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Mathematical Modeling on Assessing the Impact of Screening on HIV/AIDS Transmission Dynamics

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ABSTRACT: Human Immunodeficiency Virus /Acquired Immune Deficiency Syndrome is a globally prevalent and deadly sexually transmitted disease that has had a profound impact on human history, causing widespread fatalities and devastating economic consequences. In this model, we presents a four compartmental class of susceptible S(t), symptomatic infective I(t), asymptomatic infective I(t), and full blown AIDS class model for the transmission dynamics of HIV/AIDS in which we considered the significant role that screening played among those who are symptomatic and asymptomatic infective for the disease control and its management. We determined the positivity and boundedness of the model and the existence of its unique solution which showed clearly that the model is epidemiological meaningful and well posed. The disease-free and endemic equilibrium states were identified, and their stability is analyzed which reveals that if $R_0 < 1$ the disease free equilibrium is locally asymptotically stable and unstable if otherwise. Sensitivity analysis was also carried out using normalized forward sensitivity index and result showed that the recruitment rate (π), and transmission rate (β), is the most sensitive parameter. However, it is observed from the numerical simulation that the importance of screening is evident in its ability to detect and reduce the number of asymptomatic infective individuals, which in turn leads to an increase among the symptomatic population highlighting the importance of early detection of their status and preventing the spread of HIV/AIDS. The susceptible should also exercise caution to avoid interactions with those who are infectious, further reducing the risk of transmission.

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HIV/AIDS is a sexually transmitted disease worldwide. It was first identified in 1981 among the heterosexual men and intravenous drug users in New York and California (Sharp and Beatrice, 2011). Evidence of AIDS epidemics later grew among heterosexual men, women, and children in the Saharan Africa. In 2022, statistics reveals that approximately 39 Million people died from AIDS worldwide, about 740 children became infected with HIV and approximately 274 children died from AIDS related causes every day (Global and Regional Trend (2023)). Research has indicated that the risk of contracting the

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infection rises in correlation with the amount of sexual partners and engaging in anal intercourse (Gaodart *et al*., 1994). AIDS is classified based on clinical symptoms, viral load, and $CD4^+T$ cell count, with 2-6 stages of infection identified before the development of AIDS (Stodart and Reyes, 2006). The health of an HIV positive person will depend on the health of his or her immune system as well as on the exposure to diseases in the environment. An individual may advance through several infective stages before developing full blown AIDS WHO (2007).Once the virus enters the body, the main target is to destroy the white blood cell called $CD4^+T$ cells. The virus steadily destroys $CD4^+T$ cells over a period of years, diminishing the cells' protective ability and weakening the immune system. As the immune system becomes compromised, the HIV opportunistic diseases such as meningitis, Cancers, kidney and Tuberculosis do easily attack the body (Lamptey *et al*., 2003).HIV attacks and destroys a significant portion of $CD4^+T$ cells which are a type of immune cells. This leads to a decline in the number of $CD4^+T$ cells and weakens the immune system's ability to fight off infections and diseases (HIV/AIDS facts, 2004;Odebiyi and Ayeni, 2012).Screening refers to the process of evaluating or testing individuals or things to identify specific characteristics, qualities, or potential issues. It aims to detect, filter out, or categorize things based on predetermined criteria. There is a great development because Routine screening of unaware infectious individuals has become a crucial component of healthcare programs in low and middle-income countries. This proactive approach helps identify undiagnosed cases, prevent further transmission, provide timely treatment and care, and reduce the burden of diseases on individuals and communities. Early detection and intervention can significantly improve health outcomes and save lives. Antiretroviral medication has enabled many people who were severely ill with HIV and AIDS to recover and regain their health, achieving a significant reduction in viral load, often to undetectable levels, and restoring their ability to lead normal lives (UNAIDS 2002).Some researchers have worked on HIV/AIDS with Screening. (Issa and Massawe 2011) examined the effect of screening and variable inflow of infected immigrants on the spread of HIV/AIDS showed that sensitivity of the key parameter revealed an increase in the screening rate coupled with decrease in the progression of infective to AIDS class thereby leading to a decline in the spread of HIV/AIDS. (Odebiyi, 2024) also investigated the stability analysis of HIV/AIDS model with saturated incidence. The outcome of their results showed that screening and treatment of the infective have a significant effect in reducing the transmission of the disease control. (Al-Sheikh *et al*., 2011) worked on stability analysis of HIV/AIDS epidemic with screening. According to the research findings, it was suggested that the most efficient approach to reducing the infection rates is to consistently screen high-risk individuals and educate the general population about the infection. (Marsudi, 2014)Worked on sensitivity analysis considering the effect of screening and HIV therapy on the dynamics of the spread of HIV and gave the assertion that screening of unaware infective and placing the screened infective on therapy has a reducing effect on transmission of the disease. Also, (Srinivasa, 2023) presented a theoretical framework for transmission of HIV/AIDS epidemic in India. It was observed that screening of the infective has a significant effect on the spread of the disease. The significance of counselling and treatment in preventing the spread of HIV/AIDS was emphasized by (Ibrahim *et al*., 2021). It was revealed that providing guidance to individuals with the virus and administering anti-retroviral medications can effectively manage or eradicate HIV. They also emphasized that those strategies could avert 9-12million new cases over a period of 40years. (Ratera *et al*., 2012) examined the effect of screening and treatment on the transmission of HIV/AIDS infection in a population. Their model exhibit forward bifurcation at threshold parameter equal to unity and analysis shows that screening of unaware HIV infective and treatment of screened infective have the effect of reducing the transmission of the disease. Therefore, the objective of this paper is to investigate the impact of screening on the dynamics of HIV/AIDS model and the study is organized as follows. The formulation, the positivity and boundedness of the model and proof of the existence and uniqueness of solutions is obtained in section 2. In section 3, Analysis of the model was shown including the sensitivity indices of parameters of the basic reproduction number. Section 4 presents a numerical simulation of the model systems followed by a conclusion.

