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Mathematical Modeling of HIV Investigating the Effect of Inconsistent Treatment

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Abstract

HIV is a retrovirus that infects and impairs the cells and functions of the immune system. It has caused a great challenge to global public health systems and leads to Acquired Immunodeficiency Syndrome (AIDS), if not attended to in good time. Antiretroviral therapy is used for managing the virus in a patient's lifetime. Some of the symptoms of the disease include lean body mass and many opportunistic infections. This study has developed a SIAT mathematical model to investigate the impact of inconsistency in treatment of the disease. The arising non-linear differential equations have been obtained and analyzed. The DFE and its stability have been obtained and the study found that it is locally asymptotically stable when the basic reproduction number is less than unity. The endemic equilibrium has been obtained and found to be globally asymptotically stable when the basic reproduction number is greater than unity. Numerical solutions have been obtained and analyzed to give the trends in the spread dynamics. The inconsistency in treatment uptake has been analyzed through the numerical solutions. The study found that when the treatment rate of those infected increases, it leads to an increase in treatment population, which slows down the spread of HIV and vice versa. An increase in the rate of treatment of those with AIDS leads to a decrease in the AIDS population, the reverse happens when this rate decreases. The study recommends that the community involvement in advocating for consistent treatment of HIV to curb the spread of the disease.

Keywords

HIV Modeling, Mathematical Modeling, Reproduction Number, Inconsistent Treatment

1. Introduction

Human Immunodeficiency Virus (HIV) is a deadly virus which when not treated

results to AIDS, which is incurable. It leads to damage to body organs like, the kidney, heart, and the brain [1]. The person infected who does not take medicine for HIV has a survival time of 9 - 11 years. It is impossible to eradicate the virus during the lifetime of a host [2]. Usually, HIV appears to be associated with other diseases [3]. It can be transmitted through blood transfusion, mother to child during birth or by breastfeeding, heterosexual or homosexual relations (WHO, 2008). The HIV virus falls to low levels when treatment is adhered to, but when treatment is withdrawn the viral load increases to the levels that existed before the administration. Compared to most other infections, HIV interacts with the immune system in a very complicated manner.

HIV has three main stages. Stage one is the one in which people have acute HIV infection, which entails patients having a high viral load in their blood and is highly contagious. Many people at this stage have flu-like symptoms. Stage two involves chronic HIV infection. It is an asymptomatic and has clinical latency. HIV is still active and continues to increase in the body. Those who fail to take prescribed medicine may move to stage three (NSDCC)

Most of the people infected by HIV come from developing countries like, South Africa, India, Kenya, Mozambique, Uganda, China, Zimbabwe, Zambia, Malawi, Indonesia and Thailand. In South Africa, 7 m people, among people with high-risk behavour, are infected with HIV/AIDS (WHO, 2016).

In 2015, the strategy for treatment of HIV enhanced testing and immediate enrolment into ART treatment in order to reduce viral road and achieve the 90-90-90-plan (WHO, 2015). Promoting healthy living among all people is one of the goals of the World Health Organization (WHO). Sustainable development goals include reducing the number of deaths from disease to less than 70 per 100,000 live births.

In 2021, there were 1.5 m new cases of HIV worldwide. 650,000 died of AIDS, in the same year (UNAIDS). This is a very alarming situation that would need attention from health facilities. Laikipia County was leading in reduction of HIV infection between the years 2020 and 2021.

Adherence to medication, ongoing monitoring and follow-up care are very critical for the successful management of HIV. Patients may require additional support, including reminders, counselling, and pill boxes to help them adhere to treatment regimes and most importantly co-ordinated and comprehensive approach. Proper adherence and management of treatment reduce the risk of complications (WHO, 2015).

1.1. Literature Review

In 2018, Omondi *et al.* conducted a study on factors affecting acceptability of Isoniazid preventive therapy among healthcare providers in selected HIV clinics in Nairobi, Kenya. He found that this therapy's implementation remains optimal in Sub-Saharan Africa, despite being globally accepted.

The study provided insight into the complexity of this factor in Kenya [4]. He found out that policymakers had to be involved in this endeavor together with pro-

gramme managers (of IPT), providers and patients, for quality delivery of the intervention. A mathematical model was not used.

In the year 2018, Maimuna and Aldila from the University of Indonesia, conducted a study and used a mathematical model for HIV spread control programme with ART treatment [5]. They found out that with an increased number of infected humans who follow treatment programmes with ART, the basic reproductive number reduces.

Odhambo *et al.* conducted a study, in 2016 on antiretrovial treatment scale-up among persons living with HIV in Kenya, using results from a nationally representative survey. The results showed that 11,626 people (aged between 15 - 64 years) provided a blood sample. 648 out of those people were infected with HIV. 58.8 percent were eligible for treatment using Kenya eligibility criteria (referring to WHO (2013)).

1.2. Research Objective

The objective of this paper was to develop and analyze a SIAT mathematical model for the transmission dynamics of HIV, with an inclusion of Inconsistent treatment.

