**Research Article** 



# **Stability Analysis for Co-Dynamics of COVID-19 and HIV-AIDS with Public Sensitization and Education as Controls**

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Abstract: Infectious diseases have denied humanity joy and resources over the centuries. Major diseases such as rabies (1885), whooping cough (1914), flu influenza (1945), and COVID-19 changed the course of life. Hence, a need for alternative methods of combating infections with optimal cost. The coronavirus has shocked the world and it was devastating. The human immunodeficiency virus (HIV) epidemic has caused individuals to rethink their sexual lives, especially in Africa, where the disease has plagued several souls. The authors developed a compartmental model for co-infection of COVID-19 and HIV infection with optimal control. The following controls were incorporated into the model: education/sensitization of susceptible populations, use of anti-retroviral therapy, treatment of co-infected populations, and treatment of the COVID-19-infected population are essential in the fight against HIV and COVID-19 infections. Qualitative and quantitative solutions to the model were determined and analyzed. The HIV-COVID-19-free equilibrium of the co-dynamics model was found to be locally asymptotically stable whenever the reproductive rate was less than one and unstable otherwise. The co-dynamic model was extended to include some controls. This was to establish which strategy is suitable for combating the spread of the COVID-19-infected population. The results of our numerical simulations revealed that in combating COVID-19 and HIV spread, education of susceptible populations and treatment of COVID-19-infected populations are the best options. There has been a reduction in COVID-19 infection, an increase in the COVID-19 recovery population, and a substantial reduction in COVID-19 populations due to this control strategy. The findings of this study are an important step in the fight against HIV and COVID-19. Hence, policymakers should place priority on public education on HIV/COVID-19 infections and treatment of the COVIDinfected population when combating these diseases.

*Keywords*: COVID-19, human immunodeficiency virus-acquired immunodeficiency syndrome (HIV-AIDS), co-infection, optimal control, equilibrium points, stability analysis

**MSC:** 00A71

# **1. Introduction**

Infectious disease has been the monster that has plagued humanity with joy and resources over the century. Major diseases such as rabies (1885), whooping cough (1914), flu influenza (1945), and COVID-19 changed the course of life. The coronavirus disease has been devastating since it was reported in Wuhan, China. The number of confirmed cases and deaths reported across the world is alarming. Further, the HIV epidemic has caused individuals to rethink their sexual lives, especially in Africa, where the disease has plagued several souls.

The viral infection that thrives in the systems of other living organisms, especially humans, was detected in 1981 in the bloodstreams of mostly gay men. HIV accounts for more than 33 million deaths and 37.7 million infected cases since the disease's inception [1]. The disease unfolds in three stages: chronic, acute, and full-blown AIDS.

The authors in [2] formulated a model that focuses on the initial stages of an outbreak whenever pathogens have been observed in a new environment. They provided two methods for determining the risk associated with new cases leading to prolonged spread as opposed to outbreaks fading with a small number of cases.

Omicron spread has infected approximately 600,000 individuals in Shanghai between March and May 2022. Coupled with different control mechanisms by countries in different periods, a model was formulated to determine the impact of medical resources, shelter hospitals, and aerosol transmission generated on the spread of Omicron [3].

HIV and COVID-19 have been in several pieces of literature as academia has sought to find solutions to the spreading process of these epidemics since their inception. The diseases have been considered separately in different literature, with most reviews focusing on COVID-19 due to the devastating implications it induces in the host and its fast-killing effects. Recently, HIV-COVID-19 co-infection has been considered to understand their combined effects in the host's body [4]. Persons with HIV and COVID-19 infections have a higher risk of death as a result of the diseases due to the constant weakening of their white blood cells, which fight other foreign pathogens.

However, people with low CD4 cell counts outnumbered those not receiving anti-retroviral treatment. Those who have underlying medical issues have been found to have a greater risk of COVID-19 in people with HIV [5].

According to [6], co-infection with HIV-COVID-19 produces devastating complications in the host as a result of the disease's ability to significantly decrease the person's immune system. [7] discovered that HIV infection has the ability to make the T-cells not function properly in the affected individuals.

A report by the World Health Organization (WHO) showed that co-infections of HIV and COVID-19 have the ability to produce severe side effects in the infected person and may cause premature death [4].

Smith et al. [8] used a standard kinetic model to investigate the effectiveness of several influenza A virus immune mechanisms, bacteria co-infection, proteins, and anti-viral actions. The authors sought to further define the kinetic influenza A virus infection was measured through infected mice as the study measured the level of precision and frequency of the infection.

Researchers in [9] developed a model involving pneumonia-typhoid co-dynamics to better understand the characteristics of the relationship among the two diseases. The authors conducted an analysis of the model and determined the reproductive rate in terms of the existence and stability of equilibria.

The authors in [10] formulated, developed, and analyzed a sex co-infection transmission model on the dynamics of tuberculosis (TB) and human papilloma virus (HPV) within a population to gain insight into factors that lead to the spread of each disease within the population. Their results showed that whenever the associated reproduction number of the human papilloma virus (HPV-only sub-model) was less than unity, backward bifurcation was observed in the human papilloma virus. The authors in [11] considered an SIS-B deterministic model by incorporating fear and treatment effects. This was done by considering that the disease is transmitted from an infected population to a susceptible population. After some theoretical analysis and computing basic results such as positiveness and boundedness, the basic reproduction number  $R_0$  was computed. The equilibrium points of the model were determined.

Scientists in [12] considered a fractional multi-delay model with control for the co-dynamics of HIV/AIDS and malaria, as proposed by Carvalho and Pinto. The authors conducted numerical simulations to back up their theoretical findings. The proposed dynamical model was shown to be more appropriate and general in describing biological processes. In addition, the model's dynamics were improved and its complexity raised by combining fractional derivatives with a time delay and optimum controls.

The study seeks to add knowledge to:

• The existing studies on HIV-AIDS epidemics and their associated controls.

• The fight against the COVID-19 pandemic and the cost-benefit analysis associated with future preparedness.

• The existing compartmental models that have been formulated in the fight against both HIV-AIDS and COVID-19 diseases.

The study was organized systematically as an introduction to HIV-AIDS, COVID-19, and a review of their related literature. The model formulation, description, qualitative, and stability analysis The optimal control analysis and numerical simulations were used to determine the best optimal control strategy for combating HIV-AIDS and COVID-19.

# 1.1 HIV-AIDS data

Tables 1, 2, and 3 show the number of people tested for viral suppression, the number of people living with HIV with a suppressed viral load, the HIV population of new infections, and total deaths, respectively.

Disaggregation	2019	2020	2021	2022
Children (0-14 years)	4,651	4,734	4,827	4,842
Males (15+ years)	21,393	21,778	22,163	22,555
Females (15+ years)	66,969	68,175	69,381	70,597
Total	93,013	94,687	96,371	97,994

Table 1. Number of people tested for viral suppression among those on treatment

Source: Ghana health service

Disaggregation	2019	2020	2021	2022
Children (0-14 years)	3,176	3,455	3,734	4,023
Males (15+ years)	14,611	15,891	17,181	18,471
Females (15+ years)	45,739	49,745	53,551	57,367
Total	63,526	69,091	74,466	79,861

#### Table 2. Number of people living with HIV with suppressed viral load

Source: Ghana health service

### Table 3. HIV population, new infections and total deaths

Year	Populations	New infections	AIDS death
2019	342,054	21,206	15,922
2020	346,120	18,928	12,758
2021	349,362	15,323	9,886
2022	352,498	12,383	6,974

Source: Ghana health service

# 2. Co-infection model description and formulation

Variables	Description
S	Susceptible population.
$E_H$	Exposed HIV individuals.
$E_{HC}$	Exposed HIV and COVID-19 individuals.
$E_{c}$	Exposed COVID-19 individuals.
$I_H$	Infected HIV individuals.
$I_{HC}$	Infected HIV and COVID-19 individuals.
$I_C$	Infected COVID-19 individuals.
$A_H$	Asymptomatic HIV individuals.
$T_{C}$	Treated COVID-19 individuals.
$R_{C}$	Recovered COVID-19 individuals.

Table 4. Variable description

### Table 5. Parameter description

Parameter	Description
Λ	Recruitment rate of the host population.
β	Transmission rate.
$\alpha_1$	Rate of progression to HIV.
δ	Recruitment rate into full shown HIV compartment.
$\phi$	Recruitment rate from full blown HIV in co-infection.
φ	Recruitment rate from exposed to HIV and COVID-19 into co-infection.
$f_1$	Recruitment rate from exposed HIV into exposed HIV/COVID-19.
$f_2$	Recruitment rate from exposed COVID-19 into exposed HIV and COVID-19.
μ	Natural death.
$\delta_1$	HIV related death.
$\delta_2$	COVID-19 death rate.
$\delta_3$	HIV-COVID-19 death rate.
$\psi_1$	Co-infection recruitment rate.
$\psi_2$	Recruitment rate from COVID-19 infections into co-infection.
$\omega_1$	COVID-19 recovery rate.
$\omega_2$	Recruitment rate from COVID-19 infection into treatment.
σ	Recruitment rate from COVID-19 infection into recovery.
$\delta_4$	Fully blown HIV related death.
$\alpha_2$	Progression rate from exposed to infectious COVID-19 state.

A deterministic model for the co-infection of HIV-COVID-19 was formulated. The model is subdivided into 10 compartments based on the status of infections at any given time. Compartments consist of the following: susceptible (S) population, exposed HIV ( $E_H$ ) population, exposed COVID-19 ( $E_C$ ) population, exposed HIV-COVID-19 ( $E_{HC}$ ) population, infected HIV ( $I_H$ ) population, infected COVID-19 ( $I_C$ ) population, infected HIV-COVID-19 ( $I_{HC}$ ) population, asymptomatic HIV ( $A_H$ ) population, recovered COVID-19 ( $R_C$ ) population, and treated COVID-19 ( $T_C$ ) population. The model assumes homogeneous interactions between the populations and is non-linear in nature. The co-infection model is not a modification of any existing model.

Table 4 and Table 5 show the variables, parameters, and their descriptions used in the model formulation. Table 4 shows a gradual increase in number of people tested for viral suppression among those on treatment. Table 5 shows a gradual increase in number of people living with HIV with suppressed viral load. Figure 1 shows the schematic diagram of the co-infection model.



