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Mathematical Analysis of Sensitive Parameters on the Dynamical Spread of HIV

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ABSTRACT: Sensitivity analysis was performed on a mathematical model of Human Immunodefiency Virus (HIV) to determine the gauge and importance of each parameter to basic reproduction number in the dynamical spread of the disease.

The threshold basic reproduction number R_0, which is the average number of secondary infection generated by infected individuals in his or her infectious period was calculated using next generation matrix method, which shows that, the disease dies out when $R_0 < 1$, and the disease will persist and spread when $R_0 > 1$.The relative sensitivity analysis was computed for all the parameters in the basic reproduction number, which shows the influence of each parameter in the dynamical spread of the disease. Numerical sensitivity reveals that effective contact rate and progressor rate are the most sensitive parameters in the basic reproduction number. This analysis will help the medical practitioners and policy health makers to know the best control intervention strategies to be adopted in order to reduce dynamical spread of Human Immunodefiency Virus (HIV) in the community.

KEYWORDS: HIV, Critical Points, Basic Reproduction Number, Local Stability, Sensitivity

I INTRODUCTION

HIV/AIDS is one of the deadliest epidemic diseases the world is still battling with. The onset of the HIV epidemic in sub-Saharan Africa was extremely rapid, with estimated prevalence rate being doubled i.e. each HIV infected person annually infects, directly or indirectly, another (susceptible) individual [26]. Globally there were 36.9 million people living with HIV in 2014 with 2 million people becoming newly infected. Also, 1.2 million people die from AIDS – related illness [22]. In 2015, almost 16 people were receiving antiretroviral therapy [25].

HIV is classified as lentivirus, which attacks and depletes CD4 + T – cells, an important constituent of immune system which fights against foreign invasion. This HIV thereafter leads to AIDS. [8,20,5]. The virus is known to have a slow acting behaviour, long latency period and never wholly eliminated from the body [16]. There are two major types of HIV which include HIV-1 and HIV-2. HIV-1 is more common and can be found worldwide while HIV-2 is pre-dominantly found in West Africa. The attack on CD4+ weakens immune system which makes it harder for body to fight infectious disease such as meningitis, pneumonia, cancers and tuberculosis e.t.c. [16]. Body fluids like blood, semen, pre-seminal fluid, rectal fluids, vaginal fluid and breast milk from a person who has HIV can transmit HIV. These fluids have to come in contact with mucous membrane or damaged tissue or be directly injected into the blood stream for transmission to occur [4, 1]. The modes of transmission of HIV include, sexual intercourse, sharing needles

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or sharp objects with HIV–infected person, blood transfusion (HIV–Contaminated blood), and mother to child transmission through birth or breast feeding [18].

When a person first infected, he or she may have some symptoms. These symptoms are different from person to person; some may be experiencing cold or flu, fever, headache, sore throat, swollen lymph nodes, fatigue, rash, sores in the mouth especially at the early stage of the disease. However, the only way to tell if your symptoms are from a cold, flu or HIV is to have an HIV tests [21]. One can prevent HIV through the use of condom for sex and by avoiding sharing of needles, sharp objects or other injecting equipments [14]. Antiretroviral (ARV) therapy is the potent treatment for HIV, capable of reducing the virus concentration in the blood to an undetectable level after few weeks of treatment and to sustain the CD4 + T – cell [13, 10]. The use of HAART which is the use of at least 3 antiretroviral drugs has decrease the mortality in HIV – infected patients [15]. When there is no antiviral therapy, the time between HIV – 1 infection and the development of AIDS varies from 5 to 15 years. This depends on the genetic factor or composition of the infected person. There would be rapid multiplication of the virus in the first 2months, after initial infection while the CD4+ T cell population decreases [17]. This makes infected persons become vulnerable to opportunistic infection.

There are many factors that could fuel the spread of HIV. It is important to know these different factors responsible for its transmission and prevalence to determine how best to reduce mortality due to HIV. Thus, in this paper, we examined the most sensitive parameters that play important role in the dynamical spread and control of the disease. We tried to calculate the sensitivity indices of the basic reproduction number that determines whether the disease will die out or become endemic.

II. MATHEMATICAL MODEL FORMULATION

A non linear mathematical model is formulated and analyzed to study the sensitivity of parameters involved in basic reproduction number on the dynamical spread of HIV.

In modeling the dynamics, the population at time t, denoted by N(t), is divided into (5) five compartments of Susceptible (S(t)) individuals, Latently HIV $(L_H(t))$ individuals, HIV Undetected $(H_U(t))$ individuals, HIV Detected $(H_D(t))$ individuals, Treated HIV $(H_W(t))$ individuals. So that,

$$N(t) = S + L_H + H_U + H_D + H_W \quad (1)$$

The Susceptible population is increased by the recruitment of individuals into the population at rate π.the population decrease by natural death rate μ . We assumed that susceptible individuals acquire HIV infection, following effective contact with people infected with HIV only (i.e., those in the $(L_H, \eta_U H_U, \eta_{dH} H_D \ and \ \eta_W H_W)$ classes at a rate λ_H , given by

$$\lambda_H = \beta_H \frac{(L_H + \eta_U H_U + \eta_{dH} H_D + \eta_W H_W)}{N} \quad (2)$$

Where, β_H is the effective contact rate for HIV transmission.

