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Understanding the Transmission Dynamics and Control of HIV Infection: A Mathematical Model Approach

Saheed Ajao[a,*], Isaac Olopade[b], Titilayo Akinwumi[a], Sunday Adewale[c], Adelani Adesanya[a]

[a]*Department of Mathematics and Computer Science, Elizade University, Ilara−mokin, Ondo State. Nigeria*
[b]*Department of Mathematics and Statistics, Federal University Wukari, Wukari, Taraba State. Nigeria.*
[c]*Department of Pure and Applied Mathematics, Ladoke Akintola University of Technology, Ogbomoso, Oyo State. Nigeria.*

Abstract

New challenges like the outbreak of new diseases, government policies, war and insurgency etc. present distortion, delay and denial of persons' access to ART, thereby fuelling the spread and increasing the burden of HIV/AIDS. A mathematical model is presented to study the transmission dynamics and control of HIV infection. The qualitative and quantitative analyses of the model are carried out. It is shown that the disease-free equilibrium of the model is globally asymptotically stable whenever the basic reproduction number is less than unity. It is also shown that a unique endemic equilibrium exists whenever the basic reproduction number exceeds unity and that the model exhibits a forward bifurcation. Furthermore, the Lyapunov function is used to show that the endemic equilibrium is globally asymptotically stable for a special case of the model whenever the associated basic reproduction number is greater than unity. The model is calibrated to the data on HIV/AIDS prevalence in Nigeria from 1990 to 2019 and it represents reality. The numerical simulations on the global stability of disease-free equilibrium and endemic equilibrium justify the analytic results. The fraction of the detected individuals who are receiving treatment and stay in the treatment class plays a significant role as it influences the population of the latently-infected individuals and AIDS class as the treatment prevents the individuals from progressing into the AIDS class.

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1. Introduction

Efficient and effective testing is a gateway to HIV treatment and it is an important element of efforts to stop the AIDS epidemic [1]. A positive diagnosis allows an HIV-infected person to receive antiretroviral therapy (ART) [2]. The ART suppresses the replication of the virus and this prevents transmission to one's sexual partner. Thus, early access to antiretroviral therapy (ART) and support for continued treatment is important not only to improve the health status of HIV-infected individuals but also to prevent the transmission of HIV [3].

In 2021, 28.7 million people were receiving ART globally, and the global ART coverage was 75%. At the end of 2021, only 52% of children aged 0 to 14 years had received ART and World Health Organization recommends that more efforts should be put in place to scale up treatment, most especially for

*Corresponding author tel. no: +2348060548485
Email address: saheed.ajao@elizadeuniversity.edu.ng (Saheed Ajao)

children and adolescents [3].

In the WHO African region, where two-thirds of the disease burden exist, access to treatment is an issue for people living with HIV due to many factors. In South Africa, discrimination and stigmatization still remain major obstacles to HIV response efforts which also affect children. According to a UN report, African children are neglected when it comes to HIV treatment [4]. UNAIDS reported that despite the global scientific advances in providing better treatment for children and adults, children with HIV in Indonesia have difficulties accessing antiretroviral therapy. The deeply rooted societal and gender inequalities create barriers to women, children, and adolescents accessing quality prevention and care services and thereby making the situation worse [5]. The report of [6] explained how government policy creates a barrier to HIV treatment. Several factors have been identified as barriers to HIV treatment (see [7-10]).

The modelling of the transmission of infectious diseases is now influencing the theory and practice of disease management and control. Mathematical modelling now plays a significant role in policy decision-making regarding the epidemiology of diseases in many countries [12]. Several models have been developed to study the dynamics of HIV transmission (see [13-16]). Apentang *et al* [17] studied the impact of the implementation of HIV prevention policies therapy and control strategies among HIV/AIDS new cases in Malaysia. The study revealed that the use of condoms and uncontaminated needle-syringes are important intervention control strategies. Yang et al. [18] studied the global dynamics of an HIV model, which incorporates senior male clients and their results showed that diagnosis, treatment and education have a positive impact on controlling HIV transmission, while senior male clients increase the number of new cases of HIV and prolong the time of the outbreak. Dubey et al. [19] modelled the role of acquired immune response and antiretroviral therapy in the dynamics of HIV infection. Ghosh et al. [20] worked on an HIV/AIDS model of an SI-type with the inclusion of media and self-imposed psychological fear and their results revealed that awareness is more effective in eradicating HIV infection.

The existing models in the literature failed to consider the partitioning of detected individuals who are receiving treatment and those who do not access treatment. Hence, we proposed a mathematical model to study the transmission dynamics of HIV in Nigeria. We assume that a fraction of individuals that are detected moves to the treatment class while the remaining fraction moves to the AIDS class.

The paper is structured as follows: section 2 contains the method used in the study, Section 3 has the numerical simulations and discussion of results, while the conclusion follows in section 4.

2. Method

2.1. Model Formulation

Here, we give the description of how the HIV model is designed and formulated. The overall population of humans at the time (t) is denoted by $N(t)$ and is partitioned into six distinct classes namely: the susceptible population $S(t)$, the HIV-latently infected L, the HIV-infected undetected class H_U, the HIV-infected detected class H_D, the treatment class H_W, and the AIDS class A. Thus

$$N(t) = S(t) + L(t) + H_U(t) + H_D(t) + H_W(t) + A(t) \quad (1)$$

The susceptible individuals are assumed to be recruited into the population at the rate π and get infected after effective contact with HIV-infected people in the latent, undetected, detected, treatment and AIDS classes at the rate λ, which is given by

$$\lambda = \frac{\beta(L + \eta_1 H_U + \eta_2 H_D + \eta_3 H_W + \eta_4 A)}{N} \quad (2)$$

where β is the contact rate, $\eta_1, \eta_2, \eta_4 \geq 1$ and $\eta_3 \leq 1$ are the modification parameters which compare the level of transmissibility of the disease in $H_U, H_D, A,$ and H_W classes with respect to people in L class.

