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STABILITY ANALYSIS OF HIV/AIDS EPIDEMIC MODEL WITH VERTICAL TRANSMISSION

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ABSTRACT. In this study, a new mathematical model of HIV/AIDS with vertical transmission is presented and analysed. The well posedness of the model is analysed using the theory of positivity and boundedness of solutions. Analysis of the model shows that the disease-free equilibrium is locally and globally asymptotically stable when $\mathcal{R}_0 < 1$. Furthermore, the global asymptotic stability of the endemic equilibrium is examined using a quadratic Lyapunov function and there would be prevalence of HIV/AIDS in the population when the basic reproduction number is greater than unity. Numerical simulations are carried out to support the analytical solutions of the model.

1. INTRODUCTION

The lentivirus, also referred to as the Human Immunodeficiency Virus (HIV), is the causative agent of Acquired Immunodeficiency Syndrome (AIDS). The human immunodeficiency virus (HIV) attacks $CD4^+T$ lymphocytes, which are normally white blood cells thought to be responsible for clearing invasive microorganisms, so seriously impairing the body's immune system. A normal, healthy person's $CD4^+T$ cell level is between 800 and $1200mm^3$ and patients with HIV infection

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are considered to have acquired immunodeficiency syndrome (AIDS) if this number drops below $200mm^3$ [1–3].The HIV infection can spread from one individual to another by the use of a person's blood, semen, vaginal fluids or breast milk. The most common routes to spread HIV are through unprotected sexual contact and sharing injecting equipment, including needles and syringes, with an HIV-positive individual [4]. If the mother is HIV positive, the infant may contract the virus after birth or through breastfeeding; this is referred to as vertical transmission [5–8]. Adults with HIV infection may appear healthy for years before developing AIDS [9]. Symptoms of human immunodeficiency differ according to the infection's stage. People who are HIV positive may not exhibit any clinical signs of the virus during the early stages of the infection, which begin a few weeks after the first infection and include fever, headaches, and sore throats. Other indications and symptoms of immunosuppression include fever, diarrhea, coughing, enlarged lymph nodes, and weight loss [3]. HIV continues to be a problem for public health worldwide [10]. Over 79.4 million people have contracted the virus since it first appeared and 40.4 million of those cases have resulted in deaths. The virus is still spreading throughout the world [3]. Individuals from low- and middle-income nations make up the bulk of the global HIV infection [11].HIV infection does not have a cure. Nonetheless, one of the suggested treatments for HIV infection is the use of antiretroviral therapy (ART) [12]. ART is taking a combination of two or more anti-HIV drugs daily or as directed by a doctor, depending on the number of $CD4^+ - T$cells. Highly active anti-retroviral therapy (HAART) is the process of mixing two or more antiretroviral drugs. Among the drugs are emtricitabine (FIC), zidovudine (AZT) and highly active anti-retroviral therapy (HAART) [13]. Ninety-five percent of all persons living with HIV (PLHIV) should be diagnosed by 2025, ninety-five percent of those diagnosed should be receiving life-saving antiretroviral therapy (ART) and ninety-five percent of PLHIV receiving treatment should have a suppressed viral load for their own health and to prevent HIV from spreading to others [14].

Mathematical modelling has become an important tool in studying the transmission dynamics of infectious diseases and more and more researchers worldwide are using it as an invaluable tool to study health-related problems connected to the dynamics of infectious disease [15–22]. Numerous models have been developed