Figure 1. Schematic diagram of co-infection of model

$$\begin{cases} \frac{d}{dt}S = \Lambda - \beta (I_{c} + I_{H} + I_{Hc})S - \mu S - (1 - \beta (I_{c} + I_{H} + I_{Hc})S) + \rho R_{c} \\ \frac{d}{dt}E_{H} = \beta (I_{c} + I_{H} + I_{Hc})S - (\mu + f_{1} + \alpha_{1})E_{H} \\ \frac{d}{dt}E_{Hc} = f_{1}E_{H} + f_{2}E_{c} - (\mu + \phi)E_{Hc} \\ \frac{d}{dt}E_{c} = (1 - \beta (I_{c} + I_{H} + I_{Hc})S) - (\mu + f_{2} + \alpha_{2})E_{c} \\ \frac{d}{dt}I_{H} = \alpha_{1}E_{H} - (\mu + \delta_{1} + \gamma + \psi_{1})I_{H} \\ \frac{d}{dt}I_{H_{c}} = \psi_{1}I_{H} + \phi E_{Hc} + \psi_{2}I_{c} + \theta A_{H} - (\mu + \delta_{3})I_{Hc} \\ \frac{d}{dt}I_{c} = \alpha_{2}E_{c} - (\mu + \delta_{2} + \omega_{1} + \omega_{2} + \psi_{2})I_{c} \\ \frac{d}{dt}I_{c} = \alpha_{2}I_{c} - (\mu + \sigma)T_{c} \\ \frac{d}{dt}R_{c} = \omega_{1}I_{c} + \sigma T_{c} - (\mu + \rho)R_{c} \end{cases}$$
(1)

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(1) shows the system of differential equations obtained from the HIV-COVID-19 co-infection in Figure 1.

# 3. Co-infection model analysis

## 3.1 Positivity of solutions

The positivity of variables was proven based on the following theorem.

**Theorem 3.1** Let the initial values  $S_0$ ,  $E_{H_0}$ ,  $E_{C_0}$ ,  $I_{H_0}$ ,  $I_{H_0}$ ,  $I_{C_0}$ ,  $T_{C_0}$ , and  $R_{C_0}$  be positive, then the solution set  $\{S, E_H, E_{HC}, E_C, I_H, I_{HC}, A_H, I_C, T_C$  and  $R_c(t)\}$  of 1 is equally positive and bounded for all t > 0, if they existed. **Proof.** 

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \lambda SI_H - \mu S - (1 - \lambda)I_c + \rho R_c \\ \frac{dS}{dt} &\geq -S \left(\lambda I_H - \mu\right) \\ \frac{dS}{dt} &\geq -\lambda \left(I_H - \mu\right) dt \\ \ln S &\geq -\lambda \left(I_H - \mu\right) t + c \\ \text{At } t &= 0, S = S_0, \\ \ln S_0 &\geq c \\ \ln S &\geq -\lambda \left(I_H - \mu\right) t + \ln S_0 \\ \ln \left(\frac{S}{S_0}\right) &\geq -\lambda \left(I_H - \mu\right) t \\ \frac{S}{S_0} &\geq e^{-\lambda (I_H - \mu) t} \\ \text{As } t \to \infty, \\ S &\geq 0 \end{aligned}$$

Hence, applying same in the system of differential equation in as  $t \rightarrow \infty$ 

$$E_{H} \ge 0, E_{HC} \ge 0, E_{c} \ge 0, I_{H} \ge 0, I_{HC} \ge 0, A_{H} \ge 0, I_{c} \ge 0, T_{c} \ge 0, R_{c} \ge 0$$

# 3.2 Feasibility region

Theorem 3.2 The positively invariant set of the model solution is given by

$$\begin{split} \Sigma &= \left\{ S, E_{H}, E_{Hc}, E_{c}, I_{H}, I_{Hc}, A_{H}, I_{c}, T_{c}, R_{c}, \in R_{*}^{10} : N \leq \frac{\Lambda}{\mu}, \mu \neq 0 \right\}, \\ N &= S + E_{c} + I_{c} + T_{c} + R_{c} + E_{H} + I_{H} + A_{H} + I_{Hc} + E_{Hc}, \\ &\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE_{c}}{dt} + \frac{dI_{c}}{dt} + \frac{dT_{c}}{dt} + \frac{dR_{c}}{dt} + \frac{dE_{H}}{dt} + \frac{dI_{H}}{dt} + \frac{dI_{Hc}}{dt} + \frac{dE_{Hc}}{dt} + \frac{dE_{Hc}}{dt}, \\ &\frac{dN}{dt} = \Lambda - \mu \left( S + E_{c} + I_{c} + T_{c} + R_{c} + E_{H} + I_{H} + A_{H} + I_{Hc} + E_{Hc} \right) \\ &- \left( 1 - S + \lambda S + \delta_{2} - \lambda \right) I_{c} - \delta_{4} A_{4} - \delta_{3} I_{Hc}, \\ &\frac{dN}{dt} \leq \Lambda - \mu N, \\ &\frac{dN}{\Lambda - \mu N} \leq dt, \\ &\frac{dN}{-\mu \left( N - \frac{\Lambda}{\mu} \right)} \leq dt, \\ &\frac{dN}{\left( N - \frac{\Lambda}{\mu} \right)} \leq -\mu dt, \\ &\ln \left( N - \frac{\Lambda}{\mu} \right) \leq -\mu t + c. \\ &\operatorname{At} t = 0, N = N_{0}, \\ &\ln \left( N - \frac{\Lambda}{\mu} \right) \leq -\mu t + \ln \left( N_{0} - \frac{\Lambda}{\mu} \right), \end{split}$$

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$$\ln\left(\frac{N-\frac{\Lambda}{\mu}}{N_{0}-\frac{\Lambda}{\mu}}\right) \leq -\mu t,$$
$$\frac{N-\frac{\Lambda}{\mu}}{N_{0}-\frac{\Lambda}{\mu}} \leq e^{-\mu t}.$$
As  $t \to \infty$ ,
$$\frac{N-\frac{\Lambda}{\mu}}{N_{0}-\frac{\Lambda}{\mu}} \leq 0,$$
$$N-\frac{\Lambda}{\mu} \leq 0,$$
$$N \leq \frac{\Lambda}{\mu}.$$

Therefore, a positive solution is the invariant set and is given by

$$\Sigma = \left(S, E_H, E_{HC}, E_C, I_H, I_{HC}, A_H, I_C, T_C, R_C \varepsilon R_+^s : N \leq \frac{\Lambda}{\mu}\right).$$

# 3.3 HIV-COVID-19 free equilibrium

The HIV-COVID-19 free equilibrium of model system (1) is given by

# 3.4 HIV-COVID-19 reproductive rate

Considering state equation (1), the infection compartments can be deduced as:

$$\begin{cases} \frac{d}{dt} E_{H} = \beta (I_{C} + I_{H} + I_{HC}) S - (\mu + f_{1} + \alpha_{1}) E_{H} \\ \frac{d}{dt} E_{HC} = f_{1} E_{H} + f_{2} E_{C} - (\mu + \phi) E_{HC} \\ \frac{d}{dt} E_{C} = (1 - \beta (I_{C} + I_{H} + I_{HC}) S) - (\mu + f_{2} + \alpha_{2}) E_{C} \\ \frac{d}{dt} I_{H} = \alpha_{1} E_{H} - (\mu + \delta_{1} + \gamma + \psi_{1}) I_{H} \\ \frac{d}{dt} I_{H_{C}} = \psi_{1} I_{H} + \phi E_{HC} + \psi_{2} I_{C} + \theta A_{H} - (\mu + \delta_{3}) I_{HC} \\ \frac{d}{dt} A_{H} = \gamma I_{H} - (\theta + \mu + \delta_{4}) A_{H} \\ \frac{d}{dt} I_{C} = \alpha_{2} E_{C} - (\mu + \delta_{2} + \omega_{1} + \omega_{2} + \psi_{2}) I_{C} \\ \frac{d}{dt} T_{C} = \omega_{2} I_{C} - (\mu + \sigma) T_{C} \end{cases}$$

$$(2)$$

From (2), the matrices  $\mathcal{F}$  and  $\mathcal{V}$  are separated as follows:

Thus, the matrix  $\mathcal{F}$  becomes

Evaluating  $\mathcal{F}$  at the disease-free equilibrium

Thus, the matrix  ${\mathcal F}$  becomes

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(3)

In the same way, the Jacobian of the  $\mathcal{V}$  gives

$$\mathcal{V} = \begin{pmatrix} (\mu + f_1 + \alpha_1) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -f_1 & (\mu + \phi) & -f_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (\mu + f_2 + \alpha_2) & 0 & 0 & 0 & 0 & 0 \\ \alpha_1 & 0 & 0 & z_1 & 0 & 0 & 0 & 0 \\ 0 & -\phi & 0 & -\psi_1 & (\mu + \delta_3) & -\theta & -\psi_2 & 0 \\ 0 & 0 & 0 & -\gamma & 0 & (\theta + \mu + \delta_4) & 0 & 0 \\ 0 & 0 & -\alpha_2 & 0 & 0 & 0 & z_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\omega_2 & (\mu + \sigma) \end{pmatrix}$$
(5)

where  $z_1 = (\mu + \delta_1 + \gamma + \psi_1), z_2 = (\mu + \delta_2 + \omega_1 + \omega_2 + \psi_2).$ 

$$\mathcal{V} = \begin{pmatrix} (\mu + f_1 + \alpha_1) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -f_1 & (\mu + \phi) & -f_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (\mu + f_2 + \alpha_2) & 0 & 0 & 0 & 0 & 0 \\ \alpha_1 & 0 & 0 & z_1 & 0 & 0 & 0 & 0 \\ 0 & -\phi & 0 & -\psi_1 & (\mu + \delta_3) & -\theta & -\psi_2 & 0 \\ 0 & 0 & 0 & -\gamma & 0 & (\theta + \mu + \delta_4) & 0 & 0 \\ 0 & 0 & 0 & -\alpha_2 & 0 & 0 & 0 & z_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\omega_2 & (\mu + \sigma) \end{pmatrix}.$$
(6)

Hence, finding the  $V^{-\prime}$  gives

$$V^{-\prime} = \begin{pmatrix} \frac{1}{a_1} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{a_2}{a_1a_3} & \frac{1}{a_3} & \frac{a_4}{a_3a_5} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{a_5} & 0 & 0 & 0 & 0 & 0 \\ -\frac{a_6}{a_1a_7} & -\frac{a_6}{a_1a_7} & 0 & 0 & \frac{1}{a_7} & 0 & 0 & 0 \\ p_1 & \frac{a_8}{(a_3b_1)} & p_2 & p_3 & \frac{1}{b_1} & \frac{b_2}{(b_1b_5)} & \frac{b_3}{(b_1b_7)} & 0 \\ \frac{-(a_6b_4)}{(a_1a_7b_5)} & 0 & 0 & \frac{b_4}{(a_7b_5)} & 0 & \frac{1}{b_5} & 0 & 0 \\ 0 & 0 & \frac{b_6}{(a_5b_7)} & 0 & 0 & 0 & \frac{1}{b_7} & 0 \\ 0 & 0 & \frac{b_6b_8}{(a_5b_7b_9)} & 0 & 0 & 0 & \frac{b_8}{(b_7b_9)} & \frac{1}{b_9} \end{pmatrix}$$

with

$$\begin{aligned} a_{1} &= (\mu + f_{1} + \alpha_{1}), \ a_{2} = -f_{1}, \ a_{3} = (\mu + \phi), \\ a_{4} &= -f_{2}, \ a_{5} = (\mu + f_{2} + \alpha_{2}), \ a_{6} = \alpha, \\ a_{7} &= z_{1}, \ a_{8} = -\phi, \ a_{9} = -\psi_{1}, \\ b_{1} &= (\mu + \delta_{3}), \ b_{2} = -\theta, \ b_{3} = -\psi_{2}, \ b_{4} = -\gamma, \\ b_{5} &= (\theta + \mu + \delta_{4}), \ b_{6} = -\alpha_{2}, \ b_{7} = z_{2}, \\ b_{8} &= -\omega_{2}, \ b_{9} = (\mu + \sigma), \\ p_{1} &= \frac{-(a_{3}a_{6}a_{9}b_{5} - a_{2}a_{7}a_{8}b_{5} + a_{3}a_{6}b_{2}b_{4})}{(a_{1}a_{3}a_{7}b_{1}b_{5})}, \\ p_{2} &= \frac{(a_{4}a_{8}b_{7} + a_{3}b_{3}b_{6})}{(a_{3}a_{5}b_{1}b_{7})}, \ p_{3} = \frac{(a_{9}b_{5} + b_{2}b_{4})}{(a_{7}b_{1}b_{5})}. \end{aligned}$$

