Research Article

Global Dynamics of a Within-Host COVID-19/AIDS Coinfection Model with Distributed Delays

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Acquired immunodeficiency syndrome (AIDS) is a spectrum of conditions caused by infection with the human immunodeficiency virus (HIV). Among people with AIDS, cases of COVID-19 have been reported in many countries. COVID-19 (coronavirus disease 2019) is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). In this manuscript, we are going to present a within-host COVID-19/AIDS coinfection model to study the dynamics and influence of the coinfection between COVID-19 and AIDS. The model is a six-dimensional delay differential equation that describes the interaction between uninfected epithelial cells, infected epithelial cells, free SARS-CoV-2 particles, uninfected CD4+ T cells, infected CD4+ T cells, and free HIV-1 particles. We demonstrated that the proposed model is biologically acceptable by proving the positivity and boundedness of the model solutions. The global stability analysis of the model is carried out in terms of the basic reproduction number. Numerical simulations are carried out to investigate that if COVID-19/AIDS coinfected individuals have a poor immune response or a low number of CD4+ T cells, then the viral load of SARS-CoV-2 and the number of infected epithelial cells will rise. On the contrary, the existence of time delays can rise the number of uninfected CD4+ T cells and uninfected epithelial cells, thus reducing the viral load within the host.

1. Introduction

In December 2019, the first case of the emergence of the severe acute respiratory syndrome coronavirus 2 (COVID-19) occurred in Wuhan, China. In March 2020, the World Health Organization (WHO) declared COVID-19 a worldwide epidemic. Globally, as of 27 April 2022, over 500 million people were infected with COVID-19, including 6 million deaths [1]. Old age and its accompanying symptoms such as diabetes, heart disease, and high blood pressure are considered risk factors for developing severe COVID-19 infection and are associated with a high death rate [2, 3]. Some other risk factors are associated when infection with COVID-19 occurs in people with chronic diseases such as acquired immunodeficiency syndrome (AIDS) [4]. In 2020, there were 37.7 million persons living with HIV-1 (PLWH) worldwide; HIV-1 causes acquired immunodeficiency syndrome (AIDS) with 680,000 of them dying from HIV-1-related diseases, and only 73% of them were on antiretroviral medication (ART) [5]. Because their immune systems are impaired, PLWH who do not receive ART or whose condition is poorly managed could be more susceptible to developing COVID-19. If infected with COVID-19, such people are at a greater risk of developing acute symptoms and dying. The coinfection cases are challenging due to the scarcity of data on the outcomes and consequences of SARS-CoV-2 infection in HIV-1 positive individuals [3, 6, 7].

HIV-1 and SARS-CoV-2 are both RNA viruses. SARS-CoV-2 attacks upper respiratory epithelial cells, and the
virus generated by infected cells goes down to the lower airway, infecting bronchial and alveolar epithelial cells [4, 8]. On the other hand, HIV-1 targets CD4+ lymphocytes, which are the immune system’s most plentiful white blood cells (referred to as CD4+ T cells). A great effort is being made in many areas of the world to create measures to battle these viruses and study their biological and immunological features and clinical outcomes. Some of these studies indicate that COVID-19 pandemic has caused disruptions in HIV-1 care facilities in many countries [9, 10]. However, it is unclear whether people infected with HIV-1 having an increased incidence of COVID-19 and significant clinical signs, despite a controversial suggestion that antiretroviral therapy or HIV-1-related immunosuppression could protect HIV-1 infected people from severe COVID-19. A number of HIV-1 and SARS-CoV-2 coinfection cases have been documented throughout the world [11, 12]. Most studies of COVID-19/AIDS coinfection reported that there is a lack of clarity on what constitutes the primary illness and what constitutes comorbiditiy in the context of coinfection. Few studies inferred that SARS-CoV-2 infection does not increase the course of HIV-1 infection in PLWH [13–15]. However, Wang et al. [16] published a case report of an HIV-1/COVID-19 patient with such a lower CD4+ T cell count, and as a result, the patient had a prolonged COVID-19 course and decreased antibody levels. Moreover, COVID-19/AIDS coinfection has been observed to cause pneumonia problems more frequently than COVID-19 alone [17]. This study aims to give a comprehensive picture of SARS-CoV-2 infection in persons having HIV-1/AIDS.

Mathematical models that consist of a system of differential equations have proven their effectiveness in studying the interactions between viruses and their hosts and the common interactions between diseases (see e.g., [12, 18–24]). HIV within-host models have been widely investigated and great results have been reached [18, 19, 25–28]. On the other side, SARS-CoV-2 within-host modeling has received less attention ([24, 29–32]). Some coinfection models between SARS-CoV-2 and other viruses have been developed. For example, Pinky and Dobrovolny [33] used a within-host model to investigate SARS-CoV-2 coinfections with several viruses types such as influenza A virus (IAV), parainfluenza virus (PIV), and human rhinovirus (HRV). In fact, the models of coinfection are essential to grasp the coinfection dynamics between SARS-CoV-2 and HIV, to assist the experimental studies and save time, and to develop effective treatments for coinfected people. Ahmed et al. [34] created a fractional epidemiological model to analyze the pandemic scenario in numerous HIV and COVID-19 affected countries, including South Africa and Brazil. Then, to the best of our knowledge, the first ordinary differential within-host SARS-CoV-2/HIV coinfection system is presented by Al Agha et al. [20]. The formulation of their model is based on Nowak and Bangham’s model that was used widely to model HIV monoinfection and SARS-CoV-2 monoinfection. Al Agha et al. used the same principals to model SARS-CoV-2/HIV coinfection and connect the two infections together. The model is formulated as follows:

\[
\begin{align*}
\dot{X}(t) &= \rho - \alpha X(t) - \eta X(t)V(t), \\
\dot{Y}(t) &= \eta X(t)V(t) - kY(t) - \mu Y(t)S(t), \\
\dot{V}(t) &= aY(t) - bV(t), \\
\dot{S}(t) &= \xi + \omega Y(t)S(t) - \gamma S(t) - \theta S(t)H(t), \\
\dot{W}(t) &= \delta S(t)H(t) - \beta W(t), \\
\dot{H}(t) &= \lambda W(t) - \omega H(t),
\end{align*}
\]

where \( X(t), Y(t), \) and \( V(t) \) represent the healthy epithelial cells, infected epithelial cells, and free SARS-CoV-2 particles, respectively, whilst \( S(t), W(t), \) and \( H(t) \) depict healthy CD4+ T cells, infected CD4+ T cells, and free HIV particles concentrations at time \( t \), respectively. Epithelial cells are recruited at rate \( \rho \) and turned into infected cells at pace \( \eta X(t)V(t) \). Infected produce SARS-CoV-2 particles at rate \( aY(t) \). CD4+ T cells are recruited at rate \( \xi \), eliminate infected epithelial cells at a proportion \( \rho Y(t)S(t) \), and proliferate at rate \( \omega Y(t)S(t) \). HIV particles infect CD4+ T cells at rate \( \theta S(t)H(t) \). The infected cells produces HIV at rate \( \lambda W(t) \). The components \( X(t), Y(t), \) and \( W(t) \) of the model are terminated at rates \( \alpha X(t), kY(t), \) and \( \beta W(t) \), respectively. Then, Elaiw et al. [21] adopted the same previous model with the addition of the effect of latent cells, and then, they made a comprehensive study of the proposed model. Ringa et al. [22] presented a new mathematical model for COVID-19 and HIV/AIDS. The dynamics of the full model is driven by that of its submodels. Also, they studied the impact of intervention measures by incorporating it into the model using time-dependent controls.

Most of the previous publications are the assumptions that cells produce viruses immediately after they are infected. It is commonly observed that in many biological processes, a time delay is inevitable. For HIV-1 infection, it roughly takes about one day for a newly infected cell to become productive and then to be able to produce new virus particles. Therefore, mathematicians have frequently used different types of delays to make biological models more realistic. In [26, 28, 35], HIV models with time delay were introduced, whilst modeling and analysis of COVID-19 based on a time delay dynamic model are presented in [12, 23]. Although there are some publications that combine the coinfection between viruses in the presence of time delay, there are still no models of coinfection between SARS-CoV-2 and HIV with time delay. Due to the decisive role of time delays in dynamic systems, the objective of this work is to expand model (1) to accommodate distributed delays. This can help comprehend the coinfection dynamics between SARS-CoV-2 and HIV-1 from a different perspective. A continuous distribution function is used to represent the delay in case of distributed time delay. This makes distributed delays more realistic than discrete time delays which presume that each individual in the population has the same delay period. Thus, we have investigated a model with six delay differential equations, and we have established the solutions nonnegativity and boundedness, listed the
prospective equilibrium points and the conditions of existence, discussed the global stability of the equilibria, and examined time delay impact on the model’s dynamics. The document includes the following sections: the model is presented in Section 2. Section 3 confirms the basic properties of the model. Section 4 exhibits the global properties of the model. Section 5 lists the numerical simulations. Finally, Section 6 debates the results and some potential next directions.

