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A Mathematical Model for the Transmission of HIV/AIDS with Early Treatment

T. O. Akinwumi[1], I. A. Olopade[1*], A. O. Adesanya[1] and M. O. Alabi[2]

[1]Department of Mathematics and Computer Science, P.M.B. 002, Elizade University, Ilara-Mokin.Nigeria.
[2]Department of Physical Sciences, Chrisland University, Abeokuta, Ogun State. Nigeria.

Authors' contributions

This work was carried out in collaboration among all the authors. Authors TOA and IAO formulate the models for the study. The general analysis and numerical simulation was carried out by authors TOA, IAO, AOA and MOA. All the authors managed the literature and all the authors read and approved the final manuscript.

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Abstract

In this paper, a mathematical model for the transmission of HIV/AIDS with early treatment is developed and analyzed to gain insight into early treatment of HIV/AIDS and other epidemiological features that cause the progression from HIV to full blown AIDS. We established the basic reproduction number which is the average number of new secondary infection generated by a single infected individual during infectious period. The analysis shows that the disease free equilibrium is locally and globally asymptotically stable whenever the threshold quantity is less than unity i.e. Numerical analysis shows that the early treatment of latently infected individuals reduces the dynamical progression to full blown AIDS. The result also showed that immunity boosted substances increase the red blood cells, sensitivity analysis of basic reproduction number with respect to parameters showed that effective contact rate must not exceed 0.3 to avoid endemic stage.

Keywords: HIV/AIDS; treatment; reproduction number; equilibrium points and stability.

**Corresponding author: Email: isaac.olopade@elizadeuniversity.edu.ng;*

1 Introduction

The human immunodeficiency virus (HIV) is a virus that affects the body's immune system and leads to Acquired immunodeficiency Syndrome (AIDS). It is an infectious disease which has led to the death of millions of people in both developing and developed countries, 38.0 million [31.6 million–44.5 million] people globally were living with HIV in 2019 while 26 million [25.1 million–26.2 million] people were accessing antiretroviral therapy as of the end of June 2020 [1]. The symptoms include but not limited to: fever, chills, fatigue, swollen lymph nodes, sore throat, and night sweats [2].

One of the major problems caused by the HIV to the body is the destruction of $CD4^+$T cells which play an important role in the regulation of the body immune system. HIV causes a reduction in the number of functional $CD4^+$T cells thereby making the body unable to fight and prevent cell infections. A lot of mathematical models have been formulated to study the interactions between $CD4^+$T cells and HIV [3-6].

Early treatment of HIV/AIDS in infected persons can reduce the rate of sexual transmission in humans [7,8]. Recent control trials have found that treating HIV-positive individuals with antiretroviral drugs reduces the risk of them transmitting the disease to their heterosexual partners by more than 90% [7]. These antiretroviral drugs help in building the immune system of the infected individual against more cell infections. The antiretroviral drugs are categorized into two groups which are reverse transcriptase inhibitors (RTIs) and protease inhibitors (PIs). RTIs disrupt the conversion of RNA of the virus to DNA so that new HIV infection of cells is prevented. On the other hand, PIs hinder the production of the virus particles by the actively infected $CD4^+$T cells [3].

Since early treatment have been proven to reduce the rate of transmission, it is important to identify infected persons and put them on treatment. Individuals should also be encouraged to go for voluntary testing that will increase case detection, thereby reducing the number of secondary infections.

A lot of mathematical models have been developed extensively in the study of HIV/AIDS transmission dynamics [9-12]. Also, models incorporating treatment have also been developed by some mathematicians.

HUO et al [8], developed a simple epidemic model of HIV/AIDS with treatment compartment. They introduced a treatment compartment T where infected individuals received different kinds of treatments. It is noted that these treatment do not completely eliminate HIV from the body and the effects of treatment on the HIV/AIDS transmission dynamics was studied.

In [13], HIV- infection transmission in a male homosexual population was studied. The model considered two types of infected individuals. Those who are infected but do not know their status and are not under any clinical treatment and those who are under treatment. The analytical results show that there exists a unique endemic equilibrium which is globally asymptotically stable under a range of parameter values whenever a detection /treatment rate and an indirect measure of the level of infection risk are sufficiently large.

Adewale et.al, [14], Presented and analyzed five (5) non-linear differential compartmental models, to have better understanding on the parameters that influence the dynamical spread of HIV in the society. Numerical simulations of the model were analyzed to determine the effects of parameters on the dynamical spread of the disease. The effective contact rate and the fast progressor are the major key parameters that enhanced the dynamical spread of HIV in the society.

In [4], a mathematical model for an effective management of HIV was presented. The model presents two control variables where the uninfected $CD4^+$T cells follow the logistic growth function and the incidence term is saturated with free virus.

In this paper, we modified the work done by Huo et.al. by incorporating latently infected compartment with early treatment and treated compartment with fractions of those that failed treatment that moved to aids class, therefore, five (5) compartmental epidemiological model is developed and analyzed for the transmission of HIV/AIDS with early treatment to study the effect of early treatment on the progression from HIV to full blown AIDS.

