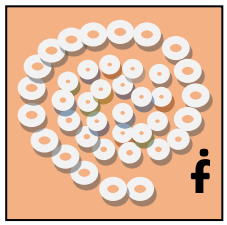

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Stability analysis of an HIV/AIDS model with saturated incidence

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Abstract

Human Immunodeficiency Virus – Acquired Immune Deficiency Syndrome HIV/AIDS stands as one of the most prevalent sexually transmitted disease globally and is regarded as one of the deadliest epidemic in human history. This study presents a mathematical model for understanding the dynamics of HIV/AIDS transmission, incorporating a saturated incidence rate. The model employs a system of ordinary differential equations, comprising various group of individuals including susceptible $S(t)$, asymptomatic infective $I_1(t)$, symptomatic infective $I_2(t)$, treated $T(t)$ and AIDS $A(t)$ class. The validity of the solution states affirms that the model is well-defined and holds epidemiological significance. The disease-free and endemic equilibrium states are identified, and their stability is analyzed using Routh Hurwitz criteria and Descartes rule of signs. Sensitivity analysis was carried out using normalized forward sensitivity index and result showed that the contact rate β_1 is the most sensitive parameter. Existence of backward bifurcation analysis is equally carried out. More so, it is observed from the numerical simulation that screening and treatment of the infective play a significant role in reducing the transmission of the disease. The outcome of the stability analysis for both disease-free and endemics equilibrium states indicates the potential for HIV/AIDS control.

Keywords and phrases: basic reproduction number; bifurcation analysis; saturated incidence; stability, sensitivity analysis;

1 Introduction

HIV/AIDS is seen as a chronic disease nowadays, because HIV-positive people can live with infection for many years provided their immune systems are checked. It gradually destroys the immune system until it is unable to fight infections that would normally have been prevented. With deterioration of the immune system, the body develops opportunistic diseases that lead to AIDS [1].

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As the immune system becomes compromised, the HIV opportunistic diseases such as meningitis, Cancers and Tuberculosis do easily attack the body [2].

In 2022, approximately 39million people died from AIDS worldwide, about 740 children became infected with HIV and approximately 274 children died from AIDS related causes every day [3]. More so, in the countries hardest hit, AIDS has sapped the population of young men and women who form the foundation of the labor force. Most die while in the peak of their reproductive years. Moreover, the epidemic has overwhelmed health care systems, increases the number of orphans, and caused life expectancy rates to plummet. It therefore constitutes a serious threat to future development in Africa.

At the end of 2022, approximately 39.0 million (33.1million-45.7million) people were living with HIV globally. 1.3million [1million-1.7million] people became newly infected with HIV while 630000 [480000-880000] people died from AIDS-related illnesses. 1.5 million (1.2-2.1million) children were living with HIV (0-14 years old) [4]. More so, according to HIV/AIDS prevalence estimates between 2023-2024, African countries with HIV/AIDS adult prevalence rates among adults in various countries according to CIA world fact book are Eswatini with 28.30%, Lesotho 24.10%, Botswana 22.60% and Zimbabwe 22.10% and outside Africa, Bahamas has the highest prevalence rate [5].

Transmission of HIV/AIDS causing virus occurs most commonly through sexual intercourse. HIV can also be transmitted through transfusions of HIV-contaminated blood or by using a contaminated needle or syringe to inject drugs into the blood stream. Infection with HIV does not necessarily mean that a person has AIDS. Some people who have HIV infection may not develop any of the clinical illnesses that define the full-blown disease of AIDS for ten years or more. Physicians prefer to use the term AIDS for cases where a person has reached the final, life-threatening stage of HIV infection [6]. In a person infected with HIV, the virus steadily destroys $CD4^{+}T$ cells over a period of years, diminishing the cells protective ability and weakening the immune system. HIV infects some other cells and wrecks the largest part of the $CD4^{+}T$ cells and this causes the $CD4^{+}T$ cells destruction and decline, hence reducing the confrontation of the susceptible system [7, 8].

Screening is the process of performing the HIV-antibody test to all individuals within a defined population. Routine screening of unaware infective has now become an integral part of programs in low and middle income countries. People can get HIV tests done at a health clinic, at special HIV voluntary counseling and testing (VCT) sites [9]. Antiretroviral medication has made it possible for many individuals who have been very sick with HIV/AIDS to become fully functioning again, with a low or even undetectable viral load. Without antiretroviral therapy, someone who has AIDS typically dies within a year [10]. Transmission of the disease and influence of the infection can be curbed by taking adequate treatment and administering suitable vaccine to infected class [11-14].

In the context of mathematical modeling of HIV/AIDS dynamics, Saturation incidence refers to a situation where the rate of new infections reaches a plateau, indicating that the epidemic has stable and under control and therefore no longer growing exponentially. This simply means that the number of new infections is roughly balanced by the number of individuals recovering or dying from the infection.

Various mathematical models were formulated for developing strategies to control the outbreak of disease and thereby making a trade-offs in choosing a best possible treatment for curbing and controlling the infection. Ross Ronald derived a threshold quantity called basic reproduction number. This helps the planners such as medical and health practitioners to determine and conclude when infection will fade out in a system. Incidence rate play a vital and significance role in discussing nature of a disease in epidemiology. In early, bilinear incidence rate in the form βxy and standard incidence rate in the form $\frac{\beta xy}{N}$ are usually used in epidemic model where β is the transmission of the disease per contact. x and y are the number of susceptible population and infected population respectively. N is the total number of population [15]. Many authors studied nonlinear types of incidence rate. Few among them are [15-18, 20]. [20] introduced saturated types of infection rate in the form $\frac{\beta xy}{(1+\alpha y)}$ where βy denotes force of infection and $\frac{\beta xy}{(1+\alpha y)}$ measures inhibition factors due to crowding effect of infected class and also an appropriate social awareness given by the population. α is the coefficient of inhibition. Later, a non-monotone type of infection rate in the form $\frac{\beta xy}{(1+\alpha y^2)}$ was also introduced to include the population psychological [11, 20]. [21] also introduced Saturated incidence rate $\frac{\beta SI}{(1+\alpha I)}$, tends to a saturation level when it gets large, where βI measures the infection force when the disease is entering a fully susceptible population and $\frac{1}{(1+\alpha I)}$ measures the inhibition effect from the behavioral change of susceptible. The saturated incidence rate of the form $\frac{1}{(1+\alpha_1 S)}$ where α_1 is a positive constant. Here, the effect of saturation factor α_1 stems from epidemic control taking appropriate percussive measures. The function forms for disease incidence with the susceptible population in the denominator of the saturating function is simply to know when the population become saturated and affected with the virus. The saturated incidence function of the form $\frac{1}{(1+\alpha_2 I)}$, where α_2 is a positive constant. Here, the number of effective contacts between infective individuals or due to the protective measures by the susceptible individuals.

From an epidemiological standpoint, saturation incidence suggests that the epidemic has reached a steady state, with the disease prevalence remaining relatively constant over time. This could simply be due to various factors such as behavioral changes, implementation of prevention strategies, availability of treatment, or natural progression of the epidemic within a population. Understanding saturation incidence therefore helps public health officials and policy makers assess the effectiveness of interventions and plan future strategies for controlling the spread of the menace.

Researchers such as [19, 22-23] among others have worked on the transmission and prevention of HIV/AIDS incorporating various stages of HIV infection and AIDS, screening and treatment of HIV/AIDS, vaccination and saturated incidence rate. Although no cure has been found for AIDS

[14], new drugs are available that can prolong the life span and improve the quality of life of infected people [12, 22,38].

2 Model formulation

In this research, the susceptible and infectious epidemic model (SI) is considered as presented by [19, 24]. A population size of $N(t)$ was partitioned into 5 subclasses of individuals which are; susceptible, asymptomatic infective, symptomatic infective, treated infective, and AIDS with sizes denoted by $S(t), I_1(t), I_2(t), T(t),$ and $A(t)$, respectively such that $N = S + I_1 + I_2 + T + A,$ as shown in figure 1.

