An SEIAT Endemic Model for the Control of HIV/AIDS in Ethiopia

S. Shalini Priya and K. Ganesan*

***Abstract*—In this article, we formulate a multi-compartmental mathematical model for the transmission of HIV/AIDS in Ethiopia. We construct a system of differential equations for an SEIAT(susceptible, exposed, infected, diagnosed and under treatment) and we investigate the outbreak of HIV/AIDS and effect on Ethiopians. The analysis of the compartmental model is carried out using stability analysis. Our model exhibits two equilibrium points, disease free equilibrium points and endemic equilibrium points. The next generation matrix is used to determine the basic reproduction number R_0. Numerical example are provided to validate our results for both the disease free state and endemic state of each sub-model. We believe that this investigation will be more effective on reducing HIV/AIDS infection and stop spreading in Ethiopia. Finally, we have attempt to calibrate our proposed model with the real-world data and estimate future HIV transmission in Ethiopia after 2031.**

***Index Terms*—HIV/AIDS, mathematical model, next generation matrix and stability analysis.**

I. Introduction

HIV is caused by human immunodeficiency virus and it can spread through breast feeding, having sex, sharing injection and drug related equipments such as needle infected with positive HIV individuals. The most extreme stage of HIV infection is AIDS. During 2018 in Ethiopia, there were 690000 individuals with HIV, 23000 new HIV infected individuals and 11000 individual died from HIV/AIDS related disease [7].

HIV spread over three main pathways through disclosure to diseased bodily tissues, fluids, sexual contact and during breastfeeding or pregnancy from mother to child. There is no high threat of transmitting HIV/AIDS if treated to sputum, nasal secretion, spurus, saliva, tears, urine or vomit unless they are infected with blood. The main method of HIV transmission is through sexual interaction. The frequent method of transmission of HIV is through blood related products. Blood can be transmitted through sharing needles while using drugs, getting injured with a needle, donating blood or providing injections using non-sterile equipment. HIV infection contains three stages they are acute, chronic and AIDS(acquired immunodeficiency syndrome). Although there is no cure for HIV but ART(antiretroviral therapy) can suppress or prevent HIV from spreading one stage to the next stage. HIV drugs allow patients to have longer and better life. Main goal of ART is to lower a patients virus load that is unnoticeable. A virus load test can detect if the blood level of HIV infection is low.

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To reduce the impact of HIV, many protective and therapeutic options such as HIV/AIDS self-protection and ART are available. However, no significant achievements have been made particularly in Sub-Saharan Africa and Ethiopia. To acquire a better understanding of HIV infection, mathematical models have been developed. A mathematical model is a mathematical representation of a system of dynamics. It is critical for forecasting, analyzing and managing the dynamic system of HIV/AIDS infection. Several assumptions and parameters must be considered when building a mathematical model, which may be recreated using governing equations. Several models have been constructed to investigate the dynamics of HIV transmission.

Aaqid Mohi-Ud Din Bhat et al. [1] constructed multi-compartment mathematical model of HIV/AIDS transmission and dynamics. They produced a solution to use as the foundation for analysing the dynamics and spread of HIV/AIDS in India, as well as the results of simulations, which were also performed, with model outcomes compared to actual health data in India.

Amare Deribew et al. [2] suggested HIV or tuberculosis infection control programmes in combination with other partnership, engage more in community mobilisation of society and education to correct prevalent, misinformation and prejudice. They carried out a cross sectional survey in Southwest Ethiopia Gilgel Gibe research to investigate Urban and Rural residents towards HIV or tuberculosis.

Espitia et al. [4] discussed about cases of HIV/AIDS in 2019 detected in San Juan de Pasto. They formulated a system of nonlinear differential equation for HIV/AIDS. The model estimation parameters are evaluated with the total number of cases in HIV and AIDS. They applied Latin Hypercube Sampling Method to analyze the sensitivity to changes in their initial condition for bisexual, homosexual and heterosexual contacts with varying sexual behaviors.

Tucker et al. [6] discussed about ethical and social implications in order to cure HIV infection, model analysis for HIV treatment, historical analysis of HIV cure, ethics and sociological analysis. They suggested the HIV cure research theme based on theoretical research and empirical research.

Muhammad et al. [15] framed an SIR mathematical model for Covid-19 infection in Indonesia using the fuzzy parameters. They suggested that the treatment has a great impact in stopping or lowering the infection transmission, but not as much as implementing health protocols and the effect of vaccination.

Omondi et al. [17] formulated a model for HIV in Kenya for the two heterosexual age groups. Their findings can influence condom distribution, ART and HIV interventions. In particular, the findings can be used to teach young adults about practising safe and secure intercourse with their partners to be able to prevent the infection spread.

Sharp et al. [18] discussed the progression of HIV and the formation of AIDS and discovered that HIV is originated in closest living relatives, which creates a number of difficulties. Human and chimpanzee gene sequences frequently differ by less than two percentage points of nucleotides. Furthermore, a comparison of HIV(human immunodeficiency virus) and SIV(simian immunodeficiency virus) may yield host and virion factors which cause disease progression, leading the path for novel treatment strategies.

Shalini priya et al. [22] framed an $SEIR$ mathematical model and analyzed the epidemic of Monekeypox spread and its impact on population of United States. They had attempt to fit their model by using the real world data and their study is more efficient on controlling Monkeypox and also anticipated the future Monkeypox infection in US. They found that Monkeypox is controllable and can be fully eradicated in disease free state by vaccination and in endemic state, monkeypox cannot be destroyed by vaccination alone.

Tigabu Kasia Ayele et al. [24] framed an $S_U S_A LITA$ mathematical model for HIV/AIDS in Ethiopia. Their approach is expanded to include interventions such as therapy, screening and prevention. Furthermore, the optimal system is formulated and numerically solved by using forward and backward sweep approach. Finally, the affordability of these integrated control techniques is determined.

