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# A Mathematical Model for the Prevention of HIV/AIDS in the Presence of Undetectable Equals Untransmittable Viral Load

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**Abstract**

Recent advancement in medicine has brought about the use of Antiretroviral Therapy (ART) treatment regime to reduce the viral load of a Human Immunodeficiency Virus (HIV) or Acquired Immune Deficiency Syndrome (AIDS) patients to an Undetectable equals Untransmittable (U=U) level. While half of HIV-positive individuals in the United States have achieved an undetectable viral load, African countries face distinct challenges, including unawareness of the possibility of attaining the U=U viral load. This paper presents a novel mathematical model for HIV/AIDS transmission in Africa, using Cape Verde as a case study, by incorporating the ART treatment, resulting in U=U. The qualitative properties of the model, including the boundedness and positivity of its solution were obtained. Local and global stability analyses of the Disease-Free Equilibrium (DFE) point of the model were performed using the next generation matrix approach and the direct Lyapunov method respectively. The result indicated that the DFE of the model is stable and the disease cannot invade the studied population. The model equations were solved through the implementation of MATLAB ODE45 algorithm and simulations were performed to visualize the effects of the ART on attaining a U=U viral load.

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Values of the parameters which are highly significant to the spread and control of the disease were varied and graphs were obtained to visualize the effects of these variations on each model compartment. Results of the simulations indicate that it is possible to attain a U=U viral load in Africa if the ART treatment is followed religiously. Implementation of the findings of this research will contribute to curbing transmission and strengthening control efforts towards ending the HIV/AIDS epidemic.

**Keywords:** Mathematical Model, Stability Analyses, Human Immunodeficiency Virus, Acquired Immune Deficiency Syndrome, Undetectable Equals Untransmittable.
**MSC2010: 00A71.**

# 1 Introduction

The Human Immunodeficiency Virus (HIV) is a virus that attacks the body's immune system, specifically the CD4 cells (T-cells) whose function is to help the immune system fight off infections, and renders it inactive. Infection with the HIV is very dangerous and fatal if left untreated and/or uncontrolled [1]. Over the years, HIV has become one of the leading global cause of death from an infectious disease. According to the World Health Organization [2], by April 2023 HIV had claimed the lives of 40.1 million individuals. The virus continues to spread in all countries worldwide, with some nations that had previously experienced a decline in transmission now witnessing a resurgence of new infections. The end product of the HIV, if left untreated, is the Acquired Immunodeficiency syndrome (AIDS), generally referred to as the most severe phase of the HIV infection. Individuals living with AIDS experience severely compromised immune systems, leading to a growing occurrence of severe illnesses known as opportunistic infections. The progression of HIV infection moves through stages, starting from acute infectiousness, then entering a phase of clinical latency/chronic infectiousness, and ultimately culminating in AIDS if left untreated [3].
Chimpanzees were known to transmit a version of HIV called simian immunodeficiency virus in the late 1800s. Prior to 1920, HIV remained unknown, and its transmission did not exhibit noticeable symptoms since it was limited to chimpanzees. The virus, however, crossed over from chimpanzees to humans during the 1920s in what is currently known as the Democratic Republic of Congo, following the killing and consumption of chimps by hunters [4]. There is no evidence supporting the possibility of animals such as dogs, cats, and other non-primate animals contracting and transmitting the virus to humans [5]. Prior to the 1980s, no precise records were available regarding the number of individuals infected with HIV or those who developed AIDS. Nonetheless, the virus had already disseminated to five continents, including North America, by that time. The first documented report of HIV was made on June 5, 1981, by the U.S. Centers for Disease Control and Prevention (CDC) [6]. The report documented instances of a rare lung infection, referred to at the time as, Pneumocystis Carinii pneumonia (PCP), affecting five previously healthy young gay men of Caucasian origin in Los Angeles. This devastating infection subsequently came to be recognised as AIDS. Since that time, HIV and AIDS have continued to proliferate globally, with a distressing escalation in fatality rates.
Transmission of the HIV is via contact with some certain body fluids of an infected person or animal. These fluids include the blood, semen, seminal fluid, rectal fluids, vaginal fluids, and breast milk of an infected person or animal. Transmission of the virus, however, is not possible via contact with the tears, sweat or saliva of an infected person, due to its very short lifespan outside the host

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in addition to the fact that it cannot be replicated without a human host [7]. Hence, the transmission of the virus is significantly by sexual contact with an infected individual or from mother to child during pregnancy or breast feeding. Transmission is also possible by the immediate sharing of unsterile sharp or medical objects such as needle, syringe, shaving clippers or blades used by an infected person.

Currently, no effective cure exists against HIV, however, with proper medical care, the virus can be controlled [8]. The major means of controlling the disease include, abstinence from sexual activities, use of condom, treatment of infectives and blood screening. It is important to note that, in order to effectively control the spread of HIV, the susceptible individuals must be prevented from being infected and the already infected individuals must be adequately informed of the available measures to ensure that they do not spread the disease any further. It is to this end that the benefits of an untransmittable status of an HIV infected individual cannot be over-emphasized as it not only help prevent the susceptible population from being infected but it also serves as a lasting and effective treatment for the infected population.

The Antiretroviral Therapy (ART) exists as a combination of three or more Antiretroviral drugs (ARVs) taken by an HIV positive individual in order to block replication (reproduction) of HIV [9]. Suppressing viral replication prevents HIV from infecting and damaging the white blood cells that the body needs to combat infections and diseases. As it stands, an individual diagnosed with HIV and treated with the ART at an early/acute stage can live just about as long as someone who does not have HIV.

The U=U is a shorthand for the campaign launched in 2016 by the Prevention Access Campaign, a health equity initiative with the goal of ending HIV/AIDS pandemic, highlighting new and updated clinical findings that demonstrate that if someone is on ART and has an undetectable viral load for at least six months, then it is not possible to transmit the virus sexually or to one's offspring: hence, Undetectable = Untransmittable (U=U). An undetectable viral load attainment does not mean that a person's HIV is cured but it does mean that the person has so little of the virus in their blood that a test cannot identify it and it cannot be transmitted.

There has been a growing interest among scientists and researchers on how to develop mathematical models that will help in understanding the endemic nature of HIV/AIDS. Igobi et al. [10] developed a mathematical model used to describe the transmission of HIV/AIDS in the presence of a single preventive parameter. In this study, the developed model was such that a single infected individual was introduced into an HIV/AIDS free population and the effects of this introduction was observed for various values of the model parameters. It was found that when there was no contact between infected and non-infected persons, there was no transmission of the virus and when there was a contact, transmission increased and the number of persons infected increased proportionately to the number of contacts made. It was also observed that the number of non-infected persons increased by introducing the preventive measure.

In a similar vein, [11] studied the impact of optimal control on the treatment of HIV/AIDS and screening of unaware infectives. In this paper, the optimal control analysis for HIV/AIDS model was performed. The conditions for optimal control of the disease with effective use of condoms, treatment regime and screening of infectives as control parameters were derived and analyzed. It was observed that the successful screening of unaware infectives has a significant impact on reducing the endemicity of HIV/AIDS. By performing numerical simulations, it was discovered that the impact and implications of unaware infectives in the community is very high.

