



Original Study

A mathematical and sensitivity analysis of an HIV/AIDS infection model

Idris Ahmed^{1†}, Jessada Tariboon², Mubarak Muhammad³, Muhammad Jamilu Ibrahim¹

¹Department of Mathematics, Faculty of Natural and Applied Sciences, Sule Lamido University, Kafin Hausa, Jigawa, 00048, Nigeria

²Department of Mathematics, Faculty of Science, King Mongkut's University of Technology North Bangkok, Bangkok, 10800, Thailand

³Khon Kaen University, Khon Kaen, 40002, Thailand

Communicated by Carlo Cattani; Received: 25.07.2023; Accepted: 01.01.2024; Online: 02.06.2024

Abstract

This study presents a mathematical model to investigate the dynamics of Human Immunodeficiency Virus infection and Acquired Immunodeficiency Syndrome (HIV/AIDS) transmission. Employing mathematical analysis, non-negativity, boundedness, the basic reproduction number $\mathcal{R}_0$, and the stability of both the disease-free and endemic equilibrium of the proposed model were derived. Normalized forward sensitivity techniques are used to determine the significance and importance of sensitive parameters associated with $\mathcal{R}_0$. To gain insights into the dynamical behavior of each compartment, an effective numerical scheme was utilized, and the results obtained suggest that there is a need, even if individuals are infected with the virus, to use non-pharmaceutical interventions as control strategies.

Keywords: HIV/AIDS, mathematical model, sensitivity analysis, numerical simulations.

AMS 2020 codes: 34B15; 34D20; 65D30; 65L05.

1 Introduction

Epidemiological models have played a crucial role in understanding the dynamics of disease transmission and developing effective control strategies for infectious diseases in real-world situations. Over the past decade, HIV/AIDS have become deadly infectious diseases, particularly in developing countries. This challenge has led to the development of some important HIV/AIDS treatment strategies, such as antiretroviral therapy (ART), among many others. Human immunodeficiency virus (HIV), the causative agent of the disease condition referred to as acquired immunodeficiency syndrome (AIDS), was regarded as the deadliest global epidemic before the emergence of coronavirus 2019 (COVID-19) on December 31, 2019 [1]. As of the end of the year 2021, the global HIV and AIDS statistics compiled by the Joint United Nations Programme on HIV/AIDS (UNAIDS) revealed that there were 38.4 million people living with HIV in the world, and AIDS-related illnesses accounted for the deaths of 40.1 million people since the beginning of the disease [2].

A plethora of numerous scientific advancements have been made concerning HIV/AIDS treatments, care, and preventive measures. HIV treatment and care usually follow the guidelines provided by the World Health Organization (WHO) to conform to global evidence-based best practices [3]. However, despite such advancements, the bottleneck that serves as the greatest hindrance and challenge in the global HIV response remains the

[†]Corresponding author.

Email address: idris.ahmed@slu.edu.ng

stigmatization and marginalization of the affected people [4]. For instance, new cases were observed to be more prevalent among individuals that remain unaware, disadvantaged, or neglected. As such, there is still a need for more global efforts if the global vision to end AIDS by the year 2030 is to come to fruition [5].

In line with the global objective to end AIDS by the year 2030, one important aspect that needs to be strengthened is the dynamics of HIV/AIDS transmission. Ensuring the halt of the HIV/AIDS epidemic is of utmost importance, and this objective can be attained through the implementation of efficient and effective strategies, prominently including HIV/AIDS treatment. One such crucial treatment available to infected individuals is the antiretroviral therapy (ART) [6, 7]. By preventing transmission between sexual partners, these therapeutic interventions play a pivotal role. Therefore, early access to ART is highly recommended, as it not only enhances the health status of individuals living with HIV/AIDS but also serves as a preventive measure against further dissemination of the virus. In the present era, modeling techniques have become indispensable in both theoretical and practical aspects of controlling and managing the spread of infectious diseases. This development has significantly influenced policy-making decisions related to disease epidemiology in numerous countries worldwide [8]. The field of HIV/AIDS research has witnessed the emergence of several models aimed at comprehending the transmission dynamics of the disease. Notable research on such models can be found in references [9–14].

Moreover, investigating the dynamics of HIV/AIDS is of great importance from many perspectives. For instance, with the recent success in the availability of the HIV vaccine [15], disease dynamic studies can be of great value in making informed decisions on priorities for vaccine distribution and recommendations. Additionally, studies into the dynamics of HIV/AIDS transmission can be used to establish evidence that predicts the spread of the disease locally, or more generally, across the globe. Although recent evidence points to an impressive decline in global HIV transmission owing to many international and local measures [16, 17], the risk of an HIV epidemic is still present, especially in economically disadvantaged countries, and the measures in place need to be sustained, intensified, and, to some extent, reviewed and appropriately modified. The current trend towards mitigating HIV/AIDS transmission recommends a multifrontal research approach based on the expertise of diverse scientists, including the basic, epidemiological, clinical, and social sciences, among others [18–31].

The rest of the paper is composed as follows. In Section 2, the formulation of the proposed model is studied. In Section 3, the theoretical analysis of the model is investigated. In Section 4, sensitivity and numerical analysis of the model is extracted. In Section 5, the important novelties of this paper are reported.