In this study, Our goal is to see if we can control HIV/AIDS using our assumptions. We also estimated the HIV/AIDS spread after 2022 in Ethiopia. We suggest the optimal strategy to prevent the HIV spread of the disease in susceptible individuals, as well as when ART and pre-exposure prophylaxis(PrEP) should be initiated to prevent patients from developing to the pre-AIDS stage. Graphs are provided to demonstrate the efficacy of the proposed technique in terms of both disease free equilibrium stability and endemic equilibrium stability.

In section II, we examine a normalized mathematical model of SEIAT with some of the control parameters. In section III, we investigate the equilibrium points of the HIV/AIDS model. In section IV, we determine the basic reproduction number R_0. In section V, we investigate the stability analysis of both the disease free state and endemic state and in section VI and VII, we engage with our model and come to a quick conclusion about it.

II. Method

We propose a five compartment mathematical model on spread of HIV/AIDS infection among human in both men and women. The human population is classified into five different groups susceptible $S(t)$, exposed $E(t)$, infected $I(t)$, diagnosed $A(t)$ and under treatment $T(t)$. The total population is $N(t) = S(t) + E(t) + I(t) + A(t) + T(t)$. The compartment model is used to develop an SEIAT model by taking consideration of HIV/AIDS infection, people who follow the health protocols, people who take preventive measures, force of infection and the rate of being exposed to infected. A SEIAT model analysis use the next generation matrix to obtain the basic reproduction number R_0 and as well as a stability study of the SEIAT model for the spread of HIV/AIDS. A discussion is held for SEIAT model and we gather data on the number of susceptible, exposed, infected, diagnosed and under treatment cases of HIV/AIDS in Ethiopia from 2021 to 2022. The important parameters are intercourse without protection, exposed person stop taking preventative measures and become unaware due to carelessness, diagnosed individual under treatment and HIV/AIDS infection.

The movement of the person from one compartment to another is shown in the following figure 1:

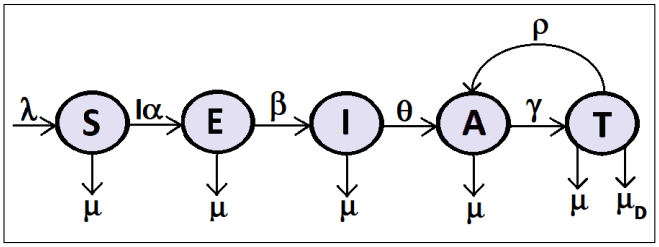

Fig. 1. Mathematical model of HIV/AIDS

This SEIAT model is expressed in the form of a system of linear differential equations:

$$\frac{dS}{dt} = \lambda - \mu S - I\alpha S \tag{1}$$

$$\frac{dE}{dt} = I\alpha S - \mu E - \beta E \tag{2}$$

$$\frac{dI}{dt} = \beta E - \mu I - \theta I \tag{3}$$

$$\frac{dA}{dt} = \theta I - \mu A - \gamma A + \rho T \tag{4}$$

$$\frac{dT}{dt} = -\rho T\gamma A - \mu T - \mu_D T \tag{5}$$

TABLE I
Parameters description

Parameters	Description
λ	Natural birth rate
μ	Natural mortality rate
μ_D	HIV/AIDS mortality rate
α	Rate of intercourse without protection
β	Rate of exposed person stop taking precaution and become unaware due to carelessness
θ	Rate of infected person undergo screening to check whether they have HIV/AIDS infection
γ	Rate of diagnosed person under treatment
ρ	Diagnosed rate of treatment for HIV infected person without clinical symptoms of AIDS

III. The Equilibrium Points

There are two types of equilibrium points, disease free and endemic. To determine these two equilibrium points every one of the equation (1) - (5) should be equal to zero,

$$0 = \lambda - \mu S - I\alpha S \tag{6}$$

$$0 = I\alpha S - \mu E - \beta E \tag{7}$$

$$0 = \beta E - \mu I - \theta I \tag{8}$$

$$0 = \theta I - \mu A - \gamma A + \rho T \tag{9}$$

$$0 = -\rho T + \gamma A - \mu T - \mu_D T \tag{10}$$

It has a certain initial conditions, where $S(0) \geq 0$, $E(0) \geq 0$, $I(0) \geq 0$, $A(0) \geq 0$ and $T(0) \geq 0$.

A. Disease free equilibrium for SEIAT model

The disease free equilibrium has some certain conditions, when there is lack of HIV/AIDS spread, then $E = E^0 = 0$, $I = I^0 = 0$, $A = A^0 = 0$ and $T = T^0 = 0$. From (6) - (10), we get our disease free equilibrium points for SEIAT model as follows:
From (6), $0 = \lambda - \mu S - I\alpha S \Rightarrow S = S^0 = \lambda\mu$.
From (7), (8), (9) and (10), we have $E = E^0 = 0, I = I^0 = 0, A = A^0 = 0$ and $T = T^0 = 0$.

B. Endemic equilibrium for SEIAT model

The endemic equilibrium has certain conditions, where there is possibilities of HIV/AIDS spread, then $S = S^* \neq 0$, $E = E^* \neq 0$, $I = I^* \neq 0$, $A = A^* \neq 0$ and $T = T^* \neq 0$. From (6) - (10), we get our endemic equilibrium points for SEIAT model as follows:
From (6), $0 = \lambda - \mu S - I\alpha S \Rightarrow \mu S + I\alpha S = \lambda$

$$\Rightarrow \boxed{S = S^* = \frac{\lambda}{\mu + I\alpha}} \tag{11}$$

From (7), $0 = I\alpha S - \mu E - \beta E \Rightarrow \mu E + \beta E = I\alpha S$

$$\Rightarrow \boxed{E = E^* = \frac{I\alpha\lambda}{(\mu + I\alpha)(\mu + \beta)}} \tag{12}$$

