Finite- and Multi-Dimensional State Representations and Some Fundamental Asymptotic Properties of a Family of Nonlinear Multi-Population Models for HIV/AIDS with ART Treatment and Distributed Delays

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Abstract. A multipopulation HIV/AIDS deterministic epidemic model is studied. The population structure is a multihuman behavioral structure composed of humans practicing varieties of distinct HIV/AIDS preventive measures learnt from information and education campaigns (IEC) in the community. Antiretroviral therapy (ART) treatment is considered, and the delay from HIV exposure until the onset of ART is considered. The effects of national and multilateral support providing official developmental assistance (ODAs) to combat HIV are represented. A separate dynamics for the IEC information density in the community is derived. The epidemic model is a system of differential equations with random delays. The basic reproduction number (BRN) for the dynamics is obtained, and stability analysis of the system is conducted, whereby other disease control conditions are obtained in a multi- and a finite dimensional phase space. Numerical simulation results are given.

1. Introduction. AIDS (acquired immunodeficiency syndrome) is a disease caused by HIV (human immunodeficiency virus). The body’s special defensive cells, CD4 cells (T cells), are attacked by HIV, thereby weakening the human immune system against infection. As the number of T-cells in the body is greatly reduced, the human body becomes vulnerable to secondary infections [2]. HIV has no cure, and infected persons live with the virus for life. According to the WHO [9], nearly 37.4 million people lived with HIV by the end of 2018. Globally, homosexuals, drug users, prisoners and people living in closed settings, sex workers and their clients, and also transgender people etc. are the most affected populations by the disease; over two thirds of the global HIV infected population lives in sub-Saharan Africa.

There is biomedical treatment against HIV. The medicine used to treat HIV is called antiretroviral therapy or ART [2]. With proper ART, the viral load reduces and becomes undetectable. People treated properly with ART, and with undetectable viral loads, live healthy long lives, and exhibit effectively no risks of transmitting the virus to HIV-negative persons [2]. Moreover, if HIV is diagnosed

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early and properly treated, the individuals live nearly natural lives as uninfected individuals. However, without proper treatment, or in the absence of treatment, the infected persons progress to AIDS, and this can occur in 2 to 15 years [9]. Thus, there is a critical time delay $\tau_2$ (cf. [19]) to diagnosis and the onset of proper treatment necessary for a healthy longer lifespan; there is also the natural time delay of 2 to 15 years, $\tau_1$, until the onset of AIDS.

ART treatment contributed to save about 13.6 million lives between 2000 and 2018 [9]. Moreover, because of national HIV programs supported by civil society and international organizations and partners, the number of new HIV infections decreased by 37%, and HIV related deaths also decreased by 45%. National and multinational campaigns against HIV have a success history in many developing countries, e.g. Uganda has succeeded to reduce the prevalence of HIV over the years since the late 1980s. The success is attributed to governmental information, education and treatment campaigns against HIV (cf. [15, 25, 12]). Other studies [22, 10] investigate the roles of information and education campaigns (IEC) on the prevalence of HIV/AIDS.

National, multinational and multilateral assistance to poor countries in provision of aids, is another measure to combat HIV/AIDS. Top multilateral organizations fighting against HIV/AIDS such as Global Fund [3], and PEPFAR [8] etc. provide official development assistance (ODA) (cf. [23]), funding and supporting large and small scale projects globally designed to prevent and treat HIV/AIDS. Such assistance in treatment or prevention has saved many lives against the disease (cf. [8, 3, 11, 23]). On the downside, the supply of ODAs are sometimes sporadic and limited, and this can upset disease control. For instance, recently, the American government discussed options to cutback funds and spending on ODAs against HIV/AIDS (cf. [4]). This announcement has led to studies (cf. [27]), to forecast long-term effects of such policy change on the global HIV/AIDS prevalence. The studies project insignificant monetary savings by the US government, when the new policy is applied; the studies rather project devastating clinical and epidemiological impacts on the global spread of HIV/AIDS. Thus, it is necessary to mathematically model and study the effects of ODAs provided by national, multinational and multilateral support, to combat HIV/AIDS.

Mathematical models have certainly advanced understanding about the dynamics of HIV/AIDS epidemics [15, 14, 22, 10, 20, 36]. Mathematical epidemic models with information intervention also advance understanding about the role of information and education in changing attitudes and behavior that lead to disease control [21, 18, 15, 22, 10]. Joshi et. al. [15] studied a SIRE model for HIV/AIDS epidemic in Uganda. Two susceptible classes with change of behavior namely- practicing abstinence or condom use, emerge from a general uneducated susceptible class via interaction with information in the community from information and education campaigns (IEC). The three susceptible classes nevertheless experience the disease from interactions with infected persons, but with different per capital disease transmission rates. In their model, the density of education in the community has a separate dynamics. Huo et.al. [14] also studied a HIV/AIDS epidemic model with a treatment class with ART. The treated class does not transmit the disease, and it can either relapse to the active infectious class, if treatment is discontinued, or progress into full blown AIDS.

1 All forms of domestic or foreign monetary support and other aids provided to combat HIV are referred to as ODAs in this paper.
Using some ideas in the studies [15, 14], a more general deterministic HIV/AIDS epidemic model is studied in this paper. It is assumed that the IECs in the community educate the adult population with multiple preventive measures (more than the two in [15]) against HIV. Moreover, the response to the education results in multiple distinct behavioral changes that can be visibly characterized into distinct sub-susceptible classes $S_j, j = 0, 1, \ldots, n, \quad n \in \mathbb{N}$, exhibiting lowered disease vulnerabilities. Furthermore, the density of information or education, $Z(t)$, at any time $t$ has a separate dynamics. The epidemic model is a system of differential equations with distributed delays representing the delay time until a newly infected person begins ART treatment, and the time until the T-cells of an infected person are sufficiently depleted, and lead to full blown AIDS.

In this paper, the impacts of IECs, the limited supply of ODAs and delayed ART treatment on HIV/AIDS dynamics are investigated from a deterministic perspective, that is, in the absence of noise in the disease dynamics. Deterministic Lyapunov functionals stability analytic techniques are applied to find disease control conditions. Moreover, the numerical simulation results are given using partial UGANDA HIV/AIDS data, to justify the results of this study.

It is important to note that certain strong fundamental properties of epidemic dynamics systems are unique to deterministic systems, and these properties are lost with the occurrence of noises in the system. For instance, the existence and stability results for the steady states of a deterministic differential equation system can easily cease to exist with the occurrence of some types of noises in the system (cf.[31, 32, 28]).

Thus, it is important that a deterministic epidemic system is thoroughly investigated to understand the fundamental properties of the system that would be affected by the occurrence of noises in the system. Thus, this paper is designed to understand the fundamental properties of the system in a deterministic environment, that emerge from the interactions between the IECs, supply of ODAs, and delays in the disease dynamics, and leading to HIV/AIDS disease control.

This paper is significantly different from [33] in that: (1) it addresses the HIV/AIDS disease dynamics in the absence of noises in the system, (2) deterministic Lyapunov functionals techniques are applied to analyze the global stability of the equilibrium, and (3) the stability analysis of the equilibrium is conducted (a) on a multidimensional phase space that gives a microscopic view of the impacts of human behavior on the asymptotic properties of the disease dynamics, and (b) on a finite dimensional phase space that gives a general view of the impacts of human behavior on the disease dynamics.

This paper is organized as follows. In Sections 2-3, the epidemic model is derived. In Section 4 model validation results are presented. In section 5, the BRN is computed and stability of the model is conducted to find disease control conditions. In Section 6, a discussion of the results is presented to determine disease eradication conditions. Numerical simulation results for UGANDA HIV/AIDS epidemic are presented in Section 7.

2. Description of the epidemic model. The HIV/AIDS epidemic model is based on the following assumptions summarized into definitions.

**Definition 2.1. Notations and abbreviations**

1. The notation $I(k, n), k \geq 0$ represents the set of consecutive natural numbers between $k$ and $n$. E.g. $I(0, n) = \{0, 1, 2, \ldots, n\}$. 

2. The following abbreviations are used:
- “IECs” abbreviates “information and education campaigns”;
- “ODA” abbreviates “official development assistance”;
- “ART” abbreviates “antiretroviral therapy”;
- “DFE” abbreviates “disease free equilibrium”;
- “SSP” abbreviates “stochastic solution process”;
- “BRN R_0” abbreviates “basic reproduction number”;
- “PrEP” abbreviates “Pre-exposure prophylaxis”.

Definition 2.2. Population structures and human behavioral categories:
(A) Sexually active adults in a community are considered. The primary means of
HIV transmission is sexual contact. Vertical transmission is not considered, and
alternative means of transmission such as contact with infected needles are not
considered. The IECs are designed to change adult sexual behaviors, especially of
the susceptible population.

It is assumed that the IECs lead to \( n \in \mathbb{N} > 1 \) distinct behavioral categories based
on safe-sex measures that are taught and learnt via the IECs (cf. [12, 24, 7, 6, 37]).
Examples of the safe-sex behavioral categories include preventive measures such as:
abstinence, mutually monogamous relationships (i.e. be faithful to partners), condom
use, use of lubricants, voluntary medical male circumcision, counseling, harm
reduction interventions for people who use drugs and all other distinct preventive
measures that reduce vulnerability and transmission rates of HIV. For a comprehen-
sive WHO recommended HIV prevention package and other HIV preventive
measures, see [12, 24, 7, 6, 37]. It is assumed that nearly all adults learn and ac-
tively practice at most one distinct measure at a time. That is, everyone practices a
\( j^{th} \) measure, \( j \in I(0, n) \), where the category \( j = 0 \) represents the state of “naivety”,
where no safe-sex measure is practiced. All persons who practice at least two mea-
sures at a time are collectively grouped into one of the \((n + 1)\) behavioral categories
\( j \in I(0, n) \).

The total sexually active human population \( N(t) \) is decomposed into five major
states namely: the susceptible state \( S(t) \), which is vulnerable to HIV infection; the
HIV infected individuals not receiving ART \( I(t) \); the treatment state \( T(t) \), repre-
senting all HIV infected individuals receiving ART treatment; the AIDS state \( A(t) \),
representing all HIV infected persons in the advanced stages of their HIV infection,
and experiencing full symptoms of AIDS; the removed state \( R(t) \), representing all
those who practice safer sexual behavior, and fully protected from HIV infection.
The removed state can consist of individuals who are adhering to HIV prevention
measures such as PrEP (see Model-Assumption 2.3 ). The susceptible state \( S(t) \) is
further decomposed into \((n + 1)\) distinct susceptible states \( S_j(t), j \in I(0, n) \), based
on the \( n + 1 \) IECs behavioral categories above. Hence,

\[
N(t) = S(t) + I(t) + T(t) + A(t) + R(t),
\]

where

\[
S(t) = \sum_{j=0}^{n} S_j(t).
\]

Indeed, the state \( S_0(t) \) represents all susceptible individuals at time \( t \), who do not
practice any HIV preventive measures, either due to negligence or limited knowl-
dge about HIV preventive measures. The states \( S_j(t), j \in I(1, n) \) represent all
susceptible individuals who through the IECs, learn and actively practice the \( j^{th} \)
major HIV/AIDS preventive measure, where \( j \in I(1,n) \). Note that for obvious reasons, it is not necessary to decompose the other states \( I, T, A, R \) into the \((n + 1)\) behavioral categories.

It is also assumed that there is a constant influx \( B \) per unit time of susceptible adults in the population. Moreover, all new individuals into the population are susceptible of type \( S_0 \).

Definition 2.3. Information density and interaction rates:

(B) The density of information at anytime \( t \) is denoted \( Z(t) \). The “naive” susceptible individuals \( S_0(t) \) modify their behavior to become at most one of \( S_j(t) \), \( j \in I(1,n) \) after receiving education, \( Z(t) \), about the disease at time \( t \), at the effective response rate \( \gamma_j = \gamma_{S_0 S_j} \), \( j \in I(1,n) \).

The rate per unit time at which the susceptible individuals in class \( S_0(t) \) change their behavior into class \( S_j(t) \) is given by the expression \( \gamma_j S_0(t) H_j(Z(t)) \), where \( H_j, j \in (1,n) \) is a nonlinear function describing the response of the susceptible class \( S_0(t) \) to the density of information \( Z(t) \) in the population.

It is also assumed that some individuals in the susceptible class \( S_0(t) \) experience the highest impacts of the IECs after interacting with information \( Z(t) \) at effective contact rate \( \gamma_0 = \gamma_{S_0 R} \). The impacts of the education obtained from the IECs result to reform their sexual behavior, and produce actions all through their lives that never result in contracting the disease. In other words, these individuals are considered to be immune to the disease and removed, \( R(t) \), at time \( t \). Indeed, according to WHO and CDC (cf. [7, 6, 37]), proper training in the use of HIV preventive medications such as Pre-exposure prophylaxis (PrEP), leads to reduction of HIV infection risk by 99%. Thus, individuals in the population who are properly trained and adhere to correct daily use of PrEP can be considered into the removal class \( R(t) \).

Thus, \( \gamma_0 S_0(t) H_0(Z(t)) \) is the rate per unit time at which \( S_0(t) \) individuals are removed \((R)\), by fully reforming their sexual behaviors and attitudes via interacting with information \( Z(t) \), and practicing safe-sex measures that will never lead to HIV infection.

