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A model of HIV/AIDS transmission dynamics with treatment: The case of the DRC

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Abstract. In this paper, we propose a novel HIV/AIDS epidemic treatment model to reduce the number of HIV cases. We divide infected individuals into four compartments, that is, a compartment of infected individuals who are unaware of their HIV status and do not show any symptoms, a compartment of infected individuals who are aware of their status but do not show any symptoms, infected individuals in the asymptomatic compartment and a treatment compartment that receives infected individuals who are aware of their status, whether they are sick or not. The basic reproduction number $\mathcal{R}_0$ for the proposed model is computed using the next generation matrix (NGM). Using a corollary of Gershgorin's circle theorem, the results show that the disease-free equilibrium (DFE) is locally asymptotically stable (LAS) if $\mathcal{R}_0 < 1$ and the endemic equilibrium (EE) is locally asymptotically stable if $\mathcal{R}_0 > 1$. We also proved by means of the Lyapunov method that the disease-free equilibrium is globally asymptotically stable if $\mathcal{R}_0 < 1$. Finally, numerical simulations of the model are conducted to support the theoretical results and also to investigate the sensitivity of certain parameters using HIV/AIDS data from the Democratic Republic of the Congo (DRC).

Keywords. HIV/AIDS model, antiretroviral, stability analysis, sensitivity analysis, basic reproduction number

1 Introduction

Mathematical modeling has been used to improve our understanding of the dynamics of infectious disease transmission and to find ways to control them. This application of mathematics dates back to the work of Daniel Bernoulli, who used mathematical and statistical approaches to assess the possible impact of smallpox vaccination in 1760 [5]. In the 1920s, the medical professional Sir Ronald Ross used mathematical modeling to suggest efficient ways to eliminate malaria. He specifically proved that the disease can be eliminated if the mosquito population is maintained below a specific level, a finding for which he was awarded the Nobel Prize in Medicine [10]. More recently, mathematics has contributed to the creation of efficient public health policies aimed

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at preventing the transmission of diseases that present a serious risk to public health, such as HIV/AIDS, influenza (a recent outbreak of avian and swine flu), malaria, SARS (Severe Acute Respiratory Syndrome), tuberculosis, and others [10].

The infectious disease known as HIV adversely affects the normal function of the immune system. It is caused by the human immunodeficiency virus (HIV), which damages the body's ability to fight infections. The first recorded incidence of HIV/AIDS was in 1981. Since then, HIV has spread throughout the world [13]. Therefore, in order to reduce the mortality rate, more aggressive antiretroviral therapies must be adopted [13, 15, 25].

Due to the lack of a vaccine, there are several difficulties associated with the treatment of HIV. Recently, the most common treatment approach for HIV patients is highly active antiretroviral therapies (HAART), which can prolong life expectancy and improve the quality of life of these patients [12]. Currently, antiretroviral therapy for HIV/AIDS involves the use of two or more antivirally active drugs, typically classified into two main groups: reverse transcriptase inhibitors (RTIs) and protease inhibitors (PIs) [8, 18]. With the help of antiretroviral therapy, infected individuals can extend the duration in which they remain without HIV-related symptoms, thus improving their quality of life. To reduce the risk of transmission to future generations, HIV-positive individuals can be treated before the development of AIDS, which can greatly decrease the risk of transmission to future generations. At least three millions of people have been saved in the United States, which highlights the significant advances made in the treatment of HIV disease [28]. Because of this, it is crucial and urgent to treat AIDS immediately after diagnosis.

Research on HIV/AIDS has attracted many scientists over the years. In terms of mathematical modeling of this infection, we report some of the works performed. Meng *et al.* [21] examined a more general HIV/AIDS model that incorporates a nonlinear incidence rate, humoral immunity, and antiretroviral therapy. The authors proved the existence of a global attractor by employing the method of characteristics and defining an auxiliary function. Yusuf and Benyah [30] described a deterministic model to control the spread of the disease using change in sexual habits and antiretroviral (ARV) therapy as control measures. They estimated the initial conditions and parameter values from the demographic and HIV/AIDS data of South Africa. Their results showed that the optimal way to reduce the transmission of the disease is for susceptible individuals to consistently practice safe sex as much as possible, while ARV treatment should be initiated for patients as soon as they progress to the pre-AIDS stage of the disease.

Huo and Feng [14] examined a model of the HIV/AIDS epidemic that includes slow and fast latent compartments, demonstrating that treatment for individuals in both slow and fast compartments is quite significant. Hao *et al.* [11] presented a stochastic HIV/AIDS model that combines commercial heterosexual behavior with a ln-type Ornstein–Uhlenbeck process, describing the transmission dynamics of HIV/AIDS between female sex workers and male clients. In addition, the authors demonstrated the existence and uniqueness of global solutions, as well as the existence of a stationary distribution using Itô's lemma. Finally, they constructed suitable Lyapunov functions to analyse the global stability of the model. With respect to the basic reproduction number, they authors provided conditions for the persistence or not of the disease.

Huo *et al.* [13] introduced a new HIV/AIDS epidemic model in which they incorporated a new compartment, that is, the treatment compartment and established the idea that the global dynamics of HIV/AIDS is determined by the basic reproduction number $\mathcal{R}_0$. Their results showed that early treatment for AIDS is necessary and meaningful. Huo and Chen [12] presented a model of HIV/AIDS with different stages and established the idea that the global dynamics of the HIV/AIDS model is determined by the basic reproduction number $\mathcal{R}_0$. Their results showed that early treatment for individuals in the asymptomatic stage of HIV infection or in the pre-AIDS stage is very important. The HIV/AIDS model with voluntary counseling and

testing (VCT) awareness and ART treatment is examined by Luo *et al.* [20]. To apply the theory of global exponential attractors to non-autonomous dissipative evolution equations, the authors first investigated the well-posedness of the model before determining the existence of the principal eigenvalue and the global attractor of the system. Their findings indicated that increasing awareness of VCT, ART treatment rates and treatment levels can play a crucial role in controlling the spread of HIV/AIDS.

Gumel [10] examined a model that accounts for the use of condoms, pharmaceutical treatments, incorporates awareness and psychological monitoring programs using HIV data from Nigeria during the period 1991-2005. His results showed that the disease (number of infections) peaked around the year 2000 and declined thereafter. This improved after considering the use of pharmaceutical treatments and preventive measures. Safiel *et al.* [26] examined the impact of screening and treatment on the transmission of HIV/AIDS infection in a population. In order to reduce HIV transmission, they recommended that more HIV/AIDS centres for voluntary screening and ARV treatment be established throughout each country to ensure that more people have access to the facilities. Wu *et al.* [29] integrated spatial heterogeneity and ART in a nonlocal dispersal HIV/AIDS epidemic model and examined the combined effects of spatial heterogeneity and ART treatment on HIV/AIDS dynamics in China, taking into account the significant role of spatial heterogeneity in epidemiological compartmental models.

Nsuami and Witbooi [22] presented a new model for HIV/AIDS transmission including antiretroviral treatment (ART) and oral pre-exposure prophylaxis (PrEP) in the context of South Africa. Their results showed that early treatment can decrease transmission and possibly decrease the number of AIDS-related deaths. Luo *et al.* [19] presented a stochastic HIV/AIDS model with ART and PrEP as treatments to describe the transmission dynamics of HIV/AIDS among men who have sex with men (MSM). The basic reproduction number $\mathcal{R}_0$ for the deterministic model is computed using the next-generation matrix to study the global stability of the disease-free equilibrium and the endemic equilibrium. Moreover, the existence of global positive solutions and the uniqueness of an ergodic stationary distribution for the stochastic model are investigated. Their results showed that both ART and PrEP treatments can effectively control the spread of AIDS within the MSM population.