The rest of the paper is organized as follows: In section 2, we developed a mathematical model where the population size N(t) is divided into five compartments; Susceptible S(t), Latently infected L(t), Infected I(t), Treated T(t), Aids A(t). In section 3, we presented the positivity of solution, disease free equilibrium point, Basic reproduction ratio, Local Stability of Disease Free Equilibrium Point, Global stability of the Disease Free Equilibrium, Endemic Equilibrium and Sensitivity Analysis. The numerical simulation was discussed in section 4

2 Model Formulation

The population size $N(t)$ of human is sub–divided into sub–classes of individuals who are Susceptible $S(t)$, Latently infected L (t), Infected $I(t)$, Treated $T(t)$ and Aids $A(t)$, So that;

$$N(t) = S(t) + L(t) + I(t) + T(t) + A(t) \tag{1}$$

The susceptible population is increased by the recruitment of individuals into the population (either by birth or immigration at the rate Λ). The population decreases by the newly infected individuals that move to latently infected class. The population also decreases by natural death (at the rate μ). Thus;

$$\frac{dS}{dt} = \Lambda - \beta SI - \mu S \tag{3}$$

The population of the latently infected class consist newly infected individuals following a contact with the infected human/object (at the rate β). The population decreases due to progression to infectious class (at the rate κ), natural and disease induced death (at the rate $\mu \ and \ \delta$) respectively, also decreases due to early treatment at the rate σ_1 .The population later increased by the help of immunity boosted from treated compartment when the CD4 counts rise above 50%. Thus;

$$\frac{dL}{dt} = \beta SI - (\kappa + \mu + \delta + \sigma_1)L + \theta T \tag{4}$$

The population of infected individual increases by progression from latently infected individual due to lack of treatment or treatment failure (at the rate κ). The population decreases due to treatment (at the rate σ_2), natural death (at the rate μ) and disease induced death (at the rate δ). Thus;

$$\frac{dI}{dt} = \kappa L - (\mu + \sigma_2 + \delta)I \tag{5}$$

The population of the treated individuals increases by the treatments of those that are latently and fully infected by HIV (at the rate $\sigma_1 and \ \sigma_2$). The population decreases due to natural death (at the rate μ), death due to the disease (at the rate δ), treatment failure due to drug resistance or inadequate dosing at the rate α and the immunity boosted after treatment and CD4 count rises above 50% (at the rate θ). Then,

$$\frac{dT}{dt} = \sigma_1 L + \sigma_2 I - (\mu + \delta + \alpha + \theta)T \tag{6}$$

Full blown AIDS compartment increases by treated individuals that failed treatment due to one medical reason or the other at the rate α The acquire immuno-deficiency syndrome individuals suffer natural death and death due to the disease (at the rate $\mu and \ \delta$) respectively. Hence;

$$\frac{dA}{dt} = \alpha T - (\mu + \delta)A \tag{7}$$

In summary, we have the following system of differential equations.

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta SI - \mu S \\
\frac{dL}{dt} &= \beta SI - (\kappa + \mu + \delta + \sigma_1)L + \theta T \\
\frac{dI}{dt} &= \kappa L - (\mu + \sigma_2 + \delta)I \\
\frac{dT}{dt} &= \sigma_1 L + \sigma_2 I - (\mu + \delta + \alpha + \theta)T \\
\frac{dA}{dt} &= \alpha T - (\mu + \delta)A
\end{aligned} \tag{8}$$

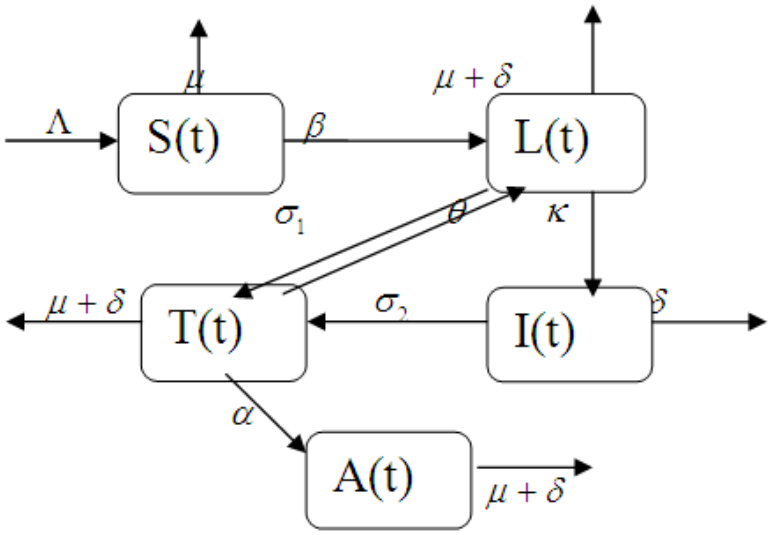

Chart 1. Flow chat

3 Positivity of Solution

Lemma 1: The closed set $D = \{(S, L, I, T, A) \in R_+^5 : N \leq \frac{\Lambda}{\mu}\}$ is positively invariant and attracting to model equation (8).

Proof: Consider the biologically-feasible region D defined above. The rate of change of the total population obtained by adding all the equations of the model is given by

$$\frac{dN}{dt} = \Lambda - \mu N - \delta(L + I + T + A) \tag{9}$$

It follows that $\frac{dN}{dt} =< 0$ whenever $N > \frac{\Lambda}{\mu}$,

Further, since $\frac{dN}{dt} \leq \Lambda - \mu N$, it is clear that $N(t) \leq \frac{\Lambda}{\mu}$

If $N(0) \leq \frac{\Lambda}{\mu}$. Therefore all solution of the model with initial condition in D remains in D for all t>0. Thus D is positively-invariant and attracting in the region D, the model can be considered as being epidemiologically and mathematically well posed.

