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# Analysis and simulation of HIV/AIDS transmission dynamics and control strategies

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## Abstract

HIV/AIDS is a fatal illness that weakens the immune system of the body, making the victim susceptible to several opportunistic infections. The analysis and numerical simulation of the proposed epidemic model of HIV/AIDS with vertical transmission was carried out. The disease free and endemic equilibrium of the model were obtained and the basic reproduction number was established. Numerical simulations using the homotropy perturbation method was employed to demonstrate the efficacy of the key findings. The results of the simulation were graphically presented and interpreted.

***Keywords:*** HIV/AIDS, disease free equilibrium, endemic equilibrium, Laplace Adomian decomposition.

## Introduction

The human immune virus (HIV) infection which can lead to acquired immune deficiency syndrome (AIDS) has become a serious infectious disease in the entire world especially in developing countries. It is a deadly disease, which breaks down the body's immune system leaving the victim vulnerable to a host of life-threatening infections, neurological disorders or unusual malignancies (Bashiru, 2014). Mathematical models, more often non – linear type for the spread of the HIV/AIDS epidemic have been studied extensively since the first case was recognized in the late 80's (Tan & Zhi, 1997; Sani *et al*, 2006; Bashiru *et al*, 2017a; Bashiru *et al*, 2017b).

In a normal healthy individual's peripheral blood, the level of $CD4^+$ T-cells is between 800 and 1200 / $mm^3$ and once it is $\leq 200$ the infected person is classified as having AIDS. The two main modes of transmission of the virus are through unsafe sex (horizontal transmission) and from an infected mother to her child (0 to 5) years, (vertical transmission). Mother to Child Transmission (MTCT) of HIV primarily takes place during the perinatal period (from the $20^{th}$ to $28^{th}$ week of gestation and ends 1st to 4th week after birth) and

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post-natal during breastfeeding. (Tan & Zhi, 1997; Bashiru, 2014; Bashiru *et al,* 2017a; Bashiru *et al,* 2017b). Numerous research studies on how to combat the HIV/ AIDS epidemics have been undertaken as a result of the frightening HIV epidemic (Medan, 2007; Bashiru & Fasoranbaku , 2009; Mukandavire *et al,* 2009; Cassels *et al*, 2012; Kateme *et al*, 2012; Fasoranbaku *et al* (2015), Ogunlaran & Oukouomi, 2016; Bashiru *et al*, 2017) , and mathematical modelling research has not lagged behind (Ongun, 2011; Yuzbas, 2012; Zhiming *et al*, 2017).

Through numerical simulations, mathematical modelling has proven an extremely important tool for tracking, control and development of the dynamic of HIV infections. It is evidently impossible to do clinical research on the HIV transmission dynamics using human patients. To this end, a number of mathematical models that described the epidemiological processes of HIV/AIDS infections have been proposed (Wang & Song, 2007; Ghoreishi *et al,* 2011; Merdan *et al*, 2011; Cassels *et al,* 2012; Dogan, 2012; Kateme *et al*, 2012; Malik *et al,* 2014; Jia & Qin, 2017; Zhiming *et al,* 2017; Glass *et al,* 2020; Ayele *et al,* 2021;).

The various models' various underlying assumptions range from those based on the mode of HIV transmission, contact patterns, latent and infectious period, to social, cultural, economic, demographic, or geographic factors. Some important researches have been published for sub-Saharan Africa (in general) and some chosen South and East African countries. (Medan, 2007; Wang & Song, 2007; Ghoreishi *et al* 2011; Merdan *et al,* 2011; Ongun, 2011; Dogan, 2012; Yüzbas, 2012). A mathematical model for HIV prevention and control among Chinese males who have sex with other men was also taken into consideration Cassels *et al* (2012).

This paper seeks to develop a mathematical model to study the impact of irresponsible infectives on the spread of HIV/AIDS infection with vertical transmission and then offer possible intervention strategies. The structure of this work is as follows. In section 2, we established a modified HIV/AIDS model and analyse some properties of disease free and endemic equilibria, reproductive number was also obtained. In section 3, the study numerical simulation with the aid of Laplace Adomian, the result and discussions are given in 4 and conclusion was elucidated in 5.

## Material and Methods

### Model Description

The population is divided into four compartments, the susceptible $S(t)$, the Infectives unaware $Q(t)$, the Infectives aware through medical screening $H(t)$ and the HIV/AIDS patient $A(t)$. $\psi$ is the constant immigration rate of susceptible, $\gamma$ is the birth rate of the new-born, $\varepsilon$ is the fraction of infected new-born joining the unaware infective class.

$$\left.\begin{aligned}
\frac{dS}{dt} &= \psi - \left(\frac{\beta_1 Q + \beta_2 H}{N}\right)S - \mu S \\
\frac{dQ}{dt} &= \left(\frac{\beta_1 Q + \beta_2 H}{N}\right)S + \gamma\varepsilon Q - (\mu + \delta + \theta)Q \\
\frac{dH}{dt} &= \theta Q - (\mu + \tau)H \\
\frac{dA}{dt} &= \delta Q + \tau H - (\mu + d)A
\end{aligned}\right\} \quad (1)$$

