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Original Article

Mathematical model analysis on the transmission of HIV/AIDS dynamic model with treatment

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ABSTRACT

In this paper, a non-linear mathematical model is proposed based on the mathematical model analysis of the transmission of HIV/AIDS in a dynamic model with treatment. We consider a compartmental mode together with non-linear differential equations in five-dimensional systems to detect the spread and control of the disease. We use the next-generation matrix technique to drive the formula for the basic reproduction number. The disease-free equilibrium of the HIV/AIDS model is stable if the basic reproduction number is less than unity; if the basic reproduction number is greater than unity, the disease-free equilibrium is unstable. We also utilize numerical simulation to explain how each parameter affects the basic reproduction number.

Keywords: HIV/AIDS, Vertical transmission, Basic reproduction number, stability, equilibrium point, Sensitivity Analysis

1. INTRODUCTION

The study of the prevalence and causes of both infectious and non-infectious diseases is known as epidemiology. The word was first restricted to the study of infectious epidemic diseases (Narasimhamurthy, 2012). Mathematical models are now crucial tools for understanding how infectious diseases spread and are managed. The assumptions, variables, and parameters are made clear in the model formulation. Additionally, models offer conceptual outcomes like thresholds, fundamental reproduction numbers, contact information, and alternative numbers. Better strategies for reducing the transmission of these diseases can result from an understanding of the transmission characteristics of infectious diseases in communities and health facilities.

One of the greatest public health disasters that has been a serious challenge to the global population for a long time is HIV/AIDS. AIDS was first recognized in the USA among homosexual males in 1981 (CDC,1982). In 1984, the virus was clearly demonstrated to be the causative agent for AIDS. The HIV/AIDS pandemic consists of many separate epidemics. Each epidemic has its own distinct origin, in terms of geography and specific populations affected, and involves different types and frequencies of risk behaviours and practices, for example, unprotected sex with multiple partners or sharing drug injection equipment (Dunne, 1995, UNAIDS, 2003).

The spread of the HIV virus can occur through several media, such as sexual contact with HIV-positive individuals, sharing needles, blood transfusions from HIV-positive individuals, and the vertical transmission process, which is mother-to-child HIV transmission (Ulfa, 2018). HIV/AIDS transmission in Africa is primarily through heterosexual sex and vertical transmission (MTCT); in Africa, 40% of all HIV/AIDS cases result from mother-to-child transmission (Adelman, 2001).

According to Ify (2004), sexual intercourse alone accounts for over 80% of reported cases of HIV infection. Vertical transmission of HIV/AIDS is also known as mother-to-child transmission (MTCT). MTCT is the transmission of HIV from an infected mother to her baby. The viruses may be transmitted to new-born babies during pregnancy (in utero), labour and delivery (through contamination by blood or other fluids during birth), and breastfeeding (Madubueze, 2015). MTCT is also referred to as "vertical transmission" or "perinatal transmission". It is the main cause of HIV infection in children.

Note that not all HIV-positive women will transmit the virus to their children. There are medicines that a woman living with HIV should take during pregnancy, labour, and delivery to protect her health and greatly reduce the chance that she will pass the virus to her baby. Medication is also given to the infant right after birth and in the first weeks of life (for 42 days). It is very important for all women to know their HIV status before they become pregnant, or very early in their pregnancy. HIV is also found in breast milk; HIV-positive women should not breastfeed their babies. In sub-Saharan Africa (SSA), the risk of transmission through this means ranges between 10 and 30%. The impact of vertical transmission of HIV/AIDS has been felt mainly in Africa (Chibaya, 2013), where the level of literacy is low, the poverty level is very high, and the quality of health services is generally very poor.

HIV infection, or AIDS, is a global pandemic, with cases reported from virtually every country. Globally, 34 million people were living with HIV at the end of 2011. More than 95% of people living with HIV/AIDS reside in low-income countries like Ethiopia (Tadesse, 2014). In Ethiopia, the first confirmed cases of HIV were detected in 1984, and the first hospitalized AIDS patients were reported in 1986, suggesting that the HIV epidemic probably began in the late 1970s or early 1980s (Kloos, _2001). Initially, the epidemic was localized in urban areas, along major commercial routes, and among certain occupational groups (Kloos, 2001). Antiretroviral therapy (ART) is an extremely expensive form of treatment, and not all nations have universal access to healthcare.

We intend to provide a mathematical model that characterizes and defines HIV/AIDS. Compartmental models significantly influenced the way epidemiological modelling in the population developed. In this study, we extend the model by Bashiru *et al*. (2017), a nonlinear mathematical model evaluated using the Susceptible - Infected - Treatment - AIDS patients (SITA) model of HIV/AIDS, in which population birth and death rates are not equal and the whole population is divided into four compartments. However, in our research, we expanded the model into Susceptible – Exposed - Infected - Treatment - AIDS patients (SEITA), which includes the exposed class (E).

2. THE MATHEMATICAL MODEL

Our initial model (Bashiru *et al*., 2017) is represented by four nonlinear ordinary differential equations. Our extended model is represented by five nonlinear ordinary differential equations by adding one more compartment based on the following basic assumptions: For this dynamical system, we considered Susceptible class S(t), exposed class E(t), infected (assumed infectious) class I(t), treatment class T(t), and AIDS patients class A(t). The susceptible individuals are those who are healthy but susceptible to diseases; exposed individuals are those who are infected but not yet infectious; infected individuals are those who are capable of transmitting the disease; individuals who are receiving treatment; and AIDS individuals are those who are seriously infected but not fully blown AIDS.

