ISSN 2581-3463

AJMS
Asian Journal of Mathematical Sciences

RESEARCH ARTICLE

Analysis of Global Stability of Endemic Equilibrium of HIV/AIDS with Drug-resistant Compartment Using Lasalle's Invariant Principle

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Received: 20-03-2021; Revised: 25-04-2022; Accepted: 20-05-2022

ABSTRACT

All over the world, human immunodeficiency virus (HIV)/AIDS has continued to be a major threat to humanity resulting in death of millions annually across the globe despite countless research works and readiness of anti-retroviral drugs as means of combating the menace. This research formulated a mathematical model to study the spread dynamics of HIV/AIDS in the presence of highly effective antiretroviral treatment (HART) incorporating HART resistance compartment. The basic reproduction number was obtained using the next generation matrix method. The endemic equilibrium of the model was analyzed using the Lasalle's invariant principle and it was found to be globally asymptotically stable.

Key words: Effective reproduction number R_0, Endemic Equilibria, Globally asymptotically stable, Simulation, Lasalle's invariant principle

INTRODUCTION

Human immunodeficiency virus (HIV) is an infection which terminates the body resistant systems, increases the risk of certain pathologies, harms body organs such as the brain, kidney, and heart, and causes death. AIDS which is the late stage of untreated HIV is one of the leading epidemics in the world.[1,2] As at 2016, about 36.7 million people were living with HIV/AIDS globally, out of which only 20.9 million people were having access to the antiretroviral treatment. 1.18 million people became newly infected. Totally, about 78 million people have been infected with HIV since the discovery of epidemics and about 35 million people died from HIV/AIDS related cases. Globally, there are 2.1 million people who have recently been infected. In Nigeria, about 3.5 million persons are reported to be carrying the deadly virus. Mortality tolls at 180000 individuals both old and young, with a prevalent rate of 3.1% among adults aged 15 and above.[3] HIV/AIDS can be contracted through unprotected sex with an infected person, breast milk of an infected woman, blood transfusion from an infected person, and sharing of sharp objects with infected persons.[4,5]

Developed and studied the distribution model of the human immunity deficiency virus (HIV) that includes dynamics in the information of risk groups due to the rise in HIV cases in Russia. In their work, they proposed a model of virus transmission in a population with a dynamic risk due to alcoholism. Their models comprises eight compartments, namely, socially adapted receptive group S_G, increased risk of addiction receptive group S_B, chronic alcoholism receptive group S_A, drug addiction receptive group S_D, socially adapted infected group I_G, increased risk of addiction infected group I_B, chronic alcoholism infected group I_A, and drug addiction receptive group I_D. The result shows that there is need to take into consideration the social processes in the description of the epidemiology of such infections as HIV. Tentative research proposes that coinfection may be accountable for upsurges seen in set-point viral

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load (spVL) at the within-host level over time.[6] These increase due to coinfection.[7] Furthermore, the concentrations of coinfection in high-risk groups versus low-risk groups may affect how HIV spreads in the general population.[8] To study the tools responsible for these effects of coinfection, an immune epidemiological model was developed.[9] They found that populations with higher spVL lead to higher increase in viral load due to coinfection, whereas populations with lower spVL lead to lower increases in viral load due to coinfection. This leads to a greater chance of coinfection increasing the occurrence of HIV in populations with high average spVL.[9] Therefore, the effects of coinfection may be alleviated by detecting the viral factors that can shrink the spVL in the population.[10]

Developed HIV transmission dynamics between drug-sensitive and drug-resistant infected individuals. Their model consisted of six compartments, namely, susceptible population S, number of individuals infected with drug-resistant strain and do not receive treatment I_{DRO}, number of individuals infected with drug-resistant strain and receive treatment I_{DRT}, number of individuals infected with drug-sensitive strain and do not receive treatment I_{DSO}, number of individuals infected with drug-sensitive strain and receive treatment I_{DST}, and number of individuals infected with drug-sensitive strain, receive treatment, and develop resistance I_{DSTr}. Their result shows that HIV immune epidemiological models simulate viral immune dynamics at the within-host scale and the epidemiological transmission dynamics at the between-host scale. They account for longitudinal changes in the immune viral dynamics of HIV positive individuals and their corresponding impact on the spread in the population.

