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Fractional-Order Mathematical Modeling of HIV/AIDS Transmission Dynamics via a Generalized Adams–Bashforth–Moulton Numerical Scheme

[1,2]Enejoh Jalija, *[1]Jeremiah Amos, [1]David Omale, [1]William Atokolo, [1]Emmanuel Abah, [3]Johnson Juwon Orugun, and[1]Bolarinwa Bolaji

[1]Department of Mathematical Sciences, Prince Abubakar Audu University, Anyigba, Kogi State, Nigeria.
[2]Department of Mathematics and statistics, The Federal Polytechnic Idah, Kogi State, Nigeria.
[3]Department of Business Administration, Prince Abubakar Audu University, Anyigba, Kogi state, Nigeria.
*Corresponding Author's email: amosjeremiah1997@gmail.com

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ABSTRACT
HIV/AIDS remains one of the major global public health challenges due to its high transmission rate, long-term health complications, and socio-economic impact on affected populations. Despite several intervention strategies, the disease continues to spread, particularly in regions with inadequate access to treatment and preventive measures. Motivated by the need to better understand the transmission dynamics of HIV/AIDS and the impact of treatment strategies, this paper focuses on the dynamics of HIV/AIDS transmission using a fractional-order mathematical model to assess the effect of treatment and contact rates on HIV/AIDS. The model presents the existence and uniqueness of the solution in the fractional-order sense, demonstrating the well-posedness of the model. The model's stability is analysed to understand the dynamics of the disease, including the basic reproduction number. These findings show treatment rates of exposed, asymptomatic, and symptomatic individuals play a critical role in reducing the reproduction number below one, implying control of the disease, whereas contact rates increase disease transmission and maintain the disease's presence. Sensitivity and simulation analyses show that transmission-related parameters have a positive impact on disease transmission, while treatment-related parameters have a negative sensitivity, leading to a reduction in the disease burden. The dynamics of population states for different treatment and contact rates are discussed using the fractional Adams–Bashforth–Moulton numerical scheme. Further contour and surface plots reveal that increased treatment and lower contact rates result in a significant reduction in the HIV/AIDS incidence, while poor control measures and increased contact rates increase the infection. The results highlight that early treatment of exposed individuals and treatment of infected individuals (asymptomatic and symptomatic) are key to reducing the disease burden. In summary, the study underlines the need for effective control measures incorporating treatment expansion and reduction in transmission pathways for the successful control and possible eradication of HIV/AIDS in the population.

CITATION
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INTRODUCTION

The Human Immunodeficiency Virus (HIV), functions as a lentivirus that creates the development of Acquired Immunodeficiency Syndrome (AIDS), which causes deteriorating immunological functions in the human body. The weakened immune system of people diagnosed with AIDS creates opportunities for fatal opportunistic infections to invade their bodies. HIV directs its harmful attack toward CD4-T cells of the body, while these cells remain essential for the immune system defense. The symptoms HIV infection produces include chills, persistent diarrhea, fatigue, fever, substantial weight loss and night sweats and sore throat, oral ulcers and muscle pain. HIV spreads through the transmission of blood as well as semen and vaginal secretions and breast milk. People living with HIV receive extended life expectancy through antiretroviral therapy, although an HIV cure or vaccination remains unavailable.

Medical professionals confirmed the first AIDS cases within Nigeria during 1985, while the official report about AIDS came from Lagos in 1986. A 13-year-old female sex worker, who operated in a neighboring West African country, was among the first Nigerian AIDS patients diagnosed in 1985. The first AIDS case announcements spread panic throughout the general population, because the prevailing belief was that the disease mainly affected homosexual communities in the US. Nigerians rejected the information about AIDS by forming beliefs that declared the condition was an American strategy to reduce sexual activity leading to the development of "American Idea for Discouraging Sex" acronyms. The incorrect understanding held by the public regarding both the disease's source and its worldwide consequences received detailed analysis in the doctoral thesis Modeling HIV/AIDS Epidemic in Nigeria (Eze, 2009).

At first, fears of HIV/AIDS had a minimal effect on Nigeria but it turned out to be a serious health concern. In 2014, the statistics showed 220,000 new HIV infections and older adults were the largest number of HIV in Nigeria. Children (under 15 years) found positive for HIV, 58,000 during the year but many of the children acquired the disease through transmission from their HIV positive mothers. The number of orphaned children remained at 1.6 million in 2014 and 747000 people received ART (antiretroviral treatment) Odiba et al.(2024) Nigerian is the second country with the highest number of HIV cases (3.2 million) after South Africa but their HIV prevalence rate remains at around 3.4% among 200 million people.

The focus of researchers now is on fractional calculus since fractional derivatives support expert knowledge of the dynamics of physical systems as indicated by Atokolo et al. (2022). The fractional order system model describes total systems and not local systems like the integer order system models. Such modelling gives better description of systems with memory Atokolo et al (2022).

Fractional order models are preferred in terms of realistic and practicality compared to integer order models. Singular kernel fractional derivatives such as the Riemann-Liouville derivative and Caputo derivative are used in biological problems. The Atangana-Baleanu operator as well as Mittag-Leffler operator is part of the non-singular type of derivative. Atokolo et al (2022), applied fractional order sterile insect technology (SIT) in the control of Zika virus infection using LADM which produced solutions in the form of converging infinite series that yielded exact solutions. Atokolo et al (2024), illustrated a fractional order mathematical model for the epidemiological parameters of Lassa fever viral infection and investigated the effects of treatment and vaccination on the dynamics of Lassa fever disease transmission using a fractional order derivative with power law function. Yunus et al (2023), investigated the COVID-19 spread in Nigeria using LADM and Caputo fractional-order derivative, which confirmed that recovery rates in normal situations were higher due to the provision of treatment and vaccinations. Omede et al (2024), developed a dynamic model for soil-transmitted helminth infection using the Caputo derivative for fractional order systems solved using LADM series solutions. Their study confirmed that the series convergence results in the accuracy solution and the fractional order model is more flexible than the standard model. Amos et al (2024), formulated a fractional mathematical model of the hepatitis C transmission dynamics through contact rates and treatment; and also established the Adams-Bash-forth Moulton method, which confirmed that the disease decreases when the contact rates are lowered and treatment rates are increased and the fractional model is more flexible than the standard model. Ahmed et al (2021), created a prediction model for the co-epidemic of HIV and COVID-19 transmissions based on ABC-fractional order derivative.

Omame et al (2022), investigated a fractional order model for a co- infection of hepatitis B virus and COVID-19 using the Atangana-Baleanu derivative and found that prevention is key to controlling the disease. Acheneje et al (2024), developed a fractional order transmission dynamics model for the co- infection of COVID-19 and Monkey pox and used LADM for an approximate solution and real data fitting as well. The investigation showed that the development of additional treatment facilities would decrease the disease cases. Smith et al (2023), studied modeling on the Co- infection Dynamics of Hepatitis C and COVID-19 by reviewing recent mathematical modeling studies of hepatitis C and COVID-19 co-infection. The review outlines the standard modeling protocol and key findings of interest with the need for more research to be done in new areas. Atokolo et al. (2023), created a mathematical model for the transmission of vector-borne diseases with vertical transmission and prevention.

Their advantages are mainly attributed to the simultaneous flexibility and non-locality aspects of fractional order models. Data can be better modelled by using fractional derivatives because of their flexibility, compared to classical derivatives. These derivatives have benefits through their capability to assess non-locality that classical derivatives cannot. Models of fractional order possess a memory property which classical models do not show by virtue of their fractional operator. Contemporary scientists use fractional differential equations as a technique to solve problems. Ullah et al (2020), presented research which Das et al (2024), described about the solution of fuzzy Volterra integral equations with degenerate kernels, by employing a combination method. Researchers have combined the Laplace transform method with Adomian Decomposition for advancing the theory of fuzzy analytical dynamic equations due to their great impact in this area.

The study is of keen interest because of the discovery of insights for fuzzy analytical dynamic equation theory. The existence and stability analysis of a three-point boundary value problem were studied by analysts, as outlined by Ali et al (2017). The authors studied several types of Ulam stability through a non-linear fractional approach.

There are various mathematic models used for control and analysis of infectious diseases, in particular, HIV. Researchers have used the Laplace-Adomian Transformation Method to set up approximate solutions of Lassa fever models of fractional orders and have constructed fractional-order models for studying dengue and COVID-19 disease spread dynamics. Fractional-order models are used to study the impacts of antiretroviral therapy (ART) using novel type-2 fuzzy logic controllers in HIV models.

Fractional-order models are highly valuable as they have superior flexibility, non-locality and memory effects. Fractional derivatives result in better models in data fitting as they are more flexible and accurate than classical derivatives. Models show proficiency in exhibiting memory effects which classical models cannot. In recent studies, researchers Ullah et al (2020), and Ali et al (2017) have shown that there is a growing interest in applications of fractional calculus for modeling of complex systems due to their research on fuzzy Volterra integral equations and stability in three-point boundary value problems.

This paper aims to provide sufficient conditions for the existence and uniqueness of solutions of a fractional-order HIV/AIDS model, carry out a stability analysis of the endemic equilibrium using the Lyapunov function method, numerically solve the model using the fractional Adams–Bashforth–Moulton method, and perform simulations to illustrate the dynamics of the model.

A thorough review of existing literature reveals that the literature has not incorporated the fractional calculus with Adams-Bashforth-Moulton method for the study of the HIV/AIDS disease and its control.

