FUDMA Journal of Sciences (FJS)
ISSN online: 2616-1370
ISSN print: 2645 - 2944
Vol. 8 No. 6, December, 2024, pp 264 - 276
DOI: https://doi.org/10.33003/fjs-2024-0806-2929

FRACTIONAL MATHEMATICAL MODEL FOR THE TRANSMISSION DYNAMICS AND CONTROL OF HIV/AIDS

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ABSTRACT

This paper investigates various epidemiological aspects of HIV/AIDS through a fractional-order mathematical model, emphasizing the role of treatment in the disease's transmission dynamics. Given the ongoing global impact of HIV/AIDS, with millions of people affected and significant mortality rates, understanding the complexities of its transmission and control is crucial for effective public health strategies. We establish conditions for the existence and uniqueness of the model's solutions within the fractional framework and perform a stability analysis of the endemic equilibrium using the Lyapunov function method. Numerical simulations, executed via the fractional Adams–Bashforth–Moulton method, demonstrate the effects of model parameters and fractional-order values on HIV/AIDS dynamics and control. Additional simulations employing surface and contour plots reveal that higher contact rates and reduced treatment efficacy correlate with increased HIV/AIDS prevalence. Our findings suggest that optimizing treatment strategies can significantly lower the prevalence of HIV/AIDS within the population, ultimately contributing to enhanced health outcomes and resource allocation in combating this critical public health issue.

Keywords: HIV/AIDS, Fractional calculus, Adams-Bashforth-Moulton method, Transmission dynamics, Control measures, Strategies

INTRODUCTION

The Human Immunodeficiency Virus (HIV), which leads to Acquired Immunodeficiency Syndrome (AIDS), primarily compromises the immune system by targeting CD4 (T) cells, thus diminishing the body's ability to combat infections (WHO, 2012). Since its discovery in 1981, AIDS has claimed over 25 million lives by 2006, with HIV affecting around 0.6% of the global population (Overview of the Global AIDS Epidemic, 2006). By 2018, an estimated 37.9 million people were living with HIV/AIDS globally, resulting in approximately 1.2 million deaths, with about 62% of those infected receiving Antiretroviral Therapy (ART) (WHO, 2019). The African continent bears the highest burden of HIV/AIDS.

HIV transmission primarily occurs through three routes: sexual intercourse, exposure to contaminated blood through transfusions or shared needles, and from mother to child during pregnancy, childbirth, or breastfeeding. While homosexual transmission is a significant factor in the United States, heterosexual transmission remains the leading method of HIV spread worldwide (Kapila et al., 2016).

The progression of HIV infection brings about varying symptoms. Individuals may be highly infectious in the early stages but frequently remain unaware of their condition until it worsens. Early symptoms can resemble flu-like signs such as fever, headache, rash, or sore throat. As the infection advances and the immune system weakens, additional symptoms may appear, including persistent fever, swollen lymph nodes, diarrhea, weight loss, and chronic cough. Without treatment, individuals face severe health threats, including tuberculosis, cryptococcal meningitis, and certain cancers (WHO, 2022). In sub-Saharan Africa, heterosexual and mother-to-child transmissions account for the majority of HIV cases, with the latter constituting 40% of infections (Adelman, 2001). Tragically, over 25 million children under the age of 15 in this region have died from AIDS, many contracting HIV during childbirth or breastfeeding. Overall, HIV/AIDS remains a critical challenge to global development initiatives.

Recently, fractional calculus has garnered attention for modeling complex systems, including biological processes. Fractional-order models, incorporating Caputo and Riemann-Liouville derivatives, provide more accurate representations of systems with memory effects. These models are increasingly applied to various diseases, such as Zika virus and Lassa fever, offering new insights into transmission dynamics and control strategies (Atokolo et al., 2022, 2024).
evaluating the impact of treatment and vaccination using fractional derivatives. Yunus et al. (2022) used the Caputo fractional derivative to study COVID-19 spread in Nigeria, revealing higher recovery rates due to treatment and vaccination. Omede et al. (2024) created a fractional model to describe soil-transmitted helminth infections, showing that fractional-order models offer greater flexibility. Ahmed et al. (2022) proposed an ABC-fractional model for HIV and COVID-19 co-epidemic transmission. Omame et al. (2022) explored a fractional model for hepatitis B and COVID-19, emphasizing prevention as key to controlling both diseases.

Amos *et al* (2024) presented a fractional mathematical model for the transmission dynamics and control of hepatitis C, using Adams-Bash-forth Moulton method, their findings showed that reducing the contact rate and increasing the treatment help to curb the disease from the population and the fractional order model offers greater flexibility than the classical model.

Acheneje et al. (2024) formulated a model for COVID-19 and monkeypox co-infection, showing that increased treatment capacity reduces disease burden. Smith et al. (2023) reviewed co-infection modeling between hepatitis C and COVID-19, identifying key findings and research gaps.

Atokolo et al. (2023) also studied the spread of vector-borne diseases, incorporating preventive strategies like Insecticide-Treated Nets (ITNs), Indoor Residual Spraying (IRS), and condom use. Their model demonstrated that full intervention, combined with treatment, can significantly reduce disease spread.

Fractional-order models offer distinct advantages over traditional models due to their increased flexibility and

capacity to incorporate non-locality and memory effects, which enhance their accuracy in approximating real-world phenomena. These characteristics make fractional models particularly suitable for complex systems. For example, Ullah et al. (2020) utilized fractional calculus in fuzzy Volterra integral equations, while Ali et al. (2017) explored boundary value problems and Ulam stability through non-linear fractional analysis, advancing the understanding of fuzzy dynamic equations.

The primary objectives of this paper include:

i. Defining conditions that ensure the existence and uniqueness of the model's solution within a fractional framework.
ii. Performing a stability analysis of the endemic equilibrium by employing the Lyapunov function method.
iii. Solving numerically using the fractional Adams–Bashforth–Moulton technique.
iv. Conducting simulations of the model for validation and analysis.

A review of the existing literature on HIV/AIDS mathematical models shows that no prior studies have combined fractional calculus with the Adams–Bashforth–Moulton method for examining HIV/AIDS transmission dynamics and control. This paper seeks to address that gap.

The structure of the paper is as follows: Section 2 discusses the model formulation, Section 3 examines the model's stability, Section 4 presents the numerical findings, and Section 5 concludes with key insights. In addition, foundational concepts from fractional calculus, such as right and left Caputo derivatives, based on the work of Podlubny et al. (1998) and Bonyah et al. (2020), are also introduced. The manuscript highlights the broad applicability of fractional calculus in fields like physics, engineering, and biomathematics, emphasizing its relevance in solving real-world problems.

Definition 1: Let $f \in \Lambda^{\infty}(R)$, then the left and right Caputo fractional derivative of the function f is given by

$$ {}^{c}D_{t}^{\gamma} f(t) = \left(t^{0}D_{t}^{-(m-\gamma)} \left(\frac{d}{dt}\right)^{m} f(t) \right)$$

$$ {}^{c}D_{t}^{\gamma} f(t) = \frac{1}{\Gamma(m-\gamma)} \int_{0}^{t} \left((t-\lambda)^{m-\gamma-1} f^{m}(\lambda)\right) d\lambda \quad (1)$$

The same way ,

$$ {}_{t}^{c}D_{T}^{\gamma} f(t) = \left({}_{t}D_{T}^{-(m-\gamma)} \left(\frac{-d}{dt}\right)^{m} f(t) \right)$$

$$ {}_{t}^{c}D_{T}^{\gamma} f(t) = \frac{(-1)^{m}}{\Gamma(m-\gamma)} \int_{t}^{T} \left((\lambda - t)^{m-\gamma-1} f^{m}(\lambda)\right) d\lambda$$

Definition 2: The generalized Mittag-Leffler function $E_{\alpha,\beta}(x)$ for $x \in R$ is given by

$$E_{\alpha,\beta}(x) = \sum_{m=0}^{\infty} \frac{x^{m}}{\Gamma(\alpha m+\beta)}, \alpha, \beta > 0 \quad (2)$$

Which can also be represented as

$$E_{\alpha,\beta}(x) = xE_{\alpha,\alpha+\beta(x)} + \frac{1}{\Gamma(\beta)} \quad (3)$$

$$E_{\alpha,\beta}(x) = L\left[t^{\beta-1}E_{\alpha,\beta(\pm\psi t^{\alpha})}\right] = \frac{S^{\alpha-\beta}}{S^{\alpha}\pm\psi} \quad (4)$$

Proposition 1.1.