2 Formulation of the proposed model

In this section, we propose a nonlinear HIV/AIDS epidemic model with optimal control strategies. The aim is to illustrate the significant role that treatment and contact rate play in effectively regulating the future course of HIV/AIDS transmission. At a given time t , the total human population represented as $\mathcal{N}(t)$ is divided into four classes: susceptible individuals denoted as $\mathcal{S}(t)$, HIV/AIDS protected individuals denoted as $\mathcal{S}_p(t)$, HIV/AIDS infected individuals denoted as $\mathcal{I}(t)$, and HIV/AIDS treated individuals denoted as $\mathcal{T}(t)$. The total human population is calculated as

$$\mathcal{N}(t) = \mathcal{S}(t) + \mathcal{S}_p(t) + \mathcal{I}(t) + \mathcal{T}(t).$$

Let

$$\lambda(t) = \frac{\beta}{\mathcal{N}} \mathcal{I}(t), \quad (1)$$

be defined as the standard incidence rate at which susceptible individuals ($\mathcal{S}$) get HIV/AIDS, and β is the HIV/AIDS transmission rate. The quantities π and $(1 - \pi)$ represent the proportions of individuals recruited into the HIV/AIDS-protected ($\mathcal{S}_p$) and susceptible ($\mathcal{S}$) groups, respectively. Within the susceptible class ($\mathcal{S}$), the number of individuals increases as individuals enter from the HIV/AIDS protected class ($\mathcal{S}_p$), where they lose their immunity at a rate of α . Susceptible individuals ($\mathcal{S}$) are at risk of contacting HIV/AIDS and becoming

infected at a rate of λ , after which they transition to the infected class ($\mathcal{I}$). HIV/AIDS-infected individuals ($\mathcal{I}$) receive treatment and transition to the treated class ($\mathcal{T}$) at a rate of γ . It is worth mentioning that in all four classes, natural mortality occurs at a rate of μ . However, individuals in the HIV/AIDS infected category $\mathcal{I}$ and treated classes $\mathcal{T}$ experience additional disease-induced mortality at rates of d , respectively.

Considering all the aforementioned assumptions, the model describing HIV/AIDS virus transmission in a sexually active population is as follows:

$$\begin{aligned}\frac{d\mathcal{S}(t)}{dt} &= (1 - \pi)\Delta + \alpha\mathcal{S}_P - (\mu + \lambda)\mathcal{S}, \\ \frac{d\mathcal{S}_P(t)}{dt} &= \pi\Delta - (\alpha + \mu)\mathcal{S}_P, \\ \frac{d\mathcal{I}(t)}{dt} &= \lambda\mathcal{S} - (\mu + d + \gamma)\mathcal{I}, \\ \frac{d\mathcal{T}(t)}{dt} &= \gamma\mathcal{I} - \mu\mathcal{T},\end{aligned}\tag{2}$$

with the initial conditions:

$$\mathcal{S}(0) = \mathcal{S}_0 \geq 0, \mathcal{S}_P(0) = \mathcal{S}_{P0} \geq 0, \mathcal{I}(0) = \mathcal{I}_0 \geq 0, \text{ and } \mathcal{T}(0) = \mathcal{T}_0 \geq 0.\tag{3}$$

Remark 1. It's important to note that this model assumes a well-mixed population, meaning there is no consideration for age, gender, sexual behavior, or other factors that may affect the transmission dynamics.

Table 1 Interpretation of state variables and parameters for the HIV/AIDS model (2).

Compartment	Description
$\mathcal{S}$	Susceptible individuals
$\mathcal{S}_P$	HIV/AIDS Protected individuals
$\mathcal{I}$	Individuals infected with HIV/AIDS
$\mathcal{T}$	Individuals treated with HIV/AIDS
Parameters	Meanings
π	Portion of recruitment individuals entered to HIV/AIDS protected class
Δ	Recruitment rate of human being
α	Loose of immunity
μ	Natural death rate
d	Death rate due to HIV/AIDS
γ	HIV/AIDS infection treatment rate

3 Theoretical analysis of the model

We present in this section the theoretical analysis of the model (2). This analysis comprises the positivity, boundedness, and stability analyses of the equilibrium points, respectively.

3.1 Positivity and boundedness of solution

Now, from system (2) and summing-up the equations and initial condition (3), gives

$$\Psi = \left\{ (\mathcal{S}, \mathcal{S}_P, \mathcal{I}, \mathcal{T}) \in \mathbb{R}_+^4 : \mathcal{N} \leq \frac{\Delta}{\mu} \right\}.\tag{4}$$

The positive invariant can be easily verified, and this ensures that the solutions remain in the set Ψ . Thus, model (2) is epidemiologically and mathematically well-posed in Ψ .

3.2 Stability analysis of the disease-free equilibrium

Now, to obtain the disease-free equilibrium (DFE) of model (2), we set each and every equation in system (2) equals to zero. Thus, DFE is

$$E_0 = (\mathcal{S}^0, \mathcal{S}_P^0, \mathcal{I}^0, \mathcal{T}^0) = \left(\frac{\Delta}{\mu} \left(\frac{\alpha + \mu(1 - \pi)}{\alpha + \mu} \right), \frac{\Delta\pi}{\alpha + \mu}, 0, 0 \right). \quad (5)$$

Using the concepts of next-generation matrix as stated in [32]. The basic reproduction number denoted by $\mathcal{R}_0$ of the model (2) is determined as

$$\mathcal{R}_0 = \frac{\beta(\mu(1 - \pi) + \alpha)}{(\gamma + \mu + d)(\mu + \alpha)}. \quad (6)$$

Note that, the basic reproduction number is defined as the expected number of secondary cases generated by one infected individual during its entire period of infectiousness in a fully susceptible population. Therefore, the DFE $E_0 = (S_0, P_0, I_0, T_0) = \left(\frac{\Delta}{\mu} \left(\frac{\alpha + \mu(1 - \pi)}{\alpha + \mu} \right), \frac{\Delta\pi}{\alpha + \mu}, 0, 0 \right)$ is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.

