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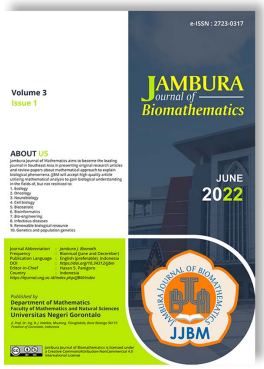

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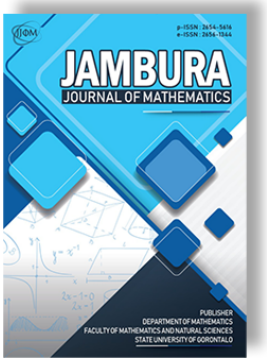

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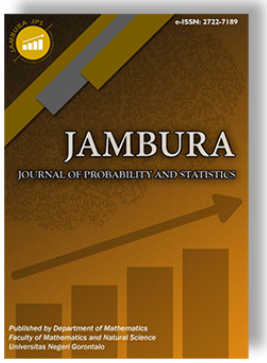

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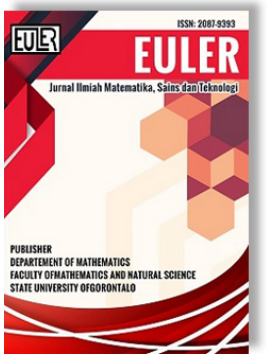

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Research Article

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Analysis of HIV/AIDS Model with risk compensation effects among Pre-Exposure Prophylaxis users and infectious immigrants

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ABSTRACT. *Pre-Exposure Prophylaxis (PrEP) is a promising HIV prevention strategy, and its provision has grown rapidly in several countries, including those in Sub-Saharan Africa. However, lingering concerns remain that introducing PrEP may lead to unintended consequences, such as decreased adherence to other prevention methods and increased risky sexual behaviour, culminating in risk compensation. This study employs a six-compartment mathematical model to investigate the effects of risk compensation behaviour among PrEP users in a population with an influx of infectious immigrants. The model exhibits only disease-free equilibrium points in the absence of infective immigrants and endemic equilibrium with the influx of infected immigrants. The disease-free equilibrium point exists and is locally and globally asymptotically stable in the absence of infective immigrants when the basic reproduction number is less than one. In contrast, the model exhibits only endemic equilibrium in the presence of infective immigrants, which is asymptotically stable when basic reproduction number exceeds unity. A sensitivity analysis of the parameters associated with R_1 was performed using the normalized forward sensitivity index to determine the most influential parameter. The analysis revealed that the number of sexual partners had the greatest influence on disease endemicity. Numerical simulations supported the analytical findings, showing that risk compensation undermines PrEP effectiveness and that multiple sexual partners increase new HIV infections. However, PrEP can significantly reduce new infections in a population with varying immigrant influx and no risk compensation behaviour, highlighting its potential impact in controlling HIV spread. The effectiveness of PrEP depends on strict adherence to usage in combination with other preventive measures. The disease persists with the inflow of infective immigrants.*

1. Introduction

Human Immunodeficiency Virus (HIV) infection and Acquired Immunodeficiency Syndrome (AIDS) are recognized as a global pandemic. Since the virus was first identified, approximately 91.4 million people have contracted HIV. By the end of 2024, 40.8 million individuals worldwide were living with HIV. West and Central Africa account for 5.2 million people living with HIV/AIDS, making it the region with the third-highest burden globally [1, 2]. Nigeria holds the highest number of cases in West and Central Africa, with 2 million people affected. It also ranks third globally in terms of HIV prevalence [1]

Migration plays a significant role in the transmission of HIV. This is largely due to increased sexual risk behaviors that connect regions with low and high infection rates. Frequent travel by migrants especially when combined with risky sexual practices is a major contributing factor [3, 4]. Migrants may be vulnerable to infection or capable of transmitting the virus. The persistence of HIV within a population is often sustained by the direct immigration of infected individuals [3–8]

The scientific consensus is clear: ART is a game-changer in the fight against HIV/AIDS. Its widespread adoption has transformed the trajectory of the epidemic [4, 9]. Additionally, the use of Pre-Exposure Prophylaxis (PrEP) whether oral or long-acting injection is recommended by WHO [10–13]. PrEP involves the use of antiretroviral (ARV) medication by uninfected individuals at substantial risk of HIV infection. It serves as an additional preventive option to block the acquisition of the virus when combined with other HIV prevention approaches. PrEP is highly effective when taken as prescribed. Studies have shown that it can reduce the risk of HIV infection from sexual activity by 99%, and the risk from injection drug use by at least 74% [9, 14]. Individuals at substantial risk include young people and key populations such as sex workers, men who have sex with men, people who inject drugs, incarcerated individuals, and transgender persons. Together with their sexual partners, these groups account for 70% of HIV infections globally [15].

Although there is currently no cure for HIV, numerous prevention strategies are available. These include the consistent and correct use of condoms during sexual activity and the adminis-

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tration of Antiretroviral Therapy (ART) for those already infected. ART helps slow the progression of the virus by lowering the viral load. This not only reduces the risk of transmission but also enables individuals to maintain good health when used properly [4]. Research indicates that ART can decrease the infectiousness of HIV-positive individuals by up to 96% [4, 9].

Despite improvements in treatment and emerging data showing a decline in newly acquired HIV cases following the implementation of Pre-Exposure Prophylaxis (PrEP) [9, 10, 12, 15, 16], concerns persist regarding potential unintended consequences. The introduction of PrEP has been associated with several troubling trends. These include but not limited to:

1. increased risky sexual behaviour [17–19],
2. decreased adherence to other prevention methods [14, 17, 20],
3. higher rates of multiple sexual partners [17–19, 21],
4. PrEP users have shown increased substance abuse [19, 22] and
5. alcohol consumption [21, 22], which have contributed to a rise in sexually transmitted infections (STIs) and HIV incidence [18, 23–26].

These factors collectively point to the phenomenon of risk compensation. This occurs when individuals adopt riskier behaviours after implementing an effective preventive measure [17, 18, 21, 25]. In the case of PrEP, risk compensation refers to the behavioural adaptation where users feel protected against HIV infection and, as a result, reduce their caution. This leads to behaviours that can undermine the protective benefits of PrEP [14, 17–19, 21, 22]. Public health personnel are concerned that risk compensation may diminish the public health impact of PrEP among key populations. This could potentially lead to an increase in HIV and other STI incidences [18, 19, 27]. Risk compensation undermines the overall effectiveness of PrEP in preventing HIV transmission, highlighting the need for comprehensive prevention strategies that address behavioural adaptations.

Mathematical models that specifically examine the impact of risk compensation on HIV/AIDS dynamics among PrEP users are scarce. However, several models have been developed to study PrEP more broadly [16, 28–31]. For instance, [32] mathematically modelled HIV prevention measures including PrEP to estimate the effects of early diagnosis, early treatment, and PrEP on the HIV epidemic in South Korea over a 40-year period, compared to the current situation. [33] also developed a mathematical model to simulate HIV incidence among men aged 15 to 65 years residing in Los Angeles County, California, who have sex with men. Their findings suggest that PrEP and Test-and-Treat strategies yield the largest reductions in HIV incidence and are highly cost-effective. Likewise, [34] proposed an epidemiological model for HIV/AIDS transmission that includes PrEP. They concluded that PrEP significantly reduces the number of new HIV infections. Similarly, [35] examined the effect of PrEP on the direct immigration of HIV/AIDS-infected individuals. Their results indicate that the disease-free equilibrium is unattainable when infected immigrants are present in the population. However, they also found that PrEP can reduce the incidence of new infections if strict adherence to medication is maintained. In similar vein, [36] presented a fractional-order mathematical model to evaluate the impact of transitioning from oral to injectable Pre-Exposure Prophylaxis (PrEP). Their findings demonstrated that the oral modality of PrEP yields better outcomes.

