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Mathematical Modelling of Dynamics of HIV Transmission Depicting the Importance of Counseling and Treatment

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ABSTRCT: Human immunodeficiency virus is an incurable disease which attacks and destroys the human immune system thereby making the body susceptible to all kinds of illnesses. If left unattended to, it can lead to the damaging of body organs such as the brain, kidney and the heart which can result to death. Unfortunately this disease has no known cure till date but through counseling and administering of antiretroviral drugs, the likelihood of dying from it becomes minimal. This study presents the deterministic HIV transmission model. The model has a unique endemic equilibrium point which is locally asymptotically stable if $R_0 > 1$, DFE of the model was obtained and is shown to be Local asymptotically stable when the associated basic reproduction number was R > 1. We established the numerical simulation of the model which shows that the effective use of condom, counseling or the use of anti-retrovirus drug can lead to effective reduction on HIV transmission. Finally, we discussed that the ART treatment rate will reduce the basic reproduction number R_0 hence, leading to the extinction of HIV/AIDS.

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Human immunodeficiency virus (HIV) infection destroys the body immune system, damages body organs such as the brain, kidney, heart etc. and causes death. Unfortunately, this disease has no cure. Human immunodeficiency virus (HIV) is thought to have originated in non-human primates in sub-Saharan Africa and was transferred to humans late in the 19th or early in the 20th century. Both HIV-1 and HIV-2 are believed to have originated in West-Central Africa and to have jumped species (a process known as zoonosis) from non-human primates to humans. Once HIV enters the body, its main target is the white blood cell, known as $CD4^+$ T cell; however, it can take two to ten weeks for an individual exposed to HIV to produce measurable quantities of antibody. When the $CD4^+$ T cell count which is normally around 1000mm reaches 200mm or below in an HIV infected patient; then that is classified as having AIDS. Because of the central role of $CD4^+$ T cells in the immune regulation, their depletion has widespread deleterious effects on the functioning of the immune system as a whole and lead to the immunodeficiency that characterized AIDS. HIV transmission as a result of sexual intercourse accounts for about three-quarters of all HIV infection world-wide (WHO 1993), HIV is therefore a sexually transmitted disease (STD). Although transmission through intercourse between men occurs in most parts of the world, the majority of the world's infections have been acquired through intercourse between men and women (heterosexual transmission) Studies have shown that the likelihood of being infected increases statistically with the number of sexual partners and with anal receptive intercourse [Gaodert F. *et al* (1944), Pickering, J. (1986)]. As with some other STDs, HIV infection can also be transmitted through blood, the transmission of HIV from mother to child includes transmission during pregnancy, during delivery and through breast-feeding (World Health Organization (2008)). Several investigations have been conducted to study the dynamics of HIV/AIDS, in particular, Srinivasa Rao Srinivasa Rao, A.S.R. (2003) presented a theoretical framework for transmission of HIV/AIDS epidemic in India. It is pointed out that the screening of infectives has substantial e§ect on the spread of AIDS. Mathematical model of disease outbreaks can be helpful by providing forecasts for the development of the epidemic that account for the complex and nonlinear dynamic of infectious disease and by rejecting the likely impact of proposed intervention before they are implemented. Therefore, the objective

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of this paper is to mathematically model the dynamics of HIV/AIDS transmission depicting the importance of counseling and treatment

Model formulation: In this model, the total population of size $N(t)$ is divided in to seven (7) epidemiological classes namely susceptible population $S(t)$ with natural death rate of μs, the population of exposed individuals $E(t)$ natural death rate μ, the infected individuals with counseling $I_C(t)$ with natural death rate μ, the non-counseling infected individuals with natural death rate μ, the AIDS individuals with counseling $A_C(t)$ with natural death rate μ and death cause by the virus ω_1, the AIDS class of non-counseling infected individual $A_N(t)$ with two death rate that is natural death rate and death by the virus at μ and ω_2 also treatment class $T(t)$ with natural death rate and death rate cause by a virus as well. The total population which is denoted $N(t)$ by is obtained as below

$$N(t) = S(t) + E(t) + I_C(t) + I_N(t) + A_C(t) + A_N(t) + T(t)$$

The susceptible population can be infected when they come in contact with infected individuals and is increased by the recruitment of individuals (assumed susceptible) into the sexually-active population at a rate π. These individuals acquire HIV infection, following effective contact with infected individuals in the E, I_C, I_N, A_C, A_N, T classes, at a rate λ. The recruitment of susceptible is assumed to occur at a constant rate for, the rate at which susceptible individuals acquire infection is given by

$$\lambda = \frac{\beta(1-a\alpha)\left[I_N + l_1 A_N + \theta_1(I_C + l_2 A_C) + l_3 T\right]}{N}$$

Is the force of infection. The population of the susceptible class is given as;

$$\frac{dS}{dt} = \pi - (\lambda + \mu)S + \theta_2 E$$

The differential equation that describes that population of exposed group is given as

$$\frac{dE}{dt} = \lambda S - (\theta_2 + \delta_1 + \delta_2 + \mu)E$$

The population of exposed group is reduced is three ways, either by natural death or they move to the infected group under counseling I_C and non-counseling infected individuals I_N at the rate of δ_1 and δ_2.