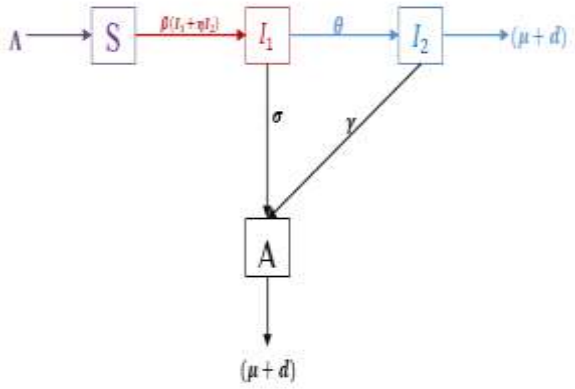

Fig 1: The model Schematic diagram of an SI model

*Formulation of the Model and its Description***:** In this research, the susceptible and infectious epidemic model (SI) is considered. A population size of $N(t)$ was partitioned into 4 subclasses of individuals who were susceptible, asymptomatic infective, symptomatic infective, and full blown AIDS population with sizes denoted by $S(t), I_1(t), I_2(t),$ and $A(t)$, respectively, as shown in figure 1.

Model description: In this section, we considered the HIV/AIDS transmission model with susceptible-infected model of the human population where the total human population at time (t) is divided in to 4 disjoint compartments. The susceptible population $S(t)$ are the number of individuals in a population who are at risk of becoming infected by a disease, and have no immunity (natural or acquired).

The asymptomatic population (I_1) are individual who are infected by a disease, but show no visible symptoms or signs of illness, yet may still transmit the disease to others. The Symptomatic population (I_2) are individuals who are infected by a disease and exhibit visible symptoms or signs of illness. AIDS population is the most advanced stage of HIV. The population includes individuals who have been characterized by a severely compromised immune system and numerous opportunistic infections. In a normal healthy individual's peripheral blood, the level of $CD4^+T$ cells is between 800 and 1200/mm^3 and once this number reaches 200 or below in an HIV infected patient, the person is classified as having AIDS (Stodart, 2006).

The infected person becomes vulnerable to AIDS-related opportunistic infections and rare cancers, which take advantage of the weakened immune defences to cause disease. As a result, total human population is given by $N(t) = S(t) + I_1(t) + I_2(t) + A(t).$ The recruitment rate of susceptible (π), occurs through either the flow of people by birth or immigration. Asymptomatic infective and Symptomatic infective infect Susceptible class at different rates $\beta,$ and $\eta\beta$ respectively. It is assumed that $\eta\beta > \beta$, they transmit at higher rate of infectivity, where $0 < \eta < 1.$ Asymptomatic infective population can be screened at a rate θ and progress to symptomatic class. Also, asymptomatic infective population and Symptomatic infective population move to full blown AIDS at different rates σ and γ respectively. Taking the above into considerations, the model dynamics is described by a set of nonlinear ordinary differential equations (ODEs), which capture the complex dynamics of the system.

Model Equation:

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \beta(I_1 + \eta I_2)S - \mu S \\
\frac{dI_1}{dt} &= \beta(I_1 + \eta I_2)S - (\theta + \mu + \sigma)I_1 \\
\frac{dI_2}{dt} &= \theta I_1 - (\mu + \gamma + d)I_2 \\
\frac{dA}{dt} &= \sigma I_1 + \gamma I_2 - (\mu + d)A
\end{aligned}\right\} \quad (1)$$

$$S(0) = S_0, I_1(0) = I_{1_0}, I_2(0) = I_{2_0}, A(0) = A_0 \quad (2)$$

The model parameters used in the model is defined as follows:

Table 1: Model variables and parameters

Parameters/ variables	Description
π	Recruitment rate of Susceptible
β	Transmission rate
d	AIDS related death rate
θ	Pace at which asymptomatic population become aware of being infected after a screening process.
μ	Natural mortality rate unrelated to AIDS
d	AIDS related death rate.
σ	Progression rate from Asymptomatic class to AID class.
γ	Progression rate from Symptomatic class to AID class.
η	Infectivity rate of transmission
$S(t)$	Susceptible population at a given time t.
$I_1(t)$	Asymptomatic population at a given time t.
$I_2(t)$	Symptomatic population at a given time t.
$A(t)$	AIDS population.

Positivity and boundedness of the model: In this section we shall show from model (1) that the state variables are non-negative and the solutions remain positive for all $t \geq 0.$ Hence, the parameters in the model are assumed to be positive.

Theorem 1: Let the initial conditions or values of the state variables be such that $\{(S(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, A(0) \geq 0) \in \Omega\},$ then the set $(S(t), I_1(t), I_2(t), A(t))$ is non-negative in Ω for all $t \geq 0.$

Proof: Considering the first equation in (1), are considered for the positivity of the state variables as

follows using the approach of (Adeyemi and Oluyo, 2023; Oladejo and Oluyo, 2022; Ong'ala *et al*., 2012; Temesgen *et al*., 2023)

$$\frac{dS}{dt} \geq -(\beta I_1 + \beta\eta I_2 + \mu)S$$

$$\frac{dS}{dt} - \int(\beta I_1 + \beta\eta I_2 + \mu)S$$

Using variable separable

$$\frac{dS}{S} \geq -\int(\beta I_1 + \beta\eta I_2 + \mu)dt$$

$$\ln s \geq -(\beta I_1 + \beta\eta I_2 + \mu)t + C$$

$$S(t) \geq e^{-(\beta I_1 + \beta\eta I_2 + \mu)t}.e^{C_1}$$

$$S(t) = S_0 e^{-(\beta I_1 + \beta\eta I_2 + \mu)t}.$$

$$S(0) = S_0 \Rightarrow A_1 = S_0$$

Since $S(t) \geq 0$, for all $t > 0$ provided that $S_0 \geq 0$.

Hence, $S(t) \geq 0$

It is possible to show using the same procedure for other state variables that:

$$I_1(t) \geq I_1(0)e^{-(\theta+\mu+\sigma)t} \geq 0, I_1(t) \geq I_1(0)e^{-(\mu+\gamma+d)t} \geq 0, A(t) \neq A(0)e^{-\mu t} \geq 0$$

This shows that all the solutions of equation (1) are positive for all $t \geq 0$. Therefore, the HIV/AIDS transmission model stated in (1) is both epidemiologically significant and numerically well posed in an attainable given region $\Omega \geq 0$

Theorem 2

Every solution in the region $\Omega = \left\{\left(S(t), I_1(t), I_2(t), A(t) \in \Omega_+^4 : N(t) \leq \frac{\pi}{\mu}\right)\right\}$ is positively invariant with respect to the HIV/AIDS model (1) in the populations. The solutions for the system are contained and remain in the region Ω for all time $t \geq 0$.