Let $f \in \Lambda^{\infty}(R) \cap C(R)$ and $\alpha \in R, m-1 < \alpha < m,$ Therefore, the conditions given below holds:

1. ${}_{t_0}^{c}D_{t}^{\gamma} I^{\gamma} f(t) = f(t)$

2. $I_{t_0}^{\gamma} D_{t}^{\gamma} f(t) = f(t) - \sum_{k=0}^{m-k} \frac{t^{k}}{k!} f^{k}(t_o).$

MATERIALS AND METHODS

Model Formulation

In developing the integer-order model for HIV/AIDS, the population is divided into six specific categories: individuals who are susceptible. (S) ,these are people who have not contracted the infection, as well as those who have been exposed to it.Individuals who are not yet infectious; asymptomatically infected individuals (I_A) Population of infected individuals who do not show clinical symptoms; symptomatic infected individuals.(I_S)Population of infected individuals exhibiting clinical symptoms; treated individuals. T_H Population of individuals undergoing treatment but not yet fully recovered R_H Recovered population.

The recruitment rate of individuals into the susceptible population is denoted as Λso that $(\beta_H)$is the effective contact rate of susceptible and infected humans with HIV/AIDS respectively. We denote (θ_H)as the progression rates from exposed HIV/AIDS classes respectively. τ_{H1} is the progression rates from infected HIV class into been symptomatically infected with the virus. τ_{H2} is the rate at which the symptomatically infected humans progresses to become infected with HIV/AIDS. The rate at which the symptomatic and HIV/AIDs infected humans move to the treatment class is denoted as σ_{IS}, σ_{A}respectively. The natural death rate of humans is denoted asμ. HIV/AIDs only classes is denoted respectively as (δ_H).

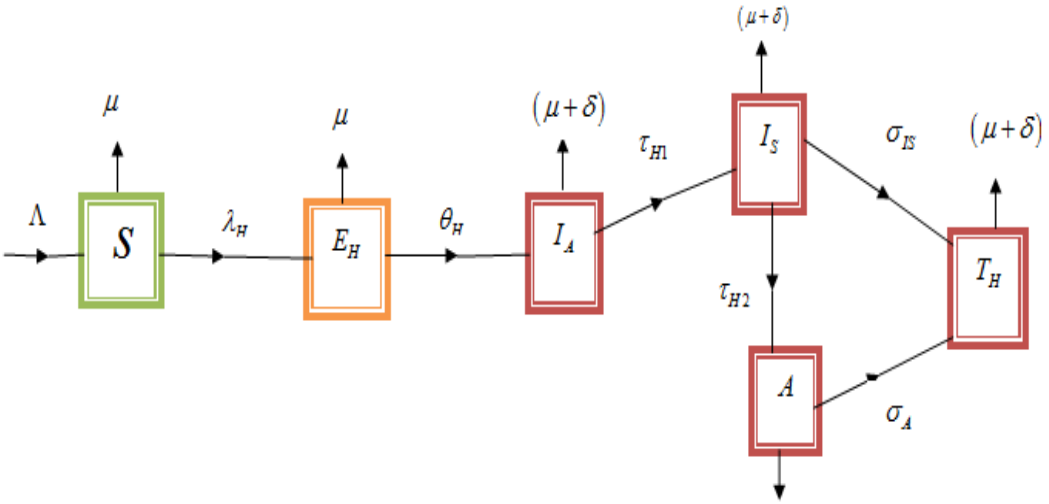

Figure 1: Model Flow Chart

Model Equations

$$\frac{dS}{dt} = \Lambda_H - \frac{\beta_H(I_A + I_S + A)}{N}S - \mu S,$$
$$\frac{dE_H}{dt} = \frac{\beta_H(I_A + I_S + A)}{N}S - (\theta_H + \mu)E_H$$
$$\frac{dI_A}{dt} = \theta_H E_H - (\tau_{H1} + \delta_H + \mu)I_A,$$
$$\frac{dI_S}{dt} = \tau_{H1} I_A - (\tau_{H2} + \sigma_{IS} + \delta_H + \mu)I_S,$$
$$\frac{dA}{dt} = \tau_{H2} I_S - (\sigma_A + \delta_H + \mu)A, \quad (5)$$
$$\frac{dT_H}{dt} = \sigma_{IS} I_S + \sigma_A A - (\delta_H + \mu)T_H.$$

Where

$\boldsymbol{\lambda_H} = \frac{\boldsymbol{\beta_H(I_A+I_S+A)}}{\boldsymbol{N}}$ Is the force of infection**.**

Table 1: Model variables and parameters

Variable	**Description**
$S(t)$	Susceptible Humans
$E_H(t)$	Exposed humans to HIV only
$\boldsymbol{I_H(t)}$	Infected humans with HIV only
$\boldsymbol{I_S(t)}$	Symptomatically infected humans with HIV
$\boldsymbol{A(t)}$	Humans with HIV/AIDS
$\boldsymbol{T_H(t)}$	Treated humans due to HIV only
Parameter	**Description**
$\boldsymbol{\Lambda}$	Recruitment rate of humans
$\boldsymbol{\mu}$	Natural death rate of humans
$\boldsymbol{\beta_H}$	Contact rate of susceptible and infected humans with HIV/AIDs
$\boldsymbol{\lambda_H}$	Force of infection of HIV/AIDS
$\boldsymbol{\delta_H}$	HIV/AIDS disease induced death rate
$\boldsymbol{\tau_{H1}}$	Progression rate from infected HIV/AIDS humans to symptomatic humans with HIV
$\boldsymbol{\tau_{H2}}$	Progression rate from symptomatic HIV humans to AIDS humans class
$\boldsymbol{\sigma_{IS}}$	Treatment rate of symptomatic HIV infected humans
$\boldsymbol{\sigma_A}$	Treatment rate of HIV/AIDS humans

Fractional HIV/AIDs mathematical model

In this section, the HIV/AIDS integer model from Eq. (5) is modified by incorporating the Caputo fractional derivative operator. By doing so, the model gains enhanced flexibility compared to its classical integer-order counterpart. This flexibility arises from the fractional-order formulation, which allows for a wider range of outputs and system behaviors, providing more nuanced insights into the dynamics of HIV/AIDS. The resulting fractional-order HIV/AIDS model is formulated as follows:

$$^cD_t^{\gamma} S_H = \Lambda_H - \frac{\beta_H(I_A + I_S + A)}{N}S - A_1 S$$
$$^cD_t^{\gamma} E_H = \lambda_H S_H - (\theta_H + \mu)\, E_H - A_2\, E_H,$$
$$^cD_t^{\gamma} I_A = \theta_H\, E_H - (\tau_{H1} + \delta_H + \mu)\, I_A - A_3\, A,$$
$$^cD_t^{\gamma} I_S = \tau_{H1} I_A - (\tau_{H2} + \sigma_{IS} + \delta_H + \mu)\, I_S, \quad (6)$$
$$^cD_t^{\gamma} A = \tau_{H2} I_S - (\sigma_A + \delta_H + \mu)A,$$
$$^cD_t^{\gamma} T_H = \sigma_{IS} I_S + \sigma_A A - (\delta_H + \mu)T_H.$$

Subject to positive initial conditions

$$S_H(0) = S_{H0}, E_H(0) = E_{H0}, I_A(0) = I_{A0}, I_S(0) = I_{S0}, A(0) = A_0, T_H(0) = T_{H0}. \quad (7)$$

Positivity of model solution

We ensured that the initial values remained non-negative throughout the analysis.$N(t) \leq \frac{\Lambda}{\mu}$ as $t \to \infty$

Secondly, $if\ limsup\, N_0\,(t) \leq \frac{\Lambda}{\mu}$, thus, the feasible domain for our model is defined as:

$$\Omega = \left\{(S_H, E_H, I_A, I_S, A, T_H) \subset R_+^6 : S + E_H + I_A + I_S + A + T_H \leq \frac{\Lambda}{\mu}, \right\},$$

so that

$\Omega = \Omega_H \subset R_+^6$,

Hence Ω is positively invariant.

If $S_0, E_{H0}, I_{A0}, I_{S0}, A_0\, T_{H0}$.

If the values are non-negative, then the solution to model (6) will remain non-negative for t>0t > 0t>0.

By examining the first equation in Eq. (6), we find that $^cD_t^{\gamma} S_H = -\lambda_H S_H - A_1 S_H$

$^cD_t^{\gamma} S_H = \Lambda - (\lambda_H + A_1)S_H$

$^cD_t^{\gamma} S_H + (\lambda_H + A_1)S_H = \Lambda$

But$\Lambda \geq 0$then

$^cD_t^{\gamma} S_H + (\lambda_H + A_1)S_H \geq 0.$

Applying the Laplace transform we obtained;

$$L\left[\,^cD_t^{\gamma} S_H\right] + L[(\lambda_H + A_1)S_H] \geq 0$$
$$S_H^{\gamma} S_H(s_H) - S_H^{\gamma-1} S_H(0) + (\omega_H + \mu)S_H(s_H) \geq 0,$$
$$S_H(s_H) \geq \frac{S_H^{\gamma-1}}{S_H^{\gamma} + (\omega_H + \mu)} S_H(0).$$

By taking the inverse Laplace transform, we obtained ;

$S_H(t) \geq E_{Hr,1}(-(\lambda_H + \mu)t^{\gamma})\, S_{H0}\ldots.$ (8)

Now since the term on the right hand side of Eq. (8) is positive, we conclude that $S_H \geq 0$for $t \geq 0$. In the same way, we also have that $E_H \geq 0, I_A \geq 0, I_S \geq 0, A \geq 0, T_H \geq 0$,to be positive, therefore, the solution will remain in R_+^6 for all $t \geq 0$ with positive initial conditions.