2. Methodology

This model has four compartments: (S), susceptible, newly infected with HIV, (I), those who have progressed into AIDS (A) and those who have been treated with antiretroviral medicine (T). Humans get recruited by immigration or natural descent to compartment (S) at the rate of Λ. They get infected and move to the (I) compartment at the rate of α_1. They may get treated at the rate of β_1 and move to the compartment (T). If they are not treated they move, after some time, to compartment (A) at the rate of ϕ_1. They may also get treated from compartment (A) at the rate of κ_1. They may also die of AIDS at the rate of σ_1.

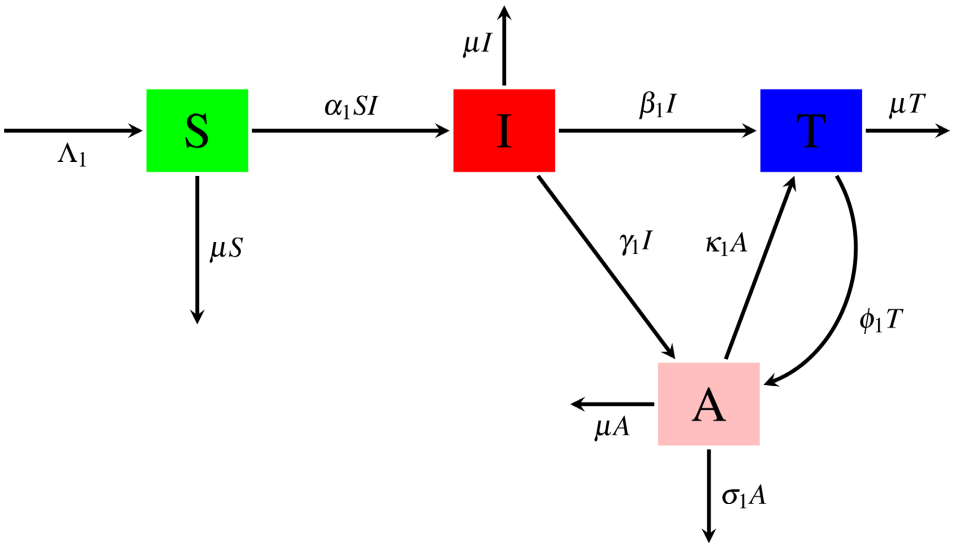

Figure 1. Model flow chart for HIV spread dynamics.

Table 1. HIV transmission dynamics parameters.

Parameter	Interpretation
Λ_1	Recruitment rate
β_1	Rate of treatment after infection
α_1	Infection rate
γ_1	Rate of progression from infected to AIDS
κ_1	Progression rate from AIDS to treatment
ϕ_1	progression rate from treatment to AIDS
μ	Natural death rate
σ_1	Death rate due to AIDS

In every compartment, μ is the rate at which people die naturally. The treatment only controls the progression of the disease since permanent immunity cannot be attained by those who are infected (Figure 1, Table 1).

The equations below are formulated from the dynamics of this model:

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} = \Lambda_1 - \alpha_1 SI - \mu S \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} = \alpha_1 SI - \left(\mu + \beta_1 + \gamma_1\right) I \\ \dfrac{\mathrm{d}A}{\mathrm{d}T} = \gamma_1 I + \phi_1 T - \left(k_1 + \sigma_1 + \mu\right) A \\ \dfrac{\mathrm{d}T}{\mathrm{d}t} = \beta_1 I + \kappa_1 A - \left(\mu + \phi_1\right) T \end{cases} \tag{1}$$

3. Basic Properties of the Model

The total population is represented by N. In a given span of time, the following equation represents the change in population:

$$\frac{\mathrm{d}N(t)}{\mathrm{d}t} = \frac{S(t)}{\mathrm{d}t} + \frac{I(t)}{\mathrm{d}t} + \frac{A(t)}{\mathrm{d}t} + \frac{T(t)}{\mathrm{d}t} \tag{2}$$

$$= \Lambda_1 - \alpha_1 SI - \mu S + \alpha_1 SI_H - \left(\mu + \beta_1 + \gamma_1\right) I + \beta_1 I + \kappa_1 A - \left(\mu + \phi_1\right) T + \gamma_1 I + \phi T - \left(k_1 + \sigma_1 + \mu\right) A \tag{3}$$

$$= \Lambda_1 - \mu S - \mu I - \mu T - \sigma_1 A - \mu A \tag{4}$$

In the absence of induced death,

$$\frac{\mathrm{d}N(t)}{\mathrm{d}t} = \Lambda_1 - \mu\left(S + I + A + T\right) \tag{5}$$

$$\frac{N(T)}{\mathrm{d}t} \le \Lambda_1 - \mu N(t) \tag{6}$$

Dividing both sides of the equation by:

$$\Lambda_1 - \mu N(t) \tag{7}$$

and integrating both sides, we have:

$$\int \frac{\mathrm{d}N(t)}{\Lambda_1 - \mu N(t)} \leq \int \mathrm{d}t \tag{8}$$