Definition 1

Let $f \in \Lambda^{\infty}(R)$,the left and right Caputo fractional derivatives of the function$f$ are then defined as

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

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

Similarly

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

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

Definition 2

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

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

Which can be denoted as;

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

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

Proposition 1

Let $f \in \Lambda^{\infty}(R) \cap C(R)$ and $\varphi \in R, n-1 < \varphi < n,$

Therefore, the conditions given below is satisfied:

1. $ {}_{t_0}^{c}D_{t}^{\varphi} I^{\varphi} f(t) = f(t), \quad (5a) $
2. $ I_{t_0}^{\varphi} D_{t}^{\varphi} f(t) = f(t) - \sum_{k=0}^{n-k} \frac{t^{k}}{k!} f^{k}(t_o). \quad (5b) $

MATERIALS AND METHODS

Mathematical Model Formulation

When formulating the HIV/AIDS integer-order model, the population is subdivided into six compartments, namely Susceptible human population (S_H): (those without the infection); Exposed individuals (E_H): (those human population infected but not yet infectious); Asymptomatic Infected population (I_A): (those infected with HIV/AIDS but without any clinical symptoms); Symptomatic Infected population (I_S) : (those infected with HIV/AIDS but not without any clinical symptoms). Those infected with HIV/AIDS with clinical symptoms); HIV/AIDS treatment population (T_H) (those receiving treatment of HIV/AIDS) and AIDS Infected human population (A).

Susceptible humans to HIV/AIDS (S_H) are recruited with the rate (π) and reduces through natural death rate (μ) and also through the proportion of individuals becoming infected after contacts with Asymptomatic infected individuals, symptomatic infected individuals, individuals on HIV/AIDS treatment and AIDS infected individuals with a rate of $\beta_1, \beta_2, \beta_3 and \beta_4$ respectively. We thus defined the dynamics of susceptible individuals as:

$$\frac{dS_H}{dt} = \pi - \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H - \mu S_H,$$

The proportion of newly infected individuals at rate $(\beta_1, \beta_2, \beta_3 and \beta_4)$ determines the rate of increase in the number of HIV/AIDS-exposed individuals (E_H) in the model. This group decreases as exposed individual's progress to the asymptomatic infected stage at rate (θ) and through natural deaths at rate (μ).

The relationships governing this population are expressed as follows:

$$\frac{dE_H}{dt} = \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H - (\theta + \mu) E_H,$$

The number of asymptomatic HIV/AIDS infected humans; (I_A) Exposed persons progress to asymptomatic infected population at rate (θ) This population decreases due to disease induced death rate (δ_1) The population die naturally at the rate of (μ), The rate at which the asymptomatic infected human population is treated with HIV/AIDS treatment (σ_1),The rate at which the asymptomatic infected human population is converted to symptomatic infected human population (α).

The dynamics of this class are:

$$\frac{dI_A}{dt} = \theta E_H - (\alpha + \sigma_1 + \delta_1 + \mu) I_A,$$

The population of symptomatic infected humans with HIV/AIDS;(I_S) Increases with the transition from asymptomatic infected humans to symptomatic infected human population at rate, (α), Decreases with the natural death rate, (μ) disease induced death rate, $(\delta_2$) rate at which symptomatic infected humans receives HIV/AIDS treatment, $(\sigma_1$) and rate at which symptomatic infected humans progresses to AIDS infected human population, (ϕ).

This class has the following dynamics:

$$\frac{dI_S}{dt} = \alpha I_A - (\phi + \sigma_2 + \delta_2 + \mu) I_S,$$

The HIV/AIDS treatment population of persons (T) is increased by the rate at which the asymptomatic persons are treated of HIV/AIDS (σ_1), the rate at which the symptomatic persons are treated of HIV/AIDS (σ_2) and the rate at which the AIDS infected persons are treated of HIV/AIDS (σ_3). The population decline due to disease related death (δ_3) and natural death (μ).

This population dynamics are given by:

$$\frac{dT}{dt} = \sigma_1 I_A + \sigma_2 I_S + \sigma_3 A - (\delta_3 + \mu) T,$$

The AIDS infected human population (A) increases at the rate of the progression of the symptomatic human to AIDS infected human population (ϕ). They decrease due to the AIDS infected human population taking the HIV/AIDS treatment at rate (σ_3) and disease induced death rate of AIDS infected human population (δ_4) and also because of the natural death rate (μ).

$$\frac{dA}{dt} = \phi I_S - (\sigma_3 + \delta_3 + \mu) A.$$

Model Assumptions

1. We assume natural death in the population (Abah et al., 2025).
2. We assume that human die due to HIV/AIDS (Odiba et al., 2025).
3. We assume that there is no vaccine for HIV/AIDS (Odiba et al., 2025).

HIV/AIDS Model Flow Chart

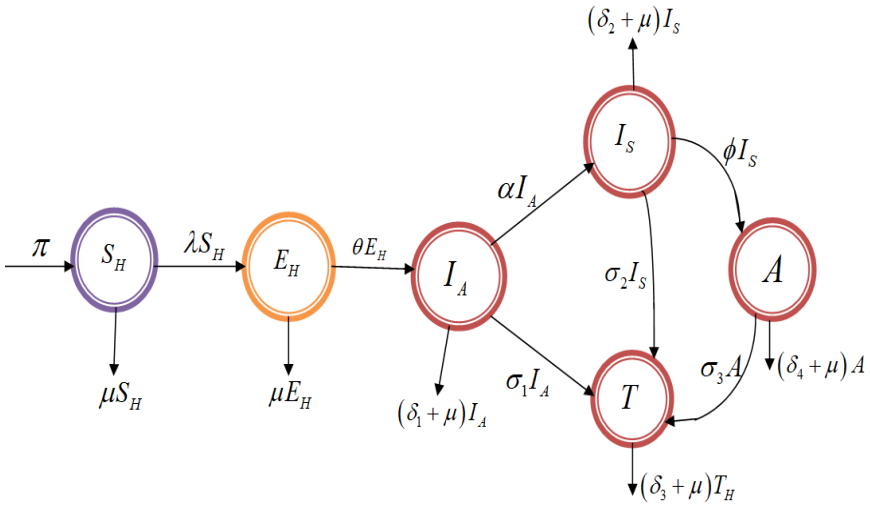

Figure 1: HIV/AIDS model flow Diagram

Figure 1 presents the flow diagram of the HIV/AIDS transmission dynamics model. It illustrates the progression of individuals through different epidemiological compartments, starting from the susceptible population, which becomes exposed through effective contact with infected individuals. The exposed

individuals progress to the infectious, and then to either the symptomatic infected class or the asymptomatic class. The model also incorporates treatment and recovery/death-related transitions, capturing both disease progression and intervention strategies. The diagram clearly shows the roles of infection, progression, treatment, and natural removal rates in the transmission dynamics of HIV/AIDS.

HIV/AIDS Model Equation

$$\frac{dS_H}{dt} = \pi - \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H - \mu S_H,$$
$$\frac{dE_H}{dt} = \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H - (\theta + \mu) E_H,$$
$$\frac{dI_A}{dt} = \theta E_H - (\alpha + \sigma_1 + \delta_1 + \mu) I_A,$$
$$\frac{dI_S}{dt} = \alpha I_A - (\phi + \sigma_2 + \delta_2 + \mu) I_S,$$
$$\frac{dT}{dt} = \sigma_1 I_A + \sigma_2 I_S + \sigma_3 A - (\delta_3 + \mu) T, \quad (6)$$
$$\frac{dA}{dt} = \phi I_S - (\sigma_3 + \delta_3 + \mu) A.$$

Where $\lambda = \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N}$.

Disease Free Equilibrium Point of HIV/AIDS

Disease free equilibrium point is a point where there is no disease in the population

At DFE $S \neq 0, E_H = 0, I_A = 0, I_S = 0, T = 0, A = 0.$

$$(S^0, E_H^0, I_A^0, I_S^0, T^0, A^0) = \left(\frac{\pi}{\mu}, 0,0,0,0,0\right). \quad (7)$$

Basic Reproduction Number of HIV/AIDS

Basic Reproduction number R_0^H is the number of secondary cases caused by individual infected people. Next generation method $R_0^H = \rho F V^{-1}$ is used to compute R_0^H using F, a non-negative matrix (other transition terms V) and finding the dominant Eigen valueρ.

$$F = \begin{pmatrix} 0 & \beta_1 & \beta_2 & \beta_3 & \beta_4 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \text{and } V = \begin{pmatrix} A_1 & 0 & 0 & 0 & 0 \\ -\theta & A_2 & 0 & 0 & 0 \\ 0 & -\alpha & A_3 & 0 & 0 \\ 0 & -\sigma_1 & -\sigma_2 & A_4 & -\sigma_3 \\ 0 & 0 & -\phi & 0 & A_5 \end{pmatrix}$$

$$R_0^H = \frac{\theta(\alpha\phi A_4\beta_4 + \alpha\phi\beta_3\sigma_3 + \alpha A_4 A_5\beta_2 + \alpha A_5\beta_3\sigma_2 + A_3 A_5 A_4\beta_1 + A_3 A_5\beta_3\sigma_1)}{A_1 A_2 A_3 A_4 A_5}. \quad (8)$$

This is the largest Eigen value.

Endemic Equilibrium of HIV/AIDS

Endemic Equilibrium signifies the permanent existence of HIV/AIDS among human population.