Model assumptions:

i. The population under this study is heterogeneous and varies with time.
ii. The susceptible become HIV-infected via sexual contact with infectives, which may also lead to the birth of infected children with a high contact rate β .
iii. The susceptible group increased with the new recruitment rate ψ .

iv. A fraction of new-borns are infected at birth and hence directly recruited into the infective class at a rate $\gamma\varepsilon$.

v. There is no direct recruitment of other infected individuals except through mother-to-child transmission.

vi. Some of the infectives move to join the treated class with rate σ_2 , while others with serious infections directly join the AIDS class with rate σ_1 .

vii. AIDS group: those who are seriously infected but not full-blown AIDS got treatment with rate v.

viii. The AIDS-related death is α .

ix. The average natural death rate of individuals in all classes is μ .

x. The exposed individuals belong to the infective class at a rate of θ .

xi. The proportion of the fast progression rate (p) of susceptible moves to join the infective class.

xii. The proportion of the slow progression rate $(1-p)$ of susceptible moves to join the exposed class.

Based on the above state variables and model assumptions, we develop the following flow chart of the dynamical system (Figure 1).

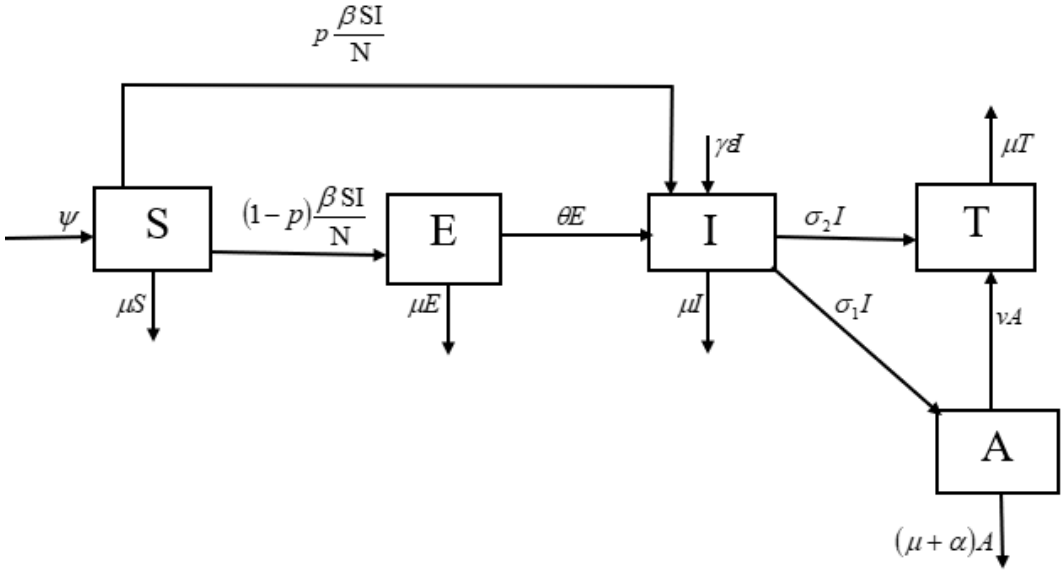


Figure 1. Schematic diagram of HIV/AIDS model

We consider the following system of five non-linear ordinary differential equations:

$$\frac{ds}{dt} = \psi - \frac{\beta \,\mathrm{SI}}{\mathrm{N}} - \mu\, S \qquad (1)$$

$$\frac{dE}{dt} = (1-p)\frac{\beta \mathrm{SI}}{\mathrm{N}} - \theta E - \mu E \qquad (2)$$

$$\frac{dI}{dt} = p\,\frac{\beta \mathrm{SI}}{\mathrm{N}} + \gamma\varepsilon I + \theta E - \sigma_1 I - \sigma_2 I - \mu I \qquad (3)$$

$$\frac{dT}{dt} = \sigma_2 I + \nu A - \mu T \qquad (4)$$

$$\frac{dA}{dt} = \sigma_1 I - \nu A - (\alpha + \mu) A \qquad (5)$$

with initial conditions: S(0)= $S_0 > 0$, E(0)= $E_0 \geq 0$,I(0)= $I_0 \geq 0$,T(0)= $T_0 \geq 0$,A(0)= $A_0 \geq 0$ and all parameters are positive.

3. **POSITIVITY OF THE SOLUTIONS**

For a dynamical system of a model to be epidemiologically meaningful, it is important to show that all the solutions with non-negative initial conditions will remain non-negative. To show this, we have taken each differential equation of the dynamical system (1) to (5) and shown that their solutions are non-negative as follows:

From (1), we have

$$\frac{ds}{dt} = \psi - \frac{\beta \,\mathrm{SI}}{\mathrm{N}} - \mu\, S$$

$$\frac{dS}{dt} + \left(\mu + \frac{\beta I}{N}\right) S = \psi \ .$$

This equation is first order linear differential equation, whose solution is

$$S(t) = \left[S_0 + \int_0^t \left(\psi e^{\mu t + \beta k(t) - \beta k(0)} \right) dt \right] e^{-(\mu t + \beta k(t) - \beta k(0))} > 0$$

Since any exponential equation with positive coefficient is always positive. Hence $S(t) > 0$

Similarly, we can show that $E(t) > 0, I(t) > 0,\ T(t) > 0$ and $A(t) > 0$ for all $t > 0$.

