

Research Article

Investigating the Effects of Intervention Strategies on Pneumonia and HIV/AIDS Coinfection Model

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HIV/AIDS and pneumonia coinfection have imposed a major socioeconomic and health burden throughout the world, especially in the developing countries. In this study, we propose a compartmental epidemic model on the spreading dynamics of HIV/AIDS and pneumonia coinfection to investigate the impacts of protection and treatment intervention mechanisms on the coinfection spreading in the community. In the qualitative analysis of the model, we have performed the positivity and boundedness of the coinfection model solutions; the effective reproduction numbers using the next-generation operator approach; and both the disease-free and endemic equilibrium points' local and global stabilities using the Routh-Hurwitz and Castillo-Chavez stability criteria, respectively. We performed the sensitivity analysis of the model parameters using both the forward normalized sensitivity index criteria and numerical methods (simulation). Moreover, we carried out the numerical simulation for different scenarios to investigate the effect of model parameters on the associated reproduction number, the effect of model parameters on the model state variables, and the solution behavior and convergence to the equilibrium point(s) of the models. Finally, from the qualitative analysis and numerical simulation results, we observed that the disease-spreading rates, protection rates, and treatment rates are the most sensitive parameters, and we recommend for stakeholders to concentrate and exert their maximum effort to minimize the spreading rates by maximizing the protection and treatment rates.

1. Introduction

Infectious diseases investigated and verified in the laboratory or in the clinic are illnesses caused by pathogenic microorganisms, and pneumonia is an infectious disease caused by microorganisms like bacteria, virus, fungus, and parasites; HIV/AIDS is also an infectious disease caused by viruses [1–3].

Acquired immunodeficiency syndrome (AIDS) caused by human immunodeficiency virus (HIV), discovered in 1981, is one of the major deadly infectious diseases that has been spreading through countries in the world [1, 4–7]. Different literatures reported that HIV/AIDS has been the major health-affected infectious disease and affected more than seventy million individuals [1, 8, 9]. HIV attacks white blood cells and is spreading through sexual contact, sharing needle, and blood contact or by fluids containing the HIV virus and by vertical transmission from mother to child at birth [5, 6].

Pneumonia caused by various pathogenic microbial agents like virus, bacteria, fungi, and parasites is a major respiratory infectious disease identified as an inflammatory condition of the lungs [10–12]. Among the pathogenic microbial agents which have potential in causing pneumonia infection, bacteria especially *Streptococcus pneumoniae* have been reported as the leading cause [10–12]. The bacteria microbial agents enter the lungs, rapidly multiply its number, and settle in the air passage called alveoli of the human being lung; the lung will be filled with fluid and pus, which makes breathing difficult [10, 12]. Pneumonia is commonly a highly transmitted disease and a major cause of morbidity and mortality in both children and adults throughout nations in the world [13, 14].

Infectious disease studies using mathematical modelling approaches have been carried out by different researchers to tackle the basic epidemic problems and for making predictions of quantitative measures of different prevention

and controlling strategies and their effectiveness; see literatures [1–13, 15–36]. Even though mathematical epidemiologists did not give attention like the common HIV/AIDS and TB coinfection [32, 34, 35] and other coinfections, the coinfection of HIV/AIDS and pneumonia in one host is a common phenomenon. Since pneumonia is one of the most common opportunistic infections for HIV/AIDS-infected individuals, some scholars have carried out few essential mathematical epidemiological research studies on the transmission dynamics of HIV/AIDS and pneumonia coinfection; see literatures [4, 5]. In this study, we have reviewed some epidemic mathematical modelling approach researches which are irrelevant to our proposed study and done by different scholars in the world. Huo et al. [1] presented a mathematical model approach study on a stage structure HIV/AIDS transmission dynamics of HIV/AIDS with treatment strategy. The finding of the study stated that the HIV/AIDS treatment strategy (ART) is the most effective strategy at the HIV asymptomatic stage of the HIV infection or before-AIDS stage to minimize its spreading in the community. Omondi et al. [9] presented a sex-structured community infection model and discuss male and female HIV infection trends with heterosexual activities. The finding of the study stated that the HIV/AIDS treatment (ART) strategy has a significant impact on controlling HIV/AIDS transmission in the community. Teklu [24] presented a mathematical modelling approach research on COVID-19 infection in the presence of prevention and control strategies. The results and findings of the study deduced that applying COVID-19 vaccination, other protection measures, home quarantine with treatment, and hospital quarantine with treatment simultaneously is the most effective strategy to minimize the COVID-19 spread in the community. Teklu and Mekonnen [4] analyzed HIV/AIDS and pneumonia coinfection model with treatment at each infection stage. From the results of the model analysis, they deduced that applying treatment mechanisms for both the single infections and coinfection individuals is the most effective strategy to minimize the coinfection disease-spreading dynamics. Teklu and Rao [5] proposed and investigated a compartmental model on the coexistence of HIV/AIDS and pneumonia with pneumonia vaccination, treatments of pneumonia, and HIV/AIDS infection control measures. The finding of the model analysis stated that to minimize the coinfection disease spread in the community, controlling pneumonia infection using vaccination and treatment is more effective than treatment of HIV/AIDS only infection.

The main purpose of this study is to investigate the impacts of pneumonia protection, pneumonia treatment, and HIV protection by using condom and HIV treatment (ART) intervention strategies simultaneously on the transmission dynamics of HIV/AIDS and pneumonia coinfection in the community. Even though researchers [4, 5] invested much effort in studying HIV/AIDS and pneumonia coinfection, they did not consider pneumonia protection, pneumonia vaccination, pneumonia treatment, HIV protection by using condom, and HIV treatment as prevention and control strategies simultaneously in their proposed coinfection model formulation and analysis. And also, the main

contributions of this study are as follows: the health stakeholders can use the findings of this modified research study to tackle the HIV/AIDS and pneumonia coinfection in the community; potential young researchers can develop their epidemiological modelling knowledge and skills; and potential senior researchers can modify the study by incorporating different modelling and intervention aspects. Based on the findings of the above-reviewed literatures, we have realized the gaps and are highly motivated to tackle the problem by modifying the research study [5]. The remaining part of this study is structured in the following sequence: the model is formulated in Section 2 and is analyzed in Section 3; sensitivity analysis, numerical simulation, and conclusions of the study are carried out in Sections 4 and 5, respectively.

2. Model Description and Formulation

Motivated by various scholars' mathematical modelling researches in real-world situations, we proposed a coinfection integer order model on HIV/AIDS and pneumonia spreading dynamics. To describe and formulate the proposed coinfection model, we divide the total human population considered in this study at a time t and represented by $N(t)$ into nine mutually distinct classifications as follows: the number of people who are susceptible to either HIV/AIDS or pneumonia infection represented by $S(t)$, the number of people who are protected against pneumonia infection represented by $P_p(t)$, the number of people who are protected against HIV infection by using condom is represented by $H_p(t)$, the number of people who are infected with pneumonia is only represented by $P_I(t)$, the number of people who are infected with HIV is only represented by $H_I(t)$, the number of people who are AIDS patients represented by $H_A(t)$, the number of people who are coinfecting with HIV/AIDS and pneumonia is represented by $C(t)$, the number of people who are treated from HIV/AIDS is represented by $T_{HA}(t)$, the number of people who are infected with pneumonia is represented by $P_T(t)$; and the total number of individuals who are considered in this study is represented by

$$N(t) = S(t) + P_p(t) + H_p(t) + H_I(t) + H_A(t) + P_I(t) + C(t) + P_T(t) + T_{HA}(t). \quad (1)$$

The force of infection where the susceptible people acquire HIV/AIDS is defined by

$$\lambda_H(t) = \frac{\beta_1}{N} (H_I(t) + \alpha H_A(t) + \vartheta C(t)). \quad (2)$$

where $1 \leq \sigma < \infty$ and $1 \leq \vartheta < \infty$ are the modification parameters which increase infectivity of individuals and β_1 is the HIV/AIDS spreading rate.

The force of infection where the susceptible people acquire pneumonia is defined by

$$\lambda_p(t) = \frac{\beta_2}{N} (P_I(t) + \omega C(t)). \quad (3)$$

where $1 \leq \omega < \infty$ is the modification parameter which increases infectivity and β_2 is the pneumonia spreading rate.

To formulate the proposed coinfection model of HIV/AIDS and pneumonia, let us assume the following: p_1, p_2 , and $(1 - p_1 - p_2)$ be portions of the total recruited people Γ who are entering to the pneumonia-protected class $P_p(t)$, to the HIV protected class $H_p(t)$, and to the susceptible class $S(t)$, respectively; pneumonia recovery by treatment is not permanent, human population is homogeneous and is not constant, there is no HIV transmission from HIV-treated people and no HIV vertical transmission, and there is no simultaneous HIV and pneumonia dual-infection transmission.

Based on Tables 1 and 2 and the model descriptions and assumptions given above, the flow chart for the spreading dynamics of HIV/AIDS and pneumonia coinfection is illustrated in Figure 1.

Based on Figure 1, we derive the system of nonlinear differential equations of the coinfection model as follows:

$$\begin{aligned}
 \frac{dS}{dt} &= (1 - p_1 - p_2)\Gamma + \varepsilon_1 P_p + \varepsilon_2 H_p + \eta P_T - (\lambda_H + \lambda_p + \mu)S, \\
 \frac{dP_p}{dt} &= p_1 \Gamma - (\delta \lambda_H + \varepsilon_1 + \mu)P_p, \\
 \frac{dH_p}{dt} &= p_2 \Gamma - (\varepsilon_2 + \mu + \sigma \lambda_p)H_p, \\
 \frac{dH_I}{dt} &= \lambda_H S + \delta \lambda_H P_p - (\mu + \tau + \xi_1 + \phi_1 \lambda_p)H_I, \\
 \frac{dH_A}{dt} &= \tau H_I - (\mu + \mu_1 + \xi_2 + \phi_2 \lambda_p)H_A, \\
 \frac{dP_I}{dt} &= \lambda_p S + \sigma \lambda_p H_p - (\mu + \mu_2 + \gamma + \phi \lambda_H)P_I, \\
 \frac{dC}{dt} &= \phi \lambda_H P_I + \phi_1 \lambda_p H_I + \phi_2 \lambda_p H_A + \rho \lambda_p T_{HA} - (\mu + \mu_3 + \theta)C, \\
 \frac{dP_T}{dt} &= \gamma P_I - (\mu + \eta)P_T, \\
 \frac{dT_{HA}}{dt} &= \xi_1 H_I + \xi_2 H_A + \theta C - (\mu + \rho \lambda_p)T_{HA},
 \end{aligned}
 \tag{4}$$

with initial data,

$$\begin{aligned}
 S(0) &> 0, \\
 P_p(0) &\geq 0, \\
 H_p(0) &\geq 0, \\
 H_I(0) &\geq 0, \\
 H_A(0) &\geq 0, \\
 H_I(0) &\geq 0, \\
 C(0) &\geq 0, \\
 P_T(0) &\geq 0, \\
 T_{HA}(0) &\geq 0.
 \end{aligned}
 \tag{5}$$

Adding all the differential equations in the system, (4) gives

$$\frac{dN}{dt} = \Delta - \mu N - (\mu_1 H_A + \mu_2 P_I + \mu_3 C). \tag{6}$$

3. Qualitative Analysis of the Model (4)

3.1. Nonnegativity and Boundedness of the Model Solutions. Since the proposed model (4) deals with human beings, we need to investigate that each of the model solution variables is nonnegative and bounded in the region.

$$\Omega = \left\{ (S, P_p, H_p, H_I, H_A, P_I, C, P_T, T_{HA}) \in \mathbb{R}_+^9, N \leq \frac{\Gamma}{\mu} \right\}. \tag{7}$$

Theorem 1. *Nonnegativity.*

Depending on the initial data given in equation (5), each of the model solutions $S(t), P_p(t), H_p(t), H_I(t), H_A(t), P_I(t), C(t), P_T(t)$, and $T_{HA}(t)$ of the system (4) is nonnegative for $t > 0$.