3.1 Disease free equilibrium point

At steady state,

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

Since there is no infection;

$$L = I = T = A = 0,$$

Therefore, $\Lambda - \mu S = 0$

Hence; $S = \frac{\Lambda}{\mu}$

$$\therefore\ E_o = (S^*, L^*, I^*, T^*, A^*) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right) \tag{10}$$

3.2 Basic reproduction number

The basic reproduction number is the number of secondary cases of infection generated from a single infection [14]. We obtained this using next generation matrix method [15]. The matrices F(new infection terms) and V (other transferring terms) are given as;

$$F = \begin{bmatrix} 0 & \frac{\beta\Lambda}{\mu} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{11}$$

And

$$V = \begin{bmatrix} K_1 & 0 & -\theta & 0 \\ -\kappa & K_2 & 0 & 0 \\ -\sigma_1 & -\sigma_2 & K_3 & 0 \\ 0 & 0 & -\alpha & K_4 \end{bmatrix} \tag{12}$$

The basic reproduction number denoted by R_o is given by $R_o = \rho\left(FV^{-1}\right)$

$$\therefore R_o = \left[\frac{\beta\Lambda\kappa K_3}{\mu\left(\kappa\theta\sigma_2 + \theta K_2\sigma_1 - K_1K_2K_3\right)}\right]$$

Where;

$K_1 = \kappa + \mu + \delta + \sigma_1$

$K_2 = \mu + \sigma_2 + \delta$

$K_3 = \mu + \delta + \alpha + \theta$

$K_4 = \mu + \delta$

The threshold quantity R_o is the basic reproduction number of the model equation above, which is the average number of new case of an infection caused by one typical infected HIV/AIDS in a population of susceptible.

3.3 Local stability of disease free equilibrium point

Theorem 1: The disease free equilibrium is locally asymptotically stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: The Jacobian matrix of the system model (8) at disease free equilibrium point E_0 (0, 0, 0, 0, 0, 0) is obtained as follows

$$J_1(E_0) = \begin{pmatrix} -\mu-\lambda & 0 & \frac{-\beta\Lambda}{\mu} & 0 & 0 \\ 0 & K_1-\lambda & \frac{\beta\Lambda}{\mu} & \theta & 0 \\ 0 & \kappa & -K_2-\lambda & 0 & 0 \\ 0 & \sigma_1 & \sigma_2 & -K_3-\lambda & 0 \\ 0 & 0 & 0 & \alpha & -K_4-\lambda \end{pmatrix} \tag{13}$$

The first and fifth column of the equation (13) have only the diagonal terms that form the first two negative eigen values i.e, $-\mu$ and $-K_4$, Hence, we have;

$$J_2(E_0) = \begin{pmatrix} K_1-\lambda & \frac{\beta\Lambda}{\mu} & \theta \\ \kappa & -K_2-\lambda & 0 \\ \sigma_1 & \sigma_2 & -K_3-\lambda \end{pmatrix} = 0 \tag{14}$$

We obtained the eigen values of the matrix $J_2(E_0)$ from the characteristics equation below;

$$A_3\lambda^3 + A_2\lambda^2 + A_1\lambda + A_0 = 0 \tag{15}$$

Where

$$A_3 = 1$$

$$A_2 = K_3 + K_2 + K_1$$

$$A_1 = \frac{\mu\theta\sigma_1 - \mu K_1 K_2 - \mu K_1 K_3 - \mu K_2 K_3 + \Lambda\beta\kappa}{\mu} \tag{16}$$

$$A_0 = -\frac{\sigma_2\kappa\theta\mu + \sigma_1\theta\mu K_2 - \mu K_1 K_2 K_3 + \Lambda\beta\kappa K_3}{\mu}$$

According to Routh Hurwitz criterion, which states that all the roots of the polynomial will have negative real parts if and only if all the coefficients A_i (i=0, 1, 2, 3) are all positive and that the matrices T_i (i=1, 2, 3) are all positive. Clearly from (16) $A_3 > 0, A_2 > 0, A_1 > 0 \, and \, A_0 > 0$ if $R_0 < 1$. Also, the Hurwitz matrix T_i is all positive which are given as below;

$$T_1 = A_2 > 0, T_2 = \begin{vmatrix} A_2 & A_3 \\ A_0 & A_1 \end{vmatrix} > 0, T_3 = \begin{vmatrix} A_2 & A_3 & 0 \\ A_0 & A_1 & A_2 \\ 0 & 0 & A_0 \end{vmatrix} > 0$$

Therefore, all the eigen-values of the polynomial (15) are negative which shows that the disease free equilibrium is locally asymptotically stable.

3.4 Global stability of the disease free equilibrium

Theorem 2: The disease free equilibrium of model (8) is globally asymptotically stable if $R_o<1$.