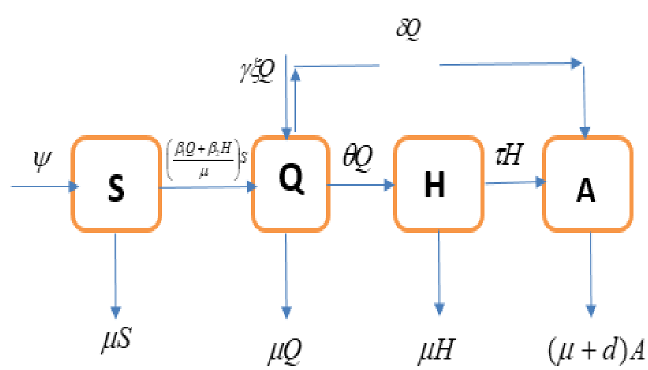


Fig.1: Schematic diagram of the proposed HIV/AIDS Model

| S/N | Parameter | Definition of the parameter | Value |
|---|---|---|---|
| 1 | $\psi$ | Rate of immigration of Susceptible | 3000 |
| 2 | $\beta_1$ | Per capital contact rate of unaware infective | 0.0009 |
| 3 | $\beta_2$ | Per capital contact rate of aware infective | $0 \leq \beta_2 \leq 1$ |
| 4 | $\mu$ | Natural death | 0.04 |
| 5 | $\delta$ | Rate at which infective unaware develop AIDS | 0.3 |
| 6 | $\varepsilon$ | Fraction of infected new-born joining unaware infected class | 0.4 |
| 7 | $\theta$ | Rate at which unaware infective become aware infective by screening | $0 \leq \theta \leq 1$ |
| 8 | $N$ | Total population. | 27000 |
| 9 | D | AIDS induced death | 0.2 |
| 10 | $\gamma$ | Birth rate of the infected new-born | $0 \leq \gamma \leq 1$ |
| 11 | $\tau$ | Rate at which infective aware develop AIDS | 0.4 |
| 12 | $s_0$ | Initial population of susceptible individuals | 15300 |
| 13 | $h_0$ | Initial population of infectives aware individuals | 4500 |
| 14 | $a_0$ | Initial population of Aids patients | 1800 |
| 15 | $q_0$ | Initial population of infectives unaware individuals | 5400 |

Since the variable A of the system does not appear in the first three equation of the model, in the subsequent analysis, we only consider the subsystem. Thus, considering the total population given $N(t) = S(t) + Q(t) + H(t)$

**Equilibrium State**

The disease-free equilibrium of the

$$DFE = \left(\frac{\psi}{\mu}, 0, 0, 0\right)$$

and endemic equilibrium given by $N(t) = S(t) + Q(t) + H(t)$ with

$$\left.\begin{aligned}
S^* &= \frac{(\gamma\varepsilon - \mu - \delta - \theta)N(\mu + \delta)}{\beta_1(\mu + \delta) + \beta_2\theta} \\
Q^* &= \frac{\psi(\beta(\mu + \delta)_1 + \beta_2\theta) + \mu N(\mu + \delta + \theta + \gamma\varepsilon)(\mu + \delta)}{(\mu + \delta + \theta + \gamma\varepsilon)(\mu + \delta)} \\
H^* &= \frac{\theta(\psi(\beta(\mu + \delta)_1 + \beta_2\theta) + \mu N(\mu + \delta + \theta + \gamma\varepsilon)(\mu + \delta))}{(\mu + \delta + \theta + \gamma\varepsilon(\beta(\mu + \delta)_1 + \beta_2\theta))(\mu + \delta)}
\end{aligned}\right\} \quad (2)$$

**Basic Reproduction Number $R_0$**

The reproduction number can be obtained using the next generation matrix, the matrices F and V, for the new infection terms and the remaining transfer terms are respectively given by

$$G = FV^{-1} \quad (3)$$

Where;

F = new infectious disease rate

V=rate of infection spread from one compartment to another

Hence,

$$F = \begin{bmatrix} \beta_1 + \gamma\varepsilon & \beta_2 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad and \quad V = \begin{bmatrix} (\mu+\delta+\theta) & 0 & 0 \\ \theta & 0 & 0 \\ 0 & 0 & -(\mu+d) \end{bmatrix} \quad (4)$$

Thus, $R_0 = \left(F^{'}\left(V^{'}\right)^{-1}\right) = \left(\dfrac{N\mu\gamma\varepsilon + \beta_1\psi}{N\mu(\mu+\delta+\theta)}\right)$ (5)

**Local Stability of Disease-Free Equilibrium**

**Proposition 1.**

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

Proof.

$$|A - \lambda Q| = \begin{pmatrix} -\mu & -\dfrac{\beta_1\psi}{N\mu} & -\dfrac{\beta_2\psi}{N\mu} \\ 0 & \dfrac{\beta_1\psi}{N\mu} + \varepsilon\gamma - \mu - \delta - \theta & \dfrac{\beta_2\psi}{N\mu} \\ 0 & \theta & -\mu-\delta \end{pmatrix} \quad (6)$$

Computing the Trace and determinant of the matrix above, thus the trace of the disease-free equilibrium is obtained as

$$\tau\left(J_{E_0}\right) = -\mu + \frac{\beta_1\psi}{N\mu} + \varepsilon\gamma - \mu - \delta - \theta \quad (7)$$

Simplifying (7) we have

$$\tau\left(J_{E_0}\right) = -N\mu^2 + (R_0 - 1)N\mu(\mu + \delta + \theta) \quad (8)$$

Also, finding the characteristic equation of the matrix in (6) we obtained

$$-(\mu + \lambda)[\lambda \in a_1\lambda + a_2] = 0$$

Where $a_1 = \theta + 2\mu + 2\delta - \dfrac{\beta_1\psi}{N\mu} - \gamma\varepsilon$ and

$a_2 = (\mu + \delta)(\theta + \mu + \delta)(1 - R_0)$

Clearly, $\lambda_1 = -\mu < 0$ and $a_1 > 0$. Hence, by Ruth – Hurwitz criteria, disease free equilibrium is locally asymptotic stable.

