ISSN No:-2456-2165

A Mathematical Model for HIV- COVID-19 Co-Infection Dynamics in Kenya

Kimulu Ancent Makau[1]
[1]Department of Mathematics and Actuarial Science, Kenyatta University, P.O. Box 43844 -00100, Nairobi, Kenya

Oke Abayomi Samuel[2]
[2]Department of Mathematical Sciences, Adekunle Ajasin University, PMB 001, Akungba Akoko, Ondo State, Nigeria

Muli Charles Ndambuki[3]
[3]Department of Mathematics and Statistics, Machakos University, *P.O. Box 136 - 90100,* Machakos, Kenya

Abstract:- The COVID-19 virus has been one of the deadly virus since it was first reported in December 2019 in Wahun, China. However vaccines have been developed for COVID-19 which were approved to be safe and reduced the infections, none such vaccine has been discovered for HIV. These two diseases are viral and this model is designed to study the co-infection dynamics in Kenya. Several models have been done on the HIV-COVID -19 co-infections in the world and our model focused on dynamics of Kenyan co-infected population. This study focused on the effect of COVID – 19 infection on HIV infected population. The equilibrium points were determined and their stability was determined using the Jacobian method. The reproduction number was obtained using the Next Generation Method. The DFE was asymptotically stable when $R_o < 1$ and unstable when $R_o > 1$. The model was solved numerically using MATLAB ode45 solver and the results shows that increase in the COVID-19 infection rate leads to increase in the co-infected population, the treated and the viral suppressed populations in the system. This implies that the co-infected population responds better to treatment to reduce their viral load in their bodies and hence reducing thedanger of death due to the co-infection.

Keywords:- *HIV, COVID-19, Co – Infection, Dynamics, Coronavirus, Next Generation Matrix.*

I. INTRODUCTION

Coronavirus started in December 2019 in the Wuhan city in Central China according to [1] and spread rapidly throughout the world becoming a global pandemic. The World Health Organization declared COVID-19 a global pandemic on Mid - March 2020. COVID -19 is caused by a virus called severe acute respiratory syndrome (SARS – CoV- 2). The COVID -19 symptoms varies but the common symptoms include fever, dry cough, fatigue, loss of taste, loss of smell and difficulties in breathing [2], [3], [4]and [5]. Older people and people living with medical conditions like asthma, cancer and HIV may experience more severe symptoms. According to [6], COVID - 19 infected individuals are infectious within the first 20 days and symptoms may appear between one to fourteen days after exposure to the virus. COVID – 19 is airborne and transmitted through contact with infected body fluids through the eyes, nose and mouth [7] and through contaminated surfaces. COVID -19 virus mutates easily causing different strains of different levels of infectivity and virulence. This has made COVID – 19 one of the deadliest virus in history. Consequently as a result of mutation there has not been a particular sure of COVID – 19 but since December 2020 different vaccines have been approved and distributed throughout the country which led to mass vaccination as a way of controlling the spread of the disease. Globally, by mid - May 2023, $13,352,935,288$ vaccine doses had been administered and 766,440,796 confirmed COVID – 19 cases with 6,932,591 confirmed deaths according to WHO. Kenya reported its first COVID-19 case on 13 March 2020 and by 23 February 2021, there were 104,500 cases, 1837 deaths and 85,665 recoveries reported in the country [8]. Also by Mid – May 2023 there has been 343,074 confirmed cases and 5,688 confirmed death cases. As at April 2023, 23,750,431 vaccine doses had been administered in Kenya. Other effective prevention methods approved were social distancing, use of quarantine method and use of face masks.

AIDS is caused by Human Immunodeficiency Virus, HIV. The virus has become one of the world's most serious health problem since the first case was reported in 1981. Since the start of the epidemic 84 million people have been infected with HIV [9] and currently 38.4 million people are living with HIV, 1.5 million new infections and 650 000 deaths in 2021 [10]. HIV is transmitted through body fluids and it attacks and weakens the body immune system by destroying the CD4+ cells [11]. Two thirds of people leaving with HIV globally are living in Sub - Saharan Africa where Kenya is part and is the hardest hit by the epidemic in the world. In 2021, Kenya had 1.4 million people leaving with HIV, 35000 new HIV infections and 22000 AIDS related deaths. The COVID – 19 epidemic led to detrimental effects on HIV and AIDS response in low and middle level countries by disrupting the access to antiretroviral medicines, preventive services and testing [9].

[12] shows that people living with HIV may be at greater risk of dying from COVID-19. Since the beginning of COVID – 19 pandemic concerns have been raised on the effect of COVID – 19 on people living with HIV due to their weakened immune systems and they are at higher risk of dying if infected with COVID – 19 more so when not receiving treatment for HIV. Two thirds of world HIV infections are found in the Sub-Saharan Africa where COVID -19 cases were raising rapidly. The COVID -19 pandemic disrupted HIV prevention and treatment strategies in many countries and UNAIDS predicted approximately 293,000 new HIV infections and 148,000 more AIDS related deaths.

A study of approximately 17 million UK adults, which included 27,480 with HIV indicated that HIV individuals were around 2.6 times more likely to die from COVID-19 than HIV negative people [13]. In a study contacted in South Africa more than three million people living in the Western Cape province were examined with 16% of them living with HIV [14]. It was found that people living with HIV were approximately twice at risk of dying of COVID – 19 compared to HIV negative individuals. This can be attributed to the reduction of CD4+ T cells counts in COVID-19 patients leading to poor clinical response. This is because CD4+ T cells are important to responding to acute and chronic viral infections by coordinating the immune system.

[15] Indicated that COVID – 19 pandemic has had negative impact on HIV care service delivery in Africa worsening health outcomes on people living with HIV. This was due to the COVID – 19 control measures imposed by the governments in Africa interrupting HIV care services which included steady supply of antiretroviral drugs (ARV), HIV counselling and testing services, TB diagnosis, condom distribution. In South Africa, it was found that retention and prescription of pre-exposure prophylaxis for pregnant women, a key prevention strategy for infant HIV transmission reduced significantly during the national COVID-19 lockdown [16]. Travel restrictions also affected the supply of ARV drugs, which significantly increased the cost of ARV drugs which were relied upon to manage HIV in Sub-Saharan Africa [17]. [18]A mathematical modelling study projected approximately up to 500,000 HIV-related deaths within a year, twice as many mother-to-child HIV transmissions and increased HIV incidence if HIV care services were interrupted for about 6 months due to the COVID-19 pandemic.

