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Mathematical Modeling of HIV/AIDS Transmission Dynamics Incorporating Pre-Exposure Prophylaxis (PrEP) Users

OA Odebiyi [1*], JK Oladejo [2], AA Taiwo [3], EO Elijah [4], OA Olajide [5], WO Salahu [6]
[1, 2, 3, 5, 6] Department of Pure and Applied Mathematics, Ladoke Akintola University of Technology, Ogbomoso, Nigeria
[4] Department of Mathematics, Federal University of Technology, Minna Niger State, Nigeria

* Corresponding Author: **OA Odebiyi**

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Abstract
HIV/AIDS continues to pose significant global health concerns, underscoring the need for mathematical modelling to inform evidence-based intervention and policy decisions. This study explores the global Stability of HIV/AIDS Dynamics with PrEP Intervention and a six-compartment model which includes susceptible, vaccinated-PrEP, asymptomatic infective, symptomatic infective, treated and AIDS population is presented. The validity of the solution states affirms that the model is well-defined and holds epidemiologically significant. The basic reproduction number (R_0) was obtained using the next generation matrix. The disease-free and endemic equilibrium points were investigated, with a comprehensive analysis of both local and global stability. Sensitivity analysis was carried out using normalized forward sensitivity index. The outcome from stability and sensitivity analysis suggest promising prospects for mitigating HIV/AIDS spread in the population. The sensitivity analyses identify critical parameters influencing disease transmission. By applying the insights gained from this analysis, stakeholders can develop evidence-based policies and interventions to reduce the spread of HIV/AIDS transmission in the population.

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1. Introduction

HIV originally came from a virus particular to chimpanzees in West Africa during the 1930s, and originally transmitted to humans through the transfer of blood through hunting. Over the decades, the virus spread through Africa, and to other parts of the world (CANFAR 2023). HIV remains a major global public health issue, having claimed 40.4 million [32.9–51.3 million] lives so far with ongoing transmission in all countries globally; with some countries reporting increasing trends in new infections. There were an estimated 39.0 million [33.1–45.7 million] people living with HIV at the end of 2022, two thirds of whom (25.6 million) are in the WHO African Region. In 2022, 630 000 [480 000–880 000] people died from HIV-related causes and 1.3 million [1.0–1.7 million] people acquired HIV. WHO, the Global Fund and UNAIDS all have global HIV strategies that are aligned with the SDG target 3.3 of ending the HIV epidemic by 2030. When considering all people living with HIV, 86% [73–>98%] knew their status, 76% [65–89%] were receiving antiretroviral therapy and 71% [60–83%] had suppressed viral loads. (CANFAR 2023; WHO 2023; UNAIDS 2023).

However, this infection has no curing medication and the Control of HIV/AIDS is not yet over (Udoo, *et al*.,2015) [2], but with access to effective HIV prevention, diagnosis, treatment and care, PrEP usage, and antiretroviral therapy (ART) or its combination has helped in halting further progression of infection but if left untreated leads to a severe stage called acquired immunodeficiency syndrome (AIDS). HIV infection has become a manageable chronic health condition, enabling people living with HIV to lead long and healthy lives. HIV infection progresses through stages as follows: (i) primary stage (asymptomatic stage): this stage faces human individuals where the virus is in the blood and cannot be diagnosed with medical instruments; (ii)

asymptomatic stage: this stage is a symptomless stage of HIV infection but diagnosable with a medical test; (iii) symptomatic stage: in this stage, the symptoms of HIV infection like tiredness, loss of weight, and extreme loss of water start to manifest in the life of HIV-infected individuals; and (iv) AIDS stage: this is an advanced stage of HIV infection where it is difficult for treatment and leads to death soon if special care is not taken. Modes of HIV transmission are through unsafe sexual practices with HIV-infected persons, through contacts of normal blood with HIV-infected blood, infected mother to child transmission through breast or at birth time, and any contacts of HIV contains fluids of human's with HIV-negative human fluids. However, in safe practice, the risk of HIV transmission can be reduced by using the principle of abstinence–be faithful–use condom (ABC). Infected individuals face disproportionately high rates of infectious diseases, compounded by restricted access to preventive measures. To understand the transmission and mitigation of diseases within infective population and their broader impact on public health, dynamic modelling approaches are employed (Martial *et al*. 2018).

Effective interventions and health care services are critical to addressing this public health concern. Criminalizing drug use traps individuals in a cycle of incarceration, exacerbating health risks for HIV, TB, and other diseases among vulnerable populations like people who inject drugs (PWID). The PrEP landscape has evolved significantly in years since the original brief was released (WHO 2015) both in scale and variety with over 75% from African region.

Pre-Exposure Prophylaxis (PrEP) is a medication regime taken daily by HIV-negative individuals to prevent HIV infection. Its primary function is to prevent HIV replication. PrEP medication such as tenofovir/emtricitabine inhibit HIV replication in the body. Also, PrEP is highly effective (up to 99%) in preventing HIV infection when taken consistently (Oladejo and Oluyo, 2022). It provides protection against HIV infection, even if other prevention methods (e.g, condoms) fail or are not used and also reduces transmission risk, especially for high-risk groups such as the sexually active individuals and injection drug users.

PrEP involves the use of antiretroviral drugs, either as oral pills or injectables, to reduce the risk of HIV infection in individuals who are at high risk of acquiring the virus. The world Health Organization (WHO) recommends PrEP as preventive measure for people who may be exposed to HIV [2015, 2019]. It is important strategy in HIV prevention efforts.

Numerous trials and studies involving some key populations have demonstrated that the use of PrEP is a secure and effective method for preventing HIV infection. Mathematical models have been employed to project the impact of early diagnosis and treatment on the HIV epidemic over an extended period, in comparison to the present scenario. Similarly, models incorporating PrEP have been utilized to illustrate a decrease in HIV incidence when provided to individuals at a high risk.

Oladejo and Oluyo (2022), proposed a mathematical model that incorporates PrEP as a control strategies to control the spread of HIV/AIDS in a population with direct inflow of infective immigrants. Result shows that the disease free is unattainable as long as there is an influx of infective immigrants.

This study present a mathematical analysis of an HIV/AIDS model that accounts for the HIV/AIDS infective population and the vaccinated susceptible population that uses PrEP as a preventive measure for people who are uninfected but are at substantial risk of been infected with the HIV infection, thereby reducing the risk of HIV infection in individuals who are at high risk of acquiring the virus. Also, to carry out sensitivity analysis in order to know which of the parameter is most sensitive and its implications to the basic reproduction number and to examine the global stability. The results of this study can provide valuable insights for policymakers and public health officials seeking to develop effective control strategies for HIV/AIDS.

2. Methodology

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \beta(I_1 + \eta I_2)S - \mu S - \alpha S + \varepsilon V \\
\frac{dV}{dt} &= \alpha S - (\mu + \varepsilon)V \\
\frac{dI_1}{dt} &= \beta(I_1 + \eta I_2)S - (\theta + \mu + \sigma)I_1 \\
\frac{dI_2}{dt} &= \theta I_1 - (\mu + \delta + d)I_2 \\
\frac{dT}{dt} &= n\delta I_2 + \sigma I_1 - (\omega + \mu)T \\
\frac{dA}{dt} &= (1-n)\delta I_2 + \omega T - (\mu + d)A
\end{aligned}\right\} \quad (1)$$

$$S(0) = S_0, V(0) = V_0, I_1(0) = I_{1_0}, I_2(0) = I_{2_0}, A(0) = A_0 \quad (2)$$

The model parameters used in the model is defined as follows

Table 1: Model variables and parameters

Parameters/ variables	Description
π	
β	Transmission rate
d	AIDS related death rate
θ	Pace at which asymptomatic population become aware of being infected after a screening process.
μ	Natural mortality rate unrelated to AIDS
ε	Fraction of vaccinated that become susceptible.
σ	Progression rate from Asymptomatic class to AIDS class.
δ	Progression rate from Symptomatic class to Treated and AID class.
η α	Infectivity rate of transmission Fraction of persons placed on prep strategy.
$S(t)$ $V(t)$	Susceptible population at a given time(t). Vaccinated susceptible
$I_1(t)$	Asymptomatic population at a given time
$I_2(t)$ $T(t)$ $A(t)$	Symptomatic population at a given time. Treated population AIDS population

In this study, the susceptible and infectious epidemic model (SI) is presented. A population of $N(t)$ was partitioned in to subclasses which are susceptible, vaccinated susceptible, asymptomatic infective population, symptomatic infective population, Treated infective population and AIDS population on the classes. The size denoted by $S(t), V(t), I\ (t), I_2(t), T(t), A(t)$ respectively as shown in figure 1 below:

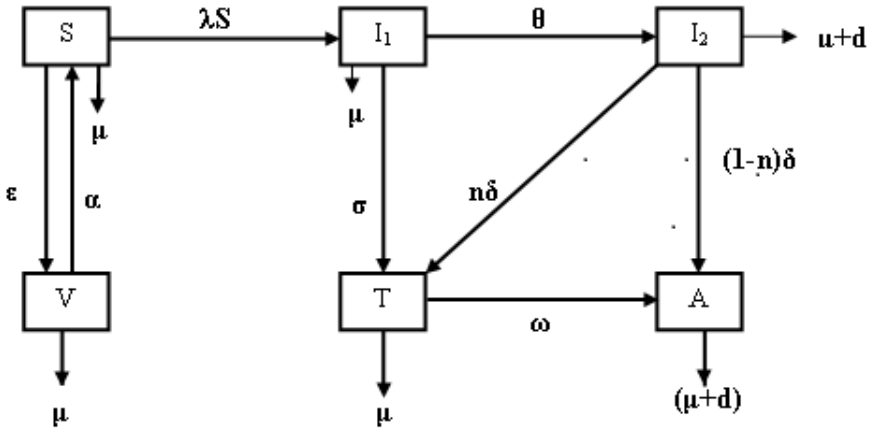

Fig 1: Transmission diagram for susceptible-infected (S-I) model

2.1 Positivity and boundedness of the model: In this section we shall show from model (1) that the state variables are non-negative and the solutions remain positive for all $t \geq 0$. Hence, the parameters in the model are assumed to be positive.

