Research Article

HIV/AIDS-Pneumonia Coinfection Model with Treatment at Each Infection Stage: Mathematical Analysis and Numerical Simulation

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In the paper, we have considered a nonlinear compartmental mathematical model *that assesses the effect of treatment on the dynamics of HIV/AIDS and pneumonia coinfection in a human population* at different infection stages. Our model revealed that the disease-free equilibrium points of the HIV/AIDS and pneumonia submodels *are both locally and globally asymptotically stable whenever the* associated basic reproduction numbers ($\mathscr{R}_H$ and $\mathscr{R}_P$) are less than unity. Both the submodel endemic equilibrium points are locally and globally asymptotically stable *whenever the* associated basic reproduction numbers ($\mathscr{R}_P$ and $\mathscr{R}_H$) are greater than unity. *The full HIV/AIDS-pneumonia coinfection model has both locally and globally asymptotically stable disease-free equilibrium points whenever the basic reproduction number of the coinfection model ($\mathscr{R}_{HP}$) is less than unity.* Using standard values of parameters collected from different kinds of literature, we found that the numerical values of the basic reproduction numbers of the HIV/AIDS-only submodel and pneumonia-only submodel are 17 and 7, respectively, and the basic reproduction number of the HIV/AIDS-pneumonia coinfection model is max $\{7, 17\} = 17$. Applying sensitive analysis, we identified the most influential parameters to change the behavior of the solution of the considered coinfection dynamical system are the HIV/AIDS and pneumonia transmission rates β_1 and β_2, respectively. *The coinfection model was numerically simulated to investigate the stability of the coinfection endemic equilibrium point, the impacts of* transmission rates, and *treatment strategies for HIV/AIDS-only, pneumonia-only, and HIV/AIDS-pneumonia coinfected individuals. Finally,* we observed that numerical simulations indicate that treatment against infection at every stage lowers the rate of infection or disease prevalence.

1. Introduction

Infectious diseases are a clinically evident illness, and commonly, they have a great influence on the human population. They are induced by a pathogenic microbial agent such as bacterial, viral, fungal, parasitic, or it can be toxic proteins, called prions. The most common ones are tuberculosis and pneumonia caused by bacteria, HIV, and influenza caused by the virus [1, 2].

HIV/AIDS is a global health issue affecting approximately 70 million people worldwide causing significant morbidity and mortality [3]. Over two-thirds of people living with HIV live in the Sub-Saharan African region [4]. Human immunodeficiency virus (HIV) is a retrovirus virus which attacks and weakens the human body immunity and the central nervous system and if untreated it continues to multiply into the host until it reaches the peak leading into a very serious disease called AIDS, the stage where the symptoms of the disease occur frequently [5–7]. HIV is transmitted through sexual intercourse, needle sharing, and direct contact of blood or other body fluids containing the virus and mother to child during childbirth [1, 8]. According to the center for disease control and prevention (CDC), when individuals get HIV and do not receive treatment known

as antiretroviral therapy (ART), they will typically progress through three stages of disease: acute HIV infection, clinical latency (HIV inactivity or dormancy), and acquired immunodeficiency syndrome (AIDS).

Pneumonia is one of airborne infectious disease caused by caused by bacteria, viruses, fungi, or parasites which attacks the human lungs or alveoli [9–11]. Most of the time pneumonia affects older adults, babies, and people with other diseases or impaired immune systems worldwide. Its most common cause is the Streptococcus pneumoniae, also known as pneumococcus [10, 12, 13]. The basic controlling strategies of pneumonia infection are treatment and vaccination interventions [11].

A coinfection is the infection of a host with two or more different pathogens or different strains of the same pathogens, leading to coexistence of strains (pathogens) at population level [14]. Mathematical modeling of infectious diseases such as coinfection of HIV/AIDS and opportunistic infection is regarded as a fundamental tool in understanding the dynamics and helpful in the decision-making processes regarding intervention strategies and measures required for disease elimination and/or control [15, 16]. Some mathematical models have been used to investigate the transmission dynamics of coinfection of two or more diseases where HIV/AIDS and pneumonia coinfection is among the diseases that infect a large number of individuals worldwide. HIV-infected persons are particularly susceptible to the development of severe pneumococcal disease, even in the setting of combination antiretroviral therapy (ART) [4, 7, 12].

Over the past, mathematical model shave been developed to analyze the population dynamics of HIV/AIDS and pneumonia single infections. Authors in [3] developed and analyzed only a sex-structured population model and studied the HIV infection trends in males and females. Their model assumed that the main mode of HIV transmission is heterosexual. They showed that prevention of HIV infection still remains the most important way of controlling further spread in the community. HIV/AIDS patients under ART treatment are possibly capable of helping the eradication of HIV by convincing their sexual partners of the need to adhere to protection via use of preexposure prophylaxis (PrEP) or any other protection means and ART treatment. Huo and Chen [5] developed and analyzed a mathematical analysis to study the spread of HIV/AIDS with treatment at different stages. Their results show that early treatment for individuals in asymptomatic stage of HIV infection or the pre-AIDS stage is very important. Rahman [2] have analyzed a seven dimension in the living organism HIV model with optimal control of in-host HIV dynamics using different control strategies such as three drug combinations, that is, FIs, RTIs, and PIs to determine the optimal treatment regime. From their results, they recommend that RTIs be used as initial therapy for HIV and FI should be introduced to the patient after the RTIs but should never be used alone.

Mbabazi et al. [13] formulated a mathematical model to study the global stability of pneumococcal pneumonia with awareness, and saturated treatment is presented. Their results showed that the family of decaying curves could help in providing mechanisms to design awareness strategies for containing pneumococcal pneumoniae threshold parameter could be reduced to less than unity if antibiotic resistance awareness and treatment are implemented simultaneously to ensure eradication of pneumococcus bacteria; thus, spread of pneumococcus pneumonia in the population will die out. Tilahun et al. [17] proposed and analyzed a nonlinear mathematical model for the transmission dynamics of pneumonia disease in a population of varying size with optimal control of pneumonia disease and cost-effective strategies. Their cost-effectiveness analysis of the adopted control strategies showed that the combination of prevention and treatment is the most cost-effective intervention strategies to fight the pneumonia pandemic. Ndelwa et al. [18] developed a mathematical model and analyzed treatment and screening strategies on pneumonia infection, and from their numerical results, they concluded screening and treating at the same time can eradicate the pneumonia epidemic from the community.

Nwankwo and Okuonghae [19] formulated and analyzed a mathematical model for the transmission dynamics of syphilis and HIV coinfection in a community to assess the impact of treatment of syphilis on the coendemicity of both diseases in a population where treatment for HIV is not readily available (or easily accessible) but with syphilis treatment sufficiently available. Their syphilis-only model and the full coinfection models undergo the phenomenon of backward bifurcation due to syphilis reinfection after recovery from a previous syphilis infection. They have got the treatment of primary and secondary syphilis in both singly and dually infected individuals; especially with high treatment rates for primary syphilis, this will result in a reduction in the incidence of HIV and its coinfection with syphilis in the community. Kaur et al. [6] formulated and analyzed a mathematical model for HIV/AIDS-TB coinfection with screening and treatment of both HIV and TB infective. Their numerical results suggested that the rates of transmission of both TB and HIV should be decreased, as an increase causes a rise in the number of infective at the equilibrium level.

However, most microbiology, epidemiology, and medical sources like [7, 12] shows the coexistence of HIV/AIDS and pneumonia infection but coinfection mathematical models of HIV/AIDS and pneumonia are rare in literature yet the coexistence between the two infections exist. In our review of literatures, we have got one mathematical model of HIV/AIDS and pneumonia infection in a population and we used it as initial literature reviewed as follows. According to Nthiiri et al. [4] maximum protection against the HIV/AIDS-pneumonia coinfection was analyzed where the maximum protection against HIV/AIDS and the maximum protection against pneumonia was the main concern of their project. In their model, they did not considered maximum protection against the coinfection rather considered maximum protection against single infections. Also, they did not considered treatment on either the submodels or the coinfection. Their analysis found that when protection is high, the number of HIV/AIDS and pneumonia cases is low.

Our paper, therefore, presents a mathematical model describing the transmission dynamics of HIV/AIDS and pneumonia coinfection in a population where treatment

for both HIV/AIDS and pneumonia are available in the community. Basically, the model will be used to evaluate the effect of treatment at every infection stage of either the single infected individuals with HIV/AIDS or pneumonia or HIV/AIDS and pneumonia coinfection as a control strategy for minimizing incidences of coinfections in the target population. In this work, we applied the center for disease control and prevention (CDC) human immunodeficiency virus (HIV) infection stages and the control measure treatment at each stage of the single infections and coinfection model. We have checked this case has never been done before. We discussed the effects of treatment for single infected individuals with either HIV/AIDS or pneumonia and the coinfected patient with HIV/AIDS and pneumonia at each infection stage. The paper is organized as follows. The model is formulated in Section 2 and is analyzed in Section 3. Sensitivity analysis, numerical results, and discussion are carried out in Section 4. Finally, conclusion and limitations of the study are carried out in Sections 5 and 6, respectively.

2. The Mathematical Model

According to the three center for disease control and prevention (CDC) HIV infection stages, we have divide the total population $N(t)$ in to eleven mutually exclusive compartments stated in Table 1, so that $N(t) = S(t) + I_P(t) + H_1(t) + H_2(t) + H_3(t) + C_1(t) + C_2(t) + C_3(t) + T_P(t) + T_H(t) + T(t)$.

We assumed that coinfected individuals can only transmit either pneumonia or HIV but not both infections simultaneously. Individuals acquire HIV infection following effective contacts with people infected with HIV only, (H_1 and H_2 classes), at the rate given by

$$\lambda_H(t) = \beta_1(H_1(t) + \rho H_2(t)), \quad (1)$$

where $\rho > 1$ is the modification parameter accounting for the assumed increased infectivity due to chronic HIV infected than acute HIV-infected one and β_1 is the HIV transmission rate.

Also, individuals acquire pneumonia infection from those in the I_P, C_1, C_2, and C_3 infectious at the rate

$$\lambda_P(t) = \beta_2\left(I_p(t) + \omega_1 C_1(t) + \omega_2 C_2(t) + \omega_3 C_3(t)\right), \quad (2)$$

where $\omega_3 > \omega_2 > \omega_1$ are modification parameters accounting for the assumed increased infectivity due to coinfections and β_2 is the pneumonia transmission rate. The derivation of the model differential equations is given in "Appendix A."

2.1. Flow Chart of the Dynamical System. Here, parameter descriptions in Table 2, state variable descriptions in Table 1 above, and based on the model assumptions that led to the formulation of the model (as stated in"Appendix A"), the flow diagram for the transmission dynamics of HIV/AIDS and pneumonia coinfection is given by Figure 1.

2.2. Dynamical System of HIV/AIDS-Pneumonia Coinfection. Based on Figure 1 above, the dynamical system of HIV/AIDS-pneumonia coinfection becomes

TABLE 1: Descriptions of state variables in model (3).

State variables	Biological meaning
S	Susceptible individuals for both HIV and pneumonia
I_P	Individuals infected with pneumonia
H_1	Acute HIV-infected individuals
H_2	Chronic HIV-infected individuals
H_2	Number of AIDS patients
C_1	Individuals coinfected with acute HIV and pneumonia
C_2	Individuals coinfected with chronic HIV and pneumonia
C_3	Individuals coinfected with AIDS and pneumonia
T_H	Individuals on treatment of HIV/AIDS at different stages
T_P	Individuals on treatment of pneumonia infection
T	Individuals on treatment of coinfections at different stages

$$\frac{dS}{dt} = \Lambda + p\gamma T_P - (\mu + \lambda_H + \lambda_P)S,$$

$$\frac{dI_P}{dt} = \lambda_P S - (\mu + \gamma + \delta_P)I_P,$$

$$\frac{dH_1}{dt} = \lambda_H S - (\mu + \gamma_1 + \alpha_1 + \varphi_1 \lambda_P),$$

$$\frac{dH_2}{dt} = \alpha_1 H_1 - (\mu + \gamma_2 + \alpha_2 + \varphi_2 \lambda_P)H_2,$$

$$\frac{dH_3}{dt} = \alpha_2 H_2 - (\mu + \gamma_3 + \delta_1 + \varphi_3 \lambda_P)H_3,$$

$$\frac{dC_1}{dt} = \varphi_1 \lambda_P H_1 - (\mu + \delta_P + \varepsilon_1 + \alpha_3)C_1, \quad (3)$$

$$\frac{dC_2}{dt} = \varphi_2 \lambda_P H_2 + \alpha_3 C_1 - (\mu + \delta_P + \varepsilon_2 + \alpha_4)C_2,$$

$$\frac{dC_3}{dt} = \varphi_3 \lambda_P H_3 + \alpha_4 C_2 - (\mu + \delta_2 + \varepsilon_3)C_3,$$

$$\frac{dT_P}{dt} = \gamma I_P - (\mu + p\gamma)T_P,$$

$$\frac{dT_H}{dt} = \gamma_1 H_1 + \gamma_2 H_2 + \gamma_3 H_3 - \mu T_H,$$

$$\frac{dT}{dt} = \varepsilon_1 C_1 + \varepsilon_2 C_2 + \varepsilon_3 C_3 - \mu T.$$

With initial conditions,

$$S(0) > 0, I_P(0) \geq 0, H_1(0) \geq 0, H_2(0) \geq 0, H_3(0) \geq 0, C_1(0)$$
$$\geq 0, C_2(0) \geq 0, C_3(0) \geq 0, T_P(0) \geq 0, T_H(0) \geq 0, T(0) \geq 0.$$
$$(4)$$

TABLE 2: Description of parameters in model (3).

| Parameter	Biological meaning	Unit
μ	Natural death rate	Time^{-1}
Λ	Recruitment rate of susceptible individuals	size $*$ Time^{-1}
α_1	The progression rate from acute HIV infection to chronic HIV	Time^{-1}
α_2	The progression rate from chronic HIV infection to AIDS	Time^{-1}
φ_1	The modification parameter accounting that acute HIV-infected individual is more susceptible to pneumonia	Time^{-1}
φ_2	The modification parameter accounting that chronic HIV-infected individual is more susceptible to pneumonia	Time^{-1}
φ_3	The modification parameter accounting that AIDS patient individual is more susceptible to pneumonia infection	Time^{-1}
λ_H	HIV/AIDS force of infection	size^{-1} $*$ Time^{-1}
λ_P	Pneumonia force of infection	size^{-1} $*$ Time^{-1}
α_3	The progression rate from acute HIV and pneumonia coinfection to chronic HIV and pneumonia coinfection	Time^{-1}
α_4	The progression rate from coinfection of chronic HIV-pneumonia to AIDS-pneumonia coinfection	Time^{-1}
δ_P	Pneumonia disease-induced death rate	Time^{-1}
δ_1	AIDS disease-induced death rate	Time^{-1}
δ_2	AIDS and pneumonia diseases-induced death rate	Time^{-1}
γ	Treatment rate for pneumonia-infected individuals	Time^{-1}
p	Portion of pneumonia infected who become susceptible again	Dimensionless
γ_1	Treatment rate for acute HIV-infected individuals	Time^{-1}
γ_2	Treatment rate for chronic HIV-infected individuals	Time^{-1}
γ_3	Treatment rate for AIDS stage-infected individuals	Time^{-1}
ε_1	Treatment rate for acute HIV and pneumonia coinfected	Time^{-1}
ε_2	Treatment rate for chronic HIV and pneumonia coinfection	Time^{-1}
ε_3	Treatment rate for AIDS and pneumonia coinfection	Time^{-1}
β_1	HIV/AIDS transmission rate	size^{-1} $*$ Time^{-1}
β_2	Pneumonia transmission rate	size^{-1} $*$ Time^{-1}
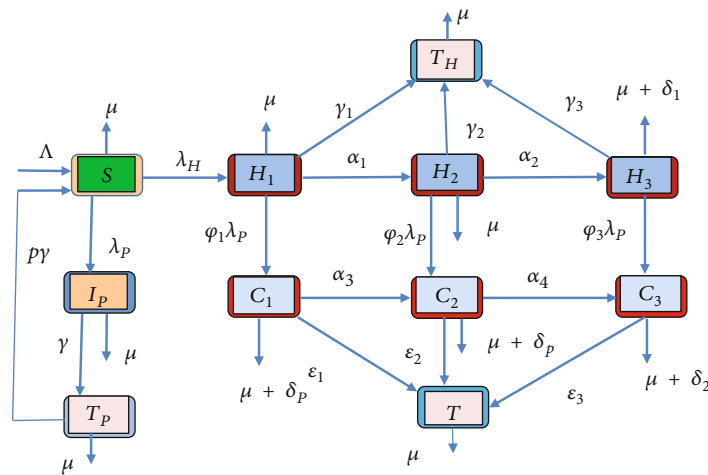
FIGURE 1: Flow chart of the HIV/AIDS-pneumonia coinfection model (3) where λ_H and λ_P are given in (1) and (2), respectively.