From (8), $0 = \beta E - \mu I - \theta I \Rightarrow I = \dfrac{\beta E}{\mu + \theta}$
Sub (12) in I, we have

$$I = \frac{\beta I\alpha\lambda}{(\mu + \beta)(\mu + I\alpha)(\mu + \theta)}$$

$$\Rightarrow \mu + I\alpha = \frac{\beta\alpha\lambda}{(\mu + \beta)(\mu + \theta)}$$

$$\Rightarrow I\alpha = \frac{\beta\alpha\lambda}{(\mu + \beta)(\mu + \theta)} - \mu$$

$$\Rightarrow I = \frac{\beta\alpha\lambda}{(\mu + \beta)(\mu + \theta)} - \frac{\mu}{\alpha}$$

$$\Rightarrow \boxed{I = I^* = \frac{\alpha\beta\lambda - \mu(\mu + \beta)(\mu + \theta)}{\alpha(\mu + \beta)(\mu + \theta)}} \tag{13}$$

From (9), $0 = \theta I - \mu A - \gamma A + \rho T$

$$\Rightarrow \mu A + \gamma A = \theta I + \rho T$$
$$\Rightarrow A(\mu + \gamma) = \theta I + \rho T \Rightarrow A = \frac{\theta I + \rho T}{\mu + \gamma}$$

Sub (13) in A, we have

$$\Rightarrow A = \frac{\theta\left(\dfrac{\alpha\beta\lambda - \mu(\mu + \beta)(\mu + \theta)}{\alpha(\mu + \beta)(\mu + \theta)}\right) + \rho T}{\mu + \gamma}$$

$$\Rightarrow \boxed{A = A^* = \frac{\theta\alpha\beta\lambda - \mu(\mu + \beta)(\mu + \theta) + \rho T}{\mu + \gamma}} \tag{14}$$

From (10), $0 = -\rho T + \gamma A - \mu T - \mu_D T$

$$\Rightarrow \rho T + \mu T + \mu_D T = \gamma A$$
$$\Rightarrow T = \frac{\gamma A}{\rho + \mu + \mu_D}$$

Sub (14) in $T = \dfrac{\gamma A}{\rho + \mu + \mu_D}$, we have

$$\Rightarrow T = \frac{\gamma\left(\dfrac{\theta\alpha\beta\lambda - \mu(\mu + \beta)(\mu + \theta)}{\mu + \gamma} + \rho T\right)}{\gamma + \mu + \mu_D}$$

$$\Rightarrow T = \frac{\gamma\theta\alpha\beta\lambda - \mu(\mu + \beta)(\mu + \theta)(\mu + \gamma) + \rho T}{(\mu + \gamma)(\gamma + \mu + \mu_D)}$$

$$\Rightarrow T((\gamma + \mu + \mu_D) - \rho) = \frac{\gamma\theta\alpha\beta\lambda - \mu(\mu + \beta)(\mu + \theta)}{\mu + \gamma}$$

$$\Rightarrow \boxed{T = T^* = \frac{\gamma\theta\alpha\beta\lambda - \mu(\mu + \beta)(\mu + \theta)}{(\mu + \gamma)(\gamma + \mu + \mu_D) - \rho}} \tag{15}$$

IV. Basic Reproduction Number

According to our proposed model, disease free state are E, I, A and T whereas endemic state are S, E, I, A and T. The basic reproduction number is denoted by R_0, an infection in which the expected number of human cases is directly caused by one to many incidents in a human population. This is a crucial parameter to consider when analysing the long-term behaviour of an endemic state. The basic reproduction number R_0 for a compartmental model of the spread of bacteria is derived from the next-generation matrix. It establishes a requirements for the stability of the disease-free equilibrium. We take into account $\mathcal{F}$ is the rate at which new infections appears in compartment, $\mathcal{V}^{-1}$ is the rate at which peoples are moved out of the compartment and $\mathcal{V}^{+1}$ is the rate at which peoples are moved into the compartment, where $\mathcal{V} = \mathcal{V}^{-1} - \mathcal{V}^{+1}$ [21].

We consider (2)-(5).

$$\begin{aligned}
\frac{dE}{dt} &= I\alpha S - \mu E - \beta E \\
\frac{dI}{dt} &= \beta E - \mu I - \theta I \\
\frac{dA}{dt} &= \theta I - \mu A - \gamma A + \rho T \\
\frac{dT}{dt} &= -\rho T + \gamma A - \mu T - \mu_D T
\end{aligned}$$

Jacobian matrix of $\mathcal{F}$ and $\mathcal{V}$ is

$$\mathcal{F} = \begin{pmatrix} I\alpha S \\ 0 \\ 0 \\ 0 \end{pmatrix} \text{ and } \mathcal{V} = \begin{pmatrix} -\mu E - \beta E \\ \beta E - \mu I - \theta I \\ \theta I - \mu A - \gamma A + \rho T \\ -\rho T + \gamma A - \mu T - \mu_D T \end{pmatrix}$$

where $F = \begin{pmatrix} 0 & \alpha S & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$.

In disease free state, since (S,E,I,A,T)=(1,0,0,0,0),

we have $F = \begin{pmatrix} 0 & \alpha & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$ and

$$V = \begin{pmatrix} -\mu-\beta & 0 & 0 & 0 \\ \beta & -\mu-\theta & 0 & 0 \\ 0 & \theta & -\mu-\gamma & \rho \\ 0 & 0 & \gamma & -\rho-\mu-\mu_D \end{pmatrix}.$$

We consider $A = (-\mu-\gamma)$, $B = (-\rho-\mu-\mu_D)$, $C = (-\mu-\theta)$ and $D = (-\mu-\beta)$.

$$adj\ V = \begin{bmatrix} AB & AB-\rho\gamma & 0 & 0 \\ -(AB-\rho\gamma) & -(AB-\rho\gamma) & 0 & -\theta\rho \\ \theta B & \theta B & CB & B\rho \\ -\theta\gamma & -\theta\gamma & C\gamma & CA \end{bmatrix}$$

$$|V| = \frac{1}{DCAB-\rho\gamma}$$

$$V^{-1} = \begin{bmatrix} \frac{1}{DC} & \frac{1}{DC} & \frac{1}{DA-\rho\gamma} & \frac{1}{D(AB-\rho)} \\ \frac{-1}{DC} & \frac{-1}{DC} & \frac{-1}{DAB-\rho} & \frac{1}{DB-\rho\gamma} \end{bmatrix}$$

$$FV^{-1} == \begin{bmatrix} \frac{-\alpha}{DC} & \frac{-\alpha}{DC} & 0 & \frac{-\alpha\theta}{D[CAB-\gamma]} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