Indeed, it is assumed that the effects of the content of the information related to HIV prevention measures from the IECs, initially rises in susceptible individuals who have not heard it, due to excitement about knowledge of new preventive measures, then saturates due to familiarity with the content of the information. The properties of \( H_j \) are given in Assumption 2.1. Using ideas in [31, 32, 30] we adopt assumptions for the nonlinear function \( H_j, j \in I(0,n) \).

Assumption 2.1. \( A1: H_j(0) = 0; A2: H_j(Z) \) is strictly monotonic on \([0, \infty)\); \( A3: H_j \in C^\infty(\mathbb{R}_+, \mathbb{R}_+) \) and \( H_j''(Z) < 0; A4. \lim_{Z \to \infty} H_j(Z) = C_1, 0 \leq C_1 < \infty; \) and \( A5: H_j(Z) \leq Z, \forall Z > 0. \)

Examples of functions in the \( H \)-family in Assumption 2.1 are \( H(x) = \frac{ax}{b + cx}, x \geq 0 \), where \( a \in [0,1] \), and \( b \geq 1 \); \( H(x) = \frac{cx}{d + cx}, x \geq 0 \), where \( c \geq 1 \), and \( d \geq 1 \). Note that \( a, c \) are the scale parameters of the functions; \( b \) is the saturation parameter, and \( d \) is a shape parameter of the function.

In addition, using ideas from [18], the rate \( \gamma_j \) can be expressed as \( \gamma_j = \nu_j a_j \), where \( a_j \) is the interaction rate by which individuals of type \( S_j \) change their behavior, and \( \nu_j \in [0,1] \) is the response intensity.
The density of information in the population $Z(t)$ at time $t$ from the IECs is assumed to grow at a rate that is proportional to the number of infected individuals $I(t), T(t), A(t)$ in the population. Furthermore, the growth rate exhibits nonlinear character in response to the number of infected individuals of all states $I(t), T(t), A(t)$ in the system. This growth rate per unit time is represented by the function $F_Z(I(t), T(t), A(t))$.

Apparently, the content of information in the IECs relates to the infected classes $I(t), T(t), A(t)$, as preventive measures are taught against these states. Moreover, the rate of supply of information, $F_Z(I(t), T(t), A(t))$, saturates over time with increase in $I(t), T(t), A(t)$ (see B). Also, it is assumed that the effectiveness or strength of the content of the information in the IECs degrades at the rate $\mu_Z$. A special form for the function $F_Z(I(t), T(t), A(t))$ is given in (17).

Note that there are several different ways to quantify HIV information density $Z(t)$ in the population over time. For instance, in [15], the amount of information present at anytime is a function of the number of national, international, governmental and non-governmental organizations involved in IECs against HIV in Uganda at any time.

**Definition 2.4. Disease transmission and generalized standard incidence rates of the disease:**

(C) The active HIV infectious class $I(t)$ passes infection to all susceptible states $S_j, j \in I(0, n)$. However, because of preventive measures learnt from the IECs, the sub-classes $S_j, j \in I(1, n)$ experience a reduced rate of transmission from $I$, than the class $S_0$. That is, at the rate $\beta_j = \beta_0, j \in I(0, n)$, the interaction between the susceptible state $S_j$ and infectious state $I(t)$ results in HIV transmission. The rate $\beta_j = \beta_0, j \in I(0, n)$ represents the average number of effective contacts (i.e. sufficient contacts to transmit disease) per person per unit time. Moreover, $\beta_0 \geq \beta_j, j \in I(1, n)$.

Recognizing the viewpoints regarding the incidence rates of human epidemics (cf. [13]), a nonlinear generalization of the standard incidence rate $\frac{\beta_j S_j(t) I(t)}{N(t)} = \beta_j S_j(t) i(t), i(t) = \frac{I(t)}{N(t)}$ is considered, where $N(t)$ is the total population at time $t$.

Indeed, $i(t) = \frac{I(t)}{N(t)}$ is the fraction of the HIV infectious persons in the population at time $t$. Observe that when $N(t)$ is a constant, then $\frac{\beta_j S_j(t) I(t)}{N(t)} = \beta_j S_j(t) i(t) = \theta(t) \beta_j S_j(t) I(t)$, where the fraction $0 < \theta(t) = \frac{1}{N(t)} < 1$, reflects that the incidence rate rises linearly with respect to the infectious state $I$, and this pattern is unsuitable for most human epidemics (cf. [17]). Also, when the total population size $N(t)$ grows or declines proportionately with a rise or a drop in disease transmission in the population, respectively, i.e. $N(t)$ and the infectious state $I(t)$ both change (increase or decrease) proportionately, then the fraction $i(t) = \frac{I(t)}{N(t)}$ is constant overtime, and the standard incidence rate $\frac{\beta_j S_j(t) I(t)}{N(t)} = \beta_j S_j(t) i(t)$ no longer reflects the true incidence rate of the disease in the population.

Thus, to increase flexibility in the standard incidence rate to represent more real life scenarios, a nonlinear incidence function $G_j$ is introduced with assumptions in Assumption 2.2. The properties of the nonlinear function $G_j$ in Assumption 2.2 signify a psychological response from the susceptible classes $S_j, j \in I(0, n)$, where more susceptibles apply more appropriate preventive measures and actions that limit contacts with infectious persons, as the HIV infectious state $I(t)$ increases in the community over time.
The modified nonlinear incidence rate of HIV in the state $S_j$, is given by the expression $\beta_j S_j(t)G_j(i(t)), j \in I(0, n)$. The function $G_j$ satisfies the assumptions in Assumption 2.2.

Using ideas in [31, 32, 30] we adopt assumptions for the nonlinear function $G_j, j \in I(0, n)$.

**Assumption 2.2.**

1. $G_j([0,1]) \subseteq \mathbb{R}^+$, $G_j(0) = 0$;  
2. $G_j(t)$ is strictly monotonic on $[0,1]$;  
3. $G_j \in C^2([0,1], \mathbb{R}_+)$ and $G_j''(i) \leq 0$;  
4. $\lim_{i \to \infty} G_j(i) = C_2, 0 \leq C_2 < \infty$; and  
5. $G_j(i) \leq i, \forall i > 0$.

An example of a function in the $G$-family in Assumption 2.2 is $G(x) = ax^{1+b}x, x \geq 0$, where $0 \leq a \leq 1$, and $b \geq 1$. Note that $G$ is expressed as follows $G(x) = \frac{ax}{1+bx} = \frac{x}{\frac{1}{b}+x}, x \geq 0$, where $\frac{1}{b}$ is the scale parameter of the function $G$ and $\frac{1}{b}$ is the saturation constant. Indeed, to illustrate further, Figure 1 depicts the behaviors of the standard incidence function $i(t) = \frac{I(t)}{N(t)}$, and a modified standard incidence function $G(i(t)) = \frac{ai(t)}{1+bi(t)}$, as the number of infectives increase, where $I(t) \in [0,1000000]$ and $a = 0.05, b = 10$. Note that Figure 1 describes the change in the incidence functions $i(t) = \frac{I(t)}{N(t)}$, and $G(i(t)) = \frac{ai(t)}{1+bi(t)}$, with respect to state $I(t)$, and not necessarily with respect to time $t$.

![Modified Standard Incidence Rate](image1)

**Figure 1.** Shows the behaviors of the modified standard incidence and the ordinary standard incidence rates as the number of infectives continually increase over time. Clearly, the modified standard incidence is more suitable for many real life scenarios where the incidence rate of the disease saturates over time as the number of infections increase in the population.

Note that disease transmission from the AIDS state $A(t)$ and from treatment state $T(t)$ are not considered (cf. [15, 14]). Indeed, it is assumed that individuals in the AIDS stage of their HIV infection exhibit the typical visible symptoms of the disease(cf. [5]), and as a result they are either aware of their disease status and take precautionary measures via abstinence to not infect others, or they are unwell due to symptoms of the disease.

**Definition 2.5. Supply of ODAs and distributed delays in the disease dynamics:**

(D) The effects of the supply of ODAs will be assessed through the parameters
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AIDS state who do not receive ART treatment, and consequently progress to the full-blown AIDS state \( A(t) \) after the time delay \( \tau_1 \), and \( \bar{\varepsilon}_j \equiv 1 - \varepsilon_j \in (0,1) \) is the other proportion per unit time of the newly infected from the class \( S_j \), \( j \in I(0,n) \), who after a time delay \( \tau_2 \) following exposure to HIV, proceed to be tested, begins ART treatment and become the state \( T(t) \), respectively.

It is expected that a constant significant supply of ODAs in a community enables more people to be easily tested and to afford ART. And as a result, the proportion \( \bar{\varepsilon}_j \equiv 1 - \varepsilon_j \in (0,1) \) increases, and the delay until ART begins, \( \tau_2 \), decreases on average. Also, if there is little or no supply of ODAs in the community, then more people cannot afford testing and ART, and consequently the proportion \( \varepsilon_j \in (0,1) \) increases.

Clearly, the time delay until the onset of ART, \( \tau_2 \), varies with available resources from ODAs, and also varies with the attitudes of newly exposed individuals in the population. Indeed, some communities attach huge social stigmas to HIV/AIDS, and as a result many people are dissuaded by such stigmas from testing and beginning early ART. Sometimes late commencing of ART may be the result of simple ignorance about the benefits of early testing and ART etc. These combinations of attitudes towards HIV can delay testing and diagnosis of HIV, and consequently, lead to delayed ART. Also, as remarked earlier, progression from HIV without treatment to full-blown AIDS can occur after time delay \( \tau_1 \) varying between 2 to 15 years. Therefore, distributed time delays \( \tau_1 \) and \( \tau_2 \) are considered to represent the variabilities in the delays, with probability density functions \( f_{\tau_1}, t_0 \leq \tau_1 \leq h_1 \) and \( f_{\tau_2}, t_0 \leq \tau_2 \leq h_2 \).

**Definition 2.6. Relapse from treatment and developing full-blown AIDS:**
(E) The only form of treatment considered is ART. In the advanced stages of HIV without treatment, the infectious individual \( I(t) \) develops full-blown AIDS \( A(t) \) after the natural incubation period \( \tau_1 \). Individuals who begin treatment \( T(t) \) in the advanced stages of HIV, where considerable damage to T-cells has occurred, can still progress to full-blown AIDS \( A(t) \) at the per capita rate \( \alpha_{TA} \), as treatment fails. Individuals diagnosed early with HIV, and receiving treatment can discontinue ART due to limited supply of ODAs in the community, or due to personal self-limiting factors against the ART. Such individuals in the state \( T(t) \) relapse to the active infectious state \( I(t) \) at the rate \( \alpha_{TI} \). Indeed, it noted that the transition from the state \( T \) to state \( I \), with withdrawal from treatment is more complex than a simple linear transfer from \( T \) to \( I \) at the rate \( \alpha_{TI} \). In fact, withdrawal from treatment can be the result of interactions that lead to discouragement from treatment, by those who have already withdrawn, and are back to being active spreaders \( I \). However, for simplicity, the linear form \( \alpha_{TI} T(t) \) is used to model withdrawal from ART treatment.

**Definition 2.7. The per capita death rates:**
(F) All individuals of all states in the population die naturally at the per capita rate \( \mu_k, k \in \{ S_j, I, T, A, R \}, j \in I(0,n) \), while the infected classes \( I, T, A \) die from the disease at the rate \( d_k, k \in \{ I, T, A \} \). Since in most natural settings the natural death causes are uniform across all disease states, then \( \mu_k = \mu, k \in \{ S_j, I, T, A, R \}, j \in I(0,n) \), where \( \mu \) is a constant. However, the distinct notation for the natural deathrates \( \mu_k, k \in \{ S_j, I, T, A, R \}, j \in I(0,n) \) will be used.
3. Derivation of the epidemic model. It follows from the assumptions (A)-(F) in Definitions 2.1-2.7 above that a compartmental framework depicting the transitions between the different states of the population is given in Figure 2, and the deterministic HIV/AIDS epidemic dynamic model with treatment and information intervention follows immediately.

\[
dS_0(t) = \left[ B - \sum_{j=1}^{n} \gamma_j S_0(t) H_j(Z(t)) - \beta_0 S_0(t) G_0(i(t)) - \gamma_0 S_0(t) H_0(Z(t)) - \mu S_0 S_0(t) \right] dt,
\]

\[
dS_j(t) = \left[ \gamma_j S_0(t) H_j(Z(t)) - \beta_j S_j(t) G_j(i(t)) - \mu S_j S_j(t) \right] dt, \quad j \in I(1, n),
\]

\[
dI(t) = \left[ \beta_0 S_0(t) G_0(i(t)) + \sum_{j=1}^{n} \beta_j S_j(t) G_j(i(t)) \right.
\]

\[ - \sum_{j=0}^{n} \int_{t_0}^{t} \varepsilon_j \beta_j S_j(t-r) G_j(i(t-r)) e^{-\mu t r} f_{\tau_j}(r) dr \]

