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Modeling the impact of biomedical prevention on HIV/AIDS transmission dynamics

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Abstract

The HIV/AIDS epidemic has significantly reshaped global public health priorities, prompting unprecedented investments in research, treatment programs, and prevention strategies. Despite these efforts, the disease continues to place substantial pressure on healthcare systems, particularly in resource limited settings. In this study, a deterministic dynamical system model is developed to assess the impact of key biomedical interventions on the transmission dynamics of HIV/AIDS. The well-posedness of the proposed model is established using the theory of positivity and boundedness of solutions. Analytical results show that the HIV/AIDS-free equilibrium is both locally and globally asymptotically stable whenever the effective reproduction number is less than unity ($\mathcal{R}_H^* < 1$). Furthermore, the analysis reveals that in the presence of antiretroviral therapy (ART) defaulting, the model may exhibit a backward bifurcation. Such a result provides a plausible explanation for the sustained transmission of HIV at the national level despite achieving the effective reproduction number less than unity. In contrast, under conditions of full adherence (100%) to ART, the model exhibits a forward bifurcation, whereby the HIV/AIDS-free equilibrium is stable whenever $\mathcal{R}_H^* < 1$ and loses stability as $\mathcal{R}_H^*$ exceeds unity. Sensitivity analysis of the effective reproduction number highlights the importance of increased ART uptake across all stages of infection, together with expanded coverage of pre-exposure prophylaxis (PrEP) and post-exposure prophylaxis (PEP), as critical strategies for reducing HIV transmission. These findings are further supported by numerical simulations. A key outcome of the study indicates that ART defaulting has the greatest impact in increasing the infectious population. While promoting ART uptake remains essential, strengthening community education on sustained adherence to ART is even more critical for achieving the World Health Organization (WHO) 90-90-90 targets.

Keywords Mathematical model, ART defaulting, PrEP and PEP interventions, Effective reproduction number, Bifurcation and stability analysis

Mathematics Subject Classification 92D30, 37N25, 34D20, 92B05

1 Introduction

More than four decades after its discovery, Human Immunodeficiency virus (HIV) infection remains a persistent global health challenge. The virus continues to affect millions of people worldwide, with profound implications for public health systems,



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socioeconomic development and population dynamics [1, 2]. The virus primarily targets CD4⁺ T lymphocytes, mononuclear macrophages, and dendritic cells, leading to progressive immune suppression and increased susceptibility to opportunistic infections and non-communicable comorbidities such as cardiovascular diseases, nephropathy, and hepatic insufficiency [3]. HIV is transmitted through contact with infected body fluids, most commonly via unprotected sexual intercourse, exposure to contaminated blood, and mother-to-child transmission during pregnancy, delivery, or breastfeeding [4–6].

The clinical course of HIV infection typically progresses through three stages: acute infection, chronic infection, and AIDS, each characterised by distinct viral dynamics and levels of infectiousness. Without treatment, immune deterioration ultimately leads to severe morbidity and death. However, early initiation of antiretroviral therapy (ART) suppresses viral replication, preserves immune function, delays disease progression, and substantially improves survival [5, 6]. Importantly, sustained viral suppression through ART effectively eliminates sexual transmission, positioning ART as both a therapeutic and preventive intervention [2, 7, 8].

Despite the transformative impact of ART, prevention remains essential to controlling HIV transmission at the population level. Traditional prevention strategies, including condom use, behavioral change interventions, and blood safety programs have been complemented by biomedical innovations that have reshaped HIV prevention. Pre-exposure prophylaxis (PrEP) and post-exposure prophylaxis (PEP) are among the most effective biomedical prevention tools currently available [9]. PrEP involves the use of antiretroviral medication by HIV-negative individuals prior to potential exposure, while PEP entails a short course of antiretroviral drugs initiated after suspected or confirmed exposure, ideally within 72 hours, to prevent seroconversion [9–11]. Daily oral PrEP based on tenofovir disoproxil fumarate (TDF) combined with emtricitabine (FTC) or lamivudine (3TC) has been shown to reduce HIV acquisition by up to 99% when adherence is high [12]. Newer options, including long acting injectable cabotegravir (CAB-LA) and event driven PrEP for selected populations, further expand prevention choices and may mitigate adherence challenges [12]. Standard PEP regimens typically involve a 28 day course of combination therapy, including tenofovir, emtricitabine, and an integrase inhibitor such as raltegravir or dolutegravir [8, 13].

In recent years, PrEP implementation has expanded rapidly in sub-Saharan Africa. By the third quarter of 2022, approximately 76% of global PrEP initiations occurred in the region, reflecting substantial programmatic scale up [11]. Nevertheless, coverage remains uneven, and challenges related to awareness, uptake, and sustained adherence persist. A meta analysis reported an average willingness to use PrEP of 64.2% across African populations, with awareness levels varying widely [14, 15]. In contrast, PEP remains markedly underutilised in most sub-Saharan African countries, particularly for non-occupational exposures, due to limited awareness, restricted access, and logistical barriers [16, 17]. Modelling studies suggest that expanding community level access to PEP could yield substantial long-term reductions in HIV incidence [18]. These disparities highlight the need to better understand how biomedical interventions interact with behavior, adherence, and stage specific infectiousness.

Mathematical modelling provides a critical framework for examining HIV transmission dynamics and evaluating intervention strategies. Early models demonstrated that

heterogeneity in contact patterns and sexual activity levels plays a decisive role in shaping epidemic outcomes, pointing out the importance of population structure and mixing patterns [19, 20]. Subsequent stochastic models incorporated random effects, mobility, and uncertainty, thereby improving realism and capturing variability inherent in HIV transmission [21–25]. As prevention and treatment options expanded, modelling efforts increasingly integrated behavioral change and biomedical interventions. Studies examining condom use, behavioral modification, and ART demonstrated their potential to reduce transmission and prevalence, emphasizing prevention alongside treatment [26–29]. Time delay models further captured the effects of incubation periods, treatment initiation and delayed AIDS related deaths on epidemic persistence [30–32]. Rivu et al. [33] developed deterministic and stochastic HIV/AIDS models to evaluate progress toward the UNAIDS 95–95–95 targets. The study derived the basic reproduction number and examined the stability of disease-free and endemic equilibria, demonstrating that stochastic models provide a more realistic representation of uncertainty in HIV transmission and intervention outcomes. In a related study, Rivu et al. [34] extended this approach by developing gender-structured stochastic and deterministic HIV/AIDS models to investigate the effects of gender-specific transmission and treatment dynamics. Their findings further confirmed the superiority of stochastic models, particularly the stochastic Runge–Kutta method, in capturing random variations in disease progression and assessing the impact of transmission and treatment parameters on epidemic control. The study in [35] examined the combined effects of drug resistance, treatment saturation, and vertical transmission, showing that treatment saturation can induce backward bifurcation, thereby complicating disease elimination. Their optimal control analysis further demonstrated that integrated strategies; enhanced screening, early treatment initiation, and adherence support are more effective than single interventions in reducing infection burden. In a related study, [37] used mathematical simulation models across 77 high-burden countries and reported that achieving the WHO 2025 targets could reduce new HIV infections by up to 83% and AIDS-related deaths by 78% relative to 2010 levels. Furthermore, [36] developed a gender stratified HIV/AIDS model incorporating PrEP, PEP, diagnosis, and treatment, highlighting that viral load undetectability under the WHO 90-90-90 strategy plays a crucial role in reducing transmission, with notable differences between male and female subpopulations.

Despite these advances, significant gaps remain in the existing modelling literature. Many studies focus on single interventions rather than evaluating their combined effects within a unified framework. Assumptions of perfect adherence, uniform access, and constant coverage remain common, despite strong evidence of variations in uptake, discontinuation, and programmatic constraints, especially in sub-Saharan Africa. Moreover, few models explicitly incorporate adherence dynamics and relatively few are calibrated using contemporary, country specific data to inform national policy decisions.

These limitations are particularly relevant in the Ugandan context. In 2024, HIV prevalence among adults aged 15–49 years was estimated at 4.9%, with approximately 1.53 million people living with HIV, 60% of whom were women [38, 39]. Uganda recorded about 37,000 new infections and 20,000 AIDS-related deaths, despite ART coverage reaching approximately 1.3 million individuals; an estimated 200,000 people remained untreated [38, 39]. Although Uganda's national HIV response adopts a combination prevention strategy, including ART, PrEP, PEP, safe male circumcision, condom promotion,

and prevention of mother to child transmission, uptake and retention remain uneven across demographic and geographic groups [39, 40]. Access to PEP is particularly limited in rural areas due to low awareness, stockouts, and logistical barriers [39, 41]. External shocks such as the COVID-19 pandemic further disrupted HIV testing, treatment continuity, and prevention services [42].

In response to these challenges, this study develops and analyzes a deterministic mathematical model of HIV/AIDS transmission parameterized using demographic and epidemiological data from Uganda. The model integrates HIV disease progression stages with key biomedical interventions; ART, PrEP, and PEP while accounting for adherence influence. The framework is used to derive and analyse the effective reproduction number under alternative intervention scenarios, conduct sensitivity analysis to identify influential parameters, and simulate realistic intervention combinations to project long term impacts on HIV incidence and prevalence.

2 Model formulation

The model includes seven classes of individuals. The susceptible population, $S(t)$ is composed of individuals at risk of contracting HIV infection. The susceptible class increases by a constant recruitment rate, Λ . It is assumed that all individuals who join the population are susceptible, this is attributed to improved health care surveillance where all expectant mothers are subjected to strict antenatal care. The induced increment of susceptible population is due to individuals who have left PrEP at a rate ω and those who have survived an HIV infection after exposure due to PEP intake at a rate τ . The protected class, $P(t)$ comprises of individuals taking PrEP to prevent HIV infection due to their sexual behaviour. The exposed class $H_E(t)$ represents individuals who have experienced a potentially infectious HIV exposure and are in the post-exposure period during which PEP may be initiated. Individuals in this compartment are assumed to have received an infectious challenge, although infection has not yet progressed to the acute HIV stage. Successful completion of PEP prevents establishment of infection and returns individuals to the susceptible class, whereas non-adherence, incomplete adherence, or PEP failure results in progression to acute HIV infection at rate β . The acute class, $H_A(t)$ consists of individuals with acute HIV infection weeks after a successful exposure to the virus. These individuals progress to the chronic stage, $H_C(t)$ at a rate δ if they are not treated. The chronic stage progresses to AIDS class, $A(t)$ if no therapy is administered at a rate θ . Administering ART in classes $H_A(t)$, $H_C(t)$ and $A(t)$ at rates ϵ_1 , ϵ_2 and ϵ_3 respectively improves the health of infected individuals and they progress to the treated class, $T(t)$. The treated class, $T(t)$ includes individuals on ART whose adherence is sufficient to achieve an undetectable viral load. Individuals in $T(t)$ class who default on ART are assumed to experience a rapid deterioration of their immune system, ultimately progressing to the AIDS class. Detection of such defaults is difficult until the onset of AIDS symptoms. The total population is given as

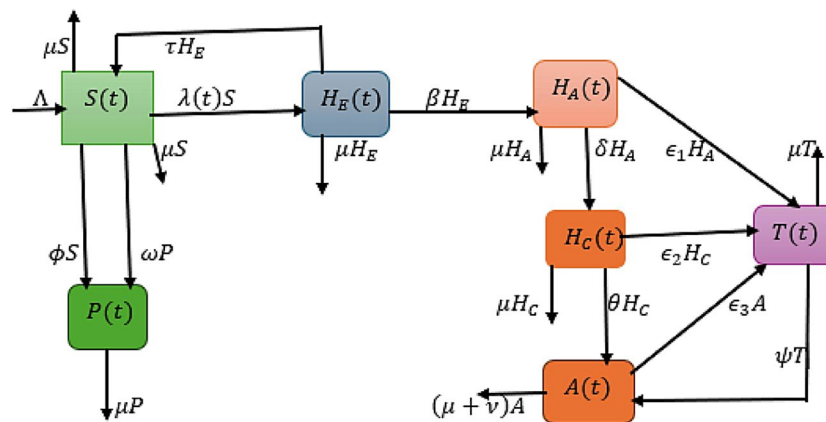
$$N(t) = S(t) + P(t) + H_E(t) + H_A(t) + H_C(t) + A(t) + T(t).$$

Table 1 below shows the parameters of the model and their description.

From compartmental diagram 1, the HIV/AIDS model equations are outlined below.