In the context of HIV evolution, while the transmission rate varies through time depending on the viral load,[7] formulated a model to distinguish between different strains. The transmission rate depends on a predefined infectivity profile which changes depending on the stage of infection, and the frequency of the different viral strains in an infected population. They made the transmission rate depend on the frequency of viral strains that were only in actively infected CD4+ T-cells. Their result shows that the presence of a latent reservoir can severely delay within-host evolutionary dynamics. This delay increases with the relative size of the reservoir and the rate at which latently infected cells proliferate. Hence, after nesting the within-host model into an epidemiological model of population dynamics, it was observed that the presence of latent reservoirs can also influence the population-level evolution of the virus.[11]

Similarly[7] researched on the stability analysis for a two-sex mathematical model of HIV where they were interested in the mathematical analysis of the equilibrium points of their model equations. They also formulated theorems based on the basic reproduction number R_0. They found out in their result that virus infection is temporal and can be cleared if $R_0<1$ and other measures such as improved HIV vaccines, ART, and awareness programs are intensified simultaneously to control the transmission rate of the infection. Hence, this research lauds all those who have researched on HIV/AIDS model and extends their works by bridging one of the gaps they left, by introducing drug resistance class into the model.

Model Formulation

Following,[10] the total human population is divided into five compartments: Susceptible individuals (S), infectious individuals who are not receiving HART (I_1), infectious individuals who receives treatment but are HART resistant (I_2), infectious individual who receives treatment and are HART non-resistant (I_3), and individuals who are down with AIDS compartment (A).

In formulating our model, the following assumptions were made:

i. It is assumed that every human who are sexually active coming into the population are susceptible to HIV
ii. A person can only get HIV through unprotected sex with HIV infected human
iii. Only three HIV infected classes are capable of spreading the virus within the population: Infectious individuals (I_1), infectious individuals who receives treatment but are HART resistant (I_2), and infectious individuals who receives treatment and are HART non-resistant (I_3). AIDS (A) individuals are too weak to have sex, hence cannot transmit the virus.

The model schematic diagram is as shown in Figure 1:

The susceptible individuals (S) are generated through constant recruitment of sexually active individuals by immigration at the rate Λ, decreases due to effective contact with I_1, I_2, and I_3 given by the infection force

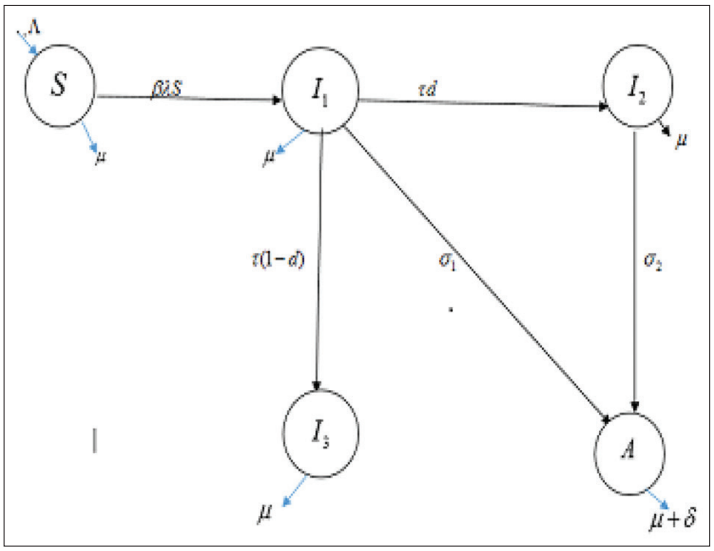

Figure 1: Schematic diagram of HIV/AIDS transmission dynamics with drug resistance compartment

$$\lambda = \frac{\beta(1-\varepsilon c)[(I_1 + I_2) + \eta I_3]}{N} \tag{1}$$

Where β is the effective contact rate, ε is the efficacy of HART, c is the compliance level to HART usage, and η is the modification parameter associated with reduced infectiousness of I_3 individuals compared to I_1 and I_2. The compartment, further, decreases due to natural death at the rate μ.

The infectious individuals who are not receiving treatment I_1 are generated through effective contact between S, I_1, I_2, and I_3 as earlier explained. They decrease when they started receiving treatment, or due to progression to AIDS at the rate τ and σ_1, respectively. They, further, decrease due to natural mortality at the rate μ.

The HART resistant population I_2 are generated when the infectious class I_1 started receiving treatment at the rate τ, but certain individuals in the compartment are resistant to the HART at the rate d. They decreases due to progression to AIDS at the rate σ_2 and further decreases due to natural mortality.

The HART non-resistant population I_3 is generated when the infectious class I_1 is treated at the rate τ as earlier mentioned and they are not resistant to the treatment at the rate (1-d). They only decrease due to natural death.

