

Modelling the role of denial in sustaining HIV transmission in South Africa

Francis Mugabi¹, Piero Manfredi², Kevin Jan Duffy^{3,4,*}

¹Faculty of Science and Technology,
International University of East Africa, Kampala, Uganda
mugabikanywa@yahoo.com

²Department of Economics and Management,
University of Pisa, Italy
piero.manfredi@unipi.it

³Department of Mathematics,
School of Agriculture and Science,
University of KwaZulu-Natal, Durban, South Africa
duffy@ukzn.ac.za

⁴National Institute for Theoretical and Computational Sciences (NITheCS),
South Africa

Received: 23 January 2026, Published: 24 July 2026

Abstract: HIV/AIDS remains a major public health challenge in South Africa, where denialism continues to undermine prevention and treatment efforts. This study develops a mathematical transmission model that explicitly incorporates denial behaviours, including refusal of preventive measures, refusal to initiate antiretroviral therapy (ART), and discontinuation of ART. Analytical and simulation results show that denial substantially increases HIV incidence, accelerates progression to AIDS, and raises the likelihood of a backward bifurcation, an occurrence where HIV may persist even when the basic reproduction number falls below one. Sensitivity analysis identified denial-related transmission as a key driver of epidemic dynamics. These findings underscore the need to address denialism by strengthening HIV testing, ART uptake and retention, and community-level education to counter misinformation and stigma.

Keywords: HIV transmission, denialism, mathematical modelling, antiretroviral therapy, backward bifurcation

I. INTRODUCTION

HIV/AIDS remains a major public health challenge in South Africa, which continues to bear one of the highest burdens globally. In 2023, the country accounted for 11.5% of global HIV infections and 7.9% of AIDS-related deaths [1]. Despite major progress in scaling up antiretroviral therapy (ART) and implementing prevention measures such as condom distribution and HIV testing campaigns, prevalence remains high, and the epidemic continues to strain the health system.

One important but often overlooked driver of this persistence is denialism. Denial of the HIV virus and its role in causing AIDS, initiated by virologist Peter Duesberg, challenges the scientific consensus by rejecting the link between HIV and AIDS, instead attributing the disease to alternative causes like drug use or mal-

nutrition. Although this approach has been thoroughly discredited, misinformation associated with it led to the promotion of alternative therapies with severe public health consequences. Psychological, political, and behavioural factors are crucial in shaping the dynamics of HIV transmission, as well as the effectiveness of prevention and treatment strategies [2].

Among these factors, denial, characterised by the refusal to acknowledge one's HIV status or the seriousness of the disease, represents a significant barrier to effective HIV prevention, care, and treatment [3]. Denial can manifest in various ways, such as reluctance to undergo HIV testing, avoidance of disclosing one's HIV status to sexual partners or healthcare providers, and failure to adhere to antiretroviral therapy (ART) regimens. After the work of Peter Duesberg, AIDS denialists argue, for instance, that HIV is harmless and cannot cause AIDS [4]. Some even claim there is no proof that HIV exists, and they view HIV treatments as toxic substances that cause more harm than good [4].

The population-level consequences of AIDS denialism can be severe, significantly hindering efforts to control and treat the disease. In South Africa, the impact of denialism was particularly pronounced during the presidency of Thabo Mbeki, when AIDS denialists, led by Peter Duesberg, were afforded undue credibility in comparison to legitimate scientists. As the nation's leader, Mbeki's stance significantly undermined the public health system and responses to the crisis. This contributed to the South African government's delay in promoting HIV testing and obstructed the distribution of antiretroviral medications [3]. As a result, many South Africans died prematurely from AIDS. In the early 2000s, at least 330,000 people are estimated to have died from the disease, and more than 35,000 babies were needlessly infected with HIV because medications to prevent mother-to-child transmission were withheld [3, 5]. Despite its profound negative impact on both individual health outcomes and broader HIV prevention efforts, the role of denial in driving HIV transmission remains inadequately explored, especially within the context of mathematical modelling.

Mathematical modelling offers a powerful framework for understanding the complex interplay between behavioural, epidemiological, and healthcare factors in shaping the trajectory of infectious disease dynamics [6, 7]. In the context of HIV in Sub-Saharan Africa, several mathematical models [8–14] have been developed to investigate different issues from a theoretical viewpoint, including HIV transmission dynamics, evaluation of the impact of interventions aimed at reducing

incidence and mortality, population-level side effects of HIV therapies, etc. Also, several highly detailed epidemiological modelling studies have focused on the specificity of the HIV epidemic in South Africa and related intervention policies [15–19]. For instance, at the onset the COVID-19 pandemic, strong concerns were raised about the potentially severe impact of the interruption of HIV services [18]. Surprisingly, such concerns were not specifically raised for HIV denial.

Additionally, in the last epoch, an entire discipline has emerged that investigates the different facets of the effects of human behaviour on infection control [20–22]. Despite these efforts, HIV denialism, a multi-dimensional behavioural phenomenon recognised for its significant impact on individual risk and healthcare-seeking behaviours with a complicated influence on HIV transmission dynamics, has received limited attention in mathematical modelling studies.

This study addresses that gap. We develop a compartmental model that explicitly incorporates denialist behaviour alongside HIV transmission and treatment. The model includes refusal of preventive measures, ART initiation, and ART discontinuation and allows us to explore how denialism alters epidemic trajectories. Using analytical and numerical approaches, we assess the potential for a backward bifurcation, evaluate parameter sensitivities, and quantify the impact of denial on HIV spread.

To the best of our knowledge, this is the first mathematical model to formalise denialism as a structural driver of HIV persistence in South Africa. By linking behavioural dynamics with epidemic thresholds and intervention outcomes, this study underscores that conventional biomedical measures may prove insufficient in the presence of mass denialist behaviours and highlights the need for integrated behavioural and biomedical strategies in HIV control.

II. MODEL DYNAMICS

The model includes various transitions between compartments, which are governed by behavioural, epidemiological, and treatment-related factors.

A. Recruitment into the population (π)

Individuals are recruited into the (S_n) group at a constant rate π .

B. Behavioural transition between S_n and S_d

- Rate of moving into denial (ε_n): Individuals in S_n may acquire denialist behaviour and move into compartment S_d , where they stop taking precautions.

In this scenario, denialist behaviour is assumed to be acquired at a constant rate ε_n . We motivate this choice later on in Section III.

- Rate of moving out of denial (ε_d): Individuals in S_d may switch to S_n , where they adopt protective behaviours due to education or sensitisation programmes.

C. HIV infection

- Force of infection for S_n (λ_n):

$$\lambda_n = \beta_n \frac{I_u + \phi_d I_d + \phi_n I_n}{N}.$$

Individuals in S_n are exposed to HIV infection based on their sexual contact rate (β_n) and the relative infectiousness of individuals in I_u , I_d , and I_n .

- Force of infection for S_d (λ_d):

$$\lambda_d = \beta_d \frac{I_u + \phi_d I_d + \phi_n I_n}{N}.$$

Individuals in S_d have a higher susceptibility to infection compared to those in S_n ($\beta_d > \beta_n$) due to their riskier behaviour (e.g., lack of precautions).

Where:

- β_n, β_d : Contact rates for individuals in compartments S_n and S_d , respectively.
- ϕ_d, ϕ_n : Relative infectiousness of I_d and I_n compared to I_u , with the infectiousness of I_u set to 1.

D. Screening and denial

Individuals in I_u are screened for HIV at a rate of α . A proportion ρ of those screened deny their diagnosis and transition to compartment I_d , including in-denial infective individuals. The remaining fraction $(1 - \rho)$ moves to compartment I_n of not-in-denial infected individuals, where they initiate antiretroviral therapy (ART). Therefore, newly infected individuals can begin ART immediately after screening, at an effective rate of $(1 - \rho)\alpha$. We also consider individuals who refuse screening and move to I_d at a rate of ω . This formulation allows the model to capture the behavioural dynamics of treatment acceptance and denialism following HIV screening.