**Figure 2.** shows the different states of the population in the HIV/AIDS epidemic, and the transition rates between the states. Note that the operators \(E_{\tau_1}[\cdots]\) and \(E_{\tau_2}[\cdots]\) represent expectations with respect to the random variables \(\tau_1\) and \(\tau_2\), respectively.
\[-\sum_{j=0}^{n} \int_{t_0}^{h_2} (1 - \varepsilon_j) \beta_j S_j(t - u) e^{-\mu_T u} f_{\tau_j}(u) du - \mu_T I(t) - d_I I(t) + \alpha_T I(t) \right] dt, \tag{5}\]

dT(t) = \left[ \sum_{j=0}^{n} \int_{t_0}^{h_2} (1 - \varepsilon_j) \beta_j S_j(t - u) G_j(i(t - u)) e^{-\mu_T u} f_{\tau_j}(u) du \right.
- \alpha_T I(t) - d_T T(t) - \alpha_T A(t) \right] dt, \tag{6}\]

dA(t) = \left[ \sum_{j=0}^{n} \int_{t_0}^{h_1} \varepsilon_j \beta_j S_j(t - r) G_j(i(t - r)) e^{-\mu_T r} f_{\tau_j}(r) dr \right.
- \mu_A A(t) - d_A A(t) + \alpha_T A(t) \right] dt, \tag{7}\]

dR(t) = [\gamma_0 S_0(t) H_0(Z(t)) - \mu_R R(t)] dt, \tag{8}\]

and

dZ(t) = [F_Z(I(t), T(t), A(t)) - \mu_Z Z(t)] dt, \tag{9}\]

where the initial conditions are given in the following: define \( h = \max(h_1, h_2), \)
\( (S_j(t), I(t), T(t), A(t), R(t), Z(t)) = (\vartheta_j(t), \varphi_1(t), \varphi_2(t), \varphi_3(t), \varphi_4(t), \varphi_5(t)), \)
\( t \in (t_0 - h, t_0], \)
\( \vartheta_j(t), \varphi_k \in C((t_0 - h, t_0], \mathbb{R}_+), \forall j \in I(0, n), k \in I(1, 5) = \{I, T, A, R, Z\}, \)
\( \vartheta_j(t_0), \varphi_k(t_0) > 0, \forall j \in I(0, n), k \in I(1, 5), \tag{10}\]

and \( C((t_0 - h, t_0], \mathbb{R}_+) \) is a Banach space of continuous functions endowed with the uniform norm

\[ ||\varphi||_{\infty} = \sup_{t_0 - h \leq t \leq t_0} |\varphi(t)|. \tag{11}\]

Indeed, the delay terms in (3)-(9) are derived as follows. The term \( \beta_j S_j(t) G_j(i(t)) \), \( j \in I(0, n) \) is the newly infected individuals per unit time from the susceptible state \( S_j(t) \) at any time \( t \geq t_0 \). The fraction \( \varepsilon_j, j \in I(0, n) \) of the newly infected population fail to get ART and progresses into full-blown AIDS after \( \tau_1 \) time units. Also, the other proportion \( \bar{\varepsilon}_j = 1 - \varepsilon_j, j \in I(0, n) \) of the newly infected population will get ART after \( \tau_2 \) time units, and enter into the treatment state \( T(t) \).

Let \( h = \max(\tau_1, \tau_2) \). Thus, assuming exponential survival lifetime during HIV infectiousness, the total number of infectious persons at any time \( t \geq t_0 \) is

\[ I(t) = I_0 e^{-\mu_T (t-t_0)} p(t-t_0) \]

\[ + \int_{t_0}^{t} \left( \varepsilon_0 \beta_0 S_0(\theta) G_0(i(\theta)) e^{-\mu_T (t-\theta)} p_{\varepsilon_0}(t-\theta) + \sum_{j=1}^{n} \varepsilon_j \beta_j S_j(\theta) G_j(i(\theta)) e^{-\mu_T (t-\theta)} p_{\varepsilon_j}(t-\theta) \right) d\theta \]

\[ + \int_{t_0}^{t} \left( \varepsilon_0 \beta_0 S_0(\theta) G_0(i(\theta)) e^{-\mu_T (t-\theta)} p_{\varepsilon_0}(t-\theta) + \sum_{j=1}^{n} \bar{\varepsilon}_j \beta_j S_j(\theta) G_j(i(\theta)) e^{-\mu_T (t-\theta)} p_{\bar{\varepsilon}_j}(t-\theta) \right) d\theta, \tag{12}\]

where \( I_0 \) is the initial infectious population, and \( p_{\varepsilon_j}(t), j \in I(0, n) \) is the probability that a newly infected person who will not receive ART, remains infectious for \( t \) time
and units. Also, \( p_{\epsilon_j}(t), j \in I(0, n) \) is the probability that a newly infected person who will receive ART, remains infectious for \( t \) time units. Clearly,

\[
p_{\epsilon_j}(t) = \begin{cases} 
1, t < \tau_1, & \text{if } j \in I(0, n) \\
0, \text{otherwise,} \quad \text{if } j \in I(0, n)
\end{cases}
\]

and

\[
p_{\epsilon_j}(t) = \begin{cases} 
1, t < \tau_2, & \text{if } j \in I(0, n) \\
0, \text{otherwise,} \quad \text{if } j \in I(0, n)
\end{cases}
\]

and

\[
p(t) = \sum_{j=0}^{n} p_{\epsilon_j}(t) + \sum_{j=0}^{n} p_{\epsilon_j}(t).
\]

It is easy to see from (12)-(15) that for any time \( t \geq h \), where all initial perturbations have already been converted, then \( I(t) \) becomes

\[
I(t) = \int_{t - \tau_1}^{t} \left( \varepsilon_0 \beta_0 S_0(\theta) G_0(i(\theta)) e^{-\mu_I(t-\theta)} + \sum_{j=1}^{n} \varepsilon_j \beta_j S_j(i(\theta)) G_j(i(\theta)) e^{-\mu_I(t-\theta)} \right) d\theta
\]

\[
+ \int_{t - \tau_2}^{t} \left( \varepsilon_0 \beta_0 S_0(\theta) G_0(i(\theta)) e^{-\mu_I(t-\theta)} + \sum_{j=1}^{n} \varepsilon_j \beta_j S_j(i(\theta)) G_j(i(\theta)) e^{-\mu_I(t-\theta)} \right) d\theta.
\]

Recall Definition 2.5, the delays \( \tau_1 \) and \( \tau_2 \) are random variables with probability densities \( f_{\tau_1}, t_0 \leq \tau_1 \leq t_1 \) and \( f_{\tau_2}, t_0 \leq \tau_2 \leq t_2 \). Therefore, taking the average of (16) with respect to the delays \( \tau_1 \) and \( \tau_2 \), and differentiating the result, and also assuming the absence of disease related death rate \( d_I(t) \), and relapse from treatment at rate \( \rho T(t) \), we obtain (5).

Hence, in the model (3)-(9), the delay term \( \sum_{j=0}^{n} \int_{t_0}^{t_1} \varepsilon_j \beta_j S_j(t - r) G_j(i(t - r)) e^{-\mu_I(r)} f_{\tau}(r) dr \) represents the average number per unit time of individuals newly infected at earlier times \( t - r, r \in [t_0, h_1] \), who survived natural death rate over the incubation period, \( \tau_1 \), of HIV, with exponential survival distribution \( e^{-\mu_I(r)} \), \( \forall r \in [t_0, h_1] \), and are currently converted into full-blown AIDS class.

The other delay term \( \sum_{j=0}^{n} \int_{t_0}^{t_2} (1 - \varepsilon_j) \beta_j S_j(t - u) G_j(i(t - u)) e^{-\mu_I u} f_{\tau}(u) du \) represents the average number per unit time of individuals newly infected at earlier times \( t - u, u \in [t_0, h_2] \), who survived natural death rate over the time lapse between initial infection and the onset of treatment, with exponential survival distribution \( e^{-\mu_I u}, \forall u \in [t_0, h_1] \), and are now newly converted into the treatment class. All other parameters of the model are non-negative and defined in Assumptions (A)-(F) in Definitions 2.1-2.7.

To complete the model formulation, applying some ideas in [18] to Definition 2.3, we take the function

\[
F_Z(I(t), T(t), A(t)) = \frac{\phi_I I(t) + \phi_T T(t) + \phi_A A(t)}{1 + \phi_I I(t) + \phi_T T(t) + \phi_A A(t)},
\]

where \( \phi_i \) is the growth rate of the information and \( \hat{\phi}_i \) is the saturation constant owing to the \( i^{th} \) class \( i \in \{I, T, A\} \).

Observe from (17) that for \( I(t), T(t), A(t) \in \mathbb{R}_+ \),

\[
\frac{\phi_{\min}(I(t) + T(t) + A(t))}{1 + \phi_{\max}(I(t) + T(t) + A(t))} \leq F_Z(I(t), T(t), A(t)) \leq \frac{\phi_{\max}(I(t) + T(t) + A(t))}{1 + \phi_{\min}(I(t) + T(t) + A(t))},
\]

where \( \phi_{\min} = \min(\phi_k), k \in \{I, T, A\} \), and \( \phi_{\max} = \max(\phi_k), k \in \{I, T, A\} \).
The form for $F_Z$ in (17)-(18) signifies that the growth rate of the information in the population saturates as the infected population increases. Furthermore, it is assumed that there is relatively more preventive information related to getting infected (becoming $I$ state) and getting treatment (becoming $T$ state) than progressing from HIV infectious ($I$) state into full-blown AIDS ($A$). That is,

$$\phi_A \approx 0 \quad \text{and} \quad \hat{\phi}_A \approx 0.$$  \hfill (19)

Observe that for $F_Z$ in (17) with (19), the equations (7) and (8) decouple from the system (3)-(9). The following vectors defined below, will be used.

$$Y(t) = (S_0(t), \ldots, S_n(t), I(t), T(t), A(t), R(t), Z(t))^T \in \mathbb{R}^{n+6}_{+},$$

$$X(t) = (S_0(t), \ldots, S_n(t), I(t), T(t), Z(t))^T \in \mathbb{R}^{n+4}_{+},$$

$$N(t) = S(t) + I(t) + T(t) + A(t) + R(t), S(t) = \sum_{j=0}^n S_j(t),$$

$$Y_{com}(t) = (S(t), I(t), T(t), A(t), R(t), Z(t))^T$$

$$= \left( \sum_{j=0}^n S_j(t), I(t), T(t), A(t), R(t), Z(t) \right)^T \in \mathbb{R}^6_+,$$

$$X_1(t) = (S(t), I(t), T(t), Z(t))^T = \left( \sum_{j=0}^n S_j(t), I(t), T(t), Z(t) \right)^T \in \mathbb{R}^4_+.$$  \hfill (20)

It is important to note from (20) and the existence result in Theorem 4.1 that the deterministic model (3)-(9) describes the dynamic behavior of the population and the HIV/AIDS epidemic on the $n+1$ multi-dimensional phase space $\mathbb{R}^{n+6}_+$. Observe that by summing the equations for the susceptible states (3)-(4), and applying the notation $S(t) = \sum_{j=0}^n S_j(t)$, leads to (21).

$$d \left[ \sum_{j=0}^n S_j(t) \right] = \left[ B - \sum_{j=0}^n \beta_j S_j(t) G_j(i(t)) - \gamma_0 S_0(t) H_0(Z(t)) - \sum_{j=0}^n \mu_j S_j(t) \right] dt,$$

$$dI(t) = \left[ \beta_0 S_0(t) G_0(i(t)) + \sum_{j=1}^n \beta_j S_j(t) G_j(i(t)) \right. - \sum_{j=0}^n \int_0^1 \epsilon_j \beta_j S_j(t-r) G_j(i(t-r)) e^{-\mu r} f_{r_2}(r) dr$$

$$\left. - \sum_{j=0}^n \int_{-r}^0 \beta_j S_j(t-u) e^{-\mu t} f_{r_2}(u) du \right. - \mu I(t) - d_I I(t) + \alpha T I(t) dt,$$

$$dT(t) = \sum_{j=0}^n \int_0^{1/2} \left[ (1-\epsilon_j) \beta_j S_j(t-u) G_j(i(t-u)) e^{-\mu t} f_{r_2}(u) du$$

$$- \alpha T I(t) - \mu T T(t) - d_T T(t) - \alpha_A T(t) dt, \right.$$

$$dA(t) = \sum_{j=0}^n \int_0^{1/2} \left[ \epsilon_j \beta_j S_j(t-r) G_j(i(t-r)) e^{-\mu r} f_{r_1}(r) dr$$

$$- \mu A A(t) - d_A A(t) + \alpha T A(t) dt, \right.$$

$$dR(t) = [\gamma_0 S_0(t) H_0(Z(t)) - \mu R(t)] dt,$$

$$dZ(t) = [F_Z(I(t), T(t), A(t)) - \mu_Z Z(t)] dt.$$  \hfill (21)

It is easy to see that the multi-dimensional system (3)-(9) which lies on the phase space $\mathbb{R}^{n+6}_+$ is simply a decomposition of the composite system (21) lying on the finite dimensional phase space $\mathbb{R}^6_+$, with solution $(S(t), I(t), T(t), A(t), R(t), Z(t))^T \in \mathbb{R}^6_+$.

Thus, a more microscopic examination of the impacts of human behavior in the HIV/AIDS epidemic is obtained by studying the decomposed system (3)-(9), while a general examination of the aggregated human behavior in the HIV/AIDS epidemic is obtained by studying the finite dimensional system (21).
4. Model validation results. Applying standard techniques in [30, 36, 29, 32, 31], it is shown that there exists a unique positive solution that satisfies (3)-(9).