3.3 Stability analysis of endemic equilibrium

By setting the right-hand side of each equations in system (2) to zero, we get

$$\begin{cases} \mathcal{S}^* = \frac{\Delta(1 - \pi)(\alpha + \mu) + \alpha\pi\Delta}{(\alpha + \mu)(\mu + \lambda^*)}, \\ \mathcal{S}_P^* = \frac{\pi\Delta(\alpha + \mu)\lambda^* + \alpha\pi\Delta\lambda^*}{(\mu + d + \gamma)(\alpha + \mu)(\mu + \lambda^*)}, \\ \mathcal{I}^* = \frac{(1 - \pi)\mu\Delta\gamma(\alpha + \mu)\lambda^* + \alpha\pi\mu\Delta\gamma\lambda^*}{(\mu + d + \gamma)(\alpha + \mu)(\mu + \lambda^*)}. \end{cases} \quad (7)$$

Let's denote $a_1 = \alpha + \mu$, $a_2 = \mu + d + \gamma$ and insert I^* in equation (1), gives

$$\lambda^* = \frac{\beta(1 - \pi)\Delta a_1 \lambda^* + \beta\alpha\pi\Delta\lambda^*}{(1 - \pi)\Delta a_1 a_2 + \alpha\pi\Delta a_2 + \pi\Delta a_2(\mu + \lambda^*) + (1 - \pi)\Delta a_1 \lambda^* + \alpha\pi\Delta\lambda^*(1 + \mu\gamma) + (1 - \pi)\mu\Delta\gamma a_1 \lambda^*},$$

upon simplification, we obtain

$$x_1 \lambda^* + x_0 = 0, \quad (8)$$

such that

$$x_1 = -\pi\Delta a_1 - (1 - \pi)\Delta a_1 - \alpha\pi\Delta - (1 - \pi)\mu\Delta\gamma a_1 - \alpha\pi\mu\Delta\gamma < 0,$$

and $x_0 = a_1 a_2 [\mathcal{R}_0 - 1] > 0$ if $\mathcal{R}_0 > 1$ since all parameters are positive. Thus,

$$\lambda^* = \frac{-x_0}{x_1} = \frac{-a_1 a_2 [\mathcal{R}_0 - 1]}{-[\pi\Delta a_2 + (1 - \pi)\Delta a_1 + \alpha\pi\Delta + (1 - \pi)\mu\Delta\gamma a_1 + \alpha\pi\mu\Delta\gamma]},$$

and gives

$$\lambda^* = \frac{-a_1 a_2 [\mathcal{R}_0 - 1]}{\pi\Delta a_2 + (1 - \pi)\Delta a_1 + \alpha\pi\Delta + (1 - \pi)\mu\Delta\gamma a_1 + \alpha\pi\mu\Delta\gamma} > 0,$$

if and only $\mathcal{R}_0 > 1$, this implies that there is a unique positive endemic equilibrium for the HIV/AIDS model (2) if and only if $\mathcal{R}_0 > 1$.

Lemma 1. *There exists a unique endemic equilibrium for HIV/AIDS infection model (2) if and only if $\mathcal{R}_0 > 1$.*

3.4 DFE global asymptotic stability

In this part, making use of the Castillo-Chavez et al. [33] condition, we analyze the global asymptotic stability of the DFE. The following lemma is important.

Lemma 2. (Castillo-Chavez et al. [17]) *Suppose that the HIV/AIDS infection model (2) can be written as*

$$\begin{aligned}\frac{d\mathcal{Y}}{dt} &= \mathcal{G}(\mathcal{Y}, \mathcal{W}), \\ \frac{d\mathcal{Z}}{dt} &= \mathcal{H}(\mathcal{Y}, \mathcal{W}), \quad \mathcal{H}(\mathcal{Y}^0, 0) = 0,\end{aligned}\tag{9}$$

such that $\mathcal{Y} \in \mathbb{R}^m$ is the components of non-infected individuals and $\mathcal{W} \in \mathbb{R}^n$ is the components of infected individuals which include treated class and $E^0 = (\mathcal{Y}^0, 0)$ denotes the DFE point of system (2).

Assume (i) For $(\frac{d\mathcal{Y}}{dt}) = \mathcal{G}(\mathcal{Y}^0, 0)$, $\mathcal{Y}^0$ is globally asymptotically stable (GAS)

(ii) $\mathcal{H}(\mathcal{Y}, \mathcal{W}) = \mathcal{B}\mathcal{W} - \check{\mathcal{H}}(\mathcal{Y}, \mathcal{W})$, $\check{\mathcal{H}}(\mathcal{Y}, \mathcal{W}) \geq 0$ for $(\mathcal{Y}, \mathcal{W}) \in \Omega_1$ where $\mathcal{B} = \mathcal{D}_{\mathcal{W}}\mathcal{H}(\mathcal{Y}^0, 0)$ is a M -matrix i.e. the off diagonal elements of $\mathcal{B}$ are non-negative and Ω_1 is the region in which the system makes biological sense. Then the fixed point $E^0 = (\mathcal{Y}^0, 0)$ is GAS equilibrium point of the system (2) whenever $\mathcal{R}_0 < 1$.

