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Stability Analysis of HIV/AIDS Epidemic Model in the Presence of Vertical Transmission and Treatment

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Abstract: HIV/AIDS is a serious health problem that continues to present a significant health concern in underdeveloped nations and may be mostly brought on via unprotected sex. This study is designed and analyzed using a dynamic modeling approach to investigate the dynamic of HIV/AIDS model with vertical transmission and the impact of knowledge on its treatment. Our proposed model exhibit disease free and the endemic equilibrium. The uniqueness and the exactness of the model were investigated and the basic reproduction number using next generation matrix was obtained, Stability analysis was also carried out. The model analysis shows that the disease free equilibrium is locally asymptomatically stable (LAS) when $R_0 < 1$. Our research suggests that treatment and awareness campaigns, when combined with other crucial control measures, may help keep the HIV/AIDS virus from spreading.

Keywords: HIV/AIDS, Vertical transmission, Basic reproductive number, Local Stability, Enlightenment campaign

I. Introduction

AIDS stand for acquired immunodeficiency syndrome, a disease that makes it difficult for the body to fight off infectious disease. The human immunodeficiency virus known as HIV causes AIDS by infecting and damaging the $CD4^+$ T-cells, which are a type of white blood cells in the body immune system that is supposed to fight off invading germs [1 – 3]. In a normal healthy individual's peripheral blood, the level of $CD4^+$ T-cells is between 800 and 1200/mm^3 and once this number reaches 200 or below in HIV infected patient, the person is classified as having AIDS [4]. HIV can be transmitted through direct contact with the blood or body fluid of someone who is infected with the virus, that contact usually comes from sharing needles or by having unprotected sex with an infected person [2–6]. An infant could get HIV from a mother who is infected, not everyone with HIV has AIDS, and in fact adults who become infected with HIV may appear healthy for years before they get sick with AIDS [7].

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Globally, more than 50 million people are living with HIV/AIDS and over 30 million have died since 1981. About 95% of people with HIV live in developing and moderate in-come nations and over 28million people with HIV living in poor and moderate in-come countries should be on antiretroviral medication [8]. Mathematical modeling has emerged as a crucial technique for monitoring the dynamics, management, and progression of HIV Clinical studies on the process of HIV transmission utilizing human subjects are obviously not possible. For this reason, a number of mathematical models that explained the epidemiological dynamics of HIV/AIDS infections have been presented. [10–18, 19, 21]. The underlying assumptions of the various models range from those based on the mode of HIV transmission, contact patterns, latent and infectious period, as well as social, cultural, economic, demographic, or geographic factors. Several significant works have been presented and published for sub-Saharan Africa (generally) and a few selected south and east African nations [19 - 26].

The aim of this research is to propose and develop a deterministic mathematical model to investigate how vertical transmission affects the spread of HIV/AIDS infection and then suggest potential intervention techniques.

The structure of this work is as follows. In section 2, we established a modified HIV/AIDS model and analyze some properties of infections through numerical simulations [2–9].

disease free and endemic equilibria, reproductive number was also obtained, numerical simulation was also carried. In section 3, result and discussion were presented. In section 4, conclusions were presented.

II. Materials and Methods

A. Model Formulation

Considering the classical assumption of [8–19] by introducing vertical transmission and treatment, we assumed that the fraction of new born baby that are infected during birth join the infective asymptomatic with the rate $(1-\varepsilon)\theta$ and others die at the birth $(0 \le \varepsilon \le 1)$.

i. modified model:

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda - c\beta(I + bJ)S - \mu S \\
\frac{dI}{dt} &= c\beta(I + bJ)S + (1-\varepsilon)\theta I - (\eta + \rho + \mu)I \\
\frac{dJ}{dt} &= \eta I - \delta(1+y)J - (\xi + \mu + \alpha)J \\
\frac{dT}{dt} &= \delta(1+y)J - (\mu + d + \alpha)T \\
\frac{dA}{dt} &= dT + \rho I + \xi J - (\mu + \alpha)A
\end{aligned}\right\} \tag{1}$$