2. COVID-19/AIDS Coinfection Model with Distributed Delay

In this section, we extend model (1) by considering a variety of distributed time delays as follows:

\[
\begin{align*}
\dot{X}(t) &= \rho - aX(t) - \eta X(t)V(t), \\
\dot{Y}(t) &= \eta \int_0^\infty g_1(\epsilon)e^{-m_1\epsilon}X(t-\epsilon)V(t-\epsilon)d\epsilon - kY(t) - \omega Y(t)S(t), \\
\dot{V}(t) &= a \int_0^\infty g_2(\epsilon)e^{-m_2\epsilon}Y(t-\epsilon)d\epsilon - \varphi V(t), \\
\dot{S}(t) &= \xi + uY(t)S(t) - \gamma S(t) - \mathcal{A}S(t)H(t), \\
\dot{W}(t) &= \mathcal{A} \int_0^\infty g_3(\epsilon)e^{-m_3\epsilon}S(t-\epsilon)H(t-\epsilon)d\epsilon - \beta W(t), \\
\dot{H}(t) &= \lambda \int_0^\infty g_4(\epsilon)e^{-m_4\epsilon}W(t-\epsilon)d\epsilon - \omega H(t).
\end{align*}
\]  

(2)

Thus, we have a system of six delay differential equations where \(X(t), Y(t), V(t), S(t), W(t), \) and \(H(t)\) stand for the concentrations of uninfected epithelial cells, infected epithelial cells, free SARS-CoV-2 particles, uninfected CD4\(^+\) T cells, infected CD4\(^+\) T cells, and HIV-1 particles at time \(t\), respectively. A scheme describing the coinfection between SARS-CoV-2 and HIV in host without time delay is shown in Figure 1. The factor \(g_1(\epsilon)e^{-m_1\epsilon}\) designates the likelihood that uninfected epithelial cells were in touch with SARS-CoV-2 particles at time \(t - \epsilon\) survived \(\epsilon\) time units, and infection occurs at time \(t\). The term \(g_2(\epsilon)e^{-m_2\epsilon}\) simulates the probability of new immature SARS-CoV-2 particles at time \(t - \epsilon\) survived \(\epsilon\) time units and mature at time \(t\). Moreover, the factor \(g_3(\epsilon)e^{-m_3\epsilon}\) symbolizes the probability that uninfected CD4\(^+\) T cells contacted by HIV-1 particles at time \(t - \epsilon\) survived \(\epsilon\) time units and become infected at time \(t\). The term \(g_4(\epsilon)e^{-m_4\epsilon}\) represents the probability that new immature HIV-1 particles at time \(t - \epsilon\) persisted \(\epsilon\) time units and mature at time \(t\), where \(m_i\) and \(i = 1, 2, 3, 4\), are and the positive constants. The delay parameter \(\epsilon\) is a random variable picked from probability distribution functions \(g_i(\epsilon)\) during time interval \([0, \infty)\). The functions \(g_i(\epsilon)\) \((i = 1, 2, 3, 4)\) satisfy \(g_i(\epsilon) > 0\) and

\[
\begin{align*}
\int_0^\infty g_i(\epsilon)d\epsilon &= 1, \quad \int_0^\infty g_i(\epsilon)e^{-m_\epsilon}d\epsilon < \infty,
\end{align*}
\]  

(3)

where \(n > 0\). Let us denote the following model:

\[
\mathcal{F}_i(\epsilon) = g_i(\epsilon)e^{-m_\epsilon},
\]

\[
L_i = \int_0^\infty \mathcal{F}_i(\epsilon)d\epsilon,
\]

(4)

where \(i = 1, 2, 3, 4\). This implies that \(0 < L_i \leq 1\). The initial conditions of model (2) are specified as follows:

\[
\begin{align*}
X(\omega) &= \varphi_1(\omega), & Y(\omega) &= \varphi_2(\omega), & V(\omega) &= \varphi_3(\omega), \\
S(\omega) &= \varphi_4(\omega), & W(\omega) &= \varphi_5(\omega), & H(\omega) &= \varphi_6(\omega),
\end{align*}
\]

(5)

where \(\varphi_i(\omega) \in \mathbb{C} = \{\xi \in C([-\infty, 0], \mathbb{R}) : \xi(\theta)e^{\alpha\theta} \text{ is uniformly continuous for } \theta \in [-\infty, 0], \|\xi\| < \infty\}, \) and \(\|\xi\| = \sup_{\theta \in [0, \infty]}|\xi(\theta)|e^{\alpha\theta}\) such that \(\alpha\) is a positive constant. Here, \(\mathbb{C}\) is the Banach space of fading memory type [36]. Therefore, using the standard theory of differential equations with infinitely distributed delays [37, 38], model (2) with initial constraints (3) has a single solution.

3. Basic Characteristics

This section proves that model (2) solutions are non-negative and ultimately bounded. Additionally, it computes whole potential equilibria and the threshold numbers.

3.1. Non-Negativity and Boundedness

Proposition 1. All of model (2) solutions with beginning conditions (3) are non-negative and eventually bounded.

Proof. Starting with model (2) first equation, we obtain \(X(t)|_{t=0} > 0\), which yields that \(X(t) > 0\) for all \(t \geq 0\). From fourth equation of the model, we get \(S(t)|_{t=\xi} > 0\); then, \(S(t) > 0\) for all \(t \geq 0\). Furthermore, the rest of the model equations give us the following model:
For all $t \in [0, \infty)$, as a result of the recursive argument, we obtain $X(t), Y(t), V(t), S(t), W(t), H(t) \geq 0$ for all $t \geq 0$. Hence, system (2) solutions with initial conditions (3) realize $(X(t), Y(t), V(t), S(t), W(t), H(t)) \in \mathbb{R}_{\geq 0}^6$ for all non-negative values of $t$.

Now, we establish the boundedness of the model’s solutions. Based on model (2) first equation, we gain $\lim_{t \to \infty} \sup X(t) \leq \Omega_1$, where $\Omega_1 = \rho/\alpha$. We define the following model:

$$\Psi_1(t) = \int_0^\infty \mathcal{F}_1(\varepsilon)X(t-\varepsilon)\varepsilon \, d\varepsilon + Y(t) + \frac{\nu}{\mu} S(t).$$

Then, we get the following model:

$$\dot{\psi}_1(t) = \int_0^\infty \mathcal{F}_1(\varepsilon) \left[ \rho - \alpha X(t-\varepsilon) - \eta X(t-\varepsilon) V(t-\varepsilon) \right] \varepsilon \, d\varepsilon + \eta \int_0^\infty \mathcal{F}_1(\varepsilon) X(t-\varepsilon) V(t-\varepsilon) \varepsilon \, d\varepsilon$$

$$- kY(t) - \nu Y(t) S(t) + \frac{\nu}{\mu} \left[ \xi + \nu Y(t) S(t) - \gamma S(t) - \mathcal{F}(t) H(t) \right]$$

$$\leq \rho \int_0^\infty \mathcal{F}_1(\varepsilon) \varepsilon \, d\varepsilon + \frac{\nu \xi}{\mu} - \alpha \int_0^\infty \mathcal{F}_1(\varepsilon) X(t-\varepsilon) \varepsilon \, d\varepsilon - kY(t) - \frac{\nu}{\mu} S(t)$$

$$\leq \rho + \frac{\nu \xi}{\mu} - \phi_1 \left[ \int_0^\infty \mathcal{F}_1(\varepsilon) X(t-\varepsilon) \varepsilon \, d\varepsilon + Y(t) + \frac{\nu}{\mu} S(t) \right]$$

$$= \rho + \frac{\nu \xi}{\mu} - \phi_1 \Psi_1(t),$$
where \( \phi_1 = \min\{a, k, \gamma\} \). This implies that \( \lim_{t \to \infty} \sup Y(t) \leq \Omega_2 \), where \( \Omega_2 = \rho / \phi_1 + \nu / u \phi_1 \). Since \( Y(t) \) and \( S(t) \) are non-negative, then \( \lim_{t \to \infty} \sup Y(t) \leq \Omega_2 \) and \( \lim_{t \to \infty} \sup S(t) \leq \Omega_3 \), where \( \Omega_3 = \xi \Omega_2 / w \). Using model (2) third equation, we get the following model:

\[
\dot{V}(t) = a \int_{0}^{\infty} \mathcal{F}_2(\epsilon) Y(t - \epsilon) d\epsilon - \rho V(t) \leq a L_2 \Omega_2 - \rho V(t) \leq a \Omega_2 - \rho V(t).
\]

(9)

This implies that \( \lim_{t \to \infty} \sup V(t) \leq \Omega_4 \), where \( \Omega_4 = a \Omega_2 / \rho \).