**Theorem 4.1.** Given the initial conditions (10)-(11), there exists a unique positive solution for the system (3)-(9). That is, \( S_j(t) > 0, I(t) > 0, T(t) > 0, A(t) > 0, R(t) > 0, \) and \( Z(t) > 0, t \geq t_0 \). Moreover, the solution of the system (3)-(9) lies in a positive self-invariant space \( \mathbb{B}_{\mathbb{R}^{n+6}}(0, r) \) in (27).

**Proof.** Observe from (3)-(9) and (20) that

\[
\frac{dN(t)}{dt} = \left[ B - \sum_{j=0}^{n} \mu S_j(t) - \mu_I I(t) - \mu_T T(t) - \mu_A A(t) - \mu_R R(t) \right] dt. \tag{22}
\]

When the solution of (3)-(9) is positive, i.e. \( Y(t) \in \mathbb{R}_+^{n+6} \), then it is easy to see from (20) that

\[
\limsup_{t \to \infty} N(t) \leq \frac{B}{\mu_{\text{min}}}, \tag{23}
\]

where

\[
\mu_{\text{min}} = \min \left( \sum_{j=0}^{n} \mu S_j, \mu_I, \mu_T, \mu_A, \mu_R \right). \tag{24}
\]

Furthermore, it is easy to see from (23), (17) and (9) that for \( Z(t_0) \in [0, \frac{1}{\mu_Z \min(1, \phi_{\text{max}}) \mu_{\text{min}}}] \),

\[
\limsup_{t \to \infty} Z(t) \leq \frac{1}{\mu_Z \min(1, \phi_{\text{min}}) \mu_{\text{min}}}, \tag{25}
\]

where \( \phi_{\text{max}} = \max(\phi_I, \phi_T, \phi_A) \), and \( \phi_{\text{min}} = \min(\hat{\phi}_I, \hat{\phi}_T, \hat{\phi}_A) \).

Thus, it follows from (23) and (25) that the closed ball centered at the origin with radius

\[
r = \frac{B}{\mu_{\text{min}}} + \frac{1}{\mu_Z \min(1, \phi_{\text{min}}) \mu_{\text{min}}}, \tag{26}
\]

defined as follows

\[
D(\infty) = \mathbb{B}_{\mathbb{R}^{n+6}}(0, r) = \{ Y(t) \in \mathbb{R}_+^{n+6} | N(t) + Z(t) = ||Y(t)||_1 \leq r \}, \tag{27}
\]
is positive self-invariant with respect to the system (3)-(9). \( \Box \)

**Remark 4.1.** Theorem 4.1 signifies that the system (3)-(9) always has a positive solution. Moreover, any trajectory of (3)-(9) that start in the closed ball \( \mathbb{B}_{\mathbb{R}^{n+6}}(0, r) \) on the phase space \( \mathbb{R}^{n+6} \), continues to oscillate in the closed ball, for all time. That is, the set \( \mathbb{B}_{\mathbb{R}^{n+6}}(0, r) \subset \mathbb{R}^{n+6} \) is positive self invariant with respect to the system (3)-(9). This result suggests that the maximum spread of the disease that starts in \( \mathbb{B}_{\mathbb{R}^{n+6}}(0, r) \) cannot exceed the bounds of the set \( \mathbb{B}_{\mathbb{R}^{n+6}}(0, r) \subset \mathbb{R}^{n+6} \) defined in (27).

5. Stability of the disease-free steady state in the multidimensional phase space \( \mathbb{R}^{n+6} \). In this section, the behavior of the trajectories of the system (3)-(9) in the neighborhood of a DFE in the multi-dimensional phase space \( \mathbb{R}^{n+6} \) are examined. The results of this section lead to an understanding of the importance of the human behavior change and ART in controlling the spread of HIV/AIDS.

Denote by \( E = X^* = (S_0^*, \ldots, S_n^*, I^*, T^*, Z^*)^T \) the steady states of the system (3)-(9).
Theorem 5.1. Let Theorem 4.1 hold. The DFE $E_0$ of the system (3)-(9) on the phase space $\mathbb{R}^{n+6}$ is given by
\[ E_0 = X_0^* = (S_0^*, \ldots, S_n^*, I^*, T^*, Z^*) = \left( \frac{B}{\mu S_0}, 0, \ldots, 0, 0, 0, 0 \right). \] (28)
Moreover the DFE $E_0$ lies in $\mathbb{R}^{n+6}(0, r) \subset \mathbb{R}^{n+6}$.

Proof. The proof of this result applies standard techniques of finding steady states of systems of differential equations (cf.\cite{30, 36, 32}).

The next result presents the basic reproduction numbers (BRN) of the family of deterministic models (3)-(9).

Theorem 5.2. Define
\[ R_1 = \beta_0 S_0^* \frac{1}{\mu_I + d_I}, \] (29)
\[ P_1 = \frac{1}{\epsilon_0 R_1 E[e^{-\mu_I t_1}](1 - \epsilon_0) R_1 E[e^{-\mu_I t_2}] + \frac{1}{C'(0)}}. \] (30)
The BRN of the deterministic model (3)-(9) is given by
\[ R_0 = R_1 P_1 = P_1 \beta_0 S_0^* \frac{1}{\mu_I + d_I}. \] (32)

Proof. The proof is easily obtained by applying the method of next generation matrix.

Remark 5.1. 1. The BRN in (32) is interpreted in the following. Indeed, given one infectious individual placed in the disease free population $E_0$, the term $\epsilon_0 R_1 E(e^{-\mu_I t_1})$ represents all newly infected individuals who fail to receive treatment and just turn into full-blown AIDS after the period $t_1$; the term $(1 - \epsilon_0) R_1 E(e^{-\mu_I t_2})$ represents the net number of newly infected individuals who remain in treatment (note that $\alpha_{TI}(1 - \epsilon_0) R_1 E(e^{-\mu_I t_2})$ represents all newly infected individuals who have either stopped treatment and currently returning to the infectious state, or those in whom treatment fails, and they are currently changing into full-blown AIDS); the term $\frac{1}{C'(0)}$ is the effect of nonlinearity in the incidence rate of the disease. Therefore, $P_1$ is approximately the probability of finding an infectious individual who would either become infectious, receive treatment and remain treated, or progress without treatment into full-blown AIDS.

Thus, the basic reproduction number $R_0$ in (32) is the average number of secondary infections in the complete disease free population $E_0$, that would proceed to the treatment class or to the full-blown AIDS class, over the average lifespan of $\frac{1}{\mu_I + d_I}$ of an infectious person in the population.

The following lemmas will be used to show the stability results for the DFE $E_0 = X_0^* = (S_0^*, \ldots, S_n^*, I^*, T^*, Z^*) = \left( \frac{B}{\mu S_0}, 0, \ldots, 0, 0, 0, 0 \right)$ of the system (3)-(9). The decoupled system with positive solution $X(t), t \geq t_0$ in (20) is utilized. Observe that the HIV positive states in $X(t)$ are $I$ and $T$. Also, observe from Assumptions 2.1&2.2 that for each $j \in I(0, n)$, $H_j$ and $G_j$ are continuous and bounded over
their domains. Moreover, denote by

$$H_j^* = \sup_{Z(t) \geq 0} H_j(Z(t)) \quad \text{and} \quad G_j^* = \max_{i(t) \in [0,1]} G_j(i(t)).$$

(33)

Clearly, from Assumption 2.1 (A5) and Assumption 2.2(A5), it is easy to see that for any \( t \geq t_0, \)

$$H_j(Z(t)) \leq \min(H_j^*, Z(t)), \forall Z(t) \geq 0 \quad \text{and} \quad G_j(i(t)) \leq \min(G_j^*, i(t)) \leq \min(G_j^*, 1),$$

\( \forall i(t) \in [0,1], \) and

$$G_j(i(t)) \leq i(t) \leq \min(1, I(t)), \forall I(t) \geq 0.$$  

(34)

**Lemma 5.1.** Let Theorem 4.1 hold, and define the \( C^{2,1} \)-function \( V : \mathbb{R}^{n+4}_+ \times \mathbb{R}_+ \to \mathbb{R}_+ \), where

$$V(t) = V_1(t) + V_2(t) + V_3(t) + V_4(t),$$

(35)

and

$$V_1(t) = (S_0(t) - S_j^0)^2, \quad V_2(t) = \sum_{j=1}^{n} S_j^2(t),$$

(36)

$$V_3(t) = (I(t) + T(t))^2, \quad V_4(t) = Z^2(t).$$

(37)

Also, let \( \phi_j, j \in I(0, n) \) and \( \varphi_k, k \in \{I, T, Z\} \) be defined as follows:

$$\phi_0 = 2\mu S_0 - \left[ \sum_{j=0}^{n} \gamma_j (1 + (H_j^*)^2) + \sum_{j=0}^{n} \gamma_j S_j^* + \beta_0 S_0^* + 2\beta_0 + 2\beta_0 \frac{1}{\lambda(\mu)} \right],$$

(38)

$$\phi_j = 2\mu S_j - \left[ \gamma_j (1 + (H_j^*)^2) + \gamma_j S_j^* + 2\beta_j + 2\beta_j \frac{1}{\lambda(\mu)} \right], j \in I(1, n),$$

(39)

and

$$\varphi_I = (1 - \frac{1}{2} \lambda(\mu))(\mu_I + d_I) - \left[ \beta_0 S_0^* (1 + 2\frac{1}{\lambda(\mu)} + \lambda(\mu)) \right]$$

$$+ (1 - \frac{1}{2} \lambda(\mu))(\mu_I + d_I)$$

$$- \left[ \sum_{j=0}^{n} (1 + \varepsilon_j)\beta_j \lambda(\mu) + \phi_I \lambda(\mu) + (\mu_T + d_T + \alpha_T - \frac{1}{\lambda(\mu)}) \right].$$

(40)

$$\varphi_T = (2 - \lambda(\mu))(\mu_T + d_T + \alpha_T - \frac{1}{\lambda(\mu)})$$

$$- \left[ \sum_{j=0}^{n} (1 + \varepsilon_j)\beta_j \lambda(\mu) + \beta_0 S_0^* \lambda(\mu) + \phi_T \lambda(\mu) + (\mu_I + d_I) \frac{1}{\lambda(\mu)} \right].$$

(41)

$$\varphi_Z = 2\mu Z - \left[ \gamma_0 S_0^* + \sum_{j=0}^{n} \gamma_j S_j^* + (\phi_I + \phi_T) \frac{1}{\lambda(\mu)} \right].$$

(42)
The differential operator $\dot{V}$ applied to $V(t)$ with respect to the system (3)-(9) satisfies the following:

$$
\dot{V}(t) \leq - \left( \phi_0 (S_0(t) - S_0^*)^2 + \sum_{j=1}^{n} \phi_j S_j^2(t) + \phi_T I^2(t) + \varphi_T T^2(t) + \varphi_Z Z^2(t) \right) + \sum_{j=0}^{n} \left( 2 \varepsilon \beta_j \frac{1}{\lambda(\mu)} \right) \mathbb{E}_{\tau_1} \left[ S_j^2(t - \tau_1) G_j^2(i(t - \tau_1)) e^{-2\mu_T \tau_1} \right],
$$

(43)

where $\mathbb{E}_{\tau_1}$ is the expectation operator with respect to the random variable $\tau_1$.