In Kibera informal settlement in Kenya, HIV patients experienced challenges accessing health facilities and ART, especially during the month of July 2020. This was occasioned by issues such as fear of contracting COVID-19, closure of health facilities, government lockdown measures and lack of personal protective equipment. Also HIV testing and ART services also reduced since the start of the COVID-19 pandemic [19].

[20] found that COVID-19 prevention could reduce the burden of co-infections with HIV and also HIV prevention control could greatly reduce the burden of co-infections with COVID-19. Effective treatment of COVID-19 could improve the immune system of those infected with the disease, therefore reducing co-infections with opportunistic infections such as HIV/AIDS. From the literature there are no clear information on the effect of COVID-19 on HIV infected individuals therefore this research will study the effects of COVID-19 on the dynamics of HIV of the infected individuals.

II. METHODOLOGY

- *The Model Flow chart is as Shown in Figure 1 below. The Model was Formulated based on the following Assumptions;*

- Every individual is susceptible to both HIV and Covid-19.
- A COVID-19 recovered individual cannot develop immunity and cannot contract COVID-19 again.
- Populations S, I_H and T_H and V_H have the equal rate (α) of contracting COVID-19.
- Populations S, I_c, T_{HC}, V_{HC} and R_c have the equal rate (β) of contracting HIV.
- No individual can have AIDS without first having HIV.
- Recovering from Covid-19 becomes susceptible to HIV only.
- V_H and V_{HC} populations cannot die of HIV and HIV-COVID-19 because the virus load has been suppressed.

- *State Variables*

Table 1 State Variables

S	**Susceptible population**
I_H	Population infected with HIV
I_c	Population infected with COVID-19
I_{HC}	Population co-infected with both HIV and COVID-19
T_H	Population treated for AIDS on
T_{HC}	Population treated for both AIDS and COVID-19
T_C	Population treated for COVID-19
V_H	Population with HIV virus suppression
V_{HC}	Population with both HIV and COVID-19 virus suppression
R_c	Population that has recovered from COVID-19

Total population, $N = S + I_H + I_c + I_{HC} + T_H + T_{HC} + T_C + V_H + V_{HC} + R_c$

➢ *Definition of Parameters*

Table 2 Definition of Parameters

$\boldsymbol{\pi}$	**Recruitment rate into the susceptible population**
β	Rate of contracting HIV by any individual
α	Rate of contracting COVID-19 by any individual
η_H	Rate of treatment of HIV infected population
η_{HC}	Rate of treatment of HIV and COVID-19 co- infected population
η_C	Rate of treatment of COVID-19 infected population
ω_H	Rate of HIV virus suppression
ω_{HC}	Rate of HIV and COVID-19 virus suppression in the co-infected population
ϵ	Rate at which COVID-19infected population recovers
δ_c	Rate of COVID-19 induced death
δ_H	Rate of HIV and AIDS-induced death
δ_{HC}	Rate of HIV and AIDS- COVID-19 induced death
μ	Rate of natural death

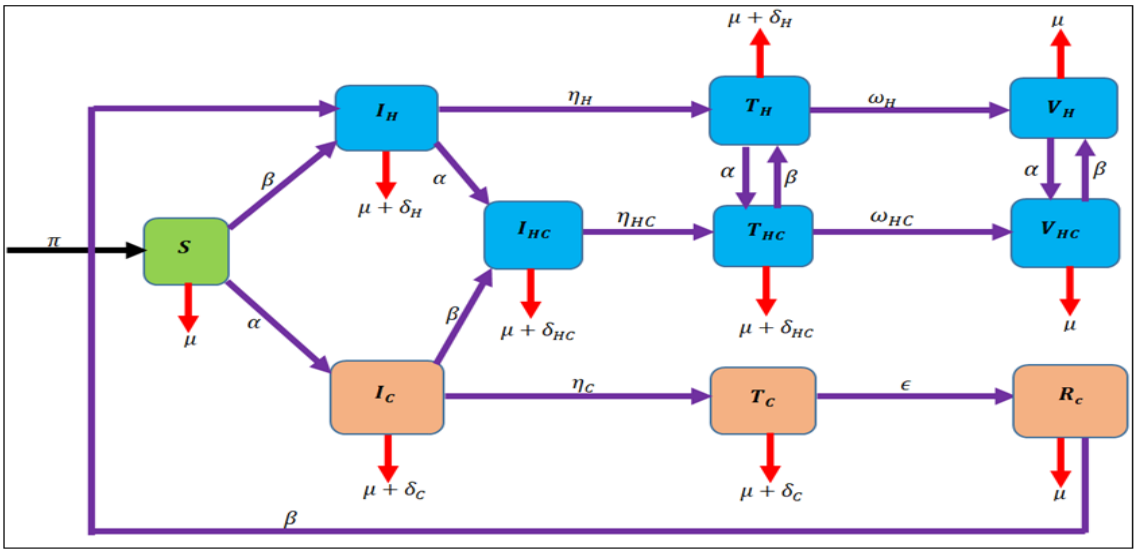

Fig 1 Model Flowchart

The governing equations for the above model used to study the HIV and COVID – 19 co-infection dynamics are given as;