We let:

$$\Lambda_1 - \mu N(t) = \phi \tag{9}$$

$$\frac{\mathrm{d}\phi}{\mathrm{d}N} = -\mu \tag{10}$$

Which implies that,

$$\mathrm{d}N = \frac{\mathrm{d}\phi}{-\mu} \tag{11}$$

Then,

$$\int \frac{\frac{\mathrm{d}\phi}{-\mu}}{\phi} = \frac{1}{-\mu}\int \frac{\mathrm{d}\phi}{t} = \frac{1}{-\mu}\ln\phi \tag{12}$$

$$\frac{1}{-\mu}\ln\phi \leq t + c \tag{13}$$

Solving we have:

$$\phi = \mathrm{e}^{-\mu(t+c)} = \mathrm{e}^{-\mu t}\mathrm{e}^{0} \tag{14}$$

Taking logs on both sides of the equation, we have:

$$\ln\phi \geq -\mu(t+c) \tag{15}$$

which implies that,

$$\mathrm{e}^{\ln\phi} \geq \mathrm{e}^{-\mu(t+c)} \tag{16}$$

Hence,

$$t \geq A\mathrm{e}^{-\mu t} \tag{17}$$

But,

$$\phi = \mathrm{e}^{-\mu(t+c)} = \mathrm{e}^{-\mu t}\mathrm{e}^{c} \tag{18}$$

which implies that,

$$\phi \geq A\mathrm{e}^{-\mu t} \tag{19}$$

Also, considering Equation (9),

$$\phi = \Lambda_1 - \mu N(t) \tag{20}$$

We can therefore write,

$$\Lambda_1 - A\mathrm{e}^{-\mu t} \geq \mu N(t) \tag{21}$$

$$\frac{\mu}{\mu}N(t) \leq \Lambda_1 - \frac{A\mathrm{e}^{-\mu t}}{\mu} \tag{22}$$

$$N(t) \leq \frac{\Lambda_1}{\mu} - \frac{A\mathrm{e}^{-\mu t}}{\mu} \tag{23}$$

at t is equal to zero. Hence,

$$N(0) \le \frac{\Lambda_1}{\mu} - \frac{A}{\mu} e^0 \tag{24}$$

$$N(0) \le \frac{\Lambda_1}{\mu} - \frac{A}{\mu} \tag{25}$$

Multiplying by:

$$\mu \tag{26}$$

on both sides of the equation, we have:

$$\mu N(0) = \Lambda_1 - A \tag{27}$$

Therefore,

$$A = \Lambda_1 - \mu N(0) \tag{28}$$

$$N(t) = \frac{\Lambda_1}{\mu} - \frac{\mu N(0)}{\mu} e^{-\mu t} \tag{29}$$

as,

$$t \to \infty \tag{30}$$

$$\left(\frac{\Lambda_1}{\mu} - N(0)\right) e^{-\mu t} \to 0 \tag{31}$$

Hence,

$$N(t) \le \frac{\Lambda_1}{\mu} \tag{32}$$

if

$$N(0) \le \frac{\Lambda_1}{\mu} \tag{33}$$

This kind of flow generates a region that is positively invariant; we could call it Π.

3.1. The Disease Free Equilibrium

We shall look at the model equations and consider what happens at the point where no disease exists. We shall equate all equations in (1) to zero because we are referring to an equilibrium point, where there is no disease:

$$0 = \Lambda_1 - \alpha_1 SI - \mu S \tag{34}$$

$$0 = \Lambda_1 SI - (\mu + \beta_1 + \gamma_1) I \tag{35}$$

$$0 = \gamma_1 I + \phi T - (\kappa_1 + \sigma_1 + \mu) A \tag{36}$$

$$0 = \beta_1 I + \kappa_1 A - (\mu + \phi_1) T \tag{37}$$

At the time when there is no disease,

$$A = 0, I = 0, T = 0 \tag{38}$$

This implies that,

$$0 = \Lambda_1 - \mu S \tag{39}$$

Making Λ_1 the subject:

$$\Lambda_1 = \mu S \tag{40}$$

$$S^* = \frac{\Lambda_1}{\mu} \tag{41}$$

At DFE,

$$S^*, I^*, A^*, T^* = \left(\frac{\Lambda_1}{\mu}, 0, 0, 0\right) \tag{42}$$

3.2. The Endemic Equilibrium

Considering the model Equation (1),

At the equilibrium point,

$$\frac{\mathrm{d}}{\mathrm{d}t} = 0 \tag{43}$$

We therefore obtain these equations:

$$0 = \Lambda_1 - \alpha_1 SI - \mu S \tag{44}$$

$$0 = \alpha_1 SI - \left(\mu + \beta_1 + \gamma_1\right) I \tag{45}$$

$$0 = \gamma_1 I + \phi T - \left(\kappa_1 + \sigma_1 + \mu\right) A \tag{46}$$

$$0 = \beta_1 I + \kappa_1 A - \left(\mu + \phi_1\right) T \tag{47}$$

From Equation (45),

$$\begin{aligned} 0 &= \alpha_1 SI - \left(\mu + \beta_1 + \gamma_1\right) I \\ 0 &= I\left(\alpha S - \mu + \beta_1 + \gamma_1\right) \end{aligned} \tag{48}$$

Either $I = 0$ or $\alpha SI - \left(\mu + \beta_1 + \Upsilon_1\right) = 0$.