Furthermore, [12] investigated the epidemiological effects of AIDS and the rate of spread of HIV/AIDS in any given population through the numericalization of the basic reproductive rate of infection

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($R_0$). The study was used to explain the application of deterministic models, classic endemic model (SIR), commercial sex workers (CSW) model, dynamic model and stability analysis on the spread of HIV/AIDS. The models showed that the AIDS disease increases progressively with years and it was thus concluded that if the current trend is unchecked, a catastrophic AIDS epidemic (pandemic) will occur in the near future. Subsequently, [13] investigated the transmission of HIV/AIDS under treatment structured by age of infection. In this study, a mathematical model which is structured according to the age of infection was formulated. By obtaining the equilibria points and performing the stability analyses of these points, it was discovered that the disease can be eradicated from the population only if on average one infected individual infects less than one person (i.e $R_0 < 1$) in his or her infectious period, otherwise the disease will persist.

In a study conducted by [14] on the stability and optimal control of a delayed HIV model, a new model for the optimal control of HIV at cell level which considers not only an intracellular delay but also a pharmacological delay was proposed. Local stability of the equilibria and the extremal control were further investigated. It was found that the extremal control for the optimal control problem attains alternately the boundary values 0 and 1. It was then suggested that this type of control is easier to implement, from a medical point of view, and leads to better results for a non-delayed problem.

Meanwhile, [15] studied the effects of undetectable viral load in association with sexual risk by considering HIV serodiscordant gay couples in Sydney as case study. It was discovered that approximately 40% of HIV serodiscordant gay couples in Sydney engaged in some unprotected anal intercourse when the HIV-positive partner's reported viral load is undetectable while approximately 20% of HIV serodiscordant gay couples engaged in some unprotected anal intercourse when the HIV-positive partner's reported viral load is detectable. Interestingly, according to a 2017 CDC report by [16], of the estimated 1.1 million people living with HIV in the United States, 85% were diagnosed and knew they had HIV while 50% have attained an undetectable viral load. The same report, however, can not be given about developing African countries such as Cape Verde whose health care facility and availability is known to be significantly limited with medical facilities unavailable and some medicines in short supply. Due to this reason, the U=U viral load attainment has not been reported in this country since its inception in 2016 with 50% of the HIV cases in the country between 25 and 49 years old and also among teens [17].

[18] studied a SICA compartmental model in epidemiology with application to HIV/AIDS in Cape Verde. In this paper, a nonlinear mathematical model for HIV/AIDS transmission with varying population size in a homogeneously mixed population was proposed. The developed model was analyzed in such a way that only HIV-infected individuals with no AIDS symptoms and who are not under ART treatment transmit HIV. It was observed that the number of individuals with HIV-infection and AIDS disease was increasingly big and does not converge to the Joint United Nations Programme on HIV/AIDS (UNAIDS) worldwide goal of ending the AIDS epidemic by 2030. It was also observed that the most significant parameter to the basic reproduction number, $R_0$, was the disease transmission rate. It was therefore concluded that it is important to invest in providing conditions to HIV-infected individuals to correctly maintain the ART treatment.

As a follow up on this study, silva2017modeling made an optimal control application on the developed HIV/AIDS model with prevention through pre-exposure prophylaxis (PrEP). PrEP is a HIV-prevention strategy in which an HIV-negative individual takes an oral pill once a day to reduce the possibility of HIV infection contraction if they come in contact with an infected individual. In this study, two different values for the relative infectiousness of HIV-infected individuals under antiretroviral treatment were considered. Through numerical simulations, it was demonstrated that

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the use of PrEP helps reduce HIV transmission significantly but does not lead to the eradication of the virus.
The aim of this paper, therefore, is to extend the SICA model with PrEP in [19] to include treatment of infected individuals with ART such as to attain a viable Undetectable=Untransmittable viral load for HIV/AIDS. The application of mathematical modeling, analyses and simulations in predicting and measuring the effect of ART, as a preventive measure, on the eradication of HIV/AIDS shall be made using Cape Verde as a case study. This work is commensurate to achieving the sustainable development goal of good health and wellbeing and also the UNAIDS worldwide goal of ending the AIDS epidemic by 2030.

## 2 Model Description and Formulation

Mathematical modeling is one of the most important tools used in understanding the dynamics of disease transmission and control. In this paper, we considered a five mutually exclusive compartments in relation to HIV/AIDS disease transmission in Cape Verde. These compartments are: the Susceptible compartment; the infected (acute stage) compartment; the infected (chronic stage) compartment; the AIDS (fully blown infected) compartment and the Treated compartment; thereafter referred to as SICAT.
The susceptible compartment consist of individuals who have a chance/probability of contracting the infection because they live within a population in which the virus exists or has been previously reported. The infected (acute stage) compartment consists of individuals who are at the earliest stage or onset of the HIV infectiousness. At this stage, an infected individual may show some flu-like symptoms of infectiousness which are not unique to HIV. During this stage, the level of HIV in the blood is very high.
The infected (chronic stage) compartment is made up of individuals who are at the chronic stage of the HIV infectiousness i.e they have survived the first 2-4 weeks of the infection onset. During this stage, an individual may not show any clinical/noticeable symptoms of the infection even though they are infectious, hence this stage is referred to as the clinical latency period. The AIDS is the final and most severe stage of the HIV infection. The AIDS (fully blown infected) compartment consist of individuals with AIDS clinical symptoms. These are individuals who may not have received any treatment against the disease or whose treatment was not effective. At this stage, it is difficult for the body to fight off opportunistic infections. Lastly, the treated compartment is made up of infected individuals who have been isolated from the other members of the studied population and are being treated with ART. The treated individuals are therefore not infectious. Table 1 below represents the five mutually exclusive compartments (state variables) of the model together with their mathematical representations.

| Variables | Description |
|---|---|
| $S(t)$ | Susceptible Population |
| $I(t)$ | Infected (acute) Population |
| $C(t)$ | Infected (chronic) Population |
| $A(t)$ | AIDS (fully blown infected) Population |
| $T(t)$ | Treated Population |

Table 1: The SICAT Model State Variables

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| Parameters | Description |
|---|---|
| $\alpha$ | Recruitment rate into the susceptible population |
| $\mu$ | Natural death rate of the entire population |
| $\beta_1$ | HIV transmission rate via interaction with the infected (acute) population |
| $\beta_2$ | HIV transmission rate via interaction with the infected (chronic) population |
| $\beta_3$ | HIV transmission rate via interaction with the AIDS-infected population |
| $\delta_1$ | Disease-induced death rate of the infected (acute) population |
| $\delta_2$ | Disease-induced death rate of the infected (chronic) population |
| $\delta_3$ | Disease-induced death rate of the AIDs-infected population |
| $\delta$ | Average disease-induced death rate of the population |
| $\gamma$ | Rate of progression from the infected (acute) to the infected (chronic) population |
| $\sigma$ | Rate of progression from the infected (chronic) to the AIDS-infected population |
| $u_1$ | Treatment Rate of the infected (acute) population |
| $u_2$ | Treatment Rate of the infected (chronic) population |
| $u_3$ | Treatment Rate of the AIDS-infected population |
| $\omega$ | Treatment efficacy |

**Model Assumptions**

1. Members of the treated population are not infectious.
2. Treatment administered is strictly ART leading to a U=U state.
3. Each compartment in the model is made up of individuals with homogeneous characteristic (the disease status).
4. Transmission of HIV is strictly via sexual interactions and by mother-to-child during pregnancy or breastfeeding.
5. Only human-to-human transmission of HIV is considered.

Figure 1 is a diagrammatic expression of the transmission and treatment processes of HIV/AIDS between all 5 model compartments.