**Global Stability of Equilibrium Point**

The Castillo-Chavez approach is used to demonstrate global stability Consider a model of the form

$$\left.\begin{aligned} \frac{dF}{dt} &= F(x,Y) \\ \frac{dI}{dt} &= G(x,Y)\ , G(x,0) = 0 \end{aligned}\right\} \quad (9)$$

Where $x \in \Re^m$ represents individuals that are not infected in the population and $Y \in R^n$ represent infected individuals. Following the above representation, it is possible to express the disease-free equilibrium of this system as $T^0 = (x^*, 0)$. The following two conditions $(H1)\, and\, (H2)$ below must be met to guarantee global asymptotic stability.

$(H1)\; For\; \dfrac{dx}{dt} = F(x,0),\, x^*$ is globally asymptotically stable.

$(H2).\, G(x,Y) = AY - \hat{G}(x,Y), \hat{G}(x,Y) \geq 0 \; for \; (x,Y) \in \Omega$

where, $A = D_Y G(x^*, 0)$ is an M-matrix (the off-diagonal components of A are non-negative), and $\Omega$ is where the model makes biological sense.

If system (2) satisfies the above two conditions, then the following theorems hold:

**Theorem 1:** The fixed point $T^0 = (x^*, 0)$ is globally asymptotically stable (g.a.s) provided that $R_0 \le 1$ and assumption that $(H_1)$ *and* $(H_2)$ are satisfied.

**Proof:** (See Jia *et al* 2017)

**Theorem 2:** The DFE $T^0$ of the model system (1) is globally asymptotically stable if $R_0 < 1$ .

**Proof:** The model equation (1) above is re-written by setting $x = (S,)$,

$$Y = (Q, H, A), T^0 = (x^*, 0) = \left(\frac{\psi}{\mu}, 0\right) \tag{10}$$

And the system $\frac{dx}{dt} = F(x, 0)$ becomes;

$$\left\{ \dot{S} = \psi - \mu S \right.$$

This equation has a unique equilibrium point

$$x^* = \left(\frac{\psi}{\mu}\right) \tag{11}$$

Which is asymptotically stable, therefore $(H1)$ is satisfied. For $(H2)$ can be verified. The model system (1), has

$$D_Y G(x^*, 0) = \begin{pmatrix} \frac{\beta_1}{N} S + \gamma\varepsilon - (\mu + \delta + \theta) & \frac{\beta_2}{N} S & 0 \\ \theta & -(\mu + \tau) & 0 \\ \delta & \tau & -(\mu + d) \end{pmatrix} \tag{12}$$

Clearly, $A = D_Y G(x^*, 0)$ is a M-Matrix. On the other hand,

$$\Rightarrow \hat{G}(x, Y) = AY - G(x, Y) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

Hence, $\hat{G} = (x, Y) = 0$ *for all* $(x, Y) \in \Omega$, therefore conditions $(H_1)$ and $(H_2)$ are satisfied. With lemma 3, the global stability of DFE is obtained and which complete the proof.

**Local Stability of Endemic Equilibrium**

**Theorem 3**. The endemic equilibrium $E_*$ of the model (1) is locally asymptotically stable if $R_0 > 1$.

**Proof.**

Let the endemic equilibrium $E_* = (S_*, Q_*, H_*)$, using linearization method by setting $X = S - S_*$, $Y = Q - Q_*$, $Z = H - H_*$

Then the resulting Jacobian matrix of system of (1) is obtained as

$$|A - \lambda Q| = \begin{pmatrix} -\mu - \frac{H\beta_2 + Q\beta_1}{N} & -\frac{\beta_1 S}{N} & -\frac{\beta_2 S}{N} \\ \frac{H\beta_2 + Q\beta_1}{N} & \frac{\beta_1 S}{N} + \varepsilon\gamma - \mu - \delta - \theta & \frac{\beta_2 S}{N} \\ 0 & 0 & 0 \end{pmatrix} \tag{13}$$

Obtainning the determinant (13), the following characteristic polynomial is obtained;

$$H(\lambda) = \frac{1}{N}\left(N\lambda^4 + a_0\lambda^3 - a_1\lambda^2 + a_2\lambda\right) \tag{14}$$

where

$$a_0 = ((\delta - \varepsilon\gamma + 2\mu + \theta)N + (Q - S)\beta_1 + H\beta_2) - \mu N + (\varepsilon - 1)\gamma + dN$$

$$a_1 = ((\delta - \varepsilon\gamma + 2\mu + \theta)N + (Q - S)\beta_1 + H\beta_2)((\mu + d + (\varepsilon - 1)\gamma)) \\ - (\mu N(\varepsilon\gamma - \delta - \mu - \theta) - \mu(Q - S)\beta_1 + H\beta_2) + (\varepsilon\gamma - \delta - \theta)(H\beta_2 + Q\beta_1)$$

$$a_2 = (\mu - d + (\varepsilon - 1)\gamma)(\mu N(\varepsilon\gamma - \delta - \mu - \theta) - \mu(Q - S)\beta_1 + H\beta_2) + (\varepsilon\gamma - \delta - \theta)(H\beta_2 + Q\beta_1)$$

Taking into account the Routh-Hurwitz criterion, which required that every characteristic's root have a negative real part. If and only if $a_2 > 0, a_2 a_1 - a_0 > 0$, then the roots of (14) have negative real parts. As a result, endemic equilibrium is asymptotically stable locally.

**Theorem 4.** If $R > 1$, the global asymptotically stable of endemic equilibrium point of the model equation (1) is found.