The AIDS population A is generated from the progression of non-treated infectious individuals and HART resistant individuals at the rate σ_1 and σ_2, respectively. They are reduced due to natural death or disease induced death at the rate μ and δ, respectively. Table 1 present the baseline values for variables of the HIV/AIDS in Nigeria. The Table 2 shows the baseline values for parameters of the HIV/AIDS.

The Model Equations are as Shown

$$\frac{dS}{dt} = \Lambda - \frac{\beta(1-\varepsilon c)[I_1 + \eta(I_2 + I_3)]\mathrm{S}}{N} - \mu S \tag{2}$$

$$\frac{dI_1}{dt} = \frac{\beta(1-\varepsilon c)[I_1 + \eta(I_2 + I_3)]\mathrm{S}}{N} - (\tau + \sigma_1 + \mu)I_1 \tag{3}$$

$$\frac{dI_2}{dt} = (\tau + \phi)dI_1 - (\sigma_2 + \mu)I_2 \tag{4}$$

$$\frac{dI_3}{dt} = (\tau + \phi)(1-d)I_1 - \mu I_3 \tag{5}$$

$$\frac{dA}{dt} = \sigma_1 I_1 + \sigma_2 I_2 - (\mu + \delta)\,\mathrm{A} \tag{6}$$

Where

$$N = S + I_1 + I_2 + I_3 + A \tag{7}$$

So that

$$\frac{dN}{dt} = \Lambda - \mu N - \delta A \tag{8}$$

Since the model is dealing with populations, all the variables and parameters of the model are positive with the natural death rates positive, that is, ($\mu > 0$), thus considering the region Ω where:

$$\Omega = \{(\mathrm{S}, \mathrm{I}_1, \mathrm{I}_2, \mathrm{I}_3, \mathrm{A}) \in \Re^5 : \mathrm{S}, \mathrm{I}_1, \mathrm{I}_2, \mathrm{I}_3, \mathrm{A} \geq 0,\} \tag{9}$$

It can be established that all solutions of the system starting in Ω remain in Ω for all t>0. In this region, the usual existence, uniqueness, and continuation of results hold for the system.

Basic Reproduction Number, R_0

The basic reproduction number is the average number of secondary infections caused by a single infectious individual during his/her entire infectious life time. Using the next generation matrix operator to compute the model's Basic Reproduction Number, as used by[5][4] and improved by[13][14]. The basic reproduction number is obtained by dividing the whole population into n compartments, in which there are $m<n$ infected compartments. Let $x_i, i=1,2,3,\ldots,m$ be the number of infected individuals in the i^{th} infected compartment at time t. The largest eigenvalue or spectra radius of FV^{-1} is the basic reproduction number of the model.

$$FV^{-1} = \left[\frac{\partial F_i(E^0)}{\partial x_i}\right]\left[\frac{\partial V_i(E^0)}{\partial x_i}\right]^{-1} \tag{10}$$

Where F_i is the rate of appearance of new infection in compartment i to another and E^0 is the disease-Free Equilibrium.

$$F = \begin{pmatrix} \beta(1-\varepsilon c) & \beta(1-\varepsilon c) & \beta(1-\varepsilon c)\eta & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{11}$$

$$V = \begin{pmatrix} K_1 & 0 & 0 & 0 \\ -\tau d & K_2 & 0 & 0 \\ -\tau(1-d) & 0 & \mu & 0 \\ -\sigma_1 & -\sigma_2 & 0 & K_3 \end{pmatrix} \tag{12}$$

$$FV^{-1} = \begin{pmatrix} \dfrac{\beta(1-\varepsilon c)[\mu K_2 + \tau d\mu + \tau\eta K_2(1-d)]}{\mu K_1 K_2} & \dfrac{\beta(1-\varepsilon c)}{K_2} & \dfrac{\beta(1-\varepsilon c)\eta}{\mu} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{13}$$

$$\left| fv^{-1} - \lambda I \right| = 0 \tag{14}$$

$$[FV^{-1}-\lambda I]=\begin{pmatrix} \dfrac{\beta(1-\varepsilon c)[\mu K_2+\tau d\mu+\tau\eta K_2(1-d)]}{\mu K_1 K_2}-\lambda & \dfrac{\beta(1-\varepsilon c)}{K_2} & \dfrac{\beta(1-\varepsilon c)\eta}{\mu} & 0 \\ 0 & -\lambda & 0 & 0 \\ 0 & 0 & -\lambda & 0 \\ 0 & 0 & 0 & -\lambda \end{pmatrix}=0 \quad (15)$$

$$R_C=\frac{\beta(1-\varepsilon c)\{\mu K_2+\tau d\mu+\tau\eta K_2(1-d)\}}{\mu K_1 K_2} \quad (16)$$

Hence, the basic reproduction number of our model is given by (16) which is the average number of secondary infections caused by a single infectious individual during his/her entire infectious life time.