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \beta S I_H - \alpha S I_C - \mu S \\
\frac{dI_H}{dt} &= \beta S I_H + \beta R_C I_H - \eta_H I_H - \alpha I_H I_{HC} - (\mu + \delta_H) I_H \\
\frac{dI_{HC}}{dt} &= \alpha I_H I_{HC} + \beta I_C I_{HC} - \eta_{HC} I_{HC} - (\mu + \delta_{HC}) I_{HC} \\
\frac{dI_C}{dt} &= \alpha S I_C - \beta I_C I_{HC} - \eta_C I_C - (\mu + \delta_C) I_C \\
\frac{dT_H}{dt} &= \eta_H I_H + \beta T_{HC} - (\alpha + \mu + \omega_H + \delta_H) T_H \\
\frac{dT_{HC}}{dt} &= \eta_{HC} I_{HC} + \alpha T_H - (\mu + \beta + \delta_{HC} + \omega_{HC}) T_{HC} \\
\frac{dT_C}{dt} &= \eta_C I_C - (\mu + \epsilon + \delta_C) T_C \\
\frac{dV_H}{dt} &= \omega_H T_H + \beta V_{HC} - (\mu + \alpha) V_H \\
\frac{dV_{HC}}{dt} &= \omega_{HC} T_{HC} + \alpha V_H - (\mu + \beta) V_{HC} \\
\frac{dR_C}{dt} &= \epsilon I_C - (\mu + \beta I_H) R_C
\end{aligned}\right\} \quad (1)$$

All parameters lies between 0 and 1, i.e, $0 < \pi, \beta, \alpha, \eta_H, \ \eta_{HC}, \ \eta_C, \omega_H, \omega_{HC}, \epsilon, \mu, \delta_H, \delta_{HC}, \delta_C < 1$

III. QUALITATIVE ANALYSIS OF THE MODEL

According to Kimulu *et al*., (2022) [21] the total population N satisfies the equation;

$N = S + I_H + I_{HC} + I_C + T_H + T_{HC} + T_C + V_H + V_{HC} + R_C$, whose time derivative is given by,

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI_H}{dt} + \frac{dI_{HC}}{dt} + \frac{dI_C}{dt} + \frac{dT_H}{dt} + \frac{dT_{HC}}{dt} + \frac{dT_C}{dt} + \frac{dV_H}{dt} + \frac{dV_{HC}}{dt} + \frac{dR_C}{dt}$$

$$\Longrightarrow \frac{dN}{dt} \leq \pi - \mu N - (\delta_H I_H + \delta_{HC} I_{HC} + \delta_C I_C + \delta_H T_H + \delta_{HC} T_{HC} + \delta_C I_C) \leq \pi - \mu N \quad (2)$$

Using the method of separation of variables to integrate and comparison theorem then as $t \longrightarrow \infty, \ N(t) \longrightarrow \frac{\pi}{\mu}$ and therefore the solution of system (1) enters and remains in the feasible region;

$\Phi = \left\{(S, I_{HC}, I_C, T_H, T_{HC}, T_C, V_H, V_{HC}, R_C): S, I_{HC}, I_C, T_H, T_{HC}, T_C, V_H, V_{HC}, R_C \in R_+^{10} \mid N \leq \frac{\pi}{\mu}\right\}$ is bounded and positively invariant.

➢ *Disease Free and Endemic Equilibrium Points*

Disease free equilibrium is the state at which there are no HIV and COVID – 19 infections in the population. This also implies that there are no HIV and COVID – 19 co-infections in the population as well. It is obtained by setting;

$I_H = I_{HC} = I_C = T_H = T_{HC} = T_C = V_H = V_{HC} = R_C = 0$ and from system (1) we obtain $S = \frac{\pi}{\mu}$.

The $DFE = \left(S^0, {I_H}^0, \ {I_{HC}}^0, \ {I_C}^0, {T_H}^0, {T_{HC}}^0, {T_C}^0, \ {V_H}^0, {V_{HC}}^0, {R_C}^0\right) = \left(\frac{\pi}{\mu}, 0,0,0,0,0,0,0,0\right)$.

The endemic equilibrium point, EEP is the state at which HIV and COVID – 19 viruses and the HIV and COVID – 19 co-infections remains and persists within the population. It is obtained by solving the system (3) below;

$$\left.\begin{array}{c} \pi - \beta S^* {I_H}^* - \alpha S^* {I_C}^* - \mu S^* = 0 \\ \beta S^* {I_H}^* + \beta {R_C}^* {I_H}^* - \eta_H {I_H}^* - \alpha {I_H}^* {I_{HC}}^* - (\mu + \delta_H) {I_H}^* = 0 \\ \alpha {I_H}^* {I_{HC}}^* + \beta {I_C}^* {I_{HC}}^* - \eta_{HC} {I_{HC}}^* - (\mu + \delta_{HC}) {I_{HC}}^* = 0 \\ \alpha S^* {I_C}^* - \beta {I_C}^* {I_{HC}}^* - \eta_C {I_C}^* - (\mu + \delta_C) {I_C}^* = 0 \\ \eta_H {I_H}^* + \beta {T_{HC}}^* - (\alpha + \mu + \omega_H + \delta_H) {T_H}^* = 0 \\ \eta_{HC} {I_{HC}}^* + \alpha {T_H}^* - (\mu + \beta + \delta_{HC} + \omega_{HC}) {T_{HC}}^* = 0 \\ \eta_C {I_C}^* - (\mu + \epsilon + \delta_C) {T_C}^* = 0 \\ \omega_H {T_H}^* + \beta {V_{HC}}^* - (\mu + \alpha) {V_H}^* = 0 \\ \omega_{HC} {T_{HC}}^* + \alpha {V_H}^* - (\mu + \beta) {V_{HC}}^* = 0 \\ \epsilon {I_C}^* - (\mu + \beta) {R_C}^* = 0 \end{array}\right\} \quad (3)$$