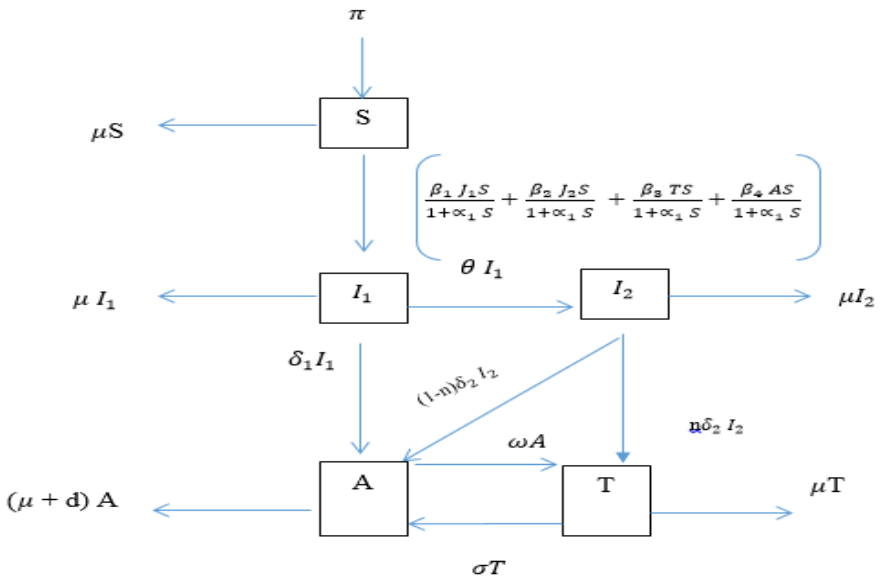

Figure 1: Transmission diagram for susceptible, infected model with the saturation term to the susceptible $(1+\alpha_1 S)$

Following the transmission diagram, in Figure 1, which incorporates the saturation term to the susceptible, asymptomatic infective, symptomatic infective, treated infective and AIDS individual. The following assumptions are taken into consideration in formulating our proposed model:

People are recruited into the Susceptible population by birth . All parameters are positive. Asymptomatic infective class (I_1) can move to symptomatic class (I_2) and full blown AIDS and asymptomatic infective population can be screened at a rate θ and progress to symptomatic class. Asymptomatic infective, Symptomatic infective, Treated class (T) and full blown AIDS can infect Susceptible class at different rates $\beta_1, \beta_2, \beta_3$ and β_4 respectively. Asymptomatic infective, Symptomatic infective, Treated class (T) will move to full blown AIDS at different rates $\delta_1, \delta_2,$ and σ respectively . Infective can be treated with ARV therapy (i.e can move to treated class) at rate ω . Considering all the assumptions made, the following system of ordinary differential equations of the proposed model is therefore considered.:

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \frac{\beta_1 I_1 S}{(1+\alpha_1 S)} - \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} - \frac{\beta_3 TS}{(1+\alpha_1 S)} - \frac{\beta_4 AS}{(1+\alpha_1 S)} - \mu S \\
\frac{dI_1}{dt} &= \frac{\beta_1 I_1 S}{(1+\alpha_1 S)} + \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} + \frac{\beta_3 TS}{(1+\alpha_1 S)} + \frac{\beta_4 AS}{(1+\alpha_1 S)} - (\theta + \mu + \delta_1) I_1 \\
\frac{dI_2}{dt} &= \theta I_1 - (\mu + \delta_2) I_2 \\
\frac{dT}{dt} &= n\delta_2 I_2 - (\sigma + \mu) T + \omega A \\
\frac{dA}{dt} &= (1-n)\delta_2 I_2 + \delta_1 I_1 - (\mu + d + \omega) A + \sigma T
\end{aligned}\right\} \quad (1)$$

As initial condition, we choose

$$S(0) = S_0, I_1(0) = I_{1_0}, I_2(0) = I_{2_0}, T(0) = T_0, A(0) = A_0$$

The model parameters are defined as follows

$\pi > 0$ is the constant number of recruitment of susceptible, $\beta_1, \beta_2, \beta_3, \beta_4$ are the transmission rates for susceptible individuals with asymptomatic infective, susceptible individuals with symptomatic infective, susceptible individuals with treated infective, and susceptible individuals with AIDS respectively, α is the Saturation term to the susceptible individual. μ is the natural mortality rate unrelated to AIDS, σ is rate at which treated individual develops to full blown AIDS, ω is the rate at which AIDS patients get treatment, δ_1 is rate of movement from asymptomatic class I_1 (t) to full blown AIDS A (t). δ_2 is rate of movement from symptomatic class I_2 (t) to treated and AIDS class respectively, n is the fraction of symptomatic infective that moved to treated class, d is the AIDS related death rate/Disease related death rate and θ is the screening rate.

2.1 Positivity and Boundedness of Solutions of the Model

We can show from model (1) that the state variables are non-negative and the solutions remain positive for all time $t \geq 0$. Here, the parameters in the model are assumed to be positive. We equally show that the feasible solutions are bounded in a region $\Omega: \Omega = \left\{ (S, I_1, I_2, T, A) \in \Omega_+^5 : N(t) \leq \frac{\pi}{\mu} \right\}$ at any time corresponding to the system (1)

Theorem 2.1: Let the initial values of the state variables be such that

$$\left\{ (S(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, T(0) \geq 0, A(0) \geq 0) \in \Omega_+^5 \right\}$$

then, the set $\{S(t), I_1(t), I_2(t), T(t), A(t), \quad and \quad N(t)\}$ is non-negative in Ω for all time $t \geq 0$.

Proof: Considering the first equation of system (1) are considered for the positivity of the state variables as follows;

Using the approach of [25, 26], we let

$$\lambda = \frac{\beta_1 I_1 S}{(1+\alpha_1 S)} + \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} + \frac{\beta_3 TS}{(1+\alpha_1 S)} + \frac{\beta_4 AS}{(1+\alpha_1 S)} \tag{2}$$

Then,

$$\frac{dS}{dt} \geq -(\lambda + \mu)S$$

$$\int \frac{1}{S} dS \geq -\int (\lambda + \mu) dt$$

$$S \geq S_0 e^{-(\lambda+\mu)t} \geq 0$$

Hence, $S \geq 0$ (3)

Also considering the second equation in (1)

$$\frac{dI_1}{dt} = \frac{\beta_1 I_1 S}{(1+\alpha_1 S)} + \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} + \frac{\beta_3 TS}{(1+\alpha_1 S)} + \frac{\beta_4 AS}{(1+\alpha_1 S)} - (\theta + \mu + \delta_1) I_1$$

$$\frac{dI_1}{dt} \geq -(\theta + \mu + \delta_1) I_1$$

$$\int \frac{1}{I_1} dI_1 \geq \int -(\theta + \mu + \delta_1) dt$$

$$I_1 \geq I_{1_0} e^{-(\theta+\mu+\delta_1)t} \geq 0$$

Hence, $I_1 \geq I_{1_0}$ (4)

We can proceed in a similar approach to proof the rest of the positivity of solutions of other state variables of I_2, T, A.

Theorem 2.2: Every solution in the region

$\Omega = \left\{(S(t), I_1(t), I_2(t), T(t), A(t) \in \Re_+^5, : S(t) + I_1(t) + I_2(t) + T(t) + A(t) \leq N(t)\right\}$ is positively invariant with respect to the HIV/AIDS model (1) in population. The solutions for the system in (1) are contained and remain in the region Ω for all time $t \geq 0$.

Proof: Considering the equation of the model (1), and taking the derivative with respect to time t of N we obtained

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

$$\Rightarrow \frac{dN}{dt} \leq \pi - \mu N(t)$$

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

where N_0 is the initial size of the population,

$$\text{Therefore, } \lim_{t \to \infty} N(t) \leq \frac{\pi}{\mu} \tag{5}$$

Using this result together with theorem 1 and model (1), we have $0 \leq N(t) \leq \frac{\pi}{\mu}$. This result implies that HIV/AIDS model (1) has non-negative and bounded solutions 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.

3 Mathematical Analysis of the Model

3.1 Disease free equilibrium point

This is the equilibrium point at which population remains in the absence of disease. In this case, no strain of the disease is present in the entire population.