**Proof.** It is easy to see that the differential operator applied to $V_1$ with respect to (3)-(9), leads to

$$
\dot{V}_1(t) = - \sum_{j=1}^{n} 2 \gamma_j (S_0(t) - S_0^*)^2 H_j(Z(t)) - 2 \gamma_0 (S_0(t) - S_0^*)^2 H_0(Z(t)) - 2 \gamma_0 (S_0(t) - S_0^*)^2 H_0(Z(t)) - 2 \gamma_0 (S_0(t) - S_0^*)^2 H_0(Z(t)) - 2 \gamma_0 (S_0(t) - S_0^*)^2 H_0(Z(t)) - 2 \gamma_0 (S_0(t) - S_0^*)^2 H_0(Z(t)) - 2 \gamma_0 (S_0(t) - S_0^*)^2 H_0(Z(t)) - 2 \gamma_0 (S_0(t) - S_0^*)^2 H_0(Z(t)).
$$

(44)

Similarly,

$$
\dot{V}_2(t) = \sum_{j=1}^{n} \left[ 2 \gamma_j S_j(t)(S_0(t) - S_0^*) H_j(Z(t)) + 2 \gamma_j S_j(t) S_0^* H_j(Z(t)) - 2 \gamma_j S_j^2(t) G_j(i(t)) - 2 \mu S_j S_j^2(t) G_j(i(t)) \right].
$$

(45)

Also,

$$
\dot{V}_3(t) = 2 \beta_0 G_0(i(t)) I(t) S_0(t) + 2 \beta_0 G_0(i(t)) T(t) S_0(t) + \sum_{j=1}^{n} [2 \beta_j G_j(i(t)) I(t) S_j(t) + 2 \beta_j G_j(i(t)) T(t) S_j(t)]
$$

$$
+ \sum_{j=0}^{n} \left[ - 2 \varepsilon \beta_j I(t) \mathbb{E}_{\tau_1} \left[ S_j(t - \tau_1) G_j(i(t - \tau_1)) e^{-\mu_T \tau_2} \right] - 2 \varepsilon \beta_j T(t) \mathbb{E}_{\tau_1} \left[ S_j(t - \tau_1) G_j(i(t - \tau_1)) e^{-\mu_T \tau_2} \right] - 2 (\mu_T + d_I) I^2(t) - 2 (\mu_T + d_I) T(t) I(t) - 2 (\mu_T + d_I + \alpha_T A) I(t) T(t)
$$

$$
- 2 (\mu_T + d_T + \alpha_T A) T^2(t).
$$

(46)

And

$$
\dot{V}_4 = [2Z(t)F_Z(I, T) - 2 \mu Z^2(t)], \quad F_Z(I, T) = \frac{\phi_I I(t) + \phi_T T(t)}{1 + \phi_I I(t) + \phi_T T(t)}.
$$

(47)

In the following, Cauchy-Schwartz inequality, Holder inequality, $(a+b)^2 \leq 2a^2 + 2b^2$, and the following algebraic inequality (48) will be used to obtained the results.

$$
2ab \leq \frac{a^2}{g(c)} + \frac{b^2}{g(c)}, g(c) > 0.
$$

(48)
Indeed, it follows from (44) and (45) that
\[ 
\dot{V}_1(t) + \dot{V}_2(t) \leq \\
\left[ -\sum_{j=1}^{n} 2\gamma_j H_j(Z(t)) - 2\gamma_0 H_0(Z(t)) + \sum_{j=1}^{n} \gamma_j S_0^j + \beta_0 S_0^j + \gamma_0 S_0^j - 2\beta_0 G_0(i(t)) \\
-2\mu S_j](S_0(t) - S_0^j)^2 + \sum_{j=1}^{n} [\gamma_j H_j(Z(t)) + \gamma_j S_0^j - 2\beta_j G_j(i(t)) - 2\mu S_j] S_0^j(t) \\
+ \sum_{j=1}^{n} 2\gamma_0 S_0^j H_j^2(Z(t)) + \gamma_0 S_0^j H_0^2(Z(t)) + \beta_0 S_0^j G_0^2(i(t)). \right] 
\]

Also, it follows from (46) that
\[ 
\dot{V}_3 \leq 2\beta_0 \frac{1}{\lambda(\mu)} (S_0(t) - S_0^j)^2 + \sum_{j=1}^{n} 2\beta_j \frac{1}{\lambda(\mu)} S_0^j(t) \\
+ \left[ \beta_0 \lambda(\mu) + \beta_0 S_0^j \frac{1}{\lambda(\mu)} + \beta_0 S_0^j \lambda(\mu) + \beta_0 S_0^j \frac{1}{\lambda(\mu)} + \sum_{j=1}^{n} \beta_j \lambda(\mu) \\
+ \sum_{j=0}^{n} \varepsilon_j \beta_j \lambda(\mu) + (\mu T + d_1) \lambda_2(\mu) + (\mu T + d_T + \alpha T) \lambda(\mu) \frac{1}{\lambda_2(\mu)} \\
-2(\mu T + d_1)] I^2(t) \\
+ \left[ \beta_0 \lambda(\mu) + \beta_0 S_0^j + \lambda(\mu) + \sum_{j=1}^{n} \beta_j \lambda(\mu) + \sum_{j=0}^{n} \varepsilon_j \beta_j \lambda(\mu) \\
+ (\mu T + d_1) \frac{1}{\lambda_2(\mu)} + (\mu T + d_T + \alpha T) \lambda_2(\mu) - 2(\mu T + d_T + \alpha T) \lambda(\mu) \right] T^2(t) \\
+ \sum_{j=0}^{n} \left( 2\varepsilon_j \beta_j \frac{1}{\lambda(\mu)} \right) E_{\tau_1} \left[ S_j^2 (t - \tau_1) G^2 (i(t - \tau_1)) e^{-2\mu T \tau_1} \right], \]
\]
where \(\lambda(\mu)\) and \(\lambda_2(\mu)\) are positive real valued functions of \(\mu\).

Also, from (47), it is easy to see that
\[ 
\dot{V}_4(t) = \left[ \frac{2\phi T Z(t) I(t)}{1 + \phi I(t) + \phi T^2(t)} + \frac{2\phi T Z(t) I(t)}{1 + \phi I(t) + \phi T^2(t)} - 2\mu Z^2(t) \right] \\
\leq \left[ \phi T \lambda(\mu) I^2(t) + \phi T \lambda(\mu) T^2(t) + \left( (\phi T + \phi T) \frac{1}{\lambda(\mu)} - 2\mu Z \right) Z^2(t) \right]. 
\]

From (49) and (34), observe that for each \(j \in I(0, n)\), the terms \(-2\gamma_j H_j(Z(t)) \leq \gamma_j \left( 1 + (H_j)^2 \right), \gamma_0 S_0^j H_j^2(Z(t)) \leq \gamma_0 S_0^j Z^2(t), \) and \(\beta_0 S_0^j G_0^2(i(t)) \leq \beta_0 S_0^j(i(t))^2 \leq \beta_0 S_0^j I^2(t)\). Now, combining (44)-(51) and grouping like-terms, and also applying (34) and some further algebraic manipulations and simplifications, it is easy to see
that
\[
\dot{V}(t) = \dot{V}_1(t) + \dot{V}_2(t) + \dot{V}_3(t) + \dot{V}_4(t)
\]
\[
\leq -\left(\phi_0(S_0(t) - S_0^*)^2 + \sum_{j=1}^{n} \phi_j S_j^2(t) + \varphi_I I^2(t) + \varphi_T T^2(t) + \varphi_Z Z^2(t)\right)
\]
\[
+ \sum_{j=0}^{n} \left(2\varepsilon_j \beta_j \frac{1}{\lambda(\mu)} \right) \mathbb{E}_r \left[ S_j^2(t) (t - \tau_1) G_j^2(i(t - \tau_1)) e^{-2\mu_I \tau_1} \right], \tag{52}
\]
where \(\phi_j, j \in I(0, n)\) and \(\varphi_k, k \in \{I, T, Z\}\) are defined in (38)-(42).

\[\square\]

Remark 5.2. (1.) Recall Theorems 28&4.1 assert that the DFE exists in \(D(\infty) \subset \mathbb{R}_{+}^{n+6}\). Furthermore, the delay-term in (52), \(\sum_{j=0}^{n} \left(2\varepsilon_j \beta_j \frac{1}{\lambda(\mu)} \right) \mathbb{E}_r \left[ S_j^2(t - \tau_1) G_j^2(i(t - \tau_1)) e^{-2\mu_I \tau_1} \right]\) is estimated in \(D(\infty) \subset \mathbb{R}_{+}^{n+6}\) in a manner that conveys meaningful interpretation of the HIV/AIDS disease dynamics in the space.

Indeed, in \(D(\infty)\), using (26) and (34), we estimate
\[
\sum_{j=0}^{n} \left(2\varepsilon_j \beta_j \frac{1}{\lambda(\mu)} \right) \mathbb{E}_r \left[ e^{-2\mu_I \tau_1} S_j^2(t) G_j^2(i(t)) \right]
\]
\[
\leq \sum_{j=0}^{n} \left(2\varepsilon_j \beta_j \frac{1}{\lambda(\mu)} \right) \mathbb{E}_r \left[ e^{-2\mu_I \tau_1} r^2(i(t)) \right]
\]
\[
\leq \sum_{j=0}^{n} \left(2\varepsilon_j \beta_j \frac{1}{\lambda(\mu)} \right) \mathbb{E}_r \left[ e^{-2\mu_I \tau_1} r^2(I(t)) \right], \tag{53}
\]
where \(r\) is defined in (26).

In the next subsection, we examine the stability of the DFE \(E_0\) in \(D(\infty) \subset \mathbb{R}_{+}^{n+6}\). The next result presents conditions for global stability of the DFE \(E_0\), when the basic reproduction number \(R_0 < 1\).

Lemma 5.2. Let the assumptions for Lemma 5.1 be satisfied and let \(R_0\) be defined in (32). Also, let Lemma 5.1 hold. Then the DFE, given by (28), for the system (3)-(9) exists in \(D(\infty)\). Moreover, in \(D(\infty)\), let \(V(t)\) be as defined in (35) and define the functional
\[
V_5(t) = \sum_{j=0}^{n} \left(2\varepsilon_j \beta_j \frac{1}{\lambda(\mu)} \right) \mathbb{E}_r \left[ \int_{0}^{t} S_j^2(\theta) G^2(i(\theta)) e^{-2\mu_I \tau_1} d\theta \right]. \tag{54}
\]

Also define
\[
R_1(\mu) = \sum_{j=0}^{n} \left(1 + \varepsilon_j \beta_j \lambda(\mu) + \phi_I \lambda(\mu) + (\mu_T + dr + \alpha_T A) \frac{1}{\lambda_2(\mu)} + r^2 \mathbb{E}_r \left[ e^{-2\mu_I \tau_1} \right] \right) \sum_{j=0}^{n} 2\varepsilon_j \beta_j \frac{1}{\lambda(\mu)} \left(\frac{1}{1 - \frac{1}{2} \lambda_2(\mu)} \right) \tag{55}
\]

Furthermore, let
\[
U_0 = \sum_{j=0}^{n} \gamma_j \left(1 + (H_j^*)^2\right) + \sum_{j=0}^{n} \gamma_j S_j^0 + \beta_0 S_0^* + 2\beta_0 + 2\beta_0 \frac{1}{\lambda(\mu)} \tag{56}
\]
and for each \( j \in I(1,n) \),

\[
U_j = \frac{1}{2\mu_{S_j}} \left[ \gamma_j \left( 1 + (H_j^*)^2 \right) + \gamma_j S^*_0 + 2\beta_j + 2\beta_j \frac{1}{\lambda(\mu)} \right].
\]

(57)

Define

\[
V_0 = \frac{1}{(2 - \lambda_2(\mu)) (\mu_T + d_T + \alpha T_A)} \left[ \sum_{j=0}^n (1 + \varepsilon_j) \beta_j \lambda(\mu) + \beta_0 S^*_0 \lambda(\mu) + \phi_T \lambda(\mu) + (\mu_I + d_I) \frac{1}{\lambda(\mu)} \right],
\]

and

\[
W_0 = \frac{1}{2\mu_Z} \left[ \gamma_0 S^*_0 + 2 \sum_{j=1}^n \gamma_j S^*_j + (\phi_I + \phi_T) \frac{1}{\lambda(\mu)} \right].
\]

(58)

(59)

It follows that in \( D(\infty) \), there exists positive constant \( m > 0 \), such that

\[
(\dot{V} + \dot{V}_0)(t) \leq -m||X(t) - E_0||^2_{R^n_{+6}}
\]

(60)

whenever \( R_0 < 1 \), \( R_1(\mu) < 1 + (1 - R_0) \), \( \max \{ U_j \}_{j \in I(0,n)} < 1 \), and \( \max \{ V_0, W_0 \} < 1 \), where the vector \( X(t) \) is defined in (20) and \( ||.||_{R^n_{+6}} \) is the natural Euclidean norm in \( R^n_{+6} \).

Proof. Let \( R_0 < 1 \), \( R_1(\mu) < 1 + (1 - R_0) \), \( \max \{ U_j \}_{j \in I(0,n)} < 1 \), and \( \max \{ V_0, W_0 \} < 1 \). Observe from (54), (43) and (53) that

\[
\dot{V}(t) + \dot{V}_0(t) \leq - \left( \phi_0(S_0(t) - S_0^*)^2 + \sum_{j=1}^n \phi_j S^*_j(t) + \phi_I I^2(t) + \varphi_T T^2(t) + \varphi_Z Z^2(t) \right)
\]

\[
+ \sum_{j=0}^n \left( 2\varepsilon_j \beta_j \frac{1}{\lambda(\mu)} \right) E_{r_{11}} \left[ (S^*_j(t)C^*_j(i(t))e^{-2\mu r_{11}}) \right]
\]

\[
\leq - \left( \phi_0(S_0(t) - S_0^*)^2 + \sum_{j=1}^n \phi_j S^*_j(t) + \phi_I I^2(t) + \varphi_T T^2(t) + \varphi_Z Z^2(t) \right)
\]

\[
+ \sum_{j=0}^n \left( 2\varepsilon_j \beta_j \frac{1}{\lambda(\mu)} \right) E_{r_{11}} \left[ e^{-2\mu r_{11}} \right] r^2(I(t))^2
\]

\[
= - \left( \phi_0(S_0(t) - S_0^*)^2 + \sum_{j=1}^n \phi_j S^*_j(t) + \phi_I I^2(t) + \varphi_T T^2(t) + \varphi_Z Z^2(t) \right).
\]

(61)

where \( \phi_j, j \in I(0,n) \) and \( \varphi_k, k \in \{ I, T, Z \} \) are given in (38)-(42). Moreover, it is easy to see from (38)-(42) and (61) that

\[
\phi_0 = 2\mu_{S_0} - \left[ \sum_{j=0}^n \gamma_j (1 + (H_j^*)^2) + \sum_{j=0}^n \gamma_j S^*_0 + \beta_0 S^*_0 + 2\beta_0 + 2\beta_0 \frac{1}{\lambda(\mu)} \right]
\]

\[
= 2\mu_{S_0} (1 - U_0) > 0
\]