Additionally, Chazuka et al. [37] investigated the stability and bifurcation effects of an HIV model incorporating both PrEP and treatment. These studies, through mathematical modeling, have shown that PrEP can significantly reduce the incidence of HIV. In a model simulated by Lyons et al. [38], the impact of PrEP on HIV transmission among men who have sex with men (MSM) in Yaoundé and Douala, Cameroon, was assessed. The study found that PrEP interventions at 50% coverage could reduce HIV prevalence from 43.2% to 35.4% in Yaoundé and from 26.5% to 20.1% in Douala over a 20-year period. When combined with increased HIV testing, the number of undiagnosed MSM declined further, supporting the implementation of PrEP as part of a comprehensive HIV prevention strategy in Cameroon. In China, Li et al. [39] evaluated the effectiveness of PrEP and enhanced biomedical interventions in reducing HIV transmission among MSM. Their model projected that implementing PrEP could prevent between 12.1% and 25.7% of new HIV infections over 20 years. When combined with test-and-treat strategies, new infections could potentially be eliminated. PrEP was also found to be cost-effective, with estimated costs ranging from $17,277$ to $18,452$ per quality-adjusted life year gained.

Although these studies indicate that PrEP is a promising prevention strategy, targeted interventions are essential. In line with WHO recommendations [10, 12], PrEP should be prioritized for high-risk groups such as sex workers, MSM, transgender individuals, and particularly in Sub-Saharan Africa young men and women aged 15 to 24, who are at heightened risk of acquiring HIV through sexual contact [40]. This study aims to model the impact of risk compensation among PrEP users on the spread of HIV/AIDS and its implications for a population experiencing a constant influx of infected immigrants. Migration patterns play a significant role in HIV transmission, primarily due to increased sexual risk behaviours that connect low- and high-risk regions. Frequent travel between countries of origin and destination creates a complex web of transmission dynamics. Key factors contributing to HIV transmission among migrants include:

1. increased exposure to high-risk environments,
2. separation from spouses or partners, leading to casual sex,
3. limited access to healthcare and HIV testing,
4. cultural and social norms that encourage risky behaviours,
5. economic vulnerability, which may lead to transactional sex.

Repeated movement between low and high-risk areas can introduce HIV into previously low-prevalence regions, sustain transmission chains in high-prevalence areas, and exacerbate existing health disparities. It is well established that immigrants may be either susceptible or infective. Disease persistence is often linked to the direct immigration of infected individuals into the population [3, 5–8, 41, 42].

Building upon the models of Oladejo and Oluyo [35], this study incorporates PrEP to investigate how risk compensation behaviour among PrEP users affects its effectiveness in a population with an influx of infected immigrants. A six-compartment deterministic HIV/AIDS transmission model is developed, accounting for: direct inflow of infectious HIV immigrants, treatment for infected individuals and PrEP for high-risk HIV-negative persons. This model evaluates the impact of risk compensation among

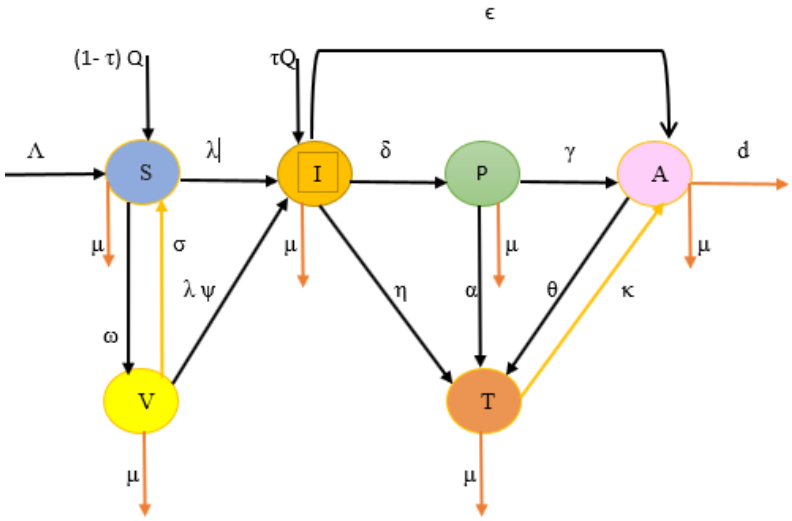

Figure 1. Flow Chart of model (1) with $\lambda = \frac{\beta c(I+a_P P+a_T T)(S+\psi V)}{N}$

PrEP users on HIV/AIDS transmission. The novelty of this work lies in the inclusion of a transmission term within the PrEP class and the consideration of immigration recruitment, enabling a detailed examination of how direct immigration influences HIV infectives. The remaining part of the paper is structured as follows: model formulation and assumptions (Section 2); model analysis and sensitivity analysis (Section 3); numerical simulation and discussion (Section 4); conclusion (Section 5).

2. Model Formulation

The model proposed is grouped into six different classes including the PrEP class $V(t)$ as prevention method for uninfected individuals at risk of possible infection and another class $T(t)$ for HIV positive individuals receiving ART treatment. The following assumptions were made in developing the model (1):

1. The heterogeneous human population varies with time t and it is grouped into six compartments as follows : the Susceptible S(t), the PrEP V(t), the Infective I(t), the Pre-AIDS P(t), the Treatment T(t), and the AIDS A(t) compartments. The total population per unit time is given by

$$N(t) = S(t) + V(t) + I(t) + P(t) + T(t) + A(t).$$

2. There is an inflow of new members Λ, who are susceptible and Q immigrants into the population per unit time, where a fraction τ, $(0 < \tau < 1)$ are infected immigrants [3, 7, 8].
3. For this study, it is assumed that infections are transmitted through sexual intercourse, contact with infected needle or blood [3, 42].
4. Persons with full blown AIDS are weak to be sexually active.
5. The model considers the effect of persons under the PrEP strategy who feel a form of protection by reducing their concern about having unsafe sexual practices and do not adhere to the drug regime. Such persons engage in high risk behaviour (risk compensation behaviour) [18, 19] thereby making PrEP ineffective.
6. The factor by which PrEP prevents transmission is ψ such that the incidence rate is:
$$\frac{\beta c(I + a_P P + a_T T)(S + \psi V)}{N},$$
when $\psi = 0$,that means the drug is effective in preventing transmission of the HIV infection and there is complete adherence to usage and other prevention strategy, [10, 12, 43]. While $\psi = 1$ means that the drug is ineffective in preventing transmission of the HIV infection because of risk compensation among PrEP users, $\forall(0 \leq \psi \leq 1)$.
7. This work considers when $\psi = 1$,(the effect of persons under the (PrEP strategy) who engages in risk compensation). This stems from the fact that risk compensation accounts for the ineffectiveness of vaccine (PrEP strategy) [18, 19]. It also justifies the presence, of the incidence rate
$$\frac{\beta c(I + a_P P + a_T T)(S + V)}{N}$$
in the compartment V(t).
8. a_T accounts for the reduction of HIV viral load consequently reducing the rate of the onward transmission of infection of individuals that use ART correctly [9, 15]. While a_P accounts for relative increase in infectiousness of individual at the Pre-AIDs stage as a result of high viral load [4, 7].