It is assumed that a fraction ϵ of newly infected individuals progresses to the latently-infected class and the remaining fraction with compromised immunity moves to the HIV-undetected class. A fraction ω of people in the latent class who are detected progresses to the detected class while the other fraction $1 - \omega$ proceeds to the undetected class. The population of the HIV-detected class increases as a result of the detection of the undetected individuals at the rate γ and diminishes as a result of fraction α of detected individuals who are receiving treatment that progresses to treatment class and the remaining fraction $1 - \alpha$ that progresses to AIDS class. We assume that those that are receiving treatment move to the latent class at the rate ϕ. The AIDS class reduces as a result of death due to the disease at the rate δ. Each population size reduces as a result of natural death which occurs in all classes. The flow chart of the model showing the interaction among the classes is depicted in figure 1. Thus, with the assumptions above, we present a deterministic model of HIV infection as follows:

$$\begin{aligned}
\frac{dS}{dt} &= \pi - \lambda S - \mu S \\
\frac{dL}{dt} &= \epsilon\lambda S + \phi H_W - (\kappa + \mu)L \\
\frac{dH_U}{dt} &= (1 - \epsilon)\lambda S + (1 - \omega)\kappa L - (\gamma + \mu)H_U \\
\frac{dH_D}{dt} &= \omega\kappa L + \gamma H_U - (\tau + \mu)H_D \\
\frac{dH_W}{dt} &= \alpha\tau H_D - (\phi + \mu)H_W \\
\frac{dA}{dt} &= (1 - \alpha)\tau H_D - (\mu + \delta)A
\end{aligned} \quad (3)$$

where

$$\lambda = \frac{\beta(L + \eta_1 H_U + \eta_2 H_D + \eta_3 H_W + \eta_4 A)}{N} \quad (4)$$

$$N = S + L + H_U + H_D + H_W + A \quad (5)$$

For convenience, we re-write the above equation (3) as thus:

$$\frac{dS}{dt} = \pi - \lambda S - \mu S$$

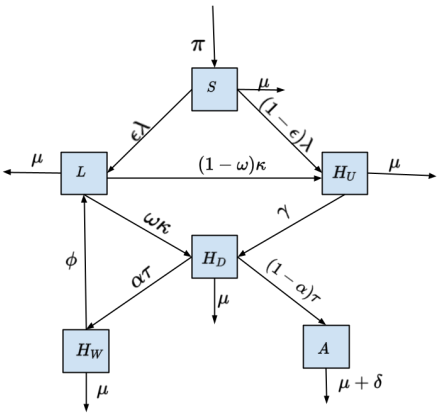

Figure 1. Schematic diagram of the model

$$\begin{aligned}
\frac{dL}{dt} &= \epsilon\lambda S + \phi H_W - T_1 L \\
\frac{dH_U}{dt} &= (1-\epsilon)\lambda S + WL - T_2 H_U \\
\frac{dH_D}{dt} &= XL + \gamma H_U - T_3 H_D \\
\frac{dH_W}{dt} &= YH_D - T_4 H_W \\
\frac{dA}{dt} &= ZH_D - T_5 A
\end{aligned} \tag{6}$$

where

$$\begin{aligned}
&T_1 = \kappa + \mu \qquad T_2 = \gamma + \mu \qquad T_3 = \tau + \mu \qquad T_4 = \phi + \mu \\
&T_5 = \mu + \delta \qquad W = (1-\omega)\kappa \qquad X = \omega\kappa \qquad Y = \alpha\tau \\
&Z = (1-\alpha)\tau
\end{aligned}$$

2.2. Model analysis

2.2.1. Basic properties

This section explores the basic dynamical features of the model (3). We claim the following:

Lemma 2.1. *The closed set*

$$D = \left\{(S, L, H_U, H_D, H_W, A) \in R_+^6 : N \le \frac{\pi}{\mu}\right\}$$

is positively invariant with non-negative initial values in R_+^6.

Proof. Summing up all the compartments of (3) with $\delta = 0$ we have

$$\frac{dN}{dt} = \pi - \mu N$$

It follows that

$$\frac{dN}{dt} \le \pi - \mu N$$

Then

$$N(t) \le N(0)e^{-\mu t} + \frac{\pi}{\mu}(1 - e^{-\mu t})$$

If $N(0) \le \frac{\pi}{\mu}$, then $N(t) \le \frac{\pi}{\mu}$. Hence, all solutions of the model having their starting values in D stay there for $t > 0$. This means that D is positively invariant and in this region, the model is considered to be epidemiologically meaningful and mathematically well-posed. Hence, we can study the dynamics of the basic model (3) in D. □

2.2.2. Stability of the disease-free equilibrium

The disease-free equilibrium of the model (6) denoted by E_1 is given by

$$E_1 = (S_0, L_0, H_{U0}, H_{D0}, H_{W0}, A_0) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0\right) \tag{7}$$

By adopting the approach of the Next Generation Matrix method as given by [25], the matrices F (new infection terms) and V (Transition terms) are as given below:

$$F = \begin{pmatrix}
\epsilon\beta & \epsilon\beta\eta_1 & \epsilon\beta\eta_2 & \epsilon\beta\eta_3 & \epsilon\beta\eta_4 \\
(1-\epsilon)\beta & (1-\epsilon)\beta\eta_1 & (1-\epsilon)\beta\eta_2 & (1-\epsilon)\beta\eta_3 & (1-\epsilon)\beta\eta_4 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0
\end{pmatrix} \tag{8}$$

and

$$V = \begin{pmatrix}
T_1 & 0 & 0 & -\phi & 0 \\
-W & T_2 & 0 & 0 & 0 \\
-X & -\gamma & T_3 & 0 & 0 \\
0 & 0 & -Y & T_4 & 0 \\
0 & 0 & -Z & 0 & T_5
\end{pmatrix} \tag{9}$$

Then $R_0 = \rho(FV^{-1})$, is given by

$$R_0 = \frac{\beta\begin{pmatrix}(1-\epsilon)[T_1T_3T_4T_5\eta_1 - XYT_5\phi\eta_1 + T_1T_4T_5\gamma\eta_2 \\ +T_1T_5Y\gamma\eta_3 + T_1T_4Z\gamma\eta_4 + YT_5\gamma\phi] \\ +\epsilon(XT_2 + \gamma W)[T_4T_5\eta_2 + YT_5\eta_3 + ZT_4\eta_4] \\ +\epsilon T_3T_4T_5(T_2 + W\eta_1)\end{pmatrix}}{T_5(T_1T_2T_3T_4 - XYT_2\phi - WY\gamma\phi)} \tag{10}$$

where ρ is the spectral radius of the dominant eigenvalue of the matrix FV^{-1}. Hence, using the Theorem 2 of [25], the following result is established:

Lemma 2.2. *The disease-free equilibrium of model (6) is locally asymptotically stable whenever the basic reproduction number* $R_0 < 1$ *and otherwise if* $R_0 > 1$

The threshold R_0 represents the basic reproduction number of the disease, which is the average number of secondary infections emanating from a single infection source in a population consisting of only the susceptible people [26]. The implication of lemma 2.2 is that a small introduction of infected individuals into the community/population will not produce a substantial outbreak of the disease when the basic reproduction number is less than unity and therefore the disease vanishes. In the next theorem, we show that the disease can be eradicated irrespective of the initial sizes of the sub-populations when $R_0 < 1$ through the exploration of the global stability of the disease-free equilibrium.