E. Transitions related to ART

We also assume flows from I_n to I_d due to embracing denial and vice versa, much in the same way as for compartments S_n and S_d . However, we do not assume the same rates acting therein (that is, ε_n and ε_d) because further processes can be in place; e.g., I_n

individuals might escape towards I_d because of the onset of adverse effects from ART.

- ART drop-out (δ_n): Individuals in I_n may discontinue treatment and join the denialist group I_d due to misinformation or side effects of ART.
- ART enrolment (δ_d): Individuals in I_d may accept treatment and move to I_n due to awareness campaigns or education.

F. Progression to AIDS

Individuals in I_d and I_n progress to AIDS at rates γ_d and γ_n , respectively. The progression rate for I_n is lower than that for I_d due to the effectiveness of ART in delaying disease progression.

G. Mortality

- All compartments experience natural mortality at rate μ .
- Individuals in the AIDS compartment A experience additional disease-induced mortality at rate ν .

The model structure is summarised compactly in the reported flowchart (Figure 1).

H. Model equations

From Figure 1, the system of ODEs governing the population dynamics is given as follows:

$$\begin{aligned} \frac{dS_n}{dt} &= \pi + \varepsilon_d S_d - (\lambda_n + \mu + \varepsilon_n) S_n, \\ \frac{dS_d}{dt} &= \varepsilon_n S_n - (\lambda_d + \mu + \varepsilon_d) S_d, \\ \frac{dI_u}{dt} &= \lambda_n S_n + \lambda_d S_d - (\alpha + \omega + \mu) I_u, \\ \frac{dI_d}{dt} &= (\rho\alpha + \omega) I_u + \delta_n I_n - (\delta_d + \gamma_d + \mu) I_d, \\ \frac{dI_n}{dt} &= (1 - \rho)\alpha I_u + \delta_d I_d - (\delta_n + \gamma_n + \mu) I_n, \\ \frac{dA}{dt} &= \gamma_d I_d + \gamma_n I_n - (\mu + \nu) A, \end{aligned} \quad (1)$$

with $S_n(0) > 0$, $S_d(0) \geq 0$, $I_u(0) \geq 0$, $I_d(0) \geq 0$, $I_n(0) \geq 0$, and $A(0) \geq 0$.

III. MODEL ASSUMPTIONS

The model describes heterosexual transmission of HIV. Following a common standard in theoretical studies of sexually transmitted infections, we assumed perfect symmetry in the behaviour of males and females, thereby avoiding gender structure [6].

Building on this, the model further assumes two heterogeneous risk groups: individuals *not in denial* (low-risk) and those *in denial* (high-risk). This places

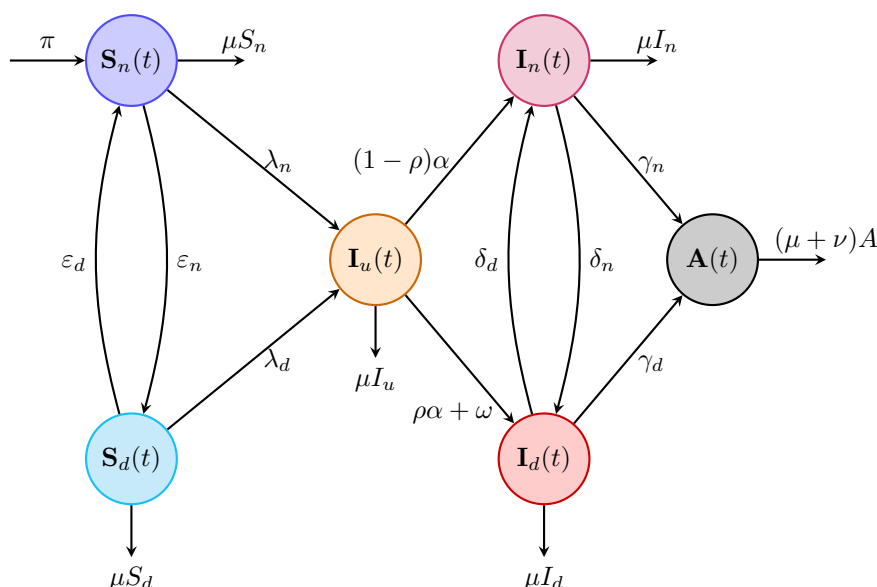


Fig. 1: Transmission dynamics of HIV in the context of denial.

the framework within the class of epidemic models with heterogeneity [6]. To the best of our knowledge, this is the first time that such heterogeneity has been used as the key ingredient in a model for HIV denialism. Recruitment is assumed to occur only into the not-in-denial susceptible group. This assumption could be relaxed, for example, by allowing a fraction of new entrants to join the denialist group due to family or social influence, but such an extension does not materially affect the qualitative results.

Another key assumption concerns the force of infection. AIDS individuals are included in the denominator, meaning that they are eligible as sexual partners, but excluded from the numerator, implying that they do not engage in risky sex. This hypothesis was less appropriate in the early epidemic when AIDS progression was rapid and untreated individuals quickly succumbed to illness and death [6]. However, the assumption becomes increasingly valid in the ART era, as therapy improves health status and quality of life [23, 24].

The model formulation of screening and ART access reflects current South African policy, where enrolment in ART requires only a positive test result. Following WHO recommendations, South Africa has adopted the “Test and Treat All” approach, which provides ART to all individuals diagnosed with HIV, regardless of CD4 count or clinical stage [25].

We do not split HIV-unaware individuals into sub-compartments based on their attitude toward denialism,

i.e., we do not differentiate between $I_{u,n}$ (unaware but not in denial) and $I_{u,d}$ (unaware and in denial). Though this should be in principle correct, given that HIV-infected but unaware individuals should behave as susceptible ones, this leads to unnecessary complexity that does not meaningfully affect the key model dynamics. The adopted formulation keeps screening (related diagnosis) as the key process through which infected individuals can enact their ideological behaviour by refusing ART.

The most important modelling choice concerns the representation of denialist behaviour. In this first effort, we opted for parsimony (Occam’s razor) and modelled transitions between denial and non-denial states as constant rates. We argue that this is appropriate for the period when denialism was actively promoted by South African President Thabo Mbeki, under the influence of virologist Peter Duesberg. In this period, adherence to denialism was largely driven by top-down political and media pressure. Classical models of information diffusion, such as the Bass model [26], describe diffusion processes driven by institutional or media communication as a linear process driven by constant rates. Pairwise, we assume that also the reverse flow from denial to non-denial was also mediated by ‘external’, top-down, communication, e.g., from non-denialist public health officers, who largely opposed the denialist stance [3, 5, 27, 28].

While ideological commitment rather than rational

cost-benefit reasoning explains much of the denialist adherence in the president Mbeki's era, we do not claim that other mechanisms of information diffusion, such as word-of-mouth imitation dynamics, were absent [20, 21, 26, 29–31]. Rather, we consider the major drivers sufficient for this first formulation, given the rich dynamics they already generate. Extensions to include word-of-mouth dynamics would be particularly useful for modelling the post-Mbeki period, when denialism was no longer state-promoted but persisted within specific communities.

Summing up in relation to the behavioral epidemiology literature, our approach represents AIDS denial as a loss of awareness implying a return to high-risk behaviors due to public communication [30]. This is mainly modelled by additional compartments of individuals losing awareness of HIV risks. This approach was used both during the early wave of behavioral change models for AIDS but also in the modern behavioral epidemiology literature [20, 30].

IV. ANALYTICAL RESULTS

A. Feasible region

To determine the feasible region where the model solutions are epidemiologically meaningful, the following theorem for positivity and boundedness is established.