The characteristic equation of FV^{-1} is

$$= \begin{bmatrix} \frac{-\alpha}{DC}-\lambda & \frac{-\alpha}{DC} & 0 & \frac{-\alpha\theta}{D[CAB-\gamma]} \\ 0 & 0-\lambda & 0 & 0 \\ 0 & 0 & 0-\lambda & 0 \\ 0 & 0 & 0 & 0-\lambda \end{bmatrix} = 0$$

$$\left[\frac{-\alpha}{DC}-\lambda\right](-\lambda)(\lambda^2-0)-0+0-0=0$$

$$\lambda_1 = \frac{-\alpha}{(-\mu-\beta)(-\mu-\theta)},\ \lambda_2 = 0,\ \lambda_3 = 0,\ \lambda_4 = 0$$

Since λ_1 is the dominant eigen value and the basic reproduction number is,

$$R_0 = \frac{\alpha}{(\mu+\beta)(\mu+\theta)} \tag{16}$$

V. Stability Analysis

Theorem 1: If $R_0 < 1$ then the disease free equilibrium is asymptotically stable and if $R_0 > 1$ then it is unstable.
Proof: From the equation (1)-(5), we frame the Jacobian matrix J as follows,

$$J = \begin{bmatrix} -\mu-I\alpha & 0 & -\alpha S & 0 \\ I\alpha & -\mu-\beta & \alpha S & 0 \\ 0 & \beta & -\mu-\theta & 0 \\ 0 & 0 & \theta & -\mu-\gamma \\ 0 & 0 & 0 & \gamma \end{bmatrix} \begin{bmatrix} 0 \\ 0 \\ 0 \\ \rho \\ -\rho-\mu-\mu_D \end{bmatrix}$$

Since S=1 and E=I=A=T=0 in disease free equilibrium, then we obtain Jacobian matrix J as,

$$J = \begin{bmatrix} -\mu & 0 & -\alpha & 0 & 0 \\ 0 & -\mu-\beta & \alpha & 0 & 0 \\ 0 & \beta & -\mu-\theta & 0 & 0 \\ 0 & 0 & \theta & -\mu-\gamma & \rho \\ 0 & 0 & 0 & \gamma & -\rho-\mu-\mu_D \end{bmatrix}$$

The characteristic equation of the Jacobian matrix J becomes,

$$\begin{vmatrix} -\mu-\lambda & 0 & -\alpha & 0 \\ 0 & -\mu-\beta-\lambda & \alpha & 0 \\ 0 & \beta & -\mu-\theta-\lambda & 0 \\ 0 & 0 & \theta & -\mu-\gamma-\lambda \\ 0 & 0 & 0 & \gamma \end{vmatrix} \begin{matrix} 0 \\ 0 \\ 0 \\ \rho \\ -\rho-\mu-\mu_D-\lambda \end{matrix} \Bigg| = 0$$

$$\begin{aligned} \Rightarrow &(-\mu-\lambda)(-\mu-\beta-\lambda)(-\mu-\theta-\lambda)[(-\mu- \\ &\gamma-\lambda)(-\rho-\mu-\mu_D-\lambda)-\rho\gamma]-\alpha(\beta)[(-\mu \\ &-\gamma-\lambda)(-\rho-\mu-\mu_D-\lambda)-\rho\gamma]=0 \end{aligned} \tag{17}$$

Solving (17), we have $\lambda_1 = -\mu$, $\lambda_2 = -\mu-\beta$, $\lambda_3 = -\mu-\theta$ and $[(-\mu-\gamma-\lambda)(-\rho-\mu-\mu_D-\lambda)-\rho\gamma]-\alpha(\beta)[(-\mu-\gamma-\lambda)(-\rho-\mu-\mu_D-\lambda)-\rho\gamma]=0$

$$\begin{aligned} &[(-\mu-\gamma-\lambda)(-\rho-\mu-\mu_D-\lambda)-\rho\gamma]-\alpha(\beta) \\ &[(-\mu-\gamma-\lambda)(-\rho-\mu-\mu_D-\lambda)-\rho\gamma]=0 \end{aligned} \tag{18}$$

Substitute (16) in (18) for α,

$$[(-\mu-\gamma-\lambda)(-\rho-\mu-\mu_D-\lambda)-\rho\gamma]-(\mu+\beta)(\mu+\theta)$$
$$R_0[\beta(-\mu-\gamma-\lambda)(-\rho-\mu-\mu_D-\lambda)-\rho\gamma]=0$$

When $R_0 < 1$,

$$\lambda_4 = -\mu-\gamma\ and\ \lambda_5 = -\rho-\mu-\mu_D$$

When $R_0 > 1$,

$$\Rightarrow[(-\mu-\gamma-\lambda)(-\rho-\mu-\mu_D-\lambda)-\rho\gamma]-(\mu+\beta)(\mu+\theta)2[\beta(-\mu-\gamma-\lambda)(-\rho-\mu-\mu_D-\lambda)-\rho\gamma]=0$$
$$\Rightarrow(-\mu-\gamma-\lambda)(-\rho-\mu-\mu_D-\lambda)[1-(\mu+\beta)(\mu+\theta)2\beta]=0$$
$$\Rightarrow(\mu\rho+\mu^2+\mu\mu_D+\mu\lambda+\gamma\rho+\gamma\mu+\gamma\mu_D+\gamma\lambda+\lambda\rho+\lambda\mu+\lambda\mu_D+\lambda^2)[1-(\mu^2+\mu\theta+\mu\beta+\beta\theta)2\beta]=0$$
$$\Rightarrow\lambda^2+\lambda(2\mu+\gamma+\rho+\mu_D)+\mu\rho+\mu\mu_D+\gamma\rho+\gamma\mu+\gamma\mu_D[(1-\mu\theta-\mu\beta-\beta\theta)2\beta]=0$$