The EEP $(S^*, {I_H}^*, \ {I_{HC}}^*, {I_C}^*, {T_H}^*, {T_{HC}}^*, {T_C}^*, \ {V_H}^*, {V_{HC}}^*, {R_C}^*)$ is implicitly obtained in terms of ${I_C}^*$ and ${V_{HC}}^*$ from the system (2) as;

$$S^* = \frac{\alpha\pi}{\alpha\mu + \beta(\mu + \eta_{HC} + \delta_{HC}) - (\beta^2 - \alpha^2){I_C}^*}, {I_H}^* = \frac{1}{\alpha}(\mu + \eta_{HC} + \delta_{HC} - \beta {I_C}^*),$$

$${I_{HC}}^* = \frac{1}{\beta}\left(\frac{\alpha^2\pi}{\alpha\mu + \beta(\mu + \eta_{HC} + \delta_{HC}) - (\beta^2 - \alpha^2){I_C}^*} - (\mu + \eta_C + \delta_C)\right), {I_C}^*, {T_H}^* = \frac{\eta_H {I_H}^* + \beta {T_{HC}}^*}{\alpha + \mu + \omega_H + \delta_H},$$

$${T_{HC}}^* = \frac{\eta_{HC}(\alpha + \mu + \omega_H + \delta_H) + \alpha(\eta_H {I_H}^* + \beta {T_H}^*)}{(\mu + \beta + \delta_{HC} + \omega_{HC})(\alpha + \mu + \omega_H + \delta_H)}, {T_C}^* = \frac{\eta_C {I_C}^*}{\mu + \epsilon + \delta_C},$$

$${V_H}^* = \frac{\omega_H {T_H}^*(\mu + \beta) + \beta\omega_{HC}{T_{HC}}^*}{\mu^2 + \mu(\mu + \beta)}, {V_{HC}}^*, {R_C}^* = \frac{\epsilon {I_C}^*}{\mu + \beta} \quad (4)$$

➢ *Reproduction Number*

The Reproduction number (R_o) is defined as the number of infections an infectious individual can cause when introduced in a susceptible population in the entire infectious period according to Kimulu *et al*., (2022) [22]. It is determined using the next generation matrix method where $\mathcal{F}_i$ and V_i are defined as matrices that represent the rate at which new infections occur and the rate of transfer of individuals out of infected compartments respectively. The R_o was obtained from the spectral radius of the $\mathcal{F}_i V_i^{-1}$ evaluated at the DFE. From system (1) then;

$$\mathcal{F} = \begin{pmatrix} \beta S I_H + \beta R_C I_H \\ \alpha I_H I_{HC} + \beta I_C I_{HC} \\ \alpha S I_C - \beta I_C I_{HC} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} (\mu + \alpha + \eta_H + \delta_H) I_H \\ (\mu + \eta_{HC} + \delta_{HC}) I_{HC} \\ (\mu + \eta_C + \delta_C) I_C \\ -\eta_H I_H - \beta T_{HC} + (\mu + \alpha + \omega_H + \delta_H) T_H \\ -\eta_{HC} I_{HC} - \alpha T_H + (\mu + \beta + \delta_{HC} + \omega_{HC}) T_{HC} \\ -\eta_C I_C + (\mu + \epsilon + \delta_C) T_C \\ -\omega_H T_H - \beta V_{HC} + (\mu + \alpha) V_H \\ -\omega_{HC} T_{HC} - \alpha V_H + (\mu + \beta) V_{HC} \end{pmatrix}$$

Let $\mathcal{F}_{DFE}$ and V_{DFE} be the Jacobian matrices of $\mathcal{F}$ and V at DFE respectively so that;

$$\mathcal{F}_{DFE} = \begin{pmatrix} \frac{\beta\pi}{\mu} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\alpha\pi}{\mu} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V_{DFE} = \begin{pmatrix} (\mu + \eta_H + \delta_H) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & (\mu + \eta_{HC} + \delta_{HC}) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (\mu + \eta_C + \delta_C) & 0 & 0 & 0 & 0 & 0 \\ -\eta_H & 0 & 0 & (\mu + \alpha + \omega_H + \delta_H) & -\beta & 0 & 0 & 0 \\ 0 & -\eta_{HC} & 0 & -\alpha & (\mu + \beta + \delta_{HC} + \omega_{HC}) & 0 & 0 & 0 \\ 0 & 0 & -\eta_C & 0 & 0 & (\mu + \epsilon + \delta_C) & 0 & 0 \\ 0 & 0 & 0 & -\omega_H & 0 & 0 & (\mu + \alpha) & -\beta \\ 0 & 0 & 0 & 0 & -\omega_{HC} & 0 & -\alpha & (\mu + \beta) \end{pmatrix}$$

Hence for system (1)), the next generation matrix is by,

$$\mathcal{F}_{DFE} V_{DFE}^{-1} = \begin{pmatrix} \frac{\beta\pi}{\mu(\mu+\eta_H+\delta_H)} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\alpha\pi}{\mu(\mu+\eta_C+\delta_C)} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$, whose characteristic polynomial equation at DFE is given by;

$$\lambda^6 \left(\frac{\beta\pi}{\mu(\mu + \eta_H + \delta_H)} - \lambda \right) \left(\frac{\alpha\pi}{\mu(\mu + \eta_C + \delta_C)} - \lambda \right) = 0 \quad (5)$$

The eigenvalues of (5) are;

$$\lambda_1 = \lambda_2 = \lambda_3 = \lambda_4 = \lambda_5 = \lambda_6 = 0, \lambda_7 = \frac{\beta\pi}{\mu(\mu+\eta_H+\delta_H)}, \lambda_8 = \frac{\alpha\pi}{\mu(\mu+\eta_C+\delta_C)}, \text{ hence } R_{01} = \frac{\beta\pi}{\mu(\mu+\eta_H+\delta_H)} \text{ and}$$

$$R_{02} = \frac{\alpha\pi}{\mu(\mu+\eta_C+\delta_C)} \text{ therefore } R_o = \max(R_{01}, R_{02}).$$

➢ *Local Stability Analysis of Disease Free Equilibrium (DFE)*

- *Theorem 3.1*

Define $R_0 = (R_{01}, R_{02})$, then the disease-free equilibrium (DFE) of the system (1) is locally asymptotically stable if $R_o < 1$.