Table 1: Parameter Description

Parameter description	Variable
Susceptible	$S(t)$
Asymptotic class	$I(t)$
Symptomatic class	$J(t)$
Treatment class	$T(t)$
AIDS class	$A(t)$

Table 2: Parameter Description with Symbol

Parameter description	Parameter
Average number of contact per unit of time	C
Recruitment rate	Λ
Progression rate from Symptomatic to AIDS	ξ
Progression rate from Asymptomatic to AIDS	ρ
Progression rate from treatment to AIDS	d
Probability of disease transmission per contact by Asymptomatic infective	β
Fraction of newborns infected with HIV who dies immediately	ε
Rate of newborn infected with HIV	θ
Disease induced dearth	α
Treatment rate from symptomatic class to treatment	δ
Natural death	μ
Enlightenment rate for the treatment	y

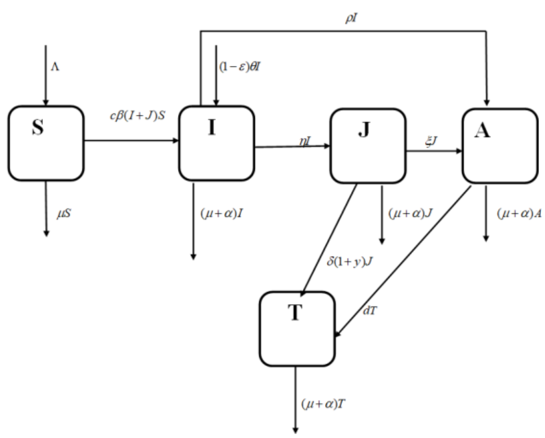

Figure 1: Schematic Diagram of an HIV/AIDS Model

B. Existence and Uniqueness of Solution for the Model

For the model to predict the future of the system from its current state at the time t_0, the initial value problem (IVP) Must have a solution that exist and also unique. In this subsection, we will establish the condition for the model's existence and uniqueness of solution.

$$X^i = F(t,x)$$

Let,

$$f_1(t,x) = \mu\kappa - c\beta(I + bJ)S - \mu S$$

$$f_2(t,x) = \alpha J - c\beta(I + bJ)S - (k - \mu)I + (1-\varepsilon)\theta I - \gamma(1+y)I, \quad I(t) = I_0$$

$$f_3(t,x) = k_1 I - (\mu + \delta)J, \quad J(t) = J_0$$

$$f_4 = \delta J + \gamma(1+y)I - (\mu + d)T, \quad T(t) = T_0$$

$$f_5 = dT - (\mu + \alpha)A, \quad A(t) = A_0$$

So that,

$$x^i = f(t,x) = f(x)$$

i. theorem

Let D^i denote the region

$$|t - t_0| \le a_i \|x - x_0\| \le b, \qquad x = (x_1, x_2 \ldots x_n), \; x_0(x_{10}, x_{20}, \ldots x_{n0})$$

And suppose that $f(t,x)$ satisfies the Lipchitz condition

$$\|f(t,x_1) - f(t,x_2)\| \le R\|x_1 - x_2\|$$

$$X(t_0) = X_0$$

Whenever the pair (t, x_1) and (t, x_2) belong to D^i, where K is a positive constant. Then there exist a constant $\sigma > 0$ such that there exist a uniqueness continuous vector solution $\bar{x}(t)$ of the system in the interval $|t - t_0| \le \sigma$ (Derrick and Grossman)

It is important to note that the condition is satisfied by requirement that $\frac{\partial f_i}{\partial x_j}, i, j = 1,2 \ldots n$ be continuous and bounded in D^i