At the equilibrium,

$$\frac{dS}{dt} = \frac{dI_1}{dt} = \frac{dI_2}{dt} = \frac{dT}{dt} = \frac{dA}{dt} = 0 \tag{6}$$

Equation (1) becomes

$$\pi - \frac{\beta_1 I_1 S}{(1+\alpha_1 S)} - \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} - \frac{\beta_3 T S}{(1+\alpha_1 S)} - \frac{\beta_4 A S}{(1+\alpha_1 S)} - \mu S = 0 \tag{7}$$

$$\frac{\beta_1 I_1 S}{(1+\alpha_1 S)} + \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} + \frac{\beta_3 T S}{(1+\alpha_1 S)} + \frac{\beta_4 A S}{(1+\alpha_1 S)} - (\theta + \mu + \delta_1) I_1 = 0 \tag{8}$$

$$\theta I_1 - (\mu + \delta_2) I_2 = 0 \tag{9}$$

$$n\delta_2 I_2 - (\sigma + \mu) T + \omega A = 0 \tag{10}$$

$$(1-n)\delta_2 I_2 + \delta_1 I_1 + \sigma T - (\mu + d + \omega) A = 0 \tag{11}$$

At disease free,

$S \neq 0, I_1 = I_2 = T = A = 0$ Substituting these to equations (7), (8), (9), (10) and (11), and solving gives the infection – free equilibrium as:

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

3.2 Endemic equilibrium point

The endemic equilibrium state is the state where the disease cannot be totally eradicated but remains in the population. For the endemic equilibrium, $S \neq 0, I_1 \neq 0, I_2 \neq 0, T \neq 0, A \neq 0$

Solving equations (7)-(11) 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;

$$E^* = \left(S^*, I_1^*, I_2^*, T^*, A^*\right) = \left\{ \begin{array}{l} \dfrac{\pi}{R_0 I_1 + \mu} \\ \dfrac{\pi R_0 - \mu K_1}{K_1 R_0}, \\ \dfrac{\theta I_1}{K_2}, \\ \dfrac{\left(\delta_2\theta\left(\eta\left(\mu K_4 + \sigma(\mu + d)\right) + \omega\left((1-n)+\sigma\right)\right) + \omega K_2 K_3 \delta_1\right) I_1}{K_2\left(\mu K_4 + \sigma(\mu + d)\right)}, \\ \dfrac{\left(\delta_2\theta\left((1-n)+\sigma\right) + K_2 K_3 \delta_1\right) I_1}{K_2\left(\mu G_4 + \sigma(\mu + d)\right)} \end{array} \right\} \tag{12}$$

respectively, where,

$K_1 = (\theta + \mu + \delta), K_2 = (\mu + \delta_2), K_3 = (\sigma + \mu), K_4 = (\mu + d + \omega)$

3.3 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 effective number of secondary infections caused by infected individual during his entire period of infectiousness [26]. To determine the next generation matrix for the model, the following are considered:

1. The number of ways that new infections can arise or be created;
2. The number of ways that infections can be transferred between compartments. Thus, the I_1, I_2, T, A compartment of system (1).

Then F_i and V_i are computed as follows using the approach of [21]:

$$F_i = \begin{pmatrix} \dfrac{\beta_1 S I_1}{(1+\alpha_1 S)} + \dfrac{\beta_2 S I_2}{(1+\alpha_1 S)} + \dfrac{\beta_3 S T}{(1+\alpha_1 S)} + \dfrac{\beta_4 S A}{(1+\alpha_1 S)} \\ 0 \\ 0 \\ 0 \end{pmatrix}, \; V_i = \begin{pmatrix} (\theta + \mu + \delta_1) I_1 \\ -\theta I_1 + (\mu + \delta_2) I_2 \\ -n\delta_2 I_2 + (\sigma + \mu) T - \omega A \\ -(1-n)\delta_2 I_2 - \delta_1 I_1 - \sigma T + (\mu + d + \omega) A \end{pmatrix} \tag{13}$$

Let G be the next generation matrix. It comprises of two parts F and V^{-1} where

$$F = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right] = \begin{pmatrix} \dfrac{\beta_1\pi}{(\mu+\alpha_1\pi)} & \dfrac{\beta_2\pi}{(\mu+\alpha_1\pi)} & \dfrac{\beta_3\pi}{(\mu+\alpha_1\pi)} & \dfrac{\beta_4\pi}{(\mu+\alpha_1\pi)} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{14}$$

$$V = \left[\frac{\partial V_1(x_0)}{\partial x_j}\right] = \begin{pmatrix} \theta+\mu+\delta_1 & 0 & 0 & 0 \\ -\theta & (\mu+\delta_2) & 0 & 0 \\ 0 & -n\delta_2 & \sigma+\mu & -\omega \\ -\delta_1 & -(1-n)\delta_2 & -\sigma & \mu+d+\omega \end{pmatrix} \quad (15)$$

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

$$R_0 = \frac{\pi\left(\left(\beta_1 K_2 + \theta\beta_2\right)\left(\mu K_4 + \sigma\left(\mu+d\right)\right) + \beta_3\left(\delta_2\theta\left(\omega + n\left(\mu+d\right)\right) + \omega\delta_1 K_2\right) + \beta_4\left(\delta_2\theta\left(\sigma + \mu\left(1-n\right)\right) + \delta_1 K_2 K_3\right)\right)}{K_1 K_2\left(\mu K_4 + \sigma\left(\mu+d\right)\right)} \quad (16)$$

3.4 Computing Numerical Sensitivity

The [26,37] normalized forward sensitivity index approach which defines a variable "P" that depends on a differentiable parameter "q" is defined as

$$X_q^p := \frac{\partial p}{\partial q} * \frac{q}{p} \quad (17)$$

As we have an explicit formula for R_0 in equation (17), we derive an analytical expression for the sensitivity of R_0, as $X_q^p := \frac{\partial p}{\partial q} * \frac{q}{p}$ with respect to each of the parameters involved in R_0 as follows:

Table 3.1: Numerical values of sensitivity indices of R_0

Parameter	Value
β_1	+ 0.847905341
θ	+ 0.0102753420
μ	- 0.4949636613
ω	+0.0768207412
α_1	--0.7142857144
δ_1	-0.7712241608
σ	-0.0037923097
d	-0.08936914870

β_2	+0.0049296821
β_3	+0.0088462045
β_4	+ 0.0143357909
π	+0.2857142856
d	-0.0893691487
θ	+0.0102753420
δ_2	-0.2692217346

3.4.1 Interpretation of Sensitivity Indices

The Table 3.1 represents the sensitivity index for the base line parameter values and it shows that when the parameters $\beta_1, \pi, \omega, \beta_4, \theta, \beta_3$ and β_2 increase while the other parameters remain constant, the value of R_0 will also increase implying that they increase the endemicity of the disease as they have positive indices and should be targeted by intervention strategies.

More so, when the parameters $\delta_1, \alpha_1, \mu, \delta_2, d,$ and σ increase while keeping other parameters constant, the value of R_0 will decrease, implying that they decrease the endemicity of the disease as they have negative indices and should equally be targeted by intervention strategies. From the table, it was revealed that the most sensitive parameter is the transmission rates for the susceptible individuals with asymptomatic infective (β_1) followed by rate of movement from asymptomatic class to AIDS class (δ_1) when we take absolute values of the sensitivity index. For instance, $X_{\beta_1}^{R_0} = +0.8479053410$ means that increasing or decreasing β_1 by 10% increases or (decreases) R_0 by 8.479053410% while $X_{\delta_2}^{R_0} = -0.2692217346$ means that increasing or (decreasing) δ_2 by 10% decreases (or increases) R_0 by 2.692217346%. we can easily check for the other parameters following similar trend.

3.5 Stability analysis of the disease free equilibrium

3.5.1 Local Stability

The Nonlinear equation (1) needs to be linearized to obtain a simpler description that approximates the true system near the equilibrium point. The following conditions are said to be strictly adhered to when linearizing.