Hence, the reproductive rate is given by

$$R_0 = P_{aa} + P_{bb} + P_{cc} + P_{dd}$$
(8)

where

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(7)

$$\begin{split} P_{aa} &= \frac{\beta \alpha \psi_1 \Lambda}{\mu \big(\mu + f_1 + \alpha_1\big) z_1 \theta}, \\ P_{bb} &= \frac{\beta f_1 \phi \Lambda}{\mu \big(\mu + f_1 + \alpha_1\big) (\mu + \phi) \theta}, \\ P_{cc} &= \frac{\beta (\mu + \phi) \alpha \gamma \Lambda}{\mu \big(\mu + f_1 + \alpha_1\big) (\mu + \phi) \big(\theta + \mu + \delta_4\big) z_1}, \\ P_{dd} &= \frac{\beta \alpha \Lambda}{\mu \big(\mu + f_1 + \alpha_1\big) z_1}. \end{split}$$

# 3.5 Local stability HIV-COVID-19 free equilibrium

Local stability of model system (1) was examined at HIV-COVID-19 free equilibrium:

Hence, Jacobian of model system (1) is given by

$$\begin{pmatrix} C_{11} & 0 & 0 & 0 & \beta S & 0 & 0 & \beta S & 0 & \rho \\ C_{22} & C_{33} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & f_1 & -(\mu + \phi) & f_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ (1 - \lambda)I_C & 0 & 0 & C_{66} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_1 & 0 & 0 & C_{44} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi & 0 & \psi_1 & C_{77} & \theta & \psi_2 & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma & 0 & C_{88} & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha_2 & 0 & 0 & 0 & C_{55} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \omega_2 & -(\mu + \sigma) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \omega_1 & \sigma & -(\mu + \rho) \end{pmatrix}$$

(9)

with

$$\begin{split} &u_{11} = -\beta \left( I_C + I_H + I_{HC} \right), \\ &C_{11} = \beta \left( I_C + I_H + I_{HC} \right) - \mu - (1 - \lambda), \\ &C_{22} = \beta \left( I_C + I_H + I_{HC} \right), \\ &C_{33} = - \left( \mu + f_1 + \alpha_1 \right), \\ &C_{44} = - \left( \mu + \delta_1 + \gamma + \psi_1 \right), \end{split}$$

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$$\begin{split} C_{55} &= - \big( \mu + \delta_2 + \omega_1 + \omega_2 \psi_2 \big), \\ C_{66} &= - \big( \mu + f_2 + \alpha_2 \big), \\ C_{77} &= - \big( \mu + \delta_3 \big), \\ C_{88} &= - \big( \theta + \mu + \delta_4 \big), \end{split}$$

when matrix (9) is evaluated at the disease-free equilibrium

$$\left(S_{0}, E_{H_{0}}, E_{HC_{0}}, E_{C_{0}}, I_{H_{0}}, I_{HC_{0}}, A_{H_{0}}, I_{C_{0}}, T_{C_{0}}, R_{C_{0}}\right) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0\right).$$

The Jacobian matrix is given by

$(-\mu$	0	0	0	$\beta S$	0	0	$\beta S$	0	$\rho$
0	$C_{33}$	0	0	0	0	0	0	0	0
0	$f_1$	$-(\mu + \phi)$	$f_2$	0	0	0	0	0	0
0	0	0	$C_{66}$	0	0	0	0	0	0
0	$\alpha_1$	0	0	$C_{44}$	0	0	0	0	0
0	0	$\phi$	0	$\psi_1$	$C_{77}$	$\theta$	$\psi_2$	0	0
0	0	0	0	γ	0	$C_{88}$	0	0	0
0	0	0	$\alpha_{2}$	0	0	0	$C_{55}$	0	0
0	0	0	0	0	0	0	$\omega_2$	$-(\mu + \sigma)$	0
( 0	0	0	0	0	0	0	$\omega_{l}$	$\sigma$	$-(\mu+\rho)$

Now,  $J(A - \rho I)$  becomes

(	$-\mu- ho$	0	0	0	$\beta S$	0	0	$\beta S$	0	$\rho$
	0	$C_{33} - \rho$	0	0	0	0	0	0	0	0
	0	$f_1$	$V_{11} - \rho$	$f_2$	0	0	0	0	0	0
	0	0	0	$V_{12} - \rho$	0	0	0	0	0	0
	0	$\alpha_{_1}$	0	0	$C_{44} - \rho$	0	0	0	0	0
	0	0	$\phi$	0	$\psi_1$	$V_{13} - \rho$	$\theta$	$\psi_2$	0	0
	0	0	0	0	γ	0	$V_{14} - \rho$	0	0	0
	0	0	0	$lpha_2$	0	0	0	$C_{55} - \rho$	0	0
	0	0	0	0	0	0	0	$\omega_2$	$V_{15} - \rho$	0
	0	0	0	0	0	0	0	$\omega_{l}$	$\sigma$	$V_{16} - \rho$

$$V_{11} = -(\mu + \phi),$$
  

$$V_{12} = -(\mu + f_2 + \alpha_2),$$
  

$$V_{13} = -(\mu + \delta_3),$$
  

$$V_{14} = -(\theta + \mu + \delta_4),$$
  

$$V_{15} = -(\mu + \sigma),$$
  

$$V_{16} = -(\mu + \rho).$$

According to the Gershgorin circle theorem, the co-infection model system in (1) is unstable at HIV-COVID-19-free equilibrium since the matrix is not diagonally dominant. Thus,  $|-\mu - \rho| \ge (\beta S + \beta S + \rho)$ ,  $|V_{13} - \rho| \ge (\phi + \psi_1 + \theta + \psi_2)$ .

# 3.6 Existence of endemic equilibrium

Model system (1) has unique endemic equilibrium and is given by  $C_{EE} = (S^*, E_H^*, E_{HC}^*, E_C^*, I_H^*, I_{HC}^*, A_H^*, I_C^*, R_C^*)$  where

$$\begin{split} S^{*} &= \frac{\Lambda + \rho R_{c}^{*}}{\beta \left( I_{c}^{*} + I_{H}^{*} + I_{Hc}^{*} \right) + \left( 1 - \beta \left( I_{c}^{*} + I_{H}^{*} + I_{Hc}^{*} \right) \right)}, \\ E_{H}^{*} &= \frac{\beta \left( I_{c}^{*} + I_{H}^{*} + I_{Hc}^{*} \right) S^{*}}{\left( \mu + f_{1} + \alpha_{1} \right)}, \\ E_{Hc}^{*} &= \frac{f_{1} E_{H}^{*} + f_{2} E_{c}^{*}}{\left( \mu + \phi \right)}, \\ E_{c}^{*} &= \frac{\left( 1 - \beta \left( I_{c}^{*} + I_{H}^{*} + I_{Hc}^{*} \right) \right) S^{*}}{\left( \mu + f_{2} + \alpha_{2} \right)}, \\ I_{H}^{*} &= \frac{\alpha_{1} E_{H}^{*}}{\left( \mu + \delta_{1} + \gamma + \psi_{1} \right)}, \\ I_{Hc}^{*} &= \frac{\psi_{1} I_{H}^{*} + \phi I_{Hc}^{*} + \psi_{2} I_{c}^{*} + \theta A_{H}^{*}}{\left( \mu + \delta_{3} \right)}, \\ A_{H}^{*} &= \frac{\gamma I_{H}^{*}}{\theta + \mu + \delta_{4}}, \end{split}$$

$$\begin{split} I_C^* &= \frac{\alpha_2 E_C^*}{\left(\mu + \delta_2 + \omega_1 + \omega_2 \psi_2\right)}, \\ T_C^* &= \frac{\omega_2 I_C^*}{\left(\mu + \sigma\right)}, \\ R_C^* &= \frac{\omega_1 I_C^* + \sigma T_C^*}{\left(\mu + \rho\right)}. \end{split}$$

# 3.7 Global stability of HIV-COVID-19 free equilibrium

Using approaches in [9, 13, 14] on the model system (1), we established global stability.

$$\frac{dk_1}{dt} = W(k_1, k_2)$$
$$\frac{dk_2}{dt} = A(k_1, k_2)$$

 $k_1$  and  $k_2$  denote the numbers of uninfected and infected individuals, respectively.  $k_1 = (S) \in \mathbb{R}^2$  and  $p_2 = (E_H, E_{HC}, E_C I_H, I_{HC}, A_H, I_C, T_C) \in \mathbb{R}^8$ . The disease-free equilibrium  $D_f$  for the model system (1) is given by  $D_f = (k_1^0, 0)$ . Thus, the global stability at  $D_f$  exists based on these conditions:

• Given  $\frac{dk_1}{dt} = Y(k_1^0, 0), z_1^0$  is globally asymptotically stable.

• 
$$Q(k_1, k_2) = Gx_2 - \widehat{C}(k_1, k_2)$$
 where  $\widehat{C}(k_1, k_2) \ge 0$  for  $(k_1, k_2) \in \in$ .

 $G = Hy_2C(z_1^0, 0)$  is an *M*-matrix with a +ve off-diagonal, and  $\tau$  is a feasible biological region of model (1). Whenever these conditions are met by system (1), then this underlying theorem holds.

**Theorem 3.3** Whenever  $R_0 < 1$  and these two conditions are met by model (1), then equilibrium point  $D_f = (k_1^0, 0)$  is said to be globally asymptotically stable.