Lemma 3. *Suppose that $\mathcal{R}_0 < 1$, and the sufficient conditions (i) – (ii) given in Lemma 2 are true. Then, DFE point $E^0 = \left(\frac{(1-\pi)\Delta(\alpha+\mu)+\alpha\pi\Delta}{\mu(\alpha+\mu)}, \frac{\pi\Delta}{\alpha+\mu}, 0, 0\right)$ of the HIV/AIDS infection model (2) is GAS.*

Proof. In view of Lemma 2 and model (2), the following matrices were derived

$$\begin{aligned}\frac{d\mathcal{Y}}{dt} &= \mathcal{G}(\mathcal{Y}, \mathcal{W}) = \begin{bmatrix} (1-\pi)\Delta + \alpha\mathcal{S}_P - (\lambda + \mu)\mathcal{S} \\ \pi\Delta - (\alpha + \mu)\mathcal{S}_P \end{bmatrix}, \\ \frac{d\mathcal{W}}{dt} &= \mathcal{H}(\mathcal{Y}, \mathcal{W}) = \begin{bmatrix} \lambda\mathcal{S} - (\mu + d + \gamma)\mathcal{I} \\ \gamma\mathcal{I} - \mu\mathcal{T} \end{bmatrix}, \\ \mathcal{G}(\mathcal{Y}^0, 0) &= \begin{bmatrix} (1-\pi)\Delta + \alpha\mathcal{S}_P^0 - \mu\mathcal{S}^0 \\ \pi\Delta - (\alpha + \mu)\mathcal{S}_P^0 \end{bmatrix},\end{aligned}$$

where $\mathcal{Y}^0 = (\mathcal{S}^0, \mathcal{S}_P^0) = \left(\frac{(1-\pi)\Delta(\alpha+\mu)+\alpha\pi\Delta}{\mu(\alpha+\mu)}, \frac{\pi\Delta}{\alpha+\mu}\right)$ is globally stable which satisfies condition (i) of Lemma 2 and

$$\mathcal{B} = \mathcal{D}_{\mathcal{W}}(\mathcal{Y}^*, 0) = \begin{bmatrix} \beta - (\mu + d + \gamma) & 0 \\ \gamma & -\mu \end{bmatrix}.$$

Upon simplification, yields

$$\check{\mathcal{H}}(\mathcal{Y}, \mathcal{W}) = \begin{bmatrix} \check{\mathcal{H}}_1(\mathcal{Y}, \mathcal{W}) \\ \check{\mathcal{H}}_2(\mathcal{Y}, \mathcal{W}) \end{bmatrix} = \begin{bmatrix} \beta\mathcal{I} - \frac{\beta\mathcal{I}}{\mathcal{N}}\mathcal{S} \\ 0 \end{bmatrix} = \begin{bmatrix} \beta\mathcal{I}\left(1 - \frac{\mathcal{S}}{\mathcal{N}}\right) \\ 0 \end{bmatrix}.$$

From the condition $\mathcal{S} \leq \mathcal{N}$ implies that $\frac{\mathcal{S}}{\mathcal{N}} \leq 1$ and $\check{\mathcal{H}}(\mathcal{Y}, \mathcal{W}) \geq 0$, which satisfies condition (ii) of Lemma 2, thus, the DFE point $E^0 = \left(\frac{(1-\pi)\Delta(\alpha+\mu)+\alpha\pi\Delta}{\mu(\alpha+\mu)}, \frac{\pi\Delta}{\alpha+\mu}, 0, 0\right)$ of the HIV/AIDS infection model (2) is GAS if $\mathcal{R}_0 < 1$. Biologically, whenever $\mathcal{R}_0 < 1$ the HIV/AIDS virus will decline whereas the total population increases [18].

4 Sensitivity and numerical analysis

In this section, we conduct a sensitivity analysis and numerical simulations in order to reinforce the qualitative analysis presented in the preceding sections. To determine the appropriate model parameters, as depicted in Table 1, we examine various relevant literature sources authored by different scholars. Subsequently, we assign some realistic values to these parameters for the purpose of numerical demonstration.

Definition 1. The normalized forward sensitivity index is defined as

$$\Upsilon_{\kappa}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \kappa} * \frac{\kappa}{\mathcal{R}_0},$$

where the variable κ depends differentially on $\mathcal{R}_0$.

This analysis enables to explore the relative significance of different parameters in model (2). The parameter with the highest magnitude surpasses that of all other parameters, indicating its utmost importance.

Table 2 Numerical values for parameters of model (2).

Parameters	Parameters value	Source
Δ	3.2millions humans/day	[34]
π	0.5813/day	Assume
α	0.039/day	Assume
μ	0.5/day	[34]
d	0.47/day	[34]
γ	0.25/day	[35]
β	0.4325/day	[36]

Table 3 Forward normalized sensitivity indices.

Parameters	Indices
β	+1
α	+0.12603
π	−1.1703
μ	−0.085673
d	−0.38525
γ	−0.20492

Table 2 provides the baseline parameters and is based on these parameters. We introduce the sensitivity indices of the model parameters in Table 3. Furthermore, we calculate the basic reproduction number $\mathcal{R}_0 = 0.1633 < 1$, suggesting that the infection is not self-sustaining and will ultimately diminish. As a result, the incidence of new HIV/AIDS cases is projected to decrease over time due to various techniques of interventions, including widespread vaccination, effective treatment, public health measures, and improved hygiene practices. Preventative and control measures can effectively manage the transmission rate (β), which has the significant influence on $\mathcal{R}_0$.

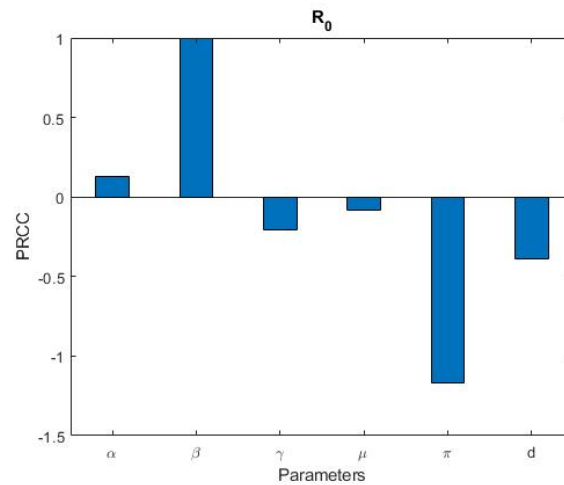


Fig. 1 PRCC sensitivity indices of model parameters on $\mathcal{R}_0$.