Boundedness of fractional model solution.

The total population of individuals from our model is given by ;

$N(t) = S_H(t) + E_H(t) + I_A(t) + I_S(t) + A(t) + T_H(t).$

So from our fractional model (6), we now obtain

$$cD_t^{\gamma} N(t) = {}^c D_t^{\gamma} S_H(t) + {}^cD_t^{\gamma} E_H(t) + {}^cD_t^{\gamma} I_A(t) + {}^cD_t^{\gamma} I_S(t) + {}^cD_t^{\gamma} A(t) + {}^cD_t^{\gamma} T_H(t),$$
$$cD_t^{\gamma} N(t) = \Lambda - \mu N(t) \quad (9)$$

Taking the Laplace transformation of (10) we obtained;

$$L\left[cD_t^{\gamma} N(t)\right] = L[\Lambda - \mu N(t)]$$
$$S_H^{\gamma} N(s_H) - S_H^{\gamma-1} N(0) + \mu N(s) \leq \frac{\Lambda}{\mu},$$
$$N(s_H) \leq \frac{S_H^{\gamma-1}}{(S^{\gamma} + \mu)} N(0) + \frac{\Lambda}{S_H(S_H^{\gamma} + \mu)} \quad (10)$$

By taking the inverse Laplace transform of Eq. (10) we obtained ;

$N(t) \leq E_{Hr,1}(-\mu t^{\gamma})N(0) + \Lambda E_{Hr,r+1}(-\mu t^{\gamma})$ (11)

At $t \to \infty$, the limit of Eq. (11) becomes

$\underset{t\to\infty}{limSup} N(t) = \frac{\Lambda}{\mu}$. (12)

This means that, if $N_0 \leq \frac{\Lambda}{\mu}$ then $N(t) \leq \frac{\Lambda}{\mu}$ which implies that,$N(t)$ is bounded.

We now conclude that, this region $\Omega = \Omega_H$, is well posed and equally feasible epidemiologically.

Existence and uniqueness of our model solution

Let the real non-negative be P, we consider $W = [0, K[]$

The set of all continuous function that is defined on M is represented by $N_e^0(W)$ with norm as;

$\|X\| = Sup\{|K(t)|, t \in W\}$.

Considering model (6) with initial conditions presented in (7) which can be denoted as an initial value problem (IVP) in (12).

${}^{c}D_t^{\gamma}(t) = Z\big(t, X(t)\big), 0 < t < P < \infty,$

$X(0) = X_0$.

Where $Y(t) = \big(S_H(t), E_H(t), I_A(t), I_S(t), A(t), T_H(t)\big)$.represents the classes and Z be a continuous function defined as follows;

$$Z\big(t, X(t)\big) = \begin{pmatrix} Z_1\big(t, S_H(t)\big) \\ Z_2(t, E_H(t)) \\ Z_3(t, I_A(t)) \\ Z_4(t, I_S(t)) \\ Z_5\big(t, A(t)\big) \\ Z_6(t, T_H(t)) \end{pmatrix} = \begin{pmatrix} \Lambda - \left(\frac{\beta_H(I_H+I_S+A)}{N} + \mu\right) S_H. \\ \left(\frac{\beta_H(I_A+I_S+A)}{N} + \mu\right) S_H - (\theta_H + \mu)\, E_H. \\ \theta_H E_H - (\tau_{H1} + \delta_H + \mu) I_A \\ \tau_{H1} I_H - (\tau_{H2} + \sigma_{IS} + \delta_H + \mu) I_S \\ \tau_{H2} I_S - (\sigma_A + \delta_H + \mu) A \\ \sigma_{IS} I_S + \sigma_A A - (\delta_H + \mu) T_H \end{pmatrix} \quad (13)$$

Using proposition (2.1), we have that,

$$\begin{aligned} S_H(t) &= S_{H0} + I_t^{\gamma}\left[\Lambda - \left(\frac{\beta_H(I_H + I_S + A)}{N} + \mu\right) S_H.\right], \\ E_H(t) &= E_{H0} + I_t^{\gamma}\left[\left(\tfrac{\beta_H(I_A+I_S+A)}{N} + \mu\right) S_H - (\theta_H + \mu)\, E_H \quad\right], \\ I_A(t) &= I_{A_0} + I_t^{\gamma}[\theta_H E_H - (\tau_{H1} + \delta_H + \mu) I_A], \\ I_S(t) &= I_{S_0} + I_t^{\gamma}[\tau_{H1} I_H - (\tau_{H2} + \sigma_{IS} + \delta_H + \mu) I_S], \\ A(t) &= A_0 + I_t^{\gamma}[\tau_{H2} I_S - (\sigma_A + \delta_H + \mu) A], \\ T_H(t) &= T_{H0} + I_t^{\gamma}[\sigma_{IS} I_S + \sigma_A A - (\delta_H + \mu) T_H]. \end{aligned} \quad (14)$$

We obtain the Picard iteration of (12) as follows;

$$\begin{aligned} S_{Hn}(t) &= S_{H0} + \frac{1}{\Gamma(\gamma)}\int_0^t (t - \lambda_H)^{\gamma-1} Z_1\left(\lambda_H, S_{Hn-1}(\lambda_H)\right) d\,\lambda_H, \\ E_{Hn}(t) &= E_{H0} + \frac{1}{\Gamma(\gamma)}\int_0^t (t - \lambda_H)^{\gamma-1} Z_2\left(\lambda_H, E_{H(n-1)}(\lambda_H)\right) d\,\lambda_H, \\ I_{An}(t) &= I_{A0} + \frac{1}{\Gamma(\gamma)}\int_0^t \left(t - \lambda_H\right)^{\gamma-1} Z_3\left(\lambda_H, I_{A(n-1)}\left(\lambda_H\right)\right) \mathrm{d}\,\lambda_H, \\ I_{Sn}(t) &= I_{S0} + \frac{1}{\Gamma(\gamma)}\int_0^t (t - \lambda_H)^{\gamma-1} Z_4\left(\lambda_H, I_{S_{(n-1)}}(\lambda_H)\right) d\,\lambda_H, \\ A(t) &= A_0 + \tfrac{1}{\Gamma(\gamma)}\textstyle\int_0^t (t - \lambda_H)^{\gamma-1} Z_5\left(\lambda_H, A_{(n-1)}(\lambda_H)\right) d\,\lambda_H. \\ T_{Hn}(t) &= T_{H0} + \frac{1}{\Gamma(\gamma)}\int_0^t (t - \lambda_H)^{\gamma-1} Z_6\left(\lambda_H, T_{H_{(n-1)}}(\lambda_H)\right) d\,\lambda_H, \end{aligned} \quad (15)$$

Lemma 2. The initial value problem (6), (7) in Eq. (15 exists and will have a unique solution $X(t) \in A_c^0(f)$.

Using Picard-Lindelof and fixed point theory, we consider the solution of

$X(t) = S_H(X(t))$,

where S is defined as the Picard operator expressed as ;

$S_H : A_c^0(f, R_+^6) \to A_c^0(f, R_+^6)$.

Therefore,

$S_H\big(X(t)\big) = X(0) + \frac{1}{\Gamma(\gamma)}\int_0^t (t - \lambda_H)^{\gamma-1} Z\,(\lambda_H, X(\lambda_H))\, d\,\lambda_H.$

which becomes

$$\begin{aligned} &\|S_H(X_1(t)) - S_H(X_2(t))\| \\ &= \left\|\frac{1}{\Gamma(\gamma)}\left[\int_0^t (t - \lambda_H)^{\gamma-1} Z(\lambda_H, X_1(\lambda_H)) - Z(\lambda_H, X_2(\lambda_H))\, d\,\lambda_H\right]\right\| \\ &\leq \frac{1}{\Gamma(\gamma)}\int_0^t (t - \lambda_H)^{\gamma-1}\, \|Z(\lambda_H, X_1(\lambda_H)) - Z(\lambda_H, X_2(\lambda_H))\, d\,\lambda_H\|. \\ &\leq \frac{\psi}{\Gamma(\gamma)}\int_0^t (t - \lambda_H)^{\gamma-1}\, \|X_1 - X_2 \quad\| d\lambda_H. \\ &\|S_H(X_1(t)) - S_H(X_2(t))\| \leq \frac{\psi}{\Gamma(\gamma + 1)S_H}. \end{aligned}$$

When $\frac{\psi}{\Gamma(\gamma+1)} S_H \leq 1$,

The application of the Picard operator leads to a contradiction, which confirms that the solutions to Eq. (5) and Eq. (6) are indeed unique.