Based on the enlisted assumptions and description depicted in the flowchart shown in Figure 1, we have the mo del equations below:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda + (1-\tau)Q - \frac{\beta c(I + a_P P + a_T T)S}{N} - (\omega + \mu)S \\
&\quad + \sigma V, \\
\frac{dV}{dt} &= \omega S - \frac{\beta c(I + a_P P + a_T T)\psi V}{N} - (\mu + \sigma)V, \\
\frac{dI}{dt} &= \frac{\beta c(I + a_P P + a_T T)(S + \psi V)}{N} - (\delta + \eta + \epsilon + \mu)I \\
&\quad + \tau Q, \\
\frac{dP}{dt} &= \delta I - (\alpha + \gamma + \mu)P,
\end{aligned} \tag{1}$$

Table 1. Table of Parameters Description

Parameter	Interpretation
Λ	inflow of susceptible individual into the population
Q	the constant influx of immigrants into the population
τ	fraction of infected immigrants
$(1-\tau)$	fraction of uninfected immigrants
β	contact rate
a_P	relative infectiousness of individual at the Pre-AIDs stage
a_T	viral suppression in individuals that use ART correctly
ψ	factor by which PrEP is effective $(\psi = 0)$ and ineffective $(\psi = 1)$ accounting for risk compensation
ω	fraction of individuals under then PrEP strategy
σ	rate at which individuals taking PrEP become susceptible
δ	fraction of the infective class that get to the Pre-AIDS class
α	fraction of the Pre-AIDS class that get treatment
θ	rate of treatment for AIDS patients
d	death due to AIDS
c	average number of sexual partners in a unit time
μ	natural mortality rate of individuals in all the classes
η	fraction of the infective class that get treatment
k	fraction of those receiving treatment who develop full blown AIDS
γ	fraction of pre-AIDS patients that develop full blown AIDS
ϵ	fraction of the infective class that develop AIDS

$$\frac{dT}{dt} = \eta I + \alpha P + \theta A - (k+\mu)T,$$
$$\frac{dA}{dt} = \epsilon I + \gamma P + \kappa T - (\theta + \mu + d)A,$$

with initial conditions

$$S(0) > 0,\ V(0) \geqslant 0,\ I(0) \geqslant 0,\ P(0) \geqslant 0,\ T(0) \geqslant 0,\ A(0) \geqslant 0. \tag{2}$$

3. Analytical Results

All analysis for the risk compensation model is considered when $\psi = 1$, The factor that PrEP is ineffective (as a result of risk compensation).

3.1. Positive Invariant Region

It is important to show that the present model is epidemiologically meaningful in the feasible and bounded region D, such that the state variables of the model is non negative for all time $t > 0$. Thus, we state the following theorem following the standard technique method given in [44–46].

Theorem 1. *The solutions* $[S(t), V(t), I(t), P(t), T(t), A(t)]$ *of the model (1) for which* $S(0) > 0$, $V(0) \geqslant 0$, $I(0) \geqslant 0$, $P(0) \geqslant 0$, $T(0) \geqslant 0$, $A(0) \geqslant 0$ *are non negative for all* $t \geqslant 0$.

Proof. Total size of the population $N(t)$ given by $N(t) = S(t) + V(t) + I(t) + P(t) + T(t) + A(t)$ implies that

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dI}{dt} + \frac{dP}{dt} + \frac{dT}{dt} + \frac{dA}{dt},$$
$$\frac{dN}{dt} = \tag{3}$$
$$Lambda + Q - \mu N.$$

Let $D = \Lambda + Q$, we solved eq. (3) using integrating factor method with the initial conditions $t = 0$ and $N(t) = N(0) = N_0$ to have

$$N(t) \leqslant \frac{D}{\mu} + \frac{N_0 - D}{\mu} e^{-dt} ast \to \infty. \tag{4}$$

Thus, whenever $N > \frac{D}{\mu}$, then $\frac{dN}{dt} < 0$. It follows from the inequality above that $\frac{dN}{dt}$ is bounded by $D - \mu N$. A standard comparison theorem [47, 48] can be used to show that $N(t) \leqslant N_0\, e^{-dt} + \frac{D}{\mu}(1 - e^{-dt})$. If $N_0 \leqslant \frac{D}{\mu}$, then $N \leqslant \frac{D}{\mu}$. Thus Ω is positively invariant set under the model described in model (1). Consequently, all the solutions are in the boundary of Ω. Hence, the model is well posed and epidemiologically meaningful and all the feasible solutions enter the region

$$\Omega = \left((S, V, I, P, T, A) \in R_+^6 : 0 \leqslant N \leqslant \frac{\Lambda + Q}{\mu} \right),$$

with non-negative initial condition. □

3.2. Equilibrium Points of the Model and Basic Reproduction Number

Whenever, infective immigrant come into the population, the disease free equilibrium is unobtainable for constant flow of infective immigrants [5–8, 41, 42]. To attain a disease free state, we assume that there are no infective immigrants by setting $\tau = 0$ in the second equation of model (1) to have:

$$E_0 = (S, V, 0, 0, 0, 0)$$
$$= \left(\frac{(\Lambda + Q)K_2}{(\mu + \omega + \sigma)\mu}, \frac{(\Lambda + Q)\omega}{(\mu + \omega + \sigma)\mu}, 0, 0, 0, 0 \right), \tag{5}$$
$$N(t) = \frac{(\Lambda + Q)}{\mu}.$$

The basic reproduction number R_1 is defined as the number of secondary cases produced by an infected person during the period of infection [49, 50]. $R_1 = \rho(\mathcal{F}\mathcal{V}^{-1})$, is the dominant spectral radius of the $(\mathcal{F}\mathcal{V}^{-1})$ largest eigenvalues given that the reproduction number,R_1 when $(\psi = 1$, The factor that PrEP is ineffective), for model (1) is given by :

$$R_1 = \frac{\beta c}{K_4 K_3} \left[\frac{(\kappa(\mu + d) + \mu K_6)(K_4 + a_P(gK_3 + q\delta))}{(\mu + d)\kappa + \mu K_6} \right.$$
$$\left. + \frac{a_T((gK_3 + q\delta)(\alpha K_6 + \gamma\theta) + K_4(\eta K_6 + \epsilon\theta))}{(\mu + d)\kappa + \mu K_6} \right], \tag{6}$$

such that

$$R_1 = R_0 + \frac{\beta c g K_3[(\kappa(\mu+d)+\mu K_6)a_P + a_T(\alpha K_6+\gamma\theta)]}{K_4K_3((\mu+d)\kappa+\mu K_6)}, \tag{7}$$

where R_0 is the basic reproduction number of model (1) when ($\psi = 0$, The factor that PrEP is effective) given by

$$R_0 = \frac{\beta c q}{K_4K_3}\left[\frac{(\kappa(\mu+d)+\mu K_6)(a_P\delta+K_4)}{(\mu+d)\kappa+\mu K_6} + \frac{a_T((\delta)(\alpha K_6+\gamma\theta)+K_4(\eta K_6+\epsilon\theta))}{(\mu+d)\kappa+\mu K_6}\right], \tag{8}$$

Considering

$$\begin{aligned} K_1 &= \omega+\mu, \quad K_5 = \mu+\kappa,\\ K_2 &= \mu+\sigma, \quad K_6 = \theta+\mu+d,\\ K_3 &= \delta+\epsilon+\eta+\mu, \quad q = \frac{S}{N} = \frac{K_2}{\omega+\sigma+\mu},\\ K_4 &= \alpha+\gamma+\mu, \quad g = \frac{V}{N} = \frac{\omega}{\omega+\sigma+\mu}. \end{aligned}$$

eq. (7) shows that $R_1 > R_0$ indicating that increase in the number of secondary case for R_1 is as a result of risk compensation by careless PrEP users. Epidemiologically, the threshold quantity R_1 is the control reproduction number (also known as effective reproduction number) of the model (1) at disease free state. we use the control reproduction number to establish the local stability of the disease-free E_0 and result is given in the Theorem 2.