(62)
\[
\phi_j = 2\mu_{S_j} - \left[ \gamma_j (1 + (H_j^*)^2) + \gamma_j S_0 + 2\beta_j + 2\beta_j \frac{1}{\lambda(\mu)} + \sigma_\lambda^2 \right], \quad j \in I(1, n)
\]

\[
= 2\mu_{S_j} [1 - U_j] > 0
\]

and

\[
\varphi_{I, 1} = \varphi_I - \sum_{j=0}^n \left( 2\varepsilon_j \beta_j \frac{1}{\lambda(\mu)} \right) E_{r_1} [e^{-2\mu_{Ib_1}}] r^2
\]

\[
= \left( 1 - \frac{1}{2} \lambda_2(\mu) \right) (\mu_I + d_I) - \left[ \beta_0 S^*_0 (1 + 2 \frac{1}{\lambda(\mu)} + \lambda(\mu)) \right] + \left( 1 - \frac{1}{2} \lambda_2(\mu) \right) (\mu_I + d_I)
\]

\[
- \sum_{j=0}^n \left( 1 + \varepsilon_j \right) \beta_j \lambda(\mu) + \phi_I \lambda(\mu) + (\mu_T + d_T + \alpha_{T, 2}) \frac{1}{\lambda_2(\mu)}
\]

\[
- \sum_{j=0}^n \left( 2\varepsilon_j \beta_j \frac{1}{\lambda(\mu)} \right) E_{r_1} [e^{-2\mu_{Ib_1}}] r^2
\]

\[
= \left( 1 - \frac{1}{2} \lambda_2(\mu) \right) (\mu_I + d_I) \left[ 1 - \frac{\beta_0 S^*_0}{(\mu_I + d_I)} \left( \frac{1 + 2 \frac{1}{\lambda(\mu)} + \lambda(\mu)}{1 - \frac{1}{2} \lambda_2(\mu)} \right) \right]
\]

\[
+ \left( 1 - \frac{1}{2} \lambda_2(\mu) \right) (\mu_I + d_I) [1 - R_1(\mu)].
\]

Select the functions \( \lambda(\mu) \) and \( \lambda_2(\mu) \) such that in (64) and (30)-(32), \( P_1 \) is proportional to \( \frac{[(1 + 2 \frac{1}{\lambda(\mu)} + \lambda(\mu))]}{(1 - \frac{1}{2} \lambda_2(\mu))} \). That is,

\[
P_1 = \frac{1}{\left[ \varepsilon_0 R_1 E(e^{-\mu_{Ib_1}}) + (1 - \varepsilon_0) R_1 E(e^{-\mu_{Tb_2}}) - \alpha_{T, 2} (1 - \varepsilon_0) R_1 E(e^{-\mu_{Tb_2}}) \frac{1}{\lambda_2(\mu)} + \frac{1}{\lambda_2(\mu)} \right]}
\]

\[
= K \left[ 1 + 2 \frac{1}{\lambda(\mu)} + \lambda(\mu) \right]
\]

\[
= \frac{K}{(1 - \frac{1}{2} \lambda_2(\mu))},
\]

where

\[
0 < K < \frac{P_1 (1 - \frac{1}{2} \lambda_2(\mu))}{1 + 2\sqrt{2}}.
\]

and \( \lambda(\mu) \) satisfies

\[
K \lambda^2(\mu) - \lambda(\mu) \left[ P_1 (1 - \frac{1}{2} \lambda_2(\mu)) - K \right] + 2K = 0.
\]

Note that when \( \lambda_2(\mu) \in (0, 2) \) is given, then the solutions of (67) are real and positive. Also, clearly from (64) and (30)-(32),

\[
R_0 = P_1 \frac{\beta_0 S^*_0}{(\mu_I + d_I)} = K \frac{1 + 2 \frac{1}{\lambda(\mu)} + \lambda(\mu)}{(1 - \frac{1}{2} \lambda_2(\mu))} \frac{\beta_0 S^*_0}{(\mu_I + d_I)}.
\]

It is easy to see from (64) that

\[
\varphi_{I, 1} = \left( 1 - \frac{1}{2} \lambda_2(\mu) \right) (\mu_I + d_I) \left[ (1 - R_0) + (1 - R_1(\mu)) \right] > 0
\]

\[
\varphi_T = (2 - \lambda_2(\mu)) (\mu_T + d_T + \alpha_{T, 2})
\]

\[
- \left[ \sum_{j=0}^n (1 + \varepsilon_j) \beta_j \lambda(\mu) + \beta_0 S^*_0 \lambda(\mu) + \phi_T \lambda(\mu) + (\mu_I + d_I) \frac{1}{\lambda_2(\mu)} \right]
\]

\[
= (2 - \lambda_2(\mu)) (\mu_T + d_T + \alpha_{T, 2}) [1 - V_0] > 0.
\]
\[ \varphi_Z = 2\mu_Z - \gamma_0 S_0^* + \sum_{j=0}^{n} \gamma_j S_0^* + (\phi_I + \phi_T) \frac{1}{\lambda(\mu)} \]

\[ = 2\mu_Z [1 - W_0] > 0. \] (71)

Thus, it follows from (61) that

\[ (\dot{V} + \dot{V}_5)(t) \leq -m||X(t) - E_0||_{R_+^{n+6}}^2, \] (72)

where

\[ m = \min_{j \in I(1,n)} (\phi_0, \phi_j, \varphi_{I,1}, \varphi_T, \varphi_Z) > 0. \] (73)

**Theorem 5.3.** Suppose the conditions of Lemma 5.2 are satisfied, then it follows that the DFE \( E_0 \) of the system (3)-(9) is uniformly, globally asymptotically stable in the region \( D(\infty) \) of the phase space \( R_+^{n+6} \). That is, the disease is eliminated from the population asymptotically, whenever the conditions \( R_0 < 1, R_1(\mu) < 1 + (1 - R_0) \), max \( (U_j)_{j \in I(0,n)} < 1 \), and max \( (V_0, W_0) < 1 \) in Lemma 5.2 are satisfied.

**Proof.** The result follows trivially by applying comparable theorems for stability using Lyapunov second method (cf.[30, 36, 29, 32, 35]). \( \square \)

### 5.1. Stability of the DFE on the finite dimensional phase space \( R_+^4 \).

Recall (28) asserts that the DFE for all susceptible states \( S_j, j \in I(1,n) \) are 0, except for the DFE of \( S_0 \) which is given by \( \frac{B}{\mu S_0} \). Moreover, the Lemma 5.2 and Theorem 5.3 assert that all trajectories of the susceptible states \( S_j, j \in I(0,n) \) that start near the DFE \( E_0 \), remain near and converge asymptotically to the DFE \( E_0 \) in the multidimensional subspace \( D(\infty) \subset R_+^{4} \).

Since asymptotically nearly all of the susceptible states \( S_j, j \in I(1,n) \) of individuals who have responded positively to the IECs, and modified their sexual behaviors become 0 (see Definition 2.2), except for the state \( S_0 \) of susceptible individuals who have not yet modified their sexual behaviors, it is easy to misinterpret the result of Theorem 5.3 as suggesting that the IECs lead to no apparent gain in establishing a significant sexually educated stable positive disease-free population asymptotically. Such misinterpretations of Theorem 5.3 are dispelled by examining the trajectories of the total susceptible state \( S(t) = \sum_{j=0}^{n} S_j(t) \) in the composite system (21).

It is easy to see from (21) that the DFE of \( X_1(t) = (S(t) = \sum_{j=0}^{n} S_j(t), I(t), T(t), Z(t)) \) is \( (\frac{B}{\mu S_0}, 0, 0, 0) \). However, note that it is not obvious that the combined susceptible state \( S(t) = \sum_{j=0}^{n} S_j(t) \) converges to the corresponding DFE \( \frac{B}{\mu S_0} \), since the trajectories of \( S(t) = \sum_{j=0}^{n} S_j(t) \) exist on a different phase space \( R_+^{4} \), than exhibited in Theorem 5.3. Thus, it is necessary to show that the trajectories of \( S(t) = \sum_{j=0}^{n} S_j(t) \) that start near the DFE \( S^* = \sum_{j=0}^{n} S_j^*(t) = \frac{B}{\mu S_0} \), remain near \( S^* = \sum_{j=0}^{n} S_j^*(t) = \frac{B}{\mu S_0} \) for all time; they never completely die out over time or grow arbitrarily beyond \( S_0^* = \frac{B}{\mu S_0} \); they also converge asymptotically to \( S_0^* = \frac{B}{\mu S_0} \).

Let \( ||.||_{R_+^{n+6}} \) and \( ||.||_{R_+^{4}} \) be the natural Euclidean norms on \( R_+^{n+6} \) and \( R_+^{4} \), respectively. On the finite dimensional phase space \( R_+^{4} \), the solution of the decoupled system (3)-(9), obtained by combining the equations for \( S_j, j \in I(0,n) \), is given by
the vector $X_1(t) = (S(t), I(t), T(t), Z(t))^T \in D_1(\infty) \subset \mathbb{R}_+^4$, where similarly to (27)

$$D_1(\infty) = \mathbb{B}_{\mathbb{R}_+^4}(0, r) = \left\{ X_1(t) \in \mathbb{R}_+^4 : \|X_1(t)\|_{\mathbb{R}_+^4} \leq r \right\},$$

is the corresponding positive self invariant space for the reduced system (21) on the phase space $\mathbb{R}_+^4$. Moreover, $r$ is defined in (26). Also, the DFE in (28) denoted by $E_0 = X_0^* = (S_0^*, 0, 0, 0, 0) \in \mathbb{R}_+^{n+4}$, corresponds to the DFE $E_{10} = X_{10}^* = (S^*, I^*, T^*, Z^*) = (\frac{B}{\mu S_0}, 0, 0, 0) \in \mathbb{R}_+^4$ for the system (21), where $S^* = \sum_{j=0}^n S_j^* = \frac{\mu}{\mu S_0}$.

**Theorem 5.4.** Suppose the conditions of Lemma 5.2 and Theorem 5.3 are satisfied. That is, $R_0 < 0$, $R_1(\mu) < 1 + (1 - R_0)$, max$(U_j)_{j \in \ell(0, n)} < 1$, and max$(V_0, W_0) < 1$ in Lemma 5.2 are satisfied. And let $S(t) = \sum_{j=0}^n S_j(t)$. Also, let $V(t)$ be as defined in (35) and define the functional $V_2(t)$ as in (54). There exists a positive real number $m_0 > 0$ such that the differential operator $\dot{V}$ applied to $(V + V_2)(t)$ with respect to the system (3)-(9) satisfies

$$\dot{V} + \dot{V}_2(t) \leq -m_0 \left( \frac{1}{(n+1)} \right) \|X_1(t) - E_{10}\|^2_{\mathbb{R}_+^4}.$$

That is, the DFE $E_{10} = X_{10}^* = (S^*, I^*, T^*, Z^*) = (\frac{B}{\mu S_0}, 0, 0, 0) \in \mathbb{R}_+^4$ is globally uniformly asymptotically stable in $D_1(\infty)$, and all trajectories of the combined susceptible state $S(t) = \sum_{j=0}^n S_j(t)$ converge asymptotically to the DFE $S^* = \sum_{j=0}^n S_j^* = \frac{\mu}{\mu S_0}$.

**Proof.** It is easy to see from (61) that

$$\dot{V} + \dot{V}_2(t) \leq - \left( \phi_0(S_0(t) - S_0^2) + \sum_{j=1}^n \phi_j S_j^2(t) + \varphi_{1,1} I^2(t) + \varphi_T T^2(t) + \varphi_Z Z^2(t) \right)$$

$$\leq -m_0 \|X(t) - E_0\|^2_{\mathbb{R}_+^{n+4}}$$

$$\leq -m_0 \left( \frac{1}{(n+1)} \right) \left( \sum_{j=0}^n S_j(t) - S_0^* \right)^2 + I^2(t) + T^2(t) + Z^2(t)$$

$$= -m_0 \left( \frac{1}{(n+1)} \right) \|X_1(t) - E_{10}\|^2_{\mathbb{R}_+^4},$$

where

$$m_0 = \min_{j \in \ell(1,n)} (\phi_0, \phi_j, \varphi_{1,1}, \varphi_T, \varphi_Z) > 0.$$

The stability results follow from comparable theorems for stability using Lyapunov second method (cf.[30, 36, 29, 32, 35]).

**Remark 5.3.** Disease eradication conditions on the phase spaces $\mathbb{R}_+^{n+4}$ and $\mathbb{R}_+^4$

Theorem 5.3 and Lemma 5.2 signify that when the conditions of Lemma 5.2 hold, then all trajectories of the system (3)-(9) on the multidimensional phase space $\mathbb{R}_+^{n+4}$ originate, oscillate and remain bounded in the closed ball $D(\infty) \subset \mathbb{R}_+^{n+4}$, where $D(\infty)$ is given in (27). Moreover, there exists a DFE $E_0 \in D(\infty) \subset \mathbb{R}_+^{n+4}$ for the system, and any trajectory that starts near $E_0$, continues oscillating near $E_0$, and over time, converges to $E_0$. \qed
Biologically, these results suggest that the maximum spread of the disease on \( \mathbb{R}^{n+4}_+ \) can not exceed the bounds of \( D(\infty) \). Furthermore, the disease is eliminated, as all disease-related states \( I, T \) and \( D \) of the decoupled system (3)-(9) die out asymptotically. And this disease eradication occurs, whenever the BRN is taken to determine the effects of the supply of ODAs, delayed ART treatment, and withdrawal from treatment on the BRN. The sensitivity of the BRN to random fluctuations and noises in the HIV/AIDS disease eradication. This would imply that the IECs effects are limited in controlling the disease.