Theorem 1. *The solutions to the system (1) that start with initial conditions that are non-negative, remain non-negative and bounded for all $t > 0$.*

Proof: The proof of the first part (non-negativeness) immediately follows from the model equations. Indeed, in the event any state variable reaches its zero boundary, it will either remain there or will increase. As for the second part of the Theorem, by summing the equations in (1), we get:

$$\frac{dN}{dt} = \pi - \mu N - \nu A \leq \pi - \mu N.$$

Which in the limit has to be larger than zero and so we have the upper bound:

$$\limsup_{t \rightarrow \infty} N(t) \leq \frac{\pi}{\mu} = N^*,$$

where N^* represents the population level in the absence of the infection.

This shows that the solutions of the model, starting from non-negative initial values, are bounded. ■

Therefore, the feasible region for the system is given by:

$$\Omega = \left\{ (S_n, S_d, I_u, I_d, I_n, A) \in \mathbb{R}_+^6 \mid N(t) \leq \frac{\pi}{\mu} = N^* \right\},$$

which is positively invariant with respect to the dynamics of (1).

B. Disease-free equilibrium and the basic reproduction number

The disease-free equilibrium point is given by $(S_n^*, S_d^*, 0, 0, 0, 0) \in \Omega$, where the total population N is at its disease-free steady state (given by $N^* = \pi/\mu$) and it holds:

$$S_n^* = \frac{\pi(\mu + \varepsilon_d)}{(\mu + \varepsilon_d)(\mu + \varepsilon_n) - \varepsilon_d \varepsilon_n},$$

$$S_d^* = \frac{\pi \varepsilon_n}{(\mu + \varepsilon_d)(\mu + \varepsilon_n) - \varepsilon_d \varepsilon_n}.$$

Quantities S_n^* and S_d^* represent the equilibrium levels balancing the populations of the denialist and non-denialist groups in a disease-free setting where pro- and anti-denial messages circulate but the infection is absent.

The next-generation method is used to determine the basic reproduction number $\mathcal{R}_0$, and to analyse the local stability of the disease-free equilibrium. This method requires the calculation of the transmission matrix (F) and the transition matrix (V) [32, 33]:

$$F = \frac{\beta_n S_n^* + \beta_d S_d^*}{N^*} \begin{pmatrix} 1 & \phi_d & \phi_n & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} \alpha + \mu & 0 & 0 & 0 \\ -(\rho\alpha + \omega) & \delta_d + \gamma_d + \mu & -\delta_n & 0 \\ -(1 - \rho)\alpha & -\delta_d & \delta_n + \gamma_n + \mu & 0 \\ 0 & -\gamma_d & -\gamma_n & \mu + \nu \end{pmatrix}$$

The basic reproduction number, is obtained by calculating the dominant eigenvalue of the matrix FV^{-1} . After simplification,

$$\mathcal{R}_0 = \mathcal{R}_u + \mathcal{R}_d + \mathcal{R}_n,$$

where

$$\mathcal{R}_u = \frac{\beta_n S_n^* + \beta_d S_d^*}{N^*(\alpha + \mu)} \quad (2)$$

represents the contribution to $\mathcal{R}_0$ from typical infective individuals who are unaware of their HIV status,

$$\mathcal{R}_d = \frac{\phi_d(\beta_n S_n^* + \beta_d S_d^*)}{N^*(\alpha + \mu)((\delta_n + \gamma_n + \mu)(\delta_d + \gamma_d + \mu) - \delta_n \delta_d)} \times ((\delta_n + \gamma_n + \mu)(\rho\alpha + \omega) + \delta_n(1 - \rho)\alpha). \quad (3)$$

represents the contribution from individuals who are in denial and not on ART, and

$$\begin{aligned} \mathcal{R}_n &= \frac{\phi_n(\beta_n S_n^* + \beta_d S_d^*)}{N^*(\alpha + \mu) \left((\delta_n + \gamma_n + \mu)(\delta_d + \gamma_d + \mu) - \delta_n \delta_d \right)} \\ &\quad \times \left((\delta_d + \gamma_d + \mu)(1 - \rho)\alpha + \delta_d(\rho\alpha + \omega) \right). \end{aligned} \quad (4)$$

represents the contribution from individuals under ART who are not in denial. These components of $\mathcal{R}_0$ corresponding to different subgroups of the population (unaware, denialists, and those under treatment), and reflect how each group contributes to the overall spread of HIV. By analysing these components, we can assess the impact of interventions targeting each group, such as improving screening rates, reducing denialism, or enhancing ART uptake, on controlling the HIV epidemic. Based on Theorem 2 in the study given in [33], the following theorem has been established:

Theorem 2. *The disease-free equilibrium is locally-asymptotically stable when $\mathcal{R}_0 < 1$, and unstable when $\mathcal{R}_0 > 1$.*

This theorem emphasises the importance of reducing $\mathcal{R}_0$ below 1 to control HIV/AIDS spread within a population. In the absence of a backward bifurcation (discussed in the next section), if $\mathcal{R}_0$ is less than 1, the disease will gradually fade, leading to a stable, disease-free state. On the other hand, if $\mathcal{R}_0$ remains above 1, the risk of widespread transmission persists, leading to an endemic equilibrium, and significant effort may be necessary to manage the number of new infections.

C. Endemic equilibrium

Endemic states of the model $(S_n^*, S_d^*, I_u^*, I_d^*, I_n^*, A^*) \in \text{int}(\Omega)$ fulfill:

$$\begin{aligned} S_n^* &= \frac{\pi(\lambda_d^* + \mu + \varepsilon_d)}{(\lambda_n^* + \mu + \varepsilon_n)(\lambda_d^* + \mu + \varepsilon_d) - \varepsilon_n \varepsilon_d}, \\ S_d^* &= \frac{\pi \varepsilon_n}{(\lambda_n^* + \mu + \varepsilon_n)(\lambda_d^* + \mu + \varepsilon_d) - \varepsilon_n \varepsilon_d}, \\ I_u^* &= \frac{\pi \left(\lambda_n^* (\lambda_d^* + \mu + \varepsilon_d) + \lambda_d^* \varepsilon_n \right)}{(\alpha + \mu) \left((\lambda_n^* + \mu + \varepsilon_n)(\lambda_d^* + \mu + \varepsilon_d) - \varepsilon_n \varepsilon_d \right)}, \\ I_d^* &= \frac{\mathcal{R}_d I_u^*}{\phi_d \mathcal{R}_u}, \\ I_n^* &= \frac{\mathcal{R}_n I_u^*}{\phi_n \mathcal{R}_u}, \\ A^* &= \frac{(\gamma_d \phi_n \mathcal{R}_d + \gamma_n \phi_d \mathcal{R}_n) I_u^*}{\phi_d \phi_n \mathcal{R}_u (\mu + \nu)}, \end{aligned} \quad (5)$$

where $\mathcal{R}_u$, $\mathcal{R}_d$ and $\mathcal{R}_n$ are as given in (2), (3), and (4), respectively, and the forces of infection are:

$$\lambda_n^* = \beta_n \left(\frac{I_u^* + \phi_d I_d^* + \phi_n I_n^*}{S_n^* + S_d^* + I_u^* + I_n^* + I_d^* + A^*} \right), \quad (6)$$

$$\lambda_d^* = \beta_d \left(\frac{I_u^* + \phi_d I_d^* + \phi_n I_n^*}{S_n^* + S_d^* + I_u^* + I_n^* + I_d^* + A^*} \right). \quad (7)$$