- *Proof :(Method 1)*

At DFE then the Infected HIV and Infected COVID-19 individuals responsible for the co -infection should less than zero i.e $I_H < 0$ and $I_C < 0$,

$\Longrightarrow \frac{dI_H}{dt} = \beta S I_H + \beta R_C I_H - \eta_H I_H - \alpha I_H I_{HC} - (\mu + \delta_H) I_H < 0$,
$\Longrightarrow I_H(\beta S + \beta R_C - \eta_H - \alpha I_{HC} - \mu - \delta_H) < 0$,
$\Longrightarrow I_H\big(\beta S + \beta R_C - (\eta_H + \alpha I_{HC} + \mu + \delta_H)\big) < 0$,
$\Longrightarrow I_H(\mu + \eta_H + \alpha I_{HC} + \delta_H)\left(\frac{\beta S + \beta R_C}{\mu + \eta_H + \alpha I_{HC} + \delta_H} - 1\right) < 0$, at DFE we obtain;
$\Longrightarrow I_H(\mu + \eta_H + \delta_H)\left(\frac{\beta \pi}{\mu(\mu + \eta_H + \delta_H)} - 1\right) < 0$,
$\Longrightarrow \frac{\beta \pi}{\mu(\mu + \eta_H + \delta_H)} - 1 < 0, \Longrightarrow R_{01} - 1 < 0, \Longrightarrow R_{01} < 1.$

In a similar manner it can be shown that from $\frac{dI_C}{dt} = \alpha S I_C - \beta I_C I_{HC} - \eta_C I_C - (\mu + \delta_C) I_C$, then $\frac{\alpha \pi}{\mu(\mu + \eta_C + \delta_C)} - 1 < 0, \Longrightarrow R_{02} - 1 < 0, \Longrightarrow R_{02} < 1.$

Hence the DFE is locally asymptotically stable when $R_{01} < 1$ and $R_{02} < 1 \Longrightarrow R_0 < 1$. ■

- *Proof: Next Generation Method (Method 2)*

The Jacobian matrix of system (1) is given by

$$J = \begin{pmatrix} -(\beta I_H + \alpha I_C + \mu) & -\beta S & 0 & -\alpha S & 0 & 0 & 0 & 0 & 0 & 0 \\ \beta I_H & \beta(S + R_C) - P - \alpha I_{HC} & -\alpha I_H & 0 & 0 & 0 & 0 & 0 & 0 & \beta I_H \\ 0 & \alpha I_{HC} & \alpha I_H - \beta I_c - Q & \beta I_{HC} & 0 & 0 & 0 & 0 & 0 & 0 \\ \alpha I_C & 0 & -\beta I_c & \alpha S - \beta I_{HC} - R & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \eta_H & 0 & 0 & -(U + \omega_H + \delta_H) & \beta & 0 & 0 & 0 & 0 \\ 0 & 0 & \eta_{HC} & 0 & \alpha & -(W + \omega_{HC} + \delta_{HC}) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \eta_C & 0 & 0 & -(\mu + \epsilon + \delta_C) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \omega_H & 0 & 0 & -U & \beta & 0 \\ 0 & 0 & 0 & 0 & 0 & \omega_{HC} & 0 & \alpha & -W & 0 \\ 0 & -\beta R_C & 0 & \epsilon & 0 & 0 & 0 & 0 & 0 & -\mu \end{pmatrix}$$

Where $P = \mu + \eta_H + \delta_H$, $Q = \mu + \eta_{HC} + \delta_{HC}$, $R = \mu + \eta_C + \delta_C$, $U = \mu + \alpha$ and $W = \mu + \beta$

At DFE the Jacobian matrix becomes;

$$J_{DFE} = \begin{pmatrix} -\mu & -\beta S^0 & 0 & -\alpha S^0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta S^0 - P & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -Q & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha S^0 - R & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \eta_H & 0 & 0 & -(U + \omega_H + \delta_H) & \beta & 0 & 0 & 0 & 0 \\ 0 & 0 & \eta_{HC} & 0 & \alpha & -(W + \omega_{HC} + \delta_{HC}) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \eta_C & 0 & 0 & -(\mu + \epsilon + \delta_C) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \omega_H & 0 & 0 & -U & \beta & 0 \\ 0 & 0 & 0 & 0 & 0 & \omega_{HC} & 0 & \alpha & -W & 0 \\ 0 & 0 & 0 & \epsilon & 0 & 0 & 0 & 0 & 0 & -\mu \end{pmatrix}$$

The first six eigenvalues are $\lambda_1 = \lambda_2 = -\mu$, $\lambda_3 = -(\mu + \epsilon + \delta_C)$ and $\lambda_4 = -(\mu + \eta_{HC} + \delta_{HC})$.

$\lambda_5 = \alpha S^0 - (\mu + \eta_C + \delta_C)$ which is negative if $\alpha S^0 - (\mu + \eta_C + \delta_C) < 0$,

$$\Rightarrow \frac{\alpha S^0}{\mu+\eta_C+\delta_C} = \frac{\alpha\pi}{\mu(\mu+\eta_C+\delta_C)} < 1 \Rightarrow R_{02} < 1 \quad (6)$$

Similarly, $\lambda_6 = \beta S^0 - (\mu+\eta_H+\delta_H)$ which is negative if $\beta S^0 - (\mu+\eta_H+\delta_H) < 0$,

$$\Rightarrow \frac{\beta S^0}{\mu+\eta_H+\delta_H} = \frac{\beta\pi}{\mu(\mu+\eta_H+\delta_H)} < 1 \Rightarrow R_{01} < 1 \quad (7)$$

The other four eigenvalues are obtained by solving the equations;

$$\lambda^2 + (2\mu+\alpha+\beta)\lambda + (\mu+\alpha)(\mu+\beta) - \beta\alpha = 0 \quad (8)$$

$$\lambda^2 + \big((\mu+\alpha+\omega_H+\delta_H) + (\mu+\beta+\omega_{HC}+\delta_{HC})\big)\lambda + (\mu+\alpha+\omega_H+\delta_H)(\mu+\beta+\omega_{HC}+\delta_{HC}) - \beta\alpha = 0 \quad (9)$$

λ_7 and λ_8 are obtained from (6) and are negative if ;

$2\mu+\alpha+\beta > 0$ and $(\mu+\alpha)(\mu+\beta) - \beta\alpha > 0$, using R-H criterion for a polynomial of degree 2.

$\Rightarrow 2\mu+\alpha+\beta > 0$, is satisfied since all parameters are positive and,

$\Rightarrow (\mu+\alpha)(\mu+\beta) - \beta\alpha > 0, \Rightarrow 1 > \frac{\beta\alpha}{(\mu+\alpha)(\mu+\beta)} \Rightarrow \frac{\beta\alpha}{(\mu+\alpha)(\mu+\beta)} > 0$, since all parameters are positive hence satisfied.

