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A Scenario-Based Mathematical Modeling of HIV Control in Nigeria Integrating Superinfection, Drug Resistance, and Future Vaccine Introduction

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ABSTRACT

This study develops a mathematical model of HIV transmission in Nigeria to assess how scaling up antiretroviral therapy, promoting behavioral change, and managing drug resistance could shape epidemic outcomes, while also exploring the potential added impact of introducing a future HIV vaccine. The model incorporates eight interconnected compartments representing susceptible, infected with primary and secondary infections, treated individuals, drug-resistant cases, vaccinated, recovered with partial immunity, and behavioral intervention. Parameterized with epidemiological data for Nigeria, the model explores Nigerian epidemic pattern under distinct and combined control measures. Simulation results show that early and high-coverage vaccination, specifically with high-efficacy and slow waning, reduces both primary and secondary infections. ART coverage is observed to be critical in curbing the resistant strain, while behavioral reinforcement amplifies the effectiveness of biomedical interventions. However, scenarios with poor ART adherence or high superinfection potential reveal resurgence in resistant infections, emphasizing the danger posed by superinfection. Combined strategy simulations produced the most significant and sustained reductions in HIV prevalence and resistance burden. The findings in this study underscore the importance of coordinated, multi-pronged strategies for HIV control in Nigeria. The model offers a valuable policy tool for evaluating trade-offs between intervention options and guiding data-driven public health planning.

1. Introduction

Understanding the potential impact of HIV vaccine introduction in a population with existing control strategies such as drug therapy, drug resistance and behavioural interventions, and management requires a systematic exploration of future possibilities (Bartsch *et al*., 2020). Scenario analysis is a useful approach in mathematical epidemiology that helps researchers and policymakers to simulate and compare the results of various intervention strategies under controlled assumptions (Garske *et al.*, 2022; Chakraborty *et al.*, 2022). In Nigeria, where Human Immunodeficiency Virus (HIV) persists, with no vaccine, mathematical modeling provides a significant tool to study the long-term results of intervention strategies and guide public health decisions (Garske *et al*., 2022; Silal *et al*., 2024).

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HIV is a virus which causes Acquired Immune Deficiency Syndrome (AIDS), a disease that affects the immune system of humans, making it vulnerable to other opportunistic infections if not treated (Centers for Disease Control and Prevention, 2024; World Health Organization, 2023). HIV remains a significant health threat, especially in Africa with approximately 26.3 million persons living with the virus (PLWH), accounting for about 65% of all world cases (World Health Organization, 2025). Nigeria alone has an approximate figure of 2 million PLWH (Onyedika-Ugoeze & Adegoke, 2025). As at today, there is no licensed vaccine for HIV (NIH HIVinfo, 2024; HIV.gov, 2024). However, researches are ongoing to understand immune correlates of protection and to leverage on new technologies as to overcome setbacks on previous efforts on HIV vaccines development (IAVI & Scripps Research, 2025; National Institute of Allergy and Infectious Diseases, 2023).

Current studies advancing HIV vaccine development, such as mRNA delivery platforms, germline-targeting immunogens, and mosaic antigen design, has marked a significant shift in the journey of HIV research as discussed in the studies of Shim *et al*. (2025), Boomgarden and Upadhyay (2025), Govindan and Stephenson (2024), and Scott and Worku (2024). While their works and findings bring new advances in HIV vaccines research, their successful transition from laboratory to public health impact requires more than just biological approach (Anderson & Garnett, 2000; Eaton *et al.*, 2012). This is where mathematical modeling becomes an indispensable tool. Mathematical models are important tools for examining the relationship between vaccination, drug resistance, partial immunity and behavioural interventions, for simulating HIV vaccine rollout effects on the population, and predicting HIV vaccine long-term impacts on HIV transmission (Anderson & Garnett, 2000). In settings like Nigeria where the HIV burden remains significantly high, and clinical trials are scarce, mathematical modeling affords the chance to explore "what-if" scenarios, providing insight into uncertain conditions and thereby, guiding policy decisions in the face of future eventualities. By integrating epidemiological, demographic and health system data, coupled with hypothetical vaccine data, models can be developed to identify potential vaccination strategies and forecast threshold for herd immunity.

Vaccines have become a viable strategy in disease control, prevention and transmissions (Eguavoen et al., 2023). The HIV vaccine is proposed in order to enhance the individual immune system, which is expected to significantly inhibit the HIV progression rate (Omale, 2021). In the absence of specific vaccine to inhibit HIV, ART is being used to strengthen the immune system (Rasheed *et al*., 2025).

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A mathematical model for HBV and HIV was developed by Rasheed *et al*., (2025). The study incorporated vertical transmission and intervention measures such as vaccine. Upon simulation, their result showed that an increase in vaccine rate and/or vaccine efficacy leads to a reduction in disease prevalence.

Omale (2021) formulated a non-linear deterministic epidemic model on the control of HIV/AIDS with the incorporation of campaign on vaccination and therapy. A vaccination class was incorporated in the model diagram. This was done in order to study the effect of public health campaign on vaccination and treatment for HIV/AIDS control within a population. Their model analysis showed that public health campaign on vaccination reduces HIV transmission.

Eguavoen *et al*. (2023) used a mathematical model to study the effect of vaccination on infectious diseases dynamics. An SEIR epidemic model was developed. Upon analysis based on existing theories, vaccination of population before disease outbreak reduces transmission rate of an epidemic. On the other hand, vaccination of the population after an outbreak of epidemic increases recovery rate. The work recommended vaccination before disease outbreak called pre-exposure prophylaxis in endemic areas of particular infectious disease.

Current advances in HIV vaccine development have stirred renewed efforts to use mathematical modeling to study the future impact of HIV vaccines. These models forecast population-level impact, helping to optimize rollout plans under several behavioural and epidemiological assumptions. In the next discussions, we examine key findings from recent modeling works, along with how this current study advances this line of research.

Dimitrov *et al*. (2021), formulated a model specifically calibrated to South African HIV data to evaluate the population-level impact of a partially effective vaccine of HIV. In their work, they examined different scenarios by varying vaccine potency, coverage, booster adherence, and behavioural evolutions. They adopted an individual-based model, called EMO-HIV v2.5 to simulate different scenarios. The results of their findings indicate that even vaccines with modest potency could cause a significant decline in HIV incidence when vaccination is implemented with early rollout and strong booster adherence. However, their work only captures South Africa and does not consider the possibility of superinfection. The role of partial immunity, and drug resistance were also ignored.