We define the following model:

\[
\Psi_2(t) = \int_{0}^{\infty} \mathcal{F}_3(\epsilon) [\xi + u Y(t - \epsilon) S(t - \epsilon) - \gamma S(t - \epsilon) - \gamma S(t - \epsilon) H(t - \epsilon)] d\epsilon + \mathcal{F}_4(\epsilon) W(t - \epsilon) d\epsilon - \beta W(t) \leq 0
\]

(11)

where \( \phi_2 = \min\{\gamma, \beta\} \). Thus, we have \( \lim_{t \to \infty} \sup W(t) \leq \Omega_5 \), where \( \Omega_5 = (\xi + \mu \Omega_2 / \phi_2) / \phi_2 \). Finally, the last equation of model (2) gives the following model:

\[
\dot{H}(t) = \lambda \int_{0}^{\infty} \mathcal{F}_4(\epsilon) W(t - \epsilon) d\epsilon - \omega H(t) \leq \lambda \Omega_5 - \omega H(t).
\]

(12)

Then, doing the same for second and third equations, we obtain another two possibilities as follows:

\[
V = 0 \text{ or } \eta L_1 X - \frac{\kappa p}{a L_2} + \frac{w y S}{a L_2} = 0.
\]

(17)

Equations (16) and (17) provide us with four possibilities. Accordingly, model (4) has four equilibrium points:

(i) Uninfected equilibrium \( EP_0 = (X_0, 0, 0, S_0, 0, 0) \), where \( X_0 = \rho / \alpha \) and \( S_0 = \xi / \gamma \)

(ii) The HIV-1 monoinfection equilibrium \( EP_1 = (X_1, 0, 0, S_1, W_1, H_1) \), where

\[
X_1 = \frac{\beta \omega}{\alpha L_2}, \quad S_1 = \frac{\beta \omega}{\alpha L_2}, \quad \frac{\beta \omega}{\alpha L_2}, \quad W_1 = \frac{\gamma \omega}{\beta \gamma \omega}, \quad H_1 = \frac{\gamma}{\beta \gamma \omega}.
\]

(18)

It follows that \( W_1 > 0 \) and \( H_1 > 0 \) only when \( \xi \alpha L_2 / \beta \gamma \omega > 1 \). Thus, we have the following model:
Here, \( R_1 = \frac{\xi \beta \eta L_2}{\alpha \eta L_1} \). Here, \( R_1 \) is the basic reproduction number for HIV-1 infection. It sets start of HIV-1 infection in host body. We note that \( W_1 > 0 \) and \( H_1 > 0 \) if \( R_1 > 1 \). Therefore, \( EP_H \) exists when \( R_1 > 1 \).

\[
\begin{align*}
\frac{u \alpha^2 k V_2}{2} + & \left( u \alpha^2 k - a \alpha \eta \rho L_2 - a \alpha \eta \eta \rho L_2 - \alpha \alpha \eta \eta \rho L_2 \right) V - a \alpha \eta \rho L_2 - a \alpha \eta \eta \rho L_2 + \alpha \eta \rho L_2^2 \right)
\right)
= 0.
\end{align*}
\] (21)

To prove that equation (21) has a positive root, we introduce a function \( B(V) \) as follows:

\[
B(V) = \frac{u \alpha^2 k V^2 + \left( u \alpha^2 k - a \alpha \eta \rho L_2 - a \alpha \eta \eta \rho L_2 - \alpha \alpha \eta \eta \rho L_2 \right) V - a \alpha \eta \rho L_2 - a \alpha \eta \eta \rho L_2 + \alpha \eta \rho L_2^2 \right)}{a \eta L_2 (a \eta L_2 - u \alpha V)}.
\] (22)

Then, we have the following equation:

\[
B(0) = \frac{-a \alpha \eta \rho L_2 - a \alpha \eta \eta \rho L_2 + \alpha \eta \rho L_2^2}{a \alpha \eta L_2 L_2^2} = \frac{a \alpha \eta k + a \alpha \eta \xi}{a \eta L_2 L_2} (R_2 - 1).
\] (23)

Here, \( R_2 = a \eta \rho L_2 / a \rho (\alpha \eta \rho + \eta \xi) \). This implies that \( B(0) > 0 \) when \( R_2 > 1 \). In addition, we find that

\[
\lim_{V \to -\frac{a \eta L_2}{u \alpha} \to -\infty} B(V) = -\infty.
\] (24)

It follows that there exists \( 0 < V_2 < u \alpha L_2 / a \rho \) such that \( B(V_2) = 0 \). From equation (20), we get \( Y_2 > 0 \),

(iii) SARS-CoV-2 monoinfection equilibrium \( EP_V \) = \((X_2, Y_2, V_2, S_2, 0, 0)\), where

\[
\begin{align*}
X_2 &= \frac{Y_2 k + S_2 Y_2 u}{\eta V_2 L_1}, \\
Y_2 &= \frac{g V_2}{a L_2}, \\
S_2 &= \frac{\xi}{V - u Y_2},
\end{align*}
\] (20)

\( V_2 \) satisfies the following equation:

(iv) COVID-19/AIDS coinfection equilibrium \( EP_{VH} \) = \((X_3, Y_3, V_3, S_3, W_3, H_3)\), where

\[
\begin{align*}
X_3 &= \frac{\varphi(k \beta \eta L_2 + k \beta \omega)}{a \eta L_2 L_2 L_4}, \\
Y_3 &= \frac{\varphi}{a \eta L_2} \left( \frac{a \eta \beta \eta L_2 L_2 L_4}{a \rho (5 k \beta \eta L_2 L_2 + k \beta \omega)} - 1 \right), \\
V_3 &= \frac{\alpha}{\eta} \left( \frac{a \eta \beta \eta L_2 L_2 L_4}{a \rho (5 k \beta \eta L_2 L_2 + k \beta \omega)} - 1 \right), \\
S_3 &= \frac{\beta \omega}{3 \beta L_2}, \\
W_3 &= \frac{\omega (a \varphi + a \eta L_2)}{a \eta L_2 L_2 L_4} \left[ \frac{\omega}{5 k \beta \eta L_2 L_2 + 5 \beta \omega} \frac{a \eta \beta \eta L_2 L_2 L_4}{a \rho (5 k \beta \eta L_2 L_2 + k \beta \omega)} - 1 \right], \\
H_3 &= \frac{a \varphi + a \eta L_2}{a \eta L_2} \left[ \frac{\omega}{5 k \beta \eta L_2 L_2 + 5 \beta \omega} \frac{a \eta \beta \eta L_2 L_2 L_4}{a \rho (5 k \beta \eta L_2 L_2 + k \beta \omega)} - 1 \right].
\end{align*}
\] (25)
It follows that $W_3 > 0$ and $H_3 > 0$ only when $(\lambda \xi / \beta \omega + u \lambda \rho L_4 / 3 \lambda L_L L_4 + \beta \omega \eta) \alpha L_2 L_3 L_4 / \alpha \varphi + \eta \alpha L_2 > 1$. On the other hand, $Y_3 > 0$ and $V_3 > 0$ only when $\eta \alpha \lambda L_L L_3 L_4 / \alpha \varphi (3 \lambda L_L L_4 + \beta \omega \eta) > 1$. Thus, we can rewrite the components of $EP_{VH}$ as follows:

\[
X_3 = \frac{X_0}{R_4}, \\
Y_3 = \frac{\alpha \varphi}{\alpha L_2} (R_4 - 1), \\
V_3 = \frac{\alpha}{\eta} (R_4 - 1), \\
S_3 = \frac{\beta \omega}{3 \lambda L_L L_4}, \\
W_3 = \frac{\omega (\alpha \varphi + \alpha \eta L_2)}{\eta \alpha \lambda L_L L_4} (R_3 - 1), \\
H_3 = \frac{\alpha \varphi + \alpha \eta L_2}{\alpha \lambda L_L L_4} (R_3 - 1),
\]

where

\[
R_3 = \left( \frac{\lambda \xi}{\beta \omega} + \frac{u \lambda \rho L_1}{3 \lambda L_L L_4 + \beta \omega \eta} \right) \frac{\eta \alpha \lambda L_L L_4}{\alpha \varphi + \alpha \eta L_2} \\
R_4 = \frac{\eta \alpha \lambda L_L L_3 L_4}{\alpha \varphi (3 \lambda L_L L_4 + \beta \omega \eta)}
\]

Therefore, $EP_{VH}$ exists when $R_3 > 1$ and $R_4 > 1$. Here, $R_3$ and $R_4$ are the threshold parameters that mark the COVID-19/AIDS coinfection incidence.

The threshold parameters are defined as follows:

\[
\begin{align*}
R_1 &= \frac{\xi \lambda L_L L_4}{\beta \omega}, \\
R_2 &= \frac{\alpha \eta \alpha L_4 L_2}{\alpha \varphi (\eta \lambda + \xi)}, \\
R_3 &= \left( \frac{\lambda \xi}{\beta \omega} + \frac{u \lambda \rho L_1}{3 \lambda L_L L_4 + \beta \omega \eta} \right) \frac{\eta \alpha \lambda L_L L_4}{\alpha \varphi + \alpha \eta L_2} \\
R_4 &= \frac{\eta \alpha \lambda L_L L_3 L_4}{\alpha \varphi (3 \lambda L_L L_4 + \beta \omega \eta)}
\end{align*}
\]

For simplicity, the contractions listed will be used in the parts that follow

\[
\begin{align*}
X(t) &\equiv X, Y(t) \equiv Y, V(t) \equiv V, \\
S(t) &\equiv S, W(t) \equiv W, H(t) \equiv H,
\end{align*}
\]

and

\[
\begin{align*}
X(t - \varepsilon) &\equiv X_\varepsilon, Y(t - \varepsilon) \equiv Y_\varepsilon, V(t - \varepsilon) \equiv V_\varepsilon, \\
S(t - \varepsilon) &\equiv S_\varepsilon, W(t - \varepsilon) \equiv W_\varepsilon, H(t - \varepsilon) \equiv H_\varepsilon.
\end{align*}
\]

### 4. Global Properties

We demonstrate the global asymptotic stability of all equilibria in this section by building Lyapunov functions using the approach described in [39]. We define $F(\Delta) = \Delta - 1 - \ln \Delta$, where $\Delta$ can be any variable for the model.

**Theorem 1.** Globally asymptotically stable (G.A.S) of equilibrium $EP_0$ is satisfied when $R_1 \leq 1$ and $R_2 \leq 1$.