On solving this quadratic equation to λ_4 and λ_5, we have

$$\lambda=\frac{-(2\mu+\gamma+\rho+\mu_D)\pm\sqrt{(2\mu+\gamma+\rho+\mu_D)^2-\frac{4(\mu\rho+\mu\mu_D+\gamma\rho+\gamma\mu+\gamma\mu_D(1-\mu\theta-\mu\beta-\beta\theta)2\beta)}{2}}}{2}$$

Let us assume $\beta = 0$, because there is no exposed individual to become infected in the disease free state.

$$\Rightarrow-\frac{1}{2}\left[-(2\mu+\gamma+\mu+\mu_D)\pm\sqrt{(2\mu+\gamma+\mu+\mu_D)^2}\right]$$
$$\Rightarrow-\frac{1}{2}[-(2\mu+\gamma+\mu+\mu_D)\pm(2\mu+\gamma+\mu+\mu_D)]$$

There are two possible ways,

$$\lambda=-\frac{1}{2}[-(2\mu+\gamma+\mu+\mu_D)+(2\mu+\gamma+\mu+\mu_D)$$
$$\lambda=0$$
$$(or)$$
$$\lambda=-\frac{1}{2}[-(2\mu+\gamma+\mu+\mu_D)-(2\mu+\gamma+\mu+\mu_D)$$
$$=-\frac{1}{2}(-2\mu-\gamma-\mu-\mu_D-2\mu-\gamma-\mu-\mu_D)$$
$$=-\frac{1}{2}2(-2\mu-\gamma-\mu-\mu_D)$$
$$\lambda=2\mu+\gamma+\mu+\mu_D$$

The disease free equilibrium is asymptotically stable when $R_0 < 1$ and unstable if $R_0 > 1$.

Theorem 2: If $R_0 > 1$ then the endemic equilibrium is stable and if $R_0 < 1$ then it is unstable.
Proof: From equation (1)-(5), we frame the Jacobian matrix J_1 as follows,

$$J=\begin{bmatrix}-\mu-I\alpha & 0 & -\alpha S & 0 & 0\\ I\alpha & -\mu-\beta & \alpha S & 0 & 0\\ 0 & \beta & -\mu-\theta & 0 & 0\\ 0 & 0 & \theta & -\mu-\gamma & \rho\\ 0 & 0 & 0 & \gamma & -\rho-\mu-\mu_D\end{bmatrix}$$

To simplify our work, let us assume $a_1 = -\mu - I\alpha$, $a_2 = -\mu-\beta$, $a_3 = -\mu-\theta$, $a_4 = -\mu-\gamma$ and $a_5 = -\rho-\mu-\mu_D$.

Then the Jacobian matrix J_1 becomes:

$$J_1=\begin{bmatrix}a_1 & 0 & -\alpha S & 0 & 0\\ I\alpha & a_2 & \alpha S & 0 & 0\\ 0 & \beta & a_3 & 0 & 0\\ 0 & 0 & \theta & a_4 & \rho\\ 0 & 0 & 0 & \gamma & a_5\end{bmatrix}$$

The characteristic equation of the Jacobian matrix J_1 becomes,

$$\begin{vmatrix}a_1-\lambda & 0 & -\alpha S & 0 & 0\\ I\alpha & a_2-\lambda & \alpha S & 0 & 0\\ 0 & \beta & a_3-\lambda & 0 & 0\\ 0 & 0 & \theta & a_4-\lambda & \rho\\ 0 & 0 & 0 & \gamma & a_5-\lambda\end{vmatrix}=0$$

$$(a_1-\lambda)(a_2-\lambda)(a_3-\lambda)[(a_4-\lambda)(a_5-\lambda)-\rho\gamma]-\alpha S\beta[(a_4-\lambda)(a_5-\lambda)-\rho\gamma]-I\alpha\alpha S\beta[(a_4-\lambda)(a_5-\lambda)-\rho\gamma]=0 \quad (19)$$

Solving (19), we have $\lambda_1 = a_1$, $\lambda_2 = a_2$, $\lambda_3 = a_3$ and $[(a_4-\lambda)(a_5-\lambda)-\rho\gamma]-\alpha S\beta[(a_4-\lambda)(a_5-\lambda)-\rho\gamma]-I\alpha\alpha S\beta[(a_4-\lambda)(a_5-\lambda)-\rho\gamma]=0$.

Where $\lambda_1 = -(\mu+I\alpha)$, $\lambda_2 = -(\mu+\beta)$, $\lambda_3 = -(\mu+\theta)$.

$$[(a_4-\lambda)(a_5-\lambda)-\rho\gamma]-\alpha S\beta[(a_4-\lambda)(a_5-\lambda)-\rho\gamma]-I\alpha\alpha S\beta[(a_4-\lambda)(a_5-\lambda)-\rho\gamma]=0$$
$$(a_4-\lambda)(a_5-\lambda)[-\alpha S\beta(1+I\alpha)-\rho\gamma]=0$$

In the endemic equilibrium, $S = S^* \neq 0$ and $I = I^* \neq 0$, so we take $S = 1$ and $I = 1$ and we substitute in the above equation,

$$(a_4-\lambda)(a_5-\lambda)[-\alpha\beta(1+\alpha)-\rho\gamma]=0 \quad (20)$$

Substitute (16) in (20) for α, we have

$$(a_4-\lambda)(a_5-\lambda)[-(\mu+\beta)(\mu+\theta)R_0\beta (1+(\mu+\beta)(\mu+\theta)R_0)-\rho\gamma]=0$$

When $R_0 > 1$, $\lambda = a_4$ and $\lambda = a_5$, where $\lambda_4 = -(\mu+\gamma)$ and $\lambda_5 = -(\rho+\mu+\mu_D)$ When $R_0 < 1$, $\lambda = 0$.

The endemic equilibrium is stable when $R_0 > 1$ and unstable if $R_0 < 1$.

VI. Result and discussion

A discussion is done by using initial values for S, E, I, A and T which determined based on the data of Ethiopia from January 2021 to December 2022 and it is given in table II.