The population of counseling infected individuals is reduced in three ways either moved to treatment class, death naturally or moved to AIDS class of non-counseling infected individuals.

$$\frac{dI_C}{dt} = \delta_1 E - (k + \rho + \mu)I_C + \nu I_N$$

The population of non-counseling infected individuals is reduced in three ways either moved to treatment class, death naturally or moved to AIDS class of non-counseling infected individuals

$$\frac{dI_N}{dt} = \delta_2 E - (\gamma + \mu + e + \nu)I_N$$

The population of AIDS class with counseling infected individuals is reduced either by natural death rate, death caused by virus or moved to treatment class.

$$\frac{dA_C}{dt} = \gamma I_N - (\omega_1 + \mu + \xi)A_C$$

The non-counseling infected AIDS class is reduced in three ways whether death caused virus, natural death or they move to treatment class

$$\frac{dA_N}{dt} = \gamma I_N - (\omega_2 + \mu + \varepsilon)A_N$$

The treatment class is reduced in two ways, either by natural death rate or death caused by virus

$$\frac{dT}{dt} = kI_C + eI_N + \zeta A_C + \varepsilon A_N - (\omega_3 + \mu)T$$

AIDS and treated individuals suffer additional disease-induced mortality at a rate ω_1, ω_2 and ω_3 respectively and natural mortality occurs in all classes at a rate μ.

Table 1: Variable of the Model

Variable Variable	Description
$S(t)$	Susceptible population at a given time (t)
$E(t)$	Population of exposed individuals at a given time (t)
$I_C(t)$	Population of infected individuals with counseling at a given time (t)
$I_N(t)$	Population of non-counseling infected individuals at given time
$A_C(t)$	Population of AIDS individuals with counseling at a given time (t)
$A_N(t)$	Population of AIDS individuals with non-counseling at a given time (t)
$T(t)$	Treatment class at a given time (t)

Table 2: Parameters of the Model and their Description

Parameter	Description
π	Recruitment rate
β	Effective rate contact
δ_1	Progression rate from $E(t)$ to $I_C(t)$
δ_2	Progression rate from $E(t)$ to $I_N(t)$
V	Progression rate from $I_N(t)$ to $I_C(t)$
e	Progression rate from $I_N(t)$ to $T(t)$
γ	Progression rate from $I_N(t)$ to $A_N(t)$
ρ	Progression rate from $I_C(t)$ to $A_C(t)$
ε	Progression rate from $A_N(t)$ to $T(t)$
ξ	Progression rate from $A_C(t)$ to $T(t)$
θ_2	Progression rate from $E(t)$ to $S(t)$
k	Progression rate from I_C to T
μ	Natural mortality rate
N	Total population
ω_1, ω_2 and ω_3	Death rate caused by virus
l_1, l_2 and l_3	Are relative risk of infection by A_N, A_C, T
θ_1	Modification parameter which is assumed to be infected individuals that modify their sexual behavior positively $\theta_1 < 1$
α	Measures complience in condom use
a	Condom efficacy

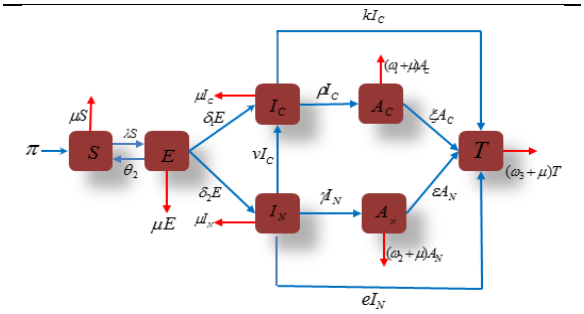

Fig. 1. Schematic Diagram of the Model

The model equations: In summary, the model for HIV transmission consists of the following deterministic system of non-linear differential equations (see Figure 1 above for a flow chart diagram of the model; the associated variables and parameters are described in Tables 1 and 2, respectively). HIV transmission dynamic between the compartments will be illustrated by the system of DE which we solved to obtain both the disease free equilibrium state and endemic equilibrium state. Hence, using the schematic flow diagram of HIV in figure 1 we obtained the following system of equation which illustrates the dynamics of HIV.

$$\frac{dS}{dt} = \pi - (\lambda + \mu)S + \theta_2 E$$

$$\frac{dE}{dt} = \lambda S - (\theta_2 + \delta_1 + \delta_2 + \mu)E$$

$$\frac{dI_C}{dt} = \delta_1 E - (k + \rho + \mu)I_C + vI_N$$

$$\frac{dA_C}{dt} = \rho I_C - (\omega_1 + \mu + \xi)A_C$$

$$\frac{dI_N}{dt} = \delta_2 E - (\gamma + \mu + e + v)I_N$$

$$\frac{dA_N}{dt} = \gamma I_N - (\omega_2 + \mu + \varepsilon)A_N$$

$$\frac{dT}{dt} = kI_C + eI_N + \xi A_C + \varepsilon A_N - (\omega_3 + \mu)T$$

STABILITY ANALYSIS OF DFE

Theorem 1 The disease-free equilibrium E_Q of the system (2.1) is locally asymptotically stable whenever $R_0 < 1$ and unstable when $R_0 > 1$.

Proof

The Jacobian matrix of the system (3.1), evaluated at J_E, is given by

$$J_E = \begin{bmatrix} -(\lambda+\mu) & \theta_2 & \frac{-\beta(1-a\alpha)\theta_1 S^*}{N} & \frac{\beta(1-a\alpha)\theta_1 l_2 S^*}{N} & \frac{-\beta(1-a\alpha)S^*}{N} & \frac{-\beta(1-a\alpha)l_1 S^*}{N} & \frac{-\beta(1-a\alpha)l_2 S^*}{N} \\ \lambda & -k_1 & \frac{\beta(1-a\alpha)\theta_1 S^*}{N} & \frac{\beta(1-a\alpha)\theta_1 l_2 S^*}{N} & \frac{\beta(1-a\alpha)S^*}{N} & \frac{\beta(1-a\alpha)S^*}{N} & \frac{\beta(1-a\alpha)l_2 S^*}{N} \\ 0 & \delta_1 & -k_2 & 0 & \nu & 0 & 0 \\ 0 & 0 & \rho & -k_3 & 0 & 0 & 0 \\ 0 & \delta_2 & 0 & 0 & -k_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma & -k_5 & 0 \\ 0 & 0 & k & \xi & e & \varepsilon & -k_6 \end{bmatrix}$$