Proof. Referring to model system (1), we can deduce

$$\begin{cases} \frac{dk_1}{dt} = Y(k_1, k_2) \\ \frac{kp_1}{dt} = \begin{pmatrix} U(t) \\ \omega_1 I_C + \sigma T_C - (\mu + \rho) R_C \end{pmatrix}, \end{cases}$$
(10)

where

$$U(t) = \Lambda - \beta (I_{C} + I_{H} + I_{HC})S - \mu S - (1 - \beta (I_{C} + I_{H} + I_{HC})S) + \rho R_{c}.$$

Hence,  $Y(k_1, 0)$  becomes

$$H(y_{1},0) = \begin{pmatrix} \Lambda - \beta (I_{C_{0}} + I_{H_{0}} + I_{HC_{0}}) S_{0} - \mu S_{0} - (1 - \beta (I_{C_{0}} + I_{H_{0}} + I_{HC_{0}}) S_{0}) + \rho R_{c_{0}} \\ \omega_{1} I_{C_{0}} + \sigma T_{C_{0}} - (\mu + \rho) R_{C_{0}} \end{pmatrix},$$

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$$Q(k_{1},k_{2}) = \begin{cases} \beta(I_{C}+I_{H}+I_{HC})S - (\mu+f_{1}+\alpha_{1})E_{H} \\ f_{1}E_{H}+f_{2}E_{C} - (\mu+\phi)E_{HC} \\ (1-\beta(I_{C}+I_{H}+I_{HC})S) - (\mu+f_{2}+\alpha_{2})E_{C} \\ \alpha_{1}E_{H} - (\mu+\delta_{1}+\gamma+\psi_{1})I_{H} \\ \psi_{1}I_{H}+\phi E_{HC}+\psi_{2}I_{C}+\theta A_{H} - (\mu+\delta_{3})I_{HC} \\ \gamma I_{H} - (\theta+\mu+\delta_{4})A_{H} \\ \alpha_{2}E_{C} - (\mu+\delta_{2}+\omega_{1}+\omega_{2}+\psi_{2})I_{c} \\ \omega_{2}I_{C} - (\mu+\sigma)T_{C} \end{cases} \end{cases}$$

The Jacobian of  $Q(k_1, k_2)$  is given by

$$J_{D_{f}} = \begin{cases} -U_{1} & 0 & 0 & \beta S & \beta S & 0 & \beta S & 0 \\ f_{1} & -(\mu + \phi) & f_{2} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -U_{2} & -\beta S & -\beta S & 0 & -\beta S & 0 \\ \alpha_{1} & 0 & 0 & 0 & -U_{3} & 0 & 0 & 0 & 0 \\ 0 & \phi & 0 & \psi_{1} & -(\mu + \delta_{3}) & \theta & \psi_{2} & 0 \\ 0 & 0 & 0 & \gamma & 0 & -U_{4} & 0 & 0 \\ 0 & 0 & \alpha_{2} & 0 & 0 & 0 & -U_{5} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \omega_{2} & -(\mu + \sigma) \end{cases}$$
(11)

where

$$U_{1} = (\mu + f_{1} + \alpha_{1}), U_{2} = (\mu + f_{2} + \alpha_{2}), U_{3} = (\mu + \delta_{1} + \gamma + \psi_{1}),$$

$$U_4 = (\theta + \mu + \delta_4), \ U_5 = (\mu + \delta_2 + \omega_1 + \omega_2 + \psi_2).$$

Hence, using the expression

$$Q(p_1, p_2) = Gx_2 - \tilde{W}(k_1, k_2),$$
(12)

we deduce the following  $Q(k_1, k_2)$  is given by

$\left[-U_{1}\right]$	0	0	$\beta S_0$	$\beta S_0$	0	$\beta S_0$	0		$\left( E_{H} \right)$	$\left(D_1\tilde{w}(k_1,k_2)\right)$	
$f_1$	$-(\mu + \phi)$	$f_2$	0	0	0	0	0	ł	$E_{HC}$	$D_2 \tilde{w}(k_1, k_2)$	
0	0	$-U_2$	$-\beta S_0$	$-\beta S_0$	0	$-\beta S_0$	0		$E_C$	$D_3 \tilde{w}(k_1, k_2)$	
$\alpha_1$	0	0	0	$-U_3$	0	0	0	0	$\int I_H$	$D_4 \tilde{w}(k_1,k_2)$	
0	$\phi$	0	$\psi_1$	$-(\mu+\delta_3)$	$\theta$	$\psi_2$	0	ſ	$]I_{HC}$	$D_5 \tilde{w}(k_1,k_2)$	ſ
0	0	0	γ	0	$-U_4$	0	0		$A_{H}$	$D_6 \tilde{w}(k_1, k_2)$	
0	0	$\alpha_{2}$	0	0	0	$-U_{5}$	0		$I_{C}$	$D_7 \tilde{w}(k_1, k_2)$	
0	0	0	0	0	0	$\omega_2$	$-(\mu + \sigma)$	J	$T_C$	$D_8 \tilde{w}(k_1, k_2)$	J

Applying the equation (12), and solving for the expression  $\tilde{w}(k_1, k_2)$  gives T is equal to

$$\begin{cases} \beta (I_{C} + I_{H} + I_{HC}) S - (\mu + f_{1} + \alpha_{1}) E_{H} \\ f_{1}E_{H} + f_{2}E_{C} - (\mu + \phi)E_{HC} \\ (1 - \beta (I_{C} + I_{H} + I_{HC}) S) - (\mu + f_{2} + \alpha_{2}) E_{C} \\ \alpha_{1}E_{H} - (\mu + \delta_{1} + \gamma + \psi_{1}) I_{H} \end{cases} = \begin{cases} -(\mu + f_{1} + \alpha_{1}) E_{H} + \beta S_{0}I_{H} + \beta S_{0}I_{HC} + \beta S_{0}I_{C} \\ f_{1}E_{H} + f_{2}E_{C} - (\mu + \phi)E_{HC} \\ -(\mu + f_{2} + \alpha_{2}) E_{C} - \beta S_{0}I_{H} - \beta S_{0}I_{HC} - \beta S_{0}I_{C} \\ \alpha_{1}E_{H} - (\mu + \delta_{1} + \gamma + \psi_{1}) I_{H} \end{cases} = \begin{cases} -(\mu + f_{1} + \alpha_{1}) E_{H} + \beta S_{0}I_{H} + \beta S_{0}I_{HC} + \beta S_{0}I_{C} \\ f_{1}E_{H} + f_{2}E_{C} - (\mu + \delta_{1} + \gamma + \psi_{1}) I_{H} \\ \gamma I_{H} - (\theta + \mu + \delta_{4}) A_{H} \\ \alpha_{2}E_{C} - (\mu + \delta_{2} + \omega_{1} + \omega_{2} + \psi_{2}) I_{C} \\ \omega_{2}I_{C} - (\mu + \delta_{1} + \gamma + \psi_{1}) T_{C} \end{cases} = \begin{cases} \gamma I_{H} - (\mu + \delta_{1} + \gamma + \psi_{1}) I_{H} \\ \gamma I_{H} - (\theta + \mu + \delta_{4}) A_{H} \\ \alpha_{2}E_{C} - (\mu + \delta_{2} + \omega_{1} + \omega_{2} + \psi_{2}) I_{C} \\ \omega_{2}I_{C} - (\mu + \sigma) T_{C} \end{cases} \end{cases}$$

where

$$T = \begin{cases} D_1 \tilde{w}(k_1, k_2) \\ D_2 \tilde{w}(k_1, k_2) \\ D_3 \tilde{w}(k_1, k_2) \\ D_4 \tilde{w}(k_1, k_2) \\ D_5 \tilde{w}(k_1, k_2) \\ D_6 \tilde{w}(k_1, k_2) \\ D_7 \tilde{w}(k_1, k_2) \\ D_8 \tilde{w}(k_1, k_2) \end{cases}$$

Hence,

It can be seen that the total population of model (1) is bound by  $S_0$ . This follows that S,  $E_H$ ,  $E_{HC}$ ,  $E_C$ ,  $I_H$ ,  $I_{HC}$ ,  $I_C$ ,  $T_H$ ,  $A_H$ ,  $R_C \leq S_0$ , and  $\beta I_H S \leq \beta I_H S_0$ ,  $\beta I_{HC} S \leq \beta I_C S_0$ ,  $\beta I_C S \leq \beta I_C S_0$  which implies  $\widehat{W}(k_1, k_2)$  is +ve definite. Matrix  $J_d f$  is an undoubted *M*-matrix with +ve off-diagonal entries. Therefore, two conditions are met, which is a proof of the globally asymptotically stable  $D_f$  [15-17].

# 3.8 Local stability of endemic equilibrium

The local stability of model system (1) at endemic equilibrium,  $C_{EE}$  = is investigated. Given the Jacobian matrix of system (1) as

$$\begin{pmatrix} t_{11} & 0 & 0 & 0 & \beta S & 0 & 0 & \beta S & 0 & \rho \\ t_{22} & t_{33} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & f_1 & P_{11} & f_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ (1-\lambda)I_C & 0 & 0 & P_{12} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha_1 & 0 & 0 & C_{44} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi & 0 & \psi_1 & P_{13} & \theta & \psi_2 & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma & 0 & P_{14} & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha_2 & 0 & 0 & 0 & C_{55} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \omega_2 & P_{15} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \omega_1 & \sigma & P_{16} \end{pmatrix}$$

with

 $t_{11} = \beta (I_C + I_H + I_{HC}) - \mu - (1 - \lambda),$   $t_{22} = \beta (I_C + I_H + I_{HC}),$   $t_{33} = -(\mu + f_1 + \alpha_1),$   $t_{44} = -(\mu + \delta_1 + \gamma + \psi_1),$   $t_{55} = -(\mu + \delta_2 + \omega_1 + \omega_2 \psi_2),$   $P_{11} = -(\mu + \phi), P_{12} = -(\mu + f_2 + \alpha_2),$   $P_{13} = -(\mu + \delta_3), P_{14} = -(\theta + \mu + \delta_4),$  $P_{15} = -(\mu + \sigma), P_{16} = -(\mu + \rho).$ 

Hence, evaluating (13) at the endemic equilibrium  $C_{EE}$  gives

(14)

(13)

with

$$p_{11} = -(\mu + \phi), P_{12} = -(\mu + f_2 + \alpha_2), P_{13} = -(\mu + \delta_3),$$
$$p_{14} = -(\theta + \mu + \delta_4), P_{15} = -(\mu + \sigma), p_{16} = -(\mu + \rho).$$

Hence, (A - lI) becomes

	$(t_{11} - l)$	0	0	0	$\beta S^*$	0	0	$\beta S^*$	0	$\rho$	
	<i>t</i> <sub>22</sub>	$t_{33} - l$	0	0	0	0	0	0	0	0	
	0	$f_1$	$P_{11} - l$	$f_2$	0	0	0	0	0	0	
	$(1-\lambda)I_c^*$	0	0	$P_{12} - l$	0	0	0	0	0	0	
I –	0	$\alpha_{_1}$	0	0	$C_{44} - l$	0	0	0	0	0	(15)
5 -	0	0	$\phi$	0	$\psi_1$	$P_{13} - l$	$\theta$	$\psi_2$	0	0	(13)
	0	0	0	0	γ	0	$p_{14} - l$	0	0	0	
	0	0	0	$\alpha_2$	0	0	0	$C_{55} - l$	0	0	
	0	0	0	0	0	0	0	$\omega_2$	$P_{15} - l$	0	
	0	0	0	0	0	0	0	$\omega_{\rm l}$	$\sigma$	$p_{16} - l$	

Using Gershgorin's circle theorem, model system (1) is not locally asymptotically stable since  $|t_{33} - l| \ge t_{22}$ ,  $|p_{11} - l| f_1 + f_2$ ,  $|p_{13} - l| \ge \phi + \psi_1 + \psi_2$  and  $|p_{16}| \ge \omega_1 + \sigma$ .