At endemic equilibrium point $(S_H \neq 0, E_H \neq 0, I_A \neq 0, I_S \neq 0, T_H \neq 0, A \neq 0)$.

We obtain the following endemic equilibrium points:

$$S_H^* = \frac{\pi}{\lambda^* + A_1},$$
$$E_H^* = \frac{\lambda^* \pi}{(\lambda^* + A_1)A_2},$$
$$I_A^* = \frac{\theta\lambda^* \pi}{(\lambda^* + A_1)A_2 A_3},$$
$$I_S^* = \frac{\theta\lambda^* \pi\alpha}{(\lambda^* + A_1)A_2 A_3 A_4}, \quad (9)$$
$$T^* = \frac{\theta(\alpha\phi\sigma_3 + \alpha A_6\sigma_2 + A_4 A_6\sigma_1)\pi\lambda^*}{(\lambda^* + A_1)A_6 A_2 A_3 A_4 A_5},$$
$$A^* = \frac{\theta\lambda^* \pi\alpha\phi}{(\lambda^* + A_1)A_6 A_2 A_3 A_4}.$$

Substituting into the force of infection $\lambda = \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N}$.

We have;

$$Q_1\lambda + Q_2 = 0$$
$$Q_1 = (\alpha\phi\theta A_5 + \alpha\phi\theta\sigma_3 + \alpha\theta A_5 A_6 + \alpha\theta A_6\sigma_2 + \theta A_4 A_5 A_6 + \theta A_4 A_6\sigma_1 + A_3 A_4 A_5 A_6)$$
$$Q_2 = (1 - R_0^H) \quad (10)$$

This shows that the endemic equilibrium point of the model is stable and has a unique positive solution if $R_0^H > 1$.

Sensitivity analysis of HIV/AIDS

Sensitivity analysis is an important component of epidemiological modelling, used to pinpoint and measure the impact of different parameters on disease spread. Through the process of parameter variation it is possible to assess which parameters exert the greatest influence on the basic reproduction number, prevalence of infection or other epidemiological measures of interest. This technique can identify the parameters that positively affect disease transmission, and those that can reduce transmission when reduced, thereby informing interventions and decision-making practices. It can be determine using: $S_x^{R_0} = \left(\frac{\partial R_0}{\partial x}\right)\left(\frac{x}{R_0}\right)$.

$$S_\theta^{R_0^T} = \frac{\mu}{\theta + \mu} = 0.10534,$$
$$S_\phi^{R_0^T} = 0.085,$$

$$S_{\beta_1}^{R_0^T} = \frac{(\phi+\sigma_2+\delta_2+\mu)(\delta_3+\mu)(\sigma_3+\delta_3+\mu)\beta_1}{\alpha\phi(\delta_3+\mu)\beta_4+\alpha\phi\beta_3\sigma_3+\alpha(\delta_3+\mu)(\sigma_3+\delta_3+\mu)\beta_2+\alpha(\sigma_3+\delta_3+\mu)\beta_3\sigma_2 \\ +(\phi+\sigma_2+\delta_2+\mu)(\delta_3+\mu)(\sigma_3+\delta_3+\mu)\beta_1+(\phi+\sigma_2+\delta_2+\mu)(\sigma_3+\delta_3+\mu)\beta_3\sigma_1} = 0.371,$$

$$S_{\beta_2}^{R_0^T} = \frac{\alpha(\delta_3+\mu)(\sigma_3+\delta_3+\mu)\beta_2}{\alpha\phi(\delta_3+\mu)\beta_4+\alpha\phi\beta_3\sigma_3+\alpha(\delta_3+\mu)(\sigma_3+\delta_3+\mu)\beta_2+\alpha(\sigma_3+\delta_3+\mu)\beta_3\sigma_2 \\ +(\phi+\sigma_2+\delta_2+\mu)(\delta_3+\mu)(\sigma_3+\delta_3+\mu)\beta_1+(\phi+\sigma_2+\delta_2+\mu)(\sigma_3+\delta_3+\mu)\beta_3\sigma_1} = 0.351,$$

$$S_{\beta_3}^{R_0^T} = \frac{(\alpha\phi\sigma_3+\alpha(\sigma_3+\delta_3+\mu)\sigma_2+(\phi+\sigma_2+\delta_2+\mu)(\sigma_3+\delta_3+\mu)\sigma_1)\beta_3}{\alpha\phi(\delta_3+\mu)\beta_4+\alpha\phi\beta_3\sigma_3+\alpha(\delta_3+\mu)(\sigma_3+\delta_3+\mu)\beta_2+\alpha(\sigma_3+\delta_3+\mu)\beta_3\sigma_2 \\ +(\phi+\sigma_2+\delta_2+\mu)(\delta_3+\mu)(\sigma_3+\delta_3+\mu)\beta_1+(\phi+\sigma_2+\delta_2+\mu)(\sigma_3+\delta_3+\mu)\beta_3\sigma_1} = 0.8983,$$

$$S_{\beta_4}^{R_0^T} = \frac{\alpha\phi(\delta_3+\mu)\beta_4}{\alpha\phi(\delta_3+\mu)\beta_4+\alpha\phi\beta_3\sigma_3+\alpha(\delta_3+\mu)(\sigma_3+\delta_3+\mu)\beta_2+\alpha(\sigma_3+\delta_3+\mu)\beta_3\sigma_2 \\ +(\phi+\sigma_2+\delta_2+\mu)(\delta_3+\mu)(\sigma_3+\delta_3+\mu)\beta_1+(\phi+\sigma_2+\delta_2+\mu)(\sigma_3+\delta_3+\mu)\beta_3\sigma_1} = 0.2954,$$

$$S_{\sigma_1}^{R_0^T} = -0.101, S_{\sigma_2}^{R_0^T} = -0.03822, S_{\sigma_3}^{R_0^T} = -0.034, \quad (11)$$

$$S_{\delta_1}^{R_0^T} = -\frac{\delta_1}{\alpha+\sigma_1+\delta_1+\mu} = -0.0125,$$

$$S_{\delta_2}^{R_0^T} = -0.0193, S_{\delta_3}^{R_0^T} = -0.5535, S_{\alpha}^{R_0^T} = 0.123, S_{\mu}^{R_0^T} = -1.789.$$

HIV/AIDS Sensitivity Bar chart

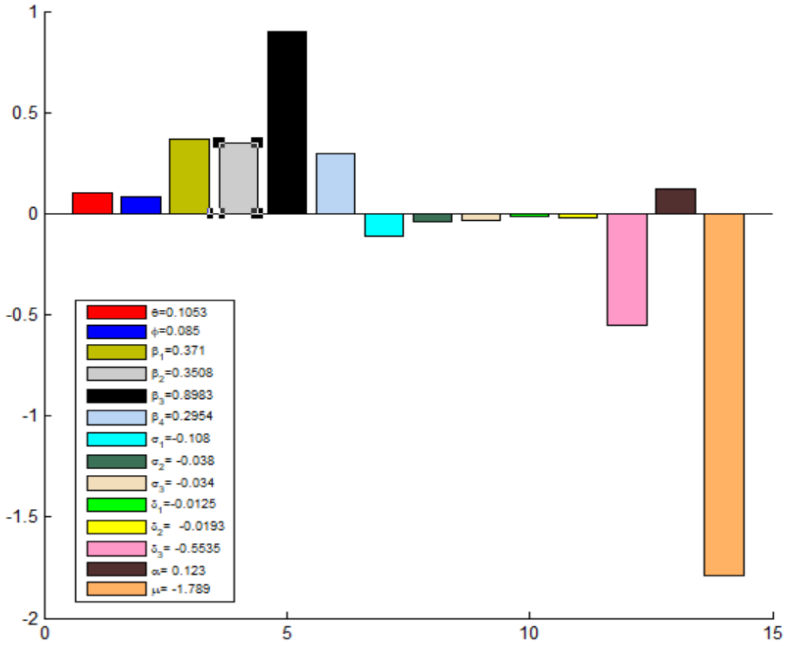

Figure 2: HIV/AIDS sensitivity Bar chart

Interpretation of Sensitivity Bar chart

Figure 2 presents the sensitivity analysis of the model parameters using a bar chart representation. The results show the relative influence of each parameter on the basic reproduction number and disease dynamics. It is observed that transmission-related parameters such as the infection rates $\beta_1, \beta_2, \beta_3, \beta_4$exhibit positive sensitivity indices, indicating that an increase in these parameters enhances the spread of HIV/AIDS. In contrast, treatment-related parameters such as $\sigma_1, \sigma_2, \sigma_3, \sigma_4$and recovery-related rates display negative sensitivity indices, implying that an increase in treatment efforts reduces disease transmission. Among all parameters, the natural death rate and treatment-related parameters show the strongest negative sensitivity, highlighting their importance in controlling the disease. Therefore, Figure 2 clearly demonstrates that reducing transmission rates while increasing treatment rates is crucial for effective disease control.

Global Stability Analysis at Endemic Equilibrium State

To study the global stability of the equilibrium point, we use the direct Lyapunov method. We find the endemic equilibrium point is globally stable if $R_0 < 1$. This suggests that, from a health perspective, the disease will be

transmitted in the population regardless of the size of the population. Using the fractional model (6).