Global Stability of Endemic Equilibrium

From equations (1) to (6)

$$\frac{dS}{dt}=\Lambda-\frac{\beta(1-\varepsilon c)[I_1+\eta(I_2+I_3)]\mathrm{S}}{N}-\mu S \quad (17)$$

$$\frac{dI_1}{dt}=\frac{\beta(1-\varepsilon c)[I_1+\eta(I_2+I_3)]\mathrm{S}}{N}-(\tau+\sigma_1+\mu)I_1 \quad (18)$$

$$\frac{d(I_2+\eta I_3)}{dt}=\left(\frac{\tau}{N}\right)I_1-\left(\frac{\mu}{N}\right)(I_2+\eta I_3) \quad (19)$$

$$\frac{dA}{dt}=\sigma_1 I_1+\sigma_2 I_2-(\mu+\delta)\,\mathrm{A} \quad (20)$$

Let

$$E^{**}=S^{**}+I_1^{**}+I_2^{**}+I_3^{**}+A^{**} \quad (21)$$

Denote an endemic equilibrium (*EE*) of model (2) – (6) and

$$H^{**}=I_1^{**}+\eta I_2^{**} \quad (22)$$

To establish the global stability E^{**}, we make the following assumptions:

$$\left(1-\frac{\beta(1-\varepsilon c)I_1}{\beta(1-\varepsilon c)I_1^{**}}\right)\left(1-\frac{A\beta(1-\varepsilon c)I_1^{**}}{A^{**}\beta(1-\varepsilon c)I_1}\right)\geq 0 \quad (23)$$

For $0\leq I_1\leq N$ and $A^{**}\leq A\leq A_{\max}$, and

$$\left(1-\frac{\beta(1-\varepsilon c)H}{\beta(1-\varepsilon c)H^{**}}\right)\left(1-\frac{A\beta(1-\varepsilon c)H^{**}}{A^{**}\beta(1-\varepsilon c)H}\right)\geq 0 \quad (24)$$

For $A^{**}\leq A\leq A_{\max}$ and $0\leq H\leq H_{\max}$,
Where

$$A_{\max} = \left(\frac{\sigma_1 + \sigma_2}{\mu + \delta} \right) N \tag{25}$$

And

$$H_{\max} = \left(\frac{\tau}{\mu} \right) N \tag{26}$$

Theorem: Suppose that

i. Assumptions (23) and (24) are satisfied;
ii. σ_2=0. If R_C > 1, them system (2) and (6) has a unique endemic equilibrium E^{**} that is globally asymptotically stable in Ω.

Proof: For system (17) – (20), motivated by,[15] we consider a Lyapounov function

$$\begin{aligned} &c_1\left(S - S^{**} - S^{**} \ln\left(\frac{S}{S^{**}} \right) \right) + c_1\left(I_1 - I_1^{**} - I_1^{**} \ln\left(\frac{I_1}{I_1^{**}} \right) \right) + c_2\left(H - H^{**} - H^{**} \ln\left(\frac{H}{H^{**}} \right) \right) \\ &+ c_3\left(A - A^{**} - A^{**} \ln\left(\frac{A}{A^{**}} \right) \right) \end{aligned} \tag{27}$$

Where c_i >0, (i=1,2,3) are constants to be determined.
It is ease to verify that L≥0 for all (S,I_1,H,A>0), and L=0 iff (S,I_1,H,A)=($S^{**},I_1^{**},H^{**},A^{**}$). Differentiating L along solutions (2) – (6) and applying all equations (23) - (27), we obtain

$$L' = c_1\left(1 - \frac{S^{**}}{S} \right) S' + c_1\left(1 - \frac{I_1^{**}}{I_1} \right) I_1' + c_2\left(1 - \frac{H^{**}}{H} \right) H' + c_3\left(1 - \frac{A^{**}}{A} \right) A' \tag{28}$$