# 3.9 Global stability of HIV-COVID-19 endemic equilibrium

This section examines the global stability of the HIV-COVID-19 endemic equilibrium of the system (1). Constructing the Lyapunov function for system (1):

**Theorem 3.4** Given that  $S = S^*$ ,  $E_H = E_H^*$ ,  $E_{HC} = E_{HC}^*$ ,  $E_C = E_C^*$ ,  $I_H = I_H^*$ ,  $I_{HC} = I_{HC}^*$ ,  $A_H = A_H^*$ ,  $I_C = I_C^*$ ,  $T_C = T_C^*$ , and  $R_C = R_C^*$  then  $E_d^*$  of model (1) is said to be globally asymptotically stable in whenever  $R_0 > 1$ .

**Proof.** Defining Lyapunov function as

$$L: \left\{ \left(S, E_{H}, E_{HC}, E_{C}, I_{H}, I_{HC}, A_{H}, I_{C}, T_{C}, R_{C}\right) \in \Delta \mid S, E_{H}, E_{HC}, E_{C}, I_{H}, I_{HC}, A_{H}, I_{C}, T_{C}, R_{C} > 0 \right\} \rightarrow R$$

given by

$$\begin{split} L = & \left( S - S^* - S^* \ln \frac{S}{S^*} \right) + \left( E_H - E_H^* - E_H^* \ln \frac{E_H}{E_H^*} \right) + \left( E_{HC} - E_{HC}^* - E_{HC}^* \ln \frac{E_{HC}}{E_{HC}^*} \right) \\ & + \left( E_C - E_C^* - E_C^* \ln \frac{E_C}{E_C^*} \right) + \left( I_H - I_H^* - I_H^* \ln \frac{I_H}{I_H^*} \right) + \left( I_{HC} - I_{HC}^* - I_{HC}^* \ln \frac{I_{HC}}{I_{HC}^*} \right) \\ & + \left( A_H - A_H^* - A_H^* \ln \frac{A_H}{A_H^*} \right) + \left( I_C - I_C^* - I_C^* \ln \frac{I_C}{I_C^*} \right) + \left( T_C - T_C^* - T_C^* \ln \frac{T_C}{T_C^*} \right) \\ & + \left( R_C - R_C^* - R_C^* \ln \frac{R_C}{R_C^*} \right). \end{split}$$

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Time derivative of L becomes

$$\begin{split} \frac{dL}{dt} &= \left(\frac{S-S^*}{S}\right) \frac{dS}{dt} + \left(\frac{E_{H}-E_{H}'}{E_{H}}\right) \frac{dE_{H}}{dt} + \left(\frac{E_{HC}-E_{HC}'}{E_{HC}}\right) \frac{dE_{HC}}{dt} \\ &+ \left(\frac{E_{C}-E_{C}'}{E_{C}}\right) \frac{dE_{H}}{dt} + \left(\frac{I_{H}-I_{H}'}{I_{H}}\right) \frac{dI_{H}}{dt} + \left(\frac{I_{HC}-I_{HC}'}{I_{DC}}\right) \frac{dI_{HC}}{dt} \\ &+ \left(\frac{A_{H}-A_{H}'}{A_{H}}\right) \frac{dA_{H}}{dt} + \left(\frac{I_{C}-I_{C}'}{I_{C}}\right) \frac{dI_{C}}{dt} + \left(\frac{T_{C}-T_{C}'}{T_{C}}\right) \frac{dI_{C}}{dt} + \left(\frac{R_{C}-R_{C}'}{R_{C}}\right) \frac{dR_{C}}{dt} \\ &- \left(1-\beta((I_{C}-I_{C}')+(I_{H}-I_{H}')+(I_{HC})-I_{HC})(S-S^{*}) - \mu(S-S^{*})\right) \\ &- \left(1-\beta((I_{C}-I_{C}')+(I_{H}-I_{H}')+(I_{HC})-I_{HC})(S-S^{*}) - (\mu+f_{1}+a_{1})(E_{H}-E_{H}')\right) \\ &+ \left(\frac{E_{HC}-E_{H}'}{E_{H}}\right) \left\{\beta((I_{C}-I_{C}')+(I_{H}-I_{H}')+(I_{HC}-I_{HC}')(S-S^{*}) - (\mu+f_{1}+a_{1})(E_{H}-E_{H}')\right) \right\} \\ &+ \left(\frac{E_{HC}-E_{HC}'}{E_{C}}\right) \left\{I_{1}(E_{H}-E_{H}') + f_{2}(E_{C}-E_{C}') - (\mu+\phi)(E_{HC}-E_{HC}')\right\} \\ &+ \left(\frac{E_{HC}-E_{HC}'}{E_{C}}\right) \left\{I_{1}(-\beta((I_{C}-I_{C}')+(I_{H}-I_{H}')+(I_{HC}-I_{HC}'))(S-S^{*}) - (\mu+f_{2}+\alpha_{2})(E_{C}-E_{C}')\right\} \\ &+ \left(\frac{I_{H}-I_{H}'}{I_{H}}\right) \left\{\alpha_{1}(E_{H}-E_{H}') - (\mu+\delta_{1}+\gamma+\psi_{1})(I_{H}-I_{H}')\right\} \\ &+ \left(\frac{I_{H}-I_{H}'}{I_{H}}\right) \left\{\alpha_{1}(E_{H}-E_{H}') - (\mu+\delta_{1}+\gamma+\psi_{1})(I_{H}-I_{H}')\right\} \\ &+ \left(\frac{I_{H}-I_{H}'}{I_{H}}\right) \left\{\beta((I_{L}-I_{H}') - (\mu+\delta_{1}+\gamma+\psi_{2})(I_{C}-I_{C}') + \theta(A_{H}-A_{H}') - (\mu+\delta_{3})(I_{HC}-I_{HC}')\right\} \\ &+ \left(\frac{I_{H}-I_{H}'}{I_{H}}\right) \left\{\beta((I_{H}-I_{H}') - (\mu+\delta_{2}+\alpha_{1}+\alpha_{2}+\psi_{2})(I_{C}-I_{C}') + \theta(A_{H}-A_{H}') - (\mu+\delta_{3})(I_{HC}-I_{HC}')\right\} \\ &+ \left(\frac{I_{H}-I_{H}'}{A_{H}}\right) \left\{\beta((I_{L}-I_{L}') - (\mu+\delta_{2}+\alpha_{1}+\alpha_{2}+\psi_{2})(I_{C}-I_{C}')\right\} \\ &+ \left(\frac{I_{L}-I_{C}'}{I_{C}}\right) \left\{\alpha_{2}(I_{C}-E_{C}') - (\mu+\delta_{2}+\alpha_{1}+\alpha_{2}+\psi_{2})(I_{C}-I_{C}')\right\} \\ &+ \left(\frac{I_{L}-I_{C}'}{I_{C}}\right) \left\{\alpha_{2}(I_{C}-I_{C}') - (\mu+\sigma)(T_{C}-T_{C}')\right\} \end{aligned}$$

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$$\begin{split} &+ \left(\frac{R_{c} - R_{c}^{*}}{R_{c}}\right) [ e_{0}(l_{c} - l_{c}^{*}) + \sigma(l_{c}^{*} - T_{c}^{*}) - (\mu + \rho)(R_{c} - R_{c}^{*}) \} \\ &\frac{dL}{dt} = \left\{ \Lambda \left( \frac{(S - S^{*})}{S} \right) - \beta \left( I_{c} - l_{c}^{*} \right) \left( \frac{(S - S^{*})^{2}}{S} \right) - \beta \left( I_{H} - I_{H}^{*} \right) \left( \frac{(S - S^{*})^{2}}{S} \right) - \beta \left( I_{Hc} - I_{Hc}^{*} \right) \left( \frac{(S - S^{*})^{2}}{S} \right) \right) \\ &- \mu \left( \frac{(S - S^{*})^{2}}{S} \right) - (1 - \beta) \left( (I_{c} - I_{c}^{*}) + (I_{H} - I_{H}^{*}) + (I_{Hc}) - I_{Hc} \right) \left( \frac{(S - S^{*})^{2}}{S} \right) + \rho \left( R_{c} - R_{c}^{*} \right) \left( \frac{S - S^{*}}{S} \right) \right\} \\ &+ \left\{ \rho \left( I_{c} - I_{c}^{*} \right) (S - S^{*}) \left( \frac{E_{H} - E_{H}^{*}}{E_{H}} \right) + \beta \left( I_{H} - I_{H}^{*} \right) (S - S^{*}) \left( \frac{E_{H} - E_{H}^{*}}{E_{H}} \right) \right) \right\} \\ &+ \beta \left( I_{Hc} - I_{Hc}^{*} \right) (S - S^{*}) \left( \frac{E_{H} - E_{H}^{*}}{E_{Hc}} \right) - (\mu + f_{1} + \alpha_{1}) \left( \frac{(E_{H} - E_{H}^{*})^{2}}{E_{Hc}} \right) \right\} \\ &+ \left\{ f_{1} \left( E_{H} - E_{H}^{*} \right) \left( \frac{E_{Hc} - E_{Hc}^{*}}{E_{Hc}} \right) + f_{2} \left( E_{C} - E_{C}^{*} \right) \left( \frac{E_{Hc} - E_{H}^{*}}{E_{Hc}} \right) - (\mu + \phi) \left( \frac{(E_{Hc} - E_{Hc}^{*})^{2}}{E_{Hc}} \right) \right\} \\ &+ \left\{ (1 - \beta) \left( I_{c} - I_{c}^{*} \right) (S - S^{*}) \left( \frac{E_{c} - E_{C}^{*}}{E_{c}} \right) + \left( I_{H} - I_{H}^{*} \right) (S - S^{*} \right) \left( \frac{E_{c} - E_{C}^{*}}{E_{c}} \right) \right\} \\ &+ \left\{ q_{1} \left( I_{Hc} - I_{Hc}^{*} \right) \left( S - S^{*} \right) \left( \frac{E_{c} - E_{C}^{*}}{E_{c}} \right) - (\mu + f_{2} + \alpha_{2}) \left( \frac{(E_{c} - E_{C}^{*})^{2}}{E_{c}} \right) \right\} \\ &+ \left\{ q_{1} \left( I_{Hc} - I_{H}^{*} \right) \left( \frac{I_{Hc} - I_{Hc}^{*}}{I_{Hc}} \right) - (\mu + \delta_{1} + \gamma + \psi_{1}) \left( \frac{(I_{H} - I_{Hc}^{*})^{2}}{I_{Hc}} \right) \right\} \\ &+ \left\{ \theta \left( A_{H} - A_{H}^{*} \right) \left( \frac{I_{Hc} - I_{Hc}^{*}}{I_{Hc}} \right) - (\mu + \delta_{3} \left( \frac{(I_{Hc} - I_{Hc}^{*})^{2}}{I_{Hc}} \right) \right\} \right\}$$