**The SICAT Model**

$$\frac{dS}{dt} = \alpha - \beta_1 IS - \beta_2 CS - \beta_3 AS - \mu S \tag{2.1}$$

$$\frac{dI}{dt} = (\beta_1 I + \beta_2 C + \beta_3 A)S - \gamma I - u_1 I - \delta_1 I - \mu I \tag{2.2}$$

$$\frac{dC}{dt} = \gamma I - \sigma C - u_2 C - \delta_2 C - \mu C \tag{2.3}$$

$$\frac{dA}{dt} = \sigma C - u_3 A - \delta_3 A - \mu A \tag{2.4}$$

$$\frac{dT}{dt} = u_1 I + u_2 C + u_3 A - (1-\omega)\delta T - \mu T \tag{2.5}$$

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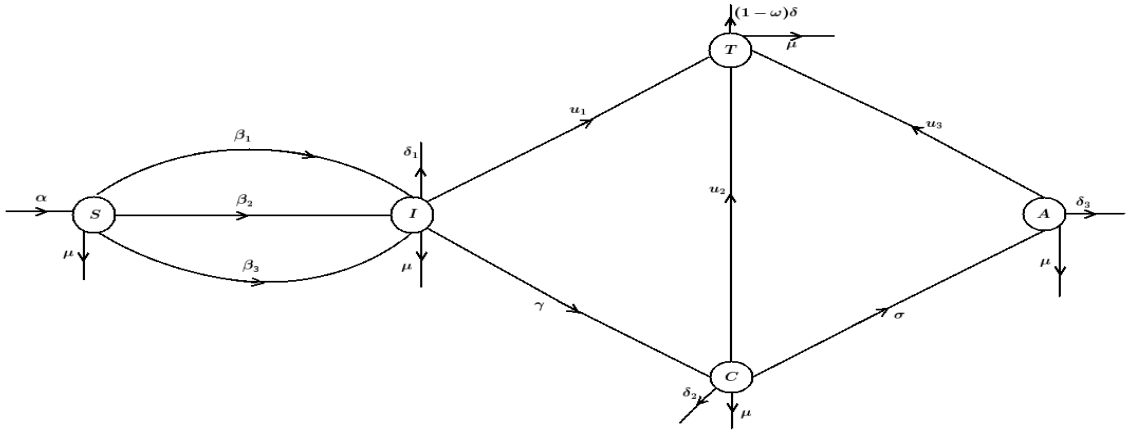


Figure 1: Schematic flow diagram of the model

# 3 The Model Analyses

## 3.1 The Invariant Region

The Invariant Region of the developed model refers to a region within which the solutions to the model are uniformly bounded as a set $\Omega \subset R^5$. The total human population is defined as $N(t) = S(t) + I(t) + C(t) + A(t) + T(t)$ such that $\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dC}{dt} + \frac{dA}{dt} + \frac{dT}{dt}$. From equations 2.1 to 2.5;

$$\frac{dN}{dt} = \alpha - \mu N - \delta_1 I - \delta_2 C - \delta_3 A - (1-\omega)\delta T$$

Thus,

$$\frac{dN}{dt} \leq \alpha - \mu N \tag{3.1}$$

Integrating both sides of (3.1), we have;

$$\frac{-1}{\mu} \ln(\alpha - \mu N) \leq t + K$$

where K is a constant of integration. Therefore,

$$(\alpha - \mu N) \geq K_1 e^{-\mu t}$$

where $K_1 = e^{\mu K}$ is a constant of integration.
Let $N(t=0) = N_0$, then;

$$(\alpha - \mu N_0) \geq K_1$$

Accordingly,

$$(\alpha - \mu N) \geq (\alpha - \mu N_0)e^{-\mu t} \implies N(t) \leq \frac{\alpha}{\mu} - \frac{(\alpha - \mu N_0)}{\mu} e^{-\mu t}$$
$$\implies N(t) \to \frac{\alpha}{\mu} \quad as \quad t \to \infty$$

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Thus, $N(t) \in [0, \frac{\alpha}{\mu}]$.
Hence, the feasible set of the solution of the system equations of the model enter and remain in the region:

$$\Omega = \{(S, I, C, A, T) \in R_+^5 \quad : N(t) \leq \frac{\alpha}{\mu}\} \tag{3.2}$$

## 3.2 Positivity of the Solution

**The Positivity Theorem:**
Let $\Omega_1 = \{(S, I, C, A, T) \in R_+^5 : S_0 > 0, I_0 > 0, C_0 > 0, A_0 > 0, T_0 > 0\}$, then the solution of $\{S, I, C, A, T\}$ are positive for $t \geq 0$.
**Proof:**
Considering equation 2.1,

$$\begin{aligned} \frac{dS}{dt} &= \alpha - (\beta_1 I + \beta_2 C + \beta_3 A + \mu)S \\ &\geq -(\beta_1 I + \beta_2 C + \beta_3 A + \mu)S \end{aligned}$$

thus,

$$\begin{aligned} \int \frac{dS}{S} \geq -\int (\beta_1 I + \beta_2 C + \beta_3 A + \mu) &\implies \ln S(t) \geq -A(t) + K_2 \\ &\implies S(t) \geq Be^{-A(t)} \end{aligned}$$

Where $A(t) = \int (\beta_1 I + \beta_2 C + \beta_3 A + \mu)dt$ and $K_2$ is a constant of integration.
At $t = 0, S_0 > 0 \implies B = e^{K_2} \geq 0$
Accordingly,

$$S(t) \geq S_0 e^{-A(t)} \geq 0 \quad , \forall \ t \geq 0 \tag{3.3}$$

Similarly, considering equation 2.2,

$$\begin{aligned} \frac{dI}{dt} &= (\beta_1 I + \beta_2 C + \beta_3 A + \mu)S - \gamma I - u_1 I - \delta_1 I - \mu I \\ &\geq -(\gamma + u_1 + \delta_1 + \mu)I \end{aligned}$$

Thus,

$$\begin{aligned} \int \frac{dI}{I} \geq -\int (\gamma + u_1 + \delta_1 + \mu I)dt &\implies \ln I(t) \geq -(\gamma + u_1 + \delta_1 + \mu I)t + D, (\text{with } D \text{ a constant}) \\ &\implies I(t) \geq Be^{-At} \end{aligned}$$

where $A = (\gamma + u_1 + \delta_1 + \mu I) \geq 0$
At $t = 0, C_0 > 0 \implies B = e^D \geq 0$
Accordingly,

$$I(t) \geq I_0 e^{-At} \geq 0 \ \forall \ t \geq 0 \tag{3.4}$$

Next, considering equation 2.3,

$$\frac{dC}{dt} = \gamma I - \sigma C - u_2 C - \delta_2 C - \mu C$$

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$$\geq -(\sigma + u_2 + \delta_2 + \mu)C$$

At $t = 0, C(t = 0) = C_0 > 0$. Hence,

$$C(t) \geq C_0 e^{-At} \geq 0 \ \ \forall \ \ t \geq 0 \tag{3.5}$$

where $A = (\sigma + u_2 + \delta_2 + \mu) \geq 0$
In the same way, considering equation 2.4 we have;

$$\frac{dA}{dt} = \sigma C - u_3 A - \delta_3 A - \mu A$$

$$\geq -(u_3 + \delta_3 + \mu)A$$

At $t = 0, A(t = 0) = A_0 > 0$. Therefore,

$$A(t) \geq A_0 e^{-Dt} \geq 0 \ \ \forall \ \ t \geq 0 \tag{3.6}$$

Where $D = (u_3 + \delta_3 + \mu) \geq 0$
Lastly, considering equation 2.5, we have;

$$\frac{dT}{dt} = u_1 I + u_2 C + u_3 A - (1 - \omega)\delta T - \mu T$$

$$\geq (\omega_1 + \mu)T$$

Where $w_1 = (1 - \omega)$. At $t = 0, T(t = 0) = T_0 > 0$.

$$\implies T(t) \geq T_0 e^{-At} \geq 0 \ \ , \forall \ \ t \geq 0 \tag{3.7}$$

Where $A = (\omega_1 + \mu) \geq 0$
This completes the proof of the theorem.