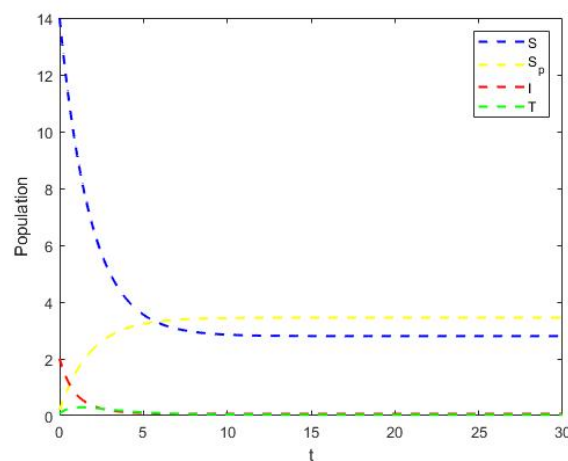


Fig. 2 Solutions behavior of model (2) at $\mathcal{R}_0 < 1$.

Figure 1 depicts the sensitivity indices of the model parameters. It is noteworthy that the transmission rates (β) and loss of immunity (α) manifest the highest biological sensitivity and have a direct correlation with the basic reproduction number as ($\mathcal{R}_0$). Correspondingly, the recruitment rate into the HIV protected group (β), the natural death rate (μ), the disease-induced death rate (d), and the treatment rate (γ) exhibit the highest sensitivity, but in an inverse relationship with the basic reproduction numbers.

The dynamic behavior of model (2) over time is depicted in Figure 2 when the value of $\mathcal{R}_0 < 1$. This numerical result serves to validate the theoretical proof stated in Lemma 3. The basic reproduction number ($\mathcal{R}_0$), is calculated as $\mathcal{R}_0 = 0.1633$. As shown in Figure 2, when $\mathcal{R}_0 = 0.1633 < 1$, the solutions of the HIV/AIDS model 2 converge to DFE after 30 days. From a biological point of view, this implies that the HIV/AIDS infection within the community will be eradicated in the future. Ultimately, the numerical simulation and the theoretical analysis in Lemma 3 coincide.

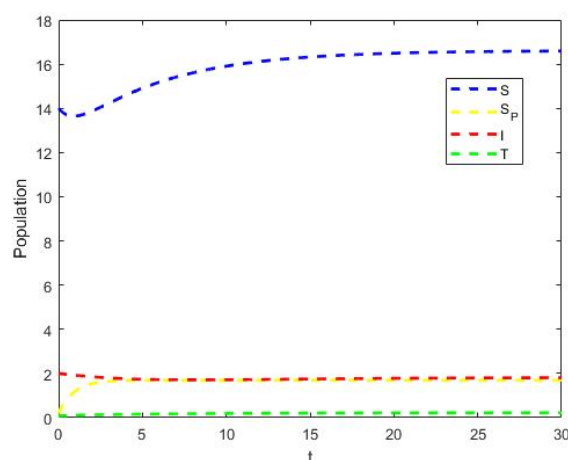
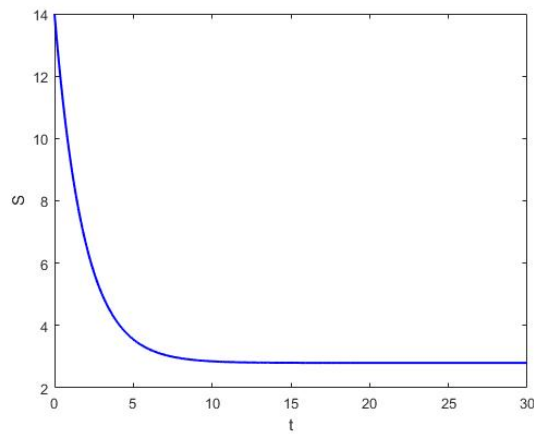
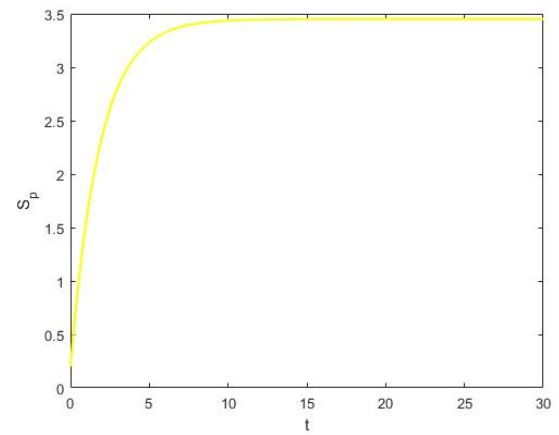
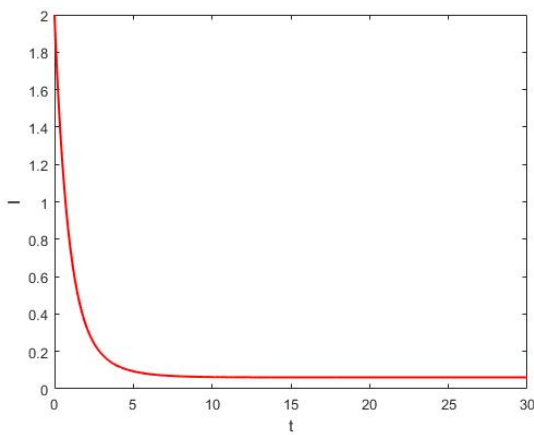
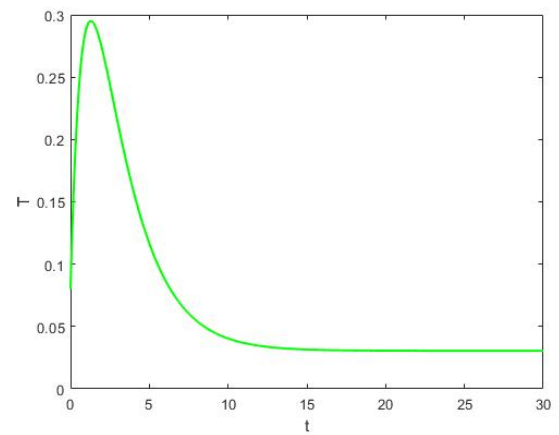
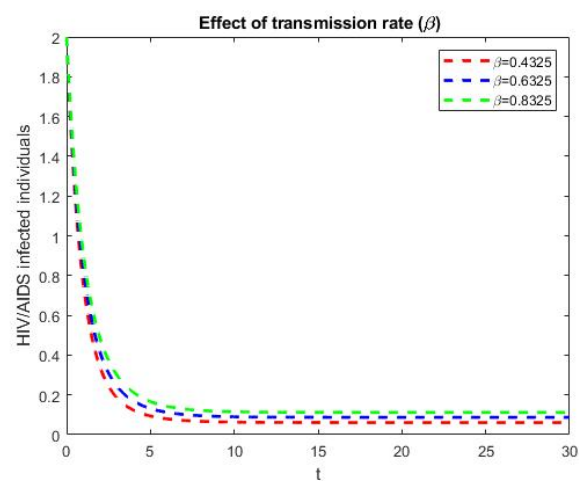