Theorem 2.3. *The disease-free equilibrium (7) of the HIV model is globally asymptotically stable whenever* $R_0 < 1$

Table 1. Description of the parameters used in the model (3)

Parameter	Description	Value	Source
π	Recruitment rate	618893	[21]
β	Contact rate	0.0785	Estimated
μ	Natural death rate	0.022	[22]
α	Fraction of detected individuals that are treated	0.29	Assumed
ϕ	Progression rate from treatment class to latent class	0.201	Assumed
γ	Detection rate of undetected individuals	0.392	Estimated
ϵ	Fraction of newly infected people with uncompromised immunity	0.9	Assumed
κ	Progression rate of people in the latent class	0.01	[15]
$\eta_1, \eta_2, \eta_3, \eta_4$	Modification parameters	1.17,1.2,0.04,0.18	Assumed
δ	Death due to the disease	0.33	[23]
τ	Treatment rate	0.89	[24]
ω	Fraction of latent individuals that are detected	0.1	Assumed

Proof. Using the Lyapunov function defined by

$$F = E_1 L + E_2 H_U + E_3 H_D + E_4 H_W + E_5 A \tag{11}$$

By differentiating (11), we have

$$F' = E_1 L' + E_2 H_U' + E_3 H_D' + E_4 H_W' + E_5 A' \tag{12}$$

where

$$E_1 = (1-\epsilon)\beta(X\eta_1 - \gamma) - N(T_2 X + \gamma W)$$
$$E_2 = \epsilon\beta(\gamma - X\eta_1) - \gamma T_1 N$$
$$E_3 = \epsilon\beta S(W\eta_1 + T_2) + T_1[(1-\epsilon)\eta_1\beta S + T_2 N]$$
$$E_4 = \frac{\begin{pmatrix}\phi[(1-\epsilon)\beta S(X\eta_1 - \gamma) - N(T_2 X + \gamma W)] \\ -\beta S\eta_1[\epsilon(T_2 X + \gamma W) + \gamma(1-\epsilon)]\end{pmatrix}}{T_4}$$
$$E_5 = \frac{\begin{pmatrix}\epsilon\beta S[(T_2 X + \gamma W)(T_4\eta_2 + Y\eta_3) + T_4 T_3(W\eta_1 + T_2)] \\ +(1-\epsilon)\beta S[T_1 T_4(\eta_2\gamma + T_3\eta_1) - Y\phi(X\eta_1 - \gamma) \\ +Y\eta_3\gamma] + N[Y\phi(T_2 X + \gamma W) - T_1 T_2 T_3 T_4]\end{pmatrix}}{ZT_4}$$

Then by substituting (6) into (12), it becomes

$$F' = E_1(\epsilon\lambda S - T_1 L + \phi H_W) + E_2((1-\epsilon)\lambda S + WL - T_2 H_U) + E_3(XL + \gamma H_U - T_3 H_D) + E_4(YH_D - T_4 H_W) + E_5(ZH_D - T_5 A) \tag{13}$$

Simplifying (13) further leads to

$$F' = \frac{\begin{pmatrix}-NT_5(Y\phi T_2 X + Y\phi\gamma W - T_1 T_2 T_3 T_4) \\ -\epsilon\beta S(T_2 X + \gamma W)[ZT_4\eta_4 + T_5 T_4\eta_2 + T_5 Y\eta_3] \\ -(1-\epsilon)\beta S[T_5 T_1 T_4\eta_4\gamma + T_5 T_3\eta_1 \\ -YT_5\phi(X\eta_1 - \gamma) + T_5 Y\eta_3\gamma + ZT_4\gamma T_1\eta_4] \\ -\epsilon\beta S T_5 T_4 T_3(W\eta_1 + T_2)\end{pmatrix} A}{ZT_4}$$

$$F' = \frac{T_5 N[Y\phi(T_2 X + \gamma W) - T_1 T_2 T_3 T_4]}{ZT_4}\left(\frac{S}{N}R_0 - 1\right)A$$
$$F' \leq \left(\frac{T_5 N[Y\phi(T_2 X + \gamma W) - T_1 T_2 T_3 T_4]}{ZT_4}\right)(R_0 - 1)A$$

Since $S \leq N$ in D, Therefore,$F' \leq 0$ if $R_0 \leq 1$ with $F' = 0$ if and only if $A = 0, L = 0, H_U = 0, H_D = 0, H_W = 0$. Also, the largest invariant set in $(S, L, H_U, H_D, H_W, A) \in D : F' = 0$ is the singleton E_1. By LaSalle Invariance Principle [27], every solution having its starting values in D, approaches E_1 as $t \to \infty$, and therefore, the disease-free equilibrium is globally asymptotically stable whenever $R_0 < 1$. □

The theorem implies that the disease can be eliminated irrespective of the initial sizes of the subpopulations of the model whenever the basic reproduction number does not exceed unity.

2.2.3. Existence of endemic equilibrium

The existence of endemic equilibrium is being investigated here and the condition for the persistence of the disease is being explored.

Let $E_2{}^* = (S^{**}, L^{**}, H_U^{**}, H_D^{**}, H_W^{**}, A^{**})$ represents the endemic equilibrium state. Also, let the force of infection at endemic equilibrium be represented by

$$\lambda^{**} = \frac{\beta(L^{**} + \eta_1 H_U^{**} + \eta_2 H_D^{**} + \eta_3 H_W^{**} + \eta_4 A^{**})}{N^{**}} \tag{14}$$

Then solving the model equations in terms of the λ (force of infection) at the steady state, we will get the following:

$$S^{**} = \frac{\pi}{\lambda^{**} + \mu} \tag{15}$$

$$L^{**} = \frac{[\epsilon T_2 T_3 T_4 + \phi Y\gamma(1-\epsilon)]\lambda^{**} S^{**}}{T_2(T_1 T_3 T_4 - \phi YX) - \phi Y\gamma W} = Q_1\lambda^{**} S^{**} \tag{16}$$

$$H_U^{**} = \frac{[(1-\epsilon)(T_1T_3T_4 - \phi YX) + \epsilon WT_3T_4]\lambda^{**}S^{**}}{T_2(T_1T_3T_4 - \phi YX) - \phi Y\gamma W} = Q_2\lambda^{**}S^{**} \tag{17}$$

$$H_D^{**} = \frac{T_4[T_1\gamma(1-\epsilon) + \epsilon(XT_2 + W\gamma)]\lambda^{**}S^{**}}{T_2(T_1T_3T_4 - \phi YX) - \phi Y\gamma W} = Q_3\lambda^{**}S^{**} \tag{18}$$

$$H_W^{**} = \frac{Y[T_1\gamma(1-\epsilon) + \epsilon(XT_2 + W\gamma)]\lambda^{**}S^{**}}{T_2(T_1T_3T_4 - \phi YX) - \phi Y\gamma W} = Q_4\lambda^{**}S^{**} \tag{19}$$

$$A^{**} = \frac{ZT_4[T_1\gamma(1-\epsilon) + \epsilon(XT_2 + W\gamma)]\lambda^{**}S^{**}}{T_5[T_2(T_1T_3T_4 - \phi YX) - \phi Y\gamma W]} = Q_5\lambda^{**}S^{**} \tag{20}$$