We now transformed the initial value problem of Eq. (13) to obtain ;

$$X(t) = X(0) + \frac{1}{\Gamma(\gamma)} \int_0^t (t - \lambda_H)^{\gamma-1} Z\left(\lambda_H, X(\lambda_H)\right) d\,\lambda_H. \quad (16)$$

Lemma 1, The Lipchitz condition described from Eq. (13) is satisfied by vector $Z\big(t, X(t)\big)$ on a set $[0, W[]_+^6]$ with the Lipchitz constant given as;

$$\psi = max\Big(\big(\beta_{H_1}^* + \beta_{H_2}^* + \beta_{H_3}^* + \mu\big), (\theta_H + \mu), (\tau_{H1} + \delta_H + \mu), (\tau_{H2} + \sigma_{IS} + \delta_H + \mu), (\sigma_A + \delta_H + \mu), (\delta_H + \mu)\Big).$$

Proof.

$$\|Z_1(t, S_H) - Z_1(t, S_{H1})\|$$
$$= \left\| \Lambda - \left(\frac{\beta_H (I_H + I_S + A)}{N} + \mu \right) S_H - \Lambda - \left(\frac{\beta_H (I_H + I_S + A)}{N} + \mu \right) S_{H1} \right\|$$
$$= \left\| -\Lambda - \left(\frac{\beta_H (I_H + I_S + A)}{N} + \mu \right) (S_H - S_{H_1}) + \mu (S_H - S_{H_1}) \right\|$$
$$\leq \left(\beta_{H_1}{}^* + \beta_{H_2}{}^* + \beta_{H_3}{}^*\right) \|S_H - S_{H1}\| + \mu \|S_H - S_{H1}\|$$
$$\therefore \|Z_1(t, S_H) - Z_1(t, S_{H1})\| \leq (\beta_{H1}{}^* + \beta_{H2}{}^* + \beta_{H3}{}^* + \mu) \|S_H - S_{H1}\|$$

Similarly we obtained the following;

$$\left\| Z_2\left(t, \mathrm{E}_H\right) - Z_2\left(t, \mathrm{E}_{H1}\right) \right\| \leq \left(\theta_H + \mu\right) \left\| \mathrm{E}_H - \mathrm{E}_{H1} \right\|,$$
$$\left\| Z_3\left(t, \mathrm{I}_A\right) - Z_3\left(t, \mathrm{I}_{A1}\right) \right\| \leq \left(\tau_{H1} + \delta_H + \mu\right) \left\| \mathrm{I}_A - \mathrm{I}_{A1} \right\|,$$
$$\left\| Z_4\left(t, \mathrm{I}_S\right) - Z_4\left(t, \mathrm{I}_{S1}\right) \right\| \leq \left(\tau_{H2} + \sigma_{IS} + \delta_H + \mu\right) \left\| \mathrm{I}_S - \mathrm{I}_{S1} \right\|$$
$$\|Z_5(t, A) - Z_5(t, A_1)\| \leq (\delta_H + \mu) \|A - A_1\|, \quad (17)$$
$$\left\| Z_6\left(t, \mathrm{T}_H\right) - Z_6\left(t, \mathrm{T}_{H1}\right) \right\| \leq \left(\sigma_A + \delta_H + \mu\right) \left\| \mathrm{T}_H - \mathrm{T}_{H1} \right\|.$$

Where we obtained

$$\|Z(t, X_1(t)) - Z(t, X_2(t))\| \leq \psi \|X_1 - X_2 \quad \|,$$
$$\psi = max\Big(\big(\beta_{H_1}^* + \beta_{H_2}^* + \beta_{H_3}^* + \mu\big), (\theta_H + \mu), (\tau_{H1} + \delta_H + \mu), (\tau_{H2} + \sigma_{IS} + \delta_H + \mu), (\sigma_A + \delta_H + \mu), (\delta_H + \mu)\Big). \quad (18)$$

Lemma 2. The initial value problem (5), (6) in Eq. (18) exists and will have a unique solution

$X(t) \in A_c^0(f)$.

Using Picard-Lindelof and fixed point theory, we consider the solution of

$X(t) = S_H(X(t))$,

where S is defined as the Picard operator expressed as ;

$S_H : A_c^0(f, R_+^6) \to A_c^0(f, R_+^6)$.

Therefore,

$$S_H\big(X(t)\big) = X(0) + \frac{1}{\Gamma(\gamma)} \int_0^t (t - \lambda_H)^{\gamma-1} Z\left(\lambda_H, X(\lambda_H)\right) d\,\lambda_H.$$

which becomes

$$\|S_H(X_1(t)) - S_H(X_2(t))\|$$
$$= \left\| \frac{1}{\Gamma(\gamma)} \left[\int_0^t (t - \lambda_H)^{\gamma-1} Z(\lambda_H, X_1(\lambda_H)) - Z(\lambda_H, X_2(\lambda_H)) \, d\,\lambda_H \right] \right\|$$
$$\leq \frac{1}{\Gamma(\gamma)} \int_0^t (t - \lambda_H)^{\gamma-1} \|Z(\lambda_H, X_1(\lambda_H)) - Z(\lambda_H, X_2(\lambda_H)) \, d\,\lambda_H\|.$$
$$\leq \frac{\psi}{\Gamma(\gamma)} \int_0^t (t - \lambda_H)^{\gamma-1} \|X_1 - X_2 \quad \| d\lambda_H.$$
$$\|S_H(X_1(t)) - S_H(X_2(t))\| \leq \frac{\psi}{\Gamma(\gamma + 1) S_H}.$$

When $\frac{\psi}{\Gamma(\gamma+1)} S_H \leq 1$, then the Picard operator gives a contradiction , so Eq.(5) , (6) solution is unique.

The basic reproduction number (R_0) and model equilibrium points:

The disease-free equilibrium (DFE) point in a mathematical model represents a steady state where no infection persists in the population, meaning that the number of infected individuals is zero. In epidemiological models, this equilibrium occurs when the disease is either eradicated or prevented from spreading within a population

The disease free equilibrium point of the model (5) is expressed as:

$$(\mathrm{HDFEP}) = \left((S^*, E_H^*, I_A{}^*, I_S{}^*, A^*, T_H^*, R_H^*) = \left(\frac{\Lambda}{\mu}, 0,0,0,,0,0\right) \right) \quad (19)$$

$$Letn = (E_H, I_A, I_S, T_H)$$

$$Sothat \frac{dn}{dt} = F - V.$$

$$F_H = \begin{bmatrix} 0 & \beta_{H1} & \beta_{H2} & \beta_{H3} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \quad V_H = \begin{bmatrix} P_1 & 0 & 0 & 0 & 0 \\ -\theta_H & P_2 & 0 & 0 & 0 \\ 0 & -\tau_{H1} & P_3 & 0 & 0 \\ 0 & 0 & -\tau_{H2} & P_4 & 0 \\ 0 & 0 & -\sigma_{IS} & -\sigma_A & P_5 \end{bmatrix}$$

Where $P_1 = (\theta_H + \mu)$, $P_2 = (\tau_{H1} + \delta_H + \mu)$,$P_3 = (\tau_{H2} + \sigma_{IS} + \delta_H + \mu)$,$P_4 = (\sigma_A + \delta_H + \mu)$,$P_5 = (\delta_H + \mu)$
In mathematical terms, the basic reproduction number is calculated as$R_0 = \rho(FV^{-1})$ where ρ is the dominant Eigen value of the system (FV^{-1}). Where R_0^H is the basic reproduction number associated with the individuals in the population.

$$R_0^H = \frac{\beta_H \theta_H \left((P_3 + \tau_{H1})P_4 + \tau_{H2}\tau_{H1}\right)}{P_3 P_2 P_1 P_4}$$

$$R_0^H = \frac{\beta_H \theta_H \left(((\tau_{H2}+\sigma_{IS}+\delta_H+\mu)+\tau_{H1})(\sigma_A+\delta_H+\mu)+\tau_{H2}\tau_{H1}\right)}{(\tau_{H2}+\sigma_{IS}+\delta_H+\mu)(\tau_{H1}+\delta_H+\mu)(\theta_H+\mu)(\sigma_A+\delta_H+\mu)} \quad (20)$$