Proof. Let the initial data be $S(0) > 0, P_p(0) > 0, H_p(0) > 0, H_I(0) > 0, H_A(0) > 0, P_I(0) > 0, C(0) > 0, P_T(0) > 0$, and $T_{HA}(0) > 0$. Then, $t > 0$, we need to prove that all the model solutions $S(t) > 0, P_p(t) > 0, H_p(t) > 0, H_I(t) > 0, H_A(t) > 0, P_I(t) > 0, C(t) > 0, P_T(t) > 0$, and $T_{HA}(t) > 0$.

Now, let us define the following set: $\tau = \sup \{t > 0 : S(t) > 0, P_p(t) > 0, H_p(t) > 0, H_I(t) > 0, H_A(t) P_I(t) > 0, C(t) > 0, P_T(t) > 0 \text{ and } T_{HA}(t) > 0\}$. Because the model state variables $S(t), P_p(t), H_p(t), H_I(t), H_A(t) P_I(t), C(t), R_T(t)$ and $T_{HA}(t)$ are continuous, we deduce that $\tau > 0$.

If $\tau = \infty$, then nonnegativity holds. But, if $0 < \tau < \infty$, $S(\tau) = 0$ or $P_p(\tau) = 0$ or $H_p(\tau) = 0$ or $H_I(\tau) = 0, H_A(\tau) = 0$ or $P_I(\tau) = 0$ or $C(\tau) = 0$ or $P_T(\tau) = 0$ or $T_{HA}(\tau) = 0$.

Rearranging the first equation of the model (4) gives us

$$\frac{dS}{dt} + (\lambda_H + \lambda_p + \mu)S = (1 - p_1 - p_2)\Gamma + \varepsilon_1 P_p + \varepsilon_2 H_p + \eta P_T. \tag{8}$$

We apply the method of integrating factors, and after some computations, we determined the result $S(\tau) = M_1 S(0)$

$$+ M_1 \int_0^\tau \exp^{\int_0^t (\lambda_H + \lambda_p + \mu) dt} ((1 - p_1 - p_2)\Gamma + \varepsilon_1 P_p(t) + \varepsilon_2 H_p(t) + \eta P_T(t)) dt > 0,$$

where-

$$M_1 = \exp^{-\left(\mu\tau + \int_0^\tau (\lambda_H(w) + \lambda_p(w)) dw\right)} > 0, S(0) > 0, P_p(t) > 0, H_p(t) > 0, P_T(t) > 0, \text{ and by the meaning of } \tau, S(\tau) > 0, \text{ hence } S(\tau) \neq 0.$$

Similarly, rearranging the second equation of the system (4) gives us $(dP_p/dt) + (\delta \lambda_H + \varepsilon_1 + \mu)P_p = \varepsilon_1 \Gamma$, and we have got

$$P_p(\tau) = M_1 P_p(0) + M_1 \int_0^\tau \exp^{\int_0^t (\varepsilon_1 + \mu + \delta \lambda_H(t)) dt} p_1 \Delta dt > 0,$$

where $M_1 = \exp^{-(\varepsilon_1 \tau + \mu \tau + \int_0^\tau (\delta \lambda_H(w)) dw)} > 0, P_p(0) > 0$, by definition of $\tau, P_p(\tau) > 0$, hence $P_p(\tau) \neq 0$.

Similarly, one can determine the results; $H_p(\tau) > 0$ hence $H_p(\tau) \neq 0, H_I(\tau) > 0$ hence $H_I(\tau) \neq 0, P_I(\tau) > 0$ hence

TABLE 1: Parameters used in the model formulation.

Parameter	Interpretation
μ	Natural mortality rate
Γ	People recruitment rate
ε_1	Pneumonia protection loss rate
ε_2	HIV protection loss rate
θ	HIV/AIDS and pneumonia coinfecting people treatment rate
Φ_1, Φ_2	Modification parameters
φ	Modification parameter
μ_2	Death rate by pneumonia disease
μ_1	Death rate by AIDS disease
γ	Pneumonia treatment rate
ξ_1	HIV-infected treatment rate
ξ_2	AIDS patient treatment rate
β_1	HIV/AIDS spreading rate
β_2	Pneumonia spreading rate
P_1	Portion of recruitment rate
P_2	Portion of recruitment rate
η	Pneumonia immunity loss rate
$\rho, \omega, \varphi, \vartheta$	Modification parameters
μ_3	Death rate by the coinfection

TABLE 2: Variables used in the coinfection model.

Variable	Epidemiological meaning
S	Susceptible people
P_p	People who are protected against pneumonia
H_p	People who are protected against HIV/AIDS
H_I	HIV-infected individuals
H_A	AIDS patients
P_I	Pneumonia-infected people
C	People coinfecting with HIV/AIDS and pneumonia
P_T	People treated from pneumonia
T_{HA}	HIV/AIDS-treated people

$P_I(\tau) \neq 0, C(\tau) > 0$ hence $C(\tau) \neq 0, P_T(\tau) > 0$ hence $P_T(\tau) \neq 0$, and $T_{HA}(\tau) > 0$ hence $T_{HA}(\tau) \neq 0$. Thus, by definition of τ given above, $\tau = \infty$, and hence, each of the model (3) solutions is nonnegative. \square

Theorem 2 Boundedness. *Each of the model solutions given in the region Ω (7) is bounded in \mathbb{R}_+^9 .*

Proof. In the absence of infections, the sum of all the differential equations given in (6), and by the nonnegativity condition in Theorem 1, we have $(dN/dt) \leq \Gamma - \mu N$. Based on the concept of the standard comparison theorem, we

determined the result $\int (dN/\Gamma - \mu N) \leq \int dt$, and integrating both sides gives us the result $-(1/\mu) \ln(\Gamma - \mu N) \leq t + c$, where c is some constant, and after some computations, we have the result

$0 \leq N(t) \leq (\Gamma/\mu)$. Therefore, the model (4) solutions with positive initial data given in (5) are bounded. \square

3.2. Qualitative Analysis HIV/AIDS Infection Submodel. Now, make the state variables corresponding to pneumonia-only infection of the coinfection model (4) as $P_p = P_I = C = P_T = 0$, we have the HIV/AIDS infection submodel given by

$$\begin{aligned} \frac{dS}{dt} &= (1 - p_2) \Gamma + \varepsilon_2 H_p - (\lambda_H + \mu) S, \\ \frac{dH_p}{dt} &= p_2 \Gamma - (\varepsilon_2 + \mu) H_p, \\ \frac{dH_I}{dt} &= \lambda_H S - (\mu + \xi_1 + \tau) H_I, \\ \frac{dH_A}{dt} &= \tau H_I - (\mu + \mu_1 + \xi_2) H_A, \\ \frac{dT_{HA}}{dt} &= \xi_1 H_I + \xi_2 H_A - \mu T_{HA}, \end{aligned} \tag{9}$$

where $\lambda_H(t) = (\beta_1/N_1)(H_I(t) + \alpha H_A(t))$ and $N_1 = S + H_p + H_I + H_A + T_{HA}$.

3.2.1. Local Stability of Disease-Free Equilibrium Point. The HIV/AIDS submodel (8) disease-free equilibrium (DFE) is computed by making each equation of the dynamical system (8) equal to zero where there are no infections and treated groups. Therefore, the submodel (8) DFE is given by $E_H^0 = (S^0, H_p^0, H_I^0, H_A^0, T_{HA}^0) = ((\Gamma/\mu)(\varepsilon_2 + \mu(1 - p_2)/\varepsilon_2 + \mu), (p_2 \Gamma/\varepsilon_2 + \mu), 0, 0, 0)$.

Using the same method stated in [22] on the HIV/AIDS submodel (8), we have computed the matrices F and V by

$$\begin{aligned} F &= \begin{pmatrix} \frac{\beta_1}{N_1^0} S^0 & \frac{\beta_1}{N_1^0} \alpha S^0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \\ &= \begin{pmatrix} \frac{\beta_1 \varepsilon_2 + \beta_1 \mu (1 - p_2)}{\varepsilon_2 + \mu} & \frac{\beta_1 \varepsilon_2 \alpha + \beta_1 \mu \alpha (1 - p_2)}{\varepsilon_2 + \mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \\ V &= \begin{pmatrix} \mu + \xi_1 + \tau & 0 & 0 \\ -\tau & \mu + \mu_1 + \xi_2 & 0 \\ -\xi_1 & -\xi_2 & \mu \end{pmatrix}. \end{aligned} \tag{10}$$

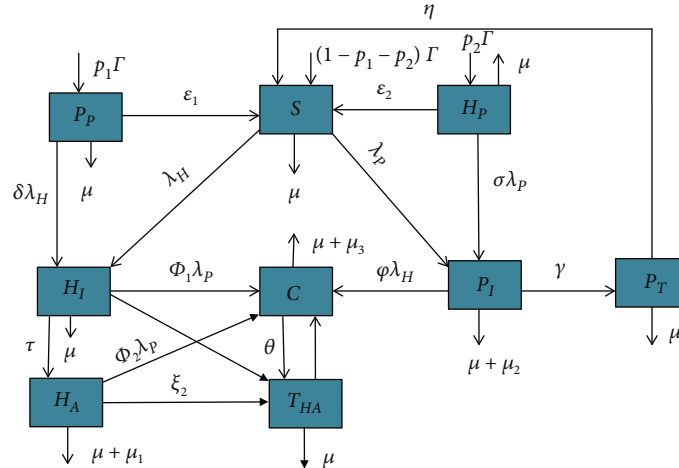


FIGURE 1: The flow chart of the HIV/AIDS and pneumonia codynamics spreading dynamics with forces of infections $\lambda_H(t)$ and $\lambda_p(t)$ given in (2) and (3), respectively.

The HIV/AIDS submodel (8) basic reproduction number is the largest eigenvalue in magnitude of the next generation matrix FV^{-1} and is computed as

$$\mathcal{R}_H^0 = \frac{\beta_1(\mu(1-p_2) + \epsilon_2)}{(\mu + \xi_1 + \tau)(\mu + \epsilon_2)} + \frac{\beta_1\mu\alpha(1-p_2) + \beta_1\epsilon_2\alpha}{(\mu + \mu_1 + \xi_2)(\mu + \epsilon_2)} \quad (11)$$

The threshold quantity \mathcal{R}_H^0 (basic reproduction number) of the HIV/AIDS submodel (8) is the expected number of secondary HIV infections produced by single infected human during its entire period of infectiousness throughout the whole susceptible community, and the HIV/AIDS submodel disease-free equilibrium point $E_H^0 = (S^0, H_p^0, H_I^0, H_A^0, T_{HA}^0) = (\Gamma/\mu((\epsilon_2 + \mu(1-p_2))/\epsilon_2 + \mu), (p_2\Gamma/\epsilon_2 + \mu), 0, 0, 0)$ has a local asymptotic stability whenever $\mathcal{R}_H^0 < 1$, and unstable whenever $\mathcal{R}_H^0 > 1$.