By the substitution of (16), (17), (18), (19), and (20) into (14), we have

$$\lambda^{**} = \frac{\beta(Q_1 + \eta_1Q_2 + \eta_2Q_3 + \eta_3Q_4 + \eta_4Q_5)\lambda^{**}S^{**}}{S^{**} + Q_2\lambda^{**}S^{**} + Q_3\lambda^{**}S^{**} + Q_4\lambda^{**}S^{**} + Q_5\lambda^{**}S^{**}} \tag{21}$$

$$\lambda^{**} = \frac{\beta(Q_1 + \eta_1Q_2 + \eta_2Q_3 + \eta_3Q_4 + \eta_4Q_5)\lambda^{**}S^{**}}{S^{**}(1 + V\lambda^{**})} \tag{22}$$

where

$$\begin{aligned}
V &= Q_1 + Q_2 + Q_3 + Q_4 + Q_5\\
Q_1 &= \frac{[\epsilon T_2T_3T_4 + \phi Y\gamma(1-\epsilon)]}{T_2(T_1T_3T_4 - \phi YX) - \phi Y\gamma W}\\
Q_2 &= \frac{[(1-\epsilon)(T_1T_3T_4 - \phi YX) + \epsilon WT_3T_4]}{T_2(T_1T_3T_4 - \phi YX) - \phi Y\gamma W}\\
Q_3 &= \frac{T_4[T_1\gamma(1-\epsilon) + \epsilon(XT_2 + W\gamma)]}{T_2(T_1T_3T_4 - \phi YX) - \phi Y\gamma W}\\
Q_4 &= \frac{Y[T_1\gamma(1-\epsilon) + \epsilon(XT_2 + W\gamma)]}{T_2(T_1T_3T_4 - \phi YX) - \phi Y\gamma W}\\
Q_5 &= \frac{ZT_4[T_1\gamma(1-\epsilon) + \epsilon(XT_2 + W\gamma)]}{T_5[T_2(T_1T_3T_4 - \phi YX) - \phi Y\gamma W]}
\end{aligned}$$

Hence, $\lambda^{**} = \dfrac{R_0 - 1}{V} > 0$ when $R_0 > 1$

$\therefore \lambda^{**}$ has a positive unique solution when $R_0 > 1$. Hence, the following result is obtained:

Lemma 2.4. *There exists a unique endemic equilibrium of the HIV model equation (6) whenever the basic reproduction number $R_0 > 1$.*

The above result indicates the existence of forward bifurcation which is verified in the next analysis.

2.2.4. Bifurcation analysis

The bifurcation is a phenomenon that describes the changes in the behaviour of a dynamical system as a result of changes in the parameter values or initial conditions of the model. This helps to determine if the disease can be cleared off when the basic reproduction number is less than unity. We will adopt the Center Manifold Theory [28] as described by [29][see Appendix B] to establish the kind of bifurcation that the model exhibits. The Center Manifold Theory is used to determine the stability of equilibrium and plays a vital role in bifurcation theory because of the changes in behaviour of the system that take place on the center manifold.

If β is chosen as the bifurcation parameter for model (6), then at $R_0 = 1$, we have that

$$\beta = \beta^* = \frac{T_5(T_1T_2T_3T_4 - XYT_2\phi - WY\gamma\phi)}{\begin{pmatrix}(1-\epsilon)[T_1T_3T_4T_5\eta_1 - XYT_5\phi\eta_1 + T_1T_4T_5\gamma\eta_2\\ +T_1T_5Y\gamma\eta_3 + T_1T_4Z\gamma\eta_4 + YT_5\gamma\phi]\\ +\epsilon(XT_2 + \gamma W)[T_4T_5\eta_2 + YT_5\eta_3 + ZT_4\eta_4]\\ +\epsilon T_3T_4T_5(T_2 + W\eta_1)\end{pmatrix}} \tag{23}$$

If the variables of (6) are changed as follows: $S = x_1, L = x_2, H_U = x_3, H_D = x_4, H_W = x_5, A = x_6$ and we use the vector notation $\mathbf{x} = (x_1, x_2, x_3, x_4, x_5, x_6)^T$, then (6) can be re-written in the form $\frac{dx}{dt} = F(\mathbf{x})$ where $F = (f_1, f_2, f_3, f_4, f_5, f_6)^T$ such that (6) becomes

$$\begin{aligned}
\frac{dx_1}{dt} &= \pi - \lambda x_1 - \mu x_1 = f_1\\
\frac{dx_2}{dt} &= \epsilon\lambda x_1 + \phi x_5 - T_1x_2 = f_2\\
\frac{dx_3}{dt} &= (1-\epsilon)\lambda x_1 + Wx_2 - T_2x_3 = f_3\\
\frac{dx_4}{dt} &= Xx_2 + \gamma x_3 - T_3x_4 = f_4\\
\frac{dx_5}{dt} &= Yx_4 - T_4x_5 = f_5\\
\frac{dx_6}{dt} &= Zx_4 - T_5x_6 = f_6
\end{aligned} \tag{24}$$

$$\tag{25}$$

The Jacobian (24) at disease-free equilibrium E_1 is given by

$$J(E_1) = \begin{pmatrix}
-\mu & -\beta^* & -\beta^*\eta_1 & -\beta^*\eta_2 & -\beta^*\eta_3 & -\beta^*\eta_4\\
0 & \epsilon\beta^* - T_1 & \epsilon\beta^*\eta_1 & \epsilon\beta^*\eta_2 & \epsilon\beta^*\eta_3 + \phi & \epsilon\beta^*\eta_4\\
0 & (1-\epsilon)\beta^* + W & (1-\epsilon)\beta^*\eta_1 - T_2 & (1-\epsilon)\beta^*\eta_2 & (1-\epsilon)\beta^*\eta_3 & (1-\epsilon)\beta^*\eta_4\\
0 & X & \gamma & -T_3 & 0 & 0\\
0 & 0 & 0 & Y & -T_4 & 0\\
0 & 0 & 0 & 0 & Z & -T_5
\end{pmatrix} \tag{26}$$