4. **BOUNDEDNESS OF THE SOLUTIONS**

To show the solution of the system bounded, let us summing up all the five equations. That is

$N(t) = S(t) + E(t) + I(t) + T(t) + A(t)$. At any time and differentiating $N(t)$ with respect to time and substitute the corresponding values from the dynamical system (1) to (5), we obtain

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dT}{dt} + \frac{dA}{dt} = \psi - \mu N + \gamma\varepsilon - \alpha A$$

Assuming that $\gamma\varepsilon < \alpha A$ the inequality, we obtain

$$\frac{dN}{dt} = \psi - \mu N + \gamma\varepsilon - \alpha A < \psi - \mu N$$

$$\frac{dN}{dt} < \psi - \mu N$$

After evaluating we obtain

$$N(t) \le \left(N_0 - \frac{\psi}{\mu} \right) e^{-\mu t} + \frac{\psi}{\mu}$$

As $t \to \infty, N(t) \le \frac{\psi}{\mu}$

$N(t) \le \frac{\psi}{\mu}$ .If and only if $N(t) \le \frac{\psi}{\mu}$ and $N(t) > 0$

Hence $0 < N(t) \le \frac{\psi}{\mu}$.

Clearly all the solutions of the dynamical system are bounded in the interval $\left(0, \frac{\psi}{\mu} \right]$

5. THE DISEASE-FREE EQUILIBRIUM POINT

To find the equilibrium point of the dynamical system setting equations (1) to (5) equals to zero. This means, $\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dT}{dt} = \frac{dA}{dt} = 0$. Furthermore, no one in the population is contagious at the disease-free equilibrium point, that is $E = I = T = A = 0$. Therefore, the disease-free equilibrium point is $\left(S^0, E^0, I^0, T^0, A^0 \right) = \left(\frac{\psi}{\mu}, 0, 0, 0, 0 \right)$.

6. THE ENDEMIC EQUILIBRIUM POINT

To find the endemic equilibrium point of the dynamical system, it is obtained by setting the right-hand side of the system (1) to (5) equal to zero, so that

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

The endemic equilibrium point of the dynamical system is $\left(S^*,E^*,I^*,T^*,A^*\right)$

Where,

$$S^*=\frac{\psi N}{\beta I^*+\mu N}$$

$$E^*=(1-p)\frac{\beta\psi I^*}{(\theta+\mu)\left(\beta I^*+\mu N\right)}$$

$$I^*=\frac{\beta\psi(\theta+p\mu)}{\beta(\theta+\mu)(\sigma_1+\sigma_2+\mu-\gamma\varepsilon)}-\frac{\mu N}{\beta}=\frac{\mu N}{\beta}(R_0-1)$$

$$T^*=\frac{1}{\mu}\left(\sigma_2+\frac{\sigma_1\nu}{\nu+\mu+\alpha}\right)I^*$$

$$A^*=\frac{\sigma_1 I^*}{\nu+\mu+\alpha}$$

7. **REPRODUCTION NUMBER**

To find the reproduction number, we will use the method of the next-generation matrix (Van den Driessche, 2002), which is defined as the spectral radius (or dominant eigenvalue) of the model. The first step is rewriting the model equations, starting with the newly infected classes:

$$\frac{dE}{dt}=(1-p)\frac{\beta SI}{N}-(\theta+\mu)E$$

$$\frac{dI}{dt}=p\frac{\beta SI}{N}+\theta E-(\sigma_1+\sigma_2+\mu-\gamma\varepsilon)I\ .$$

Then system can be written as

$$\frac{dx}{dt}=f-v$$

Here the new infection matrix f and the transmission matrix v are defined by

$$f\left(E,I,A,T,S\right)=\begin{pmatrix}\left(1-p\right)\dfrac{\beta SI}{N}\\ p\dfrac{\beta SI}{N}\end{pmatrix} \quad \text{And } v=\begin{pmatrix}\left(\theta+\mu\right)E\\ \left(\sigma_1+\sigma_2+\mu-\gamma\varepsilon\right)I-\theta E\end{pmatrix}.$$

Then, by the principle of the next-generation matrix, the Jacobian matrices at each point are given by

$$F\left(E,I,A,T,S\right)=\begin{bmatrix}0 & \left(1-p\right)\dfrac{\beta S}{N}\\ 0 & p\dfrac{\beta S}{N}\end{bmatrix},\ V=\begin{bmatrix}\theta+\mu & 0\\ -\theta & \sigma_1+\sigma_2+\mu-\gamma\varepsilon\end{bmatrix}$$

The Jacobian matrices at the DFE point is given by

$$F=\begin{bmatrix}0 & \left(1-p\right)\dfrac{\beta\psi}{\mu N}\\ 0 & p\dfrac{\beta\psi}{\mu N}\end{bmatrix},V=\begin{bmatrix}\theta+\mu & 0\\ -\theta & \sigma_1+\sigma_2+\mu-\gamma\varepsilon\end{bmatrix}\text{and}$$

$$V^{-1}=\begin{bmatrix}\dfrac{1}{\theta+\mu} & 0\\ \dfrac{\theta}{\left(\theta+\mu\right)\left(\sigma_1+\sigma_2+\mu-\gamma\varepsilon\right)} & \dfrac{1}{\sigma_1+\sigma_2+\mu-\gamma\varepsilon}\end{bmatrix}$$

$$\text{Then } FV^{-1}=\begin{bmatrix}\dfrac{\left(1-p\right)\beta\psi\theta}{\mu N\left(\theta+\mu\right)\left(\sigma_1+\sigma_2+\mu-\gamma\varepsilon\right)} & \dfrac{\left(1-p\right)\beta\psi}{\mu N\left(\sigma_1+\sigma_2+\mu-\gamma\varepsilon\right)}\\ \dfrac{p\beta\psi\theta}{\mu N\left(\theta+\mu\right)\left(\sigma_1+\sigma_2+\mu-\gamma\varepsilon\right)} & \dfrac{p\beta\psi}{\mu N\left(\sigma_1+\sigma_2+\mu-\gamma\varepsilon\right)}\end{bmatrix}$$