Substituting (5) and (6) into (7) leads to a characteristic polynomial

$$\lambda_d^* \left(c_2 (\lambda_d^*)^2 + c_1 \lambda_d^* + c_0 \right) = 0, \quad (8)$$

where

$$\begin{aligned} c_2 &= \beta_n \mathcal{R}_u (\alpha + \mu)^2 \left(\mathcal{R}_u \phi_n \phi_d (\mu + \nu) \right. \\ &\quad \left. + (\mu + \nu + \gamma_d) \phi_n \mathcal{R}_d + (\mu + \nu + \gamma_n) \phi_d \mathcal{R}_n \right), \\ c_1 &= \mathcal{R}_u (\alpha + \mu)^2 \left(\beta_n (\mu + \varepsilon_d) + \beta_d \varepsilon_n \right) \\ &\quad \times \left(\mathcal{R}_u \phi_n \phi_d (\mu + \nu) + (\mu + \nu + \gamma_d) \phi_n \mathcal{R}_d \right. \\ &\quad \left. + (\mu + \nu + \gamma_n) \phi_d \mathcal{R}_n \right) \\ &\quad + \phi_n \phi_d \mathcal{R}_u \beta_d (\mu + \nu) (\alpha + \mu)^2 \\ &\quad \times ((\alpha + \mu) \mathcal{R}_u - \mathcal{R}_0 \beta_n), \\ c_0 &= \phi_n \phi_d \beta_d \mathcal{R}_u (\mu + \nu) (\mu + \varepsilon_n + \varepsilon_d) \\ &\quad \times (\alpha + \mu)^3 (1 - \mathcal{R}_0). \end{aligned}$$

From the polynomial in equation (8), the root $\lambda_d^* = 0$ corresponds to the disease-free equilibrium since it implies a null force of infection. The remaining roots determine endemic equilibria, contingent upon the signs of the coefficients. Specifically, the signs of c_1 and c_0 are influenced by the magnitude $\mathcal{R}_0$, whereas $c_2 > 0$ irrespective of the value of $\mathcal{R}_0$. The model allows for various bifurcation scenarios at the endemic equilibrium, depending on the number of positive roots and the magnitude of $\mathcal{R}_0$. One such scenario is a backward bifurcation, which occurs when multiple positive roots (representing both endemic and disease-free equilibria) exist for $\mathcal{R}_0 < 1$. In the presence of a backward bifurcation, reducing $\mathcal{R}_0$ below unity is insufficient to control the disease. To investigate the potential for a backward bifurcation in the system described by model (1), the number of positive roots of equation (8) for different cases of $\mathcal{R}_0$ are analysed using Descartes's rule of signs and summarised in Table 1.

From Table 1, the following theorem is derived:

Theorem 3. *The HIV/AIDS model (1) has*

- (i) *an endemic equilibrium if $\mathcal{R}_0 > 1$,*
- (ii) *two endemic equilibria if $\mathcal{R}_0 < 1$ and whenever the second scenario holds.*

Scenario	c_2	c_1	c_0	$\mathcal{R}_0$	Possible number of positive roots
1	+	-	-	$\mathcal{R}_0 > 1$	1
	+	+	+	$\mathcal{R}_0 < 1$	0
2	+	+	-	$\mathcal{R}_0 > 1$	1
	+	-	+	$\mathcal{R}_0 < 1$	2,0

Table 1: Number of possible positive roots for the polynomial (8).

Figure 2 and Theorem 3 highlight that reducing $\mathcal{R}_0$ below unity may not guarantee HIV elimination if denial-driven heterogeneity in transmission or treatment response sustains the epidemic. This underscores the need to combine biomedical strategies (testing, ART scale-up) with psychosocial and behavioural interventions that address denialism directly. Without such integration, HIV may persist even after the classical control threshold is achieved.

Although the mechanisms underlying backward bifurcation in epidemiological models have been well characterised [34, 35], this study is, to the best of our knowledge, the first to identify denial, through its combined effects on transmission and therapy efficacy, as an important driver of HIV persistence.

To further explore how individual parameters influence $\mathcal{R}_0$ and disease outcomes, numerical simulations are conducted in the next section.

V. NUMERICAL RESULTS

To obtain the numerical results, a set of parameter values is required. These values are estimated in the following subsection.

A. Parameter assignments

In this section, we assign numerical values to the main parameters that characterise the dynamics of HIV transmission and progression in South Africa. These parameters are derived from various data sources, including demographic information, epidemiological studies, and UNAIDS reports. The following is a description of each parameter and its corresponding value range:

1) *Natural mortality (μ):* The natural mortality rate is based on life expectancy data from the World Health Organization [36], which reports an average life expectancy of 58.9 years for males and 64 years for females in South Africa. Additionally, according to Richter et al. [37], the average age of sexual debut is 15 years for females and 16 years for males. Thus, the range for the natural mortality rate corresponding to the life expectancy after sexual debut is estimated as $\mu = [1/(64 - 16), 1/(58.9 - 15)]$.

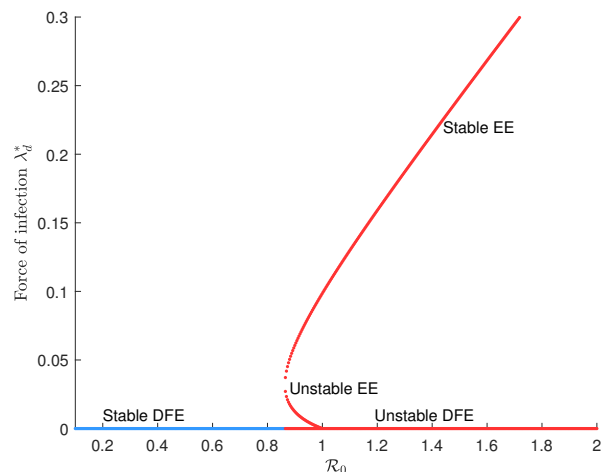


Fig. 2: Backward bifurcation diagram for model (1), showing that HIV can persist even when $\mathcal{R}_0 < 1$.

2) *Disease mortality (ν):* AIDS-related mortality is derived from UNAIDS [1] data, which reports approximately 7.5 million adults living with HIV in South Africa in 2023. Deaths due to AIDS among adults aged 15 years and older ranged from 4×10^3 to 6.3×10^4 annually. Thus, the disease mortality rate is taken as $\nu = [4 \times 10^3/7.5 \times 10^6, 6.3 \times 10^4/7.5 \times 10^6]$.

3) *Proportion of HIV individuals not accepting ART (ρ):* The proportion of HIV-positive adults who are in denial (i.e., not receiving ART) is based on UNAIDS [1] data indicating that 72–86% of adults aged 15 and older are receiving ART. Assuming that those not receiving ART are in denial, the proportion of individuals in denial is estimated to range from 16% to 28%. Thus, $\rho = [0.16, 0.28]$.

4) *Screening rate (α) and screening denial (ω):* The screening process is decomposed into two components: a screening invitation rate (K) and an acceptance probability (A). Hence, the effective screening and refusal (denial) rates are given by: $\alpha = KA$, $\omega = K(1 - A)$, where $1/K$ represents the average interval between successive testing opportunities, and A denotes the probability of accepting testing once invited.

Model-based estimates for sub-Saharan Africa indicate that the average time from HIV infection to diagnosis has decreased substantially over the past two decades, from nearly 10 years in 2000 to about 1.5 years in Southern Africa by 2020 [38]. We therefore set $1/K$ to range between 1.5 and 10 years, corresponding to $K \in [0.10, 0.67]$. Based on Mabuto et al. [39], 74–87% of adults accept HIV testing when offered, giving $A \in [0.74, 0.87]$. Substituting these ranges

Par.	Unit	Average [min, max]	Source
π	Humans year ⁻¹	8.8×10^5 [$8.5 \times 10^5, 9.3 \times 10^5$]	Estimated
β_d	Year ⁻¹	0.5 [0, 1]	[8]
β_n	Year ⁻¹	0.04 [0, 0.08]	Estimated
ϕ_n	Year ⁻¹	0.29 [0.23, 0.35]	Estimated
ϕ_d	Year ⁻¹	1.5 [1, 2]	[8]
ε_n	Year ⁻¹	0.125 [0, 0.25]	Assumed
ε_d	Year ⁻¹	0.125 [0, 0.25]	Assumed
μ	Year ⁻¹	0.022 [0.021, 0.023]	Estimated
α	Year ⁻¹	0.325 [0.07, 0.58]	Estimated
ρ	Dimens- ionless	0.22 [0.16, 0.28]	Estimated
δ_d	Year ⁻¹	0.1 [0, 0.2]	Assumed
δ_n	Year ⁻¹	0.1 [0, 0.2]	Assumed
γ_n	Year ⁻¹	0.061 [0.022, 0.1]	Estimated
γ_d	Year ⁻¹	0.084 [0.067, 0.1]	Estimated
ν	Year ⁻¹	0.007 [0.005, 0.008]	Estimated
ω	Year ⁻¹	0.095 [0.02, 0.17]	Estimated

Table 2: Model parameters: units, average values (minimum, maximum), and sources. The estimates are as in the text.

yields $\alpha \in [0.07, 0.58]$ and $\omega \in [0.02, 0.17]$.