3.2.2. Endemic Equilibrium Point (S) Existence and Uniqueness. In this subsection setting, the right-hand side of the HIV/AIDS-only dynamical system given in equation (9) is equal to

zero, and after a number of steps of computations, we have determined the endemic equilibrium point(s) given by

$$\begin{aligned} S^* &= \frac{D_1}{D_2(\mu + \lambda_H^*)}, H_p^* \\ &= \frac{p_2\Gamma}{D_2}, H_I^* \\ &= \frac{D_1\lambda_H^*}{D_2D_3(\mu + \lambda_H^*)}, H_A^* \\ &= \frac{D_1\tau\lambda_H^*}{D_2D_3D_4(\mu + \lambda_H^*)}, \text{ and } T_{HA}^* \\ &= \frac{D_1D_4\xi_1\lambda_H^* + D_1\tau\xi_2\lambda_H^*}{D_2D_3\mu(\mu + \lambda_H^*)}, \end{aligned} \quad (12)$$

where $D_1 = \epsilon_2\Gamma + \mu\Gamma(1-p_2)$, $D_2 = (\epsilon_2 + \mu)$, $D_3 = (\mu + \xi_1 + \tau)$, $D_4 = (\mu + \mu_1 + \xi_2)$.

Now, substitute H_I^* and H_A^* in the HIV/AIDS force of infection given by $\lambda_H^* = (\beta_1/N_1^*)(H_I^* + \alpha H_A^*)$, and computing for λ_H^* , we have determined that

$$\begin{aligned} \lambda_H^* &= \frac{-D_3D_4\mu}{D_1D_2\tau\mu\Gamma(1 - (\beta_1(\mu(1-p_2) + \epsilon_2)/(\mu + \xi_1 + \tau)(\mu + \epsilon_2)) - (\beta_1\mu\alpha(1-p_2) + \beta_1\epsilon_2\alpha)/(\mu + \mu_1 + \xi_2)(\mu + \epsilon_2))} \\ &= \frac{-D_3D_4\mu}{D_1D_2\tau\mu\Gamma(1 - \mathcal{R}_H^0)} > 0 \end{aligned} \quad (13)$$

if and only if $\mathcal{R}_H^0 > 1$.

Thus, based on the final result $\lambda_H^* > 0$, there is a unique positive endemic equilibrium for the HIV/AIDS submodel given in equation (9) if and only if $\mathcal{R}_H^0 > 1$.

Lemma 3. *The HIV/AIDS monoinfection model given in equation (9) has a unique endemic equilibrium solution if and only if $\mathcal{R}_H^0 > 1$.*

3.2.3. DFE Global Asymptotic Stability

Lemma 4 (The Castillo-Chavez et al. criteria stated in [23]). *If the HIV/AIDS submodel can be written as*

$$\begin{aligned} \frac{dX}{dt} &= I(X, V), \\ \frac{dV}{dt} &= J(X, V), H(X^0, 0) = 0, \end{aligned} \tag{14}$$

where $X \in \mathbb{R}^m$ be the components of noninfected individuals and $V \in \mathbb{R}^n$ be the components of infected individuals including treated class, and $E_H^0 = (X^0, 0)$ denotes the disease-free equilibrium point of the dynamical system (7).

Assume (i) for $(dX/dt) = I(X^0, 0)$, Y^0 is globally asymptotically stable (GAS). (ii) $J(X, V) = BV - \dot{J}(X, V)$, $\dot{J}(X, V) \geq 0$ for $(X, V) \in \Omega_1$ where $B = D_V J(X^0, 0)$ is an M-matrix, i.e., the off-diagonal elements of B are nonnegative, and Ω_1 is the region in which the system makes biological sense. Then, the fixed point $E_H^0 = (X^0, 0)$ is globally asymptotically stable equilibrium point of the system (8) whenever $\mathcal{R}_H^0 < 1$.

Lemma 5. *The HIV/AIDS submodel disease-free equilibrium point $E_H^0 = (\Gamma/\mu(\epsilon_2 + \mu(1 - p_2)/\epsilon_2 + \mu), (p_2\Gamma/\epsilon_2 + \mu), 0, 0, 0)$ is globally asymptotically stable if $\mathcal{R}_H^0 < 1$ and the two sufficient conditions given in Lemma 4 are satisfied.*

Proof. To prove Lemma 5, let us apply Lemma 4 on the HIV/AIDS infection submodel (8), and we have determined the following matrices:

$$\begin{aligned} \frac{dX}{dt} &= I(X, V) = \begin{bmatrix} (1 - p_2)\Gamma + \epsilon_2 H_P - (\lambda_H + \mu)S \\ \epsilon_2 \Gamma - (\epsilon_2 + \mu)H_P \end{bmatrix}, \\ \frac{dV}{dt} &= J(X, V) = \begin{bmatrix} \lambda_H S - (\mu + \xi_1 + \tau)H_I \\ \tau H_I - (\mu + \mu_1 + \xi_2)H_A \\ \xi_1 H_I + \xi_1 H_A - \mu T_{HA} \end{bmatrix}, \\ I(X^0, 0) &= \begin{bmatrix} (1 - p_2)\Gamma + \epsilon_2 H_P^0 - \mu S^0 \\ \epsilon_2 \Gamma - (\epsilon_2 + \mu)H_P^0 \end{bmatrix}, \end{aligned} \tag{15}$$

where

$$X^0 = (S^0, H_P^0) = \left(\frac{\Gamma}{\mu} \left(\frac{\epsilon_2 + \mu(1 - p_2)}{\epsilon_2 + \mu} \right), \frac{p_2 \Gamma}{\epsilon_2 + \mu} \right) \tag{16}$$

is globally stable which satisfies condition (i) of Lemma 4 and

$$B = D_V J(X^0, 0) = \begin{bmatrix} \frac{\beta_1 S^0}{N_1^0} - (\mu + \xi_1 + \tau) & \frac{\beta_1 \alpha S^0}{N_1^0} & 0 \\ \tau & -(\mu + \mu_1 + \xi_2) & 0 \\ \xi_1 & \xi_2 & -\mu \end{bmatrix}. \tag{17}$$

After a number of steps of computations, we have determined the result given by

$$\begin{aligned} \dot{J}(X, V) &= \begin{bmatrix} \dot{J}_1(X, V) \\ \dot{J}_2(X, V) \\ \dot{J}_3(X, V) \end{bmatrix} = \begin{bmatrix} \frac{\beta_1 S^0}{N_1^0} H_I + \frac{\beta_1 \alpha S^0}{N_1^0} H_A - \frac{\beta_1}{N_1} H_I S - \frac{\beta_1 \alpha}{N_1} H_A S \\ 0 \\ 0 \end{bmatrix} \\ &= \begin{bmatrix} \beta_1 H_I \left(\frac{S^0}{N_1^0} - \frac{S}{N_1} \right) + \beta_1 \alpha H_A \left(\frac{S^0}{N_1^0} - \frac{S}{N_1} \right) \\ 0 \\ 0 \end{bmatrix}. \end{aligned} \tag{18}$$

From the definitions of state variables and total population, we can justify the inequality $S \leq N_1$ that implies $S/N_1 \leq S^0/N_1^0$ and hence $\dot{J}_1(X, V) \geq 0$, which satisfies criteria (ii) of Lemma 4; thus, the HIV/AIDS submodel (8) disease-free equilibrium point $E_H^0 = (S^0, H_P^0, H_I^0, H_A^0, T_{HA}^0) = (\Gamma/\mu(\epsilon_2 + \mu(1 - p_2)/\epsilon_2 + \mu), (p_2\Gamma/\epsilon_2 + \mu), 0, 0, 0)$ is globally asymptotically stable if $\mathcal{R}_H^0 < 1$.

Epidemiologically, it means whenever $\mathcal{R}_H^0 < 1$, the HIV/AIDS-only disease dies out while the total population increases. \square

3.3. Qualitative Analysis of Pneumonia Infection Submodel.

Now, making all the state variables corresponding to HIV/AIDS infection in the full model (4) as $H_P = H_I = H_A = C = T_{HA} = 0$, we have the pneumonia submodel given by

$$\begin{aligned} \frac{dS}{dt} &= (1 - p_1)\Gamma + \epsilon_1 P_P + \eta P_R - (\lambda_P + \mu)S, \\ \frac{dP_P}{dt} &= p_1 \Gamma - (\epsilon_1 + \mu)P_P, \\ \frac{dP_I}{dt} &= \lambda_P S - (\mu + \mu_2 + \gamma)P_I, \\ \frac{dP_T}{dt} &= \gamma P_I - (\mu + \eta)P_T, \end{aligned} \tag{19}$$

with force of infection illustrated by

$$\lambda_p = \frac{\beta_2}{N_2} P_I(t), \tag{20}$$

and with initial data $S(0) > 0, P_p(0) \geq 0, P_I(0) \geq 0, P_T(0) \geq 0$, total number of human beings involved is given by $N_2(t) = S(t) + P_p(t) + P_I(t) + P_T(t)$.

3.3.1. Local Stability of Disease-Free Equilibrium Point. The pneumonia submodel (19) disease-free equilibrium point is computed by making the model equations equal to zero, where $P_I = P_T = 0$. Thus, the pneumonia submodel disease-free equilibrium point is given by $E_p^0 = (S^0, P_p^0, P_I^0, P_T^0) = (\Gamma / \mu(\varepsilon_1 + \mu(1 - p_1) / \varepsilon_1 + \mu), p_1 \Gamma / \varepsilon_1 + \mu, 0, 0)$.

The pneumonia submodel (19) basic reproduction number is the estimated number of new pneumonia-infected individuals produced by one infectious individual in a community. Similarly, using the same criteria stated in [22], we computed the pneumonia reproduction number given by $\mathcal{R}_p^0 = \beta_2(\mu(1 - p_1) + \varepsilon_1) / (\mu + \mu_2 + \gamma)(\mu + \varepsilon_1)$.

The pneumonia submodel (19) basic reproduction number is defined as the estimated number of secondary infected individuals produced by single infectious individual during its entire period of infectiousness throughout the whole susceptible population, and using the same criteria, the disease-free equilibrium point given by $E_p^0 = (S^0, P_p^0, P_I^0, P_T^0) = (\Gamma / \mu(\varepsilon_1 + \mu(1 - p_1) / \varepsilon_1 + \mu), (p_1 \Gamma / \varepsilon_1 + \mu), 0, 0)$ is locally asymptotically stable whenever $\mathcal{R}_p^0 < 1$ and unstable whenever $\mathcal{R}_p^0 > 1$.