In another related study, Gilbert *et al*. (2016), proposed a model that highlighted the use of modeling in optimizing HIV vaccine trial designs, specifically in identifying immune correlates of protection and adapting to shifting prevention baselines. Theirs was a mix of mathematical and statistical models to design

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future vaccine trials. Though their model was conceptually robust, the study is not empirical and does not capture epidemiological movements such as superinfection, treatment failures and behavioural evolution.

Recent works have begun examining region-based modeling, though there is sparse literature specifically modeling the Nigerian context. Several studies in the recent have embraced the use of system dynamics and compartmental models to Africa, examining vaccine rollout strategies under existing treatment programs. However, these studies rarely include partial immunity, resistance evolution and superinfection in their studies. The current work incorporates major epidemiological factors, in a combined structure not seen in previous models, including drug resistance, partial immunity, treatment coverage, behavioural intervention and HIV superinfection. It also integrates future vaccine, specially applied to Nigeria's context.

2. Methods

In this section, we present, discuss and analyse a futuristic HIV superinfection-vaccine model. Methods used in the study were also discussed.

2.1 Model description and development

The model incorporates major epidemiological dynamics including HIV superinfection, treatment coverage, partial immunity, behavioural intervention, drug resistance and introduction of future vaccine, specifically tailored towards the Nigeria context. The model comprise eight compartments of ordinary differential equations (ODEs) namely, susceptible population (S), vaccinated population (V), population infected with primary strain (I_1), superinfected population (I_2), drug-resistant infected population (I_R), treated population (T), recovered population with partial immunity (R) and behavioural intervention level (B).

The susceptible compartment comprises individuals who are yet to be infected with HIV and are not vaccinated. Individuals are recruited into the S compartment through a constant recruitment rate Λ, and leaves through natural death μ, vaccination v or infection by any of the infectious individuals from compartments I_1, I_2 and I_R, with a modulated-infection pressure by behavioural change through $(1-\kappa B)$. The vaccinated populations are individuals who take a hypothetical HIV vaccine and have partial immunity against the virus. The compartment grows at the rate v from recruitment of vaccinated individuals from S compartment. They loss immunity over time at the rate ω_v, get infected via breakthrough infection with minimal susceptibility σ_v, or leave through natural death μ.

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The I_1 compartment consists of populations who have been infected by the primary strain of HIV. Individuals move into the compartment through infection of S, R, or V individuals with the primary strain. Individuals in this compartment may initiate treatment at the rate θ, die from HIV-related diseases at δ_1 and natural death at μ, or become superinfected with the second strain at the rate η. Individuals in the I_2 compartment were initially infected with the first strain and later infected with the second strain of HIV. They leave through ART initiation at the rate θ, or HIV-related and naturally death, δ_2 and μ respectively. The drug-resistant infected compartment includes individuals infected with HIV strains but who have developed resistance to ART. Individuals enter the compartment either from direct contact with HIV or as a result of ART failure at the rate ω. Individuals move out of the compartment through HIV-related death δ_R or natural death μ.

The treatment compartment contains the population of those undergoing ART. Individuals enter from both I_1 and I_2 compartments at the rate θ. They develop resistance and transit to I^R compartment at the rate ω. They recover partially and proceed to R compartment at rate ρ, or die naturally or from disease at the rate $(\mu + \delta_t)$. The recovered individual with partial immunity compartment consist those who partially recover immune function as a result of ART. They move into the compartment at rate ρ and leaves through natural death μ or are re-infected at a minimal susceptibility σ. The behavioural intervention compartment was introduced to track the level of behavioural change in the population that reduces the transmission of the disease. This level increases through public health intervention ψ and decay due to fatigue or behavioral relapse ξ. Effective contact rates are reduced by higher value of B through the factor $(1 - \kappa B)$.

We define the per-capita force of infection as the combined hazard contributed by the three infectious classes:

$$\lambda(t) = \big(1 - \kappa B(t)\big)\big(\beta_1 I_1(t) + \beta_2 I_2(t) + \beta_R I_R(t)\big)$$

where β_1, β_2 and β_R are strain-specific transmission coefficients, I_1, I_2 and I_R are the prevalence (proportions) of infectious individuals, $B(t)$ is the behavioural-intervention level and κ its transmission-reducing effect. New infections occur at rates λS for susceptible, $\sigma_v \lambda V$ for vaccinated individuals and $\sigma \lambda R$ for recovered individuals; σ_v and σ represent reduced susceptibility (partial immunity) of vaccinated and recovered persons respectively. The schematic diagram showing movements from one compartment to the other is presented in Figure 1.

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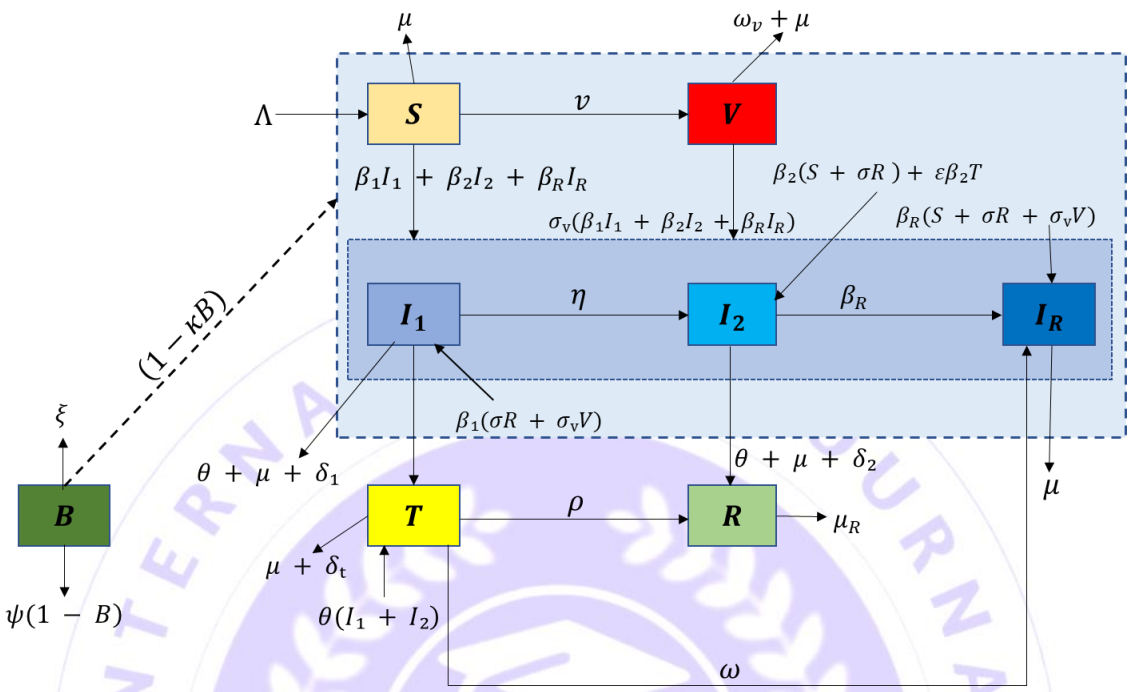