$WhereN = \frac{\pi}{\mu} ast \to \infty$

Our fractional model now becomes

$$
\begin{aligned}
&{}^{c}D_t^{\varphi} S_H = \pi - \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H - \mu S_H,\\
&{}^{c}D_t^{\varphi} E_H = \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H - (\theta + \mu) E_H,\\
&{}^{c}D_t^{\varphi} I_A = \theta E_H - (\alpha + \sigma_1 + \delta_1 + \mu) I_A,\\
&{}^{c}D_t^{\varphi} I_S = \alpha I_A - (\phi + \sigma_2 + \delta_2 + \mu) I_S,\\
&{}^{c}D_t^{\varphi} T = \sigma_1 I_A + \sigma_2 I_S + \sigma_3 A - (\delta_3 + \mu) T, \qquad (12)\\
&{}^{c}D_t^{\varphi} A = \phi I_S - (\sigma_3 + \delta_3 + \mu) A.
\end{aligned}
$$

Let $P_1 = \mu$, $P_2 = (\theta + \mu)$, $P_3 = \alpha + \sigma_1 + \delta_1 + \mu)$, $P_4 = (\phi + \sigma_2 + \delta_2 + \mu)$, $P_5 = \delta_3 + \mu)$, $P_6 = (\sigma_3 + \delta_3 + \mu)$.

We now have:

$$
\begin{aligned}
&{}^{c}D_t^{\varphi} S_H = \pi - \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H - P_1 S_H,\\
&{}^{c}D_t^{\varphi} E_H = \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H - P_2 E_H,\\
&{}^{c}D_t^{\varphi} I_A = \theta E_H - P_3 I_A,\\
&{}^{c}D_t^{\varphi} I_S = \alpha I_A - P_4 I_S,\\
&{}^{c}D_t^{\varphi} T = \sigma_1 I_A + \sigma_2 I_S + \sigma_3 A - P_5 T, \qquad (13)\\
&{}^{c}D_t^{\varphi} A = \phi I_S - P_6 A.
\end{aligned}
$$

At equilibrium point Eq. (13) has the following results

$$
\begin{aligned}
&{}^{c}D_t^{\varphi} S_H + \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H + P_1 S_H = \pi,\\
&{}^{c}D_t^{\varphi} E_H + P_2 E_H = \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H,\\
&{}^{c}D_t^{\varphi} I_A + P_3 I_A = \theta E_H, \qquad {}^{c}D_t^{\varphi} I_S + P_4 I_S = \alpha I_A,\\
&{}^{c}D_t^{\varphi} T + P_5 T = \sigma_1 I_A + \sigma_2 I_S + \sigma_3 A, \ {}^{c}D_t^{\varphi} A + P_6 A = \phi I_S.
\end{aligned}
$$

Theorem 1

Model (6) is globally asymptotically stable if $R_0^H < 1$

Whenever

$$\left(6 - \frac{S_H^*}{S_H} + \frac{\lambda_1}{\lambda_1^{**}}\left(1 - \frac{S_H E_H^*}{S_H^* E_H}\right) - \frac{I_A^* A^* I_S^* E_H}{I_A A I_S E_H^*} - \frac{T_H}{T_H^*} - \frac{T_H^* A I_A I_S}{T_H A^* I_A^* I_S^*}\right) \le 0.$$

$$Let L(t) = L_h(t)$$

Be a non-linear Lyaponov function as presented in (27) below:

$$
\begin{aligned}
&L(t) = L_1\left(S_H - S_H^* - S_H^* \ln\frac{S_H}{S_H^*}\right) + L_2\left(E_H - E_H^* - E_H^* \ln\frac{E_H}{E_H^*}\right) + L_3\left(I_A - I_A^* - I_A^* \ln\frac{I_A}{I_A^*}\right)\\
&+L_4\left(I_S - I_S^* - I_S^* \ln\frac{I_S}{I_S^*}\right) + L_5\left(T - T^* - T^* \ln\frac{T}{T^*}\right) + L_6\left(A - A^* - A^* \ln\frac{A}{A^*}\right). \qquad (14)
\end{aligned}
$$

Taking the Caputo Fractional order derivative of Eq. (14), we have

$$
\begin{aligned}
&{}^{c}D_t^{\varphi} L(t) = {}^{c}D_t^{\varphi} L_h(t) \le L_1\left(1 - \frac{S_H^*}{S_H}\right) {}^{c}D_t^{\varphi} S_H(t) + L_2\left(1 - \frac{E_H^*}{E_H}\right) {}^{c}D_t^{\varphi} E_H(t) + L_3\left(1 - \frac{I_A^*}{I_A}\right) {}^{c}D_t^{\varphi} I_A(t)\\
&+L_4\left(1 - \frac{I_S^*}{I_S}\right) {}^{c}D_t^{\varphi} I_S(t) + L_5\left(1 - \frac{T^*}{T}\right) {}^{c}D_t^{\varphi} T(t) + L_6\left(1 - \frac{A^*}{A}\right) {}^{c}D_t^{\varphi} A(t),\\
&= \lambda_1^* S_H^* \begin{pmatrix} \left(1 - \frac{S_H^*}{S_H}\right) {}^{c}D_t^{\varphi} S_H(t) + \left(1 - \frac{E_H^*}{E_H}\right) {}^{c}D_t^{\varphi} E_H(t) + \left(1 - \frac{I_A^*}{I_A}\right) {}^{c}D_t^{\varphi} I_A(t) \\ + \left(1 - \frac{I_S^*}{I_S}\right) {}^{c}D_t^{\varphi} I_S(t) + \left(1 - \frac{T^*}{T}\right) {}^{c}D_t^{\varphi} T(t) + \left(1 - \frac{A^*}{A}\right) {}^{c}D_t^{\varphi} A(t), \end{pmatrix}.\\
&\left(1 - \frac{S_H^*}{S_H}\right) {}^{c}D_t^{\varphi} S_H = \left(1 - \frac{S_H^*}{S_H}\right)(\lambda_1^* S_H^* + \mu S_H^* - \lambda_1 S_H - \mu S_H),\\
&= \lambda_{h1}^* S_H^*\left(1 - \frac{S_H \lambda_1}{\lambda_1^* S_H^*} - \frac{S_H^*}{S_H} + \frac{\lambda_1}{\lambda_1^*}\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^{\varphi} E_H = \left(1 - \frac{E_H^*}{E_H}\right)\left(\lambda_1^* S_H - \lambda_1^* S_H^* \frac{E_H}{E_H^*}\right),\\
&= \lambda_1^* S_H^*\left(1 - \frac{S_H \lambda_1^*}{\lambda_1^* S_H^*} \frac{E_H^*}{E_H} - \frac{E_H^*}{E_H} + \frac{S_H^* \lambda_1}{\lambda_1^* S_H^*}\right),\\
&\frac{P_1}{\theta}\left(1 - \frac{I_A^*}{I_A}\right) {}^{c}D_t^{\varphi} I_A = \frac{P_1}{\theta}\left(1 - \frac{I_A^*}{I_A}\right)\left(\theta E_H - P_2 \frac{I_A}{I_A^*} I_A^*\right), \qquad (15)\\
&= \lambda_1^* 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{P_3}{\alpha}\left(1 - \frac{I_S^*}{I_S}\right) {}^{c}D_t^{\alpha} I_S = \frac{P_3}{\alpha}\left(1 - \frac{I_S^*}{I_S}\right)\left(\alpha I_S - P_4 \frac{I_S}{I_S^*} I_S^*\right),
\end{aligned}
$$

$$= \lambda_1^* S_H^* \left(1 - \frac{E_H}{E_H^*} - \frac{I_H}{I_H^*} - \frac{E_H I_S^* A^* I_A^*}{E_H^* I_S A^* I_A}\right),$$

$$\frac{P_3 P_4}{\alpha\theta}\left(1 - \frac{T_H^*}{T_H}\right) {}^cD_t^{\varphi} T_H = \frac{P_3 P_4}{\alpha\theta}\left(1 - \frac{T_H^*}{T_H}\right)\left(\alpha\theta I_S I_A - P_5 \frac{T_H}{T_H^*} T_H^*\right),$$

$$= \lambda_1^* S_H^* \left(1 - \frac{T_H^*}{T_H} - \frac{I_S A I_A T_H^*}{T_H^* A^* I_S^* I_A^*} + \frac{I_S A I_A}{I_S^* A^* I_A^*}\right),$$

Hence, Eq. (13) now becomes:

$${}^cD_t^{\varphi} L(t) \leq \lambda_1^* S_H^*$$

$$\left(6 - \frac{S_H^*}{S_H} + \frac{\lambda_1}{\lambda_1^*}\left(1 - \frac{S_H E_H^*}{S_H^* E_H}\right) - \left(\frac{I_S^* I_A^* E_H}{I_S I_A E_H^*}\right) - \left(\frac{T_H^* E_H}{T_H E_H^*}\right)\right) \leq 0.$$

$$Which implies that, \; {}^cD_t^{\varphi} L(t) \leq \lambda_1^* S_H^* \theta (R_0^H - 1)\lambda S_H^*$$

$$\left(6 - \frac{S_H^*}{S_H} + \frac{\lambda_1}{\lambda_1^{**}}\left(1 - \frac{S_H E_H^*}{S_H^* E_H}\right) - \frac{I_S^* I_A^* E_H}{I_S I_A E_H^*} - \frac{T_H}{T_H^*} - \frac{T_H^* I_S I_A}{T_H I_S^* I_A^*}\right) \leq 0.$$

$$-\theta(R_0^H - 1)\lambda S_H^* \left[P_2 S_H^* \left(\frac{S_H^*}{S_H} - 1 - ln\frac{S_H^*}{S_H}\right)\right],$$

Therefore ${}^cD_t^{\varphi} L(t) \leq 0 for R_0^H < 1$.