**Proof.** Take a Lyapunov function $\delta_0(X, Y, V, S, W, H)$ as follows:

\[
\begin{align*}
\delta_0 &= X \varphi F \left( \frac{X}{X_0} \right) + \frac{1}{L_1} Y + \frac{\eta X_0}{\varphi} V + \frac{v}{u L_4} S \varphi F \left( \frac{S}{S_0} \right) + \frac{v}{u L_1 L_3} W + \frac{v \beta}{u \lambda L_4 L_5 L_4} H \\
&\quad + \frac{\eta}{L_1} \int_0^t \mathcal{F}_3(\ell) \int_{t-\varepsilon}^\ell X(\ell)Y(\ell)d\ell d\ell + \frac{\eta X_0}{\varphi} \int_0^t \mathcal{F}_2(\ell) \int_{t-\varepsilon}^\ell Y(\ell)d\ell d\ell \\
&\quad + \frac{v \varphi}{u L_1 L_3} \int_0^t \mathcal{F}_3(\ell) \int_{t-\varepsilon}^\ell S(\ell)H(\ell)d\ell d\ell + \frac{v \beta}{u L_4 L_5 L_4} \int_0^t \mathcal{F}_4(\ell) \int_{t-\varepsilon}^\ell W(\ell)d\ell d\ell.
\end{align*}
\]
Clearly, $\theta_0(X, Y, V, S, W, H) > 0$ for all $X, Y, V, S, W, H > 0$ and $\theta_0(X_0, 0, S_0, 0, 0) = 0$. Calculating $\frac{d\theta_0}{dt}$ along the solutions of system (2) gives the following equation:

$$
\frac{d\theta_0}{dt} = \left(1 - \frac{X_0}{X}\right)[\rho - \alpha X - \eta XV + \frac{1}{L_1} \xi] + \left[\int_0^{\infty} \frac{\mathcal{F}_1(\epsilon)}{\varphi} X, Y, V, d\epsilon \right] - kY - \nu YS - \frac{a\eta X_0}{\varphi} + \int_0^{\infty} \frac{\mathcal{F}_2(\epsilon)}{\varphi} (Y - Y_c) d\epsilon dt
$$

(32)

Adding up the terms in equation (32), we obtain the following equation:

$$
\frac{d\theta_0}{dt} = \left(1 - \frac{X_0}{X}\right)\left(\rho - \alpha X - \eta XV + \frac{1}{L_1} kY - \frac{1}{L_1} \nu YS - \eta X_0 V + \frac{\nu}{uL_1} \left(1 - \frac{S_0}{S}\right) \xi - \nu S\right)
$$

$$
+ \frac{1}{L_1} \nu YS_0 - \frac{\nu}{uL_1} \xi + \frac{\nu}{uL_1} \xi H - \frac{\nu}{uL_1L_3} \eta V + \frac{\nu}{uL_1L_3L_4} \xi H + \frac{\nu}{uL_1L_3L_4} L_2 Y
$$

(33)

Using $\rho = \alpha X_0$ and $\xi = \nu S_0$, we obtain the following equation:

$$
\frac{d\theta_0}{dt} = \frac{\alpha}{X} (X - X_0)^2 - \frac{\nu \eta V}{uL_1S} (S - S_0)^2 - \frac{\nu \eta X_0}{\varphi} aL_2 - \frac{1}{L_1} kL_1 - \frac{1}{L_1} vS_0) Y + \nu \xi + \frac{\nu}{uL_1} (\xi S_0 - \frac{\beta \omega \xi}{L_3L_4}) H
$$

(34)

Since $R_1 \leq 1$ and $R_2 \leq 1$, we get $\frac{d\theta_0}{dt} \leq 0$ for all $X, Y, V, S, W, H > 0$. Also, $\frac{d\theta_0}{dt} = 0$ when $X = X_0$, $S = S_0$, and $Y = H = 0$. Set $T_0 = \{(X, Y, V, S, W, H): \theta_0 = 0\}$ and the largest invariant subset (L.I.S) of $T_0$ by $T_0$. Then, the model solutions converge to $T_0'$. The set $T_0'$ contains elements with $X(t) = X_0$, $S(t) = S_0$, and $Y(t) = H(t) = 0$, and hence, $\dot{Y}(t) = H(t) = 0$. The second and last equations of the model (2) give the following equation:
0 = Y (t) = \eta \int_0^\infty \mathcal{F}_1 (e) X_0 V_\epsilon \, de, \\
0 = H (t) = \lambda \int_0^\infty \mathcal{F}_4 (e) W_\epsilon \, de. 

(35)

Thus, we get V (t) = W (t) = 0 for all t. Then, \( T^*_0 = |EP_0| \), and using Lyapunov–Lasalle asymptotic stability theorem [40–42], \( EP_0 \) is G.A.S.

\[ \square \]

In the following theorems, we need to use the equalities:

\[
\begin{align*}
\ln \left( \frac{S_j H_j}{S} \right) &= \ln \left( \frac{S_j H_j W_j}{S H_j W} \right) + \ln \left( \frac{S_j}{S} \right) + \ln \left( \frac{W_j H_j}{W_j} \right), \\
\ln \left( \frac{W_j}{W} \right) &= \ln \left( \frac{W_j H_j}{W_j} \right) + \ln \left( \frac{W_j}{W} \right), \quad j = 1, 3.
\end{align*}
\]

(36)

Furthermore,

\[
\begin{align*}
\ln \left( \frac{X V_e}{XV} \right) &= \ln \left( \frac{X V_e Y_i}{X V_i Y} \right) + \ln \left( \frac{X}{X} \right) + \ln \left( \frac{Y V_i}{Y V} \right), \\
\ln \left( \frac{Y_i}{Y} \right) &= \ln \left( \frac{Y V_e}{Y V} \right) + \ln \left( \frac{Y V_i}{Y V} \right), \quad i = 2, 3.
\end{align*}
\]

(37)

**Theorem 2.** If \( R_1 > 1 \) and \( R_4 \leq 1 \), the equilibrium \( EP_{44} \) is G.A.S.

**Proof.** Consider a Lyapunov function \( \vartheta_1 (X, Y, S, W, H) \) as follows:

\[
\begin{align*}
\vartheta_1 &= X_1 F \left( \frac{X}{X_1} \right) + \frac{1}{L_1} Y + \frac{\eta X_1}{\varphi} V + \frac{v}{u L_1} S_i F \left( \frac{S_j}{S} \right) + \frac{v}{u L_1 L_3} W_1 F \left( \frac{W}{W_1} \right) + \frac{v \beta W_1}{u L_1 L_3 L_4} \left( \frac{W}{W_1} \right) \\
&\quad + \frac{v \mathcal{G}_S H_1}{u L_1 L_3} \int_0^\infty \mathcal{F}_3 (e) \left( \frac{S H}{S H_1} \right) \, de + \frac{v \beta W_1}{u L_1 L_3 L_4} \int_0^\infty \mathcal{F}_4 (e) \left( \frac{W}{W_1} \right) \, de.
\end{align*}
\]

(38)

Differentiating \( \vartheta_1 \), we obtain the following equation:

\[
\begin{align*}
\frac{d \vartheta_1}{d t} &= (1 - \frac{X_1}{X}) [\rho - \alpha X - \eta X V] + \frac{1}{L_1} \left[ \eta \int_0^\infty \mathcal{F}_1 (e) X_\epsilon V_\epsilon \, de - k Y - v Y S + \frac{\eta X_1}{\varphi} \right] \int_0^\infty \mathcal{F}_2 (e) Y_\epsilon \, de - \frac{\eta X_1}{\varphi} \int_0^\infty \mathcal{F}_2 (e) Y_\epsilon \, de - \eta V \right] \\
&\quad + \frac{v}{u L_1} \left( 1 - \frac{S_j}{S} \right) [\xi + u Y S - \gamma S H + \frac{v}{u L_1 L_3} \left( 1 - \frac{W}{W_1} \right) \int_0^\infty \mathcal{F}_3 (e) S_\epsilon H_\epsilon \, de - \beta W \\
&\quad + \frac{v \beta W_1}{u L_1 L_3 L_4} \int_0^\infty \mathcal{F}_4 (e) W_\epsilon \, de - \omega W \right] + \frac{v \beta W_1}{u L_1 L_3 L_4} \left( \frac{W}{W_1} \right) \\
&\quad + \frac{v \mathcal{G}_S H_1}{u L_1 L_3} \int_0^\infty \mathcal{F}_3 (e) \left( \frac{S H}{S H_1} \right) \, de + \frac{v \beta W_1}{u L_1 L_3 L_4} \int_0^\infty \mathcal{F}_4 (e) \left( \frac{W}{W_1} \right) \, de.
\end{align*}
\]

(39)

Summing the terms of equation (39), we obtain the following equation:

\[
\begin{align*}
\frac{d \vartheta_1}{d t} &= \left( 1 - \frac{X_1}{X} \right) (\rho - \alpha X) Y + \frac{v}{u L_1} \left( 1 - \frac{S_j}{S} \right) (\xi - \gamma S) - \frac{1}{L_1} v Y S_1 + \frac{v}{u L_1} \mathcal{G}_S H \\
&\quad - \frac{v}{u L_1 L_3} \mathcal{G}_3 (e) S H_1 \frac{W_1}{W} \, de + \frac{v}{u L_1 L_3} v H_1 \frac{W_1}{W} \, de - \frac{v \beta w}{u L_1 L_3 L_4} \int_0^\infty \mathcal{F}_3 (e) H \, de - \frac{v \beta w}{u L_1 L_3 L_4} \int_0^\infty \mathcal{F}_4 (e) H \, de \\
&\quad + \frac{v \beta w}{u L_1 L_3 L_4} \int_0^\infty \mathcal{F}_3 (e) \ln \left( \frac{S H}{S H_1} \right) \, de + \frac{v \beta w}{u L_1 L_3 L_4} \int_0^\infty \mathcal{F}_4 (e) \ln \left( \frac{W}{W_1} \right) \, de.
\end{align*}
\]