5) *Progression rate of untreated to AIDS (γ_d):* The progression rate of untreated HIV-positive individuals to the AIDS stage is assumed to range between 10 and 15 years, based on WHO [36] guidelines. Consequently, the progression rate is given by: $\gamma_d = [1/15, 1/10]$.

6) *Progression rate of treated to AIDS (γ_n):* The progression rate for individuals on ART is assumed to range from 10 years (if ART fails) to 45.9 years (if ART is fully effective), with the difference representing the average life expectancy of 61.4 years minus the average age of first sexual debut, 15.5 years. Therefore, $\gamma_n = [1/45.9, 1/10]$.

7) *Measure of infectiousness for those not on ART (ϕ_d):* The amplification factor represents the increased risk of HIV transmission for individuals with symptomatic HIV infection. Based on the study by Nyabadza et al. [8], we assume that the amplification factor for individuals in denial (who are not on ART and have uncontrolled viral loads) lies within the range: $\phi_d = [1, 2]$.

8) *Measure of infectiousness for those on ART (ϕ_n):* UNAIDS [1] reports that between 65% and 77% of people living with HIV have suppressed viral loads due to ART. Therefore, the amplification factor for individuals on ART (those with suppressed viral loads) is assumed to range between 0.23 and 0.35. Thus, $\phi_n = [0.23, 0.35]$.

9) *Recruitment rate (π):* Based on South Africa's demographic data [36], the total population of individ-

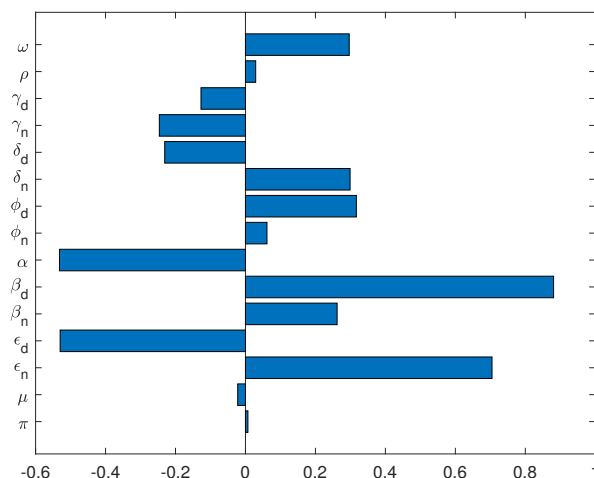


Fig. 3: Partial rank correlation coefficients for the model parameters.

uals aged 15 years and older is approximately 48.2 million, of whom about 7.5 million are living with HIV [1]. The susceptible population is therefore taken as approximately 40.7 million. Assuming demographic steady state, so that recruitment balances natural mortality to maintain a constant adult population, the recruitment rate is modelled as $\pi = \mu S = [8.5 \times 10^5, 9.3 \times 10^5]$, where S denotes the susceptible population.

10) *Transmission rate for those not in denial (β_n):* The transmission rate for individuals not in denial is related to the transmission rate for individuals in denial. It is assumed that the transmission rate for individuals not in denial is reduced by a factor of $(1 - \eta)$, where η represents the effectiveness of condom use. The effectiveness of condom use is taken to range from 0.9 to 0.94 [40]. Thus, the transmission rate is modelled as $\beta_n = (1 - \eta)\beta_d$.

These parameter estimates are summarised in Table 2. For parameters with limited data, values are assumed based on previous studies, as indicated in the table. The resulting $\mathcal{R}_0$ is calculated to be 1.6. When $\eta = 0$ or $\beta_n = \beta_d$, $\mathcal{R}_0 = 3.2$, which falls within the range of 2 to 5 as reported in the literature [41].

B. Sensitivity index

Sensitivity analysis is conducted to assess how variations in the parameters of the HIV transmission model impact the overall transmission dynamics, particularly the estimated basic reproduction number ($\mathcal{R}_0$). The Latin Hypercube Sampling (LHS) method is used to carry out the sensitivity analysis [42–44]. In applying LHS, a baseline value is established for each parameter

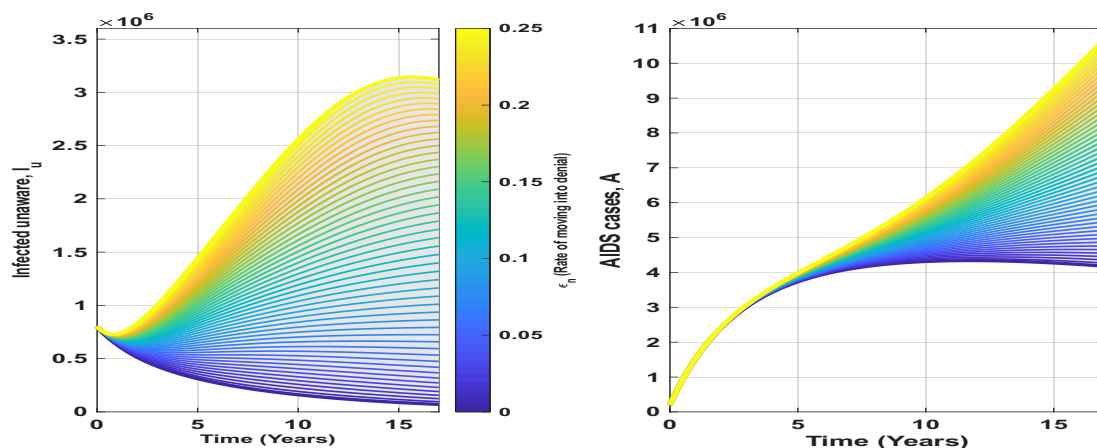


Fig. 4: Effects of denial, in the form of refusal to adopt preventive measures, on the number of new HIV infections and AIDS cases for $\varepsilon_n \in [0, 0.25]$. The remaining parameters are set to the average values listed in Table 2.

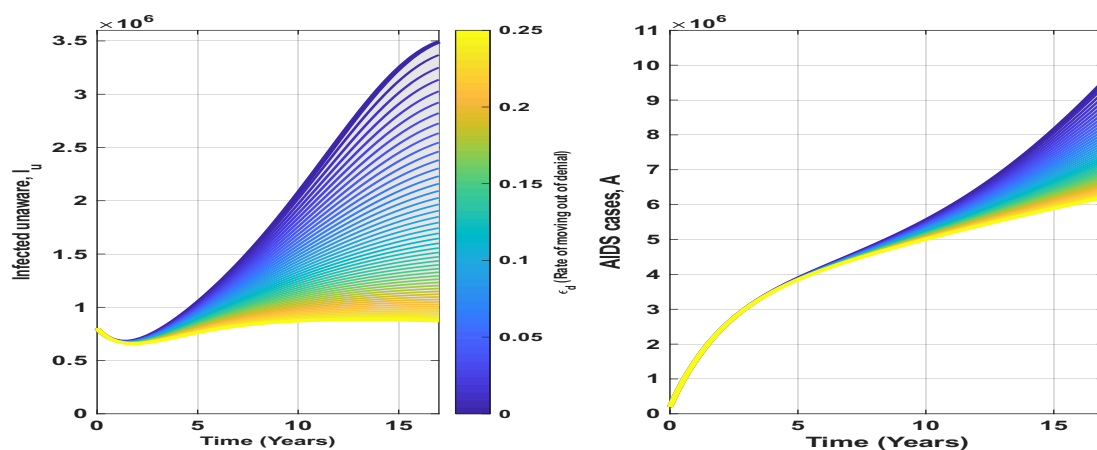


Fig. 5: Effects of dropping the denial behaviour of not using preventive measures on the number of new HIV infections and AIDS cases for $\varepsilon_d \in [0, 0.25]$. The remaining parameters are set to the average values listed in Table 2.

along with its corresponding range, followed by generating N distinct model runs to evaluate the outcome. As outcome parameter, we focus on $\mathcal{R}_0$ [42,43]. The value of N is determined to satisfy the condition $N > (4/3)K$, where K represents the number of uncertain parameters in the model [42,43]. It is assumed that the model parameters are uniformly distributed. The sensitivity of $\mathcal{R}_0$ to these parameter variations is quantified through the calculation of partial rank correlation coefficients (PRCCs) [42]. The sign of the PRCC determines the direction of the influence of a parameter on the output ($\mathcal{R}_0$), while its magnitude reflects the relative strength of that influence.