**Proof**. In order to prove that the model's endemic equilibrium is globally stable, we use a Lyapunov function.

$$V(S^*, V^*, E^*, I^*, Q^*, R^*) = \left(S - S^* - S^* \ln\frac{S^*}{S}\right) + \left(Q - Q^* - Q^* \ln\frac{Q^*}{Q}\right) + \left(H - H^* - H^* \ln\frac{H^*}{H}\right) + \left(A - A^* - A^* \ln\frac{A^*}{A}\right) \tag{15}$$

The by-product of V along this solution of (1) by direct calculation gives:

$$\frac{dV}{dt} = \left(\frac{S - S^*}{S}\right)\frac{dS}{dt} + \left(\frac{Q - Q^*}{Q}\right)\frac{dQ}{dt} + \left(\frac{H - H^*}{H}\right)\frac{dH}{dt} + \left(\frac{A - A^*}{A}\right)\frac{dA}{dt} \tag{16}$$

where;

$$\frac{dS}{dt} = \psi - \frac{(\beta_1 Q + \beta_2 H)}{N} S - \mu S$$
$$\frac{dQ}{dt} = \frac{(\beta_1 Q + \beta_2 H)}{N} S + \gamma\varepsilon Q - (\mu + \delta + \theta) Q$$
$$\frac{dH}{dt} = \theta Q - (\mu + \tau) H$$
$$\frac{dA}{dt} = \delta Q + \tau H - (\mu + d) A$$

That is;

$$\frac{dV}{dt} = \left(\frac{S - S^*}{S}\right)\left[\psi - \frac{(\beta_1 Q + \beta_2 H)}{N} S - \mu S\right] + \left(\frac{Q - Q^*}{Q}\right)\left[\frac{(\beta_1 Q + \beta_2 H)}{N} S + \gamma\varepsilon Q - (\mu\right] + \left(\frac{H - H^*}{H}\right)\left[\theta Q - (\mu + \tau) H\right] + \left(\frac{A - A^*}{A}\right)\left[\delta Q + \tau H - (\mu + d) A\right] \tag{17}$$

Then, we obtain;

$$\frac{dV}{dt} = \frac{(S - S^*)\psi - \frac{\beta_1}{N}(S - S^*)^2 (Q - Q^*) - \frac{\beta_2}{N}(S - S^*)^2 (H - H^*) - \mu (S - S^*)^2}{S} + \frac{\frac{\beta_1}{N}(Q - Q^*)^2 (S - S^*) + \frac{\beta_2}{N}(Q - Q^*)(H - H^*)(S - S^*) + \gamma\varepsilon (Q - Q^*)^2 - (\mu + \delta + \theta)(Q - Q^*)^2}{Q} + \frac{\theta (H - H^*)(Q - Q^*) - (\mu + \tau)(H - H^*)^2}{H} + \frac{\delta (A - A^*)(Q - Q^*) + \tau (A - A^*)(H - H^*) - (\mu + d)(A - A^*)^2}{A}$$

$$\frac{dV}{dt} = -\frac{(S - S^*)^2}{S}\frac{\beta_1}{N}(Q - Q^*) - \frac{(S - S^*)^2}{S}\left[\frac{\beta_2}{N} + \mu\right] + \frac{(Q - Q^*)^2}{Q}\gamma\varepsilon - \frac{(Q - Q^*)^2}{Q}(\mu + \delta + \theta) + \frac{(Q - Q^*)^2}{Q}\left(\frac{\beta_1}{N}\right)(S - S^*) - \frac{(H - H^*)^2}{H}(\mu + \tau) - \frac{(S - S^*)^2}{S}\psi + \frac{(Q - Q^*)}{Q}\left(\frac{\beta_2}{N}\right)(H - H^*)(S - S^*) - \frac{(H - H^*)}{H}\theta (Q - Q^*) + \frac{(A - A^*)}{A}\delta (Q - Q^*) + \frac{(A - A^*)}{A}\tau (H - H^*)$$

both positive and negative terms; $\frac{dV}{dt} = M - N$, such that M can be re written as;

$$= \frac{(S - S^*)}{S}\psi + \frac{(Q - Q^*)}{Q}\left(\frac{\beta_2}{N}\right)(S - S^*)(H - H^*) + \frac{(H - H^*)}{H}\theta (Q - Q^*) + \frac{(A - A^*)}{A}\delta (Q - Q^*) + \frac{(A - A^*)}{A}\tau (H - H^*) \tag{18}$$

Also,

$$N = \frac{(S - S^*)^2}{S}\left[\frac{\beta_1}{N}(Q - Q^*) + \frac{\beta_2}{N}(H - H^*) + \mu\right] + \frac{(Q - Q^*)^2}{Q}\left[\gamma\varepsilon - (\mu + \delta + \theta) + \frac{\beta_1}{N}(S - S^*)\right] + \frac{(H - H^*)^2}{H}(\mu + \tau) + \frac{(A - A^*)^2}{A}(\mu + d) \tag{19}$$

If $M < N$, then $\frac{dV}{dt}$ will be negative, $\frac{dV}{dt} = 0$, if and only if $S = S^*, Q = Q^*, H = H^*, A = A^*$

Thus, the largest compact invariant set is $\left\{(S^*, Q^*, H^*) \in \Omega : \frac{dV}{dt} = 0\right\}$ is just the singleton set $\{T^*\}$ is the Endemic Equilibrium, by LaSalle's Invariant principle, it implies that $T^*$ is globally asymptotically stable (g.a.s) in $\Omega$ if $M < N$.