Figure 1: Schematic representation of the HIV transmission model in Nigeria integrating superinfection, drug resistance, behavioral intervention, treatment, and future vaccine introduction

Based on the descriptions and assumptions above, we present an HIV model described by a system of eight ODEs, incorporating a futuristic vaccination control as shown in Eqs. (1)-(8).

$$\frac{dS}{dt} = \Lambda - \nu S - (1 - \kappa B)(\beta_1 S I_1 + \beta_2 S I_2 + \beta_R S I_R) - \mu S \quad (1)$$

$$\frac{dV}{dt} = \nu S - \omega_v V - (1 - \kappa B)\sigma_v(\beta_1 V I_1 + \beta_2 V I_2 + \beta_R V I_R) - \mu V \quad (2)$$

$$\frac{dI_1}{dt} = (1 - \kappa B)\beta_1(S + \sigma R + \sigma_v V)I_1 - (\theta + \mu + \delta_1)I_1 - \eta I_1 I_2 \quad (3)$$

$$\frac{dI_2}{dt} = \eta I_1 I_2 + (1 - \kappa B)[\beta_2(S + \sigma R + \sigma_v V)I_2 + \varepsilon\beta_2 T I_2] - (\theta + \mu + \delta_2)I_2 \quad (4)$$

$$\frac{dI_R}{dt} = \omega T + (1 - \kappa B)\beta_R(S + \sigma R + \sigma_v V)I_R - (\mu + \delta_R)I_R \quad (5)$$

$$\frac{dT}{dt} = \theta(I_1 + I_2) - (\mu + \delta_t + \rho + \omega)T \quad (6)$$

$$\frac{dR}{dt} = \rho T - \mu R \quad (7)$$

$$\frac{dB}{dt} = \psi(1 - B) - \xi B \quad (8)$$

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Eqs. (1)-(8) are subject to the following initial conditions: $S(0) > 0, V(0) \geq 0, I_1(0) \geq 0,\ I_2(0) \geq 0,\ I_R(0) \geq 0, T(0) \geq 0, R(0) \geq 0$ and $B(0) \geq 0$.

2.1.1 Feasibility and positivity of the model

Theorem 2.1: Let $\mathcal{H} = \{S,\ V,\ I_1,\ I_2,\ I_R,\ T, R, B \in \mathbb{R}_+^8\}$ denote the feasible region of the HIV model (1)-(8). Then, $\mathcal{H}$ is positively invariant and attracting. In addition, for $S(0) > 0, V(0) \geq 0, I_1(0) \geq 0,\ I_2(0) \geq 0, I_R(0) \geq 0, T(0) \geq 0, R(0) \geq 0$ and $B(0) \geq 0$ in $\mathcal{H}$, the solutions of the model remain non-negative for all time $t > 0$.

Proof: We show that all the state variables in each of the ODEs in model (1)-(8) remain non-negative for all time $t > 0$, given that they start with non-negative initial conditions. To do this, we apply the method of integrating factor (IF) to each equation of model (1)-(8).

Recall the first equation, representing susceptible population S:

$$\frac{dS}{dt} = \Lambda - \nu S - (1 - \kappa B)(\beta_1 S I_1 + \beta_2 S I_2 + \beta_R S I_R) - \mu S$$

$$\Longrightarrow \frac{dS}{dt} = \Lambda - \lambda_S S - \nu S - \mu S \tag{9}$$

where $\lambda_S = (1 - \kappa B)(\beta_1 S I_1 + \beta_2 S I_2 + \beta_R S I_R)$ (10)

From Eq. (9), we take $\theta_S = \lambda_S + \nu + \mu$, as the total loss term, so that Eq. (9) becomes,

$$\frac{ds}{dt} + \theta_S S = \Lambda \tag{11}$$

The IF for Eq. (11) is given as $e^{\int \theta_S dt}$. Multiply Eq. (11) by IF. This yield:

$$e^{\int \theta_S dt}\frac{ds}{dt} + \theta_S S = e^{\int \theta_S dt}\Lambda e^{\int \theta_S dt} \tag{12}$$

$$\frac{d}{dt}\left(S e^{\int \theta_S dt}\right) = \Lambda e^{\int \theta_S dt} \tag{13}$$

Integrating both sides of Eq. (13) gives:

$$S(t)e^{\int_0^t \theta_S(\tau)d\tau} = S(0) + \int_0^t \Lambda e^{\int_0^s \theta_S(\tau)d\tau} ds$$

$$\Longrightarrow S(t) = e^{-\int_0^t \theta_S(\tau)d\tau}\left[S(0) + \int_0^t \int_0^t \Lambda e^{\int_0^s \theta_S(\tau)d\tau} ds\right] \geq 0 \tag{14}$$

Obviously, $\Lambda \geq 0,\ \theta_S \geq 0$, and $S(0) \geq 0$ (from definition), hence we conclude that $S(t)$ for all time $t \geq 0$. Following similar procedures as in Eqs. (9) - (14), it can be shown that $V(t) \geq 0,\ I_1(t) \geq 0,\ I_2(t) \geq 0,$

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$I_R(t) \geq 0, T(t) \geq 0, R(t) \geq 0$ and $B(t) \geq 0$ for all time $t \geq 0$. Since we have established that each state variable has a solution expressed as non-negative terms, we conclude that all solution starting from $\mathcal{H}$ remain non-negative for all time $t \geq 0$. Thus, the model is feasible and biologically meaningful.

2.2 Equilibrium States of the Model

In this section, we discuss the states of equilibrium of the HIV model (1)-(8). The model has two equilibrium states, namely the disease-free equilibrium (DFE) and the endemic equilibrium (EE) states.

2.2.1 Disease-free equilibrium DFE

The DFE is the state where there is no HIV infection in the population. All compartments associated with infection are zero, hence they cancel out. We denote the DFE state by $\boldsymbol{\mathcal{E}_0}$. Hence, $I_1^* = I_2^* = I_R^* = T^* = R^* = 0$, and $S^* > 0, V^* \geq 0,\ B^* \geq 0$.

$$\boldsymbol{\mathcal{E}_0} = \left(\frac{\Lambda}{\omega+\mu} \cdot \frac{\omega\Lambda}{(\omega+\mu)(\mu+\sigma)}, 0, 0, 0, 0, 0, \frac{\psi}{\psi+\xi}\right) \tag{15}$$

$\boldsymbol{\mathcal{E}_0}$ represents a population that is completely healthy where infection has not invaded or has been eliminated from human population. It helps in analyzing the criteria for disease invasion through a threshold called the basic reproduction number. Moreover, stability analysis around $\boldsymbol{\mathcal{E}_0}$ helps to determine whether the disease can be controlled or eradicated under the current strategies.

