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ORIGINAL ARTICLE

Analysis of a virus-resistant HIV-1 model with behavior change in non-progressors

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***Abstract*—We develop a virus-resistant HIV-1 mathematical model with behavior change in HIV-1 resistant non-progressors which was analyzed for both partial and total abstinence cases. The model has both disease-free and endemic equilibrium points that are locally asymptotically stable depending on the value of the associated threshold quantities $\mathcal{R}_T$ and $\mathcal{R}_T^{'}$. In both cases, a non-linear Goh–Volterra Lyapunov function was used to prove that the endemic equilibrium point is globally asymptotically stable for special case while the method of Castillo-Chavez was used to prove the global asymptotic stability of the disease-free equilibrium point. In both the analytic and numerical results, this study shows that in the context of resistance to HIV/AIDS, total abstinence can also play an important role in protection against this notorious infectious disease.**

***Keywords*-Resistance; Behavior change; Partial & Total Abstinence; Goh–Volterra Lyapunov function.**

AMS Subject Classification: 92Bxx, 92B05.

I. Introduction

As it was reported in the 1980s, the human immunodeficiency virus (HIV), and the later stage of infection through cell depletion known as AIDS has continue to play a leading role in the series of the greatest ever infectious disease. United Nations Program on HIV/AIDS (UNAIDS) and the World Health Organization (WHO) have already provided the estimates of the number of cases since the 1980s.

More than 30 million people are currently HIV positive. According to the current trends, at least 7300 people are infected with HIV and a minimum of 5000 die from AIDS-related causes including at-least 690 children on a daily basis (UNAIDS, 2009). This means that for every five HIV positive individuals, at least four of them including adults and children die from the infection daily [10], [32]. The two main types of HIV are HIV-1 and HIV-2. The most dangerous that has spread worldwide is HIV-1 while the latter is less pathogenic and less spread since it's confined to West African countries. The test carried out on one can not

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sufficiently detect the other due to large genetic differences between them.

Immediately after HIV infection, the lymphocytes, or white blood cells, known as CD4+ T cells are the major target. Therefore, the anti-HIV antibody and cytotoxic T cell production by the immune system is consequently initiated. An HIV positive individual is not classified as having AIDS until CD4+T cell count which is approximately around $1000mm^{-3}$ depletes to $150mm^{-3}$ or thereabout. Since CD4+T plays a very important role in the body immune mechanism, deterioration and depletion result in acquired immunodeficiency syndrome called AIDS. The average number of times it takes HIV to develop to AIDS is dependent on the strength of the immune system of the victim [23].

It is therefore pertinent to study methods of HIV prevention. Different control strategies such as behavior change due to HIV awareness campaign, reduction in sexual partners, anti retroviral treatment ART etc. have collectively played important roles in combating the menace. They are still very much relevant due to unavailability of vaccine. The use of condom has also played an important role and can possibly prevent HIV transmission almost perfectly. Other intervention methods that can concurrently prevent both sexes are still very much needed. Recently, an experimental product containing a drug that can prevent rectal and vaginal transmission of HIV and other sexually transmitted diseases was detected but unfortunately did not see the light of the day due to the fact that the gel is ineffective with high HIV infection risk [24]. Other efforts such as the HIV vaccine and diaphragm technique fail to manifest to any meaningful impact [6], [21].

From biological point of view, HIV resistance is known as the genetic mutation in the DNA that delays AIDS progression or aids production of permanent immunity (i.e. no progression) to AIDS. This kind of mutation which is known as CCR5-delta 32 plays an important role in the development of the two kinds of HIV resistance known. This CCR5-delta 32 breaks and distorts the HIV's ability to deplete and destroy the immunity of the CD4+T cells. The mutation makes the CCR5 co-receptor on the outside of cells to develop at a smaller rate than usual and no longer sit outside of the cell. This co-receptor is similar to a door that allows HIV passage into the cell where within a second locks "the door" which consequently prevents HIV entrance into the CD4+T cells [14]. This genetic mutation has been reported to be inborn. There are very few paper on resistant mathematical model, some of them are [25], [13] and [15] but the resistance was modeled on influenza and SARS which is quite different from HIV/AIDS. This still remains a biological research question needed to be answered.

Research has shown that some people develop resistance to the killer HIV-1 virus [22], [28]. In fact, a report in [18] shows that though this resistance is rare but actually exists. Virus resistance can be understood in two scenarios. First, there are cases of individuals that are exposed to HIV but after a long period of times, diagnosis shows that they are uninfected. This case of exposed uninfected have been detected from among infants of infected mothers, health workers during treatment of infected individuals, commercial sex workers, individuals having unprotected sex with seropositive partners etc. The second category is HIV infected individuals with low or no progression to AIDS as expected under normal circumstances. They live with the virus for many years with an absolutely low level of HIV-1 RNA or no loss of CD4+ cells that has been identified among various individuals such as children and homosexual men and women mutation [18], [8].

In 2014, the report in [12] confirms that some people show partial or absolutely complete inborn resistance to the HIV virus . The major or main contributor to this strange development is a mutation of the gene encoding CCR5 which acts as a co-receptor for HIV. CCR5 may even be defective in some individuals which will enhance protection against disease. These individuals live a normal life since the HIV-1 virus cannot bind itself to it and its perhaps here that the key to

overcome the disease lies hidden. Estimation later shows that the proportion of individuals under this category is less than 1%. Similar occurrences make leading Oxford University researcher Sarah Rowland-Jones to believe continual exposure is a requirement for maintaining immunity after which 15 proteins were identified to be unique to those virus-free sex workers [3], [2]. A genetic mutation that blocks HIV which may hold the key to future treatment was also studied in 2016 by [9].

In 2010, [4] identified factors such as APOBEC3G, Toll-like receptors, acute-phase amyloid A protein, interleukin-22, APOBEC3G and natural killer cells as the main reason why some people do not even seroconvert let alone progressing to AIDS despite multiple HIV exposure. More interesting reasons behind this strange occurrence has been examined by the university of Minnesota in 2014 [33] and by [17] in 2013. Another interesting factor that influence the spread of HIV/AIDS is change in sexual behavior towards sex. This is caused by the infectiousness nature, high death rate and stigmatization encountered by victims of HIV/AIDS. This has subsequently affect the transmission of the disease in recent years.

Behavior change intervention will help individuals change their drug-using behaviors and sexual behavior that put them at a high risk of contracting HIV. It also creates skills and knowledge that can influence their motivation and ability to kick start behavior change. Couples, peer groups, individuals, communities or institutions can be targeted on a multiple level. This behavior change can also be motivated through skills-building, motivational or educational approach. Interventions can target different kind of behaviors such as condom usage, number of sexual partners, correct use of best prevention approach etc. Though many researchers have developed different models to examine the dynamics of the virus, HIV-1 mathematical model where infected individuals gain resistance to acquisition of HIV and resistance to deterioration of HIV incorporating behavior change in form of partial and total abstinence is still a biological question needed to be answered.

Researchers like [31], [19] have done commendable work in tackling the menace of the deadly virus, in this research, we present a new virus-resistant HIV-1 model with behavior change. This behavior change to avoid infection happens as a result of the wide spread of the agony and death caused by HIV/AIDS. This change happens either partially or totally. Those who show partial abstinence are those that only reduced their sexual partners but still involve in HIV-risk activities or live in endemic environment while those who totally abstain are those who maintain only one sexual partner and do away from all HIV-risk activities or exposed and endemic environment.

Mathematical modeling has become an effective tool in studying infectious disease by many researchers. It shall be used again here to study the dynamics of resistance in HIV-1 transmission and how it produce significant reduction rate in the community. We hope it helps policy-makers and public health workers in the epidemic control.

Several researchers like [20], [19], [1] and references therein have published commendable research output about transmission dynamics of HIV/AIDS. They have also studied control and prevention strategies of this notorious epidemic. In order to further extend, compliment and contribute to the work of the aforementioned researchers, a new comprehensive model has been designed. The model extends the work of the aforementioned researchers by, for instance,

1) Considering the influence of virus-resistance i.e. resistance to acquisition and resistance to deterioration.
2) Incorporating the change of behavior class whose rate of progression is either through partial abstinence or total abstinence.
3) Including a compartment (I_1) for slow progressors. These are the category of people with partial resistance to the virus.
4) Including a compartment (I_2) for non progressors. These are the category of people with complete resistance to the virus and do not move to AIDS compartment (A).
5) Including a compartment (I_3) for fast pro-

gressors. These are the category of people with no resistance to the virus.

All these instances have not been considered before.

The paper is organized as follows. Section 2 entails model formulation and assumptions while section 3 contains basic properties of the model. This is followed by the analysis of the sub-model (model with total abstinence) and that of the full model (model with partial abstinence) in section 4. Section 5 presents the numerical simulation and discussion of results while the last section contains the conclusion, acknowledgment and disclosure statement.