**The Laplace Adomian Decomposition Method**

Here, we apply the Laplace –Adomian decomposition method to obtain the approximate result of the HIV/AIDS model. We represent the nonlinear terms by $V_n=\sum_{n=0}^{\infty}V_n(t)$ and $W_n=\sum_{n=0}^{\infty}W_n(t)$. Adding Laplace operator to both sides, we obtained;

$$\left.\begin{aligned}L\left(\frac{dS}{dT}\right)&=L(\psi)-\frac{\beta_1}{N}L(V)-\frac{\beta_2}{N}L(W)-\mu L(S)\\ L\left(\frac{dQ}{dT}\right)&=\frac{\beta_1}{N}L(V)+\frac{\beta_2}{N}L(W)+\gamma\varepsilon L(Q)-(\mu+\delta+\theta)L(Q)\\ L\left(\frac{dH}{dT}\right)&=\theta L(Q)-(\mu+\tau)L(H)\\ L\left(\frac{dA}{dT}\right)&=\delta L(Q)+\tau L(H)-(\mu+d)L(A)\end{aligned}\right\} \quad (20)$$

The Laplace transform of a function $f(t)$ with order n is defined as

$$L(f^n(t))=s^nL(f(t))-s^{n-1}L(f(0))-s^{n-2}L(f'(0))\cdots$$

$$\left.\begin{aligned}L(S(t))&=\frac{s_0(0)}{\alpha}+\frac{\psi}{\alpha}-\frac{\beta_1}{\alpha N}L(V)-\frac{\beta_2}{\alpha N}L(W)-\frac{\mu}{\alpha}L(S)\\ L(Q(t))&=\frac{q_0(0)}{\alpha}+\frac{\beta_1}{\alpha N}L(V)+\frac{\beta_2}{\alpha N}L(W)+\frac{(\gamma\varepsilon)}{\alpha}L(Q)-\frac{(\mu+\delta+\theta)}{\alpha}L(Q)\\ L(H(t))&=\frac{h_0(0)}{\alpha}+\frac{(\theta)}{\alpha}L(Q)-\frac{(\mu+\tau)}{\alpha}L(H)\\ L(A(t))&=\frac{a_0(0)}{\alpha}+\frac{\delta}{\alpha}L(Q)+\frac{\tau}{\alpha}L(H)-\frac{(\mu+d)}{\alpha}L(A)\end{aligned}\right\} \quad (21)$$

We assume the following series as the solution of each component

$$S_n=\sum_{n=0}^{\infty}S_n(t),Q_n=\sum_{n=0}^{\infty}Q_n(t),H_n=\sum_{n=0}^{\infty}H_n(t),A_n=\sum_{n=0}^{\infty}A_n(t)$$

Substituting into (11),

$$L\left(\sum_{n=0}^{\infty}S(t)\right)=\frac{s_0(0)}{\alpha}+\frac{\psi}{\alpha}-\frac{\beta_1}{\alpha N}L\left(\sum V_n(t)\right)-\frac{\beta_2}{\alpha N}L\left(\sum W_n(t)\right)-\frac{\mu}{\alpha}L\left(\left(\sum S_n(t)\right)\right)$$

$$L\left(\sum_{n=0}^{\infty}Q(t)\right)=\left\{\begin{aligned}&\frac{q_0(0)}{\alpha}+\frac{\beta_1}{\alpha N}L\left(\sum V_n(t)\right)+\frac{\beta_2}{\alpha N}L\left(\sum W_n(t)\right)\\ &+\frac{(\gamma\varepsilon)}{\alpha}L\left(\sum Q_n(t)\right)-\frac{(\mu+\delta+\theta)}{\alpha}L\left(\sum Q_n(t)\right)\end{aligned}\right\} \quad (22)$$

$$L\left(\sum_{n=0}^{\infty}H(t)\right)=\frac{h_0(0)}{\alpha}+\frac{(\theta)}{\alpha}L\left(\sum Q_n(t)\right)-\frac{(\mu+\tau)}{\alpha}L\left(\sum H_n(t)\right)$$

$$L\left(\sum_{n=0}^{\infty}A(t)\right)=\frac{a_0}{\alpha}+\frac{\delta}{\alpha}L\left(\sum Q_n(t)\right)+\frac{\tau}{\alpha}L\left(\sum H_n(t)\right)-\frac{(\mu+d)}{\alpha}L\left(\sum A_n(t)\right)$$

The Adomian polynomial for the nonlinear case can be represented as an infinite series whose sum are

$$V_n=\sum_{n=0}^{\infty}V_n(t)\ and\ W_n=\sum_{n=0}^{\infty}W_n(t)$$

Such that the Adomian polynomial obtained for each of the nonlinear class are

$$V_0=Q_0S_0,V_1=Q_0S_1+Q_1S_0,V_2=Q_0S_2+Q_1S_1+Q_2S_0$$

And

$$W_0=Q_0H_0,W_1=Q_0H_1+Q_1H_0,W_2=Q_0H_2+Q_1H_1+Q_2H_0$$

Inputting the non-linear terms

$$L\left(\sum_{n=0}^{\infty}S(t)\right)=\frac{s_0(0)}{\alpha}+\frac{\psi}{\alpha}-\frac{\beta_1}{\alpha N}L\left(\sum Q_nS_n(t)\right)-\frac{\beta_2}{\alpha N}L\left(H_nS_n(t)\right)-\frac{\mu}{\alpha}L\left(\left(\sum S_n(t)\right)\right) \quad (23)$$

$$L\left(\sum_{n=0}^{\infty}Q(t)\right)=\left\{\begin{aligned}&\frac{q_0(0)}{\alpha}+\frac{\beta_1}{\alpha N}L\left(\sum Q_nS_n(t)\right)+\frac{\beta_2}{\alpha N}L\left(\sum H_nS_n(t)\right)+\\ &\frac{(\gamma\varepsilon)}{\alpha}L\left(\sum Q_n(t)\right)-\frac{(\mu+\delta+\theta)}{\alpha}L\left(\sum Q_n(t)\right)\end{aligned}\right\} \quad (24)$$