## 3.3 The Equilibrium Points

**The Disease Free Equilibrium (DFE) Point**
The DFE of the model is defined as $(S^*(t), 0, 0, 0, 0)$ satisfying

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dC}{dt} = \frac{dA}{dt} = \frac{dT}{dt} = 0$$

By equating equations (2.1) to (2.5) to 0 and substituting $I = C = A = T = 0$, We obtain $S^* = \frac{\alpha}{\mu}$
Accordingly, the DFE of the model is defined as:

$$E_0 = (S^*, 0, 0, 0, 0) \tag{3.8}$$

**The Endemic Equilibrium Point (EEP)** The EEP of the model is defined as $(S^*(t), C_h^*(t), I_h^*(t), A^*(t), T^*(t))$ satisfying

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dC}{dt} = \frac{dA}{dt} = \frac{dT}{dt} = 0$$

Equating equation (2.3) to 0, we obtain:

$$C^* = \frac{\gamma I^*}{Z} \tag{3.9}$$

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where $Z = \sigma + u2 + \delta_2 + \mu$
Similarly, equating equation (2.4) to 0, we obtain:

$$\begin{aligned} A^* &= \frac{\sigma C^*}{B} \\ &= \frac{\sigma\gamma I^*}{BZ} \end{aligned} \tag{3.10}$$

where $B = u_3 + \delta_3 + \mu$
Similarly, equating equation (2.5) to 0, we obtain:

$$\begin{aligned} T^* &= \frac{u_1 I^* + u_2 C^* + u_3 A^*}{Y} \\ &= \frac{DI^*}{BZY} \end{aligned} \tag{3.11}$$

where $Y = b\delta + \mu \quad , b = 1 - \omega$ and $\quad D = u_1 BZ + u_2\gamma B + u_3\sigma\gamma$
Next, equating equation (2.1) to 0, we obtain:

$$\begin{aligned} A^* &= \frac{\sigma C^*}{B} \\ S^* &= \frac{\alpha}{\beta_1 I^* + \beta_2 C^* + \beta_3 A^* + \mu} \end{aligned} \tag{3.12}$$

Substituting equations 3.9 and 3.10 into 3.12, we obtain:

$$S^* = \frac{\alpha BZ}{EI^* + \mu BZ} \tag{3.13}$$

where $E = \beta_1 BZ + \beta_2\gamma B + \beta_3\sigma\gamma$. Lastly, equating equation (2.2) to 0, we obtain:

$$S^* = \frac{FI^*}{\beta_1 I^* + \beta_2 C^* + \beta_3 A^*} \tag{3.14}$$

where $F = \gamma + u_1 + \delta_1\mu$
Substituting equations 3.9 and 3.10 into 3.14, we obtain:

$$S^* = \frac{BZF}{\beta_1 BZ + \beta_2 B\gamma + \beta_3\sigma\gamma} \tag{3.15}$$

Clearly, all the state variables, $(S^*, I^*, C^*, A^*, T^*)$, are strictly positive, thus there exist an EEP, $E^* = (S^*(t), C_h^*(t), I_h^*(t), A^*(t), T^*(t))$.

## 3.4 Local Asymptotic Stability Analysis

**The Basic Reproduction Number**
According to the principle of next generation matrix, the basic reproduction number is the spectral radius of the next generation matrix $FV^{-1}$ of the system (2.1) to (2.5) [20]. The infectious classes are defined as:

$$f_i - v_i = \begin{pmatrix} I' \\ C' \\ A' \end{pmatrix}$$

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$$= \begin{pmatrix} (\beta_1 I + \beta_2 C + \beta_3 A)S - (\gamma + u_1 + \delta_1 + \mu)I \\ \gamma I - \sigma C - u_2 C - \delta_2 C - \mu C \\ \sigma C - u_3 A - \delta_3 A - \mu A \end{pmatrix} \tag{3.16}$$

where,

$$f_i = \begin{pmatrix} (\beta_1 I + \beta_2 C + \beta_3 A)S \\ 0 \\ 0 \end{pmatrix} \tag{3.17}$$

and:

$$v_i = \begin{pmatrix} (\gamma + u_1 + \delta_1 + \mu)I \\ (\sigma + u_2 + \delta_2 + \mu)C - \gamma I \\ (u_3 + \delta_3 + \mu)A - \sigma C \end{pmatrix} \tag{3.18}$$

where $f_i$ is the rate of appearance of new infection(s) in compartment i and $v_i$ represents the rate of transfer of individuals into compartment i, with $i \in [1, 3]$.
The matrix F and V are obtained as follows:

$$F = \begin{pmatrix} \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial C} & \frac{\partial f_1}{\partial A} \\ \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial C} & \frac{\partial f_2}{\partial A} \\ \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial C} & \frac{\partial f_3}{\partial A} \end{pmatrix}$$

$$= \begin{pmatrix} \beta_1 S & \beta_2 S & \beta_3 S \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{3.19}$$

and;

$$V = \begin{pmatrix} \frac{\partial v_1}{\partial I} & \frac{\partial v_1}{\partial C} & \frac{\partial v_1}{\partial A} \\ \frac{\partial v_2}{\partial I} & \frac{\partial v_2}{\partial C} & \frac{\partial v_2}{\partial A} \\ \frac{\partial v_3}{\partial I} & \frac{\partial v_3}{\partial C} & \frac{\partial v_3}{\partial A} \end{pmatrix}$$

$$= \begin{pmatrix} (\gamma + u_1 + \delta_1 + \mu) & 0 & 0 \\ -\gamma & (\sigma + u_2 + \delta_2 + \mu) & 0 \\ 0 & -\sigma & (u_3 + \delta_3 + \mu) \end{pmatrix} \tag{3.20}$$

Lastly, $V^{-1} = \begin{pmatrix} \frac{1}{\delta_1+\gamma+\mu+u_1} & 0 & 0 \\ \frac{\gamma}{(\delta_1+\gamma+\mu+u_1)(\delta_2+\mu+\sigma+u_2)} & \frac{1}{\delta_2+\mu+\sigma+u_2} & 0 \\ \frac{\gamma\sigma}{(\delta_1+\gamma+\mu+u_1)(\delta_2+\mu+\sigma+u_2)(\delta_3+\mu+u_3)} & \frac{\sigma}{(\delta_2+\mu+\sigma+u_2)(\delta_3+\mu+u_3)} & \frac{1}{\delta_3+\mu+u_3} \end{pmatrix}$