Theorem 1: Let the initial conditions or values of the state variables be such that $\{(S(0) \geq 0, V(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, T(0) \geq 0, A(0) \geq 0) \in \Omega\}$, then the set $(S(t), I_1(t), I_2(t), T(t), A(t))$ is non-negative in Ω for all $t \geq 0$.

Proof: Considering the first equation in (1), are considered for the positivity of the state variables as follows using the approach of (Adeyemi and Oluyo, 2023; Oladejo and Oluyo, 2022; Odebiyi *et al*., 2024; Temesgen *et al*., 2023) [12, 1, 18]

$$\frac{dS}{dt} \geq -(\beta(I_1 + \eta I_2) + \mu + \alpha)S$$

$$\frac{dS}{dt} - \int (\beta(I_1 + \eta I_2) + \mu + \alpha)S$$

Using variable separable

$$\frac{dS}{S} \geq -\int \left(\beta\left(I_1 + \eta I_2\right) + \mu + \alpha\right)dt$$

$$\ln s \geq -\left(\beta\left(I_1 + \eta I_2\right) + \mu + \alpha\right)t + C$$

$$S(t) \geq e^{-(\beta(I_1+\eta I_2)+\mu+\alpha)t}.e^{C_1}$$

$$S(t) = S_0 e^{-(\beta(I_1+\eta I_2)+\mu+\alpha)t}.$$

$$S(0) = S_0 \Rightarrow A_1 = S_0$$

Since $S(t) \geq 0,$ for all $t > 0$ provided that $S_0 \geq 0.$

Hence, $S(t) \geq 0$

It is possible to show using the same procedure for other state variables that:

$$I_1(t) \geq I_1(0)e^{-(\theta+\mu+\sigma)t} \geq 0, V(t) \geq V(0)e^{-(\mu+\varepsilon)t} \geq 0, I_2(t) \geq I_2(0)e^{-(\mu+\gamma+d)t} \geq 0,$$
$$T(t) \geq T(0)e^{-(\sigma+\mu)t} \geq 0, A(t) \geq A(0)e^{-(\mu+d)t} \geq 0$$

This shows that all the solutions of equation (1) are positive for all $t \geq 0$. Therefore, the HIV/AIDS transmission model stated in (1) is both epidemiologically significant and numerically well posed in an attainable given region $\Omega \geq 0$

Theorem2: Every solution in the region $\Omega = \left\{\left(S(t), V(t), I_1(t), I_2(t), T(t), A(t) \in \Omega_+^6 : N(t) \leq \frac{\pi}{\mu}\right)\right\}$ is positively invariant with respect to the HIV/AIDS model (1) in the populations. The solutions for the system are contained and remain in the region Ω for all time $t \geq 0.$

Proof: Considering the equation of the model, and adding up all the derivatives with respect to time $t,$ we obtained

$$\frac{dN(t)}{dt} = \pi + \mu\left(S + V + I_1 + I_2 + T + A\right) - dA$$

Let d=0

$$\frac{dN(t)}{dt} = \pi + \mu N$$

$$N \leq \frac{\pi}{\mu} + \left(N_0 - \frac{\pi}{\mu}\right)e^{-\mu t}$$

Where N_0 is the initial size of the population
Therefore,

$$\lim_{t\to\infty} N(t) \leq \frac{\pi}{\mu}$$

This result implies that HIV/AIDS model (1) has non-zero negative and bounded solution in the region Ω and all the solutions starting in Ω approach, enter or stay in Ω . Hence, it is sufficient to conclude that the model is epidemiologically well posed.

2.2 Existence and uniqueness of solution of the model

In this section, we establish conditions for the existence and uniqueness of a solution of our model. We shall rigorously employ Picard theorem to achieve this.

Theorem 3: There exists a unique solution for the transmission of HIV model (1).

Consider the system of equations below:

$$\begin{aligned} x_1' &= f_1(x_1, x_2, \cdots, x_n, t), x_1(t_0) = (x_1)_0 \\ x_2' &= f_2(x_1, x_2, \cdots, x_n, t), x_2(t_0) = (x_2)_0 \\ &\vdots \\ x_n' &= f_n(x_1, x_2, \cdots, x_n, t), x_n(t_0) = (x_n)_0 \end{aligned} \tag{3}$$

The model can be written in a compact form as follows

$$x' = f(t, x), x(t_0) = x_0 \tag{4}$$

Theorem 4: Let f be continuous in a domain

$$D = \left\{(x,t) : |t - t_0| \le a, \|x - x_0\| \le b, x = (x_1, x_2, \ldots x_n), x_0 = \{(x_1)_0, (x_2)_0, \cdots, (x_n)_0\}\right\} \tag{5}$$

Suppose that $f(t,x)$ satisfies Lipschitz condition

$$\|f(x_1,t) - f(x_2,t)\| \le L\|x_1 - x_2\| \tag{6}$$

Then, system (3) has a unique solution in D, where the pairs $f(x_1,t)$ and $f(x_2,t)$ belongs to D and L (Lipschitz constant) is a positive constant.

It is of great importance to note that the Lipschitz condition is satisfied by the requirement that $\frac{\partial f_1}{\partial f_j}, i, j = 1,2,\cdots,n$ be continuous and bounded in D.

Proof:
It is suffices to show Lipschitz condition property satisfied by the model and this is done as follow.
From the first compartment of equation (1), it can be shown that

$$\frac{dS}{dt} = f_1(S,t) = \frac{dS}{dt} = \pi - \beta(I_1 + \eta I_2)S - \mu S - \alpha S + \varepsilon V \tag{7}$$

$$\|f_1(S_1,t) - f_1(S_2,t)\| \le L_1\|S_1 - S_2\| \tag{8}$$

Then, from the first compartment in equation (1),
Using the inequality, $\|f_1(S_1,t) - f_1(S_2,t)\| \le L_1\|S_1 - S_2\|$,
Then (7) becomes

$$\begin{aligned} &= \pi - \beta(I_1 + \eta I_2)S_1 - \mu S_1 - \alpha S_1 + \varepsilon V - (\pi - \beta(I_1 + \eta I_2)S_2 - \mu S_2 - \alpha S_2 + \varepsilon V) \\ &= \pi - \beta(I_1 + \eta I_2)S_1 - \mu S_1 - \alpha S_1 + \varepsilon V - \pi + (\beta(I_1 + \eta I_2)S_2 + \mu S_2 + \alpha S_2 + \varepsilon V) \end{aligned} \tag{9}$$

Further simplification of (9) gives

$$= \|-(\beta(I_1 + \eta I_2) + \mu + \alpha)S_1 + (\beta(I_1 + \eta I_2) + \mu + \alpha)S_2\|$$

$$= \|(-(\beta(I_1 + \eta I_2) + \mu + \alpha)S_1 + (\beta(I_1 + \eta I_2) + \mu + \alpha)S_2)\|$$

$$= |-1|\|(\beta(I_1 + \eta I_2) + \mu + \alpha)(S_1 - S_2)\|$$

$$\le |-1|\|[(\beta(I_1 + \eta I_2) + \mu + \alpha)](S_1 - S_2)\|$$

$$\leq \|[(\beta(I_1 + \eta I_2) + \mu + \alpha)](S_1 - S_2)\| \tag{10}$$

Using the limiting value where $I_1 \leq I_2 \leq T \leq A \leq \frac{\pi}{\mu}$,then it follows from (10) that

$$\leq \|[(\beta(I_1 + \eta I_2) + \mu + \alpha)](S_1 - S_2)\|$$

$$\|f_1(S_1,t) - f_1(S_2,t)\| \leq \left\| \left(\beta\left(\frac{\pi}{\mu} + \eta\frac{\pi}{\mu}\right) + \mu + \alpha \right)(S_1 - S_2) \right\| \tag{11}$$

$$\leq \left\| \left(\beta\left(\frac{\pi}{\mu} + \eta\frac{\pi}{\mu}\right) + \mu + \alpha \right) \right\| \|(S_1 - S_2)\| \tag{12}$$

Therefore, it follow from equation (8), $\|f_1(S_1,t) - f_1(S_2,t)\| \leq L_1 \|S_1 - S_2\|$

This implies that $f_1(S_1,t)$ is Lipschitz continuous with Lipschitz constant where

$$L_1 = \left(\beta\left(\frac{\pi}{\mu} + \eta\frac{\pi}{\mu}\right) + \mu + \alpha \right) \tag{13}$$