(1) The constant terms and the product term must be neglected (Nonlinear Terms)
(2) The higher order terms must as well be neglected

The system of equation (1) was linearized about ($S^0, I_1^0, I_2^0, T^0, A^0$), by setting

$$\left.\begin{aligned} S &= S^0 + x \\ I_1 &= I_1^0 + y \\ I_2 &= I_2^0 + z \\ T &= T^0 + m \\ A &= A^0 + p \end{aligned}\right\} \tag{18}$$

where at DFE we have $S^0, I_1^0, I_2^0, T^0, A^0$.

Then,

$$\left.\frac{dx}{dt} = \frac{dS}{dt}, \frac{dy}{dt} = \frac{dI_1}{dt}, \frac{dz}{dt} = \frac{dI_2}{dt}, \frac{dm}{dt} = \frac{dT}{dt}, \frac{dp}{dt} = \frac{dA}{dt}\right\} \tag{19}$$

Substituting (18) and (19) into system (1) yields

$$\begin{aligned} \frac{dx}{dt} &= \pi - \frac{\beta_1(I_1^0 + y)(S^0 + x)}{1 + \alpha_1(S^0 + x)} - \frac{\beta_2(I_2^0 + z)(S^0 + x)}{1 + \alpha_1(S^0 + x)} - \frac{\beta_3(T^0 + m)(S^0 + x)}{1 + \alpha_1(S^0 + x)} - \frac{\beta_4(A^0 + p)(S^0 + x)}{1 + \alpha_1(S^0 + x)} \\ &- \mu(S^0 + x) \\ \frac{dy}{dt} &= \frac{\beta_1(I_1^0 + y)(S^0 + x)}{1 + \alpha_1(S^0 + x)} + \frac{\beta_2(I_2^0 + z)(S^0 + x)}{1 + \alpha_1(S^0 + x)} + \frac{\beta_3(T^0 + m)(S^0 + x)}{1 + \alpha_1(S^0 + x)} + \frac{\beta_1(A^0 + p)(S^0 + x)}{1 + \alpha_1(S^0 + x)} \\ &- (\theta + \mu + \delta_1)(I_1^0 + y) \\ \frac{dz}{dt} &= \theta(I_1^0 + y) - (\mu + \delta_2)(I_2^0 + z) \\ \frac{dm}{dt} &= n\delta_2(I_2^0 + z) - (\sigma + \mu)(T^0 + m) + \omega(A^0 + p) \\ \frac{dp}{dt} &= (1 - n)\delta_2(I_2^0 + z) + \delta_1(I_1^0 + y) - (\mu + d + \omega)(A^0 + p) + \sigma(T^0 + m) \end{aligned} \tag{20}$$

we expand and separate the linear terms and neglect the higher order terms and other nonlinear to obtain,

$$\left.\begin{aligned}
\frac{dx}{dt} &= \left(\begin{matrix} 2\beta_1 I_1^0 S^0\alpha_1 - \beta_1 I_1^0 + 2\beta_2 I_2^0 S^0\alpha_1 - \beta_2 I_2^0 + 2\beta_3 T^0 S^0\alpha_1 - \beta_3 T^0 + 2\beta_4 T^0 S^0\alpha_1 \\ -\beta_4 A^0 - \mu \end{matrix}\right)x - \\
&\left(\beta_1 S^0\right)y - \left(\beta_2 S^0\right)z - \left(\beta_3 S^0\right)m - \left(\beta_4 S^0\right)p + Nonlinear terms \\
\frac{dy}{dt} &= \left(\begin{matrix} \beta_1 I_1^0 - 2\beta_1 I_1^0 S^0\alpha_1 - 2\beta_2 I_2^0\alpha_1 S^0 + \beta_2 I_2^0 + \beta_3 T^0 S^0\alpha_1 + \beta_3 T^0 - \beta_3 T^0 S^0\alpha_1 - \beta_4 A^0 S^0\alpha_1 + \\ \beta_4 A^0 - \beta_4 A^0 S^0\alpha_1 \end{matrix}\right)x + \\
&\left(\beta_1 S^0 - (\theta+\mu+\delta_1)\right)y + \left(\beta_2 S^0\right)z + \left(\beta_3 S^0\right)m + \left(\beta_4 S^0\right)p + Nonlinear terms \\
\frac{dz}{dt} &= \theta y - (\mu+\delta_2)z \\
\frac{dm}{dt} &= n\delta_2 z - (\sigma+\mu)m + \omega p \\
\frac{dp}{dt} &= \delta_1 y + ((1-n)\delta_2)z + \sigma m - (\mu+d+\omega)p
\end{aligned}\right\} \quad (21)$$

Theorem 3.1

Let $(K_1+K_2+K_3+K_4) > \dfrac{\beta_1\pi}{\mu}$,

$$\left(K_3(K_1+K_2+K_4) + K_4(K_1+K_2) + K_2K_1\right) > \left(\frac{\beta_1(K_2+K_3+K_4)+\beta_2\theta}{\mu}\right),$$

$$\left(K_4(K_1+K_2)+K_1K_2)K_3 + K_4K_2K_1\right)\mu > \frac{\pi}{\mu}\left[\begin{matrix}\beta_1\left((K_2+K_4)K_3 + K_2K_4\right) + \beta_2\theta(K_3+K_4) + \\ \delta_2\theta\left(\beta_4(1-n)+\beta_3 n\right)\end{matrix}\right],$$

$$K_4K_3K_2K_1\left(1-R_0\right) > 0$$

Then the disease free equilibrium is locally asymptotically stable.

Proof:

The Jacobian matrix of the above linearized equation (21) at the disease free equilibrium $\left(\dfrac{\pi}{\mu},0,0,0,0\right)$ is

$$E^0 = \begin{pmatrix} -\mu & -\beta_1 q & -\beta_2 q & -\beta_3 q & -\beta_4 q \\ 0 & \beta_1 q - K_1 & \beta_2 q & \beta_3 q & \beta_4 q \\ 0 & \theta & -K_2 & 0 & 0 \\ 0 & 0 & n\delta_2 & -K_3 & 0 \\ 0 & 0 & (1-n)\delta_2 & \sigma & -K_4 \end{pmatrix} \quad (22)$$

where,

$$q = \frac{\pi}{(\mu+\alpha_1\pi)}$$
$$K_1 = (\theta+\mu+\delta_1)$$
$$K_2 = (\mu+\delta_2)$$

$$K_3 = (\sigma + \mu)$$
$$K_4 = (\mu + d + \omega)$$

The characteristic equation is obtained as

$$(\lambda + \mu)\left(P_0\lambda^4 + P_1\lambda^3 + P_2\lambda^2 + P_3\lambda + P_4\right) = 0 \tag{23}$$

Clearly, $\lambda = -\mu$

and,

$$P_0 = 1,$$

$$P_1 = (K_1 + K_2 + K_3 + K_4) - \frac{\beta_1\pi}{\mu},$$

$$P_2 = \left(K_3(K_1 + K_2 + K_4) + K_4(K_1 + K_2) + K_2K_1\right) - \left(\frac{\beta_1(K_2 + K_3 + K_4) + \beta_2\theta}{\mu}\right),$$

$$P_3 = \left(K_4(K_1 + K_2) + K_1K_2)K_3 + K_4K_2K_1\right) - \frac{\pi}{\mu}\left[\begin{array}{l}\beta_1\left((K_2 + K_4)K_3 + K_2K_4\right) + \\ \beta_2\theta(K_3 + K_4) + \delta_2\theta\left(\beta_4(1-n) + \beta_3 n\right)\end{array}\right]$$

$$P_4 = K_4K_3K_2K_1\left(1 - R_0\right) > 0$$

$$P_0 > 0, P_1 > 0, P_2 > 0, P_3 > 0, P_4 > 0 \Rightarrow R_0 > 1,$$

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

3.5.2 Stability of the Endemic equilibrium

The stability of the endemic equilibrium was carried out by linearizing (1) about $\left(S^*, I_1^*, I_2^*, T^*, A^*\right)$ Keeping in mind the conditions guiding linearization