Thus, the next generation matrix: $G = FV^{-1}$

$$= \begin{pmatrix} \frac{S\beta_1}{\delta_1+\gamma+\mu+u_1} + \frac{S\beta_2\gamma}{(\delta_1+\gamma+\mu+u_1)(\delta_2+\mu+\sigma+u_2)} + \frac{S\beta_3\gamma\sigma}{(\delta_1+\gamma+\mu+u_1)(\delta_2+\mu+\sigma+u_2)(\delta_3+\mu+u_3)} & \frac{S\beta_2}{\delta_2+\mu+\sigma+u_2} + \frac{S\beta_3\sigma}{(\delta_2+\mu+\sigma+u_2)(\delta_3+\mu+u_3)} & \frac{S\beta_3}{\delta_3+\mu+u_3} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

The eigenvalues of the matrix $G$, are:

$$\left[\frac{S^*\beta_1}{\delta_1+\gamma+\mu+u_1} + \frac{S^*\beta_2\gamma}{(\delta_1+\gamma+\mu+u_1)(\delta_2+\mu+\sigma+u_2)} + \frac{S^*\beta_3\gamma\sigma}{(\delta_1+\gamma+\mu+u_1)(\delta_2+\mu+\sigma+u_2)(\delta_3+\mu+u_3)}, 0, 0\right]$$

Hence, the spectral radius of G is;

$$R_0 = R_1 + R_2 + R_3$$

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where,

$$R_1 = \frac{S^*\beta_1}{\delta_1 + \gamma + \mu + u_1}$$
$$R_2 = \frac{S^*\beta_2\gamma}{(\delta_1 + \gamma + \mu + u_1)(\delta_2 + \mu + \sigma + u_2)}$$
$$R_3 = \frac{S^*\beta_3\gamma\sigma}{(\delta_1 + \gamma + \mu + u_1)(\delta_2 + \mu + \sigma + u_2)(\delta_3 + \mu + u_3)}$$

Accordingly, by substituting the values in table (2) below the basic reproduction number;

$$R_0 = 0.791630939 \tag{3.21}$$

**Table of values** According to the principle of next generation matrix, the DFE of an infectious

Table 2: Table of Values

| Parameters | Values | Sources |
|---|---|---|
| $\alpha$ | 0.0316357 | [18] |
| $\mu$ | 0.0001667 | Estimate |
| $\beta_1$ | 0.0005 | [21] |
| $\beta_2$ | 0.00015 | [21] |
| $\beta_3$ | 0.00012 | Estimate |
| $\delta_1$ | 0.00115 | Estimate |
| $\delta_2$ | 0.0115 | Estimate |
| $\delta_3$ | 0.115 | [13] |
| $\delta$ | 0.04255 | Estimate |
| $\gamma$ | 0.167 | [13] |
| $\sigma$ | 0.1 | [11] |
| $u_1$ | 0.01 | [21] |
| $u_2$ | 0.01 | [21] |
| $u_3$ | 0.33 | [18] |
| $\omega$ | 0.45 | Estimate |

disease is locally asymptotically stable if the basic reproduction number, $R_0 < 1$ and the EEP is unstable. Hence, the DFE of the developed HIV/AIDS model is stable and thus the disease can not invade the population.

## 3.5 Global Asymptotic Stability Analysis

In order to examine the DFE for global stability, we employed the Lyapunov stability procedure [22, 23]. The developed HIV/AIDS model was denoted by:

$$\begin{cases} \frac{dX}{dt} = F(X,Y) \\ \frac{dY}{dt} = G(X,Y) \end{cases} \tag{3.22}$$

Where $X = (S)$ denotes the uninfected population and $Y = (I, C, A, T)$ denotes the Infected population.
The point $E_0 = (X^*, 0)$ is said to be globally asymptotically stable if $R_0 < 1$ and in addition the following two conditions hold:

C1: For $\frac{dX}{dt} = F(X, 0)$, $E_0$ is globally asymptotically stable.

C2: $G(X,Y) = AY - G^*(X,Y)$ , $G^*(X,Y) \geq 0$ for $(X,Y) \in \Omega$

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**C1:**

$$F(X,0) = \alpha - \mu S \tag{3.23}$$

Clearly, $E_0 = (\frac{\alpha}{\mu}, 0, 0, 0, 0)$ is globally asymptotically stable for $\frac{dX}{dt} = F(X,0)$. This can be verified as shown below: By solving equation (3.23) using the method of separation of variables, we have:

$$\begin{aligned} \frac{dS}{dt} = \alpha - \mu S \implies \int \frac{dS}{\alpha - \mu S} &= \int dt \\ \implies -\frac{1}{\mu}\ln(\alpha - \mu S) &= t + C \end{aligned}$$

where C is a constant of integration.

Accordingly,

$$-\ln(\alpha - \mu S) = \mu t + C$$

Thus

$$\mu S = \alpha - Ae^{-\mu t}$$

where A is a constant of integration. Hence,

$$S = \frac{\alpha}{\mu} - Ae^{-\mu t} \implies S \to \frac{\alpha}{\mu} \quad as \quad t \to \infty$$

and this implies the global convergence of 3.23 in $\Omega$.

**C2:**

$$G(X,Y) = AY - G^*(X,Y) = \begin{cases} (\beta_1 I + \beta_2 C + \beta_3 A)S - (\gamma + u_1 + \delta_1 + \mu)I \\ \gamma I - (\sigma + u_2 + \delta_2 + \mu)C \\ \sigma C - (u_3 + \delta_3 + \mu)A \\ u_1 I + u_2 C + u_3 A - (b\delta + \mu)T \end{cases} \tag{3.24}$$

Where:

$$A = \begin{pmatrix} -(\gamma + u_1 + \delta_1 + \mu) & 0 & 0 & 0 \\ \gamma & -(\sigma + u_2 + \delta_2 + \mu) & 0 & 0 \\ 0 & \sigma & -(u_3 + \delta_3 + \mu) & 0 \\ u_1 & u_2 & u_3 & -(b\delta + \mu) \end{pmatrix}$$

and;

$$G^*(X,Y) = \begin{bmatrix} -(\beta_1 I + \beta_2 C + \beta_3 A)S \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

Clearly, $G^*(X,Y) \leq 0$. Hence, condition 2 is not satisfied. Thus, $E_0 = (X^*, 0)$ may not be GAS for $R_0 < 1$.

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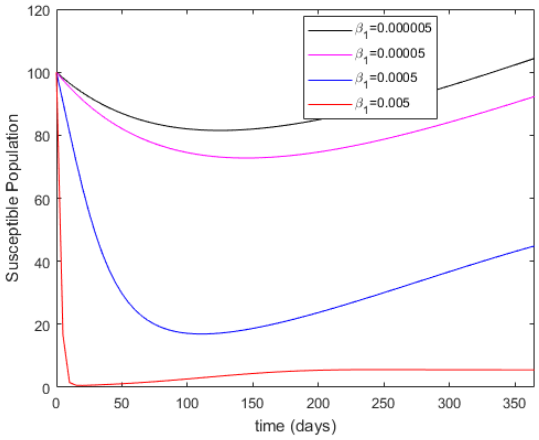
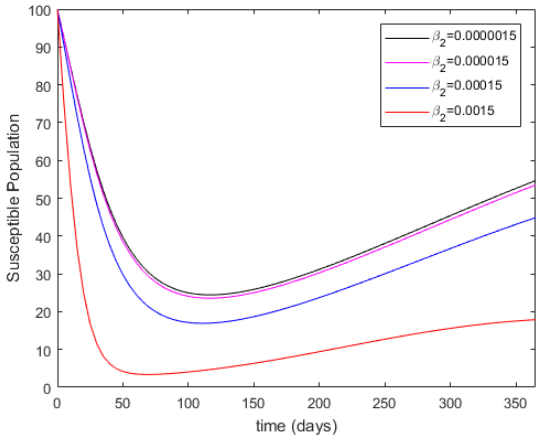
# 4 Results and Discussion