to examine the dynamics of HIV/AIDS with vertical transmission in relation to efforts for treatment and control [23–30]. [31] considered counseling, vaccination and ART as control measures for the lentivirus. [32] developed a mathematical model for an efficient management of the HIV epidemic while according to [33] analysis, raising public awareness about HIV/AIDS can be beneficial in lowering the disease's prevalence within the general population.An ideal control framework for the dynamics of HIV/AIDS transmission was provided by [34]. A mathematical model featuring drug resistance compartment was developed by [35] to explain HIV/AIDS transmission. Research on the effects of delayed diagnosis on HIV/AIDS was conducted by [36]. In order to lower the prevalence of HIV infection, the results collected indicated that significant effort should be focused on encouraging early ART initiation. [37] used Ethiopia as a case study to examine the best ways to prevent HIV/AIDS transmission. Their findings showed that various control strategy combinations reduced the number of infectious people with AIDS symptoms who were both diagnosed and undetected. Early treatment of people who are latently infected lowers the dynamical development to full-blown AIDS, according to [38]. [11] offered mathematical analysis for HIV infections with public knowledge and detectability of viral load. According to their study, raising public knowledge about HIV infection can help stop its transmission. Additionally, when therapy is given to an infected person with a detectable viral load, the virus can be readily suppressed to become undetectable, preventing HIV from being shared through sexual activity. A nonlinear fractional order for HIV transmission dynamics with optimal control was suggested by [39]. The study recommended that in order to reduce the spread of HIV infections there is need for personal precaution and periodic monitoring by medical practitioners.The dynamics of HIV/AIDS transmission for both vertical and non-linear treatment were examined in the [24] study. Their study found that reducing the prevalence of HIV transmission would be greatly aided by early identification of HIV infection.

The organization of the work is as follows: Section (2) presents the full description of model. The analysis of the model is carried out in Section (3), while in Section (4), the numerical simulations of the system are performed. Section (5) wraps up the work with concluding remarks.

2. THE HIV/AIDS MODEL

The total human population denoted $N(t)$ is sub-divided into four compartments of individuals namely, susceptible class $S(t)$ (those who are not infected but are prone to contracting HIV infection), asymptomatic infected class $I(t)$ (those who have contracted HIV infection but have not shown sysmptoms), symptomatic infected class $I_s(t)$ (individuals who have contracted HIV and have shown symptoms) and the full blown AIDS class $A(t)$ (those who have developed AIDS). Then the total population is obtained as $N(t) = S(t) + I(t) + I_s(t) + A(t)$

Let π be the recruitment rate of susceptible individuals into the population. The effective contact rate with the probability that susceptible individuals are being infected with HIV per contact with infected and AIDS individuals are $c\beta$ and $d\beta$. μ represents the natural mortality rate experienced by every compartment of the population. The incidence rate $(cI + dA)\beta S$ increases the asymptomatic infectious human population and reduces it due to progression to the full blown AIDS class at the rate σ. The symptomatic infectious population is increased due to progression from asymptomatic infectious population and vertical transmission at the rates α and a. The full blown AIDS compartment increases at the rate γ and reduced due to disease induced death at the rate δ. Then the mathematical model governing the dynamics of HIV/AIDS is given as:

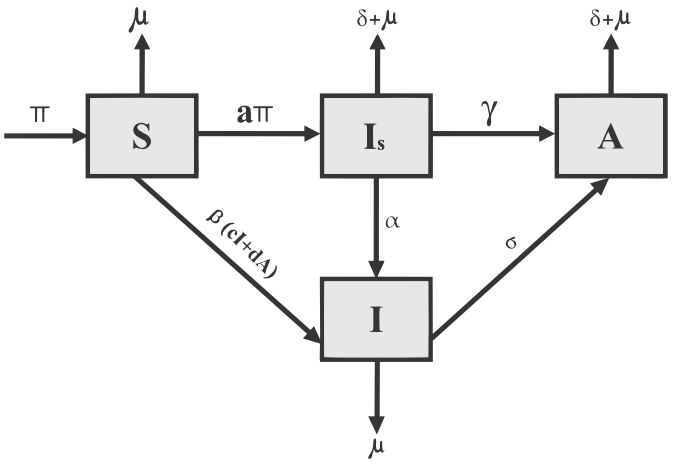

FIGURE 1. The schematic diagram describing the dynamical spread of the HIV/AIDS model

$$
\begin{aligned}
\frac{dS}{dt} &= \pi - a\pi I_s - (cI + dA)\beta S - \mu S \\
\frac{dI}{dt} &= (cI + dA)\beta S - (\sigma + \alpha + \mu)I \\
\frac{dI_s}{dt} &= \alpha I + a\pi I_s - (\delta + \gamma + \mu)I_s \\
\frac{dA}{dt} &= \gamma I_s + \sigma I - (\delta + \mu)
\end{aligned} \tag{1}
$$