The sum of all the differential equations in (3) is given by

$$\frac{dN}{dt} = \Lambda - \mu N - (\delta_P I_P + \delta_1 H_3 + \delta_P C_1 + \delta_P C_2 + \delta_2 C_3). \quad (5)$$

Since model (3) monitors human population, it is assumed that all variables and parameters are nonnegative. The dynamics of model (3) will be analyzed in the following invariant region:

$$\Omega = \left\{ (S, H_1, H_2, H_3, I_P, C_1, C_2, C_3, T_P, T_H, T) \in \mathbb{R}^{11}{}_+, N \leq \frac{\Lambda}{\mu} \right\}. \quad (6)$$

Then, we have proved the positivity and boundedness of solutions of (3) in Ω in "Appendix B."

3. Mathematical Model Analysis

Before we analyzed the HIV/AIDS-pneumonia coinfection model (3), it is useful to gain some background about the HIV-only submodel and pneumonia-only submodel transmission dynamics.

3.1. HIV/AIDS-Only Submodel Analysis. The HIV submodel of (3) (obtained by setting $I_P = C_1 = C_2 = C_3 = T_P = T = 0$) is given by

$$\frac{dS}{dt} = \Lambda - (\mu + \lambda_H)S,$$

$$\frac{dH_1}{dt} = \lambda_H S - (\mu + \gamma_1 + \alpha_1)H_1,$$

$$\frac{dH_2}{dt} = \alpha_1 H_1 - (\mu + \gamma_2 + \alpha_2)H_2, \quad (7)$$

$$\frac{dH_3}{dt} = \alpha_2 H_2 - (\mu + \gamma_3 + \delta_1)H_3,$$

$$\frac{dT_H}{dt} = \gamma_1 H_1 + \gamma_2 H_2 + \gamma_3 H_3 - \mu T_H,$$

where the total population is $N_1(t) = S(t) + H_1(t) + H_2(t) + H_3(t) + T_H(t)$ and the HIV/AIDS force of infection is given by $\lambda_H = \beta_1(H_1 + \rho H_2)$ with initial conditions:

$$S(0) > 0, H_1(0) \geq 0, H_2(0) \geq 0, H_3(0) \geq 0, T_H(0) \geq 0. \quad (8)$$

The sum of all the differential equations in (7) is obtained as

$$\frac{dN_1}{dt} = \Lambda - \mu N_1 - \delta_1 H_3. \quad (9)$$

Consider the region $\Omega_1 = \{ (S, H_1, H_2, H_3, T_H) \in \mathbb{R}^5{}_+, N_1 \leq \Lambda/\mu \}$. It is easy to show that the set Ω_1 is positively invariant and a global attractor of all positive solution of submodel. Hence, it is sufficient to consider the dynamics of model (7) in Ω_1. In this region, the model is considered epidemiologically and mathematically well posed.

3.1.1. Disease-Free Equilibrium Point of the HIV/AIDS-Only Submodel. The disease-free equilibrium point (DFE) of the HIV/AIDS-only submodel (7) is denoted by $E_H^0 = (S^0, H_1^0, H_2^0, H_3^0, T_H^0)$ and obtained by making the right hand side of the system as zero and setting all the infectious classes and treatment class to zero as $H_1 = H_2 = H_3 = T_H = 0$ we have got $S^0 = \Lambda/\mu$ such that $E_H^0 = (\Lambda/\mu, 0, 0, 0, 0)$.

3.1.2. The Basic Reproduction Number of the Submodel. The basic reproduction number of HIV/AIDS-infected individuals denoted by $\mathscr{R}_H$ is defined as the average number of secondary infections produced by a single HIV/AIDS infectious individual introduced in a wholly susceptible population during his or her entire infectious period [14, 20]. This definition is given for the dynamical system that represents the spread of infection in a population. We calculate the basic reproduction number by using the next-generation operator method on the dynamical system (7). The basic reproduction number is obtained by taking the largest (dominant) eigenvalue (spectral radius) of the matrix: $FV^{-1} = [\partial \mathscr{F}_i(E_H^0)/\partial x_j][\partial v_i(E_H^0)/\partial x_j]^{-1}$ [21, 22] where $\mathscr{F}_i$ is the rate of appearance of new infection in compartment i, v_i is the transfer of infections from one compartment i to another, and E_H^0 is the disease-free equilibrium point.

After some calculations we have got $F =$

$$\begin{bmatrix} \beta_1(\Lambda/\mu) & \beta_1\rho(\Lambda/\mu) \\ 0 & 0 \end{bmatrix}, V = \begin{bmatrix} \mu + \gamma_1 + \alpha_1 & 0 \\ -\alpha_1 & \mu + \gamma_2 + \alpha_2 \end{bmatrix},$$

$$V^{-1} = \begin{bmatrix} 1/(\mu + \alpha_1 + \gamma_1) & 0 \\ \alpha_1/((\mu + \alpha_1 + \gamma_1)(\mu + \alpha_2 + \gamma_2)) & 1/(\mu + \alpha_2 + \gamma_2) \end{bmatrix},$$

and $FV^{-1} = [(\beta_1\Lambda/(\mu(\mu + \gamma_1 + \alpha_1))\mu(\mu + \gamma_1 + \alpha_1)) + (\beta_1\rho\Lambda \alpha_1/(\mu(\mu + \gamma_1 + \alpha_1)(\mu + \gamma_2 + \alpha_2))\mu(\mu + \gamma_1 + \alpha_1)(\mu + \gamma_2 + \alpha_2)) \beta_1\rho\Lambda/(\mu(\mu + \gamma_2 + \alpha_2)) 0 0]$.

Then, the spectral radius (reproduction number $\mathscr{R}_H$) of FV^{-1} of the HIV/AIDS subdynamical system (7) is $\rho(FV^{-1}) = \mathscr{R}_H = (\beta_1\Lambda/\mu(\mu + \gamma_1 + \alpha_1)) + (\beta_1\rho\Lambda\alpha_1/(\mu(\mu + \gamma_1 + \alpha_1)(\mu + \gamma_2 + \alpha_2))) = \mathscr{R}_{H_1} + \mathscr{R}_{H_2}$.

3.1.3. Local Stability of the Submodel Disease-Free Equilibrium Point

Theorem 1. *The disease-free equilibrium point E_H^0 of the HIV/AIDSonly submodel is locally asymptotically stable (LAS) if $\mathscr{R}_H < 1$, and unstable if $\mathscr{R}_H < 1$.*

Proof. "Appendix C" $\qquad \square$

Biologically speaking, Theorem 1 implies that HIV can be eliminated from the population when $\mathscr{R}_H < 1$ if the initial sizes of the subpopulation of the submodel are in the region of attraction of E_H^0.

3.1.4. Existence and Stability of Endemic Equilibrium Point of the Submodel. Before investigating the global asymptotic stability of the DFE, it is instructive to determine the number of

endemic equilibrium solutions of the model (7). Let an arbitrary equilibrium point of a HIV/AIDS-only dynamical system (7) is denoted by $E_H^* = (S^*, H_1^*, H_2^*, H_3^*, T_H^*)$. Moreover, let $\lambda_H^* = \beta_1(H_1^* + \rho H_2^*)$ be the associated infection rate ("force of infection") at endemic equilibrium point. After some calculations, we have got $\lambda_H^* = \mu(\mathscr{R}_H - 1)$.

$\Longrightarrow \lambda_H^* > 0$ if $\mathscr{R}_H > 1$ and hence an endemic equilibrium point $E_H^* = (S^*, H_1^*, H_2^*, H_3^*, T_H^*)$ of the HIV/AIDS submodel (7) exist whenever $\mathscr{R}_H > 1$

where $S^* = \Lambda/\mu R_{0H}, H_1^* = \Lambda(\mathscr{R}_H - 1)/d_1 R_{0H}$ with $d_1 = \mu + \gamma_1 + \alpha_1, H_2^* = (\alpha_1 \Lambda(\mathscr{R}_H - 1)/d_1 d_2 R_{0H})$ with $d_2 = \mu + \gamma_2 + \alpha_2, H_3^* = (\alpha_2 \mu(\mathscr{R}_H - 1)/d_3)$ with $d_3 = \mu + \gamma_3 + \delta_1$ and $T_H^* = ((\Lambda(\mathscr{R}_H - 1)\gamma_1/d_1 R_{0H})\Lambda(\mathscr{R}_H - 1)\gamma_1/d_1 R_{0H} + (\alpha_1 \Lambda(\mathscr{R}_H - 1)\gamma_2/d_1 d_2 R_{0H})\alpha_1 \Lambda(\mathscr{R}_H - 1)\gamma_2/d_1 d_2 R_{0H} + (\alpha_2 \mu(\mathscr{R}_H - 1)\gamma_3/d_3))/\mu$ all are positives if $\mathscr{R}_H > 1$.

Theorem 2. *The submodel (7) has a unique endemic equilibrium point if and only if* $\mathscr{R}_H > 1$.

3.1.5. Global Asymptotic Stability (GAS) of the Disease-Free Equilibrium Point

Theorem 3. *The disease-free equilibrium point* $E_H^0 = (\Lambda/\mu, 0, 0, 0, 0)$ *of the dynamical system (7) is globally asymptotically stable if* $\mathscr{R}_H \leq 1$ *otherwise unstable.*

Proof. To show global stability of the DFE applied Lyapunov function method as [13, 23]. □

Let the Lyapunov function $L : R_+^5 \longrightarrow R_+$ is defined by: $L(S, H_1, H_2, H_3, T_H) = a_2 H_1 + a_3 H_2$

where $E_H^0 = (\Lambda/\mu, 0, 0, 0, 0)$ disease-free equilibrium is point where $a_2 = 1/((\mu + \gamma_1 + \alpha_1)(\mu + \gamma_2 + \alpha_2))$ and $a_3 = (1 - \mathscr{R}_{H_1})/(\mu + \gamma_2 + \alpha_2)$ are positive constants. Then, $dL/dt = a_2(dH_1/dt) + a_3(dH_2/dt)$:

$$
\begin{aligned}
\Longrightarrow \frac{dL}{dt} &= a_2(\lambda_H S - (\mu + \gamma_1 + \alpha_1)H_1) + a_3(\alpha_1 H_1 - (\mu + \gamma_2 + \alpha_2)H_2) \\
&\leq \left(a_2 \beta_1 \frac{\Lambda}{\mu} - a_2(\mu + \gamma_1 + \alpha_1) + a_3 \alpha_1\right)H_1 \\
&\quad + \left(a_2 \beta_1 \rho \frac{\Lambda}{\mu} - a_3(\mu + \gamma_2 + \alpha_2)\right)H_2.
\end{aligned}
\tag{10}
$$

Since $S \leq S^0 = \Lambda/\mu$ to find values of a_2 and a_3 take $a_2 \beta_1(\Lambda/\mu) - a_2(\mu + \gamma_1 + \alpha_1) + a_3 \alpha_1 = 0 \Longrightarrow a_3 = (-a_2 \beta_1(\Lambda/\mu)\Lambda/\mu + a_2(\mu + \gamma_1 + \alpha_1))/\alpha_1$; then, we obtained as $dL/dt \leq (a_2 \beta_1 \rho(\Lambda/\mu) + a_2 \beta_1(\Lambda(\mu + \gamma_2 + \alpha_2)/\mu\alpha_1) - (a_2(\mu + \gamma_1 + \alpha_1)(\mu + \gamma_2 + \alpha_2))/\alpha_1)H_2$.

Here, we can take $a_2 = 1/((\mu + \gamma_1 + \alpha_1)(\mu + \gamma_2 + \alpha_2))$ and $a_3 = (1 - \mathscr{R}_{H_1})/(\mu + \gamma_2 + \alpha_2)$ with $\mathscr{R}_{H_1} = \beta_1 \Lambda/(\mu(\mu + \gamma_1 + \alpha_1))$ which is the reproduction number for acute HIV infection (H_1) and a_3 is positive for $\mathscr{R}_{H_1} < 1$.

Then, we obtained

$$
\frac{dL}{dt} \leq \frac{\beta_1 \rho \Lambda}{\mu(\mu + \gamma_1 + \alpha_1)(\mu + \gamma_2 + \alpha_2)} + \frac{\beta_1 \Lambda}{\mu\alpha_1(\mu + \gamma_1 + \alpha_1)} - \frac{1}{\alpha_1} = \alpha_1(\mathscr{R}_H - 1),
\tag{11}
$$

$\Longrightarrow dL/dt \leq \alpha_1(\mathscr{R}_H - 1) \Longrightarrow dL/dt \leq 0$ if $\mathscr{R}_H \leq 1$ and $dL/dt = 0$ if and only if $S = S^0, H_1 = H_1^0, H_2 = H_2^0, H_3 = H_3^0$, and $T_H = T_H^0$. Therefore, the largest compact invariant set in $\{(S, H_1, H_2, H_3, T_H) \in \Omega_1 : dL/dt = 0\}$ is the singleton $\{E_H^0\}$ where E_H^0 is the disease-free equilibrium point of the model (7).

Thus, by LaSalle's invariance principle [24], it implies that the disease-free equilibrium point $E_H^0 = (\Lambda/\mu, 0, 0, 0, 0)$ is globally asymptotically stable in Ω_1 if $\mathscr{R}_H \leq 1$.

3.1.6. Local and Global Stabilities of Endemic Equilibrium Point of the HIV/AIDS-Only Submodel

Theorem 4. *The endemic equilibrium point* $E_H^* = (S^*, H_1^*, H_1^*, H_1^*, H_1^*)$ *is locally asymptotically stable for the basic reproduction number* $\mathscr{R}_H > 1$.

Proof. Appendix D □

Theorem 5. *The endemic equilibrium point* $E_H^* = (S^*, H_1^*, H_2^*, H_3^*, T_H^*)$ *is globally asymptotically stable for the basic reproduction number* $\mathscr{R}_H > 1$, *otherwise unstable.*

Proof. Let the Lyapunov function $V : R_+^5 \longrightarrow R_+$ is defined by

$$
\begin{aligned}
L(S, H_1, H_2, H_3, T_H) &= a_1\left(S - S^* - S^* \ln\left(\frac{S}{S^*}\right)\right) \\
&\quad + a_2\left(H_1 - H_1^* - H_1^* \ln\left(\frac{H_1}{H_1^*}\right)\right) \\
&\quad + a_3\left(H_2 - H_2^* - H_2^* \ln\left(\frac{H_2}{H_2^*}\right)\right).
\end{aligned}
\tag{12}
$$

□

Here, we have $dL/dt = a_1((dS/dt) - ((S^*/S)(dS/dt))) + a_2((dH_1/dt) - ((H_1^*/H_1)H_1^*/H_1 dH_1/dt)) + a_3((dH_2/dt) - ((H_2^*/H_2)H_2^*/H_2(dH_2/dt)))$

$$
\begin{aligned}
\Longrightarrow \frac{dL}{dt} &= a_1\left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} + a_2\left(1 - \frac{H_1^*}{H_1}\right)\frac{dH_1}{dt} \\
&\quad + a_3\left(1 - \frac{H_2^*}{H_2}\right)\frac{dH_2}{dt}.
\end{aligned}
\tag{13}
$$

At the endemic equilibrium point $E_H^* = (S^*, H_1^*, H_2^*, H_3^*, T_H^*)$, we obtain from the system (7): $\Lambda = (\mu + \lambda_H^*)S^*, \lambda_H^* S^* = (\mu + \gamma_1 + \alpha_1)H_1^*$ and $\alpha_1 H_1^* = (\mu + \gamma_2 + \alpha_2)H_2^*$

$\Longrightarrow \Lambda = (\mu + \lambda_H^*)S^*, (\mu + \gamma_1 + \alpha_1)H_1 = \lambda_H^* S^*(H_1/H_1^*)$ and $(\mu + \gamma_2 + \alpha_2)H_2 = \alpha_1 H_1^*(H_2/H_2^*)$.