In addition to the individuals who changed sexual behaviour, in this paper, we divide the groups of infected individuals into four compartments: (i) infected individuals who are unaware of their HIV status and do not show any symptoms, (ii) infected individuals who are aware of their HIV status but do not display any symptoms, (iii) infected individuals who display HIV symptoms, and (iv) a treatment class that receives infected individuals who are aware of their status, whether they present HIV symptoms or not. Moreover, we investigate the sensitivity of certain parameters that influence the basic reproduction number $\mathcal{R}_0$. None of the work done earlier on the mathematical modeling of HIV/AIDS dynamics has ever considered this split, to the best of our knowledge. We compute the basic production number using the next generation matrix and determine the equilibrium points of the model and their stability, using a corollary of Gershgorin's circle theorem and the Lyapunov theorem. Moreover, we conduct the numerical simulations using the demography and HIV/AIDS data from DRC.

This paper is organized as follows. In Section 2, we derived an HIV/AIDS model, followed by the model analysis in Section 3. In Section 4, numerical simulations are presented. Some discussion is given in Section 5.

2 Mathematical model

In this section, we describe a basic model HIV/AIDS epidemic with treatment and some properties based on the model.

2.1 Model formulation

This section presents a simple mathematical model of HIV/AIDS with treatment. The human population $N(t)$ is divided into six compartments, namely $S(t)$ representing susceptible individuals; $I_1(t)$ representing the new infected individuals who are unaware of their status and do not show any symptoms of AIDS; $I_2(t)$ infected individuals who do not show symptoms of AIDS, but who know their status, $A(t)$ representing individuals with AIDS and who show clinical symptoms of AIDS; $T(t)$ represents the number of individuals receiving pharmaceutical treatment (antiretroviral: ARV) and $R(t)$ is the number of individuals who have changed their sexual behavior to reduce the risk of infection [13, 30]. It is assumed that these individuals (in the $R(t)$ class) will maintain this behaviour for the rest of their lives. Note that some of the infected individuals in class I_1 may move to the compartment I_2, while others may move to the compartment A. Certain infected individuals in I_2 can transfer to compartment A and others into compartment T. In addition, some infected individuals in A can be moved to the compartment T. Also to note is the fact that some individuals in the compartment T can be transferred back to the compartment I_2 after treatment. Therefore, the total human population $N(t)$ for the proposed model is given by

$$N(t) = S(t) + I_1(t) + I_2(t) + A(t) + T(t) + R(t). \tag{2.1}$$

Thus, the model (2.2) is given by (see Fig 1):

$$\frac{dS}{dt} = \Lambda - \frac{S\beta}{N}(I_1 + cI_2 + dA) - \mu S - \phi S \tag{2.2a}$$
$$\frac{dI_1}{dt} = \frac{S\beta}{N}(I_1 + cI_2 + dA) - a_1 I_1 \tag{2.2b}$$
$$\frac{dI_2}{dt} = \lambda I_1 - a_2 I_2 + \kappa_2 T \tag{2.2c}$$
$$\frac{dA}{dt} = \sigma_1 I_1 + \sigma_2 I_2 - a_3 A \tag{2.2d}$$
$$\frac{dT}{dt} = \alpha I_2 + \kappa_1 A - a_4 T \tag{2.2e}$$
$$\frac{dR}{dt} = \phi S - \mu R, \tag{2.2f}$$

where $a_1 = \sigma_1 + \lambda + \mu$, $a_2 = \sigma_2 + \mu + \alpha$, $a_3 = \mu + \delta_1 + \kappa_1$ and $a_4 = \kappa_2 + \mu + \delta_2$. The parameters used in the model (2.2) are described in Table 1.

2.2 Positive invariant region

This section is devoted to proving that the variables of the model (2.2) are positive and bounded using the following lemmas.

Lemma 2.1. *The solution* $(S(t), I_1(t), I_2(t), A(t), T(t)\ R(t))$ *of the system* (2.2) *with initial conditions* $S(0) > 0, I_1(0) \geq 0, I_2(0) \geq 0, A(0) \geq 0, T(0) \geq 0$ *et* $R(0) \geq 0$ *is positive for all time* $t > 0$.

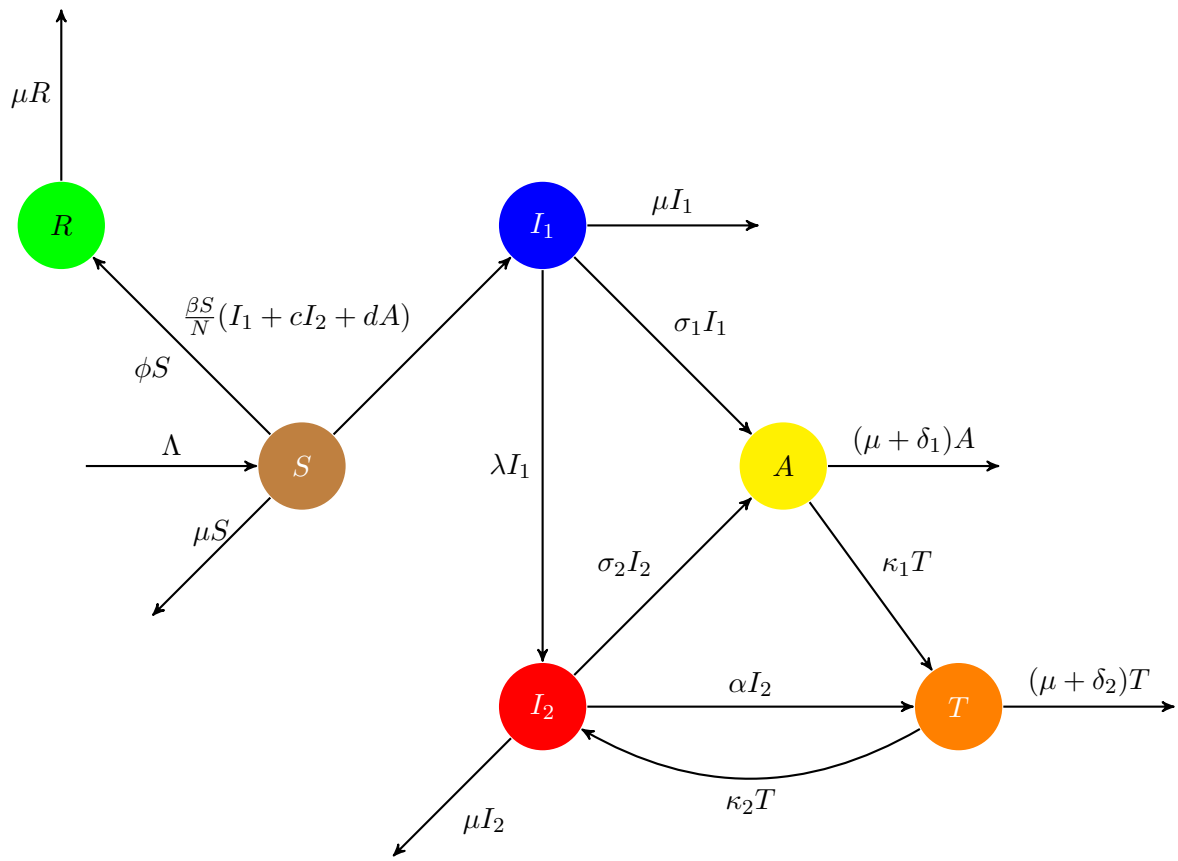

Figure 1: **HIV/AIDS with treatement**

Table 1: Description of parameters for the model (2.2)

Symbol	Parameter Description
Λ	Recruitment rate of the population
β	Effective contact rate
μ	Natural death rate
ϕ	The rate of susceptible individuals who changed their habits
c	Infection rate for individuals of class $I_2(t)$
d	Infection rate for individuals of class $A(t)$
σ_1	Progression rate from the $I_1(t)$ class into the $A(t)$ class
σ_2	Progression rate from the $I_2(t)$ class into the $A(t)$ class
λ	Progression rate from the I_1 class into the I_2 class
δ_1	The disease-related death rate of the AIDS $A(t)$
δ_2	The disease-related death rate of being treated $T(t)$, $\delta_2 < \delta_1$
α	Proportion of the $I_2(t)$ class receiving treatment
κ_1	Proportion of the $A(t)$ class receiving treatment
κ_2	Proportion of treated individuals who leave compartment $T(t)$.