The state variables (1) are subject to the initial conditions:

$$S(t) > 0, \quad I(t) \geq 0, \quad I_s \geq 0, \quad A(t) \geq 0.$$

TABLE 1. The description of variables of the HIV/AIDS model

Variable	Definition
$S(t)$	Susceptible Individuals
$I(t)$	Asymptomatic Infected Individuals
$T_s(t)$	Symptomatic Infected Individuals
$A(t)$	Full Blown AIDS Individuals

TABLE 2. The description of parameters of the HIV/AIDS model

Parameter	Description
π	Recruitment rate
β	Transmission rate
δ	HIV/AIDS induced death rate
μ	Natural mortality rate
σ	Rate at which symptomatic individuals becomes AIDS individuals
α	Rate at which asymptomatic individuals become symptomatic
γ	Rate at which symptomatic individuals become AIDS individual
a	ertical transmission rate
c	Probability of disease transmission per contact with an asymptomatic individual

3. Analysis Of The HIV/AIDS model

3.1. **Invariant region.**

Theorem 3.1. *The feasible region of the HIV model* (1) *given by* $D \subset \mathbb{R}^4_+$ *where* $\{D = (S, I, I_s, A) \in \mathbb{R}^4 : S + I + I_s + A\}$ *is positively invariant and attracting.*

Proof. To establish the invariant region of the HIV model, the total human population is added together such that the rate of change of total population denoted by $N(t)$ is defined as

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dI_s}{dt} + \frac{dA}{dt}. \tag{2}$$

Then the rate of change of total population becomes

$$\frac{dN}{dt} = \pi - \mu(S + I + I_s + A) - \delta(I_s + A). \tag{3}$$

In the absence of HIV induced death and solving by standard technique, it follows that

$$N(t) \leq N(0)e^{-\mu t} + \frac{\pi}{\mu}(1 - e^{\mu t}). \tag{4}$$

Therefore if $N(0) \leq \frac{\pi}{\mu}$, then $N(t) \leq \frac{\pi}{\mu}$ for all $t > 0$. Hence, the biologically feasible region D of the HIV model is positively invariant. Furthermore, if $N(0) \geq 0$, then the solution enters D in finite time or $N(t)$ approaches $\frac{\pi}{\mu}$ asymptotically as $t \to \infty$. Hence the region D attracts all solutions in $\mathbb{R}^4_+$ □

3.2. **Positivity and Boundedness of solutions.**

The mathematical model (1) considers only human population, then it is pertinent that all its state variables and associated parameters are positive for all time t. Therefore, the following result holds for all the state variables in the mathematical model (1).

Theorem 3.2. *The solution set* $\{S, I, I_s, A\}$ *of the HIV model* (1) *with positive initial conditions* $S(0), I(0), I_s(0), A(0)$ *in the region* D *remain positive in* D *for all time* $t > 0$.

Proof. Considering the first compartment of the model (1) so that

$$\frac{dS}{dt} + \beta((cI + dA) + \mu)S. \tag{5}$$

This implies that

$$S(t) \geq S(0)exp\left[-\int_0^t (cI(w) + dA(w))dw + \mu(t)\right] > 0, \quad \text{for all} \quad t > 0. \tag{6}$$

□

Following the same procedure, it can be shown that the remaining state variables $I(0) > 0, I_s > 0, A(0) > 0 \; \forall \; t > 0$.

3.3. **Equilibrium points and Stability Analysis.**

3.3.1. *Disease-free equilibrium.* The HIV-free equilibrium point of the model (1) is a state where there is absence of HIV infection in the population. It is determined by setting the disease related variables to zero. Solving the related system of equation, the HIV model (1) has a disease-free equilibrium point