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$$\begin{split} &+ \left\{ \gamma \left( I_{H} - I_{H}^{*} \right) \left( \frac{A_{H} - A_{H}^{*}}{A_{H}} \right) - \left( \theta + \mu + \delta_{4} \right) \left( \frac{\left( A_{H} - A_{H}^{*} \right)^{2}}{A_{H}} \right) \right\} \\ &+ \left\{ \alpha_{2} \left( E_{c} - E_{c}^{*} \right) \left( \frac{I_{c} - I_{c}^{*}}{I_{c}} \right) - \left( \mu + \delta_{2} + a_{4} + a_{2} + \psi_{2} \right) \left( \frac{\left( I_{c} - I_{c}^{*} \right)^{2}}{I_{c}} \right) \right\} \\ &+ \left\{ \alpha_{4} \left( I_{c} - I_{c}^{*} \right) \left( \frac{T_{c} - T_{c}^{*}}{T_{c}} \right) - \left( \mu + \sigma \right) \left( \frac{\left( T_{c} - T_{c}^{*} \right)^{2}}{T_{c}} \right) \right\} \\ &+ \left\{ \alpha_{4} \left( I_{c} - I_{c}^{*} \right) + \sigma \left( T_{c} - T_{c}^{*} \right) \left( \frac{R_{c} - R_{c}^{*}}{R_{c}} \right) - \left( \mu + \rho \right) \left( \frac{\left( R_{c} - R_{c}^{*} \right)^{2}}{R_{c}} \right) \right\} \\ &+ \left\{ \alpha_{4} \left( I_{c} - I_{c}^{*} \right) + \sigma \left( T_{c} - T_{c}^{*} \right) \left( \frac{\left( S - S^{*} \right)^{2}}{R_{c}} \right) - \left( \mu + \rho \right) \left( \frac{\left( S - S^{*} \right)^{2}}{R_{c}} \right) \right\} \\ &+ \left\{ \alpha_{4} \left( I_{c} - I_{c}^{*} \right) + \sigma \left( T_{c} - T_{c}^{*} \right) \left( \frac{\left( S - S^{*} \right)^{2}}{R_{c}} \right) - \left( \mu + \rho \right) \left( \frac{\left( S - S^{*} \right)^{2}}{R_{c}} \right) \right\} \\ &+ \left\{ \alpha_{4} \left( I_{c} - I_{c}^{*} \right) \left( I_{c} - I_{c}^{*} \right) \left( \left( I_{c} - I_{c}^{*} \right) + \left( I_{H} - I_{H}^{*} \right) \right) \left( \left( S - S^{*} \right)^{2} \right) \right\} \\ &- \mu \left( \frac{\left( S - S^{*} \right)^{2}}{S} \right) - \beta \left( I_{c} - I_{c}^{*} \right) \right\} \\ &+ \left\{ \beta \left( I_{c} - I_{c}^{*} \right) \left( S - S^{*} \right) \left( \frac{E_{\mu} - E_{\mu}^{*}}{E_{\mu}} \right) + \beta \left( I_{H} - I_{H}^{*} \right) \left( S - S^{*} \right) \left( \frac{E_{\mu} - E_{\mu}^{*}}{E_{\mu}} \right) \right\} \\ &+ \left\{ \beta \left( I_{icc} - I_{cc}^{*} \right) \left( S - S^{*} \right) \left( \frac{E_{\mu} - E_{\mu}^{*}}{E_{\mu}} \right) - \left( \mu + f_{1}^{*} + \alpha_{1} \right) \left( \frac{E_{\mu} - E_{\mu}^{*}}{E_{\mu}} \right) \right\} \\ &+ \left\{ f_{1} \left( I_{\mu} - I_{\mu}^{*} \right) \left( S - S^{*} \right) \left( \frac{E_{\mu} - E_{\mu}^{*}}{E_{\mu}} \right) - \left( \mu + \phi_{\mu} \left( \frac{E_{HC} - E_{\mu}^{*} \right)^{2} \right) \right\} \\ &+ \left\{ I_{i} - I_{i}^{*} \right) \left( S - S^{*} \right) \left( \frac{E_{\mu} - E_{\mu}^{*}}{E_{\mu}} \right) - \left( \mu + \phi_{\mu} \left( \frac{E_{\mu} - E_{\mu}^{*} \right)^{2} \right) \right\} \\ \\ &+ \left\{ I_{\mu} - I_{\mu}^{*} \right) \left( S - S^{*} \right) \left( \frac{E_{\mu} - E_{\mu}^{*}}{E_{\mu}} \right) - \left( \mu + \sigma_{\mu} \left( S - S^{*} \right) \left( \frac{E_{\mu} - E_{\mu}^{*}}{E_{\mu}} \right) \right) \\ \\ &+ \left\{ I_{\mu} - I_{\mu}^{*} \right) \left( S - S^{*} \right) \left( \frac{E_{\mu} - E_{\mu}^{$$

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$$+ \left\{ \psi_{1} \left( I_{H} - I_{H}^{*} \right) \left( \frac{I_{HC} - I_{HC}^{*}}{I_{HC}} \right) + \phi \left( E_{HC} - E_{HC}^{*} \right) \left( \frac{I_{HC} - I_{HC}^{*}}{I_{HC}} \right) + \psi_{2} \left( I_{C} - I_{C}^{*} \right) \left( \frac{I_{HC} - I_{HC}^{*}}{I_{HC}} \right) \right) \right\}$$

$$+ \theta \left( A_{H} - A_{H}^{*} \right) \left( \frac{I_{HC} - I_{HC}^{*}}{I_{HC}} \right) - \left( \mu + \delta_{3} \right) \left( \frac{\left( I_{HC} - I_{HC}^{*} \right)^{2}}{I_{HC}} \right) \right) \right\}$$

$$+ \left\{ \gamma \left( I_{H} - I_{H}^{*} \right) \left( \frac{A_{H} - A_{H}^{*}}{A_{H}} \right) - \left( \theta + \mu + \delta_{4} \right) \left( \frac{\left( A_{H} - \overline{A}_{H}^{*} \right)^{2}}{A_{H}} \right) \right) \right\}$$

$$+ \left\{ \alpha_{2} \left( E_{C} - E_{C}^{*} \right) \left( \frac{I_{C} - I_{C}^{*}}{I_{C}} \right) - \left( \mu + \delta_{2} + \omega_{1} + \omega_{2} + \psi_{2} \right) \left( \frac{\left( I_{C} - I_{C}^{*} \right)^{2}}{I_{C}} \right) \right\}$$

$$+ \left\{ \omega_{2} \left( I_{C} - I_{C}^{*} \right) \left( \frac{T_{C} - T_{C}^{*}}{T_{C}} \right) - \left( \mu + \sigma \right) \left( \frac{\left( T_{C} - T_{C}^{*} \right)^{2}}{T_{C}} \right) \right\}$$

$$+ \left\{ \omega_{1} \left( I_{C} - I_{C}^{*} \right) + \sigma \left( T_{C} - T_{C}^{*} \right) \left( \frac{R_{C} - R_{C}^{*}}{R_{C}} \right) - \left( \mu + \rho \right) \left( \frac{\left( R_{C} - R_{C}^{*} \right)^{2}}{R_{C}} \right) \right\}.$$

Using the expression  $t = t_1 - t_2$  gives

$$\begin{split} t_{1} &= \Lambda + \rho \Big( R_{c} - R_{c}^{*} \Big) \Big( \frac{S - S^{*}}{S} \Big) + \beta \Big( I_{c} - I_{c}^{*} \Big) \Big( S - S^{*} \Big) \Big( \frac{E_{H} - E_{H}^{*}}{E_{H}} \Big) + \beta \Big( I_{H} - I_{H}^{*} \Big) \Big( S - S^{*} \Big) \Big( \frac{E_{H} - E_{H}^{*}}{E_{H}} \Big) \\ &+ f_{1} \Big( E_{H} - E_{H}^{*} \Big) \Big( \frac{E_{HC} - E_{HC}^{*}}{E_{HC}} \Big) + f_{2} \Big( E_{C} - E_{C}^{*} \Big) \Big( \frac{E_{HC} - E_{HC}^{*}}{E_{HC}} \Big) + (1 - \beta) \Big( I_{C} - I_{c}^{*} \Big) \Big( S - S^{*} \Big) \Big( \frac{E_{C} - E_{C}^{*}}{E_{C}} \Big) \\ &+ \Big( I_{H} - I_{H}^{*} \Big) \Big( S - S^{*} \Big) \Big( \frac{E_{C} - E_{C}^{*}}{E_{C}} \Big) + \Big( I_{HC} - I_{HC}^{*} \Big) \Big( S - S^{*} \Big) \Big( \frac{E_{C} - E_{C}^{*}}{E_{C}} \Big) + \alpha_{1} \Big( E_{H} - E_{H}^{*} \Big) \Big( \frac{I_{H} - I_{H}^{*}}{I_{H}} \Big) \\ &+ \psi_{1} \Big( I_{H} - I_{H}^{*} \Big) \Big( \frac{I_{HC} - I_{HC}^{*}}{I_{HC}} \Big) + \phi \Big( E_{HC} - E_{HC}^{*} \Big) \Big( \frac{I_{HC} - I_{HC}^{*}}{I_{HC}} \Big) + \psi_{2} \Big( I_{C} - I_{C}^{*} \Big) \Big( \frac{I_{HC} - I_{HC}^{*}}{I_{HC}} \Big) \\ &+ \theta \Big( A_{H} - A_{H}^{*} \Big) \Big( \frac{I_{HC} - I_{HC}^{*}}{I_{HC}} \Big) + \gamma \Big( I_{H} - I_{H}^{*} \Big) \Big( \frac{A_{H} - A_{H}^{*}}{A_{H}} \Big) \\ &+ \alpha_{2} \Big( E_{C} - E_{C}^{*} \Big) \Big( \frac{I_{L} - I_{C}^{*}}{I_{C}} \Big) + \omega_{2} \Big( I_{C} - I_{C}^{*} \Big) \Big( \frac{T_{C} - T_{C}^{*}}{T_{C}} \Big) + \omega_{1} \Big( I_{C} - I_{C}^{*} \Big) \end{split}$$

