0) = I_0, \quad A(0) = A_0, \quad T(0) = T_0,$$

Existence and uniqueness of Solutions

Solving nonlinear equations continues to be a challenging task in differential calculus. The fractional-order model we are studying exhibits considerable nonlinearity, which makes finding exact solutions to such a complex system particularly difficult. Therefore, our main focus is on tackling the issues concerning the existence and uniqueness of solutions for model (2). To do this, we use the fixed point theorem, a widely recognized technique for demonstrating the existence of solutions to nonlinear equations across different mathematical contexts (Atangana and Baleanu, 2016). This approach enables us to better understand the system's behavior and characteristics. On the interval q, suppose that $p = K(q) \times K(q),$, where the Banach space $K(q)$ of continuous real value functions is defined with the norm

$$\|S, E, I, A, T\| = \|S\| + \|E\| + \|I\| + \|A\| + \|T\|,$$

where,

$$\begin{aligned}
\|S\| &= \sup\{|S(\xi)| : \xi \in q\}, \\
\|E\| &= \sup\{|E(\xi)| : \xi \in q\}, \\
\|I\| &= \sup\{|I(\xi)| : \xi \in q\}, \\
\|A\| &= \sup\{|A(\xi)| : \xi \in q\}, \\
\|T\| &= \sup\{|T(\xi)| : \xi \in q\},
\end{aligned}$$

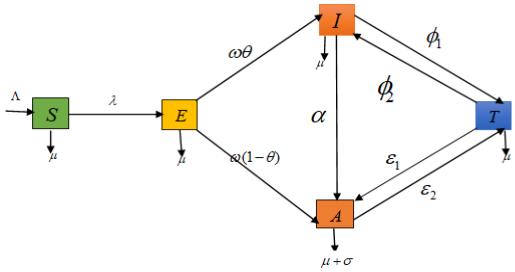


Figure 1: Schematic diagram for the HIV-AIDS model

The model (2) is transformed using the Atangana-Baleanu fractional derivatives, resulting in the following differential equations

$$\begin{aligned}
E(\xi) - E(0) &= \frac{1-\chi}{\mathcal{A}(\chi)}\{\lambda S - (\omega + \mu)E\} + \\
&\quad \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^{\xi}(\xi - y)^{\chi-1}\{\lambda S - (\omega + \mu)E\}\,dy \\
I(\xi) - I(0) &= \frac{1-\chi}{\mathcal{A}(\chi)}\{\omega\theta E + \phi_2 T - (\alpha + \phi_1 + \mu)I\} + \\
&\quad \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^{\xi}(\xi - y)^{\chi-1}\{\omega\theta E + \phi_2 T - (\alpha + \phi_1 + \mu)I\}\,dy
\end{aligned} \tag{3}$$

$$S(\xi)-S(0)=\frac{1-\chi}{\mathcal{A}(\chi)}\{\Lambda-(\lambda+\mu)S\}+\frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^{\xi}(\xi-y)^{\chi-1}\{\Lambda-(\lambda+\mu)S\}dy$$

$$A(\xi)-A(0)=\frac{1-\chi}{\mathcal{A}(\chi)}\{\omega(1-\theta)E+\alpha I+\varepsilon_2 T-(\varepsilon_1+\sigma+\mu)A\}+\frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^{\xi}(\xi-y)^{\chi-1}\{\omega(1-\theta)E+\alpha I+\varepsilon_2 T-(\varepsilon_1+\sigma+\mu)A\}dy$$

$$T(\xi)-T(0)=\frac{1-\chi}{\mathcal{A}(\chi)}\{\phi_1 I+\varepsilon_1 A-(\varepsilon_2+\phi_2+\mu)T\}+\frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^{\xi}(\xi-y)^{\chi-1}\{\phi_1 I+\varepsilon_1 A-(\varepsilon_2+\phi_2+\mu)T\}dy$$

Simplifying (3), we have

$$K_1(\xi,\ S)=\Lambda-(\lambda+\mu)S$$
$$K_2(\xi,\ E)=\lambda S-(\omega+\mu)E$$
$$K_3(\xi,\ I)=\omega\theta E+\phi_2 T-(\alpha+\phi_1+\mu)I$$
$$K_4(\xi,\ A)=\omega(1-\theta)E+\alpha I+\varepsilon_2 T-(\varepsilon_1+\sigma+\mu)A$$
$$K_5(\xi,\ T)=\phi_1 I+\varepsilon_1 A-(\varepsilon_2+\phi_2+\mu)T$$

Theorem 4.1. If the aforementioned inequality holds:

A_T

$0\le\beta_i<1,\quad \text{for } i=1,2,3,...,5$

Then the kernels $\kappa_1, \kappa_2, \kappa_3, \kappa_4, \kappa_5$ satisfy the Lipschitz condition.