Proof: Considering the equation of the model, and adding up all the derivatives with respect to time t, we obtained

$$\frac{dN(t)}{dt} = \pi + \mu(S + I_1 + I_2 + A) + dI_2 + dA$$

Let d=0

$$\frac{dN(t)}{dt} = \pi + \mu N$$

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

Where N_0 is the initial size of the population

Therefore,

$$\lim_{t\to\infty} N(t) \leq \frac{\pi}{\mu}$$

This result implies that HIV/AIDS model (1) has non-zero negative and bounded solution in the region Ω and all the solutions starting in Ω approach, enter or stay in Ω . Hence, it is sufficient to conclude that the model is epidemiologically well posed.

Existence and Uniqueness of solution of the Model: In this section, we establish conditions for the existence and uniqueness of a solution of our model. We shall rigorously employ Picard theorem to achieve this.

Theorem 3: Picard Theorem

Suppose

$$y' = f(t, y), y(t_0) = y_0 \qquad (3)$$

Is given system of ordinary differential equations and suppose $f(t, x)$ is continuous and satisfies a Lipschitz condition in the closed and bounded domain $\|x - x_0\| \leq \zeta, \|t - t_0\| \leq \tau$. Let $\|f(t, x)\| < M$

Then the IVP (3) has a unique solution in the interval $\|t - t_0\| \leq h$, where h=Min $\{t, \zeta / M\}$.

Using the approach of (Odebiyi *et al.*, 2024),

Considering our system of equations

$$\left.\begin{aligned} f_1(t,x) &= \frac{dS}{dt} = \pi - \beta(I_1 + \eta I_2)S - \mu S \\ f_2(t,x) &= \beta(I_1 + \eta I_2)S - (\theta + \mu + \sigma)I_1 \\ f_3(t,x) &= \theta I_1 - (\mu + \gamma + d)I_2 \\ f_4(t,x) &= \sigma I_1 + \gamma I_2 - (\mu + d)A \end{aligned}\right\} \qquad (4)$$

$$S(0) = S_0, I_0(0) = I_0, I_2(0) = I_0, T(0) = T_0, A(0) = A_0 \quad (5)$$

So that our system of equations has the form

$$x' = f(t, x) = f(x,), \qquad x(t_0) = x_0 \quad (6)$$

Define

$$D = \{x = (S, I_1, I_2, A) : S, I_1, I_2 A \leq 1\}, \quad (7)$$

And let

$\|x - x_0\| \leq \zeta, \|t\| \leq \tau$, with $t_0 = 0, x_0 = (S, I_1, I_2, A)$

Now, we shall show using Picard theorem that (6) has a unique solution, by proving the following:

1. f is continuous
2. f satisfies a Lipschitz condition, and
3. $|f| \leq M$

Now, the function $f(t, x)$ is continuous as each component $f_i, i = 1,2,3,4,$ of $f(t, x)$ is a continuous function of the variable $x = (S, I_1, I_2, A)^T$.

Let us establish the Lipschitz condition. We do this by showing that each component of $f_i, i = 1..4$ satisfies a Lipschitz condition.

Let $x = (S, I_1, I_2, A)^T$,

then $f(x) = (f_1(x), f_2(x), f_3(x), f_4(x))^T$,

Now, noting that $(S, I_1, I_2, A)^T \le 1$, we have that

$$f_1(x) - f_1(y) = \left|(\pi - \pi) + (-\beta_1)\left[S(I_1 - I_1^*) + I_1(S - S^*)\right] + (-\beta\eta)\left[S(I_2 - I_2^*) + I_2(S - S^*)\right] + (-\mu)(S - S^*)\right|$$

$$\le \beta_1\left|I_1 - I_1^*\right| + \beta_1\left|S - S^*\right| + \beta\eta\left|I_2 - I_2^*\right| + \beta\eta\left|S - S^*\right| + \mu\left|S - S^*\right|$$

$$= (\mu + \beta_1 + \eta\beta_2)\left|S - S^*\right| + \beta_1\left|I_1 - I_1^*\right| + \beta\eta\left|I_2 - I_2^*\right|$$

$$= L_{11}\left|S - S^*\right| + L_{21}\left|I_1 - I_1^*\right| + L_{31}\left|I_2 - I_2^*\right|$$

Therefore,

$$\left|f_1(x) - f_1(y)\right| \le L_1\left\|x - y\right\| \qquad (8)$$

Where $L_1 = \max\left(l_{11}, l_{22}, l_{31}\right]$ and

$l_{11} = (\mu + \beta_1 + \eta\beta_2)$

$l_{21} = \beta_1,$

$l_{31} = \beta\eta$

Similarly,

$$f_2(x) - f_2(y) = \left|\beta_1\left[S(I_1 - I_1^*) + I_1(S - S^*)\right] + \beta\eta\left[S(I_2 - I_2^*) + I_2(S - S^*)\right] + \left[-(\theta + \mu + \delta_1)(I_1 - I_1^*)\right]\right|$$

$$\le \beta_1\left|I_1 - I_1^*\right| + \beta_1\left|S - S^*\right| + \beta\eta\left|I_2 - I_2^*\right| + \beta\eta\left|S - S^*\right| + (\theta + \mu + \delta_1)\left|I_1 - I_1^*\right|$$

$$= (\beta_1 + \beta\eta)\left|(S - S^*) + [\beta_1 + (\theta + \mu + \delta_1)]\right|I_1 - I_1^*\right| + (\beta\eta)\left|I_2 - I_2^*\right|$$

$$= l_{12}\left|S - S^*\right| + l_{22}\left|\beta_1 + (\theta + \mu + \delta_1)\right| + l_{32}\left|I_2 - I_2^*\right|$$

Therefore,

$$\left|f_2(x) - f_2(y)\right| \le L_2\left\|x - y\right\| \qquad (9)$$

Where $L_2 = \max\left(L_{12}, L_{22}, L_{32}, L_{42}\right]$ and

$L_{12} = (\beta_1 + \beta\eta); L_{22} = \beta_1 + (\theta + \mu + \delta); L_{32} = \beta\eta$

are constant depending on the parameter values of the model.