The matrix (26) has a simple zero eigenvalue at $\beta = \beta^*$ and hence Center Manifold Theory [28] as described by [29] can be used to analyse the dynamics of the system. The Jacobian matrix (26) has a right eigenvector denoted by $W = (w_1, w_2, w_3, w_4, w_5, w_6)^T$ and a left eigenvector $V = (v_1, v_2, v_3, v_4, v_5, v_6)^T$ corresponding to the zero eigenvalue. Then

$$w_1 = \frac{\beta^*\begin{pmatrix}T_3T_4T_5[w_2 + \eta_1w_3] + (Xw_2 + \gamma w_3)\\ \times[T_4T_5\eta_2 + T_5Y\eta_3 + T_4Z\eta_4]\end{pmatrix}}{T_3T_4T_5\mu},$$

$$\begin{aligned}
w_2 &= w_2 > 0, & w_3 &= w_3 > 0\\
w_4 &= \frac{Xw_2 + \gamma w_3}{T_3}, & w_5 &= \frac{Y[Xw_2 + \gamma w_3]}{T_3T_4},\\
w_6 &= \frac{Z[Xw_2 + \gamma w_3]}{T_3T_5}
\end{aligned}$$

and

$$v_1 = 0, \qquad v_2 = v_2 > 0, \qquad v_3 = v_3 > 0,$$

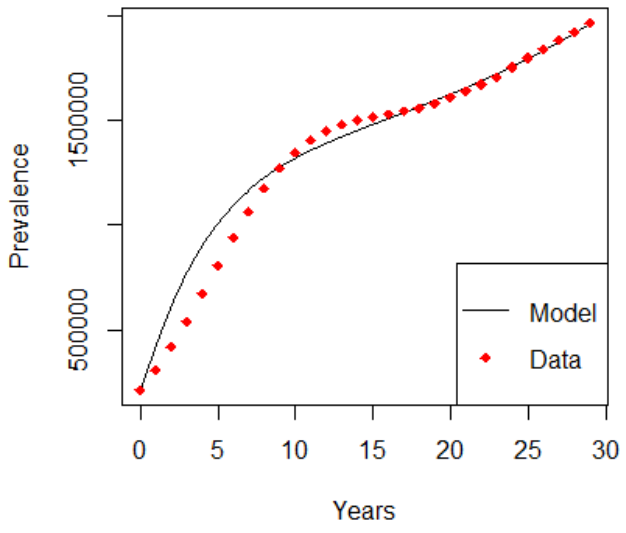

Figure 2. Fitting of HIV model (3) to the data of prevalence cases of HIV/AIDS infection in Nigeria between 1990 and 2019 [30].

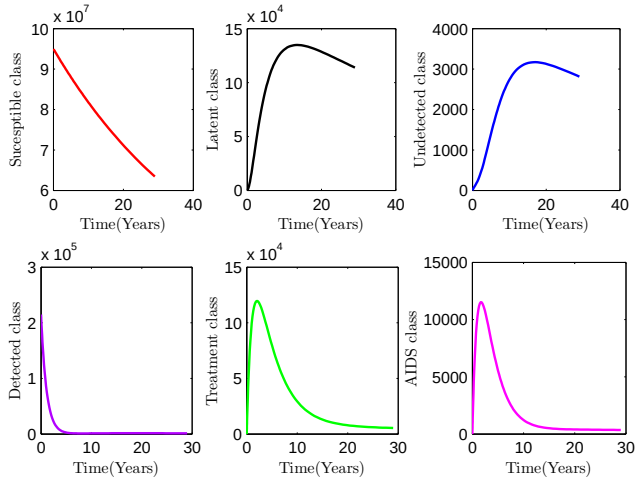

Figure 3. Plot of the different classes of the model when $R_0 < 1$ ($R_0 = 0.4$) with $\beta = 0.0085$ and other values used are in table 1

$$v_4 = \frac{\begin{pmatrix}(T_4T_5\eta_2 + YT_5\eta_3 + T_4Z\eta_4)[\epsilon\beta^* v_2 + (1-\epsilon)\beta^* v_3] \\ +\phi Y T_5 v_2\end{pmatrix}}{T_3T_4T_5},$$

$$v_5 = \frac{\beta^*\eta_3(\epsilon v_2 + (1-\epsilon)v_3) + \phi v_2}{T_4}, \quad v_6 = \frac{\beta^*\eta_4(\epsilon v_2 + (1-\epsilon)v_3)}{T_5}$$

Computation of a and b

By finding the associated non-zero partial derivatives of $F(x)$ at disease-free equilibrium, the associated bifurcation coefficients a and b as given by the Center Manifold Theory [28][see Appendix B], are defined by

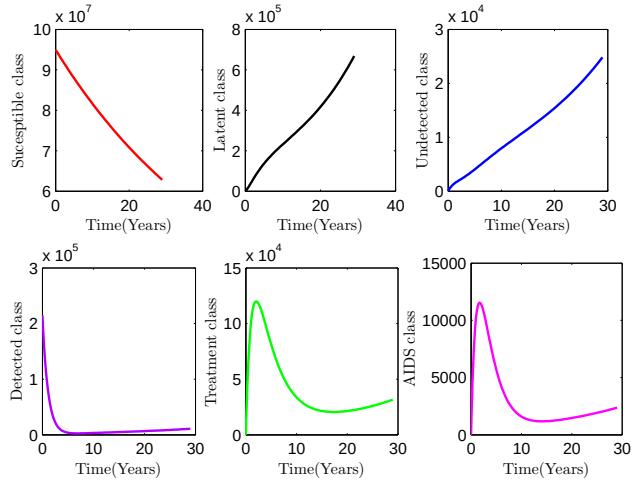

Figure 4. Plot of the different classes of the model when $R_0 > 1$ ($R_0 = 3.3$) using the values of the parameters in table 1

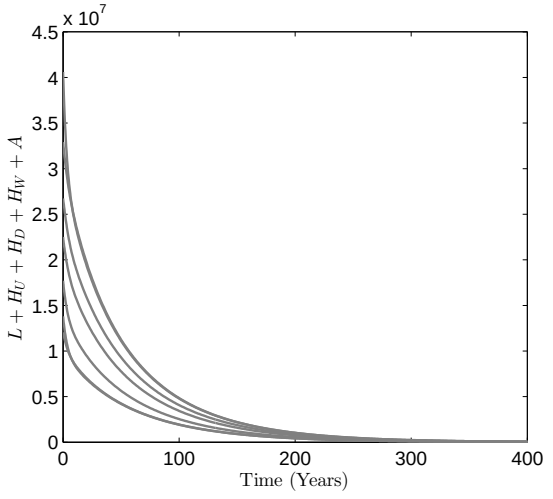

Figure 5. Plot of the total number of the infected classes ($L+H_U+H_D+H_W+A$) at different initial conditions when $R_0 = 0.4$, with $\beta = 0.0085$ and other values of the parameters used are given in Table 1