Table 1 Parameters used in the model formulation and their description

Parameter	Definition
Λ	Constant increase rate of the susceptible population
μ	Natural mortality rate across all subdivisions
τ	Rate of PEP intake to prevent HIV infection after exposure
ϕ	Rate of PrEP intake to prevent HIV infection before exposure
β	Progression rate to acute HIV infection after exposure
ω	Rate at which the protected individuals abandon PrEP to become susceptible to HIV
ϵ_1	ART intake rate of acute infected individuals
ϵ_2	ART intake rate of chronic infected individuals
ϵ_3	ART intake rate of AIDS patients
δ	Rate of progression from acute infection to chronic infection
θ	Rate of progression from chronic infection to AIDS stage
ψ	Rate at which individuals default ART
ν	AIDS induced mortality rate
$\lambda(t)$	HIV force of infection

**Fig. 1** A compartmental diagram for HIV/AIDS model

$$\begin{aligned}
 \frac{dS(t)}{dt} &= \Lambda + \tau H_E(t) + \omega P(t) - (\lambda(t) + \mu + \phi)S(t), \\
 \frac{dP(t)}{dt} &= \phi S(t) - (\omega + \mu)P(t), \\
 \frac{dH_E(t)}{dt} &= \lambda(t)S(t) - (\tau + \beta + \mu)H_E(t), \\
 \frac{dH_A(t)}{dt} &= \beta H_E(t) - (\delta + \epsilon_1 + \mu)H_A(t), \\
 \frac{dH_C(t)}{dt} &= \delta H_A(t) - (\theta + \epsilon_2 + \mu)H_C(t), \\
 \frac{dA(t)}{dt} &= \theta H_C(t) + \psi T(t) - (\nu + \epsilon_3 + \mu)A(t), \\
 \frac{dT(t)}{dt} &= \epsilon_1 H_A(t) + \epsilon_2 H_C(t) + \epsilon_3 A(t) - (\psi + \mu)T(t), \\
 \frac{dN(t)}{dt} &= \Lambda - \mu N(t) - \nu A(t).
 \end{aligned} \tag{1}$$

The rate at which an individual acquires HIV is defined as

$$\lambda(t) = k\xi\left(\frac{H_C(t) + aH_A(t) + bA(t)}{N}\right),$$

where k is the contact rate, ξ is the probability that a contact is efficient to cause an infection, a and b are amplification parameters implying increased infectivity in H_A and A classes compared to H_C class. HIV infectivity is highest during the acute stage up to 10 to 26 times greater than in the chronic phase, moderate and stable during the chronic stage, and increases again during the AIDS stage, though generally remaining lower than the acute phase [43, 44].

3 Analytical solution of the HIV/AIDS model

The theoretical analysis of the model aims to establish its feasibility, identify the HIV/AIDS equilibrium points, calculate the effective reproduction number, and assess the stability of the equilibria.

3.1 Basic properties of the model

The model's solutions are analyzed for positivity and boundedness to confirm the epidemiological relevance and stability of the system.

Lemma 3.1 The HIV/AIDS model solution $(S(t), P(t), H_E(t), H_A(t), H_C(t), A(t), T(t))$ uniquely exists and is positive for all $t \geq 0$ for a given initial population $(S_0, P_0, H_{E0}, H_{A0}, H_{C0}, A_0, T_0)$.

Proof From equation one of system (1)

$$\frac{dS(t)}{dt} + (\lambda(t) + \mu + \phi)S(t) = \Lambda + \tau H_E(t) + \omega P(t). \quad (2)$$

If $S(t_1)$ tends to zero in equation (2) for some time t_1 , where $0 < t < t_1$, such that

$$\frac{dS(t_1)}{dt} = \Lambda + \tau H_E(t_1) + \omega P(t_1) > 0. \quad (3)$$

This implies that $S(t)$ is positive. Applying the same technique we can show that all other state solutions are positive. $\square$

Lemma 3.2 The closed set $\Omega = \{(S(t), P(t), H_E(t), H_A(t), H_C(t), A(t), T(t)) \in \mathbb{R}_+^7 : N(t) \leq \frac{\Lambda}{\mu}\}$ is a biologically feasible region.

Proof From the eighth equation of system (1)

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - \nu A. \quad (4)$$

By Lemma 3.1, $A(t) \geq 0$, which leads to the inequality

$$\frac{dN(t)}{dt} + \mu N(t) \leq \Lambda. \quad (5)$$

With initial condition $N(0) = N_0$, integrate (5) to get

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N_0 - \frac{\Lambda}{\mu}\right)e^{-\mu t}. \quad (6)$$

As $t \rightarrow \infty$, we have $0 \leq N(t) \leq \frac{\Lambda}{\mu}$. The positive solution set is an invariant set of model (1) given by $\Omega = \{(S(t), P(t), H_E(t), H_A(t), H_C(t), A(t), T(t)) \in \mathbb{R}_+^7 : N(t) \leq \frac{\Lambda}{\mu}\}$. Thus, the solution of model (1) is bounded. $\square$

Hence, all state variables remain non-negative and bounded for all $t \geq 0$. Therefore, the model is mathematically and epidemiologically well posed, and its solutions exist uniquely and remain within the biologically feasible region Ω for all time.

3.2 Disease-free equilibrium point of the HIV/AIDS model

The HIV/AIDS-free equilibrium state is possible when the community is free of HIV infection, that is in absence of HIV infected individuals and AIDS patients. This could be due to maximum protection effort (PrEP) or due to complete absence of HIV pathogens in a community. To obtain HIV/AIDS-free state, equate system (1) to zero and solve;

$$\begin{aligned}\Lambda + \tau H_E(t) + \omega P(t) - (\lambda(t) + \mu + \phi)S(t) &= 0, \\ \phi S(t) - (\omega + \mu)P(t) &= 0, \\ \lambda(t)S(t) - (\tau + \beta + \mu)H_E(t) &= 0, \\ \beta H_E(t) - (\delta + \epsilon_1 + \mu)H_A(t) &= 0, \\ \delta H_A(t) - (\theta + \epsilon_2 + \mu)H_C(t) &= 0, \\ \theta H_C(t) + \psi T(t) - (\nu + \epsilon_3 + \mu)A(t) &= 0, \\ \epsilon_1 H_A(t) + \epsilon_2 H_C(t) + \epsilon_3 A(t) - (\psi + \mu)T(t) &= 0.\end{aligned}\quad (7)$$

In absence of infected individuals, that is, $H_A = H_C = A = 0$, we obtain $H_E = T = \lambda(t) = 0$, such that system (7) reduces to

$$\Lambda + \omega P(t) - (\mu + \phi)S(t) = 0, \quad (8)$$

$$\phi S(t) - (\omega + \mu)P(t) = 0. \quad (9)$$

Solving equations (8) and (9) simultaneously leads to $S = \frac{\Lambda(\omega + \mu)}{\mu(\omega + \mu + \phi)}$ and $P = \frac{\phi\Lambda}{\mu(\omega + \mu + \phi)}$.

Thus, the HIV/AIDS-free state is

$$\mathcal{H}_0 = \left(\frac{\Lambda(\omega + \mu)}{\mu(\omega + \mu + \phi)}, \frac{\phi\Lambda}{\mu(\omega + \mu + \phi)}, 0, 0, 0, 0, 0 \right). \quad (10)$$

3.2.1 Derivation of the HIV/AIDS effective reproduction number

In this context, the effective reproduction number, $\mathcal{R}_{\mathcal{H}}^*$ is the average number of secondary HIV infections caused by an acute HIV or chronic HIV or AIDS infectious individual during his or her entire period of infectiousness in presence of ART, PEP and PrEP interventions. We use the next-generation matrix method as applied in [29, 45, 46], to determine the effective reproduction number $\mathcal{R}_{\mathcal{H}}^*$ of system (1). Let $\mathcal{F}$ denote the matrix of HIV infection terms and $\mathcal{V}$ be the matrix of the remaining transfer terms from one compartment to the other in system (1). Now, define the HIV/AIDS dynamics with the following infected subsystem;

$$\begin{aligned}
\frac{dH_E(t)}{dt} &= \lambda(t)S(t) - (\tau + \beta + \mu)H_E(t), \\
\frac{dH_A(t)}{dt} &= \beta H_E(t) - (\delta + \epsilon_1 + \mu)H_A(t), \\
\frac{dH_C(t)}{dt} &= \delta H_A(t) - (\theta + \epsilon_2 + \mu)H_C(t), \\
\frac{dA(t)}{dt} &= \theta H_C(t) + \psi T(t) - (\nu + \epsilon_3 + \mu)A(t), \\
\frac{dT(t)}{dt} &= \epsilon_1 H_A(t) + \epsilon_2 H_C(t) + \epsilon_3 A(t) - (\psi + \mu)T(t).
\end{aligned} \quad (11)$$

The exposed class $H_E(t)$ is included in the infected subsystem because it represents individuals who have entered the infection progression process, even though some may eventually return to susceptibility through successful PEP intervention. From subsystem (11),

$$\mathcal{F} = \begin{bmatrix} \frac{k\xi(H_C + aH_A + bA)S}{N} \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix} (\tau + \beta + \mu)H_E \\ -\beta H_E + (\delta + \epsilon_1 + \mu)H_A \\ -\delta H_A + (\theta + \epsilon_2 + \mu)H_C \\ -\theta H_C - \psi T + (\nu + \epsilon_3 + \mu)A \\ -\epsilon_1 H_A(t) - \epsilon_2 H_C(t) - \epsilon_3 A(t) + (\psi + \mu)T(t) \end{bmatrix}. \quad (12)$$

Evaluating the Jacobian matrix of $\mathcal{F}$ at HIV/AIDS-free state, we obtain

$$F = \begin{bmatrix} 0 & k\xi a \frac{S^0}{N^0} & k\xi \frac{S^0}{N^0} & k\xi b \frac{S^0}{N^0} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}. \quad (13)$$

At HIV/AIDS-free state $N^0 = S^0 + P^0$, where $S^0 = \frac{\Lambda(\omega + \mu)}{\mu(\omega + \mu + \phi)}$, $P^0 = \frac{\phi\Lambda}{\mu(\omega + \mu + \phi)}$ and thus, the expression $\frac{S^0}{N^0}$ reduces to $\frac{\omega + \mu}{\omega + \mu + \phi}$. Jacobian (13) leads to

$$F = \begin{bmatrix} 0 & k\xi a \frac{(\omega + \mu)}{\omega + \mu + \phi} & k\xi \frac{(\omega + \mu)}{\omega + \mu + \phi} & k\xi b \frac{(\omega + \mu)}{\omega + \mu + \phi} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}. \quad (14)$$

In the same way the Jacobian matrix of $\mathcal{V}$ is evaluated at HIV/AIDS-free state to obtain

$$V = \begin{bmatrix} (\tau + \beta + \mu) & 0 & 0 & 0 & 0 \\ -\beta & (\delta + \epsilon_1 + \mu) & 0 & 0 & 0 \\ 0 & -\delta & (\theta + \epsilon_2 + \mu) & 0 & 0 \\ 0 & 0 & -\theta & (\nu + \epsilon_3 + \mu) & -\psi \\ 0 & -\epsilon_1 & -\epsilon_2 & -\epsilon_3 & (\psi + \mu) \end{bmatrix}. \quad (15)$$

The next generation matrix FV^{-1} is obtained from matrices (14) and (15) and used to give the characteristic polynomial

$$P(\lambda) = \lambda^4 \left(\lambda - \frac{k\xi\beta(\omega + \mu)(ak_3k_4(\psi + \mu) + k_4\delta(\psi + \mu) + b\mu\delta\theta + b\psi(\epsilon_1k_1 + \epsilon_2\delta + \theta\delta) - \psi\epsilon_3(ak_3 + \delta))}{(\omega + \phi + \mu)k_1k_2k_3(k_4(\psi + \mu) - \epsilon_3\psi)} \right), \quad (16)$$

where $k_1 = (\tau + \beta + \mu)$, $k_2 = (\delta + \epsilon_1 + \mu)$, $k_3 = (\theta + \epsilon_2 + \mu)$ and $k_4 = (\epsilon_3 + \nu + \mu)$. The leading eigenvalue of characteristic polynomial (16) gives the HIV/AIDS effective reproduction number

$$\mathcal{R}_{\mathcal{H}}^* = \mathcal{R}_A + \mathcal{R}_C + \mathcal{R}_D, \quad (17)$$

where

$$\begin{aligned} \mathcal{R}_A &= \frac{k\xi\beta(\omega + \mu)(ak_3k_4(\psi + \mu))}{(\omega + \phi + \mu)k_1k_2k_3(k_4(\psi + \mu) - \epsilon_3\psi)}, \\ \mathcal{R}_C &= \frac{k\xi\beta(\omega + \mu)(k_4\delta(\psi + \mu))}{(\omega + \phi + \mu)k_1k_2k_3(k_4(\psi + \mu) - \epsilon_3\psi)}, \\ \mathcal{R}_D &= \frac{k\xi\beta(\omega + \mu)(b\mu\delta\theta + b\psi(\epsilon_1k_3 + \epsilon_2\delta + \delta\theta) - \psi\epsilon_3(ak_3 + \delta))}{(\omega + \phi + \mu)k_1k_2k_3(k_4(\psi + \mu) - \epsilon_3\psi)}. \end{aligned}$$

$\mathcal{R}_A$ is the expected number of secondary infections generated by individuals in the acute infection stage, $\mathcal{R}_C$ is the contribution from individuals in the chronic infection stage, and $\mathcal{R}_D$ is the contribution from individuals in the AIDS stage, including the effects of treatment interruption.