The LHS method is used to derive the PRCCs for the parameters of the HIV transmission model (Table 2) and depicted in Figure 3.

From Figure 3, it is evident that the parameters exerting the greatest influence on HIV transmission are the transmission rate within the susceptible denial group (β_d), the rate at which individuals transition into denialism (ε_n), the rate of disengagement from denialist behaviour (ε_d), and the screening rate (α). This indicates that the transmission risks from the susceptible denial group (S_d) are considerably higher than those from the infected denial group (I_d), since denialists often forgo protective measures. A lower PRCC value of 0.3 for β_n compared to 0.9 for β_d illustrates that effectively controlling denialism within the population can markedly reduce HIV transmission rates.

The parameters ε_n and ε_d highlight potential strategies for managing this high-risk group, while those associated with the infected denial group – specifically,

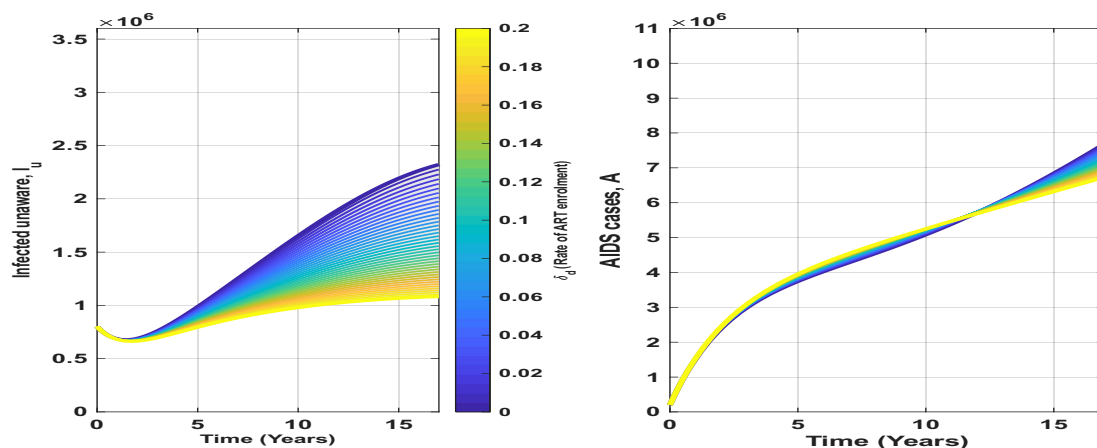


Fig. 6: Effects of accepting to join ART for those previously in denial on the number of new HIV infections and AIDS cases for $\delta_d \in [0, 0.2]$. The remaining parameters are set to the average values listed in Table 2.

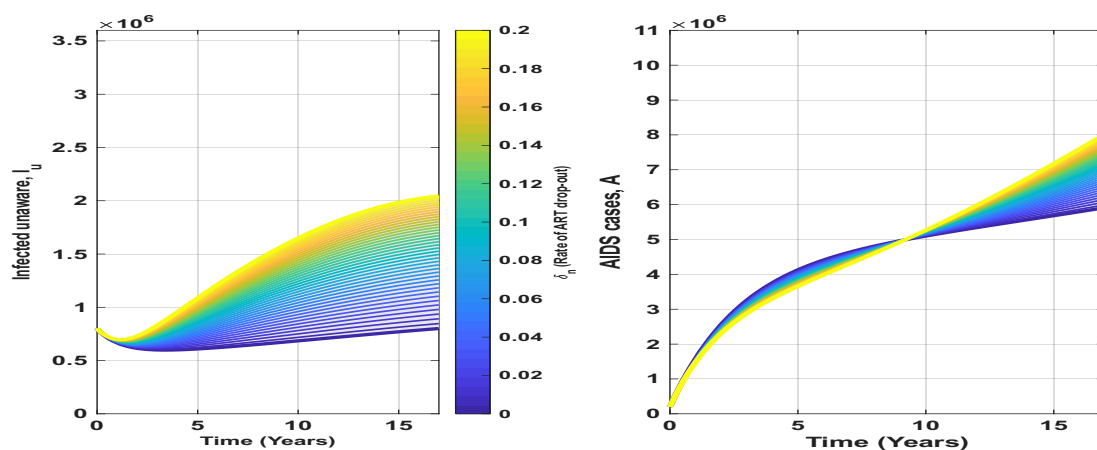


Fig. 7: Effects of ART discontinuation on the number of new HIV infections and AIDS cases for $\delta_n \in [0, 0.2]$. The remaining parameters are set to the average values listed in Table 2.

the rate of individuals dropping from ART to join the denial group (δ_n) and the rate at which individuals leave denialism to re-engage with ART (δ_d), also exert significant effects on disease dynamics. Notably, ε_d and δ_d have negative PRCC values, whereas the others are positive.

The high sensitivity of α underscores the pivotal role of early HIV diagnosis in epidemic control. Increased screening shortens the period during which individuals remain unaware of their infection, thereby reducing the duration of untreated, high-transmission states and limiting onwards spread. These results highlight that interventions directly targeting denial-related behaviours, such as reducing entry into denial and supporting re-engagement with care, could have a disproportionately large impact on epidemic control, especially when

complemented by intensified screening that promotes early diagnosis and treatment initiation.

In the following subsections, the effects of denial are further explored by analysing the numerical model solutions as the respective parameters vary within the ranges specified in Table 2. The simulations are assumed to begin in 2023, as most of the data used to derive the parameter values correspond to that year, with the final time set to 2040, covering a period of 17 years.

C. Effects of denial: refusal of preventive measures

As shown in Figure 4, denial of preventive measures can significantly increase both the number of new infections and the number of individuals progressing to the AIDS stage. In the absence of individuals joining the denial class, the annual incidence rate continues

to decline, along with the number of people reaching the AIDS stage. Denial of preventive measures directly undermines HIV control. Scaling up prevention campaigns, including condom promotion, HIV education, and stigma reduction, remains essential, particularly for high-risk denialist groups.

D. Effects of dropping the denial behaviour before infection

As observed in Figure 5, if individuals abandon the denial behaviour of not using preventive measures, both the incidence rate and the number of AIDS cases will significantly decrease. Encouraging individuals to abandon denialism early, through trust-building, counselling, and stigma reduction, can substantially cut HIV incidence, reinforcing the importance of behavioural and community-based interventions.

E. Effects of accepting to join ART for those previously in denial

As observed in Figure 6, when individuals who have been in denial accept to join ART, there is a reduction in both the number of new HIV infections and the number of people progressing to the AIDS stage. Expanding linkage-to-care and treatment re-engagement programmes targeting denialists could markedly reduce transmission, supporting South Africa's "Test and Treat All" policy.