Therefore, FV^{-1} is the next generation matrix of the model, then the dominant eigenvalue of FV^{-1} represents $R_0=\left(FV^{-1}\right)$, which is $R_0=\dfrac{\beta\psi\left(\theta+\mu p\right)}{\mu N\left(\theta+\mu\right)\left(\sigma_1+\sigma_2+\mu-\gamma\varepsilon\right)}$.

8. STABILITY ANALYSIS OF DISEASE-FREE EQUILIBRIUM POINT

The Jacobian matrix at the disease-free equilibrium point $\left(S^0,E^0,I^0,T^0,A^0\right)=\left(\dfrac{\psi}{\mu},0,0,0,0\right)$

$$J\left(\frac{\psi}{\mu},0,0,0,0\right)=\begin{bmatrix} -\mu & 0 & \frac{-\beta\psi}{\mu N} & 0 & 0 \\ 0 & -(\theta+\mu) & \frac{(1-p)\beta\psi}{\mu N} & 0 & 0 \\ 0 & \theta & \frac{p\beta\psi}{\mu N}-(\sigma_1+\sigma_1+\mu-\gamma\varepsilon) & 0 & 0 \\ 0 & 0 & \sigma_2 & -\mu & \nu \\ 0 & 0 & \sigma_2 & 0 & -(\nu+\mu+\alpha) \end{bmatrix}$$

The corresponding Characteristic polynomial is

$$\begin{vmatrix} -\mu-\lambda & 0 & \frac{-\beta\psi}{\mu N} & 0 & 0 \\ 0 & -(\theta+\mu)-\lambda & \frac{(1-p)\beta\psi}{\mu N} & 0 & 0 \\ 0 & \theta & \frac{p\beta\psi}{\mu N}-(\sigma_1+\sigma_1+\mu-\gamma\varepsilon)-\lambda & 0 & 0 \\ 0 & 0 & \sigma_2 & -\mu-\lambda & \nu \\ 0 & 0 & \sigma_1 & 0 & -(\nu+\mu+\alpha)-\lambda \end{vmatrix}=0$$

$$\Rightarrow\begin{vmatrix} -\mu-\lambda & 0 & x & 0 & 0 \\ 0 & y-\lambda & b & 0 & 0 \\ 0 & \theta & q-\lambda & 0 & 0 \\ 0 & 0 & \sigma_2 & -\mu-\lambda & \nu \\ 0 & 0 & \sigma_1 & 0 & m-\lambda \end{vmatrix}=0$$

Where,

$$x=\frac{-\beta\psi}{\mu N}, y=-(\theta+\mu), b=\frac{(1-p)\beta\psi}{\mu N}, q=\frac{p\beta\psi}{\mu N}-(\sigma_1+\sigma_2+\mu-\gamma\varepsilon) and\, m=-(\nu+\mu+\alpha)$$

The Jacobian evaluated at the DFE point has five eigenvalues, three of which are $\lambda_1=-\mu$, $\lambda_2=-\mu$ and $\lambda_3=-(\nu+\mu+\alpha)$ which are negative. The remaining two eigenvalues are the roots of the polynomial equation $a_2\lambda^2+a_1\lambda+a_0=0$, Where $a_2=1$, $a_1=-(y+q)$ and $a_0=yq-b\theta$.

It is easy to show that the eigenvalues are both negative (or have negative real parts). By Routh-Hurwitz stability criteria for polynomials of degree two, the eigenvalues are all negative if, $a_2>0$, $a_1>0$ and $a_0=yq-b\theta>0$.

$$a_0=yq-b\theta>0 \Rightarrow -(\theta+\mu)\left[\frac{p\beta\psi}{\mu N}-(\sigma_1+\sigma_2+\mu-\gamma\varepsilon)\right]-\frac{(1-p)\beta\psi}{\mu N}\theta>0$$

$$\Rightarrow (\theta+\mu)(\sigma_1+\sigma_2+\mu-\gamma\varepsilon) > \frac{\beta\psi(\mu\rho+\theta)}{\mu N}$$

Both sides divided by $(\theta+\mu)(\sigma_1+\sigma_2+\mu-\gamma\varepsilon)$, we obtain

$$\frac{\beta\psi(\mu\rho+\theta)}{\mu N(\theta+\mu)(\sigma_1+\sigma_2+\mu-\gamma\varepsilon)} < 1 \Rightarrow R_0 < 1.$$

Based on the above description, if $R_0 < 1$, then all eigenvalues are negative. It indicates that the disease-free equilibrium point is stable; otherwise, it is unstable.