$$\mathcal{E}_0 = (S, I, I_s, A) = (\frac{\pi}{\mu}, 0, 0, 0). \tag{7}$$

After establishing the disease-free equilibrium, we proceeded to calculating the basic reproduction number, denoted by $\mathcal{R}_0$. The basic reproduction number is defined as the average number of new cases of secondary infection which is caused by an infectious individual during the period of infectiousness in the population of susceptible. The basic reproduction number is used to obtain the threshold conditions under which the incidence of human immunodeficiency virus persists or dies out if a small number of HIV actively infected human is introduced into a completely susceptible environment. The $\mathcal{R}_0$ is calculated using the next generation matrix approach [40, 41], where the infected compartments of the model (1) are considered at $\mathcal{E}_0$. The transmission matrix F and transition matrix V obtained at $\mathcal{E}_0$ are given as follows

$$F = \begin{pmatrix} \frac{c\beta\pi}{\mu} & 0 & \frac{d\beta\pi}{k_1} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} k_1 & 0 & 0 \\ -\alpha & k_2 - a\pi & 0 \\ -\sigma & -\gamma & k_{3s} \end{pmatrix},$$

where $k_1 = (\sigma + \alpha + \mu)$, $k_2 = (\gamma + \delta + \mu)$, $k_3 = (\delta + \mu)$. It therefore follows that the spectral radius of FV^{-1} is the basic reproduction number given as

$$\mathcal{R}_0 = \frac{\beta\pi\{(k_2 - a\pi)(ck_3 + d\sigma) + d\alpha\gamma\}}{\mu k_1 k_3 (k_2 - a\pi)}. \tag{8}$$

Lemma 3.1. *The HIV-free equilibrium of the model* (1) *is locally asymptotically stable whenever the threshold parameter* $\mathcal{R}_0 < 1$ *and unstable otherwise.*

Proof. The Jacobian matrix of the HIV model (1) evaluated at $\mathcal{E}_0$ is obtained as

$$V = \begin{pmatrix} -\mu & -\frac{c\beta\pi}{\mu} & -a\pi & -\frac{d\beta\pi}{\mu} \\ 0 & \frac{c\beta\pi}{\mu} - k_1 & 0 & \frac{d\beta\pi}{\mu} \\ 0 & \alpha & a\pi - k_2 & 0 \\ 0 & \sigma & \gamma & -k_3 \end{pmatrix}. \tag{9}$$

Clearly, one of the eigenvalues of (9) is obtained as $\lambda_1 = -\mu$ and the remaining are obtained from the polynomial given by

$$\lambda^3 + B_1\lambda^2 + B_2\lambda + B_3 = 0, \tag{10}$$

where

$$B_1 = k_1 + k_2 + k_3 - \frac{c\beta\pi}{\mu} > 0,$$

$$B_2 = k_1(k_2 + k_3) + k_3(k_2 - a\pi) - \frac{\beta\pi\{(k_2 + k_3 - d\sigma)\}}{\mu} > 0,$$

$$B_3 = \mu k_1 k_2 (k_2 - a\pi)(1 - \mathcal{R}_0).$$

Apparently, all the coefficients of the eigenvalues of the polynomial (3.9) are positive. Then by Descarte's rule of sign, it follows that all the eigenvalues are negative, real and distinct. Hence the disease-free equilibrium of the HIV model (1) is locally asymptotically stable. □

3.3.2. *Endemic equilibrium.* The endemic equilibrium point of the model (1) is determined to provide more insights into the long term effects of the spread dynamics of HIV/AIDS in the population. It is a steady state solution where there is presence of disease in the population. Let the endemic equilibrium point of the model be denoted by $\mathcal{E}^{**} = (S^*, I^*, I_s^*, A^*)$ and $\lambda^{**} = \beta(cI^* + dA^*)$ represent the force of infection of the model. Then solving (1) simultaneously at steady state

yields the following:

$$\mathcal{E}^{**} = \begin{matrix} S^* = \frac{\pi k_1(k_2-a\pi)}{k_1(k_2-a\pi)(\lambda^{**}+\mu)+a\pi\alpha\lambda^{**}} \\ I^* = \frac{\pi(k_2-a\pi)\lambda^{**}}{k_1(k_2-a\pi)(\lambda^{**}+\mu)+a\pi\alpha\lambda^{**}} \\ I_s^* = \frac{\alpha\pi\lambda^{**}}{k_1(k_2-a\pi)(\lambda^{**}+\mu)+a\pi\alpha\lambda^{**}} \\ A^* = \frac{\lambda^{**}(\gamma\alpha+\sigma(k_2-a\pi))}{k_3\{k_1(k_2-a\pi)(\lambda^{**}+\mu)+a\pi\alpha\lambda^{**}\}} \end{matrix} . \tag{11}$$