II. Model Formulation and Model Assumptions

We formulate an HIV-1 resistant and behavior change model by splitting the total human population at time t, denoted by $N(t)$, into six mutually-exclusive compartments of susceptible individuals $S(t)$, slow progressor HIV-1 infected class $I_1(t)$, non progressor HIV-1 infected class $I_2(t)$, fast progressor HIV-1 infected class $I_3(t)$, behavior change class I_4 and AIDS class A such that

$$N(t) = S(t)+I_1(t)+I_2(t)+I_3(t)+I_4(t)+A(t).$$

It is worth noting that the AIDS class consists of weak and unhealthy infected individuals that are assumed to be sexually inactive.

Sexually active individuals are recruited into the susceptible population at a constant rate B. The susceptible individuals acquire the virus through effective contact with an HIV-1 positive and infectious individuals at the rate λ given by

$$\lambda = \frac{\beta(I_3 + \sigma_1 I_1 + \sigma_2 I_2 + \sigma_3 I_4)}{N}, \qquad (1)$$

where β in (1) denotes the effective contact rate that is capable of leading to infection, $0 \leq \sigma_1 \leq 1$ denotes the modification parameter that account for the assumed reduction in the transmission of virus by the slow progressor HIV-1 infected class I_1 in comparison to the fast progressor HIV-1 infected individuals in I_3, $0 \leq \sigma_2, \sigma_3 \leq 1$ are the modification parameters accounting for the assumed reduction of infectiousness by I_2 and I_4 classes in comparison to the slow-progressor and fast progressor classes I_1 and I_3 respectively. So that

$$\sigma_3 < \sigma_2 < \sigma_1 < 1, \quad \sigma_3 \geq 0. \qquad (2)$$

The acquisition of infection by the slow progressor HIV-1 infected individuals I_1 occur at the rate $\alpha_1\lambda$, that of I_2 occur at the rate $\alpha_2\lambda$ and that of I_3 at the rate $\alpha_3\lambda$. Natural death occur constantly to anybody at the rate μ and rate of progression from I_1 to AIDS class A at the rate ρ_1. Therefore, the rate of change of the total population of the susceptible and and slow progressor classes is respectively given by

$$\begin{aligned}\dot{S}(t) =& B - (\alpha_1 + \alpha_2 + \alpha_3)\lambda S - \mu S,\\ \dot{I}_1(t) =& \alpha_1\lambda S - \rho_1 I_1 - \mu I_1,\end{aligned}$$

where $\dot{}$ represents derivative with respect to time. The non-progressor HIV-1 infected class is generated by the break-through of infection of susceptible class at the rate $\alpha_2\lambda$, total abstinence due to behavior change at the rate γ_1, partial abstinence from I_4 due to behavior change at the rate γ_2 and natural death at the rate μ so that we have

$$\dot{I}_2(t) = \alpha_2\lambda S - \gamma_1 I_2 + \gamma_2 I_4 - \mu I_2.$$

Similarly, we compose the fast progressor class by the break-through of infection of the susceptible class at the rate $\alpha_3\lambda$, AIDS acquisition at the rate ρ_2 so that the class is given by

$$\dot{I}_3(t) = \alpha_3\lambda S - \rho_2 I_3 - \mu I_3.$$

The behavior change class is formulated through the total abstinence of non progressors at the rate γ_1 and partial abstinence at the rate γ_2 given by

$$\dot{I}_4(t) = \gamma_1 I_2 - \gamma_2 I_4 - \mu I_4.$$

While incorporating the behavior change in the model, we deliberately focused on the behavior change of the non-progressors HIV-1 infected individuals even though, it is imperative that all individuals can change their behavior at any given time. This is because this class of individuals are the most dangerous class just that they won't show

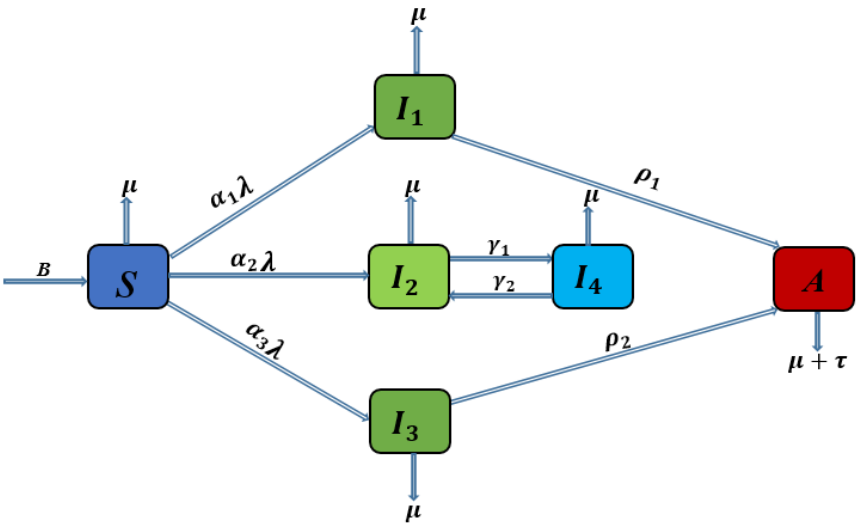


Fig. 1. Flow chart of the model.

any sign of AIDS.
And finally, the AIDS class is given by

$$\dot{A}(t) = \rho_1 I_1 + \rho_2 I_3 - (\mu + \tau)A,$$

where τ is the AIDS-induced death rate. Since progression are not the same, we have

$$\alpha_3 > \alpha_1 > \alpha_2, \ \alpha_1 + \alpha_2 + \alpha_3 = 1 \qquad (3)$$

where $0 < \alpha_1, \ \alpha_2, \ \alpha_3 < 1$. The resultant mathematical model for the transmission dynamics of HIV-1 incorporating virus resistance and behavior change through partial and total abstinence using a set of non-linear autonomous set of differential equations is given by:

$$\frac{dS}{dt} = B - (\alpha_1 + \alpha_2 + \alpha_3)\lambda S - \mu S, \qquad (4)$$
$$\frac{dI_1}{dt} = \alpha_1 \lambda S - K_1 I_1, \qquad (5)$$
$$\frac{dI_2}{dt} = \alpha_2 \lambda S + \gamma_2 I_4 - K_2 I_2, \qquad (6)$$
$$\frac{dI_3}{dt} = \alpha_3 \lambda S - K_3 I_3, \qquad (7)$$
$$\frac{dI_4}{dt} = \gamma_1 I_2 - K_4 I_4, \qquad (8)$$
$$\frac{dA}{dt} = \rho_1 I_1 + \rho_2 I_3 - K_5 A, \qquad (9)$$

where

$$K_1 = \rho_1 + \mu, K_2 = \gamma_1 + \mu, K_3 = \rho_2 + \mu,$$
$$K_4 = \gamma_2 + \mu, K_5 = \mu + \tau,$$

with initial condition

$$S(0) > 0, \ I_1(0) > 0, \ I_2(0) > 0,$$
$$I_3(0) > 0, \ I_4(0) > 0, \ A(0) > 0. \qquad (10)$$

The flow chart of this model is given in Figure 1.

III. Basic Properties of the Model

Since the model is a dynamical system, it it is therefore imperative to ensure that it is biologically meaningful through the establishment of its positivity solution and boundedness at all time $t \geq 0$.

A. Positivity and boundedness of the Model.

Lemma III.1. *The closed set*

$$\Gamma = \left\{(S, I_1, I_2, I_3, I_4, A) \in \mathbb{R}^6_+ \,|\, S + I_1 + \ldots + I_4 + A \leq \frac{B}{\mu}\right\}$$

is attracting and positively invariant with respect to the model equation (4)-(9).

Proof: From (4), we define an integrating factor as

$$\xi(t) = \exp\left\{\int_o^t [\mu + (\alpha_1 + \alpha_2 + \alpha_3)\lambda(\eta)]d\eta\right\},$$

where $\lambda(\eta) = \lambda(I_1, I_2, I_3, I_4)$. So that the solution of (4) is given by

$$S(t)\xi(t) = B\int_o^t \xi(t)dt,$$

which can be re-written as

$$S(t)\exp\left\{\int_o^t [\mu+(\alpha_1+\alpha_2+\alpha_3)\lambda(\eta)]d\eta\right\} = S(0) + B\int_o^t \left[\exp\left\{\int_o^s [\mu+(\alpha_1+\alpha_2+\alpha_3)\lambda(\eta)]d\eta\right\}\right] ds,$$

which implies

$$S(t)\exp\left\{\mu t+\int_o^t (\alpha_1+\alpha_2+\alpha_3)\lambda(\eta)d\eta\right\} = S(0) + B\int_o^t \left[\exp\left\{\mu s+\int_o^s (\alpha_1+\alpha_2+\alpha_3)\lambda(\eta)d\eta\right\}\right] ds$$

so that

$$\begin{aligned} S(t) = B\int_o^t \left[\exp\left\{\mu s+\int_o^s (\alpha_1+\alpha_2+\alpha_3)\lambda(\eta)d\eta\right\}\right] ds \\ \times \exp\left\{-\mu t-\int_o^t (\alpha_1+\alpha_2+\alpha_3)\lambda(\eta)d\eta\right\} \\ + S(0)\exp\left\{-\mu t-\int_o^t (\alpha_1+\alpha_2+\alpha_3)\lambda(\eta)d\eta\right\}, \end{aligned}$$

where $S(0)$ is an initial condition for $S(t)$ and hence it is a constant. This expression guarantees the positivity of the state variable $S(t)$ under the condition that $S(0) > 0$ which consequently ensures the positivity of $I_1(t)$, $I_2(t)$, $I_3(t)$, $I_4(t)$ and $A(t)$ provided that (10) is satisfied for all time $t \geq 0$.