For initial Approximation, at n=0

$$L(S(t))=\frac{s_0(0)}{\alpha}+\frac{\psi}{\alpha},L(Q(t))=\frac{q_0(0)}{\alpha},L(H(t))=\frac{h_0(0)}{\alpha},L(A(t))=\frac{a_0}{\alpha}-\frac{(\mu+\delta)}{\alpha} \quad (25)$$

Applying Laplace inverse operator, $S(t)=s_0+\psi,q_0=q_0,h_0=h_0,q_0=a_0$

$$L\left(\sum_{n=0}^{\infty}A(t)\right)=\frac{a_0}{\alpha}+\frac{\delta}{\alpha}L\left(\sum Q_n(t)\right)+\tau\frac{L\left(\sum H_n(t)\right)}{\alpha}-\frac{(\mu+d)}{\alpha}L\left(\sum A_n(t)\right)$$

Thus, comparing coefficient of first term and adding Laplace inverse for

The first iteration

$$S_1(t)=\left(-\frac{1}{2}\frac{\psi\beta_1q_0}{N}-\frac{1}{2}\frac{\psi\beta_2h_0}{N}-\frac{1}{2}\psi\mu\right)t^2+\left(-\frac{s_0\beta_1q_0}{N}-\frac{s_0\beta_2q_0}{N}-s_0\mu\right)t$$

$$Q_1(t)=\left(\frac{1}{2}\frac{\psi\beta_1q_0}{N}+\frac{1}{2}\frac{\psi\beta_2h_0}{N}\right)t^2+\left(\frac{s_0\beta_1q_0}{N}-q_0\mu+\frac{s_0\beta_2h_0}{N}-q_0\theta+q_0\gamma\varepsilon-q_0\delta\right)t$$

$$H_1(t)=(q_0\theta-h_0\mu-h_0\tau)t$$

and

$$A_1(t)=(q_0\delta+h_0\tau-a_0d-a_0d)t$$

Three iterations were performed to obtain the approximate results such that;

$$S(t) = s_0(t) + s_1(t) + s_2(t) + s_3(t)\ldots$$

$$Q(t) = Q_0(t) + Q_1(t) + Q_2(t) + Q_3(t)\ldots$$

$$H(t) = H_0(t) + H_1(t) + H_2(t) + H_3(t)\ldots$$

$$A(t) = A_0(t) + A_1(t) + A_2(t) + A_3(t) + \ldots$$

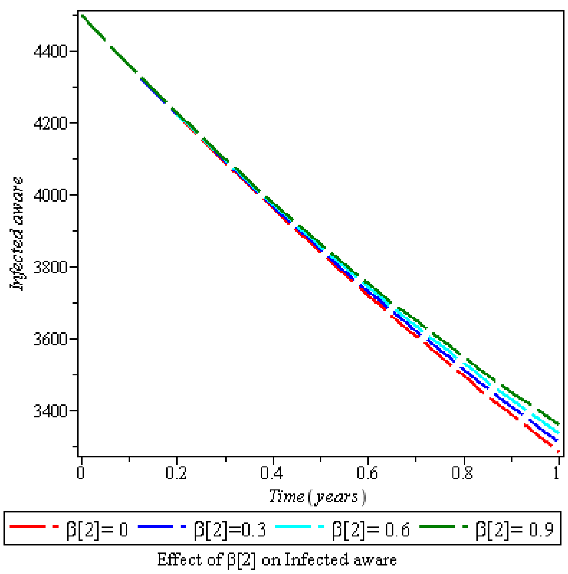


Figure 2: Effect of capital contact rate of aware infected $\beta_2$ on aware population

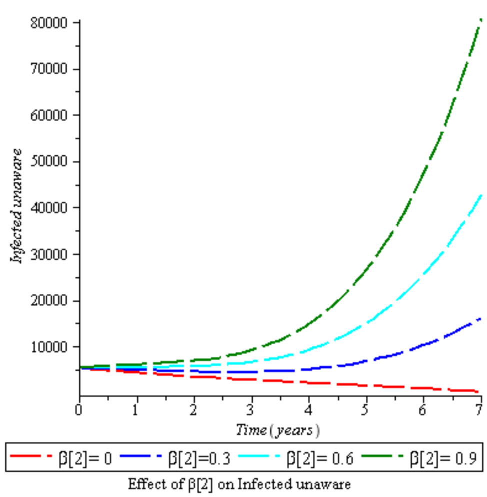


Figure 3: Effect of capital contact rate of aware infected $\beta_2$ on unaware population