Then,

$$\frac{dS}{dt} = \pi - \lambda_H S - \mu S \quad (3)$$

A fraction ε_1 of the newly infected individuals with HIV are assumed to show no disease symptoms initially. These individuals (known as “slow progressor” for HIV) are moved to the latently HIV class (L_H). The remaining fraction, $(1-\varepsilon_1)$ of the newly infected individuals are assumed to immediately display disease symptoms (Fast progressors) and are moved to the undetected infectious class H_U. The population of latent class is decreased by the

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progression of latent HIV individual to active undetected HIV H_U (at a rate κ_H) and also reduced by natural death rate (μ) and finally increased by the fraction of Treated HIV at the rate (ϕ) that moves from treated class to latently HIV compartment. Thus,

$$\frac{dL_H}{dt} = \varepsilon_1\lambda_H S - (\kappa_H + \mu)L_H + \phi H_W \quad (4)$$

The population of undetected infected individuals is increased by the infection of fast progressors (at the rate $(1-\varepsilon_1)$ and the development of symptoms by latently individual at the rate $(1-\omega_1)\kappa_H$ where ω_1 is the endogenous reactivation rate. This population is decreased by natural death rate (μ) and disease induced death (at a rate δ_{UH}) and further decreased by detection rate (γ_{UH}) of HIV undetected infected individuals. Hence

$$\frac{dH_U}{dt} = (1-\varepsilon_1)\lambda_H S + (1-\omega_1)\kappa_H L_H - (\gamma_{UH} + \mu + \delta_{UH})H_U \quad (5)$$

The population of detected infected HIV individual increases by the fraction of latently individuals who develop disease symptoms (at the rate $\omega_1\kappa_H$), where ω_1 is the endogenous reactivation rate and the detection of undetected individual at the rate γ_{UH}. The population later decreases by treatment rate (τ_1) for HIV detected individual and finally reduces by the natural death rate, induced mortality death rate at μ and δ_{UH} respectively. Hence we have.

$$\frac{dH_D}{dt} = \omega_1\kappa_H L_H + \gamma_{UH}H_U - (\tau_1 + \mu + \delta_{UH})H_D \quad (6)$$

The population of Treated HIV individuals is increased by those that have received treatment from HIV detected infected individual at the rate (τ_1) this population reduces by fraction of treated individual that moved back to latently HIV individuals at the rate, (ϕ) since treatment does not completely clears the virus and finally reduced by natural death rate (μ).

Hence,

$$\frac{dH_W}{dt} = \tau_1 H_D - (\phi + \mu + \delta_{HW})H_W \quad (7)$$

III. HIV MODEL

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \lambda_H S - \mu S \\
\frac{dL_H}{dt} &= \varepsilon_1\lambda_H S - K_1 L_H + \phi H_W \\
\frac{dH_U}{dt} &= (1-\varepsilon_1)\lambda_H S + K_2 L_H - K_3 H_U \\
\frac{dH_D}{dt} &= \omega_1\kappa_H L_H + \gamma_{UH}H_U - K_4 H_D \\
\frac{dH_W}{dt} &= \tau_1 H_D - K_5 H_W
\end{aligned}\right\} \quad (8)$$

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$$K_1 = (\kappa_H + \mu), K_2 = (1-\omega_1)\kappa_H, K_3 = (\gamma_{UH} + \mu + \delta_{UH}), K_4 = (\tau_1 + \mu + \delta_{dH}), K_5 = (\phi + \mu + \delta_{HW}),$$

Where $\lambda_H = \beta_H \dfrac{(L_H + \eta_U H_U + \eta_{dH} H_D + \eta_W H_W)}{N}$ (9)

For this model, it can be shown that the region,

$$D = \{(S + L_H + \eta_U H_U + \eta_{dH} H_D + \eta_W H_W) \in R^5_+ : N \le \pi / \mu \quad (10)$$

For HIV model only to be epidemiologically meaningful and well posed, we need to prove that all state variables are non-negative for all $t > 0$

Lemma 1.

The closed set $D = \{(S + L_H + \eta_U H_U + \eta_{dH} H_D + \eta_W H_W) \in R^5_+ : N \le \pi / \mu \}$ is positively-invariant and attracting with respect to the model (8)

Proof: Consider the biologically-feasible region D, defined above. The rate of change of the total population, obtained by adding all equations of the model (8), is given by

$$\frac{dN}{dt} = \pi - \mu N - \delta_{UH} - \delta_{dH} - \delta_{HW} \quad (11)$$

It follows that $\dfrac{dN}{dt} < 0$ whenever $N > \dfrac{\pi}{\mu}$. Furthermore,

Since $\dfrac{dN}{dt} \le \pi - \mu N$, it is clear that $N(t) \le \dfrac{\pi}{\mu}$ if $N(0) \le \dfrac{\pi}{\mu}$.