3.3.2. Existence and Uniqueness of Endemic Equilibrium Point. The endemic equilibrium points of the pneumonia submodel given in equation (19) are computed by making the right-hand side of the system as zero, and after some computations, we have determined that

$$\lambda_p^* = -\frac{B_0}{B_1} = \frac{\Gamma K_2 K_3^2 (p_2 K_1 K_2 + \varepsilon_1 p_1 K_2) [\mathcal{R}_p^0 - 1]}{p_1 \Gamma K_2 K_3 (K_2 K_3 - \eta \gamma) + p_2 \Gamma K_1 K_2 K_3 (K_3 + \gamma) + \varepsilon_1 p_1 \Gamma K_2 K_3 (K_3 + \gamma)} > 0 \tag{26}$$

if and only if $\mathcal{R}_p^0 > 1$ since each of the parameters is positive. Thus, the pneumonia submodel given in equation (19) has a unique positive endemic equilibrium point only whenever $\mathcal{R}_p^0 > 1$.

Lemma 6. *The pneumonia submodel given in equation (19) has a unique positive endemic equilibrium if and only if $\mathcal{R}_p^0 > 1$.*

3.3.3. Global Asymptotic Stability of Disease-Free Equilibrium Point

$$\begin{aligned} S^* &= \frac{(1 - p_1) \Gamma K_1 K_2 K_3 + \varepsilon_1 p_1 \Gamma K_2 K_3}{K_1 K_2 K_3 (\lambda_p^* + \mu) - K_1 \eta \gamma \lambda_p^*}, \\ P_p^* &= \frac{p_1 \Gamma}{K_1}, P_I^* = \frac{(1 - p_1) \Gamma K_1 K_2 K_3 \lambda_p^* + \varepsilon_1 p_1 \Gamma K_2 K_3 \lambda_p^*}{K_1 K_2^2 K_3 (\lambda_p^* + \mu) - K_1 K_2 \eta \gamma \lambda_p^*}, \\ P_T^* &= \frac{(1 - p_1) \Gamma K_1 K_2 K_3 \gamma \lambda_p^* + \varepsilon_1 p_1 \Gamma K_2 K_3 \gamma \lambda_p^*}{K_1 K_2^2 K_3^2 (\lambda_p^* + \mu) - K_1 K_2 K_3 \eta \gamma \lambda_p^*}, \end{aligned} \tag{21}$$

where $K_1 = \varepsilon_1 + \mu, K_2 = \gamma + \mu + \mu_2$, and $K_3 = \mu + \eta$.

We substitute P_I^* stated in equation (21) in equation (20), we computed as $N_2^* \lambda_p^* = \beta_2 P_I^*$ and gives us the result

$$\begin{aligned} &(1 - p_1) \Gamma K_1 K_2^2 K_3^2 + \varepsilon_1 p_1 \Gamma K_2^2 K_3^2 + p_1 \Gamma K_2^2 K_3^2 \lambda_p^* \\ &+ p_1 \Gamma K_2^2 K_3^2 \mu + (1 - p_1) \Gamma K_1 K_2 K_3^2 \lambda_p^* - p_1 \Gamma K_2 K_3 \eta \gamma \lambda_p^* \\ &+ \varepsilon_1 p_1 \Gamma K_2 K_3^2 \lambda_p^* + (1 - p_1) \Gamma K_1 K_2 K_3 \gamma \lambda_p^* + \varepsilon_1 p_1 \Gamma K_2 K_3 \gamma \lambda_p^* \\ &- \beta_2 (1 - p_1) \Gamma K_1 K_2 K_3^2 - \beta_2 \varepsilon_1 p_1 \Gamma K_2 K_3^2 = 0. \end{aligned} \tag{22}$$

Rearranging (22), we have derived the nonzero linear equation.

$$B_1 \lambda_p^* + B_0 = 0. \tag{23}$$

where

$$\begin{aligned} B_1 &= p_1 \Gamma K_2 K_3 (K_2 K_3 - \eta \gamma) \\ &+ (1 - p_1) \Gamma K_1 K_2 K_3 (K_3 + \gamma) \\ &+ \varepsilon_1 p_1 \Gamma K_2 K_3 (K_3 + \gamma) > 0, \end{aligned} \tag{24}$$

$$B_0 = \Gamma K_2 K_3^2 ((1 - p_1) K_1 K_2 + \varepsilon_1 p_1 K_2) [1 - \mathcal{R}_p^0] < 0 \tag{25}$$

if and only if $\mathcal{R}_p^0 > 1$ since each parameter has a positive value. Computing the expression in equation (23), we have obtained the result given by

Lemma 7. *The pneumonia submodel (19) disease-free equilibrium point given by the expression*

$E_p^0 = ((1 - p_1) \Gamma (\varepsilon_1 + \mu) + \varepsilon_1 p_1 \Gamma / \mu (\varepsilon_1 + \mu), (p_1 \Gamma / \varepsilon_1 + \mu), 0, 0)$ *is globally asymptotically stable if and only if $\mathcal{R}_p^0 < 1$ and the two sufficient conditions given in Lemma 4 holds.*

Proof. Using the criteria stated by Lemma 4 above on the pneumonia submodel (19) and setting $X \in \mathbb{R}^2$ be the components of noninfected individuals and $V \in \mathbb{R}^2$ be the

components of infected individuals including recovery class. Then, we have determined the following matrices:

$$\begin{aligned} \frac{dX}{dt} &= I(X, V) = \begin{bmatrix} (1-p_1)\Gamma + \varepsilon_1 P_p + \eta P_T - (\lambda_p + \mu)S \\ p_1\Gamma - (\varepsilon_1 + \mu)P_p \end{bmatrix}, \\ \frac{dV}{dt} &= J(X, V) = \begin{bmatrix} \lambda_p S - (\gamma + \mu + \mu_2)P_I \\ \gamma P_I - (\mu + \eta)P_T \end{bmatrix}, \\ I(X, 0) &= \begin{bmatrix} (1-p_1)\Gamma + \varepsilon_1 P_p - \mu S \\ p_1\Gamma - (\varepsilon_1 + \mu)P_p \end{bmatrix}, \\ B = D_V J(X^0, 0) &= \begin{bmatrix} \frac{\beta_2 S^0}{S^0 + P_p^0} - (\gamma + \mu + \mu_2) & 0 \\ \gamma & -(\mu + \eta) \end{bmatrix}. \end{aligned} \tag{27}$$

After we perform some calculations, we have determined that

$$\begin{aligned} \dot{J}(X, V) &= \begin{bmatrix} \dot{J}_1(X, V) \\ \dot{J}_2(X, V) \end{bmatrix} \\ &= \begin{bmatrix} \frac{\beta_2 S^0 P_I}{S^0 + P_p^0} - \frac{\beta_2 P_I S}{N_2} \\ 0 \end{bmatrix} \\ &= \begin{bmatrix} \beta_2 P_I \left(\frac{S^0}{S^0 + P_p^0} - \frac{S}{N_2} \right) \\ 0 \end{bmatrix}. \end{aligned} \tag{28}$$

Since $S \leq S^0$, $P_p < P_p^0$, one can show that $S - S^0 \leq 1$, $P_p - P_p^0 \leq 1$, and $\dot{J}_1(X, V) \geq 0$; thus, the disease-free equilibrium point $E_p^0 = ((1-p_1)\Gamma(\varepsilon_1 + \mu) + \varepsilon_1 p_1 \Gamma / \mu(\varepsilon_1 + \mu), (p_1 \Gamma / \varepsilon_1 + \mu), 0, 0)$ of the pneumonia monoinfection model (19) is globally asymptotically stable if $\mathcal{R}_p^0 < 1$. Epidemiologi-

cally, it means whenever $\mathcal{R}_p^0 < 1$, the pneumonia-only disease dies out while the total population increases. \square

3.4. Qualitative Analysis of Pneumonia and HIV/AIDS Coinfection Model. In Sections 3.2 and 3.3, we analyzed the HIV/AIDS and pneumonia single infection models, respectively, and based on the results on these submodels now considered and analyzed the full HIV/AIDS and pneumonia coinfection model in the bounded region Ω illustrated in equation (7).

3.4.1. Stability of Disease-Free Equilibrium Point. The full coinfection model (4) disease-free equilibrium point is computed by setting each of the equations in the model equal to zero in the absence of infections and treatment such that $H_I = H_A = P_I = C = P_T = T_{HA} = 0$. Thus, after some calculations, we have determined the HIV/AIDS and pneumonia coinfection disease-free equilibrium point given by

$$\begin{aligned} E_{HP}^0 &= (S^0, P_p^0, H_p^0, H_I^0, H_A^0, P_I^0, C^0, P_T^0, T_{HA}^0) \\ &= \frac{(1-p_1-p_2)\Gamma(\varepsilon_1 + \mu)(\varepsilon_2 + \mu) + \varepsilon_1 p_1 \Gamma + \varepsilon_2 p_2 \Gamma(\varepsilon_1 + \mu)}{\mu(\varepsilon_1 + \mu)(\varepsilon_2 + \mu)}, \\ &\quad \frac{\varepsilon_1 \Gamma}{\varepsilon_1 + \mu}, \frac{p_2 \Gamma}{\varepsilon_2 + \mu}, 0, 0, 0, 0, 0, 0, 0. \end{aligned} \tag{29}$$

Similarly, using the same criteria stated in [22], the coinfection model (4) basic reproduction number denoted by \mathcal{R}_{HP}^0 is to be determined as

$$FV^{-1} = \begin{bmatrix} \frac{\beta_1(\mu(1-p_2) + \alpha_2)}{(\mu + \xi_1 + \tau)(\mu + \varepsilon_2)} + \frac{\beta_1 \mu \alpha(1-p_2) + \beta_1 \varepsilon_2 \alpha}{(\mu + \mu_1 + \xi_2)(\mu + \varepsilon_2)} & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_2(\mu(1-p_1) + \varepsilon_1)}{(\mu + \mu_2 + \gamma)(\mu + \varepsilon_1)} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}. \tag{30}$$

The coinfection model (4) basic reproduction number is the dominant eigenvalue in magnitude of the next generation matrix $F.V^{-1}$ given by

$$\begin{aligned} \mathcal{R}_{HP}^0 &= \max \{ \mathcal{R}_H^0, \mathcal{R}_P^0 \} \\ &= \max \left\{ \frac{\beta_1(\mu(1-p_2) + \alpha_2)}{(\mu + \xi_1 + \tau)(\mu + \varepsilon_2)} + \frac{\beta_1 \mu \alpha(1-p_2) + \beta_1 \varepsilon_2 \alpha}{(\mu + \mu_1 + \xi_2)(\mu + \varepsilon_2)}, \frac{\beta_2(\mu(1-p_1) + \varepsilon_1)}{(\mu + \mu_2 + \gamma)(\mu + \varepsilon_1)} \right\}, \end{aligned} \tag{31}$$

where \mathcal{R}_H^0 represent the HIV/AIDS-only basic reproduction number, \mathcal{R}_P^0 represents the pneumonia only basic reproduction number, and \mathcal{R}_{HP}^0 represents the coinfection basic reproduction numbers, respectively.