In order to solve the model equations numerically, we implemented the MATLAB ODE45 algorithm for the developed model, and plotted the graphs of each model compartment against time, with time ranging from 0 to 365 days. The following results were obtained:

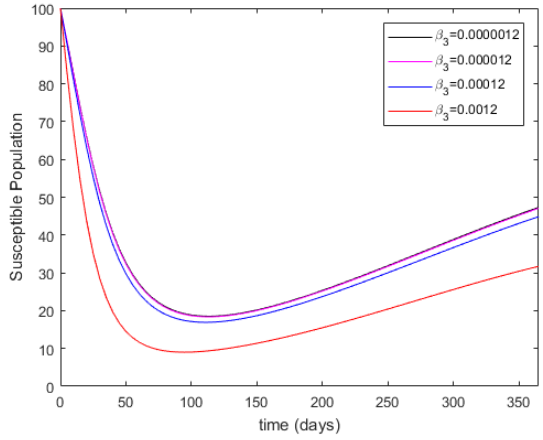
## 4.1 The Susceptible Population




(a) Susceptible Population against Time varying $\beta_1$




(b) Susceptible Population against Time varying $\beta_2$




(c) Susceptible Population against Time varying $\beta_3$

Figure 2: Effects of the transmission rates, $\beta s$, on the Susceptible Population over Time

Figure 2 represents the behavior of the Susceptible population over a period of 365 days. The

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Susceptible population is seen to experience a continuous decrease in population during the first 100 days. This decrease can be associated to the progression of people out of this class into the infected (acute) class at the rates $\beta_1$, $\beta_2$ and $\beta_3$. Figure 2a indicates that an increase in the transmission rate of HIV via interaction with the infected (acute) population leads to a faster decrease in the population of the susceptible class compared to an increase in its transmission rate via interaction with the infected (chronic) population or with the AIDS (fully blown infected) population as indicated in Figures 2b and 2c respectively. These imply that the infected (acute) population are relatively more infectious than the two other infected classes.
After about 100 days, however, the susceptible class is seen to experience a steady increase in it's size due to the recruitment of individuals into this class from outside the studied population. It can be observed that a decrease in $\beta_1$ leads to a faster increase in the population of this class compared to a decrease in $\beta_2$ and $\beta_3$ which gives a more steady and consistent increase in this class. We can also observe that the susceptible population does not increase up to its initial size over a period of one year because of the relatively low recruitment rate as the outside population becomes more aware of the presence of the virus within the studied population.

## 4.2 The Infected (acute) Population

Figure 3 represents the behavior of the Infected (acute) Population over time. The infected (acute) population is seen to experience a slight increase in its population during the first 50 days due to the population of this class by the susceptible population. Figure 3a indicates that an increase in the transmission rate of HIV via interaction with the infected (acute) population, $\beta_1$, leads to an increase in the rate at which the population of the Infected (acute) class increases. However, Figures 3b and 3c indicate that an increase in the rate of progression from the infected (acute) to the infected (chronic), $\gamma$, and in the treatment rate of the infected (acute) population, $u_1$, lead to a downward slope in the rate at which the population of the Infected (acute) class increases.
Subsequently, after a period of about 50 days, the infected (acute) class is seen to undergo a decrease in its population. This decrease can be associated with the effectiveness of the ART treatment given to the members of this class and also the recruitment of the members of this population into the infected (chronic) population. It can be seen that, at the end of 1 year, members of the infected (acute) population has dropped to a relatively low number ($\leq 10$) as most of them are now receiving the ART treatment and are thus undetectable and untransmittable. It can also be seen from 3c that the higher the treatment rate of this population the lesser the number of individuals in the population over time. Hence, it can be concluded that the ART treatment, is in fact, efficacious and that an increase in its availability to the infected (acute) class will lead to a drastic drop in the member of this population over a period of time.

## 4.3 Infected (chronic) Population

Figure 4 represents the behavior of the infected (chronic) population against time. The infected (chronic) population is seen to experience a slight increase in its population during the first 70 days due to the population of this class by the infected (acute) class. Figure 4a indicates that an increase in the transmission rate of HIV via interaction with the infected (chronic) population, $\beta_2$, leads to a slight increase in the rate at which the population of the Infected (chronic) class increases. However, Figures 4b and 4c indicate that an increase in the rate of progression from the infected (chronic) to the AIDS (fully blown infected) class, $\sigma$, and in the treatment rate of the infected

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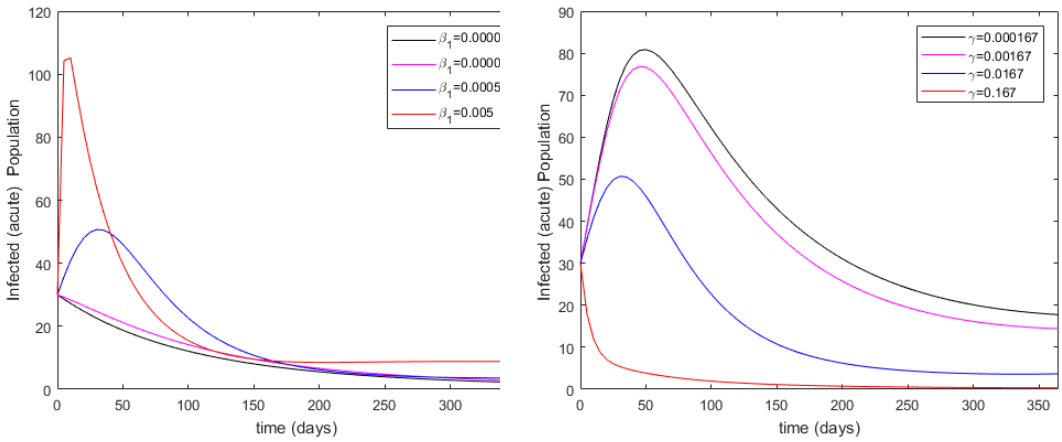
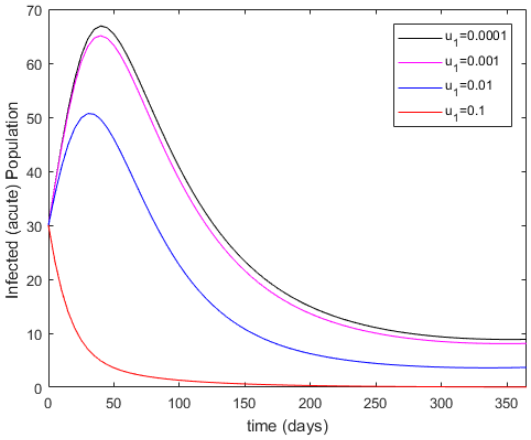



(a) Infected (acute) Population against Time varying $\beta_1$ (b) Infected (acute) Population against Time varying $\gamma$




(c) Infected (acute) Population against Time varying $u_1$

Figure 3: Effects of some parameters on the Infected (acute) Population over Time

(chronic) population, $u_2$, lead to a decrease in the rate at which the population of the Infected (chronic) class increases. It can also be observed from Figure 4d that an increase in the rate of progression from the infected (acute) to the infected (chronic) population, $\gamma$, results in an increase in the population of the infected (chronic) class.
Subsequently, after this period of about 70 to 80 days, the infected (chronic) class is seen to undergo a decrease in its population. This decrease can be associated with the effectiveness of the ART