Then, we obtained

$$\frac{dL}{dt} = a_1\left(1 - \frac{S^*}{S}\right)\left((\mu + \lambda_H{}^*)S^* - (\mu + \lambda_H)S\right)$$

$$+ a_2\left(1 - \frac{H_1^*}{H_1}\right)\left(\lambda_H S - \lambda_H^* S^* \frac{H_1}{H_1^*}\right)$$

$$+ a_3\left(1 - \frac{H_2^*}{H_2}\right)\left(\alpha_1 H_1 - \alpha_1 H_1{}^* \frac{H_2}{H_2^*}\right)$$

$$\implies \frac{dL}{dt} = -a_1\mu\frac{(S - S^*)^2}{S}$$

$$+ [a_1 + a_2]\beta_1 H_1^* S^* + [a_1 + a_2]\beta_1\rho H_2{}^* S^*$$

$$+ [a_2 - a_1]\beta_1 H_1 S + [a_2 - a_1]\beta_1\rho H_2 S \qquad (14)$$

$$- a_1\frac{S^{*2}}{S}\beta_1 H_1{}^* - a_1\frac{S^{*2}}{S}\beta_1\rho H_2^*$$

$$+ \left[a_3\alpha_1 - a_2\beta_1\rho S^* \frac{H_2^*}{H_1^*}\right]H_1$$

$$+ \left[a_1\beta_1\rho S^* - a_3\alpha_1 \frac{H_1^*}{H_2^*}\right]H_2$$

$$+ a_1\beta_1 H_1 S^* - a_2\beta_1 H_1 S^* - a_2\beta_1 H_1^* S$$

$$- a_2\beta_1\rho H_2 S \frac{H_1^*}{H_1} - a_3\alpha_1 H_1 \frac{H_2^*}{H_2} + a_3\alpha_1 H_1^*.$$

Choose a_1, a_2, and a_3 such that the expressions in the brackets vanish and take $a_1 = a_2$ and solve for a_3, by making expressions in the closed bracket zero, we obtain as $a_3\alpha_1 - a_2\beta_1\rho S^*(H_2^*/H_1^*) = 0$

$$\implies a_3 = a_1\beta_1\rho S^* \frac{H_2^*}{\alpha_1 H_1^*} \text{ for } a_1 = a_2,$$

$$\implies \frac{dL}{dt} = -a_1\mu\frac{(S - S^*)^2}{S} + 2a_1\beta_1 H_1^* S^* + 2a_1\beta_1\rho H_2^* S^*$$

$$- a_1\frac{S^{*2}}{S}\beta_1 H_1^* - a_1\frac{S^{*2}}{S}\beta_1\rho H_2{}^* + a_1\beta_1 H_1 S^*$$

$$- a_1\beta_1 H_1 S^* - a_1\beta_1 H_1^* S - a_1\beta_1\rho H_2 S \frac{H_1^*}{H_1}$$

$$- a_1\beta_1\rho S^* \frac{H_2^{*2}}{H_1^*}\frac{H_1}{H_2} + a_1\beta_1\rho S^* H_2^*.$$

$$(15)$$

Grouping some terms in the expression above yields

$$\frac{dL}{dt} = -a_1\mu\frac{(S - S^*)^2}{S} + a_1\beta_1 H_1^* S^*\left[2 - \frac{S}{S^*} - \frac{S^*}{S}\right]$$

$$+ a_1\beta_1\rho H_2^* S^*\left[3 - \frac{S^*}{S} - \frac{S H_2 H_1^*}{S^* H_2^* H_1} - \frac{H_2^* H_1}{H_2 H_1^*}\right]. \qquad (16)$$

Using the arithmetic-geometric mean inequality property, we have $2 - (S/S^*) - (S^*/S) \leq 0$ and $3 - (S^*/S) - (SH_2 H_1^*/S^* H_2^* H_1) - (H_2^* H_1/H_2 H_1^*) \leq 0$.

Hence, we conclude that $dL/dt \leq 0$, and hence, L is the representative Lyapunov function.

Furthermore, $dL/dt = 0$ if and only if $(S, H_1, H_2, H_3, T_H) = (S^*, H_1^*, H_2^*, H_3^*, T_H^*)$, and the largest invariant subset contained in the set $E_\infty = \{(S, H_1, H_2, H_3, T_H) \in \Omega_1 : dL/dt = 0\}$ is the set contained only the endemic equilibrium point:

$$E_H^* = (S^*, H_1^*, H_2^*, H_3^*, T_H^*). \qquad (17)$$

Therefore, we conclude by LaSalle's invariance principle [24] that $E_H^* = (S^*, H_1^*, H_2^*, H_3^*, T_H^*)$ is globally asymptotically stable (GAS) if $\mathcal{R}_H > 1$.

3.2. Pneumonia Submodel Analysis. We have the pneumonia submodel of (3) when $H_1 = H_2 = H_3 = C_1 = C_2 = C_3 = T_H = T = 0$, which is given by

$$\frac{dS}{dt} = \Lambda + p\gamma T_P - (\mu + \lambda_P)S,$$

$$\frac{dI_P}{dt} = \lambda_P S - (\mu + \gamma + \delta_P)I_P, \qquad (18)$$

$$\frac{dT_P}{dt} = \gamma I_P - (\mu + p\gamma)T_P,$$

where the total population is $N_2(t) = S(t) + I_p(t) + T_p(t)$ and the pneumonia force of infection is given by $\lambda_P = \beta_2 I_P$ with initial conditions

$$S(0) > 0, I_P(0) \geq 0, T_P(0) \geq 0. \qquad (19)$$

The sum of all the differential equations in (18) above is obtained as

$$\frac{dN_2}{dt} = \Lambda - \mu N - \delta_P I_p. \qquad (20)$$

Consider the region $\Omega_2 = \{(S, I_P, T_P) \in \mathbb{R}^3_+, N_2 \leq \Lambda/\mu\}$. It is easy to show that the set Ω_2 is positively invariant and a global attractor of all positive solution of submodel (18). Hence, it is sufficient to consider the dynamics of model (18) in Ω_1. In this region, the model is epidemiologically and mathematically well posed.

3.2.1. Disease-Free Equilibrium Point (DFE) of the Pneumonia-Only Submodel. The disease-free equilibrium point (DFE) of the system (18) is obtained by making the right hand side of the system as zero and setting all the infectious classes and treatment class to zero as $I_P = T_P = 0$ we have got $S^0 = \Lambda/\mu$ such that $E_P^0 = (\Lambda/\mu, 0, 0)$.

3.2.2. The Reproduction Number of the Pneumonia-Only Submodel. We calculate the basic reproduction number denoted by $\mathcal{R}_P$ using the van den Driesch and Warmouth next-generation matrix approach from [22]. The basic reproduction number is obtained by taking the largest (dominant) eigenvalue (spectral radius) of the matrix: $FV^{-1} = [\partial \mathcal{F}_i(E_P^0)/\partial x_j][\partial v_i(E_P^0)/\partial x_j]^{-1}$, where $\mathcal{F}_i$ is the rate of appearance of new infection in compartment i, v_i is the transfer of infections from one compartment i to another, and E_P^0 is

the disease-free equilibrium point. The reproduction number $\mathscr{R}_P$ of the pneumonia-only dynamical system (18) is obtained by rearrange the differential equation of the dynamical system (18) above in terms of $dX_i/dt = \mathscr{F}_i - v_i = \mathscr{F}_i - (v_i^- - v_i^+)$. Then, after some calculations, we have got $FV^{-1} = \begin{bmatrix} \beta_2\Lambda/(\mu(\mu+\gamma+\delta_P)) & 0 \\ 0 & 0 \end{bmatrix}$ and the spectral radius (reproduction number $\mathscr{R}_P$) of FV^{-1} of the pneumonia subdynamical system (18) is $\mathscr{R}_P = \beta_2\Lambda/(\mu(\mu+\gamma+\delta_P))$.

3.2.3. Local Stability of the Submodel Disease-Free Equilibrium Point

Theorem 6. *The disease-free equilibrium point of the pneumonia-only submodel is locally asymptotically stable (LAS) if $\mathscr{R}_P < 1$, otherwise unstable.*

Proof. The local stability of the disease-free equilibrium of the system (18) can be studied from its Jacobian matrix at the disease-free equilibrium point $E_P^0 = (S^0, I_P{}^0, T_P{}^0) = (\Lambda/\mu, 0, 0)$ and Routh-Hurwitz stability criteria. Then, the Jacobian matrix of the dynamical system (18) at $E_P^0 = (\Lambda/\mu, 0, 0)$ is given by

$$J\left(E_P^0\right) = \begin{pmatrix} -\mu & \dfrac{\beta_2\Lambda}{\mu} & p\gamma \\ 0 & -(\mu+\gamma+\delta_P) & 0 \\ 0 & \gamma & -(\mu+p\gamma) \end{pmatrix}. \quad (21)$$

$\square$

Then, the characteristic equation of the above Jacobian matrix is given by

$$\begin{vmatrix} -\mu-\lambda & \dfrac{\beta_2\Lambda}{\mu} & p\gamma \\ 0 & \left[\dfrac{\beta_2\Lambda}{\mu} - (\mu+\gamma+\delta_P)\right]-\lambda & 0 \\ 0 & \gamma & -(\mu+p\gamma)-\lambda \end{vmatrix} = 0,$$

$$\Longrightarrow (-\mu-\lambda)\left(\left[\dfrac{\beta_2\Lambda}{\mu}-(\mu+\gamma+\delta_P)\right]-\lambda\right)(-(\mu+p\gamma)-\lambda) = 0,$$

$$\Longrightarrow \lambda_1 = -\mu, \lambda_2 = \dfrac{\beta_2\Lambda}{\mu}-(\mu+\gamma+\delta_P), \lambda_3 = -(\mu+p\gamma),$$

$$\Longrightarrow \lambda_2 = \dfrac{\beta_2\Lambda}{\mu}-(\mu+\gamma+\delta_P) = (\mu+\gamma+\delta_P)\left[\dfrac{\beta_2\Lambda}{\mu(\mu+\gamma+\delta_P)}-1\right],$$

$$\Longrightarrow \lambda_2 = (\mu+\gamma+\delta_P)[\mathscr{R}_P-1] < 0 \text{ if } \mathscr{R}_P < 1. \quad (22)$$

Therefore, since all the eigenvalues of the characteristics polynomial of the system (18) are negative for $\mathscr{R}_P < 1$, the disease-free equilibrium point of the system (18) is locally asymptotically stable.

3.2.4. Existence of Endemic Equilibrium Point of the Pneumonia Submodel.

Before investigating the global asymptotic stability of the DFE, it is instructive to determine the number of endemic equilibrium solutions of the model (18). Let an arbitrary endemic equilibrium point of the pneumonia-only dynamical system (18) be denoted by $E_P^* = (S^*, I_P{}^*, T_P{}^*)$. Now, after some calculations, we have got a unique endemic equilibrium point $E_P^* = (S^*, I_P^*, T_P^*)$ where $S^* = (d_2\Lambda m_2 + d_2\Lambda m_3 D_1[\mathscr{R}_P-1] + D_2 p\gamma[\mathscr{R}_P-1])/((d_2 m_2 + d_2 m_3 D_1[\mathscr{R}_P-1])(\mu+D_1[\mathscr{R}_P-1])) > 0$, $I_P^* = (d_2\Lambda D_1[\mathscr{R}_P-1])/(m_2+m_3 D_1[\mathscr{R}_P-1]) > 0$ and $T_P^* = (D_2[\mathscr{R}_P-1])/(d_2 m_2 + d_2 m_3 D_1[\mathscr{R}_P-1]) > 0$ if $\mathscr{R}_P > 1$ where $D_1 = ((\mu+\gamma+\delta_P)(\mu+p\gamma)\mu)/((\mu+\gamma+\delta_P)(\mu+p\gamma)-p\gamma^2)$ and $D_2 = d_2\gamma\Lambda D_1$.

Theorem 7. *The model (18) has a unique endemic equilibrium point if $\mathscr{R}_P > 1$.*

3.2.5. Globally Asymptotically Stability (GAS) of the Disease-Free Equilibrium Point

Theorem 8. *The disease-free equilibrium point $E_P^0 = (S^0, I_P{}^0, T_P{}^0) = (\Lambda/\mu, 0, 0)$ of the dynamical system (18) is globally asymptotically stable if $\mathscr{R}_P \leq 1$, otherwise unstable.*

Proof. Let the Lyapunov function $L : R_+^3 \longrightarrow R_+$ is defined by: $L(S, I_P, T_P) = a_1 I_P$
where $E_P^0 = (S^0, I_P{}^0, T_P{}^0) = (\Lambda/\mu, 0, 0)$ is disease-free equilibrium point where $a_1 = 1$ is a positive constant. $\square$

Then $dL/dt = a_1(dI_P/dt)$

$$\Longrightarrow \dfrac{dL}{dt} = a_1 \dfrac{dI_P}{dt} = a_1(\lambda_P S - (\mu+\gamma+\delta_P)I_P)$$
$$= a_1[\beta_2 I_P S - (\mu+\gamma+\delta_P)I_P]$$

$$\Longrightarrow \dfrac{dL}{dt} = a_1[\beta_2 S - (\mu+\gamma+\delta_P)]I_P$$
$$\leq a_1\left[\beta_2 S^0 - (\mu+\gamma+\delta_P)\right]I_P \text{ since } S \leq S^0 = \dfrac{\Lambda}{\mu}, \quad (23)$$

$$\Longrightarrow \dfrac{dL}{dt} \leq a_1\left[\beta_2\dfrac{\Lambda}{\mu} - (\mu+\gamma+\delta_P)\right]I_P$$
$$= a_1(\mu+\gamma+\delta_P)\left[\beta_2\dfrac{\Lambda}{\mu(\mu+\gamma+\delta_P)} - 1\right],$$

$$\Longrightarrow \dfrac{dL}{dt} \leq a_1(\mu+\gamma+\delta_P)[\mathscr{R}_P - 1]$$
$$= (\mu+\gamma+\delta_P)[\mathscr{R}_P - 1], \quad (24)$$

$\Longrightarrow dL/dt \leq 0$ if $\mathscr{R}_P \leq 1$. Hence, the disease-free equilibrium point $E_P^0 = (\Lambda/\mu, 0, 0)$ is globally asymptotically stable whenever $\mathscr{R}_P \leq 1$.

3.2.6. Local and Global Stabilities of Endemic Equilibrium Point.

The following theorem studies the local stability of

the endemic equilibrium. The result is obtained by means of the Routh-Hurwitz stability criteria.