Furthermore, addition of (4)-(9) gives

$$\begin{aligned} \tfrac{dN(t)}{dt} &= B - \mu N(t) - \tau A \\ &\Downarrow \\ \tfrac{dN(t)}{dt} &\leq B - \mu N(t), \end{aligned} \tag{11}$$

whose solution is

$$N(t) \leq \frac{B}{\mu} + \left[N(0) - \frac{B}{\mu}\right]\exp(-\mu t), \tag{12}$$

$$\begin{aligned} \lim_{t\to\infty} N(t) &\leq \frac{B}{\mu} + \lim_{t\to\infty}\left[N(0) - \frac{B}{\mu}\right]\exp(-\mu t) \\ &= \frac{B}{\mu}. \end{aligned}$$

This shows the boundedness of the solution above by $\frac{B}{\mu}$ in the domain defined by the provision of Lemma III.1. Therefore, the model is epidemically well-posed and mathematically meaningful since all the state variables are non-negative for all $t \geq 0$. Hence, it is sufficient to study and analyze the model in Γ [26], [27]. This completes the proof. ■

IV. Analysis of the Model

A. Analysis of the Model with Total Abstinence of Non-progressors

Here, we analyze the model for non-progressors that change their behavior through total abstinence from all means of contracting HIV-1 and from all HIV-1 endemic environments i.e. $\gamma_2 = 0, \sigma_3 = 0$ so that equation (4)-(9) becomes

$$\frac{dS}{dt} = B - (\alpha_1 + \alpha_2 + \alpha_3)\lambda_1 S - \mu S, \tag{13}$$

$$\frac{dI_1}{dt} = \alpha_1\lambda_1 S - K_1 I_1, \tag{14}$$

$$\frac{dI_2}{dt} = \alpha_2\lambda_1 S - K_2 I_2, \tag{15}$$

$$\frac{dI_3}{dt} = \alpha_3\lambda_1 S - K_3 I_3, \tag{16}$$

$$\frac{dI_4}{dt} = \gamma_1 I_2 - \mu I_4, \tag{17}$$

$$\frac{dA}{dt} = \rho_1 I_1 + \rho_2 I_3 - K_5 A, \tag{18}$$

where

$$\lambda_1 = \frac{\beta(I_3 + \sigma_1 I_1 + \sigma_2 I_2)}{N}. \tag{19}$$

All model parameters are positive.

B. Local Stability of Disease-Free equilibrium (DFE)

The disease-free equilibrium of (13)-(18) is given by

$$\begin{aligned} \psi_1^* &= (S^*, I_1^*, I_2^*, I_3^*, I_4^*, A^*) \\ &= \left(\frac{B}{\mu}, 0, 0, 0, 0, 0\right). \end{aligned} \tag{20}$$

This shows that

$$N^* = S^* = \frac{B}{\mu} \text{ and } \frac{S^*}{N^*} = 1$$

at disease-free equilibrium point ψ_1^*. By employing the next generation method [7], [34], $\mathcal{F}_1$ (the new infection terms) and $\mathcal{V}_1$ (transfer terms) are expressed as

$$\mathcal{F}_1 = \begin{bmatrix} \alpha_1\sigma_1\beta & \beta\sigma_2\alpha_1 & \beta\alpha_1 & 0 & 0 \\ \alpha_2\sigma_1\beta & \beta\sigma_2\alpha_2 & \beta\alpha_2 & 0 & 0 \\ \alpha_3\sigma_1\beta & \beta\sigma_2\alpha_3 & \beta\alpha_3 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

$$\mathcal{V}_1 = \begin{bmatrix} K_1 & 0 & 0 & 0 & 0 \\ 0 & K_2 & 0 & 0 & 0 \\ 0 & 0 & K_3 & 0 & 0 \\ 0 & -\gamma_1 & 0 & \mu & 0 \\ -\rho_1 & 0 & -\rho_2 & 0 & K_5 \end{bmatrix}.$$

Taking ρ as the spectral radius (magnitude of the dominate eigenvalue) of the next generation matrix $\mathcal{F}_1\mathcal{V}_1^{-1}$, the reproduction number is given by

$$\mathcal{R}_T^{'} = \frac{\beta(\alpha_1 K_2 K_3\sigma_1 + \alpha_2 K_1 K_3\sigma_2 + \alpha_3 K_1 K_2)}{K_1 K_2 K_3}. \tag{21}$$

The quantity $\mathcal{R}_T^{'}$ represents the measure of average number of new virus infection of HIV-1 developed by a single HIV-1 infected individual in a population where there are people who practice total abstinence and are completely susceptible. Hence, we present the following Lemma.

Lemma IV.1. *The DFE of the reduced model* (13)-(18) *with total abstinence is locally asymptotically stable (LAS) if* $\mathcal{R}_T^{'} < 1$*, and unstable if* $\mathcal{R}_T^{'} > 1$.

The proof is standard and can be established using theorem 2 of [34].

C. Existence of Endemic Equilibrium

The reduced model with total abstinence has a unique positive endemic equilibrium point (EEP). This is the point where at least one of the virus infected compartments is non-zero. Let

$$\psi_1^{**} = (S^{**}, I_1^{**}, I_2^{**}, I_3^{**}, I_4^{**}, A^{**}) \tag{22}$$

be the endemic equilibrium point. We further define the force of infection as

$$\lambda_1^{**} = \frac{\beta(I_3^{**} + \sigma_1 I_1^{**} + \sigma_2 I_2^{**})}{N^{**}}. \tag{23}$$

Solving equation (13)-(18) in terms of the force of infection λ_1^{**} at steady-state gives:

$$\begin{aligned} S^{**} &= \frac{B}{\mu + (\alpha_1+\alpha_2+\alpha_3)\lambda_1^{**}}, \\ I_1^{**} &= \frac{\alpha_1 B\lambda_1^{**}}{K_1[\mu + (\alpha_1+\alpha_2+\alpha_3)\lambda_1^{**}]}, \\ I_3^{**} &= \frac{B\lambda_1^{**}}{K_3[\mu + (\alpha_1+\alpha_2+\alpha_3)\lambda_1^{**}]}, \\ I_4^{**} &= \frac{\gamma_1 B\alpha_2\lambda_1^{**}}{K_2\mu[\mu + (\alpha_1+\alpha_2+\alpha_3)\lambda_1^{**}]}, \\ A^{**} &= \frac{B\lambda_1^{**}(\rho_1\alpha_1 K_3 + K_1\rho_2)}{K_1 K_3 K_5[\mu + (\alpha_1+\alpha_2+\alpha_3)\lambda_1^{**}]}, \\ I_2^{**} &= \frac{\alpha_2 B\lambda_1^{**}}{K_2[\mu + (\alpha_1+\alpha_2+\alpha_3)\lambda_1^{**}]}, \\ N^{**} &= \frac{BK_1K_3K_5f_1 - \tau B\lambda_1^{**}(\rho_1\alpha_1K_3 + K_1\rho_2)}{\mu K_1K_3K_5f_1}, \end{aligned} \tag{24}$$

where $f_1 = \mu + (\alpha_1+\alpha_2+\alpha_3)\lambda_1^{**}$. Substituting all the equations in (24) into (23), it can be shown that the non-zero equilibria of the model satisfy the following linear equation in terms of λ_1^{**}:

$$a_o\lambda_1^{**} + a_1 = 0, \tag{25}$$

where

$$\begin{aligned} a_o &= \alpha_1\mu K_2K_3(\mu+\tau+\rho_1) \\ &\quad + K_1K_2[\mu\alpha_3(\rho_2+\mu+\tau) + K_3K_5\alpha_2], \end{aligned} \tag{26}$$

$$a_1 = \mu K_1K_2K_3K_5(1-\mathcal{R}_T^{'}). \tag{27}$$

Clearly, $a_o > 0$, $a_1 \geq 0$ if and only if $\mathcal{R}_T^{'} \leq 1$ so that $\lambda_1^{**} = -\frac{a_1}{a_o} \leq 0$. This shows that no existence of positive endemic equilibrium whenever $\mathcal{R}_T^{'} \leq 1$. Hence, the endemic equilibrium point ψ_1^{**} exists and unique whenever $\mathcal{R}_T^{'} > 1$. We claim the following result.