Contrary to logic, it appears that, on the multidimensional phase space \( D(\infty) \subset \mathbb{R}^{n+4}_+ \), only susceptible individuals of category \( S \) of magnitude \( S_0 = \frac{B}{\mu S_0} \), who have not yet reformed their sexual behaviors due to the IECs remain over time, whenever the disease is eradicated. This would imply that the IECs effects are limited in controlling the disease.

This misconception is corrected in Theorem 5.4 that asserts that, on the finite dimensional phase space \( D_1(\infty) \subset \mathbb{R}^4_+ \), the trajectories of the combined susceptible state \( S(t) = \sum_{j=0}^{n} S_j(t) \) converge asymptotically to the DFE \( S^* = \sum_{j=0}^{n} S_j^*(t) = \frac{B}{\mu S_0} \), whenever the conditions \( R_1(\mu) < 1 + (1 - R_0), \max(U_j)_{j\in I(0,n)} < 1 \), and \( \max(V_0, W_0) < 1 \) in Theorem 5.3 and Lemma 5.2 are satisfied. In other words, the susceptible population that remains asymptotically whenever the disease is eradicated is a combination \( S^* = \sum_{j=0}^{n} S_j^*(t) = \frac{B}{\mu S_0} \) of susceptible individuals of all categories \( j \in I(0,n) \) in Definitions 2.2.

6. Discussion of results. In this section, a more critical look at the BRN \( R_0 \) in (32) is taken to determine the effects of the supply of ODAs, delayed ART treatment, and withdrawal from treatment on the BRN \( R_0 \), in order to find conditions for disease eradication. The reader is directed to the studies \([33, 34]\) for more results on the sensitivity of the BRN (32) to random fluctuations and noises in the HIV/AIDS disease dynamics.

Remark 6.1. Effects of the supply of ODAs, delayed ART treatment and relapse from treatment on disease eradication: Recall Definitions 2.1-2.7, the effects of the supply of ODAs, IECs and delayed ART treatment are assessed via the parameters \( \varepsilon_j, \tilde{\varepsilon}_j, j \in I(0,n), \gamma_j \equiv \gamma S_0 S_j, j \in I(1,n) \), and \( \tau_2 \), respectively. We examine the effects of continuous changes in these parameters on the BRN in (32).

Clearly, from (30)-(32) the magnitude of the probability term \( P_j \) determines the magnitude of the BRN \( R_0 \). Furthermore,

\[
\frac{\partial P_j}{\partial \varepsilon_0} = (-1) \frac{(a_2 - a_1 - a_3)}{(a_1 + \varepsilon_0(a_2 - a_1 - a_3) + \frac{1}{G'(0)})^2},
\]

where

\[
a_1 = R_1 E(e^{-\mu_1 \tau_1}), \quad a_2 = R_1 E(e^{-\mu_1 \tau_2}),
\]

\[
a_3 = \alpha_{TI} R_1 E(e^{-\mu_1 \tau_2}) \frac{1}{(\alpha_{TI} + \mu_T + d_T + \alpha_{TA})}.
\]

It is easy to see that \( \frac{\partial P_j}{\partial \varepsilon_0} < 0 \), if

\[
E(e^{-\mu_1 \tau_2}) \left( 1 - \frac{1}{(\alpha_{TI} + \mu_T + d_T + \alpha_{TA})} \right) > E(e^{-\mu_1 \tau_1}),
\]

and \( \frac{\partial P_j}{\partial \varepsilon_0} > 0 \), otherwise.
That is, when (80) holds, then $P_1$ continuously decreases to zero as $\bar{\varepsilon}_0 \to 1$, and as a result the BRN $R_0$ continuously decreases to zero, and vice versa. This fact is exhibited in Figure 3.

From (80), the left-hand side (LHS), $E(e^{-\mu \tau_2}) \left(1 - \alpha_{TI} \frac{1}{\alpha_{TI} + \mu_T + d_T + \alpha_{TA}}\right)$, is the expected conditional probability that a newly exposed HIV person, survives natural death (with probability $E(e^{-\mu \tau_2})$) and remains in ART treatment without relapsing to infectiousness (with probability $\left(1 - \alpha_{TI} \frac{1}{\alpha_{TI} + \mu_T + d_T + \alpha_{TA}}\right)$), given that the infected person is tested and begins ART treatment after $\tau_2$ time units. That is, the LHS is the expected probability that an individual infected with HIV will remain healthy, and alive during the ART treatment.

The right-hand side (RHS) term, $E(e^{-\mu \tau_1})$, of (80) is clearly the expected conditional probability that an exposed person will progress into full-blown AIDS after $\tau_1$ time units, given that the person fails to get ART treatment.

Thus, (78)-(80) suggest that when ODAs are readily available, and more people tend to get ART treatment (i.e. $\bar{\varepsilon}_0 \to 1$), then the BRN $R_0 << 1$ and the disease is more easily eliminated, since.

Also, observe from (80) that when either $\tau_2$ is small (i.e. $\tau_2 \to 0$), or $\alpha_{TI}$ is small (i.e. $0 < \alpha_{TI} << 1$), then $LHS >> RHS$, assuming all other constants are fixed. This implies that when either $\tau_2 \to 0$, or $0 < \alpha_{TI} << 1$, then $P_1 \to 0$, and consequently $R_0 << 1$. In other words, when newly infected people get tested soon after infection, and begin early ART treatment (i.e. $\tau_2 \to 0$), then increasing the supply of ODAs (i.e. $\bar{\varepsilon}_0 \to 1$) will result in more people getting tested and paying for ART treatment, which makes the disease eradication process easier. This fact is illustrates in the example in Figure 4.

The alternative when $\alpha_{TI}$ is small and $R_0 << 1$, signifies that when fewer number of people who are receiving ART treatment relapse from treatment, and back into the infectious state, then increasing the supply of ODAs (i.e. $\bar{\varepsilon}_0 \to 1$), so that more people get tested and pay for ART treatment, will make the disease eradication process easier.

Remark 6.2. The effects of different distributions of the delays on disease eradication: Some information is known about the delay, $\tau_1$, after infection until full-blown AIDS occurs [9, 2]. In fact, over 2 to 15 years, untreated HIV individuals develop full-blown AIDS [9, 2]. Using (80), more specific sufficient conditions for $\tau_2$ leading to $R_0 << 1$ are derived, whenever the distributions of the random delays $\tau_1$ and $\tau_2$ are specified. Recall, the delays $\tau_1$ and $\tau_2$ are distributed with densities $f_{\tau_1}, f_{\tau_2}$. Three different distributions for $\tau_1$ and $\tau_2$ are considered below.

\begin{enumerate}
\item[(1)] Delays $\tau_1$ and $\tau_2$ are constant:
Assume $f_{\tau_1}(r) = \delta(r-\tau_1), r \in [t_0, h_1]$ and $f_{\tau_2}(u) = \delta(u-\tau_2), u \in [t_0, h_2]$, where $\delta(.)$ is the Dirac-Delta function. This scenario implies that after exposure to HIV, the delay until treatment $\tau_2$, and the delay until AIDS $\tau_1$, are constant for everyone. It is easy to see that (80) holds provided that
\[ \tau_2 < \tau_1 + \frac{1}{\mu} \log \left(1 - \alpha_{TI} \frac{1}{\alpha_{TI} + \mu_T + d_T + \alpha_{TA}}\right) < \tau_1. \]  
(81)

Note that $\frac{1}{\mu} \log \left(1 - \alpha_{TI} \frac{1}{\alpha_{TI} + \mu_T + d_T + \alpha_{TA}}\right) < 0$.

**Example.** Using $\tau_1 \in [2, 15]$ years, it follows that the RHS of (81) satisfies the inequality $\tau_1 + \frac{1}{\mu} \log \left(1 - \alpha_{TI} \frac{1}{\alpha_{TI} + \mu_T + d_T + \alpha_{TA}}\right) < 2$ years. This inequality
implies that the LHS of (81) also satisfied the inequality $\tau_2 < 2$ years. Thus, if every newly infected HIV person is tested and begins ART treatment before 2 years, then increasing the supply of ODAs (i.e. $\bar{\varepsilon}_0 \to 1$) so that more people get tested and begin ART treatment, makes it easier to eradicate the disease.

: (2.) **Delays $\tau_1$ and $\tau_2$ have exponential distributions:** Assuming that those who are newly infected proceed into the ART class or AIDS class independently at constant rates $\theta_2$ and $\theta_1$, respectively, then the time until treatment begins $\tau_2$, and the time until full-blown AIDS develops $\tau_1$, follow exponential distributions with means $E[\tau_2] = \frac{1}{\theta_2}$ time units, and $E[\tau_1] = \frac{1}{\theta_1}$ time units, respectively. In this scenario, two independent Poisson processes describe the number of people getting ART treatment and developing full-blown AIDS over time.

It is easy to see that (80) becomes

$$
\frac{\theta_2 \frac{1}{\mu} (1 - \alpha TI) \left(1 + \frac{1}{\alpha TI + \mu_T + d_T + \alpha TA}\right)}{\theta_1 \frac{1}{\mu}} > \frac{\theta_1 \frac{1}{\mu}}{\theta_1 \frac{1}{\mu} + \mu \frac{1}{\mu}}.
$$

(82)

where given that a newly infected person will begin ART treatment, $\frac{\theta_2 \frac{1}{\mu}}{\theta_1 \frac{1}{\mu} + \mu \frac{1}{\mu}}$ is the conditional fraction of the newly infected population that successfully gets into ART treatment. This implies that the fraction $1 - \frac{\theta_2 \frac{1}{\mu}}{\theta_2 \frac{1}{\mu} + \mu \frac{1}{\mu}}$ will die naturally.

Also, $\frac{\theta_1 \frac{1}{\mu}}{\theta_1 \frac{1}{\mu} + \mu \frac{1}{\mu}}$ is the conditional fraction of those who will turn into full-blown AIDS, given that they do not receive ART, and do not die naturally. Therefore, the LHS of (82) is the fraction of the newly infected population that survives natural death, gets ART treatment, and does not relapse from treatment. The RHS is the fraction of the newly infected population that fails to get ART treatment, and develops full-blown AIDS.

This special example confirms the observation that, when more newly infected people are likely to get tested, begin early ART treatment and do not relapse from treatment, then increasing the supply of ODAs (i.e. $\bar{\varepsilon}_0 \to 1$) so that more people are able to afford ART treatment, makes it easier to control the disease.

: (3) **Delays $\tau_1$ and $\tau_2$ have Gamma distributions:** Assuming that those who are newly infected proceeds into the ART class or AIDS class independently, and suppose certain internal or physically changes occur in those infected before they get into ART treatment or develop full-blown AIDS. That is, assume $\alpha_2$ and $\alpha_1$ average changes occur in the newly infected population that receives ART treatment, and the population that does not receive ART which ultimately becomes full-blown AIDS state, respectively. Also, the changes occur at constant rates $\theta_2$ and $\theta_1$, respectively. It follows that the time until ART treatment begins $\tau_2$, and the time until full-blown AIDS $\tau_1$ begins follow the Gamma distributions with means $E[\tau_2] = \frac{\alpha_2}{\theta_2}$ and $E[\tau_1] = \frac{\alpha_1}{\theta_1}$, respectively.

Applying similar technique in (82) and some algebraic simplifications, it is easy to see that (80) leads to

$$
E[\tau_2] < \frac{\alpha_2}{\mu_1} \left[\left(1 - \alpha TI \frac{1}{\alpha TI + \mu_T + d_T + \alpha TA}\right)\right]^\frac{1}{\alpha_2} \left(1 + \frac{\mu_1}{\alpha_1} E[\tau_1]\right)^\frac{1}{\alpha_1} - 1.
$$

(83)
The result in (83) suggests that if every newly infected person is certain to begin ART on average \( E[\tau_2] \) before
\[
\frac{\alpha_2}{\mu_1} \left[ \left( 1 - \alpha_{TI} \right) \frac{1}{\alpha_{TI} + \mu_T + d_T + \alpha_{TA}} \right] \frac{1}{\bar{\tau}_2} 
\]
\[
\left( 1 + \frac{\mu_1 E[\tau_1]}{\alpha_1} \right)^{\frac{\bar{\tau}_1}{\bar{\tau}_2}} - 1 \]
units of time, then increasing the supply of ODAs (i.e. \( \bar{\tau}_0 \rightarrow 1 \)) so that more people are able to afford ART, leads to a greater chance of controlling the disease from the population.

7. Numerical simulation results for HIV/AIDS epidemic using partial Uganda data. In this section some ideas and statistics in [15] for UGANDA HIV/AIDS epidemic over 1992 through 2006 are used to design parameter estimates for the HIV/AIDS model (3)-(9) for a hypothetical population with comparable characteristics as in the study [15]. Note that the example in this section is completely theoretical, and the primary objective of this section is to numerically explore the impacts of the factors studied in the model (3)-(9), namely: ART treatment, the supply of ODAs, and early HIV testing-and-onset of ART treatment, in the absence of noise in the HIV/AIDS epidemic dynamics. The reader is directed to [33] for a more practical and comprehensive application of the HIV/AIDS model (3)-(9) in a highly random environment with white noise perturbations.