Theorem 9. *The endemic equilibrium point of the system (18) is locally asymptotically stable if $\mathscr{R}_P > 1$.*

Proof. To show the local stability of the endemic equilibrium point, we use the method of the Jacobian matrix and Routh Hurwitz stability criteria. Then, the Jacobian matrix of the dynamical system (18) at the endemic equilibrium point $E_P^* = S^*, I_P^*, T_P^*$ where $S^* = (d_2\Lambda m_2 + d_2\Lambda m_3 D_1[\mathscr{R}_P - 1] + D_2 p\gamma[\mathscr{R}_P - 1])/((d_2 m_2 + d_2 m_3 D_1[\mathscr{R}_P - 1])(\mu + D_1[\mathscr{R}_P - 1]))$, $I_P^* = (d_2\Lambda D_1[\mathscr{R}_P - 1])/(m_2 + m_3 D_1[\mathscr{R}_P - 1])$ and $T_P^* = (D_2[\mathscr{R}_P - 1])/(d_2 m_2 + d_2 m_3 D_1[\mathscr{R}_P - 1])$, $D_2 = d_2\gamma\Lambda D_1$ for the system (18) whenever $\mathscr{R}_P > 1$ is given by

$$J(E_P^*) = \begin{pmatrix} -\mu - \beta_2 I_P^* & -\beta_2 S^* & p\gamma \\ \beta_2 I_P^* & \beta_2 S^* - d_1 & 0 \\ 0 & \gamma & -d_2 \end{pmatrix}, \qquad (25)$$

$$\Longrightarrow J(E_P^*) = \begin{pmatrix} A_1 & A_4 & p\gamma \\ A_5 & A_2 & 0 \\ 0 & \gamma & A_3 \end{pmatrix} \qquad \text{where } A_1 = -\mu - \beta_2 I_P^*$$

, $A_2 = \beta_2 S^* - d_1, A_3 = -d_2, A_4 = -\beta_2 S^*$, and $A_5 = \beta_2 I_P^*$. $\qquad\square$

Then, the characteristic equation of the above Jacobian matrix is given by

$$\begin{vmatrix} A_1 - \lambda & A_4 & p\gamma \\ A_5 & A_2 - \lambda & 0 \\ 0 & \gamma & A_3 - \lambda \end{vmatrix} = 0, \qquad (26)$$

$\Longrightarrow a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0$ where $a_3 = 1, a_2 = -(A_1 + A_2 + A_3), a_1 = (A_1 A_2 + A_1 A_3 + A_2 A_3 - A_4 A_5)$, and $a_0 = -A_1 A_2 A_3 + A_3 A_4 A_5 - p\gamma^2 A_5$.

Here, we apply the necessary condition of Routh-Hurwitz stability criteria since $a_3 = 1$ is positive in sign, indicating all a_2, a_1, and a_0 should be positive.

Hence, $a_2 = -(A_1 + A_2 + A_3) = \mu + \beta_2 I_P^* - \beta_2 S^* + d_1 + d_2$

$$\Longrightarrow a_2 = \frac{\mu(d_2 m_2 + d_2 m_3 D_1[\mathscr{R}_P - 1])(\mu + D_1[\mathscr{R}_P - 1]) + \beta_2 D_2[\mathscr{R}_P - 1]((\mu + D_1[\mathscr{R}_P - 1])) - \beta_2 d_2\Lambda m_2 - \beta_2 d_2\Lambda m_3 D_1[\mathscr{R}_P - 1] - \beta_2 D_2 p\gamma[\mathscr{R}_P - 1]}{(d_2 m_2 + d_2 m_3 D_1[\mathscr{R}_P - 1])(\mu + D_1[\mathscr{R}_P - 1])}$$

$$+ (d_1 + d_2)(d_2 m_2 + d_2 m_3 D_1[\mathscr{R}_P - 1])(\mu + D_1[\mathscr{R}_P - 1]) > 0 \text{ if } \mathscr{R}_P > 1,$$

$$a_1 = A_1 A_2 + A_1 A_3 + A_2 A_3 - A_4 A_5 = \left(\mu + \beta_2 \frac{d_2\Lambda D_1[\mathscr{R}_P - 1]}{m_2 + m_3 D_1[\mathscr{R}_P - 1]}\right)\left(\beta_2 \frac{d_2\Lambda m_2 + d_2\Lambda m_3 D_1[\mathscr{R}_P - 1] + D_2 p\gamma[\mathscr{R}_P - 1]}{(d_2 m_2 + d_2 m_3 D_1[\mathscr{R}_P - 1])(\mu + D_1[\mathscr{R}_P - 1])} - d_1\right)$$

$$+ d_2\left(\mu + \beta_2 \frac{d_2\Lambda D_1[\mathscr{R}_P - 1]}{m_2 + m_3 D_1[\mathscr{R}_P - 1]}\right) + \beta_2 \frac{d_2\Lambda m_2 + d_2\Lambda m_3 D_1[\mathscr{R}_P - 1] + D_2 p\gamma[\mathscr{R}_P - 1]}{(d_2 m_2 + d_2 m_3 D_1[\mathscr{R}_P - 1])(\mu + D_1[\mathscr{R}_P - 1])}\left(\beta_2 \frac{d_2\Lambda D_1[\mathscr{R}_P - 1]}{m_2 + m_3 D_1[\mathscr{R}_P - 1]}\right) > 0 \text{ if } \mathscr{R}_P > 1,$$

$$(27)$$

$$a_0 = -A_1 A_2 A_3 + A_3 A_4 A_5 - p\gamma^2 A_5$$
$$= (\mu + \beta_2 I_P^*)(\beta_2 S^* - d_1)d_2 + d_2\beta_2 S^*(\beta_2 I_P^*) - \beta_2 I_P^* p\gamma^2$$
$$= \left(\mu + \beta_2 \frac{d_2\Lambda D_1[\mathscr{R}_P - 1]}{m_2 + m_3 D_1[\mathscr{R}_P - 1]}\right)$$
$$\cdot \left(\beta_2 \frac{d_2\Lambda m_2 + d_2\Lambda m_3 D_1[\mathscr{R}_P - 1] + D_2 p\gamma[\mathscr{R}_P - 1]}{(d_2 m_2 + d_2 m_3 D_1[\mathscr{R}_P - 1])(\mu + D_1[\mathscr{R}_P - 1])} - d_1\right)d_2$$
$$+ d_2\beta_2 \frac{d_2\Lambda m_2 + d_2\Lambda m_3 D_1[\mathscr{R}_P - 1] + D_2 p\gamma[\mathscr{R}_P - 1]}{(d_2 m_2 + d_2 m_3 D_1[\mathscr{R}_P - 1])(\mu + D_1[\mathscr{R}_P - 1])}$$
$$\cdot \left(\beta_2 \frac{d_2\Lambda D_1[\mathscr{R}_P - 1]}{m_2 + m_3 D_1[\mathscr{R}_P - 1]}\right)$$
$$- \beta_2 \frac{d_2\Lambda D_1[\mathscr{R}_P - 1]}{m_2 + m_3 D_1[\mathscr{R}_P - 1]}p\gamma^2 > 0 \text{ if } \mathscr{R}_P > 1. \qquad (28)$$

Thus, all the coefficients of the characteristic's polynomial a_0, a_1, a_2, and a_3 have the same sign. Then, applying the Routh-Hurwitz criteria to determine the sign of the root without calculating their values of the root of the characteristics equation $a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0$ and we have got the Routh-Hurwitz array that has no sign change, indicating the roots of the characteristics equation of the dynamical system (18) are negatives. Hence, the endemic equilibrium point $E_P^* = (S^*, I_P^*, T_P^*)$ of the dynamical system (18) is locally asymptotically stable.

3.2.7. Global Stability of Endemic Equilibrium of Pneumonia Submodel

Theorem 10. *If $\mathscr{R}_P > 1$ the unique endemic equilibrium point $E_P^* = (S^*, I_P^*, T_P^*)$ is globally asymptotically stable in the interior of Ω_2.*

Proof. Let us take the Lyapunov function $L : \mathbb{R}_+^3 \longrightarrow \mathbb{R}$ by $L(S, I_P, T_P) = A_1[(S - S^*) + (I_P - I_P^*) + (T_P - T_P^*)]^2 + A_2[I_P - I_P^* - I_P^* \ln (I_P/I_P^*)] + A_3[T_P - T_P^*]^2$ for positive constants $A_1 = 1/2$, $A_2 = (\delta_P + 2\mu)/\beta_2$ and $A_3 = (\delta_P + 2\mu)/2\gamma$. Then, L is C^1 on the interior of Ω_2, $E_P^* = (S^*, I_P^*, T_P^*)$ is the global minimum of L on Ω_2, and $L(S^*, I_P^*, T_P^*) = 0$.

Then, the time derivative of L is given by

$$
\begin{aligned}
\frac{dL}{dt} &= 2A_1[(S - S^*) + (I_P - I_P^*) + (T_P - T_P^*)] \frac{d(S + I_P + T_P)}{dt} \\
&\quad + A_2 \left[1 - \frac{I_P^*}{I_P} \right] \frac{dI_P}{dt} + 2A_3[T_P - T_P^*] \frac{dT_P}{dt}, \\
\Longrightarrow \frac{dL}{dt} &= [(S - S^*) + (I - I_P^*) + (T_P - T_P^*)] \left\{ \frac{d(S + I_P + T_P)}{dt} \right\} \\
&\quad + \frac{\delta_P + 2\mu}{\beta_2} \left[1 - \frac{I_P^*}{I_P} \right] \frac{dI_P}{dt} + \frac{\delta_P + 2\mu}{\gamma} [T_P - T_P^*] \frac{dT_P}{dt}.
\end{aligned}
\tag{29}
$$

Using expressions at the endemic equilibrium,

$$
\begin{aligned}
\Lambda &= \mu(S^* + I_P^* + T_P^*) + \delta_P I_P^*, \\
\beta_2 S^* &= \mu + \gamma + \delta_P, \\
0 &= (\mu + p\gamma) T_P^* - \gamma I_P^*,
\end{aligned}
\tag{30}
$$

we have got

$$
\begin{aligned}
\frac{dL}{dt} &= [(S - S^*) + (I - I_P^*) + (T_P - T_P^*)] \{ \mu(S^* + I_P^* + T_P^*) \\
&\quad + \delta_P I_P^* - \mu(S + I_P + T_P) - \delta_P I_P \} \\
&\quad + \frac{\delta_P + 2\mu}{\beta_2} \left[\frac{I_P - I_P^*}{I_P} \right] \{ \beta_2 I_P S - \beta_2 k_2 S^* I_P \} \\
&\quad + \frac{\delta_P + 2\mu}{\gamma} [T_P - T_P^*] \{ \gamma I_P - (\mu + p\gamma) T_P \\
&\quad + (\mu + p\gamma) \mathrm{T}_P^* - \gamma I_P^* \}, \\
\Longrightarrow \frac{dL}{dt} &= -\mu[(S - S^*) + (T_P - T_P^*)]^2 - (\delta_P + \mu)(I_P - I_P^*)^2 \\
&\quad - (\delta_P + 2\mu)(\mu + p\gamma)(T_P - T_P^*)^2 \leq 0.
\end{aligned}
\tag{31}
$$

Hence, dL/dt is negative. Note that $dL/dt = 0$ if and only if $S = S^*$, $I_P = I_P^*$ and $T_P = T_P^*$. Therefore, the largest compact invariant set in $\{(S, I_P, T_P) \in \Omega_2 : dL/dt = 0\}$ is the singleton $\{E_P^*\}$ set where E_P^* the endemic equilibrium point. Then, by LaSalle's invariant principle, [24] then implies that E_P^* is globally asymptotically stable in the interior of Ω_2 if $\mathscr{R}_P > 1$.

3.3. Analysis of the HIV-Pneumonia Coinfection Model (3).

In this section, we analyze the main dynamical system (3). Epidemiologically, the HIV/AIDS-pneumonia coinfection dynamical system (3) will have four equilibrium points, namely, HIV/AIDS-pneumonia coinfection model disease-free equilibrium point E_{HP}^0, HIV/AIDS-only endemic equilibrium point E_H^*, pneumonia-only endemic equilibrium point E_P^*, and the endemic equilibrium point of the coexistence of HIV-pneumonia denoted by E_{HP}^*.

3.3.1. Disease-Free Equilibrium Point of the HIV-Pneumonia Coinfection Model.

The disease-free equilibrium point $E_{HP}^0 = (S^0, H_1^0, H_2^0, H_3^0, C_1^0, C_2^0, C_3^0, T_P^0, T_H^0, T^0)$ of the system (3) is obtained by setting all the infectious classes and treatment classes to zero and making the right hand side of the system as zero. Then, the disease-free equilibrium of the HIV/AIDS coinfection model is $E_{HP}^0 = (\Lambda/\mu, 0, 0, 0, 0, 0, 0, 0, 0, 0)$.

3.3.2. The Basic Reproduction Number $(\mathscr{R}_{HP})$ of the HIV-Pneumonia Coinfection Model.

The basic reproduction number of the dynamical system (3) by applying the next-generation operator method [21, 22] is the largest (dominant) eigenvalue (spectral radius) of the matrix: $FV^{-1} = [\partial \mathscr{F}_i(E_{HP}^0)/\partial x_j][\partial \nu_i(E_{HP}^0)/\partial x_j]^{-1}$, where $\mathscr{F}_i$ is the rate of appearance of new infection in compartment i, ν_i is the transfer of infections from one compartment i to another, and E_{HP}^0 is the disease-free equilibrium point. Here, we obtained the following matrices:

$$
F = \begin{bmatrix}
\beta_2 \dfrac{\Lambda}{\mu} & 0 & 0 & 0 & \beta_2 \omega_1 \dfrac{\Lambda}{\mu} & \beta_2 \omega_2 \dfrac{\Lambda}{\mu} & \beta_2 \omega_3 \dfrac{\Lambda}{\mu} & 0 & 0 & 0 \\
0 & \beta_1 \dfrac{\Lambda}{\mu} & \beta_1 \rho \dfrac{\Lambda}{\mu} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0
\end{bmatrix},
$$

$$
V = \begin{bmatrix}
m_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & m_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & -\alpha_1 & m_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & -\alpha_2 & m_4 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & m_5 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & -\alpha_3 & m_6 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & -\alpha_4 & m_7 & 0 & 0 & 0 \\
-\gamma & 0 & 0 & 0 & 0 & 0 & 0 & m_8 & 0 & 0 \\
0 & -\gamma_1 & -\gamma_2 & -\gamma_3 & 0 & 0 & 0 & 0 & \mu & 0 \\
0 & 0 & 0 & 0 & -\varepsilon_1 & -\varepsilon_2 & -\varepsilon_3 & 0 & 0 & \mu
\end{bmatrix}.
\tag{32}
$$

Then, using Mathematica, we have got the spectral radius (dominant eigenvalue) of the matrix FV^{-1} is $\mathcal{R}_{HP}$ = max $\{(\Lambda\beta_1/\mu(\mu + \gamma_1 + \alpha_1)) + (\Lambda\rho\alpha_1\beta_1/(\mu(\mu + \gamma_1 + \alpha_1)(\mu + \gamma_2 + \alpha_2))), \Lambda\beta_2/\mu(\mu + \gamma + \delta_P)\}$ where $\mathcal{R}_P = \Lambda\beta_2/\mu(\mu + \gamma + \delta_P)$ is the basic reproduction number for pneumonia infection and $\mathcal{R}_H = (\Lambda\beta_1/\mu(\mu + \gamma_1 + \alpha_1)) + (\Lambda\rho\alpha_1\beta_1/(\mu(\mu + \gamma_1 + \alpha_1)(\mu + \gamma_2 + \alpha_2)))$ is the basic reproduction number for HIV/AIDS infection, and hence, $\mathcal{R}_{HP}$ = max $\{\mathcal{R}_P, \mathcal{R}_H\}$ is the basic reproduction number of the HIV and pneumonia co-infection.