Therefore, all solutions of the model with initial conditions in D remain in D for all $t > 0$ (i.e., the ω-limits sets of the system (8) are contained in D). Thus, D is positively-invariant and attracting. In this region, the model can be considered as been epidemiologically and mathematically well posed

III.I Disease Free Equilibrium

For critical points, we set

$$\frac{dS}{dt} = \frac{dL_H}{dt} = \frac{dH_U}{dt} = \frac{dH_D}{dt} = \frac{dH_W}{dt} = 0 \quad (12)$$

At disease free equilibrium, it is assumed that there is no infection; Hence (DFE) is given as

$$\varepsilon_0 = (S, L_H, H_U, H_D, H_W) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0\right)$$

III.II DERIVATION OF BASIC REPRODUCTION NUMBER (R_O) FOR HIV

The Next Generation Matrix ($F.V^{-1}$) Method

One of fundamental questions of mathematical epidemiology is to find the threshold conditions that determine whether an infectious disease will spread in a population when the disease is introduced into the population [14].

The basic reproduction number of the model (8) R_H is calculated by using the next generation matrix [31]. Using their approach [31], we have,

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$$F = \begin{pmatrix} \varepsilon_1 \lambda_H S \\ (1-\varepsilon_1)\lambda_H S \\ 0 \\ 0 \end{pmatrix} \quad and \quad V = \begin{pmatrix} (K_H + \mu)L_H - \phi H_W \\ (1-\omega_1)K_H L_H + (\gamma_{UH} + \mu + \delta_{UH})H_U \\ -\omega_1 K_H L_H - \gamma_{UH} H_U + (\tau_1 + \mu + \delta_{UH})H_D \\ -\tau_1 H_D + (\phi + \mu + \delta_{HW})H_W \end{pmatrix}$$

After taking partial derivative F and V, we have

$$F = \begin{pmatrix} \varepsilon_1\beta_H & \varepsilon_1\beta_H\eta_U & \varepsilon_1\beta_H\eta_{dH} & \varepsilon_1\beta_H\eta_W \\ (1-\varepsilon_1)\beta_H & (1-\varepsilon_1)\beta_H\eta_U & (1-\varepsilon_1)\beta_H\eta_{dH} & (1-\varepsilon_1)\beta_H\eta_W \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad (13)$$

$$V = \begin{pmatrix} K_1 & 0 & 0 & -\phi \\ K_2 & K_3 & 0 & 0 \\ -\omega_1 K_H & -\gamma_{UH} & K_4 & 0 \\ 0 & 0 & -\tau_1 & K_5 \end{pmatrix} \quad (14)$$

Thus,

$$R_H = \left\{ \left(\frac{1}{\phi\tau_1 K_2 \gamma_{UH} + \phi\tau_1\omega_1 K_H K_3 - K_4 K_5 K_1 K_3} \right) \begin{pmatrix} \beta_H(-(1-\varepsilon_1)\gamma_{UH}\phi\tau_1 + (1-\varepsilon_1)\eta_U\phi\tau_1\omega_1\kappa_H \\ -(1-\varepsilon_1)\eta_U K_5 K_4 K_1 - (1-\varepsilon_1)\gamma_{UH}\eta_{dH} K_5 K_1 \\ -(1-\varepsilon_1)\gamma_{UH}\eta_W\tau_1 K_1 - \varepsilon_1 K_4 K_5 K_3 - \varepsilon_1 K_4 K_5 K_2 \eta_U \\ -\eta_{dH}\omega_1\kappa_H\varepsilon_1 K_5 K_3 - \eta_{dH}\gamma_{UH}\varepsilon_1 K_5 K_2 - \tau_1\omega_1\kappa_H\varepsilon_1\eta_W K_3 \\ -\eta_W\gamma_{UH}\varepsilon_1\tau_1 K_2 \end{pmatrix} \right\}^T \quad (15)$$

he threshold quantity R_H is the basic reproduction number of the normalized model system (8) for HIV infection in a population. It measures the average number of new secondary infections generated by a single infected individual in his or her infectious period in a susceptible population [1].

III.III GLOBAL STABILITY OF DISEASE FREE EQUILIBRIUM (HIV)

Here, the global asymptotic stability (GAS) property of the DEF of the HIV model only will be explored.

Theorem3.4: The disease free-equilibrium of the system (8) is globally asymptotically stable whenever $R_H < 1$ and unstable if $R_H > 1$.

Proof: It follows that $S = N^* - L_H - H_U - H_D - H_W$ at steady state. The proof is based on using the comparison theorem [21] to prove the global stability.