Endemic equilibrium point

We explored the possibility of an endemic equilibrium point, which represents a positive steady state where HIV/AIDS continues to exist within the population. At this equilibrium, all model variables reach constant values, indicating that the disease maintains a consistent presence rather than disappearing. This analysis is crucial for understanding how the disease can sustain itself over time and the factors influencing its persistence in a given population
non-zero.$(S_H^* \neq 0, E_H^* \neq 0, I_A^* \neq 0, I_S^* \neq 0, A^* \neq 0 \; and \quad T_H^* \neq 0)$.
To analyze the endemic equilibrium point, the model equations are examined in relation to the force of infection affecting human populations. In the context of the fractional HIV/AIDS model (6), the endemic equilibrium state is characterized by the specific values of the model variables that indicate a sustained presence of the disease within the population: $E_{**} = (S_H^{**}, E_H^{**}, I_A^{**}, I_S^{**}, A^{**}T_H^{**},)$,
Defined as;

$$S^{**} = \frac{\Lambda}{\lambda_H^{**} + \mu}$$

$$E_H^{**} = \frac{\lambda_H^{**}\Lambda}{(\lambda_H^{**} + \mu)(\theta_H + \mu)} = \frac{\Lambda\lambda_H^{**}}{(\lambda_H^{**} + \mu)P_1}$$

$$I_A^{**} = \frac{\Lambda\lambda_H^{**}\theta_H}{(\lambda_H^{**} + \mu)P_1P_2}$$

$$I_S^{**} = \frac{\Lambda\lambda_H^{**}\theta_H\tau_{H1}}{(\lambda_H^{**} + \mu)P_1P_2P_3} \quad (21)$$

$$A^{**} = \frac{\Lambda\lambda_H^{**}\theta_H\tau_{H2}\tau_{H1}}{(\lambda_H^{**} + \mu)P_1P_2P_3P_4}$$

$$T_H^{**} = \frac{\Lambda\lambda_H^{**}\theta_H\left(\sigma_{IS}\tau_{H1} + \sigma_A\tau_{H2}\right)}{(\lambda_H^{**} + \mu)P_1P_2P_3P_4}$$

Where$P_1 = (\theta_H + \mu)$,
$P_2 = (\tau_{H1} + \delta_H + \mu)$,$P_3 = (\tau_{H2} + \sigma_{IS} + \delta_H + \mu)$,$P_4 = (\sigma_A + \delta_H + \mu)$,$P_5 = (\delta_H + \mu)$
Substituting into the force of infection $\lambda_H = \frac{\beta_H(I_A+I_S+A)}{N}$

$$\lambda_H^{**} = \frac{P_1P_2P_3\left(P_5P_4 - \sigma_A\phi_H\right)\left(R_0^H - 1\right)}{\left[\left(\left(-\phi_H + \tau_{H2}\right)\sigma_A + \left(\tau_{H2} + P_4\right)P_5\right)P_3 + \tau_{H1}\left(P_4\left(\sigma_S + P_5\right)\right)\right]\theta_H + P_2P_3\left(P_5P_4 - \sigma_A\phi_H\right)} \quad (22)$$

$R_0^H - 1 > 0$

Which implies that , the endemic equilibrium point of model (5) is stable.

Global stability analysis at endemic equilibrium state

The global stability of the equilibrium point is assessed using the direct Lyapunov method. The endemic equilibrium point is considered globally stable when the basic reproduction number exceeds one, indicating that the disease will disseminate through the population, irrespective of the initial conditions. This analysis applies to the fractional model (6), providing insights into the conditions under which the disease maintains its presence within the population.

Where $N \leq \frac{\Lambda}{\mu} ast \to \infty$, and

then$\lambda_H = \beta_{H1}I_A + \beta_{H2}I_S + \beta_{H3}T_H$
our fractional model now becomes

$${}^{c}D_t^{\gamma} S_H = \Lambda_H - \frac{\beta_H(I_A + I_S + A)}{N}S - A_1S$$

$${}^{c}D_t^{\gamma} E_H = \lambda_H S_H - (\theta_H + \mu)\, E_H - A_2\, E_H,$$

$${}^{c}D_t^{\gamma} I_A = \theta_H\, E_H - (\tau_{H1} + \delta_H + \mu)\, I_A - A_3\, A,$$

$$
\begin{aligned}
&{}^{c}D_t^{\gamma} I_S = \tau_{H1} I_A - (\tau_{H2} + \sigma_{IS} + \delta_H + \mu)\, I_S,\\
&{}^{c}D_t^{\gamma} A = \tau_{H2} I_S - (\sigma_A + \delta_H + \mu) A,\\
&{}^{c}D_t^{\gamma} T_H = \sigma_{IS} I_S + \sigma_A A - (\delta_H + \mu) T_H.,
\end{aligned} \tag{23}
$$

.

At equilibrium point Eq. (23) has the following results

$\Lambda = \lambda_{H1}^{*} S_H^{*} + \mu S_H^{*}$, $A_2 E_H^{*} = \lambda_{H1}^{*} S^{*}$, $A_3 I_A^{*} = \theta_H E_H^{*}$, $A_4 I_S^{*} = \tau_{H1} I_A^{*}$, $A_5 A^{*} = \tau_{H2} I_S^{*}$,

$A_6 A^{*} = \sigma_{IS} I_S^{*} + \sigma_A A^{*}$.

Theorem 1

Model (20) is globally asymptotically stable if $R0 >$

whenever

$$
\left(6 - \frac{S_H^{*}}{S_H} + \frac{\lambda_{H1}}{\lambda_{H1}^{**}}\left(1 - \frac{S_H E_H^{*}}{S_H^{*} E_H}\right) - \frac{I_A^{*} E_H}{I_A E_H^{*}} - \frac{I_S^{*} E_H}{I_S E_H^{*}} - \frac{A^{*} E_H}{A E_H^{*}} - \frac{T_H}{T_H^{*}} - \frac{T_H^{*} I_A I_S A}{T_H I_A^{*} I_S^{*} A^{*}}\right) \le 0.
$$

$$
Let L(t) = L_H(t)
$$

be a non-linear Lyaponov function as presented in (21) below:

$$
\begin{aligned}
L(t) &= L_1\left(S_H - S_H^{*} - S_H^{*} ln\frac{S_H}{S_H^{*}}\right) + L_2\left(E_H - E_H^{*} - E_H^{*} ln\frac{E_H}{E_H^{*}}\right) + L_3\left(I_A - I_A^{*} - I_A^{*} ln\frac{I_A}{I_A^{*}}\right)\\
&+L_4\left(I_S - I_S^{*} - I_S^{*} ln\frac{I_S}{I_S^{*}}\right) + L_5\left(A - A^{*} - A^{*} ln\frac{A}{A^{*}}\right) + L_6\left(T_H - T_H^{*} - T_H^{*} ln\frac{T_H}{T_H^{*}}\right).
\end{aligned} \tag{24}
$$