Similarly re-writing the second compartment of the equation model

$$\frac{dV}{dt} = f_2(t,x) = \alpha S - (\mu + \varepsilon)V \tag{14}$$

It is important to show that $f_2(V_2,t)$ satisfies the Lipschitz condition

$$\|f_2(V_1,t) - f_2(V_2,t)\| \leq L_2 \|V_1 - V_2\| \tag{15}$$

Where L_2 is the Lipschitz constant greater than zero, then equation (15) become

$$\|f_1(V_1,t) - f_1(V_2,t)\| = \|\alpha S - (\mu + \varepsilon)V_1 - \alpha S - (\mu + \varepsilon)V_2\| \tag{16}$$

$$= \|\alpha S - (\mu + \varepsilon)V_1 - (\alpha S - (\mu + \varepsilon)V_2)\| \tag{17}$$

Further simplification of (16) gives

$$\begin{aligned} &= \|-(\mu + \varepsilon)V_1 + ((\mu + \varepsilon)V_2)\| \\ &= \|(-1)(\mu + \varepsilon)[V_1 - V_2]\| \\ &\leq |-1|\|(\mu + \varepsilon)[V_1 - V_2]\| \\ &\leq \|(\mu + \varepsilon)[V_1 - V_2]\| \end{aligned} \tag{18}$$

Therefore, it follow from (18), $\|f_2(V_1,t) - f_2(V_2,t)\| \leq \|(\mu + \varepsilon)(V_1 - V_2)\|$

This implies that $f_2(V_2,t)$ is Lipschitz continuous with Lipschitz constant

$$L_2 = (\mu + \varepsilon) \tag{19}$$

Similarly re-writing the third compartment of the equation model

$$\frac{dI_1}{dt} = f_3(t,x) = \beta(I_1 + \eta I_2)S - (\theta + \mu + \sigma)I_1 \tag{20}$$

It is important to show that $f_2(I_{12},t)$ satisfies the Lipschitz condition

$$\|f_3(I_{11},t) - f_3(I_{12},t)\| \leq L_3 \|I_{11} - I_{12}\|$$

Where L_3 is the Lipschitz constant greater than zero, then equation (20) become

$$\|f_3(I_{11},t)-f_3(I_{12},t)\|=\|\beta(I_1+\eta I_2)S-(\theta+\mu+\sigma)I_1-\beta(I_1+\eta I_2)S-(\theta+\mu+\sigma)I_2\| \quad (21)$$

Further simplification of (21) gives

$$=\|f_3(I_{11},t)-f_3(I_{12},t)\|=\|\beta(I_1+\eta I_2)S-(\theta+\mu+\sigma)I_{11}-(\beta(I_1+\eta I_2)S+(\theta+\mu+\sigma)I_{12}\|$$

$$=\|-((\theta+\mu+\sigma)I_{11})+(\theta+\mu+\sigma)I_{12}\|$$

$$=\|(-1)[(\theta+\mu+\sigma)](I_{11}-I_{12})\|$$

$$\leq|(-1)|\|[(\theta+\mu+\sigma)](I_{11}-I_{12})\|$$

$$\leq\|(\theta+\mu+\sigma)(I_{11}-I_{12})\| \quad (22)$$

Therefore, it follow from equation $\|f_3(I_{11,}t)-f_3(I_{12,}t)\|\leq\|(\theta+\mu+\sigma)(I_{11}-I_{12})\|$

This implies that $f_3(I_{13},t)$ is Lipschitz continuous with Lipschitz constant

$$L_2=(\theta+\mu+\sigma) \quad (23)$$

In a Similar manner, re-writing the forth compartment of equation(1)

$$\frac{dI_2}{dt}=f_4(I_2,t)=\theta I_1-(\mu+\delta+d)I_2 \quad (24)$$

It is important to show that $f_3(I_2,t)$ satisfies the Lipschitz condition

$$\|f_4(I_{21},t)-f_4(I_{22},t)\|\leq L_4\|I_{21}-I_{22}\| \quad (25)$$

Where L_4 is the Lipschitz constant greater than zero, then equation (24) become

$$\|f_3(I_{21},t)-f_3(I_{22},t)\|=\|\theta I_1-(\mu+\gamma+d)I_{21}-(\theta I_1-(\mu+\gamma+d)I_{22}\| \quad (26)$$

Further simplification of (26) gives

$$\|f_3(I_{21},t)-f_3(I_{22},t)\|=\|\theta I_1-(\mu+\delta+d)I_{21}-\theta I_1+(\mu+\delta+d)I_{22}\|$$
$$=\|(-(\mu+\delta+d))[I_{21}-I_{22}]\|$$
$$\leq\|(-1)((\mu+\delta+d))[I_{21}-I_{22}]\|$$
$$\leq|-1|\|(\mu+\delta+d)[I_{21}-I_{22}]\| \quad (27)$$

Therefore, it follows from equation (27)

$f_4(I_{21},t)-f_4(I_{22},t)\leq(\mu+\delta+d)\|I_{21}-I_{22}\|$

$$\|f_4(I_{21},t)-f_4(I_{22},t)\|\leq L_4\|I_{12}-I_{22}\|$$

This implies that $f_4(I_{24,}t)$ is Lipschitz continuous with Lipschitz constant

$$L_4=(\mu+\delta+d) \quad (28)$$

In a Similar manner, re-writing the fifth compartment of equation (1)

$$\frac{dT}{dt}=f_5(t,x)=n\delta_2 I_2+\sigma I_1-(\varpi+\mu)T \quad (29)$$

It is important to show that $f_5(T_5,t)$ satisfies the Lipschitz condition

$$\|f_5(T_1,t)-f_5(T_2,t)\|\leq L_5\|T_1-T_2\| \quad (30)$$

Where L_5 is the Lipschitz constant greater than zero, then equation (29) become

$$\|f_5(T_1,t)-f_5(T_2,t)\|=\|n\delta_2 I_2+\sigma I_1-(\omega+\mu)T-(n\delta_2 I_2+\sigma I_1-(\omega+\mu)T)\| \quad (31)$$

Further simplification of (31) gives

$$\|f_5(T_1,t)-f_5(T_2,t)\|=\|-(\omega+\mu)T_1+(\omega+\mu)T_2\|$$

$$=\|-(\omega+\mu)T_1+(\omega+\mu)T_2\|$$

$$\leq(|-1|)\|(\omega+\mu)[T_1-T_2]\|$$

$$\leq\|(\omega+\mu)[T_1-T_2]\| \quad (32)$$

Therefore, it follow from equation (31)

$$\|f_5(T_1,t)-f_5(T_2,t)\|\leq(\omega+\mu)\|T_1-T_2\|$$

$$\|f_5(T_1,t)-f_5(T_2,t)\|\leq L_5\|T_1-T_2\|$$

This implies that $f_5(T_5,t)$ is Lipschitz continuous with Lipschitz constant where,

$$L_5=(\omega+\mu) \quad (33)$$

Re-writing the sixth compartment of the equation model

$$\frac{dA}{dt}=f_6(A,t)=(1-n)\delta_2 I_2+\sigma T+\omega A-(\mu+d)A \quad (34)$$

It is important to show that $f_6(A_6,t)$ satisfies the Lipschitz condition

$$\|f_6(A_1,t)-f_6(A_2,t)\|\leq L_6\|A_1-A_2\| \quad (35)$$

Where L_6 is the Lipschitz constant greater than zero, then equation (34) become

$$\|f_6(A_1,t)-f_6(A_2,t)\|=\|(1-n)\delta_2 I_2+\sigma T+\omega A-(\mu+d)A-((1-n)\delta_2 I_2+\sigma T+\omega A-(\mu+d)A)\| \quad (36)$$

Further simplification of (36) gives

$$\|f_5(A_1,t)-f_5(A_2,t)\|=\|(1-n)\delta_2 I_2+\sigma T+\omega A_1-(\mu+d)A_1-(1-n)\delta_2 I_2+\sigma T-\omega A_2+(\mu+d)A_2\|$$

$$=\|-(\mu+d)A_1+\omega A_1+(\mu+d)A_2+\omega A_2\|$$

$$=\|(-1)(\mu+d+\omega)(A_1-A_2)\|$$

$$\leq|-1|\|(\mu+d+\omega)(A_1-A_2)\|$$

$$\leq\|(\mu+d+\omega)(A_1-A_2)\| \quad (37)$$

Therefore, it follow from equation $\|f_6(A_1,t)-f_6(A_2,t)\|\leq(d+\mu)\|A_1-A_2\|$

This implies that $f_6(A_6,t)$ is Lipschitz continuous with Lipschitz constant

$$L_6=(\mu+d+\omega) \quad (38)$$

Hence, the HIV model (1) has a unique solution in the region D .

2.3 Mathematical Analysis of the Model

2.3.1 **Disease free equilibrium point***:* This is a state where the disease is completely eliminated from the population, that is, there is no infected individuals, no disease transmission and population is entirely susceptible.