Similarly, λ_9 and λ_{10} are obtained from (7) and are negative if ;

$2\mu+\alpha+\beta+\omega_H+\delta_H + \omega_{HC}+\delta_{HC} > 0$ and $(\mu+\alpha+\omega_H+\delta_H)(\mu+\beta+\omega_{HC}+\delta_{HC}) - \beta\alpha > 0$, using R-H criterion for a polynomial of degree 2.

$\Rightarrow 2\mu+\alpha+\beta+\omega_H+\delta_H + \omega_{HC}+\delta_{HC} > 0$, is satisfied since all parameters are positive and,

$\Rightarrow (\mu+\alpha+\omega_H+\delta_H)(\mu+\beta+\omega_{HC}+\delta_{HC}) - \beta\alpha > 0, \Rightarrow 1 > \frac{\beta\alpha}{(\mu+\alpha+\omega_H+\delta_H)(\mu+\beta+\omega_{HC}+\delta_{HC})}$

$\Rightarrow \frac{\beta\alpha}{(\mu+\alpha+\omega_H+\delta_H)(\mu+\beta+\omega_{HC}+\delta_{HC})} > 0$, since all parameters are positive hence satisfied.

Hence the DFE is locally asymptotically stable if $R_{01} < 1$ and $R_{02} < 1 \Rightarrow R_0 < 1$ ■

IV. NUMERICAL SIMULATION AND DISCUSSION OF RESULTS

The system of differential equations (1) was solved using MATLAB ode45 solver to study the effects of α and β on the HIV – COVID -19 co-infection dynamics in Kenya. The table below shows the model default parameters used in the simulations. Parameters missing from literature were assumed within reasonable range.

Table 3 Numerical Simulation

Parameter	Description	Value	Reference
π	Recruitment rate into the susceptible population	0.947	Assumed
β	Rate of contracting HIV by any individual	0.3425	[23]
α	Rate of contracting COVID-19 by any individual	0.1175	[20]
η_H	Rate of treatment of HIV infected population	0.75	Assumed
η_{HC}	Rate of treatment of HIV and COVID-19 co- infected population	0.534	Assumed
η_C	Rate of treatment of COVID-19 infected population	0.6744	Assumed
ω_H	Rate of HIV virus suppression	0.715	Assumed
ω_{HC}	Rate of HIV and COVID-19 virus suppression in the co-infected population	0.234	Assumed
ϵ	Rate at which COVID-19 infected population recovers	0.066667	[24]
δ_c	Rate of COVID-19 induced death	0.15	[20]
δ_H	Rate of HIV and AIDS-induced death	0.016	[22]
δ_{HC}	Rate of HIV and AIDS- COVID-19 induced death	0.21	Assumed
μ	Natural death rate	0.0539	[21]

➢ *Discussion of Results*

Figures 1 – 4 below shows the effects of rate of contracting COVID – 19 on HIV and HIV- COVID -19 co-infected individuals. Figure 1 shows that increase in the rate of contracting COVID-19 leads to decrease in the HIV infected population. This is attributed to the fact that more HIV infected individuals are migrating to the HIV- COVID -19 co-infected population. Consequently this leads to increase in HIV- COVID -19 co-infected population with increase in the rate of COVID-19 infection within the first half of the first year and thereafter reducing exponentially as time increases. From figure 3, the treated HIV- COVID -19 co-infected population increases with increase in α. This can be attributed to the fact that once diagnosed with the co-infection, the individuals seek treatment in fear of death mostly attributed to COVID-19 infection. The HIV- COVID -19 co-infected population who are virally suppressed increases with increase in α. This implies that treatment of the HIV- COVID -19 co-infected population leads to viral suppression of HIV virus and recovery from COVID-19 virus which is likely to reduce the mortality rate among the infected population.

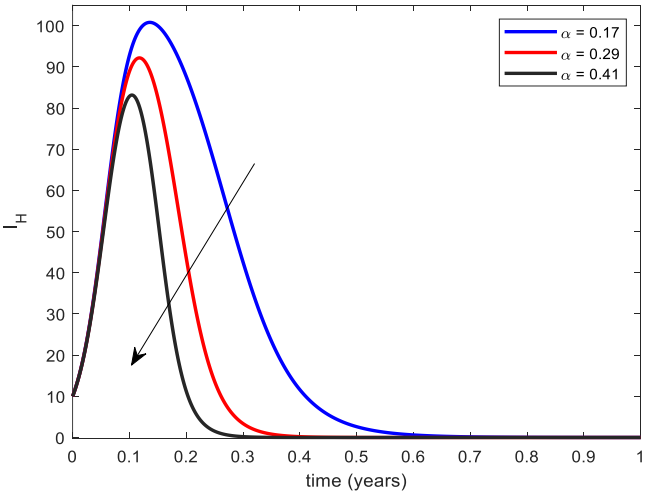

Fig 1 Effect of Rate of Contracting COVID-19 on HIV Infected Individuals

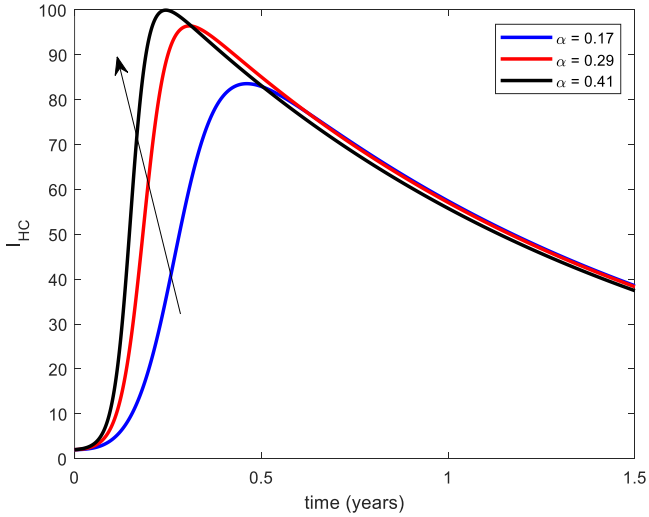

Fig 2 Effect of Rate of Contracting COVID-19 on HIV-COVID-19 Co-Infected Individuals