3.3.3. Local Stability of the Disease-Free Equilibrium Point

Theorem 11. *The disease-free equilibrium point of the model (3) above is locally asymptotically stable if $\mathcal{R}_{HP} < 1$ and unstable if $\mathcal{R}_{HP} > 1$.*

Proof. The Jacobian matrix $J(E^0_{HP})$ of the model at E^0_{HP} is given by

$$J(E^0_{HP}) = \begin{bmatrix} -\mu & \beta_2\dfrac{\Lambda}{\mu} & -\beta_1\dfrac{\Lambda}{\mu} & -\beta_1\rho\dfrac{\Lambda}{\mu} & 0 & -\beta_2\omega_1\dfrac{\Lambda}{\mu} & -\beta_2\omega_2\dfrac{\Lambda}{\mu} & -\beta_2\omega_3\dfrac{\Lambda}{\mu} & p\gamma & 0 & 0 \\ 0 & Z_2 & 0 & 0 & 0 & \beta_2\omega_1\dfrac{\Lambda}{\mu} & \beta_2\omega_2\dfrac{\Lambda}{\mu} & \beta_2\omega_3\dfrac{\Lambda}{\mu} & 0 & 0 & 0 \\ 0 & 0 & Z_3 & \beta_1\rho\dfrac{\Lambda}{\mu} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_1 & Z_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha_2 & Z_5 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & Z_6 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \alpha_3 & Z_7 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha_4 & Z_8 & 0 & 0 & 0 \\ \gamma & 0 & 0 & 0 & 0 & 0 & 0 & 0 & Z_9 & 0 & 0 \\ 0 & 0 & \gamma_1 & \gamma_2 & \gamma_3 & 0 & 0 & 0 & 0 & -\mu & 0 \\ 0 & 0 & 0 & 0 & 0 & \varepsilon_1 & \varepsilon_2 & \varepsilon_3 & 0 & 0 & -\mu \end{bmatrix}. \qquad (33)$$

The eigenvalues of the above Jacobian matrix are $\lambda_1 = \lambda_2 = \lambda_3 = -\mu < 0$ or $\lambda_4 = Z_2 = \beta_2(\Lambda/\mu) - (\mu + \gamma + \delta_P)$ or $\lambda_5 = Z_5 = -(\mu + \gamma_3 + \alpha_3) < 0$ or $\lambda_6 = Z_6 = -(\mu + \delta_P + \varepsilon_1 + \alpha_3) < 0$ or $\lambda_7 = Z_7 = -(\mu + \delta_P + \varepsilon_2 + \alpha_4) < 0$ or $\lambda_8 = Z_8 = -(\mu + \delta_2 + \varepsilon_3) < 0$ or $\lambda_9 = Z_9 = -(\mu + p\gamma) < 0$ or $a_2\lambda^2 + a_1\lambda + a_0 = 0$ for $a_2 = 1, a_1 = -Z_3 - Z_4$ and $a_0 = Z_3Z_4 - \beta_1\rho\alpha_1(\Lambda/\mu)$.

Here, $\lambda_4 = Z_2 = \beta_2(\Lambda/\mu) - (\mu + \gamma + \delta_P) = (\mu + \gamma + \delta_P)[(\beta_2\Lambda/\mu(\mu + \gamma + \delta_P)) - 1] = (\mu + \gamma + \delta_P)[\mathcal{R}_P - 1] \Longrightarrow \lambda_4 = (\mu + \gamma + \delta_P)[\mathcal{R}_P - 1] < 0$ if $\mathcal{R}_P < 1$.

To check the remaining eigenvalues are negatives for the quadratic equation $a_2\lambda^2 + a_1\lambda + a_0 = 0$, we can apply Routh-Hurwitz stability criteria since $a_2 = 1 > 0$ both a_1 and a_0 should be positives.

Now, $a_1 = (\mu + \gamma_2 + \alpha_2) + (\mu + \gamma_1 + \alpha_1)[1 - \mathcal{R}_{H_1}] \Longrightarrow a_1 > 0$ if $\mathcal{R}_{H_1} < 1$ and $a_0 = (\mu + \gamma_2 + \alpha_2)(\mu + \gamma_1 + \alpha_1) [1 - (\beta_1\rho\alpha_1\Lambda/(\mu(\mu + \gamma_2 + \alpha_2)(\mu + \gamma_1 + \alpha_1)))] \Longrightarrow a_0 = (\mu + \gamma_2 + \alpha_2)(\mu + \gamma_1 + \alpha_1)[1 - \mathcal{R}_{H_2}) > 0$ if $\mathcal{R}_{H_2} < 1$.

Hence, the last two eigenvalues are negatives if $\mathcal{R}_H < 1$ and thus all the eigenvalues are negatives if $\mathcal{R}_{HP}$ = max $\{\mathcal{R}_H, \mathcal{R}_P\} < 1$. Thus, since all the eigenvalues are negatives, the disease-free equilibrium point of the HIV/AIDS-pneumonia coinfection dynamical system (3) above is locally asymptotically stable if

$$\mathcal{R}_{HP} = \max \{\mathcal{R}_H, \mathcal{R}_P\} < 1. \qquad (34)$$

□

3.3.4. Existence of Endemic Equilibrium Point for the Full HIV/AIDS-Pneumonia Coinfection Model.

The endemic equilibrium point (EEP) of the model (3) above is denoted by $E^*_{HP} = (S^*, I^*_P, H^*_1, H^*_2, H^*_3, C^*_1, C^*_2, C^*_3, T^*_P, T^*_H, T^*)$ which occurs when the disease persist in the community. From the analysis of the HIV-only submodel (7) and the pneumonia-only submodel (18), we have shown that there is no endemic equilibrium point if $\mathcal{R}_H < 1$ and $\mathcal{R}_P < 1$ implies that there is no endemic equilibrium point if $\mathcal{R}_{HP} < 1$ for the coinfection model. The endemic equilibrium of the system (3) above can be obtained as

$$S^* = \frac{\Lambda + p\gamma T_P^*}{(\mu + \lambda_H^* + \lambda_P^*)}, I_P^* = \frac{\lambda_P^* S^*}{(\mu + \gamma + \delta_P)}, H_1^*$$

$$= \frac{\lambda_H^* S^*}{(\mu + \gamma_1 + \alpha_1 + \varphi_1 \lambda_P^*)}, H_2^* = \frac{\alpha_1 H_1^*}{(\mu + \gamma_2 + \alpha_2 + \varphi_2 \lambda_P^*)}, H_3^*$$

$$= \frac{\alpha_2 H_2^*}{(\mu + \gamma_3 + \delta_1 + \varphi_3 \lambda_P^*)}, C_1^* = \frac{\varphi_1 \lambda_P^* H_1^*}{(\mu + \delta_P + \varepsilon_1 + \alpha_3)}, C_2^*$$

$$= \frac{\varphi_2 \lambda_P^* H_2^* + \alpha_3 C_1^*}{(\mu + \delta_P + \varepsilon_2 + \alpha_4)}, C_3^* = \frac{\varphi_3 \lambda_P^* H_3^* + \alpha_4 C_2^*}{(\mu + \delta_2 + \varepsilon_3)}$$

$$\tag{35}$$

$$T_P^* = \frac{\gamma I_P^*}{(\mu + p\gamma)}, T_H^* = \frac{\gamma_1 H_1^* + \gamma_2 H_2^* + \gamma_3 H_3^*}{\mu}, T = \frac{\varepsilon_1 C_1^* + \varepsilon_2 C_2^* + \varepsilon_3 C_3^*}{\mu}.$$

$$\tag{36}$$

Summary of endemic equilibrium point. The explicit computation of the endemic equilibrium of the full model (3) given in equations (35) and (36) in terms of model parameters is difficult analytically; however, the model (3) above endemic equilibrium $E_{HP}^* = (S^*, I_P^*, H_1^*, H_2^*, H_3^*, C_1^*, C_2^*, C_3^*, T_P^*, T_H^*, T^*)$ exists if $\mathscr{R}_H > 1$ and $\mathscr{R}_P > 1$, i.e., $\mathscr{R}_{HP} > 1$. We will give an explanation of E_3^* in our numerical simulations.

4. Sensitivity Analysis of the Model Parameters and Numerical Simulations

Results of sensitivity analysis and the numerical simulation are given in this section, and the set of parameters used are given in Table 3 below. The full model (3) is now simulated, using the parameter estimates in Table 3 (unless otherwise stated), to assess the potential impact of treatment strategies against pneumonia and HIV/AIDS coinfection, as follows.

4.1. Sensitivity Analysis of the Model Parameters. Sensitivity is performed to identify the most dominant parameters for the spreading out as well as control of infection in the community. To go through sensitivity analysis, we follow the technique described in [9, 17]. Results of sensitivity analysis are given in this section, and the set of parameters used are given in Table 3 below where N_0 is the total number of the assumed initial population under consideration in the model numerical simulation part.

We are able to know how important each parameter is to the spread of the disease through sensitivity indices of $\mathscr{R}_{HP}$ to all different parameters. We carried out the sensitivity analysis to determine the model robustness to parameter values.

The normalized forward sensitivity index of a variable $\mathscr{R}_{HP}$ that depends differentiably on a parameter p is defined as $\text{SI}(p) = (\partial \mathscr{R}_{HP}/\partial p) * (p/\mathscr{R}_{HP})$ [9].

These sensitivity indices allow us to determine the relative importance of different parameters in pneumonia and HIV/AIDS transmission and prevalence. The most sensitive parameter has the magnitude of the sensitivity index larger than that of all other parameters. We can calculate the sensitivity index in terms of $\mathscr{R}_H$ and $\mathscr{R}_P$ since

TABLE 3: Standard parameter values from literatures.

Parameter	Nominal value	Source
Λ	$0.0413*N_0$	Estimated
μ	0.01	Estimated
$\alpha_1, \alpha_2, \alpha_3, \alpha_4$	0.498, 0.08, 0.2885, and 0.3105, respectively	[5], Assumed
$\varphi_1, \varphi_2, \varphi_3$	6, 7, and 10, respectively	Assumed
δ_P	0.135	[18]
δ_1	0.333	Assumed
δ_2	0.42	Assumed
γ	0.2	[25]
$\gamma_1, \gamma_2, \gamma_3$	0.2, 0.15, and 0.13, respectively	[5, 25], Assumed
$\varepsilon_1, \varepsilon_2, \varepsilon_3$	0.20, 0.201, and 0.230	[25], Assumed
β_1	Variable	[26]
β_2	Variable	Assumed
$\rho, \omega_1, \omega_2, \omega_3$	1.2, 1, 1, and 1	Assumed

TABLE 4: Sensitivity indices of $\mathscr{R}_{HP} = \mathscr{R}_H$.

Sensitivity index	Value
$\text{SI}(\Lambda) = \left[\dfrac{\partial \mathscr{R}_H}{\partial \Lambda}\right] \times \left[\dfrac{\Lambda}{\mathscr{R}_H}\right]$	+1
$\text{SI}(\beta_1) = [\partial \mathscr{R}_H/\partial \beta_1] \times [\beta_1/\mathscr{R}_H]$	+1
$\text{SI}(\rho) = \left[\dfrac{\partial \mathscr{R}_H}{\partial \rho}\right] \times \left[\dfrac{\rho}{\mathscr{R}_H}\right]$	+0.6134
$\text{SI}(\alpha_1) = [\partial \mathscr{R}_H/\partial \alpha_1] \times [\alpha_1/\mathscr{R}_H]$	-0.0639
$\text{SI}(\mu) = \left[\dfrac{\partial \mathscr{R}_H}{\partial \mu}\right] \times \left[\dfrac{\mu}{\mathscr{R}_H}\right]$	-0.3150
$\text{SI}(\gamma_1) = [\partial \mathscr{R}_H/\partial \gamma_1] \times [\gamma_1/\mathscr{R}_H]$	-0.1371
$\text{SI}(\gamma_2) = [\partial \mathscr{R}_H/\partial \gamma_2] \times [\gamma_2/\mathscr{R}_H]$	-0.0264
$\text{SI}(\alpha_2) = [\partial \mathscr{R}_H/\partial \alpha_2] \times [\alpha_2/\mathscr{R}_H]$	-0.0141

TABLE 5: Sensitivity indices of $\mathscr{R}_{HP} = \mathscr{R}_P$.

Sensitivity index	Value
$\text{SI}(\Lambda) = \left[\dfrac{\partial \mathscr{R}_P}{\partial \Lambda}\right] \times \left[\dfrac{\Lambda}{\mathscr{R}_P}\right]$	+1
$\text{SI}(\beta_2) = [\partial \mathscr{R}_P/\partial \beta_2] \times [\beta_2/\mathscr{R}_P]]$	+1
$\text{SI}(\mu) = \left[\dfrac{\partial \mathscr{R}_P}{\partial \mu}\right] \times \left[\dfrac{\mu}{\mathscr{R}_P}\right]$	-0.4421
$\text{SI}(\gamma) = [\partial \mathscr{R}_P/\partial \gamma] \times [\gamma/\mathscr{R}_P]$	-0.6559
$\text{SI}(\delta_P) = [\partial \mathscr{R}_P/\partial \delta_P] \times [\delta_P/\mathscr{R}_P]$	-0.3852

$$\mathscr{R}_{HP} = \max\{\mathscr{R}_H, \mathscr{R}_P\}. \tag{37}$$

The sensitivity indices in terms of $\mathscr{R}_{HP} = \mathscr{R}_H = (\Lambda\beta_1/\mu(\mu + \gamma_1 + \alpha_1)) + (\Lambda\rho\alpha_1\beta_1/(\mu(\mu + \gamma_1 + \alpha_1)(\mu + \gamma_2 + \alpha_2)))$ and the sensitivity indices in terms of $\mathscr{R}_{HP} = \mathscr{R}_P = \Lambda\beta_2/\mu(\mu + \gamma + \delta_P)$ are given in Tables 4 and 5, respectively.
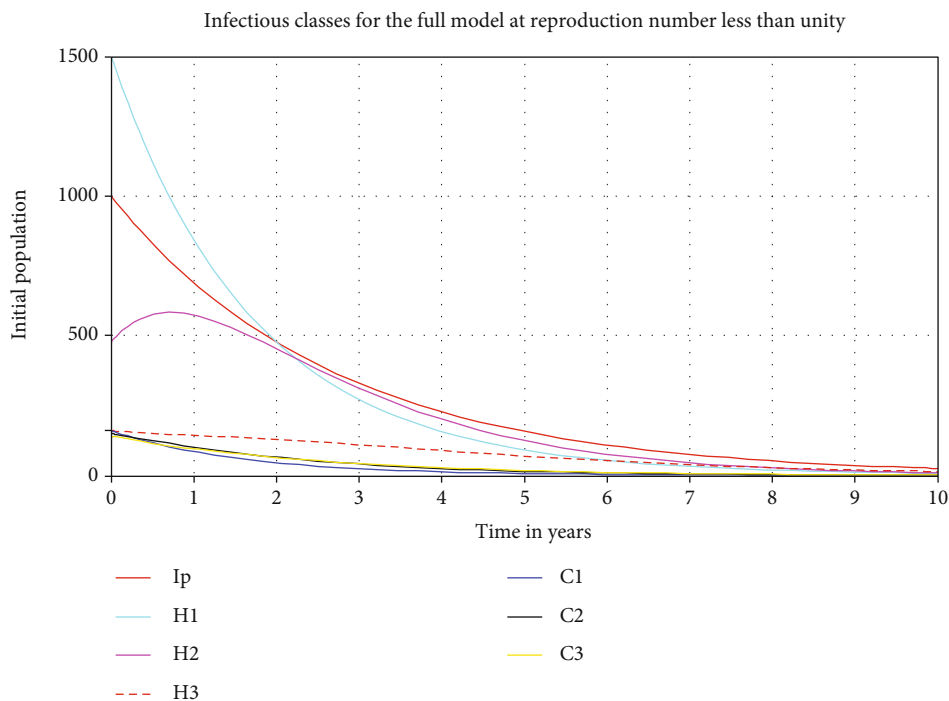
FIGURE 2: Behavior of infected classes of the full HIV/AIDS-pneumonia coinfection model at $\mathcal{R}_H < 1$ and $\mathcal{R}_P < 1$ where $\beta_1 =0.00000289$ and $\beta_2 =0.0000079$.
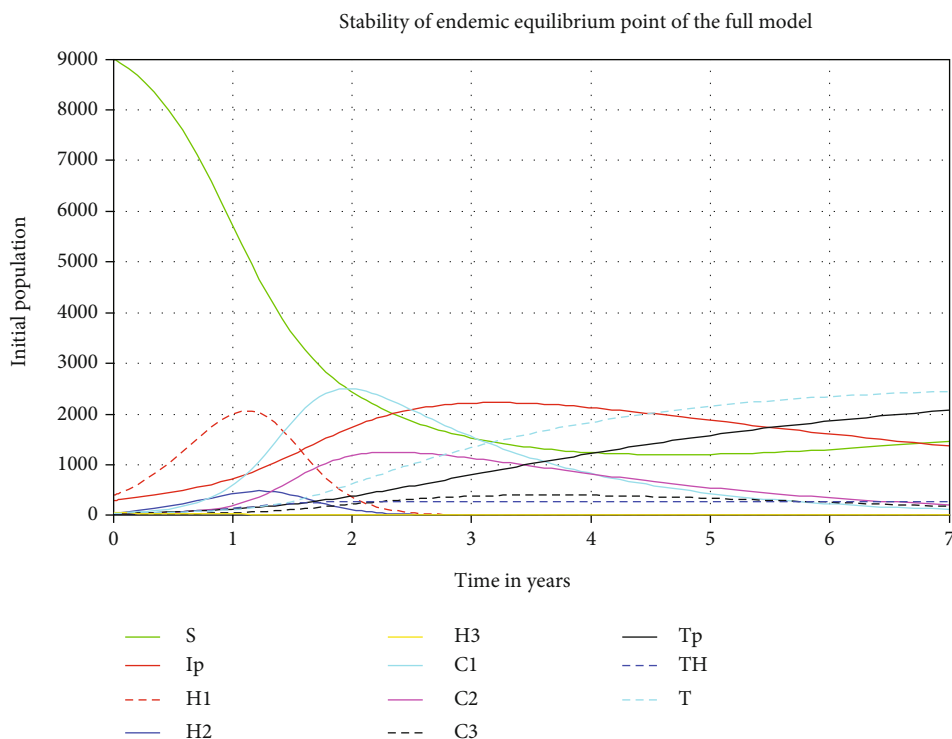