Fig. 3 Solutions behavior of model (2) at $\mathcal{R}_0 > 1$.

Next, we proceed to determine the stability of the endemic equilibrium point of model (2) with a basic reproduction number of $\mathcal{R}_0 = 1.6310 > 1$. As illustrated in Figure 3, all numerical routines consistently indicate that the solution trajectories converge towards the stable endemic equilibrium of the system. Figure 3 presents the time series results of model (2) using the baseline parameter values. We computed the value of the basic reproduction number as $\mathcal{R}_0 = 1.6310 > 1$, indicating that the solutions of model (2) converge towards the endemic equilibrium point when $\mathcal{R}_0 = 1.6310 > 1$ (Figure 3).

We conduct the numerical simulations of model (2) which describe distinct compartments within the human population, including susceptible individuals ($\mathcal{S}$), protected individuals ($\mathcal{S}_P$), infected individuals ($\mathcal{I}$), and treated individuals ($\mathcal{T}$), which examine the temporal changes occurring within these compartments. The results reveal a consistent decrease in the number of susceptible individuals accompanied by an increase in the population of individuals protected against HIV/AIDS over time. This observation is supported by the inverse relationship observed between the susceptible and HIV/AIDS-protected populations, as depicted in Figures 4 and 5. This trend can be attributed to the direct correlation between the number of susceptible individuals and the population protected against HIV/AIDS. Additionally, a decline in the number of individuals infected with HIV/AIDS is observed, as shown in Figure 6. This phenomenon can be attributed to the contributions of HIV/AIDS protected individuals to the susceptible population. Additionally, there is a slight increase in the number of HIV/AIDS treated individuals, which eventually stabilizes and decreases over time. This factor could contribute to the decline in the number of HIV/AIDS infected individuals within the population, as indicated in Figure 7.

Numerical analysis of the rate of transmission between susceptible and infected individuals is conducted to see whether or not the transmission rate contributed significantly to the epidemics of HIV/AIDS infections in the populations. The diagram in Figure 8 shows the positive effects of the transmission rate on HIV/AIDS transmission. An increase in human interactions contributes significantly to the spread of HIV/AIDS infections.

**Fig. 4** Susceptible individuals**Fig. 5** Protected individuals**Fig. 6** Infected individuals**Fig. 7** Treated individuals**Fig. 8** Transmission rate against HIV/AIDS infected individuals.

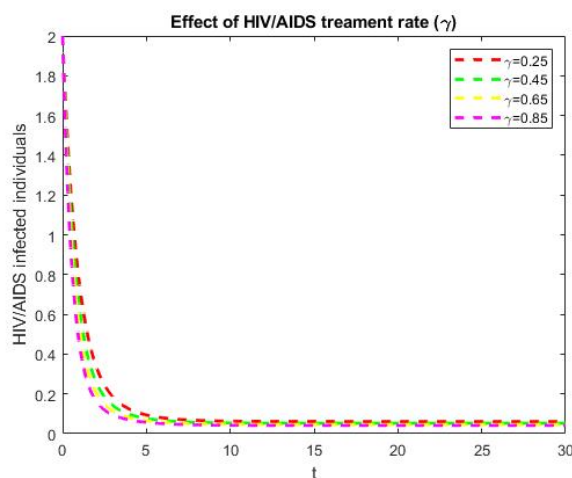


Fig. 9 Treatment rate against HIV/AIDS infected individuals.

Figure 9 illustrates the effect of treatment rate γ on the number of HIV/AIDS infectious population ($\mathcal{I}$). The result shows that an increasing of the value of γ decreases the number of HIV/AIDS infectious individuals in the community. Consequently, increasing treatment intervention measures against HIV/AIDS highly decreases the number of HIV/AIDS infectious people.