Proof. By taking the kernel $\kappa_1(\xi,\ S)=\Lambda-(\lambda+\mu)S$. Let S and S_1 be any two functions, so that :

$$\left\|\kappa_1(\xi,\ S(\xi))-\kappa_1(\xi,\ S_1(\xi))\right\|=\left\|\Lambda-\lambda S+\mu S-(\Lambda-\lambda S_1+\mu S_1)\right\|$$

$$\le\left\|-(\lambda+\mu)\right\|\left\|S(\xi)-S_1(\xi)\right\|$$

$$\le\left\|-\left(\frac{\beta I}{N}+\frac{\beta A}{N}+\mu\right)\right\|\left\|S(\xi)-S_1(\xi)\right\|$$

$$\le\left(\beta\left\|\frac{I(\xi)}{N}\right\|+\beta\left\|\frac{A(\xi)}{N}\right\|+\mu\right)\left\|S(\xi)-S_1(\xi)\right\|$$

$$\le(\beta P_3+\beta P_4+\mu)\left\|S(\xi)-S_1(\xi)\right\|$$

$$\le\beta_1\left\|S(\xi)-S_1(\xi)\right\|$$

$\beta_1=(\beta P_3+\beta P_4+\mu)$,where $P_1=\max_{\xi\in J}\left\|S(\xi)\right\|$, $P_2=\max_{\xi\in J}\left\|E(\xi)\right\|, P_3=\max_{\xi\in J}\left\|I(\xi)\right\|$, $P_4=\max_{\xi\in J}\left\|A(\xi)\right\|$, $P_5=\max_{\xi\in J}\left\|R(\xi)\right\|$, Are bounded functions, we have

$$\left\|\kappa_1(\xi,\ S(\xi))-\kappa_1(\xi,\ S_1(\xi))\right\|=\le\beta_1\left\|S(\xi)-S_1(\xi)\right\|$$

Thus κ_1 satisfies the Lipschitz condition, and if $0\le\beta_1<1$, then it is also a contraction for κ_1. In the same manner, the Lipschitz condition is satisfied by other kernels:

$$\left\|\kappa_2(\xi,\ E(\xi))-\kappa_2(\xi,\ E_1(\xi))\right\|=\le\beta_2\left\|E(\xi)-E_1(\xi)\right\|,$$
$$\left\|\kappa_3(\xi,\ I(\xi))-\kappa_3(\xi,\ I_1(\xi))\right\|=\le\beta_3\left\|I(\xi)-I_1(\xi)\right\|,$$
$$\left\|\kappa_4(\xi,\ A(\xi))-\kappa_4(\xi,\ A_1(\xi))\right\|=\le\beta_4\left\|A(\xi)-A_1(\xi)\right\|,$$
$$\left\|\kappa_5(\xi,\ R(\xi))-\kappa_5(\xi,\ R_1(\xi))\right\|=\le\beta_5\left\|R(\xi)-R_1(\xi)\right\|,$$

By taking the kernel for the model into consideration, we write (3) as follows :

$$S(\xi)=S(0)+\frac{1-\chi}{\mathcal{A}(\chi)}\kappa_1(\xi,\ S)+\frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^{\xi}(\xi-y)^{\chi-1}\kappa_1(y,\ S)\,dy$$
$$E(\xi)=E(0)+\frac{1-\chi}{\mathcal{A}(\chi)}\kappa_2(\xi,\ E)+\frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^{\xi}(\xi-y)^{\chi-1}\kappa_2(y,\ E)\,dy$$
$$I(\xi)=I(0)+\frac{1-\chi}{\mathcal{A}(\chi)}\kappa_3(\xi,\ I)+\frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}+\int_0^{\xi}(\xi-y)^{\chi-1}\kappa_3(y,\ I)\,dy$$
$$A(\xi)=A(0)+\frac{1-\chi}{\mathcal{A}(\chi)}\kappa_4(\xi,\ A)+\frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^{\xi}(\xi-y)^{\chi-1}\kappa_4(y,\ A)\,dy$$
$$R(\xi)=R(0)+\frac{1-\chi}{\mathcal{A}(\chi)}\kappa_5(\xi,\ R)+\frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^{\xi}(\xi-y)^{\chi-1}\kappa_5(y,\ R)\,dy \quad (4)$$