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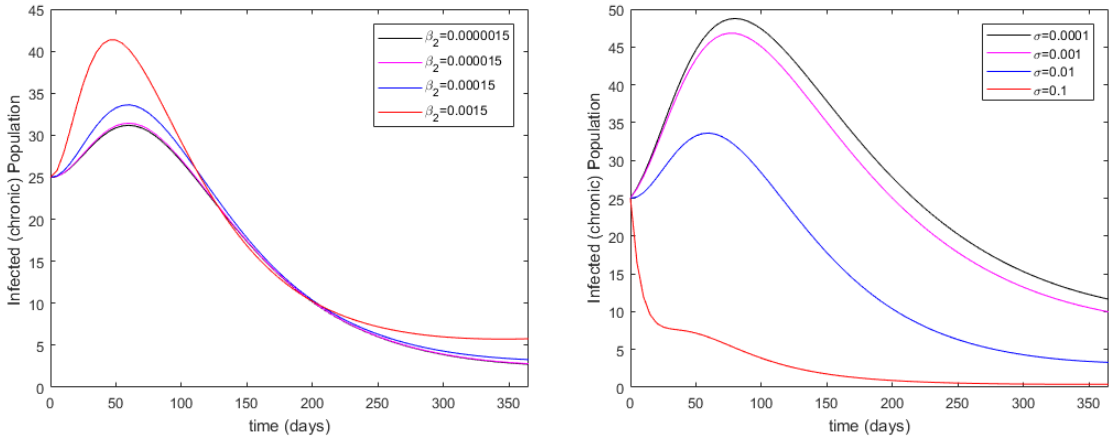
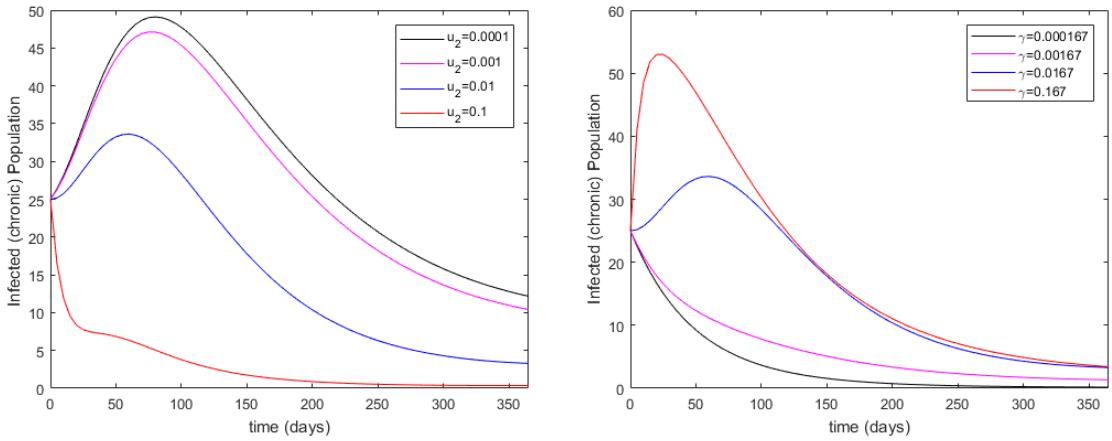



(a) Infected (chronic) Population against Time varying $\beta_2$ (b) Infected (chronic) Population against Time varying $\sigma$




(c) Infected (chronic) Population against Time varying $u_2$ (d) Infected (chronic) Population against Time varying $\gamma$

Figure 4: Effects of some parameters on the Infected (chronic) Population over Time

treatment given to the members of this class and also because of the recruitment of the members of this population into the AIDS (fully blown infected) population. It can be seen that, at the end of 1 year, the members of the infected (chronic) population has dropped to a relatively low number ($\leq 15$) as most of them are now receiving the ART treatment and are thus undetectable and untransmittable. It can also be seen from Figure 4c that the higher the treatment rate of this population the lesser the number of individuals in the population over time. Hence, it can be concluded that an increase in the availability of the ART treatment to the infected (chronic) class will lead to a drastic drop in the member of this population over a period of time.

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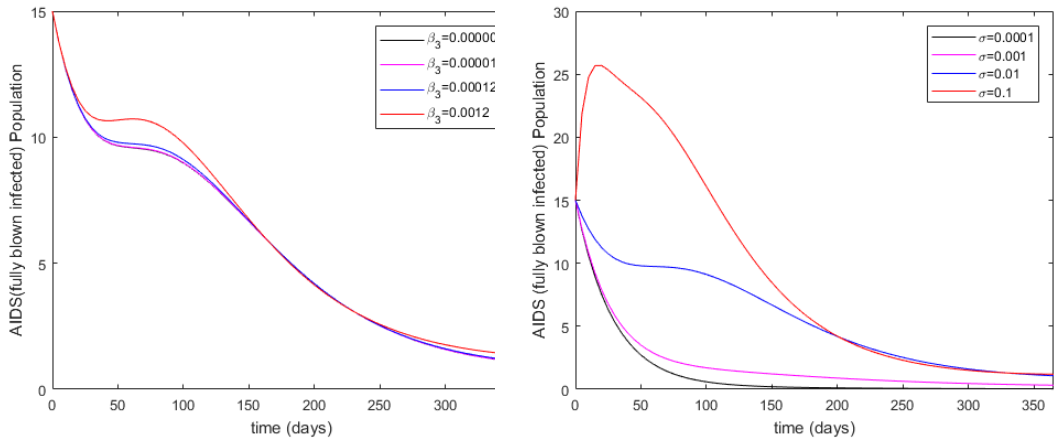
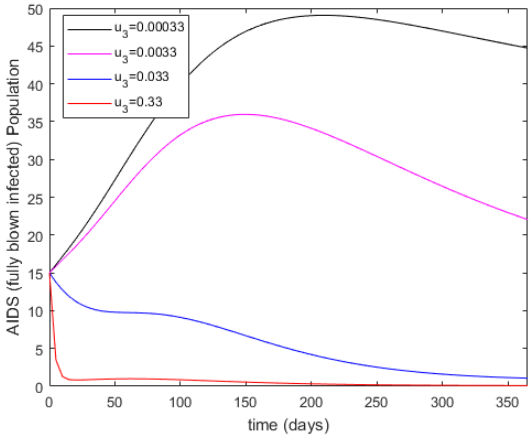
### 4.4 AIDS (fully blown infected) Population




(a) AIDS (fully blown infected) Population against Time varying $\beta_3$

(b) AIDS (fully blown infected) Population against Time varying $\sigma$




(c) AIDS (fully blown infected) Population against Time varying $u_3$

Figure 5: Effects of some parameters on the AIDS (fully blown infected) Population over Time

Figure 5 represents the behavior of the AIDS (fully blown infected) population against time. The AIDS class is seen to experience a slight decrease in its population over a period of 50 days due to the de-population of this class associated with its high death rate. After this short period, however, there was a slight increase in the members of this class due to its population by the few

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infected (chronic) members who are not being treated. Thereafter, this class continues to experience a steady decrease in its population associated with the effectiveness of the ART treatment being administered. It can be seen that, at the end of 1 year, the members of this population has dropped to a relatively low number ($\leq 5$) as most of them are now receiving the ART treatment and are thus undetectable and untransmittable. It can, therefore, be concluded that the ART treatment, is very effective and that an increase in its availability to the AIDS (fully blown infected) class will lead to a drastic drop in the member of this population over a period of time.
Moreover, Figure 5a indicates that an increase in the transmission rate of HIV via interaction with the AIDS (fully blown infected)) class, $\beta_3$, has no significant effect on its population size as people do not move into this class directly from the susceptible class. However, Figures 5b and 5c indicate that an increase in the rate of progression from the infected (chronic) class to the AIDS (fully blown infected) class, $\sigma$, and in the treatment rate of the AIDS (fully blown infected) class, $u_3$, lead to an increase and a decrease in the population of this class respectively.