With 100% ART adherence ($\psi = 0$), expression (17) reduces to

$$\mathcal{R}_{\mathcal{H}} = \frac{k\xi\beta(\omega + \mu)}{(\omega + \phi + \mu)k_1k_2k_3k_4}(ak_3k_4 + \delta k_4 + b\delta\theta). \quad (18)$$

The terms k_1 , k_2 , k_3 and k_4 represent the total exit rates from exposed, acute, chronic and AIDS stages respectively. The infection generating potential is represented by the term $k\xi\beta(\omega + \mu)$ whereas $(\omega + \phi + \mu)$ is the total rate out of susceptible or protected class. The weighted contributions to secondary infections from acute infections, transitions from acute to chronic and chronic to AIDS are ak_3k_4 , δk_4 and $b\delta\theta$ respectively. The relative importance of each parameter in $\mathcal{R}_{\mathcal{H}}^*$ will be examined through sensitivity analysis, as presented in Sect. 3.4.

In absence of PrEP ($\phi = 0, \omega = 0$), (18) simplifies to

$$\mathcal{R}_{\mathcal{H}1} = \frac{k\xi\beta}{k_1k_2k_3k_4}(ak_3k_4 + \delta k_4 + b\delta\theta), \quad (19)$$

representing the reproduction potential when only PEP and ART are implemented.

Proposition 3.3 For any positive parameters, $\mathcal{R}_{\mathcal{H}} < \mathcal{R}_{\mathcal{H}1}$.

Proof Since $0 < \frac{\omega + \mu}{\omega + \phi + \mu} < 1$. It follows that,

$$\mathcal{R}_{\mathcal{H}} = \frac{\omega + \mu}{\omega + \phi + \mu} \mathcal{R}_{\mathcal{H}1} < \mathcal{R}_{\mathcal{H}1}.$$

□

When PEP is absent ($\tau = 0$), (18) reduces to

$$\mathcal{R}_{\mathcal{H}2} = \frac{k\xi\beta(\omega + \mu)}{(\omega + \phi + \mu)k_1^*k_2k_3k_4}(ak_3k_4 + \delta k_4 + b\delta\theta) \quad (20)$$

with $k_1^* = (\beta + \mu)$, representing the reproduction threshold under PrEP and ART interventions only. If both PrEP and PEP are absent, the threshold further reduces to

$$\mathcal{R}_{\mathcal{H}_0} = \frac{k\xi\beta}{k_1^*k_2k_3k_4}(ak_3k_4 + \delta k_4 + b\delta\theta), \quad (21)$$

representing the baseline reproduction number under ART alone.

Proposition 3.4 For any positive parameters, $\mathcal{R}_{\mathcal{H}_2} < \mathcal{R}_{\mathcal{H}_0}$.

Proof Since $0 < \frac{\omega+\mu}{\omega+\phi+\mu} < 1$. It follows that,

$$\mathcal{R}_{\mathcal{H}_2} = \frac{\omega+\mu}{\omega+\phi+\mu}\mathcal{R}_{\mathcal{H}_0} < \mathcal{R}_{\mathcal{H}_0}.$$

□

By propositions 3.3 and 3.4, it follows that;

$$\mathcal{R}_{\mathcal{H}} < \mathcal{R}_{\mathcal{H}_0}.$$

This ordering demonstrates that the simultaneous implementation of PEP, PrEP, and ART achieves the greatest reduction in HIV transmission potential. The absence of either prevention strategy leads to a higher reproduction number, while the absence of both results in the maximum transmission potential for HIV infection.

3.2.2 Local and global stability of HIV/AIDS-free equilibrium point

Lemma 3.5 The HIV/AIDS-free equilibrium $\mathcal{H}_0$ is locally asymptotically stable if $\mathcal{R}_{\mathcal{H}}^* < 1$ and unstable otherwise.

Proof To prove that $\mathcal{H}_0$ is locally asymptotically stable, it is prudent to identify the relevant subsystem, that consists of the infected compartments; $x = (H_E, H_A, H_C, A, T)^T$. The subsystem is

$$\frac{dx}{dt} = Jx, \quad (22)$$

where J is the Jacobian evaluated at $\mathcal{H}_0$ to give

$$J_{\mathcal{H}_0} = \begin{bmatrix} -k_1 & k\xi a \frac{(\omega+\mu)}{(\omega+\mu+\phi)} & k\xi \frac{(\omega+\mu)}{(\omega+\mu+\phi)} & k\xi b \frac{(\omega+\mu)}{(\omega+\mu+\phi)} & 0 \\ \beta & -k_2 & 0 & 0 & 0 \\ 0 & \delta & -k_3 & 0 & 0 \\ 0 & 0 & \theta & -k_4 & \psi \\ 0 & \epsilon_1 & \epsilon_2 & \epsilon_3 & -(\psi + \mu) \end{bmatrix}. \quad (23)$$

We decompose $J_{\mathcal{H}_0}$ as

$$J_{\mathcal{H}_0} = F - V, \quad (24)$$

where F and V are defined in equations (14) and (15) respectively.

It is clear that $F \geq 0$. Now, observe that all off-diagonal entries of V are non-positive. Hence V is a Z -matrix and its leading principal minors are $D_1 = k_1 > 0$, $D_2 = k_1k_2 > 0$, $D_3 = k_1k_2k_3 > 0$ and $D_4 = k_1k_2k_3k_4 > 0$. Thus,

$$\det(V) = k_1k_2k_3(k_4(\psi + \mu) - \epsilon_3\psi) = k_1k_2k_3(k_4\mu + \psi(\nu + \mu)) > 0. \quad (25)$$

Therefore, all principal minors of V are positive, and hence V is a nonsingular M -matrix implying that $V^{-1} \geq 0$. Since $F \geq 0$ and $V^{-1} \geq 0$, it follows that $FV^{-1} \geq 0$ and the associated reproduction number is given by $\mathcal{R}_{\mathcal{H}}^* = \rho(FV^{-1})$, where $\rho(\cdot)$ denotes the spectral radius.

By Theorem 2 of [46], for a matrix of the form $F - V$ with $F \geq 0$ and V a nonsingular M -matrix,

$$s(F - V) < 0 \iff \rho(FV^{-1}) < 1, \quad (26)$$

where $s(F - V) = \max\{\Re(\lambda) : \lambda \in \sigma(F - V)\}$. Since $J_{\mathcal{H}_0} = F - V$, (26) leads to

$$s(J_{\mathcal{H}_0}) < 0 \iff \mathcal{R}_{\mathcal{H}}^* < 1.$$

Hence, all eigenvalues of $J_{\mathcal{H}_0}$ have negative real parts whenever $\mathcal{R}_{\mathcal{H}}^* < 1$, and therefore $\mathcal{H}_0$ is locally asymptotically stable. $\square$

Theorem 1 The HIV/AIDS-free equilibrium point $\mathcal{H}_0$ is globally asymptotically stable if $\mathcal{R}_{\mathcal{H}}^* < 1$ and unstable otherwise.

Proof Following the method of Castillo-Chavez et al. [48], system (1) is partitioned into the uninfected and infected subsystems. Let $X = (S, P)$ represent the uninfected compartments and $Z = (H_E, H_A, H_C, A, T)$ represent the infected compartments. The uninfected subsystem is given by

$$\begin{aligned} \frac{dS(t)}{dt} &= \Lambda + \tau H_E(t) + \omega P(t) - (\lambda(t) + \mu + \phi)S(t), \\ \frac{dP(t)}{dt} &= \phi S(t) - (\omega + \mu)P(t), \end{aligned} \quad (27)$$

which can be written as $\frac{dX}{dt} = F(X, Z)$. The infected subsystem is

$$\begin{aligned} \frac{dH_E(t)}{dt} &= \lambda(t)S(t) - k_1 H_E(t), \\ \frac{dH_A(t)}{dt} &= \beta H_E(t) - k_2 H_A(t), \\ \frac{dH_C(t)}{dt} &= \delta H_A(t) - k_3 H_C(t), \\ \frac{dA(t)}{dt} &= \theta H_C(t) + \psi T(t) - k_4 A(t), \\ \frac{dT(t)}{dt} &= \epsilon_1 H_A(t) + \epsilon_2 H_C(t) + \epsilon_3 A(t) - (\psi + \mu)T(t), \end{aligned} \quad (28)$$

denoted by $\frac{dZ}{dt} = G(X, Z)$. For $Z = 0$, subsystem (27) reduces to

$$F(X, 0) = \begin{bmatrix} \Lambda + \omega P - (\mu + \phi)S \\ \phi S - (\omega + \mu)P \end{bmatrix} \quad (29)$$

Solving subsystem (29) yields the unique equilibrium $X^0 = (S^0, P^0)$, where $S^0 = \frac{\Lambda(\omega + \mu)}{\mu(\omega + \mu + \phi)}$ and $P^0 = \frac{\Lambda\phi}{\mu(\omega + \mu + \phi)}$. Standard linear stability analysis shows that all eigenvalues of Jacobian (29) have negative real parts. Therefore X^0 is globally asymptotically stable which satisfies the first condition (H1) of Castillo-Chavez method [48].

Next we verify condition two. The infected subsystem can be written as

$$G(X, Z) = AZ - \hat{G}(X, Z), \quad (30)$$

where $A = D_Z G(X^0, 0)$. Since $\frac{S^0}{N^0} = \frac{\omega + \mu}{\omega + \mu + \phi}$, with $N^0 = \frac{\Lambda}{\mu}$, it follows that

$$A = \begin{bmatrix} -k_1 & k\xi a \frac{(\omega + \mu)}{(\omega + \mu + \phi)} & k\xi \frac{(\omega + \mu)}{(\omega + \mu + \phi)} & k\xi b \frac{(\omega + \mu)}{(\omega + \mu + \phi)} & 0 \\ \beta & -k_2 & 0 & 0 & 0 \\ 0 & \delta & -k_3 & 0 & 0 \\ 0 & 0 & \theta & -k_4 & \psi \\ 0 & \epsilon_1 & \epsilon_2 & \epsilon_3 & -(\psi + \mu) \end{bmatrix}. \quad (31)$$

Since all off-diagonal entries of A are non-negative, A is a Metzler matrix. Furthermore,

$$\hat{G}(X, Z) = AZ - G(X, Z) = \begin{bmatrix} k\xi \left(\frac{\omega + \mu}{\omega + \mu + \phi} - \frac{S}{N} \right) (aH_A + H_C + bA) \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}.$$

Since the feasible region Ω is positively invariant and $S \leq S^0$, we obtain $\frac{S}{N} \leq \frac{S^0}{N^0} = \frac{\omega + \mu}{\omega + \mu + \phi}$. Hence $\hat{G}(X, Z) \geq 0$, for all $(X, Z) \in \Omega$. Therefore, condition (H2) of [48] is satisfied. Since conditions (H1) and (H2) hold, and $\mathcal{R}_{\mathcal{H}}^* < 1$, it follows from Theorem 4.1 [48] that HIV/AIDS-free equilibrium $\mathcal{H}_0$ is globally asymptotically stable in Ω . $\square$

3.3 Existence of the HIV/AIDS endemic state

The endemic state of HIV/AIDS corresponds to a situation where the disease remains continuously present in the population. Refer to system (7) together with $\frac{dN}{dt} = 0$ and solve to obtain the endemic equilibrium point $\mathcal{H}_e = (S^*, P^*, H_E^*, H_A^*, H_C^*, A^*, T^*)$, where

$$\begin{aligned} S^* &= \frac{\phi(\omega + \mu) \left(\Lambda \beta \delta \nu (k_3 \epsilon_1 + (\epsilon_2 + \theta(\psi + \mu))\delta) + \tau \delta (\Lambda - \mu N^*) (\epsilon_3 + (\psi + \mu)k_4) k_2 k_3 \right)}{\phi \beta \delta \nu ((\lambda + \mu + \phi)(\omega + \mu) - \omega) (k_3 \epsilon_1 + (\epsilon_2 + \theta(\psi + \mu))\delta)}, \\ P^* &= \frac{\phi \left(\Lambda \beta \delta \nu (k_3 \epsilon_1 + (\epsilon_2 + \theta(\psi + \mu))\delta) + \tau \delta (\Lambda - \mu N^*) (\epsilon_3 + (\psi + \mu)k_3) k_2 k_3 \right)}{\beta \delta \nu ((\lambda + \mu + \phi)(\omega + \mu) - \omega) (k_3 \epsilon_1 + (\epsilon_2 + \theta(\psi + \mu))\delta)}, \\ H_E^* &= \frac{\delta (\Lambda - \mu N^*) (\epsilon_3 + (\psi + \mu)k_4) k_2 k_3}{\beta \delta \nu (k_3 \epsilon_1 + (\epsilon_2 + \theta(\psi + \mu))\delta)}, H_A^* = \frac{\delta (\Lambda - \mu N^*) (\epsilon_3 + (\psi + \mu)k_4) k_2}{\delta \nu (k_3 \epsilon_1 + (\epsilon_2 + \theta(\psi + \mu))\delta)}, \\ H_C^* &= \frac{\delta (\Lambda - \mu N^*) (\epsilon_3 + (\psi + \mu)k_4)}{\nu (k_3 \epsilon_1 + (\epsilon_2 + \theta(\psi + \mu))\delta)}, A^* = \frac{\Lambda - \mu N^*}{\nu}, \\ T^* &= \frac{k_4 (\Lambda - \mu N^*)}{\nu} - \frac{\theta \delta (\Lambda - \mu N^*) (\epsilon_3 + k_4(\psi + \mu))}{\nu (k_3 \epsilon_1 + (\epsilon_2 + \theta(\psi + \mu))\delta)}, \end{aligned}$$

where

$$\begin{aligned} N^* &= \frac{\eta \Lambda \beta (\omega + \mu) \mathcal{C}}{\eta \mu \beta (\omega + \mu) \mathcal{C} + \nu (k_1 k_2 k_3 (\omega + \mu + \phi) - \nu \beta \eta (\omega + \mu) \mathcal{C})} \text{ and} \\ \mathcal{C} &= \frac{b(k_3 \epsilon_1 + \delta(\epsilon_2 + \theta(\psi + \mu))) + (a + \delta)(\epsilon_3 + (\psi + \mu)k_4)}{k_3 \epsilon_1 + \delta(\epsilon_2 + (\psi + \mu))\theta}. \end{aligned} \quad (32)$$