Hence, presenting the subsection of (4) recursively, we obtain:

$$S_v(\xi) = \frac{1-\chi}{\mathcal{A}(\chi)}\kappa_1(\xi,\ S_{v-1}) + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)} + \int_0^\xi (\xi - y)^{\chi-1}\kappa_1(\mathrm{y},\ S_{v-1})\,dy$$

$$E_v(\xi) = \frac{1-\chi}{\mathcal{A}(\chi)}\kappa_2(\xi,\ E_{v-1}) + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^\xi (\xi - y)^{\chi-1}\kappa_2(\mathrm{y},\ E_{v-1})\,dy$$

$$I_v(\xi) = \frac{1-\chi}{\mathcal{A}(\chi)}\kappa_3(\xi,\ I_{v-1}) + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^\xi (\xi - y)^{\chi-1}\kappa_3(\mathrm{y},\ I_{v-1})\,dy$$

$$A_v(\xi) = \frac{1-\chi}{\mathcal{A}(\chi)}\kappa_4(\xi,\ A_{v-1}) + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^\xi (\xi - y)^{\chi-1}\kappa_4(\mathrm{y},\ A_{v-1})\,dy$$

Subject to the initial conditions:

$$S_0(\xi) = S(0),\ E_0(\xi) = E(0),$$

$$A_0(\xi) = A(0),\ I(\xi) = I(0),\ R_0(\xi) = R(0)$$

The system (5) is obtained by using the initial conditions and the difference between the successive terms.

$$\Delta_v(\xi) = S_v(\xi) - S_{v-1}(\xi) = \frac{1-\chi}{\mathcal{A}(\chi)}\kappa_1(\xi,\ S_{v-1}) - \kappa_1(\xi,\ S_{v-2}) + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^\xi (\xi - y)^{\chi-1}\left(\kappa_1(\xi,\ S_{v-1}) - \kappa_1(\xi,\ S_{v-2})\right)dy$$

$$\aleph_v(\xi) = E_v(\xi) - E_{v-1}(\xi) = \frac{1-\chi}{\mathcal{A}(\chi)}\kappa_2(\xi,\ E_{v-1}) - \kappa_2(\xi,\ E_{v-2}) + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^\xi (\xi - y)^{\chi-1}\left(\kappa_2(\xi,\ E_{v-1}) - \kappa_2(\xi,\ E_{v-2})\right)dy$$

$$\Upsilon_v(\xi) = I_v(\xi) - I_{v-1}(\xi) = \frac{1-\chi}{\mathcal{A}(\chi)}\kappa_3(\xi,\ I_{v-1}) - \kappa_3(\xi,\ I_{v-2}) + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^\xi (\xi - y)^{\chi-1}\left(\kappa_3(\xi,\ I_{v-1}) - \kappa_3(\xi,\ I_{v-2})\right)dy \quad (5)$$

$$\Xi_v(\xi) = A_v(\xi) - A_{v-1}(\xi) = \frac{1-\chi}{\mathcal{A}(\chi)}\kappa_4(\xi,\ A_{v-1}) - \kappa_4(\xi,\ A_{v-2}) + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^\xi (\xi - y)^{\chi-1}\left(\kappa_4(\xi,\ A_{v-1}) - \kappa_4(\xi,\ A_{v-2})\right)dy$$

$$\Phi_v(\xi) = R_v(\xi) - R_v(\xi) = \frac{1-\chi}{\mathcal{A}(\chi)}\kappa_5(\xi,\ R_{v-1}) - \kappa_5(\xi,\ R_{v-2}) + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^\xi (\xi - y)^{\chi-1}\left(\kappa_5(\xi,\ R_{v-1}) - \kappa_5(\xi,\ R_{v-2})\right)dy$$

Where:

$$S_v(\xi) = \sum_{i=1}^{v}\Delta_i(\xi),$$

$$E_v(\xi) = \sum_{i=1}^{v}\aleph_i(\xi),$$

$$I_v(\xi) = \sum_{i=1}^{v}\Upsilon_i(\xi), \quad (6)$$

$$A_v(\xi) = \sum_{i=1}^{v}\Xi_i(\xi),$$

$$R_v(\xi) = \sum_{i=1}^{v}\Phi_i(\xi),$$

Applying the triangular inequality and taking norm to (6), we obtain equation (7)

$$\|\Delta_v(\xi)\| = \|S_v(\xi) - S_{v-1}(\xi)\| \le \frac{1-\chi}{\mathcal{A}(\chi)}\|\kappa_1(\xi,\ S_{v-1}) - \kappa_1(\xi,\ S_{v-2})\| + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\left\|\int_0^\xi (\xi - y)^{\chi-1}\left(\kappa_1(\xi,\ S_{v-1}) - \kappa_1(\xi,\ S_{v-2})\right)dy\right\| \quad (7)$$