Using comparison method, we have,

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$$\begin{pmatrix} \frac{dL_H}{dt} \\ \frac{dH_U}{dt} \\ \frac{dH_D}{dt} \\ \frac{dH_W}{dt} \end{pmatrix} = (F - V) \begin{pmatrix} L_H \\ H_U \\ H_D \\ H_W \end{pmatrix} - Fi \begin{pmatrix} L_H \\ H_U \\ H_D \\ H_W \end{pmatrix} \tag{16}$$

$$(\text{F - V}) = \begin{pmatrix} \varepsilon_1\beta_H - K_1 & \varepsilon_1\beta_H\eta_U & \varepsilon_1\beta_H\eta_{dH} & \varepsilon_1\beta_H\eta_W + \phi \\ (1-\varepsilon_1)\beta_H + K_2 & (1-\varepsilon_1)\beta_H\eta_U - K_3 & (1-\varepsilon_1)\beta_H\eta_{dH} & (1-\varepsilon_1)\beta_H\eta_W \\ \omega_1\kappa_H & \gamma_{UH} & -K_4 & 0 \\ 0 & 0 & \tau_1 & -K_5 \end{pmatrix}$$

According to [6, 31], all eigen values of the matrix (F - V) have negative real parts.
Hence, we have

$$\begin{bmatrix} ((\varepsilon_1\beta_H - K_1) - \lambda)((1-\varepsilon_1)\beta_H\eta_U - K_3 - \lambda)(-K_4 - \lambda)(-K_5 - \lambda) \\ -(\varepsilon_1\beta_H\eta_U)((1-\varepsilon_1)\beta_H + K_2)(-K_4 - \lambda)(-K_5 - \lambda) + (\varepsilon_1\beta_H\eta_{dH})((1-\varepsilon_1)\beta_H + K_2)(\gamma_{UH}(-K_5 - \lambda)) \\ -(\varepsilon_1\beta_H\eta_W + \phi)((1-\varepsilon_1)\beta_H + K_2)(\gamma_{UH} + \tau_1) \end{bmatrix} = 0 \tag{17}$$

Thus, from equation (17), the characteristic equation is given by

$$\lambda^4 + a_4\lambda^3 + a_3\lambda^2 + a_2\lambda - a_1$$

Where

$$a_4 = (-\eta_U\beta_H(1-\varepsilon_1) - \varepsilon_1\beta_H + K_4K_5K_1K_3)$$

$$\begin{aligned} a_3 = (&-(1-\varepsilon_1)K_5\eta_U\beta_H - K_5\varepsilon_1\beta_H + K_5K_1 + K_5K_3 + K_4K_5 - \eta_{dH}\omega_1\kappa_H\varepsilon_1\beta_H - (1-\varepsilon_1)\gamma_{UH}\eta_{dH}\beta_H \\ &-(1-\varepsilon_1)K_4\eta_U\beta_H - K_4\varepsilon_1\beta_H + K_4K_1 + K_4K_3 - (1-\varepsilon_1)K_1\eta_U\beta_H - \varepsilon_1K_2\eta_U\beta_H - \varepsilon_1K_3\beta_H + K_1K_3) \end{aligned}$$

$$\begin{aligned} a_2 = (&-\tau_1\omega_1\kappa_H\varepsilon_1\eta_W\beta_H - (1-\varepsilon_1)\gamma_{UH}\eta_W\tau_1\beta_H - \phi\tau_1\omega_1\kappa_H - \eta_{dH}\omega_1\kappa_H\varepsilon_1K_5\beta_H - (1-\varepsilon_1)\gamma_{UH}\eta_{dH}K_5\beta_H \\ &-(1-\varepsilon_1)\eta_UK_5K_4\beta_H - K_4K_5\varepsilon_1\beta_H + K_4K_5K_1 + K_4K_5K_3 - (1-\varepsilon_1)\eta_U\beta_HK_5K_1 - \varepsilon_1K_2K_5\eta_U\beta_H \\ &-\varepsilon_1K_3K_5\beta_H + -K_3K_2K_5 - \eta_{dH}\gamma_{UH}\varepsilon_1K_2\beta_H - (1-\varepsilon_1)K_1\gamma_{UH}\eta_{dH}\beta_H - \eta_{dH}\kappa_H\varepsilon_1K_3\beta_H\omega_1 \\ &-(1-\varepsilon_1)\eta_U\beta_HK_4K_1 - \eta_U\varepsilon_1K_2K_4\beta_H - \varepsilon_1K_3K_4\beta_H + K_4K_1K_3) \end{aligned}$$