C. Basic Reproduction Number (R_0)

$$\frac{dI}{dt} = \alpha J - c\beta(I + bJ)S - (k - \mu)I + (1-\varepsilon)\theta I - \gamma(1+y)I$$

$$\frac{dJ}{dt} = k_1 I - (\mu + \delta)J$$

$$R_0 = F \times V^{-1}$$

$$F = \begin{pmatrix} c\beta\kappa & c\beta b\kappa \\ 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} (k_1+\mu) - (1-\varepsilon) + \gamma(1+y) & -\alpha \\ -k_1 & (\mu+\delta) \end{pmatrix}$$

$$F = \begin{pmatrix} \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial J} \\ \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial J} \end{pmatrix}$$

$$V = \begin{pmatrix} \frac{\partial v_1}{\partial I} & \frac{\partial v_1}{\partial J} \\ \frac{\partial v_2}{\partial I} & \frac{\partial v_2}{\partial J} \end{pmatrix}$$

$$|V| = [(k_1+\mu) - (1-\varepsilon)\theta + \gamma(1-y)][\mu+\delta] - \alpha k_1$$

Finding the adjoint of V,

The cofactors are- $a_{11} = (\mu + \delta)$ $a_{12} = k_1$

$a_{21} = \alpha$

$a_{22} = (k_1 + \mu) - (1 - \varepsilon) + \gamma(1 + y)$

Transpose

$$\begin{pmatrix} (\mu+\delta) & \alpha \\ k_1 & (k_1+\mu) - (1-\varepsilon) + \gamma(1+y) \end{pmatrix}$$

$$V^{-1} = \frac{adjoint\ of\ V}{|V|}$$

$$= \begin{pmatrix} \frac{(\mu+\delta)}{[(k_1+\mu)-(1-\varepsilon)\theta+\gamma(1-y)][\mu+\delta]-\alpha k_1} & \frac{\alpha}{[(k_1+\mu)-(1-\varepsilon)\theta+\gamma(1-y)][\mu+\delta]-\alpha k_1} \\ \frac{k_1}{[(k_1+\mu)-(1-\varepsilon)\theta+\gamma(1-y)][\mu+\delta]-\alpha k_1} & \frac{(k_1+\mu)-(1-\varepsilon)+\gamma(1+y)}{[(k_1+\mu)-(1-\varepsilon)\theta+\gamma(1-y)][\mu+\delta]-\alpha k_1} \end{pmatrix}$$

$$R_0 = F \times V^{-1}$$

$$R_0 = \frac{c\beta k(\mu+\delta) + k_1 c\beta bk}{[(k_1+\mu)-(1-\varepsilon)\theta+\gamma(1-y)][\mu+\delta]-\alpha k_1}$$

D. Equilibrium and their Stability

In the absence of disease infection in the population, $I = 0$. Solving equation (1) the disease – free equilibrium was obtained as $(S, I, J, T, A) = \left(\frac{\Lambda}{\mu}, 0,0,0,0\right)$

Also, in the presence of disease infection in the population, $I \ne 0$. Solving (1) admits a unique solution

$$E_* = (S_*, I_*, J_*, T_*, A_*)$$

Where;

$$S^* = \frac{(\eta + \mu + \rho - \theta\varepsilon)(\delta y + \xi + \delta + \mu)}{c\beta(b\eta + \delta y + \xi + \delta + \mu)}$$

$$I^* = -\frac{\left(\begin{pmatrix} (1+y)(c\beta\Lambda + \theta\varepsilon - \eta - \mu - \rho)\delta - \mu^2 + (c\beta\Lambda + \theta\varepsilon - \xi - \eta - \rho)\mu \\ + (cb\beta\Lambda - \xi)\eta + \xi(c\beta\Lambda + \theta\varepsilon - \rho) \end{pmatrix}\mu\right)}{(\beta(\theta\varepsilon - \eta - \mu - \rho)(\delta(1+y) + b\eta + \mu + \xi)c)}$$

$$J^* = -\frac{\left(\eta\begin{pmatrix} (1+y)(c\beta\Lambda + \theta\varepsilon - \eta - \mu - \rho)\delta - \mu^2 + (c\beta\Lambda + \theta\varepsilon - \xi - \eta - \rho)\mu \\ + (cb\beta\Lambda - \xi)\eta + \xi(c\beta\Lambda + \theta\varepsilon - \rho) \end{pmatrix}\mu\right)}{(\beta(\theta\varepsilon - \eta - \mu - \rho)(\delta(1+y) + \mu + \xi)(\delta(1+y) + b\eta + \mu + \xi)c)}$$