3.3. Local Stability of the Disease-Free Equilibrium Point

Theorem 2. *The disease-free equilibrium of model (1) is locally asymptotically stable in the absence of infective immigrants if $R_1 < 1$ and is unstable if $R_1 > 1$.*

Proof. To find the DFE, it is assumed that there are no infective immigrants by setting ($\tau = 0$,fraction of infected immigrants) in the model (1), to investigate local stability of DFE, the variational matrix, $J(E_0)$ is constructed as follows:

$$J(E_0) = \begin{pmatrix} -K_1 & \sigma & -\beta cq & -\beta a_P cq & -\beta a_T cq & 0\\ \varpi & -k_2 & -\beta cg & -\beta a_P cg & -\beta a_T cg & 0\\ 0 & 0 & -(K_3-\beta c) & \beta c a_P & \beta c a_T & 0\\ 0 & 0 & \delta & -K_4 & 0 & 0\\ 0 & 0 & \eta & \alpha & -K_5 & \theta\\ 0 & 0 & \epsilon & \gamma & \kappa & -K_6 \end{pmatrix}. \tag{9}$$

The characteristic equation corresponding with $J(E_0)$ is

$$(\lambda^2 b_0 + \lambda b_1 + b_2)(\lambda^4 a_0 + \lambda^3 a_1 + \lambda^2 a_2 + \lambda a_3 + a_4) = 0, \tag{10}$$

where

$$\begin{aligned} b_0 &= 1, \quad b_1 = K_1+K_2, \quad b_2 = \mu(\omega+\mu+\sigma),\\ a_0 &= 1, \quad a_1 = K_1+K_3+K_4+K_7-c\beta, \end{aligned}$$

$$\begin{aligned} a_2 &= (K_3+K_4)(K_5+K_6)+K_3K_4+\kappa(\mu+d)\\ &\quad +\mu K_6-\beta c(\delta a_P+\eta a_T+K_4+K_5+K_6),\\ a_3 &= K_3(K_5+K_6)+(\kappa(\mu+d)+K_6\mu)(K_3+K_4)\\ &\quad -\beta c\big((\delta a_P+K_4)(K_5+K_6)+\kappa(\mu+d)+\mu K_6\\ &\quad +a_T(\eta(K_4+K_6)+\delta\alpha+\epsilon\theta)\big),\\ a_4 &= K_4K_3((\mu+d)\kappa+\mu K_6)(K_3(\kappa(\mu+d)+\mu K_6)(K_4\\ &\quad +\beta cga_p)+a_T(\kappa K_6+\gamma\theta)-R_1). \end{aligned}$$

Now applying the Routh-Hurwitz criterion to the polynomial (10), for $R_1 < 1$ the coefficient $a_i > 0$ for $i = 0,1,2,3,4$. Thus, all the eigen-values will have non positive real part. $b_1 > 0$, $b_2 > 0$, then $b_1b_2 > 0$ for the quadratic equation. In the fourth root equation for $a_1 > 0$, then $(K_1+K_3+K_4+K_7) > c\beta$ also for $a_2>0$, then $(K_3+K_4)(K_5+K_6)+K_3K_4+\kappa(\mu+d)+\mu K_6 > \beta c(\delta a_P+\eta a_T+K_4+K_5+K_6)$. Likewise for $a_3 > 0$, then

$$\begin{aligned} a_3 &= K_3(K_5+K_6)+(\kappa(\mu+d)+K_6\mu)(K_3+K_4)\\ &> \beta c\big((\delta a_P+K_4)(K_5+K_6)+\kappa(\mu+d)+\mu K_6\\ &\quad +a_T(\eta(K_4+K_6)+\delta\alpha+\epsilon\theta)\big). \end{aligned}$$

From

$$\begin{aligned} a_4 &= K_4K_3((\mu+d)\kappa+\mu K_6)(K_3(\kappa(\mu+d)+\mu K_6)(K_4\\ &\quad +\beta cga_p)+a_T(\kappa K_6+\gamma\theta)-R_1)\\ &> 0, \end{aligned}$$

when

$$R_1 < K_3(\kappa(\mu+d)+\mu K_6)(K_4+\beta cga_p)+a_T(\kappa K_6+\gamma\theta).$$

After careful simplification of $a_1a_2a_3 > a_3^2+a_1^2a_4$ is true, all the roots will be negative or have negative real parts. It follows that all the eigenvalues $J(E_0)$ have negative real parts. This implies that the disease-free equilibrium is locally asymptotically stable if $R_1 < 1$. Hence, in the absence of infective immigrant the disease-free equilibrium point, E_0 is locally asymptotically stable, if $R_1 < 1$ unstable if $R_1 > 1$. □

Epidemiologically, the implication of Theorem 2 is that a small influx of infected individual will not generate an out break of the disease in the population if the threshold quantity R_1 is less than unity. In other words the disease will rapidly die out when the control reproduction number $R_1 < 1$ if the initial size of the infected individual is in the basin of attraction of E_0. To maintain $R_1 < 1$ testing and treatment must be enhanced, adherence counsellings be provided for those under the PrEP strategy and treatment to increase the effectiveness of interventions.

3.4. Stability Analysis of the Endemic Equilibrium

However, as long as the infective immigrant are entering the population, the disease-free equilibrium will become unattainable, this is in agreement with Brauer and Van den Driessche [5], Ram et al. [7] and Issa et al. [6]. Consequentially the model (1) posses only the Endemic equilibrium point in the presence of infected immigrants into the population, the endemic equilibrum of the HIV/AIDS model (1) designated as E^* is defined

Table 2. Cases of multiple roots using Descartes rule of sign

Case	C_3	C_2	C_1	C_0	R_1	No. of changing sign	Real root (positive)
1	+	+	+	-	$R_1 > 1$	1	1
2	+	-	-	-	$R_1 > 1$	1	1
3	+	+	-	-	$R_1 > 1$	1	1
4	+	-	+	-	$R_1 > 1$	3	1,3
5	-	+	+	+	$R_1 > 1$	1	1

as $E^* = (S^*, V^*, I^*, P^*, T^*, A^*)$, which were obtained after calculation to be

$$\begin{aligned} P^* &= \frac{\delta I^*}{K_4}, \\ A^* &= \frac{(K_5(\epsilon K_4 + \delta\gamma) + \kappa(\eta K_4 + \delta\alpha))I^*}{K_4(\kappa(\mu + d) + \mu K_6)}, \\ T^* &= \frac{(\theta(\epsilon K_4 + \delta\gamma) + K_6(\eta K_4 + \delta\alpha))I^*}{K_4(\kappa(\mu + d) + \mu K_6)}, \\ V^* &= \frac{\omega(\Lambda + (1-\tau)Q)}{((MI^*)^2 + M(\omega + 2\mu + \sigma)I^* + \mu(\omega + \mu + \sigma))}, \\ S^* &= \frac{\omega(\Lambda + (1-\tau)Q)(MI^* + K_2)}{((MI^*)^2 + M(\omega + 2\mu + \sigma)I^* + \mu(\omega + \mu + \sigma))}, \end{aligned} \tag{11}$$

and the value of I^* are the positive roots of the polynomial equation:

$$C_3 I^{*3} + C_2 I^{*2} + C_1 I^* - C_0 = 0, \tag{12}$$

where

$$\begin{aligned} C_3 &= K_3 M^2, \\ C_2 &= M(K_3(\omega + 2\mu + \sigma) - M(Q + \Lambda)), \\ C_1 &= K_3\mu(\omega + \mu + \sigma) - M\big((\omega + 2\mu + \sigma)\tau Q \\ &\quad + (\Lambda + (1-\tau)Q(\omega + \mu + \sigma))\big), \\ C_0 &= \tau Q\mu(\omega + \sigma + \mu), \\ M &= \frac{\beta c q((\mu + d)\kappa + \mu K_6)(a_P\delta + K_4)}{NK_4K_3((\mu + d)\kappa) + \mu K_6} \\ &\quad + \frac{a_T(\theta(\epsilon K_4 + \delta\gamma) + K_6(\eta K_4 + \delta\alpha))}{NK_4K_3((\mu + d)\kappa) + \mu K_6}. \end{aligned}$$

By Descartes' rule of sign in [51, 52], a sign change means there is either one or zero real positive root of the polynomial (12), so one of the root will be positive. This implies that $S^*, V^*, I^*, P^*, T^*, A^*$ will have multiple roots. The model (1) will have positive endemic solution E^* if $R_1 > 1$. There are several possibilities of equilibra as shown by the instances of sign change from 1-5 in Table 2 for the polynomial (12)

This suggests that forward bifurcation is possible as this is a situation where the disease-free is unattainable (losses its stability) due to the presence of infected immigrants in the population and a stable endemic equilibrium exists as R_1 increase through one. Thus, this phenomenon has notable indication for the control disease.The epidemiological implication of this is that there is a continuous influx of infective immigrants ensuring the transmission chain remains unbroken. Forward bifurcation occurs when the basic reproduction number exceeds 1, making the transition from disease-free to endemic as disease free becomes unstable.