Proof. The proof follows similar lines as in Yusuf *et al.* [30]. □

Lemma 2.2. *The feasible region* Γ *of the model* (2.2) *defined by*

$$\Gamma = \left\{ (S(t),\ I_1(t),\ I_2(t),\ A(t),\ T(t),\ R(t)) \in \mathbb{R}^6_+ / 0 \leq S(t) + I_1(t) + I_2(t) + A(t) + R(t) \leq \frac{\Lambda}{\mu} \right\}$$

is positively invariant, which means that its parameters and variables of the system (2.2) *are always positive since it concerns the human population model [3].*

Proof. By adding the equations of the system (2.2) and an application of the theorem of integral inequalities proposed by [16], we obtain

$$\begin{aligned} \frac{dN}{dt} =& \Lambda - \mu N - \delta_1 A - \delta_2 T \\ \leq& \Lambda - \mu N. \end{aligned} \tag{2.3}$$

Using the integrating factor method for first-order linear ODEs on (2.3), we obtain:

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}), \tag{2.4}$$

where $N(0)$ represents the initial value of the total population. Thus

$$\lim_{t \to \infty} sup\ N(t) \leq \frac{\Lambda}{\mu}.$$

This implies that the region

$$\Gamma = \left\{ (S(t),\ I_1(t),\ I_2(t),\ A(t),\ T(t),\ R(t)) \in \mathbb{R}^6_+ / 0 \leq S(t) + I_1(t) + I_2(t) + A(t) + R(t) \leq \frac{\Lambda}{\mu} \right\}$$

is positively invariant for the system (2.2). In particular,

$$N(t) \leq \frac{\Lambda}{\mu} \text{ if } N(0) \leq \frac{\Lambda}{\mu}.$$

$$\text{Thus, } 0 \leq N(t) \leq \frac{\Lambda}{\mu}.$$

□

3 Analysis of the mathematical model

In this section, we determine the equilibrium of the model, analyse their stability and calculate the basic reproduction number.

3.1 Disease-free equilibrium and basic reproduction number $\mathcal{R}_0$

We obtain the DFE equilibrium of the system (2.2) as follows

$$E_0(S_0,\ I_{1,0},\ I_{2,0},\ A_0,\ T_0,\ R_0) = \left(\frac{\Lambda}{\mu+\phi},\ 0,\ 0,\ 0,\ 0,\ \frac{\Lambda\phi}{\mu(\mu+\phi)}\right). \tag{3.1}$$

We use the next generation matrix (NGM) approach proposed by [27] to derive the basic reproduction number, noted $\mathcal{R}_0$ of the system (2.2). We rearrange (2.2) as follows:

$$\frac{dI_1}{dt} = \frac{S\beta}{N}(I_1 + cI_2 + dA) - (\sigma_1 + \lambda + \mu)I_1 \tag{3.2a}$$

$$\frac{dI_2}{dt} = \lambda I_1 - (\sigma_2 + \mu + \alpha)I_2 + \kappa_2 T \tag{3.2b}$$

$$\frac{dA}{dt} = \sigma_1 I_1 + \sigma_2 I_2 - (\mu + \delta_1 + \kappa_1)A \tag{3.2c}$$

$$\frac{dT}{dt} = \alpha I_2 + \kappa_1 A - (\kappa_2 + \mu + \delta_2)T \tag{3.2d}$$

$$\frac{dS}{dt} = \Lambda - \frac{S\beta}{N}(I_1 + cI_2 + dA) - \mu S - \phi S \tag{3.2e}$$

$$\frac{dR}{dt} = \phi S - \mu R \tag{3.2f}$$

Letting $x = (I_1,\ I_2,\ A,\ T,\ S,\ R)^t$ denote the states of a population with respect to their pathological (disease) status. Thus, the system (3.2) can be written as:

$$\frac{dx}{dt} = \mathcal{F}(x) - \mathcal{V}(x), \tag{3.3}$$

where $\mathcal{F}(x)$ is the rate of new infections and $\mathcal{V}(x)$ is the rate of transfer (by other means) between compartments. The Jacobian matrices of $\mathcal{F}$ et $\mathcal{V}$ at the disease free equilibrium E_0 are given by

$$F = \begin{pmatrix} \frac{\beta\mu}{\mu+\phi} & c\frac{\beta\mu}{\mu+\phi} & d\frac{\beta\mu}{\mu+\phi} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{3.4}$$

and

$$V = \begin{pmatrix} \sigma_1+\lambda+\mu & 0 & 0 & 0 \\ -\lambda & \sigma_2+\mu+\alpha & 0 & -\kappa_2 \\ -\sigma_1 & -\sigma_2 & \mu+\delta_1+\kappa_1 & 0 \\ 0 & -\alpha & -\kappa_1 & \kappa_2+\mu+\delta_2 \end{pmatrix}, \tag{3.5}$$

respectively. Then, the matrix of $M = FV^{-1}$ is given by

$$M = \frac{1}{\det \mathrm{V}} \begin{pmatrix} A_1 & A_2 & A_3 & A_4 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{3.6}$$

where

$$\begin{aligned}
\det \mathrm{V} =& (\sigma_1+\lambda+\mu)[(\sigma_2+\mu+\alpha)(\mu+\delta_1+\kappa_1)(\kappa_2+\mu+\delta_2) - \kappa_1\kappa_2\sigma_2 - (\mu+\delta_1+\kappa_1)\alpha\kappa_2], \\
A_1 =& \frac{\beta\mu}{\mu+\phi}\left[(\sigma_2+\mu+\alpha)(\mu+\delta_1+\kappa_1)(\kappa_2+\mu+\delta_2) - \kappa_1\kappa_2\sigma_2 - \alpha\kappa_2(\mu+\delta_1+\kappa_1)\right] \\
&+\frac{c\beta\mu}{\mu+\phi}\left[\lambda(\mu+\delta_1+\kappa_1)(\kappa_2+\mu+\delta_2) + \kappa_1\kappa_2\sigma_1\right] \\
&+\frac{d\beta\mu}{\mu+\phi}\left[\lambda\sigma_2(\kappa_2+\mu+\delta_2) - \sigma_1\alpha\kappa_2 + \sigma_1(\sigma_2+\mu+\alpha)(\kappa_2+\mu+\delta_2)\right], \\
A_2 =& \frac{c\beta\mu}{\mu+\phi}\left[(\sigma_1+\lambda+\mu)(\mu+\delta_1+\kappa_1)(\kappa_2+\mu+\delta_2)\right] + \frac{d\beta\mu}{\mu+\phi}\left[\sigma_2(\sigma_1+\lambda+\mu)(\kappa_2+\mu+\delta_2)\right], \\
A_3 =& \frac{c\beta\mu}{\mu+\phi}\left[\kappa_1\kappa_2(\sigma_1+\lambda+\mu)\right] + \frac{d\beta\mu}{\mu+\phi}(\sigma_1+\lambda+\mu)[(\sigma_2+\mu+\alpha)(\kappa_2+\mu+\delta_2) - \alpha\kappa_2]
\end{aligned}$$

and

$$A_4 = \frac{c\beta\mu}{\mu+\phi}\left[\kappa_2(\sigma_1+\lambda+\mu)(\mu+\delta_1+\kappa_1)\right] + \frac{d\beta\mu}{\mu+\phi}\sigma_2\kappa_2(\sigma_1+\lambda+\mu).$$