At E_0 DFE we have,

$$J_{E_0} = \begin{bmatrix} -\mu & \theta_2 & -T_1 & -T_2 & -T_3 & -T_4 & -T_5 \\ 0 & -k_1 & T_1 & T_2 & T_3 & T_4 & T_5 \\ 0 & \delta_1 & -k_2 & 0 & \nu & 0 & 0 \\ 0 & 0 & \rho & -k_3 & 0 & 0 & 0 \\ 0 & \delta_2 & 0 & 0 & -k_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma & -k_5 & 0 \\ 0 & 0 & k & \xi & e & \varepsilon & -k_6 \end{bmatrix}$$

Reduce the Jacobian above in upper triangular matrix, we have,

$$J_{E_0} = \begin{bmatrix} -\mu & \theta_2 & -T_1 & -T_2 & -T_3 & -T_4 & -T_5 \\ 0 & -k_1 & T_1 & T_2 & T_3 & T_4 & T_5 \\ 0 & 0 & -\frac{k_1k_2 - T_1\delta_1}{k_1} & \frac{\delta_1 T_2}{k_1} & \frac{\nu k_1 + T_3\delta_1}{k_1} & \frac{\delta_1 T_4}{k_1} & \frac{\delta_1 T_5}{k_1} \\ 0 & 0 & 0 & -T_6 & \frac{\rho(\nu k_1 + T_3\delta_1)}{k_1k_2 - T_1\delta_1} & \frac{\rho\delta_1 T_4}{k_1k_2 - T_1\delta_1} & \frac{\rho\delta_1 T_5}{k_1k_2 - T_1\delta_1} \\ 0 & 0 & 0 & 0 & -T_7 & -T_8 & -T_9 \\ 0 & 0 & 0 & 0 & 0 & -T_{10} & -T_{11} \\ 0 & 0 & 0 & 0 & 0 & 0 & -T_{12} \end{bmatrix}$$

Whose eigenvalues are;

$$\lambda_1 = -\mu, \lambda_2 = -k_1, \lambda_3 = \frac{-(k_1k_2 - T_1\delta_1)}{k_1}, \lambda_4 = -T_6, \lambda_5 = -T_7,$$

$$\lambda_6 = -T_{10}, \lambda_7 = -T_{12}$$

Now,

$$\lambda_3 = \frac{(T_1\delta_1 - k_1k_2)}{k_1}, \lambda_4 = \frac{-\left[\rho T_2\delta_1 + k_3\left(T_1\delta_1 - k_1k_2\right)\right]}{T_1\delta_1 - k_1k_2}$$

$$\lambda_5 = -\frac{\left[v\rho T_2\delta_2 + vk_3T_6\delta_1 - k_1k_2k_3k_7 + k_3k_4(T_1\delta_1 - k_1k_2) + k_2k_3T_3\delta_2\right]}{\rho T_2\delta_1 + k_3(T_1\delta_1 - k_1k_2)}$$

$$\lambda_6 = \frac{\gamma k_2k_3\delta_2T_4 + v\rho k_5T_2\delta_2 + vk_3k_5T_1\delta_2 + \rho k_4k_5T_2\delta_1 + k_2k_3k_4T_3\delta_2 + k_3k_4k_5(T_1\delta_1 - k_1k_2)}{v\rho T_2\delta_2 + vk_3T_1\delta_2 + \rho k_4T_2\delta_1 + k_2k_3T_3\delta_2 + k_3k_4(T_1\delta_1 - k_1k_2)}$$

$$\lambda_7 = \frac{\left(\begin{array}{l} ek_2k_3k_5T_5\delta_2 + \varepsilon\gamma k_2k_3T_5\delta_2 + kvk_3k_5T_5\delta_2 + kk_3k_4k_5T_5\delta_1 + \rho k_4k_5k_6T_2\delta_1 + vk_3k_5k_6T_1\delta_2 \\ + v\rho\xi k_5T_5\delta_2 + \rho\xi k_4k_5T_5\delta_1 + \gamma k_2k_3k_6\delta_2T_4 + k_2k_3k_4k_5k_6T_3\delta_2 + + \rho vk_5k_6T_2\delta_2 + \rho\xi k_4k_5T_5\delta_1 + \\ k_3k_4k_5k_6(T_1\delta_1 - k_1k_2) \end{array}\right)}{\left(\gamma k_2k_3\delta_2T_4 + v\rho k_5T_2\delta_2 + vk_3k_5T_1\delta_2 + \rho k_4k_5T_2\delta_1 + k_3k_4k_5(T_1\delta_1 - k_1k_2) + k_2k_3k_5T_3\delta_2\right)}$$

Hence, if $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5 < 0$ if $R_0 < 1$ and $T_1\delta_1 > k_1k_2$ then the DFE is LAS

where, $T_1 = \beta(1-a\alpha)\theta_1,\ T_2 = \beta(1-a\alpha)\theta_1l_1,\ T_3 = \beta(1-a\alpha)\theta_1l_2,$

$$T_4 = \beta(1-a\alpha)l_1,\ T_5 = \beta(1-a\alpha)l_2,\ T_8 = \frac{k_3\delta_2T_4k_2}{\rho T_2\delta_1 - k_1k_2k_3 + k_3T_1\delta_1}$$

$$T_9 = \frac{k_3\delta_2T_5k_2}{\rho T_2\delta_1 - k_1k_2k_3 + k_3T_1\delta_1}$$

$$T_{11} = \frac{\gamma k_3\delta_2T_5k_3}{\gamma k_2k_3\delta_2T_4 + v\rho k_5T_7\delta_2 + vk_3k_5T_1\delta_2 + \rho k_4k_5T_2\delta_1 - k_1k_2k_3k_4k_5 + k_2k_3k_4T_3\delta_2 + k_3k_4k_5T_1\delta_1}$$