Taking the Caputo Fractional order derivative of Eq. (25), we have

$$
\begin{aligned}
&{}^{c}D_t^{\gamma} L(t) = {}^{c}D_t^{\gamma} L_H(t) \le L_1\left(1 - \frac{S_H^{*}}{S_H}\right) {}^{c}D_t^{\gamma} S_H(t) + L_2\left(1 - \frac{E_H^{*}}{E_H}\right) {}^{c}D_t^{\gamma} E_H(t) + L_3\left(1 - \frac{I_A^{**}}{I_A}\right) {}^{c}D_t^{\gamma} I_A(t)\\
&+L_4\left(1 - \frac{I_S^{*}}{I_S}\right) {}^{c}D_t^{\gamma} I_S(t) + L_5\left(1 - \frac{A^{*}}{A}\right) {}^{c}D_t^{\gamma} A(t) + L_6\left(1 - \frac{T_H^{*}}{T_H}\right) {}^{c}D_t^{\gamma} T_H(t),\\
&= \lambda_{H1}^{*} S_H^{*}\left(\begin{array}{l}\left(1 - \frac{S_H^{*}}{S}\right) {}^{c}D_t^{\gamma} S_H(t) + \left(1 - \frac{E_H^{*}}{E_H}\right) {}^{c}D_t^{\gamma} E_H(t) + \left(1 - \frac{I_A^{*}}{I_{AH}}\right) {}^{c}D_t^{\gamma} I_A(t)\\ +\left(1 - \frac{I_S^{*}}{I_S}\right) {}^{c}D_t^{\gamma} I_S(t) + \left(1 - \frac{A^{*}}{A}\right) {}^{c}D_t^{\gamma} A(t)\left(1 - \frac{T_H^{*}}{T_H}\right) {}^{c}D_t^{\gamma} T_H(t),\end{array}\right).\\
&\left(1 - \frac{S_H^{*}}{S_H}\right) {}^{c}D_t^{\gamma} S_H = \left(1 - \frac{S_H^{*}}{S_H}\right)(\lambda_{H1}^{*} S_H^{*} + \mu S_H^{*} - \lambda_{H1} S_H - \mu S_H),\\
&= \lambda_{H1}^{*} S_H^{*}\left(1 - \frac{S_H \lambda_{H1}}{\lambda_{H1}^{*} S_H^{*}} - \frac{S_H^{*}}{S_H} + \frac{\lambda_{H1}}{\lambda_{H1}^{*}}\right) + \mu S_H^{*}\left(2 - \frac{S_H}{S_H^{*}} - \frac{S_H^{*}}{S_H}\right),\\
&\left(1 - \frac{E_H^{*}}{E_H}\right) {}^{c}D_t^{\gamma} E_H = \left(1 - \frac{E_H^{*}}{E_H}\right)\left(\lambda_{H1}^{*} S_H - \lambda_{H1}^{*} S_H^{*} \frac{E_H}{E_H^{*}}\right),\\
&= \lambda_{H1}^{*} S_H^{*}\left(1 - \frac{S_H \lambda_{H1}^{*}}{\lambda_{H1}^{*} S_H^{*}} \frac{E_H^{*}}{E_H} - \frac{E_H^{*}}{E_H} + \frac{S_H^{*} \lambda_{H1}}{\lambda_{H1}^{*} S_H^{*}}\right),\\
&\frac{A_2}{\theta_H}\left(1 - \frac{I_A^{*}}{I_A}\right) {}^{c}D_t^{\gamma} I_A = \frac{A_2}{\theta_H}\left(1 - \frac{I_A^{*}}{I_A}\right)\left(\theta_H E_H - A_3 \frac{I_A}{I_A^{*}} I_A^{*}\right),\\
&= \lambda_{H1}^{*} S_H^{*}\left(1 + \frac{E_H}{E_H^{*}} - \frac{I_A}{I_A^{*}} - \frac{E_H I_A^{*}}{E_H^{*} I_A}\right),\\
&\frac{A_2 A_3}{\tau_{H1}\theta_H}\left(1 - \frac{I_S^{*}}{I_S}\right) {}^{c}D_t^{\gamma} I_S = \frac{A_2 A_3}{\tau_{H1}\theta_H}\left(1 - \frac{I_S^{*}}{I_S}\right)\left(\tau_{H1} E_H - A_4 \frac{I_S}{I_S^{*}} I_S^{*}\right),\\
&= \lambda_{H1}^{*} S_H^{*}\left(1 - \frac{I_S^{*}}{I_S} - \frac{I_A I_S^{*}}{I_S^{*} I_S} + \frac{I_A}{I_A^{*}}\right),\\
&\frac{A_1}{\tau_{H2}}\left(1 - \frac{A^{*}}{A}\right) {}^{c}D_t^{\gamma} A = \frac{A_1}{\tau_{H2}}\left(1 - \frac{A^{*}}{A}\right)\left(\tau_{H2} E_H - A_5 \frac{A}{A^{*}} A^{*}\right),\\
&= \lambda_{H1}^{*} S_H^{*}\left(1 - \frac{E_H}{E_H^{*}} - \frac{A}{A^{*}} + \frac{E_H A^{*}}{E_H^{*} A}\right).\\
&\frac{A_6}{\sigma_A}\left(1 - \frac{T_H^{*}}{T_H}\right) {}^{c}D_t^{\gamma} T_S = \frac{A_6}{\sigma_A}\left(1 - \frac{T_H^{*}}{T_H}\right)\left(\sigma_A E_H - A_6 \frac{T_H}{T_H^{*}} T_H^{*}\right),\\
&= \lambda_{H1}^{*} S_H^{*}\left(1 + \frac{E_H}{E_H^{*}} - \frac{T_H}{T_H^{*}} - \frac{E_H T_H^{*}}{E_H^{*} T_H}\right).
\end{aligned} \tag{25}
$$

Hence, Eq. (26) now becomes;

$$
{}^{c}D_t^{\gamma} L(t) \le \lambda_{H1}^{*} S_H^{*}
$$

$$
\left(6 - \frac{S_H^{*}}{S_H} + \frac{\lambda_{H1}}{\lambda_{H1}^{**}}\left(1 - \frac{S_H E_H^{*}}{S_H^{*} E_H}\right) - \frac{I_A^{*} E_H}{I_A E_H^{*}} - \frac{I_S^{*} E_H}{I_S E_H^{*}} - \frac{A^{*} E_H}{A E_H^{*}} - \frac{T_H}{T_H^{*}} - \frac{T_H^{*} I_A I_S A}{T_H I_A^{*} I_S^{*} A^{*}}\right) \le 0.
$$

$$
Which implies that,\ {}^{c}D_t^{\gamma} L(t) \le \lambda_{H1}^{*} S_H^{*} \psi (R_0 - 1) \lambda_H S_H^{*}
$$

$$\left(6-\frac{S_H^*}{S_H}+\frac{\lambda_{H1}}{\lambda_{H1}^{**}}\left(1-\frac{S_H E_H^*}{S_H^* E_H}\right)-\frac{I_A^* E_H}{I_A E_H^*}-\frac{I_S^* E_H}{I_S E_H^*}-\frac{A^* E_H}{A E_H^*}-\frac{T_H}{T_H^*}-\frac{T_H^* I_A I_S A}{T_H I_A^* I_S^* A^*}\right).$$

$$-\psi(R_0-1)\lambda_H S_H^*\left[A_1 S_H^*\left(\frac{S_H^*}{S_H}-1-ln\frac{S_H^*}{S_H}\right)\right] \quad (29)$$

Therefore ${}^cD_t^\gamma L(t)\le 0 for R_0>1$.This implies that ${}^cD_t^\gamma L(t)=0$. If $E_*=(S^*,E_H^*,I_A^*,I_S^*,A^*,T_H^*)$, is the endemic equilibrium point. , then by LaSalle's invariance principle, the endemic equilibrium point is globally asymptotically stable in Ω whenever $R_0>1$.

Fractional order model numerical results

We numerically solved the fractional-order HIV/AIDS model using the generalized fractional Adams-Bashforth–Moulton method described by Chan *et al* (2020). The parameter values utilized in the model are detailed in Table 1, which also presents simulations that incorporate various fractional-order values. This approach allows for a comprehensive analysis of the model's behavior under different conditions and provides insights into the dynamics of HIV/AIDS transmission (γ).

Implementation of the Fractional Adams–Bashforth–Moulton Method

We utilized the approach outlined by Baskonus et al(2015)., Diethelm, as detailed in NCDC (2019), Diethelm (1999), Baskonus et al. (2015), and Liu et al. (2023) for this study. The solution to the fractional HIV/AIDS model presented in (6) was approximated using the fractional Adams–Bashforth–Moulton method. This fractional model (6) is expressed by Chan *et al* (2020). as follows:

$${}^cD_t^\gamma P(t)=Q(t,q(t)),0<t<\beta, \quad (26)$$

$$P^{(n)}(0)=P_0^{(n)},n=1,0,\dots,q,q=[\gamma]. \quad (27)$$

Where $P=(S^*,E_H^*,I_A{}^*,I_S^*,A^*,T_H^*)\in R_+^6$ and $M(t,q(t))$ is a real valued function that is continuous.

Eq. (27) can be therefore be represented using the concept of fractional integral as follows;

$$P(t)=\sum_{n=0}^{m-1}P_0^{(n)}\frac{t^n}{n!}+\frac{1}{\Gamma(\gamma)}\int_0^t(t-y)^{\gamma-1}R(y,m(y))dy. \quad (28)$$

Using the method described by Baskonus et al. (2015), we let the step size $g=\frac{\beta}{N},N\in N$ with a grid that is uniform on $[0,\beta]$. Where $t_c=cr,c=0,1,1,\dots N$. Therefore, the fractional order model of HIV/AIDs model presented in (6) can be approximated as :

$$S_{Hk+1}(t)=S_{H0}+\frac{g^\gamma}{\Gamma(\gamma+2)}\left\{\lambda_H-\left(\beta_{H1}I_A^n+\beta_{H2}I_S^n+\beta_{H_3}T_H^n\right)\frac{S_H^n}{N_H^n}-\mu S_H^n\right\}+$$

$$\frac{g^\gamma}{\Gamma(\gamma+2)}\sum_{y=0}^{k}dy,k+1\left\{\lambda_H-\left(\beta_{H1}I_A^n+\beta_{H2}I_S^n+\beta_{H_3}T_H^n\right)\frac{S_y}{N_{Hy}}-\mu S_y\right\}$$

$$E_{H_{k+1}}(t)=E_{H_0}+\frac{g^\gamma}{\Gamma(\gamma+2)}\left\{\left(\beta_{H1}I_A^n+\beta_{H2}I_S^n+\beta_{H_3}T_H^n\right)\frac{S^n}{N^n}-A_2E_H^n\right\}+$$

$$\frac{g^\gamma}{\Gamma(\gamma+2)}\sum_{y=0}^{k}dy,k+1\left\{\left(\beta_{H1}I_A^n+\beta_{H2}I_S^n+\beta_{H_3}T_H^n\right)\frac{S_y}{N_{Hy}}-A_2E_{Hy}\right\},$$

$$I_{Ak+1}(t)=I_{AH0}+\frac{g^\gamma}{\Gamma(\gamma+2)}\{\theta_H E_H^n-(\tau_{H1}+\delta_H+\mu)I_A^n-A_3A^n\ \}+$$

$$\frac{g^\gamma}{\Gamma(\gamma+2)}\sum_{y=0}^{k}dy,k+1\{\theta_H E_{Hy}-\left(\tau_{H1}+\delta_{Hy}+\mu\right)I_{Ay}-A_3A_{AHy}\ \},$$

$$I_{S_{k+1}}(t)=I_{S_0}+\frac{g^\gamma}{\Gamma(\gamma+2)}\{\tau_{H1}I_A^n-(\tau_{H2}+\sigma_{IS}+\delta_H+\mu)I_S^n\ \}+$$

$$\frac{g^\gamma}{\Gamma(\gamma+2)}\sum_{y=0}^{k}dy,k+1\{\tau_{H1}I_{Ay}-(\tau_{H2}+\sigma_{IS}+\delta_H+\mu)I_{Sy}\ \},$$

$$A_{k+1}(t)=A_0+\frac{g^\gamma}{\Gamma(\gamma+2)}\{\tau_{H2}I_S^n-(\sigma_A+\delta_H+\mu)A^n\}+$$

$$\frac{g^\gamma}{\Gamma(\gamma+2)}\sum_{y=0}^{k}dy,k+1\{\tau_{H2}I_{Sy}-(\sigma_A+\delta_H+\mu)A_y\}, \quad (29)$$

$$T_{H_{k+1}}(t)=T_{H_0}+\frac{g^\gamma}{\Gamma(\gamma+2)}\{\sigma_{IS}I_S^n+\sigma_AA^n-(\delta_H+\mu)T_H^n\}+$$

$$\frac{g^\gamma}{\Gamma(\gamma+2)}\sum_{y=0}^{k}dy,k+1\{\sigma_{IS}I_{Sy}+\sigma_{Ay}A-(\delta_H+\mu)T_y\},$$