2.2.2 Endemic equilibrium EE

We denote the endemic equilibrium state by $\boldsymbol{\mathcal{E}_1}$. This is the state where infection persists in the population at constant levels over time. All compartments at $\boldsymbol{\mathcal{E}_1}$ are non-zero and constant. That is, $S^* > 0, V^* > 0, I_1^* > 0, I_2^* > 0, I_R^* > 0, T^* > 0, R^* > 0, B^* > 0$. Equating equations of model (1)-(8) to zero and solving the resulting system simultaneously yields:

$$\boldsymbol{\mathcal{E}_1} = \left(\frac{\Lambda}{v+\mu+B^*(\beta_1 I_1^* + \beta_2 I_2^* + \beta_R I_R^*)}, \frac{vS^*}{\mu+\omega_v+B^*\sigma_v(\beta_1 I_1^* + \beta_2 I_2^* + \beta_R I_R^*)}, I_1^*, I_2^*, T^*, R^*, B^*\right) \tag{16}$$

where,

$$I_1^* = \frac{\theta+\mu+\delta_2-(1-\kappa B^*)[\beta_2(S^*+\delta R^*+\sigma_v V^*)+\varepsilon\beta_2 T^*]}{\eta}$$

$$I_2^* = \frac{(1-\kappa B^*)\beta_1(S^*+\delta R^*+\sigma_v V^*)-(\theta+\mu+\delta_2)}{\eta},\ T^* = \frac{\theta(I_1^*+I_2^*)}{\mu+\delta_T+\rho+\omega},\ \ R^* = \frac{\rho\theta(I_1^*+I_2^*)}{\mu(\mu+\delta_T+\rho+\omega)}$$

$$I_R^* = \frac{\omega T^*}{(\mu+\delta_R)-(1-\kappa B^*)\beta_R(S^*+\delta R^*+\sigma_v V^*)},$$

$\boldsymbol{\mathcal{E}_1}$ describes a long-term equilibrium state where HIV infection continues to grow even in the presence of control measures. It provides further understanding on HIV epidemic level, the impact of drug resistance,

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vaccination plans and behavioural change. Stability analysis of the HIV endemic states provides information that helps to determine whether the infection remains or transition to eradication.

Table 1 and Table 2 present a summary of parameters and state variables used in this study for quick comprehension. The values and sources of these variables and parameters were also provided.

Table 1: Summary of parameters used and their values

Symbol	Description	Value	Source
Λ	Recruitment/birth rate	0.00005 per person/day (birth rate ≈ 33 per 1000/year)	World Bank (2025)
μ	Natural death rate	0.00005 per person/day (death rate ≈ 10 per 1000/year)	World Bank (2025)
β_1	Contact rate (primary infection)	0.05 per person/day (Calibrated from $R_0 \approx 2$ for HIV)	Granich et al. (2009)
β_2	Contact rate (superinfection)	0.045 per person/day	Koelle & Rasmussen (2021)
β_R	Contact rate (drug-resistant)	0.03 per person/day	Castro et al. (2017)
ν	Vaccination rate	0 per person/day	WHO. (2025b)
ω_v	Vaccine waning rate	0 per person/day	WHO. (2025b)
σ_v	Vaccine efficacy	0%	WHO. (2025b)
σ	Partial immunity modifier from R	0.5 (dimensionless)	Conway (2018)
η	Superinfection rate modifier	0.0025 per person²/day	Nowak et al. (1990)
ε	Infectivity of treated individuals	1 (dimensionless)	Eaton et al. (2012)
κ	Behavioral intervention impact	0.5 (dimensionless)	Rhodes & Simic (2020).
θ	ART initiation rate	0.001 per person/day	Fitted to ART coverage (~77%)
ρ	Recovery rate (immune restoration)	0.0001 per person/day	Mendicino et al. (2021).

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ω	Resistance emergence rate	0.0005 per person/day	WHO. (2023b)
δ_1	Death rate from primary infection	0.0002 per person/day	CDC (2023)
δ_2	Death rate from superinfection	0.00025 per person/day	Whiley et al. (2014)
δ_R	Death rate from drug-resistant infection	0.0003 per person/day	Feder (2021)
δ_t	Death rate while on treatment	0.0001 per person/day	WHO. (2023b)
ψ	Rate of behavioral gain	0.01 per day	Mavedzenge et al. (2019)
ξ	Loss rate of behavior change	0.005 per day	Gillman et al. (2022)

Table 2: Summary of variables used and their values

Symbol	**Description**	**Value**	**Source**
$S(0)$	Susceptible population	234,444,000	CDC (2025)
$V(0)$	Vaccinated population	0	WHO. (2025b)
$I_1(0)$	Primary infected population	1,660,000	CDC (2025); AIDSResTher, (2025)
$I_2(0)$	Superinfected population	118,800	Smith & Koelle (2024)
$I_R(0)$	Drug-resistant infected population	118,800	WHO. (2023a)
$T(0)$	Treated population (on ART)	1,460,000	CDC (2025); WHO (2025b)
$R(0)$	Recovered (partial immunity)	0	Douek et al. (2009)
$B(0)$	Behavioral intervention level	0.3 (dimensionless)	Bekker et al. (2022)
N	Total population	237,528,000	Macrotrends (2025), Worldometer (2025)

3. Numerical Simulation and Discussion of Results

In this study, we employ scenario-based simulations to examine the impact of future vaccination rollout, spread of drug resistance, and improvements in behavioural modifications on the long-term dynamics of HIV. The scenarios are implemented using R software and parametrized forms of the model (1)-(8) tailored to Nigeria's epidemiological data and patterns. We consider different vaccination rollout

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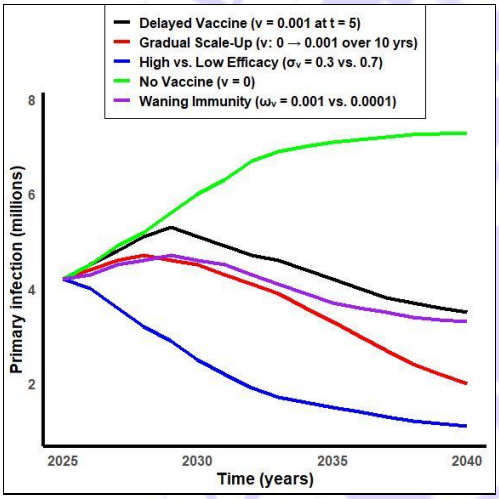
scenarios under different vaccine coverage rates and efficacy levels. Drug-resistance scenarios assess the increasing proportion of treatment failures due to resistant strains; while behavior-improvement scenarios focus on enhanced preventive measures that reduce transmission. The values of variables and parameters used to simulate the model (1) - (8) are contained in Table 1 and Table 2.