Let

$$\left.\begin{array}{l} S = S^* + w \\ I_1 = I_1^* + f \\ I_2 = I_2^* + g \\ T = T^* + h \\ A = A^* + q \end{array}\right\} \tag{24}$$

where $S^*, I_1^*, I_2^*, T^*, A^*$ are the endemic equilibrium

$$\left.\frac{dS}{dt} = \frac{dw}{dt}, \frac{dI_1}{dt} = \frac{df}{dt}, \frac{dI_2}{dt} = \frac{dg}{dt}, \frac{dT}{dt} = \frac{dh}{dt}, \frac{dA}{dt} = \frac{dq}{dt}\right\} \tag{25}$$

Substituting (24) and (25) into (1) gives

$$\frac{dw}{dt}=\pi-\frac{\beta_1(f+I_1^*)(w+S^*)}{1+\alpha_1(w+S^*)}-\frac{\beta_2(g+I_2)(w+S^*)}{1+\alpha_1(w+S^*)}-\frac{\beta_3(h+T^*)(w+S^*)}{1+\alpha_1(w+S^*)}-\frac{\beta_4(q+A^*)(w+S^*)}{1+\alpha_1(w+S^*)}$$

$$-\mu(w+S^*)$$

$$\Rightarrow\frac{dw}{dt}=\pi-\beta_1(f+I_1^*)(w+S^*)\left[1+\alpha_1(w+S^*)\right]^{-1}-\beta_2(g+I_2^*)(w+S^*)\left[1+\alpha_1(w+S^*)\right]^{-1}$$

$$-\beta_3(h+T^*)(w+S^*)\left[1+\alpha_1(w+S^*)\right]^{-1}-\beta_4(q+A^*)(w+s^*)\left[1+\alpha_1((w+S^*)\right]^{-1}-\mu(w+S^*)$$

Following binomial expansion and neglecting all higher order terms and nonlinear terms to obtain

$$\frac{dw}{dt}=\begin{pmatrix}\beta_1I_1^*\alpha_1S^*+\beta_1I_1^*S^*\alpha_1-\beta_2I_2^*+2\beta_2I_2^*\alpha_1S^*-\beta_1I_1^*+\beta_3T^*\alpha_1S^*-\\ \beta_3T^*+\beta_3T^*S^*\alpha_1-\beta_4A^*+2\beta_4A^*\alpha_1S^*-\mu\end{pmatrix}w+ \tag{26}$$

$$\left(-\beta_1S^*\right)f+\left(-\beta_2S^*\right)g+\left(-\beta_3S^*\right)h-\left(\beta_4S^*\right)q+Nonlinearterms$$

And also,

$$\frac{df}{dt}=\begin{pmatrix}\beta_1I_1^*-\beta_1I_1^*\alpha_1S^*+\beta_4A^*-2\beta_4A^*\alpha_1S^*+\beta_2I_2^*-2\beta_2I_2^*\alpha_1S^*\\ +\beta_3T^*-\beta_3T^*\alpha_1S^*-2\beta_3T^*\alpha_1S^*\end{pmatrix}w+ \tag{27}$$

$$\left(\beta_1S^*-(\theta+\mu+\delta_1)\right)f+\left(\beta_2S^*\right)g+\left(\beta_3S^*\right)h+\left(\beta_4S^*\right)q+\text{Nonlinear Terms}$$

Also,

$$\frac{dg}{dt}=\theta f-(\mu+\delta_2)g \tag{28}$$

Also,

$$\frac{dh}{dt}=n\delta_2g-(\sigma+\mu)h+\omega q \tag{29}$$

Likewise,

$$\frac{dq}{dt}=\delta_1f+((1-n)\delta_2)g+\sigma h-(\mu+d+\omega)q \tag{30}$$

Therefore, the linearized equations are:

$$\frac{dw}{dt}=\begin{pmatrix}\beta_1I_1^*\alpha_1S^*+\beta_1I_1^*S^*\alpha_1-\beta_2I_2^*+2\beta_2I_2^*\alpha_1S^*-\beta_1I_1^*+\beta_3T^*\alpha_1S^*-\\ \beta_3T^*+\beta_3T^*S^*\alpha_1-\beta_4A^*+2\beta_4A^*\alpha_1S^*-\mu\end{pmatrix}w+ \tag{31}$$

$$\left(-\beta_1S^*\right)f+\left(-\beta_2S^*\right)g+\left(-\beta_3S^*\right)h-\left(\beta_4S^*\right)q+Nonlinearterms$$

$$\frac{df}{dt}=\left(\beta_1I_1^*-\beta_1I_1^*\alpha_1S^*+\beta_4A^*-2\beta_4A^*\alpha_1S^*+\beta_2I_2^*-2\beta_2I_2^*\alpha_1S^*+\beta_3T^*-\beta_3T^*\alpha_1S^*-2\beta_3T^*\alpha_1S^*\right)w \tag{32}$$

$$+\left(\beta_1S^*-(\theta+\mu+\delta_1)\right)f+\left(\beta_2S^*\right)g+\left(\beta_3S^*\right)h+\left(\beta_4S^*\right)q+\text{Nonlinear Terms}$$

$$\frac{dg}{dt}=\theta f-(\mu+\delta_2)g \tag{33}$$

$$\frac{dh}{dt}=n\delta_2g-(\sigma+\mu)h+\omega q \tag{34}$$

$$\frac{dq}{dt} = \delta_1 f + \left((1-n)\delta_2\right) g + \sigma h - (\mu + d + \omega) q \tag{35}$$

Theorem 3.2: Let $K_1 + K_2 + K_3 + K_4 + A > \beta_1 S$,

$$(K_1 + K_2)(A + K_3 + K_4) + A(K_4 + K_3)K_1K_2 + \mu K_4 + \sigma(\mu + d) + \beta SB > \beta S(A + K_4 + K_3 + K_2)$$

$$\begin{pmatrix} B\beta_2\theta + (K_4 + K_3)((K_1 + K_2)A + K_1K_2 + \beta_1 B) + (\mu K_4 + \sigma(\mu + d))(A + (K_1 + K_2)) \\ + AK_1K_2 \end{pmatrix} >$$

$$S\begin{pmatrix} \beta_1(\mu K_4 + \sigma(\mu + d)) + BK_2 + (\beta_1 A + \beta_2\theta + K_2)(K_3 + K_4) + (\beta_2\theta + K_2)A + \\ \delta_2\theta(\beta_3 n + \beta_4(1-n)) \end{pmatrix}$$

$$\begin{pmatrix} (\mu K_4 + \sigma(\mu + d))((K_1 + K_2)A + K_1K_2)AK_1K_2(K_3 + K_4) + \\ BS((\beta_1 + \beta_2\theta)K_2(K_3 + K_4) + \beta_1(\mu K_4 + \sigma(\mu + d)) + +(\beta_3 n + \beta_4(1-n))\delta_2\theta) \end{pmatrix} >$$

$$S\begin{Bmatrix} \beta_1[(\mu K_4 + \sigma(\mu + d))(K_2 + A) + K_2(K_3 + K_4)A] \\ \beta_2\theta((K_3 + K_4)(A) + (\mu K_4 + \sigma(\mu + d))) + \beta_3(n(A + K_4) + (1-n)\omega)\theta\delta_2 + \\ \beta_4((1-n)A + \delta_2\theta[(1-n)K_3 + n\theta]) \end{Bmatrix} \quad \text{and}$$

$$K_1K_2(\mu K_4 + \sigma(\mu + d))(A + R_0 B)S + \beta_3 B(1-n)\omega\theta S > (K_1K_2(\mu K_4 + \sigma(\mu + d))R_0 + \beta_3(1-n)\omega\theta)AS$$

Then the endemic equilibrium point $\left(S^*, I_1^*, I_2^*, T^*, A^*\right)$ is locally asymptotically stable.