In the similar manner of the single infections, the basic reproduction number of HIV/AIDS and pneumonia coinfection is defined as the estimated number of secondary infectious produced by one coinfecting individual during its entire period of infectiousness in the whole susceptible population, and the disease-free equilibrium point given by

$$E_{HP}^0 = (S^0, P_p^0, H_p^0, H_I^0, H_A^0, P_I^0, C^0, P_T^0, T_{HA}^0) = \frac{(1 - p_1 - p_2)\Gamma(\varepsilon_1 + \mu)(\varepsilon_2 + \mu) + \varepsilon_1 p_1 \Gamma + \varepsilon_2 p_2 \Gamma(\varepsilon_1 + \mu)}{\mu(\varepsilon_1 + \mu)(\varepsilon_2 + \mu)}, \left(\frac{\varepsilon_1 \Gamma}{\varepsilon_1 + \mu}, \frac{p_2 \Gamma}{\varepsilon_2 + \mu}, 0, 0, 0, 0, 0\right) \tag{32}$$

is locally asymptotically stable if and only if $\mathcal{R}_{HP}^0 < 1$ and unstable if $\mathcal{R}_{HP}^0 > 1$.

3.4.2. *Endemic Equilibrium of the Model (4).* The full coinfection model (4) endemic equilibrium points are determined by setting each differential equation equal to zero, and we obtained the result given by

$$\begin{aligned} S^* &= \frac{(1 - p_1 - p_2)\Gamma + \varepsilon_1 P_p^* + \varepsilon_2 H_p^* + \eta P_T^*}{(\lambda_H^* + \lambda_P^* + \mu)}, \\ P_p^* &= \frac{p_1 \Gamma}{(\delta \lambda_H^* + \varepsilon_1 + \mu)}, \\ H_p^* &= \frac{p_2 \Gamma}{(\delta \varepsilon_2 + \mu + \sigma \lambda_P^*)}, \\ H_I^* &= \frac{\lambda_H^* S^* + \delta \lambda_H^* P_p^*}{(\mu + \xi_1 + \tau + \phi_1 \lambda_P^*)}, \\ H_A^* &= \frac{\tau H_I^*}{(\mu + \mu_1 + \xi_2 + \phi_2 \lambda_P^*)}, \\ P_I^* &= \frac{\lambda_P^* S^* + \delta \lambda_P^* H_p^*}{(\gamma + \mu + \mu_2 + \varphi \lambda_H^*)}, \\ C^* &= \frac{\varphi \lambda_H^* P_I^* + \phi_1 \lambda_P^* H_I^* + \phi_2 \lambda_P^* H_A^* + \rho \lambda_P^* T_{HA}^*}{(\mu + \mu_3 + \theta)}, \\ P_T^* &= \frac{\gamma P_I^*}{(\mu + \eta)}, \\ T_{HA}^* &= \frac{\xi_1 H_I^* + \xi_2 H_A^* + \theta C^*}{(\rho \lambda_P^* + \mu)}. \end{aligned} \tag{33}$$

The coinfection model (4) we proposed is highly non-linear, and hence, the explicit computation of the endemic equilibrium point(s) in terms of the illustrated model parameters is difficult analytically; however, based on the

previous analyses of the HIV/AIDS and pneumonia sub-models, the endemic equilibrium point(s) represented by $E_{HP}^* = (S^*, P_p^*, H_p^*, H_I^*, H_A^*, P_I^*, C^*, P_T^*, T_{HA}^*)$ exists whenever $\mathcal{R}_H^0 > 1$ and $\mathcal{R}_P^0 > 1$, i.e., $\mathcal{R}_{HP}^0 > 1$. The stability is shown in the numerical simulation part.

3.4.3. *Possibility of Existence of Backward Bifurcation for the Coinfection Dynamical System (4).* Let $S = v_1, P_p = v_2, H_p = v_3, H_I = v_4, H_A = v_5, P_I = v_6, C = v_7, P_T = v_8$, and $T_{HA} = v_9$, and the total human population is given by $N = v_1 + v_2 + v_3 + v_4 + v_5 + v_6 + v_7 + v_8 + v_9$.

Moreover, by the vector representation $V = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9)^T$, the dynamical system (4) will be rewritten as $(dV/dt) = H(V)$ with $H = (h_1, h_2, h_3, h_4, h_5, h_6, h_7, h_8, h_9)^T$ and

$$\begin{aligned} \frac{dv_1}{dt} &= h_1 = (1 - p_1 - p_2)\Gamma + \varepsilon_1 v_2 + \varepsilon_2 v_3 + \eta v_8 - (\lambda_H + \lambda_P + \mu)v_1, \\ \frac{dv_2}{dt} &= h_2 = p_1 \Gamma - (\delta \lambda_H + \varepsilon_1 + \mu)v_2, \\ \frac{dv_3}{dt} &= h_3 = p_2 \Gamma - (\varepsilon_2 + \mu + \sigma \lambda_P)v_3, \\ \frac{dv_4}{dt} &= h_4 = \lambda_H v_1 + \delta \lambda_H v_2 - (\mu + \tau + \xi_1 + \phi_1 \lambda_P)v_4, \\ \frac{dv_5}{dt} &= h_5 = \tau v_4 - (\mu + \mu_1 + \xi_2 + \phi_2 \lambda_P)v_5, \\ \frac{dv_6}{dt} &= h_6 = \lambda_P v_1 + \sigma \lambda_P v_3 - (\gamma + \mu + \mu_2 + \varphi \lambda_H)v_6, \\ \frac{dv_7}{dt} &= h_7 = \varphi \lambda_H v_6 + \phi_1 \lambda_P v_4 + \phi_2 \lambda_P v_5 + \rho \lambda_P v_9 - (\mu + \mu_3 + \theta)v_7, \\ \frac{dv_8}{dt} &= h_8 = \gamma v_6 - (\mu + \eta)v_8, \\ \frac{dv_9}{dt} &= h_9 = \xi_1 v_4 + \xi_2 v_5 + \theta v_7 - \rho \lambda_P v_9 - \mu v_9, \end{aligned} \tag{34}$$

where $\lambda_H = (\beta_1/N)[v_4 + \alpha v_5 + \vartheta v_7]$ for $1 \leq \rho_1 < \infty$ and $\lambda_P = (\beta_2/N)[v_6 + \omega v_7]$ for $1 \leq \omega < \infty$.

Then, the Jacobian matrix of the new dynamical system given in (22) at E_{HP}^0 , represented by $J(E_{HP}^0)$ and determined by

$$J(E_{HP}^0) = \begin{pmatrix} -\mu & \varepsilon_1 & \varepsilon_2 & E_1 & E_2 & E_3 & E_4 & \eta & 0 \\ 0 & -(\varepsilon_1 + \mu) & 0 & E_5 & E_6 & 0 & E_7 & 0 & 0 \\ 0 & 0 & -(\varepsilon_2 + \mu) & 0 & 0 & E_8 & E_9 & 0 & 0 \\ 0 & 0 & 0 & E_{10} & E_{11} & 0 & E_{12} & 0 & 0 \\ 0 & 0 & 0 & \tau & -(\mu + \mu_1 + \xi_2) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & E_{13} & E_{14} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(\mu + \mu_3 + \theta) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma & 0 & -(\mu + \eta) & 0 \\ 0 & 0 & 0 & \xi_1 & \xi_2 & 0 & \theta & 0 & -\mu \end{pmatrix}. \tag{35}$$

where $E_1 = -(\beta_1/N^0)v_1^0, E_2 = -(\beta_1 \alpha/N^0)v_1^0, E_3 = -(\beta_2/N^0)v_1^0, E_4 = -(\beta_1/N^0)\vartheta v_1^0 - (\beta_2/N^0)\omega v_1^0, E_5 = -(\beta_1/N^0)v_2^0, E_6 = (\beta_1/$

$$N^0)\delta\alpha v_2^0, E_7 = -(\beta_1/N^0)\rho_1 v_2^0, E_8 = -(\beta_2/N^0)v_3^0, E_9 = -(\beta_2/N^0)\omega v_3^0, E_{10} = (\beta_1/N^0)v_1^0 + (\beta_1/N^0)\delta v_2^0 - (\mu + \tau + \xi_1), E_{11} = (\beta_1/N^0)\alpha v_1^0 + (\beta_1/N^0)\delta v_2^0, E_{12} = (\beta_1/N^0)\vartheta v_1^0 + (\beta_1/N^0)\delta\vartheta v_2^0, E_{13} = (\beta_2/N^0)v_1^0 + (\beta_2/N^0)v_3^0 - (\gamma + \mu + \mu_2), E_{14} = (\beta_2/N^0)\omega v_3^0 + (\beta_2/N^0)\omega v_3^0.$$

Let us assume $\mathcal{R}_p^0 > \mathcal{R}_H^0$ without loss of the generality, and $\mathcal{R}_{HP}^0 = 1$, i.e., $\mathcal{R}_p^0 = 1$. Moreover, let $\beta_2 = \beta^*$ be a bifurcation parameter. Solving for β_2 using $\mathcal{R}_p^0 = 1$ as $\mathcal{R}_p^0 = (\beta_2(\mu(1 - p_1) + \varepsilon_1)/(\mu + \mu_2 + \gamma)(\mu + \varepsilon_1)) = 1$, we determined as $\beta^* = \beta_2 = ((\mu + \mu_2 + \gamma)(\mu + \varepsilon_1))/(\mu(1 - p_1) + \varepsilon_1)$.

Then, we compute the eigenvalues of the Jacobian matrix $J(E_{HP}^0)$ at E_{HP}^0 for $\beta_2 = \beta^*$, and we determined the eigenvalues given by $\lambda_1 = -\mu < 0$ or $\lambda_2 = -(\varepsilon_1 + \mu) < 0$ or $\lambda_3 = -(\varepsilon_2 + \mu) < 0$ or $\lambda_4 = E_{10} = (\beta_1/N^0)v_1^0 + (\beta_1/N^0)\delta v_2^0 - (\mu + \tau + \xi_1) = (\mu + \tau + \xi_1)[\mathcal{R}_H^0 - 1] < 0$ if $\mathcal{R}_H^0 < 1$ or $\lambda_5 = 0$ or $\lambda_6 = E_{13} = (\beta_2/N^0)v_1^0 + (\beta_2/N^0)v_3^0 - (\gamma + \mu + \mu_2) = (\gamma + \mu + \mu_2)[\mathcal{R}_p^0 - 1] < 0$ if $\mathcal{R}_p^0 < 1$ or $\lambda_7 = -(\mu + d_3 + \theta) < 0$ or $\lambda_8 = -(\mu + \eta) < 0$ or $\lambda_9 = -\mu < 0$. From the computations, we observed that all the eigenvalues are negative if $\mathcal{R}_{HP}^0 < 1$. We apply the centre manifold theory stated in [31], to illustrate that the dynamical system (4) undergoes the phenomenon of forward bifurcation at $\mathcal{R}_p^0 = 1$. For the eigenvectors of the Jacobian J_{β^*} , for the case $\mathcal{R}_p^0 = 1$, the right eigenvectors at $\beta_2 = \beta^*$ corresponding to the zero eigenvalue given by $y = (y_1, y_2, y_3, y_4, y_5, y_6, y_7, y_8, y_9)^T$ are

$$y_1 = \frac{\varepsilon_2 E_7 (\mu + \eta) y_5 + (\varepsilon_2 + \mu) (\mu + \eta) E_2 y_5 + (\varepsilon_2 + \mu) \eta \gamma y_5}{\mu (\varepsilon_2 + \mu) (\mu + \eta)}, y_2 = 0, y_3 = \frac{E_7}{\varepsilon_2 + \mu} y_5, y_4 = 0, y_5 = y_5 > 0, y_6 = 0, y_7 = \frac{\kappa}{\mu + \eta} u_5, y_8 = 0, y_9 = 0. \tag{36}$$