The baseline scenario reflects the current epidemic context in Nigeria without an HIV vaccine ($v = 0$), vaccine efficacy ($\sigma_v = 0$), and waning ($\omega_v = 0$), representing present-day dynamics under ART and behavioral interventions alone. Vaccination scenarios are assessed relative to this baseline to illustrate the potential added value of future vaccine introduction. Values other than $v = 0$, $\sigma_v = 0$ and $\omega_v = 0$ illustrate different levels of intervention.



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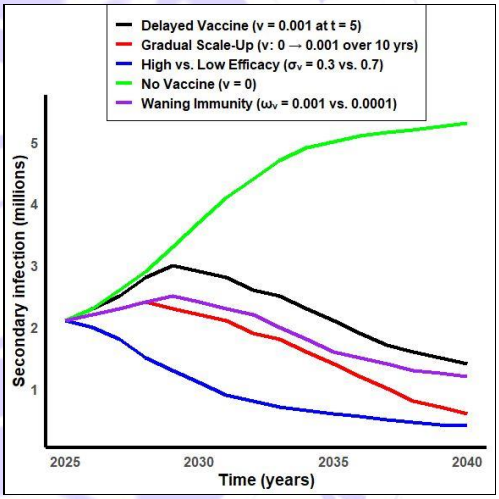

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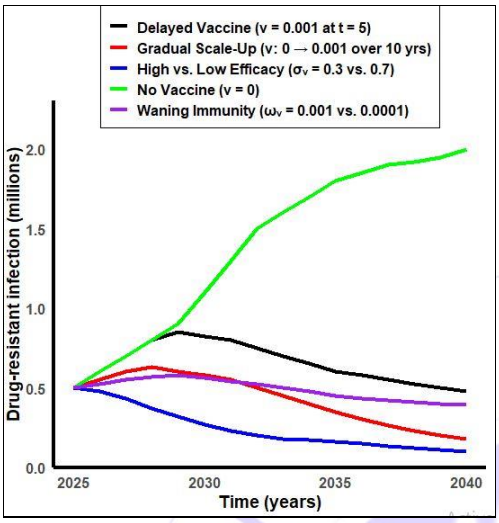

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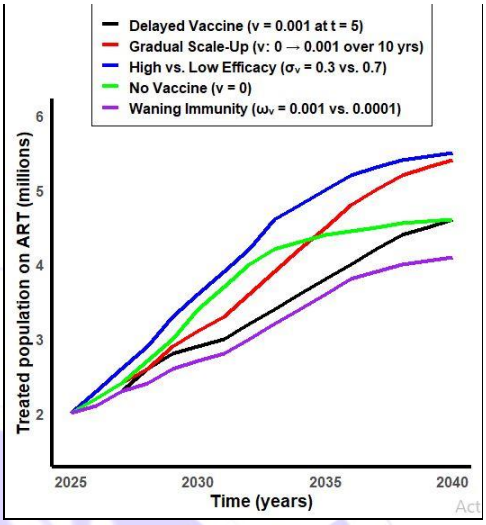

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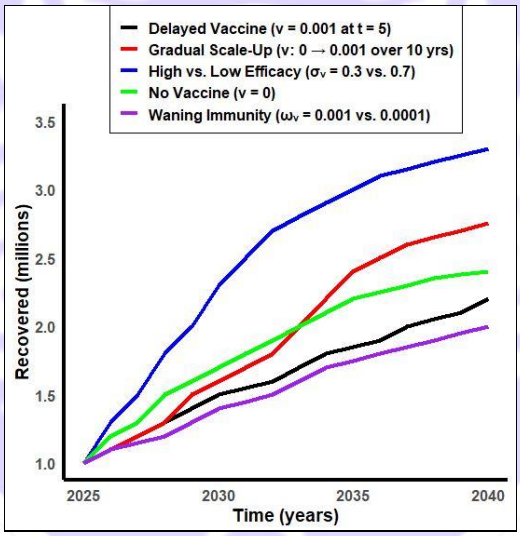

(e)

Figure 1. Projected dynamics of HIV infection and control under future vaccination rollout scenarios in Nigeria

Figure 1(a) illustrates the impact of future-vaccination rollout for HIV primary infection in Nigeria from 2025 to 2040. We observe significant trend movements in epidemic profiles based on the scale, effectiveness of the vaccination intervention plans and timing of vaccination. In the absence of vaccination, that is, when $v = 0$, infection of the primary strain rises gradually from 4.2 million in 2025 to approximately 7.3 million by 2040. This reflects the baseline scenario of unchecked transmission. A delayed-vaccine

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rollout was introduced at $v = 0.001$ at year 5. Initially at this point, we observe a similar increase, however a steady decrease was observed, reducing infections to approximately 3.5 million by 2040. A more notable decline was observed when there was a steady scale-up vaccination intervention, causing infection to drop to 2 million. This gradual scale-up strategy emphasizes the value of phased vaccination implementation even in resource-constrained scenarios. The most significant impact of vaccination rollout was noticed with varying vaccine efficacy at $\sigma_v = 0.3 \ \ vs \ \ 0.7$. At this point, infections drop to around 1 million, amplifying the need to invest in high-efficacy HIV vaccines.

On the other hand, the waning immunity scenario ($\omega_{\mathrm{v}} = 0.001 \ vs. 0.0001$), reveals a modest peak which is followed by a gradual decrease to 3.3 million, highlighting the importance for booster intervention plans to sustain vaccine-induced immunity. Overall, the results highlight that even with logistical constraints, timely deployment of effective vaccines, can significantly reverse HIV transmission trends, with higher efficacy and sustained protection being major determinants of long-term success.

The simulation profile for secondary HIV infection is shown in **Figure 1(b)**. It reveals distinct epidemiological trend that underscore the significance of effective vaccine development and early rollout. We observe a steady rise in HIV secondary infection in the absence of vaccine, peaking at 5 million cases by 2040. This shows continued transmission and susceptibility within a population that has partial immunity. In the case where vaccination rollout was delayed, a small curb to the growth of the infection was observed, with infection decreasing marginally after an initial rise. Conversely, we notice a distinct decline in secondary infection in the gradual scale-up scenarios over time, with cases declining to below 1 million by 2024. This illustrates the long-term importance of increasing and sustaining vaccination efforts. The most impactful case is observed in the high-efficacy vaccination profile, where aggressive and effective vaccination results in a consistent and sharp decrease in secondary infections, reaching near-eradication levels by the end of 2040. Finally, the waning immunity profile reveals a modest decrease in infections, though the decline is not as significant when compared to the high-efficacy and scale-up scenarios. This indicates that to sustain control, a vaccine-induced immunity has to be maintained. Generally, findings show that high-efficacy vaccination interventions coupled with early and sustained deployment are important to minimizing secondary transmission in the population.