but $I \neq 0$ therefore $\alpha SI - \left(\mu + \beta_1 + \Upsilon_1\right) = 0$ giving us:

$$\frac{\alpha_1 S}{\alpha_1} = \frac{\mu + \beta_1 + \gamma_1}{\alpha_1} \tag{49}$$

$$\dot{S} = \frac{\mu + \beta_1 + \gamma_1}{\alpha_1} \tag{50}$$

Replacing in Equation (51) in Equation (44),

$$S^{**} = \frac{\mu + \beta_1 + \gamma_1}{\alpha_1} \tag{51}$$

$$0 = \Lambda_1 - \alpha_1 S^{**} I - \mu S^{**}$$

but,

$$S^{**} = \frac{\mu + \beta_1 + \gamma_1}{\alpha_1} \tag{52}$$

Therefore,

$$I = \frac{\Lambda_1 - \mu S^{**}}{\alpha_1 S^{**}} = \frac{\Lambda_1}{\alpha_1 S^{**}} - \frac{\mu}{\alpha_1} \tag{53}$$

We have:

$$\frac{\Lambda_1}{\alpha_1(\mu+\beta_1+\gamma_1)}-\frac{\mu}{\alpha_1} \tag{54}$$

$$I^{**}=\frac{\Lambda_1}{\alpha_1(\mu+\beta_1+\gamma_1)}-\frac{\mu}{\alpha_1}=\frac{\Lambda_1\alpha_1-\mu(\mu+\beta_1+\gamma_1)}{\alpha_1(\mu+\beta_1+\gamma_1)} \tag{55}$$

Considering Equation (44) and replacing in Equation (45),

$$0=\beta_1 I+\kappa_1 A-(\mu+\phi_1)T \tag{56}$$

$$\kappa_1 A=(\mu+\phi_1)T-\beta_1 I \tag{57}$$

$$A^{**}=\frac{(\mu+\phi_1)T^{**}}{\kappa_1}-\frac{\beta_1}{\kappa}\frac{\Lambda_1\alpha_1-\mu(\mu+\beta_1+\gamma_1)}{\alpha_1(\mu+\beta_1+\gamma)} \tag{58}$$

Considering Equation (46),

$$0=\Upsilon_1 I+\phi T-(\kappa_1+\sigma_1+\mu)A \tag{59}$$

$$\frac{\phi_1 T^{**}}{\phi_1}=\frac{(\kappa_1+\sigma_1+\mu)A^{**}}{\phi_1}-\frac{\gamma_1 I}{\phi_1} \tag{60}$$

Therefore,

$$T^{**}=\frac{\kappa_1+\sigma_1+\mu}{\phi_1}T^{**}-\frac{\gamma_1}{\phi_1}I^{**} \tag{61}$$

Endemic equilibrium is equal to:

$$\left(S^{**},I^{**},A^{**},T^{**}\right) \tag{62}$$

Which is equal to,

$$\begin{Bmatrix}\frac{\mu+\beta_1+\gamma_1}{\alpha_1},\frac{\Lambda_1\alpha_1-(\mu+\beta_1+\gamma_1)}{\alpha_1(\mu+\beta_1+\gamma_1)},\frac{(\mu+\phi_1)T^{**}}{\kappa_1}-\frac{\beta_1}{\kappa_1}\frac{\Lambda_1\alpha_1-\mu(\mu+\beta_1+\gamma_1)}{\alpha_1(\mu+\beta_1+\gamma_1)},\\ \frac{(\kappa_1+\sigma_1+\mu)A^{**}}{\phi_1}-\frac{\gamma_1}{\phi_1}I^{**}A^{**}\end{Bmatrix} \tag{63}$$

3.3. The Basic Reproduction Number

This number denotes the secondary infections arising from one infected individual. The next generation matrix will be used to determine its value. We shall consider the four non-linear compartments in this model as "infectious", represented by "X" the non-Infectious compartments represented by "Y" [6].

$$\dot{I}=\alpha_1 SI-(\mu+\beta_1+\gamma_1)I \tag{64}$$

$$\dot{A}=\gamma_1 I+\phi_1 T-(\kappa_1+\sigma_1+\mu)A \tag{65}$$

$$f=\begin{bmatrix}\alpha_1 SI\\ \gamma_1 I+\phi_1 T\end{bmatrix}\Rightarrow\begin{bmatrix}f_1\\ f_2\end{bmatrix}=\begin{bmatrix}\alpha_1 SI\\ \gamma_1 I+\phi_1 T\end{bmatrix} \tag{66}$$

$$\upsilon=\begin{bmatrix}(\mu+\beta_1)I\\ (\kappa_1+\delta_1+\mu)A-\gamma_1 I\end{bmatrix} \tag{67}$$

$$\mathscr{F} = \begin{bmatrix} \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial A} \\ \frac{\partial f_1}{\partial A} & \frac{\partial f_2}{\partial A} \end{bmatrix} = \begin{bmatrix} \alpha_1 S & 0 \\ \gamma_1 & 0 \end{bmatrix} \tag{68}$$

$$v = \begin{bmatrix} \frac{\partial \upsilon_1}{\partial I} & \frac{\partial \upsilon_1}{\partial A} \\ \frac{\partial \upsilon_2}{\partial I} & \frac{\partial \upsilon_2}{\partial A} \end{bmatrix} = \begin{bmatrix} \mu + \beta_1 & 0 \\ -\gamma_1 & \kappa_1 + \sigma_1 + \mu \end{bmatrix} \tag{69}$$