Where

$$S_{k+1}^n(t)=S_0+\frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k}f_{y,k+1}\left\{\lambda-\left(\beta_{H1}I_{Ay}+\beta_{H1}I_{Sy}+A_y\right)\frac{S_y}{N_y}-\mu S_y\right\},$$

$$E_{Hk+1}^n(t)=E_{H0}+\frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k}f_{y,k+1}\left\{\left(\beta_{H1}I_{Ay}+\beta_{H1}I_{Sy}+A_y\right)\frac{S_y}{N_{Hy}}-\mu E_{Hy}\right\},$$

$$I_{Ak+1}^n(t)=I_{A0}+\frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k}f_{y,k+1}\left\{\rho E_{Hy}-A_2I_{Ay}\right\}, \quad \dots(30)$$

$$I^n_{Sk+1}(t) = I_{S0} + \frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k} f_{y,k+1}\{\theta_H I_{Ay} - A_3 I_{Sy}\},$$
$$T^n_{Hk+1}(t) = T_{H0} + \frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k} f_{y,k+1}\{\sigma_{IS} I^n_S + \sigma_A A^n - (\delta_H + \mu)T^n_H\}. \quad (30)$$
$$A^n_{k+1}(t) = A_0 + \frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k} f_{y,k+1}\{\tau_{H2} I^n_S - (\sigma_A + \delta_H + \mu)A^n\},$$

From (29) and (30) obtained;
$dy_{,K+1} = K^{\gamma+1} - (k-\gamma)(k+\gamma)^{\gamma}, y = 0$
$(k-y+2)^{\gamma+1} + (k-\gamma)^{\gamma+1} - 2(k-y+1)^{\gamma+1}, 1 \le y \le k$
$1, y = k+1$
and
$$f_{y,k+1} = \frac{g^{\gamma}}{\gamma}[(k-y+1)^{\gamma}(k-y)^{\gamma}], 0 \le y \le k.$$

Table 2: Parameter values and sources

Parameter	Description	Value	Source
Λ	Recruitment rate human	0.007	Ngungu et al. (2023)
β_H	Contact rate of susceptible and infected humans with HIV/AIDs	0.3425	Odiba et al. (2024)
λ_h	Force of infection of HIV/AIDS	0.05	Shah et al. (2022)
μ	Natural death rate of humans	0.012	Ngungu et al. (2023)
θ_H	Progression rate from exposed human to HIV/AIDS to infected human with HIV/AIDS	0.4	Odiba et al. (2024)
δ_H	HIV/AIDS disease induced death rate	0.01	Ngungu et al. (2023)
τ_{H2}	Progression rate from symptomatic HIV humans to AIDS humans class	0.07	Ayele et al. (2021)
σ_{IS}	Treatment rate of symptomatic HIV infected humans	0.34	Assumed
σ_A	Treatment rate of HIV/AIDS humans	0.41	Assumed
$\boldsymbol{\tau_{H1}}$	Progression rate from infected HIV/AIDS humans to symptomatic humans with HIV	0.43	Assumed

Numerical Simulation

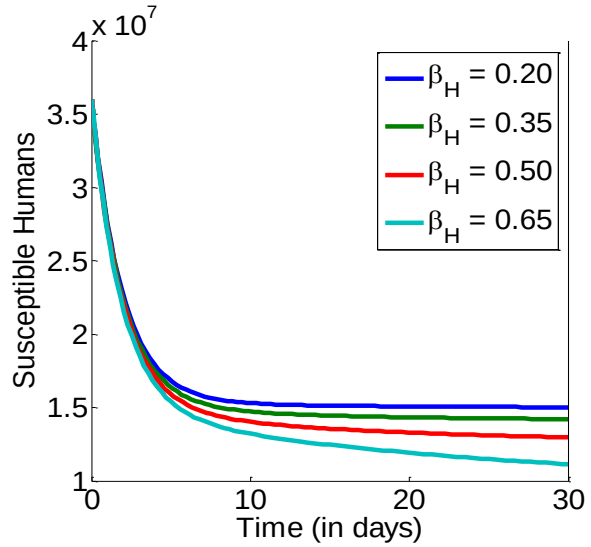

Figure 1(a): Simulation of susceptible Human to HIV/AIDS

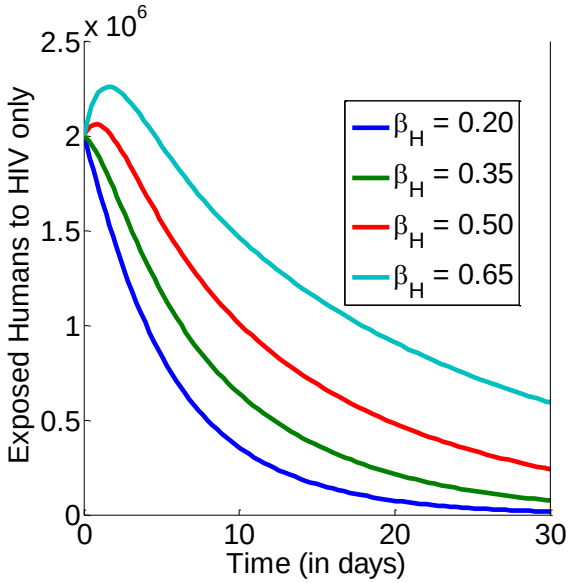

Figure 1(b): Simulation of Exposed Human to HIV/AIDS

Fig (1a) depicts the simulation of the effect of the Contact rate (β_H) on HIV/AIDs in Susceptible Human population. It is observed that, as the Contact rate (β_H) increases, the number of Susceptible Human population decreases. (1b) depicts the simulation of the effect of the Contact rate (β_H) on HIV/AIDs in the Exposed Human population. It is observed that, as the Contact rate (β_H) increases, the numberof Exposed individualsincreases.

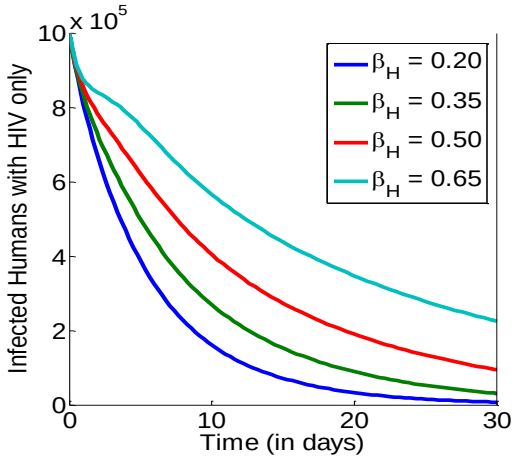

Figure 1(c): Simulation Infected Asymptomatic Human with HIV/AIDS

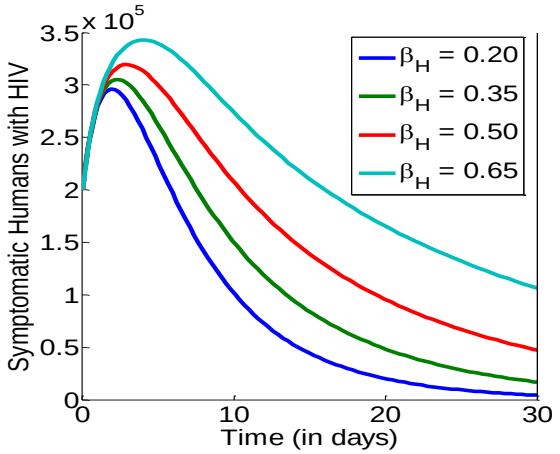

Figure 1(d): Simulation Infected Symptomatic Human with HIV/AIDS

Fig (1c) depicts the simulation of the effect of the Contact rate (β_H) on HIV/AIDs in Infected Asymptomatic Human population. It is observed that, as the Contact rate (β_H) increases, the number of Infected Asymptomatic Human population decreases. (1d) depicts the simulation of the effect of the Contact rate (β_H) on HIV/AIDs in the Infected Symptomatic Human population. It is observed that, as the Contact rate (β_H) increases, the number of Infected Symptomatic increases.