Proof: We will use comparison theorem [16] to prove the global stability. The rate of change of variables representing the infected components of equation (8) can be re-written as;

$$\begin{pmatrix} \frac{dL}{dt} \\ \frac{dI}{dt} \\ \frac{dT}{dt} \\ \frac{dA}{dt} \end{pmatrix} = (F - V)\begin{pmatrix} L \\ I \\ T \\ A \end{pmatrix} - F_i\begin{pmatrix} L \\ I \\ T \\ A \end{pmatrix} \tag{17}$$

Where;

$$\begin{pmatrix} \frac{dL}{dt} \\ \frac{dI}{dt} \\ \frac{dT}{dt} \\ \frac{dA}{dt} \end{pmatrix} = (F-V)\begin{pmatrix} \beta S-\kappa-\mu-\delta-\sigma_1-\theta \\ \kappa-\mu-\sigma_2-\delta \\ \sigma_1+\sigma_2-\mu-\delta-\alpha-\theta \\ \alpha-\mu-\delta \end{pmatrix} - F_i \begin{pmatrix} L \\ I \\ T \\ A \end{pmatrix} \tag{18}$$

Then,

$$\begin{pmatrix} \frac{dL}{dt} \\ \frac{dI}{dt} \\ \frac{dT}{dt} \\ \frac{dA}{dt} \end{pmatrix} (F-V)\begin{pmatrix} \beta S-\kappa-\mu-\delta-\sigma_1-\theta \\ \kappa-\mu-\sigma_2-\delta \\ \sigma_1+\sigma_2-\mu-\delta-\alpha-\theta \\ \alpha-\mu-\delta \end{pmatrix} \tag{19}$$

All the eigen values of the matrix F – V have negative real parts. It follows that the linearized differential inequality system above is stable whenever $R_o < 1$.Consequently, by comparison theorem [15] we have that $E_h = I_h = J_h = I_R = 0, \to (0,0,0,0,0)$ as $t \to \infty$. Substituting $E_h = I_h = J_h = I_R = 0$ into (8) we have that $S(t) \to S(0)$ as $t \to \infty$. Hence, we have a positive invariant region. It follows that disease free equilibrium is globally asymptotically stable whenever $R_0 < 1$.

3.5 Endemic equilibrium

Let $\varepsilon_1^{**} = (S^{**}, L^{**}, I^{**}, T^{**}, A^{**})$ represents any arbitrary endemic equilibrium of the model equation (8). Solving equations in the system simultaneously yield;

$$S^{**} = -\frac{\kappa\theta\sigma_2 + \theta K_2\sigma_1 - K_1K_2K_3}{\beta\kappa K_3} \tag{20}$$

$$L^{**} = -\frac{(\mu\kappa\theta\sigma_2 + \mu\theta K_2\sigma_1 - \mu K_1K_2K_3 + \Lambda\beta\kappa K_3)K_2}{\beta(\kappa\theta\sigma_2 + \theta K_2\sigma_1 - K_1K_2K_3)\kappa} \tag{21}$$

$$I^{**} = -\frac{(\mu\kappa\theta\sigma_2 + \mu\theta K_2\sigma_1 - \mu K_1K_2K_3 + \Lambda\beta\kappa K_3)}{\beta(\kappa\theta\sigma_2 + \theta K_2\sigma_1 - K_1K_2K_3)} \tag{22}$$

$$T^{**} = -\frac{(\mu\kappa\theta\sigma_2 + \mu\theta K_2\sigma_1 - \mu K_1K_2K_3 + \Lambda\beta\kappa K_3)(\kappa\sigma_2 + K_2\sigma_1)}{\beta(\kappa\theta\sigma_2 + \theta K_2\sigma_1 - K_1K_2K_3)\kappa K_3} \tag{23}$$

$$A^{**} = -\frac{\alpha(\mu\kappa\theta\sigma_2 + \mu\theta K_2\sigma_1 - \mu K_1K_2K_3 + \Lambda\beta\kappa K_3)(\kappa\sigma_2 + K_2\sigma_1)}{\beta(\kappa\theta\sigma_2 + \theta K_2\sigma_1 - K_1K_2K_3)\kappa K_3K_4} \tag{24}$$

The endemic equilibrium point of the HIV/AIDS model equation (8) exists whenever the threshold quantity $R_o > 1$

3.6 Sensitivity analysis

Sensitivity analysis investigates the relations between parameters of a model and its threshold quantity basic reproduction number Ro which determines the spread/eradication of a disease in a community at a particular time. [17] Sensitivity Analysis has been used for different parameterization tasks of models of biological systems, such as finding necessary parameters for research prioritization [18], identifying less influenced parameters or parameters clustering [19].

Sensitivity analysis of the model is determined by the partial derivatives of the basic reproduction number with respect to its parameters;

$$\text{“}P\text{”}: \; X_P^{R_O} = \frac{\partial R_O}{\partial P} \times \frac{P}{R_O}.$$

The results of the sensitivity indices of $\mathrm{R_O}$ are as shown in the table below;

Table 1. Signs of sensitivity index (s. I) of R_0

Parameter	S. I.
β	Positive
μ	Negative
α	Positive
θ	Negative
κ	Positive
σ_1	Negative
σ_2	Negative
δ	Negative

4 Numerical Simulation

Table 2 Numerical behavior of equation (8) is studied using MAPLE 19 software with parameters values in the table below;

Table 2. Description of Parameters with Values

Parameter	Descriptions	Values
Λ	Recruitment into Population	2000
β	Effective Contact Rate	0.2
μ	Natural Death Rate	0.02
δ	Disease Induced Death Rate	0.01
α	Treatment Failure	0.01
θ	Immunity Boost	0.1
κ	Progression Rate	0.12
σ_1	Treatment Rate	0.2
σ_2	Treatment Rate	0.1