FIGURE 3: The stability analysis of the endemic equilibrium of the HIV/AIDS-pneumonia coinfection model at $\beta_1 =0.00029$ and $\beta_2 =0.00079$.

Using the parameter values in Table 3, the sensitivity indexes are computed in Tables 4 and 5 as above.

4.2. Numerical Results and Discussion. Using the data provided in Table 3 and different initial conditions, the numerical results are generated for the dynamics of model (3) using MATLAB numerical solver (ode45) which generate results for ode45. The ode45 was chosen because of its computational speed and increased level of accuracy for solving non-stiff ordinary differential equations and we simulated the

Effect of treatment on pneumonia reproduction number (Rp)
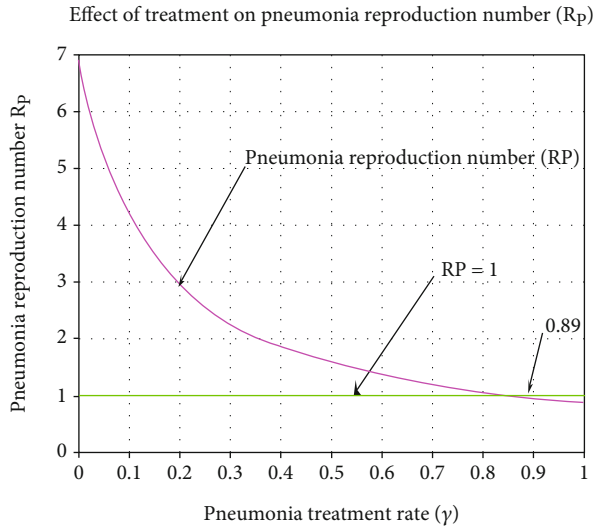
FIGURE 4: Pneumonia reproduction number. Simulation at variable treatment rate (γ).
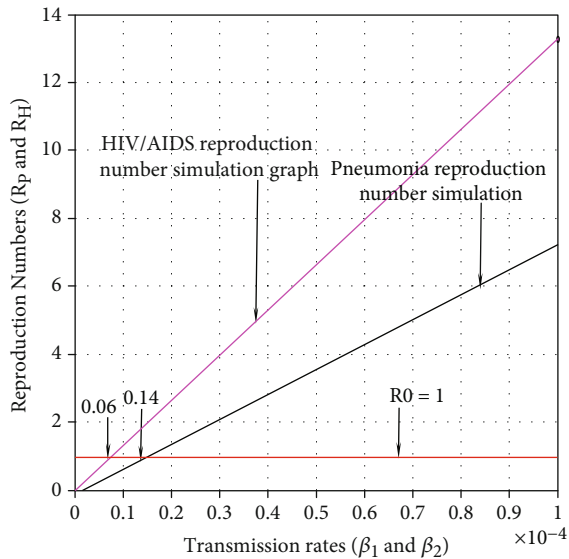

FIGURE 5: Graphs of $\mathcal{R}_H$ and $\mathcal{R}_P$ at different values of β_1 and β_2, respectively.

model using different initial conditions for different values of β_1 and β_2. It shows the effect of β_1 on $\mathcal{R}_H$ and the effect of β_2 on $\mathcal{R}_P$. Here, we have done numerical simulation for the HIV/AIDS-pneumonia coinfection dynamical system (3) for the purpose of verifying some of the analytical results and results which are difficult to be done analytically. This is done by using a set of parameter values in Table 3 above whose sources are mainly calculated from literatures in order to have realistic simulation results. In this analysis, we also discussed the effect of parameters change on the basic reproduction number graphically using MATLAB ode45 software.

4.2.1. HIV/AIDS-Pneumonia Coinfection Model Simulation for Various Thresholds. Here, simulations are carried out to monitor the dynamics of the HIV/AIDS-pneumonia coin-
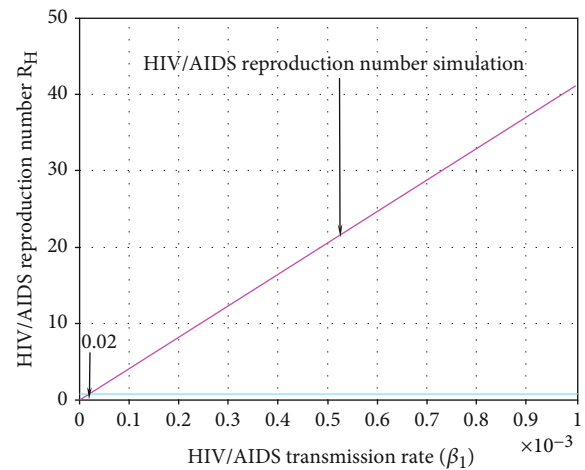

FIGURE 6: Pneumonia reproduction number. Simulation at variable transmission rate β_2.

FIGURE 7: HIV/AIDS reproduction number simulation at variable transmission rate (β_1).

fection model (3) for different values of the associated reproduction thresholds $\mathcal{R}_H$ and $\mathcal{R}_P$ and we plot the graphs of the time verses infected population for different values of reproduction numbers.

Figure 2 above was plotted using MATLAB ode45 program under consideration of the basic reproduction numbers being less than a unity and shows the behavior of the infectious classes of the HIV/AIDS-pneumonia coinfection model (3) at $\mathcal{R}_H < 1$ and $\mathcal{R}_P < 1$ (i.e., $\mathcal{R}_{HP} < 1$). The simulation given in Figure 2 above shows that each of infectious classes $(I_P, H_1, H_2, H_3, C_1, C_2, C_3)$ is converging to the disease-free equilibrium point of the model. This was obtained when $\mathcal{R}_P = 0.1445$ at $\beta_2 = 0.0000079$ and $\mathcal{R}_H = 0.1374$ at $\beta_1 = 0.00000289$ with all other parameters are given as in Table 3. This indicates that the disease-free equilibrium point of the full HIV/AIDS-pneumonia coinfection model is globally asymptotically stable.
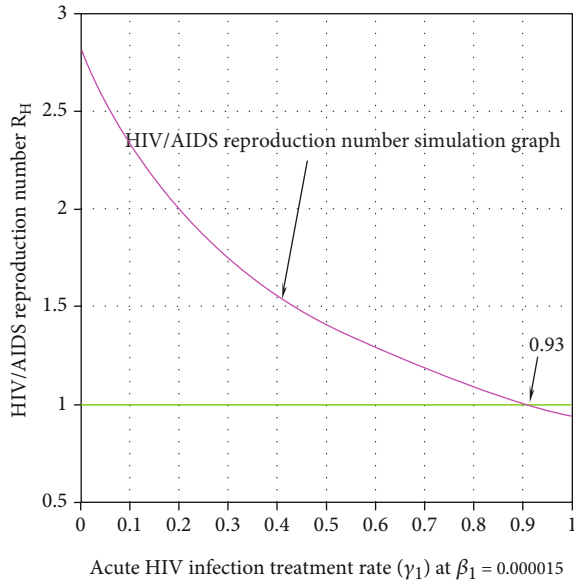
Figure 8: HIV reproduction number simulation at variable acute infection treatment rate (γ_1).
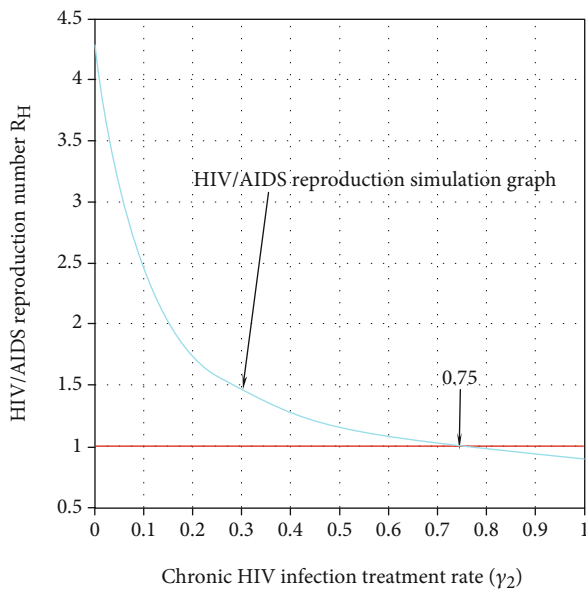

Figure 9: HIV/AIDS reproduction number simulation at variable chronic Infection treatment rate.

Figure 3 was plotted using the values $\mathscr{R}_P = 7$ at $\beta_2 = 0.00079$ and $\mathscr{R}_H = 17$ at $\beta_1 = 0.00029$ with all other parameters are given in Table 3. The simulation in Figure 3 above shows the stability of the HIV/AIDS-pneumonia coinfection model endemic equilibrium point. From Figure 3 above, in the long run, the convergence of the solutions is observed at the values greater than 7 years. The plot shows that the HIV/AIDS-pneumonia coinfection model (3) endemic equilibrium point is locally asymptotically stable.

4.2.2. Reproduction Number Simulations with Variable Parameter Values. Here, we have taken parameters from Table 3, and we have done numerical simulations of repro-
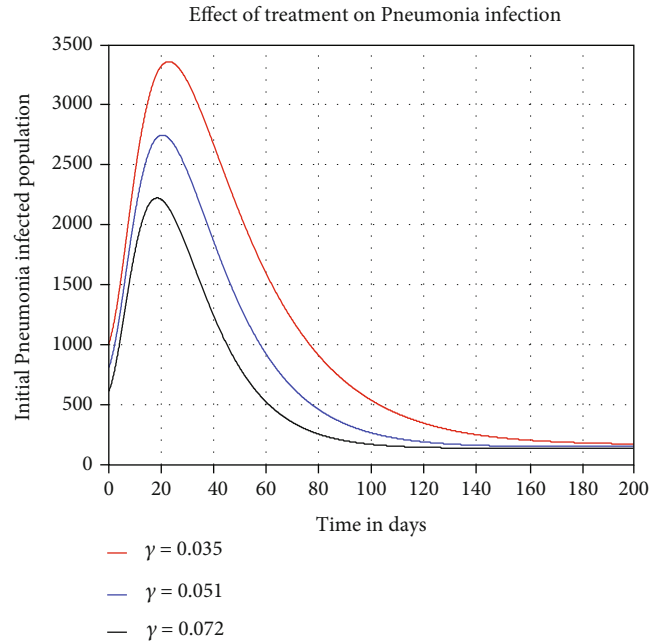

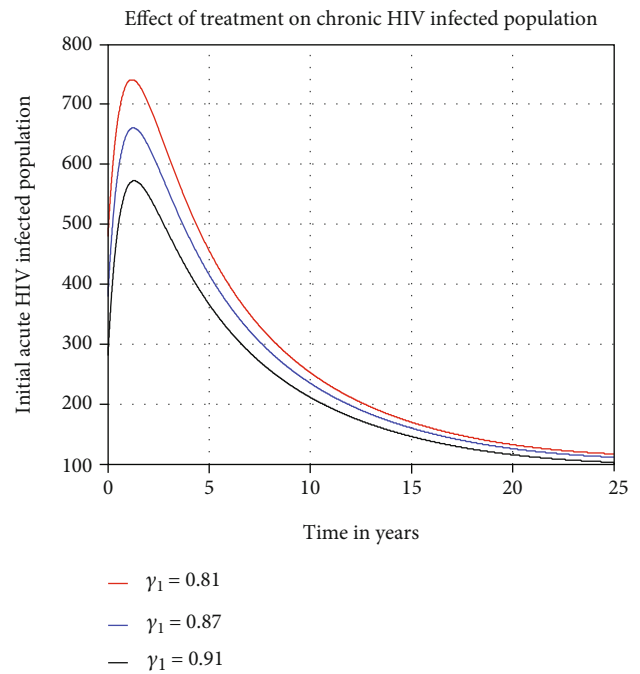Figure 10: The population of pneumonia-infected individual's response to treatment.

Figure 11: The population of acute-infected Individual's response to treatment.

duction numbers with variable parameter values using ode45 method, and we obtained figures from Figures 4–9. Thus, Figure 4 shows the pneumonia-only submodel reproduction number simulation at variable treatment rate, and from the graph, we see that pneumonia infection dies out at pneumonia treatment rate $\gamma > 0.89$. Figure 6 showed that pneumonia infection dies out whenever $\beta_2 < 0.00003$. Figure 7 shows that in the long run the HIV/AIDS
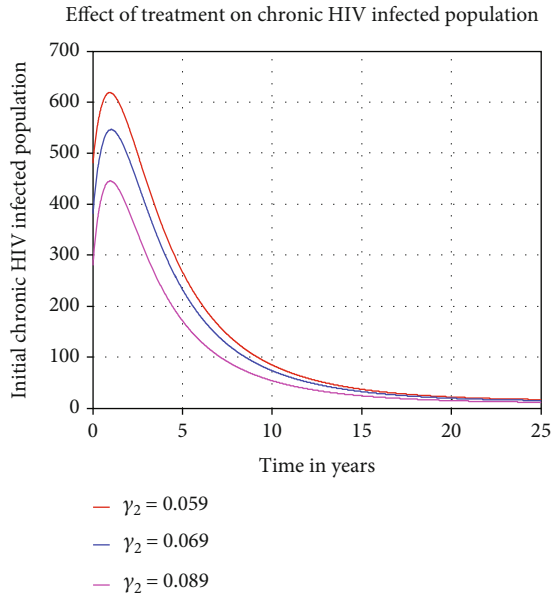
FIGURE 12: The population of chronic HIV-infected individual's response to treatment.