Lemma IV.2. *The endemic equilibrium point (EEP) of the reduced model* (13)-(18) *with total abstinence is locally asymptotically stable (LAS) if* $\mathcal{R}_T^{'} > 1$.

D. Global Stability of DFE

To establish the global stability of DFE points, we adopt the approach of [5] to re express (13)-(18) in the following vector form

$$\dot{X} = L(X, Y), \tag{28}$$

$$\dot{Y} = M(X,Y), M(X,0) = 0, \qquad (29)$$

where the vector $X = (S)$ denotes the HIV-1 uninfected compartment of the system and $Y = (I_1, I_2, I_3, I_4, A) \in \mathbb{R}^5_+$ represents the HIV-1 infected compartments. Using the DFE point to establish the stability analysis, the following two conditions must be satisfied:

$\mathbf{N}_1$: For $\dot{X}(t) = L(X^o, 0)$, X^o is globally asymptotically stable.

$\mathbf{N}_2 : M(X,Y) = JY - \hat{M}(X,Y)$, $\hat{M}(X,Y) \geq 0$ for $X, Y \in \Omega_m$ where $J = \frac{\partial M}{\partial Y}(X^o, 0)$.

For this analysis, the expressions for $J_1, Y_1, \hat{M}_1$ and M_1 are for the reduced model with the same definition as above while expressions for $J, Y, \hat{M}$ and M are for the full model with the same definition. From our model equation, we obtain the Jacobian matrix of only the infected compartment at DFE as follows:

$$J_1 = \begin{bmatrix} \frac{\alpha_1\sigma_1\beta S^*}{N^*} - K_1 & \frac{\beta\sigma_2\alpha_1 S^*}{N^*} & \frac{\beta\alpha_1 S^*}{N^*} & 0 & 0 \\ \frac{\alpha_2\sigma_1\beta S^*}{N^*} & \frac{\alpha_2\sigma_2\beta S^*}{N^*} - K_2 & \frac{\beta\alpha_2 S^*}{N^*} & 0 & 0 \\ \frac{\alpha_3\sigma_1\beta S^*}{N^*} & \frac{\alpha_3\sigma_2\beta S^*}{N^*} & \frac{\beta\alpha_3 S^*}{N^*} - K_3 & 0 & 0 \\ 0 & \gamma_1 & 0 & -\mu & 0 \\ \rho_1 & 0 & \rho_2 & 0 & -K_5 \end{bmatrix}$$

$$J_1 Y_1 = J_1 \begin{bmatrix} I_1 \\ I_2 \\ I_3 \\ I_4 \\ A \end{bmatrix} = \begin{bmatrix} \frac{\beta\alpha_1(\sigma_1 I_1 + \sigma_2 I_2 + I_3)S^*}{N^*} - K_1 I_1 \\ \frac{\beta\alpha_2(\sigma_1 I_1 + \sigma_2 I_2 + I_3)S^*}{N^*} - K_2 I_2 \\ \frac{\beta\alpha_3(\sigma_1 I_1 + \sigma_2 I_2 + I_3)S^*}{N^*} - K_3 I_3 \\ \gamma_1 I_2 - \mu I_4 \\ \rho_1 I_1 + \rho_2 I_3 - K_5 A \end{bmatrix}$$

$\hat{M}_1(X,Y) = J_1 Y_1 - M_1(X,Y) \geq 0$ where

$$M_1(X,Y) = \begin{bmatrix} \frac{\beta\alpha_1(\sigma_1 I_1 + \sigma_2 I_2 + I_3)S}{N} - K_1 I_1 \\ \frac{\beta\alpha_2(\sigma_1 I_1 + \sigma_2 I_2 + I_3)S}{N} - K_2 I_2 \\ \frac{\beta\alpha_3(\sigma_1 I_1 + \sigma_2 I_2 + I_3)S}{N} - K_3 I_3 \\ \gamma_1 I_2 - \mu I_4 \\ \rho_1 I_1 + \rho_2 I_3 - K_5 A \end{bmatrix}$$

and

$$\hat{M}_1(X,Y) = \begin{bmatrix} \beta\alpha_1(\sigma_1 I_1 + \sigma_2 I_2 + I_3)\left(1 - \frac{S}{N}\right) \\ \beta\alpha_2(\sigma_1 I_1 + \sigma_2 I_2 + I_3)\left(1 - \frac{S}{N}\right) \\ \beta\alpha_3(\sigma_1 I_1 + \sigma_2 I_2 + I_3)\left(1 - \frac{S}{N}\right) \\ 0 \\ 0 \end{bmatrix},$$

Since $S \leq N$, this shows that $\hat{M}_1(X,Y) \geq 0$. It can be seen that $\lim_{t\to\infty} X(t) = X^o$ and J is an M-matrix, thus X^o is globally asymptotically stable, hence, $\mathbf{N}_1$ is satisfied. Also, $\hat{M}_1(X,Y) \geq 0$ for $(X,Y) \in \Omega_m$. Hence, $\mathbf{N}_2$ is satisfied and E^o is globally asymptotically stable whenever $\mathcal{R}_T' < 1$.

E. Global Stability of Endemic Equilibrium Point

Following the provision of Lemma IV.2, we establish the following theorem.

Theorem IV.3. *The endemic equilibrium point of the reduced model* (13)-(18) *is globally asymptotically stable (GAS) whenever* $\mathcal{R}_T' > 1$.

Proof: Using the idea of [1], we construct the Lyapunov function:

$$B = \sum_{k=1}^{6} A_k B_k, \quad A_k > 0, \qquad (30)$$

where A_k is a constant and B_k is given by

$$B_k = \int_{f_k^{**}}^{f} \left(1 - \frac{f_k^{**}}{x}\right) dx, \qquad (31)$$

for

$$f_k^{**} \in W = \{S, I_1, I_2, I_3, I_4, A\},$$

where $k = 1, 2, 3, 4, 5, 6$. This vividly shows that B_k is positive definite, continuous and differentiable in Γ. Hence, $B_k \in C'[\Gamma, \mathbb{R}^+]$. Differentiating B partially with respect to each f_k we have

$$\frac{\partial B}{\partial f_k} = A_k \left(1 - \frac{f_k^{**}}{f_k}\right), \qquad (32)$$

so that

$$\frac{\partial B}{\partial f_k} = 0 \implies A_k \left(1 - \frac{f_k^{**}}{f_k}\right) = 0.$$

Differentiating (32) again partially with respect to each f_k gives

$$\frac{\partial^2 B}{\partial f_k^2} = \frac{A_k f_k^{**}}{f_k^2},\ k = 1, ..., 6. \tag{33}$$

From (32), if $f_k = f_k^{**}$, then $S = S^{**}, I_1 = I_1^{**}, I_2 = I_2^{**}, I_3 = I_3^{**}, I_4 = I_4^{**}, A = A^{**}$. This clearly shows that the endemic equilibrium point is the only stationary point of B. Since (30) is always positive, it means that the endemic equilibrium is a global minimum point of the function B for all $f_k \in \Gamma \subseteq \mathbb{R}_+^6$. Next is to establish that the function B is a Lyapunov function which can be done by proving that B is negative definite. The time derivative of B is given by

$$\frac{dB}{dt} = \sum_{k=1}^{6} A_k \left(1 - \frac{f_k^{**}}{f_k}\right) \dot{f}_k, \tag{34}$$

which is negative definite for all time $t > 0$. It is worth noting here that for all $f_k^{**} \in \Gamma$, $\dot{f}_k \leq \dot{N}$ which makes equation (34) to be

$$\frac{dB}{dt} \leq \sum_{k=1}^{6} A_k \left(1 - \frac{f_k^{**}}{f_k}\right) \dot{N}. \tag{35}$$

From equation (12), we obtain the derivative

$$\frac{dN}{dt} = \mu \left(\frac{B}{\mu} - N(0)\right) \exp(-\mu t). \tag{36}$$

Substituting (36) in (35), we have

$$\frac{dB}{dt} \leq \sum_{k=1}^{6} A_k \left(1 - \frac{f_k^{**}}{f_k}\right) \mu \left(\frac{B}{\mu} - N(0)\right) \exp(-\mu t). \tag{37}$$

When $t \to \infty$, $\frac{dB}{dt} \leq 0$ which means that the total initial population $N(0)$ is within the basin Γ i.e. $N(0) \leq \frac{B}{\mu}$. Also when the initial population is outside the basin of attraction i.e. $N(0) \geq \frac{B}{\mu}$ as $t \to \infty$, then $\frac{dB}{dt} \leq 0$ and hence, the right-hand side of (37) is negative definite. This proves that irrespective of the size of the initial population $N(0)$, the left hand side is always less or equal to zero as $t > 0$. This consequently clarifies that the constructed function B is a Lyapunov type and can be used to establish the global stability of the system. Moreover, $\frac{dB}{dt} = 0$ if and only if

$$S = S^{**}, I_1 = I_1^{**}, I_1 = I_1^{**}, I_2 = I_2^{**},$$
$$I_3 = I_3^{**}, I_4 = I_4^{**}, A = A^{**},$$

and the largest positive invariant subset of Γ that satisfies $\frac{dB}{dt} = 0$ is the singleton ψ_1^{**}. Hence, ψ_1^{**} is a unique endemic equilibrium point of the system (13)-(18) which is GAS in Γ. ■