The determinant of this function will be:

$$\upsilon = (\mu + \beta_1)(\kappa_1 + \delta_1 + \mu) \tag{70}$$

$$\upsilon^{-1} = \frac{1}{det v} \begin{bmatrix} \kappa_1 + \sigma_1 + \mu & 0 \\ \gamma_1 & \mu + \beta_1 \end{bmatrix} \tag{71}$$

$$\frac{1}{(\mu + \beta_1)(\kappa_1 + \delta_1 + \mu)} \begin{bmatrix} \kappa_1 + \sigma_1 + \mu & 0 \\ \gamma_1 & \mu + \beta_1 \end{bmatrix} \tag{72}$$

$$v^{-1} = \begin{bmatrix} \frac{1}{\mu + \beta_1} & 0 \\ \frac{\gamma_1}{(\mu + \beta_1)(\kappa_1 + \delta_1 + \mu)} & \frac{1}{\kappa_1 + \delta_1 + \mu} \end{bmatrix} \tag{73}$$

$$Fv^{-1} = \begin{bmatrix} \alpha_1 & 0 \\ \gamma_1 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\mu + \beta_1} & 0 \\ \frac{\gamma_1}{\mu + \beta_1} & 0 \end{bmatrix} \tag{74}$$

The eigenvalues are:

$$A - PI = \begin{bmatrix} \frac{\alpha_1}{\mu + \beta_1} S^* - p & 0 \\ \frac{\gamma_1}{\mu + \beta_1} & 0 - P \end{bmatrix} \tag{75}$$

This is the characteristic polynomial.

$$det(A - PI) = \left(\frac{\alpha_1 S^*}{\mu + \beta_1} - P \right)(-P) - 0 = P^2 - \frac{\alpha_1 S^*}{\mu + \beta_1} \tag{76}$$

But,

$$S^* = \frac{\Lambda_1}{\mu} \tag{77}$$

Therefore,

$$\left(\frac{\alpha_1 \Lambda_1}{(\mu + \beta_1)\mu} - P \right)(-P) = 0 \tag{78}$$

This implies that,

$$P = \frac{\alpha_1 \Lambda_1}{(\mu + \beta_1)\mu} \tag{79}$$

This means that, either,

$$P = 0$$

or

$$P - \frac{\alpha_1 \Lambda_1}{(\mu + \beta_1)\mu} = 0 \tag{80}$$

In this case, then:

$$P = \frac{\alpha_1 \Lambda_1}{(\mu + \beta_1)\mu} \tag{81}$$

Therefore,

$$R_0 = \frac{\alpha_1 \Lambda_1}{(\mu + \beta_1)\mu} \tag{82}$$

which the basic reproduction number R_0 of the SIAT model.

3.4. The Stability of Disease Free Equilibrium

In order to solve Equation (1), we need to linearize the model equations by using a Jacobian Matrix as follows [7]:

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial A} & \frac{\partial f_1}{\partial T} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial I} & \frac{\delta f_2}{\partial A} & \frac{\partial f_2}{\partial T} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial A} & \frac{\partial f_3}{\partial T} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial A} & \frac{\partial f_4}{\partial T} \end{bmatrix} \tag{83}$$

substituting the values of the corresponding parameters, we have:

$$J = \begin{bmatrix} -\alpha_1 I - \mu & -\alpha_1 S & 0 & 0 \\ \alpha_1 I & \alpha_1 S - (\mu + \beta_1 + \gamma_1) & 0 & 0 \\ 0 & \gamma_1 & -(\kappa_1 + \sigma_1 + \mu) & \phi_1 \\ 0 & \beta_1 & \kappa_1 & -(\mu + \phi_1) \end{bmatrix} \tag{84}$$

We know that at DFE,

$$(S^*, I^*, A^*, T^*) = \left(\frac{\Lambda_1}{\mu}, 0, 0, 0\right) \tag{85}$$

We shall now obtain the Jacobian Matrix at DFE as:

$$J = \begin{bmatrix} -\mu & \frac{-\alpha_1 \Lambda_1}{\mu} & 0 & 0 \\ 0 & \frac{\alpha_1 \Lambda_1}{\mu}(\mu + \beta_1 + \gamma_1) & 0 & 0 \\ 0 & \gamma_1 & -(\kappa_1 + \sigma_1 + \mu) & \phi_1 \\ 0 & \beta_1 & \kappa_1 & -(\mu + \phi_1) \end{bmatrix} - \begin{bmatrix} B & 0 & 0 & 0 \\ 0 & B & 0 & 0 \\ 0 & 0 & B & 0 \\ 0 & 0 & 0 & B \end{bmatrix} \tag{86}$$