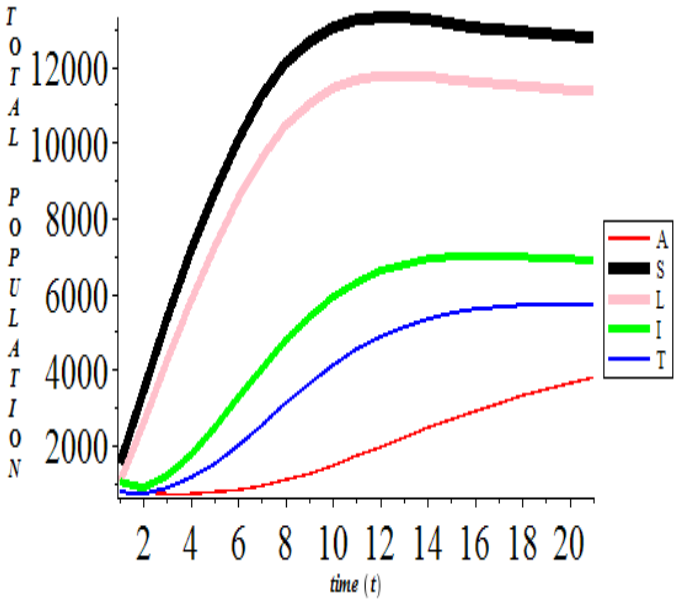

Fig. 1. Graph of total population when
$\Lambda = 2000, \beta = 0.2, \mu = 0.02, \delta = 0.01, \alpha = 0.01,$
$\theta = 0.1, \kappa = 0{,}12, \sigma_1 = 0.2, \sigma_2 = 0.1$

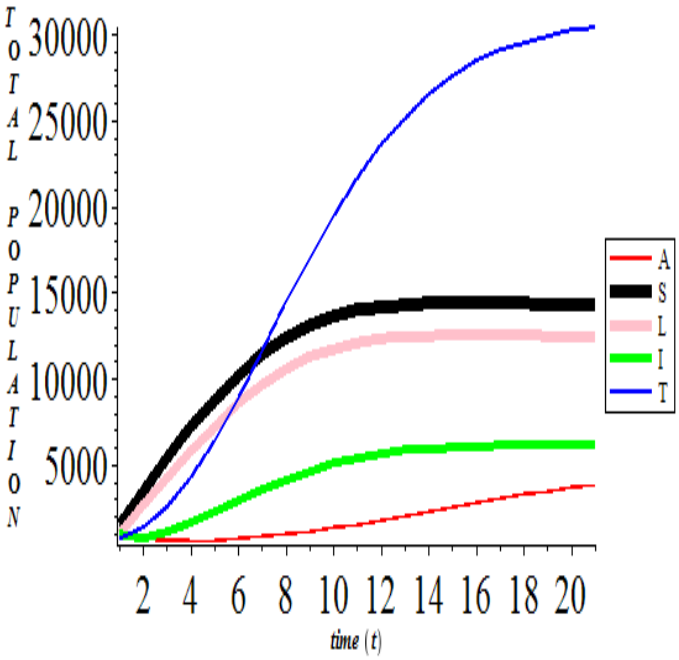

Fig. 2. Graph of total population when
$\Lambda = 2000, \beta = 0.2, \mu = 0.02, \delta = 0.01, \alpha = 0.01,$
$\theta = 0.1, \kappa = 0{,}12, \sigma_1 = 0.5, \sigma_2 = 0.1$

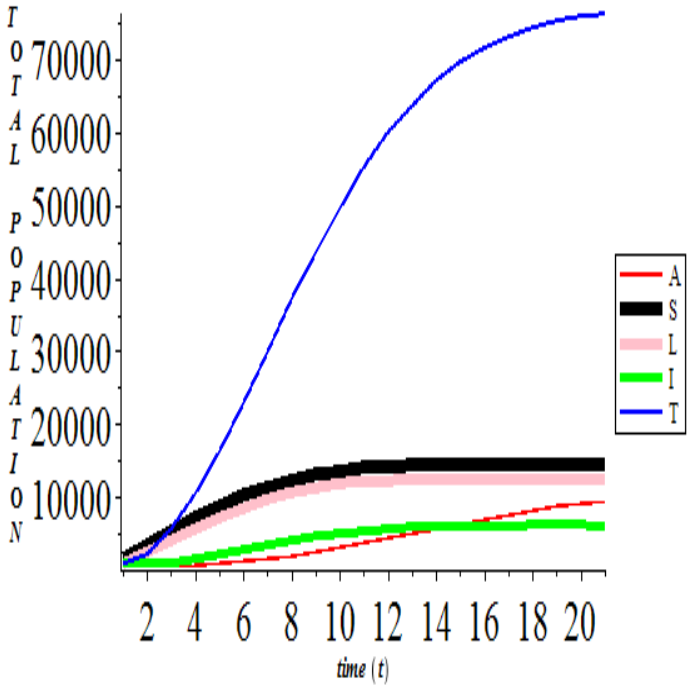

Fig. 3. Graph of total population when
$\Lambda = 2000, \beta = 0.2, \mu = 0.02, \delta = 0.01, \alpha = 0.01,$
$\theta = 0.1, \kappa = 0,12, \sigma_1 = 1, \sigma_2 = 0.1$