Also,

$$\left|f_3(x) - f_3(y)\right| = \left|\theta(I_1 - I_1^*) + \left[-(\mu + \gamma + d)\right]\left(I_2 - I_2^*\right)\right|$$

$$\le \theta\left|I_1 - I_1^*\right| + (\mu + \gamma + d)\left|I_2 - I_2^*\right|$$

$$= 0\left|S - S^*\right| + \theta\left|I_1 - I_1^*\right| + \left[(\mu + \delta_2)\right]\left|I_2 - I_2^*\right| + 0\left|A - A^*\right|$$

$$= L_{13}\left|S - S^*\right| + L_{23}\left|I_1 - I_1^*\right| + L_{33}\left|I_2 - I_2^*\right| + l_{43}\left|A - A^*\right|$$

$$\left|f_3(x) - f_3(y)\right| \le L_3\left\|x - y\right\| \qquad (10)$$

Where $L_3 = \max\left(L_{13}, L_{23}, L_{33}, L_{43}\right)$;

And $L_{13} = 0; L_{23} = \theta; L_{33} = (\mu + \gamma + d), L_{43} = 0$

are constant depending on the parameter values of the model. Also,

$$\left|f_4(x) - f_4(y)\right| \le \left|\sigma(I_1 - I_1^*) + \gamma(I_2 - I_2^*) + \left[-(\mu + d)\right](A - A^*)\right| \le \sigma\left|I_1 - I_1^*\right| + \delta\left|I_2 - I_2^{**}\right| + (\mu + d)\left|A - A^*\right|$$

$$= \sigma\left|I_1 - I_1^*\right| + \delta\left|I_2 - I_2^*\right| + (\mu + d)\left|A - A^*\right| = L_{14}\left|S - S^*\right| + L_{24}\left|I_1 - I_1^*\right| + L_{34}\left|I_2 - I_2^*\right| + L_{44}\left|A - A^*\right|$$

$$= \left|f_4(x) - f_4(y)\right| \le L_4\left\|x - y\right\| \qquad (11)$$

where $L_4 = \max\left(L_{14}, L_{24}, L_{34}, L_{44}\right)$

$L_{14} = 0; L_{24} = \sigma; L_{34} = \delta; L_{44} = (\mu + d)$

are constants depending on the parameter values of the model.

Therefore, $\left|f_4(x) - f_4(y)\right| \le L_4\left\|x - y\right\|$,

To obtain the bound for $f(t, x)$: clearly $(S, I_1, I_2, A) \le 1$

$$\left|f_1(x)\right| \le \lambda + \beta\left|I\right\|S\right| + \beta\eta\left|I_2\right\|S\right| + \mu\left|S\right|$$

$$\le \lambda + \beta + \beta\eta + \mu = M_1$$

$$\left|f_2(x)\right| \le \lambda + \beta\left|I_1\right\|S\right| + \beta\eta\left|I_2\right\|S\right| + (\theta + \mu + \delta_1)\left|I_1\right|$$

$$\le \beta_1 + \beta\eta + (\theta + \mu + \delta_1) = M_2$$

$$\left|f_3(x)\right| \le \theta\left|I_1\right| + (\mu + \gamma + d)\left|I_2\right| = \theta + (\mu + \gamma + d)$$

$$= M_3$$

$$\left|f_4(x)\right| \le \sigma\left|I_1\right| + \gamma\left|I_2\right| + (\mu + d)\left|A\right| \le \sigma + \gamma + (\mu + d)$$

$$= M_4$$

Therefore, $\left|f(x)\right| = \max\left\{M_1, M_2, M_3, M_4\right\} \le M$

Therefore, we have shown that a unique solution exists for the initial value problem (IVP) in equation (6) within the specified domain $\left|x - x_0\right| \le \zeta$ and $\left|t\right| \le h$. Thus completing the proof.

Mathematical Analysis of the Model

Disease free equilibrium point: This refers to a state where a disease is no longer present or prevalent in a population. That means that it is a state where the disease has been completely eliminated or eradicated. At the equilibrium,

$$\frac{dS}{dt} = \frac{dI_1}{dt} = \frac{dI_2}{dt} = \frac{dA}{dt} = 0$$

$$\left.\begin{aligned}\frac{dS}{dt} &= \pi - \beta(I_1 + \eta I_2)S - \mu S \\ \frac{dI_1}{dt} &\beta(I_1 + \eta I_2)S - (\theta + \mu + \sigma)I_1 \\ \frac{dI_2}{dt} &= \theta I_1 - (\mu + \gamma + d)I_2 \\ \frac{dA}{dt} &= \sigma I_1 + \gamma I_2 - (\mu + d)A\end{aligned}\right\} \quad (12)$$

Disease free equilibrium: This is the equilibrium point at which population remains in the absence of disease. $S \neq 0, I_1 = I_2 = T = A = 0$ Substituting these to equation (12) and solving gives the infection – free equilibrium as

$$E^0 = \left(S^0, I_1^0, I_2^0, A^0\right) = \left(\frac{\pi}{\mu}, 0, 0, 0\right)$$

Endemic equilibrium point: The endemic equilibrium state is the state where the disease cannot be totally eradicated but remains in the population. For the endemicequilibrium,

$S \neq 0, I_1 \neq 0, I_2 \neq 0, T \neq 0, A \neq 0$

Solving equations (12) in terms of I_1^* simultaneously when $S \neq 0, I_1 \neq 0, I_2 \neq 0, T \neq 0, A \neq 0$, we have the endemic equilibrium points respectively as;

$$E^* = \left(S^*, I_1^*, I_2^*, A^*\right) = \left[\frac{\pi}{\mu R_0}, \frac{\pi(R_0 - 1)}{R_0 G_1}, \frac{\theta I_1}{G_2}, \frac{(G_2\sigma + \gamma\theta)I_1}{G_2 G_3}\right] \quad (13)$$

respectively, where,

$$G_1 = (\theta + \mu + \sigma), G_2 = (\mu + \sigma + d), G_3 = (\mu + d)$$

Derivation of Basic Reproduction Number, R_0*:* The computation of the basic reproduction number is essential. The basic reproduction number R_0 is defined as the average number of new cases of an infectious disease that a single infected person can generate in a population that is fully susceptible to the disease. In other words, it measures how easily a disease can spread through a population. To determine the next generation matrix for the model, the number of ways that new infections can arise or be created and also the number of ways that infections can be transferred between compartments are put into consideration.