5 Conclusions

This study aimed to explore potential control measures against HIV/AIDS, specifically focusing on protective strategies and treatments. The qualitative analysis of the model was thoroughly examined, and it was determined that the model solutions are non-negative and bounded. Utilizing the concept of the next-generation matrix, the basic reproduction number of the model was computed. Furthermore, sufficient conditions for local and global stability of the models' equilibrium points were highlighted, and it is found that when the corresponding basic reproduction number falls below one, the disease-free and endemic equilibrium points are locally and globally asymptotically stable. This shown the significance of implementing effective measures to ensure the basic reproduction number remains below one, thereby effectively controlling the spread of HIV/AIDS.

The sensitivity analysis of the model reveals that the HIV/AIDS transmission rate and the loss of immunity from HIV/AIDS are the most influential parameters impacting the basic reproduction numbers of the model, which directly correlate with the disease burden. This implies that increasing treatment rates will lead to a decrease in HIV/AIDS transmission within the community.

The theoretical findings are validated through numerical simulations, and they can be summarized as follows: The solutions of the HIV/AIDS infection model (2), tend to converge to the disease-free equilibrium point when the basic reproduction number $\mathcal{R}_0 = 0.1633 < 1$. Conversely, the solutions converge to the endemic equilibrium point when $\mathcal{R}_0 = 1.6310 > 1$. These results highlight the importance of focusing on maximizing intervention mechanisms to effectively prevent and control co-infection within the community. Stakeholders should prioritize these measures to combat the spread of HIV/AIDS.

The present study is considered as a preliminary one and opens several potential research avenues for future scholars to explore. These avenues include incorporating an optimal control framework, utilizing a stochastic approach, employing fractional order derivative methods, considering environmental effects, incorporating stages of HIV infection, and validating the model using real-world infection data. Scholars interested in this field can pursue these avenues to contribute to the existing body of knowledge.

6 Declarations

6.1 Conflict of interest:

The authors declare no conflict of interest.

6.2 Funding:

Not applicable.

6.3 Author's contribution:

I.A.-Writing-Review and Editing, Resources, Supervision, Writing-original Draft Preparation. J.T.-Writing-Review and Editing, Conceptualization. M.M.-Methodology, Data Curation. M.J.I.-Writing-Original Draft Preparation, Validation.

6.4 Acknowledgement:

The first author acknowledges the financial support provided by the Tertiary Education Trust Fund (TeT-Fund) and Sule Lamido University Kafin Hausa, P.M.B 048, Kafin Hausa, Jigawa State, Nigeria.

6.5 Data availability statement:

All data that support the findings of this study are included within the article.

6.6 Using of AI tools:

The authors declare that they have not used Artificial Intelligence (AI) tools in the creation of this article.

References

- [1] Wang D., Hu B., Hu C., Zhu F., Liu X., Zhang J., Wang B., Xiang H., Cheng Z., Xiong Y., Zhao Y., Li Y., Wang X., Peng A.Z., Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus-infected pneumonia in Wuhan/China, *JAMA Network*, 323(11), 1061–1069, 2020.
- [2] Reshadat-Hajiabad T., Khajavi A., Hosseinpour A.M., Bojdy A., Hashemi-Meshkini A., Varmaghani M., Determinants and economic burden of HIV/AIDS in Iran: A prospective study, *BMC Health Services Research*, 23(1), 251, 2023.
- [3] WHO, Chan M., Consolidated guidelines on "The use of Antiretroviral Drugs for Treating and Preventing HIV Infection" Recommendations for a Public Health Approach, (2nd Ed.), Switzerland, 1–480, 2016.
- [4] Easterbrook P., Johnson C., Figueroa C., Baggaley R., HIV and Hepatitis testing: global progress challenges and future directions, *AIDS Reviews*, 18(1), 3–14, 2016.
- [5] WHO, Chan M., Global health sector strategy on HIV 2016–2021, Switzerland, 1–60, 2016.
- [6] Dimitrov D., Wood D., Ulrich A., Swan D.A., Adamson B., Lama J.R., Sanchez J., Duerr A., Projected effectiveness of HIV detection during early infection and rapid ART initiation among MSM and transgender women in Peru: a modeling study, *Infectious Disease Modelling*, 4, 73–82, 2019.
- [7] Espitia C.C., Botina M.A., Solarte M.A., Hernandez I., Riascos R.A., Meyer J.F., Mathematical model of HIV/AIDS considering sexual preferences under antiretroviral therapy, a case study in San Juan de Pasto, Colombia, *Journal of Computational Biology*, 29(5), 483–493, 2022.
- [8] Kretzschmar M., Disease modeling for public health: added value, challenges, and institutional constraints, *Journal of Public Health Policy*, 41(1), 39–51, 2020.
- [9] Ngina P., Mbogo R.W., Luboobi L.S., HIV drug resistance: insights from mathematical modelling, *Applied Mathematical Modelling*, 75, 141–161, 2019.
- [10] Gao W., Veerasha P., Cattani C., Baishya C., Baskonus H.M., Modified predictor-corrector method for the numerical solution of a fractional-order SIR model with 2019-nCoV, *Fractal and Fractional*, 6(2), 92, 2022.
- [11] Cai L., Li X., Ghosh M., Guo B., Stability analysis of an HIV/AIDS epidemic model with treatment, *Journal of Computational and Applied Mathematics*, 229(1), 313–323, 2009.
- [12] Hassan A.S., Hussaini N., Analysis of an HIV-HCV simultaneous infection model with time delay, *Journal of the Nigerian Society of Physical Sciences*, 3, 1–11, 2021.