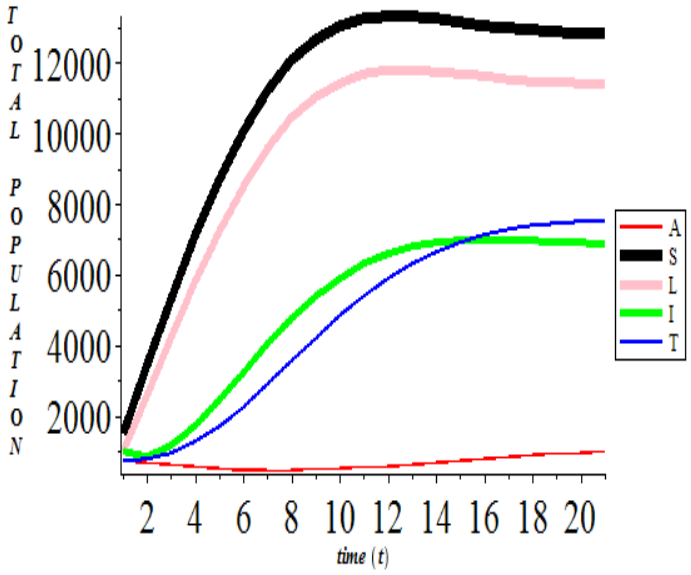

Fig. 4. Graph of total population when
$\Lambda = 2000, \beta = 0.2, \mu = 0.02, \delta = 0.01, \alpha = 0.01,$
$\theta = 0.1, \kappa = 0,12, \sigma_1 = 0.2, \sigma_2 = 0.3$

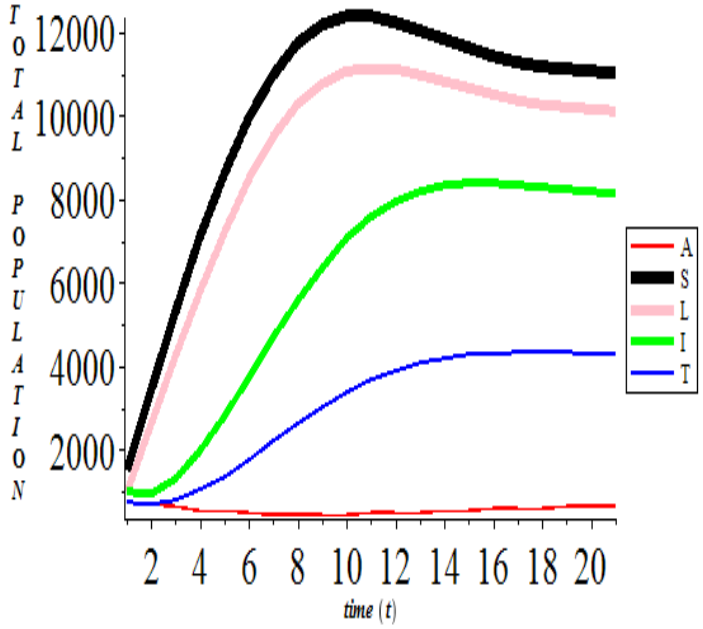

Fig. 5. Graph of total population when
$\Lambda = 2000, \beta = 0.2, \mu = 0.02, \delta = 0.01, \alpha = 0.01,$
$\theta = 0.1, \kappa = 0{,}12, \sigma_1 = 0.2, \sigma_2 = 0.6$

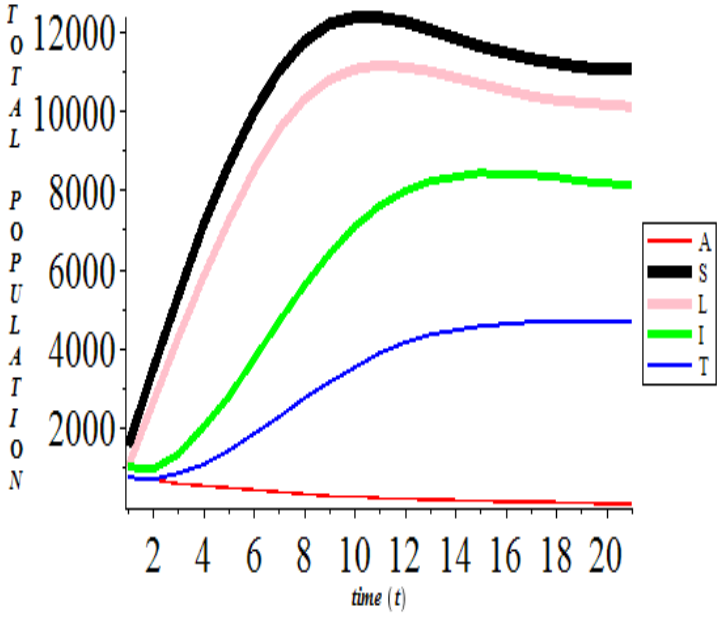

Fig. 6. Graph of total population when
$\Lambda = 2000, \beta = 0.2, \mu = 0.02, \delta = 0.01, \alpha = 0.01,$
$\theta = 0.1, \kappa = 0{,}12, \sigma_1 = 0.2, \sigma_2 = 1$