when we substitute R_0 in T_{12} we have

$$R_0 = \frac{T_1v\delta_2 + T_1k_4\delta_1}{k_1k_2k_4} + \frac{\rho T_2(v\delta_2 + k_4\delta_1)}{k_1k_2k_3k_4} + \frac{T_3\delta_2}{k_1k_4} + \frac{\gamma\delta_2T_4}{k_1k_4k_5} + \frac{T_5k_7}{k_1k_2k_3k_4k_5k_6}$$
$$+ \frac{T_5ek_2k_3k_5\delta_2 + T_5\varepsilon\gamma k_2k_3\delta_2 + T_5kvk_3k_5\delta_2 + T_5kk_3k_4k_5\delta_1 + T_5v\rho\xi k_5\delta_2 + T_5\rho\xi k_4k_5\delta_1}{k_1k_2k_3k_4k_5k_6}$$

$$T_{12} = \frac{k_1k_2k_3k_4k_5k_6}{K}\left\{\begin{array}{l} T_1v\delta_2k_3k_5k_6 + T_1\delta_1k_3k_4k_5k_6 + T_2\rho v\delta_2k_5k_6 + T_2\delta_1\rho k_4k_5k_6 + T_3\delta_2k_2k_3k_5k_6 + T_4\gamma\delta_2k_2k_3k_6 + \\ e\delta_2k_2k_3k_5T_5 + \varepsilon\gamma\delta_2k_2k_3T_5 + kv\delta_2k_3k_5\mathrm{T}_5 + k\delta_1k_3k_4k_5T_5 + v\rho\xi\delta_2k_5T_5 + \rho\xi\delta_1k_4k_5T_5 - 1 \end{array}\right\}$$

$$-T_{12} = \frac{k_1k_2k_3k_4k_5k_6}{K}\left[R_0 - 1\right]$$

Where,

$$K = \gamma k_2k_3\delta_2T_4 + v\rho k_5T_2\delta_2 + vk_3k_5T_1\delta_2 + \rho k_4k_5T_2\delta_1 - k_1k_2k_3k_4k_5 + k_2k_3k_4T_3\delta_2 + k_3k_4k_5T_1\delta_1$$
$$-K = \left(k_1k_2k_3k_4k_5 - \gamma k_2k_3\delta_2T_4 - v\rho k_5T_2\delta_2 - vk_3k_5T_1\delta_2 - \rho k_4k_5T_2\delta_1 - k_2k_3k_4T_3\delta_2 - k_3k_4k_5T_1\delta_1\right)$$
$$\Rightarrow\ -T_{12} = -\frac{k_1k_2k_3k_4k_5k_6}{K}\left[R_0 - 1\right]$$

$$\Rightarrow\ T_{12} = \frac{k_1k_2k_3k_4k_5k_6}{K}\left[R_0 - 1\right]$$

Theorem 2 The DFE of the model is GAS if $R_0 < 1$ and unstable if otherwise.

Proof

Consider the HIV model (1); the proof is based on using the comparison theorem. Since all the off- diagonal entries of the Jacobian matrix of the infected compartments of the HIV model are non-negative at DFE (E_0). We follow the approach of Abba Gumel (2010) in proving the theorem. Let $R_0 < 1,$

Then the infected compartments of the model (2.1) can be re-written as

$$\frac{dx}{dt} = (f - v)x - Jx \quad \text{.....(2.2)}$$

$$where\ x = [E, I_C, A_C, I_N, A_N, T]^T,$$

Then,

$$J_x = \left(1 - \frac{S}{N}\right)\begin{bmatrix} 0 & \beta\theta_1 & \beta\theta_1 l_1 & \beta\theta_2 l_1 & \beta l_1 & \beta l_2 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

This worth noting that fx is non-negative matrix, since $S(t) \le N(t)$ in the invariant set. Hence it follows that

$$\frac{dx}{dt} \le (f - v)x \text{.....(2.3)}$$

Since all the eigenvalues of the matrix $(f - v)$ are all having a negative real parts (from local stability, where $\rho(fv)^{-1} < 1$ if $R_0 < 1$ which is equivalent to $(f - v)$ having a negative real parts when $R_0 < 1$,hence by comparison theorem $\lim_{n\to\infty}(E, I_C, A_C, I_N, A_N, T) = (0,0,0,0,0,0)$

Now substituting $E = I_C = A_C = I_N = A_N = T = 0$ in the first equation of the model (2.1) shows that $S(t) \to S^* \ as \ t \to \infty$. Thus, $\lim_{n\to\infty}(E, I_C, A_C, I_N, A_N, T) = \varepsilon_0$.

The Existence of Endemic Equilibrium Point: This is a case where there is infection in the study population. We find the condition for the existence of endemic equilibrium for the proposed model, the endemic equilibrium are obtained in terms of infection of the force of infection.

Let $E_Q = \{S^*, E^*, I_C{}^*, A_C{}^*, I_N{}^*, A_N{}^*, T^*\}$ represent endemic equilibrium of the model (1). Solving the equations of the model (2.1) at steady state we get,

$$\pi - (\lambda + \mu)S + \theta_2 E = 0$$
$$\lambda S - (\theta_2 + \delta_1 + \delta_2 + \mu)E = 0$$
$$\delta_1 E - (k + \rho + \mu)I_C + vI_N = 0$$
$$\rho I_C - (\omega_1 + \mu + \xi)A_C = 0$$
$$\delta_2 E - (\gamma + \mu + e + v)I_N = 0$$
$$\gamma I_N - (\omega_2 + \mu + \varepsilon)A_N = 0$$
$$kI_C + eI_N + \xi A_C + \varepsilon A_N - (\omega_3 + \mu)T = 0$$