As the Lipschitz condition is satisfied by the kernel, the following equations hold:

$$\|S_v(\xi) - S_{v-1}(\xi)\| \le \frac{1-\chi}{\mathcal{A}(\chi)}\beta_1\|S_{v-1}(\xi) - S_{v-2}(\xi)\| + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_1\int_0^\xi (\xi - y)^{\chi-1}\|S_{v-1}(\xi) - S_{v-2}(\xi)\|dy$$

and

$$\|\Delta_v(\xi)\| \le \frac{1-\chi}{\mathcal{A}(\chi)}\beta_1\|\Delta_{v-1}(\xi)\| + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_1\int_0^\xi (\xi - y)^{\chi-1}\|\Delta_{v-1}(\xi)\|dy \quad (8)$$

Similarly, we obtained the following results :

$$\|\aleph_v(\xi)\| = \le \frac{1-\chi}{\mathcal{A}(\chi)}\beta_2\|\aleph_{v-1}(\xi)\| + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_2\int_0^\xi (\xi - y)^{\chi-1}\|\aleph_{v-1}(\xi)\|dy$$

$$\|\Upsilon_v(\xi)\| = \le \frac{1-\chi}{\mathcal{A}(\chi)}\beta_3\|\Upsilon_{v-1}(\xi)\| + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_3\int_0^\xi (\xi - y)^{\chi-1}\|\Upsilon_{v-1}(\xi)\|dy$$

$$\|\Xi_v(\xi)\| = \le \frac{1-\chi}{\mathcal{A}(\chi)}\beta_4\|\Xi_{v-1}(\xi)\| + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_4\int_0^\xi (\xi - y)^{\chi-1}\|\Xi_{v-1}(\xi)\|dy$$

$$\|\Phi_v(\xi)\| = \le \frac{1-\chi}{\mathcal{A}(\chi)}\beta_5\|\Phi_{v-1}(\xi)\| + \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_5\int_0^\xi (\xi - y)^{\chi-1}\|\Phi_{v-1}(\xi)\|dy$$

Theorem 4.2. A unique solution is exhibited by the proposed HIV/AIDS fractional order model with ABC operator (2) if $\xi_{\max}$ satisfies the following condition

$$\frac{1-\chi}{\mathcal{A}(\chi)}\beta_i + \frac{\xi_{\max}}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_i < 1, \quad for\ i = 1, 2, \ldots, 6.$$

Proof. It is clear that $S(\xi), E(\xi), I(\xi), A(\xi), T_T, R(\xi)$ are bounded and the kernel of these functions also satisfies the Lipschitz condition. Hence applying the succeeding relation with the application of the (8), we obtained

$$\|\Delta_v(\xi)\| \le \|S(0)\|\left[\frac{1-\chi}{\mathcal{A}(\chi)}\beta_1 + \frac{\xi_{\max}}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_1\right]^v$$

,

$$\|\aleph_v(\xi)\| \le \|E(0)\|\left[\frac{1-\chi}{\mathcal{A}(\chi)}\beta_2 + \frac{\xi_{\max}}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_2\right]^v$$

,

$$\|\Upsilon_v(\xi)\| \le \|I(0)\|\left[\frac{1-\chi}{\mathcal{A}(\chi)}\beta_3 + \frac{\xi_{\max}}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_3\right]^v$$

,

$$\|\Xi_v(\xi)\| \le \|A(0)\|\left[\frac{1-\chi}{\mathcal{A}(\chi)}\beta_4 + \frac{\xi_{\max}}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_4\right]^v,$$

$$\|\Phi_v(\xi)\| \le \|R(0)\|\left[\frac{1-\chi}{\mathcal{A}(\chi)}\beta_5 + \frac{\xi_{\max}}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_5\right]^v$$

,

Therefore, since (6) is a smooth function and it exists.

$$S(\xi) - S(0) = S_v(\xi) - \Psi_{1(v)}(\xi)$$
$$E(\xi) - E(0) = E_v(\xi) - \Psi_{2(v)}(\xi)$$
$$I(\xi) - I(0) = I_v(\xi) - \Psi_{3(v)}(\xi)$$
$$A(\xi) - A(0) = A_v(\xi) - \Psi_{4(v)}(\xi)$$
$$R(\xi) - R(0) = R_v(\xi) - \Psi_{5(v)}(\xi)$$

Note, the term $\|\Psi_\infty(\xi)\| \to 0$ at infinity. It can be shown as follows:

$$\|\Psi_\infty(\xi)\| \le \left\| \begin{matrix} \frac{1-\chi}{\mathcal{A}(\chi)}\kappa_1(\xi,\ S) - \kappa_1(\xi,\ S_{v-1}) + \\ \frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^\xi (\xi - y)^{\chi-1}\left(\kappa_1(\xi,\ S) - \kappa_1(\xi,\ S_{v-1})\right) dy \end{matrix} \right\|$$

$$\|\Psi_\infty(\xi)\| \le \frac{1-\chi}{\mathcal{A}(\chi)}\|\kappa_1(\xi,\ S) - \kappa_1(\xi,\ S_{v-1})\| +$$

$$\frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^\xi (\xi - y)^{\chi-1}\|\kappa_1(\xi,\ S) - \kappa_1(\xi,\ S_{v-1})\| dy$$

$$\le \frac{1-\chi}{\mathcal{A}(\chi)}\beta_1\|S - S_{v-1}\| +$$

$$\frac{\chi^\xi}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_1\|S_v - S_{v-1}\|$$