At the equilibrium,

$$\frac{dS}{dt}=\frac{dV}{dt}=\frac{dI_1}{dt}=\frac{dI_2}{dt}=\frac{dT}{dt}=\frac{dA}{dt}=0$$

$$\left.\begin{aligned}
&\pi-\beta(I_1+\eta I_2)S-\mu S-\alpha S+\varepsilon V=0\\
&\alpha S-(\mu+\varepsilon)V=0\\
&\beta(I_1+\eta I_2)S-(\theta+\mu+\sigma)I_1=0\\
&\theta I_1-(\mu+\delta+d)I_2=0\\
&n\delta I_2+\sigma I_1-(\omega+\mu)T=0\\
&(1-n)\delta I_2+\omega T-(\mu+d)A=0
\end{aligned}\right\}$$

At the disease free equilibrium, we set $S\neq 0, V=0, I_1=I_2=T=A=0$ Substituting these to equation (1) and solving gives the infection – free equilibrium as

$$E^0=\left(S^0,V^0,I_1^0,I_2^0,A^0\right)=\left(\frac{\pi(\mu+\varepsilon)}{\mu(\mu+\varepsilon+\alpha)},\frac{\alpha\pi}{\mu(\mu+\varepsilon+\alpha)},0,0,0,0\right) \tag{39}$$

2.3.2 **Endemic equilibrium point***:* The endemic equilibrium is a steady- state solution where the disease persists in the population at a stable level. At endemic equilibrium, $S\neq 0, V\neq 0, I_1\neq 0, I_2\neq 0, T\neq 0, A\neq 0$

Solving equations (1) simultaneously, we have the endemic equilibrium points of the HIV model (1) designated by $\varepsilon_0^*=\left(S^*,I_a{}^*,I_s{}^*,T^*\,A^*\right)$ is obtained as

$$\left\{\begin{aligned}
s^* &= \frac{1}{\left[K_1(\lambda^*+\mu+\alpha)-(\pi\alpha+\varepsilon\alpha)\right]}\\
V^* &= \frac{\alpha}{K_1}\left[\frac{1}{(K_1(\lambda^*+\mu+\alpha))-(\pi\alpha+\varepsilon\alpha)}\right]\\
I_1^* &= \frac{\lambda^*}{K_1}\left[\frac{1}{(K_1(\lambda^*+\mu+\alpha))-(\pi\alpha+\varepsilon\alpha)}\right]\\
I_2^* &= \frac{\theta}{K_2K_3}\left[\frac{\lambda^*}{(K_1(\lambda^*+\mu+\alpha))-(\pi\alpha+\varepsilon\alpha)}\right]\\
T^* &= \frac{n\delta\theta\lambda^*}{K_2K_3K_4}\left[\frac{1}{(k_1(\lambda^*+\mu+\alpha))-(\pi\alpha+\varepsilon\alpha)}\right]+\frac{\sigma\lambda^*}{K_2k_4}\left[\frac{1}{(K_1(\lambda^*+\mu+\alpha))-(\pi\alpha+\varepsilon\alpha)}\right]\\
A^* &= \frac{(1-n)\delta}{K_2K_3K_5}\left[\frac{\theta\lambda^*}{(K_1(\lambda^*+\mu+\alpha))-(\pi\alpha+\varepsilon\alpha)}\right]+\sigma\left(\begin{aligned}&\frac{n\delta\theta\lambda^*}{K_2K_3K_4}\left[\frac{1}{(K_1(\lambda^*+\mu+\alpha))-(\pi\alpha+\varepsilon\alpha)}\right]+\\&\frac{\sigma\lambda^*}{K_2k_4}\left[\frac{1}{(K_1(\lambda^*+\mu+\alpha))-(\pi\alpha+\varepsilon\alpha)}\right]\end{aligned}\right)
\end{aligned}\right. \tag{40}$$

Where,

$K_1=(\mu+\varepsilon), K_2=(\theta+\mu+\sigma), K_3=(\mu+\delta+d), K_4=(\omega+\mu), K_5=(\mu+d)$

2.3.3 **Derivation of Basic Reproduction Number,** R_0*:* This is a threshold parameter. It represents the average number of secondary infections generated by a single infected individual in a completely susceptible population. For example if $R_0<1,$ means Disease will decline and eventually die out. If $R_0=1,$ Disease will remain stable and if $R_0>1,$ Disease will spread and potentially lead to an outbreak. The basic reproduction number predicts disease spread and potential outbreaks, guides control measures (e.g., vaccination, quarantine), and helps evaluate effectiveness of interventions. The next-generation matrix is determined by analyzing new infection pathways and also infection transmission between compartments.

F_i and V_i are calculated as follows using the approach of (Van den Driessche and Watmough, 2002)

$$f_i = \begin{bmatrix} \beta(I_1 + \eta I_2)S \\ 0 \\ 0 \\ 0 \end{bmatrix}, \quad v_i = \begin{bmatrix} -(\theta+\mu+\sigma)I_1 \\ \theta I_1 - (\mu+\delta+d)I_2 \\ {}^{1} \\ n\delta I_2 + \sigma I_1 - (\omega+\mu)T \\ (1-n)\delta I_2 + \omega T - (\mu+d)A \end{bmatrix} \tag{41}$$

The Jacobian matrices of F_i and V_i at the disease free equilibrium point, $S_0 = \frac{\pi(\mu+\varepsilon)}{\mu(\mu+\varepsilon+\alpha)}$, are:

$$DF(E_0) = \left[\frac{\partial F_i(E_0)}{\partial x_j}\right] = \begin{bmatrix} \beta S_0 & \beta\eta S_0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{42}$$

$$DV(E_0) = \left[\frac{\partial V_1(E_0)}{\partial x_j}\right] = \begin{bmatrix} G_1 & 0 & 0 & 0 \\ -\theta & G_2 & 0 & 0 \\ -\sigma & -n\delta_2 & G_3 & 0 \\ 0 & -(1-n)\delta & -\omega & G_4 \end{bmatrix} \tag{43}$$

Where, $G_1 = (\theta+\mu+\sigma)$, $G_2 = (\mu+\delta+d)$, $G_3 = (\omega+\mu)$, $G_4 = (\mu+d)$

$$K_1 = (\mu+\varepsilon), K_2 = (\theta+\mu+\sigma), K_3 = (\mu+\delta+d), K_4 = (\omega+\mu), K_5 = (\mu+d)$$

$$V^{-1} = \begin{bmatrix} \frac{1}{G_1} & 0 & 0 & 0 \\ \frac{\theta}{G_1G_2} & \frac{1}{G_2} & 0 & 0 \\ \frac{n\theta\delta_2 + \sigma G_2}{G_1G_2G_3} & \frac{n\delta_2}{G_2G_3} & \frac{1}{G_3} & 0 \\ -\frac{\delta n\theta K_3 - n\omega\theta\delta_2 - \delta\theta K_3 - \omega\sigma K_2}{G_1G_2G_3G_4} & -\frac{\delta nK_3 - n\omega\delta_2 - \delta K_3}{G_2G_3G_4} & \frac{\omega}{G_3G_4} & \frac{1}{G_4} \end{bmatrix} \tag{44}$$

$$FV^{-1} = \begin{bmatrix} \frac{\beta S_0}{G_1} + \frac{\beta\eta S_0\theta}{G_1G_2} & \frac{\beta\eta S_0\theta}{G_2} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{45}$$

The basic reproduction number, which is the dominant Eigen-value of the product FV^{-1}, is therefore obtained as:

$$R_0 = \frac{\beta S_0(\eta\theta + G_2)}{G_1G_2} \tag{46}$$

2.3.4 Stability analysis of the Disease free equilibrium

Theorem 4*:* The disease-free state is locally asymptotically stable if the basic reproduction number $R_0 < 1$ and unstable if otherwise.

Proof:
We evaluate the Jacobian matrix of the model at the disease free equilibrium

$$\left(\frac{\pi(\mu+\varepsilon)}{\mu(\mu+\varepsilon+\alpha)},\frac{\alpha\pi}{\mu(\mu+\varepsilon+\alpha)},0,0,0,0\right)$$

$$J(E_0)=\begin{bmatrix} -(\mu+\alpha) & \varepsilon & -\beta S_0 & -\beta\eta S_0 & 0 & 0 \\ \alpha & -(\mu+\varepsilon) & 0 & 0 & 0 & 0 \\ 0 & 0 & \beta S_0 - G_1 & \beta\eta S_0 & 0 & 0 \\ 0 & 0 & \theta & -G_2 & 0 & 0 \\ 0 & 0 & \sigma & n\delta & -G_3 & 0 \\ 0 & 0 & 0 & K_4 & \omega & -G_5 \end{bmatrix} \quad (47)$$

Where, $G_1=(\theta+\mu+\sigma)$, $G_2=(\mu+\delta+d)$, $G_3=(\omega+\mu), G_4=(1-n)\delta, K_5=(\mu+d)$
The characteristic polynomial equation is obtained as

$$(\lambda+G_5)(\lambda+G_3)(\lambda^2+c_1\lambda+c_2)(\lambda^2+b_1\lambda+b_2)=0 \quad (48)$$

Where,

$c_1=G_1+G_2-\beta S_0$, $c_2=G_1G_2(1-R_0)$, $b_1=(2\mu+\alpha-\varepsilon)$, $b_2=\mu(\alpha+\mu)-\varepsilon(\mu+2\alpha)$,

$$S_0=\frac{\pi(\mu+\varepsilon)}{\mu(\mu+\varepsilon+\alpha)}$$

The eigenvalues $\lambda_1=-G_5, \lambda_2=-G_3,$ and the remaining will be obtained from the quadratic equations (12) and (13),i.e

$$\lambda^2+C_1\lambda+C_2=0 \quad (49)$$

$$\lambda^2+b_1\lambda+b_2=0 \quad (50)$$

Where $b_1>0$ if $2\mu+\alpha>\varepsilon,\ b_2>0$ if $\mu(\alpha+\mu)>\varepsilon(\mu+2\alpha)$
$c_1>0,$ if $G_1+G_2>\beta S_0,$ and $c_2>0\Rightarrow R_0>1$

Then by Routh Hurwitz criteria, the remaining four eigenvalues are negative. Hence, the disease free equilibrium is locally asymptotically stable.