$$S^* = \frac{\pi + \theta_2 E^*}{\lambda + \mu}$$

$$= \frac{\pi + \theta_2\left(\frac{\lambda S^*}{k_1}\right)}{\lambda + \mu} = \frac{k_1\pi + \theta_2\lambda S^*}{k_1(\lambda + \mu)}$$

$$\Rightarrow k_1\pi + \theta_2\lambda S^* = S^*\{k_1(\lambda + \mu)\}$$

$$\Rightarrow S^* = \frac{k_1\pi}{[k_1(\lambda + \mu) - \theta_2\lambda]} = \frac{k_1\pi}{P}$$

$$E^* = \frac{\lambda\pi}{[k_1(\lambda + \mu - \theta_2\lambda)]} = \frac{\lambda\pi k_1}{p}$$

$$I_C{}^* = \frac{\delta_1\left\{\frac{\lambda\pi k_1}{p}\right\} + \nu\left\{\frac{\delta_2\lambda\pi k_1}{pk_4}\right\}}{k_2} = \frac{\lambda\pi[\delta_1 k_1 k_4 + \nu\delta_2 k_1]}{pk_2k_4}$$

$$I_N{}^* = \frac{\delta_2\lambda\pi k_1}{pk_4}$$

$$A_N{}^* = \frac{\gamma\delta_2\lambda\pi k_1}{pk_4k_5}$$

$$A_C{}^* = \frac{\rho\lambda\pi k_1[k_4\delta_1 + \nu\delta_2]}{pk_2k_3k_4}$$

$$T^* = \frac{kk_1k_3k_5k_6\lambda\pi[k_4\delta_1 + \nu\delta_2]}{pk_2k_4} + \frac{e\delta_2\pi\lambda k_1}{pk_4} + \frac{\xi\rho\lambda\pi k_1[k_4\delta_1 + \nu\delta_2]}{pk_2k_3k_4} + \frac{\varepsilon\gamma\delta_2\lambda\pi k_1}{pk_4k_5}$$

$$\Rightarrow T^* = \frac{kk_1k_3k_5k_6\lambda\pi[k_4\delta_1 + \nu\delta_2] + \delta_2 e\lambda\pi k_2k_3k_5 + \xi\rho\pi\lambda k_1k_5k_6[k_4\delta_1 + \nu\delta_2] + \varepsilon\gamma\delta_2\lambda\pi k_1k_2k_3k_6}{pk_2k_3k_4k_5k_6}$$

The endemic equilibrium point in terms of infection class is written as;

$$E_Q = \left\{ \begin{array}{l} \frac{\pi k_1}{P}, \frac{\lambda^*\pi k_1}{p}, \frac{\lambda^*\pi k_1[\delta_1 k_4 + \nu\delta_2]}{pk_2k_4}, \frac{\delta_2\lambda^*\pi k_1}{pk_4}, \frac{\rho\lambda^*\pi k_1[k_4\delta_1 + \nu\delta_2]}{pk_2k_3k_4}, \frac{\gamma\delta_2 k_1\lambda^*\pi}{pk_4k_5}, \\ \\ \frac{kk_1k_3k_5k_6\lambda^*\pi[k_4\delta_1 + \nu\delta_2] + \delta_2 e\lambda^*\pi k_2k_3k_5 + \xi\rho\pi\lambda^* k_1k_5k_6[k_4\delta_1 + \nu\delta_2] + \varepsilon\gamma\delta_2\lambda^*\pi k_1k_2k_3k_6}{pk_2k_3k_4k_5k_6} \end{array} \right\}$$

where $p = Pk_1, k_2 = k + \rho + \mu, k_3 = \omega_1 + \mu + \xi, k_4 = \gamma + \mu + e + \nu,\ k_5 = \omega_2 + \mu + \varepsilon, k_6 = \omega_3 + \mu$

We have

$$N^*(t) = S^*(t) + E^*(t) + I_C{}^*(t) + I_N{}^*(t) + A_C{}^*(t) + A_N{}^*(t) + T^*(t)$$

$$\Rightarrow N^* = \left\{ \begin{array}{l} \frac{\pi k_1}{P} + \frac{\lambda \pi k_1}{p} + \frac{\lambda \pi k_1 \left[\delta_1 k_4 + \nu \delta_2\right]}{p k_2 k_4} + \frac{\delta_2 \lambda \pi k_1}{p k_4} + \frac{\rho \lambda \pi k_1 \left[k_4 \delta_1 + \nu \delta_2\right]}{p k_2 k_3 k_4}, \frac{\gamma \delta_2 k_1 \lambda^* \pi}{p k_4 k_5} + \\ \\ \frac{k k_1 k_3 k_5 k_6 \lambda \pi \left[k_4 \delta_1 + \nu \delta_2\right] + \delta_2 e \lambda \pi k_2 k_3 k_5 + \xi \rho \pi \lambda k_1 k_5 k_6 \left[k_4 \delta_1 + \nu \delta_2\right] + \varepsilon \gamma \delta_2 \lambda \pi k_1 k_2 k_3 k_6}{p k_2 k_3 k_4 k_5 k_6} \end{array} \right\}$$

$$\Rightarrow N^* = \frac{\left\{ \begin{array}{l} k_1 k_2 k_3 k_4 k_5 k_6 \pi + \lambda \pi k_2 k_3 k_4 k_5 k_6 + k_3 k_5 \lambda \pi \left[k_4 \delta_1 + \nu \delta_2\right] + \delta_2 \lambda \pi k_2 k_3 k_5 k_6 + \gamma \delta_2 \lambda \pi k_2 k_3 k_6 + \\ \rho \lambda \pi k_5 \left[k_4 \delta_1 + \nu \delta_2\right] + k k_3 k_5 \lambda \pi \left[k_4 \delta_1 + \nu \delta_2\right] + e \alpha \lambda \pi k_2 k_3 k_5 + \xi p \lambda \pi k_5 \left[k_4 \delta_1 + \nu \delta_2\right] + \\ \varepsilon \lambda \pi \delta_2 k_2 k_3 \end{array} \right\}}{k_2 k_3 k_4 k_5 k_6 p}$$