F. Analysis of the Full Model

G. Local Stability of DFE

In this section, we shall analyze the full model just as we did for the sub-model in the previous section. It is worth noting that the full model has the same DFE as the sub-model given by equation (20) which exists in the same region Γ. We employ the same next generation matrix to establish the reproduction number as follows:

$$\mathcal{F} = \begin{bmatrix} \alpha_1\sigma_1\beta & \beta\sigma_2\alpha_1 & \beta\alpha_1 & \beta\alpha_1\sigma_3 & 0 \\ \alpha_2\sigma_1\beta & \beta\sigma_2\alpha_2 & \beta\alpha_2 & \beta\alpha_2\sigma_3 & 0 \\ \alpha_3\sigma_1\beta & \beta\sigma_2\alpha_3 & \beta\alpha_3 & \beta\alpha_3\sigma_3 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

$$\mathcal{V} = \begin{bmatrix} K_1 & 0 & 0 & 0 & 0 \\ 0 & K_2 & 0 & -\gamma_2 & 0 \\ 0 & 0 & K_3 & 0 & 0 \\ 0 & -\gamma_1 & 0 & K_4 & 0 \\ -\rho_1 & 0 & -\rho_2 & 0 & K_5 \end{bmatrix}.$$

Taking ρ as the spectral radius (magnitude of the dominate eigenvalue) of the next generation matrix $\mathcal{F}\mathcal{V}^{-1}$, the reproduction number is given by

$$\mathcal{R}_T = \left[\frac{P + Q}{K_1 K_3 (K_2 K_4 - \gamma_1\gamma_2)}\right], \tag{38}$$

where

$$P = (K_2 K_4 - \gamma_1\gamma_2)(\alpha_1 K_3 \sigma_1 + \alpha_3 K_1),$$
$$Q = \alpha_2 K_1 K_3 (\gamma_1\sigma_3 + K_4\sigma_2).$$

Lemma IV.4. *The disease-free equilibrium point (DFE) of the full model* (4)-(9) *with partial abstinence is locally asymptotically stable (LAS) if* $\mathcal{R}_T < 1$ *and unstable otherwise.*

H. Existence of Endemic Equilibrium

The full model with partial abstinence has a unique positive endemic equilibrium point (EEP). This is the point where at least one of the virus infected compartments is non-zero. Let

$$\psi^{**} = (S^{**}, I_1^{**}, I_2^{**}, I_3^{**}, I_4^{**}, A^{**}) \tag{39}$$

be the endemic equilibrium point. We further define the force of infection as

$$\lambda^{**} = \frac{\beta(I_3^{**} + \sigma_1 I_1^{**} + \sigma_2 I_2^{**} + \sigma_3 I_4^{**})}{N^{**}}. \tag{40}$$

Solving equation (4)-(9) in terms of the force of infection λ^{**} at steady-state we obtain:

$$\begin{aligned}
S^{**} &= \frac{B}{\mu + (\alpha_1+\alpha_2+\alpha_3)\lambda^{**}},\\
I_1^{**} &= \frac{\alpha_1 B\lambda^{**}}{K_1[\mu + (\alpha_1+\alpha_2+\alpha_3)\lambda^{**}]},\\
I_3^{**} &= \frac{B\lambda^{**}}{K_3[\mu + (\alpha_1+\alpha_2+\alpha_3)\lambda^{**}]},\\
A^{**} &= \frac{B\lambda^{**}(\rho_1\alpha_1 K_3 + K_1\rho_2)}{K_1K_3K_5[\mu + (\alpha_1+\alpha_2+\alpha_3)\lambda^{**}]},\\
I_2^{**} &= \frac{\alpha_2 B\lambda^{**}K_4}{f_1(K_2K_4 - \gamma_1\gamma_2)},\\
N^{**} &= \frac{BK_1K_3K_5f_1 - \tau B\lambda^{**}(\rho_1\alpha_1K_3 + K_1\rho_2)}{\mu K_1K_3K_5f_1},\\
I_4^{**} &= \frac{\gamma_1 B\alpha_2\lambda^{**}K_4}{K_4[\mu+(\alpha_1+\alpha_2+\alpha_3)\lambda^{**}](K_2K_4-\gamma_1\gamma_2)},
\end{aligned} \tag{41}$$

where $f_1 = \mu + (\alpha_1+\alpha_2+\alpha_3)\lambda^{**}$. Substituting all the equations in (41) into (40), it can be shown that the non-zero equilibria of the model satisfy the following linear equation in terms of λ^{**}:

$$a_2\lambda^{**} + a_3 = 0, \tag{42}$$

where

$$\begin{aligned}
a_2 &= \alpha_3\mu K_1(\rho_2+\mu+\tau) + \alpha_1K_3\mu(\mu+\rho_1+\tau)\\
&\quad + K_1K_3K_5\alpha_2 > 0 \qquad (43)\\
a_3 &= \mu K_1K_3K_5(1-\mathcal{R}_T). \qquad (44)
\end{aligned}$$

Clearly, $a_2 > 0$, $a_3 \geq 0$ if and only if $\mathcal{R}_T \leq 1$ so that $\lambda^{**} = -\frac{a_3}{a_2} \leq 0$ which shows no existence of positive endemic equilibrium whenever $\mathcal{R}_T \leq 1$. Hence, the endemic equilibrium point ψ^{**} exists and unique whenever $\mathcal{R}_T > 1$. We claim the following result.

Lemma IV.5. *The endemic equilibrium point (EEP) of the full model* (4)-(9) *with partial abstinence is locally asymptotically stable (LAS) if* $\mathcal{R}_T > 1$.

I. Global Stability of DFE of the full model

We will establish the proof using the same approach as in Section IV.C as follows:

$$\begin{aligned}
\hat{M}(X,Y) &= JY - M(X,Y)\\
&= \begin{bmatrix}
\frac{\beta\alpha_1(\sigma_1I_1+\sigma_2I_2+I_3+\sigma_3I_4)S^*}{N^*} - K_1I_1\\
\frac{\beta\alpha_2(\sigma_1I_1+\sigma_2I_2+I_3+\sigma_3I_4)S^*}{N^*} - K_2I_2+\gamma_2I_4\\
\frac{\beta(\sigma_1I_1+\sigma_2I_2+I_3+\sigma_3I_4)S^*}{N^*} - K_3I_3\\
\gamma_1I_2 - K_4I_4\\
\rho_1I_1 + \rho_2I_3 - K_5A
\end{bmatrix}\\
&\quad - \begin{bmatrix}
\frac{\beta\alpha_1(\sigma_1I_1+\sigma_2I_2+I_3+\sigma_3I_4)S^*}{N^*} - K_1I_1\\
\frac{\beta\alpha_2(\sigma_1I_1+\sigma_2I_2+I_3+\sigma_3I_4)S^*}{N^*} - K_2I_2+\gamma_2I_4\\
\frac{\beta(\sigma_1I_1+\sigma_2I_2+I_3+\sigma_3I_4)S^*}{N^*} - K_3I_3\\
\gamma_1I_2 - K_4I_4\\
\rho_1I_1 + \rho_2I_3 - K_5A
\end{bmatrix}\\
&= \begin{bmatrix}
\beta\alpha_1(\sigma_1I_1+\sigma_2I_2+I_3+\sigma_3I_4)\left(1-\frac{S}{N}\right)\\
\beta\alpha_2(\sigma_1I_1+\sigma_2I_2+I_3+\sigma_3I_4)\left(1-\frac{S}{N}\right)\\
\beta\alpha_3(\sigma_1I_1+\sigma_2I_2+I_3+\sigma_3I_4)\left(1-\frac{S}{N}\right)\\
0\\
0
\end{bmatrix} \geq 0,
\end{aligned}$$

where $\frac{S^*}{N^*} \leq 1$ at DFE and since $S \leq N$, this shows that $\hat{M}(X,Y) \geq 0$. It can be seen that $\lim_{t\to\infty} X(t) = X^o$ and J is an M-matrix, thus X^o is globally asymptotically stable, hence, $\mathbf{N}_1$ is satisfied. Also, $\hat{M}(X,Y) \geq 0$ for $(X,Y) \in \Omega_m$. Hence, $\mathbf{N}_2$ is satisfied and E^o is globally asymptotically stable whenever $\mathcal{R}_T < 1$.

J. Global Stability of The Endemic Equilibrium

We consider the special case where the virus-induced death rate τ is negligible. This is very much realistic since HIV-1 positive individuals under treatment can live many years hail and healthy without dying of the virus. Substituting $\tau = 0$ into (11), as $t \to \infty$ gives $N \to \frac{B}{\mu}$. Putting this in equation (1), we have

$$\lambda = \beta_1(I_3 + \sigma_1 I_1 + \sigma_2 I_2 + \sigma_3 I_4) \tag{45}$$

where $\beta_1 = \dfrac{\beta\mu}{B}$.