F. Effects of ART discontinuation

As shown in Figure 7, discontinuation of ART in favour of joining the denial class leads to an increase in both the number of new HIV infections and the number of individuals progressing to the AIDS stage. Preventing ART discontinuation among denialists is critical for sustaining epidemic gains and achieving UNAIDS 95–95–95 targets. Interventions should prioritise adherence support, combating misinformation, and providing psychosocial support to minimise drop-out.

G. Overall system dynamics

To provide a complete picture of the model's long-term behaviour under denial-related dynamics, Figure 8 illustrates the trajectories of all compartments simulated over an extended period of 250 years. The 250-year simulation horizon is not intended as a realistic policy projection but rather to illustrate the asymptotic behaviour of the system and demonstrate convergence towards the endemic equilibrium predicted by the analytical results. The results indicate that the system

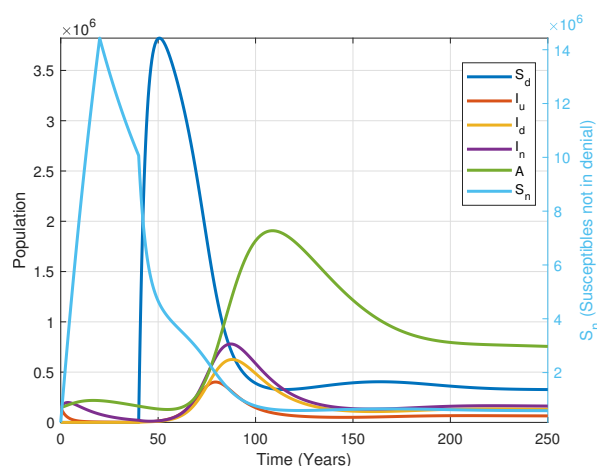


Fig. 8: Long-term dynamics of all model variables showing convergence towards endemic equilibrium. The parameter values used are given in Table 2.

eventually converges to an endemic steady state following pronounced transient oscillations across all sub-populations. In particular, the susceptible groups (S_n and S_d) exhibit large fluctuations before stabilisation, reflecting the delayed epidemiological and behavioural feedback caused by transitions into and out of denial.

These long-term dynamics reinforce the analytical results, demonstrating that denial can sustain HIV transmission for extended periods and delay epidemic stabilisation even when $\mathcal{R}_0$ is close to unity. The interplay between behavioural adaptation, treatment uptake, and denial-driven heterogeneity produces persistent epidemic behaviour that would not arise in homogeneous models.

VI. DISCUSSION

Despite substantial progress in expanding access to HIV testing and antiretroviral therapy (ART), HIV remains a major public health challenge in South Africa. A key barrier is denialism, expressed through reluctance to test, non-disclosure of status, and ART non-adherence [3]. This study highlights, through a mathematical modelling approach, how denialism alters epidemic trajectories and complicates elimination efforts.

Bifurcation analysis (Table 1) shows that the system may undergo a backward bifurcation, where disease-free and endemic equilibria coexist even when $\mathcal{R}_0 < 1$. This implies that reaching the classical threshold condition is not sufficient for elimination. Similar findings in other settings [9, 10] reinforce that conventional

biomedical interventions alone are inadequate in populations with entrenched denialism. Public health strategies must therefore pair ART scale-up with behaviour change interventions explicitly addressing denial.

The backward bifurcation observed in this model is primarily driven by the combined effects of transmission heterogeneity and treatment refusal among denialist individuals. In particular, high transmission rates within the denialist susceptible group (β_d), together with low ART uptake and increased ART discontinuation, allow a sufficiently large high-risk subpopulation to persist even when the average transmission potential falls below the classical threshold $\mathcal{R}_0 = 1$. This creates bistability, where both disease-free and endemic equilibria may coexist depending on the initial conditions. Epidemiologically, this implies that reducing transmission alone may be insufficient if denial-driven treatment avoidance remains prevalent within the population.

Sensitivity analysis (Figure 3) identifies the transmission rate among denialist susceptibles (β_d) as the most influential parameter. This finding emphasises the need for targeted education and communication efforts that promote safer sexual practices among individuals who avoid testing or reject diagnosis. Reducing β_d through stigma reduction and trust-building with health systems could substantially curb transmission.

Numerical results (Figures 4–5) demonstrate that flows into and out of denial (ε_n and ε_d) strongly shape epidemic dynamics. High rates of entry into denial drive HIV incidence upward, while high rates of exit support epidemic control. Interventions that reduce stigma, counter misinformation, and strengthen social support for disclosure and treatment-seeking are therefore central to epidemic mitigation.

The model further shows the dual impact of ART uptake and discontinuation (Figures 6–7). Sustained ART use lowers incidence and delays AIDS progression, while discontinuation among denialists reverses these gains. Sustaining ART adherence is thus critical to achieving the UNAIDS 95-95-95 targets [1]. Policies should not only promote ART initiation but also invest in adherence support, counselling, and targeted outreach to individuals at risk of denial-driven discontinuation.

A limitation of the present model is the assumption that transitions into and out of denial occur at constant rates. Although this approximation is reasonable in the context of the President Thabo Mbeki era, characterised by strong ideological commitment combined with top-down political and media influence, it does not fully capture other involved nonlinear behavioural processes. These might include a range of factors acknowledged

by the literature on coupled behavior-infection models e.g., peer imitation (that is, word-of-mouth), social network effects, prevalence-dependent risk perception or social norms [20–22, 26, 29–31]. Future work incorporating these mechanisms could enhance model realism and improve understanding of the interplay between denialism and HIV transmission dynamics, especially regarding trends in the epoch after President Mbeki.

Overall, these findings underscore that denialism is not a passive behavioural factor but an active driver of HIV transmission. Addressing denial requires embedding psychosocial and cultural interventions within existing biomedical programmes. For South Africa and other resource-constrained settings, this means expanding HIV services to include counselling, community-led education, and communication campaigns that directly counter denialist narratives. Future research should explore the long-term impact of various intervention strategies on mitigating denialism and assess how different sub-populations respond to targeted interventions. Understanding these dynamics could provide deeper insights into refining policies aimed at achieving sustainable HIV epidemic control in South Africa.

ACKNOWLEDGMENT

The authors warmly thank the editor and the anonymous referee of the journal for their valuable comments and constructive suggestions, which greatly improved the quality and clarity of this manuscript.

REFERENCES

- [1] UNAIDS, South Africa Results and Transparency Portal, 2024, [24 July 2026], <https://aidsinfo.unaids.org/>
- [2] S. C. Kalichman, The Psychology of AIDS Denialism, *European Psychologist*, 19:13–22, 2014.
- [3] S. C. Kalichman, L. Eaton, C. Cherry, “There is no proof that HIV causes AIDS”: AIDS denialism beliefs among people living with HIV/AIDS, *Journal of Behavioral Medicine*, 33:432–440, 2010.
- [4] N. Nattrass, Still Crazy After All These Years: The Challenge of AIDS Denialism for Science, *AIDS and Behavior*, 14:248–251, 2010.
- [5] P. Chigwedere, M. Essex, AIDS Denialism and Public Health Practice, *AIDS and Behavior*, 14:237–247, 2010.
- [6] R. M. Anderson, R. M. May, *Infectious Diseases of Humans: Dynamics and Control*, Oxford University Press, 1991.
- [7] M. J. Keeling, P. Rohani, *Modeling Infectious Diseases in Humans and Animals*, Princeton University Press, 2011.
- [8] F. Nyabadza, C. Chiyaka, Z. Mukandavire, S. D. Hove-Musekwa, Analysis of an HIV/AIDS model with public-health information campaigns and individual withdrawal, *Journal of Biological Systems*, 18:357–375, 2010.
- [9] T. Kaisara, F. Nyabadza, Modelling Botswana’s HIV/AIDS response and treatment policy changes: Insights from a cascade of mathematical models, *Mathematical Biosciences and Engineering*, 20:1122–1147, 2023.