2.3.5 Global asymptotic stability of endemic equilibrium

Theorem 5: The global asymptotic stability of the HIV model (1) around the endemic equilibrium point is globally asymptotically stable in the region D whenever the basic reproduction number is greater than one.

Proof: Consider a quadratic Lyapunov function $L: D\in R_+^6\rightarrow R_+$ defined by

$$L=\frac{1}{2}\left\{(S-S^{**})+(I_a-I_a^{**})+(I_s-I_s^{**})+(T-T^{**})(A-A^{**})\right\}^2 \quad (51)$$

The time derivative of the Lyapunov function (51) is given by

$$\frac{dL}{dt}=\left\{(S-S^{**})+(I_a-I_a^{**})+(I_s-I_s^{**})+(T-T^{**})+(A-A^{**})\right\}\frac{d}{dt}(S+I_a+I_s+T+A)$$

$$\frac{dL}{dt}=\left\{(S-S^{**})+(I_a-I_a^{**})+(I_s-I_s^{**})+(T-T^{**})+(A-A^{**})\right\}\left\{\pi-\mu(S+I_a+I_s+T+A)\right\}$$

$$= -\mu\left\{\left(S - S^{**}\right) + \left(I_a - I_a^{**}\right) + \left(I_s - I_s^{**}\right) + \left(T - T^{**}\right) + \left(A - A^{**}\right)\right\}\left\{\left(S + I_a + I_s + T + A\right) - \frac{\pi}{\mu}\right\} \tag{52}$$

Since $N^{**} \leq \frac{\pi}{\mu}$, then, the following result is obtained

$$\frac{dL}{dt} \leq -\mu\left\{\left(S - S^{**}\right) + \left(V - V^{**}\right) + \left(I_1 - I_1^{**}\right) + \left(I_2 - I_2^{**}\right) + \left(T - T^{**}\right) + \left(A - A^{**}\right)\right\}\left\{\begin{matrix}\left(S + V + I_1 + I_2 + T + A\right) - \\ \left(S^{**} + V + I_1^{**} + I_2^{**} + T^{**} + A^{**}\right)\end{matrix}\right\}$$

$$= -\mu\left\{\left(S - S^{**}\right) + \left(V - V^{**}\right) + \left(I_1 - I_1^{**}\right) + \left(I_2 - I_2^{**}\right) + \left(T + T^{**}\right)\left(A - A^{**}\right)\right\} \times \left\{\begin{matrix}\left(S - S^{**}\right) + \left(V - V^{**}\right) + \left(I_1 - I_1^{**}\right) + \\ \left(I_2 - I_2^{**}\right) + \left(T - T^{**}\right) + \left(A - A^{**}\right)\end{matrix}\right\}$$

$$= -\mu\left\{\left(S - S^{**}\right) + \left(V - V^{**}\right) + \left(I_1 - I_1^{**}\right) + \left(I_2 - I_2^{**}\right) + \left(T - T^{**}\right) + \left(A - A^{**}\right)\right\}^2 \tag{53}$$

Since the time derivative of the continuously differentiable function G is negative semi-definite i.e., $\frac{dL}{dt} \leq 0$, then, the function L is a Lyapunov function. Therefore, $\frac{dL}{dt} = 0$ provided that $S = S^{**}$, $V = V^{**}$, $A = A^{**}$, $I_1 = I_1^{**}$, $I_2 = I_2^{**}$, $T = T^{**}$ and $A = A^{**}$. Then, by LaSalle's invariance principle (LaSalle, 1976), the largest invariance set for which $\frac{dL}{dt} = 0$ is the singleton set $\left\{\varepsilon^{**}\right\}$, which implies that the endemic equilibrium point of the HIV/AIDS model (1) is globally asymptotically stable.

2.3.6 Global Asymptotic Stability of the Disease free Equilibrium

Theorem 6: The disease free equilibrium point of the model is globally asymptotically stable whenever the basic reproduction number is less than unity.

Proof: Following the approach given in the global asymptotic stability of the HIV model (1) is investigated. Re-writing the HIV model (1) in a compact form as follows

$$\begin{aligned}\frac{dX}{dt} &= F\left(X,\ Z\right),\\ \frac{dZ}{dt} &= G\left(X,\ Z\right),\ G\left(X,\ 0\right) = 0,\end{aligned} \tag{54}$$

Where X is the uninfected class of the HIV model and Z is the infected classes of the model i.e. $X = S \in R_+^2$ and $Z = \left(I_1, I_2, T, A\right) \in R_+^4$. Also, let the disease-free equilibrium point of the HIV model be denoted by $\varepsilon_0 = \left(X^*, 0\right)$. Then, the following properties must be satisfied

H_1 : For $\frac{dX}{dt} = F\left(X, 0\right)$, X^* is globally asymptotically stable

H_2 : $G\left(X, Z\right) = AZ - \hat{G}\left(X,\ Z\right) \geq 0$,

Where $A = \partial G / \partial Z$, which is an M-matrix evaluated at $\left(X^*,\ 0\right)$ with non-negative off diagonal entries.

Theorem 7: The disease-free $\varepsilon_0 = \left(X^*, 0\right)$ of the HIV model (1) is globally asymptotically stable if the properties H_1 and H_2 are satisfied.

Proof: $F\left(X,\ Z\right)$ and $G\left(X,\ Z\right)$ are obtained from the HIV model (1) as

$$S(0) = S_0, V(0) = V_0, I_1(0) = I_{1_0}, I_2(0) = I_{2_0}, A(0) = A_0$$

$$F(X,\ Z)=\begin{pmatrix}\pi-\beta(I_1+\eta I_2)S-\mu S-\alpha S+\varepsilon V\\ \alpha S-(\mu+\varepsilon)V\end{pmatrix} \tag{55}$$

$$G(X,\ Z)=\begin{pmatrix}\beta(I_1+\eta I_2)S-(\theta+\mu+\sigma)I_1\\ \theta I_1-(\mu+\delta+d)I_2\\ n\delta I_2+\sigma I_1-(\omega+\mu)T\\ (1-n)\delta I_2+\omega T-(\mu+d)A\end{pmatrix} \tag{56}$$

Such that,

$$F(X,\ 0)=(\pi-\ \mu S-\alpha S\ )$$

$$\frac{dS}{dt}=\pi-(\mu+\alpha)S \tag{57}$$

Simplifying Equation (57) gives

$$S(t)=\frac{\pi}{\mu}+\left(S(0)-\frac{\pi}{\mu}\right)\ell^{-(\mu+\alpha)t} \tag{58}$$

Also, for $F(X,\ 0)=-(\ \mu+\varepsilon\ )V$

$$\frac{dV}{dt}+(\ \mu+\varepsilon\ )V=0$$

$$V(t)=V(0)e^{-(\mu+\varepsilon)t}$$

Irrespective of the initial sizes of the variables as $t\to\infty$, then, $S(t)\to\frac{\pi}{\mu}, V(t)\to 0$. Therefore, the DFE $(X^*,\ 0)$ is globally asymptotically stable satisfying property H_1.

Now, to establish the second property H_2, recall that

$$G(X,\ Z)=\begin{pmatrix}\beta(I_1+\eta I_2)S-(\theta+\mu+\sigma)I_1\\ \theta I_1-(\mu+\delta+d)I_2\\ n\delta I_2+\sigma I_1-(\omega+\mu)T\\ (1-n)\delta I_2+\omega T-(\mu+d)A\end{pmatrix} \tag{59}$$

An M-matrix whose off diagonal entries are non-negative is obtained as

$$A=\frac{\partial G}{\partial Z}=\begin{pmatrix}\beta S^*-(\theta+\mu+\sigma) & \beta S^*\eta & 0 & 0\\ \theta & -(\mu+\delta+d) & 0 & 0\\ \sigma & n\delta & -(\omega+\mu) & 0\\ 0 & \xi & \omega & -(\mu+d)\end{pmatrix} \tag{60}$$

Where $S^*=\frac{\pi}{\mu}$. Then, from

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

$$\hat{G}(X, Z) = \begin{pmatrix} \beta(I_1 + \eta I_2)(S^* - S) \\ 0 \\ 0 \\ 0 \end{pmatrix} \tag{61}$$

Since $0 \le S \le \frac{\pi}{\mu}$, clearly, it is obvious that $\hat{G}(X,Y) \ge 0, \hat{G}_2(X,Y) = 0, \hat{G}_3(X,Y) = 0$, $\hat{G}_4(X,Y) = 0$

Hence, property H_2 is satisfied. Therefore, the disease-free equilibrium of the HIV model (1) is globally asymptotically stable.