Then F_i and V_i are computed as follows using the approach of (Van den Driessche and Watmough, 2002)

$$F_i = \begin{bmatrix}\beta(I_1 + \eta I_2)S \\ 0 \\ 0\end{bmatrix}, \text{ and } V_i = \begin{bmatrix}(\theta + \mu + \delta_1)I_1 \\ -\theta I_1 + (\mu + \gamma + d)I_2 \\ -\sigma I_1 - \gamma I_2 + (\mu + d)A\end{bmatrix} \quad (14)$$

The Jacobian matrices of F_i and V_i at the disease free equilibrium point x_0, are

Let K be the next generation matrix, comprising of two parts F and V^{-1} where

$$DF(x_0) = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right] = \begin{bmatrix}\frac{\beta\pi}{\mu} & \frac{\beta\eta\pi}{\mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0\end{bmatrix} \quad (15)$$

$$DV(x_0) = \left[\frac{\partial V_1(x_0)}{\partial x_j}\right] = \begin{bmatrix}G_1 & 0 & 0 \\ -\theta & G_2 & 0 \\ -\sigma & -\gamma & G_3\end{bmatrix} \quad (16)$$

Where, $G_1 = (\theta + \mu + \sigma), G_2 = (\mu + \gamma + d)$ e, $G_3 = (\mu + d)$

$$V^{-1} = \begin{bmatrix}\frac{1}{G_1} & 0 & 0 \\ \frac{\theta}{G_1 G_2} & \frac{1}{G_2} & 0 \\ \frac{\gamma\theta + \sigma G_2}{G_1 G_2 G_3} & \frac{\gamma}{G_2 G_2} & \frac{1}{G_3}\end{bmatrix} \quad (17)$$

$$FV^{-1} = \begin{bmatrix}\frac{\beta\pi}{\mu G_1} + \frac{\beta\eta\pi\theta}{\mu G_1 G_2} & \frac{\beta\eta\pi}{\mu G_2} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0\end{bmatrix} \quad (18)$$

The basic reproduction number, which is the dominant Eigen-value of the product FV^{-1}, is therefore obtained as:

$$R_0 = \frac{\beta\pi(\eta\theta + G_2)}{\mu G_1 G_2} \quad (19)$$

Stability analysis of the Disease Free Equilibrium

*Local Stability: Theorem 4:*The disease-free state is locally asymptotically stable if the basic reproduction number $R_0 < 1$ and unstable if otherwise.

Proof:

We evaluate the Jacobian Matrix of the model (12) at the disease free equilibrium $\left(\frac{\pi}{\mu},0,0,0\right)$

$$J_0=\begin{bmatrix} -\mu & -\frac{\beta\pi}{\mu} & -\frac{\beta\pi\eta}{\mu} & 0 \\ 0 & \frac{\beta\pi}{\mu}-G_1 & \frac{\beta\eta\pi}{\mu} & 0 \\ 0 & \theta & -G_2 & 0 \\ 0 & \sigma & \gamma & -G_3 \end{bmatrix} \quad (20)$$

We obtained the Characteristic polynomial as

$(\lambda+G_3)(\lambda+\mu)\left[\lambda^2 a+b\lambda+c\right]=0$

Where,

$a=1$

$b=(G_1+G_2)-\frac{\beta\pi}{\mu}$

$c=\mu G_1G_2(1-R_0)$

Clearly,

$\lambda_1=-G_3,\lambda_2=-\mu$ and λ_3,λ_4 is obtained from the quadratic equation. If

$a>0, b>0,$ If $b=(G_1+G_2)>\frac{\beta\pi}{\mu}$, $c>0\Rightarrow R_0>1$

Then by Routh Hurwitz criteria, the remaining four eigen-values are negative. Hence, the disease free equilibrium is locally asymptotically stable.

Global stability for disease free equilibrium: Theorem 3:The disease free equilibrium of system (12) is globally achievable and stable when the basic reproduction number is less than 1, but it becomes unstable and prone to disease outbreaks when the basic reproduction is greater than 1.

Proof:

Using Comparison theorem as implemented in (Mushayabasa and Bhunu, 2011) that the rate of change of the infected compartment of system (1) can be written as

$$\begin{bmatrix} \frac{dI_1}{dt} \\ \frac{dI_2}{dt} \\ \frac{dA}{dt} \end{bmatrix}=(F-V)\begin{bmatrix} I_1 \\ I_2 \\ A \end{bmatrix}-F_i\begin{bmatrix} I_1 \\ I_2 \\ A \end{bmatrix} \quad (21)$$

Where, $F-V=\begin{bmatrix} \frac{\beta\pi}{\mu}-G_1 & \frac{\beta\eta\pi}{\mu} & 0 \\ \theta & -G_2 & 0 \\ \sigma & \gamma & -G_3 \end{bmatrix}$ (22)

and, $G_1=(\theta+\mu+\sigma), G_2=(\mu+\gamma+d), G_3=(\mu+d)$

The characteristic equation is

$(\lambda+G_3)\left\{\lambda^2 a+b\lambda+c\right\}$ (23)

Where

$a=1, b=(G_1+G_2)-\frac{\beta\pi}{\mu}, c=\mu G_1G_2(1-R_0)$

Clearly,

$\lambda_1=-G_3,$ and λ_2,λ_3 is obtained from the quadratic equation. $a>0$, $b>0$ if $b=(G_1+G_2)>\frac{\beta\pi}{\mu}$

$c>0\Rightarrow R_0<1$

Therefore, by Routh-Hurwitz criteria, the remaining eigen-values are negative. Hence, the disease free equilibrium is globally asymptotically stable.

Global stability of endemic equilibrium

Theorem 4: If $R_0>1,$ then the endemic equilibrium point of the model equation (12) is globally asymptotically stable in Γ, provided $S\geq S^*, I_1\geq I_1^*, I_2\geq I_2^*,$ and $A\geq A^*$.