(40)
By utilizing the equilibrium conditions for $EP_H$, we get the following equation:

$$
\begin{align*}
\rho &= \alpha X_1, \\
\xi &= \gamma S_1 + \mathfrak{H}_1 S_1, \\
\mathfrak{H}_3 H_1 S_1 &= \beta W_1, \\
\lambda L_4 W_1 &= \omega H_1.
\end{align*}
$$

Then, we obtain the following equation:

$$
\frac{d\theta}{dt} = \frac{\alpha}{X} (X - X_1)^2 + \left( \frac{\mu X}{\nu} a L_2 - \frac{1}{L_1} k - \frac{1}{L_1} \nu S_1 \right) Y - \frac{\nu Y}{u L_1 S} (S - S_1)^2 + \frac{\nu}{u L_1} \mathfrak{H}_2 H_1 \left( 1 - \frac{S}{S_1} \right)
$$

$$
\begin{align*}
+ \frac{\nu}{u L_1} \mathfrak{H}_2 H_1 \left( \eta \mathfrak{H}_2 W_1 - \frac{\nu}{u L_1 L_3} \int_0^\infty \mathcal{F}_3 (\epsilon) S_1 H_1 W_1 \, d\epsilon + \frac{\nu}{u L_1} \mathfrak{H}_2 H_1 - \frac{\nu}{u L_1 L_3} \int_0^\infty \mathcal{F}_{4} (\epsilon) W_1 H_1 \, d\epsilon - \frac{\nu}{u L_1} \mathfrak{H}_2 H_1 
\end{align*}
$$

Using the equalities given by equation (36) in case of $j = 1$, we get the following equation:

$$
\frac{d\theta}{dt} = \frac{\alpha}{X} (X - X_1)^2 - \frac{\nu Y}{u L_1 S} (S - S_1)^2 + \frac{\nu}{u L_1} \mathfrak{H}_2 H_1 \left( \frac{\eta}{\alpha \omega} (\mathfrak{H}_2 L_3 + \beta \omega) - 1 \right) Y
$$

$$
\begin{align*}
- \frac{\nu}{u L_1} \mathfrak{H}_2 H_1 \left[ \left( \frac{S}{S_1} - 1 - \ln \left( \frac{S}{S_1} \right) \right) - \frac{\nu}{u L_1 L_3} \int_0^\infty \mathcal{F}_3 (\epsilon) \left( S_1 H_1 W_1 \right) \, d\epsilon \right]
\end{align*}
$$

Therefore, equation (43) becomes

$$
\frac{d\theta}{dt} = \frac{\alpha}{X} (X - X_1)^2 - \frac{\nu Y}{u L_1 S} (S - S_1)^2 + \frac{\nu}{u L_1} \mathfrak{H}_2 H_1 \left( \frac{\eta}{\alpha \omega} (\mathfrak{H}_2 L_3 + \beta \omega) - 1 \right) Y
$$

$$
\begin{align*}
- \frac{\nu}{u L_1} \mathfrak{H}_2 H_1 \left[ \left( \frac{S}{S_1} - 1 - \ln \left( \frac{S}{S_1} \right) \right) - \frac{\nu}{u L_1 L_3} \int_0^\infty \mathcal{F}_3 (\epsilon) \left( S_1 H_1 W_1 \right) \, d\epsilon \right]
\end{align*}
$$

Since $R_4 \leq 1$, we find that $d\theta/dt \leq 0$ for all $X, Y, V, S, W, H > 0$. Also, $d\theta/dt = 0$ when $X = X_1$, $S = S_1$, $Y = 0$, $W = W_1$, and $H = H_1$. Model (2) solutions converge to $T_1$ the L.I.S of $T_1 = \{(X, Y, V, S, W, H): d\theta/dt = 0\}$. The set $T_1$ contains elements with $Y (t) = 0$; then, $Y (t) = 0$. Second equation of system (2) implies
0 = Y(t) = \eta \int_0^\infty \mathcal{F}_1(e)X_1V_\epsilon \, d\epsilon, \quad (45)

which gives \( V(t) = 0 \) for all \( t \). Therefore, \( T'_1 = \{ EP_V \} \) and \( EP_H \) is G.A.S according to Lyapunov–LaSalle asymptotic stability theorem [40–42].

Theorem 3. If \( R_2 > 1 \) and \( R_3 \leq 1 \), then the equilibrium \( EP_V \) is G.A.S.

Proof. We introduce a Lyapunov function \( \varphi_2(X, Y, V, S, W, H) \) as follows:

\[
\varphi_2 = X_2F\left(\frac{X}{X_2}\right) + \frac{1}{L_1}Y_2F\left(\frac{Y}{Y_2}\right) + \frac{\nu L_1}{L_2}Y_2F\left(\frac{V}{V_2}\right) + \frac{\nu}{u L_1}S_1F\left(\frac{S}{S_2}\right) + \frac{\nu}{u L_1}W + \frac{v \beta}{u L_1 L_3 L_4}H
\]

\[
+ \frac{\eta X_2 V_2}{L_1} \int_0^\infty \mathcal{F}_1(e) \left( F\left(\frac{X(e)}{X_2}\right) - \mathcal{F}_1(e) \right) \, d\epsilon \, d\epsilon + \frac{\eta X_2 Y_2}{\varphi \varphi} \int_0^\infty \mathcal{F}_2(e) \left( Y(e) - \mathcal{F}_1(e) \right) \, d\epsilon \, d\epsilon
\]

\[
+ \frac{v}{u L_1 L_3 L_4} \lambda \int_0^t \mathcal{F}_3(e) \left( \frac{S_H}{S_H} \right) \, d\epsilon \, d\epsilon + \frac{v \beta}{u L_1 L_3 L_4} \lambda \int_0^t \mathcal{F}_4(e) \left( \frac{W}{W_\epsilon} \right) \, d\epsilon \, d\epsilon.
\]

Differentiating \( \varphi_2 \), we obtain the following equation:

\[
\frac{d\varphi_2}{dt} = (1 - X_2/X)\left[ \rho - aX - \eta XV \right] + \frac{1}{L_1} \left( 1 - Y_2/Y \right) \left[ \eta \int_0^\infty \mathcal{F}_1(e) X_v e - kY_\epsilon - \varphi \right] + \frac{\eta X_2}{\varphi} \left[ 1 - \frac{V_2}{V} \right]
\]

\[
\times \left[ a \int_0^\infty \mathcal{F}_2(e) Y_v e - \varphi V \right] + \frac{v}{u L_1} \left( 1 - \frac{S_2}{S} \right) \left[ \xi + uYS - \gamma S - \lambda \mathcal{F}_3(e)S_H \right] + \frac{v \beta}{u L_1 L_3 L_4} \left[ \lambda \int_0^\infty \mathcal{F}_4(e) \left( \frac{S}{S_\epsilon} \right) \, d\epsilon \, d\epsilon \right]
\]

\[
+ \frac{\eta X_2 Y_2}{\varphi} \int_0^\infty \mathcal{F}_2(e) \left( Y_\epsilon - \mathcal{F}_2(e) \right) \, d\epsilon \, d\epsilon + \frac{v}{u L_1 L_3} \lambda \int_0^t \mathcal{F}_3(e) \left( \frac{S_H}{S_H} \right) \, d\epsilon \, d\epsilon
\]

By collecting the terms of equation (47), we have the following equation:

\[
\frac{d\varphi_2}{dt} = (1 - X_2/X)\left[ \rho - aX - \eta \int_0^\infty \mathcal{F}_1(e) X_v Y_\epsilon - \frac{1}{L_1} kY_\epsilon + \frac{1}{L_1} kY_2 - \frac{1}{L_1} \nu Y_2 S - \frac{\eta X_2}{\varphi} a \int_0^\infty \mathcal{F}_2(e) Y_\epsilon \, d\epsilon \right]
\]

\[
+ \frac{\eta X_2 Y_2}{\varphi} \int_0^\infty \mathcal{F}_2(e) \left( Y_\epsilon - \mathcal{F}_2(e) \right) \, d\epsilon \, d\epsilon + \frac{v \beta}{u L_1 L_3 L_4} \lambda \int_0^t \mathcal{F}_3(e) \left( \frac{S_H}{S_H} \right) \, d\epsilon \, d\epsilon
\]

\[
+ \frac{\eta X_2 Y_2}{\varphi} \int_0^\infty \mathcal{F}_2(e) \left( Y_\epsilon - \mathcal{F}_2(e) \right) \, d\epsilon \, d\epsilon.
\]
By using the equilibrium conditions for $EP_V$, we obtain the following equation:

$$
\begin{align*}
\frac{d\theta_2}{dt} &= -\frac{\alpha}{X}(X - X_2)^2 + \eta V_2 X_2 \left(1 - \frac{X_2}{X}\right) + \left(\frac{\eta X_2}{\varphi} a L_2 - \frac{1}{L_1}\right) - \frac{1}{L_1} \nu S_2 \left(1 - \frac{X}{S}\right)
+ \frac{\eta X_2 V_2 - \nu Y_2 S_2}{L_1} \int_0^\infty \mathcal{F}_1(e) \frac{X V_2 Y_2}{X Y_2} \, de
\end{align*}
$$

$$(49)$$

Using the equalities given by equation (37) in case of $i = 2$, we get the following equation:

$$
\begin{align*}
\frac{d\theta_2}{dt} &= -\frac{\alpha}{X}(X - X_2)^2 - \frac{\nu Y_2}{L_1 S_2} (S - S_2)^2 \frac{1}{L_1} \nu Y_2 \left(2 - \frac{S_2}{S} - 2 \frac{S}{S}\right) - \frac{\eta X_2 V_2}{L_1} \left[2 - \ln\left(\frac{X_2}{X}\right)\right]
+ \frac{\nu}{L_1} \left(3 S_2 - \frac{\beta \omega}{\lambda L_3 L_4}\right) H + \frac{\eta X_2 V_2}{L_1} \int_0^\infty \mathcal{F}_1(e) \frac{X V_2 Y_2}{X Y_2} \, de - \frac{\eta X_2 V_2}{L_2} \int_0^\infty \mathcal{F}_2(e) \left[\frac{Y V_2}{Y_2 V} - 2 \ln\left(\frac{Y V_2}{Y_2 V}\right)\right] \, de.
\end{align*}
$$

$$\text{(50)}$$

Therefore, equation (51) becomes

$$
\begin{align*}
\frac{d\theta_2}{dt} &= -\frac{\alpha}{X}(X - X_2)^2 - \frac{\nu Y_2}{L_1 S_2} (S - S_2)^2 - \eta X_2 V_2 F\left(\frac{X_2}{X}\right) + \frac{\nu}{L_1} \left(3 S_2 - \frac{\beta \omega}{\lambda L_3 L_4}\right) H
- \frac{\eta X_2 V_2}{L_1} \int_0^\infty \mathcal{F}_1(e) F\left(\frac{X V_2 Y_2}{X Y_2 V}\right) \, de - \frac{\eta X_2 V_2}{L_2} \int_0^\infty \mathcal{F}_2(e) F\left(\frac{Y V_2}{Y_2 V}\right) \, de.
\end{align*}
$$

$$\text{(52)}$$

If $R_3 \leq 1$, then $EP_{V/H}$ does not exist since $H_3 \leq 0$ and $W_3 \leq 0$. This implies that

$$
\begin{align*}
\dot{H}(t) &= \lambda L_3 W - \nu H \leq 0,
\dot{W}(t) &= \lambda L_3 S - \beta W \leq 0.
\end{align*}
$$

$$\text{(53)}$$

Therefore, we get $(\mathcal{S} - \beta \omega / (\lambda L_3 L_4)) H \leq 0$ for all $H, S > 0$. Hence, we have $(\mathcal{S} - \beta \omega / (\lambda L_3 L_4)) \leq 0$, and therefore, $d\theta_2 / dt \leq 0$ for all $X, Y, V, S, W, H > 0$. In addition, $d\theta_2 / dt = 0$ when $X = X_2, S = S_2, H = 0, Y = Y_2$, and $V = V_2$. Solutions of the model (2) that converge to $T_2^*$ is the L.I.S of $T_2^*$: $d\theta_2 / dt = 0$. The set $T_2^*$ has elements with $H(t) = 0$, and thus, $\dot{H}(t) = 0$. Using system (2) last equation, we get the following equation:

$$
\dot{W}(t) = \lambda \int_0^\infty \mathcal{F}_3(e) W(e) \, de.
$$

$$\text{(54)}$$

Yield $W(t) = 0$ for all values of $t$. Therefore, $T_2^* = \{EP_V\}$ and $EP_V$ is G.A.S according to Lyapunov–LaSalle asymptotic stability theorem [40–42].
Theorem 4. If \( R_1 > 1 \) and \( 1 < R_3 \leq 1 + \eta \beta \xi L_2 L_3 \alpha \beta \omega( w \varphi + a \gamma L_2 ) \), then the equilibrium \( \theta_{3}(X, Y, V, S, W, H) \) is G.A.S.

Proof. We consider a Lyapunov function \( \theta_3 \) as follows:

\[
\theta_3 = X_3 F(X_3) + \frac{1}{L_1} Y_3 F(Y_3) + \frac{\eta X_3 V_3}{Y_3} F(V_3) + \frac{v}{uL_1} S_3 F(S_3) + \frac{v}{uL_1 L_3} W_3 F\left(\frac{W_3}{W_3}\right) + \frac{v \beta}{uL_1 L_3 L_4} L_4 F\left(\frac{H_3}{H_3}\right) + \frac{\eta X_3 V_3}{L_1} \int_0^\infty \mathcal{F}_1(e) \int_{t-e}^t F\left(\frac{X(\ell)V(\ell)}{X_3 V_3}\right) d\ell de + \frac{a \eta X_3 V_3}{\varphi} \int_0^\infty \mathcal{F}_2(e) \int_{t-e}^t F\left(\frac{Y(\ell)}{Y_3}\right) d\ell de \\
+ \frac{\beta S_3 H_3}{uL_1 L_3} \int_0^\infty \mathcal{F}_3(e) \int_{t-e}^t F\left(\frac{S(\ell)H(\ell)}{S_3 H_3}\right) d\ell de + \frac{\beta W_3}{uL_1 L_3 L_4} \int_0^\infty \mathcal{F}_4(e) \int_{t-e}^t F\left(\frac{W(\ell)}{W_3}\right) d\ell de.
\]  

By differentiating \( \theta_3 \), we obtain the following equation:

\[
\frac{d\theta_3}{dt} = \left(1 - \frac{X_3}{X} \right) \left[ \rho - \alpha X - \eta XV \right] + \frac{1}{L_1} \left(1 - \frac{Y_3}{Y}\right) \left[ \eta \int_0^\infty \mathcal{F}_1(e) X_3 V_3 de - kY - vYS \right] + \frac{\eta X_3}{\varphi} \left(1 - \frac{V_3}{V}\right) \\
\times \left[ a \int_0^\infty \mathcal{F}_1(e) Y_3 de - \varphi V \right] + \frac{u}{uL_1} \left(1 - \frac{S_3}{S}\right) \left[ \xi + uYS - \gamma S - \beta SH \right] + \frac{v}{uL_1 L_3} \left(1 - \frac{W_3}{W}\right) \\
\times \left[ \beta \int_0^\infty \mathcal{F}_3(e) S_3 H_3 de - \beta W \right] + \frac{\beta W_3}{uL_1 L_3 L_4} \left(1 - \frac{H_3}{H}\right) \left[ \lambda \int_0^\infty \mathcal{F}_4(e) W_3 de - \omega H \right] \\
+ \frac{\eta X_3 V_3}{L_1} \int_0^\infty \mathcal{F}_1(e) \left[ \frac{XV}{X_3 V_3} - \frac{XV}{X_3 V_3} + \ln \left(\frac{XV}{X_3 V_3}\right) \right] de + \frac{a \eta X_3 V_3}{\varphi} \int_0^\infty \mathcal{F}_2(e) \left[ \frac{Y}{Y_3} - \frac{Y}{Y_3} + \ln \left(\frac{Y}{Y_3}\right) \right] de \\
+ \frac{\beta S_3 H_3}{uL_1 L_3} \int_0^\infty \mathcal{F}_3(e) \left[ \frac{SH}{S_3 H_3} - \frac{SH}{S_3 H_3} + \ln \left(\frac{SH}{S_3 H_3}\right) \right] de + \frac{\beta W_3}{uL_1 L_3 L_4} \int_0^\infty \mathcal{F}_4(e) \left[ \frac{W}{W_3} - \frac{W}{W_3} + \ln \left(\frac{W}{W_3}\right) \right] de.
\]  

Collecting terms of equation (56) gives the following equation:

\[
\frac{d\theta_3}{dt} = \left(1 - \frac{X_3}{X} \right) \left[ \rho - \alpha X - \eta XV \right] - \frac{\eta X_3}{L_1} \int_0^\infty \mathcal{F}_1(e) X_3 V_3 \frac{Y_3}{Y_3} de - \frac{1}{L_1} kY - \frac{\eta X_3}{L_1} \int_0^\infty \mathcal{F}_2(e) Y_3 de + \frac{\eta X_3}{\varphi} \int_0^\infty \mathcal{F}_3(e) \frac{V_3}{V_3} \frac{Y_3}{Y_3} de \\
+ \frac{\eta X_3}{uL_1 L_3} \left(1 - \frac{S_3}{S}\right) \left[ \xi + uYS - \gamma S - \beta SH \right] - \frac{\eta X_3}{uL_1 L_3} \int_0^\infty \mathcal{F}_3(e) S_3 H_3 de + \frac{v}{uL_1 L_3} \beta W_3 \\
- \frac{v \beta}{uL_1 L_3 L_4} \int_0^\infty \mathcal{F}_4(e) W_3 \frac{H_3}{H_3} de - \frac{v \beta}{uL_1 L_3 L_4} \frac{H_3}{H_3} - \frac{v \beta}{uL_1 L_3 L_4} \frac{H_3}{H_3} \int_0^\infty \mathcal{F}_4(e) \ln \left(\frac{XV}{XV}\right) de \\
+ \frac{a \eta X_3}{\varphi} \int_0^\infty \mathcal{F}_2(e) \frac{Y_3}{Y_3} \frac{Y_3}{Y_3} de + \frac{v \beta S_3 H_3}{uL_1 L_3} \int_0^\infty \mathcal{F}_3(e) \frac{S_3 H_3}{S_3 H_3} de \\
+ \frac{v \beta W_3}{uL_1 L_3 L_4} \int_0^\infty \mathcal{F}_4(e) \frac{W_3}{W_3} \frac{W_3}{W_3} de.
\]  