The characteristic polynomial of the matrix M is

$$\lambda^2(A_1 - \lambda) = 0. \tag{3.7}$$

Hence, the basic reproduction number $\mathcal{R}_0$ of the model (2.2) is the dominant eigenvalue of M given by

$$\begin{aligned}
\mathcal{R}_0 =& \frac{\beta\mu}{\mu+\phi}\left[\frac{1}{\sigma_1+\lambda+\mu}\right] \\
&+\frac{\beta\mu}{\mu+\phi}\left[\frac{c[\lambda(\mu+\delta_1+\kappa_1)(\kappa_2+\mu+\delta_2) + \kappa_1\kappa_2\sigma_1]}{(\sigma_1+\lambda+\mu)[(\sigma_2+\mu+\alpha)(\mu+\delta_1+\kappa_1)(\kappa_2+\mu+\delta_2) - \kappa_1\kappa_2\sigma_2 - \alpha\kappa_2(\mu+\delta_1+\kappa_1)]}\right] \\
&+\frac{\beta\mu}{\mu+\phi}\left[\frac{d[(\kappa_2+\mu+\delta_2)(\sigma_1\sigma_2+\mu\sigma_1+\lambda\sigma_2) + \alpha\sigma_1(\mu+\delta_2)]}{(\sigma_1+\lambda+\mu)[(\sigma_2+\mu+\alpha)(\mu+\delta_1+\kappa_1)(\kappa_2+\mu+\delta_2) - \kappa_1\kappa_2\sigma_2 - \alpha\kappa_2(\mu+\delta_1+\kappa_1)]}\right].
\end{aligned} \tag{3.8}$$

The expression of $\mathcal{R}_0$ can be simplified as follows

$$\mathcal{R}_0 = \frac{\beta\mu}{\mu+\phi}\left[\mathcal{R}_1 + \mathcal{R}_2 + \mathcal{R}_3\right] \tag{3.9}$$

where

$$\mathcal{R}_1 = \frac{1}{\sigma_1 + \lambda + \mu}, \tag{3.10}$$

$$\mathcal{R}_2 = \frac{c[\lambda(\mu + \delta_1 + \kappa_1)(\kappa_2 + \mu + \delta_2) + \kappa_1\kappa_2\sigma_1]}{(\sigma_1 + \lambda + \mu)[(\sigma_2 + \mu + \alpha)(\mu + \delta_1 + \kappa_1)(\kappa_2 + \mu + \delta_2) - \kappa_1\kappa_2\sigma_2 - \alpha\kappa_2(\mu + \delta_1 + \kappa_1)]}, \tag{3.11}$$

$$\mathcal{R}_3 = \frac{d[(\kappa_2 + \mu + \delta_2)(\sigma_1\sigma_2 + \mu\sigma_1 + \lambda\sigma_2) + \alpha\sigma_1(\mu + \delta_2)]}{(\sigma_1 + \lambda + \mu)[(\sigma_2 + \mu + \alpha)(\mu + \delta_1 + \kappa_1)(\kappa_2 + \mu + \delta_2) - \kappa_1\kappa_2\sigma_2 - \alpha\kappa_2(\mu + \delta_1 + \kappa_1)]}. \tag{3.12}$$

Note that the basic reproduction number, $\mathcal{R}_0$, is the average number of secondary infections from a single individual throughout its entire infectious period in a population of susceptible individuals [2, 30].

The Jacobian matrix J of the system (2.2) is

$$J = \begin{pmatrix} \eta_1 & -\epsilon & -c\epsilon & -d\epsilon & 0 & 0 \\ \eta_2 & \epsilon - a_1 & c\epsilon & d\epsilon & 0 & 0 \\ 0 & \lambda & -a_2 & 0 & \kappa_2 & 0 \\ 0 & \sigma_1 & \sigma_2 & -a_3 & 0 & 0 \\ 0 & 0 & \alpha & \kappa_1 & -a_4 & 0 \\ \phi & 0 & 0 & 0 & 0 & -\mu \end{pmatrix}, \tag{3.13}$$

where

$$\epsilon = S\beta/N,\ \eta_1 = -\beta(I_1 + cI_2 + dA)/N - \mu - \phi \text{ and } \eta_2 = \beta(I_1 + cI_2 + dA)/N.$$

Corollary 3.1. *[1] Assume that the matrix B has $n \times n$ real entries. If the diagonal elements b_{ii} of B verify*

$$-b_{ii} > r_i$$

where

$$r_i = \sum_{j=1,\ j\neq i}^{n} |b_{ij}|, \tag{3.14}$$

for $i = 1,\ 2,\ \cdots,\ n$; then the eigenvalues of B are negative or have negative real parts . Therefore, the equilibrium point is locally asymptotically stable [4].

Theorem 3.2. *The disease-free equilibrium (DFE) of the model (2.2), given by E_0, is locally asymptotically stable (LAS) whenever $\mathcal{R}_0 < 1$ and unstable for $\mathcal{R}_0 > 1$.*

Proof. The Jacobian matrix J of the system (2.2) at the disease-free equilibrium point E_0 is given by

$$J(E_0) = \begin{pmatrix} -(\mu + \phi) & -\frac{\beta\mu}{\mu+\phi} & -c\frac{\beta\mu}{\mu+\phi} & -d\frac{\beta\mu}{\mu+\phi} & 0 & 0 \\ 0 & \frac{\beta\mu}{\mu+\phi} - a_1 & c\frac{\beta\mu}{\mu+\phi} & d\frac{\beta\mu}{\mu+\phi} & 0 & 0 \\ 0 & \lambda & -a_2 & 0 & \kappa_2 & 0 \\ 0 & \sigma_1 & \sigma_2 & -a_3 & 0 & 0 \\ 0 & 0 & \alpha & \kappa_1 & -a_4 & 0 \\ \phi & 0 & 0 & 0 & 0 & -\mu \end{pmatrix} \tag{3.15}$$

The matrix $J(E_0)$ has two eigenvalues that have negative real parts, $\lambda_1 = -\mu$ and $\lambda_2 = -(\mu + \phi)$. Removing rows 1 and 5 from $J(E_0)$, we end up with the sub-matrix as follows:

$$J_s(E_0) = \begin{pmatrix} \frac{\beta\mu}{\mu+\phi} - a_1 & c\frac{\beta\mu}{\mu+\phi} & d\frac{\beta\mu}{\mu+\phi} & 0 \\ \lambda & -a_2 & 0 & \kappa_2 \\ \sigma_1 & \sigma_2 & -a_3 & 0 \\ 0 & \alpha & \kappa_1 & -a_4 \end{pmatrix} \tag{3.16}$$

The matrix $J_s(E_0)$ will have negative eigenvalues in accordance with Corollary 3.1 if the following inequalities are satisfied:

$$-\frac{\beta\mu}{\mu+\phi} + a_1 > c\frac{\beta\mu}{\mu+\phi} + d\frac{\beta\mu}{\mu+\phi}. \tag{3.17a}$$

$$1 > \frac{\lambda + \kappa_2}{a_2}. \tag{3.17b}$$

$$1 > \frac{\sigma_1 + \sigma_2}{a_3}. \tag{3.17c}$$

$$1 > \frac{\alpha + \kappa_1}{a_4}. \tag{3.17d}$$

Inequality (3.17a) can be simplified as follows

$$\frac{1 - \mathcal{R}_0}{\frac{\beta\mu}{\mu+\phi}(\frac{c}{a_1} + \frac{d}{a_1} - \mathcal{R}_2 - \mathcal{R}_3)} > 1. \tag{3.18}$$