TABLE II
Initial values for HIV/AIDS used in our model

Variables	Values	References
$S(0)$	1,550,000	[25]
$E(0)$	775,000	Assumption
$I(0)$	609,349	[26]
$A(0)$	487,476	Assumption
$T(0)$	487,476	Assumption

TABLE III
VALUES OF PARAMETERS FOR HIV/AIDS USED IN OUR MODEL

Param -eters	Values	Description	References
λ	0.29	Natural birth rate	[25]
μ	0.06	Natural mortality rate	[24]
μ_D	0.016	HIV/AIDS mortality rate	[24]
α	0.5	Rate of intercourse without protection	Assumption
β	0.3	Rate of exposed person stop taking preventive measure and become unaware due to carelessness	[24]
θ	0.1	Rate of infected person undergo screening to check whether they have HIV/AIDS infection	[24]
γ	0.01	Rate of diagnosed person under treatment	[24]
ρ	0.25	Diagnosed rate of treatment for HIV infected person without clinical symptoms of AIDS	[24]

Table III displays the parameters value of the SEIAT compartmental mathematical model for HIV/AIDS in Ethiopia.

The system $(1)-(5)$ is evaluated for the different set of parameters that satisfy the conditions of the stability analysis of both disease free equilibrium points and endemic equilibrium points. In the numerical simulation, we have expressed x axis as months from January 2021 to December 2022 as 24 months and y axis as total number of individuals infected by HIV/AIDS in Ethiopia. We have taken the five compartments as susceptible, exposed, infected, diagnosed and under treatment.

Figure 2 indicates the SEIAT graph of HIV/AIDS in Ethiopia from January 2021 to December 2022. If this trend continues in 2023, both disease free equilibrium points and endemic equilibrium points changes and the disease free equilibrium stability is shown in figure 3, for the five compartmental mathematical model susceptible, exposed, infected, diagnosed and under treatment when $R_0 < 1$. The endemic equilibrium is shown in figure 4 for the five compartmental mathematical model susceptible, exposed, infected, diagnosed and under treatment when $R_0 > 1$.

Figure 5 represent the effect of α on exposed population. It clearly indicates that the exposed population decreases whenever we increases the parameter α values, figure 6 represent the effect of α on infected population. It clearly indicates that the infected population decreases whenever we increases the parameter α values, figure 7 represent the effect of α on diagnosed population. It clearly indicates that the diagnosed population gradually decreases whenever we increases the parameter α values. Figure 8 represent the effect of β on exposed population and it indicates that the exposed population decreases gradually whenever we increases β values, figure 9 represent the effect of β on infected population and it clearly indicates that the infected population decreases whenever we increases β values. Figure 10 represent the SEIAT graph of HIV/AIDS in Ethiopia from 2021 to 2031 and we can certainly notice a reduction in HIV cases.

Figure 11 and 12 shows the relationship between R_0 with other parameters(α, β and θ). Figure 11 shows that the rate of α increases with increase in R_0 and the rate of θ increases with increase in R_0. Figure 12 shows that the rate of β increases with decrease in R_0 and the rate of θ increases with decrease in R_0.

It has also been discovered that the disease becomes more endemic as a result of immigration and that the disease spread decreases when infective become aware of their infection after screening, contact tracing and do not engage in sexual behavior, however it increases when evidence of contact is not performed. In the absence of contact tracing and screening, infected people continue to spread the HIV disease since they are unaware of their infection and it is figured out clearly. We predicted that in Ethiopia there will be no new HIV cases by using our SEIAT mathematical model and by following precautions, usage of condoms and aware of HIV affected people which was mentioned by us. Following these suggestions one can see HIV less Ethiopia in 2031.