3.5. *Bifurcation*

The existence of the local asymptotic stability of the endemic equilibrium of the HIV/AIDS model (1) is carried out, using the Centre Manifold Theory illustrated by [53]. Suppose that β is taken as the bifurcation parameter such that $\beta^* = \beta$, when $R_1 = 1$ in eq. (6), to obtain

$$\begin{aligned} \beta^* &= \frac{K_4K_3((\mu + d)\kappa + \mu K_6)}{c\Psi}, \\ \Psi &= ((\mu + d)\kappa + \mu K_6)(a_P(gK_3 + q\delta) + K_4) + a_P((gK_3 \\ &\quad + q\delta)(\alpha K_6 + \gamma\delta) + K_4(\eta K_6 + \epsilon\theta)). \end{aligned} \tag{13}$$

The linearized matrix of the model (1) evaluated at the disease-free equilibrium E_0 and evaluated at β^* is given by the matrix in eq. (9). $J(E_0, \beta^*)$ has the right eigenvector corresponding to the simple zero eigenvalue given by $w = (w_1, w_2, w_3, w_4, w_5, w_6)^T$ so that $J(E_0, \beta_0^*)w = 0$. We have

$$\begin{aligned} w_1 &= -w_3\frac{w_3K_3(\omega q + gK_1)}{\mu(\omega + \mu + \sigma)}, \\ w_2 &= -\frac{w_3K_3(qK_2 + g\sigma)}{\mu(\omega + \mu + \sigma)}, \quad w_3 > 0, \\ w_4 &= \frac{\delta w_3}{K_4}, \quad w_5 = w_3\left(\frac{K_4K_3 - \beta^* c((K_4 + \delta a_P))}{\beta^* c a_T K_4}\right), \\ w_6 &= w_3\left(\frac{\beta^* c(a_T(\epsilon K_4 + \gamma\delta) - \kappa(K_4 + \delta a_P)) + \kappa K_4K_3}{\beta^* c a_T K_4 K_6}\right). \end{aligned}$$

The left eigenvector associated to $J(E_0, \beta_1^*)v = 0$ is given by

$$\begin{aligned} v_1 &= 0, \quad v_2 = 0, \\ v_3 &= v_6\frac{(\kappa(\mu + d) + \mu K_6)}{\theta\beta^* c a_T}, \\ v_4 &= v_6\frac{a_P(\kappa(\mu + d) + \mu K_6) + a_T(\alpha K_6 + \gamma\theta)}{\theta K_4 a_T}, \\ v_5 &= v_6\frac{K_6}{\theta}, \quad v_6 > 0. \end{aligned}$$

The non-vanishing partial derivatives are calculated and obtained as follows

$$\begin{aligned} \frac{\partial^2 f_1}{\partial x_1 \partial x_3}(E_0, \beta^*) &= \frac{\partial^2 f_1}{\partial x_3 \partial x_1}(E_0, \beta^*) = \frac{-x_2\beta^* c}{(x_1 + x_2)^2}, \\ \frac{\partial^2 f_1}{\partial x_1 \partial x_4}(E_0, \beta^*) &= \frac{\partial^2 f_1}{\partial x_4 \partial x_1}(E_0, \beta^*) = \frac{-x_2\beta^* c a_p}{(x_1 + x_2)^2}, \\ \frac{\partial^2 f_1}{\partial x_1 \partial x_5}(E_0, \beta^*) &= \frac{\partial^2 f_1}{\partial x_5 \partial x_1}(E_0, \beta^*) = \frac{-x_2\beta^* c a_T}{(x_1 + x_2)^2}, \\ \frac{\partial^2 f_1}{\partial x_2 \partial x_3}(E_0, \beta^*) &= \frac{\partial^2 f_1}{\partial x_3 \partial x_2}(E_0, \beta^*) = \frac{\partial^2 f_1}{\partial x_3 \partial x_6}(E_0, \beta^*) \\ &= \frac{\partial^2 f_1}{\partial x_6 \partial x_3}(E_0, \beta^*) = \frac{\partial^2 f_2}{\partial x_1 \partial x_3}(E_0, \beta^*) \end{aligned}$$

$$
\begin{aligned}
&= \frac{\partial^2 f_2}{\partial x_3 \partial x_1}(E_0,\beta^*) = \frac{x_1\beta^* c}{(x_1+x_2)^2},\\
\frac{\partial^2 f_1}{\partial x_2 \partial x_4}(E_0,\beta^*) &= \frac{\partial^2 f_1}{\partial x_4 \partial x_2}(E_0,\beta^*) = \frac{\partial^2 f_1}{\partial x_4 \partial x_6}(E_0,\beta^*)\\
&= \frac{\partial^2 f_1}{\partial x_6 \partial x_4}(E_0,\beta^*) = \frac{\partial^2 f_2}{\partial x_1 \partial x_4}(E_0,\beta^*)\\
&= \frac{\partial^2 f_2}{\partial x_4 \partial x_1}(E_0,\beta^*) = \frac{x_1\beta^* a_p c}{(x_1+x_2)^2},\\
\frac{\partial^2 f_1}{\partial x_2 \partial x_5}(E_0,\beta^*) &= \frac{\partial^2 f_1}{\partial x_5 \partial x_2}(E_0,\beta^*) = \frac{\partial^2 f_1}{\partial x_5 \partial x_6}(E_0,\beta^*)\\
&= \frac{\partial^2 f_1}{\partial x_6 \partial x_5}(E_0,\beta^*) = \frac{\partial^2 f_2}{\partial x_1 \partial x_5}(E_0,\beta^*)\\
&= \frac{\partial^2 f_2}{\partial x_5 \partial x_1}(E_0,\beta^*) = \frac{x_1\beta^* a_T c}{(x_1+x_2)^2},\\
\frac{\partial^2 f_2}{\partial x_2 \partial x_3}(E_0,\beta^*) &= \frac{\partial^2 f_2}{\partial x_3 \partial x_2}(E_0,\beta^*) = \frac{-x_1\beta^* c}{(x_1+x_2)^2},\\
\frac{\partial^2 f_2}{\partial x_2 \partial x_4}(E_0,\beta^*) &= \frac{\partial^2 f_2}{\partial x_4 \partial x_2}(E_0,\beta^*) = \frac{-x_1\beta^* a_p c}{(x_1+x_2)^2},\\
\frac{\partial^2 f_2}{\partial x_2 \partial x_5}(E_0,\beta^*) &= \frac{\partial^2 f_2}{\partial x_5 \partial x_2}(E_0,\beta^*) = \frac{-x_1\beta^* a_T c}{(x_1+x_2)^2}.
\end{aligned}
$$

The bifurcation coefficient a and b are given as

$$
\begin{aligned}
a &= \sum_{k,i,j=1}^{6} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_0,\beta^*),\\
b &= \sum_{k,i,j=1}^{6} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(E_0,\beta^*),
\end{aligned}
\tag{14}
$$

using the non zero partial derivative,

$$
\begin{aligned}
a &= -2\frac{v_3\beta^* c\mu(w_3+w_4+w_5+w_6)(w_3+w_4a_p+w_5a_T)}{(\Lambda+Q)},\\
b &= 2v_3c(w_3+w_4a_p+w_5a_T).
\end{aligned}
\tag{15}
$$

Then after some computations using the values of w_3, w_4, w_5, w_6 and v_3, we have a and b as

$$
\begin{aligned}
a &= -\frac{2V_6w_3^2K_3\mu(\kappa(\mu+d)+\mu K_6)}{\theta\beta c a_T K_4(\Lambda+Q)}\left(\frac{K_4(K_6+\epsilon)+\delta(K_6+\gamma)}{K_6}\right.\\
&\quad \left.+\frac{(K_6+\kappa)(K_3K_4-\beta c(K_4+\delta a_p))}{\beta c a_T K_6}\right),\\
b &= \frac{v_6w_3K_3(\kappa(\mu+d)+\mu K_6)}{\theta{\beta^*}^2 c a_T}.
\end{aligned}
$$

Clearly, $b>0$, from computation $w_3>0$ and $v_6>0$.To determine the value of a to be either positive or negative, we must examine the sign of $K_4K_3-\beta^*c(K_4+\delta a_P)$, because the numerator is negative.