9. STABILITY ANALYSIS OF ENDEMIC EQUILIBRIUM POINT

In this section, we discussed the stability analysis of the endemic equilibrium point. That is

$$\left(S^*, E^*, I^*, T^*, A^*\right) = \begin{pmatrix} \dfrac{\psi N(\theta+\mu)(\sigma_1+\sigma_2+\mu-\gamma\varepsilon)}{p\beta\psi(\theta+\mu)+(1-p)\theta\beta\psi}, \\ \dfrac{(1-p)\psi\left[p\beta\psi(\theta+\mu)+(1-p)\beta\psi\theta-\mu N(\theta+\mu)(\sigma_1+\sigma_2+\mu-\gamma\varepsilon)\right]}{(\theta+\mu)\left[p\beta\psi(\theta+\mu)+(1-p)\beta\psi\theta\right]}, \\ \dfrac{p\beta\psi(\theta+\mu)+(1-p)\beta\psi\theta}{\beta(\theta+\mu)(\sigma_1+\sigma_2+\mu-\gamma\varepsilon)}-\dfrac{\mu N}{\beta}, \\ \dfrac{1}{\mu}\left(\sigma_2+\dfrac{\sigma_1\nu}{\nu+\mu+\alpha}\right)\left(\dfrac{p\beta\psi(\theta+\mu)+(1-p)\beta\psi\theta}{\beta(\theta+\mu)(\sigma_1+\sigma_2+\mu-\gamma\varepsilon)}-\dfrac{\mu N}{\beta}\right), \\ \dfrac{\sigma_1}{\nu+\mu+\alpha}\left(\dfrac{p\beta\psi(\theta+\mu)+(1-p)\beta\psi\theta}{\beta(\theta+\mu)(\sigma_1+\sigma_2+\mu-\gamma\varepsilon)}-\dfrac{\mu N}{\beta}\right) \end{pmatrix}$$

The Jacobian matrix of the dynamical system of the endemic equilibrium point is

$$J\left(S^*, E^*, I^*, T^*, A^*\right) = \begin{bmatrix} -\left(\dfrac{\beta I^*}{N}+\mu\right) & 0 & \dfrac{-\beta S^*}{N} & 0 & 0 \\ \dfrac{(1-p)\beta I^*}{N} & -(\theta+\mu) & \dfrac{(1-p)\beta S^*}{N} & 0 & 0 \\ \dfrac{p\beta I^*}{N} & \theta & \dfrac{p\beta S^*}{N}-(\sigma_1+\sigma_2+\mu-\gamma\varepsilon) & 0 & 0 \\ 0 & 0 & \sigma_2 & -\mu & \nu \\ 0 & 0 & \sigma_1 & 0 & -(\nu+\mu+\alpha) \end{bmatrix}$$

To find the Eigen values of the Jacobian matrix, we make $\det(J-\lambda I_n)=0$, where I_n 5x5 identity matrix. This means

$$\begin{vmatrix} x-\lambda & 0 & y & 0 & 0 \\ k & q-\lambda & l & 0 & 0 \\ m & \theta & r-\lambda & 0 & 0 \\ 0 & 0 & \sigma_2 & -\mu-\lambda & \nu \\ 0 & 0 & \sigma_1 & 0 & z-\lambda \end{vmatrix} = 0$$

Where

$$x = -\left(\frac{\beta I^*}{N} + \mu\right), y = -\left(\frac{\beta S^*}{N}\right), k = \frac{(1-p)\beta I^*}{N}, m = \frac{p\beta I^*}{N}, l = \frac{(1-p)\beta S^*}{N}, r = \frac{p\beta S^*}{N} - (\sigma_1 + \sigma_2 + \mu - \gamma\varepsilon)$$

$$q = -(\theta + \mu), z = -(\nu + \mu + \alpha)$$

The Jacobian evaluated at the EE point has five eigenvalues, two of which are $\lambda_1 = -\mu$ and $\lambda_2 = -(\nu + \alpha + \mu)$ which are negative. The remaining three eigenvalues are the roots of the polynomial equation $a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0$. Where,

$$a_3 = 1$$

$$a_2 = 3\mu + \sigma_1 + \sigma_2 + \theta - \gamma\varepsilon - \frac{p\beta S^*}{N} + \frac{\beta I^*}{N}$$

$$a_1 = (rx + qx + rq - l\theta - my)$$

$$a_0 = -qrx - yk\theta + l\theta x + my$$

Following Routh-Hurwitz stability criteria for polynomial of degree six, the eigenvalues are all negative if $a_0 > 0$, $a_2 > 0$ and $a_2a_1 - a_0$.

$a_3 = 1 > 0$ is true, the endemic equilibrium of the model is locally asymptotically stable if

$a_0 > 0, a_2 > 0$ and the inequalities $a_1 > \frac{a_0}{a_2}$ are true.

10. **GLOBAL STABILITY AT DISEASE FREE EQUILIBRIUM**

To prove the global stability, we make use of Castillo-Chavez method (Castillo-Chavez, 2004). Consider a model of the form

$$\begin{cases} \dfrac{dF}{dt} = F(X,T) \\ \dfrac{dG}{dt} = G(X,T), G(X,0) = 0 \end{cases} \quad *$$

Where $X \in \Re^m$ represents, individuals that are not infected in the population and $T \in \Re^m$ represents infected individuals. Following the above representation, the disease-free equilibrium state can be written as $Y_0 = (X^*, 0)$, the two conditions given below are used to verify the disease-free equilibrium is globally asymptotically stable.

(A_1). For $\frac{dF}{dt} = F(X,0)$ is globally asymptotically stable.