By recursively repeating the process, we obtain

$$\|\Psi_\infty(\xi)\| \le \left[\frac{1-\chi}{\mathcal{A}(\chi)} + \frac{\xi^\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_1\right]^{v+1}\beta_1^v M$$

Apply $\xi_{\max}$, we have

$$\|\Psi_\infty(\xi)\| \le \left[\frac{1-\chi}{\mathcal{A}(\chi)} + \frac{\xi_{\max}^\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\beta_1\right]^{v+1}\beta_1^v M$$

Taking the limit on both sides as $v \to \infty$, we obtain

$$\|\Psi_\infty(\xi)\| \to 0$$

Uniqueness of Solution

Demonstrating the system's uniqueness of solution is a crucial aspect of the analysis. So via contraction, we suppose that there is another system of solution to (2) $S_1(\xi), E_1(\xi), I_1(\xi), A_1(\xi), R_1(\xi)$

$$\|S(\xi) - S_1(\xi)\| \le \frac{1-\chi}{\mathcal{A}(\chi)}\left(\kappa_1(\xi,\ S) - \kappa_1(\xi,\ S_1)\right) +$$

$$\frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^\xi (\xi - y)^{\chi-1}\left(\kappa_1(\xi,\ S) - \kappa_1(\xi,\ S_1)\right) dy \quad (9)$$

Now, we apply the norm to equation (9)

$$\|S(\xi) - S_1(\xi)\| \le \frac{1-\chi}{\mathcal{A}(\chi)}\|\kappa_1(\xi,\ S) - \kappa_1(\xi,\ S_1)\| +$$

$$\frac{\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\int_0^\xi (\xi - y)^{\chi-1}\|\kappa_1(\xi,\ S) - \kappa_1(\xi,\ S_1)\| dy,$$

Applying the kernel's Lipschitz conditional properties, obtain

$$\le \frac{1-\chi}{\mathcal{A}(\chi)}\|S(\xi) - S_1(\xi)\|\beta_1 +$$

$$\frac{\beta_1\xi^\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\|S(\xi) - S_1(\xi)\|,$$

Which yields

$$\|S(\xi) - S_1(\xi)\|\left(1 - \beta_1\frac{1-\chi}{\mathcal{A}(\chi)} + \frac{\beta_1\xi^\chi}{\mathcal{A}(\chi)\Gamma(\xi)}\right) \le 0,$$

$$\|S(\xi) - S_{H1}(\xi)\| = 0 \to S(\xi) = S_1(\xi).$$

Therefore, the system has a unique solution. Similarly, the above result can be obtained for various solutions of $E(\xi), I(\xi), A(\xi), R(\xi)$.

RESULTS AND DISCUSSION

Sensitivity analysis of the TB model

Sensitivity analysis is a crucial technique for identifying the parameters that impact the spread and management of a disease in a population. By methodically altering these parameters in a mathematical or computational model, researchers can pinpoint the factors that promote disease transmission and those that help reduce its spread. The sensitivity index of R_0 with respect to a parameter p is given by :

$$\Im_p^{R_0} = \frac{p}{R_{0T}} \cdot \frac{\partial R_0}{\partial p}$$

Given that

$$R_0 = \frac{\beta\omega\left(\left(-\phi_1 + \varepsilon_1\right)\phi_2 + \varepsilon_1\varepsilon_2 - \left(-P_2 + P_3 + \alpha\right)P_4 + \phi_1\varepsilon_2\right)\theta - \left(-\phi_1 + \varepsilon_1\right)\phi_2 - P_4P_2\right)}{\left(\phi_2\left(\phi_1P_3 + \varepsilon_1\alpha\right) + P_2\left(-P_3P_4 + \varepsilon_1\varepsilon_2\right)\right)P_1}$$

1. Parameter β : $\frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0} = 1$

2. Parameter ω : $\frac{\partial R_0}{\partial \omega} \times \frac{\omega}{R_0} = 1$

4. Parameter ϕ_1 :

$$\frac{\partial R_0}{\partial \phi_1} \times \frac{\phi_1}{R_0} = -\frac{\phi_1}{(\phi_1+\mu+\alpha)(\varepsilon_2+\phi_2+\mu)+\phi_2(\varepsilon_1+\sigma+\mu)\phi}$$