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$$\begin{split} &+\sigma \big(T_{C}-T_{C}^{*}\big) \bigg(\frac{R_{C}-R_{C}^{*}}{R_{C}}\bigg) \\ &t_{2} = \Lambda \bigg(\frac{S^{*}}{S}\bigg) - \beta \big(I_{C}-I_{C}^{*}\big) \bigg(\frac{\left(S-S^{*}\right)^{2}}{S}\bigg) + \beta \big(I_{H}-I_{H}^{*}\big) \bigg(\frac{\left(S-S^{*}\right)^{2}}{S}\bigg) + \beta \big(I_{HC}-I_{HC}^{*}\big) \bigg(\frac{\left(S-S^{*}\right)^{2}}{S}\bigg) \\ &+\mu \bigg(\frac{\left(S-S^{*}\right)^{2}}{S}\bigg) + \bigg(1-\beta \big(I_{C}-I_{C}^{*}\big) \bigg(\frac{\left(S-S^{*}\right)^{2}}{S}\bigg) + \big(I_{H}-I_{H}^{*}\big) \bigg(\frac{\left(S-S^{*}\right)^{2}}{S}\bigg) + \big(I_{HC}\big) - I_{HC}\bigg) \bigg(\frac{\left(S-S^{*}\right)^{2}}{S}\bigg) \\ &+ \big(\mu + f_{1}+\alpha_{1}\big) \bigg(\frac{\left(E_{H}-E_{H}^{*}\right)^{2}}{E_{H}}\bigg) + \big(\mu + \phi \bigg) \bigg(\frac{\left(E_{HC}-E_{HC}^{*}\right)^{2}}{E_{HC}}\bigg) + \big(\mu + f_{2}+\alpha_{2}\big) \bigg(\frac{\left(E_{C}-E_{C}^{*}\right)^{2}}{E_{C}}\bigg) \\ &+ \big(\mu + \delta_{3}\big) \bigg(\frac{\left(I_{HC}-I_{HC}^{*}\right)^{2}}{I_{HC}}\bigg) + \big(\theta + \mu + \delta_{4}\big) \bigg(\frac{\left(A_{H}-A_{H}^{*}\right)^{2}}{A_{H}}\bigg) + \mu\bigg(\frac{\left(I_{C}-I_{C}^{*}\right)^{2}}{I_{C}}\bigg) + \delta_{2}\bigg(\frac{\left(I_{C}-I_{C}^{*}\right)^{2}}{I_{C}}\bigg) \\ &+ \alpha_{b}\bigg(\frac{\left(I_{C}-I_{C}^{*}\right)^{2}}{I_{C}}\bigg) + \omega_{2}\bigg(\frac{\left(I_{C}-I_{C}^{*}\right)^{2}}{I_{C}}\bigg) + \psi_{2}\bigg(\frac{\left(I_{C}-I_{C}^{*}\right)^{2}}{I_{C}}\bigg) \\ &+ (\mu + \sigma)\bigg(\frac{\left(T_{C}-T_{C}^{*}\right)^{2}}{T_{C}}\bigg) + (\mu + \rho)\bigg(\frac{\left(R_{C}-R_{C}^{*}\right)^{2}}{R_{C}}\bigg). \end{split}$$

Now, by inspection, we observe that  $t_1 > t_2$ . Hence, system (1) is not globally asymptotically stable [18].

# 4. Co-infection model extension to control

Examining possible controls that would help reduce COVID-19 and HIV co-habiting in humans, bearing in mind that HIV-AIDS is never curable, Hence, we modified the model to include control by adding time-dependent controls for  $u_1$ , educating the community on COVID and HIV infections;  $u_2$ , administering anti-retroviral drugs to HIV-infected humans;  $u_3$ , treating COVID-19 and HIV-infected humans; and  $u_4$ , treating the COVID-19-infected population. Hence, given the co-infection model:

$$\frac{d}{dt}S = \Lambda - (1 - u_{1})\beta(I_{c} + I_{H} + I_{Hc})S - \mu S - (1 - u_{1})(1 - \beta)(I_{c} + I_{H} + I_{Hc})S + \rho R_{c}$$

$$\frac{d}{dt}E_{H} = (1 - u_{1})\beta(I_{c} + I_{H} + I_{Hc})S - (\mu + f_{1} + \alpha_{1})E_{H}$$

$$\frac{d}{dt}E_{HC} = f_{1}E_{H} + f_{2}E_{C} - (\mu + \phi)E_{HC}$$

$$\frac{d}{dt}E_{C} = (1 - u_{1})(1 - \beta)(I_{C} + I_{H} + I_{Hc})S - (\mu + f_{2} + \alpha_{2})E_{C}$$

$$\frac{d}{dt}I_{H} = \alpha_{1}E_{H} - (\mu + \delta_{1} + \gamma + \psi_{1} + u_{2})I_{H}$$

$$\frac{d}{dt}I_{H_{c}} = \psi_{1}I_{H} + \phi E_{HC} + \psi_{2}I_{C} + \theta A_{H} - (\mu + \delta_{3} + u_{3})I_{HC}$$

$$\frac{d}{dt}A_{H} = \gamma I_{H} - (\theta + \mu + \delta_{4})A_{H}$$

$$\frac{d}{dt}I_{C} = \alpha_{2}E_{C} - (\mu + \delta_{2} + \omega_{1} + \omega_{2} + \psi_{2} + u_{4})I_{c}$$

$$\frac{d}{dt}R_{c} = \omega_{1}I_{C} + \sigma T_{C} + u_{2}I_{H} + u_{3}I_{HC} + u_{4}I_{C} - (\mu + \rho)R_{C}$$
(16)

We consider a quadratic function, as in [19], that minimizes these populations through education  $u_1$ , treatment of the HIV infected humans  $u_2$ , treatment of the COVID-19 and HIV infected humans  $u_3$  and treatment of the COVID-19 infected humans  $u_4$ . Costs are non-linear in nature. We therefore use a quadratic functional for the optimal control in our study. Hence, we employed an objective functional  $\mathcal{J}$  as:

$$\mathcal{J} = \int_{0}^{t_{f}} \left[ A_{1}E + A_{2}E_{HC} + A_{3}E_{C} + A_{4}I_{H} + A_{5}I_{HC} + A_{6}A_{H} + A_{7}I_{C} + \frac{1}{2} \left( C_{1}u_{1}^{2} + C_{2}u_{2}^{2} + C_{3}u_{3}^{2} + C_{4}u_{4}^{2} \right) \right] \cdot dt$$
(17)

With (17), the quantities  $A_1, A_2, A_3, A_4, A_5, A_6$ , and  $A_7$  are weight constants. The expressions  $\frac{C_1u_1^2}{2}, \frac{C_2u_2^2}{2}, \frac{C_3u_3^2}{2}$ , and  $\frac{C_4u_4^2}{2}$  are the cost-minimizing variables in the model. We seek controls  $u_1^*, u_2^*, u_3^*, u_4^*$  such that

$$\mathcal{J}\left(u_{1}^{*}, u_{2}^{*}, u_{3}^{*}, u_{4}^{*}\right) = \min\left\{J\left(u_{1}, u_{2}, u_{3}, u_{4}\right) : \left(u_{1}, u_{2}, u_{3}, u_{4}\right) \in U\right\}$$
(18)

where

$$U = \{ (u_1, u_2, u_3, u_4) | 0 \le u_i \le 1, i = 1, 2, 3, 4, \text{ Lebesgue measurable} \}$$
(19)

Here, Pontryagin's principle [20, 21], which is an analytic method, would be used to convert systems (16) and (17) into a minimization problem. The Hamiltonian H function with respect to the controls  $u_1$ ,  $u_2$ ,  $u_3$ , and  $u_4$  is given by:

$$H = \left[ A_1 E + A_2 E_{HC} + A_3 E_C + A_4 I_H + A_5 I_{HC} + A_6 A_H + A_7 I_C + \frac{1}{2} \left( C_1 u_1^2 + C_2 u_2^2 + C_3 u_3^2 + C_4 u_4^2 \right) \right]$$

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$$+\lambda_{1} \{ \Lambda - (1-u_{1})\beta(I_{C} + I_{H} + I_{HC})S - \mu S - (1-u_{1})(1-\beta)(I_{C} + I_{H} + I_{HC})S + \rho R_{c} \}$$

$$+\lambda_{2} \{ (1-u_{1})\beta(I_{C} + I_{H} + I_{HC})S - (\mu + f_{1} + \alpha_{1})E_{H} \}$$

$$+\lambda_{3} \{ f_{1}E_{H} + f_{2}E_{C} - (\mu + \phi)E_{HC} \}$$

$$+\lambda_{4} \{ (1-u_{1})(1-\beta)(I_{C} + I_{H} + I_{HC})S - (\mu + f_{2} + \alpha_{2})E_{C} \}$$

$$+\lambda_{5} \{ \alpha_{1}E_{H} - (\mu + \delta_{1} + \gamma + \psi_{1} + u_{2})I_{H} \}$$

$$+\lambda_{6} \{ \psi_{1}I_{H} + \phi E_{HC} + \psi_{2}I_{C} + \theta A_{H} - (\mu + \delta_{3} + u_{3})I_{HC} \}$$

$$+\lambda_{7} \{ \gamma I_{H} - (\theta + \mu + \delta_{4})A_{H} \}$$

$$+\lambda_{8} \{ \alpha_{2}E_{C} - (\mu + \delta_{2} + \omega_{1} + \omega_{2} + \psi_{2} + u_{4})I_{c} \}$$

$$+\lambda_{9} \{ \omega_{2}I_{C} - (\mu + \sigma)T_{C} \}$$

$$+\lambda_{10} \{ \omega_{1}I_{C} + \sigma T_{C} + u_{2}I_{H} + u_{3}I_{HC} + u_{4}I_{C} - (\mu + \rho)R_{C} \}.$$
(20)

**Theorem 4.1** There exists an optimal control  $U^* = u_1^*, u_2^*, u_3^*, u_4^* \in U$  such that

$$\mathcal{J}(u_1^*, u_2^*, u_3^*, u_4^*) = \min_U \mathcal{J}(u_1, u_2, u_3, u_4)$$
(21)

subject to control system (16) with initial conditions.

**Proof.** The control space  $U = \{u \mid u_1, u_2, u_3, u_4 \text{ are measurable, } 0 \le u_1, u_2, u_3, u_4 \le u_m ax < \infty, t \in [0, t_f]\}$  is convex and closed by definition. The optimal system is bounded, which verifies the compactness needed for the existence of the optimal control. Also, the integrand in the functional  $\left[G_1D_p + G_2D_L + G_3D_H + \frac{1}{2}(C_1u_1^2 + C_2u_2^2 + C_3u_3^2)\right]$  is convex on the control u.