This implies that, ${}^cD_t^{\varphi} L(t) = 0$. If $E_* = \left(S_H^*, E_H^*, I_A^*, I_S^*, T^*, A^*\right)$,denotes the endemic equilibrium point. Then, according to LaSalle's invariance theory, the endemic equilibrium point is globally asymptotically stable in Ω whenever $R_0^H < 1$.

Fractional order mathematical model

$$\begin{aligned}
&{}^cD_t^{\varphi} S_H = \pi - \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H - \mu S_H,\\
&{}^cD_t^{\varphi} E_H = \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H - (\theta + \mu) E_H,\\
&{}^cD_t^{\varphi} I_A = \theta E_H - (\alpha + \sigma_1 + \delta_1 + \mu) I_A,\\
&{}^cD_t^{\varphi} I_S = \alpha I_A - (\phi + \sigma_2 + \delta_2 + \mu) I_S,\\
&{}^cD_t^{\varphi} T = \sigma_1 I_A + \sigma_2 I_S + \sigma_3 A - (\delta_3 + \mu) T,\\
&{}^cD_t^{\varphi} A = \phi I_S - (\sigma_3 + \delta_3 + \mu) A.
\end{aligned} \tag{16}$$

Subject to the initial conditions

$$S_H(0) = S_{H0}, E_H(0) = E_{H0}, I_A(0) = I_{A0}, I_S(0) = I_{S0}, T(0) = T_0, A(0) = A_0.$$

Model Analysis

Positivity of model solution

We considered the non-negativity of the initial values

$$N(t) \leq \frac{\pi}{\mu} \quad \text{as} \quad t \to \infty$$

Secondly, $if \; limsup \, N_0 \, (t) \leq \frac{\pi}{\mu}$, then our model feasible domain is given by:

$$\Omega = \left\{\left(S_H, E_H, I_A, I_S, T, A()_{+_{H_H A_S}}^6 T + A \leq \frac{\pi}{\mu}\right)\{\}\right\}, \tag{17}$$

So that,

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

Hence Ω is positively invariant.

If $(S_{H0}, E_{H0}, I_{A0}, I_{S0}, T_0, A_0$)are non-negative, then the solution of model (6) will be non-negative for t> 0. From Eq. (6), selecting the first equation, we obtained:

$${}^cD_t^{\varphi} S_H = \pi - \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H - \mu S_H,$$

$${}^cD_t^{\varphi} S_H + \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A + \mu)}{N} S_H = \pi,$$

But$\pi \geq 0$then

$${}^cD_t^{\varphi} S_H + \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A + \mu)}{N} S_H \geq 0.$$

Applying the Laplace transform we obtained:

$$L\left[{}^{c}D_t^{\varphi}S_H\right]+L\left[\frac{(\beta_1 I_A+\beta_2 I_S+\beta_3 T+\beta_4 A+\mu)}{N}S_H\right]\geq 0.$$

$$S_H^{\varphi}S_H(s)-S_H^{\varphi-1}S_H(0)+\frac{(\beta_1 I_A+\beta_2 I_S+\beta_3 T+\beta_4 A+\mu)}{N}S_H(s)\geq 0,$$

$$S_H(s)\geq \frac{S_H^{\varphi-1}}{S_H^{\varphi}+\frac{(\beta_1 I_A+\beta_2 I_S+\beta_3 T+\beta_4 A+\mu)}{N}}S_H(0). \quad (18)$$

By taking the inverse Laplace transform, we obtained:

$$S_H(t)\geq E_{t\varphi,1}\left(-\left(\frac{(\beta_1 I_A+\beta_2 I_S+\beta_3 T+\beta_4 A+\mu)}{N}\right)t^{\varphi}\right)S_{H0}. \quad (19)$$

Now, since the term on the right-hand side of Eq. (19) is positive, we conclude that $S_H\geq 0$ for$t\geq 0$. In the same way, we also have that $E_H\geq 0, I_A\geq 0, I_S\geq 0, T_H\geq 0, A\geq 0$, that are positives. Therefore, the solution will remain in R_+^6 for all $t\geq 0$ with positive initial conditions.

Boundedness of Fractional Model Solution

The total human population from our model is given by;

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

So, from our fractional model (16), we now obtain

$${}^{c}D_t^{\varphi}N(t)={}^{c}D_t^{\varphi}S_H(t)+{}^{c}D_t^{\varphi}E_H(t)+{}^{c}D_t^{\varphi}I_A(t)+{}^{c}D_t^{\varphi}I_S(t)+{}^{c}D_t^{\varphi}T_H(t)+D_t^{\varphi}A(t)$$

$${}^{c}D_t^{\varphi}N(t)=\pi-\mu N(t), \quad (20)$$

Taking the Laplace transformation of (20), we obtained:

$$L\left[{}^{c}D_t^{\varphi}N(t)\right]=L[\pi-\mu N(t)],$$

$$S_H^{\varphi}N(s)-S_H^{\varphi-1}N(0)+\mu N(s)\leq\frac{\pi}{\mu},$$

$$N(s)\leq\frac{S_H^{\varphi-1}}{(S^{\varphi}+\mu)}N(0)+\frac{\pi}{S_H(S_H^{\varphi}+\mu)} \quad (21)$$

By taking the inverse Laplace transform of Eq. (21), we obtained:

$$N(t)\leq E_{t\varphi,1}(-\mu t^{\varphi})N(0)+\pi E_{t\varphi,\varphi+1}(-\mu t^{\varphi}) \quad (22)$$

At $t\to\infty$, the limit of Eq. (22) becomes:

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

This means that, if $N_0\leq\frac{\pi}{\mu}$.

Then, $N(t)\leq\frac{\pi}{\mu}$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 V, we consider $U=[0,V[]$

The set of all continuous function that is defined on P is represented by $N_e^0(V)$ with norm as:

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

Considering model (13) along with the initial conditions specified, this can be represented as an initial value problem (IVP) in (23).

$${}^{c}D_t^{\varphi}K(t)=Z(t,K(t)),0<t<V<\infty, \quad (23)$$

$K(0)=K_0.$

Where $K(t)=\left(S_H(t),E_H(t),I_A(t),I_S(t),T(t),A(t)\right)$.represent the classes and Z be a continuous function defined as follows:

$$Z(t,K(t))=\begin{pmatrix}Z_1\left(t,S_H(t)\right)\\Z_2(t,E_H(t))\\Z_3(t,I_A(t))\\Z_4(t,I_S(t))\\Z_5\left(t,T(t)\right)\\Z_6\left(t,A(t)\right)\end{pmatrix}=\begin{pmatrix}\pi-\frac{(\beta_1 I_A+\beta_2 I_S+\beta_3 T+\beta_4 A)}{N}S_H-\mu S_H\\\frac{(\beta_1 I_A+\beta_2 I_S+\beta_3 T+\beta_4 A)}{N}S_H-(\theta+\mu)E_H\\\theta E_H-(\alpha+\sigma_1+\delta_1+\mu)I_A\\\alpha I_A-(\phi+\sigma_2+\delta_2+\mu)I_S\\\sigma_1 I_A+\sigma_2 I_S+\sigma_3 A-(\delta_3+\mu)T\\\phi I_S-(\sigma_3+\delta_3+\mu)A\end{pmatrix} \quad (24)$$

Using proposition (2.1), we have that,

$$S_H(t) = S_{H0} + I_t^{\varphi}\left[\pi - \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H - \mu S_H\right],$$
$$E_H(t) = E_{H0} + I_t^{\varphi}\left[\frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A)}{N} S_H - (\theta + \mu) E_H\right], \quad (25)$$
$$I_A(t) = I_{A0} + I_t^{\varphi}[\theta E_H - (\alpha + \sigma_1 + \delta_1 + \mu) I_A],$$
$$I_S(t) = I_{S0} + I_t^{\varphi}[\alpha I_A - (\phi + \sigma_2 + \delta_2 + \mu) I_S],$$
$$T_H(t) = T_{H0} + I_t^{\varphi}[\sigma_1 I_A + \sigma_2 I_S + \sigma_3 A - (\delta_3 + \mu) T],$$
$$A_0(t) = A_0 + I_t^{\varphi}[\phi I_S - (\sigma_3 + \delta_3 + \mu) A[]]$$

We now obtained the following;

$$S_{Hn}(t) = S_{H0} + \frac{1}{\Gamma(\varphi)}\int_0^t (t-\lambda)^{\varphi-1} Z_1\left(\lambda, S_{H(n-1)}(\lambda)\right) d\,\lambda,$$
$$E_{Hn}(t) = E_{H0} + \frac{1}{\Gamma(\varphi)}\int_0^t (t-\lambda)^{\varphi-1} Z_2\left(\lambda, E_{H(n-1)}(\lambda)\right) d\,\lambda,$$
$$I_{An}(t) = I_{A0} + \frac{1}{\Gamma(\varphi)}\int_0^t (t-\lambda)^{\varphi-1} Z_3\left(\lambda, I_{A(n-1)}(\lambda)\right) d\,\lambda,$$
$$I_{Sn}(t) = I_{S0} + \tfrac{1}{\Gamma(\varphi)}\textstyle\int_0^t (t-\lambda)^{\varphi-1} Z_4\left(\lambda, I_{S(n-1)}(\lambda)\right) d\,\lambda, \quad (26)$$
$$T_n(t) = E_0 + \frac{1}{\Gamma(\varphi)}\int_0^t (t-\lambda)^{\varphi-1} Z_5\left(\lambda, T_{(n-1)}(\lambda)\right) d\,\lambda,$$
$$A_n(t) = A_0 + \frac{1}{\Gamma(\varphi)}\int_0^t (t-\lambda)^{\varphi-1} Z_6\left(\lambda, A_{(n-1)}(\lambda)\right) d\,\lambda.$$