3.3.1 Local bifurcation analysis at $\mathcal{R}_{\mathcal{H}}^* = 1$

In this sub-section, we investigate the qualitative behavior of system (1) near the HIV/AIDS-free equilibrium and establish the existence of a bifurcation when the effective reproduction number crosses unity. Particularly, the center manifold theory [35, 47, 49, 50] is applied to determine the type of bifurcation exhibited by the model. Let the critical bifurcation value occur at $\eta = k\xi = \eta^*$ such that $\mathcal{R}_{\mathcal{H}}^* = 1$, then

$$\eta^* = \frac{(\omega + \phi + \mu)k_1k_2k_3(k_4(\psi + \mu) - \epsilon_3\psi)}{\beta(\omega + \mu)(ak_3k_4(\psi + \mu) + k_4\delta(\psi + \mu) + b\mu\delta\theta + b\psi(\epsilon_1k_1 + \epsilon_2\delta + \theta\delta) - \psi\epsilon_3(ak_3 + \delta))}.$$

The Jacobian matrix of system (1) evaluated at the HIV/AIDS-free equilibrium point $\mathcal{H}_0$ is given as

$$J_{\mathcal{H}_0} = \begin{bmatrix} -(\mu + \phi) & \omega & \tau & -\frac{\eta a(\omega + \mu)}{(\omega + \mu + \phi)} & -\frac{\eta(\omega + \mu)}{(\omega + \mu + \phi)} & -\frac{\eta b(\omega + \mu)}{(\omega + \mu + \phi)} & 0 \\ \phi & -(\omega + \mu) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -k_1 & \frac{\eta a(\omega + \mu)}{(\omega + \mu + \phi)} & \frac{\eta(\omega + \mu)}{(\omega + \mu + \phi)} & \frac{\eta b(\omega + \mu)}{(\omega + \mu + \phi)} & 0 \\ 0 & 0 & \beta & -k_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta & -k_3 & 0 & 0 \\ 0 & 0 & 0 & 0 & \theta & -k_4 & \psi \\ 0 & 0 & 0 & \epsilon_1 & \epsilon_2 & \epsilon_3 & -(\psi + \mu) \end{bmatrix} \quad (33)$$

with $k_1 = \tau + \beta + \mu$, $k_2 = \delta + \epsilon_1 + \mu$, $k_3 = \theta + \epsilon_2 + \mu$, $k_4 = \nu + \epsilon_3 + \mu$. The Jacobian (33) is block lower triangular with the uninfected block

$$J_1 = \begin{pmatrix} -(\mu + \phi) & \omega \\ \phi & -(\omega + \mu) \end{pmatrix},$$

whose eigenvalues have strictly negative real parts since $\text{tr}(J_1) = -(\phi + \omega + 2\mu) < 0$ and $\det(J_1) = \mu(\omega + \mu + \phi) > 0$.

The remaining eigenvalues are those of the infected block

$$J_2 = \begin{pmatrix} -k_1 & \frac{\eta a(\omega + \mu)}{(\omega + \mu + \phi)} & \frac{\eta(\omega + \mu)}{(\omega + \mu + \phi)} & \frac{\eta b(\omega + \mu)}{(\omega + \mu + \phi)} & 0 \\ \beta & -k_2 & 0 & 0 & 0 \\ 0 & \delta & -k_3 & 0 & 0 \\ 0 & 0 & \theta & -k_4 & \psi \\ 0 & \epsilon_1 & \epsilon_2 & \epsilon_3 & -(\psi + \mu) \end{pmatrix}. \quad (34)$$

Let $\mathcal{R}_{\mathcal{H}}^*$ be given by (17). The characteristic polynomial corresponding to Jacobian (34) evaluated at η^* is given as

$$P(\lambda) = \lambda(\lambda^4 + A_1\lambda^3 + A_2\lambda^2 + A_3\lambda + A_4), \quad (35)$$

where

$$A_1 = k_1 + k_2 + k_3 + k_4 + \psi + \mu,$$

$$A_2 = k_1(k_2 + k_3 + k_4) + k_2(k_3 + k_4) + k_3k_4 + (\psi + \mu)(k_1 + k_2 + k_3 + k_4) - \psi\epsilon_3,$$

$$A_3 = k_1k_2(k_3 + k_4) + k_3k_4(k_1 + k_2) + (\psi + \mu)(k_1(k_2 + k_3 + k_4) + k_2(k_3 + k_4) + k_3k_4) - \psi\epsilon_3(k_1 + k_2 + k_3) - \frac{k_1k_2k_3k_4M}{K}((a+1)(k_4 + \psi + \mu) + b(k_3 + \psi + \mu)),$$

$$A_4 = k_1k_2k_3(k_4(\psi + \mu) - \psi\epsilon_3 - \frac{k_4M}{K}((\psi + \mu)((a+1)k_4 + bk_3) - \psi((a+1)\epsilon_3 + b\epsilon_2) + b\theta\epsilon_1)),$$

$$M = k_4(\psi + \mu) - \epsilon_3\psi,$$

$$K = k_4\mu\delta + ak_3k_4\mu + b\mu\delta\theta + \psi(k_4\delta + b\epsilon_1k_3 + ak_3k_4 + b\epsilon_2\delta + b\delta\theta - a\epsilon_3k_3 - \epsilon_3\delta).$$

At $\mathcal{R}_H^* = 1$, the characteristic polynomial (35) possesses a simple zero eigenvalue. The remaining eigenvalues have negative real parts if and only if the associated quartic polynomial satisfies the Routh–Hurwitz stability criteria. Specifically, this occurs when $A_1 > 0$, $A_2 > 0$, $A_3 > 0$, $A_4 > 0$, $A_1 A_2 > A_3$, and $A_1 A_2 A_3 > A_3^2 + A_1^2 A_4$.

Under these conditions, all non-zero eigenvalues lie strictly in the left half of the complex plane, ensuring local asymptotic stability of the system on the center manifold. The next step is to compute the Center manifold coefficients. Let v be the right eigenvector associated with the simple zero eigenvalue of the Jacobian (33), then

$$\begin{aligned} v_7 &= \frac{\beta(k_4(\epsilon_1 k_3 + \epsilon_2 \delta) + \epsilon_3 \theta \delta)}{k_2 k_3(k_4(\psi + \mu) - \epsilon_3 \psi)} v_3, \\ v_6 &= \frac{\beta(\theta \delta(\psi + \mu) + \psi(\epsilon_1 k_3 + \delta \epsilon_2))}{k_2 k_3(k_4(\psi + \mu) - \epsilon_3 \psi)} v_3, \quad v_5 = \frac{\delta \beta}{k_2 k_3} v_3, \quad v_4 = \frac{\beta}{k_2} v_3, \quad v_3 = v_3, \\ v_2 &= \frac{\phi}{\omega + \mu} v_1, \quad v_1 = \frac{\omega + \mu}{\mu(\omega + \mu + \phi)} \left(\tau + \frac{\eta^* \beta(\omega + \mu)}{\omega + \mu + \phi} \left(\frac{a}{k_2} + \frac{\delta}{k_2 k_3} \right. \right. \\ &\quad \left. \left. + \frac{\theta \delta(\psi + \mu) + \psi(\epsilon_1 k_3 + \delta \epsilon_2)}{k_2 k_3(k_4(\psi + \mu) - \epsilon_3 \psi)} \right) \right) v_3. \end{aligned}$$

An associated left eigenvector w is chosen as such that

$$\begin{aligned} w_1 &= w_2 = 0, \quad w_3 = w_3, \quad w_4 = \frac{k_1}{\beta} w_3, \\ w_5 &= \left(\frac{k_1}{\beta} - \frac{\eta^* a(\omega + \mu)}{\omega + \mu + \phi} - \frac{(\eta^* b(\psi + \mu)(\omega + \mu))}{(\omega + \mu + \phi)(k_4(\psi + \mu) - \epsilon_3 \psi)} \right) w_3, \\ w_6 &= \frac{\eta^* b(\psi + \mu)(\omega + \mu)}{(\omega + \mu + \phi)(k_4(\psi + \mu) - \epsilon_3 \psi)} w_3, \quad w_7 = \frac{\psi \eta^* b(\omega + \mu)}{(\omega + \mu + \phi)(k_4(\psi + \mu) - \epsilon_3 \psi)} w_3. \end{aligned}$$

The normalisation condition $w^T v = 1$ gives the normalisation constant as

$$w = \frac{1}{\mathcal{N}},$$

where

$$\begin{aligned} \mathcal{N} &= 1 + \frac{\beta}{k_2} \mathcal{N}_1 + \frac{\delta \beta}{k_2 k_3} \mathcal{N}_2 + \frac{\beta(\theta \delta(\psi + \mu) + \psi(\epsilon_1 k_3 + \epsilon_2 \delta))}{k_2 k_3 M} \mathcal{N}_3 \\ &\quad + \frac{\beta(k_4(\epsilon_1 k_3 + \epsilon_2 \delta) + \epsilon_3 \theta \delta)}{k_2 k_3 M} \mathcal{N}_4, \\ \mathcal{N}_1 &= \frac{1}{k_2} \left(\frac{\eta^* a(\omega + \mu)}{\omega + \mu + \phi} + \delta \mathcal{N}_2 + \frac{\epsilon_1 \psi}{\psi + \mu} \mathcal{N}_3 \right), \\ \mathcal{N}_2 &= \frac{\eta^*(\omega + \mu)}{k_3(\omega + \mu + \phi)} \left(1 + \frac{b(\theta(\psi + \mu) + \epsilon_2 \psi)}{M} \right), \\ \mathcal{N}_3 &= \frac{\eta^* b(\omega + \mu)(\psi + \mu)}{(\omega + \mu + \phi) M}, \quad \mathcal{N}_4 = \frac{\psi}{\psi + \mu} \mathcal{N}_3. \end{aligned}$$

Following the approach of Castillo-Chavez and Song [49], the bifurcation constants are defined as

$$m_1 = \sum_{k,i,j} v_k w_i w_j \frac{\partial^2 h_k}{\partial x_i \partial x_j}(\mathcal{H}_0, \eta^*), \quad m_2 = \sum_{k,i} v_k w_i \frac{\partial^2 h_k}{\partial x_i \partial \rho}(\mathcal{H}_0, \eta^*), \quad (36)$$

where h_k denotes the k^{th} component of the vector field. The only nonlinear term in the system is the incidence function $h_3(x, \eta) = \eta \frac{(H_C + aH_A + bA)S}{N}$. At HIV/AIDS-free equilibrium point, we obtain the following expressions of the second derivatives;

$$\begin{aligned} \frac{\partial^2 h_3}{\partial S \partial H_A} &= \frac{\eta a \mu \phi}{\Lambda(\omega + \mu + \phi)}, \quad \frac{\partial^2 h_3}{\partial S \partial H_C} = \frac{\eta \mu \phi}{\Lambda(\omega + \mu + \phi)}, \quad \frac{\partial^2 h_3}{\partial S \partial A} = \frac{\eta b \mu \phi}{\Lambda(\omega + \mu + \phi)}, \\ \frac{\partial^2 h_3}{\partial H_A^2} &= \frac{-2\eta a^2 \mu(\omega + \mu)}{\Lambda(\omega + \mu + \phi)}, \quad \frac{\partial^2 h_3}{\partial H_C^2} = \frac{-2\eta \mu(\omega + \mu)}{\Lambda(\omega + \mu + \phi)}, \quad \frac{\partial^2 h_3}{\partial A^2} = \frac{-2\eta b^2 \mu(\omega + \mu)}{\Lambda(\omega + \mu + \phi)}, \\ \frac{\partial^2 h_3}{\partial H_A \partial H_C} &= \frac{-\eta \mu(a+1)(\omega + \mu)}{\Lambda(\omega + \mu + \phi)}, \quad \frac{\partial^2 h_3}{\partial H_A \partial A} = \frac{-\eta \mu b(a+1)(\omega + \mu)}{\Lambda(\omega + \mu + \phi)}, \quad \frac{\partial^2 h_3}{\partial H_C \partial A} \\ &= \frac{2b\eta \mu(\omega + \mu)}{\Lambda(\omega + \mu + \phi)}, \\ \frac{\partial^2 h_3}{\partial H_A \partial \eta} &= \frac{a(\omega + \mu)}{\omega + \mu + \phi}, \quad \frac{\partial^2 h_3}{\partial H_C \partial \eta} = \frac{\omega + \mu}{\omega + \mu + \phi}, \quad \frac{\partial^2 h_3}{\partial A \partial \eta} = \frac{b(\omega + \mu)}{\omega + \mu + \phi}. \end{aligned} \quad (37)$$