$(A_2).= G(X,T) = BT - G(X,T), G(X,T) \geq 0$ *for all* $(X,T) \in \Omega$

Where $B = D_T G(X^*,0)$ is an M-matrix (the off-diagonal elements of B are non-negative) and Ω is the region where the model makes biological sense.

Lemma 1: The fixed point $Y_0 = (X^*,0)$ is globally asymptotically stable equilibrium of $(*)$ provided that $R_0 \leq 1$ and assumption that (A_1) and (A_2) are satisfied.

Theorem 1: The disease-free equilibrium point of the model is globally asymptotically stable.

Proof: The model equation (1)-(5) is re-written as in form of (*) by setting $X = (S)$ and $T = (E, I, T, A)$. The disease-free equilibrium is given by

$U_0(X^*,0) = \left(\frac{\psi}{\mu}, 0, 0, 0, 0\right)$. And the system $\frac{dY}{dt} = F(X,0)$ becomes

$$\frac{ds}{dt} = \psi - \frac{\beta \mathrm{SI}}{\mathrm{N}} - \mu S$$

This equation has a unique equilibrium point, $Y_0 = \left(\frac{\psi}{\mu}\right)$. Which is asymptotically stable, therefore, the condition (A_1) is satisfied.

$$\text{For } A_2 : G(X,T) = \begin{pmatrix} (1-p)\frac{\beta \mathrm{SI}}{\mathrm{N}} - \theta E - \mu E \\ p\frac{\beta \mathrm{SI}}{\mathrm{N}} + \gamma \varepsilon I + \theta E - \sigma_1 I - \sigma_2 I - \mu I \\ \sigma_2 I + \nu A - \mu T \\ \sigma_1 I - \nu A - (\alpha + \mu) A \end{pmatrix}$$

$$D_T G(X^*,0) = \begin{pmatrix} -(\theta+\mu) & (1-p)\frac{\beta S}{N} & 0 & 0 \\ \theta & p\frac{\beta S}{N} + \gamma\varepsilon - (\sigma_1 + \sigma_2 + \mu) & 0 & 0 \\ 0 & \sigma_2 & -\mu & \nu \\ 0 & \sigma_1 & 0 & -(\nu + \alpha + \mu) \end{pmatrix}$$

Clearly, $B = D_T G(X^*,0)$ is an M-matrix? On the other hand, $G(X,T) = BT - G(X,T)$ this implies

$$G(X,T) = BT - G(X,T) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

$G(X,T) = (X,T) \geq 0$ *for all* $(X,T) \in \Omega$, therefore, conditions (A_1) and (A_2) are satisfied. With theorem 1, the global stability of DFE is obtained and which completes the proof.

11. GLOBAL STABILITY OF ENDEMIC EQUILIBRIUM POINT

Theorem 2: $R_0 < 1$, the endemic equilibrium point of the model is globally asymptotically stable.

Proof: To establish the global stability of the endemic equilibrium of the model, we construct the following by Lyapunov function.

$$L(S^*, E^*, I^*, T^*, A^*) = \left(S - S^* - S^* \ln \frac{S}{S^*}\right) + \left(E - E^* - E^* \ln \frac{E}{E^*}\right) + \left(I - I^* - I^* \ln \frac{I}{I^*}\right)$$
$$+ \left(T - T^* - T^* \ln \frac{T}{T^*}\right) + \left(A - A^* - A^* \ln \frac{A}{A^*}\right)$$

If we take the derivative of L we obtain,

$$\frac{dL}{dt} = \left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} + \left(1 - \frac{E^*}{E}\right)\frac{dE}{dt} + \left(1 - \frac{I^*}{I}\right)\frac{dI}{dt} + \left(1 - \frac{T^*}{T}\right)\frac{dT}{dt} + \left(1 - \frac{A^*}{A}\right)\frac{dA}{dt}$$

Substituting each, we get

$$\frac{dL}{dt} = \left(1 - \frac{S^*}{S}\right)\left(\psi = \frac{\beta SI}{N} - \mu S\right) + \left(1 - \frac{E^*}{E}\right)\left(\frac{(1-p)\beta SI}{N} - (\theta + \mu)E\right)$$
$$+ \left(1 - \frac{I^*}{I}\right)\left(\frac{p\beta SI}{N} + \gamma\varepsilon I + \theta E - (\sigma_1 + \sigma_2 + \mu)I\right)$$
$$+ \left(1 - \frac{T^*}{T}\right)(\sigma_2 I + vA - \mu T) + \left(1 - \frac{A^*}{A}\right)(\sigma_1 I - vA - (\alpha + \mu)A)$$

Expanding the above, we obtain:

$$\Rightarrow \frac{dL}{dt}=\left(1-\frac{S^*}{S}\right)\left(\psi-\frac{\beta\left(S-S^*\right)\left(I-I^*\right)}{N}-\mu\left(S-S^*\right)\right)$$

$$+\left(1-\frac{E^*}{E}\right)\left(\frac{(1-p)\beta\left(S-S^*\right)\left(I-I^*\right)}{N}-(\theta+\mu)\left(E-E^*\right)\right)$$

$$+\left(1-\frac{I^*}{I}\right)\left(\frac{p\beta\left(S-S^*\right)\left(I-I^*\right)}{N}+\gamma\varepsilon\left(I-I^*\right)+\theta\left(E-E^*\right)-\left(\sigma_1+\sigma_2+\mu\right)\left(I-I^*\right)\right)$$

$$+\left(1-\frac{T^*}{T}\right)\left(\sigma_2\left(I-I^*\right)+\nu\left(A-A^*\right)-\mu\left(T-T^*\right)\right)$$

$$+\left(1-\frac{A^*}{A}\right)\left(\sigma_1\left(I-I^*\right)-(\nu+\alpha+\mu)\left(A-A^*\right)\right)$$