$$\begin{aligned} a_1 = (&-(-\phi\tau_1\omega_1\kappa_HK_3 - \varepsilon_1K_4K_5K_3\beta_H - \phi\tau_1K_2\gamma_{UH} + K_4K_5K_1K_3 - (1-\varepsilon_1)\gamma_{UH}\eta_W\tau_1K_1\beta_H \\ &-(1-\varepsilon_1)\gamma_{UH}\eta_{dH}K_5K_1\beta_H - (1-\varepsilon_1)\eta_UK_5K_4K_1\beta_H + (1-\varepsilon_1)\eta_U\phi\tau_1\omega_1\kappa_H\beta_H - (1-\varepsilon_1)\gamma_{UH}\phi\tau_1\beta_H \\ &-\eta_{dH}\gamma_{UH}\varepsilon_1K_5K_2\beta_H - \varepsilon_1K_4K_5K_2\eta_U\beta_H - \eta_W\gamma_{UH}\varepsilon_1\tau_1K_2\beta_H - \tau_1\omega_1\kappa_H\varepsilon_1\eta_WK_3\beta_H - \eta_{dH}\omega_1\kappa_H\varepsilon_1K_5K_3)) \end{aligned}$$

Applying Routh-Hurwitz criteria of order 4;

$n = 4 : a_1 > 0$, $a_3 > 0$, $a_4 > 0$, *and* $a_1a_2a_3 > a_3^2 + a_1^2a_4$.

Then for $a_1 > 0$

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$$\frac{\left(\begin{array}{l}\beta_H(-(1-\varepsilon_1)\gamma_{UH}\phi\tau_1+(1-\varepsilon_1)\eta_U\phi\tau_1\omega_1\kappa_H-(1-\varepsilon_1)\eta_U K_5K_4K_1\\-(1-\varepsilon_1)\gamma_{UH}\eta_{dH}K_5K_1-(1-\varepsilon_1)\gamma_{UH}\eta_W\tau_1K_1-\varepsilon_1K_4K_5K_3-\varepsilon_1K_4K_5K_2\eta_U-\\\eta_{dH}\omega_1\kappa_H\varepsilon_1K_5K_3-\eta_{dH}\gamma_{UH}\varepsilon_1K_5K_2-\tau_1\omega_1\kappa_H\varepsilon_1\eta_WK_3-\eta_W\gamma_{UH}\varepsilon_1\tau_1K_2\end{array}\right)}{(\phi\tau_1K_2\gamma_{UH}+\phi\tau_1\omega_1K_HK_3-K_4K_5K_1K_3)}<1,$$

Hence, we have established that the disease free equilibrium is globally asymptotically stable whenever $R_H<1$ and unstable when $R_H>1.$

III.IV EXISTENCE OF ENDEMIC EQUILIBRIUM (EE)

Where $\varepsilon_0^* = (S^{**}, L_H^{**}, H_U^{**}, H_D^{**}, H_W^{**})$ are the endemic equilibrium points.

Equation (8) becomes

$$\frac{dS}{dt} = \pi - \lambda_H S - \mu S \tag{18.1}$$

$$\frac{dL_H}{dt} = \varepsilon_1\lambda_H S - K_1L_H + \phi H_W \tag{18.2}$$

$$\frac{dH_U}{dt} = (1-\varepsilon_1)\lambda_H S + K_2\kappa_H L_H - K_3H_U \tag{18.3}$$

$$\frac{dH_D}{dt} = \omega_1\kappa_H L_H + \gamma_{UH}H_U - K_4H_D \tag{18.4}$$

$$\frac{dH_W}{dt} = \tau_1 H_D - K_5H_W \tag{18.5}$$

Solving equation 18.1- 18.5 at steady state and re-writing in terms of $\lambda_H^{**}S^{**}$, we have

$$L_{H^{**}} = \rho_1\lambda_H^{**}S^{**} \tag{18.6}$$

$$H_{U^{**}} = \frac{(1-\varepsilon_1)\lambda_H^{**}S^{**}}{K_3} + \frac{K_{12}K_HP_1\lambda_H^{**}S^{**}}{K_3} \ldots\ldots\ldots\ldots P_2\lambda_H^{**}S^{**} \tag{18.7}$$

$$H_{D^{**}} = \frac{\omega_1K_HP_1\lambda_H^{**}S^{**}}{K_4} + \frac{\gamma_{UH}P_2\lambda_H^{**}S^{**}}{K_4} \ldots\ldots\ldots\ldots P_3\lambda_H^{**}S^{**} \tag{18.8}$$

$$H_W = \frac{\tau_1P_3\lambda_H^{**}S^{**}}{K_5} \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots P_4\lambda_H^{**}S^{**} \tag{18.9}$$

Where

$$P_1 = \frac{\varepsilon_1}{X} + \frac{\phi\tau_1\gamma_{UH}(1-\varepsilon_1)}{Y}$$

$$P_2 = \frac{(1-\varepsilon_1)}{K_3} + \frac{K_2\kappa_H}{K_3}\left[\frac{\varepsilon_1}{X} + \frac{\phi\tau_1\gamma_{UH}(1-\varepsilon_1)}{Y}\right]$$