$$T^* = -\frac{\left(\mu(1+y)\begin{pmatrix} (1+y)(c\beta\Lambda + \theta\varepsilon - \eta - \mu - \rho)\delta - \mu^2 + (c\beta\Lambda + \theta\varepsilon - \xi - \eta - \rho)\mu \\ + (cb\beta\Lambda - \xi)\eta + \xi(c\beta\Lambda + \theta\varepsilon - \rho) \end{pmatrix}\mu\delta\right)}{((d+\mu)(\delta(1+y) + b\eta + \mu + \xi)(\delta(1+y) + \mu + \xi)c\beta(\theta\varepsilon - \eta - \mu - \rho))}$$

$$A^{*}=-\frac{\left(\mu\begin{pmatrix}(1+y)(c\beta\Lambda+\theta\varepsilon-\eta-\mu-\rho)\delta-\mu^{2}+\\(c\beta\Lambda+\theta\varepsilon-\eta-\xi-\rho)\mu+(cb\beta\Lambda-\xi)\eta\\+\xi(c\beta\Lambda+\theta\varepsilon-\rho)\end{pmatrix}\begin{pmatrix}(\mu\rho+d(\eta+\rho))(1+y)\delta\\+(d+\mu)(\mu\rho+\xi(\eta+\rho))\end{pmatrix}\right)}{((\mu+\alpha)(\theta\varepsilon-\eta-\rho)(\delta(1+y)+b\eta+\mu+\xi)(d+\mu)c\beta(\delta(1+y)+\mu+\xi))}$$

E. Local Stability of Disease Free Equilibrium

Preposition 1

If $R_0<1$, then the disease free equilibrium E_0 is locally asymptotically stable.

Proof

Considering Linearization method, the resulting characteristic equation of system (1) is

$$|A-\lambda I|=0$$

If $R_0<1$, then the disease free equilibrium E_0 is locally asymptotically stable.

$$|A-\lambda I|=\begin{pmatrix}-\mu & -\frac{c\beta\Lambda}{\mu} & -\frac{c\Lambda b\beta}{\mu} & 0 & 0\\ 0 & \frac{c\beta\Lambda}{\mu}-\eta-\rho-\mu-\alpha+(1-\varepsilon)\theta & \frac{c\Lambda b\beta}{\mu} & 0 & 0\\ 0 & \eta & -\delta(1+y)-\zeta-\mu-\alpha & 0 & 0\\ 0 & 0 & \delta(1+y) & -d-\mu-\alpha & 0\\ 0 & \rho & \zeta & d & -\alpha-\mu\end{pmatrix}$$

Computing the Trace and the determinant of the matrix above, thus the trace at DFE is given by;

$$\tau(J_{E_0})=-4\mu+\left(\frac{c\beta\Lambda+(1-\varepsilon)\theta}{\mu(\eta+\rho+\mu+\alpha)}-1\right)(\eta+\rho+\mu+\alpha)-\delta(1+y)-\zeta-d$$

Simplifying we have;

$$\tau(J_{E_0})=-4\mu-3\alpha+(R_0-1)(\eta+\rho+\mu+\alpha)-\delta(1+y)-\zeta-d$$

And the determinant of the matrix is obtained as;

$$Det(J_{E_0})=\begin{pmatrix}-\mu^{3}+((-y-1)\delta-2\alpha+(1-\varepsilon)\theta-\eta-\rho-\zeta)\mu^{2}\\+\begin{pmatrix}-(1+y)(\alpha+(-1+\varepsilon)\theta+\eta+\rho)\delta-\alpha^{2}\\+((1-\varepsilon)\theta-\eta-\rho-\zeta)\alpha-\zeta(-1+\varepsilon)\theta+(-\eta-\rho)\zeta+c\beta\Lambda\end{pmatrix}\mu\\+(\delta(1+y)+b\eta+\alpha+\zeta)\beta\Lambda c\end{pmatrix}(\mu+\alpha)(d+\mu+\alpha)$$

The Trace (τ) is negative and the determinant is positive with the same condition. Thus, the disease free equilibrium is asymptotically stable provided the $R_0<1$.