Proof:

Equations (31)-(35) can be expressed in matrix form as

$$\begin{pmatrix} w' \\ f' \\ g' \\ h' \\ q' \end{pmatrix} = \begin{pmatrix} A & -\beta_1 S^* & -\beta_2 S^* & -\beta_3 S^* & -\beta_4 S^* \\ B & -\beta_1 S^* - (\theta + \mu + \delta_1) & \beta_2 S^* & \beta_3 S^* & \beta_4 S^* \\ 0 & \theta & -(\mu + \delta_2) & 0 & 0 \\ 0 & 0 & n\delta_2 & -(\sigma + \mu) & \omega \\ 0 & \delta_1 & (1-n)\delta_2 & \sigma & -(\mu + d + \omega) \end{pmatrix} \begin{pmatrix} w \\ f \\ g \\ g \\ q \end{pmatrix} + Nonlinearterms \tag{36}$$

where,

$$A = \begin{pmatrix} \beta_1 I_1^* \alpha_1 S^* + \beta_1 I_1^* S^* \alpha_1 - \beta_2 I_2^* + 2\beta_2 I_2^* \alpha_1 S^* - \beta_1 I_1^* + \beta_3 T^* \alpha_1 S^* - \beta_3 T^* + \beta_3 T^* S^* \alpha_1 \\ -\beta_4 A^* + 2\beta_4 A^* \alpha_1 S^* - \mu \end{pmatrix}$$

$$B = \left(\beta_1 I_1^* - \beta_1 I_1^* \alpha_1 S^* + \beta_4 A^* - 2\beta_4 A^* \alpha_1 S^* + \beta_2 I_2^* - 2\beta_2 I_2^* \alpha_1 S^* + \beta_3 T^* - \beta_3 T^* \alpha_1 S^* - 2\beta_3 T^* \alpha_1 S^*\right)$$

The Jacobian matrix of the equation (31)-(35), at endemic equilibrium point $\left(S^*, I_1^*, I_2^*, T^*, A^*\right)$

Let $C^* = \begin{pmatrix} A & -\beta_1 S^* & \left(-\beta_2 S^*\right) & -\beta_3 S^* & -\beta_4 S^* \\ B & \left(\beta_1 S^* - (\theta + \mu + \delta_1)\right) & \beta_2 S^* & \beta_3 S^* & \beta_4 S^* \\ 0 & \theta & -(\mu + \delta_2) & 0 & 0 \\ 0 & 0 & n\delta_2 & -(\sigma + \mu) & \omega \\ 0 & \delta_1 & \left((1-n)\delta_2\right) & \sigma & -(\mu + d + \omega) \end{pmatrix}$ (37)

Therefore the characteristic equation, $\left|C^* - \lambda I\right| = 0 \Rightarrow$

$$\begin{vmatrix} A-\lambda & -\beta_1 S^* & -\beta_2 S^* & -\beta_3 S^* & -\beta_4 S^* \\ 0 & \left(\beta_1 S^* - (\theta+\mu+\delta_1)\right)-\lambda & \beta_2 S^* & \beta_3 S^* & \beta_4 S^* \\ 0 & \theta & -(\mu+\delta_2)-\lambda & 0 & 0 \\ 0 & 0 & n\delta_2 & -(\sigma+\mu)-\lambda & \omega \\ 0 & \delta_1 & (1-n)\delta_2 & \sigma & -(\mu+d+\omega)-\lambda \end{vmatrix} \quad (38)$$

Evaluating for λ in (38) yields the characteristic equation

$$\lambda^3 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5 = 0 \quad (39)$$

where.

$$a_0 = 1$$

$$a_1 = K_1 + K_2 + K_3 + K_4 + A - \beta_1 S$$

$$a_2 = (K_1+K_2)(A+K_3+K_4) + A^*(K_4+K_3)K_1K_2 + \mu K_4 + \sigma(\mu+d) - \beta S(A+K_4+K_3+K_2-B)$$

$$a_3 = \beta_1\left[\{-(K_3+K_4+K_2)A - (K_4-B+K_2)K_3 + (K_2-B)K_4 - BK_2 - \sigma\omega\}\right]S$$
$$-\beta_2\{\theta[(A-B)+K_3+K_4]\} - \beta_3 n\delta_2\theta S - \beta_4\delta\theta(1-n)S + (K_4+K_3)[(K_1+K_2)A + K_1K_2]$$
$$+(\mu K_4 + \mu(\mu+d))[A+(K_1+K_2)]K_1K_2A$$

$$a_4 = (\mu K_4 + \sigma(\mu+d))((K_1+K_2)A + K_1K_2)AK_1K_2(K_3+K_4) + BS\begin{pmatrix}(\beta_1+\beta_2\theta)K_2(K_3+K_4)+\\ \beta_1(\mu K_4+\sigma(\mu+d))+\\ +(\beta_3 n+\beta_4(1-n))\delta_2\theta\end{pmatrix}$$

$$-S\begin{Bmatrix}\beta_1[(\mu K_4+\sigma(\mu+d))(K_2+A)+K_2(K_3+K_4)A]\\ \beta_2\theta((K_3+K_4)(A)+(\mu K_4+\sigma(\mu+d)))+\beta_3(n(A+K_4)+(1-n)\omega)\theta\delta_2+\\ \beta_4((1-n)A+\delta_2\theta[(1-n)K_3+n\theta])\end{Bmatrix}$$

$$a_5 = K_1K_2(\mu K_4+\sigma(\mu+d))(A+R_0B)S + \beta_3 B(1-n)\omega\theta S -$$
$$(K_1K_2(\mu K_4+\sigma(\mu+d))R_0 + \beta_3(1-n)\omega\theta)AS$$

Then the endemic equilibrium point $\left(S^*, I_1^*, I_2^*, T^*, A^*\right)$ is locally asymptotically stable, it follows from Descartes rule of signs, that equation (39) have no change in sign meaning there are no positive roots. Therefore, since all eigenvalues are negative then the endemic or disease equilibrium is locally asymptotically stable. Then the endemic equilibrium point $\left(S^*, I_1^*, I_2^*, T^*, A^*\right)$ is locally asymptotically stable.

3.6 Existence of backward bifurcation

The existence of backward bifurcation is explored using the centre manifold made popular by [28]. This has widely been used in the study of some epidemiological models [27,29-32,34-36].

For understanding and convenience, we consider the following change of variables

Let $S = x_1, I_1 = x_2, I_3 = x_3, T = x_4,$ and $A = x_4$ Also, Let $x = (x_1, x_2, x_3, x_4, x_5)^T$, so the system (2) can be re-written in the form $\frac{dx}{dt} = F(x),$ where $F = (f_1, f_2, f_3, f_4, f_5)^T$, as follows, then the model equations becomes

$$\begin{aligned}
\frac{dx_1}{dt} &= \pi - \frac{\beta_1 x_2 x_1}{(1+\alpha_1 x_1)} - \frac{\beta_2 x_3 x_1}{(1+\alpha_1 x_1)} - \frac{\beta_3 x_4 x_1}{(1+\alpha_1 x_1)} - \frac{\beta_3 x_5 x_1}{(1+\alpha_1 x_1)} - \mu x_1 = 0 \\
\frac{dx_2}{dt} &= \frac{\beta_1 x_2 x_1}{(1+\alpha_1 x_1)} + \frac{\beta_2 x_3 x_1}{(1+\alpha_1 x_1)} + \frac{\beta_3 x_4 x_1}{(1+\alpha_1 x_1)} + \frac{\beta_4 x_5 x_1}{(1+\alpha_1 x_1)} - (\theta + \mu + \delta_1) x_2 = 0 \\
\frac{dx_3}{dt} &= \theta x_2 - (\mu + \delta_2) x_3 = 0 \\
\frac{dx_4}{dt} &= n\delta_2 x_3 - (\sigma + \mu) x_4 + \omega x_5 = 0 \\
\frac{dx_5}{dt} &= (1-n)\delta_2 x_3 + \delta_1 x_2 + \sigma x_4 - (\mu + d + \omega) x_5 = 0
\end{aligned} \tag{40}$$

Taking $\beta_1 = \beta_1^*$, where β_1^* is the chosen bifurcation parameter and the case when $R_0 = 1.$ Then,

$$\beta_1^* = \frac{(K_1 K_2 + \pi\theta\beta_2)(\mu K_4 + \sigma(\mu + d)) - \pi(\beta_3(\delta_2\theta(\omega + n(\mu + d)) + \omega\delta_1 K_2) + \beta_4(\delta_2\theta(\sigma + \mu(1-n)) + \delta_1 K_2 K_3))}{\pi K_2(\mu K_4 + \sigma(\mu + d))}$$

The Jacobian of system (40), evaluated at the disease free equilibrium E_0, and β_1^* is given by

$$J(E_0, \beta_1^*) = \begin{pmatrix} -\mu & -\beta_1 q & -\beta_2 q & -\beta_3 q & -\beta_4 q \\ 0 & \beta_1 q - K_1 & \beta_2 q & \beta_3 q & \beta_4 q \\ 0 & \theta & -K_2 & 0 & 0 \\ 0 & 0 & n\delta_2 & -K_3 & 0 \\ 0 & 0 & (1-n)\delta_2 & \sigma & -K_4 \end{pmatrix} \tag{41}$$

The characteristic equation of the Jacobian matrix

$$(\lambda + \mu)(P_0\lambda^4 + P_1\lambda^3 + P_2\lambda^2 + P_3\lambda + P_4) = 0 \tag{42}$$

where $P_i; i = 0,1,..4 > 0$.