2.3.7 Sensitivity Analysis of the basic Reproduction number

Sensitivity analysis assesses how changes in model parameters, assumptions or input affect the outcomes and conclusions of the model, providing insights into the robustness and reliability of the results. By identifying the most influential parameters, sensitivity analysis informs policymakers and decision-makers on where to focus resources and efforts to maximize impact and mitigate uncertainty.

Sensitivity analysis also allow us to measure the relative change in a state variable when a parameter changes. The normalized forward sensitivity index of a variable to a parameter is a ratio of the relative change in the variable to the relative change in the parameter. When a variable is differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivatives.

Using the approach of (Chitnis, 2008) [9], the normalized forward sensitivity index of a variable "b" that depends differentiable on a parameter "m" is defined as

$$X_m^b := \frac{\partial b}{\partial m} * \frac{m}{b} \tag{62}$$

As we have an explicit formula for R_0 in equation (46), and we derive an analytical expression for the sensitivity of R_0, as $X_h^g := \frac{\partial g}{\partial h} * \frac{h}{g}$ with respect to each of the parameters involved in R_0 as computed in table 2 below:

Table 2: Sensitivity Result

Parameters	Values	Sensitivity Index	Source
β	0.0009	+1.0000	Alsheikh (2011)
π	300	+1.0000	Odebiyi *et.al* (2024) [1]
μ	0.02	-0.70007719	Ibrahim *et.al*. (2006) [14]
δ	0.2	-0.00031059	Ratera *et al*. (2012) [15]
d	1.0	-0.0015529	Alsheikh (2011) [15]
σ	0.2	-0.2797202	Ratera *et al*. (2012)
θ	0.015	-0.01833895	Alsheikh (2021)
η	0.3	0.002640	Odebiyi *et al.* (2024) [1]

2.3.8 **Interpretation of Sensitivity Indices***:* Table 2 represents the sensitivity index for the base line parameter values and it shows that recruitment rate (π), transmission rate (β), and infectivity rate (η) are the most sensitive parameters. When the parameters π, β and η increase while other parameters remain constant, the value of R_0 also increases. More so, when the parameters $\theta, \delta, \sigma, \mu$ and d increase while keeping other parameters constant, the value of R_0 also decreases. It should be targeted by intervention strategies in order to have a stable and disease free environment. For instance, $X_\beta^{R_0} =$ +1.0000 means that increasing or decreasing β by 5% increases or (decreases) R_0 by 5% while $X_\sigma^{R_0} =$ -0.01833895 means that increasing or (decreasing) R_0 by 0.09169475% as seen in table 2 below. Others can be calculated following same procedure.

3. Numerical simulations and discussion

Simulation of Simulation of the model was performed for better understanding of dynamical spread of transmission of HIV/AIDS infection using Maple 18.0 software. The simulation demonstrates model equations and reveals the impact of these parameters on the basic reproduction number.