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(0,0)$$

Then, we obtain

$$a = \frac{-2\beta^*\mu}{\pi}\begin{bmatrix}(v_2\epsilon + v_3(1-\epsilon)) \\ \times(w_2 + w_3 + w_4 + w_5 + w_6) \\ \times(\eta_1 w_3 + \eta_2 w_4 + \eta_3 w_5 + \eta_4 w_6 + w_2)\end{bmatrix} \tag{27}$$

and

$$b = (v_2\epsilon + v_3(1-\epsilon))[w_2 + w_3\eta_1 + w_4\eta_2 + w_5\eta_3 + w_6\eta_4] \tag{28}$$

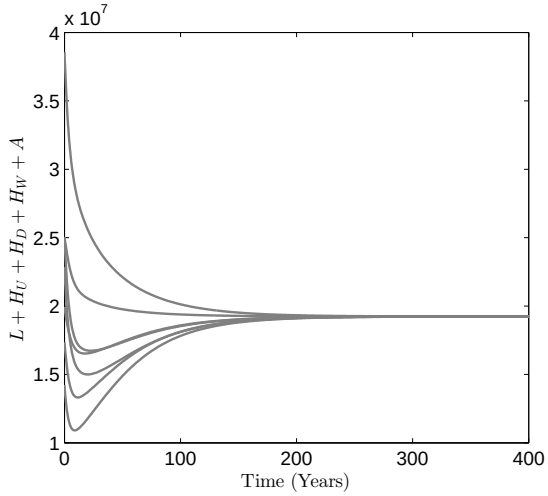

Figure 6. Plot of the total number of the infected classes ($L+H_U+H_D+H_W+A$) at different initial conditions when $R_0 = 3.3$, with $\epsilon = 1$, $\omega = 0$ and other values of the parameters used are given in Table 1

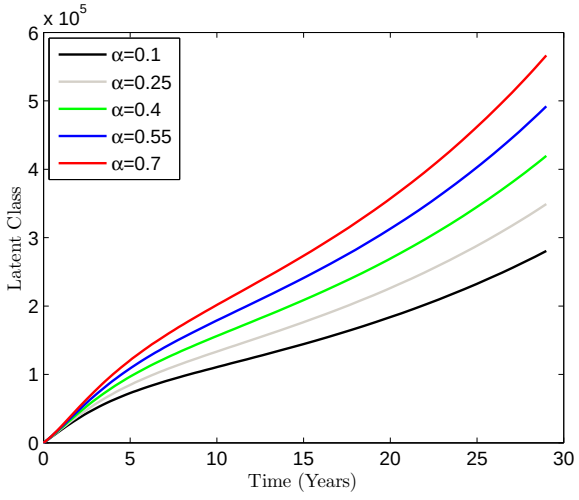

Figure 7. Graph of the population of the latent class against time when the fraction of the detected population receiving treatment is varied

From (28), b is positive as usual and a is negative from (27). Since $a < 0$ (negative), then the HIV infection model exhibits a forward bifurcation. This implies that the epidemiological condition $R_0 < 1$ is necessary and sufficient for the elimination of HIV infection.

2.2.5. *Global stability of the endemic equilibrium*

We consider the global stability of endemic equilibrium of the HIV infection model for a situation when $\epsilon = 1$ and the fraction of the latently infected people that are detected equals zero ($\omega = 0$) and also we let $\beta^* = \frac{\beta}{N}$. Then, with these assumptions the model's basic reproduction number when $\epsilon = 1$ and $\omega = 0$ is given by

$$R_{or} = \frac{\beta^* \pi \begin{pmatrix} Y\gamma\kappa T_5\eta_3 + Z\gamma\kappa T_4\eta_4 + \gamma\kappa T_4T_5\eta_2 \\ +\kappa T_3T_4T_5\eta_1 + T_4T_3T_2T_5 \end{pmatrix}}{T_5\mu\ (T_4T_3T_2T_1 - Y\gamma\kappa\phi)}$$

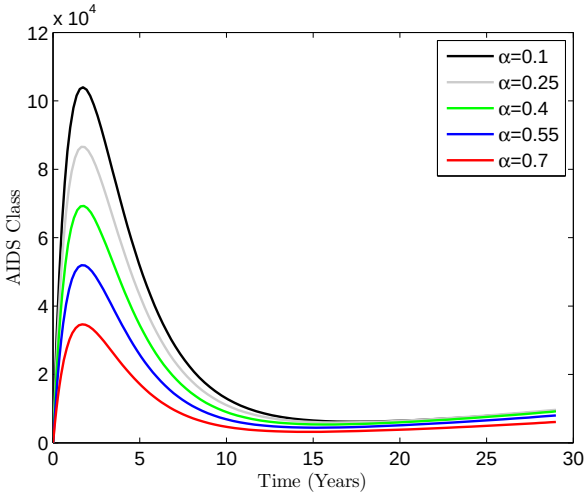

Figure 8. Graph of the population of the AIDS class against time when the fraction of the detected population receiving treatment is varied

Also, the model with $\epsilon = 1$ and $\omega = 0$ possesses a unique endemic equilibrium point represented by E^*_{2r} , which is given by

$$E_{2r}{}^*{}_{|\epsilon=1,\omega=0} = (S^{**}, L^{**}, H_U^{**}, H_D^{**}, H_W^{**}, A^{**})$$

and that

$$S^{**} > 0, L^{**} > 0, H_U^{**} > 0, H_D^{**} > 0, H_W^{**} > 0 \text{ and } A^{**} > 0$$

when $R_{or} > 1$

Theorem 2.5. *The endemic equilibrium of the reduced model having $\epsilon = 1$ and $\omega = 0$ is globally asymptotically stable whenever $R_{or} > 1$.*

Proof. Using the Goh-Volterra type of Lyapunov function, we have

$$\begin{aligned} M = {} & S - S^{**} - S^{**} ln\frac{S}{S^{**}} + L - L^{**} - L^{**} ln\frac{L}{L^{**}} \\ & + C\left(H_U - H_U^{**} - H_U^{**} ln\frac{H_U}{H_U^{**}}\right) + D\left(H_D - H_D^{**} - H_D^{**} ln\frac{H_D}{H_D^{**}}\right) \\ & + E\left(H_W - H_W^{**} - H_W^{**} ln\frac{H_W}{H_W^{**}}\right) + F\left(A - A^{**} - A^{**} ln\frac{A}{A^{**}}\right) \end{aligned} \tag{29}$$

where

$$C = \frac{\beta^* S^{**}[T_3T_4T_5\eta_1 + \gamma(T_4T_5\eta_2 + YT_5\eta_3 + ZT_4\eta_4)] + \gamma Y\phi T_5}{T_2T_3T_4T_5} \tag{30}$$