The Center manifold coefficients in (36) are obtained as

$$\begin{aligned} m_1 &= \frac{2\eta \mu}{\Lambda(\omega + \mu + \phi)} \left(\phi v_1 (av_4 + v_5 + bv_6) - (\omega + \mu) \mathcal{A} \right), \\ m_2 &= \frac{\omega + \mu}{\omega + \mu + \phi} (av_4 + v_5 + bv_6), \end{aligned} \quad (38)$$

where $\mathcal{A} = a^2 v_4^2 + v_5^2 + b^2 v_6^2 + (a+1)v_6 v_5 + b(a+1)v_4 v_6 + 2bv_5 v_6$. Since $w_3 > 0$, $v_4 > 0$, $v_5 > 0$, and $v_6 > 0$, it follows that $m_2 > 0$. The sign of m_1 is determined by the expression $\phi v_1 (av_4 + v_5 + bv_6) - (\omega + \mu) \mathcal{A}$. Consequently,

$$m_1 > 0 \quad \text{if} \quad \phi v_1 (av_4 + v_5 + bv_6) > (\omega + \mu) \mathcal{A},$$

whereas

$$m_1 < 0 \quad \text{if} \quad \phi v_1 (av_4 + v_5 + bv_6) < (\omega + \mu) \mathcal{A}.$$

Since $m_2 > 0$, the bifurcation behavior is completely determined by the sign of m_1 . By the Center Manifold Theorem of Castillo-Chavez and Song [49],

- (a) if $m_1 > 0$, then the model undergoes a backward (subcritical) transcritical bifurcation at $\mathcal{R}_H^* = 1$, and a stable endemic equilibrium may coexist with the disease-free equilibrium for some values of $0 < \mathcal{R}_H^* < 1$;
- (b) if $m_1 < 0$, then the model undergoes a forward (supercritical) transcritical bifurcation at $\mathcal{R}_H^* = 1$, and the endemic equilibrium exists only when $\mathcal{R}_H^* > 1$.

Thus, the following theorem holds.

Theorem 2 Suppose that all parameters of the model are positive such that $m_1 > 0$ and $m_2 > 0$. Then the HIV/AIDS model undergoes a backward transcritical bifurcation at $\mathcal{R}_H^* = 1$. Consequently, there exists a range of values $0 < \mathcal{R}_H^* < 1$ for which both the HIV/AIDS-free equilibrium and an endemic equilibrium coexist.

In this case, even when the effective reproduction number satisfies $\mathcal{R}_H^* < 1$, HIV/AIDS may not be eliminated from the population, as illustrated in Fig. 2. This

phenomenon arises due to the presence of backward bifurcation, where a stable endemic equilibrium coexists with the disease-free equilibrium when the reproduction number is below unity. Consequently, reducing the threshold parameter below one is not sufficient to guarantee disease eradication, which may partly explain the continued persistence and spread of HIV at the national level despite the effective reproduction number being less than one. The bifurcation Fig. 2 was generated using the parameter values listed in Table 3. For each value of the bifurcation parameter η , the corresponding reproduction number $\mathcal{R}_H^*$ was computed and the equilibrium solutions of the model were determined numerically. The endemic equilibrium branches were obtained by numerically solving the steady-state equations of system (1) using MATLAB.

The occurrence of backward bifurcation indicates that high rates of ART default relative to treatment initiation among AIDS patients can undermine disease control efforts. Consequently, HIV may persist in the population even when the reproduction number is below unity, owing to the existence of a residual pool of infectious individuals maintained through treatment interruption.

Consider the case of strict adherence to ART, that is, $\psi = 0$. Under this assumption, the effective reproduction number becomes

$$\mathcal{R}_H = \frac{\eta\beta(\omega + \mu)}{(\omega + \phi + \mu)k_1k_2k_3k_4}(\delta k_4 + ak_3k_4 + b\delta\theta).$$

At $\mathcal{R}_H = 1$, the bifurcation value is $\eta^* = \frac{(\omega + \phi + \mu)k_1k_2k_3k_4}{\beta(\omega + \mu)(ak_4 + \delta k_3k_4 + b\delta\theta)}$. The center manifold coefficients are computed and obtained as;

$$\begin{aligned} m_1 &= \frac{-2\eta(\omega + \mu)w_3}{\Lambda(\omega + \mu + \phi)} \left(\frac{\phi(\omega + \mu)}{\omega + \mu + \phi} (av_4 + v_5 + bv_6) + \mu\mathcal{B} \right), \\ m_2 &= \frac{\omega + \mu}{\omega + \mu + \phi} (av_4 + v_5 + bv_6)w_3, \end{aligned} \quad (39)$$

where

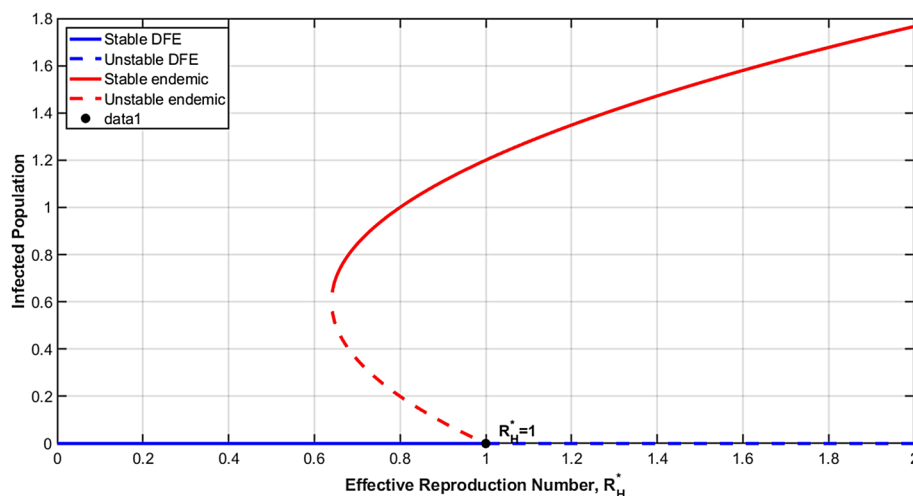


Fig. 2 Backward bifurcation in HIV/AIDS Model

$$v_4 = \frac{\beta}{k_2}v_3, \quad v_5 = \frac{\delta\beta}{k_2k_3}v_3, \quad v_6 = \frac{\theta\delta\beta}{k_2k_3k_4}v_3,$$

$$\mathcal{B} = \left(\frac{a\beta}{k_2}\right)^2 + \left(\frac{\delta\beta}{k_2k_3}\right)^2 + \left(\frac{b\theta\delta\beta}{k_2k_3k_4}\right)^2 + \frac{(a+1)\delta\beta^2}{k_2^2k_3} + \frac{b(a+1)\theta\delta\beta^2}{k_2^2k_3k_4} + \frac{2b\theta\delta^2\beta^2}{k_2^2k_3^2k_4}.$$

It is evident from (39) that $m_2 > 0$ and $m_1 < 0$, since all model parameters are positive. This implies that the system undergoes a *forward transcritical bifurcation* at $\mathcal{R}_H = 1$ for $\psi = 0$.

Theorem 3 Suppose $\psi = 0$. Then, the HIV/AIDS model undergoes a forward transcritical bifurcation at $\mathcal{R}_H = 1$. In particular, when $\mathcal{R}_H > 1$, the HIV/AIDS-free equilibrium loses stability and a unique endemic equilibrium emerges. Moreover, this endemic equilibrium is locally asymptotically stable for $\mathcal{R}_H$ sufficiently close to unity.

3.4 Sensitivity analysis

Sensitivity analysis provides a systematic way for identifying the key parameters that significantly influence HIV transmission dynamics and control. By assessing the relative contributions of these parameters, it facilitates the identification of effective strategies for mitigating HIV transmission, as well as reducing associated morbidity and mortality. In this study, the methodology applied in [36, 50, 51] is adopted. Specifically, the sensitivity index of the effective reproduction number, $\mathcal{R}_H^*$, with respect to a model parameter $\mathcal{P}$ is defined as

$$S_{\mathcal{P}}^{\mathcal{R}_H^*} = \frac{\partial \mathcal{R}_H^*}{\partial \mathcal{P}} \times \frac{\mathcal{P}}{\mathcal{R}_H^*}. \quad (40)$$

Using expression (40) together with the parameter values listed in Table 3, the corresponding sensitivity indices are computed and presented in Table 2.

The sensitivity indices in Table 2 identify the parameters that most strongly influence the effective reproduction number $\mathcal{R}_H^*$. The parameters k and ξ exhibit sensitivity indices of +1, indicating that they are the most influential drivers of HIV transmission. A proportional increase in either parameter leads to an equivalent increase in $\mathcal{R}_H^*$, pointing out the importance of interventions that reduce their associated transmission pathways.

It is also revealed that the parameter ψ has a strongly positive influence on the model outcome, implying that higher rates of ART defaulting are associated with an increase in new HIV infections. This emphasizes the critical role of sustained adherence to ART in reducing HIV transmission. Moderate positive sensitivity indices are observed for parameters a , b , and θ , implying that increases in these parameters contribute to higher

Table 2 Numerical values of sensitivity indices of $\mathcal{R}_H^*$

Parameter	Sensitivity index	Parameter	Sensitivity index
k	1.0000	ϵ_1	-0.0155
ξ	1.0000	ϵ_2	-0.4756
β	0.0014	ϵ_3	-0.7092
ω	0.0099	δ	-0.3789
a	0.3948	ϕ	-0.0099
b	0.1982	ν	-0.0616
θ	0.0715	τ	-0.0010
ψ	0.5781		

transmission potential, albeit to a lesser extent. Parameters related to prevention, treatment, and disease progression, including ϵ_1 , ϵ_2 , ϵ_3 , ϕ , τ , δ , and ν , have negative sensitivity indices, indicating that increases in these parameters reduce $\mathcal{R}_{\mathcal{H}}^*$. Among these parameters, the ART initiation rates (ϵ_2 and ϵ_3) exhibit greater magnitudes, highlighting its significant impact in reducing HIV transmission from individuals in the chronic and AIDS stages. Enhanced ART uptake across all infection stages, together with increased coverage of PrEP and PEP, represent positive results for controlling HIV transmission, progression and reducing AIDS related deaths.

4 Numerical solution of the HIV/AIDS model

In this section, numerical simulations are performed to investigate the impact of various control strategies on the transmission dynamics of HIV/AIDS. The initial conditions were chosen to reflect the HIV/AIDS situation in Uganda in mid-2024. According to the 2024 National Population and Housing Census, Uganda’s total population was estimated at $N_0 = 45,905,417$ [52]. The Uganda AIDS Commission Annual Joint AIDS Review (JAR) report for 2022/2023 estimated the number of people living with HIV (PLHIV) at 1,433,337, of whom 1,403,603 were receiving antiretroviral therapy (ART) [54]. The same report recorded approximately 51,516 new HIV infections during the reporting period. Assuming an average duration of one month in both the exposed and acute infection stages, the corresponding initial populations were taken as $H_{E0} = H_{A0} = \frac{51,516}{12} \approx 4,293$. Although no official national estimate of active PrEP users was publicly available, HIV prevention programme reports indicate substantial expansion of PrEP services, with more than 500,000 cumulative initiations by 2023 and continued growth through 2024 [54]. Consequently, the initial PrEP population was assumed to be 600,000. Furthermore, the sustained decline in AIDS-related mortality and the high ART coverage reported by national HIV programmes suggest that untreated AIDS cases constitute a relatively small proportion of PLHIV. Accordingly, the initial AIDS population was taken as 10,000. The remaining infected population was assigned to the chronic HIV compartment according to $H_{C0} = \text{PLHIV} - T_0 - H_{A0} - A_0$, which gives $H_{C0} = 15,441$. The susceptible population was then obtained from the population balance relation $S_0 = N_0 - (P_0 + H_{E0} + H_{A0} + H_{C0} + A_0 + T_0)$, yielding $S_0 = 43,867,787$. Hence, the initial conditions used in all simulations were $S_0 = 43,867,787$, $P_0 = 600,000$, $H_{E0} = 4,293$, $H_{A0} = 4,293$, $H_{C0} = 15,441$, $A_0 = 10,000$, $T_0 = 1,403,603$. These values represent the baseline distribution of the Ugandan population across the model compartments.