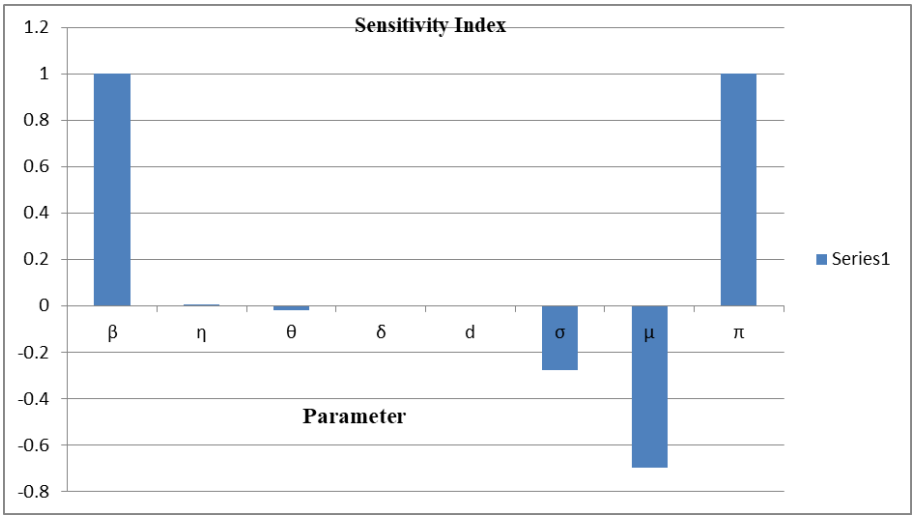

Fig 3.1: Graphical Representation of the Sensitivity indices of R_0

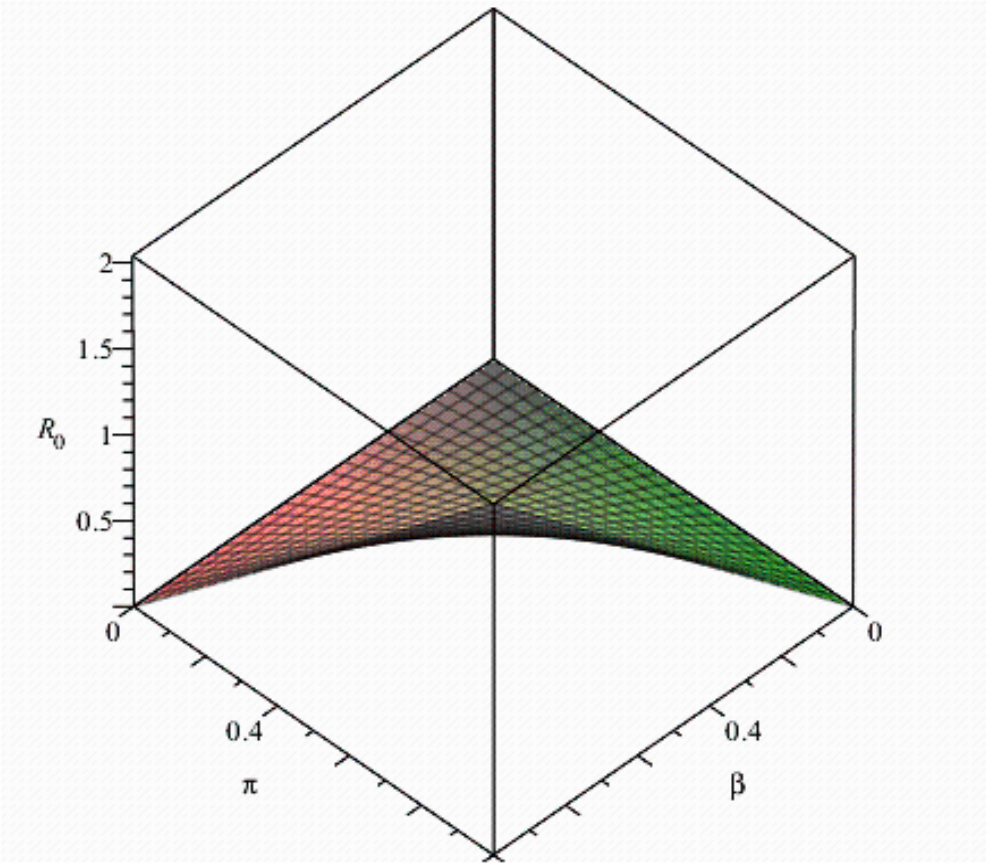

Fig 3.2: Sensitivity of the basic reproduction number to the parameters π and β

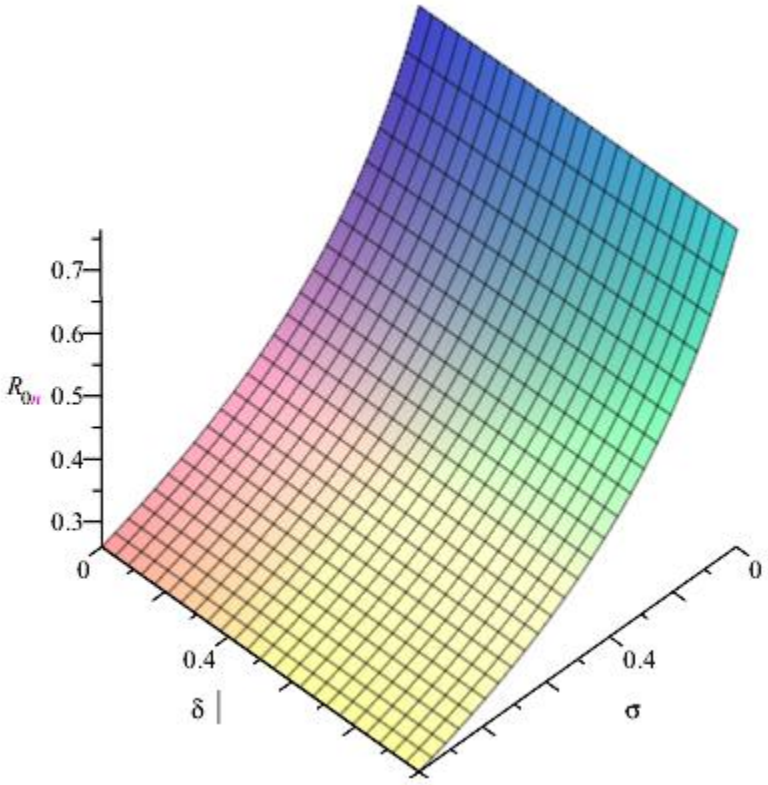

Fig 3.3: Sensitivity of the basic reproduction number to the parameters δ and σ

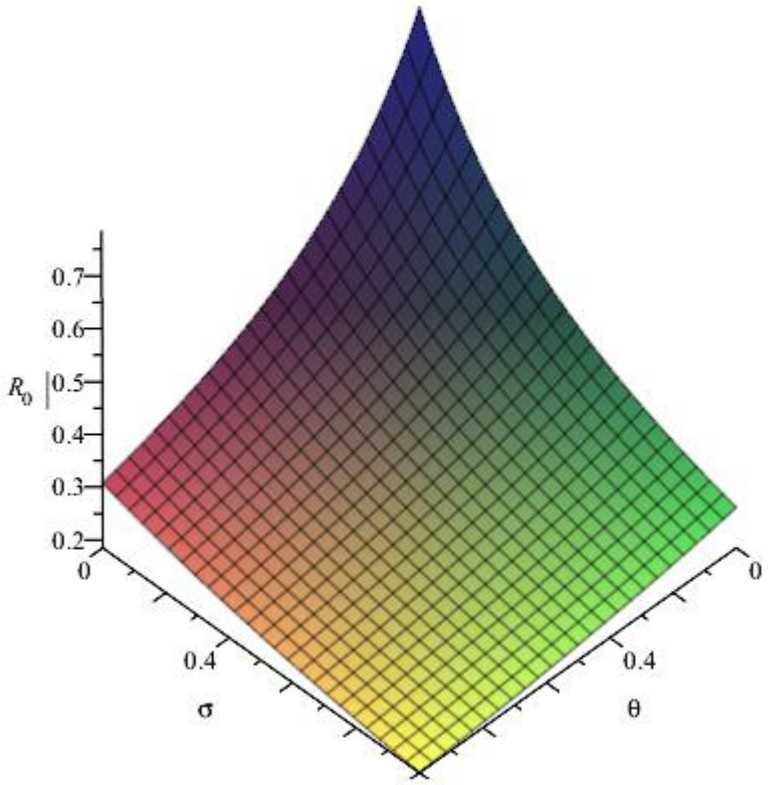

Fig 3.4: Sensitivity of the basic reproduction number to the parameters σ and θ

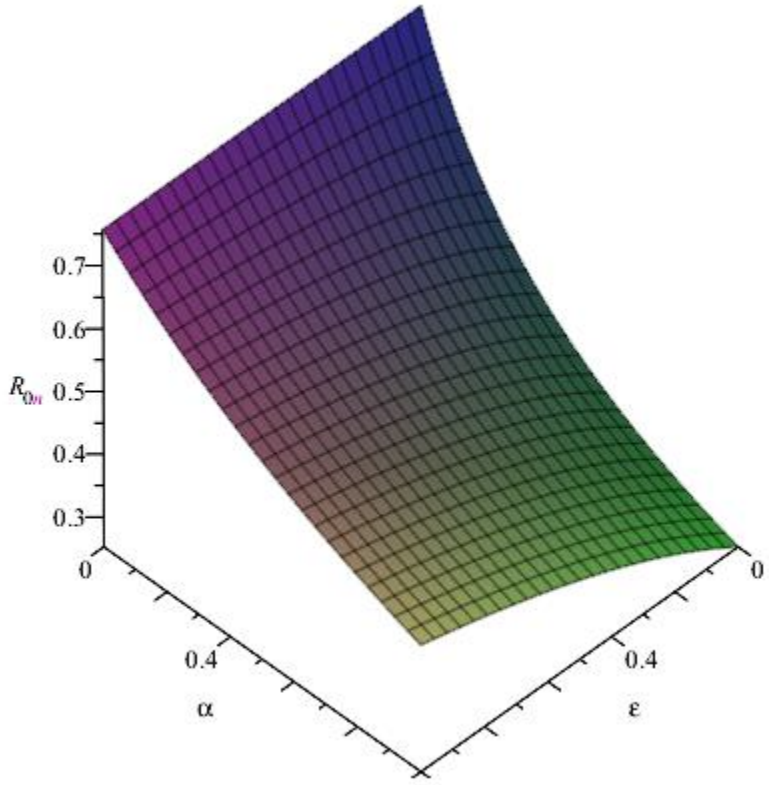

Fig 3.5: Sensitivity of the basic reproduction number to the parameters α and ε

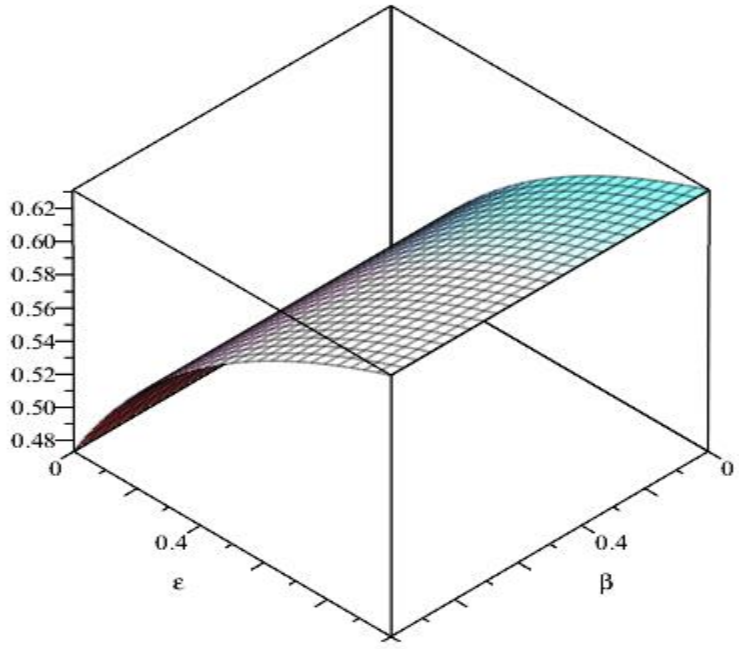

Fig 3.6: Sensitivity of the basic reproduction number to the parameters ε and β

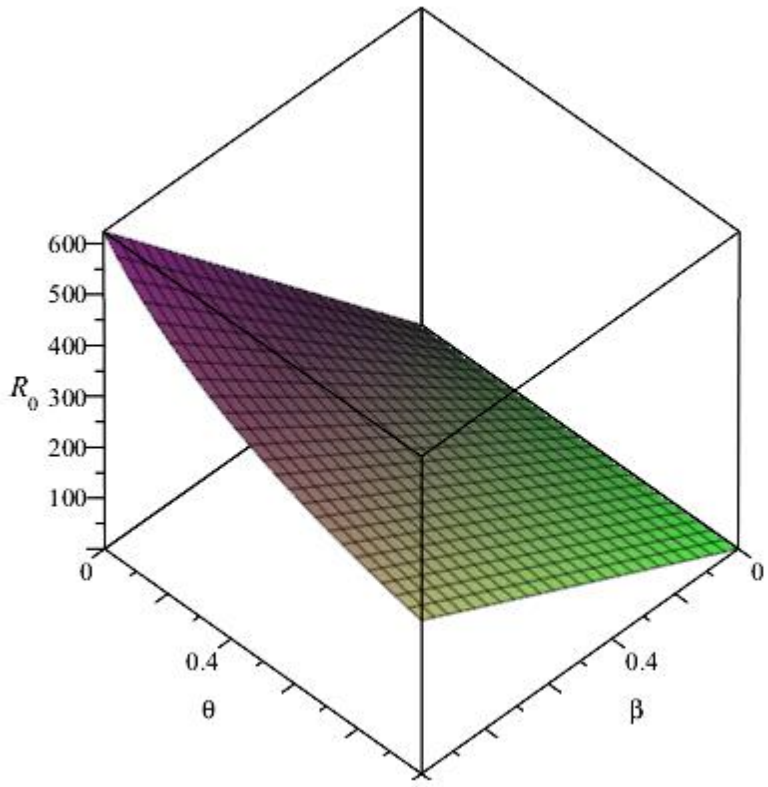

Fig 3.7: Sensitivity of the basic reproduction number to the parameters θ and β

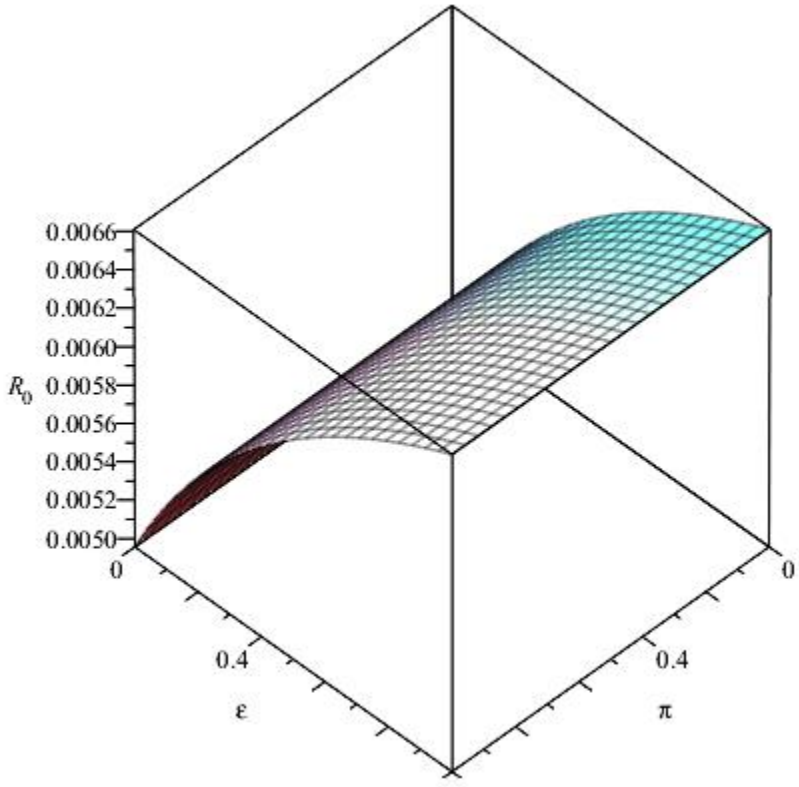

Fig 3.8: Sensitivity of the basic reproduction number to the parameters ε and π

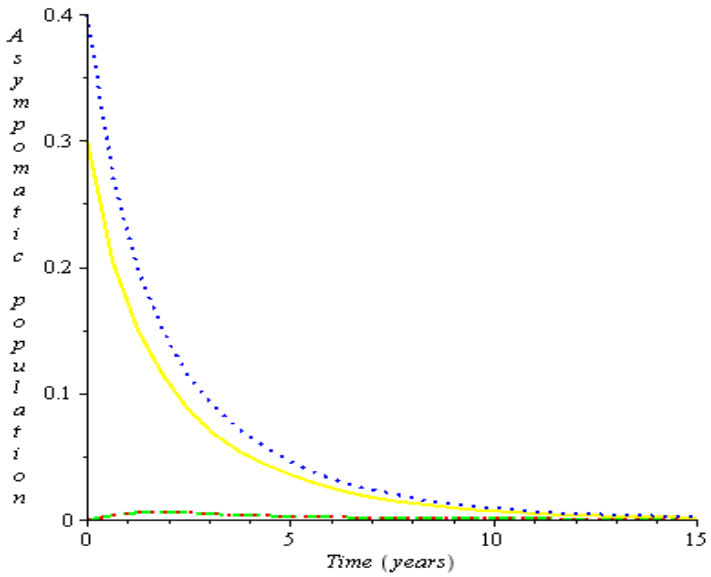

Fig 3.9: Global stability of disease free equilibrium

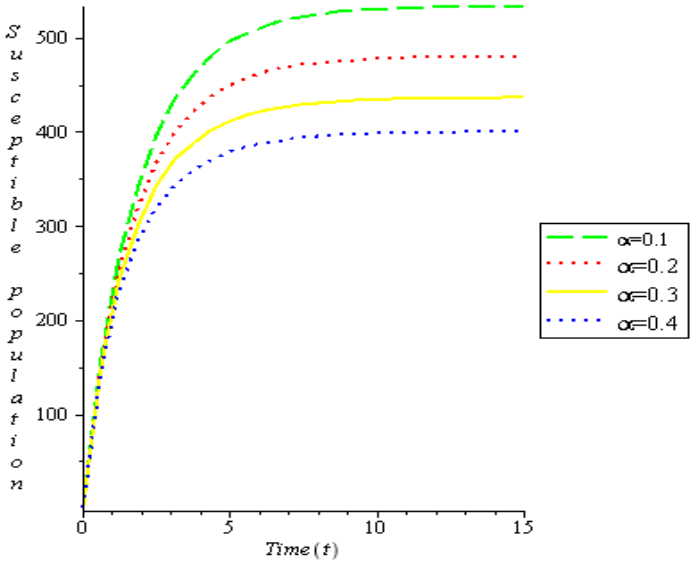

Fig 3.10: Plot of fraction of susceptible population placed on PrEP

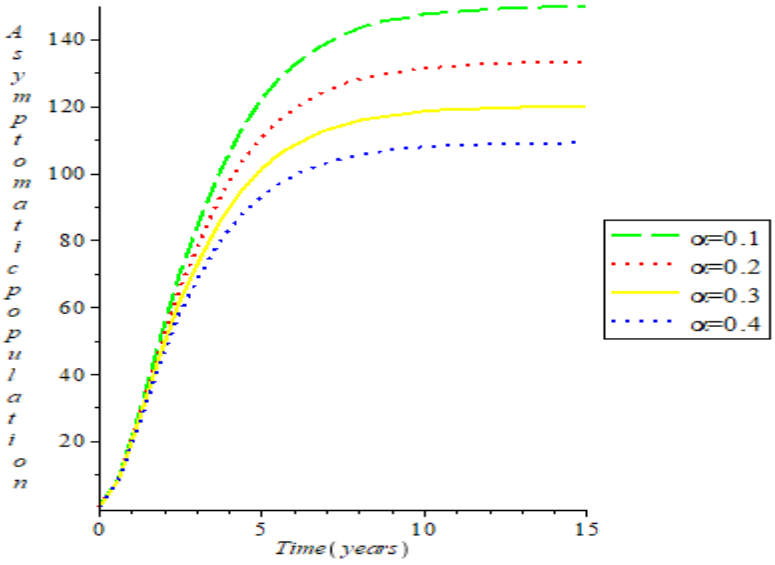

Fig 3.11: Plot of Fraction of Vaccinated susceptible placed on PrEp

Discussion

Fig 3.1 shows the graphical representation of the basic reproduction in form of a bar chart. The most sensitive parameters are recruitment rate , transmission rate and infectivity rate.

Fig 3.2 shows Both recruitment rate of susceptible π and β. An increase in these parameter values increases the value of the basic reproduction number. Similarly in fig 3.3, increases in progression rate from asymptomatic infective to AIDS class (δ), and progression rate from symptomatic class to treated and AIDS class respectively (σ), resulted in to a corresponding decrease the value of basic reproduction number.

Fig 3.4 also shows the plot of Sensitivity of the basic reproduction number to the parameters (σ) and (θ). It reveals that increase in the value of (σ) and (θ), resulted into a corresponding decrease in the basic reproduction number population. Figure 3.5 reveals the Sensitivity of the basic reproduction number to the parameters (α) and (ε). Both parameters reveals that an increase in the fraction of individuals placed on prep strategy (a) and fraction of vaccinated that become susceptible (ε), resulted into a corresponding decrease in the basic reproduction number population. An increase in the value of fraction of vaccinated that become susceptible (ε), decreases the basic reproduction number and increase in the transmission rate (β), will lead to increase in the basic reproduction number as depicted in fig 3.6.