Which gives

$$\begin{aligned} L' = {} & c_1\left(-\mu S\left(1 - \frac{S^{**}}{S} \right)^2 + \beta(1-\varepsilon c) S^{**} I_1^{**}\left(2 - \frac{S^{**}}{S} - \frac{I_1^{**}}{I_1} \right) - \frac{\beta(1-\varepsilon c) S I_1^{**} I_1}{\beta(1-\varepsilon c) S^{**} I_1^{**} I_1} + \frac{\beta(1-\varepsilon c) I_1}{\beta(1-\varepsilon c) I_1^{**}} \right) + \\ & c_1 \beta(1-\varepsilon c) S^{**} H^{**}\left(2 - \frac{S^{**}}{S} - \frac{I_1^{**}}{I_1} - \frac{\beta(1-\varepsilon c) S I_1^{**} H}{\beta(1-\varepsilon c) S^{**} I_1 H^{**}} + \frac{\beta(1-\varepsilon c) H}{\beta(1-\varepsilon c) H^{**}} \right) \\ & c_2 \tau I_1^{**}\left(\frac{I_1}{I_1^{**}} - \frac{H}{H^{**}} - \frac{H^{**} I_1}{H I_1^{**}} + 1 \right) I + c_3 \sigma_1 I_1^{**}\left(\frac{I_1}{I_1^{**}} - \frac{A}{A^{**}} - \frac{A^{**} I_1}{A I_1^{**}} + 1 \right) H' \end{aligned} \tag{29}$$

Notice that x–1≥ln(x) for any x>0, and the equality holds iff x=1. Together with (23), we find that

$$\begin{aligned} & 2 - \frac{S^{**}}{S} - \frac{I_1^{**}}{I_1} - \frac{\beta(1-\varepsilon c) S I_1^{**} I_1}{\beta(1-\varepsilon c) S^{**} I_1^{**} I_1} + \frac{\beta(1-\varepsilon c) I_1}{\beta(1-\varepsilon c) I_1^{**}} = \left(1 - \frac{\beta(1-\varepsilon c) I_1^{**}}{\beta(1-\varepsilon c) I_1} \right)\left(1 - \frac{A\beta(1-\varepsilon c) I_1^{**}}{A^{**}\beta(1-\varepsilon c) I_1} \right) \\ & + 3 - \frac{S^{**}}{S} \frac{\beta(1-\varepsilon c) S I_1^{**} I_1}{\beta(1-\varepsilon c) S^{**} I_1^{**} I_1} \frac{A\beta(1-\varepsilon c) I_1^{**}}{A^{**}\beta(1-\varepsilon c) I_1} - \frac{I_1^{**}}{I_1} + \frac{A}{A^{**}} \le \left(\frac{S^{**}}{S} - 1 \right) - \left(\frac{\beta(1-\varepsilon c) S I_1^{**} I_1}{\beta(1-\varepsilon c) S^{**} I_1^{**} I_1} - 1 \right) \\ & - \left(\frac{A\beta(1-\varepsilon c) I_1^{**}}{A^{**}\beta(1-\varepsilon c) I_1} - 1 \right) - \frac{I_1^{**}}{I_1} + \frac{A}{A^{**}} \end{aligned} \tag{30}$$

$$= -\ln\left(\frac{S^{**}}{S}.\frac{\beta(1-\varepsilon c)SI_1^{**}I_1}{\beta(1-\varepsilon c)S^{**}I_1^{**}I_1}.\frac{A\beta(1-\varepsilon c)I_1^{**}}{A^{**}\beta(1-\varepsilon c)I_1}\right)-\frac{I_1^{**}}{I_1}+\frac{A}{A^{**}} \tag{31}$$

$$= \frac{A}{A^{**}}-\ln\left(\frac{A}{A^{**}}\right)-\frac{I_1}{I_1^{**}}+\ln\left(\frac{I_1}{I_1^{**}}\right) \tag{32}$$

Likewise, using (24), yields

$$2-\frac{S^{**}}{S}-\frac{I_1}{I_1^{**}}-\frac{\beta(1-\varepsilon c)SI_1^{**}H}{\beta(1-\varepsilon c)S^{**}H^{**}I_1}+\frac{\beta(1-\varepsilon c)H}{\beta(1-\varepsilon c)H^{**}}\leq\frac{H}{H^{**}}-\ln\left(\frac{H}{H^{**}}\right)-\frac{I_1}{I_1^{**}}+\ln\left(\frac{I_1}{I_1^{**}}\right) \tag{33}$$

Meanwhile, one can verify that

$$\begin{aligned}&\frac{I_1}{I_1^{**}}-\frac{H}{H^{**}}-\frac{H^{**}I_1}{HI_1^{**}}+1=-\left(\frac{H^{**}I_1}{HI_1^{**}}-1\right)+\frac{I_1}{I_1^{**}}-\frac{H}{H^{**}}\leq-\ln\left(\frac{H^{**}I_1}{HI_1^{**}}\right)+\frac{I_1}{I_1^{**}}-\frac{H}{H^{**}}=\frac{I_1}{I_1^{**}}\\&-\ln\left(\frac{I_1}{I_1^{**}}\right)-\frac{H}{H^{**}}+\ln\left(\frac{H}{H^{**}}\right)\end{aligned} \tag{34}$$