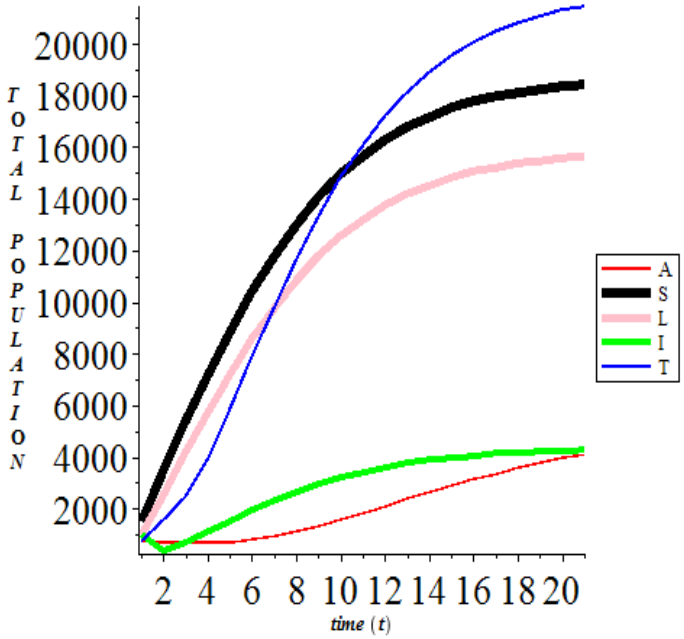

Fig. 7. Graph of Total Population when
$\Lambda = 2000, \beta = 0.2, \mu = 0.02, \delta = 0.01, \alpha = 0.01,$
$\theta = 0.1, \kappa = 0{,}12, \sigma_1 = 0.2, \sigma_2 = 0.1$

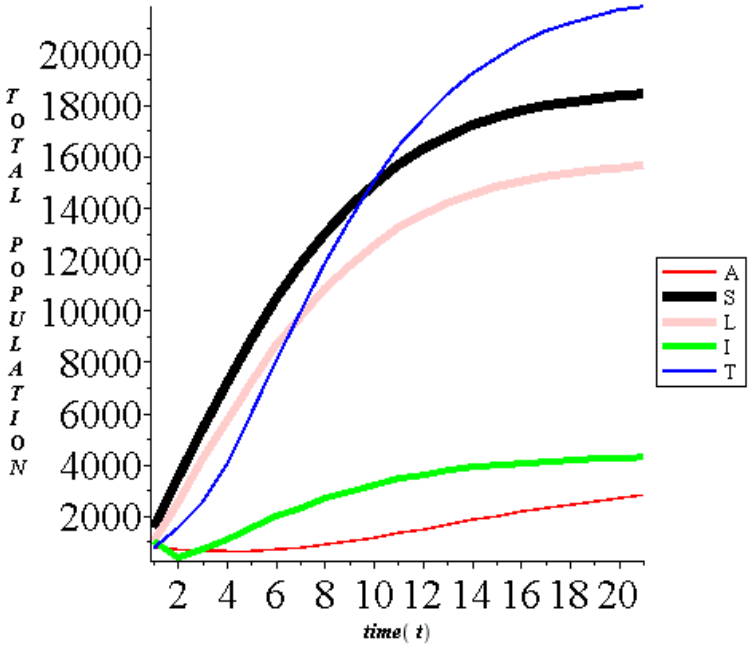

Fig. 8. Graph of total population when
$\Lambda = 2000, \beta = 0.2, \mu = 0.02, \delta = 0.01, \alpha = 0.01,$
$\theta = 0.6, \kappa = 0{,}12, \sigma_1 = 0.2, \sigma_2 = 0.1$

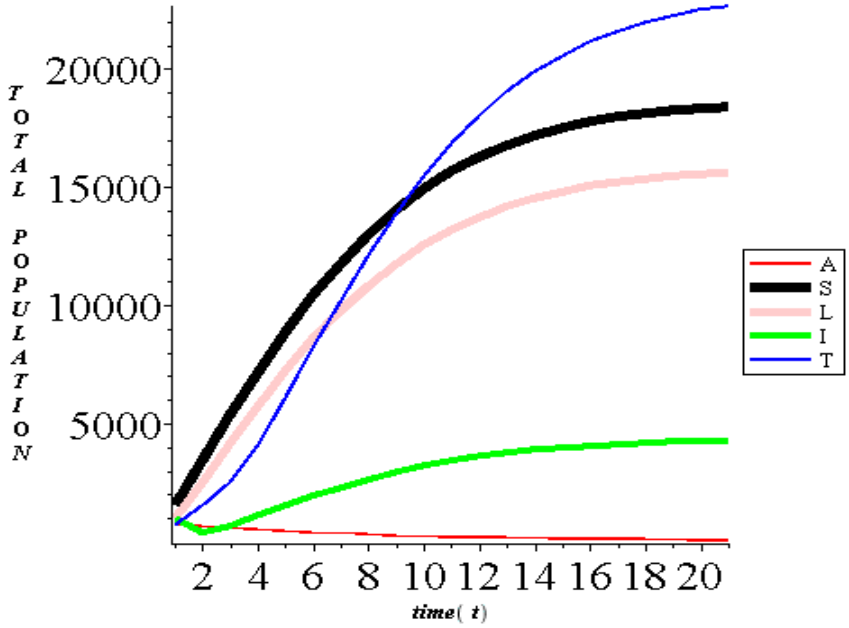

Fig. 9. Graph of total population when
$\Lambda = 2000, \beta = 0.2, \mu = 0.02, \delta = 0.01, \alpha = 0.01,$
$\theta = 1, \kappa = 0{,}12, \sigma_1 = 0.2, \sigma_2 = 0.1$