(a.) Initial conditions: Similarly to the UGANDA UN data in 1991 [15], we assume the initial population consists of 18.38 million people, and the adult population vulnerable to HIV is 32%. Thus, the 5,899,980 million adults consist of the states \( S_0, S_1, S_2, I, T, A \) and \( R \). In the 5,899,980 initial adult population, 15% is assumed initially infected or removed, i.e. about 884,997 adults in \( I, T, A \) or \( R \) states. Moreover, there is a 12% prevalence rate of HIV (i.e. \( I(0) \) is about 707,997 adults), 2% treatment rate (\( T(0) \) is about 117,999 adults), 1% of the adult population completely removed (\( R(0) \) is about 58,999 adults), and 0% full-blown AIDS (\( A(0) = 0 \)). Thus, in the remaining 85% of the adult population, all susceptibles (about 501,4983 adults), we assume 10% are \( S_1(0) \) (about 501,498 adults), susceptibles adopting safe practices such as abstinence and being faithful; 10% are \( S_2(0) \) (about 501,498 adults), susceptibles adopting all other medically advised practices such as condom use etc.; the remaining 80% are susceptibles \( S_0 \) (about 4,011,986 adults), not practicing any safe sex measures.

Also, similarly to [15], it is assumed that there are initially about 240 organizations (20%) involved in the IEC. That is, \( Z(0) = 0.2 \). Applying the percentages above and scaling the initial values (by 1 million), lead to the initial conditions (84).

\[
S_0(t) = 4.011986, \quad S_1(t) = 0.501498, \quad S_2(t) = 0.501498, \quad I(t) = 0.707997, \\
T(t) = 0.117999, \quad A(t) = 0, \quad R(t) = 0.058999, \quad Z(t) = 0.2, \quad \forall t \in [-h, 0], \\
h = \max(T_1, T_2) = 8. 
\]

(b.) Information rates: Similarly to [15], the size of information in the population \( Z(t) \) is proportional to the number of organizations giving out information on HIV. Furthermore, the breakdown into the two states \( S_1(t) \) and \( S_2(t) \) at any time depends on the proportion of organizations giving information about the respective behaviors.

Therefore, as in [15], it is assumed 10% per unit time of \( S_0(t) \) interact with \( Z(t) \) and become \( S_1(t) \), and 80% per unit time interact with \( Z(t) \) and become \( S_2(t) \). Also, another 10% per unit time of \( S_0(t) \) interact with \( Z(t) \) and become \( R(t) \). That is, \( \gamma_0 = 0.1 \) per year , \( \gamma_1 = 0.1 \) per year and \( \gamma_2 = 0.8 \) per year.
(c.) Infection rates: Some studies [12] argue that the effects of behavioral changes such as abstinence, delayed sexual initiation, mutual monogamy and correct and consistent condom use on reducing HIV prevalence rates are complex to understand and debatable. However, UNAIDS [26], asserts that condom use effectively reduces the risk of transmission of HIV by the range of 69-94%. Siding with UNAIDS, we utilize some information from [15] to estimate $\beta_j$, $j = 0, 1, 2$. That is, it is assumed that $\beta_0 \in [0.01, 0.1]$, and that $\beta_1$ and $\beta_2$ are proportional to $\beta_0$. Moreover, $\beta_1 \ll \beta_0$ and $\beta_1 \ll \beta_2 < \beta_0$, where $\beta_0$, $\beta_1$ and $\beta_2$ are respective disease transmission rates in the susceptible class $S_0$ (those not practicing any safe sex measures), in $S_1$ (those practicing abstinence and be faithful), and in $S_2$ (those practicing condom use). Based on the above relationships, it is assumed that $\beta_1 = 0.05\beta_0$, and $\beta_2 = 0.4\beta_0$. That is, it assumed that the disease transmission rate among those not practicing any sexual preventive measures ($S_0$) is 20 times more than those who are practicing abstinence or adhering to sexually monogamous relationships ($S_1$), and also 2.5 times more than those who practice the use of condoms ($S_2$).

The estimate for $\beta_0 = 0.0211$ from [15] is used for simplicity. It is easy to see that $\beta_1 = 0.05\beta_0 = 0.001055$ and $\beta_2 = 0.4\beta_0 = 0.00844$.

(d.) Influx of new adults, natural and disease related death rates: Using UN data for UGANDA [1], the natural death rate per year is estimated using the sample mean lifespan of individuals in Uganda over the years 1997-2005. The sample mean lifespan over 1997-2005 is 47.51667 years. It is easy to see that the natural death rate, given a sample mean lifespan of 47.51667 years is $\mu_k = \mu = 1/47.51667 = 0.02104525$ per year, $k \in \{S_j, I, T, A, R\}$, $j \in I(0, 2)$.

Similarly, the UNAIDS UGANDA report [26], indicates that HIV related death rates per year were 17.2% in 1997, 13.36% in 1999, 14% in 2001, 14.42% in 2003. This leads to the sample mean estimated death rate $d_I = 0.1474$ per year.

According to Kasamba et. al. [16], ART led to significant reduction of the mortality rate of HIV positive people in UGANDA. It is shown (cf. [16]) that in the same HIV positive group with death rate of 116.4 deaths per 1,000 persons per year prior to ART, a reduction by 25% mortality rate is obtained after ART, leading to 87.4 deaths per 1,000 person per year. Thus, based on this information, assume $d_T = 0.25d_A = 0.03685$ per year.

According to [2], a white blood cell (CD4) count, lower than 200 cells/mm$^3$, is classified as full blown AIDS. Furthermore, if not treated immediately, the infected person cannot survive beyond 3 years. This suggests that the average lifetimes $\frac{1}{d_A} \ll \frac{1}{d_I}$. For this reason, assume $d_A$ is twice larger than $d_I$. That is, $d_A = 0.2948$ per year.

In [15], data for new susceptible adults entering the population over years 1990 to 2005 is utilized to calculate the mean influx rate of 0.55 per year of new susceptibles into the population. Using this estimate, assume that $B = 0.55$ per year new “ignorant” adult susceptibles of type $S_0$, enter the population at any time.

(e.) Delays until treatment and AIDS: Recall [9] that without proper ART, the infected individual progresses to AIDS in 2 to 15 years. That is, $\tau_1 \in [2, 15]$ years. Thus, assume that $\tau_1 = 8$ years. Also, in [2], it is suggested that within 2 to 12 weeks a newly infected person develops HIV antibodies, and over 6 months nearly every infected person can be diagnosed for HIV. Thus, early ART is assumed between $\tau_2 \in [0.038, 0.5]$ years.
(f.) Nonlinear incidence rates and nonlinear information rates: The nonlinear functions in Assumption 2.1 and Assumption 2.2 are taken to be

\[ H_j(Z) = \frac{a_j Z(t)}{1 + d_j Z(t)}, \quad G_j(i) = \frac{b_j i(t)}{1 + c_j i(t)} \quad j = 0, 1, 2 \quad (85) \]

where \(0 \leq a_j, b_j \leq 1, c_j, d_j \geq 1\) for \(j = 0, 1, 2\) are considered. Similarly, for the growth rate function given in (17), it is assumed that \(\hat{\phi_I} = 0.05, \hat{\phi_T} = 0.1, \hat{\phi_A} = 0.15, \hat{\phi_I} = 0.03, \hat{\phi_T} = 0.06, \hat{\phi_A} = 0.09\).

Note that all parameter estimates given in (a.)-(e.) above are summarized in Table 1, the DFE \(T = 15\) years, and the trajectories of the different states \(S_0(t), S_1(t), S_2(t), I(t), T(t)\) and \(A(t)\) of (3 )-(9) are generated by applying the Euler approximation scheme over 40 years from 1991 to 2031. For the parameter estimates in Table 1, the DFE \(E_0 = (26.13417, 0, 0, 0, \ldots , 0)\).

| Parameter | Symbol(s) | Estimate(s) in years |
|-----------|-----------|----------------------|
| Effective response rate of \(S_0(t), S_1(t), S_2(t)\) | \(\gamma_0, \gamma_1, \gamma_2\) | 0.1, 0.1, 0.8 |
| Infection transmission rates | \(\beta_0, \beta_1, \beta_2\) | 0.0211, 0.001055, 0.00844 |
| Natural death rates of \(S_0(t), S_1(t), S_2(t)\) | \(\mu_{S_0}, \mu_{S_1}, \mu_{S_2}\) | 0.01568 |
| Natural death rates of \(I(t), T(t), A(t), R(t)\) | \(\mu_I, \mu_T, \mu_A, \mu_R\) | 0.01568 |
| Infection related death rates of \(I(t)\) | \(d_I\) | 0.1474 |
| Infection related death rates of \(T(t)\) | \(d_T\) | 0.03685 |
| Infection related death rates of \(A(t)\) | \(d_A\) | 0.2948 |
| Recruitment rate | B | 0.55 |
| Return rate from \(T(t)\) to \(I(t)\) | \(\alpha_{TI}\) | 0.01 |
| Failure of treatment rate from \(T(t)\) to \(A(t)\) | \(\alpha_{TA}\) | 0.01 |
| Proportion of newly infected individuals from the class \(S_j, j = 0, 1, 2\) who do not receive ART and joins full blown AIDS state \(A(t)\) | \(\epsilon_0, \epsilon_1, \epsilon_2\) | 0 - 1 |
| Time delay to progress to full blown AIDS | \(\tau_1\) | 2 - 15 |
| Time delay to begin treatment | \(\tau_2\) | 0.38-15 |

Table 1. Shows the list of model parameters, estimates and their definitions. Note that the parameters are expressed in years and converted to days for all simulations in Section 7.

Note that wherever necessary, all practical simulation interpretations will be extended by extrapolation only over 40 years from 1991 until 2031, with initial conditions in (84).

7.1. Sensitivity of the BRN and the effects of early ART treatment and supply of ODAs. Figure 3 depicts the sensitivity of the BRN in (32) to the continuous changes in (1) the delay after infection, until the onset of ART treatment \(\tau_2\), and (2) the proportion getting ART treatment \(1 - \epsilon_0\), per unit time.

Observe in Figure 3(i) that early treatment (small \(\tau_2\) values) leads to \(R_0 << 1\). In Figure 3(ii), when ODAs are readily available and more people get ART treatment (i.e. \(1 - \epsilon_0 \to 1\), then \(R_0 << 1\).

These observations suggests that HIV/AIDS is more easily eradicated, (1) whenever infected individuals begin early ART treatment and prevent the virus from damaging their T-cells; (2) whenever ODAs and other resources are readily available that encourage more people to get ART treatment.
Figure 3. (i) depicts a continuously rising relationship between the BRN and the delay $\tau_2$. (ii) shows a declining relationship between the BRN and the proportion $1 - \varepsilon_0$ of individuals who are receiving ART treatment.

7.2. Numerical results for the global stability of the DFE and disease eradication. Applying the parameters in Table 1 to (66)-(67), and selecting $0 < K < 2.683626$, leads to $\lambda(\mu) = 0.25285683449939$. Furthermore, the modified BRN and other disease control parameters in (56)-(59) are respectively, $R_0 = 0.02393198 < 1$, $U_0 = 0.08569636 < 1$, $U_1 = 0.001062895 < 1$, $U_2 = 0.001063665 < 1$, $V_0 = 0.08175263 < 1$ and $W_0 = 0.3662252 < 1$.

Thus, the conditions of Theorem 5.3 and Theorem 5.4 are satisfied and as a result, the DFE $E_0$ is globally stable, and the disease dies out asymptotically. Moreover, from Theorem 5.4, $S(t) = S_0(t) + S_1(t) + S_2(t)$ persists and converges asymptotically to $S^* = \sum_{j=0}^{\infty} S_j^*(t) = \frac{B}{\mu_0} = 26.13417$. These results are exhibited in Figure 4.

8. Conclusion. In this study, a deterministic multipopulation behavioral HIV/AIDS epidemic model is derived and studied. Human behavior is influenced by IECs in the population. The impacts of limited constant supply of ODAs on the disease dynamics are considered. Moreover, the random delay time to begin ART treatment after HIV infection is also studied. Stability analysis of the DFE is given and conditions for disease eradication are found. The model is applied to UGANDA HIV/AIDS data.

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Figure 4. (a-1)-(d-1) shows the behavior of the path of the total susceptible population $S(t) = S_0(t) + S_1(t) + S_2(t)$, over time, whenever the conditions of Theorem 5.3 and Theorem 5.4 are satisfied. Clearly, the path of $S(t)$ is persistent as proven in Theorem 5.4 and approaches the DFE state $S^*_0 = \frac{B}{\mu S_0} = 26.13417$. The dotted red-line in (d-1) is the value of $S^*_0 = 26.13417$. The figures (e-1), (f-1) and (g-1) also show the paths of the HIV related states $I, T$ and $A$. Clearly, the paths of $I, T$ and $A$ approach the corresponding coordinate 0 of the DFE $E_0$. In other words, the disease is getting extinct over time.

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