Proof: To establish the global stability of the endemic equilibrium E^*, following the approach of (Olaniyi, 2023), we analyzed by constructing the following quadratic Lyapunov function L such that

$$L=\frac{1}{2}\left[(S-S^*)+(I_1-I_1^*)+(I_2-I_2^*)+(A-A^*)\right]^2$$

By direct calculation of the time derivatives $L(t)$ along the solutions of the system (12) is obtained as

$$\frac{dL}{dt}=\left[(S-S^*)+(I_1-I_1^*)+(I_2-I_2^*)+(A-A^*)\right]\left(\frac{dS}{dt}+\frac{dI_1}{dt}+\frac{I_2}{dt}+\frac{dA}{dt}\right) \quad (24)$$

Substituting the appropriate solutions of the system (1) into the derivative of $L(t)$ gives

$$\frac{dL}{dt}\leq\left[(S-S^*)+(I_1-I_1^*)+(I_2-I_2^*)+(A-A^*)\right]\frac{dN}{dt}$$

$$\frac{dL}{dt}\leq\left[(S-S^*)+(I_1-I_1^*)+(I_2-I_2^*)+(A-A^*)\right](\Lambda-\mu N)$$

$$\leq\left(N-\frac{\pi}{\mu}\right)(\pi-\mu N) \quad (25)$$

We obtain the result by rearranging and simplifying (24) ,

$$\leq-\frac{1}{\mu}(\pi-\mu N)^2$$

Let

$$\psi=\pi-\mu N\Rightarrow\frac{dL}{dt}\leq-\frac{1}{\mu}\psi^2 \quad (26)$$

Hence, $\left(\frac{dL}{dt}\right)(S, I_1, I_2, A) \leq 0$ and $\frac{dL}{dt} = 0,$ if and only if $S = S^*, I_1 = I_1^*, I_2 = I_1^*, A = A^*$ Therefore, If $X < Y,$ then, $\frac{dL}{dt}$ will be negative definite, implying that $\frac{dL}{dt} = 0,$ if and only if $S = S^*, I_1 = I_1^*, I_2 = I_2^*,$ and $A = A^*$ .Therefore, the largest positive invariant set in $\left\{\left(S^*, I_1^*, I_2^*, , A^*\right) \in \Omega : \frac{dL}{dt} = 0\right\}$ is a singleton $\{E_1\},$ where E_1 is globally asymptotically stable in the set Γ in accordance to LaSalle's invariant principle LaSalle, JP (1976), it then implies that E_1 is globally asymptotically stable in Γ if $R_0 > 1.$

Sensitivity Analysis of the basic Reproduction number: Sensitivity indices tell us how crucial each parameter is to disease transmission and prevalence and discover parameters that have a high impact on R_0 and should be targeted by intervention strategies. More so, sensitivity analysis is commonly used to determine the robustness of model predictions to the parameter values (since there are usually errors in data collection and presumed Description of Sensitivity Analysis parameter values and more so, to discover parameters that have a high impact in R_0 .

Sensitivity analysis also allow us to measure the relative change in a state variable when a parameter changes. The normalized forward sensitivity index of a variable to a parameter is a ratio of the relative change in the variable to the relative change in the parameter. When a variable is differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivatives.

Using approach of (Chitnis, 2008), the normalized forward sensitivity index of a variable "g" that depends differentiable on a parameter "h" is defined as

$$X_h^g := \frac{\partial g}{\partial h} * \frac{h}{g} \quad (27)$$

As we have an explicit formula for R_0 in equation (19), we derive an analytical expression for the sensitivity of R_0, as $X_h^g := \frac{\partial g}{\partial h} * \frac{h}{g}$ with respect to each of the parameters involved in R_0 as computed in table 2.

Interpretation of Sensitivity Indices: Table 2 represents the sensitivity index for the base line parameter values and it shows that recruitment rate (π) and transmission rate (β) are the most sensitive parameters. When the parameters $\pi, \beta,$ and η increase while the other parameters remain constant, the value of R_0 will also increase. More so, when the parameters $\theta, \gamma, \sigma, \mu$ and d increase while keeping other parameters constant, the value of R_0 will decrease. It should be targeted by intervention strategies in order to have a stable and disease free environment. For instance, $X_\beta^{R_0}$ = +1.0000 and $X_\pi^{R_0}$ =+1.0000 means that increasing or decreasing β and π by 10% increases or (decreases) R_0 by 10% while $X_\sigma^{R_0}$ = -0.2797202797means that increasing or (decreasing) σ by 10% decreases (or increases) R_0 by 2.797202797% as seen in table 2 below. Others can be calculated following same procedure.

Table 2: parameters, values, sensitivity index and source used in the model

Parameter	Value	Sensitivity index	Source
π	300	1.0000	Estimated
β	0.0009	1.0000	Al-sheikh, (2011)
θ	0.015	-0.01833895058	Al-sheikh, (2011)
γ	0.2	-0.00031059652	Ratera, (2024)
η	0.3	0.00264007040	Assumed
σ	0.2	-0.2797202797	Ratera, (2012)
d	1.0	-0.6682196492	Odebiyi. *et al.*, 2024
μ	0.02	-0.700077190	Ibrahim*et al.*, 2021

Numerical Simulations and Discussion: Simulation of Simulation of the model was performed for better understanding of dynamical spread of transmission of HIV/AIDS infection using Maple 18.0 software. This section presents the numerical results for the model considered using Maple 18.0 software and the direct substitution method. The simulation demonstrates model equation, the global stability of the HIV/AIDS transmission and control. Also, the simulation reveals the impact of these parameters on the numerical spread of the disease. The results of the numerical simulations are given in figures 1-5 to illustrate the system's behaviour for different values of the model's parameter. The impact of screening rate on the spread of the disease is analyzed using realistic parameters, showing how changes in screening rates affect the numerical dynamics of the disease, highlighting the

importance of screening in controlling the spread of the disease.