Figure 1(c) demonstrates the simulation outcomes for long-term dynamics of drug-resistant infection under different vaccination plan scenarios of resistant strains. In the absence of vaccination, the I_R exhibits a gradual increase, rising from 0.5 million in 2025 to nearly 2.0 million by 2040, signaling an unchecked accumulation of resistant cases over time. HIV infections peaked near 0.85 million when a delayed-

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vaccination rollout plan was initiated, before experiencing a downward trend to 0.48 million by 2040. We observe a modest impact. Under the gradual scale-up scenario, where vaccination coverage rises linearly, we notice a more substantial decrease in infection, reducing resistant infections to approximately 0.18 million, underscoring the importance of early and sustained vaccination rollout. We observe the most impactful case, under the high versus low efficacy scenario when HIV infections decrease rapidly, falling below 0.1 million by the end of the simulation period. This means that higher vaccine efficacy will not only curb infections but also fights the emergence and persistence of resistant strains. The waning immunity profile indicates a slower downward trend, peaking around 0.39 million, highlighting the need for durable immunity to sustain infection on the long-run. Collectively, these findings suggest that high efficacy and durable immunity, coupled with early deployment of vaccine are crucial for reducing the burden of drug-resistant HIV infection in Nigeria.

Figure 1(d) shows infection trajectory of treated population on ART under various vaccination scenarios. In the No Vaccine case, the treated population grows gradually from 2.0 million in 2025 to nearly 4.6 million by 2040, indicating the natural upward trend in ART uptake amidst persistent transmission. The Delayed Vaccine scenario, which is initiated after five years, leads to a more steady growth in the ART population, peaking at a similar level by 2040 but with slower intermediate growth, showing a lagged benefit of late vaccination rollout. The Gradual Scale-Up profile reveals a notable upward trend in ART coverage, growing from 2.0 million to more than 5.4 million by 2040. This is driven by consistent declines in new infections that facilitate retention and broader treatment access. Clearly, we observe that the High versus Low Efficacy cases results in the largest ART population, growing to nearly 5.5 million. This underscores the amplified impact of a high-efficacy vaccine in aiding treatment enrollment by preventing transmission and lowering disease progression. Conversely, the Waning Immunity case reveals subdued decrease in ART coverage, terminating just above 4.1 million. This demonstrates a diminished long-term vaccine protection and its weaker effect on transmission control. Summarily, the patterns observed in these scenarios emphasize that early, enhanced, and sustained vaccination plans will not only reduce infections but also aid ART uptake, which is crucial in controlling infection in the long-run and improving health outcomes in the population.

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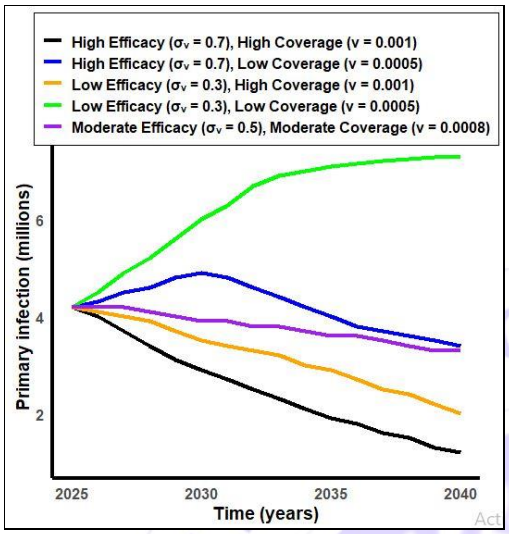
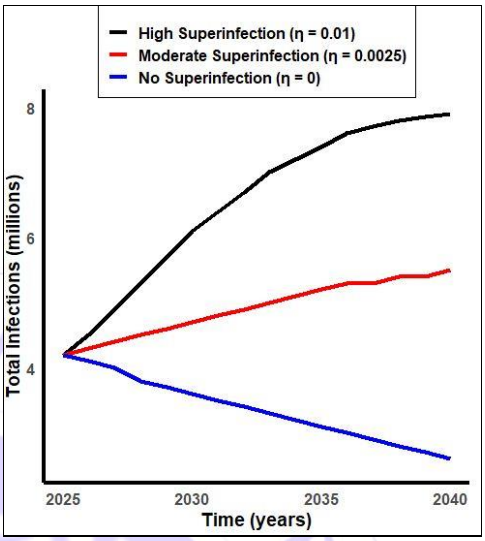


(a)



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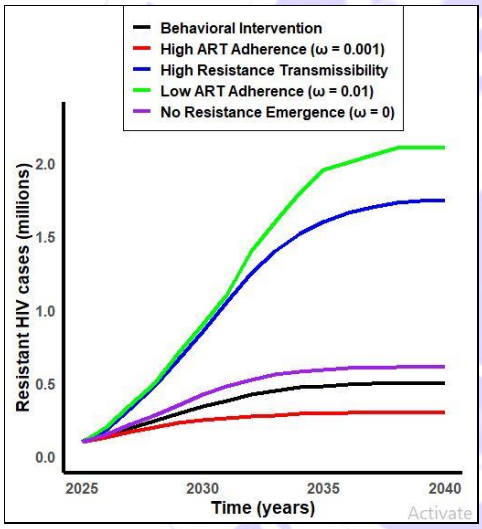

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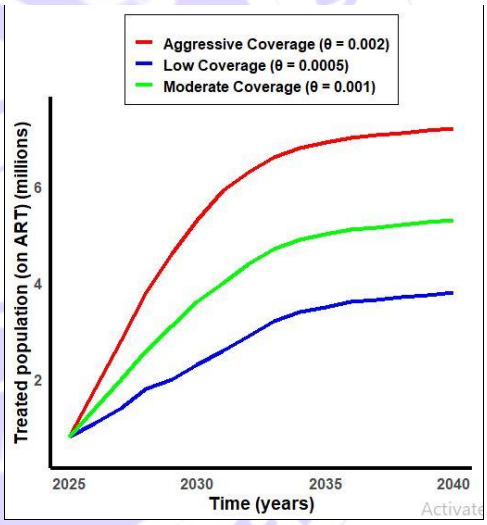

(d)