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$$P_3 = \frac{\omega_1\kappa_H}{K_4}\left[\frac{\varepsilon_1}{X} + \frac{\phi\tau_1\gamma_{UH}(1-\varepsilon_1)}{Y}\right] + \frac{\gamma_{UH}}{K_4}\left[\frac{(1-\varepsilon_1)}{K_2} + \frac{K_2\kappa_H}{K_3}\left[\frac{\varepsilon_1}{X} + \frac{\phi\tau_1\gamma_{UH}(1-\varepsilon_1)}{Y}\right]\right]$$

$$P_4 = \frac{\tau_1}{K_5}\cdot\frac{\omega_1\kappa_H}{K_4}\left[\frac{\varepsilon_1}{X} + \frac{\phi\tau_1\gamma_{UH}(1-\varepsilon_1)}{Y}\right] + \frac{\gamma_{UH}}{K_4}\left[\frac{(1-\varepsilon_1)}{K_2} + \frac{K_{12}\kappa_H}{K_3}\left[\frac{\varepsilon_1}{X} + \frac{\phi\tau_1\gamma_{UH}(1-\varepsilon_1)}{Y}\right]\right]$$

Where

$$X = K_1\left(1 - \frac{\phi\tau_1 w_1\kappa_H}{K_1K_4K_5} - \frac{\phi\tau_1\gamma_{UH}K_{12}\kappa_H}{K_1K_4K_5K_3}\right)$$

$$Y = K_1K_4K_5K_3\left(1 - \frac{\phi\tau_1\omega_1K_H}{K_1K_4K_5} - \frac{\phi\tau_1\gamma_{UH}K_{12}K_H}{K_1K_4K_5K_3}\right)$$

$$\lambda_H^{**} = \frac{\beta_H\left[L_H^{**} + \eta_U H_U^{**} + \eta_{dH}H_D^{**} + \eta_W H_W^{**}\right]}{N} \qquad (18.10)$$

Substituting the expressions in $(33.14 - 33.17)$ into (33.18) we have

$$\lambda_H^{**}\left[S^{**} + P_1\lambda_H^{**}S^{**} + P_2\lambda_H^{**}S^{**} + P_3\lambda_H^{**}S^{**} + P_4\lambda_H^{**}S^{**}\right] = \beta\lambda_H^{**}S^{**}\left[P_1 + \eta_U P_2 + \eta_{dH}P_3 + \eta_W P_4\right] \qquad (18.11)$$

Divide each term in (18.11) by $\lambda_H{}^{**}S^{**}$

$$1 + P_5\lambda^{**} = \beta\left[P_1 + \eta_U P_2 + \eta_{dH}P_3 + \eta_W P_4\right]$$

Where $P_5 = P_1 + P_2 + P_3 + P_4 \geq 0$

So that

$$1 + P_5\lambda_H^{**} = \frac{\beta}{K_1K_3K_4K_5}\left[\frac{\varepsilon_1(K_3K_4K_5)}{K_1A} + \frac{\phi\tau_1\gamma_{UH}(1-\varepsilon_1)}{(\ A\ )} + (1-\varepsilon_1)K_1K_4K_5\right.$$

$$+ \frac{K_2K_H\varepsilon_1K_4K_5}{A} + \frac{\phi\tau_1\gamma_{UH}(1-\varepsilon_1)}{A} + \frac{\omega_1K_H\varepsilon_1K_3K_5}{A} + \frac{\phi\tau_1\gamma_{UH}(1-\varepsilon_1)}{A} + \gamma_{UH}(1-\varepsilon_1)K_1K_5$$

$$+ \frac{\gamma_{UH}K_2\varepsilon_1K_HK_5}{A} + \frac{\gamma_{UH}K_2K_H\phi\tau_1\gamma_{UH}(1-\varepsilon_1)}{K_{14}K_{13}A} + \frac{\tau_1\omega_1K_H\varepsilon_1K_3}{A} + \frac{\tau_1\omega_1K_H\phi\tau_1\gamma_{UH}(1-\varepsilon_1)}{K_{15}K_1 + A} + \gamma_{UH}(1-\varepsilon_1)K_1K_5$$

$$\left. + \frac{\gamma_{UH}K_2K_H\varepsilon_1K_5}{A} + \frac{\gamma_{UH}K_2K_H\phi\tau_1\gamma_{UH}(1-\varepsilon_1)}{K_4K_3A}\right]$$

$$= R_H + Q \quad where$$

$$Q = \frac{\beta}{K_1K_3K_4K_5}\left[\frac{\varepsilon_1K_3K_5}{A}(K_4 + \omega_1K_H) + \frac{\varepsilon_1K_2K_5K_H}{A}(K_4 + 2\gamma_{UH}) + \frac{\varepsilon_1\tau_1\omega_1K_HK_3}{A}\right.$$

$$\left. + 3\frac{\phi\tau_1\gamma_{UH}(1-\varepsilon)}{A} + \frac{(1-\varepsilon_1)2\gamma_{UH}K_H}{AK_4K_3}(K_2\phi\tau_1 + K_2\phi\tau_1) + (1-\varepsilon_1)\gamma_{UH}\left(\frac{2\tau_1\omega_1K_H\phi}{K_5K_4A} + 2K_1K_5 + K_1K_4K_5\right)\right]$$