III. Results and Discussion

A. Numerical Simulation

We use the numerical software (MAPLE) to plot the graph, we obtain the following;

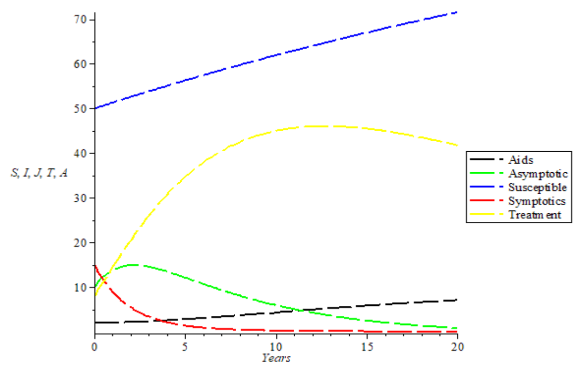

Fig 2: Behavioral Dynamic of the Compartments against time when Contact Rate =0.0005.

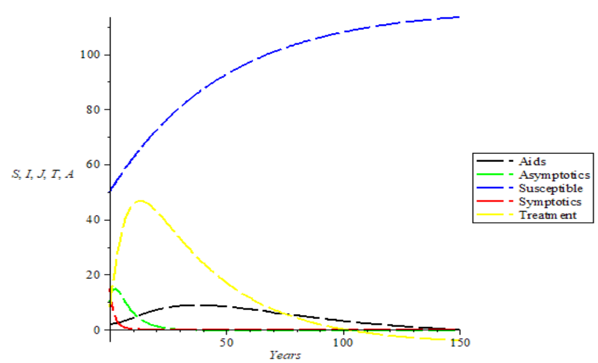

Fig 3: Behavioral Dynamic of the Compartments against time when Contact Rate =0.0002.

Fig 2 and 3 Shows the behavior of susceptible population when the contact rate is reduces from 0.0005 to 0.0002, the susceptible population increases drastically and tends to equilibrium, this could be as a result of enlightenment campaign to go for HIV/AIDS treatment and avoid been contact with the infected individual. The Treatment class rises significantly and drop drastically and attained an equilibrium position and remain steady after sometimes. This could be as a result of carefulness of susceptible individual not to have contact with infected individual. The asymptomatic Class and symptomatic class decreases significantly; also the AIDS class also reduces as the contact rate reduces from 0.0005 to 0.0002.

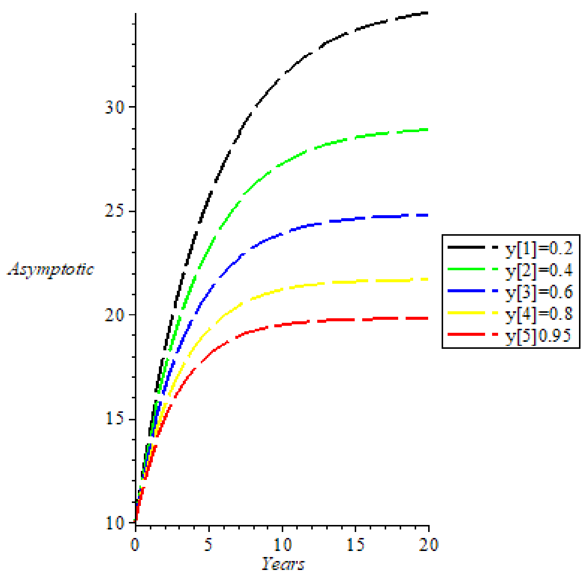

Fig 4: Dynamic behavior of Asymptomatic with Varying Value of Enlightenment Rate against Time

It is seen from figure 4 that with increase in the value of enlightenment rate y, the asymptotic class decreases.