$$D = \frac{\beta^* S^{**}[T_4T_5\eta_2 + YT_5\eta_3 + ZT_4\eta_4] + Y\phi T_5}{T_3T_4T_5} \tag{31}$$

$$E = \frac{\beta^*\eta_3 S^{**} + \phi}{T_4} \tag{32}$$

$$F = \frac{\beta^*\eta_4 S^{**}}{T_5} \tag{33}$$

Taking the derivative of (29), we have

$$\dot{M} = \dot{S} - S^{**}\frac{\dot{S}}{S} + \dot{L} - L^{**}\frac{\dot{L}}{L} + \frac{\begin{pmatrix}\beta^* S^{**}[T_3T_4T_5\eta_1 + \gamma(T_4T_5\eta_2 + YT_5\eta_3 + ZT_4\eta_4)] \\ +\gamma Y\phi T_5\end{pmatrix}}{T_2T_3T_4T_5} \times\left(\dot{H}_U - H_U^{**}\frac{\dot{H}_U}{H_U}\right) + \frac{\beta^*S^{**}[T_4T_5\eta_2 + YT_5\eta_3 + ZT_4\eta_4] + Y\phi T_5}{T_3T_4T_5} \times\left(\dot{H}_D - {H_D}^{**}\frac{\dot{H}_D}{H_D}\right) + \frac{\beta^*\eta_3S^{**}+\phi}{T_4}\left(\dot{H}_W - H_W^{**}\frac{\dot{H}_W}{H_W}\right) + \frac{\beta^*\eta_4S^{**}}{T_5}\left(\dot{A} - A^{**}\frac{\dot{A}}{A}\right)$$

$$\begin{aligned}\dot{M} &= 2\beta^*S^{**}(L^{**} + \eta_1H_U^{**} + \eta_2H_D^{**} + \eta_3H_W^{**} + A^{**}) + 2\mu S^{**} \\ &- \mu S - \frac{\beta^*{S^{**}}^2}{S}(L^{**} + \eta_1H_U^{**} + \eta_2H_D^{**} + \eta_3H_W^{**} + A^{**}) \\ &- \frac{\mu S^{**}}{S} - \frac{\beta^*SL^{**}}{L}(L + \eta_1H_U + \eta_2H_D + \eta_3H_W + A) + \phi H_W^{**} \\ &- \frac{H_WL^{**}}{L} - \frac{LH_U^{**}}{L^{**}H_U}\begin{pmatrix}\beta^*S^{**}[\eta_1H_U^{**} + \eta_2H_D^{**} \\ +\eta_3H_W^{**} + A^{**}] + \phi H_W^{**}\end{pmatrix} \\ &+ (\beta^*S^{**}[\eta_1H_U^{**} + \eta_2H_D^{**} + \eta_3H_W^{**} + A^{**}] + \phi H_W^{**}) \\ &- \frac{H_UH_D^{**}}{H_U^{**}H_D}(\beta^*S^{**}[\eta_2H_D^{**} + \eta_3H_W^{**} + A^{**}] + \phi H_W^{**}) + \\ &(\beta^*S^{**}[\eta_2H_D^{**} + \eta_3H_W^{**} + A^{**}] + \phi H_W^{**}) \\ &- \frac{(\beta^*\eta_3S^{**}+\phi)H_D{H_W^{**}}^2}{H_D^{**}H_W}\beta^*\eta_3S^{**}H_W^{**} \\ &+ \phi H_W^{**} - \frac{\beta^*\eta_4S^{**}H_D{A^{**}}^2}{FH_D^{**}} + \beta^*\eta_4S^{**}A^{**}\end{aligned}$$

Then

$$\begin{aligned}\dot{M} =& \beta^*S^{**}L^{**}\left(2 - \frac{S^{**}}{S} - \frac{S}{S^{**}}\right) + \mu S^{**}\left(2 - \frac{S}{S^{**}} - \frac{S^{**}}{S}\right) \\ &+ \beta^*S^{**}\eta_1H_U^{**}\left(3 - \frac{S^{**}}{S} - \frac{H_UL^{**}S}{H_U^{**}LS^{**}} - \frac{LH_U^{**}}{L^{**}H_U}\right) \\ &+ \beta^*S^{**}\eta_2H_D^{**}\left(4 - \frac{S^{**}}{S} - \frac{H_DL^{**}S}{H_D^{**}LS^{**}} - \frac{LH_U^{**}}{L^{**}H_U} - \frac{H_UH_D^{**}}{H_U^{**}H_D}\right) \\ &+ \beta^*S^{**}\eta_3H_W^{**}\begin{pmatrix}5 - \frac{S^{**}}{S} - \frac{H_WL^{**}S}{H_W^{**}LS^{**}} \\ -\frac{LH_U^{**}}{L^{**}H_U} - \frac{H_UH_D^{**}}{H_U^{**}H_D} - \frac{H_DH_W^{**}}{H_D^{**}H_W}\end{pmatrix} \\ &+ \beta^*S^{**}\eta_4A^{**}\begin{pmatrix}5 - \frac{S^{**}}{S} - \frac{AL^{**}S}{A^{**}LS^{**}} \\ -\frac{LH_U^{**}}{L^{**}H_U} - \frac{H_UH_D^{**}}{H_U^{**}H_D} - \frac{H_DA^{**}}{H_D^{**}A}\end{pmatrix} \\ &+ \phi H_W^{**}\left(4 - \frac{H_WL^{**}}{H_W^{**}L} - \frac{LH_U^{**}}{L^{**}H_U} - \frac{H_UH_D^{**}}{H_U^{**}H_D} - \frac{H_DH_W^{**}}{H_D^{**}H_W}\right)\end{aligned}$$

The arithmetic mean surpasses the geometric mean, then we have the following inequalities

$$2 - \frac{S}{S^{**}} - \frac{S^{**}}{S} \le 0, \qquad 2 - \frac{S^{**}}{S} - \frac{S}{S^{**}} \le 0,$$

$$3 - \frac{S^{**}}{S} - \frac{H_UL^{**}S}{H_U^{**}LS^{**}} - \frac{LH_U^{**}}{L^{**}H_U} \le 0,$$

$$4 - \frac{S^{**}}{S} - \frac{H_DL^{**}S}{H_D^{**}LS^{**}} - \frac{LH_U^{**}}{L^{**}H_U} - \frac{H_UH_D^{**}}{H_U^{**}H_D} \le 0,$$

$$5 - \frac{S^{**}}{S} - \frac{H_WL^{**}S}{H_W^{**}LS^{**}} - \frac{LH_U^{**}}{L^{**}H_U} - \frac{H_UH_D^{**}}{H_U^{**}H_D} - \frac{H_DH_W^{**}}{H_D^{**}H_W} \le 0,$$

$$5 - \frac{S^{**}}{S} - \frac{AL^{**}S}{A^{**}LS^{**}} - \frac{LH_U^{**}}{L^{**}H_U} - \frac{H_UH_D^{**}}{H_U^{**}H_D} - \frac{H_DA^{**}}{H_D^{**}A} \le 0,$$

$$4 - \frac{H_WL^{**}}{H_W^{**}L} - \frac{LH_U^{**}}{L^{**}H_U} - \frac{H_UH_D^{**}}{H_U^{**}H_D} - \frac{H_DH_W^{**}}{H_D^{**}H_W} \le 0$$

Therefore $L \le 0$ when $R_{or} > 1$. Hence, by the LaSalle Invariance Principle [27], every solution of the model tends to E_2r^* as $t \to \infty$ for $R_{0r} > 1$. □

The epidemiological implication of this is that HIV infection will persist in the community irrespective of the initial sizes of the subpopulations of the model whenever $R_{0r} > 1$.