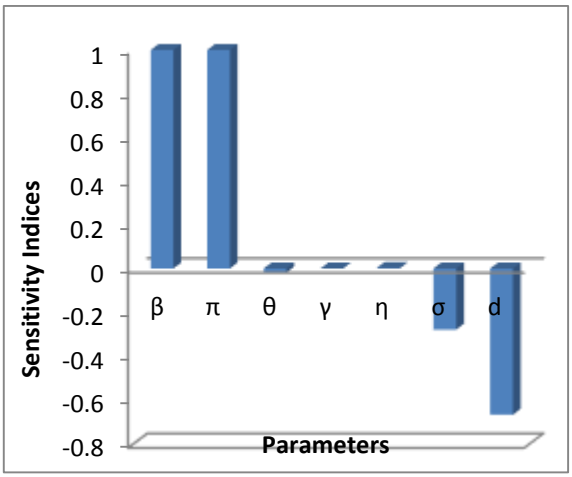

Fig 2: Graphical Representation of the Sensitivity indices of R_0

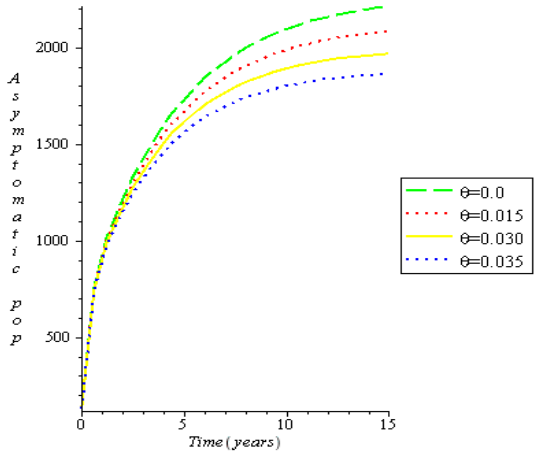

Fig 1:portrait of susceptible against θ

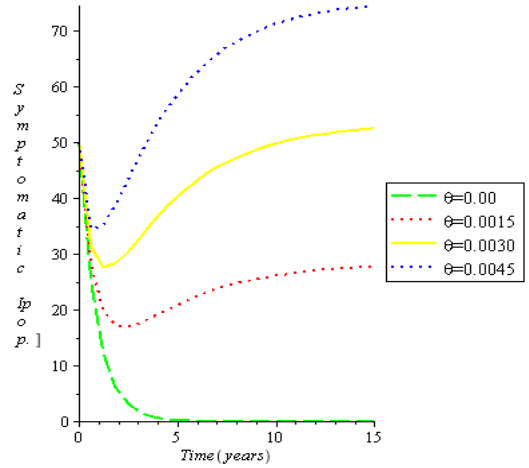

Fig2: portrait of symptomatic against θ

According to figure 1, the asymptomatic population are unaware of their status and may be more likely to engage in risky behaviours. Therefore, as they are been screened, they become aware of their status and thereby, leading to a reduction in their population. However, some of these individuals progress to the symptomatic class, causing an increase in the number of symptomatic population as illustrated in figure 2.

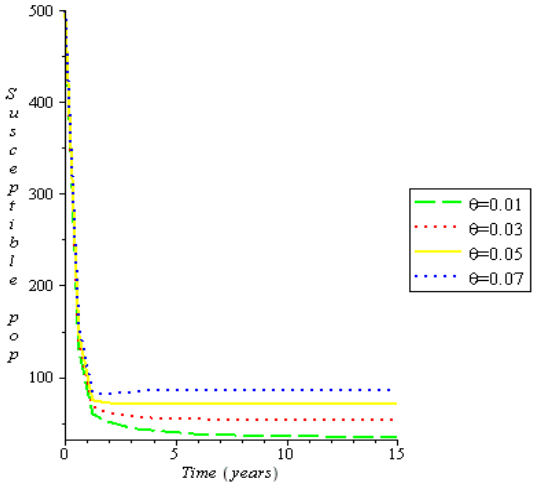

Fig 3: portrait of susceptible against θ

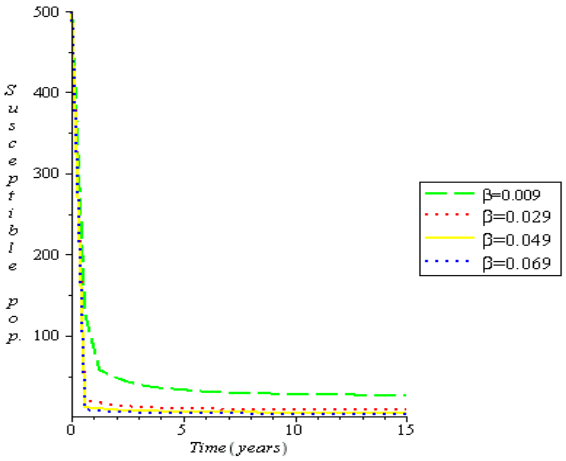

Fig 4: portrait of susceptible against β

Figure 3 illustrates the relationship between the susceptible populations against time with various values of screening rate. It was observed that as the screening rate increases, the susceptible population also grows.

This is because individuals who are screened and aware of their status are more likely to take precautionary measures, avoid risky behaviours such as sexual activities and refrain from been contact with infective individuals, thereby maintaining a healthy environment and reducing the spread of the disease

while Figure 4 reveals the portrait of Susceptible population against time with various transmission rates for susceptible individual with asymptomatic infective. The plot shows that as the transmission rate increases, the susceptible population declines, highlighting the importance of adhering to health regulations to prevent the spread of the disease.

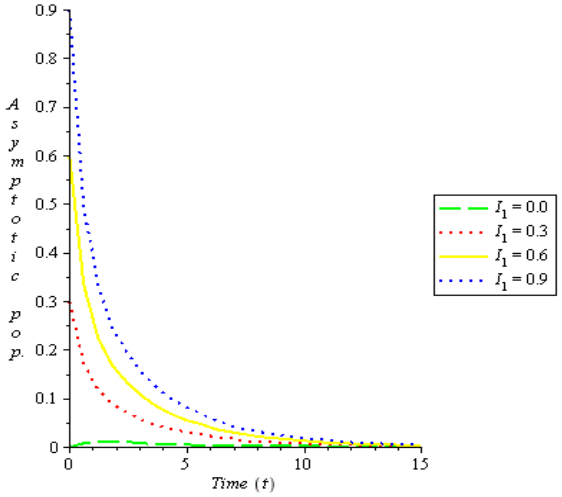

Fig 5: portrait of global stability of the disease free equilibrium with various initial conditions.

Figure 5 reveals portrait of global stability of the equilibrium with various initial conditions as illustrated. This agrees with the result of the global stability of disease free in theorem 3. It simply suggest that if the basic reproduction number $R_0 < 1$, HIV/AIDS can be eradicated from the population, regardless of how many people are initially infected. In other words, if the condition $R_0 < 1$ is met, the disease will eventually die out, regardless of the initial number of infective individuals whenever $R_0 < 1$. The solutions stabilize or converge to the HIV/AIDS-free equilibrium point, indicating that the asymptomatic infective population will eventually decrease to zero.

Conclusion: This paper presents a nonlinear mathematical model for the spread of HIV/AIDS. It was shown that the disease free is locally asymptotically stable when $R_0 < 1$ and the global stability of the endemic equilibrium was also established using a quadratic Lyapunov function. A sensitivity analysis of the basic reproduction number revealed that the recruitment rate and transmission rate are the most sensitive parameters, highlighting the need for intervention strategies to focus on reducing these parameters. The analysis also showed that increasing the rate of detection through screening can control the spread of the disease, emphasizing the importance of screening in preventing the endemicity of HIV/AIDS population and should also be key targets for intervention. The findings also underscore the need for caution among HIV infected and susceptible individuals in the environment to prevent the spread of the disease. The importance of screening is therefore evident in its ability to detect and reduce the number of infective individuals.