The eigenvalues are:

$$A - BI = 0, \text{ where } B \text{ is a constant.} \tag{87}$$

will be:

$$\begin{bmatrix} -\mu - A & \frac{-\alpha_1 \Lambda_1}{\mu} & 0 & 0 \\ 0 & \frac{-\alpha_1 \Lambda_1}{\mu} - (\mu + \beta_1 + \gamma_1) - A & 0 & 0 \\ 0 & \gamma_1 & (-(\kappa_1 + \sigma_1 & +\mu) - A)\phi_1 \\ 0 & \beta_1 & \kappa_1 & -(\mu + \phi_1) - A \end{bmatrix} \tag{88}$$

The eigenvalues are:

$$\begin{bmatrix} -\mu \\ -\mu - \frac{1}{2}\phi_1 - \frac{1}{2}\kappa_1 - \frac{1}{2}\sigma_1 + \frac{1}{2}\sqrt{\phi_1^2 + 2\phi_1\kappa_1 - 2\phi_1\sigma_1 + \kappa_1^2 + 2\kappa_1\sigma_1 + \sigma_1^2} \\ -\mu - \frac{1}{2}\phi_1 - \frac{1}{2}\kappa_1 - \frac{1}{2\sigma_1} - \frac{1}{2}\sqrt{\phi_1^2 + 2\phi_1\kappa_1 - 2\phi_1\sigma_1 + \kappa_1^2 + 2\kappa_1\sigma_1 + \sigma_1^2} \\ -\left(\frac{\beta_1\mu + \mu^2\mu\gamma_1 - \Lambda_1\alpha_1}{\mu}\right) \end{bmatrix} \tag{89}$$

All the eigenvalues in Equation (89) are negative apart from the second one. The condition for stability of DFE is that all eigenvalues must be negative [8]. This implies that:

$$-\mu - \frac{1}{2}\phi_1 - \frac{1}{2}\kappa_1 - \frac{1}{2}\sigma_1 + \frac{1}{2}\sqrt{\kappa_1^2 + (2\phi_1 + 2\sigma_1)\kappa_1 + (\phi_1 - \sigma_1)^2} < 0 \tag{90}$$

The following equation has to be satisfied for the stability of DFE:

$$\frac{1}{2}\sqrt{\kappa_1^2 + (2\phi_1 + 2\sigma_1)\kappa_1 + (\phi_1 - \sigma_1)^2} < \mu + \frac{1}{2}\phi_1 + \frac{1}{2}\kappa_1 + \frac{1}{2}\sigma_1 \tag{91}$$

which can be simplified as:

$$\sqrt{\kappa_1^2 + (2\phi_1 + 2\sigma_1)\kappa_1 + (\phi_1 - \sigma_1)^2} < 2\mu + \phi_1 + \kappa + \sigma \tag{92}$$

3.5. Numerical Simulations

Here, the parameters used in the model are contextualized (**Table 2**); numerical results are presented and discussed. In some figures, possible control strategies are investigated.

Figure 2 shows that there is a big gap between the treatment population and both the infectious and the AIDS population. The treatment population increases steadily from zero to around 18,500 people decreases to 1100, rises to around 14,900, drops to around 1300 and then levels off. The AIDS population increases from zero to around 2200, drops to around 500, rises to 1000 and then levels off during this time of study. There is a small gap between this population and the infectious one. The infectious population increases from zero to around 1200, falls to around four hundred, rises to around five hundred and then levels off for the time of study.

As can be seen from **Figure 3**, when" beta" has a value of 0.75 the treatment

Table 2. HIV transmission dynamics parameters and their references.

Parameter	Interpretation	Value	Source
Λ_1	Recruitment rate	3295	[9] [10] [11]
β_1	Rate of treatment after infection	0.0000075	[9]
α_1	Infection rate	0.000011591	[9] [10]
γ_1	Progression rate from infected to AIDS	$1-\beta$	Calculated
κ_1	Rate of progression from AIDS to treatment	0.512	[9]
ϕ_1	Rate of progression from treatment to AIDS	0.000498	Estimated
μ	Natural death rate	0.031	[9] [12] [13]
σ_1	Death rate due to AIDS	0.0821	[9] [12]

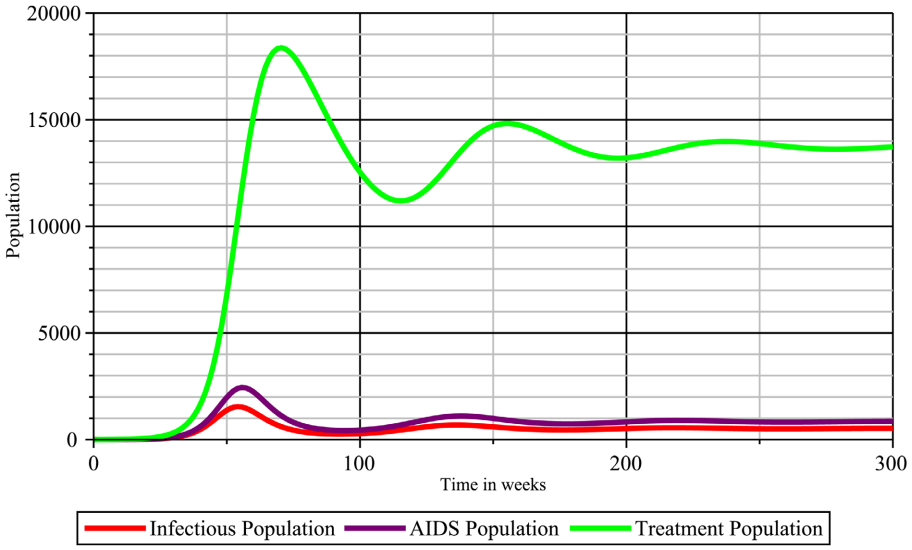

Figure 2. The disease dynamics of the SIAT model focusing on infectious treatment and AIDS compartments.