- [10] K. Afassinou, F. Chirove, K. S. Govinder, Pre-exposure prophylaxis and antiretroviral treatment interventions with drug resistance, *Mathematical Biosciences*, 285:92–101, 2017.
- [11] B. Nannyonga, J. Y. T. Mugisha, L. S. Luboobi, The role of HIV positive immigrants and dual protection in a co-infection of Malaria and HIV/AIDS, *Applied Mathematical Sciences*, 5:2919–2942, 2011.
- [12] H. Nampala, L. S. Luboobi, J. Y. T. Mugisha, C. Obua, M. Jablonska-Sabuka, Modelling hepatotoxicity and antiretroviral therapeutic effect in HIV/HBV coinfection, *Mathematical Biosciences*, 302:67–79, 2018.
- [13] J. Y. T. Mugisha, L. S. Luboobi, Modelling the effect of vertical transmission in the dynamics of HIV/AIDS in an age-structured population, *The South Pacific Journal of Natural and Applied Sciences*, 21:82–90, 2003.
- [14] F. Nyabadza, Z. Mukandavire, S. D. Hove-Musekwa, Modelling the HIV/AIDS epidemic trends in South Africa: Insights from a simple mathematical model, *Nonlinear Analysis: Real World Applications*, 12:2091–2104, 2011.
- [15] T. B. Hallett, J. M. Baeten, R. Heffron, R. Barnabas, G. de Bruyn, Í. Cremin, et al., Optimal Uses of Antiretrovirals for Prevention in HIV-1 Serodiscordant Heterosexual Couples in South Africa: A Modelling Study, *PLOS Medicine*, 8:e1001123, 2011.
- [16] J. W. Eaton, L. F. Johnson, J. A. Salomon, T. Barnighausen, E. Bendavid, A. Bershteyn, et al., HIV Treatment as Prevention: Systematic Comparison of Mathematical Models of the Potential Impact of Antiretroviral Therapy on HIV Incidence in South Africa, *PLOS Medicine*, 9:e1001245, 2012.
- [17] J. W. Eaton, N. Bacaër, A. Bershteyn, V. Cambiano, A. Cori, R. E. Dorington, et al., Assessment of epidemic projections using recent HIV survey data in South Africa: a validation analysis of ten mathematical models of HIV epidemiology in the antiretroviral therapy era, *The Lancet Global Health*, 3:e598–e608, 2015.
- [18] B. Jewell, J. Smith, T. Hallett, The Potential Impact of Interruptions to HIV Services: A Modelling Case Study for South Africa, medRxiv 2020.04.22.20075861, 2020.
- [19] R. V. Barnabas, A. A. Szpiro, X. Ntinga, M. L. Mugambi, H. van Rooyen, A. Bruce, et al., Fee for home delivery and monitoring of antiretroviral therapy for HIV infection compared with standard clinic-based services in South Africa: a randomised controlled trial, *The Lancet HIV*, 9:e848–e856, 2022.
- [20] P. Manfredi, A. D’Onofrio, *Modeling the Interplay Between Human Behavior and the Spread of Infectious Diseases*, Springer, New York, 2013.
- [21] Z. Wang, C. T. Bauch, S. Bhattacharyya, A. d’Onofrio, P. Manfredi, M. Perc, et al., Statistical physics of vaccination, *Physics Reports*, 664:1–113, 2016.
- [22] F. Verelst, L. Willem, P. Beutels, Behavioural change models for infectious disease transmission: a systematic review (2010–2015), *Journal of the Royal Society Interface*, 13:20160820, 2016.
- [23] O. O. Oguntibeju, Quality of life of people living with HIV and AIDS and antiretroviral therapy, *HIV/AIDS - Research and Palliative Care*, 4:117–124, 2012.
- [24] W. Jia, K. Jiao, J. Ma, M. Liao, C. Wang, D. Kang, et al., HIV infection disclosure, treatment self-efficacy and quality of life in HIV-infected MSM receiving antiretroviral therapy, *BMC Infectious Diseases*, 22:937, 2022.
- [25] South Africa National Department of Health, 2023 ART clinical guidelines for the management of HIV in adults, pregnancy and breastfeeding, adolescents, children, infants and neonates, 2023, [24 July 2026], https://knowledgehub.health.gov.za/system/files/elibdownloads/2023-05/National_ART_Clinical_Guideline_2023_04_28_signed_0.pdf
- [26] F. M. Bass, A New Product Growth for Model Consumer Durables, *Management Science*, 15:215–227, 1969.
- [27] M. Mbali, AIDS Discourses and the South African State: Government denialism and post-apartheid AIDS policy-making, *Transformation: Critical Perspectives on Southern Africa*, 54:104–122, 2004.
- [28] J. Wang, AIDS denialism and ‘The humanisation of the African’, *Race & Class*, 49:1–18, 2008.
- [29] C. T. Bauch, Imitation dynamics predict vaccinating behaviour, *Proceedings of the Royal Society B: Biological sciences*, 272:1669–1675, 2005.
- [30] S. Funk, M. Salathé, V. A. A. Jansen, Modelling the influence of human behaviour on the spread of infectious diseases: a review, *Journal of the Royal Society Interface*, 7:1247–1256, 2010.
- [31] A. d’Onofrio, P. Manfredi, P. Poletti, The Interplay of Public Intervention and Private Choices in Determining the Outcome of Vaccination Programmes, *PLOS ONE*, 7:e45653, 2012.
- [32] O. Diekmann, J. A. P. Heesterbeek, *Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis and Interpretation*, John Wiley & Sons, 2000.
- [33] P. van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Mathematical Biosciences*, 180:29–48, 2002.
- [34] A. B. Gumel, Causes of backward bifurcations in some epidemiological models, *Journal of Mathematical Analysis and Applications*, 395:355–365, 2012.
- [35] O. Sharomi, C. N. Podder, A. B. Gumel, B. Song, Mathematical analysis of the transmission dynamics of HIV/TB coinfection in the presence of treatment, *Mathematical Biosciences and Engineering*, 5:145–174, 2008.
- [36] WHO Data, South Africa country overview, 2024, [24 July 2026], <https://data.who.int/countries/710>
- [37] L. Richter, S. A. Norris, J. Ramjith, Early sexual debut : voluntary or coerced? Evidence from longitudinal data in South Africa - the Birth to Twenty Plus study, *South African Medical Journal*, 105:304–307, 2015.
- [38] K. Giguère, J. W. Eaton, K. Marsh, L. F. Johnson, C. C. Johnson, E. Ehui, et al., Trends in knowledge of HIV status and efficiency of HIV testing services in sub-Saharan Africa, 2000–20: a modelling study using survey and HIV testing programme data, *The Lancet HIV*, 8:e284–e293, 2021.
- [39] T. Mabuto, B. Hansoti, D. Kerrigan, N. Mshweshwe-Pakela, G. Kubeka, S. Charalambous, C. Hoffmann, HIV testing services in healthcare facilities in South Africa: a missed opportunity, *Journal of the International AIDS Society*, 22:e25367, 2019.
- [40] S. D. Pinkerton, P. R. Abramson, M. E. Turk, Updated estimates of condom effectiveness, *Journal of Association of Nurses in AIDS Care*, 9:88–89, 1998.
- [41] NETEC, Playing the Numbers Game: R_0 , 2020, [24 July 2026], <https://netec.org/2020/01/30/playing-the-numbers-game-r0/>
- [42] S. M. Blower, H. Dowlatbadi, Sensitivity and Uncertainty Analysis of Complex Models of Disease Transmission: An HIV Model, as an Example, *International Statistical Review*, 62:229–243, 1994.
- [43] M. D. McKay, R. J. Beckman, W. J. Conover, Comparison of Three Methods for Selecting Values of Input Variables in the Analysis of Output from a Computer Code, *Technometrics*, 21:239–245, 1979.
- [44] A. Saltelli, S. Tarantola, F. Campolongo, M. Ratto, *Sensitivity Analysis in Practice: A Guide to Assessing Scientific Models*, John Wiley & Sons, 2004.