3. Numerical Simulations and Discussion of Results

We fit the HIV model to data on HIV/AIDS prevalence in Nigeria from 1990 to 2019 as presented in table 2 (see Appendix. A) sourced from [30]. We use the likelihood function to estimate the values of the contact rate (β) and the detection rate of the undetected class (γ). The estimated values of β and γ are 0.0785 and 0.392 respectively. The total population of Nigeria in 1990 stood at 95214256 based on [31] and the initial conditions used for the simulations are as follow: $S(0) = 94999422, L(0) = 0, H_U(0) = 0, H_D(0) = 214834, H_W(0) = 0$ and $A(0) = 0$. The time interval of 0 to 29 corresponds to the time interval between 1990 to 2019 and the values of the parameters used for the simulations are given in table 1. The results of the numerical simulations of the model are presented in figures 2 - 8.

In figure 2, we fit the HIV model to data in table 2 and also obtain the estimated values for the contact rate (β) and the detection rate (γ). The model fits well with the real data and thus the model represents reality. Figure 3 illustrates the behaviour of each compartment when the basic reproduction number is greater than unity. The susceptible class keeps decreasing while the other infected compartments L, H_U, H_D, H_W and A are increasing which indicates the persistence of the HIV infection. Figure 4 shows the trajectories of the model when the basic reproduction number is less than unity. The population of the susceptible declines and the infected classes L, H_U, H_D, H_W and A are reducing after some period, which means that the disease can be controlled if $R_0 < 1$.

Figures 5 and 6 illustrate the verification of the global stability properties of the disease-free equilibrium and endemic

equilibrium. The long-term dynamics of the model as depicted by figure 5 shows that the trajectories converge and the disease vanishes irrespective of the initial sizes of the subpopulations when the basic reproduction number is less than unity and figure 6 shows that the disease persists when the basic reproduction number is greater than unity. In figure 7, the plot shows the population of latently infected individuals when the fraction of the HIV-detected individuals that are receiving treatment is varied. The population of the latently infected increases as this fraction increases. This is due to the fact that HIV infection has no cure but can be managed. The graph in figure 8 shows that the population of the AIDS class reduces as the fraction of detected individuals receiving treatment increases.

4. Conclusion

We propose a mathematical model to study the transmission dynamics of HIV and conduct qualitative and quantitative analyses of the model. The model's disease-free equilibrium is locally asymptotically stable whenever the basic reproduction number is less than unity. Also, there exists a unique endemic equilibrium for the model whenever the basic reproduction number is greater than unity and it is shown that the model exhibits forward bifurcation which implies that the necessary condition $R_0 < 1$ is sufficient for the elimination of the disease. Using the Lyapunov function, we further showed that the disease-free equilibrium and endemic equilibrium are globally asymptotically stable whenever the basic reproduction number is less than unity and greater than unity respectively. The proposed model fits with the data on HIV/AIDS prevalence in Nigeria from 1990 to 2019 as it represents the reality. The simulation shows that the disease can be controlled when the basic reproduction number is less than unity and persists if otherwise. The simulations that illustrate the global stability of the model justify the analytic results. The effect of increasing the fraction of the detected individuals that are receiving treatment is examined and it increases the population of the latent class and reduces the population of the AIDS class, since the disease has no cure, the treatment is meant to improve the health of a patient by reducing the viral load to an undetected level and prevent a patient from progressing into AIDS. Hence, there is a need to intensify efforts in the treatment of those who are being detected.

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Appendix A:

HIV/AIDS Prevalence in Nigeria from 1990-2019 as given by [30]

Table 2. Data of HIV/AIDS Prevalence in Nigeria from 1990-2019 [30]

Year	1990	1991	1992	1993	1994
Cases	214934	307403	417550	541812	674511
Year	1995	1996	1997	1998	1999
Cases	808728	940842	1065300	1177623	1271363
Year	2000	2001	2002	2003	2004
Case	1347177	1406166	1449357	1479819	1500481
Year	2005	2006	2007	2008	2009
Cases	1515892	1527636	1542107	1558937	1581336
Year	2010	2011	2012	2013	2014
Cases	1609292	1638694	1670713	1707410	1752498
Year	2015	2016	2017	208	2019
Cases	1797982	1841027	1882445	1922997	1963044

Appendix B:

Theorem (Castillo-Chavez and Song [29]). *Consider the following general system of ordinary differential equations with a parameter ϕ.*

$$\frac{dx}{dt} = f(x,\phi),\ f : R^n \times R \to R^n \text{ and } f \in C^2(R^n \times R)$$

where 0 *is an equilibrium point of the system(that is, $f(0,\phi) = 0$ for all ϕ) and*

1. *$A = D_x f(0,0)$ is the linearization matrix of the system around the equilibrium* 0 *with ϕ evaluated at* 0*;*

2. *Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts;*

3. *Matrix A has a right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue.*

Let f_k be the k^{th} component of f and

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0)$$

Then the local dynamics of the system around the equilibrium point 0 *is totally determined by the signs of a and b.*

i. *$a > 0, b > 0$. When $\phi < 0$ with $|\phi| \ll 1$,* 0 *is locally asymptotically stable, and there exists a positive unstable equilibrium; when $0 < \phi \ll 1$,* 0 *is unstable and there exists a negative and locally asymptotically stable equilibrium;*

ii. *$a < 0, b < 0$. When $\phi < 0$ with $|\phi| \ll 1$,* 0 *is unstable; when $0 < \phi \ll 1$,* 0 *is locally asymptotically stable, and there exists a positive unstable equilibrium;*

iii. *$a > 0, b < 0$. When $\phi < 0$ with $|\phi| \ll 1$,* 0 *is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0 < \phi \ll 1$,* 0 *is stable, and a positive unstable equilibrium appears;*

iv. *$a < 0, b > 0$. When ϕ changes from negative to positive,* 0 *changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.*