Transforming equation eq. (23) to get

$$X(t) = X(0) + \tfrac{1}{\Gamma(\varphi)}\textstyle\int_0^t (t-\lambda)^{\varphi-1} Z\left(\lambda, X(\lambda)\right) d\,\lambda. \quad (27)$$

Lemma 1

The Lipchitz condition described from Eq. (24) is satisfied by vector $Z(t, K(t))$ on a set $[0, V[]_+^6]$ with the Lipchitz constant given as:

$$\psi = max\left(\left(\beta_1^* + \beta_2^* + \beta_3^* + \beta_4^* + \mu\right), (\theta + \mu), (\alpha + \sigma_1 + \delta_1 + \mu), (\phi + \sigma_2 + \delta_2 + \mu), (\delta_3 + \mu), (\sigma_3 + \delta_4 + \mu)\right).$$

Proof.

$$\|Z_1(t, S_h) - Z_1(t, S_{h1})\|$$
$$= \left\|\pi - \left(\pi - \tfrac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A + \mu)}{N}\right) S_H - \pi - \tfrac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A + \mu)}{N} S_{H1}\right\|,$$
$$= \left\|-\pi - \frac{(\beta_1 I_A + \beta_2 I_S + \beta_3 T + \beta_4 A + \mu)}{N}\left(S_H - S_{H_1}\right) + \mu\left(S_H - S_{H_1}\right)\right\| \leq \left(\beta_1^* + \beta_2^* + \beta_3^* + \beta_4^*\right)\|S_H - S_{H1}\| + \mu\|S_H - S_{H1}\|,$$
$$\therefore \|Z_1(t, S_H) - Z_1(t, S_{H1})\| \leq \left(\beta_1^* + \beta_2^* + \beta_3^* + \beta_4^* + \mu\right)\|S_H - S_{H1}\|$$

Similarly, we obtained the following:

$$\left\|Z_2\left(t, \mathrm{E}_H\right) - Z_2\left(t, \mathrm{E}_{H1}\right)\right\| \leq (\theta + \mu)\left\|\mathrm{E}_H - \mathrm{E}_H\right\|,$$
$$\left\|Z_3\left(t, \mathrm{I}_A\right) - Z_3\left(t, \mathrm{I}_{A1}\right)\right\| \leq (\alpha + \sigma_1 + \delta_1 + \mu)\left\|\mathrm{I}_A - \mathrm{I}_{A1}\right\|, \quad (28)$$
$$\left\|Z_4\left(t, \mathrm{I}_S\right) - Z_4\left(t, \mathrm{I}_{S1}\right)\right\| \leq (\phi + \sigma_2 + \delta_2 + \mu)\left\|I_S - I_{S1}\right\|,$$
$$\|Z_5(t, T_H\quad) - Z_5(t, T_{H1}\quad)\| \leq (\delta_3 + \mu)\|T_H - T_{H1}\quad\|,$$
$$\|Z_6(t, A) - Z_6(t, A_1)\| \leq (\sigma_3 + \delta_4 + \mu)\|A - A_1\|.$$

Where we obtained:

$$\|Z(t, K_1(t)) - Z(t, K_2(t))\| \leq \varphi\|K_1 - K_2\|,$$
$$\psi = max\left(\left(\beta_1^* + \beta_2^* + \beta_3^* + \beta_4^* + \mu\right), (\theta + \mu), (\alpha + \sigma_1 + \delta_1 + \mu), (\phi + \sigma_2 + \delta_2 + \mu), (\delta_3 + \mu), (\sigma_3 + \delta_4 + \mu)\right). \quad (29)$$

Lemma 2

The initial value problem (16), (17) in Eq. (29) exists and will have a unique solution $K(t) \in D_c^0(E)$.

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

$$K(t) = S_H\left(K(t)\right),$$

Where S is defined as the Picard operator expressed as:

$S_H: D_c^0(E, R_+^6) \to D_c^0(E, R_+^6)$.
Therefore,

$$S_H\big(K(t)\big) = K(0) + \frac{1}{\Gamma(\varphi)}\int_0^t (t-\lambda)^{\varphi-1} Z\,(\lambda, K(\lambda))\, d\,\lambda.$$

Which becomes

$$\big\|S_H\big(K_1(t)\big) - S_H\big(K_2(t)\big)\big\|,$$
$$= \left\|\frac{1}{\Gamma(\varphi)}\left[\int_0^t (t-\lambda)^{\varphi-1} Z(\lambda, K_1(\lambda)) - Z(\lambda, K_2(\lambda))\, d\,\lambda\right]\right\|,$$
$$\leq \frac{1}{\Gamma(\varphi)}\int_0^t (t-\lambda)^{\varphi-1}\, \|Z(\lambda, K_1(\lambda)) - Z(\lambda, K_2(\lambda))\, d\,\lambda\|.$$
$$\leq \frac{\psi}{\Gamma(\varphi)}\int_0^t (t-\lambda)^{\varphi-1}\, \|K_1 - K_2\| d\lambda.$$
$$\big\|S_H\big(K_1(t)\big) - S_H\big(K_2(t)\big)\big\| \leq \frac{\psi}{\Gamma(\varphi+1)S_H}.$$

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

Lemma 3

The initial value problem (6), in Eq. (29) exists and will have a unique solution $X(t) \in A_c^0(f)$.
Using Picard-Lindelöf 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,

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

Which becomes:

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

When $\frac{\psi}{\Gamma(\varphi+1)} S_H \leq 1$, then the Picard operator gives a contradiction,
So, Eq. (6), (7) solution is unique.

RESULTS AND DISCUSSION

Fractional order model numerical results

Applying the generalized fractional Adams–Bashforth–Moulton scheme outlined in (31), the fractional-order HIV/AIDS model was solved numerically. The parameter values used are listed in Table 1, where various fractional-order values $\varphi$are incorporated and simulated.

Implementation of fractional Adams–Bashforth–Moulton method

The approach developed by Baskonus et al. (2015), Diethelm (1999), and freed is adopted in this study. An approximate solution to the fractional HIV/AIDS model in equation (6) is obtained using the fractional Adams–Bashforth–Moulton scheme. Consequently, the fractional model in (6) can be rewritten as follows:

$$^cD_t^{\varphi} P(t) = Q(t, q(t)), 0 < t < \psi, \qquad (30)$$
$$P^{(n)}(0) = P_0^{(n)}, n = 1,0,\ldots, q, q = [\varphi].$$

Where $P = \left(S_H^*, E_H^*, I_A^*, I_S^*, T^*, A^* \right) \in R_+^6$ and $V(t, q(t))$ is a real valued function that is continuous.
Eq. (27) can therefore be represented using the concept of fractional integral as follows:

$$P(t) = \sum_{n=0}^{m-1} P_0^{(n)} \frac{t^n}{n!} + \frac{1}{\Gamma(\varphi)} \int_0^t (t-y)^{\varphi-1} R\big(k, m(k)\big) dk \quad (31)$$

Using the method described in Amos et al. (2024), we let the step size $g = \frac{\psi}{N}, N \in N$ with a grid that is uniform on $[0, \psi]$. Where $t_c = cr, c = 0,1,1, \ldots N$. Therefore, the fractional order model of HIV/AIDs model presented in (6) can be approximated as:

$$S_{Hk+1}(t) = S_{H0} + \frac{g^\varphi}{\Gamma(\varphi+2)} \left\{ \pi - \frac{(\beta_1 I_A^n + \beta_2 I_S^n + \beta_3 T^n + \beta_4 A^n)}{N} S_H^n - \mu S_H^n \right\} + \frac{g^\varphi}{\Gamma(\varphi+2)} \sum_{y=0}^k dy, k+1 \left\{ \pi - \frac{(\beta_1 I_{Ay} + \beta_2 I_{Sy} + \beta_3 T_y + \beta_4 A_y)}{Ny} S_{Hy} - \mu S_{Hy} \right\},$$

$$E_{Hk+1}(t) = E_{H0} + \frac{g^\varphi}{\Gamma(\varphi+2)} \left\{ \frac{(\beta_1 I_A^n + \beta_2 I_S^n + \beta_3 T^n + \beta_4 A^n)}{N} S_H^n - (\theta+\mu) E_H^n \right\} + \frac{g^\varphi}{\Gamma(\varphi+2)} \sum_{y=0}^k dy, k+1 \left\{ \frac{(\beta_1 I_{Ay} + \beta_2 I_{Sy} + \beta_3 T_y + \beta_4 A_y)}{Ny} S_{Hy} - (\theta+\mu) E_{Hy} \right\}, \quad (32)$$

$$I_{A(k+1)}(t) = I_{A0} + \frac{g^\varphi}{\Gamma(\varphi+2)} \{\theta E_H^n - (\alpha + \sigma_1 + \delta_1 + \mu) I_A^n\} + \frac{g^\varphi}{\Gamma(\varphi+2)} \sum_{y=0}^k dy, k+1 \{\theta E_{Hy} - (\alpha + \sigma_1 + \delta_1 + \mu) I_{Ay}\},$$