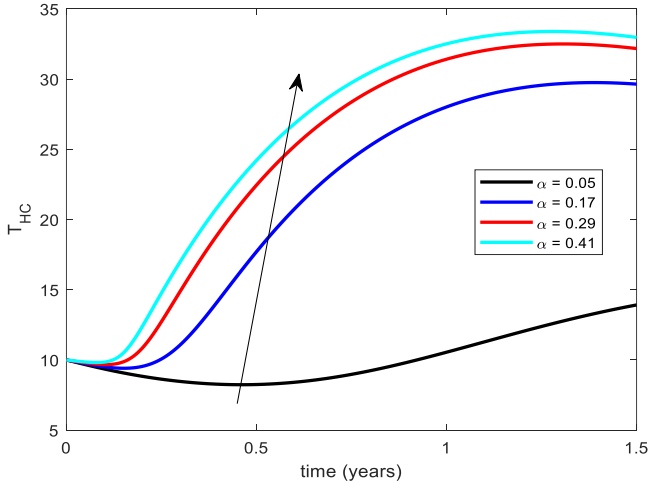

Fig 3 Effect of Rate of Contracting COVID-19 on HIV-COVID-19 Co-Infected Treated Individuals

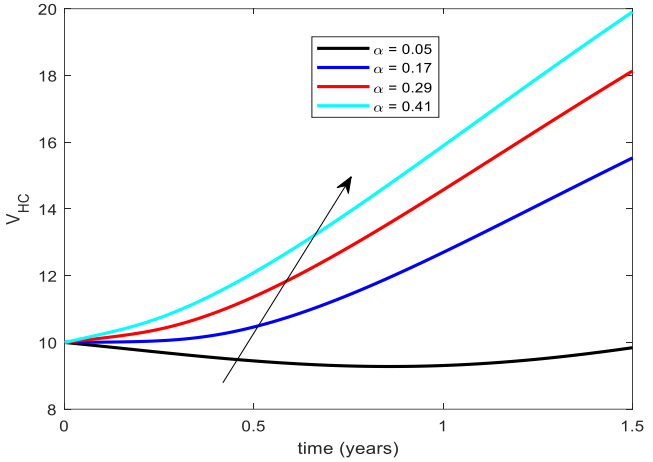

Fig 4 Effect of Rate of Contracting COVID-19 on HIV-COVID-19 Co-Infected Viral Suppressed Individuals

Figures 5 - 8 shows the effects of the rate of increase of HIV infection on HIV- COVID -19 co-infection dynamics. Figure 5 shows that increase in HIV infection leads to drastic increase of HIV infected population in the initial stages of the increase of the infection rate due to increased new infections. Afterwards, the HIV infected population decreases, which can be attributed to high level of HIV patients seeking treatment in Kenya. Increase in β leads to increase in the HIV- COVID – 19 co-infected population within the first 0.6 years after the start of the co=infection as indicated in figure 6. Afterwards, the co-infected population behaves on the converse with increase in β. This is due to the fact that more population is migrating from the HIV infected population to the HIV- COVID – 19 co-infected population. Figure 7 indicates that the treated co-infected population increases with increase in β between the inception of the co-infection and approximately 1.4 years where the co-infected population behaves differently under the same increase of β. This may be as a result of fast mutation of the COVID-19 virus producing different constraints over time. Since more co-infected population is seeking treatment which suppresses the viral load in the infected individuals the Virally suppressed population increases with increases with in β as indicated in figure 8.

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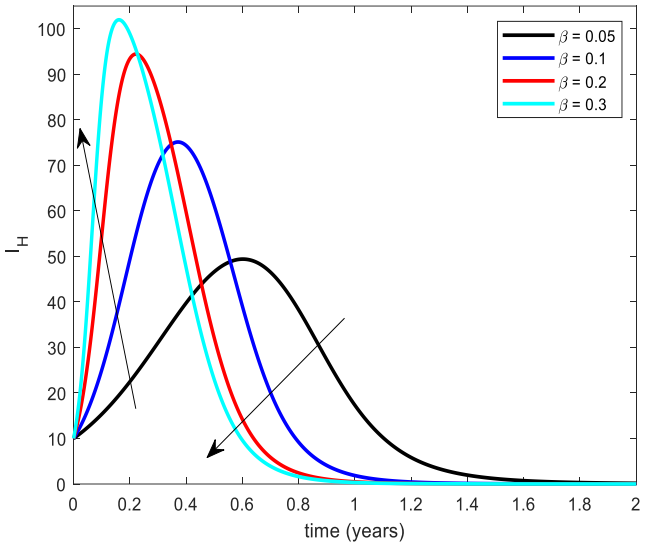