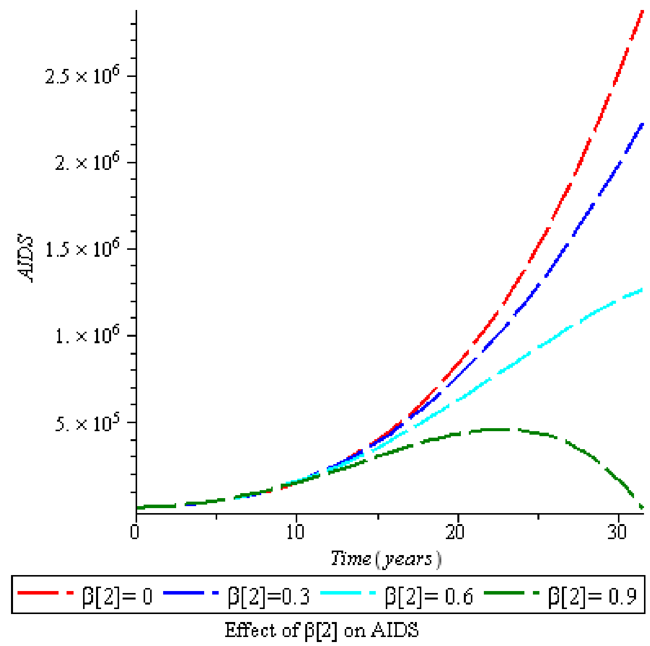


Figure 4: Effect of capital contact rate of aware infected $\beta_2$ on AIDS infected population

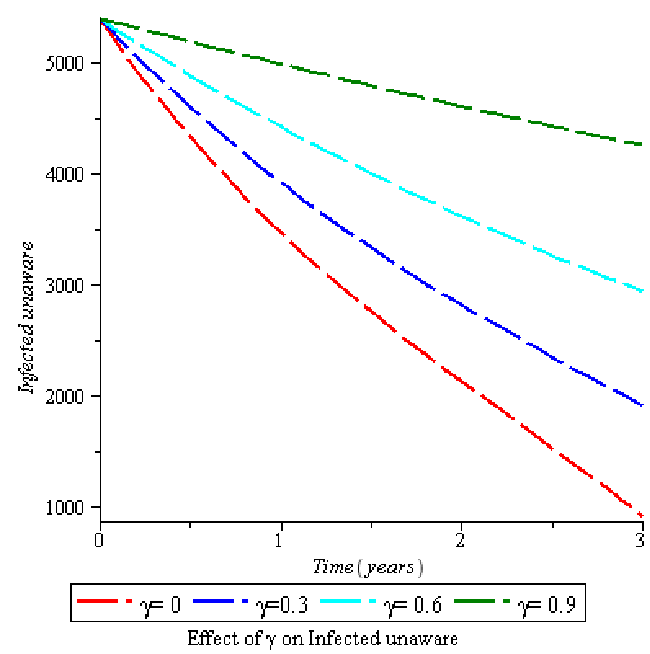


Figure 5: Effect of Birth rate of infected new born $\gamma$ on unaware population.

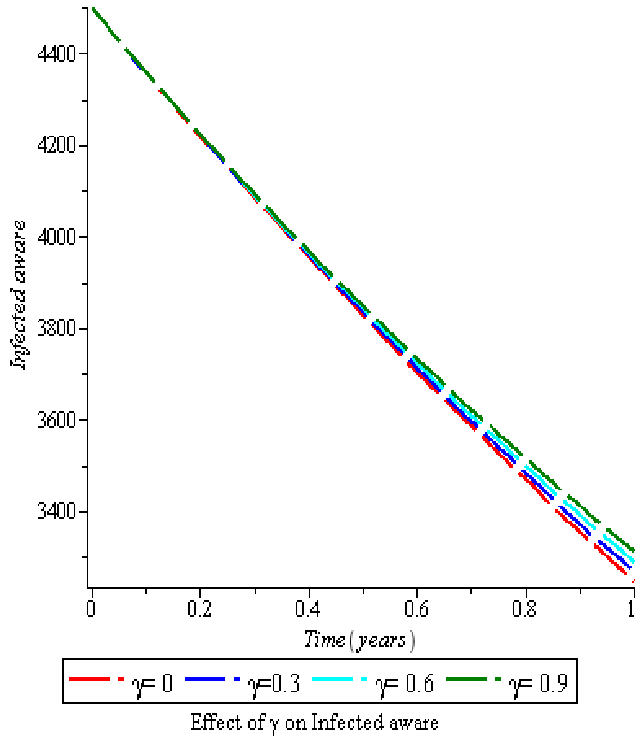


Figure 6: Effect of birth rate infected new born $\gamma$ on HIV infected aware population.