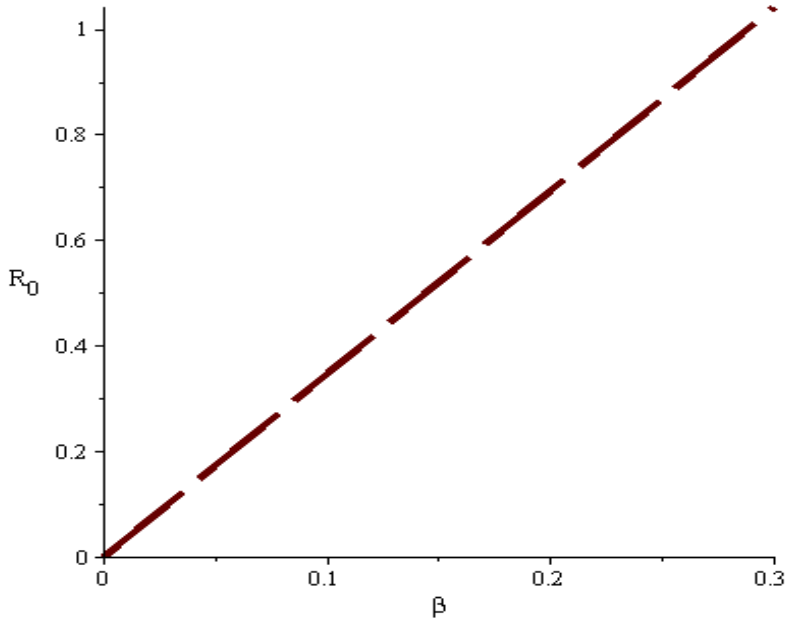

Fig. 10. Graph of basic reproduction number R_0 against effective contact rate ($\beta = 0.3$)

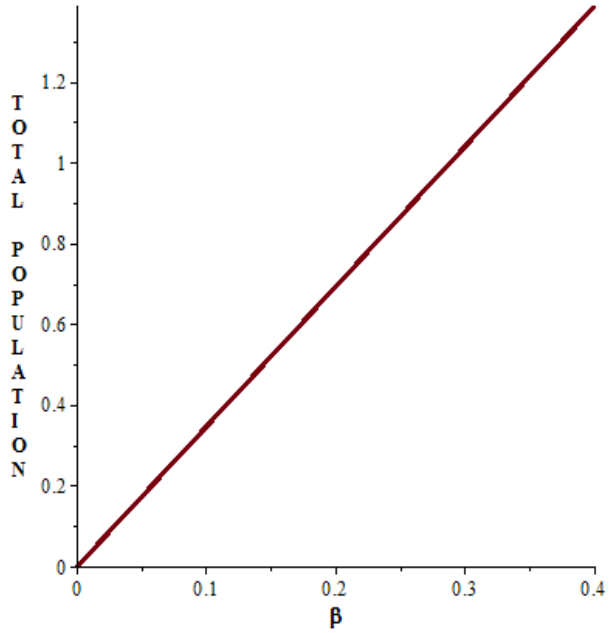

Fig. 11. Graph of basic reproduction number R_0 against effective contact rate ($\beta = 0.4$)

5 Discussion of Results

Five (5) compartmental mathematical model for the transmission of HIV/AIDS with immunity boost is presented and analyzed to gain insight into early treatment of HIV/AIDS and other epidemiological features that cause the progression from HIV to full blown AIDS. We analyzed the basic reproduction number R_o which determines whether disease dies off or spread, the result shows that the disease dies off whenever R_o is less than unity i.e. $R_o < 1$ but spread when $R_o > 1$. Sensitivity analysis of basic reproduction number R_o with respect to parameters shows the parameters that need to be checked by medical practitioners/policy health makers, parameters with positive index such as effective contact rate increases the basic reproduction number and must not exceed 0.3 to avoid endemic stage. The numerical analysis of the model shows the dynamical behavior of the epidemiological parameters used in the formulation of the model (8).

Fig. 1-6 analyzed the early treatment of latently and active infected individuals; the results show that starting treatment early is the most effective way to prevent HIV being progressed to AIDS, the higher/early the treatment of infected individuals the less the active full blown AIDS individuals. The early treatment lowers the viral load in HIV patients and prevented the progression to the last stage.

Fig. 7-9 show how immunity booster prevented HIV being progressed to full AIDS; HIV is a virus that attacks a specific type of immune system cell in the body known as T-cells, HIV-positive patients need extra vitamins and minerals to help repair and heal the damaged cells. Immunity booster plays a vital role in the dynamical control of HIV-AIDS. The result in Fig. 10 shows that when the immunity is full i.e. $\theta = 1$, it reduces the progression from HIV to full blown AIDS.

Figs. 10 and 11 depict the effect of effective contact rate on the basic reproduction number, the result shows that effective contact rate is one of the parameters that increases the spread of HIV i.e. the higher the contact rate the higher the basic reproduction number and the faster would be the spread in the community. Fig. 10 shows that R_0 is less than one (1) when effective contact rate is 0.3, while Fig. 11 shows it is greater than one (1) when contact rate is 0.4 and above.

6 Conclusion

Starting treatment as soon as possible after someone is diagnosed with HIV is better than delaying it, treatment should be started as early as possible after HIV is diagnosed and interventions that boost the immunity should be adopted by policy health makers to reduce the progression to full blown AIDS.

Competing Interests

Authors have declared that no competing interests exist.

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