Declaration of Conflict of Interest: The authors declare no conflict of interest

Data availability statement:
Data are available upon request from the first author or corresponding author

REFERENCES

Adeyemi, MO; Oluyo, TO (2023). Mathematical Modelling for the Control of Fly Borne Mastitis Disease in Cattle. Frontirrs in Applied Mathematics and Statistics, 1-19.https://doi.org/10.3389/fams.2023.1171157

Chitnis, N; Cushing, JM; Hyman, M (2008). Determining important parameters in the spread of Malaria Through the sensitivity analysis of Mathematical model. Bulleting of Mathematical Biology, 70:1272-1296. https://doi.org/10.1137/050638941

Diekmann, O; Heesterbeek, JA; Metz, JAJ (1990). On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous population. *J.Math.Bio.*28:265-382

Gaodart, F; Bailes, E; Robertson, DL (1994). 'Origin of HIV-1 in the Chimpanzee Pan troglodytestroglodytes'. *Nature* 397(6718):436--41

Global and Regional Trend (2023) .UNICEF data

HIV/AIDS facts (2004). Pediatric HIV Infection and AIDS: Point of View. https://tnsmb.org/journal/index.php/ijman/article/view/54

Ibrahim, IA; Daniel, EE; Dan-Hausa, AA; Adamu, MU; Shawalu, CJ; Yusuf, A (2021). Mathematical Modelling of Dynamics of HIV Transmission Depicting the Importance of Counselling and Treatment. *J. Appl. Sci. Environ. Manage.*25 (6) 893-903. https://www.ajol.info/index.php/jasem

ISSA, S; Massawe, S (2011). Modelling the effect of screening on the spread of HIV infection in a

population with variable inflow of infective immigrants. *Sci. Res. Essays*.20: 4397-4405.

Lamptey, P; Wigley, M; Carr, D; Collymore, Y (2003). Facing the HIV/AIDS Pandemic" Publication of the Population References Bureau (PRB), population bulleting Washington, DC, 57(1):1-43.

LaSalle, JP (1976). The stability of dynamical systems, regional conference series in Applied Mathematics (*SIAM)*, Philadelphia.

Marsudi, M; Andari, A (2014). Sensitivity Analysis of Effect of Screening and HIV Therapy on the Dynamics of Spread of HIV. *Appl. Math. Sci.* 8(155):7749-7763. DOI: https://doi.org/10.12988/ams.2014.49737.

Mushayabasa, S; Bhunu, CP(2011). Modelling Schistosomiasis and HIV/AIDS Co-dynamics. Computational and Mathematical Methods in Medicine. *J. Math. Anal. Applicat.* 379(2):852-860.

Odebiyi, OA; Oladejo, JK (2024). Stability Analysis of HIV/AIDS Model with Saturated Incidence. *Intern. J. Math. Anal. Model.* 7(1):75-98.

Odebiyi,OA; Ayeni,RO (2012).Modelling and Simulation of HIV infection of $CD4^+$ T cells with past and current History of the disease. *J. Nig. Assoc. Math. Phys.* 22:495-502.

Odebiyi, OA; Oladejo, JK; Yahaya, AA; Elijah, EO (2024). Analysis of HIV/AIDS Model with nonlinear Incidence Function. *Inter. J. Res. Innov. Appl. Sci.* 9 (4): 317-341. DOI: 10.511584/IJRIAS/

Odebiyi, OA; Areo, AO; Olajide, OA; Taiwo, AA, Yahaya, AA (2024). Investigating Picard's Existence and Uniqueness of Solution of an HIV/AIDS Epidemic Model in a Population. *Inter. J. Res. Anal. Rev.* 11(1): 790-798.

Oladejo JK, Oluyo TO (2022). Effect of Prep on HIV/AIDS dynamics with Immigration of Infective. *Inter. J. Math. Anal. Model.* 5(2):261-283. https://tnsmb.org/journal/index.php/ijman/article/view/54

Olaniyi, S; Falowo, OD; Mukamuri, OS; Obabiyi, OS; Adepoju, OA (2023) Effects of saturated treatment on malaria spread with Optimal Control intervention. *Alexandria Engineer. J.* 65: 443-459. www.elsevier.com/locate/aej

Ong'ala, JO; Mugisha J; Odahiambo, J (2012). Mathematical Model for Pneumonia Dynamics among Children. Africa Mathematical Sciences Association Conference (*SAMSA* 2012) 26th -29th Nov.2012.

Ratera, S; Estomih, SM; Daniel, OM (2012). "Modelling the Effect of Screening and Treatment on Transmission of HIV/AIDS Infection in a Population. *Am. J. Math. Stat*. 2(4):75-88.

Sarah Al-sheikh; Farida, M; Muna, A (2011). Stability Analysis of an HIV/AIDS Epidemic Model with Screening. *Inter. Math. Forum.* 6(66): 3251-3273.

Sharp, PM; Beatrice, BH (2011). Origins of HIV and AIDS the *Pandemic*. DOI:10.1101/cshperspect.a006841

Srinivase, ASR (2003). Mathematical Modelling of AIDS epidemic in India. *Curr. Sci*. 84.

Stodart, CA; Reyes, RA (2006). Models of HIV-1 disease: A Review Status. *Drug.Discov. Today Disease Models*. 3(1):113-119.

Temesgen, DK; Fekadu, ML; Ebisa, Olana, B (2023). Optimal Control Analysis of the Dynamics of Covid-19 with Application to Ethiopian Data. *Appl. Math. Inf. Sci.* 17(5): 867-880.

UNAIDS/WHO (2002). Pediatric HIV infection and AIDS: Point of view

Van den Driessche, P; Watmough, J (2002). Reproduction numbers and Sub-threshold Endemic equilibra for compartment models of disease transmission. *Math. Biosci*. 180: 29-48.

WHO (2007). 'Case definitions of HIV for surveillance and revised clinical staging and immunology classification of HIV-related disease in adults and children'. Geneva: World Health Organization. Pp.6-16. ISBN 978-92-4-159562-9