$$I_{S(k+1)}(t) = I_{S0} + \frac{g^\varphi}{\Gamma(\varphi+2)} \{\alpha I_A^n - (\phi + \sigma_2 + \delta_2 + \mu) I_S^n\} + \frac{g^\varphi}{\Gamma(\varphi+2)} \sum_{y=0}^k dy, k+1 \{\alpha I_{Ay} - (\phi + \sigma_2 + \delta_2 + \mu) I_{Sy}\},$$

$$T_{(k+1)}(t) = T_0 + \frac{g^\varphi}{\Gamma(\varphi+2)} \{\sigma_1 I_A^n + \sigma_2 I_S^n + \sigma_3 A^n - (\delta_3 + \mu) T^n\} + \frac{g^\varphi}{\Gamma(\varphi+2)} \sum_{y=0}^k dy, k+1 \{\sigma_1 I_{Ay} + \sigma_2 I_{Sy} + \sigma_3 A_y - (\delta_3 + \mu) T_y\},$$

$$A_{(k+1)}(t) = A_0 + \frac{g^\varphi}{\Gamma(\varphi+2)} \{\phi I_S^n - (\sigma_3 + \delta_3 + \mu) A^n\} + \frac{g^\varphi}{\Gamma(\varphi+2)} \sum_{y=0}^k dy, k+1 \{\phi I_{Sy} - (\sigma_3 + \delta_3 + \mu) A_y\}.$$

Where

$$S_{H(k+1)}^n(t) = S_{H0} + \frac{1}{\Gamma(\varphi)} \sum_{y=0}^k f_{y,k+1} \left\{ \pi - \frac{(\beta_1 I_{Ay} + \beta_2 I_{Sy} + \beta_3 T_y + \beta_4 A_y)}{Ny} S_{Hy} - \mu S_{Hy} \right\},$$

$$E_{H(k+1)}^n(t) = E_{H0} + \frac{1}{\Gamma(\varphi)} \sum_{y=0}^k f_{y,k+1} \left\{ \frac{(\beta_1 I_{Ay} + \beta_2 I_{Sy} + \beta_3 T_y + \beta_4 A_y)}{Ny} S_{Hy} - (\theta+\mu) E_{Hy} \right\},$$

$$I_{A(k+1)}^n(t) = I_{A0} + \frac{1}{\Gamma(\varphi)} \sum_{y=0}^k f_{y,k+1} \{\theta E_{Hy} - (\alpha + \sigma_1 + \delta_1 + \mu) I_{Ay}\}, \quad (33)$$

$$I_{S(k+1)}^n(t) = I_{S0} + \frac{1}{\Gamma(\varphi)} \sum_{y=0}^k f_{y,k+1} \{\alpha I_{Ay} - (\phi + \sigma_2 + \delta_2 + \mu) I_{Sy}\},$$

$$T_{(k+1)}^n(t) = T_0 + \frac{1}{\Gamma(\varphi)} \sum_{y=0}^k f_{y,k+1} \{\sigma_1 I_{Ay} + \sigma_2 I_{Sy} + \sigma_3 A_y - (\delta_3 + \mu) T_y\}$$

$$A_{(k+1)}^n(t) = A_0 + \frac{1}{\Gamma(\varphi)} \sum_{y=0}^k f_{y,k+1} \{\phi I_{Sy} - (\sigma_3 + \delta_3 + \mu) A_y\}.$$

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

Importance of using the fractional Adam-Bashforth Moulton method in obtaining the numerical solutions of the model

1. The fractional Adams–Bashforth–Moulton method provides high-order accuracy while requiring only one extra function evaluation at each step.
2. It features built-in error control and is widely applied in integrated solvers for ordinary differential equations.
3. Owing to its versatility across disciplines such as engineering, chemistry, and medicine, this approach is an effective tool for the numerical solution of partial and fractional-order differential equations.

Fractional Order HIV/AIDS Model Simulation

This section presents the numerical simulation of the HIV/AIDS model using the three differential operators employed. Table 2 shows the values of the model variables (parameters) used in this simulation. The sources of these parameters are cited in APA format.

Table 2: Description of parameters

Parameter	Values/day	Sources
π_h	0.0014	Abah et al., 2025
β_1	0.3425	Alkahtani et al., 2017
β_2	0.1126	Abah et al., 2025
β_3	0.03	Estimated
β_4l	0.6742	Estimated
θ	0.003	Subashini et al., 2020
ϕ	0.1540	Abah et al., 2025
σ_1	0.02	Estimated
σ_2	0.15001	Odiba et al. (2024)
σ_3	0.6	Abah et al., 2025
δ_1	0.001	Abah et al., 2025
δ_2	0.002	Estimated
δ_3	0.004	Estimated
δ_4	0.008	Estimated
A	0.01399	Odiba et al. (2024)
M	0.4	Abah et al., 2025