1. When $K_4K_3-\beta^*c(K_4+\delta a_P)>0$: If $K_4K_3>\beta^*c(K_4+\delta a_P)$, the resultant will be positive. Since the numerator is negative, the overall expression for a will be negative (because of the negative sign in the formula).
   Conclusion: $a<0$ if $K_4K_3>\beta^*c(K_4+\delta a_P)$ this indicates a forward bifurcation.
2. When $K_4K_3-\beta^*c(K_4+\delta a_P)<0$: If $K_4K_3<\beta^*c(K_4+\delta a_P)$, the resultant value will be negative. Since the numerator is negative, the overall expression will be positive (because multiplying a negative number by a negative number gives a positive result, as there already is a negative sign outside the fraction).
   Conclusion: $a>0$ if $K_4K_3<\beta^*c(K_4+\delta a_P)$ This indicates a backward bifurcation.
3. When $K_4K_3-\beta^*c(K_4+\delta a_P)=0$: If $K_4K_3=\beta^*c(K_4+\delta a_P$, the numerator will be negative. In this case, the value of a becomes negative.
   Conclusion: $a<0$ if $K_4K_3=\beta^*c(K_4+\delta a_P)$ this indicates a forward bifurcation.

When $K_4K_3>\beta^*c(K_4+\delta a_P)$, our numerical analysis reveals that a becomes negative, signaling a forward bifurcation. According to Theorem 4.1 in [53], this implies that the HIV/AIDS model (1) undergoes a forward bifurcation, as illustrated in Figure 2. Therefore $R_1<1$ is necessary and sufficient for disease elimination. Hence the endemic equilibrium is locally asymptomatically stable for $R_1>1$ or sufficiently close to one. The epidemiological implication of this is that the disease can be controlled as long as $R_1<1$. Though a little influx of infected person can make the disease endemic in the population, making control measures less effective.

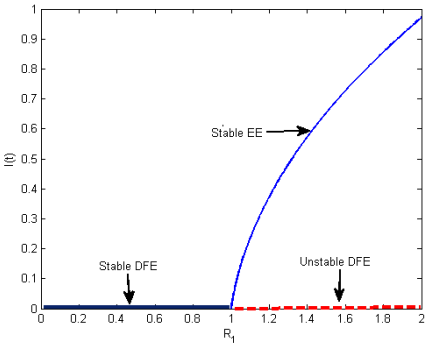

Figure 2. forward bifurcation diagram

3.6. *local Stability of the Endemic Equilibrium*

Theorem 3. *If $R_1>1$, the endemic equilibrium E^* of the model (1) is locally asymptotically stable in Ω otherwise unstable.*

Proof. It follows from the existence of forward bifurcation. □

Remark 1. Influx of infective immigrants can reduce the threshold for forward bifurcation ($R_1=1$), increase the stability of endemic equilibrium and sustain endemic transmission, even if $R_1<1$. The implications of infective immigrants and forward bifurcation on disease control are significant. Control efforts can be challenging, leading to higher disease prevalence. Furthermore, when combined with risk compensation, control measures may be less effective and potentially facilitate the development of drug resistance.

3.7. Global Stability of the Endemic Equilibrium

Theorem 4. *If $R_1 > 1$, the endemic equilibrium E^* of the model (1) is globally asymptotically stable in Ω .*

Proof. When $R_1 > 1$ and there exist endemic equilibrium points for the model (1), considering the Lyapunov function of the Goh-Volterra type [54, 55]

$$F = \frac{1}{K_1}\left(S - S^{**} - S^{**}\ln\frac{S}{S^{**}}\right) + \frac{1}{K_2}\left(V - V^{**} - V^{**}\ln\frac{V}{V^{**}}\right) + \frac{1}{K_3}\left(I - I^{**} - I^{**}\ln\frac{I}{I^{**}}\right) + \frac{1}{K_4}\left(P - P^{**} - P^{**}\ln\frac{P}{P^{**}}\right) + \frac{1}{K_5}\left(T - T^{**} - T^{**}\ln\frac{T}{T^{**}}\right) + \frac{1}{K_6}\left(A - A^{**} - A^{**}\ln\frac{A}{A^{**}}\right). \tag{16}$$

□

Taking the time derivative of eq. (16) substituting the derivative of (I, P, T, A) from model (1) to have

$$\begin{aligned}\frac{DF}{dt} = {} & \frac{1}{K_1}\left(\frac{S - S^{**}}{S}\right)(\Lambda + (1-\tau)Q + \sigma V - S(\widetilde{\beta}cI + \widetilde{\beta}a_pcP \\ & + \widetilde{\beta}a_Tscr) - K_8S) + \frac{1}{K_2}\left(\frac{V - V^{**}}{V}\right)(\omega S - V(\widetilde{\beta}cI \\ & + \widetilde{\beta}a_pcP + \widetilde{\beta}a_TcT) - K_9V) + \frac{1}{K_3}\left(\frac{I - I^{**}}{I}\right)(\tau Q \\ & + (S+V)(\widetilde{\beta}cI + \widetilde{\beta}a_pcP + \widetilde{\beta}a_TcT) - K_1I) \\ & + \frac{1}{K_4}\left(\frac{P - P^{**}}{P}\right)(\epsilon\delta I - K_3P) + \frac{1}{K_5}\left(\frac{T - T^{**}}{T}\right) \\ & \times (\eta\delta I + \gamma\alpha P + \theta A - K_4T) + \frac{1}{K_6}\left(\frac{A - A^{**}}{A}\right)((1 \\ & - \epsilon - \eta)\delta I + \alpha(1-\gamma)P + \kappa T - K_7T).\end{aligned} \tag{17}$$

Substituting the equations of model (1) at steady state into eq. (17) gives

$$\begin{aligned}\frac{DF}{dt} = {} & -\frac{(S - S^{**})^2}{S} - \frac{(V - V^{**})^2}{V} - \frac{(I - I^{**})^2}{I} \\ & - \frac{(P - P^{**})^2}{P} - \frac{(T - T^{**})^2}{T} - \frac{(A - A^{**})^2}{A}.\end{aligned} \tag{18}$$

Therefore $\frac{dF}{dt} \leq 0$ will be negative definite and $S = S^*$, $V = V^*$, $I = I^*$, $P = P^*$, $T = T^*$, $A = A^*$. Thus, the largest compact invariant $(S^*, V^*, I^*, P^*, T^*, A^*) \in \Omega : \frac{dF}{dt} = 0$ is the singleton E^*. By LaSalle's invariant principle [56], E^* is globally asymptotically stable in Ω since $\frac{dF}{dt} \leqslant 0$ for $R_1 > 1$.

3.8. Analysis of Sensitivity Index

Understanding the basic reproduction number R_1 enables public health officials to evaluate the effectiveness of interventions, informing policy decisions that optimize resource allocation and ultimately enhance disease control efforts. So the normalized forward sensitivity index of each parameter associated with the basic reproduction number is analyzed using the Chitnis et al. [57] approach which is defined as

$$H_r^{R_1} = \frac{r}{R_1}\frac{\partial R_1}{\partial r}. \tag{19}$$

The numerical values shown in Table 3 are computed to enable us know which of the parameters have a high impact on the basic reproduction number so that it will be the target point of any control and preventive strategy; as depicted by the chart in Figure 3.