### 4.5 Treated Population

Figure 6 represents the behavior of the treated population over a period of 365 days. The treated class can be seen to experience a continuous increase in its population throughout the entire 365 days. This increase can be associated with the continuous progression of people into this class from all the infected classes. Figure 6a indicates that an increase in the treatment rate of the infected (acute) population, $u_1$, leads to an increase in the growth rate of the treated class. However, it can be seen that if $u_1$ is as high as 0.1, then after some time the treated population shall be maintained at a steady state since a high treatment rate of the infected (acute) class implies a lesser number of new infections and hence a lesser number of individuals requiring treatment. Similarly, Figures 6b and 6c indicate that an increase in the treatment rate of the infected (chronic) population, $u_2$, and that of the AIDS (fully blown infected) class, $u_3$, lead to an increase in the growth rate of the treated class.
Between days 300 and 365, the number of individuals in the treated class is seen to be maintained at around 160 members. This is due to the efficiency of the ART treatment being administered to the infected classes. Besides, since the disease induced death rate of this class is relatively small, then the rate of outflow due to death and that of inflow due to treatment after a long period of time will be approximately equal as the treatment efficacy will imply that there will be less people requiring treatment since the HIV is neither detectable nor transmittable amongst the treated population.

## 5 Conclusion and Recommendations

The results of the analyses of the developed HIV/AIDS model indicate that the disease free equilibrium of the model is stable while the Endemic equilibrium point is unstable. These results imply that it is possible to curtail the growth and spread of this disease within a studied population provided the assumptions and parameters of the developed model are implemented within such population including the early detection and treatment of the infected classes with ART. Similarly, based on the results obtained from the simulations performed on the model, it can be concluded justifiably that the achievement of an HIV-free community of people is not only possible but also obtainable, in any developing country such as Cape Verde, by bringing the viral load of any HIV/AIDS positive patient to a U=U state. We make bold to say that it is viable to achieve the UNAIDS worldwide goal of ending the AIDS epidemic by 2030 if the following recommendations are highly adhered to.

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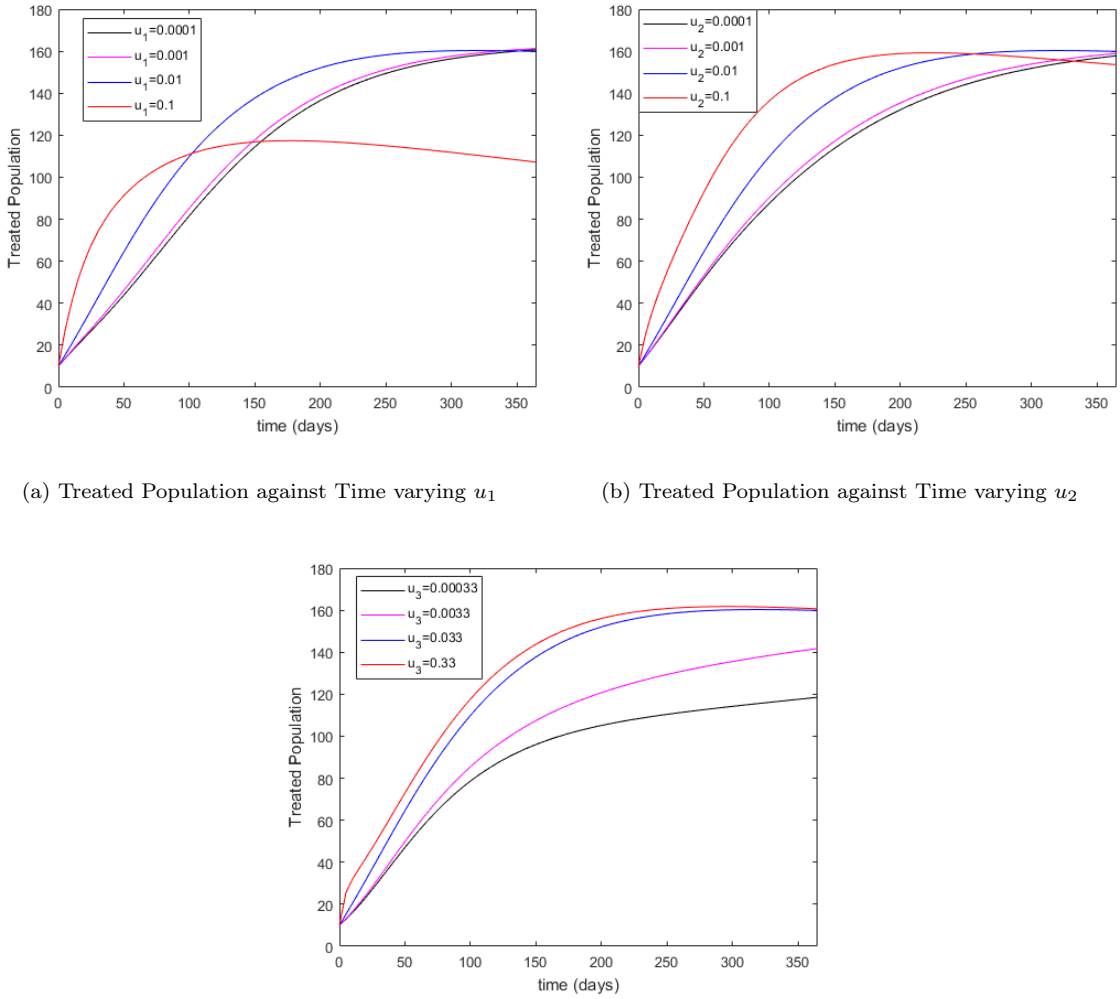


(a) Treated Population against Time varying $u_1$

(b) Treated Population against Time varying $u_2$

(c) Treated Population against Time varying $u_3$

Figure 6: Effects of the treatment rates on the Treated Population over Time

Early diagnostic kits should be made readily available by the government and health organizations to members of any community in which HIV/AIDS is reported. These diagnostic kits will aid in detecting the presence of this virus in an HIV positive person at an early stage (acute infectiousness) and this will significantly aid the commencement of an early treatment via the ART which will in turn bring about the attainment of a U=U level. It is also not only necessary but very pertinent that community members of an HIV endemic population be educated on HIV-AIDS and its fatality and most importantly the ART treatment should be provided by the government and concerned health organizations at a significantly subsidized amount to those in need of it. Awareness

campaigns should be carried out and encouraged by various governmental and non-governmental organizations at a regular interval on the prevention and treatment of HIV/AIDS via the ART treatment. Lastly, prevention of HIV via pre-exposure prophylaxis should be highly encouraged in sexual partners of an HIV/AIDS infected person, who themselves are under ART treatment regime.

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## Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper. The lead author "Mary Oluwabunmi OGUNMODIMU" was formerly known and addressed as "Mary Oluwabunmi AKINADE" but did a change of last-name due to marriage.

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