By substituting I^* and A^* into the force of infection, the following result is obtained

$$\lambda^{**} = \frac{\mu k_1 k_3(k_2 - a\pi)(\mathcal{R}_0 - 1)}{k_1 k_3(k_2 - a\pi) + a\alpha\pi}. \tag{12}$$

Hence, the endemic equilibrium point exists whenever $\mathcal{R}_0 > 1$ in (3.11).

3.3.3. *Global Stability of Disease-free equilibrium.* In this part, the global asymptotic stability of the HIV/AIDS model (1) is explored using the approach of [17, 24, 42–44]. The model can be transformed into the form:

$$\begin{matrix} \frac{dX}{dt} = F(X, Z) \\ \frac{dZ}{dt} = G(X, Z), G(X, 0) \end{matrix} , \tag{13}$$

where $X \in \mathbb{R}$ and $Z \in \mathbb{R}^3_+$. The X component represent the uninfected compartment of the HIV/AIDS model (1) and the Z components represent the infected compartments of the system respectively. Then the following properties must be satisfied in order to establish the global stability of the disease-free equilibrium of the system (1):

$R_1 :$ For $\frac{dX}{dt} = F(X^*, 0)$, X^* is globally asymptotically stable.

$R_2 :$ $G(X, Z) = AZ - \hat{G}(X, Z)$, $\hat{G}(X, G) \geq 0$ for $(X, Z) \in D$,

where $A = \frac{\partial G}{\partial Z}$ is an M-matrix evaluated at $(X^*, 0)$ whose off-diagonal elements are non-negative

Theorem 3.3. *The disease-free equilibrium point $\mathcal{E}_0$ of the HIV/AIDS system* (1) *is globally asymptotically stable provided $\mathcal{R}_0 \leq 1$ and, properties R_1 and R_2 are satisfied.*

Proof. $F(X, Z)$ and $G(X, Z)$ are obtained from the system (1) as

$$F(X, Z) = (\pi - a\pi I_s - \beta S(cI + dA) - \mu S) \tag{14}$$

and

$$G(X,Z) = \begin{pmatrix} \beta S(cI + dA) - (\sigma + \alpha + \mu) I \\ \alpha I + a\pi I_s - (\delta + \gamma + \mu) I_s \\ \sigma I + \gamma I_s - (\delta + \mu) A \end{pmatrix} \tag{15}$$

such that $F(X,0) = (\pi - \mu S)$. Then $\frac{dX}{dt} = F(X,0)$ implies that

$$\frac{dS}{dt} = \pi - \mu S. \tag{16}$$

Solving by standard technique yields

$$S(t) = \frac{\pi}{\mu}\left(1 - e^{\mu t}\right) + S(0)\, e^{-\mu t}. \tag{17}$$

Irrespective of the size of $S(0)$, as $t \to \infty$, then $S(t) \to \frac{\pi}{\mu}$. Therefore, the disease-free equilibrium point $(X^*, 0)$ is globally asymptotically stable satisfying property R_1.

Furthermore, the M-matrix with non-negative off-diagonal element is given by

$$A = \frac{\partial G}{\partial Z} = \begin{pmatrix} c\beta S^0 - (\alpha + \sigma + \mu) & 0 & d\beta S^0 \\ \alpha & a\pi - (\gamma + \delta + \mu) & 0 \\ \sigma & \gamma & -(\delta + \mu) \end{pmatrix}, \tag{18}$$

where $S^0 = \frac{\pi}{\mu}$. Then, it follows that $AZ - \hat{G}(X,Z)$ is obtained as

$$\hat{G}(X,Z) = \begin{pmatrix} \beta(cI + dA)(S^0 - S) \\ 0 \\ 0 \end{pmatrix}. \tag{19}$$

Since $0 \leq S \leq \frac{\pi}{\mu}$, it is apparent that $\hat{G} \geq 0$ satisfying property R_2. Therefore the disease-free equilibrium of the HIV/AIDS model (1) is globally asymptotically stable. This ends the proof. □

3.3.4. *Global Stability of the Endemic equilibrium.* The global asymptotic stability of the HIV/AIDS model (1) around the endemic equilibrium is explored using the approach of [22, 25].