Where $A = \left(1 - \frac{\phi\tau_1w_1K_H}{K_1K_4K_5} - \frac{\phi\tau_1\gamma_{uH}K_{12}K_H}{K_1K_4K_5K_3}\right)$

Therefore, $1 + P_5\lambda^{**} = R_H + Q$

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$$\lambda^{**} = \frac{R_H + Q - 1}{P_5} > 0$$

Whenever $R_H > 1$ (19)

III.V HIV SENSITIVITY ANALYSIS

This analysis will help us to know which of the parameters causes most reduction in R_o and parameters that have high impact on R_o and these should be targeted by intervention strategies in order to have most effective control of the disease. This analysis tells us how crucial and important each parameter is to disease transmission. We compute the normalized forward sensitivity index of the reproduction number with respect to its parameters.

Definition: If a variable 'b' depends differentiably on a parameter 'u', then, the normalized forward sensitivity index of 'b' with respect to 'u' is denoted by X_b, which is defined as $X_b = \frac{b}{u}\frac{\partial u}{\partial b}$

As we have explicit formula for R_o, we derive an analytical expression for the sensitive of R_o as

$$X_b^{R_o} = \frac{dR_o}{du} \times \frac{u}{R_o} \quad (20)$$

Sensitivity analysis for each parameter involved in R_H is therefore calculated.

The results obtained were tabulated below as follows:

Table III.I Values of Numerical Sensitivity of HIV

PARAMETERS	SENSITIVITY VALUE
τ_1	-0.141060
μ	-0.096178
δ_{dH}	-0.074683
δ_{UH}	-0.789652
ϕ	-0.223921
γ_{UH}	-0.083820
η_U	0.000401
η_W	-0.000034
η_{dH}	-0.000020
ω_1	0.090673
ε_1	0.763369
β_H	1.000000
κ_H	0.699705

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Table III.II: Variable and Description

VARIABLES	DESCRIPTIONS
S	Susceptible individuals
L_H	Latent HIV individual
H_D	Detected HIV individual
H_U	UndetectedHIV individual
H_W	Treated HIV individual

Table III.III: Parameters and Descriptions

PARAMETERS	DESCRIPTIONS
π	Recruitment rate
μ	Natural death rate
τ_1	Treatment rate
ε_1	Fast progressor
κ_H	Progression rate
γ_{UH}	Detection rate
ϕ	Loss of immunity
β_H	Effective contact rate
δ_{UH}, δ_{dH}	Disease induced rate
$\eta_U, \eta_{dH}, \eta_W$	Modification parameters

IV. NUMERICAL SIMULATIONS OF THE MODEL

In order to verify the dynamical behavior of the model, the numerical simulations is analyzed by using Runge –Kutta method using the following set of parameter values:

$\pi = 0.2,\ \mu = 0.02,\ \tau_1 = 0.2,\ \delta_{dH} = 0.1,\ \delta_{UH} = 0.3,\ \phi = 0.7,\ \gamma_{UH} = 0.2,\ \eta_U = 0.001$