The polynomial equation (41) has a zero eigenvalue when $P_4 = 0$, that is when $(R_0 = 1)$.

$$\lambda(P_0\lambda^3 + P_1\lambda^2 + P_2\lambda^1 + P_3) = 0 \tag{43}$$

such that $\lambda = 0$ is a zero eigenvalue and

$$P_0\lambda^3 + P_1\lambda^2 + P_2\lambda^1 + P_3 = 0 \tag{44}$$

So that all the eigenvalues in (44) will have negative real parts indicating that there exist bifurcation at $R_0 = 1$. Hence, the need for the centre manifold theorem [28], the associated right eigenvector w corresponding to the zero eigenvalue, where $w = (w_1, w_2, w_3, w_4, w_5)^T$, can be obtained from $J(E_0, \beta_1^*)w = 0$. Hence, we have

$$\begin{cases} w_1 = -q\left\{\dfrac{(\beta_1 K_2 + \beta_3\theta)(K_4 - \sigma w) + \delta_1 K_2(\beta_3 w + \beta_4) + \theta\delta_2(n + (1-n)(1-\sigma))(1+\omega)}{\theta(K_4 - \sigma\omega)}\right\}w_3 \\ w_2 = \dfrac{K_2 w_3}{\theta} \\ w_3 = w_3 > 0 \\ w_4 = \dfrac{\theta\delta_2(n + w(1-n+\sigma n) + w\delta_1 K_2)w_3}{K_3\theta(k_4 - \sigma w)} \\ w_5 = \dfrac{(\delta_1 K_2 + \theta\delta_2((1-n)+\sigma n))w_3}{\theta(K_4 - \sigma\omega)} \end{cases}$$

Similarly, the Jacobian, $J(E_0, \beta_1^*)$, has a left eigenvector $v = (v_1, v_2, v_3, v_4, v_5)$ satisfying $v.w = 1$, where

$$\begin{cases} v_1 = 0 \\ v_2 = \dfrac{(K_4 - \sigma\omega)v_5}{q\beta_3(K_3 + \omega)} \\ v_3 = \dfrac{\{(K_4 - \sigma)(n\delta_2\beta_3 - \omega\beta_2) + (K_3 + \omega)(\beta_2 K_4 + \beta_3(1-n)\delta_2)\}v_5}{K_2\beta_3(K_3 + \omega)} \\ v_4 = \dfrac{(k_3(K_4 - \sigma) + \sigma(K_3 + \omega))v_5}{(K_3 + \omega)} \\ v_5 = v_5 > 0 \end{cases}$$

3.6.1 Computation of a and b

The coefficients a and b as defined by Castillo-Chavez and Song [34] are given by:

$a = \sum_{i,j,k=1}^{\infty} v_k w_i w_j \dfrac{\partial^2 f_k(E_0, \beta^*)}{\partial x_i \partial x_j}$ and $b = \sum_{k,i,j=1}^{\infty} v_k w_i \dfrac{\partial^2 f_k(E_0, \beta^*)}{\partial x_i \partial \beta^*}$; and are algebraically computed as follows, considering only the nonzero components $f_i : k = 2,3,4,5.$ With these definitions, the following were obtained:

$$\Rightarrow v_2 w_1 \left\{-2\left(\frac{w_2\beta_1^* + w_3\beta_2 + w_4\beta_3 + w_5\beta_5}{(1+\alpha_1 x_2)^2}\right)\right\}$$

w_1 is non positive, while $(w_2, w_3, w_4, w_5) > 0$, and $v_2 > 0$

On substituting of necessary quantities and simplifying,

$$\Rightarrow a = \left\{ \frac{\begin{array}{c} 2qw_3v_5\left(w_2\beta_1^* + w_3\beta_2 + w_4\beta_3 + w_5\beta_5\right)\left(K_3\left(K_4 - \sigma\right) + \sigma(K_3 + \omega)\right) \\ \left(\beta_1 K_2 + \beta_3\theta\right)\left(K_4 - \sigma\omega\right) + \delta_1 K_2(\beta_3\omega + \beta_4) + \theta\delta_2\left(n + \left(1-n\right)\left(1-\sigma\right)\right)(1+\omega) \end{array}}{q\beta_3\theta\left(K_4 - \sigma\omega\right)\left(K_3 + \omega\right)\left(1 + \alpha_1\frac{\pi}{\mu}\right)} \right\} \quad (45)$$

$$b = qw_3v_5\pi\left(\frac{\begin{array}{c} \left(\beta_1 K_2 + \beta_3\theta\right)\left(K_4 - \sigma\omega\right) + \delta_1 K_2\left(\beta_3\omega + \beta_4\right) + \theta\delta_2(n + (1 - n(1-\sigma))(1+\omega) \\ \left(K_3(K_4 - \sigma) + \sigma(K_3 + \omega)\right) \end{array}}{q\beta_3\theta\left(\mu + \pi\alpha_1\right)\left(K_3 + \omega\right)\left(K_5 - \sigma\omega\right)} \right) \quad (46)$$

Clearly, from (43) and (44), $a > 0, b > 0,$ then the model exhibits a backward bifurcation around $R_0 = 1$, that is there exists bi-stability of equilibrium point (one stable disease free and stable endemic equilibrium for $R_0 < 1.$ This is implying that the disease free is not globally stable. The implication, from epidemiological view point of the existence of backward bifurcation is that the classical necessary requirement of $R_0 < 1$ is insufficient to control the spread of HIV in the population.

4 Numerical simulations and discussion

This section presents the numerical simulation results for the HIV/AIDS model.

The parameters in the model (1) shown in table 4.1 were obtained from literatures published by different researchers and referenced accordingly.

Table 4.1: **parameters, values and source used in the model**

d	1.0	[24]
θ	0.01	[33]
μ	0.02	Assumed
ω	0.1	[33]
n	0.2	Assumed
α_1	0.1	Assumed

δ_1	0.2	[19]
δ_2	0.01	[19]
σ	0.001	[19]
β_1	0.86	[19]
β_2	0.15	[19]
β_3	0.10	[19]
β_4	0.08	Assumed

4.1 Numerical simulation

Simulation of the model was performed for better understanding of dynamical spread of transmission of HIV/AIDS infection using Maple 18 software. The results of the model equations are presented below in form of graphs and are discussed in the figure below to illustrate the changes in the compartments. The screening rate, treatment rate and the saturation terms were checked in order to observe their impact on the numerical spread of the disease using a set of reasonable parameters.