FIGURE 13: The population of AIDS patient's response to treatment.
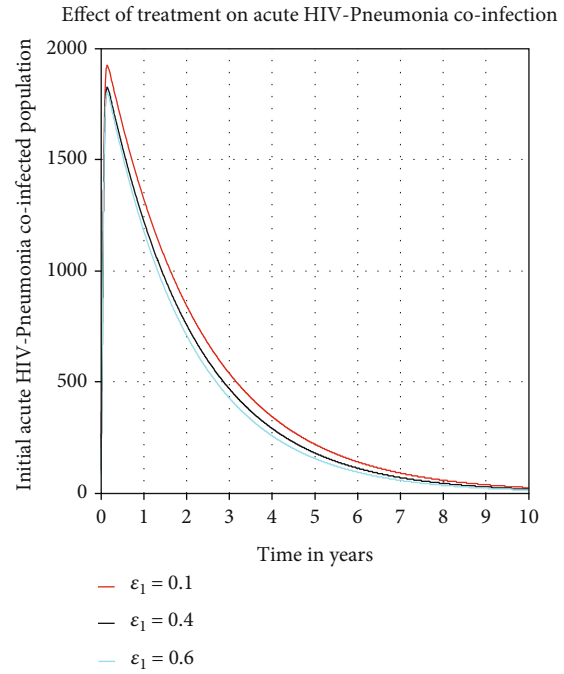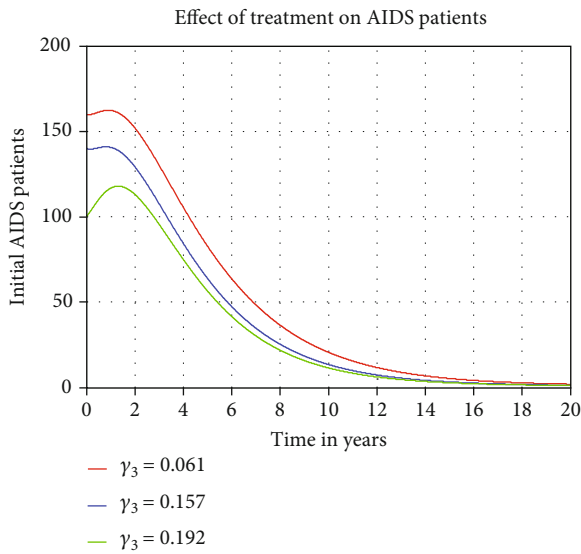

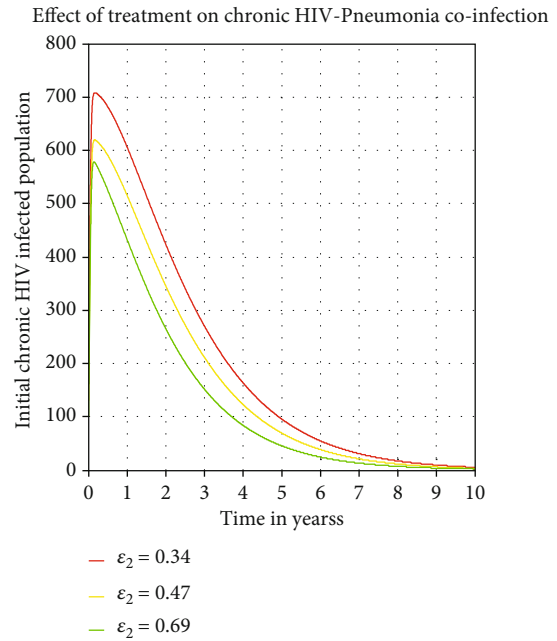FIGURE 14: The population of acute HIV-pneumonia coinfected individual's response to treatment.

FIGURE 15: The population of chronic HIV-pneumonia-infected individuals' response to treatment.

transmission decreases whenever $\beta_1 < 0.00002$. Figure 8 shows the HIV/AIDS reproduction number simulation at variable acute-infected treatment rate, and from the graph, we see that the HIV/AIDS-only submodel reproduction number is less than unity whenever $\gamma_1 > 0.93$ at $\beta_1 = 0.00015$, and Figure 9 shows the HIV/AIDS reproduction number simulation at variable chronic-infected treatment rate, and from the graph, we see that the HIV/AIDS-only submodel reproduction number is less than unity whenever the $\gamma_2 > 0.75$ at $\beta_1 = 0.00015$. Similarly, using parameter values from Table 3, the numerical simulation in Figure 5 shows that comparison of the basic reproduction numbers $\mathscr{R}_H$ and $\mathscr{R}_P$ at different values of β_1 and β_2, respectively; also, from the simulations,

we see that the basic reproduction number for HIV/AIDS is greater than the basic reproduction number of pneumonia-only infection where HIV/AIDS-only submodel reproduction number is also the basic reproduction number of the coinfection model. Also, $\mathscr{R}_H < 1$ whenever $\beta_1 < 0.000006$ and $\mathscr{R}_P < 1$ if $\beta_2 < 0.000014$. Antiretroviral therapy (ART) is used to suppress the HIV virus and stop the progression to AIDS stage,
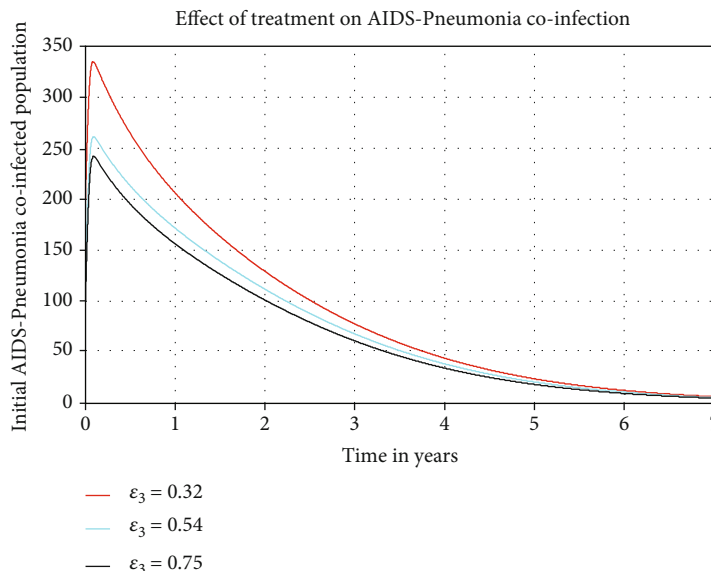
FIGURE 16: The population of AIDS-pneumonia coinfected Individuals' response to treatment.

and WHO recommends the immediate use of ART soon after diagnosis especially at the early stage in order to prevent the onward transmission and pneumonia antibiotics are used to treat bacterial pneumonia.

4.2.3. Simulations of Infected Population with Various Treatment Rates. In this subsection, from the numerical simulation, we have shown the effects of treatment at each infected stages of the model (3). Simulations from Figures 10–16 show the effects of variations of treatment rates on the infected population. Figure 10 shows that when pneumonia treatment rate γ increases from 0.035 to 0.072 the pneumonia infection decreases. Figure 11 shows that when acute HIV infection treatment rate γ_1 increases from 0.81 to 0.91 the acute HIV infection decreases. Figure 12 shows that when chronic HIV infection treatment rate γ_2 increases from 0.59 to 0.89 the chronic HIV infection decreases. Figure 13 shows that when AIDS patients treatment rate γ_3 increases from 0.061 to 0.192 the AIDS patients decreases. Figure 14 shows that when acute HIV/AIDS-pneumonia coinfection treatment rate ε_1 increases from 0.0.1 to 0.6 the acute HIV/AIDS-pneumonia coinfection decreases. Figure 15 shows that when chronic HIV/AIDS-pneumonia coinfection treatment rate ε_2 increases from 0.0.34 to 0.69 chronic HIV/AIDS-pneumonia coinfection decreases. Figure 16 shows that when AIDS-pneumonia coinfection treatment rate ε_3 increases from 0.32 to 0.75 AIDS-pneumonia coinfection decreases. Thus, our model considers treatment at every infection stages of the full HIV/AIDS-pneumonia coinfection model; all numerical simulation graphs from Figures 10–16 show those effects of treatment on the infected population in the corresponding compartments, and also, all simulated curves show that the infected population in the compartment decreases whenever the corresponding treatment rate increases which is the finding of this work.

5. Conclusion

In this work, we formulated a mathematical model of eleven nonlinear differential equations on HIV/AIDS and pneumonia coinfection with the assumption of mass action incidence and incorporating treatment at each stage of the infection. We have shown the positivity and boundedness of the solutions of the model. The threshold parameter $\mathscr{R}_{HP}$ was calculated and used to determine the conditions under which the HIV/AIDS and pneumonia could be transmitted and remained endemic in the population. We thus showed that three disease-free equilibrium points E_H^0, E_P^0, and E_{HP}^0, respectively, for the HIV submodel, pneumonia submodel, and full model are locally asymptotically stable when $\mathscr{R}_{HP} < 1$, i.e., $\mathscr{R}_H < 1$ and $\mathscr{R}_P < 1$. We also showed that the population with both HIV/AIDS and pneumonia infection have three endemic equilibrium points E_H^*, E_P^*, and E_{HP}^* respectively, for the HIVsub model, pneumonia submodel, and full model which were locally asymptotically stable when $\mathscr{R}_{HP} > 1$, i.e., $\mathscr{R}_H > 1$ or $\mathscr{R}_P > 1$. Global stability analysis of the submodels disease-free equilibrium points was established whenever $\mathscr{R}_{HP} < 1$, i.e., $\mathscr{R}_H < 1$ and $\mathscr{R}_P < 1$. To investigate, the effect of treatment at each infected compartment was considered for both the submodels and the full model, namely, treatment of pneumonia infection, treatment of acute HIV infection, treatment of chronic HIV infection, treatment of AIDS patients, treatment of acute HIV/AIDS-pneumonia coinfection, treatment of chronic HIV/AIDS-pneumonia coinfection, and treatment of AIDS-pneumonia coinfection. We have showed the most sensitive parameters of our model which can be epidemiologically controlled are the HIV/AIDS transmission rate β_1 and pneumonia transmission rate β_2 so it is reasonable to recommend the use of intervention strategy for HIV/AIDS transmission in making β_1 less than 0.00014 and treatment for pneumonia transmission in making β_2 less than 0.00006.

Numerical simulations were used to compare the endemic scenarios showed by analytical results. From the numerical results, we obtained that $\mathscr{R}_H = 17$ and $\mathscr{R}_P = 7$ at $\beta_1 = 0.00029$ and $\beta_2 = 0.00079$ and all other parameters used are from Table 3. We observed that treatment against a disease has the effect of reducing the progression rate of HIV infection to the AIDS stage and the disease prevalence. From the numerical results and discussion above, we would like to recommend the following to control the spread of HIV/AIDS and pneumonia coinfection. Thus, we can interpret the situation in an epidemiological manner that a society with some individuals infected with HIV/AIDS and without HIV/AIDS treatment is at risk of being coinfected with pneumonia which in turn creates socioeconomic effects if no intervention is implemented in time for either or both HIV/AIDS and pneumonia infection. We conclude that HIV/AIDS treatment for individuals with HIV/AIDS infections results in a significant reduction of the number of individuals progressing to AIDS stage and reduction of the coinfected individuals and reduction of the disease-induced death. Also, effective treatment of pneumonia for the coinfected individuals also reduced the number of individuals that progress to AIDS class. Here, we conclude that HIV/AIDS treatment for only HIV/AIDS individuals (HIV submodel) and coinfected individuals could be a better approach to studying the dynamics of HIV/AIDS and pneumonia and could be the best measure to reduce $\mathscr{R}_{HP}$ and coinfection. Models which incorporate other protective measures such as vaccination for pneumonia infection, education of population, and using condom for HIV/AIDS infection may be considered for further research

6. Limitations of the Study

There was a lack of literatures about HIV/ADS and pneumonia coinfection and well-organized standard parameter values for the determination of model parameters.

Appendix

A. Model Assumptions and Descriptions

We considered the three center for disease control and prevention (CDC) human immunodeficiency virus (HIV) infection stages and divide the total population $N(t)$ into eleven compartments which are the susceptible individuals to both HIV/AIDS and pneumonia denoted as $S(t)$ with recruitment by birth at a rate Λ, the acute HIV-infected individuals denoted as $H_1(t)$, the chronic HIV-infected individuals denoted as $H_2(t)$, the AIDS stage individuals denoted as $H_3(t)$, the acute HIV and pneumonia coinfected individuals denoted as $C_1(t)$, the chronic HIV and pneumonia coinfected individuals denoted as $C_2(t)$, the AIDS and pneumonia coinfected individuals denoted as $C_3(t)$, the pneumonia-infected individuals denoted as $I_P(t)$, the treatment group denoted as $T_P(t)$ which contains individuals on treatment of pneumonia infection, the treatment group denoted as $T_H(t)$ which contains individuals on treatment of HIV/AIDS entered from the three HIV stages $H_1(t)$,

$H_2(t)$, and $H_3(t)$ infected groups, and the treatment group denoted as $T(t)$ contains individuals who are on treatment of the coinfection from $C_1(t)$, $C_2(t)$, and $C_3(t)$ such that $N(t) = S(t) + I_P(t) + H_1(t) + H_2(t) + H_3(t) + C_1(t) + C_2(t) + C_3(t) + T_P(t) + T_H(t) + T(t)$ and in order to formulate the dynamical system, the following assumptions have been taken:

(i) We assume that all individuals in a given compartment are identically infectious

(ii) HIV-infected class is considered susceptible to pneumonia infection. However, we assumed that pneumonia-infected population is not susceptible to HIV infection

(iii) The susceptible class, $S(t)$, contains individuals at risk of either HIV or pneumonia infection

(iv) The susceptible population is increased by the recruitment of individuals into the population by Λ.

(v) The susceptible individual is infected with pneumonia ($I_P(t)$) at infection rate ("the force of infection") $\lambda_P(t)$ and infected with HIV at infection rate ("the force of infection") $\lambda_H(t)$

(vi) The constant α_1 be rate of progression of acute HIV-infected individuals ($H_1(t)$) to chronic HIV-infected individuals ($H_2(t)$), α_2 be the rate of progression of the chronic HIV-infected individuals ($H_2(t)$) to AIDS patients ($H_3(t)$), α_3 be the rate of progression of the acute HIV-pneumonia coinfected individuals ($C_1(t)$) to the chronic HIV-pneumonia coinfected individuals ($C_2(t)$), α_4 be the rate of progression of the chronic HIV-pneumonia coinfected individuals ($C_2(t)$) to the AIDS-pneumonia coinfected individuals ($C_3(t)$)

(vii) γ be the treatment rate for pneumonia-infected individuals $I_P(t)$, γ_1, γ_2, and γ_3 are treatment rates for the infected groups $H_1(t)$, $H_2(t)$, and $H_3(t)$ respectively; φ_1, φ_2, and φ_3 are the modification parameters of $H_1(t)$, $H_2(t)$, and $H_3(t)$ to $C_1(t)$, $C_2(t)$, and $C_3(t)$ respectively, at the force of infection rate $\lambda_P(t)$; and ε_1, ε_2, and ε_3 are treatment rates of the coinfections $C_1(t)$, $C_2(t)$, and $C_3(t)$ respectively

(viii) Infected individuals in the classes $I_P(t)$, $H_3(t)$, and all the coinfected groups have reduced daily activities due to morbidity and are less involved in HIV/AIDS transmission and we assume no HIV transmission from these classes

(ix) All individuals are subject to a natural death at the rate μ

(x) The population is not constant

(xi) No vertical transmission for HIV infection and no natural recovery for pneumonia infection

(xii) No permanent immunity for pneumonia-infected individuals and become susceptible again after treatment

(xiii) Since pneumonia is a population density-dependent transmission, we assumed the mass action incidence rate defined as the rate at which individuals acquire pneumonia $\lambda_P(t) = \beta_2(I_p(t) + \omega_1 C_1(t) + \omega_2 C_2(t) + \omega_3 C_3(t))$ where $\omega_3 > \omega_2 > \omega_1$ are modification parameters accounting for the assumed increased infectivity due to coinfection

(xiv) Also the rate at which individual acquire HIV/AIDS is defined as $\lambda_H(t) = \beta_1(H_1(t) + \rho H_2(t))$ where $\rho > 1$ is the modification parameter accounting for the assumed increased infectivity due to chronic HIV infection than the acute HIV-infected one

B. Positivity and Boundedness of Solutions

Proof of Positivity. Assume $S(0) > 0, I_P(0) > 0, H_1(0) > 0, H_2(0) > 0, H_2(0) > 0, C_1(0) > 0, C_2(0) > 0, C_3(0) > 0, T_P(0) > 0, T_H(0)$ and $T(0) > 0$ then for all $t > 0$, we have to prove that $S(t) > 0, I_P(t) > 0, H_1(t) > 0, H_2(t) > 0, H_3(t) > 0, C_1(t) > 0, C_2(t) > 0, C_3(t) > 0, T_P(t) > T_H(t) > 0$, and $T(t) > 0$. □

We define $\tau = \sup \{t > 0 : S(t) > 0, I_P(t) > 0, H_1(t) > 0, H_2(t) > 0, H_3(t) > 0, C_1(t) > 0, C_2(t) > 0, C_3(t) > 0, T_P(t) > 0, T_H(t) > 0$ and $T(t) > 0\}$.