Combining inequalities (3.17b) and (3.17d) gives

$$1 > \frac{(\lambda + \kappa_2)(\sigma_1 + \sigma_2)(\alpha + \kappa_1)}{a_2 a_3 a_4}. \tag{3.19}$$

Using inequalities (3.18) and (3.19), we obtain

$$\frac{1 - \mathcal{R}_0}{\frac{\beta\mu}{\mu+\phi}(\frac{c}{a_1} + \frac{d}{a_1} - \mathcal{R}_2 - \mathcal{R}_3)} > 1 > \frac{(\lambda + \kappa_2)(\sigma_1 + \sigma_2)(\alpha + \kappa_1)}{a_2 a_3 a_4}.$$

It follows that

$$\frac{1 - \mathcal{R}_0}{(\lambda + \kappa_2)(\sigma_1 + \sigma_2)(\alpha + \kappa_1)} > \frac{\frac{\beta\mu}{\mu+\phi}(\frac{c}{a_1} + \frac{d}{a_1} - \mathcal{R}_2 - \mathcal{R}_3)}{a_2 a_3 a_4}.$$

The above inequality holds if $\mathcal{R}_0 < 1$. This shows that the DFE is locally asymptotically stable since $\mathcal{R}_0 < 1$. □

Theorem 3.3. *The disease-free equilibrium E_0 of the model (2.2) is globally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof. Following the ideas of [7, 12], we construct the Lyapunov function as follows:

$$V(t) = I_1(t) + \theta_1 I_2(t) + \theta_2 A(t) + \theta_3 T(t), \tag{3.20}$$

where $\theta_i > 0$ $(i = 1, 2, 3)$ are constants to be determined. The differentiation of $V(t)$ with respect to time is given by

$$\frac{dV}{dt} = \frac{dI_1}{dt} + \theta_1 \frac{dI_2}{dt} + \theta_2 \frac{dA}{dt} + \theta_3 \frac{dT}{dt}. \tag{3.21}$$

Substituting equation (2.2) into equation (3.21), we obtain:

$$\begin{aligned}\frac{dV}{dt} =& S\beta/N(I_1 + cI_2 + dA) - a_1 I_1 + \theta_1(\lambda I_1 - a_2 I_2 + \kappa_2 T) + \theta_2(\sigma_1 I_1 + \sigma_2 I_2 - a_3 A) \\ &+\theta_3(\alpha I_2 + \kappa_1 A - a_4 T).\end{aligned} \tag{3.22}$$

At equilibrium without disease: $S_0 = \Lambda/(\mu+\phi)$ and $R_0 = \Lambda\phi/\mu(\mu+\phi)$ et $N = \Lambda/\mu$, equation (3.22) becomes

$$\begin{aligned}\frac{dV}{dt} \leq& \frac{\beta\mu}{\mu+\phi}(I_1 + cI_2 + dA) - a_1 I_1 + \theta_1(\lambda I_1 - a_2 I_2 + \kappa_2 T) + \theta_2(\sigma_1 I_1 + \sigma_2 I_2 - a_3 A) \\ &+\theta_3(\alpha I_2 + \kappa_1 A - a_4 T) \\ &= \left[\frac{\beta\mu}{\mu+\phi} - a_1 + \theta_1\lambda + \theta_2\sigma_1\right] I_1 + \left[\frac{c\beta\mu}{\mu+\phi} + \theta_2\sigma_2 + \theta_3\alpha - \theta_1 a_2\right] I_2 \\ &+ \left[\frac{d\beta\mu}{\mu+\phi} - \theta_2 a_3 + \theta_3\kappa_1\right] A + [\theta_1\kappa_2 - \theta_3 a_4]\, T.\end{aligned} \tag{3.23}$$

Setting the coefficients of $I_2,\ I_3,\ A$ and T to zero

$$\begin{cases} \theta_1\kappa_2 - \theta_3 a_4 = 0 \\ d\beta\mu/(\mu+\phi)\theta_2 a_3 + \theta_3\kappa_1 = 0 \\ c\beta\mu/(\mu+\phi) + \theta_2\sigma_2 + \sigma_3\alpha - \theta_1 a_2 = 0 \end{cases} \tag{3.24}$$

Solving equation (3.24), we obtain

$$\theta_1 = \frac{a_4\beta\mu(d\sigma_2 + ca_3)}{(\mu+\phi)(a_2a_3a_4 - a_3\alpha\kappa_2 - \kappa_1\kappa_2\sigma_2)},\ \theta_3 = \frac{\mu\beta\kappa_2(d\sigma_2 + ca_3)}{(\mu+\phi)(a_2a_3a_4 - a_3\alpha\kappa_2 - \kappa_1\kappa_2\sigma_2)} \text{ and}$$

$$\theta_2 = \frac{\beta\mu[(d\sigma_2 + ca_3)(a_2a_4 - \alpha\kappa_2) - c(a_2a_3a_4 - a_3\alpha\kappa_2 - \kappa_1\kappa_2\sigma_2)]}{\sigma_2(\mu+\phi)(a_2a_3a_4 - a_3\alpha\kappa_2 - \kappa_1\kappa_2\sigma_2)}.$$

By substituting θ_1 θ_2, and θ_3 into equation (3.23), we obtain

$$\begin{aligned}\frac{dV}{dt} =& [\frac{\beta\mu}{\mu+\phi} - a_1 + \frac{\lambda a_4\beta\mu(d\sigma_2 + ca_3)}{(\mu+\phi)(a_2a_3a_4 - a_3\alpha\kappa_2 - \kappa_1\kappa_2\sigma_2)} \\ &+ \frac{\sigma_1\beta\mu[(d\sigma_2 + ca_3)(a_2a_4 - \alpha\kappa_2) - c(a_2a_3a_4 - a_3\alpha\kappa_2 - \kappa_1\kappa_2\sigma_2)]}{\sigma_2(\mu+\phi)(a_2a_3a_4 - a_3\alpha\kappa_2 - \kappa_1\kappa_2\sigma_2)}]I_1.\end{aligned} \tag{3.25}$$

Rewrite equation (3.25) as follows

$$\begin{aligned}\frac{dV}{dt} \leq& \left[\frac{\beta\mu\,[a_2a_3a_4 - \kappa_1\kappa_2\sigma_2 - \alpha\kappa_2a_3 + c(\lambda a_3a_4 + \kappa_1\kappa_2\sigma_1) + d(\sigma_1a_2a_4 + \lambda\sigma_2a_4 - \sigma_1\alpha\kappa_2)]}{(\mu+\phi)[a_2a_3a_4 - \kappa_1\kappa_2\sigma_2 - \alpha\kappa_2a_3]}\right. \\ &-a_1 + \frac{\sigma_1\beta\mu ca_3(a_2a_4 - \alpha\kappa_2)}{\sigma_2(\mu+\phi)(a_2a_3a_4 - a_3\alpha\kappa_2 - \kappa_1\kappa_2\sigma_2)} \\ &\left. - \frac{\sigma_1\beta\mu ca_3(a_2a_4 - \alpha\kappa_2)}{\sigma_2(\mu+\phi)(a_2a_3a_4 - a_3\alpha\kappa_2 - \kappa_1\kappa_2\sigma_2)}\right] I_1.\end{aligned} \tag{3.26}$$

Equation (3.26) simplifies to the following:

$$\frac{dV}{dt} \leq a_1(\mathcal{R}_0 - 1)I_1. \tag{3.27}$$

□

It is evident from equation (3.27) that when $\mathcal{R}_0 \leq 1$, dV/dt is negative semi-definite with equality at $\mathcal{R}_0 = 1$. In addition, $dV/dt = 0$, $I_1 = I_2 = A = T = 0$. By inserting $I_1 = I_2 = A = T = 0$ into equation (2.2), we obtain $S \to \Lambda/(\mu+\phi)$, $A \to 0$ and $R_0 \to \Lambda\phi/\mu(\mu+\phi)$ as $t \to \infty$. Based on the invariance principle of [17], the DFE is globally asymptotically stable (GAS).