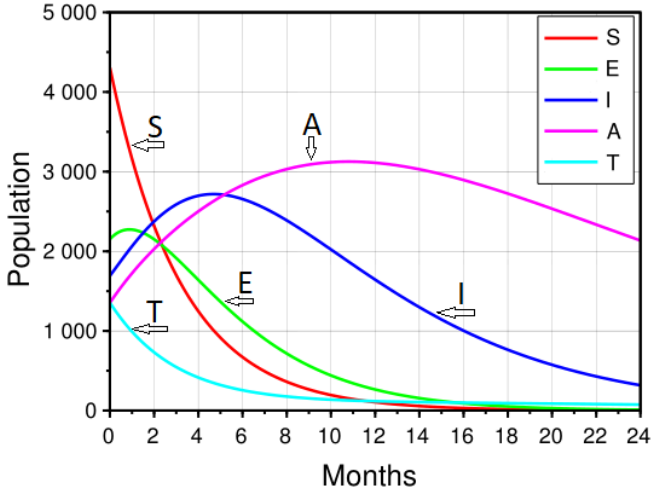

Fig. 2. SEIAT graph of HIV/AIDS in Ethiopia from January 2021 to December 2022

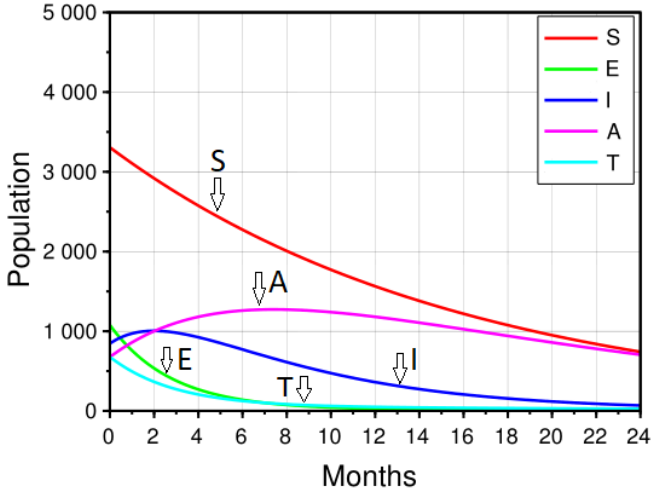

Fig. 3. Analysis of the disease free equilibrium's stability when $R_0 < 1$

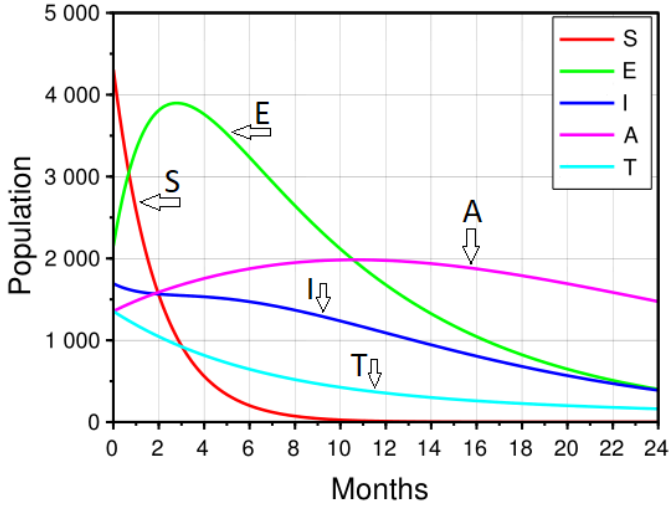

Fig. 4. Stability analysis of the endemic equilibrium when $R_0 > 1$

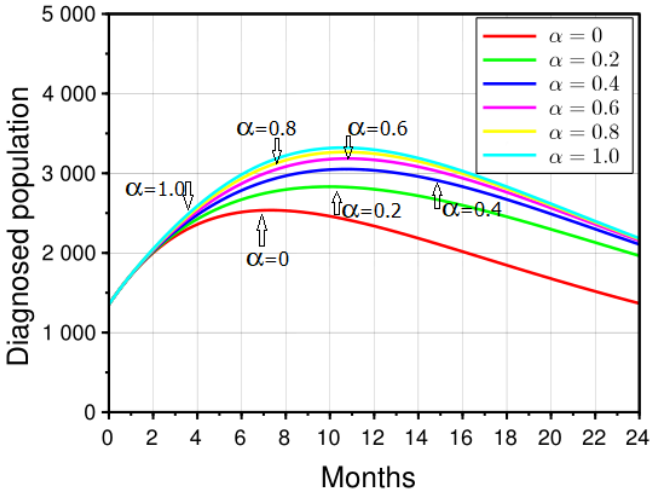

Fig. 7. The curves of the diagnosed population for different values of α

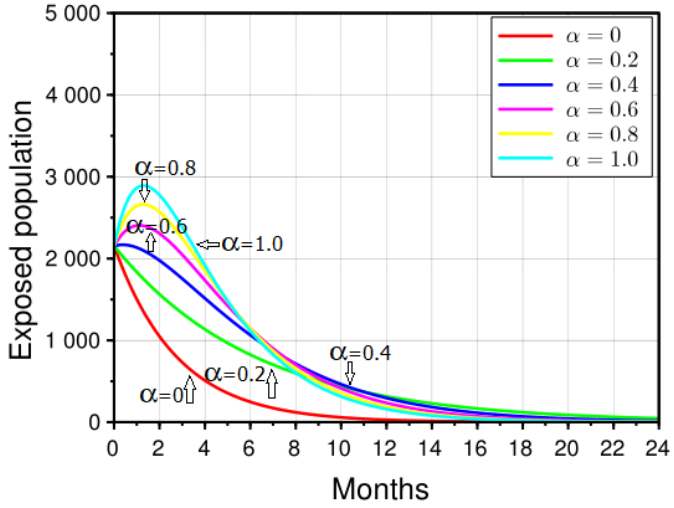

Fig. 5. The curves of the exposed population for different values of α

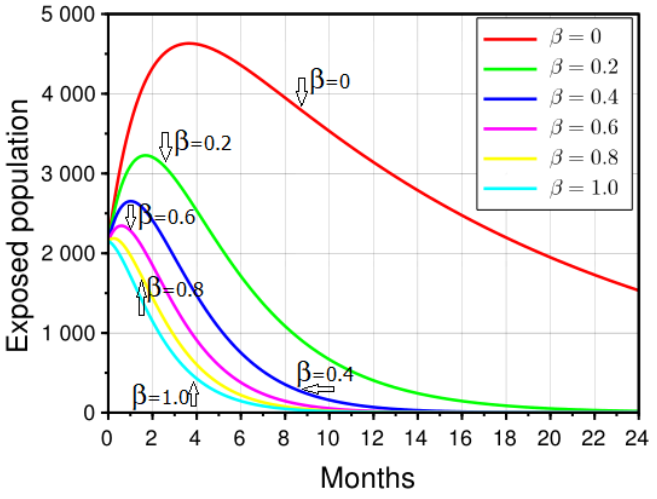

Fig. 8. The curves of the exposed population for different values of β

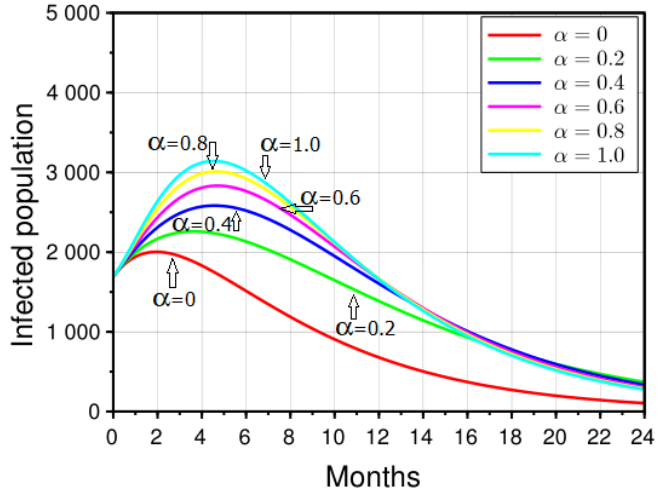

Fig. 6. The curves of the infected population for different values of α

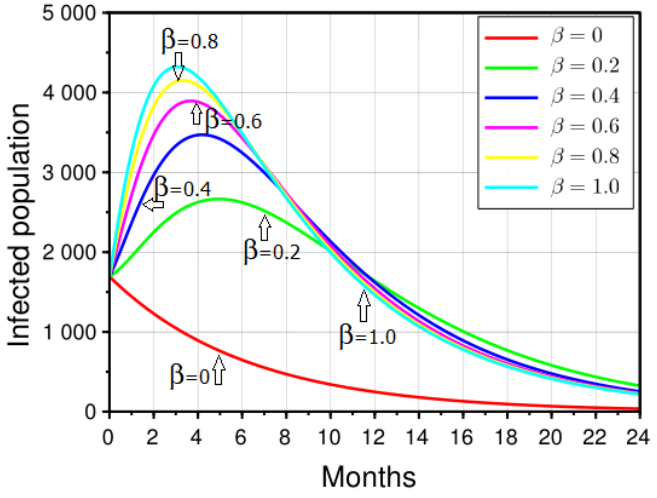

Fig. 9. The curves of the infected population for different values of β

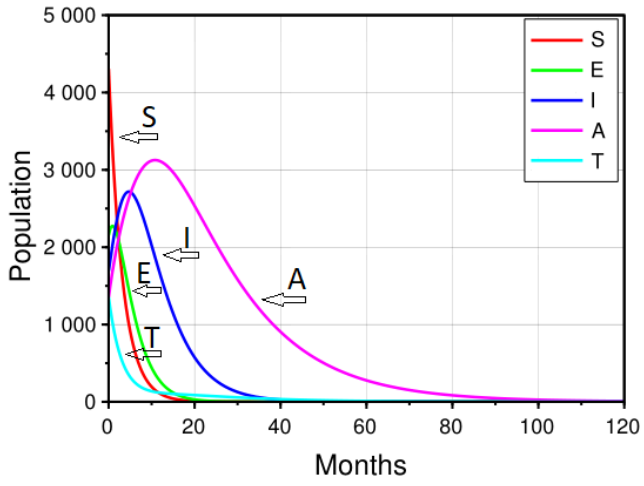

Fig. 10. SEIAT graph of HIV/AIDS in Ethiopia from 2021 to 2031

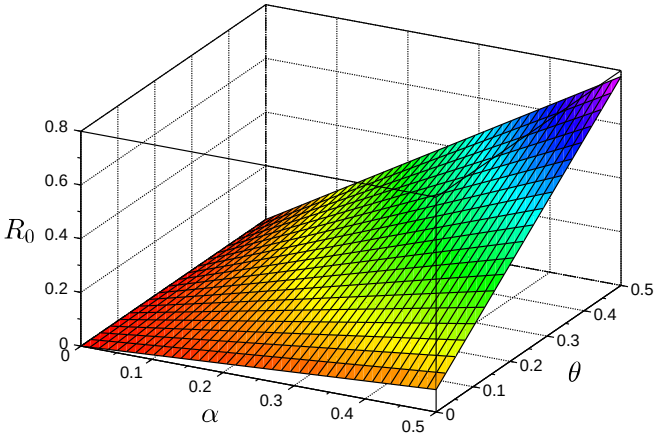

Fig. 11. Variation of α and θ in R_0

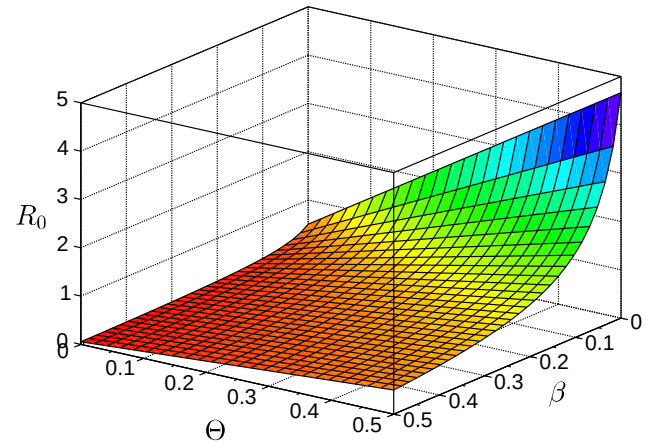

Fig. 12. Variation of β and θ in R_0

VII. Conclusion

In this article, we have discussed HIV/AIDS spread in Ethiopia during January 2021 to December 2022. We have framed SEIAT mathematical model for HIV/AIDS infection as a system of linear differential equations and examined the spread of the disease. We discovered that HIV/AIDS is controlled in disease free state by using protection and be aware of HIV infection. Whereas, in the endemic state, HIV/AIDS may not be controlled by using protection and aware of HIV infection alone and status will continue to exist for some time. More diagnosis and severe treatment will increase the life span of the affected people with HIV. We also predicted the HIV/AIDS infection after 