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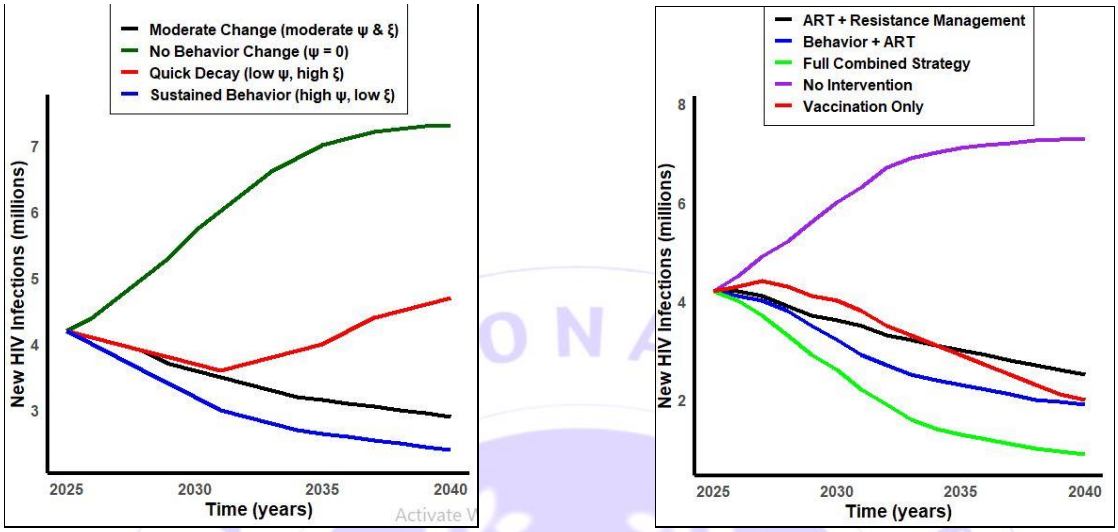

(e) **(f)**

Figure 2. Effect of strategic interventions on the HIV infection in Nigeria

Figure 2(a) demonstrates the crucial impact of vaccine efficacy and coverage in shaping the long-term pattern of infections. The combination of high efficacy at $\sigma_v = 0.7$ with high coverage at $\nu = 0.001$ produces the most significant decrease in infections, showing a sustained rise from 4.2 million in 2025 to approximately 1.2 million by 2024. This shows effective and wide-spread distribution of vaccine has the potential to significantly curb transmission of the infections. In contrast, a combination of high efficacy and low coverage reduces HIV burden more modestly, revealing that efficacy alone cannot offset limited vaccine access. Similarly, a combination of high coverage with low efficacy results to a slower and less substantial decline. At moderate efficacy and coverage, where $\sigma_v = 0.5$ and $\nu = 0.0008$, we observe gradual but steady decline, serving as a feasible middle ground in settings with logistical constraints. The worst-case scenarios occur when efficacy and coverage are both low, resulting in a continuous upward trend in infections. This highlights the risk of deploying ineffective vaccine with less coverage. These findings underscore the combine effect of vaccine efficacy and coverage in vaccination strategies, emphasizing the rationale for policies that gives priority to both vaccine quality and equitable distribution in order to obtain meaningful epidemiological impact.

The scenarios in **Figure 2(b)** explore varying levels of superinfection risk, revealing that secondary infection has the potential to undermine the gains of vaccination and ART. We observe that when $\eta = 0$, the infection pattern shows a gradual downward trend over the period 2025-2040, with total infection

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dropping from 4.2 million to nearly 2.6 million. With moderate superinfection rate, at $\eta = 0.0025$, the downward trend reverses, and infection levels steadily rise to over 5.5 million by 2040, indicating the burden of the second strain. The effect is seen to be more higher under high superinfection risk, when $\eta = 0.01$, where HIV infections escalate sharply, reaching approximately 8 million by the end of 2040. These outcomes show that superinfection can compromise both treatment-derived and vaccine-induced immunity, resulting to persistence transmission even in the presence of control strategies. Therefore, models that do not consider the risk posed by superinfection may significantly undermine the long-term burden of HIV infection and overestimate the effectiveness of control strategies. This finding demonstrates the significance of incorporating secondary infection dynamics into planning, specifically in regions with high transmission intensity, such as Nigeria, or where partial immunity is the norm.

In **Figure 2(c),** we simulated five different resistance scenarios for the simulation period to evaluate the dynamics of drug-resistant HIV under varying levels of behavioural interventions and treatment adherence. The baseline path, representing absence of resistance indicate modest rise in resistant cases, maintaining a steady point at 6 0.6 million throughout the period. However, in the case of low ART adherence, we observe a sharp and steady increase in resistant infections, going above 2 million by 2040. This indicates compounding impact of poor adherence on resistance spread. On the other hand, improved adherence significantly reduced resistance growth, resulting in less than 0.3 million cases by 2040. Increasing resistance transmissibility caused an aggressive epidemic expansion, closely moving along the curve of low-adherence. This underscores the potential danger posed by highly infectious resistant strains. However, when behavioural was integrated, we observe containment of resistance, with cases leveling off below 0.5 million. The outcomes in these scenarios underscore the dual significance of promoting preventive behaviours and sustaining high adherence to treatment to suppress the emergence and transmission of resistant HIV strains. Failure to maintain these control and preventives plans could undermine both vaccination and ART benefits in the long term.

Figure 2(d) is the profile describing projected dynamics of treated population under varying levels of ART coverage, which is determined by the ART initiation rate (θ). With treatment coverage at $\theta = 0.0005$ (low coverage), the population of persons on ART grows slowly, peaking just under 4 million by 2040. This scenario indicates suboptimal programmatic reach, potentially allowing progression and continued transmission of the infection. A moderate rise in ART initiation, at $\theta = 0.001$, significantly enhances treatment coverage, with more than 5 million persons on treatment by 2040. The most aggressive coverage case is when $\theta = 0.002$. This scenario shows the most significant effect, increasing the treated

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population to over 7 million within the same period. These outcomes underscore the importance of accelerating treatment initiation to curb the growth of infection and improve population-level health outcomes. After the first decade, the rate of expansion plateaus. This highlights the need to sustain investment in retention and adherence strategies alongside scale-up. The model reinforces that enhancing ART uptake can alter the epidemic pattern, especially when implemented early and consistently.