$\eta_W = 0.001,\ \eta_{dH} = 0.001,\ \varepsilon_1 = 0.7,\ \beta_H = 0.1$

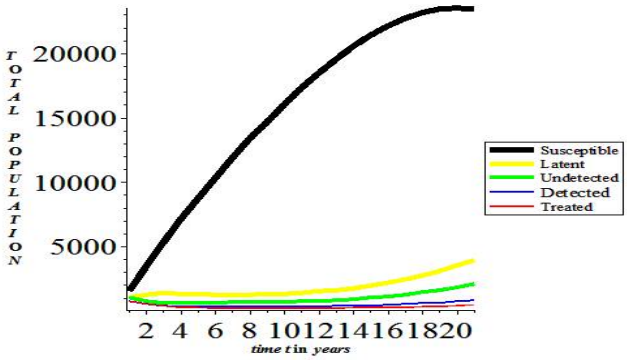

Fig. 1

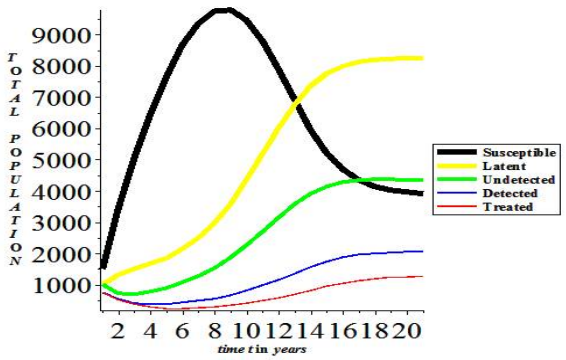

Fig.2

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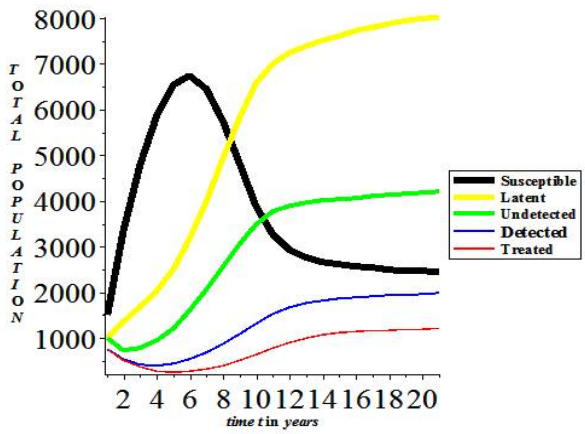

Fig.3

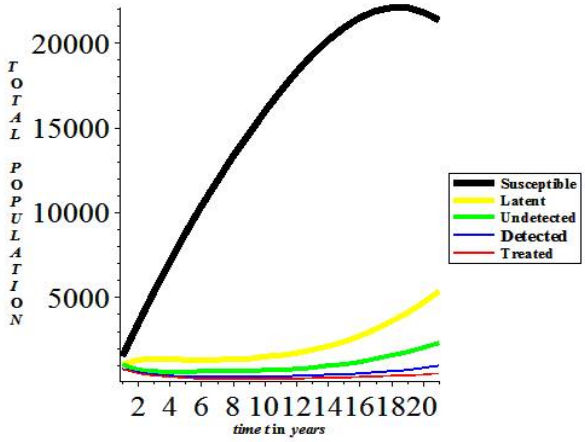

Fig.4

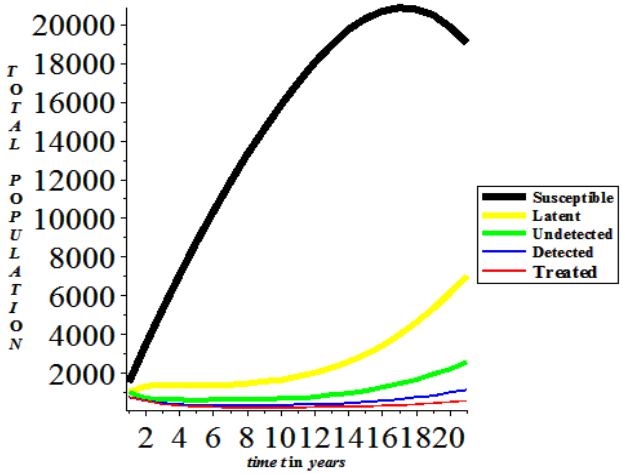

Fig.5

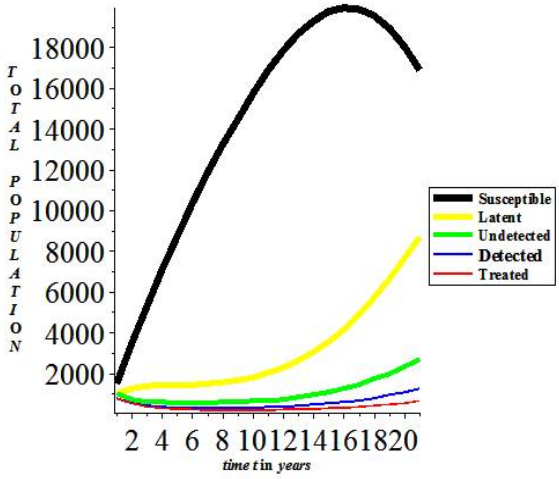

Fig.6

V. DISCUSSION AND CONCLUSIONS

It is observed that effective contact rate in **Fig. 1, 2** and **3,** reduces the susceptible individuals and increases latently infected individuals, infected undetected individuals and infected detected individuals. It reduces susceptible individuals from **23000** to **2500** due to an increase in effective contact rate from **0.1** to **0.5.** In **Fig. 4, 5** and **6,** the effect of fast progressor is shown. It can be seen that as progressor increases, the susceptible individuals decreases while the latently infected, infected undetected and infected detected individuals increases. It is noted that the fast progressor can be controlled when the immunity of an individual is very strong.

In conclusion, we presented and analyzed five (5) non-linear differential compartmental models, to have better understanding on the parameters that influence the dynamical spread of HIV in the society. Numerical simulations of the model were analyzed to determine the effects of parameters on the dynamical spread of the disease. The effective contact rate and the fast progressor are the major key parameters that enhanced the dynamical spread of HIV in the society. These need to be targeted by medical practitioners and policy health makers in order to reduce the dynamical spread of the disease in the environment.

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