2022 in Ethiopia. The results illustrate that the spread and treatment rate have the greatest impact on basic reproduction number. This indicates that HIV/AIDS control and prevention efforts should focus more on spreading preventative campaigns. Campaign aimed at those at high risk of HIV/AIDS infection to encourage them to utilise preventative measures such as PrEP and condoms will be more effective. By providing treatment for the HIV patients their immune system is strengthened and thus developing AIDS is reduced. Although increased unsafe sex behavior reduced PrEP effectiveness, PrEP use was even more favorable than the current condition. Early treatment for HIV diagnosis has also significantly reduced HIV incidence. However, early ART did not show any sufficient benefit. As predicted, it would be more successful if all measures (PrEP, early diagnosis and treatment) were done simultaneously. Furthermore, enlisting more affected people on ART would be perfect in minimizing the number of new infected people because it is known to help decrease the virus load in the human body, avoiding further spread due to HIV infections. It is therefore necessary to allocate additional materials to HIV prevention and treatment programmes that are specifically aimed to vulnerable, exposed and infected persons. Furthermore, the formulated model predicts that there will be decrease in the infectious cases, as there has yet to be a viable treatment available to eradicate the virus/disease. As a result, our proposed approach and its outputs could be extremely useful for understanding HIV/AIDS transmission dynamics in Ethiopia and around the world. Futhermore, the model is expected to help researchers and biologists from other countries in assessing their strategies for preventing and reducing the spread of this endemic.

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