The force of infection λ^{**} can be expressed, at steady state as

$$\lambda^{**} = \frac{\beta(1-a\alpha)\left(I_N{}^{**} + l_1 A_N{}^{**} + \theta_1 (I_C{}^{**} + l_2 A_C{}^{**}) + l_3 T^{**}\right)}{N^*} \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (3.2)$$

Let $H = \beta(1-a\alpha)$, Substituting the $I_N{}^{**}, A_N{}^{**}, I_C{}^{**}, A_C{}^{**}, T^{**}$ in (3.2), we have

$$\lambda^{**} = H \left\{ \frac{\left\{ \begin{array}{l} \frac{\delta_2 \lambda \pi k_1}{p k_4} + \frac{l_1 \gamma \delta_2 \lambda \pi k_1}{p k_4 k_5} + \frac{\theta_1 \lambda \pi k_1 \left[\delta_1 k_4 + \nu \delta_2\right]}{p k_2 k_4} + \frac{\theta_1 l_2 \rho \lambda \pi k_1 \left[k_4 \delta_1 + \nu \delta_2\right]}{p k_2 k_3 k_4} \\ \frac{l_3 k k_1 k_3 k_5 k_6 \lambda \pi \left[k_4 \delta_1 + \nu \delta_2\right] + l_3 \delta_2 e \lambda \pi k_2 k_3 k_5 + l_3 \xi \rho \pi \lambda k_1 k_5 k_6 \left[k_4 \delta_1 + \nu \delta_2\right] + l_3 \varepsilon \gamma \delta_2 \lambda \pi k_1 k_2 k_3 k_6}{p k_2 k_3 k_4 k_5 k_6} \end{array} \right\}}{N^*} \right\}$$

$$\Rightarrow H \left\{ \begin{array}{l} \delta_2 \pi k_2 k_3 k_5 k_6 + \gamma \delta_2 \pi l_1 k_2 k_3 k_6 + \theta_1 \pi k_1 k_3 k_5 \left[k_4 \delta_1 + \nu \delta_2\right] k_6 + \theta_1 l_2 \rho \pi k_5 \left[k_4 \delta_1 + \nu \delta_2\right] + k k_3 k_5 \pi \\ \\ \left[k_4 \delta_1 + \nu \delta_2\right] + \delta_2 e \pi k_2 k_3 k_5 + \xi \rho \pi k_5 \left[k_4 \delta_1 + \nu \delta_2\right] + \varepsilon \gamma \delta_2 \pi k_2 k_3 \end{array} \right\} =$$

$$k_1 k_2 k_3 k_4 k_5 k_6 \pi + \lambda \left\{ \begin{array}{l} \pi k_2 k_3 k_4 k_5 + k_3 k_4 \pi \left[k_4 \delta_1 + \nu \delta_2\right] \alpha_2 \pi k_2 k_3 k_4 + \gamma \delta_2 \pi k_2 k_3 + \rho \pi k_5 \left[k_4 \delta_1 + \nu \delta_2\right] + k k_3 k_5 \pi \\ \\ \left[k_4 \delta_1 + \nu \delta_2\right] + k k_3 k_5 \pi \left[k_4 \delta_1 + \nu \delta_2\right] + e \delta_2 \pi k_2 k_3 k_5 + \varepsilon \gamma \delta_2 \pi k_2 k_3 + \xi \rho \pi k_5 \left[k_4 \delta_1 + \nu \delta_2\right] \end{array} \right\}$$

When we substitutes R_0 in above we have,

$$\Rightarrow k_1 k_2 k_3 k_4 k_5 k_6 \left[R_0 - 1\right] = \lambda A^*$$

Where, $p = \left[k_1 (\lambda + \mu)\right]$

$$A^* = \left\{ \begin{array}{l} \pi k_2 k_3 k_4 k_5 + k_3 k_4 \pi \left[k_4 \delta_1 + \nu \delta_2\right] \alpha_2 \pi k_2 k_3 k_4 + \gamma \delta_2 \pi k_2 k_3 + \rho \pi k_5 \left[k_4 \delta_1 + \nu \delta_2\right] + k k_3 k_5 \pi \\ \\ \left[k_4 \delta_1 + \nu \delta_2\right] + k k_3 k_5 \pi \left[k_4 \delta_1 + \nu \delta_2\right] + e \delta_2 \pi k_2 k_3 k_5 + \varepsilon \gamma \delta_2 \pi k_2 k_3 + \xi \rho \pi k_5 \left[k_4 \delta_1 + \nu \delta_2\right] \end{array} \right\}$$

$$\lambda = \frac{k_1 k_2 k_3 k_4 k_5 k_6 \left[R_0 - 1\right]}{A^*}$$

Lemma 1 *The model* (1) *has a unique endemic (positive) equilibrium point, given by* E_Q, *whenever* $R_0 > 1$

Theorem 4 The unique endemic equilibrium point of the model 2.1 is LAS if $R_0 > 1$ and unstable if otherwise.

RESULT AND DISCUSSION

To illustrate the analytic results obtained from system (model 1), we did some simulations using the parameters values in Table (3). A numerical simulation of the model is based on demographic data

as follows. The average lifespan $\left(\frac{1}{\mu}\right)$ is assumed to be 50 years (so that, $\mu = 0.02$) and the recruitment rate into the sexually-active population is assumed to be 3 million per year.