Collecting the positive and negative terms we obtain

$$\frac{dL}{dt}=K-L, where$$

$$K=\left(1-\frac{S^*}{S}\right)\psi+\left(1-\frac{E^*}{E}\right)\left(\frac{(1-p)\beta\left(S-S^*\right)\left(I-I^*\right)}{N}\right)+\frac{\left(I-I^*\right)^2}{I}\left(\frac{p\beta\left(S-S^*\right)}{N}\right)$$

$$+\theta\left(E-E^*\right)\left(1-\frac{I^*}{I}\right)+\left(1-\frac{T^*}{T}\right)\left(\sigma_2\left(I-I^*\right)+\nu\left(A-A^*\right)\right)+\left(1-\frac{A^*}{A}\right)\left(\sigma_1\left(I-I^*\right)\right)$$

And

$$M=\frac{\left(S-S^*\right)^2}{S}\left(\frac{\beta\left(I-I^*\right)}{N}+\mu\right)+(\theta+\mu)\frac{\left(E-E^*\right)^2}{E}$$

$$+\frac{\left(I-I^*\right)^2}{I}\left(\sigma_1+\sigma_2+\mu-\gamma\varepsilon\right)+\mu\frac{\left(T-T^*\right)^2}{T}$$

$$+(\nu+\alpha+\mu)\frac{\left(A-A^*\right)^2}{T}$$

If $K<M$, then $\frac{dL}{dt}$ will be negative and $\frac{dL}{dt}=0$, if and only if $S=S^*, E=E^*, I=I^*, T=T^*$ *and* $A=A^*$.Thus, the maximum compact invariant set is $\left\{\left\{\left(S^*,E^*,I^*,T^*,A^*\right)\in\Omega:\frac{dL}{dt}=0\right\}\right.$.Which is the singleton set $\left\{X^*\right\}$, hence the endemic equilibrium, by LaSalle's Invariant principles; it implies that X^* is globally asymptotically stable (GAS) in Ω if $K<M$.

12. **SENSITIVITY ANALYSIS**

It is vital to understand the relative relevance of the various elements involved in HIV/AIDS transmission in order to decide how to reduce human mortality and morbidity due to the disease. The purpose of sensitivity analysis is to identify the parameters that have a significant influence on the reproduction number and to assess the resilience of model predictions to parameter values. These are the definitions of the normalized forward sensitivity indices of R_0 that rely differentiable on a parameter m and defined by $H_m^{R_0} = \frac{m}{R_0}\frac{\partial R_0}{\partial m}$, we take $m = \beta, \psi, \theta, \delta_1, \sigma_2, \varepsilon, \gamma$ and μ. The parameter values given in Table 2 below are used to evaluate the sensitivity indices of some parameters that are responsible for the transmission dynamics of HIV/AIDS infectious disease.

Table 1. Sensitivity indices of the basic reproduction number to model parameters.

Parameter Symbol	Sensitivity Index
μ	-0.7248
β	1
ψ	1
ε	0.0256
γ	0.0256
θ	0.9525
σ_2	-0.002
δ_1	-0.002

Each parameter value has a different impact on the sensitivity index in Table 1, which contains positive and negative values. A positive value means that the basic reproduction number will grow if the parameter value is increased while the other parameters are held constant, and will decrease if the parameter value is decreased while the other parameters are held constant. In contrast, a negative value means that increasing the parameter value while maintaining the other parameters will cause the basic reproduction number to decrease, and decreasing the parameter value while maintaining the other parameters will cause the basic reproduction number to increase. The new birth rate in the susceptible human population, the transmission coefficient from susceptible to infected, and the natural death rate are the parameters that are most sensitive to changes in value R_0 , according to sensitivity analysis.

13. **NUMERICAL SIMULATION**

In this part, we perform numerical simulations of the models (1) to (5) are carried out using the $MATLAB$ software to illustrate the results and to identifying the control parameters. From

the SEITA mathematical model with a system of five –variable differential equations (S, E, I, T, A) with parameters that affect the system. The parameters values adopted from Table 2 and the initial population levels were assumed as follows: $S(0) = 15000$, $E(0) = 1500, I(0) = 1000$, T(0)=2500 and $A(0) = 200$.

Table 2. Parameter values and descriptions.

Parameter	Parameter description	Values	Reference
β	The rate of contact between susceptible and infectives	0.5	Bashiru *et al*. (2017)
θ	Rate of exposed to infectives	0.02	Assumed
σ_1	Rate of infectives to AIDS patient	0.04	Bashiru *et al*. (2017)
σ_2	Rate of infectives to treatment	0.6	Bashiru *et al*. (2017)
α	AIDS related death rate	0.02	Bashiru *et al*. (2017)
μ	Natural death rate	0.001	Assumed
ν	Rate of AIDS to treatment	0.035	Assumed
γ	Vertical transmission rate	0.8	Assumed
ε	Fraction of infected new born rate	0.02	Bashiru *et al*. (2017)
p	Fast progression rate	0.6	Assumed
$1-p$	Slow progression rate	0.4	Assumed
ψ	New recruitment rate	0.12	Assumed