Theorem 3.4. *The endemic equilibrium point of the HIV/AIDS model* (1) *is globally asymptotically stable whenever* $\mathcal{R}_0 > 1$.

Proof. Consider the quadratic Lyapunov function $\mathcal{L} : D \in \mathbb{R}_+^4 \to \mathbb{R}_+$ defined by

$$\mathcal{L} = \frac{1}{2}\{(S - S^{**}) + (I - I^{**}) + (I_s - I_s^{**}) + (A - A^{**})\}^2. \tag{20}$$

Taking the derivative of (20) gives

$$\begin{aligned}
\dot{\mathcal{L}} &= \{(S - S^{**}) + (I - I^{**}) + (I_s - I_s^{**}) + (A - A^{**})\}\frac{d}{dt}(S + I + I_s + A) \\
&= \{(S - S^{**}) + (I - I^{**}) + (I_s - I_s^{**}) \\
&\quad + (A - A^{**})\}\{\pi - \mu(S + I + I_s + A) - \delta(I_s + A) \\
\dot{\mathcal{L}} &\leq \{(S - S^{**}) + (I - I^{**}) + (I_s - I_s^{**}) \\
&\quad + (A - A^{**})\}\{\pi - \mu(S + I + I_s + A)\} \qquad (21) \\
&= \mu\{(S - S^{**}) + (I - I^{**}) + (I_s - I_s^{**}) \\
&\quad + (A - A^{**})\}\{(S + I + I_s + A) - \frac{\pi}{\mu}\}
\end{aligned}$$

Since $N^{**} = \frac{\pi}{\mu}$, then (21) becomes

$$\begin{aligned}
\dot{\mathcal{L}} &= \{(S - S^{**}) + (I - I^{**}) + (I_s - I_s^{**}) + (A - A^{**})\} \\
&\quad \times \{(S + I + I_s + A) - (S^{**} + I^{**} + I_s^{**} + A^{**})\} \\
&= -\mu\{(S - S^{**}) + (I - I^{**}) + (I - I_s^{**}) + (A - A^{**})\} \qquad (22) \\
&\quad \times \{(S - S^{**}) + (I - I^{**}) + (I - I_s^{**}) + (A - A^{**})\} \\
&= -\mu\{(S - S^{**}) + (I - I^{**}) + (I - I_s^{**}) + (A - A^{**})\}^2.
\end{aligned}$$

Proportionately, the time derivative of the continuosly differentiable function $\mathcal{L}$ is negative semi-definite. That is $\dot{\mathcal{L}} \leq 0$. Then, the function $\mathcal{L}$ is a Lyapunov function. Therefore, $\dot{\mathcal{L}} = 0$ provided $S = S^{**}$, $I = I^{**}$, $I_s = I_s^{**}$ and $A = A^{**}$. Then, by LaSalle's invariance principle [45], the largest invariance set for which $\dot{\mathcal{L}} = 0$ is the singleton set $\{\mathcal{E}^{**}\}$, which implies that the endemic equilibrium point of the HIV/AIDS model (1) is globally asymptotically stable. □

3.3.5. *Sensitivity Analysis.* The influence of parameters of the HIV model relative to the basic reproduction number is examined through sensitivity analysis using the normalized forward sensitivity index. By definition, the normalized forward sensitivity index of a variable to a parameter is the ratio of the relative change in the variable to the relative change in the parameter [46]. Sensitivity analysis is a salient tool used for measuring the effect and contribution of each parameter of the basic reproduction number to the output of the model.The normalized forward sensitivity indices of the basic reproduction number $\mathcal{R}_0$, relative to its parameter q is given by

$$\mathcal{X}_p^{R_0} = \frac{\partial \mathcal{R}_0}{\partial q} \times \frac{q}{\mathcal{R}_0}. \tag{23}$$

The sensitivity indices of the associated parameters of the basic reproduction number is presented in Table 3 where the associated parameters are shown to have either increasing or decreasing influence on $\mathcal{R}_0$.