Left eigenvectors corresponding to the zero eigenvalue at $\beta_2 = \beta_2^*$ that holds $y.z = 1$, given by

$$z = (z_1, z_2, z_3, z_4, z_5, z_6, z_7, z_8, z_9) \text{ and } z_1 = z_2 = z_3 = z_4 = z_6 = z_7 = z_8 = z_9 = 0 \text{ and } z_5 = z_5 > 0. \tag{37}$$

After many steps of calculations and simplification, we determined the bifurcation coefficients given by a and b as

$$a = 2z_5 y_1 y_5 \frac{\partial^2 h_5(0,0)}{\partial v_1 \partial v_5} + 2z_5 y_3 y_5 \frac{\partial^2 h_5(0,0)}{\partial v_2 \partial v_5} = 2\beta_2^* z_5 y_5 [y_1 + y_3], \\ = 2\beta_2^* z_5 y_5^2 \left[\frac{-\varepsilon_2 \beta_2^* v_3^0 (\mu + \eta) - (\varepsilon_2 + \mu) (\mu + \eta) \beta_2^* v_1^0 - (\varepsilon_2 + \mu) \eta \gamma - \mu (\mu + \eta) \beta_2^* v_3^0}{\mu (\varepsilon_2 + \mu) (\mu + \eta)} \right]. \tag{38}$$

Thus,

$$a = -2\beta_2^* z_5 y_5^2 \left[\frac{\varepsilon_2 \beta_2^* v_3^0 (\mu + \eta) + (\varepsilon_2 + \mu) (\mu + \eta) \beta_2^* v_1^0 + (\varepsilon_2 + \mu) \eta \gamma + \mu (\mu + \eta) \beta_2^* v_3^0}{\mu (\varepsilon_2 + \mu) (\mu + \eta)} \right] < 0, \\ b = z_5 y_5 \frac{\partial^2 h_5(0,0)}{\partial v_5 \partial \beta_2} = z_5 y_5 (v_3^0 + v_1^0) > 0. \tag{39}$$

Therefore, using the criteria stated in [31], the HIV/AIDS and pneumonia coinfection dynamical system (4) do not exhibit the phenomenon of backward bifurcation whenever $\mathcal{R}_{HP}^0 = \mathcal{R}_p^0 = 1$. Thus, there is no positive endemic equilibrium point rather there is only the coinfection model disease-free equilibrium point in the region at which $\mathcal{R}_{HP}^0 < 1$.

4. Sensitivity and Numerical Analysis

In this section, we need to verify the qualitative analysis results performed in Section 3, and we have performed several sensitivity and numerical analyses. In this study, to obtain more relevant model parameters illustrated in Table 3, we have observed and reviewed different research studies based on the mathematical modelling on infectious diseases, and for some other parameters, we assumed realistic values for the purpose of sensitivity and numerical analyses and illustrations.

4.1. The Coinfection Model Sensitivity Analysis

Definition 8. Let z be variable; then, the normalized forward sensitivity index of z which depends differentially on a parameter ϑ is defined as $SEI D(\vartheta) = (\partial z / \partial \vartheta) * (\vartheta / z)$ [27].

The sensitivity indices we have calculated in this subsection allow to investigate the relative significance of various parameters in the proposed HIV/AIDS and pneumonia coinfection spreading dynamics. The parameter which has larger magnitude than that of all other parameters is the most sensitive parameter. Now, we can compute the sensitivity indices in terms of the model basic reproduction numbers \mathcal{R}_H^0 and \mathcal{R}_p^0 since $\mathcal{R}_{HP}^0 = \max \{ \mathcal{R}_H^0, \mathcal{R}_p^0 \}$.

Applying the baseline parameter values given in Table 3, we have derived Tables 4 and 5 to show the sensitivity indices of the model parameters.

In this study, with the baseline parameter values given in Table 3, we have computed $\mathcal{R}_H^0 = 1.91$ which implies that HIV/AIDS spreads in the community, and we also have determined the indices in Table 4. Sensitivity analysis results show that the HIV/AIDS spreading rate β_1 has the highest impact on the HIV/AIDS only infection basic reproduction number (\mathcal{R}_H^0).

Similarly, using baseline parameter values given in Table 3, we have computed $\mathcal{R}_p^0 = 3.86$ which implies that pneumonia is spreading throughout the community, and we also have computed the sensitivity indices as shown in Table 5. Sensitivity analysis results show that the foremost sensitive positive parameter is the pneumonia spreading rate β_2 . Using Tables 4 and 5, biologically, we can conclude

TABLE 3: Parameter values used for sensitivity and numerical analyses.

Parameter	Value	Reference
Γ	1000 humans/day	[21]
μ	$(1/64.5 \times 365)/\text{day}$	[6]
ε_1	0.005/day	[7]
ε_2	0.0004/day	[7]
μ_1	0.00034/day	[7]
μ_2	0.057/day	[13]
θ	0.0021/day	Assumed
Φ_1, Φ_2	1 no unit	Assumed
δ, σ	1 no unit	Assumed
ξ_1	0.0023/day	[25]
μ_3	0.15/day	Assumed
φ	1 no unit	Assumed
η	0.1/day	[13]
β_1	0.3425/day	[6]
β_2	0.0115/day	[13]
p_1	0.597/day	[7]
p_2	0.006/day	[7]
ξ_2	0.13/day	[5]
γ	0.2/day	[13]
φ, α, ω	1 no unit	Assumed

TABLE 4: Sensitivity indices for $\mathcal{R}_{HP}^0 = \mathcal{R}_H^0$.

Sensitivity index	Value
SEID(β_1)	+1
SEID(ε_2)	+0.01
SEID(p_2)	-0.67
SEID(μ_1)	+0.38
SEID(τ)	+0.31
SEID(ξ_1)	-0.56
SEID(ξ_2)	-0.45
SEID(α)	+0.37

TABLE 5: Sensitivity indices for $\mathcal{R}_{HP}^0 = \mathcal{R}_P^0$.

Sensitivity index	Values
SEID(β_2)	+1
SEID(μ_2)	+0.01
SEID(γ)	-0.60
SEID(ε_1)	0.21
SEID(p_1)	-0.72

that the most sensitive parameters are the HIV/AIDS and pneumonia spreading rates.

In this subsection, we performed numerical simulation illustrated in Figure 2 to investigate the HIV/AIDS and pneumonia coinfection model parameters sensitivity indices with respect to the coinfection reproduction number, and from the result, we observed that both the HIV and pneumonia spreading rates β_1 and β_2 , respectively, are epidemiologically the most sensitive parameters having a direct proportionality with the HIV/AIDS and pneumonia reproduction numbers, respectively. Furthermore, the HIV infection protection portion (p_2) and pneumonia infection protection portion (p_2) and treatment rates are more sensitive parameters having an indirect proportionality with the associated reproduction number.

4.2. The Coinfection Model Numerical Simulations. In this part, we carried out simulations for the HIV/AIDS and pneumonia codynamics by using the parameter baseline values given in Table 3 mainly to verify the qualitative analysis performed throughout Section 3. To investigate the numerical results of the constructed coinfection model (4), the initial data should have nonnegative values because the number of people in each class cannot be negative. In this subsection, the numerical simulations were conducted with MATLAB by applying the Runge-Kutta ODE45 method. Throughout this subsection, we examine the behavior of the coinfection model solutions and their convergence to the corresponding equilibrium points, investigate the impact of the model parameters on the diseases spreading in the community, and more specifically examine the effect of protection and treatment strategies on the diseases spreading dynamics. In order to simulate the HIV/AIDS and pneumonia coinfection model (4), set the nonnegative initial data $(S(0), P_P(0), H_P(0), H_I(0), H_A(0), P_I(0), C(0), P_T(0), T_{HA}(0)) = (1500, 350, 250, 150, 100, 200, 90, 85, 70)$.

4.2.1. Simulation to Show Behaviour Solutions Whenever $\mathcal{R}_{HP}^0 < 1$. The numerical trajectories given in Figure 3 show the behavior of the coinfection model solutions over time whenever $\mathcal{R}_{HP}^0 < 1$. From this numerical result, we can justify the qualitative results proved in Section 3.3.1. The HIV/AIDS and pneumonia dynamical system (4) effective reproduction number is calculated as $\mathcal{R}_{HP}^0 = 0.46$. We also observed that after 100 days, the coinfection dynamical system solutions converge to the disease-free equilibrium point if $\mathcal{R}_{HP}^0 = \max\{\mathcal{R}_H^0, \mathcal{R}_P^0\} = \max\{0.46, 0.87\} = 0.87 < 1$. Epidemiologically, it means that the coinfection outbreaks in the population will be eliminated in the near future.

4.2.2. Simulation to Show Solution Trajectories Whenever $\mathcal{R}_{HP}^0 > 1$. In this subsection, we have carried out the numerical simulation of the coinfection dynamical to examine the solution trajectory behavior whenever $\mathcal{R}_{HP}^0 = 3.86 > 1$. Figure 4 shows that the simulation trajectories will converge to the model endemic equilibrium point whenever the coinfection model computed effective reproduction number is $\mathcal{R}_{HP}^0 = \max\{\mathcal{R}_H^0, \mathcal{R}_P^0\} = \max\{1.91, 3.86\} = 3.86 > 1$. It means that

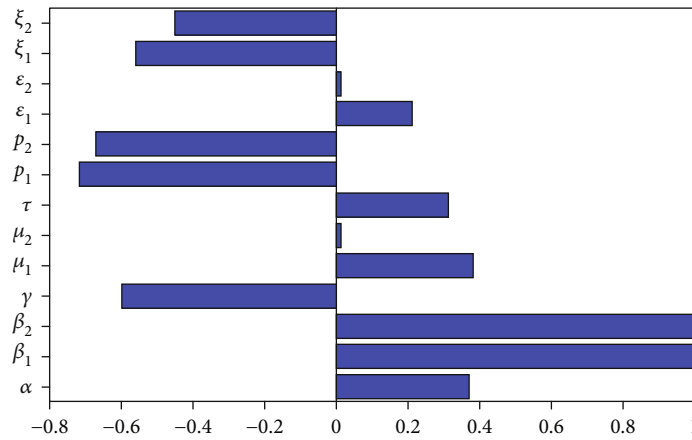


FIGURE 2: Simulation of sensitivity indices of parameters with respect to \mathcal{R}_{HP}^0 .