By using the equilibrium conditions for \( EP_{VH} \),
we get the following equation:
\[
\frac{d\theta_i}{dt} = \frac{\alpha}{X} (X - X_i)^2 + \eta V_i X_i \left( \frac{1 - X_i}{X} \right) + \left( \frac{\eta X_i}{\varphi} a L_2 - \frac{1}{L_3} \right) \frac{\varphi}{L_3} y - \frac{\eta X_i}{L_3} \int_0^\infty \mathcal{F}_1(e) \frac{X_i V_i X_i Y_i}{X_i Y_i} \, de
\]
\[
+ \eta X_i V_i - \frac{1}{L_3} \frac{\varphi}{L_3} a \int_0^\infty \mathcal{F}_2(e) \frac{Y_i V_i}{Y_i V_i} \, de + \frac{\eta X_i V_i}{v u L_3} (S - S_i)^2 \]
\[
- \frac{1}{L_3} \frac{\varphi}{L_3} a \int_0^\infty \mathcal{F}_3(e) \frac{S_i H_i W_i}{S_i H_i W_i} \, de
\]
\[
+ \frac{v}{u L_3} \mathcal{F}_4(e) \left( \frac{W_i H_i}{W_i H_i} \right) \, de + \frac{v \mathcal{F}_3(e)}{u L_3} \left( \frac{S_i H_i}{S_i H_i} \right) \, de + \frac{v \mathcal{F}_4(e)}{u L_3} \left( \frac{W_i}{W_i} \right) \, de.
\]

Using the equalities given by equations (36) and (37) in case of \( i, j = 3 \), we get the following equation:

\[
\frac{d\theta_i}{dt} = \frac{\alpha}{X} (X - X_i)^2 - \frac{v y}{u L_3} (S - S_i)^2 - \frac{1}{L_3} \frac{\varphi}{L_3} a \int_0^\infty \mathcal{F}_2(e) \frac{Y_i V_i}{Y_i V_i} \, de
\]
\[
- \frac{v \mathcal{F}_3(e)}{u L_3} \left( \frac{S_i H_i}{S_i H_i} \right) \, de + \frac{v \mathcal{F}_4(e)}{u L_3} \left( \frac{W_i}{W_i} \right) \, de
\]
\[
- \frac{\eta X_i V_i}{L_3} a \int_0^\infty \mathcal{F}_2(e) \left( \frac{W_i H_i}{W_i H_i} \right) \, de + \frac{v \mathcal{F}_3(e)}{u L_3} \left( \frac{S_i H_i W_i}{S_i H_i W_i} \right) \, de
\]
\[
- \frac{v \mathcal{F}_4(e)}{u L_3} \left( \frac{W_i H_i}{W_i H_i} \right) \, de.
\]

Therefore, equation (60) becomes the following equation:
Since $1 < R_j \leq 1 + an\tilde{\lambda}\xi L_2 L_4/\beta(\omega + a\eta L_2)$,
then $d\theta_j/dt \leq 0$ for the positive values of $X, Y, V, S, W, \text{and } H$. Moreover, $d\theta_j/dt = 0$
when $X = X_1, Y = Y_1, V = V_1, W = W_1, S = S_1, H = H_1$. The model trajectories
that converge to $T_{ij}$ be the L.I.S of $T_3 = \{(X, Y, V, S, W, H): d\theta_j/dt = 0\}$. Hence, $T_{ij} = \{\text{EPVH}\}$
and $\text{EPVH}$ is G.A.S according to Lyapunov–Lasalle stability theorem. \hfill \Box

All equilibria of model (2) with the existence conditions and global stability
constraints are summarized in Table 1.

5. Numerical Simulations

We execute numerical simulations in this part to enhance the outcomes of
Theorems 1–4. Moreover, the impact of time delays on system dynamical behavior
will be tested. To transform a model with distributed time delay (2) to a
discrete one, we choose a Dirac delta function $D(.)$ as a specific formula
of kernel $g_i(.)$ as follows:

$$
\forall \epsilon_i \in (0, \infty), \quad \epsilon_i = 1, 2, 3, 4.
$$

Then, we get the following equation:

$$
L_j = \int_0^\infty D(\epsilon - \epsilon_i)e^{-m\epsilon_i}d\epsilon = e^{-m\epsilon_i}, \quad j = 1, 2, 3, 4.
$$

Thus, model (2) is reduced as follows:

$$
\begin{align*}
\dot{X}(t) &= \rho - aX(t) - \eta X(t)V(t),
\dot{Y}(t) &= \eta e^{-m\epsilon_1}X(t) - e_1V(t) - kY(t) - \nu Y(t)S(t),
\dot{V}(t) &= a e^{-m\epsilon_2}Y(t) - \varphi V(t),
\dot{S}(t) &= \xi + uY(t)S(t) - \gamma S(t) - 3S(t)H(t),
\dot{W}(t) &= 3e^{-m\epsilon_3}S(t) - e_3H(t) - \beta W(t),
\dot{H}(t) &= \lambda e^{-m\epsilon_4}W(t) - \omega H(t).
\end{align*}
$$

For model (64), the threshold parameters are given by the following equation:

5.1. Stability of Equilibrium Points. During this part, we choose
delay parameters as follows: $\epsilon_1 = 1, \epsilon_2 = 0.8, \epsilon_3 = 1,$ and $\epsilon_4 = 0.8$. Additionally, we select three distinct starting
conditions of the model (18):

Initial-1: $X(\epsilon) = 5, \quad Y(\epsilon) = 0.0001, \quad V(\epsilon) = 0.0002, \quad S(\epsilon) = 100, \quad W(\epsilon) = 5, \quad \text{and } H(\epsilon) = 10,$

Initial-2: $X(\epsilon) = 10, \quad Y(\epsilon) = 0.001, \quad V(\epsilon) = 0.002, \quad S(\epsilon) = 200, \quad W(\epsilon) = 10, \quad \text{and } H(\epsilon) = 15,$

Initial-3: $X(\epsilon) = 15, \quad Y(\epsilon) = 0.002, \quad V(\epsilon) = 0.003, \quad S(\epsilon) = 300, \quad W(\epsilon) = 15, \quad \text{and } H(\epsilon) = 20.$

Here, $\epsilon \in [-\max(\epsilon_1, \epsilon_2, \epsilon_3, \epsilon_4), 0]$ and it is optional to
pick these values. Moreover, the initial conditions are split into three groups to provide global stability for any starting
conditions. To dissolve system (18), we utilize MATLAB solver dde23. Based on equilibrium points $\text{EP}_0, \text{EP}_P, \text{EP}_V,$
and $\text{EP}_\text{PVH}$ global stability explained in Theorems 1–4, the
simulations are divided into four cases. In these instances, we change values of $\eta, \nu, \varphi,$ and $\tilde{\lambda}$ of system (18). Other
parameters values are set and recorded in Table 2. The four scenarios are detailed as follows:

(i) Case 1 (stability of $EP_\varnothing$): we take $\eta = 0.006$, $\nu = 0.01$, $\varphi = 0.3$, and $\mathfrak{S} = 0.0001$. The thresholds in this case are given by $R_1 = 0.322 < 1$ and $R_2 = 0.008 < 1$. In harmony with Theorem 1, the equilibrium $EP_\varnothing = (22.41, 0.0, 1000, 0, 0)$ is G.A.S (Figure 2). This is the best case scenario when the person is free of SARS-CoV-2 and HIV-1 infection.

(ii) Case 2 (stability of $EP_H$): we get $\eta = 0.0006$, $\nu = 0.01$, $\varphi = 0.3$, and $\mathfrak{S} = 0.0016$. This provides us with $R_1 = 5.15 > 1$ and $R_2 = 0.004 < 1$. According to Theorem 2, the equilibrium $EP_H = (22.41, 0.0, 186.478, 13.3211, 27.266)$ is G.A.S (Figure 3). This simulates the situation in which a person has HIV-1 infection with depressed CD4$^+$ T cell levels, but SARS-CoV-2 infection is not present.

(iii) Case 3 (stability of $EP_V$): we select $\eta = 2.9$, $\nu = 0.002$, $\varphi = 0.1$, and $\mathfrak{S} = 0.0001$. This gives $R_2 = 56.997 > 1$ and $R_3 = 0.3535 < 1$. In this case, the system solutions converge globally to equilibrium $EP_V = (0.4285, 0.0087, 0.018, 1094.69, 0, 0)$. This result accords with Theorem 3 (Figure 4). This scenario simulates the case of a person infected with SARS-CoV-2 but not HIV-1 infection.

(iv) Case 4 (stability of $EP_{VH}$): we consider $\eta = 2.9$, $\nu = 0.02$, $\varphi = 0.1$, and $\mathfrak{S} = 0.0016$. This implies that $R_3 = 5.19433 > 1$, $R_4 < 1 + a\eta\mathfrak{S}\lambda L_2 L_3/L_1/\beta\varphi (u\omega + a\eta L_2) = 6.1436$, and $R_4 = 30.1279 > 1$. In agreement with Theorem 4, the equilibrium $EP_{VH} = (0.7155, 0.005, 0.0105, 186.478, 13.48, 27.59)$ is G.A.S (Figure 5). In this case, COVID-19/AIDS coinfection occurs, where an HIV-1 patient gets infected with SARS-CoV-2. CD4$^+$ T cells, which are the main target of HIV-1, are recruited to eliminate SARS-CoV-2 infection from the body. However, if the patient has low CD4$^+$ T cell counts, the clearance of SARS-CoV-2 may not be achieved. This can cause severe infection and death.