Numerical Simulations

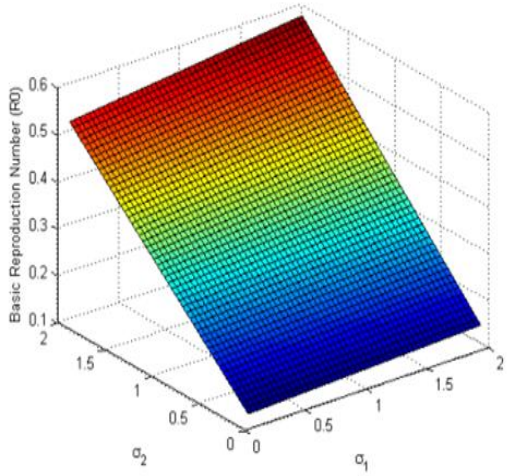

Figure 3a: Surface plot showing the impact of σ_1 and $\sigma_2$on R_0^H

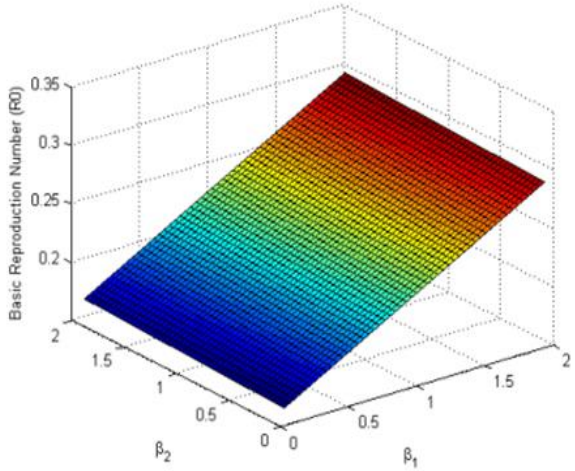

Figure 3b: Surface plot showing the impact of β_1 and $\beta_2$on R_0^H

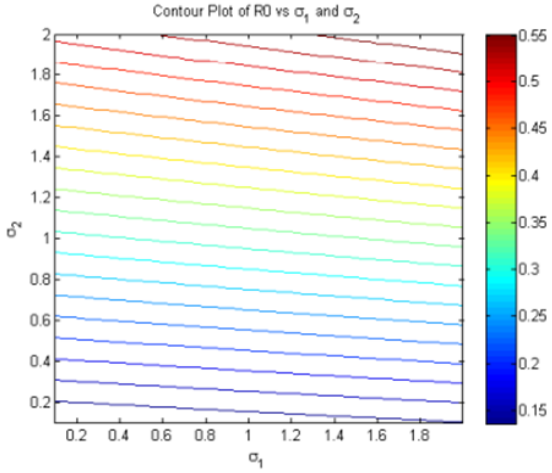

Figure 3c: Contour plot showing the impact of σ_1 and $\sigma_2$on R_0^H

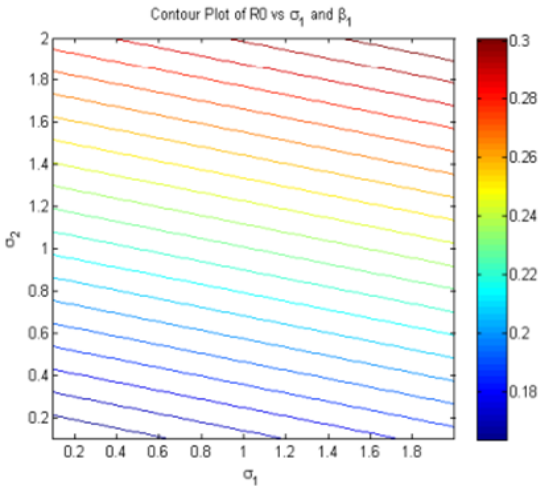

Figure 3d: Contour plot showing the impact of β_1 and $\sigma_1$on R_0^H

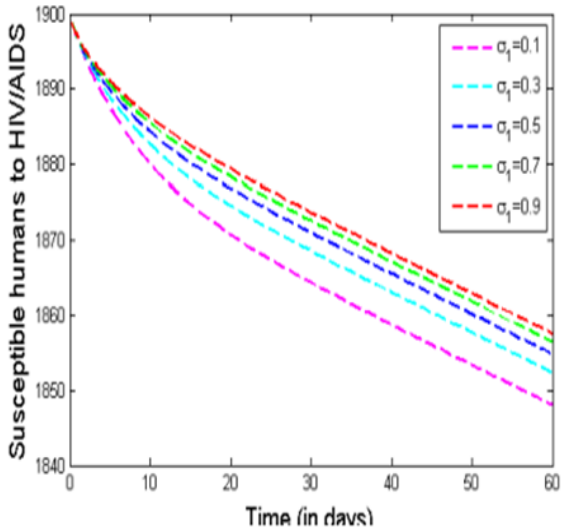

Figure 4a: Simulation of the impact of σ_1 on susceptible human population

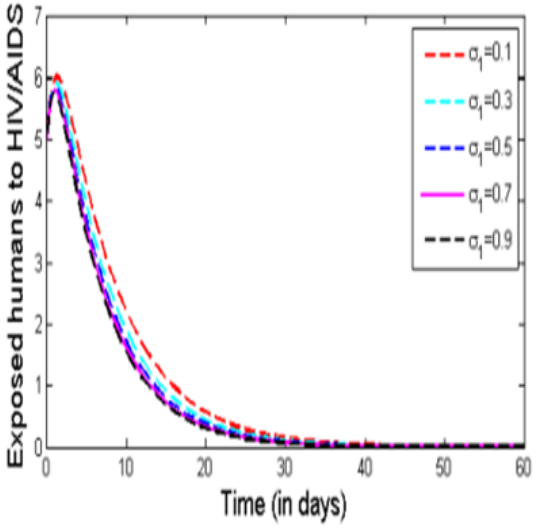

Figure 4b: Simulation of the impact of σ_1 on exposed human population

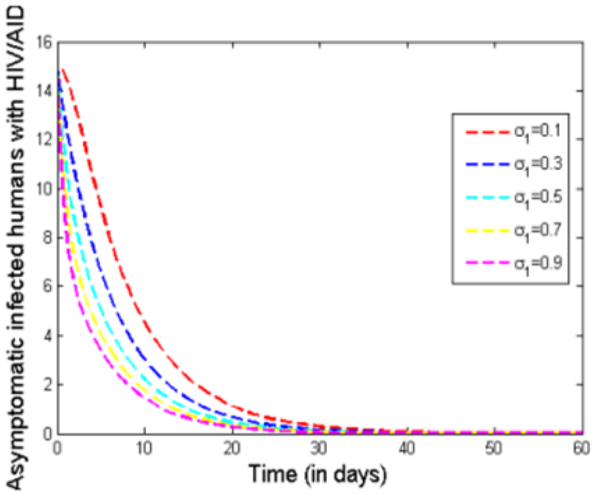

Figure 4c: Simulation of the impact of σ_1 on asymptomatic infected human population

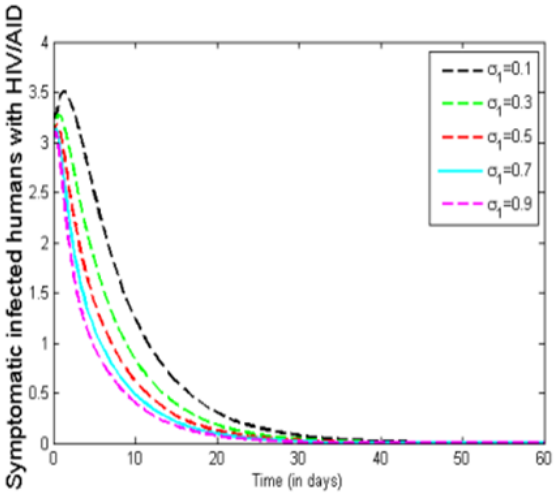

Figure 4d: Simulation of the impact of σ_1 on symptomatic infected human population

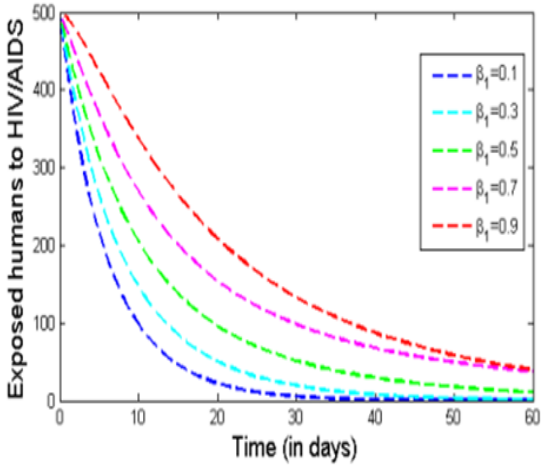

Figure 4e: Simulation of the impact of β_1 on asymptomatic infected human population

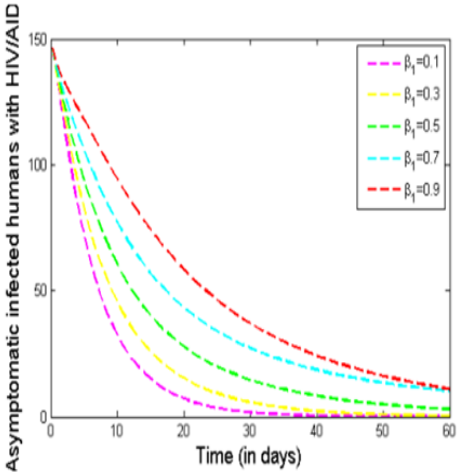

Figure 4f: Simulation of the impact of β_1 on asymptomatic infected human population

Fig (3a) shows that the basic reproduction number $R_0^H$is reduced and is less than 1 with increasing treatment rates $\sigma_1$and σ_2 . Biologically this implies that a large increase in treatment coverage among asymptomatic and symptomatic persons will significantly reduce the effect of HIV/AIDS on the human population. If the treatment measures are inadequate, the prevalence of the disease will be high. Therefore, the figure shows that the larger the fraction of infected individuals that receive effective treatment, the faster the decrease of HIV/AIDS cases in the population, thus establishing a direct link of the model results to the importance of treatment interventions in real life situations.

Fig. (3b) shows that decreases below unity R_0^H when the transmission rate $\beta_1$decrease and $\sigma_1$increase. Biologically, this means that the spread of HIV/AIDS can be reduced by lowering the rate of contact between susceptible and infected individuals and increasing the rate of treatment. Without effective control measures to reduce transmission, the prevalence of the disease would increase. This figure underscores the significance of interventions such as public awareness campaigns, safe practices and behavioral modifications, as reducing the effective contact rate directly reduces the number of new infections.

Fig.3c shows the contour plot of $\sigma_1$and $\sigma_2$with respect toR_0^H. The maximum value is 0.6, indicating the transmission rate is still high for some combinations of parameters. Increasing treatment rates $\sigma_1$and $\sigma_2$leads to a decrease in, which biologically correspond to a faster decline of the population of humans infected with HIV/AIDS. This emphasizes the need to increase treatment interventions.

(Fig. 3d) The contour plot with and shows that increasing treatment coverage σ_1 and decreasing contact rate $\beta_1$are reduces below one. Biologically, this means that higher treatment rates, along with a reduction in the interactions between susceptible and infected individuals, can lead to the eradication of HIV/AIDS from the human population.

(Fig. 4a) illustrates that as the treatment rate $\sigma_1$of infected individuals' increases, the number of humans susceptible to HIV/AIDS decreases. This emphasizes the preventive effect of treating infectious individuals to control the spread of the disease.

(Fig.4b) indicates that when a greater number of exposed individuals are treated at rateσ_1, the population of humans that develop HIV/AIDS drops. This underscores the importance of early intervention in the exposed population biologically to decrease the total disease burden.

(Fig. 4c) This figure depicts the influence of the treatment rate $\sigma_1$on the population of asymptomatic HIV/AIDS-infected individuals. The result indicates that an increase in the treatment rate leads to a decline in the number of asymptomatic infected persons. From a biological perspective, this suggests that effective treatment of asymptomatic individuals helps to slow down both the progression and transmission of HIV/AIDS within the population.

(Fig. 4d) This figure presents the impact of the treatment rate on symptomatic infected individuals. It is observed that higher treatment rates σ_1 significantly reduce the number of symptomatic cases. This highlights the importance of timely and adequate treatment for both asymptomatic and symptomatic individuals in minimizing the overall prevalence of the disease.

(Fig. 4e) This figure illustrates that an increase in the contact rate β_1 leads to a rise in the exposed human population. This indicates that more frequent interactions between susceptible and infected individuals significantly elevate the risk of exposure.

(Fig. 4f) The figure demonstrates that a higher contact rate β_1 results in an increase in the number of asymptomatic infected individuals. This underscores the importance of reducing contact rates in order to curb the hidden transmission of the disease.

CONCLUSION

A fractional order mathematical model for HIV/AIDS transmission was formulated in this research to investigate the impact of treatment and contact rates on the course of the disease. The result of the model analysis showed that the solution exists and is unique, thereby validating the model, while the stability analysis showed that the disease-free state can be achieved if the basic reproduction number is less than one. The findings show that higher treatment rates for exposed, asymptomatic and symptomatic individuals have a substantial impact on HIV/AIDS transmission. Specifically, treatment of exposed individuals and continuous treatment of infected individuals is effective in reducing the spread of the disease and the disease burden. On the other hand, increased contact rates were shown to increase the spread of HIV/AIDS, which in turn results in a higher exposure and infection rate. The contour and simulation findings also reveal the need for an optimal balance of high treatment coverage and low contact rates to manage and possibly eradicate HIV/AIDS. The study also shows that suboptimal treatment approaches or ongoing high-risk contact patterns may maintain the disease. In summary, the results highlight the need for a multifaceted approach to control the disease in the population, including access to treatment and early detection, and those interventions that reduce contact rates. These measures are essential for reducing the impact and burden of HIV/AIDS.

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