Similarly,

$$\frac{I_1}{I_1^{**}}-\frac{A}{A^{**}}-\frac{A^{**}I_1}{AI_1^{**}}+1\leq\frac{I_1}{I_1^{**}}-\ln\left(\frac{I_1}{I_1^{**}}\right)-\frac{A}{A^{**}}+\ln\left(\frac{A}{A^{**}}\right) \tag{35}$$

It follows from (32) – (35) that the equation (30) gives

$$\begin{aligned}&L'\leq c_1\beta(1-\varepsilon c)S^{**}I_1^{**}\left(\frac{A}{A^{**}}-\ln\left(\frac{A}{A^{**}}\right)-\frac{I_1}{I_1^{**}}+\ln\left(\frac{I_1}{I_1^{**}}\right)\right)+c_1\beta(1-\varepsilon c)S^{**}H^{**}\left(\frac{H}{H^{**}}-\ln\left(\frac{H}{H^{**}}\right)-\frac{I_1}{I_1^{**}}+\ln\left(\frac{I_1}{I_1^{**}}\right)\right)+\\&c_2\tau I_1^{**}\left(\frac{I_1}{I_1^{**}}-\ln\left(\frac{I_1}{I_1^{**}}\right)-\frac{H}{H^{**}}+\ln\left(\frac{H}{H^{**}}\right)\right)+c_3\sigma_1 I_1^{**}\left(\frac{I_1}{I_1^{**}}-\ln\left(\frac{I_1}{I_1^{**}}\right)-\frac{A}{A^{**}}+\ln\left(\frac{A}{A^{**}}\right)\right)\end{aligned} \tag{36}$$

Take

$$c_1=\sigma_1\tau I_1^{**},\ c_2=\sigma_1\beta(1-\varepsilon c)S^{**}H^{**}\text{ and }c_3=\tau\beta(1-\varepsilon c)S^{**}I_1^{**}$$

It could be verified by direct calculations that the right-hand side of the inequality (36) is zero, which shows $L'\leq 0$ with the chosen positive constants c_1, c_2, and c_3. Moreover, if $L'=0$, then there exists a constant E such that $S=S^{**}$, $I_1=EI_1^{**}$, $H=EH^{**}$, and $A=EA^{**}$
However, by the equations (23), (24), (29), and (32)

$$\sigma_1 EI_1^{**}-(\mu+\delta)EA^{**}=0$$

This implies that
$E\leq 1$.

Thus, the largest invariant sets for which $L'=0$ contains only EE. Therefore, bu Lasalle's principle,[16] the EE is globally asymptotically stable in $\overline{\Omega}$ when $R_C>1$.

RESULTS AND DISCUSSION

In this section, we presented some numerical simulation to monitor the dynamics of the full model (2) – (6) to have pictoral demonstration of the model dynamics using MAPLE 18 software.

Table 1: Baseline values for variables of the human immunodeficiency virus/AIDS in Nigeria

Serial number	Variables	Values	References
1	N	90,062,386	Aaron 2021
2	S	88,452,386	Calculated
3	I_1	800,000	Assumed
4	II_2	20,424	Assumed
5	III_3	160,000	Assumed
6	A	629,576	Assumed

Table 2: Baseline values for parameters of the human immunodeficiency virus/AIDS

Serial number	Parameters	Values	References
1	Λ	162,123	Calculated
2	B	0.2	Abdulrahman *et al*., (2013)
3	Δ	0.014	Abdulrahman *et al*., (2013)
4	H	0.9	Abdulrahman *et al*., (2013)
5	E	0.8	Abdulrahman *et al*., (2013)
6	σ_1	0.067	Abdulrahman *et al*., (2013)
7	σ_2	0.05	Assumed
8	M	0.018	UNICEF (2020)
9	D	0–1	Varies
10	τ	0–1	Varies
11	C	0–1	Varies

Baseline Values for the Parameters used in the Model

These data are estimated based on HIV/AIDS epidemiology and published data. Based on research carried out by,[17] they reported that there is a 20% chance of effective sexual contact rate per year for sexually active individuals. This shows that $\beta = 0.2$. Moreover, due to reduced sexual transmission rate of I_3, it is assumed that $\eta = 0.9$ so that $\eta\beta = 0.18$. Furthermore, the efficacy of condom usage is reported as 0.8.[17] The HIV/AIDS-induced death (δ) is estimated to be 0.014 and according to the[18] natural mortality rate (μ) in Nigeria is 0.018. The recruitment rate is estimated as the product of the natural mortality and the total population size as $\Lambda=\mu\times N$ that is, (Λ=0.018×90062386=162123). The progression rate of untreated HIV infected individuals was estimated based on data gotten from[17] as σ_1= 0.067 and HART resistant individuals is assumed to be σ_2=0.05. Moreover, lastly, the control parameters τ and d range between 0 and 1.