3.2 Endemic equilibrium

3.2.1 Existence of the endemic equilibrium

If $\mathcal{R}_0 > 1$, then the system of the model (2.2) possesses a unique endemic equilibrium at E^*. The system (2.2) can be written in the form

$$\begin{pmatrix} -\lambda_1 & 0 & 0 & 0 & 0 & 0 \\ \lambda_1 & -(\sigma_1+\lambda+\mu) & 0 & 0 & 0 & 0 \\ 0 & \lambda & -(\sigma_2+\mu+\alpha) & 0 & \kappa_2 & 0 \\ 0 & \sigma_1 & \sigma_2 & -(\mu+\delta_1+\kappa_1) & 0 & 0 \\ 0 & 0 & \alpha & \kappa_1 & -a_4 & 0 \\ \phi & 0 & 0 & 0 & 0 & -\mu \end{pmatrix} \begin{pmatrix} S \\ I_1 \\ I_2 \\ A \\ T \\ R \end{pmatrix} = \begin{pmatrix} -\Lambda \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \tag{3.28}$$

where

$$\lambda_1 = -\frac{S\beta}{N}(I_1 + cI_2 + dA) - \mu - \phi.$$

Solving equation (3.28), we obtain

$$S^* = \frac{\mu N}{\mathcal{R}_0(\mu+\phi)}, \tag{3.29}$$

$$I_1^* = \frac{\Lambda\mathcal{R}_0 - \mu N}{a_1\mathcal{R}_0} = \frac{\Lambda\mathcal{R}_0 - \mu N}{(\sigma_1+\lambda+\mu)\mathcal{R}_0}, \tag{3.30}$$

$$\begin{aligned} I_2^* =& \frac{[\Lambda\mathcal{R}_0 - \mu N](\lambda a_3 a_4 + \sigma_1\kappa_1\kappa_2)}{\mathcal{R}_0 a_1(a_2a_3a_4 - \alpha\kappa_2 a_3 - \kappa_1\kappa_2\sigma_2)} \\ =& \frac{[\Lambda\mathcal{R}_0 - \mu N](\lambda(\mu+\delta_1+\kappa_1)(\kappa_2+\mu+\delta_2) + \kappa_1\kappa_2)}{\mathcal{R}_0(\sigma_1+\lambda+\mu)[(\sigma_2+\mu+\alpha)(\mu+\delta_1+\kappa_1)(\kappa_2+\mu+\delta_2) - \kappa_1\kappa_2\sigma_2 - (\mu+\delta_1+\kappa_1)\alpha\kappa_2]} \\ =& \frac{[\Lambda\mathcal{R}_0 - \mu N]\mathcal{R}_2}{c\mathcal{R}_0}, \end{aligned} \tag{3.31}$$

$$\begin{aligned} A^* =& \frac{[\Lambda\mathcal{R}_0 - \mu N](\sigma_1 a_2 a_4 - \sigma_1\kappa_2\alpha + \sigma_2\lambda a_4)}{\mathcal{R}_0 a_1(a_2a_3a_4 - \alpha\kappa_2 a_3 - \kappa_1\kappa_2\sigma_2)} \\ =& \frac{[\Lambda\mathcal{R}_0 - \mu N](\sigma_1(\sigma_2+\mu+\alpha)(\kappa_2+\mu+\delta_2) - \sigma_1\kappa_2\alpha + \sigma_2\alpha(\kappa_2+\mu+\delta_2))}{\mathcal{R}_0(\sigma_1+\lambda+\mu)[(\sigma_2+\mu+\alpha)(\mu+\delta_1+\kappa_1)(\kappa_2+\mu+\delta_2) - \kappa_1\kappa_2\sigma_2 - (\mu+\delta_1+\kappa_1)\alpha\kappa_2]} \\ =& \frac{[\Lambda\mathcal{R}_0 - \mu N^*]\mathcal{R}_3}{d\mathcal{R}_0}, \end{aligned} \tag{3.32}$$

$$T^* = \frac{[\Lambda\mathcal{R}_0 - \mu N]}{\mathcal{R}_0(\kappa_2+\mu+\delta_2)}\left[\frac{\alpha\mathcal{R}_2}{c} + \frac{\kappa_1\mathcal{R}_3}{d}\right] \tag{3.33}$$

and

$$R^* = \frac{\phi}{\mu}S^* = \frac{\phi N}{\mathcal{R}_0(\mu+\phi)}. \tag{3.34}$$

3.2.2 Stability of the endemic equilibrium

Theorem 3.4. *The endemic equilibrium E^* of the model (2.2) is locally asymptotically stable if $\mathcal{R}_0 > 1$.*

Proof. An evaluation of the Jacobian matrix J at the endemic equilibrium E^* gives

$$J(E^*) = \begin{pmatrix} -\beta/N(I_1 + cI_2 + dA) - \mu - \phi & -S^*\beta/N & -cS^*\beta/N & -dS^*\beta/N & 0 & 0 \\ \beta/N(I_1 + cI_2 + dA) & S^*\beta/N - a_1 & cS^*\beta/N & dS^*\beta/N & 0 & 0 \\ 0 & \lambda & -a_2 & 0 & \kappa_2 & 0 \\ 0 & \sigma_1 & \sigma_2 & -a_3 & 0 & 0 \\ 0 & 0 & \alpha & \kappa_1 & -a_4 & 0 \\ \phi & 0 & 0 & 0 & 0 & -\mu \end{pmatrix} \tag{3.35}$$

The matrix $J(E^*)$ has one eigenvalue which is the negative real part, $\lambda = -\mu$. The remaining sub-matrix is given by

$$J_s(E^*) = \begin{pmatrix} -\beta/N(I_1 + cI_2 + dA) - \mu - \phi & -S^*\beta/N & -cS^*\beta/N & -dS^*\beta/N & 0 \\ \beta/N(I_1 + cI_2 + dA) & S^*\beta/N - a_1 & cS^*\beta/N & dS^*\beta/N & 0 \\ 0 & \lambda & -a_2 & 0 & \kappa_2 \\ 0 & \sigma_1 & \sigma_2 & -a_3 & 0 \\ 0 & 0 & \alpha & \kappa_1 & -a_4 \end{pmatrix} \tag{3.36}$$

According to Corollary [1], the matrix $J_s(E^*)$ will have negative eigenvalues if the following inequalities are fulfilled.

$$\beta/N(I_1 + cI_2 + dA) + \mu + \phi > \frac{S^*\beta}{N}(1 + c + d). \tag{3.37a}$$

$$-\frac{S^*\beta}{N} + a_1 > \beta/N(I_1 + cI_2 + dA) + cS^*\beta/N + dS^*\beta/N. \tag{3.37b}$$

$$1 > \frac{\lambda + \kappa_2}{a_2}. \tag{3.37c}$$

$$1 > \frac{\sigma_1 + \sigma_2}{a_3}. \tag{3.37d}$$

$$1 > \frac{\alpha + \kappa_1}{a_4}. \tag{3.37e}$$

By adding the inequalities (3.37a) and (3.37b), we obtain

$$\mu + \phi + a_1 > 2\beta S^*/N(1 + c + d) \tag{3.38}$$

Substituting equation (3.29) into equation (3.38) gives

$$\mathcal{R}_0 > \frac{2\mu\beta(1 + c + d)}{(\mu + \phi)(\mu + \phi + a_1)}. \tag{3.39}$$