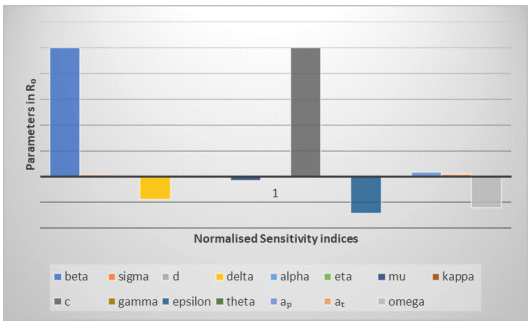

Figure 3. Sensitivity index of basic reproduction number

Table 3. Sensitivity index value and Parameter Values used in the Simulation

Parameter	Sensitivity indices	Estimated values	Source
β	+1	0.34	[59]
a_P	+0.0357535860	0.21	[34]
a_T	+0.0195805866	0.05	[9, 15]
Q	-	$1.3million$	[58]
Λ	-	$3,952,953$	Estimated
C	+1	3	[7]
δ	-0.1741107516	0.223	[60]
d	-0.000046569703	1	[7]
μ	-0.3051767674	$\frac{1}{54}$	[58]
α	-0.0003941568	0.4	Assumed
ϵ	-0.2858478846	0.34	[60]
ω	-0.2413535246	0.1	[34]
τ	-	0.05	Assumed
η	-0.0043631830	0.85	[58]
γ	-0.0012455350	0.6	Estimated
k	-0.0013297968	0.04	[58]
θ	+0.0023750547	0.1	[34]
σ	+0.0114930250	0.4	Assumed

From Figure 3 and Table 3, it is clear that the key factors influencing R_1 are the parameters β (contact rate), a_P (relative infectiousness of individual at the Pre-AIDs stage), a_T (viral suppression in individuals that use ART correctly), σ (rate at which individuals taking PrEP become susceptible) with positive value, this indicates that these parameters have the potential to increase the value of the associated basic reproduction number. When the values of these parameters are increased, there is a proportional increase in the basic reproduction number consequently increasing the prevalence of the disease in the population. The parameters with negative values have a beneficial impact on the disease burden. Specifically, increasing these parameter values, while keeping all other parameters constant, leads to a decrease in the reproduction number R_1, thereby reducing the disease transmission. This consequently reduces the spread of the disease in the population.

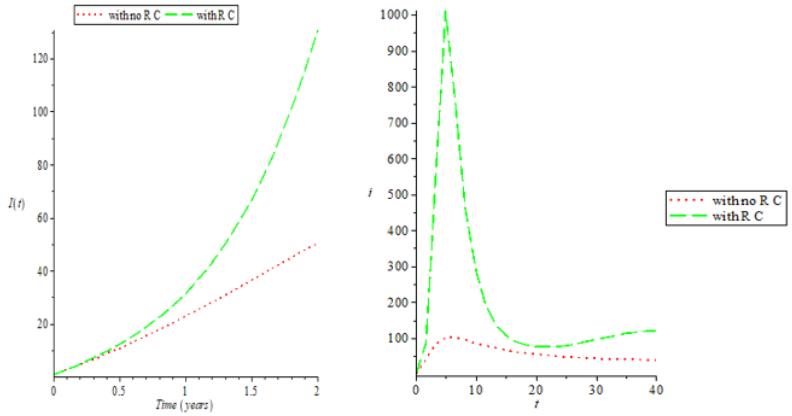

Figure 4. Comparison of model without risk compensation $\psi = 0$ and with risk compensation $\psi = 1$ among PrEP users

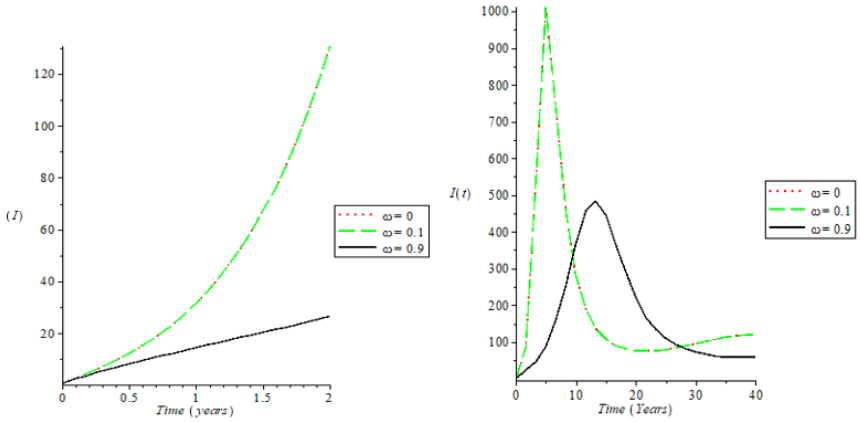

Figure 5. Different fractions of susceptible individual on PrEP in 2 years and 40 years

4. Numerical Results

To study the dynamical behaviour of the model numerically, model (1) is solved using Runge-Kunta method of order four, computed with the aid of the MAPLE 18 mathematical software, using the 2021 demography of Nigeria, the total population is 213.4 million [58] following the United Nations Nigerian country data. The natural mortality rate is estimated to be $\mu = \frac{1}{54}$ and recruitment rate was estimated to be $\Lambda = \mu N$. The a_T was considered based on studies on the efficacy of ART by [9]; d was estimated based on the HIV/AIDS factsheets [59]. According to the 2021 country report [60] 34% of individuals living with HIV had an initial CD4 cell count below 200 $cells/mm^3$ during reporting period, while 53.3% had a count below 350 cell/mm^3. The difference between these two is denoted as $\delta = 0.563 - 0.34 = 0.223$ with $\epsilon = 0.34$ representing the propotion of below 200 cells/mm^3. In similar vein, based on data from [58], the estimated fraction of individuals with unsuppressed viral load is $k = 0.04$. assumed , calculated or either estimated based on the HIV/AIDS factsheets [59]. All the set of parameter and initial values in Table 3 were taken per year.

Figure 4 shows the plot of the effect of risk compensation (RC) among PrEP users in comparison to model when there is no risk compensation. The red curve is the model when $\psi = 0$ (factor by which PrEP is effective without risk compensation), indicating the dynamics with strict adherence to the uptake in combination with other preventive strategy. While the green curve is the model when $\psi = 1$ (factor by which PrEP is ineffective with risk compensation), indicates the model dynamics where users are careless, having one form of risk compensation or another such as unprotected sex practices with multiple partners, illicit drug use etc. The effect of risk compensation among PrEP user can have a fatal effect on the HIV/AIDS dynamics as shown in Figure 4. That is why the total number of people infected in the plot remains high in comparison to the red plot. The red curve demonstrates that PrEP is effective when taken as prescribed as a preventive tool for uninfected individuals at substantial risk of HIV infection when taken with other preventive strategy. In Figure 5, we simulate various fractions of susceptible individuals under the PrEP strategy with the model ($\psi = 1$) in 2 years and 40 years, when $\omega = 0$ refers to the scenario when no person is taking PrEP, $\omega = 0.1$ is the scenario where only 10% of the susceptible individuals are on the PrEP strategy, while the $\omega = 0.9$ refers to the situation where 90% of the susceptible individual are on the PrEP strategy. We note from the graph that there is no difference between the blue and red lines that is when $\omega = 0$ and $\omega = 0.1$, respectively. Ordinarily, 10% PrEP users should show a remarkable decrease in incidences as evident in our work on the effectiveness of PrEP in reducing incidences of HIV/AIDS infection in [35] but because there are reduced concern about risky unsafe sexual practices and other vices by PrEP users. The effect of PrEP