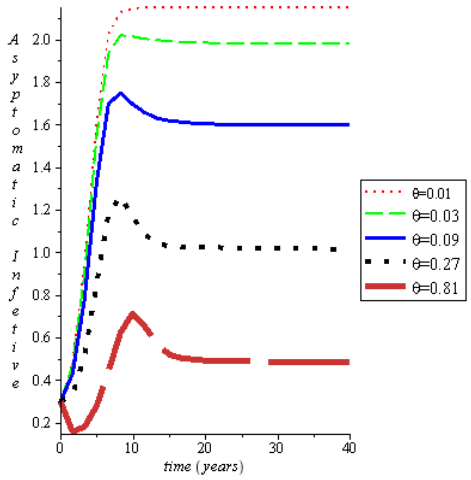

Fig.1 (a)

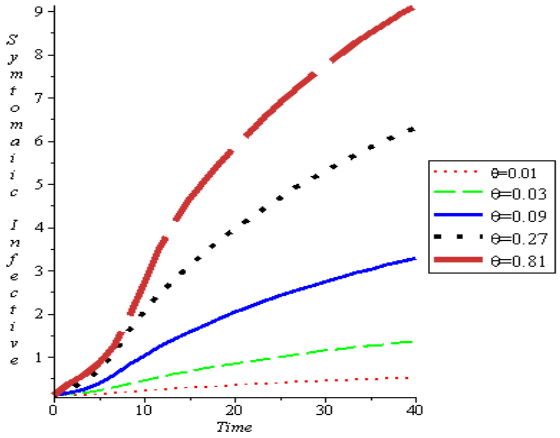

Fig. 1 (b)

Figures 1(a)-1(b) depicts the effect of screening rates (θ) on asymptomatic infective population $I_1(t)$ against time (t) and symptomatic infective $I_2(t)$ population. As the asymptomatic HIV infective population become aware of their status, there is a decrease in the population of asymptomatic infective population which ultimately bring about an increase in the population of

symptomatic infective population as seen in **Figure 1(b)**. It was observed here that there is an increase in the population of symptomatic infective as the screening rate increases. This is simply due to the population of asymptomatic infective that migrated to the symptomatic infective compartment as a result of the awareness gotten.

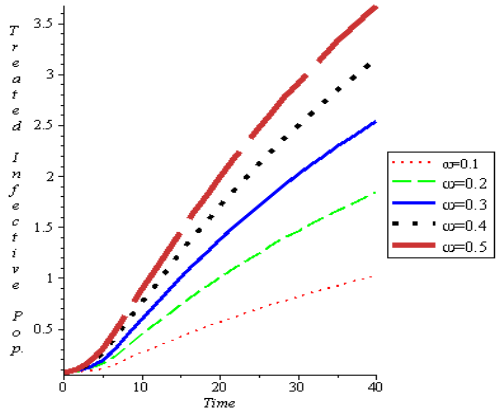

Fig.2 (a)

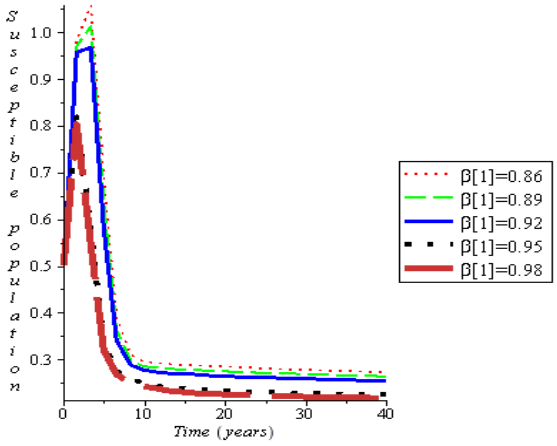

Fig.2 (b)

Figure 2(a) depicts the graph of Treated infective population $T(t)$ against Time (t) for different values of treatment rate and **Figure 2(b)** depicts the graph of susceptible Population against time (t) for different values of transmission rate for susceptible individuals with asymptomatic infective (β_1) The symptomatic infective undergo and submit to treatment with antiretroviral therapy which as a result leads to an increase in the proportion of symptomatic infective population. This therefore result to an increase in the proportion of treated infective populations as shown in **Figure (2a)** while **Figure (2b)** shows that as transmission rate increase, the susceptible population decrease significantly. It is clearly seen that as the transmission parameter values increase in the population, the number of susceptible population also reduce. As long as susceptible population continue to interact with the infective without taking any preventive measures at any of the infective stages, there is going to be a continuous reduction in the susceptible population.

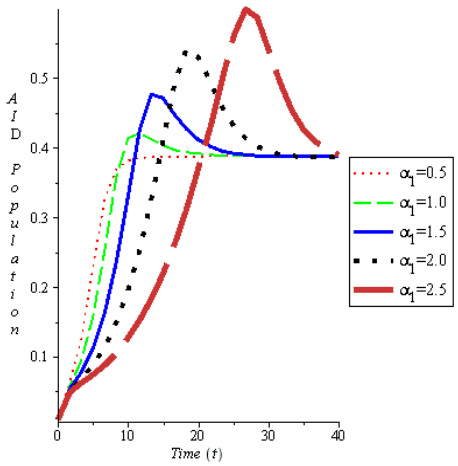

Fig.3 (a)

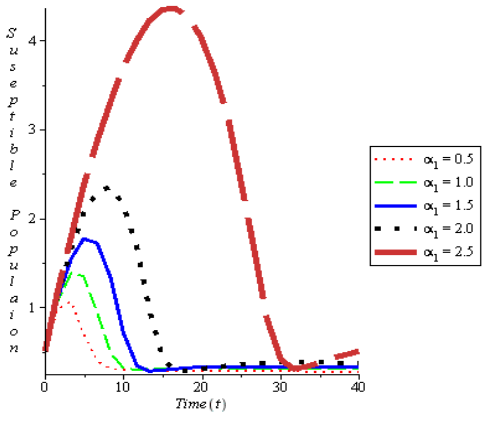

Fig.3 (b)

Fig. (3a) This depicts the graph of Aids population A(t) against time (t) for different values of saturation term (α_1) while **Fig. (3b).** depicts the graph of susceptible population against time for various values of saturation term (α_1). This shows that as the saturation term α_1 increases, the AIDS population decreases. This is simply due to the crowding effects of AIDS individual and other infective as seen in **Figure (3a)** while also, as the saturation term α_1 increases in **Figure (3b)**, the susceptible population increases. This is simply due to the precautionary measures taken by susceptible individual.

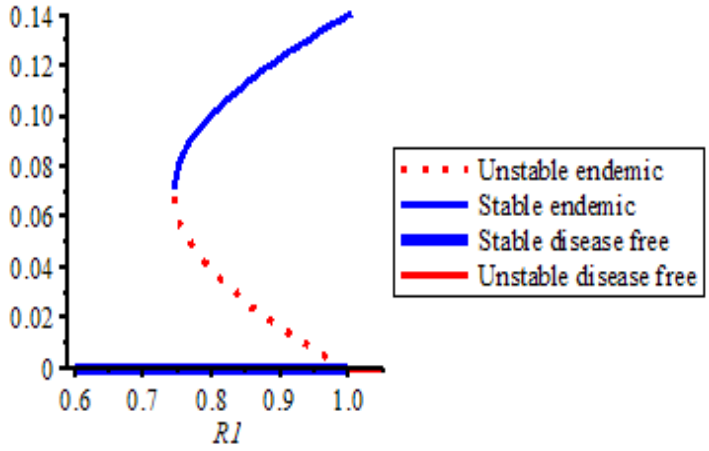

Fig.4. Backward Bifurcation Diagram

The phenomenon of Figure 7 shows that the HIV/AIDS model have multiple endemic equilibrium whenever $R_0 < 1$. In this case, the classical requirement of having $R_0 < 1$, is necessary but no longer sufficient for effective disease elimination.

5 Conclusion

In this paper, we formulated and presented a mathematical model for HIV/AIDS transmission, which incorporated saturated incidence term. It was shown that the system of equation represents a useful mathematical model of a physical system by carrying out a classical qualitative proof of the positivity of solution of the governing system of model equations. More so, the existence of both the disease-free and endemic equilibria were established and analyzed for stability, and it was shown that both equilibria were locally asymptotically stable. This implies that whenever the said conditions are satisfied, the HIV/AIDS infection can be controlled. The result of the sensitivity analysis showed the transmission rates for susceptible individuals with asymptomatic infective β_1, as the most sensitive parameter. The result of the bifurcation analysis showed that the model exhibit a backward bifurcation which indicates that $R_0 < 1$ is no longer sufficient for effective disease control. However, screening and treatment of the infective have a significant effect in reducing the transmission of the disease. Susceptible population also reduce when infective carelessly interact with other infective and also susceptible population increase as saturation increase due to the precautionary measures taken.

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