TABLE 3. The parameter values for optimality system simulations

Parameters	Value	Sensitivity Indices	References
π	0.14	+1.0000	[47]
μ	0.5 yr^{-1}	-1.9327	[26]
β	0.0003	+1.0000	[49]
γ	0.4	+0.0063	[48]
δ	0.02	-0.0009	[49]
d	1.0 yr^{-1}	+0.0178	[26,49]
σ	0.01	-0.0118	[49]
α	0.04 yr^{-1}	-0.0613	[26]
a	0.3	+0.0005	[49]
c	3.0 yr^{-1}	1.0000	[49]

4. NUMERICAL SIMULATIONS

Numerical simulations of the HIV/AIDS model (1) were performed with the aid of MATLAB computing software in order to corroborate the analytical results established in section (3) and the results are presented graphically.

As presented in Figure 2, the effect of parameters representing transmission probability of disease per contact by an AIDS individual d and transmission probability of disease per contact by an asymptomatic individual c on the basic reproduction number of the HIV. It can be seen that increase in the values of both d and c increases the value of the basic reproduction number. This is suggesting that having close contact with symptomatically infected HIV individual and full blown AIDS individual will enhance the prevalence of HIV in the population. In a similar manner, Figure 3 depicts the plot of the basic reproduction number against the parameters representing probability of disease transmission at birth a and effective contact rate β. It is observed that increment in vertical transmission rate and effective contact rate increases the value of the basic reproduction number. The implication of this from the epidemiological viewpoint is that HIV will continue to persist in the population if effective control measures are not put in place.

Figure 4 is showing how both progression of asymptomatically infected individual to full blown AIDS class σ and effective contact rate β affect the basic reproduction number of the HIV model. It can be deduced that increase in the values of these parameters will lead to a significant increase in the value of the basic reproduction number. This from the epidemiological point of view is suggesting that HIV dynamics cannot be managed in the population if efforts are not set out to curb the dynamics of HIV/AIDS in the population. As shown in Figure 5, it is observed that the population of asymptomatic HIV infected individuals converge to the HIV/AIDS-free equilibrium. The implication of this is that HIV/AIDS can be reduced in the population regardless of the initial size of the population of the model. Figure 6 depicts the global asymptotic behaviour of symptomatically infected HIV individuals around the endemic equilibrium. It is observed that irrespective of the initial size of the population, the symptomatic population of infected individuals will converge to a unique endemic equilibrium point.

5. Conclusion

A mathematical model for the dynamical spread of human immunodeficiency virus (HIV) with vertical transmission was formulated and analysed in this study. Using a system of ordinary differential equation, the model was stratified into four compartments of susceptible, asymptomatic, symptomatic and full blown AIDS

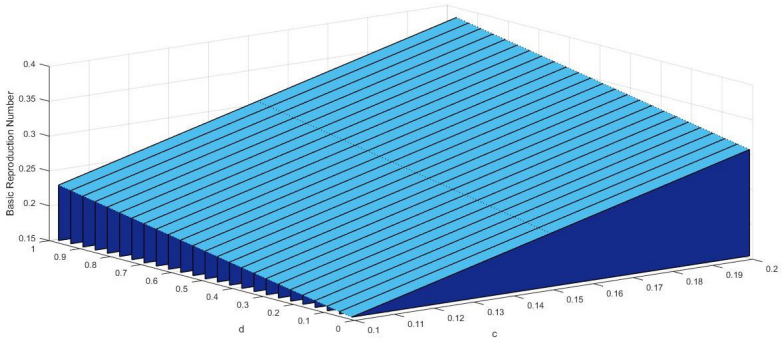

FIGURE 2. 3-D plot showing the influence of d and c on the basic reproduction number