Thus, the total population, in the absence of disease, is $\frac{\pi}{\mu} = 150$million, Furthermore, the following initial conditions is used in the numerical simulations of the model: $S(0) = 1468000$ $E(0) = 130000$ which is assumed, $I_N(0) = 2500000, I_C(0) = 200000;$ $A_N(0) = 200000;$ $A_C(0) = 120000$ and $T(0) = 450000.$

Using the parameters values in table 3 we obtained the following using MATLAB

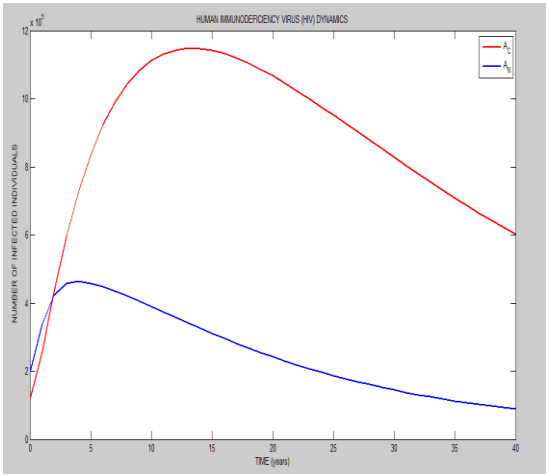

Fig. 2: Dynamics of AIDS individuals with counseling and AIDS individuals without counseling using the parameter values in table 3

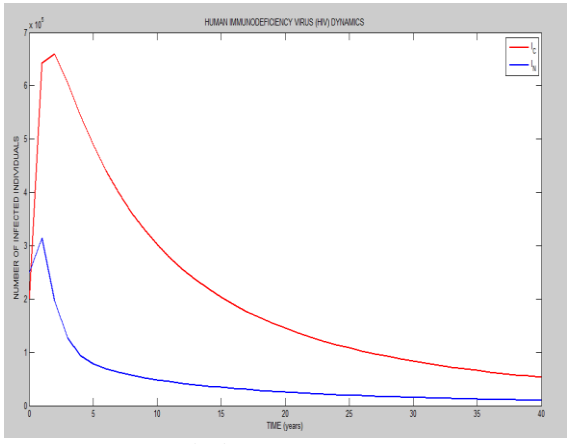

Fig. 4: Dynamics of infected individuals with counseling and infected individuals without counseling using the parameter values in table 3

Table 3: Parameters and their Values

Parameter	Value
π	$3000000\,human/\,yr$
β	$0.5yr^{-1}$
δ_1	$0.83yr^{-1}$
δ_2	$0.6yr^{-1}$
k	$0.1yr^{-1}$
ρ	$0.3yr^{-1}$
μ	$0.02yr^{-1}$
ν	$0.6yr^{-1}$
ξ	$0.10yr^{-1}$
e	$0.6yr^{-1}$
ω_1	$0.04yr^{-1}$
ω_2	$0.0835yr^{-1}$
ω_3	$0.7611yr^{-1}$
γ	$0.5yr^{-1}$
ε	$0.09yr^{-1}$
l_1	$0.02yr^{-1}$
l_2	$0.15yr^{-1}$
l_3	$0.62yr^{-1}$
a	$0.3yr^{-1}$
α	$0.830yr^{-1}$
θ_1	$0.930yr^{-1}$
θ_2	$0.02yr^{-1}$

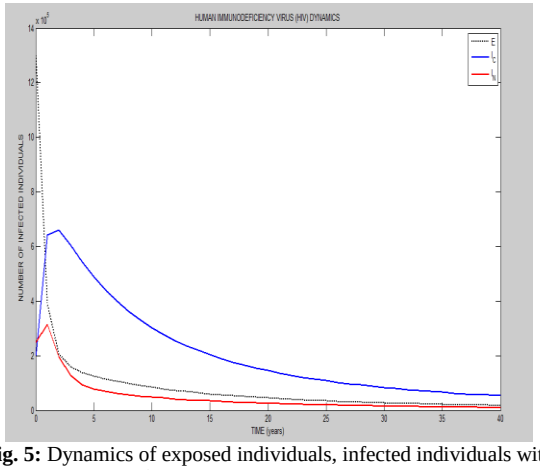

Fig. 5: Dynamics of exposed individuals, infected individuals with counseling and infected individuals without counseling using the parameter values in table 3

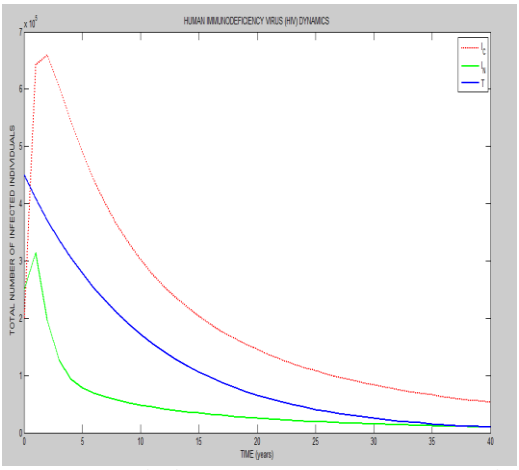

Fig. 6: Dynamics of infected individuals with counseling, infected individuals without counseling and treatment individuals using the parameter values in table 3