5.2 Impact of Time Delays on COVID-19/AIDS Dynamics. Here, we adjust parameters of delay $\epsilon_i, i = 1, 2, \ldots, 4$ and set the parameters values $\eta = 2.9$, $\nu = 0.02$, $\varphi = 0.1$, and $\mathfrak{S} = 0.0016$. Since $R_1, R_2, R_3$, and $R_4$ offered by equation (65) rely on $\epsilon_i, i = 1, 2, \ldots, 4$, varying parameters $\epsilon_i$ will convert stability of the equilibria. We consider the following cases:

- (D.P.S1) $\epsilon_1 = \epsilon_2 = \epsilon_3 = \epsilon_4 = 0$
- (D.P.S2) $\epsilon_1 = 0.3, \epsilon_2 = 0.4, \epsilon_3 = 0.5,$ and $\epsilon_4 = 0.6$
- (D.P.S3) $\epsilon_1 = 10, \epsilon_2 = 11, \epsilon_3 = 12,$ and $\epsilon_4 = 13$

With the above values, we solve model (64) with given initial conditions:

Initial-3: $(X(\epsilon), Y(\epsilon), V(\epsilon), S(\epsilon), W(\epsilon), H(\epsilon)) = (15, 0.002, 0.003, 300, 15, 20)$.

The inclusion of time delays can increase the number of uninfected epithelial and CD4$^+$ T cells while diminish the number of other compartments, as shown in Figure 6. Table 3 shows the values $R_i$ and $R_2$ for selected values of $\epsilon_i, i = 1, 2, \ldots, 4$. Clearly, $R_1$ and $R_2$ decrease when $\epsilon_i$ are increased, and accordingly, the stability of $EP_0$ can be changed. Let us compute the critical value of the time delay that changes the stability of $EP_0$. Without loss of generality, we let the parameters $\epsilon_1 = \epsilon_2 = \epsilon_3$ and $\epsilon_1 = \epsilon_2 = \epsilon_3$, and write $R_1$ and $R_2$ as functions of $\epsilon_{34}$ and $\epsilon_{12}$, respectively, as follows:

$$R_1(\epsilon_{34}) = \frac{\xi \mathfrak{S} \lambda e^{-\mu(\tau_{34}+\tau_4)}\beta\omega}{\beta\varphi},$$

$$R_2(\epsilon_{12}) = \frac{a\nu\xi e^{-\mu(\tau_{12}+\tau_4)}}{a\varphi (\gamma + \nu)}.$$
Figure 2: Continued.
Figure 2: The numerical simulations of model (64) for $\eta = 0.006$, $v = 0.01$, $\varphi = 0.3$, and $\mathcal{I} = 0.0001$ using three different initial conditions sets. Uninfected equilibrium $E_{P_0} = (22.41, 0, 0, 1000, 0, 0)$ is G.A.S. (a) Uninfected epithelial cells. (b) Infected epithelial cells. (c) SARS-CoV-2. (d) Uninfected CD4$^+$ T cells. (e) Infected CD4$^+$ T cells. (f) HIV-1.

Figure 3: Continued.
Figure 3: The numerical simulations of model (64) for $\eta = 0.0006$, $\nu = 0.01$, $\varphi = 0.3$, and $\mathfrak{I} = 0.0016$ using three different initial conditions sets. HIV-1 monoinfection equilibrium $EP_{H} = (22.41, 0, 0, 186.478, 13.3211, 27.266)$ is G.A.S. (a) Uninfected epithelial cells. (b) Infected epithelial cells. (c) SARS-CoV-2. (d) Uninfected CD4$^{+}$ T cells. (e) Infected CD4$^{+}$ T cells. (f) HIV-1.
Figure 4: Continued.
Figure 4: The numerical simulations of model (64) for $\eta = 2.9$, $\nu = 0.002$, $r = 0.1$, and $\mathcal{S} = 0.0001$ using three different initial conditions sets. SARS-CoV-2 monoinfection equilibrium $EP_V = (0.4285, 0.0087, 0.018, 1094.69, 0, 0)$ is G.A.S. (a) Uninfected epithelial cells. (b) Infected epithelial cell. (c) SARS-CoV-2. (d) Uninfected CD4$^+$ T cells. (e) Infected CD4$^+$ T cells. (f) HIV-1.

Figure 5: Continued.
Figure 5: Model (64) numerical simulations for $\eta = 2.9$, $\nu = 0.02$, $\varphi = 0.1$, and $\mathcal{F} = 0.0016$ with three different initial conditions sets. COVID-19/AIDS co-infection equilibrium $EP_{\nu H} = (0.7155, 0.005, 0.0105, 186.478, 13.48, 27.59)$ is G.A.S. (a) Uninfected epithelial cells. (b) Infected epithelial cells. (c) SARS-CoV-2. (d) Uninfected $CD4^+$ T cells. (e) Infected $CD4^+$ T cells. (f) HIV-1.
Figure 6: Model (64) numerical simulations for $\eta = 2.9$, $\nu = 0.02$, $\varphi = 0.1$, and $\mathcal{S} = 0.0016$ with three different sets of delay parameters. (a) Uninfected epithelial cells. (b) Infected epithelial cells. (c) SARS-CoV-2. (d) Uninfected CD4+ T cells. (e) Infected CD4+ T cells. (f) HIV-1.
To compel basic reproduction numbers $R_1$ and $R_2$ to verify $R_1(\epsilon_{34}) \leq 1$ and $R_2(\epsilon_{12}) \leq 1$, respectively, we choose the following equations:

$$\epsilon_{34} \geq \epsilon_{34}^{\min}, \text{ where } \epsilon_{34}^{\min} = \max \left\{ 0, \frac{1}{m_1 + m_4} \ln \frac{\xi \lambda}{\beta y \omega} \right\}.$$

(67)

And

$$\epsilon_{12} \geq \epsilon_{12}^{\min}, \text{ where } \epsilon_{12}^{\min} = \max \left\{ 0, \frac{1}{m_1 + m_2} \ln \frac{a \eta \rho}{\alpha \phi (\gamma k + v_0)} \right\}.$$

(68)

Therefore, if $\epsilon_{34} \geq \epsilon_{34}^{\min}$ and $\epsilon_{12} \geq \epsilon_{12}^{\min}$, then $E_0$ is G.A.S. Computing $\epsilon_{34}$ and $\epsilon_{12}$ gives $\epsilon_{34} = 4.15888$ and $\epsilon_{12} = 6.82823$, respectively. It follows

(i) If $\epsilon_{34} \geq 4.15888$ and $\epsilon_{12} \geq 6.82823$, then $R_1(\epsilon_{34}) \leq 1$, $R_2(\epsilon_{12}) \leq 1$, and $E_0$ is G.A.S.

(ii) If $\epsilon_{34} < 4.15888$ or $\epsilon_{12} < 6.82823$, then $R_1(\epsilon_{34}) > 1$, $R_2(\epsilon_{12}) > 1$, and $E_0$ will lose its stability.

6. Discussion

Coinfection between COVID-19/AIDS has become a serious problem during COVID-19 pandemic. Mathematical modeling represents a main tool in helping experimental studies understand new diseases. We studied a within-host COVID-19/AIDS coinfection model with distributed delays in this paper. The model explores the contacts between healthy epithelial cells, infected epithelial cells, free SARS-CoV-2 particles, uninfected CD4$^+$ T cells, infected CD4$^+$ T cells, and free HIV-1 particles. There are four equilibrium points for the model with the following listed properties:

(a) Uninfected equilibrium $E_{0}$: its existence is permanent and it is G.A.S if $R_1 \leq 1$ and $R_2 \leq 1$. This represents the situation of a person without SARS-CoV-2 or HIV-1 infections.

(b) The HIV-1 monoinfection equilibrium $E_{1}$ exists if $R_1 > 1$, and it is G.A.S if $R_4 \leq 1$. At this point, the person has only HIV-1 infection, but he is not infected by SARS-CoV-2.

(c) SARS-CoV-2 monoinfection equilibrium $E_{P_{V}}$ is appeared when $R_2 > 1$, and if $R_3 \leq 1$, then it is G.A.S. It is the instance of a person who is suffering from SARS-CoV-2 infection only.

(d) COVID-19/AIDS coinfection equilibrium $E_{P_{VI}}$ exists and G.A.S if $R_4 > 1$ and $1 < R_3 \leq 1 + a \eta \xi L_2 L_4 / \beta \omega (u_0 a + a \eta L_2)$. In this case, the patient suffers from COVID-19/AIDS coinfection.

The numerical and theoretical results were found to be in agreement. The time delays increase the concentrations of uninfected epithelial and CD4$^+$ T cells, while they decrease concentrations of free SARS-CoV-2 and HIV-1 particles. Thus, parameters of delay can be examined and used in developing effective treatments for COVID-19/AIDS coinfected patients. Moreover, the model with distributed delays confirmed the effect observed in [20] that low numbers of CD4$^+$ T cells can increase the risk of severe SARS-CoV-2 infection in coinfected patient. Thus, our model can be used to estimate the parameters required to get rid of SARS-CoV-2 in HIV-1 patients. Also, a bifurcation analysis can be executed in order to get a deeper understanding of the stability changes. Furthermore, the work can be developed by finding a better approximation of all parameters in model (2) through fitting with real data. We will keep these points in mind for future projects. [46].

## Data Availability

No underlying data were collected or produced in this study.

## Conflicts of Interest

The authors declare that they have no conflicts of interest.

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