5. Parameter ϕ_2 :

$$\frac{\partial R_0}{\partial \phi_2} \times \frac{\phi_2}{R_0} = -\frac{\phi_2((\alpha+\mu+\phi_1)(\varepsilon_1+\sigma+\mu)-\varepsilon_1\varepsilon_2)}{(\phi_1+\mu+\alpha)(\varepsilon_2+\phi_2+\mu)+\phi_2(\varepsilon_1+\sigma+\mu)\phi}$$

6. Parameter ε_1 :

$$\frac{\partial R_0}{\partial \varepsilon_1} \times \frac{\varepsilon_1}{R_0} = \frac{\varepsilon_1(\alpha+\mu+\phi_1-\phi)-\varepsilon_1\varepsilon_2\phi_2(\sigma+\mu+\varepsilon_1)\beta}{(\phi_1+\mu+\alpha)(\varepsilon_2+\phi_2+\mu)+\phi_2(\varepsilon_1+\sigma+\mu)\phi}$$

7. Parameter ε_2 :

$$\frac{\partial R_0}{\partial \varepsilon_2} \times \frac{\varepsilon_2}{R_0} = \frac{\varepsilon_2(\alpha+\mu+\phi_1)(\varepsilon_1+\sigma+\mu)+\varepsilon_2\phi_2(\alpha+\mu+\phi_1)}{(\phi_1+\mu+\alpha)(\varepsilon_2+\phi_2+\mu)+\phi_2(\varepsilon_1+\sigma+\mu)\phi}$$

8. Parameter σ :

$$\frac{\partial R_0}{\partial \sigma} \times \frac{\sigma}{R_0} = -\frac{\sigma\phi_2(\alpha+\mu+\phi_1)}{(\phi_1+\mu+\alpha)(\varepsilon_2+\phi_2+\mu)+\phi_2(\varepsilon_1+\sigma+\mu)\phi}$$

9. Parameter α :

$$\frac{\partial R_0}{\partial \alpha} \times \frac{\alpha}{R_0} = -\frac{\alpha(\varepsilon_2+\phi_2+\mu)\phi_2\beta\omega}{(\phi_1+\mu+\alpha)(\varepsilon_2+\phi_2+\mu)+\phi_2(\varepsilon_1+\sigma+\mu)\phi}$$

10. Parameter μ :

$$\frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0} = -\frac{\mu}{(\phi_1+\mu+\alpha)(\varepsilon_2+\phi_2+\mu)+\phi_2(\varepsilon_1+\sigma+\mu)\phi}$$

Final Sensitivity Analysis Results:

1. Sensitivity Index for β : 1
2. Sensitivity Index for ω : 1
4. Sensitivity Index for ϕ_1 : -1.447×10^{-5}
5. Sensitivity Index for ϕ_2 : -1.276×10^{-4}
6. Sensitivity Index for ε_1 : 3.422×10^{-5}
7. Sensitivity Index for ε_2 : 3.422×10^{-5}
8. Sensitivity Index for σ : -1.012×10^{-4}
9. Sensitivity Index for α : -5.748×10^{-5}
10. Sensitivity Index for μ : -4.228×10^{-6}