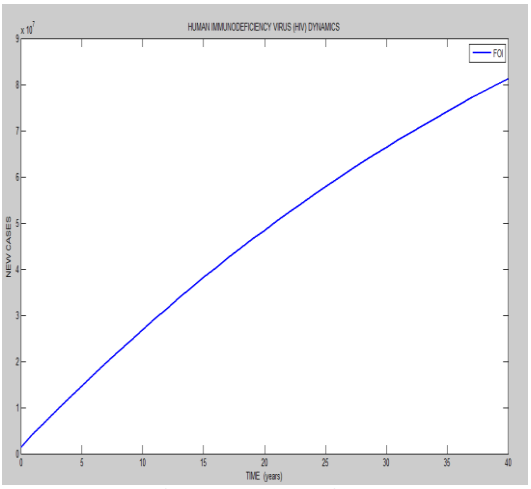

Fig. 7: simulation of new HIV cases infection when there is no treatment in each compartment, Parameter values used in table 3 with (for this treatment-only intervention, we set

$$k = v = \xi = \varepsilon = \alpha = a = 0$$ the value of

$$R_0 = 1.0323)$$

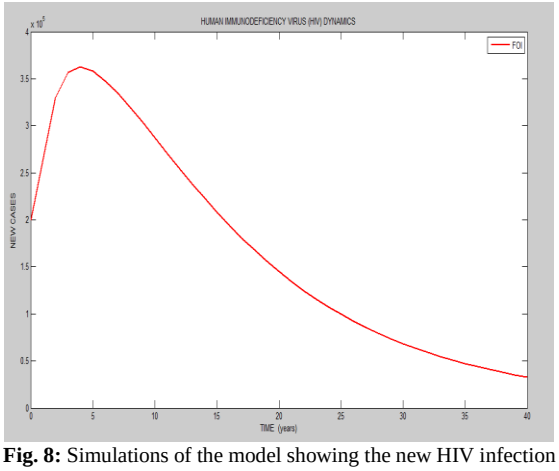

Fig. 8: Simulations of the model showing the new HIV infection cases and the time needed to reduction the virus using Parameter values given in table 3 above with $\beta = 0.64$ the value of

$$R_0 = 0.532$$

The discussion of the above simulations from Fig. 2 to 8 for the projection of 40 years It can be seen that increasing the ART treatment rate will reduce basic reproduction number R_0, which will lead us into extinction of HIV/AIDS. On the other hand, increasing of transition rate into AIDS compartment will increase basic reproduction number R_0. This means that if the individual who have failed in ART treatment do not re-follow the ART treatment will make the HIV/AIDS exist in the field from Fig. 7. The projected cases of new infection till 2059 are given in Fig. 8. We observe that the cases of new infection remain around the steady state about 500,000 cases of infection per year towards 2059. This is supported by the data that seem to fluctuate around the steady state. Our results are suggestive of the fact that interventions aimed at averting new infections will be most appropriate.

Conclusion: The results of sensitivity analysis showed that the model system is most sensitive to Infection contact rates, the testing and counseling rates as well as treatment rates. the singular use of public health counseling of infected individuals or anti-retroviral drugs can lead to the effective control or elimination of HIV in if their effectiveness levels are moderately high enough (these strategies could avert between 9 to 12 million new HIV cases over a period of 40 years).

REFERENCES

Abdallah S; Waziri I; Estomih S; Massawe I; Oluwole DM (2012). Mathematical Modelling of HIV/AIDS Dynamics with Treatment and Vertical Transmission, *Appl. Math. 2(3): 77-89*

Arazoza, HD and Lounes, RA (2002). Non-linear model for a sexually transmitted disease with contact tracing, IMA *J Math. Appl. Med. Biol.*19 (2002), 221-234

Apenteng, OO; Ismail, NA (2019). Modelling the impact of migration on HIV persistency in Ghana, department of applied statistics, faculty of economics & administration, university of Malaya, Kuala Lumpur, Malaysia

Basavapathruni, A; Anderson, KS (December 2007)."Reverse transcription of the HIV-1 pandemic". *The FASEB J.* **21** (14): 3795–3808

Basavarajaiah. DM; Narasimhamurthy B; Maheshappa KB. Leelavathy B (2012),

Mathematical model approach to HIV/AIDS transmission from mother to child, international journal of scientific & technology research volume 1, issue 9, ISSN 2277-8616

Biswas, MHA (2012). Optimal Chemotherapeutic Strategy for HIV Infections-State Constrained Case. *Proceedings of the 1st PhD Students Conference in Electrical and Computer Engineering*, University of Porto, Porto, 28-29 June 2012.

Biswas, MHA (2014). On the evolution of AIDS/HIV treatment: an optimal control approach. Current HIV Research, 12, 1-12.

Gaodert F; Bailes E; Robertson DL (1944)."*Origin of hiv-1 in the chimpanzee pan troglodytestroglodytes*". Nature 397(6718): 436–41.

Gumel, AB; Connell M and Van Den Driessche, P (2006). Mathematical analysis of a staged-progression HIV model with imperfect vaccine.

Hethcote HW and Thieme HR (1985). Stability of the Endemic Equilibrium in Endemic Models with sub-populations. Math. Biosci. 75: 205-227. https://doi.org/10.2174/1570162x120114071609 4638

Pickering, J; Wiley, JA; Padian, NS; Lieb, LE; Echenberg, DF and Walker, J (1986) Modelling the incidence of acquired immunodeficiency syndrome (AIDS) in San Francisco, Los Angeles and New York. *Math. Model.* 7, 661-688.

Karen RD and Susan CW (1999). The effectiveness of condoms in reducing heterosexual transmission of HIV, family planning perspectives. 31(6): 272-279

Mukandavire, W; Garira M; Tchuenche JM; (2009). Modeling effects of public health educational campaigns on HIV/AIDS transmission dynamics. *Appl. Math. Modelling*. 33: 2084-2095.

Srinivasa, ASR; Mathematical modeling of AIDS epidemic in India, *Curr. Sci*. 84 (2003), 1192ñ1197

World Health Organization (1993). Scaling up Antiretroviral Therapy in Resource-limited Settings: Treatment Guidelines for a Public Health Approach. www.who.int/hiv/pub/prev care/en/arvrevision2003en.pdf.

World Health Organization (2008). Progress towards Universal Access to HIV Prevention, Treatment, Care and Support. National Agency for the Control of AIDS. www.who.int/hiv/topics/universalaccess/en/inde x.html. (Accessed April 2009).