Subtracting inequality (3.37c) from inequality (3.39), we obtain

$$\mathcal{R}_0 - 1 > \frac{2\mu\beta(1 + c + d)}{(\mu + \phi)(\mu + \phi + a_1)} - \frac{\lambda + \kappa_2}{a_2}. \tag{3.40}$$

Dividing the above inequality by $\frac{2\mu\beta(1+c+d)}{(\mu+\phi)(\mu+\phi+a_1)} - \frac{\lambda+\kappa_2}{a_2}$ yields the following result:

$$\frac{\mathcal{R}_0 - 1}{\frac{2\beta(1+c+d)}{(\mu+\phi)(\mu+\phi+a_1)} - \frac{\lambda+\kappa_2}{a_2}} > 1 \tag{3.41}$$

Combining inequalities (3.37d) and (3.37e) gives

$$1 > \frac{(\sigma_1+\sigma_2)(\alpha+\kappa_1)}{a_3 a_4}. \tag{3.42}$$

From inequalities(3.41) and (3.42) we have

$$\frac{\mathcal{R}_0 - 1}{\frac{2\mu\beta(1+c+d)}{(\mu+\phi)(\mu+\phi+a_1)} - \frac{\lambda+\kappa_2}{a_2}} > 1 > \frac{(\sigma_1+\sigma_2)(\alpha+\kappa_1)}{a_3 a_4}.$$

It follows that

$$\frac{\mathcal{R}_0 - 1}{\frac{2\mu\beta(1+c+d)}{(\mu+\phi)(\mu+\phi+a_1)} - \frac{\lambda+\kappa_2}{a_2}} > \frac{(\sigma_1+\sigma_2)(\alpha+\kappa_1)}{a_3 a_4}.$$

The above mentioned inequality holds if $\mathcal{R}_0 > 1$. Therefore, the endemic equilibrium point E^* is locally asymptotically stable since $\mathcal{R}_0 > 1$. □

4 Numerical simulations

This section presents some numerical results for the system (2.2) to support the theoretical results obtained in the previous sections using the parameter values provided in Table 3.

4.1 Parameters of the model and initial conditions

We provide HIV/AIDS data for the Democratic Republic of the Congo (DRC) for 2020 as part of our analysis. According to reports [23, 24], we estimate a total population $N(0) = 89$ millions consisting of 0.484 million individuals who were infected with HIV/AIDS in 2020. There were 14000 new infections (or $0,014$ million) as the new infected individuals. Among the remaining infected individuals, there were eighty-two percent of adults over 15 years of age living with HIV who were tested and knew their HIV status, while eighteen percent of those living with HIV were asymptomatic. We assume that only 20% of the patients received ARV treatment. The initial conditions of the population are summarised in Table 2.

Table 2: Initial conditions of the population

Variable	$S(0)$	$I_1(0)$	$I_2(0)$	$A(0)$	$T(0)$	$R(0)$
Value	88. 516	0.014	0.0846	0.30832	0.07708	0

The constant recruitment rate Λ is calculated as follows [30]:

$$\begin{aligned}\Lambda =& \text{Total Population} \times \text{crude birth rate} \times \text{infant survival rate} + \text{migration}\\ =& 89 \times \frac{42.31}{1000} \times \left(1 - \frac{63.6}{1000}\right) + 5.3\\ =& 8.8261\end{aligned} \tag{4.1}$$

Table 3: Parameter values obtained using data from the DRC.

Parameter	Unity	Value	Source
Λ	Population/year	8.8261	Estimate
β	per year	0.15	[24]
μ	per year	0.0166	[24]
ϕ	per year	0.083	[24]
c	per year	0.03	Estimate
d	per year	0.001	Estimate
σ_1	per year	0.0025	[24]
σ_2	per year	0.06	[24]
λ	per year	0.0015	[24]
δ_1	per year	0.0909	[9, 30]
δ_2	per year	0.0667	[9, 30]
α	per year	0.035	[24]
κ_1	per year	0.2	[24]
κ_2	per year	0.04	[24]

The parameter values are given in Table 3.

The parameters c and d denote the respective infection potentials of the infected classes I_2 and A. It is expected that $c > d$ given that individuals in the compartment A are physically weak and not prone to transmit further infections as opposed to individuals in the compartment I_2.

4.2 Sensitivity analysis

To determine the best way to reduce the mortality rate and AIDS-related disease, we will have to use the following approach to calculate the sensitivity indices of the effective reproduction number $\mathcal{R}_0$ to the model parameters [6, 26]. These indices show us how essential each parameter is for the spread and frequency of the disease, while identifying the factors that significantly influence $\mathcal{R}_0$ and that should be targeted by intervention strategies.

Definition 1. The normalized direct sensitivity index of a variable y that depends differentiable on a parameter x is determined as follows:

$$\chi_x^y = \frac{\partial y}{\partial x} \times \frac{x}{y} \tag{4.2}$$

From (4.2), we derive an analytical expression for the sensitivity of $\mathcal{R}_0$ in the following manner:

$$\chi_x^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial x} \times \frac{x}{\mathcal{R}_0} \tag{4.3}$$

to each of the parameters involved in $\mathcal{R}_0$.

For example, the sensitivity index of $\mathcal{R}_0$ with respect to β is given by:

$$\begin{aligned}\chi_{\beta}^{\mathcal{R}_0} =& \frac{S}{N}[\mathcal{R}_1+\mathcal{R}_2+\mathcal{R}_3]\times\frac{\beta}{\mathcal{R}_0}\\ =& \frac{S}{N}[\mathcal{R}_1+\mathcal{R}_2+\mathcal{R}_3]\times\frac{\beta}{\frac{\beta S}{N}[\mathcal{R}_1+\mathcal{R}_2+\mathcal{R}_3]}\\ =& 1.\end{aligned}$$

Other indices are derived using (4.3) with the help of Matlab 2023b and they are ranked from most sensitive to least in Table 4.

Table 4 contains positive and negative sensitivity indices. The indices with positive signs increase the value of $\mathcal{R}_0$ when they are increased, which implies that they increase the endemicity of the disease. Those with negative signs decrease the value of $\mathcal{R}_0$, when they are increased, as indicated in Figure 3, implying that they reduce the endemicity of the disease.

Table 4: Numerical values of the sensitivity indices of $\mathcal{R}_0$

Parameter	Sensitivity Index
β	1
c	6.9397×10^{-4}
κ_2	2.5216×10^{-4}
κ_1	9.0606×10^{-5}
d	1.2638×10^{-5}
λ	-0.0723
σ_1	-0.1212
ϕ	-0.8333
α	-1.8857×10^{-4}
δ_2	-2.0191×10^{-4}
σ_2	-3.7302×10^{-4}
δ_1	-8.701×10^{-5}

The contact rate is the most sensitive parameter because it has the ability to increase $\mathcal{R}_0$ while keeping other parameters constant, as indicated in Figure 2.

Figure 4 demonstrates how $\mathcal{R}_0$ changes with the parameters β and σ_1. When β increases, the value of $\mathcal{R}_0$ also increases, conforming that the effective contact rate has a direct positive impact on the prevalence of the infection. On the other side, an increase in σ_1, results in a decrease in $\mathcal{R}_0$ implying that where more indivuals become less sexually active, thus $\mathcal{R}_0$ reduces. The relation between $\mathcal{R}_0$ and σ_1 is illustrated in Figure 6. This indicates that a greater number of infected individuals recognise their conditions and avoid having sexual intercourse.

A numerical simulation gives $\mathcal{R}_0 = 1.2145 > 1$, as illustrated in Figure 5. Since $\mathcal{R}_0 > 1$, the number of people eventually tends to a constant over time.