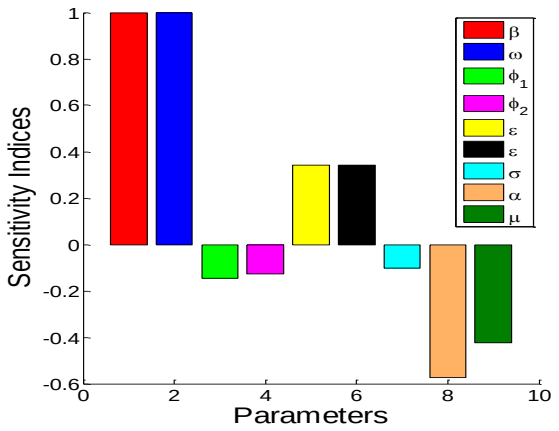


Figure 2: Sensitivity Bar chart

Numerical Simulations of the model

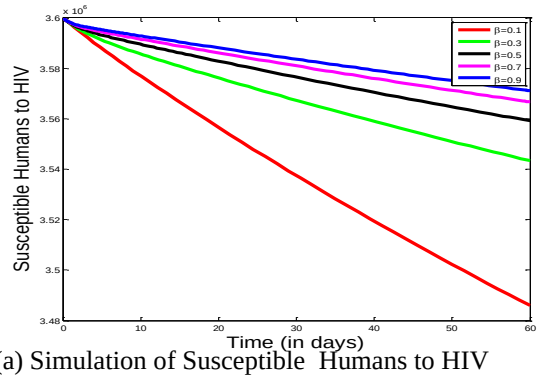
Through numerical simulations conducted using MATLAB, we obtained graphical solutions that depicted the behavior of the HIV/AIDS model. These simulations provided visual representations of how key variables, such as the number of infected and susceptible individuals, evolved over time under varying conditions. By adjusting parameters such as contact rate (β), treatment effectiveness, and intervention strategies, the simulations illustrated potential outcomes, including disease outbreaks or stabilization (Acheneje *et al*, 2024; Agbata *et al*, 2024) . The graphical solutions offered insights into real-life behavior by demonstrating how the disease might spread or be controlled in practice (El-sayed *et al*, 2023). They allowed us to observe trends, such as fluctuations in infection rates, and to evaluate the effectiveness of different public health interventions. By comparing these simulations with actual epidemiological data, we were able to refine the model and enhance predictions, ultimately contributing to more effective strategies for managing and controlling HIV/AIDS.

The parameter values used in the numerical simulations are presented in the table 2 below.

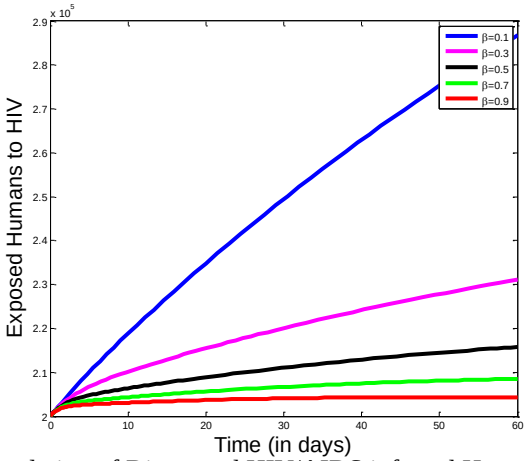
Table 2. Parameter values used in the model and their sources

Parameter	Value	Source
Λ	0.002	(Olumuyiwa *et al*, 2021)
μ	0.0000548	(Agbata *et al*, 2023)
σ	0.01	(Odeh *et al*, 2024)
β	0.01	(Agbata *et al*, 2024)
θ	0.0021	Assumed
ω	0.001	(Bolarinwa *et al*, 2024)
ε_1	0.2	(Agbata *et al*, 2024)