Table 3 Parameter values used in numerical simulations

Parameter	Value (year ^{−1})	Source	Parameter	Value (year ^{−1})	Source
Λ	$0.0352N_0$	[52]	ω	2.000	Assumed
μ	0.01471	[53]	k	106.80	Assumed
τ	0.0125	[54]	ξ	0.0011	Assumed
ϕ	0.020	[54]	a	12.000	Assumed
β	12.000	[55]	b	4.000	Assumed
ϵ_1	0.200	[54]	ν	0.500	[43]
ϵ_2	0.840	[54]	ψ	0.050	[54]
ϵ_3	0.900	[54]	θ	0.125	[55]
δ	12.000	[55]			

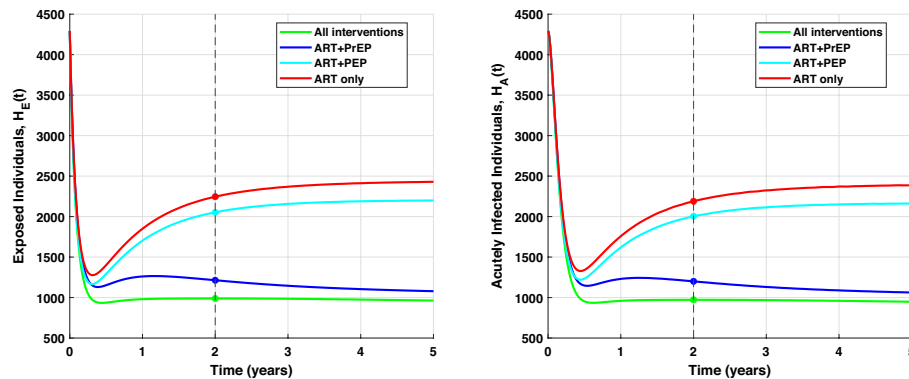


Fig. 3 Role of intervention strategies in exposed and acute classes

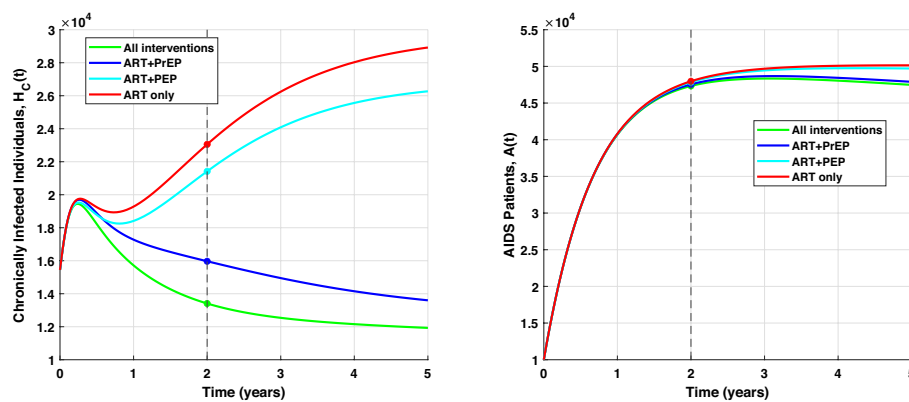


Fig. 4 Role of intervention strategies in chronic and AIDS classes

Table 4 Compartment values evaluated at $t = 2$ years under different intervention strategies

Intervention strategy	H_E	H_A	H_C	A
All interventions	988	972	13408	47331
ART + PrEP	1214	1200	15968	47548
ART + PEP	2052	2004	21416	47845
ART only	2246	2189	23053	47975

The model was parameterized using demographic and epidemiological data for Uganda obtained from the Uganda Bureau of Statistics (UBOS), the World Bank, and the Uganda AIDS Commission (UAC) [52–54]. For example, the treatment initiation rate for chronically infected individuals was estimated from Uganda’s reported ART coverage of approximately 84% among people living with HIV. Assuming that individuals who are diagnosed and linked to care initiate treatment within one year, the treatment initiation parameter was taken as $\epsilon_2 \approx 0.84 \text{ yr}^{-1}$. Similarly, the progression rate from chronic HIV infection to AIDS was derived from the average duration of untreated chronic HIV infection. Studies indicate that untreated individuals typically remain in the chronic stage for approximately 8 years before progressing to AIDS. Using the reciprocal of the mean duration, $\theta = \frac{1}{8} = 0.125 \text{ yr}^{-1}$. Parameters for which direct estimates were unavailable were assigned values based on epidemiological considerations and assumptions consistent with the HIV/AIDS literature. The parameter values used in the simulations are presented in Table 3.

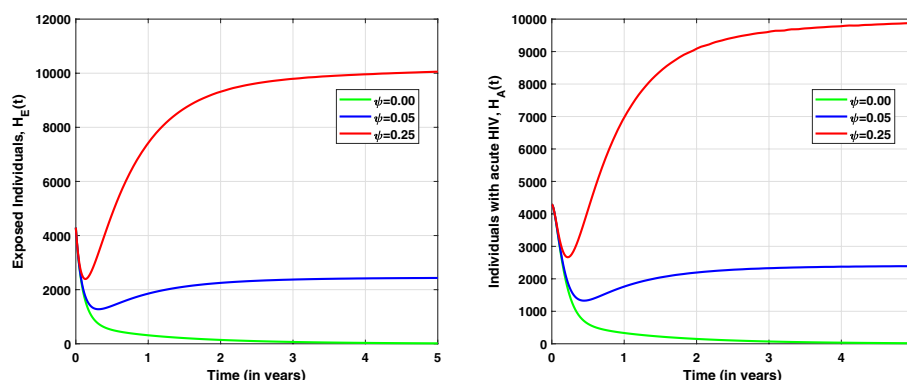


Fig. 5 Effect of defaulting ART in exposed and acute classes

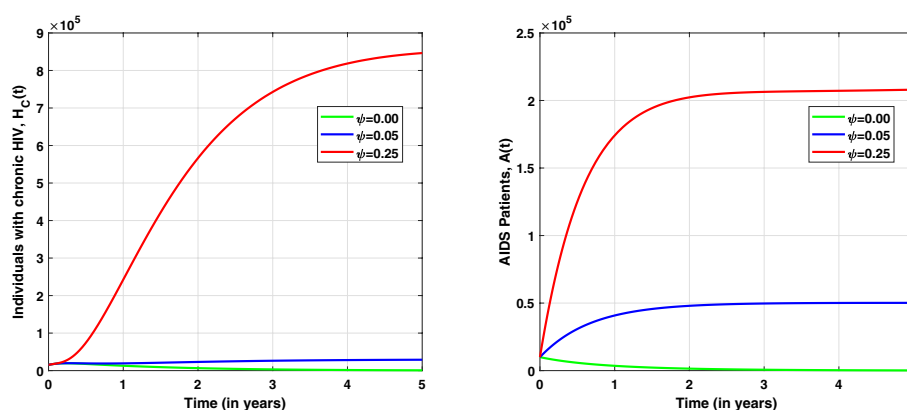


Fig. 6 Effect of defaulting ART in chronic and AIDS classes

The resulting system of ordinary differential equations was solved numerically in MATLAB using built-in ODE solvers, and the simulations were used to assess the effects of the proposed intervention strategies on HIV/AIDS transmission, disease progression, and treatment outcomes.

4.1 Simulating the role of biomedical interventions on the dynamics of HIV/AIDS

Figures 3 and 4 illustrate the effects of the different intervention strategies, while the corresponding numerical outcomes are summarized in Table 4. The results indicate that implementing multiple interventions simultaneously leads to a substantial reduction in HIV transmission compared to single-intervention strategies. In particular, the number of exposed individuals decreases markedly when at least two control measures are combined, with the greatest reduction observed when all interventions are applied concurrently. This outcome is primarily attributable to the enhanced prevention of new infections through the combined use of PrEP and PEP, together with improved treatment coverage. The simulations also demonstrate a sustained decline in the populations of acutely infected, chronically infected, and AIDS individuals under combined intervention strategies. As shown in Table 4, the simultaneous implementation of all control measures produces the largest reduction in disease burden after two years of intervention. This strategy yields the lowest numbers of exposed, acutely infected, chronically infected, and AIDS individuals, highlighting the benefits of an integrated HIV prevention and treatment approach. The next most effective strategy is the combined

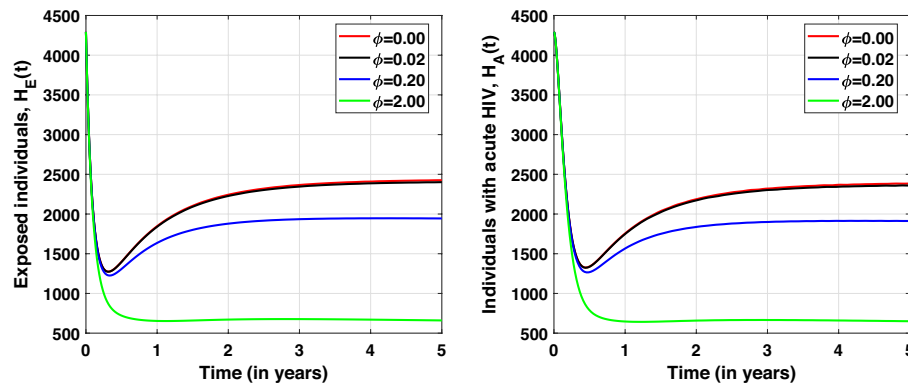


Fig. 7 Effect of PrEP on exposed and acute classes

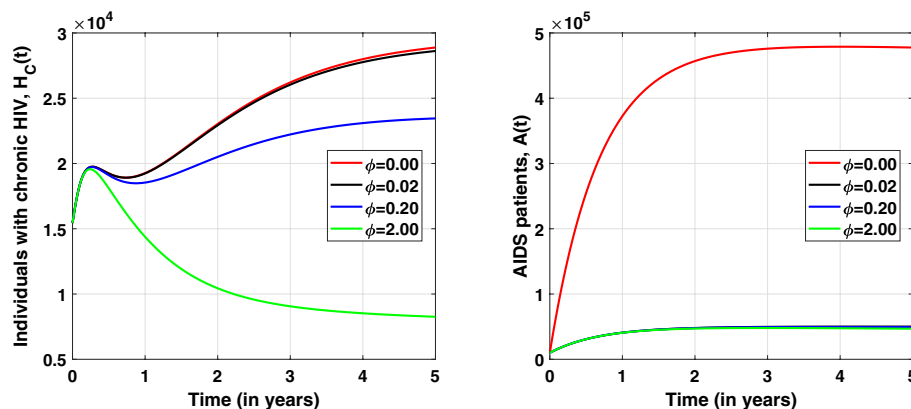


Fig. 8 Effect of PrEP on chronic and AIDS classes

implementation of ART and PrEP, which also achieves substantial reductions across the infected compartments.

4.2 Simulating the effect of defaulting ART on the dynamics of HIV/AIDS

The consequences of defaulting on HIV medication are clearly illustrated in Figs. 5 and 6. A default rate of $\psi = 0.25$ indirectly leads to an increase in the number of exposed, acutely infected, and chronically infected individuals, while directly increasing the number of individuals progressing to the AIDS class. Since ART suppresses viral load and consequently reduces the probability of HIV transmission, individuals who default on treatment experience an increase in viral load, which enhances their infectiousness and contributes to a higher rate of disease spread within the population. Conversely, when full compliance with ART ($\psi = 0$) is achieved, the number of individuals in all infected classes decline substantially. The simulation results suggest that sustained ART adherence could substantially reduce HIV transmission and progression to AIDS. Under the model assumptions, the number of AIDS cases declines to very low levels within approximately five years, indicating the potential effectiveness of long-term ART programs in reducing the burden of HIV/AIDS.