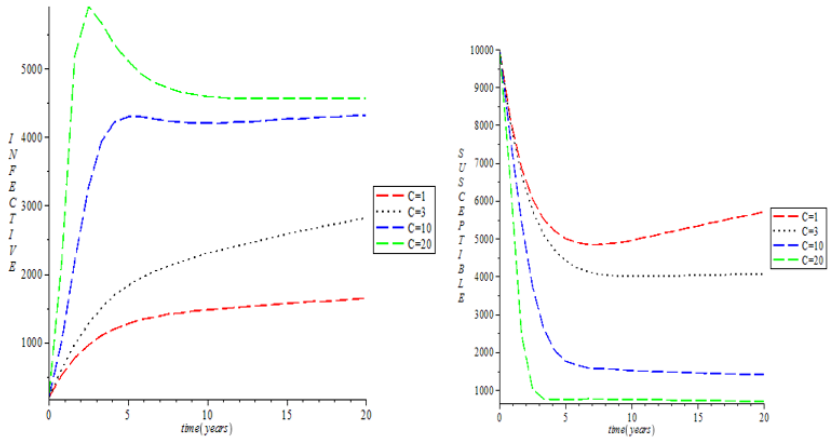

Figure 6. Graphical result of the effect of the number of sexual partners on I(t) and S(t)

is not felt in population indicated by the blue and red simply because of risk compensation therefore the infection will persist in these two cases. In the third case 90% percent PrEP users shows a remarkable reduction in HIV infection, to achieve a remarkable rolling back of the epidemic in line with the sustainable developmental goals.

The implication is that there should be a minimum or optimal number of users that will be cost effective due to the fact that the drug is still expensive in some setting. The level of protection PrEP offers strongly correlates with adherence. A voluntary decision to take PrEP is very important, without which leaves the community worse than when there is no PrEP strategy in place as indicated by Figure 4. Some PrEP users may also marginalize others through increased deceptive forms of freedom and control. Therefore, PrEP is expected to decrease public health burden of HIV/AIDS and drug resistance too. Its provision should be accompanied with different counseling on risky behaviour reduction and adherence. Additionally to mitigate risk compensation comprehensive sexual health education and awareness should be promoted, regular STI testing and treatment be encouraged among these people groups, condom promotion and distribution, encouraging safe sexual practices and PrEP monitoring and adherence follow up be provided.

Plots in Figure 6 indicate that increase in the number of sexual partners leads to decrease in the number of susceptible individuals and consequently increase in the spread of the infection. The higher the number of sexual partners, the greater the risk of getting infected as observed from Figure 6. Thus, in order to reduce the spread of the disease, it is desirable to keep the number of sexual partners at minimum, practice safe sex, maintain strict adherence to biomedical interventions and have behavioural change. Additionally, to prevent the susceptible population from being wiped out, the number of sexual partner as well as unsafe sexual interaction or contact with immigrants is to be restricted. Generally, the number of sexual partners is the strongest attributer of risk of HIV acquisition. The plot result revealed that the number of new HIV infection is largely due to the influence of increased number of sexual partners. Thus, in order to reduce the spread of the disease, the number of sexual partners as well as unsafe sexual interaction with infected individuals must be avoided.

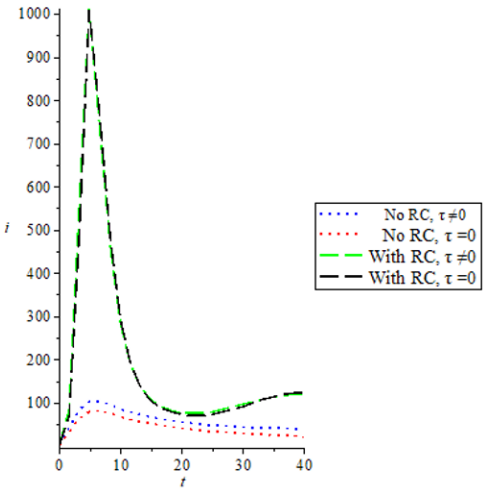

Figure 7. The combine effect of infective immigrants and risk compensation

Figure 7 shows the effect of risk compensation (RC) $\psi = 1$ on the infective class in a population with or without infected immigrant. Immigration poses a significant risk for disease dissemination, the effect may be substantial in developing countries which normally do not check the complete health status of immigrants. These immigrants place their partners in their home countries and their destination at risk of HIV/AIDS. It can be observed from Figure 7 that the infective immigrants has no much effect on the dynamic due to risk compensation as indicated by the black and green curves.

Between the red and blue curves shows a little increase due to the presence of infected immigrants on a dynamic with no RC $\psi = 0$. This means that the presence of immigrants increases the total population of the infective initially, making the infection more endemic and persistent in the population then decreasing with time. It is clear that direct inflow of infected immigrants and RC increases the numbers of infective which ultimately increase the prevalence of the disease. To check the spread of disease and prevent the endemicity in the population, effective immigration policies such as screening of immigrants should be taken seriously. Simulation results in Figure 7 shows that in the popu-

lation where the PrEP strategy is in place, the peak of epidemic is higher in the presence of infected immigrants than in the absence of infected immigrants but either way it still reduces infection. Thus PrEP can leads to reduction in the risk of sexual transmission of HIV even with constant influx of infected immigrants. The public health implications is that they should enhance screening and testing for immigrant and targeted interventions for high risk groups should be pursued radically.

5. Conclusion

In this work, a nonlinear deterministic mathematical model $\psi = 1$ was proposed and analyzed to study the effect of risk compensation among PrEP users on the dynamics of HIV/AIDS with variable inflow of infective immigrants, and also incorporating treatment at different stage of infection using the standard incidence. Analysis of the model showed that as long as the infective immigrants are entering the population, the disease free equilibrium become unattainable and the existence and uniqueness of Disease Free Equilibrium (DFE) of the model without infective immigrants and Endemic Equilibrium Point (EEP) were proved depending on the basic reproduction number R_1. It was established that DFE is locally asymptotically stable in the absence of infected immigrants if $R_1 < 1$ and for $R_1 > 1$ it is unstable. The model exhibited a forward bifurcation and the global stability of the Endemic Equilibrium point was shown to exist using the Lyapunov function and LaSalle's invariance principle when $R_1 > 1$. It was also established that the risk compensation $\psi = 1$ and multiple number of sexual partners (c) can be a barrier to the adherence of PrEP. The effectiveness of PrEP can only be harnessed by strict adherence to usage of the medication.

Numerical simulation of model (1) with PrEP ($\omega = 0.9$) or without PrEP ($\omega = 0$) were compared. It was observed that PrEP reduces the number of new HIV incidences, however, from simulation values 90% susceptible will have to be on the PrEP strategy for its effect to be felt in the population because of risk compensation among users, that is on the high side considering the cost of PrEP. Likewise, numerical results revealed that an increase in the number of sexual partner c results in the increase of the incidence of the HIV/AIDS infection. Simulation also shows that immigration of infected individuals plus high-risk behaviour of PrEP users can hinder the effectiveness of PrEP in preventing the HIV infection; the rate of spread of the diseases will be massive as in the case where there is no protection. To curb HIV transmission among migrants, policymakers must address the social determinants of health, ensure universal access to healthcare, and implement targeted interventions tailored to migrants' specific needs. Effective prevention strategies for migrants must incorporate cultural sensitivity, language accessibility, mobile health services, peer education, economic empowerment initiatives, and collaboration with local community organizations. We acknowledge several limitations in this study. Notably, mathematical models incorporating risk compensation are relatively scarce, and further research and data collection are necessary to fully capture the complexities of this phenomenon and its impact on disease transmission. Likewise the sensitivity analysis was provided but it doesn't account for uncertainty in parameter estimation. Future studies will capture the use of Latin Hypercube Sampling/Partial Rank Correlation Coefficient (LHS/PRCC). Likewise the optimal control of HIV/AIDS with respect to PrEP still need to be carried out on the effect of risk compensation in undermining the protection that PrEP has to offer should be explored.

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Abbreviations.

PrEP	:	Pre-Exposure Prophylaxis
HIV	:	Human Immunodeficiency Virus
AIDS	:	Acquired Immunodeficiency Syndrome
RC	:	Risk Compensation

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