Theorem IV.6. *The endemic equilibrium point of the full model* (4)-(9) *is globally asymptotically stable (GAS) whenever* $\mathcal{R}_T > 1$.

Proof: Since Lemma IV.5 has already been established, we construct the following non-linear Lyapunov function for the system (4)-(9) as follows:

$$\begin{aligned} L = {} & \frac{\beta_1}{f_2\mu}\int_{S^*}^{S}\left(1-\frac{S^*}{x}\right)dx + \frac{1}{\alpha_1\alpha_2}\int_{I_1^*}^{I_1}\left(1-\frac{I_1^*}{x}\right)dx \\ & + \frac{\gamma_2}{\alpha_2}\int_{I_2^*}^{I_2}\left(1-\frac{I_2^*}{x}\right)dx + \frac{\alpha_1}{\beta_1\alpha_3}\int_{I_3^*}^{I_3}\left(1-\frac{I_3^*}{x}\right)dx \\ & + \frac{\beta_1^2}{K_2K_3\gamma_1}\int_{I_4^*}^{I_4}\left(1-\frac{I_4^*}{x}\right)dx + \frac{1}{\rho_1\rho_2}\int_{A^*}^{A}\left(1-\frac{A^*}{x}\right)dx. \end{aligned}$$

The derivative of L along the solution of the system (4)-(9) is given by

$$\begin{aligned} \dot{L} = {} & \frac{\beta_1}{f_2\mu}\left(1-\frac{S^*}{S}\right)\dot{S} + \frac{1}{\alpha_1\alpha_2}\left(1-\frac{I_1^*}{I_1}\right)\dot{I}_1 \\ & + \frac{\gamma_2}{\alpha_2}\left(1-\frac{I_2^*}{I_2}\right)\dot{I}_2 + \frac{\alpha_1}{\beta_1\alpha_3}\left(1-\frac{I_3^*}{I_3}\right)\dot{I}_3 \\ & + \frac{\beta_1^2}{K_2K_3\gamma_1}\left(1-\frac{I_4^*}{I_4}\right)\dot{I}_4 + \frac{1}{\rho_1\rho_2}\left(1-\frac{A^*}{A}\right)\dot{A}. \end{aligned}$$

Using (4)-(9), we have

$$\begin{aligned} \dot{L} = {} & \frac{\beta_1}{f_2\mu}\left[B-(f_2\lambda+\mu)S - \frac{S^{**}}{S}\{B-(f_2\lambda+\mu)S\}\right] \\ & + \frac{1}{\alpha_1\alpha_2}\left[\alpha_1\lambda S - K_1I_1 - \frac{I_1^{**}}{I_1}\{\alpha_1\lambda S - K_1I_1\}\right] + \\ & \frac{\gamma_2}{\alpha_2}\left[\alpha_2\lambda S + \gamma_2 I_4 - K_2I_2 - \frac{I_2^{**}}{I_2}\{\alpha_2\lambda S + \gamma_2 I_4 - K_2I_2\}\right] \\ & + \frac{\alpha_1}{\beta_1\alpha_3}\left[\alpha_3\lambda S - K_3I_3 - \frac{I_3^{**}}{I_3}\{\alpha_3\lambda S - K_3I_3\}\right] \\ & + \frac{\beta_1^2}{K_2K_3\gamma_1}\left[\gamma_1I_2 - K_4I_4 - \frac{I_4^{**}}{I_4}\{\gamma_1I_2 - K_4I_4\}\right] \\ & + \frac{1}{\rho_1\rho_2}\left[\rho_1I_1 + \rho_2I_3 - K_5A \right. \\ & \left. - \frac{A^{**}}{A}\{\rho_1I_1 + \rho_2I_3 - K_5A\}\right], \end{aligned} \tag{46}$$

where $f_2 = \alpha_1 + \alpha_2 + \alpha_3$. At endemic equilibrium point of (4)-(9), we have the following expressions.

$$\begin{aligned} B &= \mu S^{**} + f_2(I_3^{**} + \sigma_1 I_1^{**} + \sigma_2 I_2^{**} + \sigma_3 I_4^{**})S^{**}, \\ K_1 &= \frac{\alpha_1\beta_1(I_3^{**} + \sigma_1 I_1^{**} + \sigma_2 I_2^{**} + \sigma_3 I_4^{**})S^{**}}{I_1^{**}}, \\ K_2 &= \frac{\gamma_2 I_4^{**} + \alpha_2\beta_1(I_3^{**} + \sigma_1 I_1^{**} + \sigma_2 I_2^{**} + \sigma_3 I_4^{**})S^{**}}{I_2^{**}}, \\ K_3 &= \frac{\alpha_3\beta_1(I_3^{**} + \sigma_1 I_1^{**} + \sigma_2 I_2^{**} + \sigma_3 I_4^{**})S^{**}}{I_3^{**}}, \\ K_4 &= \frac{\gamma_1 I_2^{**}}{I_4^{**}}, \\ K_5 &= \frac{\rho_1 I_1^{**} + \rho_2 I_3^{**}}{A^{**}} \end{aligned} \tag{47}$$

Substituting expressions in (47) into (46), after some simplifications and factorization, we have

$$\begin{aligned} \dot{L} = {} & \frac{\beta_1 S^{**}}{f_2}\left(2 - \frac{S}{S^{**}} - \frac{S^{**}}{S}\right) \\ & + \frac{\beta_1^2}{\mu}(I_3^{**} + \sigma_1 I_1^{**} + \sigma_2 I_2^{**} + \sigma_3 I_4^{**})S^{**}\left(2 - \frac{S}{S^{**}} - \frac{S^{**}}{S}\right) \\ & + \frac{\beta_1}{\alpha_2}\left[(I_3^{**} + \sigma_1 I_1^{**} + \sigma_2 I_2^{**} + \sigma_3 I_4^{**})S^{**}\left(2 - \frac{I_1}{I_1^{**}} - \frac{I_1^{**}}{I_1}\right)\right. \\ & \left. + (I_3 + \sigma_1 I_1 + \sigma_2 I_2 + \sigma_3 I_4)S\left(2 - \frac{I_1}{I_1^{**}} - \frac{I_1^{**}}{I_1}\right)\right] \\ & + \left(\frac{1}{\rho_2} + \frac{1}{\rho_1}\right)\left(2 - \frac{A}{A^{**}} - \frac{A^{**}}{A}\right) \end{aligned}$$

$$+\gamma_2\left[\beta_1(I_3+\sigma_1 I_1+\sigma_2 I_2+\sigma_3 I_4)S\left(2-\frac{I_2}{I_2^{**}}-\frac{I_2^{**}}{I_2}\right)\right.$$
$$\left.+\beta_1(I_3^{**}+\sigma_1 I_1^{**}+\sigma_2 I_2^{**}+\sigma_3 I_4^{**})S^{**}\left(2-\frac{I_2}{I_2^{**}}-\frac{I_2^{**}}{I_2}\right)\right]$$
$$+\left(\frac{\gamma_2^2}{\alpha_2}+\frac{\alpha_1\beta_1^2}{K_2K_3}\right)\frac{I_2^2I_3^{**}}{I_4^{**}}\left(3-\frac{I_2^{**}}{I_2}-\frac{I_2I_3^{**}}{I_2^{**}I_3}-\frac{I_2^{**}I_3I_4}{I_2I_3^{**}I_4^{**}}\right).$$

Consequently, since the arithmetic mean exceeds the geometric mean, then we have

$$2-\frac{S}{S^{**}}-\frac{S^{**}}{S}\leq 0,$$
$$2-\frac{A}{A^{**}}-\frac{A^{**}}{A}\leq 0,$$
$$2-\frac{I_1}{I_1^{**}}-\frac{I_1^{**}}{I_1}\leq 0,$$
$$2-\frac{I_2}{I_2^{**}}-\frac{I_2^{**}}{I_2}\leq 0,$$
$$3-\frac{I_2^{**}}{I_2}-\frac{I_2I_3^{**}}{I_2^{**}I_3}-\frac{I_2^{**}I_3I_4}{I_2I_3^{**}I_4^{**}}\leq 0.$$

Since $S\geq 0, I_1\geq 0, I_2\geq 0, I_3\geq 0, I_4\geq 0, A\geq 0$ and Lemma IV.5 is satisfied, it follows that $\dot{L}\leq 0$ since all other model parameters are non-negative for $\mathcal{R}_T>1$. Furthermore, $\dot{L}=0$ if and only if $S=S^{**}, I_1=I_1^{**}, I_2=I_2^{**}, I_3=I_3^{**}, I_4=I_4^{**}, A=A^{**}$. Thus, L is a Lyapunov function of the subsystem (4)-(9) on Γ. It therefore follows by LaSalle's Invariance Principle [16] that the subsystem (4)-(9) has a globally asymptotically stable endemic equilibrium point ψ^{**}. The result presented here shows that for a special case ($\tau=0$), the virus will consistently persist in the community whenever the associated reproduction number $\mathcal{R}_T>1$. ■

V. Numerical Simulation and Discussion of Results

In this section, we shall carry out the numerical simulation of the model to corroborate the analytic results. We shall solve the model equation (4)-(9) numerically and present the results graphically using Maple 18 and Python mathematical software. A 3D surface plot shall also be presented to examine the relationship between the reproduction number, the partial abstinence rate γ_2 and σ_3 which is the modification parameter which account for the assumed reduction of infectiousness by the behavior change class I_4.