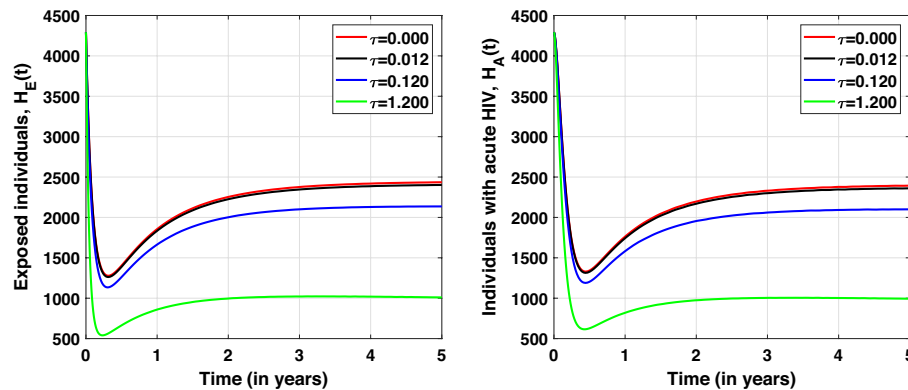


Fig. 9 Effect of PEP on exposed and acute classes

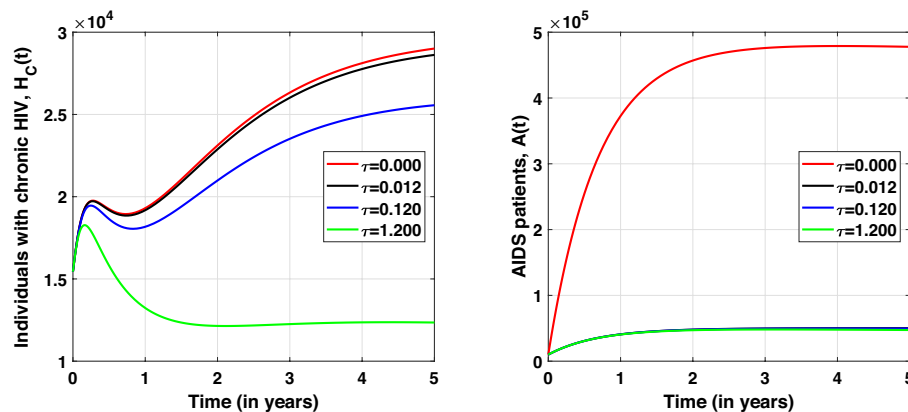


Fig. 10 Effect of PEP on chronic and AIDS classes

4.3 Simulating the role of PrEP on HIV/AIDS dynamics

Figure 7 demonstrates that increased uptake of PrEP significantly reduces HIV exposure. As a result, the number of individuals with acute and chronic HIV infections declines, which subsequently leads to a reduction in the number of AIDS cases, as illustrated in Fig. 8.

4.4 Simulating the role of PEP on HIV/AIDS dynamics

The impact of PEP is evident in Figs. 9 and 10, where its application results in a decline in the number of exposed, acutely and chronically infected individuals. Furthermore, an indirect reduction in the number of AIDS cases is observed, largely due to the prevention of infection at the exposure stage and subsequent progression through effective PEP intervention.

4.5 Simulating the role of ART in the dynamics of HIV/AIDS

Figures 11, 12, and 13 demonstrate the impact of ART initiation at different stages of HIV infection. Increasing ART initiation during the acute stage (ϵ_1) results in only a modest reduction in the infected classes. This limited impact can be attributed to the brief duration of the acute infection phase, which often remains unnoticed. When ART initiation is increased at the chronic stage (ϵ_2), a more pronounced reduction is observed in the chronically infected population and in AIDS cases, accompanied by an increase in

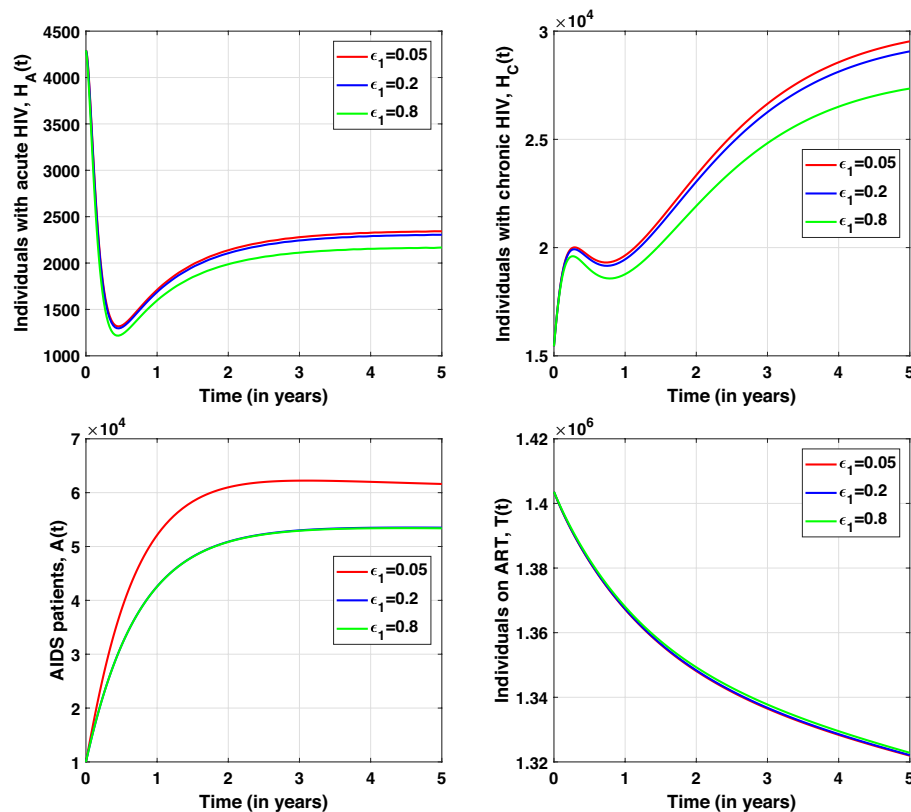


Fig. 11 Effect of ART at HIV acute stage

the number of individuals on ART. Furthermore, initiating ART at the AIDS stage (ϵ_3) significantly reduces the number of AIDS patients while increasing the number of individuals receiving treatment. This intervention also indirectly contributes to a reduction in the number of chronically infected individuals.

5 Conclusion

This study developed and analysed a mathematical model for HIV/AIDS transmission to investigate the role of ART, PrEP, and PEP in controlling the epidemic. The model was shown to be epidemiologically well posed through the establishment of the positivity and boundedness of its solutions. The effective reproduction number ($\mathcal{R}_H^*$) was derived and used as the threshold parameter for evaluating the dynamics of HIV/AIDS. Both the HIV/AIDS-free and endemic equilibrium states were established. The analysis revealed that the HIV/AIDS-free equilibrium is locally and globally asymptotically stable whenever $\mathcal{R}_H^* < 1$. The bifurcation analysis revealed the possible occurrence of a backward transcritical bifurcation when the effective reproduction number is equal to unity. This phenomenon implies that HIV infection persists in the population even when the effective reproduction number is less than one. Consequently, this result provides a possible explanation for the continued persistence and spread of HIV at the national level despite significant progress in reducing the effective reproduction number through treatment and prevention strategies. Interestingly, under full compliance with ART ($\psi = 0$), the model exhibits a forward bifurcation, indicating the existence of a unique endemic equilibrium that is stable whenever $RR_H > 1$. From a public health perspective, this result highlights the need for more aggressive and sustained control measures

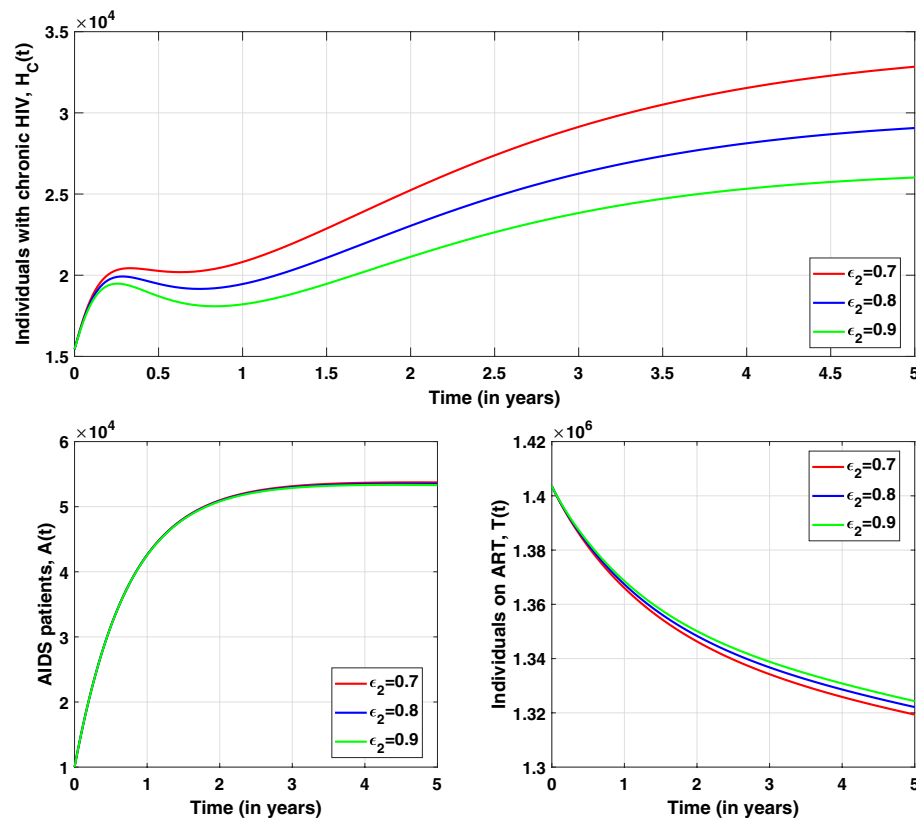


Fig. 12 Effect of ART at HIV chronic stage

capable of reducing the reproduction number well below unity while simultaneously addressing factors that sustain endemic transmission.

Sensitivity analysis identified HIV acquisition related parameters as the key drivers of disease transmission within the community. In contrast, prevention and treatment related parameters were found to play a crucial role in controlling the epidemic. Specifically, ART was shown to reduce the progression of HIV infection to AIDS, PrEP lowered the susceptibility of individuals to HIV infection, and PEP increased the likelihood of survival following potential HIV exposure. Overall, the sensitivity results suggest that public health policies should prioritize strengthening ART programs alongside preventive interventions such as PrEP and PEP. At the same time, targeting key transmission related parameters could yield significant reductions in HIV transmission, AIDS-related morbidity, and mortality.

The numerical simulations further demonstrate that ART remains central to combating the HIV/AIDS epidemic. A key finding of this study is that adherence to ART is critical if Uganda, and the global community at large, are to achieve the goal of ending the AIDS pandemic. While individual strategies ART, PrEP, and PEP each contribute to reducing the number of people living with HIV/AIDS, the results emphasize that a combined implementation of these interventions produces the greatest impact.

This study recommends strengthening interventions that enhance sustained adherence to ART, since the results demonstrate that ART plays a central role in reducing HIV progression and transmission. Public health authorities should therefore reinforce programs that promote long term ART adherence through improved patient follow up,

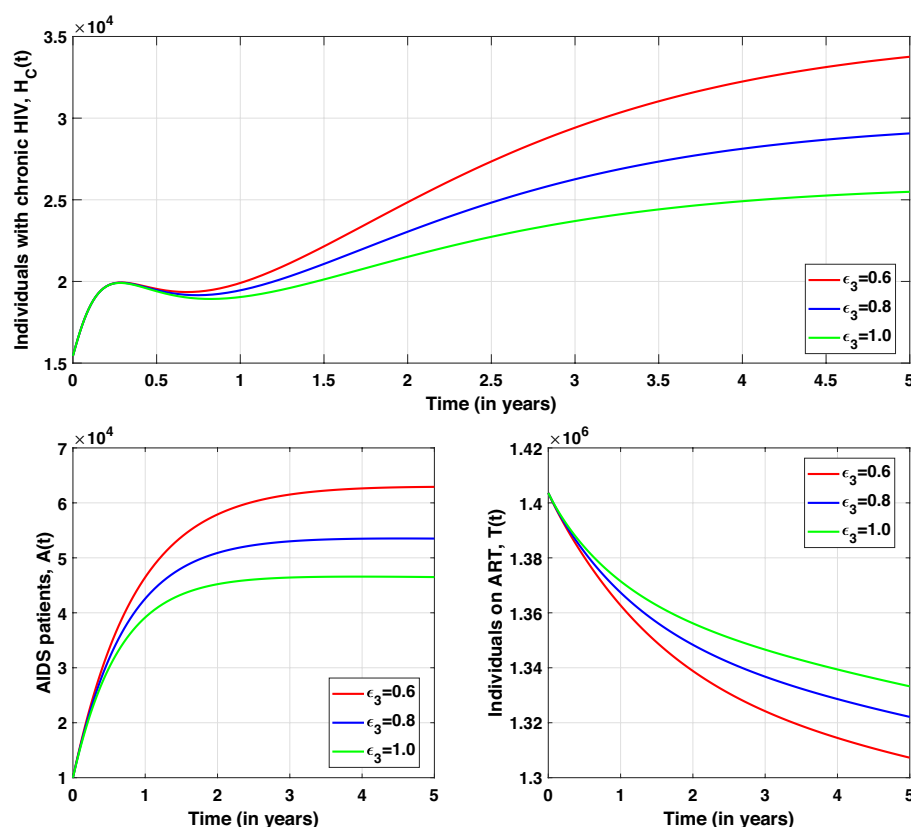


Fig. 13 Effect of ART at AIDS stage

community based adherence support systems, and uninterrupted access to antiretroviral medications. Achieving high levels of ART adherence will significantly reduce AIDS related morbidity and mortality. In addition, access to PrEP should be expanded among high risk populations, while ensuring the timely availability of PEP following any potential HIV exposure. Integrating these preventive strategies with improved treatment programs will enhance the overall effectiveness of HIV prevention and control efforts.