Fig. 3.7 depicts the Sensitivity of the basic reproduction number to the parameters θ and (β). It was shown that increase in pace at which asymptomatic infective population become aware of been infected after a screening process yields a decrease in the basic reproduction and also an increase in the basic reproduction number as transmission rate also increase. Fig. 3.8 investigated the sensitivity of the basic reproduction number to the parameters (ε) and (π), and an increase in the fraction of vaccinated that become susceptible (ε), decreases the basic reproduction number and the basic reproduction number also increases as the recruitment rate of susceptible increases.

The portrait of global stability of the disease free equilibrium with various initial conditions as illustrated in fig. 3.9. It simply suggest that if the basic reproduction number $R_0 < 1$, the virus cannot sustain itself in the population and the number of new cases will decrease over time. The HIV virus can be eradicated from the population, regardless of the number of people that are initially infected and the disease will eventually die out, regardless of the initial number of infective individuals whenever $R_0 < 1$ and the system will always converge to the disease-free state.

Fig. 3.10 shows the proportion of susceptible individuals taking PrEP. However, the plot indicates that non-adherence to PrEP leads to decrease in PrEP coverage among the susceptible population while in fig 3.11, reveals that as more individuals are enlisted for PrEP uptake, there is a notable decline in the asymptomatic class as observed.

4. Conclusion

This research utilizes mathematical modelling to evaluate the effectiveness of Pre-Exposure Prophilaxis (PrEP) in preventing HIV transmission and curtaining its spread at various stages of HIV infection. The nonlinear mathematical model provides a robust framework for understanding HIV/AIDS transmission dynamics among the susceptible population, vaccinated susceptible, asymptomatic, symptomatic, treated and AIDS infective population. The outcomes of both local and global stability analysis of the disease-free equilibrium indicate a strong potential for achieving and maintaining a stable disease-free environment. The results of this study highlight the importance of targeted interventions in achieving a disease-free environment. The most sensitive parameters influencing the dynamics of the disease were revealed. However, increasing those parameters would increase the basic reproduction number. Therefore, health policymakers and stakeholders are advised to prioritize interventions that increase parameters with negative index values to effectively reduce R_0 and achieve a disease free and healthier environment.

In conclusion, we therefore recommend based on our findings, daily pre-Exposure Prophylaxis (PrEP) for susceptible populations and vaccinated individuals at substantial risk of HIV infection. By doing so, we can significantly reduce the risk of HIV transmission and prevent further spread of the disease. It is essential to emphasize that the effectiveness of PrEP relies heavily on strict adherence to the prescribed treatment regimen.

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