Table 1: Hypothetical Value of Parameters

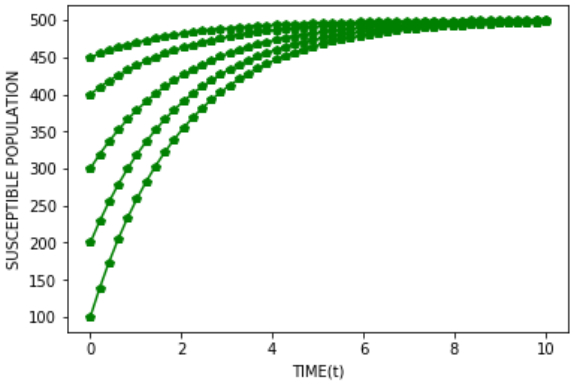
Parameter	Value (per year)	Source
B	5600	Estimated
α_1	0.25	Estimated
α_2	0.10	Estimated
β	0.015	Estimated
μ	0.016	Estimated
α_3	0.65	Estimated
ρ_1	0.12	Estimated
γ_1	1.00	[30], [20]
γ_2	0.95	Estimated
ρ_2	0.75	Estimated
τ	0.0909	[11]
σ_1	0.85	Estimated
σ_2	0.55	Estimated
σ_3	0.008	[29]

Table 2: Initial Conditions

$S(0)$	$I_1(0)$	$I_2(0)$	$I_3(0)$	$I_4(0)$	$A(0)$
450	10	8	5	10	15
400	40	25	20	10	5
300	70	50	40	30	10
200	95	65	53	47	40
100	120	88	67	65	60

To start with, we will show numerically that the disease-free equilibrium ψ^* is locally asymptotically stable. The parameter values presented in Table 1 and the initial conditions shown in Table 2 shall be used.

Considering the case when the reproduction number is less than unity i.e. $\mathcal{R}_T=0.025<1$, the graphical solution of model equation (4)-(9) is given in fig.2 - fig.7. It can be seen that only the susceptible population $S=500$ survive while the infected population in the slow progression class I_1, non progression class I_2, fast progression class I_3, behavior change class I_4 and AIDS class A goes into extinction. This confirms that the DFE of (4)-(9) as presented in Lemma (IV.4) is locally asymptotically stable whenever $\mathcal{R}_T<1$.



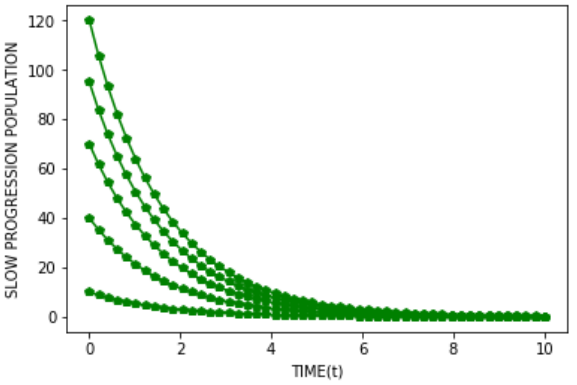
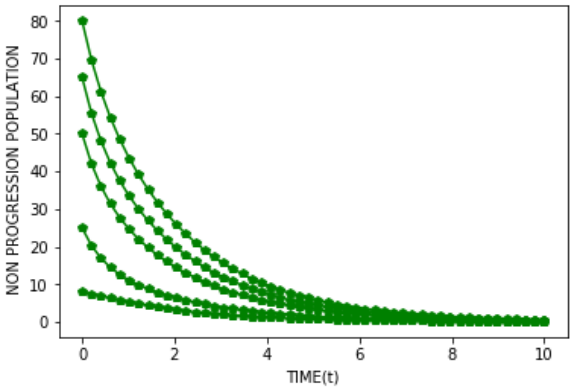


Fig. 2 and Fig. 3 Showing The Behavior of Both Susceptible and Slow Progression Populations when $\mathcal{R}_T$ is Less Than Unity.



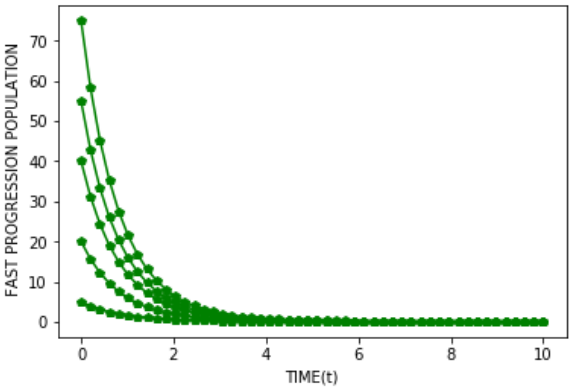
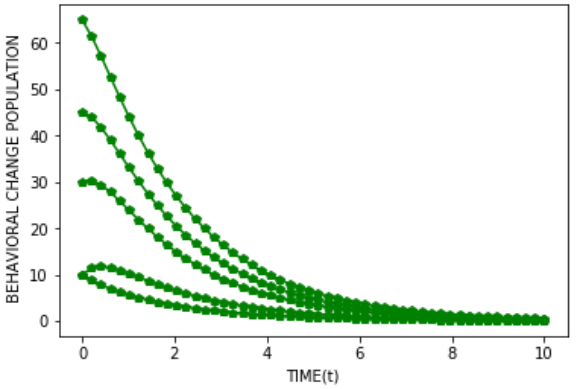


Fig. 4 and Fig. 5 Showing The Behavior of Both Non Progression and Fast Progression Populations when $\mathcal{R}_T$ is less than Unity.



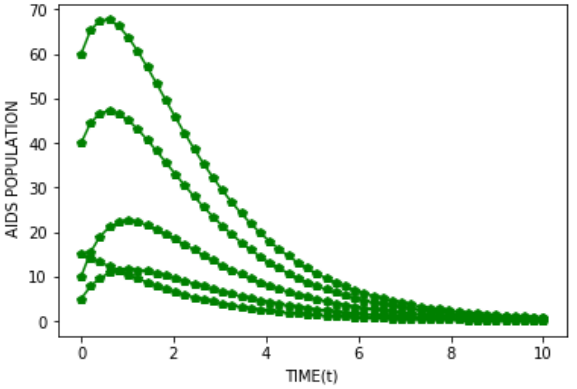
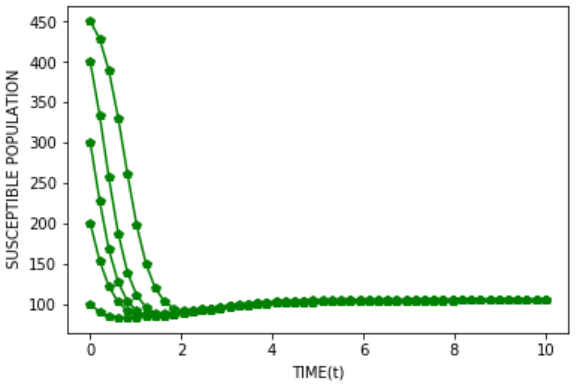


Fig. 6 and Fig. 7 Showing The Behavior of Both Fast Progression and AIDS Populations when $\mathcal{R}_T$ is Less Than Unity.



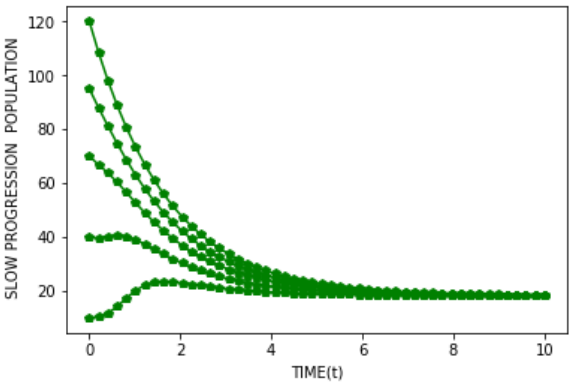
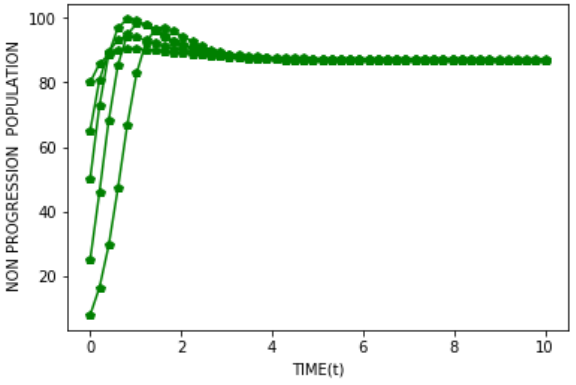


Fig. 8 and Fig. 9 Showing The Behavior of Both Susceptible and Slow Progression Populations when $\mathcal{R}_T$ is Greater Than Unity.