From the continuity of $S(t), I_P(t), H_1(t), H_2(t), H_3(t), C_1(t), C_2(t), C_3(t), T_P(t), T_H(t)$, and $T(t)$, we deduce that $\tau > 0$. If $\tau = +\infty$, then positivity holds. But, if $0 < \tau < +\infty, S(\tau) = 0$ or $I_P(\tau) = 0$ or $H_1(\tau) = 0$ or $H_2(\tau) = 0$ or $H_3(\tau) = 0$ or $C_1(\tau) = 0$ or $C_2(\tau) = 0$ or $C_3(\tau) = 0$ or $T_P(\tau) = 0$ or $T_H(\tau) = 0$ or $T(0) = 0$.

From first equation of the model (1), we have $dS/dt = \Lambda + p\gamma T_P - (\mu + \lambda_H + \lambda_P)S$ and it can be rewritten as $dS/dt + (\mu + \lambda_H + \lambda_P)S = \Lambda + p\gamma T_P$, which is a first-order linear ordinary differential equation. To solve it, first find the integrating factor IF $= \exp^{\int((\mu+\lambda_H(t)+\lambda_P(t)))dt}$, then multiplying the equation by the integrating factor, we obtain as $\exp^{\int((\mu+\lambda_H(t)+\lambda_P(t)))dt}(dS/dt) + \exp^{\int((\mu+\lambda_H(t)+\lambda_P(t)))dt}(\mu + \lambda_H + \lambda_P)S = \exp^{\int((\mu+\lambda_H(t)+\lambda_P(t)))dt}(\Lambda + p\gamma T_P)$

$$\Longrightarrow \frac{d}{dt}\left(S(t) \exp^{\int((\mu+\lambda_H(t)+\lambda_P(t)))dt}\right) = \exp^{\int((\mu+\lambda_H(t)+\lambda_P(t)))dt}(\Lambda + p\gamma T_P)$$
(B.1)

Integrating both sides from 0 to τ will give us

$$\left(\left(S(t) \exp^{\int((\mu+\lambda_H(t)+\lambda_P(t)))dt}\right)\right)\Big|_0^\tau$$
$$= \int_0^\tau \exp^{\int((\mu+\lambda_H(t)+\lambda_P(t)))dt}(\Lambda + p\gamma T_P)dt$$

$$\left(\left(S(t) \exp^{\mu t+\int_0^t(\lambda_H(u)+\lambda_P(u))du}\right)\right)\Big|_0^\tau$$
$$= \int_0^\tau \exp^{\int((\mu+\lambda_H(t)+\lambda_P(t)))dt}(\Lambda + p\gamma T_P(t))dt$$
$$\Rightarrow S(\tau) \exp^{\left(\mu\tau+\int_0^\tau(\lambda_H(w)+\lambda_P(w))dw\right)} - S(0)$$
$$= \int_0^\tau \exp^{\int((\mu+\lambda_H(t)+\lambda_P(t)))dt}(\Lambda + p\gamma T_P(t))dt$$
$$\Rightarrow S(\tau) = M_1 S(0) + M_1 \int_0^\tau \exp^{\int((\mu+\lambda_H(t)+\lambda_P(t)))dt}$$
$$\cdot (\Lambda + p\gamma T_P(t))dt > 0.$$
(B.2)

Since $M_1 = \exp^{-(\mu\tau+\int_0^\tau(\lambda_H(w)+\lambda_P(w))dw)} > 0, S(0) > 0$, and from the definition of τ, we see that $T_P(t) > 0$ and also the exponential function is always positive; then, the solution $S(\tau) > 0$, hence $S(\tau) \neq 0$. Similarly, all state variables are positive at τ. Thus, based on the definition of τ above, τ is not finite which means $\tau = +\infty$ and hence all the solutions of the system (3) above are nonnegative.

Proof of Boundedness. From equation (13) and since all the state variables are nonnegative by positivity in the absence of infections, we obtained that $(dN/dt) \leq \Lambda - \mu N$. Now, using a standard comparison theorem, we do have $\int dN/(\Lambda - \mu N) \leq \int dt$ and integrating both sides gives $-(1/\mu) \ln(\Lambda - \mu N) \leq t + c$ where c is some constant, and after some calculations, we got $0 \leq N(t) \leq \Lambda/\mu$ which means all possible feasible solutions of the system (3) with positive initial conditions will enter in the bounded region $\Omega = \{ (S, H_1, H_2, H_3, I_P, C_1, C_2, C_3, T_P, T_H, T) \in \mathbb{R}^{11}_+, N(t) \leq \Lambda/\mu\}$. □

C. Proof of Theorem 1

The local stability of the disease-free equilibrium point of the system (7) can be studied from its Jacobian matrix at the disease-free equilibrium point $E_H^0 = (S^0, H_1^0, H_2^0, H_3^0, T_H^0) = (\Lambda/\mu, 0, 0, 0, 0)$ and Routh-Hurwitz stability criteria. Then, the Jacobian matrix of the dynamical system (3) at $E_H^0 = (\Lambda/\mu, 0, 0, 0, 0, 0)$ is given by

$$J(E_H^0) = \begin{pmatrix} -\mu & A_1 & A_2 & 0 & 0 \\ 0 & M_1 & -A_2 & 0 & 0 \\ 0 & \alpha_1 & M_2 & 0 & 0 \\ 0 & 0 & \alpha_2 & M_3 & 0 \\ 0 & \gamma_1 & \gamma_2 & \gamma_3 & -\mu \end{pmatrix}, \quad \text{(C.1)}$$

where $A_1 = -\beta_1(\Lambda/\mu), A_2 = -\beta_1\rho(\Lambda/\mu), M_1 = \beta_1(\Lambda/\mu) - (\mu + \gamma_1 + \alpha_1), M_2 = -(\mu + \gamma_2 + \alpha_2)$, and $M_3 = -(\mu + \gamma_3 + \delta_1)$.

Then, the characteristic equation of the above Jacobian matrix is given by

$$\begin{vmatrix} -\mu-\lambda & A_1 & A_2 & 0 & 0 \\ 0 & M_1-\lambda & -A_2 & 0 & 0 \\ 0 & \alpha_1 & M_2-\lambda & 0 & 0 \\ 0 & 0 & \alpha_2 & M_3-\lambda & 0 \\ 0 & \gamma_1 & \gamma_2 & \gamma_3 & -\mu-\lambda \end{vmatrix} = 0,$$

$$\Longrightarrow (-\mu-\lambda)\left[(M_1-\lambda)\begin{vmatrix} M_2-\lambda & 0 & 0 \\ \alpha_2 & M_3-\lambda & 0 \\ \gamma_2 & \gamma_3 & -\mu-\lambda \end{vmatrix} + A_2 \begin{vmatrix} \alpha_1 & 0 & 0 \\ 0 & M_3-\lambda & 0 \\ \gamma_1 & \gamma_3 & -\mu-\lambda \end{vmatrix} \right]$$

$$- A_1 A_2 \begin{vmatrix} 0 & 0 & 0 \\ 0 & M_3-\lambda & 0 \\ 0 & \gamma_3 & -\mu-\lambda \end{vmatrix} = 0,$$

$$\Longrightarrow (M_3-\lambda)(-\mu-\lambda)(-\mu-\lambda)[(M_1-\lambda)(M_2-\lambda) + A_2\alpha_1] = 0, \qquad (C.2)$$

$$\Longrightarrow \lambda_1 = M_3 = -(\mu+\gamma_3+\delta_1) < 0 \text{ or } \lambda_2 = \lambda_3 = -\mu < 0$$ or $\lambda^2 - (M_1+M_2)\lambda^2 + (M_1 M_2 + A_2\alpha_1) = 0$.

For the quadratic equation $\lambda^2 - (M_1+M_2)\lambda + (M_1 M_2 + A_2\alpha_1) = 0$ with its coefficients are given by $a_2 = 1$, $a_1 = -(M_1+M_2)$, and $a_0 = (M_1 M_2 + A_2\alpha_1)$ use Routh-Hurwitz stability criteria. Since $a_2 = 1 > 0$, the sign of a_1 and a_0 should be positive, i.e., $a_1 = -(M_1+M_2) = -(\beta_1(\Lambda/\mu) - (\mu+\gamma_1+\alpha_1) - (\mu+\gamma_2+\alpha_2)) = (\mu+\gamma_1+\alpha_1)(1 - R_{H_1})$ $+ (\mu+\gamma_2+\alpha_2) > 0$ if $\mathscr{R}_{H_1} < 1$ since all parameters are positive and $a_0 = (M_1 M_2 + A_2\alpha_1) = (\mu+\gamma_1+\alpha_1)(\alpha_1 + \mu + \gamma_2 + \alpha_2)(1 - \mathscr{R}_{H_2}) > 0$ if $\mathscr{R}_{H_2} < 1$ and all the elements of the first column of the Routh-Hurwitz array have the same sign means that eigenvalues are negative. Therefore, since all the eigenvalues of the characteristics polynomial of the system (3) are negative for $\mathscr{R}_H < 1$, the disease-free equilibrium point of the system (3) is locally asymptotically stable.

D. Proof of Theorem 4

To show the local stability of the endemic equilibrium point, we use the method of Routh-Hurwitz stability criteria. The Jacobian matrix of the dynamical system (3) at the endemic equilibrium point $E_H^* = (S^*, H_1^*, H_2^*, H_3^*, T_H^*)$ is

$$J(E_H^*) = \begin{pmatrix} A_1 & A_5 & A_6 & 0 & 0 \\ \lambda_H^* & A_2 & -A_6 & 0 & 0 \\ 0 & \alpha_1 & A_3 & 0 & 0 \\ 0 & 0 & \alpha_2 & A_4 & 0 \\ 0 & \gamma_1 & \gamma_2 & \gamma_3 & -\mu \end{pmatrix}, \qquad (D.1)$$

where $A_1 = -\mu\mathscr{R}_H$, $A_2 = \beta_1 S^* - (\mu+\gamma_1+\alpha_1) = (\beta_1\Lambda/\mu\mathscr{R}_H) - (\mu+\gamma_1+\alpha_1) = (\beta_1\Lambda/\mu\mathscr{R}_H) - d_1$ with $d_1 = \mu+\gamma_1+\alpha_1$, $A_3 = -(\mu+\gamma_2+\alpha_2)$, $A_4 = -(\mu+\gamma_3+\delta_1)$, $A_5 = -\beta_1\Lambda/\mu\mathscr{R}_H$, and $A_6 = -\beta_1\rho\Lambda/\mu\mathscr{R}_H$.

Then, the characteristic equation of the above Jacobian matrix is given by

$$\begin{vmatrix} A_1-\lambda & A_5 & A_6 & 0 & 0 \\ \lambda_H^* & A_2-\lambda & -A_6 & 0 & 0 \\ 0 & \alpha_1 & A_3-\lambda & 0 & 0 \\ 0 & 0 & \alpha_2 & A_4-\lambda & 0 \\ 0 & \gamma_1 & \gamma_2 & \gamma_3 & -\mu-\lambda \end{vmatrix} = 0,$$

$$\Longrightarrow (A_1-\lambda)[(A_2-\lambda)(A_3-\lambda)(A_4-\lambda)(-\mu-\lambda)$$
$$+ A_6\alpha_1(A_4-\lambda)(-\mu-\lambda)] + A_5[\lambda_H^*(A_3-\lambda)(A_4-\lambda)(-\mu-\lambda)]$$
$$+ A_6[\lambda_H^*\alpha_1(A_4-\lambda)(-\mu-\lambda)] = 0,$$

$$\Longrightarrow [(A_4-\lambda)(-\mu-\lambda)]\{-\lambda^3 + (A_1+A_2+A_3)\lambda^2$$
$$- (A_1 A_2 + A_1 A_3 + A_2 A_3 + A_6\alpha_1 + A_5\lambda_H^*)\lambda$$
$$+ A_1 A_2 A_3 + A_1 A_6\alpha_1 + A_3 A_5\lambda_H^* + A_6\lambda_H^*\alpha_1\} = 0,$$

$$\Longrightarrow \lambda_1 = A_4 \text{ or } \lambda_2 = -\mu \text{ or } \{-\lambda^3 + (A_1+A_2+A_3)\lambda^2$$
$$- (A_1 A_2 + A_1 A_3 + A_2 A_3 + A_6\alpha_1 + A_5\lambda_H^*)\lambda \qquad (D.2)$$
$$+ A_1 A_2 A_3 + A_1 A_6\alpha_1 + A_3 A_5\lambda_H^* + A_6\lambda_H^*\alpha_1\} = 0.$$

Here, we have obtained that $\lambda_1 = A_4 = -(\mu+\gamma_3+\delta_1) < 0$, $\lambda_2 = -\mu < 0$, and $a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0$ where-

$a_3 = 1$, $a_2 = -(A_1+A_2+A_3) = [\mu + \mu(\mathscr{R}_H - 1)]^2 - \beta_1\Lambda + \mu\mathscr{R}_H(d_1 + (\mu+\gamma_2+\alpha_2))/\mu\mathscr{R}_H > 0$ if $\mathscr{R}_H > 1$,

$a_1 = (\Lambda\rho\alpha_1\beta_1)^2/\mu\mathscr{R}_H(\mu+\gamma_1+\alpha_1)(\mu+\gamma_2+\alpha_2)^2 + ((d_2\mu^2\Lambda\beta_1/\mu^2\mathscr{R}_H(\mu+\gamma_1+\alpha_1)) + (d_2\mu^2\Lambda\rho\alpha_1\beta_1/\mu^2\mathscr{R}_H(\mu+\gamma_1+\alpha_1)(\mu+\gamma_2+\alpha_2)))^2 + (\mu\beta_1\Lambda/\mu\mathscr{R}_H) > 0$ if $\mathscr{R}_H > 1$ and $a_0 = \Lambda\rho\alpha_1\beta_1 + \beta_1\rho\Lambda\alpha_1 + (d_2\beta_1\Lambda(\mathscr{R}_H - 1)/\mathscr{R}_H) + (\beta_1\rho\Lambda\alpha_1(\mathscr{R}_H - 1)/\mathscr{R}_H) > 0$ if $\mathscr{R}_H > 1$ implies all the coefficients of the characteristic's polynomial are positives if $\mathscr{R}_H > 1$.

Here, applying the Routh-Hurwitz method, we obtained the Routh-Hurwitz array that has no sign change; then, the roots of the characteristics equation of the dynamical system (7) are negative. Hence, the endemic equilibrium point of the dynamical system (7) is locally asymptotically stable.

Data Availability

No data.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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