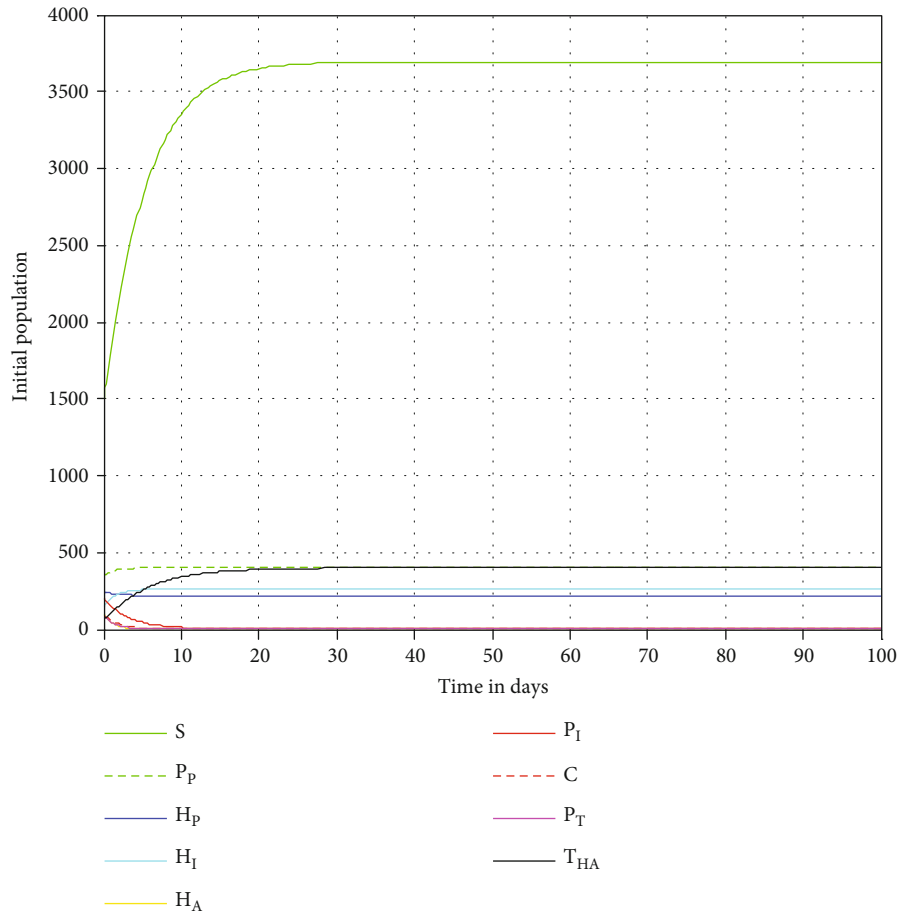


FIGURE 3: Simulation of the dynamical system (4) solutions at $\mathcal{R}_{HP}^0 = 0.46 < 1$.

the HIV/AIDS and pneumonia coinfection model (4) solutions approach to its endemic equilibrium point if $\mathcal{R}_{HP}^0 = 3.86 > 1$.

4.2.3. *Simulation to Show the Impact of HIV Spreading Rate on Pneumonia Transmission.* Numerical simulation illustrated in Figure 5 investigates the impact of HIV spreading rate β_1 on the number of coinfecting people denoted by C .

From the result, we observed that when we increase the value of β_1 , then the number of coinfecting people in the population increases. Whenever HIV spreading rate β_1 increases from a value 0.00001 to a value 0.8, then the HIV/AIDS and pneumonia coinfection population denoted by C is highly increases, and thus, we recommend for the stakeholders to exert their optimum effort on decrease the HIV spreading rate with applying suitable intervention mechanisms.

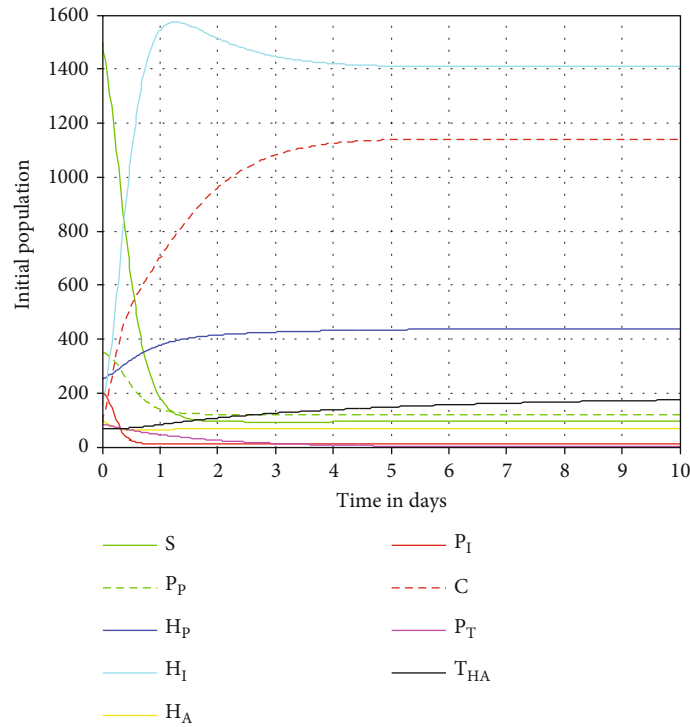


FIGURE 4: Simulation of the coinfection system (4) at $\mathcal{R}_{HP}^0 = 3.86 > 1$.

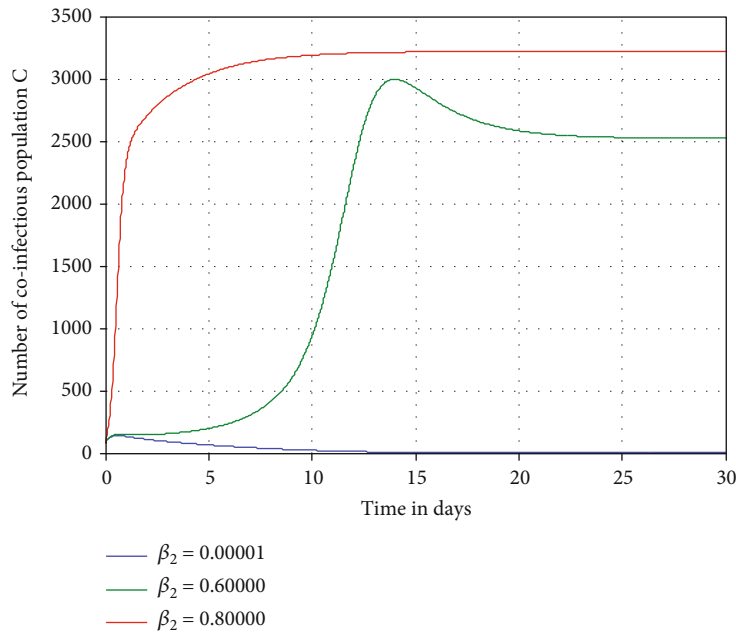


FIGURE 5: Effect of HIV spreading rate β_1 on the coinfection C.

4.2.4. *Simulation to Investigate the Impact of Pneumonia Spreading on the Coinfection.* Numerical simulation illustrated in Figure 6 investigates the impact of pneumonia spreading rate β_2 on the number of coinfectious people denoted by C. From the result, we observed that increasing the value of β_2 leads to an increase of the number of coinfectious people in the population. Consequently, increasing pneu-

monia transmission rate β_2 from 0.00001 to 0.8 leads to a highly increase of HIV/AIDS and pneumonia coinfection C.

4.2.5. *Treatment Impact on the Number of HIV-Infected Population.* In this subsection, we perform numerical simulation illustrated in Figure 7 to investigate the impact of HIV treatment (antiretroviral therapy or ART) rate (ξ_1) on the

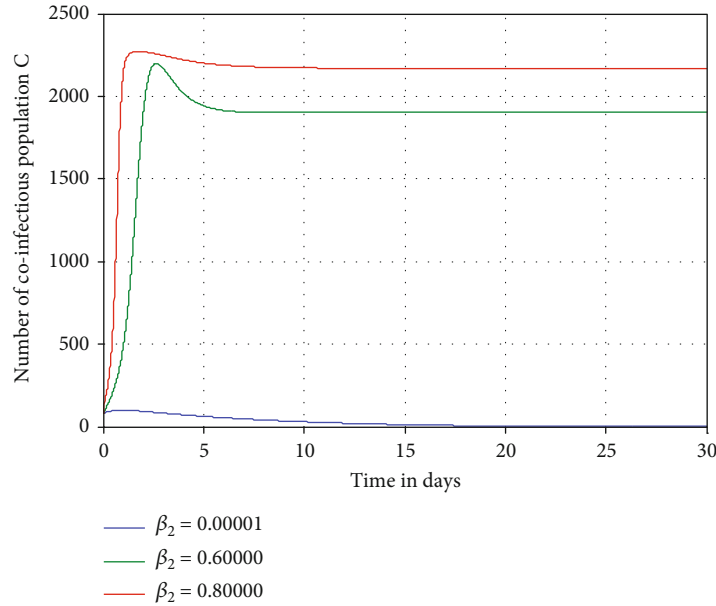


FIGURE 6: Effect of pneumonia spreading rate β_2 on the coinfection C .

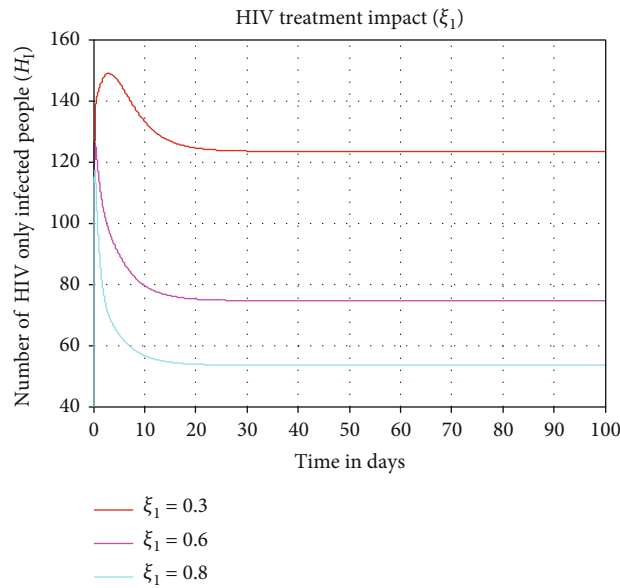


FIGURE 7: Effect of treatment on HIV-infected population.

HIV-infected population denoted by H_I . From the numerical simulation result, we observe that whenever we increase the value of HIV treatment (antiretroviral therapy or ART) rate (ξ_1) from 0.3 to 0.8, the number of HIV-infected population is going down throughout the community.

4.2.6. *Treatment Impact on the Number of AIDS Patients.* In this subsection, we perform numerical simulation illustrated in Figure 8 to investigate the impact of HIV treatment (antiretroviral therapy or ART) rate (ξ_2) on the AIDS patient population denoted by H_A . From the numerical simulation result, we observe that whenever we increase the value of

HIV treatment (antiretroviral therapy or ART) rate (ξ_2) from 0.3 to 0.8, the number of AIDS patient population is going down throughout the community.