ε_2	0.2	Assumed
ϕ_1	0.002	(Odeh *et al*, 2024)
ϕ_2	0.01	Assumed

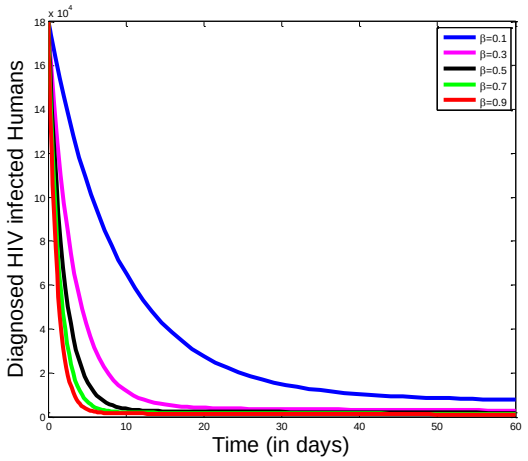


(a) Simulation of Susceptible Humans to HIV

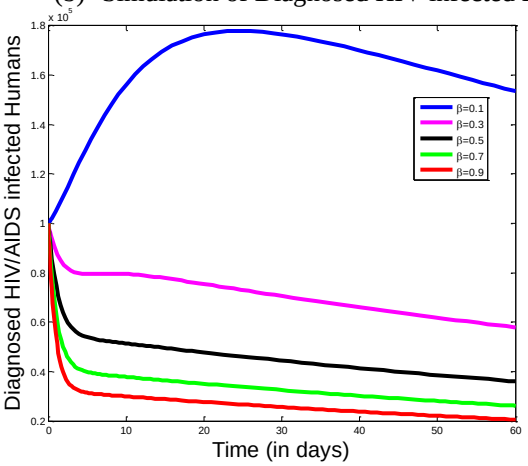


(a) Simulation of Diagnosed HIV/AIDS infected Humans

Fig: 3 Effect of β on $S(t)$ and $E(t)$

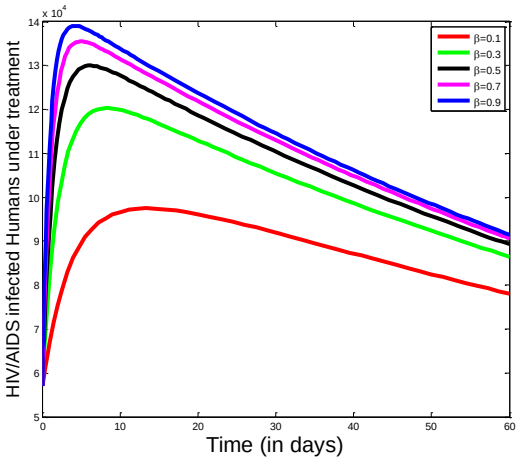


(b) Simulation of Diagnosed HIV infected Humans

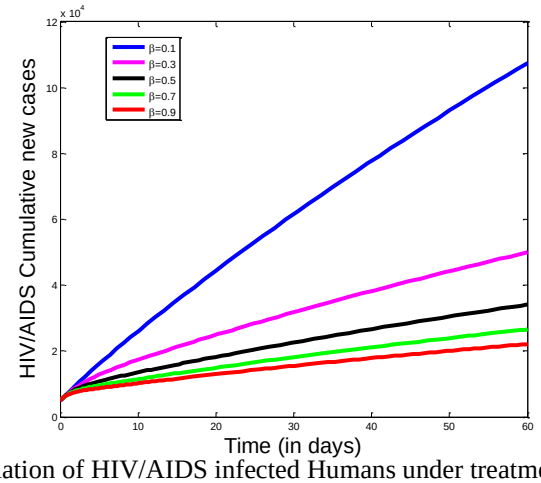


(b) Simulation of Exposed Humans to HIV

Fig: 4 Effect of β on $I(t)$ and $A(t)$



(b) Simulation of HIV/AIDS Cumulative new cases



(a) Simulation of HIV/AIDS infected Humans under treatment

Fig: 5 Effect of β on $A(t)$ and cumulative new cases

Figure 3(a) illustrates how variations in the fractional-order derivative influence the number of susceptible individuals over time. As the fractional-order parameter increases (β), the susceptible population decreases, indicating that treatment strategies are effectively reducing the number of people at risk of infection. This suggests that fractional order derivatives enhance the effectiveness of intervention measures. Similarly, Figure 3(b) shows that the exposed population initially increases before rapidly declining as infection rates rise. While the overall susceptible population is shrinking due to higher transmission rates, some individuals continue to transition into the exposed category. The initial rise in the exposed population may be attributed to delayed treatment initiation or ongoing interactions with infected individuals before preventive measures became fully effective. Figure 4(a) reveals the temporal changes in the infected population, where the number of infected individuals initially rises as people move from the exposed category to the infected class. However, after reaching a peak, this population begins to decline as more individuals progress to the AIDS stage. This peak signifies a turning point where transmission rates slow due to either effective treatment strategies or behavioral changes within the population. Figure 4(b) confirms that the number of individuals diagnosed with AIDS continues to decrease over time, not due to recovery, but rather as a result of mortality. This decline may also reflect the impact of early intervention and improved HIV management, which can delay disease progression. Meanwhile, Figure 5(a) highlights an increasing trend in the number of individuals receiving treatment, as infected individuals transition into the treatment group. This underscores the effectiveness of medical interventions and improved healthcare accessibility. Finally, Figure 5(b) reveals a steady rise in the cumulative number of HIV/AIDS cases over time, driven by fluctuations in infection. This trend emphasizes the ongoing transmission of the virus despite treatment efforts, reinforcing the need for sustained interventions to curb new infections and improve long-term disease management.

CONCLUSION

This study utilizes the Atangana–Baleanu–Caputo (ABC) fractional derivative, which incorporates the Mittag-Leffler kernel, to investigate the transmission dynamics within an HIV/AIDS framework. The results underscore the critical role of timely diagnosis, access to effective treatment, public education initiatives, and reducing stigma in controlling the spread of HIV/AIDS. Numerical simulations indicate that as the fractional-order parameter increases, the number of susceptible individuals decreases, thereby highlighting the positive impact of preventive and therapeutic interventions. The model also captures a transient rise in the exposed group before a decline, representing the shift from being at risk to becoming infected. Similarly, the infected population shows an initial increase followed by a peak and eventual reduction, mirroring the progression from HIV infection to the development of AIDS. A consistent drop in the number of individuals in the AIDS stage further supports the value of early and sustained interventions in delaying or preventing disease advancement. Despite the effectiveness of existing treatment strategies, the model also reveals a persistent growth in the cumulative number of HIV/AIDS cases. This trend points to the ongoing need for robust and continuous public health efforts. While current interventions have made significant progress in reducing transmission, long-term success will depend on expanding healthcare access, promoting comprehensive education, and addressing underlying social and economic challenges. The use of fractional calculus in this context proves to be a powerful approach, offering improved accuracy in

modeling infectious diseases by incorporating memory-dependent effects. Moving forward, future studies should integrate additional real-world factors such as demographic shifts, levels of treatment compliance, behavioral patterns, and socio-economic variables to further refine the model's predictive strength.

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