The simulation result in **Figure 2(e)** demonstrates the crucial impact of behavioural change adoption and persistence on infection incidence. When $\psi = 0$, new infections increase steadily from 4.2 million in 2025 to over 7.3 million by 2040. In the second case, when behavioral interventions are adopted rapidly and sustained over time, (that is, with high ψ and low ξ), we observe a consistent and sharp decline in new HIV infections is observed, reaching nearly 2.4 million by 2040. This demonstrates a significant long-term gain of durable behavioral change. However, with a rapid decay in behavioural interventions (low ψ, high ξ), the result we yield only transient gains; infections initially decrease but rebound after a few years, approaching pre-intervention stages by 2040. A modest scenario, where behavioral interventions are adopted at a reasonable pace and sustained to a fair extent (moderate ψ and ξ), results in a gradual and steady decrease in HIV infections, stabilizing around 2.9 million. The findings in these scenarios show that not only the scale but also the persistence of behavioral intervention is crucial to achieving and maintaining decrease in transmission. They also suggest that integrating strategies to reinforce and prolong behavior change through repeated community engagement, structural interventions, or incentive mechanisms, can significantly improve epidemic results in the long term.

Fig. 2(f) gives a pictorial summary of the cumulative impact of incorporating ART, vaccination rollout, behavioural interventions and resistance management on HIV epidemic control in Nigeria between 2025 and 2040. The case with no intervention yields a steady increase in new infections. This underscores the persistent transmission risk when there are no control measures. In the presence of only vaccine, we observe a modest decline in HIV infections over time, reflecting the significant of immunization. It also shows its limitations when deployed in isolation. When treatment is integrated with resistance management, a more profound decline in new infections is noticed, underscoring the important role of sustained treatment adherence and containment of drug-resistant strains. The coupled impact of behavioral interventions with treatment yields even greater declines, illustrating the synergistic effect of promoting risk-reducing behaviors with biomedical interventions. Most importantly, the full combined strategy, which encompasses ART, vaccination, behavioural change and resistance management, produces the sharpest and most sustained decrease in new HIV infections. It reduces incidence to below 1 million by 2040. Obviously, no

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single intervention is sufficient on its own; rather, a coordinated, multi-layered approach offers the most effective pathway for long-term HIV infection control.

The comprehensive simulation analysis across ART coverage, vaccination rollout, superinfection dynamics, behavioral interventions, drug resistance emergence, and combined strategies shows distinct but interconnected implications for HIV epidemic control in Nigeria. Vaccination alone, particularly with early and gradual scale-up, substantially reduces both primary and secondary infections and improves partial immunity, but its impact is amplified when coupled with effective treatment coverage and sustained behavioral change. Notably, the plots shows that high treatment initiation rates, that is, $\theta \geq 0.001$, and adherence reduce both infectious burden and the spread of drug-resistant strains, confirming the findings of Maduakolam et al. (2022), who emphasized the centrality of universal testing and treatment in epidemic reversal. In contrast, poor treatment adherence hastens resistance dynamics (through high ω), undermining long-term benefits and stressing the need for adherence-support programs. Superinfection at moderate to high levels ($\eta \geq 0.0025$), visibly alters the infection trajectory by reducing the efficacy of both ART and vaccine programs. This aligns with observations by Loosli et al. (2024), suggesting evolutionary feedbacks can shift epidemic thresholds. Similarly, behavioral changes that initiate long-lasting risk-averse behavior (high ψ, low ξ) significantly flatten incidence curves, though rapid behavior decay reverses these benefits. Ultimately, the combined intervention scenario reflects the greatest epidemiological benefits. It produces coupled effects that surpass the sum of individual interventions. This confirms the multifaceted nature of HIV control, as discussed in modeling studies by Hendrix et al. (2023), which advocate for integrated strategies balancing biomedical and behavioral components.

Our modeling results connect well with Nigeria's HIV control targets and the UNAIDS 95-95-95 goals. The baseline scenario without a vaccine shows that, although ART scale-up has improved outcomes, superinfection and drug resistance could weaken these gains and slow epidemic control if not tackled. Introducing vaccination further lowers new infections and deaths, strengthening the impact of ART and behavioral programs. Since Nigeria's 95-95-95 targets are still not fully met, especially in viral suppression, these findings suggest that adding a future HIV vaccine to existing strategies could speed up progress toward epidemic control. This supports UNAIDS' call for a mix of biomedical, behavioral, and structural approaches to end the epidemic (UNAIDS, 2024).

4. Conclusion

In this study, we developed a mathematical model to examine the dynamics of HIV transmission in Nigeria, integrating key epidemiological factors such as primary and secondary infections, treatment, drug

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resistance, partial recovery (with waning immunity), behavioral interventions, and importantly, future HIV vaccination. The model was developed as an eight-compartment system and parameterized using the data available for the Nigerian context (2024–2025), including ART coverage trends, vaccination rollout projections, and behavioral dynamics. The incorporation of superinfection and reinfection, resistance emergence, and the potential introduction of a future vaccine enabled a multi-layered approach that shows the complex landscape of HIV control in Nigeria.

A sequence of scenario-based simulations was carried out to evaluate the potential impact of vaccination strategies, behavioral intervention, treatment coverage, drug resistance dynamics, and superinfection on the overall epidemic trajectory. Vaccination rollout scenarios revealed that both the timing and coverage level of vaccination are crucial: delayed or gradual vaccine introduction produced modest reductions in incidence, while high-efficacy and low waning immunity vaccines were significantly more effective in reducing the primary infection burden. The model indicated that vaccine-induced protection against primary infection (I_1), secondary infection (I_2), and drug-resistant cases (I_R) not only curbed new infections but also indirectly improves treatment effectiveness and slowed the speed of resistance.

Simulations on superinfection and reinfection risk showed that higher levels of reinfection (that is, increasedη) could partially compromise treatment gains and vaccination benefits. This underscores the importance of behavioral control and population-level immunity. Similarly, drug resistance dynamics illustrated that weak treatment adherence (higher resistance emergence rate, ω) could reverse ART progress and increase the burden of the resistant strain, specifically when coupled with low behavioral adherence. On the contrary, behavioral reinforcement and strong ART adherence curbed both transmission and resistance. ART coverage scenarios revealed that scaling ART (θ) significantly reduced HIV infection prevalence, particularly when combined with preventive strategies. Behavioral dynamics further showed that sustained behavior (high ψ, low ξ) complemented biomedical interventions by lowering incidence and slowing resurgence.

Finally, the combined strategy simulations highlighted the importance of integration: scenarios that jointly scaled vaccination, ART, and behavioral intervention consistently produced the most significant reduction in all infection compartments and resistant strains. This synergy validates the model's basic assumption—that single interventions are inadequate in containing HIV infections, but combined interventions can offer transformative potential in epidemic control.

Conclusively, this model and its scenario-based simulations offer strong evidence that timely vaccination deployment**,** resistance monitoring, robust treatment programs**,** and sustained behavioral

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interventions must be pursued to achieve lasting control of HIV in Nigeria. Future work could expand this approach to integrate cost-benefit analyses, optimize resource allocation, and assess real-time intervention policies with availability of more relevant data.

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