This study has some limitations. It does not incorporate parameter fitting, likelihood-based estimation, or validation against longitudinal HIV/AIDS surveillance data. In addition, the model assumes homogeneous mixing of the population and does not account for heterogeneity in age, gender, or risk groups in HIV transmission dynamics.

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Author contributions

M.B., M.K., and A.M. conceived and designed the study and developed the mathematical model. M.B., M.K., and R.M. performed the mathematical analysis and conducted the simulations. M.B., A.M., and J.A. interpreted the results and contributed to the preparation, review, and editing of the manuscript. R.M. and J.A. provided supervision and overall guidance throughout the study.

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Data availability

The datasets generated during or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Ethical approval for this study was obtained from the Mbarara University of Science and Technology Research Ethics Committee (MUST-REC) (ID: MUST-2025-734). Additional approval was obtained from the Uganda National Council for Science and Technology (UNCST) (Approval No.: NS1268ES). Not applicable. This study did not involve direct participation of human subjects, as it was based on secondary data and mathematical modeling.

Consent for publication

Not applicable. The study did not involve identifiable individual data requiring consent for publication.

Competing interests

The authors declare no competing interests.

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References

1. Lu Y, Li H, Dong R, Zhang M, Dong X, Yang C, et al. Global and regional disease burden of HIV/AIDS from 1990 to 2021 and projections to 2030. *BMC Public Health*. 2025;25(1):1928.
2. Eisinger RW, Fauci AS. Ending the HIV/AIDS pandemic. *Emerg Infect Dis*. 2018;24(3):413.
3. Haregu TN, Oldenburg B, Sestwe G, Elliott J, Nanayakkara V. Epidemiology of comorbidity of HIV/AIDS and non-communicable diseases in developing countries: a systematic review. *J Glob Health Care Syst*. 2012;2(1):1–12.
4. De Cock KM, Jaffe HW, Curran JW. The evolving epidemiology of HIV/AIDS. *AIDS*. 2012;26(10):1205–13.
5. AIDS and Hepatitis C. Professional Group, Society of Infectious Diseases, Chinese Medical Association; Chinese Center for Disease Control and Prevention. Chinese guidelines for the diagnosis and treatment of HIV/AIDS (2021 edition). *Infect Diseases Immun* 2022;2(03):145–167.
6. Yoshimura K. Current status of HIV/AIDS in the ART era. *J Infect Chemother*. 2017;23(1):12–6.
7. Cohen MS, Chen YQ, McCauley M, Gamble T, Hosseinipour MC, Kumarasamy N, et al. Antiretroviral therapy for the prevention of HIV-1 transmission. *N Engl J Med*. 2016;375(9):830–9.
8. Rodger A, Phillips A, Lundgren J, Tao X, Shao D, Xue W, et al. Prevention of HIV-1 infection with antiretroviral therapy. *N Engl J Med*. 2011;365(20):1934.
9. Holz WA. Post-Exposure and Pre-Exposure HIV Prophylaxis. In: *Introduction to Clinical Infectious Diseases: A Problem-Based Approach*. Springer Nature Switzerland: Cham; 2025. p. 683–92.
10. Ayieko J, Petersen ML, Kanya MR, Havlir DV. PEP for HIV prevention: are we missing opportunities to reduce new infections? *J Int AIDS Soc*. 2022;5:25.
11. Centers for Disease Control and Prevention. (2023). Post-exposure prophylaxis (PEP). <https://www.cdc.gov/stophivtogether/hiv-prevention/pep.html>
12. Pan American Health Organization. (2024). Pre-exposure prophylaxis (PrEP) for HIV prevention. <https://www.paho.org>
13. National Institutes of Health. (2024). Post-exposure prophylaxis (PEP) fact sheet. <https://hivinfo.nih.gov>
14. Jimu C, Nyamaruze P, Moyo FT, Makaye J. Understanding barriers to pre-exposure prophylaxis (PrEP) uptake among young people in Lower Gweru, Zimbabwe. A qualitative study. *J HIV/AIDS Soc Serv*. 2025;1–32.
15. Nabunya R, Karis VM, Nakanwagi LJ, Mukisa P, Muwanguzi PA. Barriers and facilitators to oral PrEP uptake among high-risk men after HIV testing at workplaces in Uganda: a qualitative study. *BMC Public Health*. 2023;23(1):365.
16. Garnett GP, Godfrey-Faussett P. How plausible is it that PEP would be cost-effective in sub-Saharan Africa? *J Int AIDS Soc*. 2025;28:e26455.
17. Resar D, Mwamelo AJ, Olowu A, Drakes J, Macul H, Gusmao ED, et al. Untapped potential of post-exposure prophylaxis in sub-Saharan Africa: a comparative analysis of PEP implementation planning in Kenya, Mozambique, Nigeria, Uganda and Zambia. *J Int AIDS Soc*. 2025;28:e26471.
18. Magni S, Byamukama D, Haske MS, Mukami J, Moyo I, Auerbach JD. Implementing the new WHO guidelines on HIV post-exposure prophylaxis: perspectives from five African countries. *J Int AIDS Soc*. 2025;28:e26447.
19. Jacquez JA, Simon CP, Koopman J, Sattenspiel L, Perry T. Modeling and analyzing HIV transmission: the effect of contact patterns. *Math Biosci*. 1988;92(2):119–99.
20. Sani A, Kroese DP, Pollett PK. Stochastic models for the spread of HIV in a mobile heterosexual population. *Math Biosci*. 2007;208(1):98–124.
21. Tuckwell HC, Le Corfec E. A stochastic model for early HIV-1 population dynamics. *J Theor Biol*. 1998;195(4):451–63.
22. Di Biase G, D'Amico G, Di Girolamo A, Janssen J, Iacobelli S, Tinari N, et al. A stochastic model for the HIV/AIDS dynamic evolution. *Math Probl Eng*. 2007;2007(1):065636.
23. Din A, Li Y. Optimizing HIV/AIDS dynamics: stochastic control strategies with education and treatment. *Eur Phys J Plus*. 2024;139(9):812.
24. Kumar N, Kashif M, Chauhan TS, Chauhan IS. Modeling and Control of HIV/AIDS Epidemics: A Stochastic and Optimal Control Perspective. *J Nonlinear Math Phys*. 2025;32(1):80.
25. Nsuami MU, Witbooi PJ. A stochastic model for HIV epidemic with treatment and inflow of infectives. *Int J Appl Math*. 2018;31(5):545.
26. Baryarama F, Mugisha JYT, Luboobi LS. A mathematical model for the dynamics of HIV/AIDS with gradual behaviour change. *Comput Math Methods Med*. 2006;7(1):15–26.
27. Nyabadza F. A mathematical model for combating HIV/AIDS in Southern Africa: will multiple strategies work? *J Biol Syst*. 2006;14(03):357–72.
28. Oyovwevotu SO. Mathematical modelling for assessing the impact of intervention strategies on HIV/AIDS high risk group population dynamics. *Heliyon*. 2021;10:7.

29. Olaniyi S, Olayiwola JO, Obabiyi OS, Lebelo RS, Abimbade SF. Mathematical analysis of non-autonomous HIV/AIDS transmission dynamics with efficient and cost-effective intervention strategies. *Contemp Math*. 2025;6(2):1380–2725.
30. Raza A, Ahmadian A, Rafiq M, Salahshour S, Naveed M, Ferrara M, et al. Modeling the effect of delay strategy on transmission dynamics of HIV/AIDS disease. *Adv Differ Equ*. 2020;2020(1):663.
31. Lutambi AM. The effect of delayed death in HIV/AIDS models. *J Biol Dyn*. 2016;10(1):286–96.
32. Denu D, Ngoma S, Salako RB. Analysis of a time-delayed HIV/AIDS epidemic model with education campaigns. *Comput Appl Math*. 2021;40(6):210.
33. Rivu NNK, Kamrujjaman M, Iqbal A. HIV/AIDS suppression in North America: intervention plans and Cost-Effectiveness of UNAIDS 90–90–90 and 95–95–95 targets. *Commun Nonlinear Sci Numer Simul*. 2025:109246.
34. Rivu NNK, Kamrujjaman M, Ahmed S. Comparative analysis of stochastic and predictable models in the HIV epidemic across genders. (2025). [arXiv:2501.17259](https://arxiv.org/abs/2501.17259).
35. Peter OJ, Teklu SW, Balogun GB, Bolarin G, Asfaw MD. HIV/AIDS compartmental model analysis with drug resistance treatment, vertical transmission, and optimal control theory. *Sci Rep*. 2025;15(1):44156.
36. Moya EMD, Rodrigues DS, Pietrus A, Severo AM. A mathematical model for HIV/AIDS under pre-exposure and post-exposure prophylaxis. *Biomath*. 2022;11(2):2208319–2208319.
37. Stover J, Glaubius R, Teng Y, Kelly S, Brown T, Hallett TB, et al. Modeling the epidemiological impact of the UNAIDS 2025 targets to end AIDS as a public health threat by 2030. *PLoS Med*. 2021;18(10):e1003831.
38. Ministry of Health (MOH). HIV/AIDS in Uganda: The epidemic response. Kampala, Uganda: Ministry of Health. (2024).
39. Uganda AIDS Commission (UAC). Uganda HIV/AIDS Fact Sheet 2024. Kampala, Uganda: Uganda AIDS Commission. (2024).
40. Akullian A, Ssempijja V, Breidenbecker D, Nalugoda F, Nakigozi G, Santelli J, ..., Serwadda D. Evaluating the biomedical and behavioural drivers of HIV-1 incidence decline in adolescent girls and young women in Uganda: A mathematical modelling study. *medRxiv*. (2025).
41. Obeagu EI, Obeagu GU, Ede MO, Odo EO, Buhari HA. Translation of HIV/AIDS knowledge into behavior change among secondary school adolescents in Uganda: A review. *Medicine*. 2023;102(49):e36599.
42. UNAIDS. The Urgency of Now: AIDS at a Crossroads – Global AIDS Update 2024. Geneva: UNAIDS. (2024).
43. Byamukama M, Karuhanga M, Kajunguri D. Mathematical analysis of the role of treatment and vaccination in the management of the HIV/AIDS and pneumococcal pneumonia co-infection. *J Math*. 2025;2025(1):5879698.
44. Hernandez-Vargas EA, Middleton RH. Modeling the three stages in HIV infection. *J Theor Biol*. 2013;320:33–40.
45. Heffernan JM, Smith RJ, Wahl LM. Perspectives on the basic reproductive ratio. *J R Soc Interface*. 2005;2(4):281–93.
46. Van den Driessche P, Watmough J. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Math Biosci*. 2002;180(1–2):29–48.
47. Byamukama M, Kajunguri D, Karuhanga M. Optimal control analysis of pneumonia and HIV/AIDS co-infection model. *Math Open*. 2024;3:2450006.
48. Castillo-Chavez C, Feng Z, Huang W. Global dynamics of a plant-herbivore model with toxin-determined functional response. *SIAM J Appl Math*. 2012;72(4):1002–20.
49. Castillo-Chavez C, Song B. Dynamical models of tuberculosis and their applications. *Math Biosci Eng*. 2004;1(2):361.
50. Mbeine Asimwe A, Byamukama M, Karuhanga M. Modeling the effect of alcohol consumption on the progression dynamics of Hepatitis C infection with treatment. *Math Med Life Sci*. 2026;3(1):2620745.
51. Byamukama M, Kajunguri D, Karuhanga M. A mathematical model for Co-infection dynamics of pneumocystis pneumonia and HIV/AIDS with treatment. 2023. [arXiv:2306.04407](https://arxiv.org/abs/2306.04407).
52. Uganda Bureau of Statistics (UBOS). National Population and Housing Census 2024 – Final Report Volume 1. 2024. <https://statistics.ubos.org/nphc/reports/National-Population-and-Housing-Census-2024-Final-Report-Volume-1-Main.pdf>
53. World Bank. Uganda Country Profile: Demographic Indicators (Birth Rate and Death Rate). World Bank Data, 2024. <https://data.worldbank.org/country/uganda>.
54. Uganda AIDS Commission (UAC). Annual Joint AIDS Review Report FY 2022/2023. Uganda AIDS Commission, Kampala, Uganda, 2023. <https://uac.go.ug/images/2024/jar-2024/jar-2022-2023-annual-report.pdf>.
55. National Institutes of Health (NIH). (2024). Stages of HIV infection. HIVinfo. U.S. Department of Health and Human Services. <https://hivinfo.nih.gov/understanding-hiv/fact-sheets/stages-hiv-infection>.

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