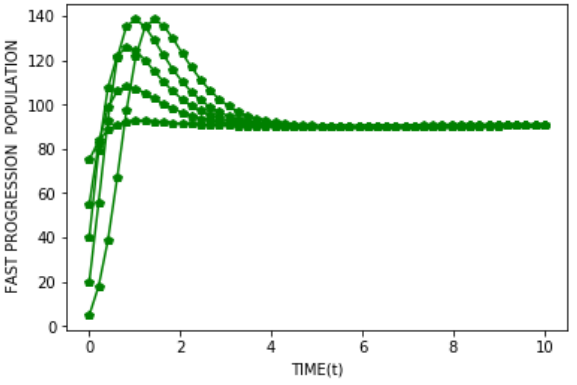
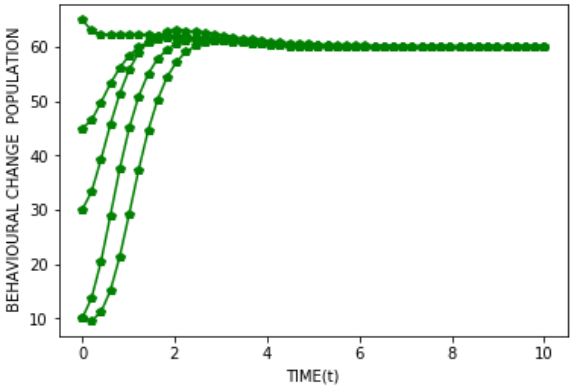


Fig. 10 and Fig. 11 Showing The Behavior of Both Non Progression and Fast Progression Populations when $\mathcal{R}_T$ is Greater Than Unity.



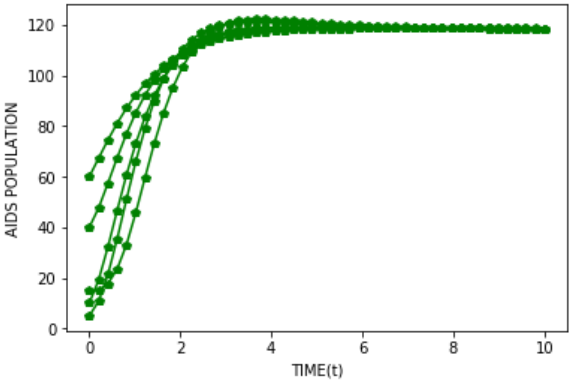


Fig. 12 and Fig. 13 Showing The Behavior of Both Fast Progression and AIDS Populations when $\mathcal{R}_T$ is Greater Than Unity.

Considering the case when the reproduction number is greater than unity i.e. $\mathcal{R}_T = 5.903 > 1$, the graphical solution of model equation (4)-(9) is given in fig.8 - fig.13. It can be observed that when the reproduction number $\mathcal{R}_T > 1$, all the populations survive that's

$$[S^{**}, I_1^{**}, I_2^{**}, I_3^{**}, I_4^{**}, A^{**}] = [0.0155, 0.6575, 0.9452, 0.8105, 0.5421, 0.7813],$$

which clearly indicates that the population converges or tends to the endemic equilibrium points ψ^{**} whenever $\mathcal{R}_T > 1$. It can also be seen that the susceptible population reduces drastically because of the reproduction number being greater than unity while all the remaining infected populations increases with time. This confirms that the endemic equilibrium points ψ^{**} is locally asymptotically stable and thus, confirms the analytic results presented in Lemma IV.5.

A. Effect of Partial and Total Abstinence in HIV/AIDS Transmission

Here, we will observe the effect of partial and total abstinence in the transmission of HIV/AIDS. We simulate the reproduction number in equation (21) and (38). For the case of partial abstinence (i.e. when $\sigma_3 = \gamma_2 \neq 0$), with the parameter values in Table 1, when $\gamma_2 = 0.95$ and $\sigma_3 = 0.008$, $\mathcal{R}_T = 0.025$. For the case of total abstinence (i.e. when $\sigma_3 = \gamma_2 = 0$), with the parameter values in Table 1, $\mathcal{R}_T^{'} = 0.020$ meaning that $\mathcal{R}_T^{'}$ for the total abstinence is less than $\mathcal{R}_T$ for the partial abstinence. Since our aim in epidemiology is to find all possible means to reduce the reproduction number of infectious disease, it means that those that changed their sexual attitude through total abstinence from HIV/AIDS and all factors that can cause its transmission are at lower or no risk of contacting HIV/AIDS. Hence, total abstinence is one of the key factors to be safe from HIV/AIDS. Figure 14 below shows a 3D surface plot to understand more about the relationship between the reproduction number and γ_2 and σ_3.

We can easily observe that the higher the value of both σ_3 and γ_2, the higher the reproduction number and the lower their values the lower the reproduction number. The lowest reproduction number 0.018 is gotten when $\sigma_3 = \gamma_2 = 0$ i.e. (total abstinence). Hence, total abstinence is essential in the protection against HIV/AIDS transmission.

VI. Conclusion, Acknowledgment and Disclosure Statement

In this study, a new virus resistant HIV-1 model with behavior change was proposed and systematically analyzed for both partial and total abstinence from HIV/AIDS. Basic analysis of the model such as positivity solution, reproduction number, invariant region, establishment of both disease-free and endemic equilibrium points for both scenarios were carried out. The local asymptotic stability of the DFE and EE for both models whenever the associated reproduction number is less than unity and greater than unity respectively were proved. A non-linear Goh–Volterra Lyapunov function is used to prove that the endemic equilibrium point is globally asymptotically stable for the case when the virus-induced death rate $\tau = 0$ while the method of Castillo-Chavez is used to prove the global asymptotic stability of the disease-free equilibrium point whenever the reproduction number is less than unity. In the numerical simulation, it was established that people with total abstinence are more protected against HIV/AIDS than those with partial abstinence and also established that the reproduction number is minimal under this same condition. Since those with resistance to HIV/AIDS do not proceed to the AIDS compartment, this also highlight the importance of HIV-resistance which plays an important role in the protection against HIV/AIDS.

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Disclosure Statement

The research work forms part of the first authors

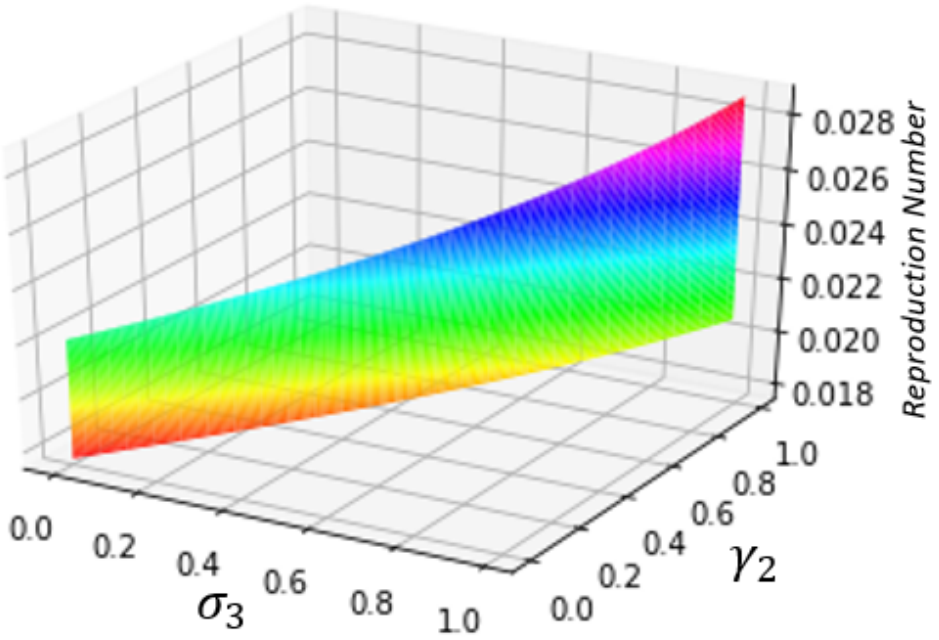

Fig. 14 Showing The Effect of σ_3 and γ_2 on the Reproduction Number Using the Parameter Values on Table 1 when $\sigma_3 = \gamma_2 = [0, 1]$.

PhD work and the co-authors are his supervisors.

Data Availability Statement

The numerical data and hypothetical value of parameters used to support the findings of this research are included within the article. They are either properly referenced, assumed or estimated in Table 1.

Conflict of Interest

No conflict of interest as far as this research is concerned.

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