A numerical simulation yields $\mathcal{R}_0 = 0.74025 < 1$, when the values of $\phi = 0.09$, $\alpha = 0.05$, $\sigma_1 = 0.0125$, $\sigma_2 = 0.16$ and $\lambda = 0.0025$ increase at the same time as shown in Figure 3 while keeping other parameters constant, then the disease-free equilibrium is globally asymptotically stable. This means that the HIV infection rate eventually decreases when more individuals become sick and display infection symptoms. It also decreases because of the change of sexual habits in the susceptible population. When we only increase the contact $\beta = 0.2$, keeping the

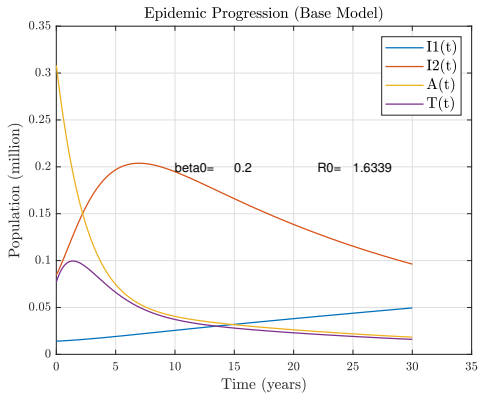

Figure 2: $\mathcal{R}_0 = 1.6339 > 1.2157$.

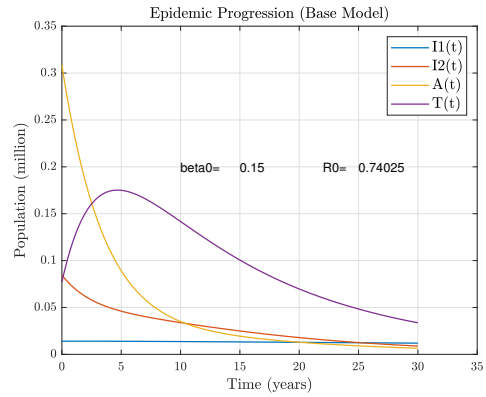

Figure 3: $\mathcal{R}_0 = 0.74025¡1$.

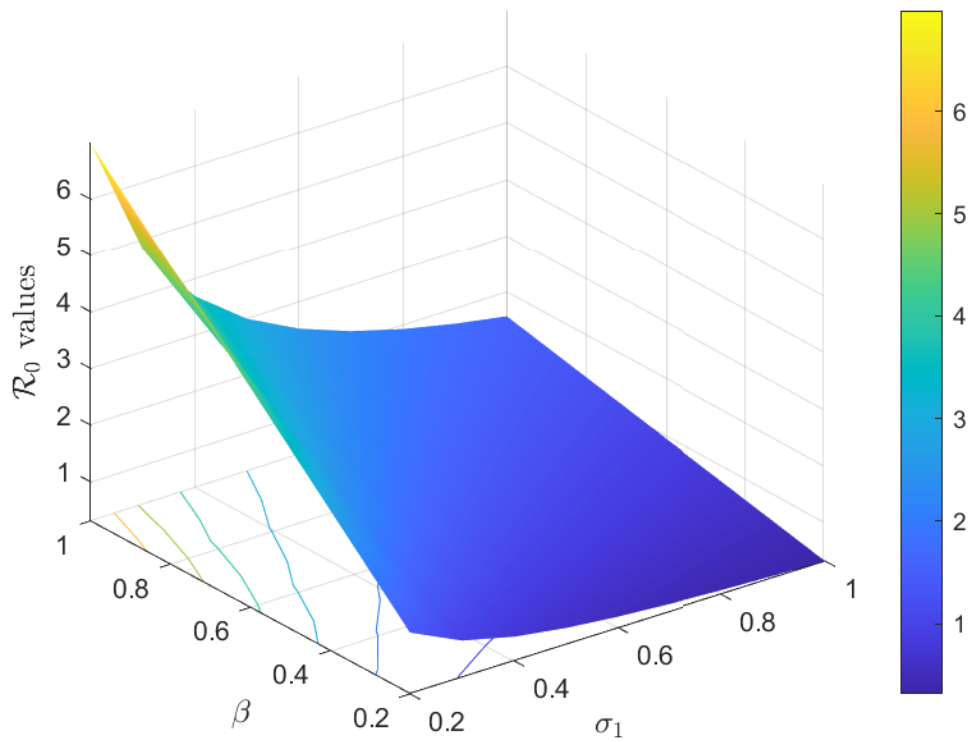

Figure 4: Plot of $\mathcal{R}_0$ against β and σ_1.

other parameters constant, the value of $\mathcal{R}_0$ also increases as indicated in Figure 2, implying the increase of the endemicity of the disease within the population.

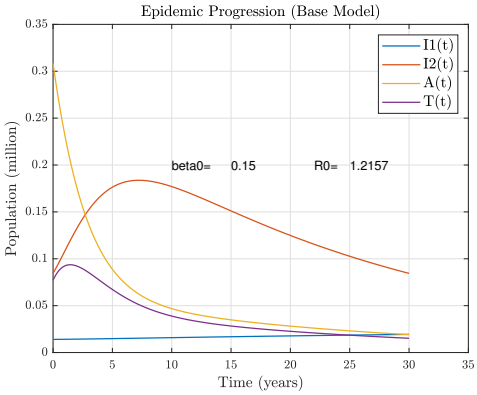

Figure 5: $\mathcal{R}_0 = 1.2157 > 1$.

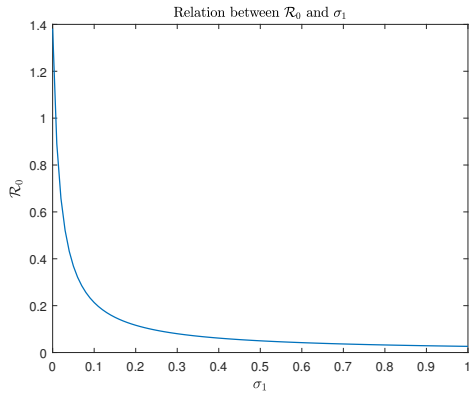

Figure 6: Variation of $\mathcal{R}_0$ with σ_1.

5 Conclusion

In this paper, we presented a model that describes the dynamic HIV/AIDS population in the Democratic Republic of the Congo (DRC), taking into account antiretroviral therapy (ARV) as treatment and susceptible individuals who change sexual habits to avoid contracting the disease. Infected individuals are subdivided into three compartments: infected individuals who are unaware of their HIV status and do not show any symptoms, infected individuals who are aware of their HIV status but do not show any symptoms, and infected individuals who show HIV symptoms. We determined the basic reproduction number $\mathcal{R}_0$ using the next-generation matrix and also analyzed the global dynamics of the model. We showed that disease-free equilibrium is locally asymptotically stable and globally asymptotically stable if $\mathcal{R}_0 < 1$, using a corollary of Gershgorin's circle theorem and the Lyapunov method, respectively. Moreover, the model analysis showed the existence of a unique endemic equilibrium that is locally asymptotically stable if $\mathcal{R}_0 > 1$ using a corollary of Gershgorin's theorem circle. Our results showed that early treatment for infected individuals can reduce disease transmission or keep the basic reproduction number below 1. In addition, it showed that the contact rate parameter is the most sensitive parameter in $\mathcal{R}_0$.

In order to reduce the HIV transmission rate, we recommend that the government of the DRC strengthens public awareness through campaigns on abstinence and safe sex. Additionally, to prolong the life expectancy of infected individuals across the entire country, we recommend universal screening and immediate initiation of antiretroviral treatment after diagnosis.

The data on HIV/AIDS that we have used in this work was reported for the year 2020. We are currently searching the data for 2021-2024 to enable us conduct a model fitting in order to determine confidently those parameters that are currently mere estimates. Results of the model fitting exercise will be reported in an upcoming manuscript.

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