Fig 5 Effect of Rate of Contracting HIV on HIV Infected Individuals

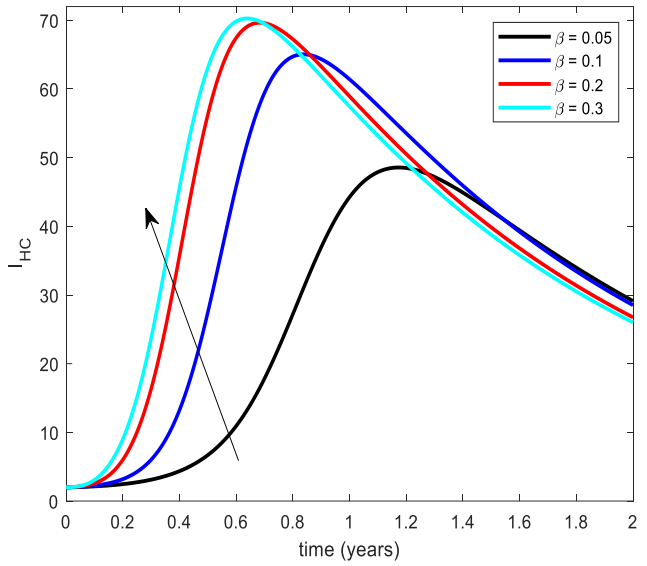

Fig 6 Effect of Rate of Contracting HIV on HIV- COVID-19 Co-Infected Individuals

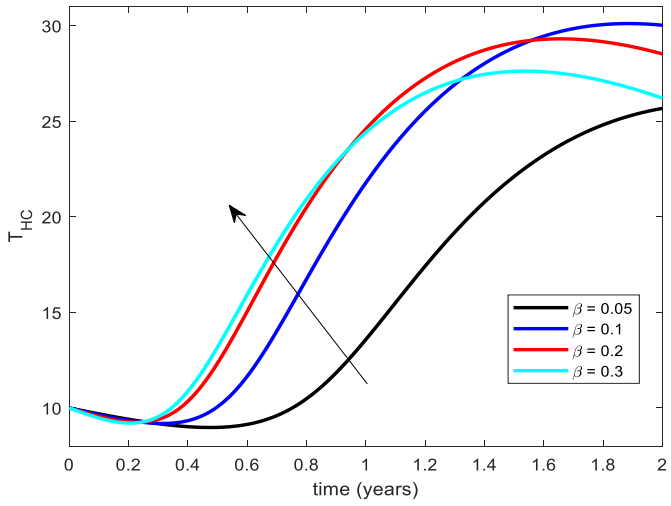

Fig 7 Effect of Rate of Contracting HIV on HIV- COVID-19 Co-Infected Treated Individuals

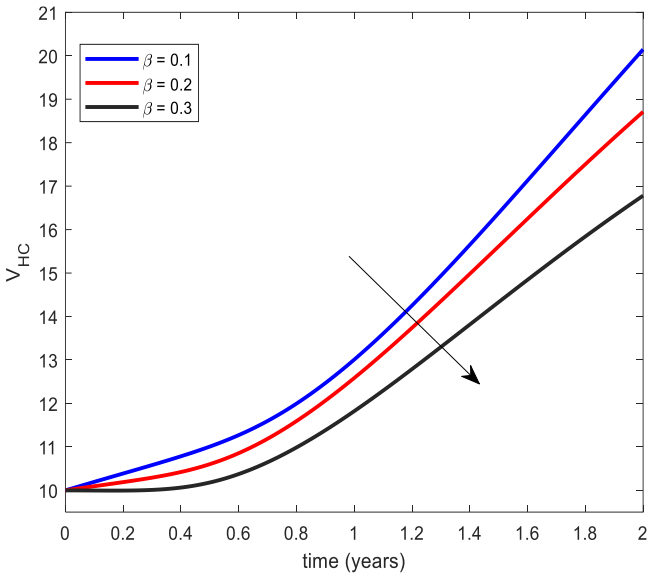

Fig 8 Effect of Rate of Contracting HIV on HIV- COVID-19 Co-Infected Viral Suppressed Individuals

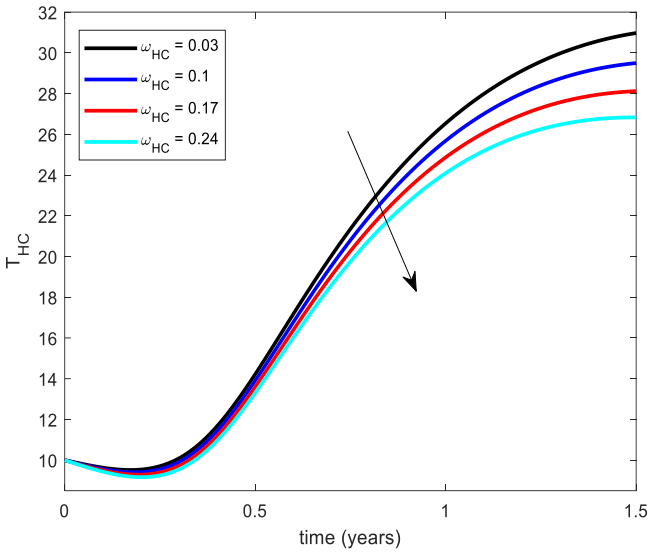

Fig 9 Effect of rate of HIV- COVID-19 viral suppression on HIV- COVID-19 Co-infected treated individuals

Figure 9 shows that treated co-infected population decreases with increase in the rate of virus suppression in the co-infected population. This is due to the fact that this reduces the viral load in the population to the levels where they may not infect susceptible population on interaction. Hence this reduces the treated population because of increased transit to the virally suppressed population leading to increase in the virally suppressed population as indicated in figure 10.

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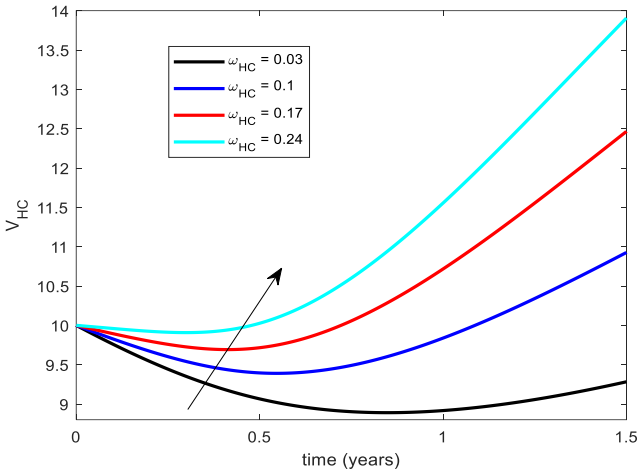

Fig 10 Effect of Rate of HIV- COVID-19 Viral Suppression on HIV- COVID-19 Co-Infected Virus Suppressed Individuals

V. CONCLUSION

This research formulates a mathematical model for the HIV – COVID-19 co-infection dynamics in Kenya. The population under study was divided into 10 compartment. The resulting system of differential equations representing the model was solved numerically using MATLAB ode45 solver. The results obtained shows that:

- Increase in the rate of infection by COVID – 19 and increase of the rate of infection by HIV increases the HIV – COVID-19 co-infected population.
- The treated co-infected population increases with the rates of infection of both COVID-19 and HIV which consequently increases the viral suppressed population.

In conclusion, increase in the COVID-19 infection rate leads to increase in the co-infected population, the treated and the viral suppressed populations in the system. This implies that the co-infected population responds better to treatment to reduce their viral load and hence danger of death due to the co-infection.

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