Simulations

In this section, some numerical simulations associated with different values of the effective reproduction number of the model were presented to further justify the analytical results were presented graphical as shown in Figures 2-6.

Total number of infected individuals with different initial variable conditions (i.e., I(0)=100000 and I(0)=1000000). (Control parameters τ=0, d=0,c=0.9 which gives (R_C=0.6588235294). It could be noted that irrespective of the initial population of the infected individuals, the infected population declines when R_C<1. Hence it is a validation of the stability analysis of the model.

Total Number of Infected Individuals with different initial variable conditions (i.e, I(0)=1000000 and I(0)=100000). The control parameters (τ=0.2, d=0.25, c=0.8) which gives (R_C=2.927554180). It could be

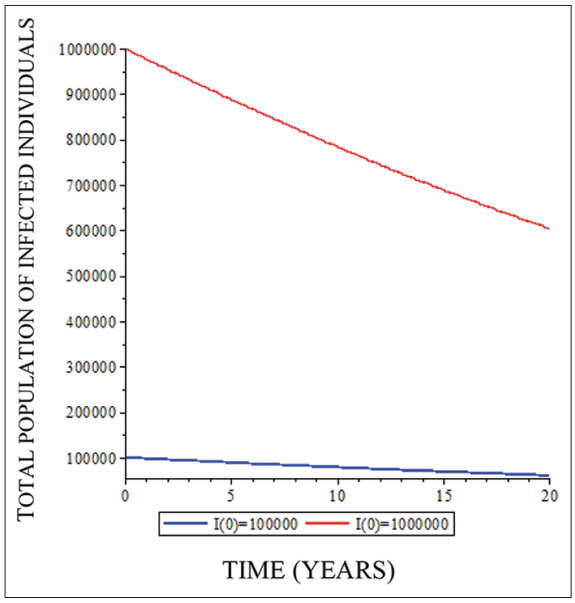

Figure 2: Validation of analytical result on stability

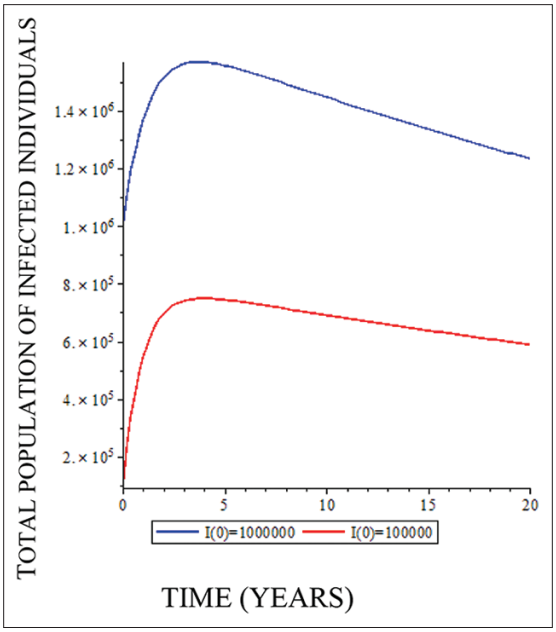

Figure 3: Validation of analytical result on stability analysis

noted that irrespective of the initial population of the infected individuals, the infected population grows when R_C>1. Hence, it is a validation of the stability analysis of the model.

Total number of infected individuals with different level of compliance to condom usage (i.e., c=0,c=0.25,c=0.6,c=0.8). The result shows that for different levels of compliance to condom usage, there is a decline in the infected population and the higher the level of condom usage the faster the decline in the infected population.

Effect of low drug resistance and medium treatment on the AIDS population (i.e., τ=0.5, d=0.25, d=0.0). It has been observed that the lower the drug resistance, the faster the AIDS population declines.

The figure shows the effect of very low contact rate on a small infected population (i.e. β=0.04,β=0.02,I(0)=100,000). It is observed that the effective contact rate has significant impact on the infected population, the lower the contact rate, the faster, and the decline in the infected population.