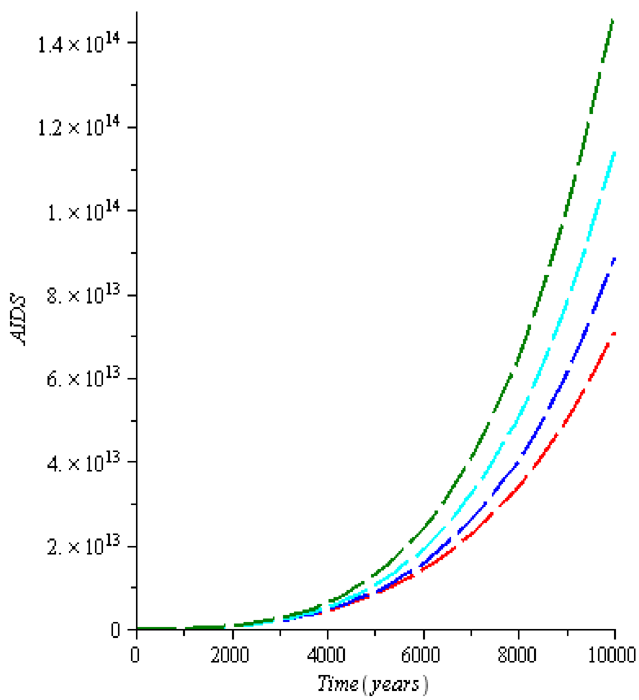


Figure 7: Effect of birth rate infected new born $\gamma$ on AIDS population

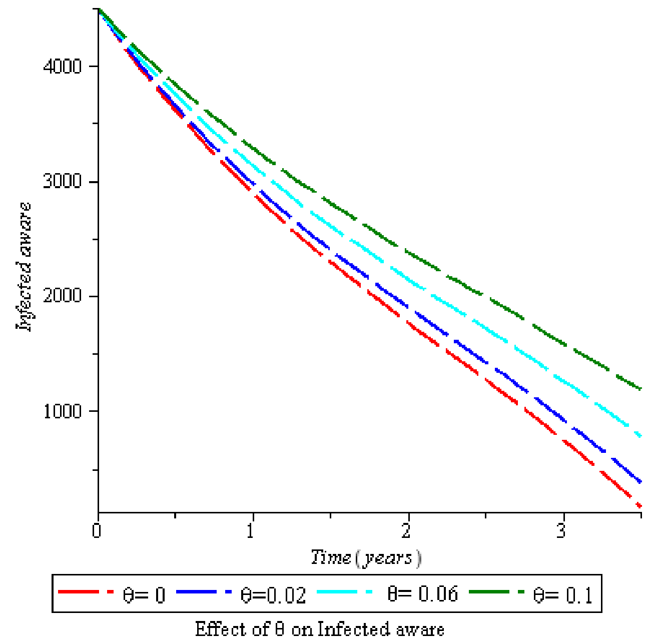


Figure 8: Effect of $\theta$ on aware infected population.

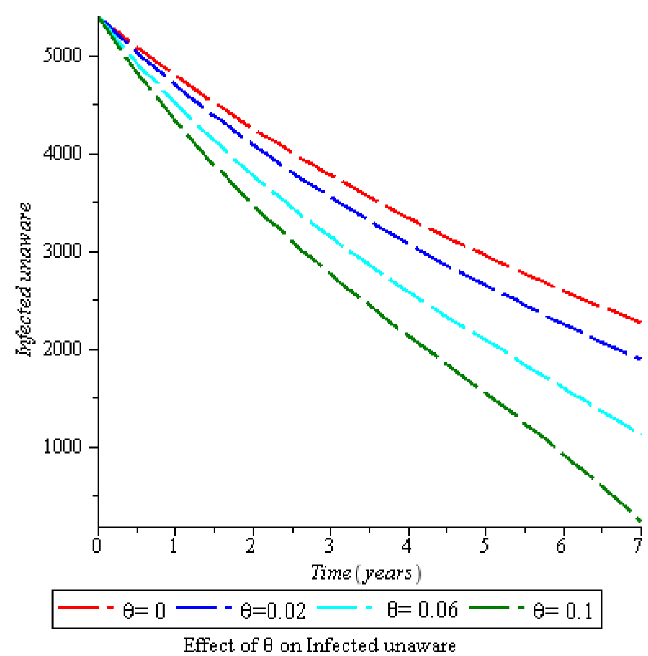


Figure 9: Effect of $\theta$ on Infected unaware population.

**Discussion**

The influence of capital contact rate of aware infected group is examined in the transmission dynamics of an HIV model. Similarly, the influence of the birth rate of infected new born is explored.

The study also evaluates the rate at which ignorant infectives' become aware infectives' by screening in the model's population.

Figure 2 depicts the effect of $\beta_2$ on the aware Infective class. When $\beta_2 = 0$, the system is stable. As $\beta_2$ rises from 0.3 to 0.6, so does the number of infected carriers. It was observed that when the effective contact rate progresses to 0.9, the population of the class reaches its peak and then drastically decreases to the minimum, this is an indication that if preventive measures are not taken to control the effective contact rate of the aware population class, the class may extinction as time progresses.

Figure 3 depicts the simulation results for the unaware infective and discovered that when $\beta_2$ increases sequentially from 0 to 0.9, the population of the group grows. This demonstrates that if preventative actions are not used to reduce the effective contact rate of the ignorant population class, the class may become extinct as time passes.

Figure 4 reveals the influence of $\beta_2$ on the AIDS class. As the contact rate grows from 0 to 0.9, the class population disappears. This shows that if HIV is to be lowered in the population, strategic steps to minimize $\beta_2$ should be implemented.

Figures 5–7 show the influence of the birth rate of infected new born babies on the aware, ignorant, and AIDS classes. The population of both the conscious and unaware classes grow as the number of infected new born babies' increases. Figure 7 depicts the AIDS class, which demonstrates that if the rate of infected new born infants does not decrease, the population of persons living with HIV/AIDS will rise. Figures 8 and 9 evaluate the effect of screening on the rate at which unaware infective become aware infective in the three specified classes. As $\theta$ goes from 0 to 0.1, the population of the class increases to its maximum level in the conscious compartment. Contrary, when the level grows from 0 to 0.1, the population of the unaware class shrinks considerably. This illustrates the relevance of HIV screening in the community's eradication, it is believed that if more people are aware that they are infected, the rate at which they transmit the illness will be significantly lowered, and the population will eventually be free of HIV/AIDS as time passes.

**Conclusion.**

In this work, a mathematical model of HIV/AIDS with vertical transmission and screening is proposed and studied the stability of the disease-free and endemic equilibrium was also examined throughout the model analysis. The next generating matrix is used to determine the basic reproductive number $R_0$. In order to successfully eradicate the presence of HIV/AIDS in the community, more individuals must be screened to determine their HIV status and preventative measures must be implemented to stop the virus from spreading. It should also be mentioned that vertical disease transmission should be monitored in order to limit the pace at which HIV-infected newborns are recruited into the population. The numerical result of this study, HIV/AIDS will be eradicated from society.

**Conflict of interest**

The manuscript was read and approved by all the authors. They therefore declare that there is no conflict of interest.

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