- [13] Apenteng O.O., Osei P.P., Oduro B., Kwabla M.P., Ismail N.A., The impact of implementing HIV prevention policies therapy and control strategy among HIV and AIDS incidence cases in Malaysia, *Infectious Disease Modelling*, 5, 755–765, 2020.
- [14] Yang W., Shu Z., Lam J., Sun C., Global dynamics of an HIV model incorporating senior male clients, *Applied Mathematics and Computation*, 311, 203–216, 2017.
- [15] Prudden H., Tatoud R., Slack C., Shattock R., Anklesaria P., Bekker L.G., Buchbinder S., Experimental medicine for HIV vaccine research and development, *Vaccines*, 11(5), 970, 2023.
- [16] Bassey A.E., Miteu G.D., A review of current trends in HIV epidemiology, surveillance, and control in Nigeria, *Annals of Medicine and Surgery*, 85(5), 1790–1795, 2023.
- [17] Bekolo C.E., Kouanfack C., Ateudjieu J., Bechem E.T., Ndeso S.A., Tendengfor N., Nsagha D.S., Choukem S.P., The declining trend in HIV prevalence from population-based surveys in Cameroon between 2004 and 2018: myth or reality in the universal test and treat era?, *BMC Public Health*, 23(1), 479, 2023.
- [18] Shete A., Wagh V., Sawant J., Shidhaye P., Sane S., Rao A., Kulkarni S., Ghatge M., Antiretroviral treatment-induced galectin-9 might impact HIV viremia in addition to contributing to inflammaging, *International Journal of Molecular Sciences*, 24(15), 12273, 2023.
- [19] Deepika S., Veerasha P., Dynamics of chaotic waterwheel model with the asymmetric flow within the frame of Caputo fractional operator, *Chaos Solitons and Fractals*, 169, 113298, 2023.
- [20] Veerasha P., A numerical approach to the coupled atmospheric ocean model using a fractional operator, *Mathematical Modelling and Numerical Simulation with Applications*, 1(1), 1–10, 2021.
- [21] Naik M.K., Baishya C., Veerasha P., Baleanu D., Design of a fractional-order atmospheric model via a class of ACT-like chaotic system and its sliding mode chaos control, *Chaos: An Interdisciplinary Journal of Nonlinear Science*, 33(2), 023129, 2023.
- [22] Raghavendra V., Veerasha P., Analysing the market for digital payments in India using the predator-prey mode, *An International Journal of Optimization and Control: Theories and Applications*, 13(1), 104–115, 2023.
- [23] Veerasha P., The efficient fractional order based approach to analyze chemical reaction associated with pattern formation, *Chaos Solitons and Fractals*, 165(2), 112862, 2022.
- [24] İlhan E., Veerasha P., Baskonus H.M., Fractional approach for a mathematical model of atmospheric dynamics of CO₂ gas with an efficient method, *Chaos Solitons and Fractals*, 152, 111347, 2021.
- [25] Naik P.A., Owolabi K.M., Yavuz M., Zu J., Chaotic dynamics of a fractional order HIV-1 model involving AIDS-related cancer cells, *Chaos Solitons and Fractals*, 140, 110272, 2020.
- [26] Ahmed I., Akgül A., Jarad F., Kumam P., Nonlaopon K., A Caputo-Fabrizio fractional-order cholera model and its sensitivity analysis, *Mathematical Modelling and Numerical Simulation with Applications*, 3(2), 170–187, 2023.
- [27] Fatima B., Yavuz M., Rahman M.U., Al-Duais F.S., Modeling the epidemic trend of middle eastern respiratory syndrome coronavirus with optimal control, *Mathematical Biosciences and Engineering*, 20(7), 11847–11874, 2023.
- [28] Evirgen F., Uçar E., Uçar S., Özdemir N., Modelling influenza a disease dynamics under Caputo-Fabrizio fractional derivative with distinct contact rates, *Mathematical Modelling and Numerical Simulation with Applications*, 3(1), 58–73, 2023.
- [29] Atede A.O., Oname A., Inyama S.C., A fractional order vaccination model for COVID-19 incorporating environmental transmission: a case study using Nigerian data, *Bulletin of Biomathematics*, 1(1), 78–110, 2023.
- [30] Joshi H., Yavuz M., Townley S., Jha B.K., Stability analysis of a non-singular fractional-order covid-19 model with nonlinear incidence and treatment rate, *Physica Scripta*, 98(045216), 2023.
- [31] Dubey P., Dubey U.S., Dubey B., Modeling the role of acquired immune response and antiretroviral therapy in the dynamics of HIV infection, *Mathematics and Computers in Simulation*, 144, 120–137, 2018.
- [32] Driessche P.V.D., Watmough J., Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Mathematical Biosciences*, 180(1-2), 29–48, 2002.
- [33] Castillo-Chavez C., Blower S., Driessche P.V.D., Kirschner D., Yakubu A.A., *Mathematical Approaches for Emerging and Reemerging Infectious Diseases: Models, Methods, and Theory*, Springer, New York, USA, 2002.
- [34] Hussaini N., *Mathematical Modelling and Analysis of HIV Transmission Dynamics*, (PhD Thesis), Brunel University, UK, 2010.
- [35] Luteria J., Mbete D., Wangila S., Co-infection model of HIV/AIDS-Pneumonia on the effect of treatment at initial and final stages, *IOSR Journal of Mathematics*, 14(5), 56–81, 2018.
- [36] Ringa N., Diagne M.L., Rwezaura H., Oname A., Tchoumi S.Y., Tchuenche J.M., HIV and COVID-19 co-infection: a mathematical model and optimal control, *Informatics in Medicine Unlocked*, 31, 100978, 2022.