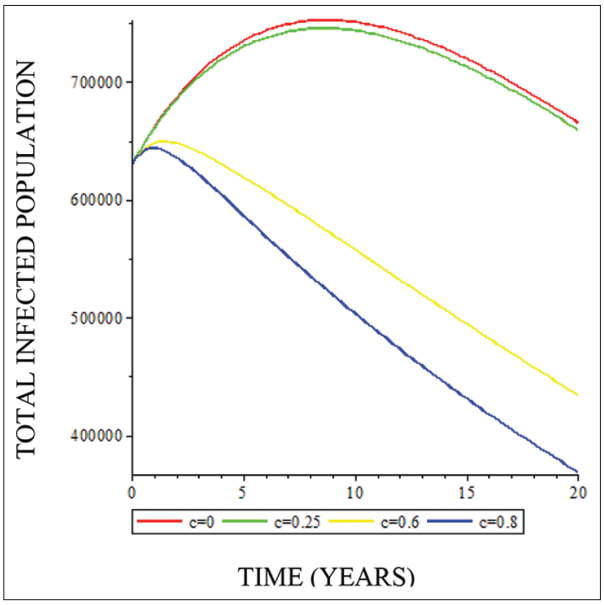

Figure 4: Effect of condom usage on infected population

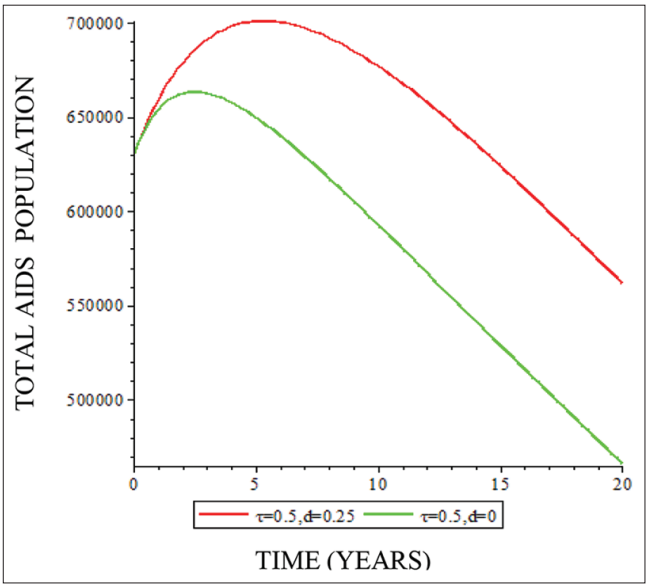

Figure 5: Effect of drug resistance on the AIDS population

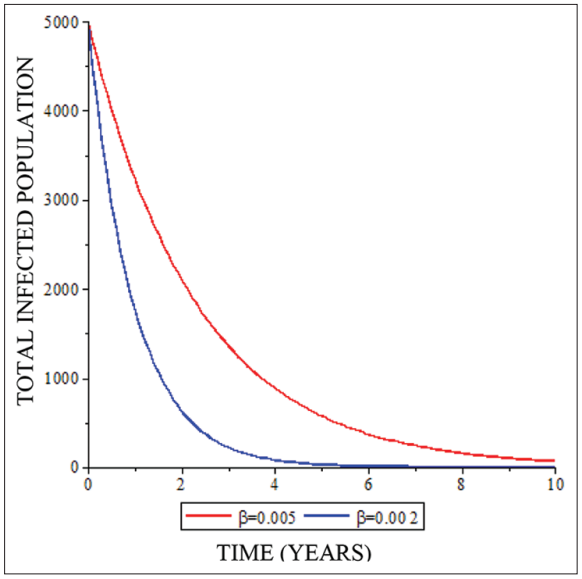

Figure 6: Validation of analytical result on stability

CONCLUSION

The research work has revealed that the model will be globally asymptotically stable if $R_C \leq 1$. Hence, HIV can be curtailed to the barest minimum if the effective reproduction number is less than one. Furthermore, the best strategy in controlling HIV/AIDS is abstinence as 0.2% effective sexual contact reduces the infected population to 500 in 5 years, while 80% compliance to condom usage reduces it to 1000 in 20 years and 50% treatment with 25% drugs resistance reduces the infected population to 5000 in 20 years.

Recommendations

The research, therefore, recommends that:

i. HIV patients undertaken treatments should go for regular check up to ascertain whether the viral load is reducing or they are resistant to HART
ii. It is also recommended to the World Health Organization, CDC and NAFDAC that the efficacy level of the produced condoms should be at 95% and above to reduce the risk of getting infected through sex. Since HIV transmission depends largely on effective contact rate.
iii. One of the limitations of this study is the unavailability of records of HIV cases; therefore, health workers should make data available for researchers.

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