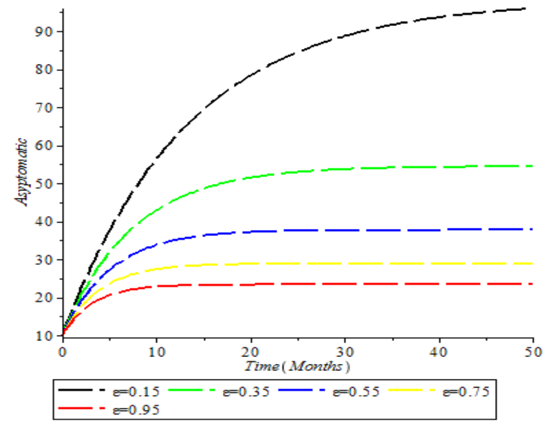

Fig 5: Dynamic behavior of Asymptomatic with Varying Value of Fraction of Newborns Infected with HIV who dies immediately against time

The asymptotic behavior versus time is depicted in Fig. 5 together with the varying percentage of HIV-positive babies that pass away at birth. It is clear that the population of people who are asymptomatic decreases as the proportion of HIV-positive neonates who die right away rises.

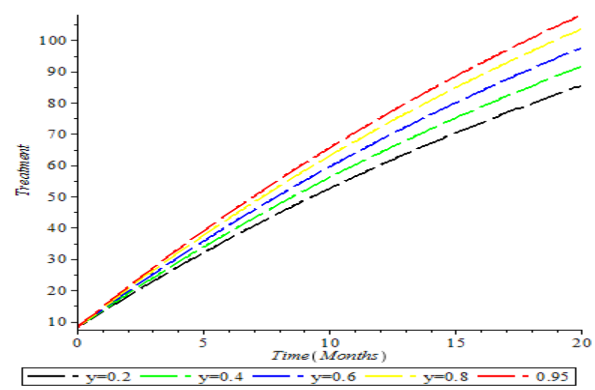

Fig 6: Dynamic behavior of Treatment against Time with Varying Value of Enlightenment Rate(y).

In Fig. 6, it can be seen that raising awareness makes it more likely for HIV-positive people to seek treatment, which increases the number of people in the treatment class. When people with HIV are made aware of the risks of delaying treatment, more will seek it, which will increase the number of people receiving it.

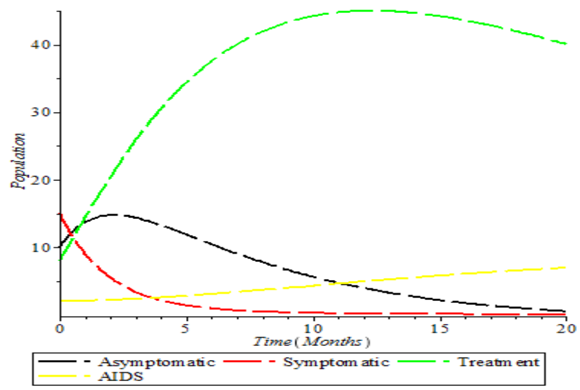

Fig 7: Graph of Asymptomatic, Symptomatic, Treatment and AIDS against Time when Enlightenment Rate(y) is 0.8 and $\varepsilon = 0.9$

Figure 7 shows that when there are few newborn HIV-positive babies in the community due to the high proportion of newborns who die from HIV infection right away $\varepsilon = 0.9$. As enlightenment rate (y) is 0.8 the proportion of symptomatic decreases continuously which leads to increasing in population receiving treatment initially but decreasing as the asymptomatic rises initially but decreases drastically and nearly goes extinct, this in turn causes an increase in full-blown AIDS.

IV. Conclusion

This study introduce a deterministic mathematical model for HIV/AIDS transmission, we discussed a stability analysis of an HIV/AIDS epidemic model with vertical transmission and treatment. We can control the disease burden by controlling the effective contact rate of the infected population and continue to enlighten people about the virus. we conclude that, using an effective educational enlightenment campaign on the spread of the disease is the most effective way to control HIV/AIDS transmission within the population, and AIDS patients should also be encouraged to seek treatment regularly in order to protect their lives and as this will lower the disease-related mortality rate.

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