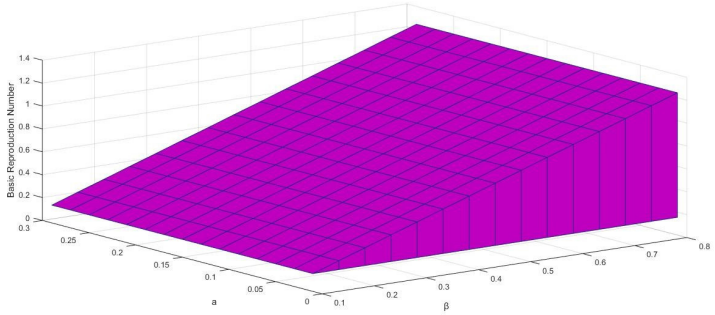

FIGURE 3. Effect of a and β on the basic reproduction number.

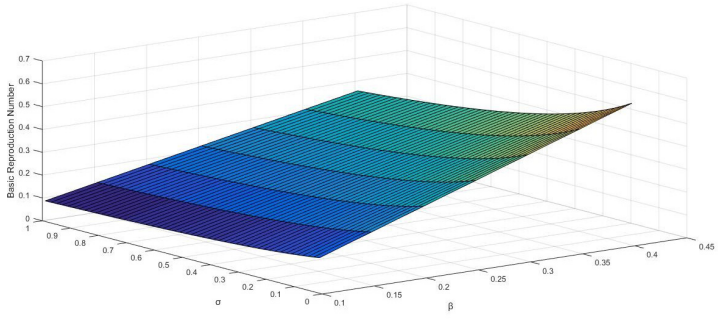

FIGURE 4. 3-D plot showing the influence of σ and β on the basic reproduction number.

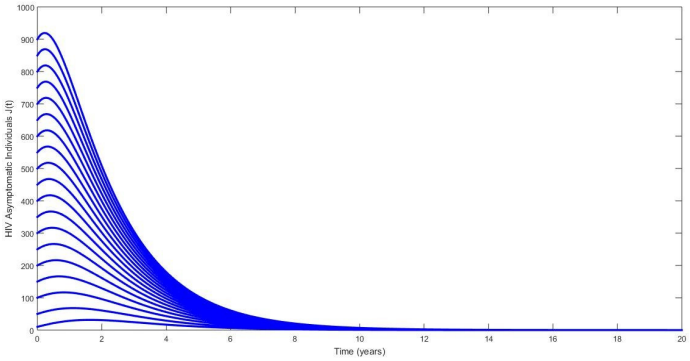

FIGURE 5. Global asymptotic behaviour of the symptomatically infected HIV individual around the disease-free equilibrium.

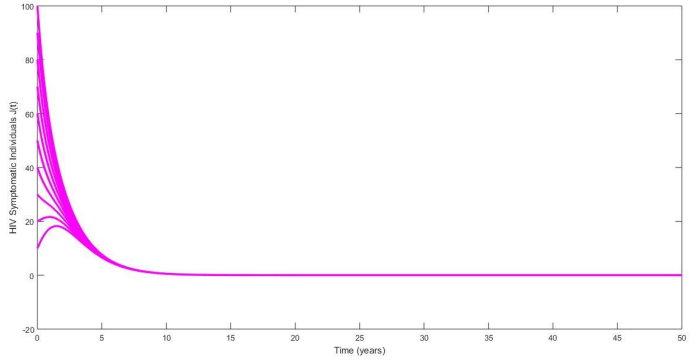

FIGURE 6. Global asymptotic behaviour of the symptomatically infected HIV individual around the endemic equilibrium point.

individuals respectively. The analytical solution of the model revealed that the equilibria states of the model were investigated and the basic reproduction number was obtained using the next generation matrix approach. The disease-free equilibrium is both locally and globally asymptotically stable when the basic reproduction number is less than unity using the linearized Jacobian and M-matrix methods. Furthermore, the global asymptotic stability of the endemic equilibrium point was established using a quadratic Lyapunov function and it was shown that HIV/AIDS will persist in the population whenever $\mathcal{R}_0 > 1$. Moreover, it is pertinent to state that the parameters used for the numerical simulation of the model

were hypothetically chosen. However, real data of HIV/AIDS cases could be used to fit the model for a more realistic study.

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