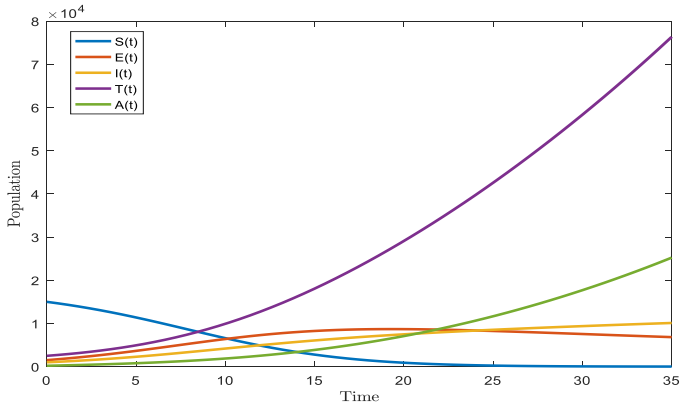


Figure 2. Transmission of HIV/AIDS with Treatment

From Figure 2, we observe that with time, the susceptible population decreases, the exposed and infected population increases, and for a long time, they decrease while the treated and HIV/AIDS patients increase.

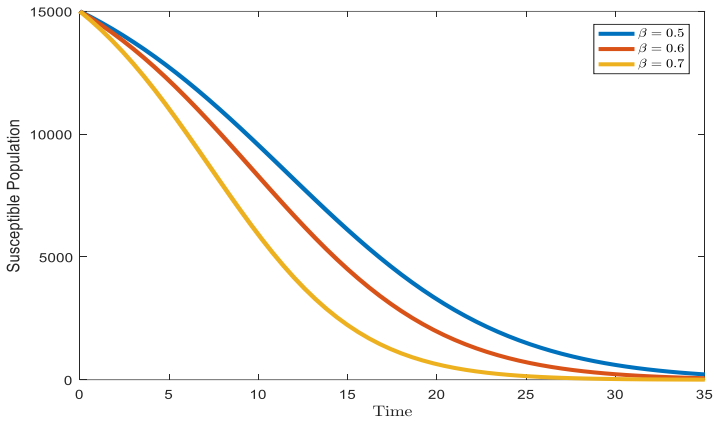


Figure 3. Effect of contact rate on susceptible population

According to figure 3, which depicts how rates affect the susceptible population, the vulnerable population declines as the rate of contact rises, as would be expected.

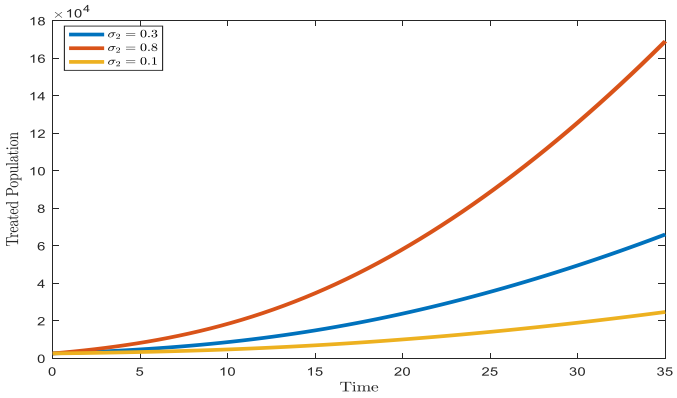


Figure 4: Effect of Treatment rate

From figure 4**,** it shows the effect of treatment rate on the treated population. We see that as the rate increases, treated population also increases as assumed.

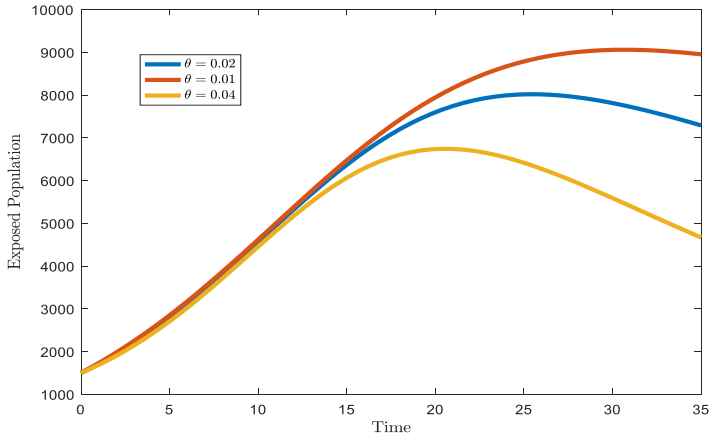


Figure 5: Effect of exposed rate

Figure 5, shows the effect of exposure rate on the exposed population. We observe that as the rate decreases, the exposed population increases, and for a long time, the population will decrease.

CONCLUSIONS

In this study, a deterministic model is used to illustrate and investigate the dynamics of HIV/AIDS. It was decided whether endemic and disease-free equilibrium stability existed. The next-generation matrix method was used to get the fundamental reproduction number of the model, which predicts that the disease will not persist since the disease-free equilibrium is stable when less than unity. The vulnerable population reduces as the transmission coefficient rises, as would be expected, and the population is also impacted by the treatment rate. As expected, the population grows as the rate rises. However, it is essential to continue efforts to prevent the spread of HIV/AIDS and provide appropriate healthcare to those affected. So, increase awareness about the modes of transmission and preventive measures through public health campaigns and educational programs. Provide psychosocial support, counselling, and information services to individuals affected by HIV/AIDS, their families, and communities.

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