population rises to around 21,000 from zero. As "beta" decreases to 0.0075 the treated population increases from zero to around to 17,000 people, falls to around 13,000, rises to around 16,000 people and then levels of. The gap between the two graphs is small and this continues for many weeks. When "beta" increases to 0.95, the treatment population increases to around than 22,000 people. It then decreases to around 15,000, increases to around 17,000 and then levels off for the rest of the months of study. The gap between the graphs of "beta" =0.75 and "beta" equal to 0.0075 is bigger that the gap between the graphs of "beta = 0.75 and "beta" = 0.95.

In **Figure 4**, we note that when "kappa" has value of 0.512 the treatment population increases drastically from zero to around 21,000 people. It then decreases to around 14,000, rises to 17,000 and then levels off. As kappa decreases to 0.0512, the treatment population rises from zero to around 18,000, falls to

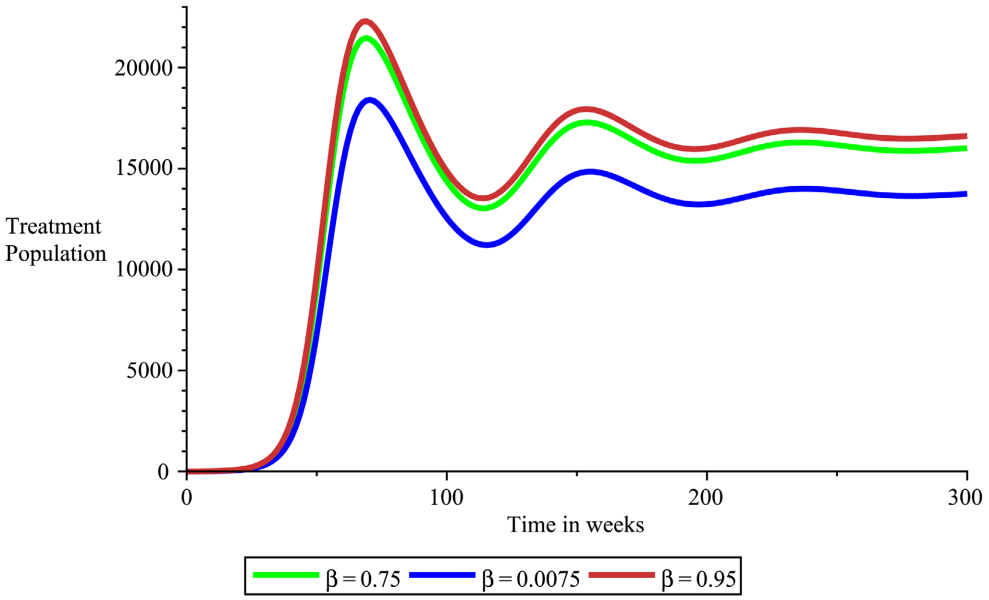

Figure 3. Treatment compartment at different values of beta.

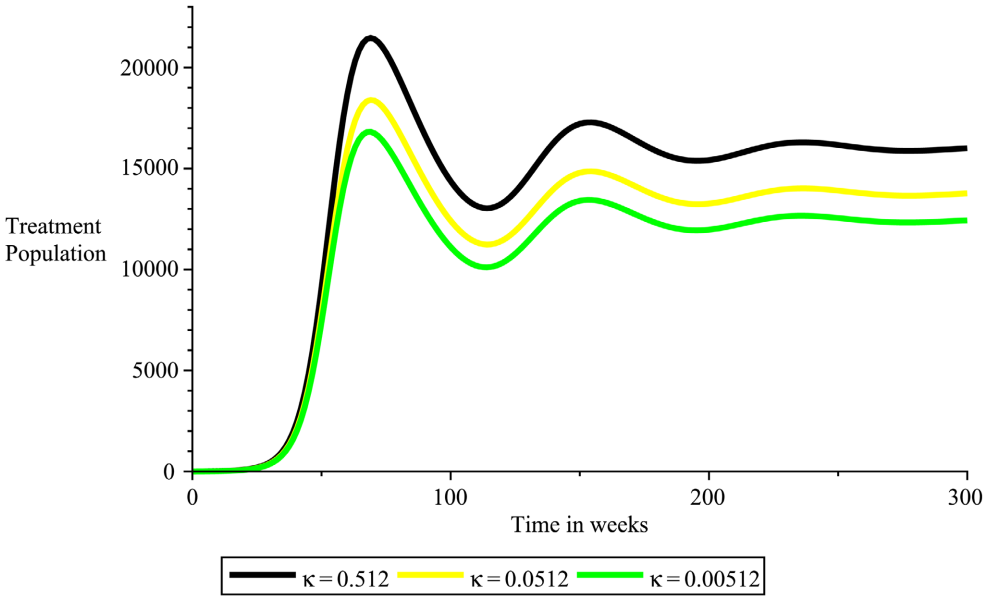

Figure 4. Treatment compartment at different values of κ.

around 12,000, rises to around 14,000 and then levels off. As kappa continues to decrease (0.00512), the treatment population rises from zero to around 1600, falls to around 10,000, rises to decreases to around 16,000, then falls to around 11,000 people, rises to around 13,000 and finally levels off for the months of study. The gap between the graphs of "kappa" = 0.0512 and "kappa"= 0.00512 is small as compared to the gap between the graphs of "kappa" = 0.512 and "kappa" = 0.00512.