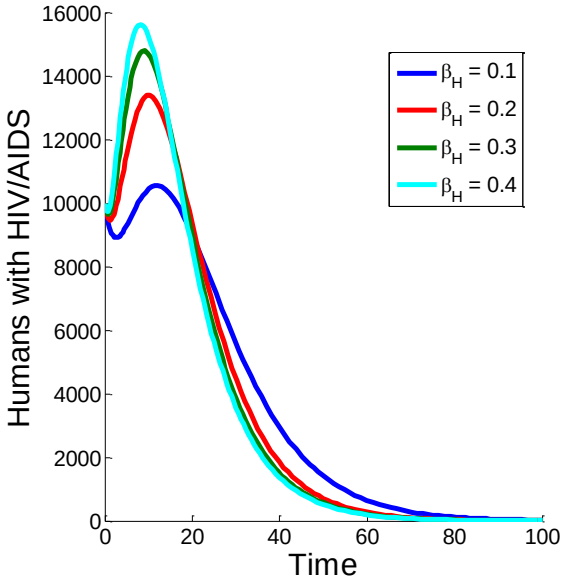

Figure 1(e): Simulation of Infected Human with AIDS only

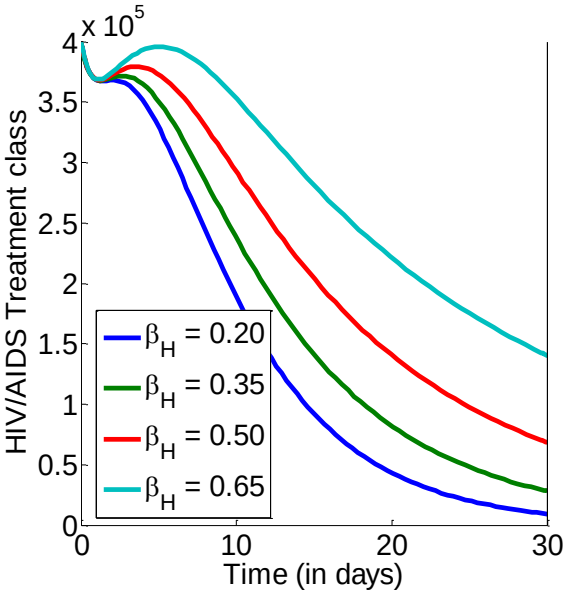

Figure 1(f): Simulation of Human on Treatment of AIDS

Fig (1e) depicts the simulation of the effect of the Contact rate (β_H) on HIV/AIDs in Infected Human with AIDs only population. It is observed that, as the Contact rate (β_H) increases, the number of Infected Human with AIDs only population increases. (1f) depicts the simulation of the effect of the Contact rate (β_H) on HIV/AIDs in the Treatment population. It is observed that, as the Contact rate (β_H) increases, the Treatment population increases.

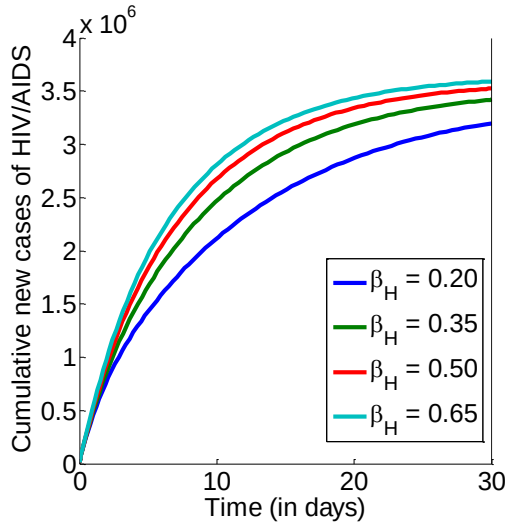

Figure 1(g): Cummulative New Cases of HIV/AIDS

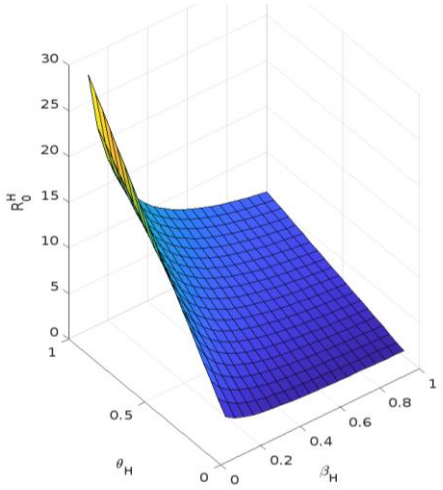

Figure 2(a): Surface plot showing the effect of $\beta_H$and $\sigma_H$on R_0^H

(1g) depicts the simulation of the effect of the Contact rate (β_H) on HIV/AIDs on the Cumulative new cases of HIV/AIDs . It is observed that, as the Contact rate (β_H) increases, the Cumulative new cases of HIV/AIDs increases. 2(a), it can be observed that the basic reproduction number R_0^H reaches a peak below one (1) as the values of $(\beta_H) and (\theta_H)$ increase. This indicates that increasing these parameters will ultimately alleviate the impact of HIV/AIDs on the population.

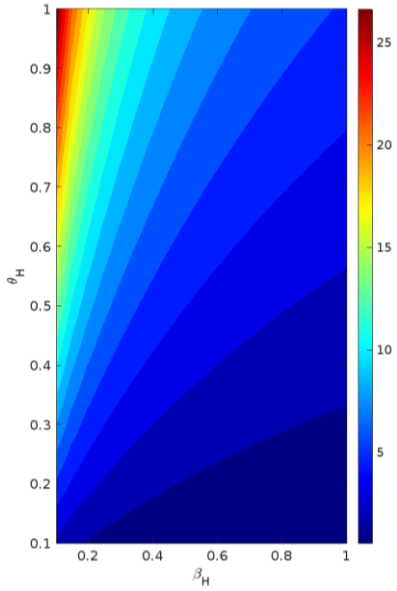

Figure 2(b): Contour plot showing the effect of $\beta_H$and $\theta_H$on R_0^H

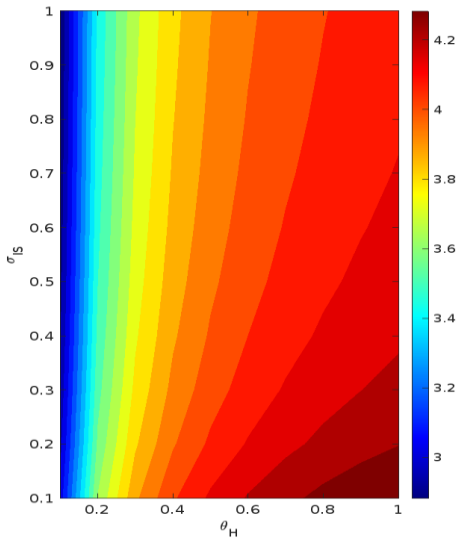

Figure 2(c): Contour plot showing the effect of $\beta_H$and $\sigma_H$on R_0^H

The graph depicted in Fig. (2b) illustrates the contour plot of $\beta_H and \theta_H$concerning R_0^H. Upon examination of the numerical streams within the graph, it is evident that the maximum value of R_0^Hattained by varying these parameters is 0.6, indicating a value below unity (1). This observation suggests that augmenting these parameters would not trigger a significant outbreak of HIV/AIDs in the population. The graph depicted in Fig. (2c) illustrates the contour plot of $\beta_H and \sigma_H$concerning R_0^H. Upon examination of the numerical streams within the graph, it is evident that the maximum value of R_0^Hattained by varying these parameters is 0.6, indicating a value below unity (1). This observation suggests that augmenting these parameters would not trigger a significant outbreak of HIV/AIDs in the population.

CONCLUSION

This paper presents a mathematical model for the transmission dynamics and control of HIV/AIDS utilizing the Caputo fractional derivative. Acknowledging the importance of fractional modeling, we begin with a comprehensive theoretical analysis of the fractional HIV/AIDS model, emphasizing the existence and uniqueness of solutions, as well as the stability of equilibrium points. To numerically solve the model, we employed the fractional Adams–Bashforth–Moulton method. The simulations highlighted the impact of disease incidence, taking into account various model parameters and different fractional orders of the Caputo operator. We also explored the effects of varying parameters, such as the contact rates between infected and susceptible individuals. The results indicate that enhancing

treatment strategies could significantly mitigate the spread of HIV/AIDS within the population. Future research could focus on addressing non-linear partial differential equations using approaches similar to those suggested by Zhang et al. (2022), which offer a general symbolic computational framework for deriving analytic solutions

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