Therefore, there exist constant q > 1, +ve values  $u_1$ ,  $u_2$ ,  $u_3$ , and  $u_4$  such

$$J(u_1, u_2, u_3, u_4) \ge u_1 \left( \left| u_1 \right|^2 + \left| u_2 \right|^2 + \left| u_3 \right|^2 \right)^{\frac{q}{2}} - u^2.$$

In the quest to find an optimal solution, Pontryagin's principle is applied to the Hamiltonian (20). If (y, u) is an optimal solution to the control problem, there exists a non-trivial vector function  $\lambda = (\lambda_1 \dots \lambda_6)$  satisfying the below equation:

$$\frac{dz}{dt} = \frac{\partial H(t, y, u, \lambda)}{\partial \lambda}, \ 0 = \frac{\partial H(t, y, u, \lambda)}{\partial u}, \ \frac{d\lambda}{dt} = \frac{\partial H(t, y, u, \lambda)}{\partial z}$$
(22)

Hence, the necessary condition associated with the Hamitonian (20) is applied.

**Theorem 4.2** Given that *S*,  $D_p$ ,  $D_L$ ,  $D_H$ ,  $R_C$  and *R* are optimal state solutions with associated optimal control variables  $(u_1^*, u_2^*, u_3^*, u_4^*)$  for the optimal control problems (16) and (17), there exist adjoint variables  $\lambda_i$  for i = 1, ..., 6, satisfying

$$\begin{split} \dot{\lambda}_{1}^{\prime} &= -\frac{\partial H}{\partial S} = (\dot{\lambda}_{1} - \dot{\lambda}_{2})(1 - u_{1})\beta(I_{c} + I_{H} + I_{HC}) + (\dot{\lambda}_{1} - \dot{\lambda}_{4})(1 - u - 1)(1 - \beta)(I_{c} + I_{H} + I_{HC}) + \mu\dot{\lambda}_{4} \\ \dot{\lambda}_{2}^{\prime} &= -\frac{\partial H}{\partial E_{H}} = A_{1} + (\dot{\lambda}_{2} - \dot{\lambda}_{3})f_{1} + (\dot{\lambda}_{2} - \dot{\lambda}_{3})\alpha_{1} + \mu\dot{\lambda}_{2} \\ \dot{\lambda}_{3}^{\prime} &= -\frac{\partial H}{\partial E_{HC}} = -A_{2} + (\dot{\lambda}_{3} - \dot{\lambda}_{6})\phi + \mu\dot{\lambda}_{3} \\ \dot{\lambda}_{4}^{\prime} &= -\frac{\partial H}{\partial E_{C}} = -A_{3} + (\dot{\lambda}_{4} - \dot{\lambda}_{7})\alpha_{2} + \dot{\lambda}_{4}f_{2} + \mu\dot{\lambda}_{4} \\ \dot{\lambda}_{3}^{\prime} &= -\frac{\partial H}{\partial I_{H}} = -A_{4} + (\dot{\lambda}_{1} - \dot{\lambda}_{2})(1 - u_{1})\beta S + (\dot{\lambda}_{2} - \dot{\lambda}_{4})(1 - \beta)S + (\dot{\lambda}_{5} - \dot{\lambda}_{6})\psi_{1} + (\dot{\lambda}_{5} - \dot{\lambda}_{7})\gamma + (\mu + \delta_{1} + u_{2})\dot{\lambda}_{5} \\ \dot{\lambda}_{6}^{\prime} &= -\frac{\partial H}{\partial I_{HC}} = -A_{5} + (\dot{\lambda}_{1} - \dot{\lambda}_{2})(1 - u_{1})\beta S + (\dot{\lambda}_{1} - \dot{\lambda}_{4})(1 - u_{1})(1 - \beta)S + (\mu + \delta_{3} + u_{3}) \\ \dot{\lambda}_{7}^{\prime} &= -\frac{\partial H}{\partial A_{H}} = -A_{6} + (\theta + \mu + \delta_{4})\dot{\lambda}_{7} \\ \dot{\lambda}_{4}^{\prime} &= -\frac{\partial H}{\partial I_{c}} = (\dot{\lambda}_{1} - \dot{\lambda}_{2})(1 - u_{1})\beta S + (\dot{\lambda}_{1} - \dot{\lambda}_{2})(1 - u_{1})(1 - \beta)S + (\dot{\lambda}_{8} - \dot{\lambda}_{6})\psi_{2} \\ &\quad + (\dot{\lambda}_{8} - \dot{\lambda}_{9})\omega_{2} + (\dot{\lambda}_{8} - \dot{\lambda}_{10})\omega_{1} + (\dot{\lambda}_{8} - \dot{\lambda}_{6})u_{4} \\ \dot{\lambda}_{5}^{\prime} &= -\frac{\partial H}{\partial I_{c}} = (\dot{\lambda}_{9} - \dot{\lambda}_{10})\sigma + \mu\dot{\lambda}_{9} \\ \dot{\lambda}_{10}^{\prime} &= -\frac{\partial H}{\partial I_{c}} = (\dot{\lambda}_{10} - \dot{\lambda}_{1})\rho + \mu\dot{\lambda}_{10} \end{split}$$
(23)

with boundary condition

$$\lambda_i(t_f) = 0, \ i = 1, 2, ..., 10$$
 (24)

The optimal control  $u_1^*$ ,  $u_2^*$ ,  $u_3^*$ , and  $u_4^*$  are given by

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$$u_{1}' = \min\left\{1, \max\left\{0, \left(\left(\lambda_{2} - \lambda_{1}\right)\frac{\beta S\left(I_{C} + I_{H} + I_{HC}\right)}{B_{1}} + \left(\lambda_{4} - \lambda_{1}\right)\frac{(1 - \beta)S\left(I_{C} + I_{H} + I_{HC}\right)}{B_{1}}\right)\right\}\right\}$$
$$u_{2}' = \min\left\{1, \max\left\{0, \left(\left(\lambda_{5} - \lambda_{10}\right)\frac{I_{H}}{B_{2}}\right)\right\}\right\}$$
$$u_{3}' = \min\left\{1, \max\left\{0, \left(\left(\lambda_{6} - \lambda_{10}\right)\frac{I_{HC}}{B_{3}}\right)\right\}\right\}$$
$$u_{4}' = \min\left\{1, \max\left\{0, \left(\left(\lambda_{8} - \lambda_{10}\right)\frac{I_{C}}{B_{4}}\right)\right\}\right\}$$

# 5. Numerical simulations

Numerical simulations regarding the effects of strategies on the HIV-COVID-19 model are conducted using parameter values in Table 6. Table 6 shows a decline in the number of new cases of HIV infections as well as the number of HIV-AIDS deaths. Optimal control is derived by solving a system consisting of a state equation (16), an objective functional equation (17), an adjoint equation (23), transversality conditions, and characterization.

Parameter	Description	Source		
Λ	(10-1,000)	[25]		
β	0.075	[26]		
$\alpha_1$	(0.025-0.075)	[25]		
δ	0.15	[25]		
$\phi$	0.001	Assumed		
$\varphi$	0.0045	Estimated		
$f_1$	0.025	Assumed		
$f_2$	0.035	Assumed		
μ	0.0025	[25]		
$\delta_1$	0.15	[25]		
$\delta_2$	0.12	[26]		
$\delta_3$	0.13	Estimated		
$\psi_1$	0.005	Assumed		
$\psi_2$	0.02	Estimated		
$\omega_1$	0.9286	[26]		
$\omega_2$	0.0714	[26]		
σ	0.0083	[26]		
$\delta_4$	0.14	[25]		
$\alpha_2$	0.01	[26]		

Table 6. Co-infection model and parameter description

Generally, an optimal system is solved by employing an iterative scheme using the Range-Kutta fourth-order approach [22-24].

# 5.1 Strategy 1: Optimal education/sensitization of susceptible population and treatment of COVID-19 infected population

Using education and treatment of COVID-19 infected as control strategies, we optimized the objective functional by setting the other control strategies to zero. Using Strategy 1, it can be established from Figure 2 that the COVID-19 recovered population has increased and the COVID-19 infected population has decreased. Moreover, the co-infected population has also decreased, as shown in Figure 3. This is so because, as the recovery population increases, there is the possibility of the infectious population reducing.

There has been a reduction in the susceptible population and an increased COVID-19-infected population, as shown in Figure 4. This is because, as the population's susceptibility reduces, more people are infected since it is an open, dynamical system.



Figure 2. Population with and without control





# 5.2 Strategy 2: Optimal education/sensitization of susceptible population and use of antiretroviral therapy

Using education and anti-retroviral therapy as control strategies, we optimized the objective function by setting the other control strategies to zero. Using this strategy, we observed from Figure 4 that COVID-19-susceptible individuals have decreased and COVID-19-treated populations have decreased. However, the co-infected population has increased, and HIV infection has reduced in a gradual process, as shown in Figure 4.

There has been a reduction in the COVID-19 susceptible population and a reduction in COVID-19 treated populations, as shown in Figure 5.









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Figure 5. Population with and without control



Figure 6. Population with and without optimal control

# 5.3 Strategy 3: Treatment of COVID-19-HIV infected population and treatment of COVID-19 infected population

Using treatment of co-infected populations and COVID-19-infected populations as control strategies, we optimized the objective functional by setting the other control strategies to zero. Using strategy 3, we can observe from Figure 6 that the COVID-19 recovered population has increased and the COVID-19 infected population has increased. However, HIV infections have reduced in a gradual process, and the co-infected population has increased, as shown in Figure 6. As there are more recoveries in COVID-19, more COVID-19 infections imply that there are substantial numbers of susceptible populations becoming infected in the system.

However, there has been a reduction in the COVID-19 susceptible population and an increase in COVID-19 infected populations, as shown in Figure 7.



Figure 7. Population with and without optimal control

# 6. Conclusion

A co-infection of HIV-COVID-19 model was developed to examine the co-existence of both diseases in humans. The study employed a non-linear differential equation in formulating the co-infection model. Positivity of solutions and boundedness were conducted to determine a feasible region.

The stability analysis of the co-infection model was determined at equilibrium. The local and global stability of the co-dynamics model's equilibria were established. The co-dynamic model was extended to optimal control.

The results of numerical simulations revealed that the best strategy to be used in combating COVID-19 infection spread is Strategy 1 (education of susceptible individuals and treatment of COVID-19-infected populations). As there are more recoveries in COVID-19, more COVID-19 infections imply that there are substantial numbers of susceptible populations becoming infected in the system. There has been a reduction in the susceptible population and an increase in the COVID-19-infected population. This is because, as the population's susceptibility reduces, more people are infected since it is an open, dynamical system.

There were reductions in COVID-19 infection, an increase in the COVID-19 recovery population, and a substantial reduction in co-infection populations due to this control strategy. Therefore, policymakers should give more priority to educating the public on COVID-19 and HIV infections and the treatment of the COVID-19-infected population when combating these diseases.

The authors recommend future research on HIV, COVID-19, and HIV-COVID-19 co-infection. The pattern of dynamics regarding these infections is relevant for policymakers in the fight against future epidemics. These future studies should target the comparison of results and findings to better understand the best control measures for addressing future epidemics.

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## Data availability statement

The data used in the analysis of this study were taken from published articles. These articles are cited at relevant places within the text as references.

# **Conflict of interest**

The authors declare that there is no conflict of interest regarding the publication of this work.

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