In Figure 5, the AIDS population starts from zero and increases to around five hundred people then falls within 100 months to around 200 people. It then rises to around 250 and then levels off in the time of study. When "kappa"

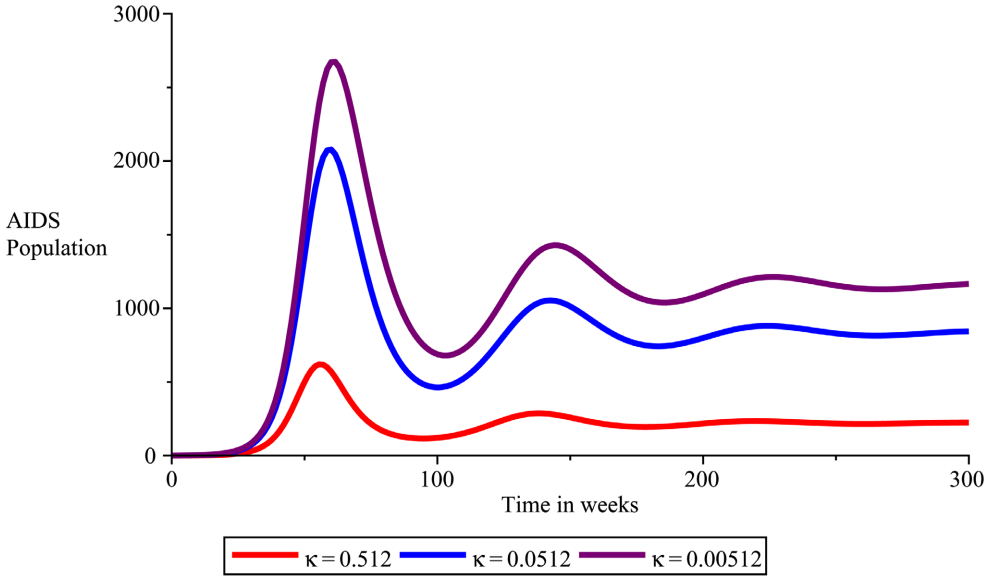

Figure 5. AIDS population at different values of κ.

decreases to 0.0512, the AIDS population increases to around 2000 people, drops to around 500, rises to 1000 and then levels off in the months of study. When "kappa" reduces to 0.00512, the AIDS population increases from zero to around 2700 people. It then drops to around 700, rises to around 1500 and levels off for the time of study. The gap between the graph of kappa = 0.0512 and kappa = 0.0512 is smaller than the gap between kappa = 0.0512 and kappa = 0.00512.

4. Conclusion and Recommendation

4.1. Conclusion

A SIAT model was formulated for the transmission dynamics of HIV. Differential equations were formulated from the model. The same equations were used to determine the basic reproduction number R_0, disease free equilibrium, and its stability established. The conclusion was that when $R_0 < 1$, the DFE is locally asymptotically stable. From the disease dynamics, the endemic equilibrium was established. Numerical solutions were also obtained and the corresponding graphs were analyzed. It was found that when the rate at which the infected go for treatment increases, β, the treatment population increases. Similarly, when the rate at which the AIDS patients go for treatment, κ, increases, the AIDS population decreases.

4.2. Recommendation

The study recommends decentralizing the testing of HIV from the health centers to the community settings, so as to increase the rate at which the infected youth go for treatment. When the community is involved, more cases will be detected and encouraged to go for treatment in good time and this will assist in maintaining $R_0 < 1$. Some of the ways in which this can be done are by sensitizing the youth in public gatherings such as churches, schools, colleges, universities, foot-

ball matches, chief barazas, and other social gatherings, where the youth are found. Social media could also be used to sensitize the youth on the importance of consistent uptake of ART. The study further recommends that the community could be trained to discourage stigmatization of those with AIDS, and create an environment that will encourage these patients to go for medication consistently. Furthermore, health centers should have programmes that encourage constant counseling of such patients. They should also secure conducive places for counseling, in order to encourage these patients to constantly come for treatment. It is also recommended that every health centre should have a chaplain who can effectively listen to the challenges that such patients encounter in their home settings. Community-based organizations such as churches and religious organizations could organize outreach programmes to the vulnerable youth such as those involved in substance abuse, street families, commercial sex workers, and those suffering from psychological problems, such as poor mental health. Further research could also be done on how to improve the human immunity system, so as to curb the devastating effect caused by HIV, in the body.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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