4.2.7. *Treatment Impact on HIV/AIDS and Pneumonia Coinfection.* In this part, we simulate the state variable which represents HIV/AIDS and pneumonia coinfection dynamics illustrated in Figure 9. From the result, we observed that whenever the treatment rate θ is going up, then the number of HIV/AIDS and pneumonia population decreases in the community. Epidemiologically, it means whenever the stakeholders of human being health increase treatment

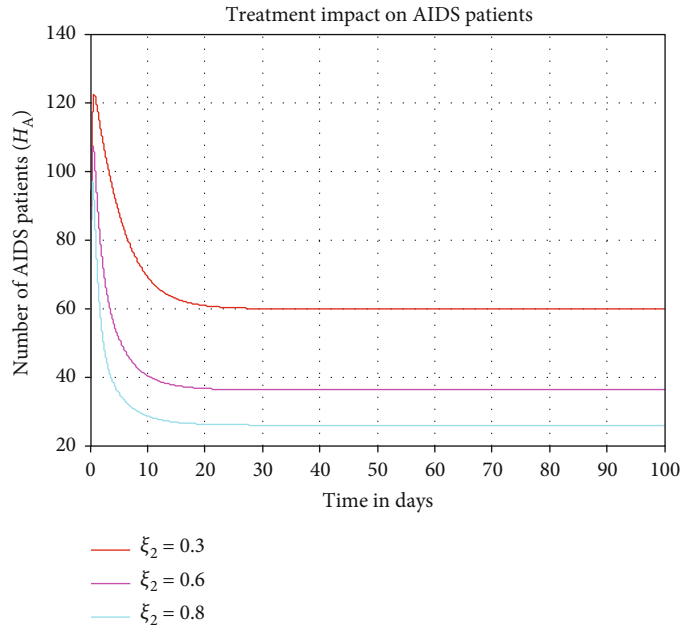


FIGURE 8: Effect of treatment on AIDS patients.

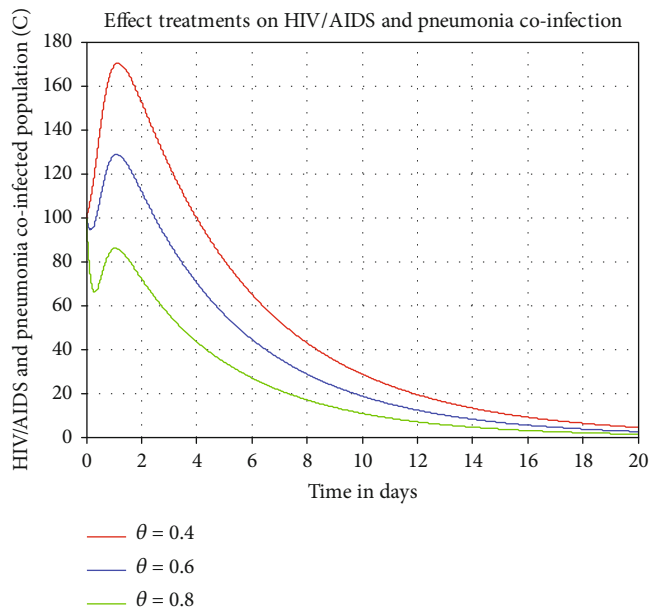


FIGURE 9: Effect of treatment rate θ on the coinfection C .

intervention strategies from the rate 0.4 to the rate 0.8, this implies that the number of HIV/AIDS and pneumonia coinfected individuals is going down.

4.2.8. *Impact of the HIV/AIDS Spreading Rate β_1 on \mathcal{R}_H^0 .* Simulation illustrated in Figure 10 investigates the influence of the HIV/AIDS spreading rate β_1 on the effective reproduction number \mathcal{R}_H^0 . Since increasing the HIV/AIDS spreading rate leads to increase, the HIV/AIDS transmission in the community health stakeholders shall introduce effective

intervention strategies to minimize the value of β_1 less than 0.829.

4.2.9. *Impact of Portion of Protection against HIV Infection p_2 on \mathcal{R}_H^0 .* The numerical simulation represented in Figure 11 illustrated that the portion p_2 of the human recruitment rate that entered to the HIV/AIDS protected class using condom intervention strategy has a significant effect on \mathcal{R}_H^0 . From the result, we observed that increasing the value of p_2 leads to a decrease in the spreading rate of

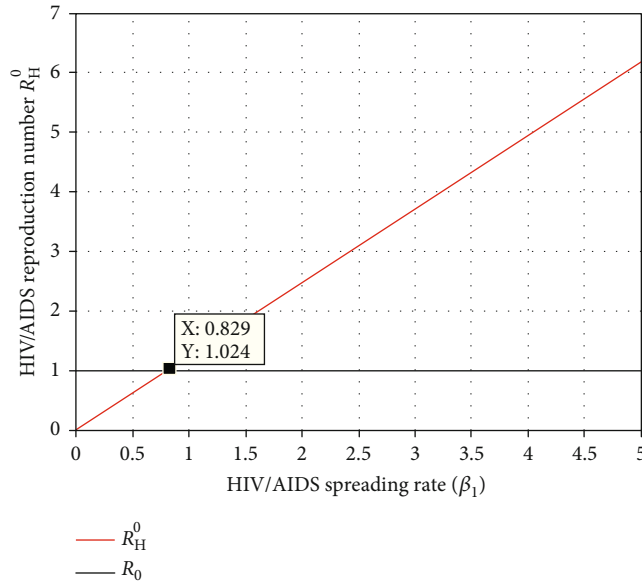


FIGURE 10: Effect of HIV/AIDS spreading rate β_1 on \mathcal{R}_H^0 .

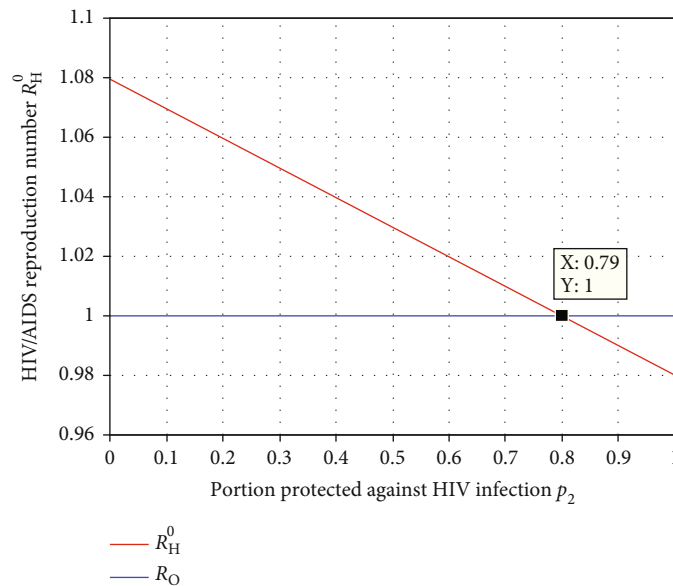


FIGURE 11: Effect of protection intervention portion p_2 on \mathcal{R}_H^0 .

HIV/AIDS in the population. And we recommend for the health stakeholders to introduce the portion of human recruitment portion p_2 ; more than 0.79 makes the effective reproduction number value \mathcal{R}_H^0 below unity.

4.2.10. *Impact of HIV Infection Treatment ξ_1 on \mathcal{R}_H^0 .* Simulation illustrated in Figure 12 shows that the HIV treatment rate ξ_1 has a significant effect on \mathcal{R}_H^0 . From the result, we observed that whenever we increase the HIV treatment rate ξ_1 , then the HIV spreading rate decreases in the population. We recommend for the health stakeholders to exert their

optimum effort to introduce the HIV treatment rate ξ_1 more than the value 0.97 to make \mathcal{R}_H^0 less than unity.

4.2.11. *Impact of Pneumonia Spreading Rate β_2 on \mathcal{R}_p^0 .* Numerical simulation illustrated in Figure 13 examined the effect of pneumonia spreading rate β_2 on the effective reproduction of pneumonia \mathcal{R}_p^0 . From the figure, we observed that increasing the value of β_2 leads to increase the effective reproduction number of the pneumonia, and whenever $\beta_2 < 0.149$, then $\mathcal{R}_p^0 < 1$. Thus, stakeholders of public health shall exert optimum effort to minimize the

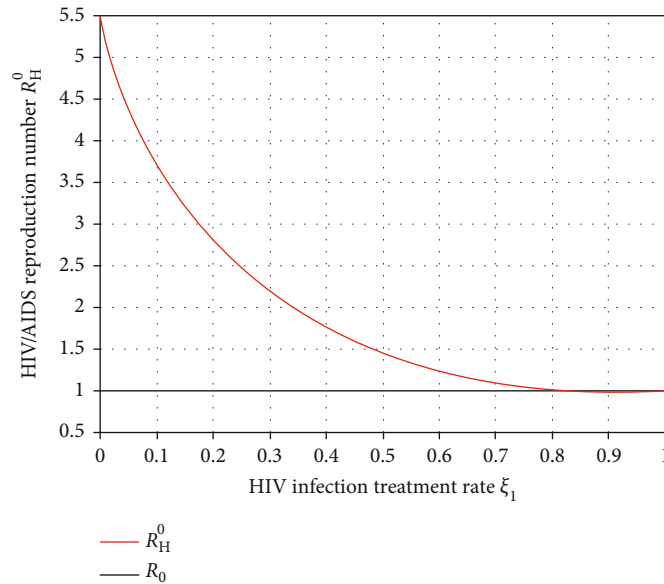


FIGURE 12: Effect of HIV treatment on \mathcal{R}_H^0 .

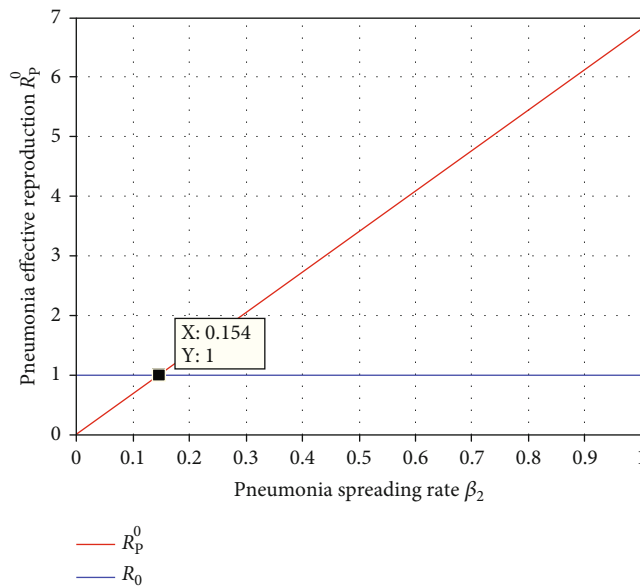


FIGURE 13: Effect of pneumonia transmission rate β_2 on \mathcal{R}_p^0 .

spreading rate β_2 for prevention and controlling of pneumonia spreading throughout the population. Epidemiologically, it means that whenever the pneumonia spreading rate increases, then the pneumonia disease increases in the community, and the disease will be eliminated from the population if $\beta_2 < 0.154$.

4.2.12. *Impact of Portion p_1 of Pneumonia Protection on \mathcal{R}_p^0 .* Numerical simulation illustrated in Figure 14 shows that the portion p_1 of pneumonia protection of the human recruitment rate has an influential impact on \mathcal{R}_p^0 . From the result, we observed that increasing the portion of

pneumonia protection decreases the pneumonia spreading throughout the population. Thus, for stakeholders, we recommend to introduce the portion p_1 of the human recruitment greater than 0.803 and to make the value of \mathcal{R}_p^0 below one.

4.2.13. *Impact of Pneumonia Treatment Rate γ on \mathcal{R}_p^0 .* Numerical simulation represented in Figure 15 illustrated that the treatment rate γ of pneumonia has a crucial indirect role on \mathcal{R}_p^0 . From the result, we observed that whenever the pneumonia treatment rate increases, then the pneumonia spreading in the population decreases. Thus, we recommend

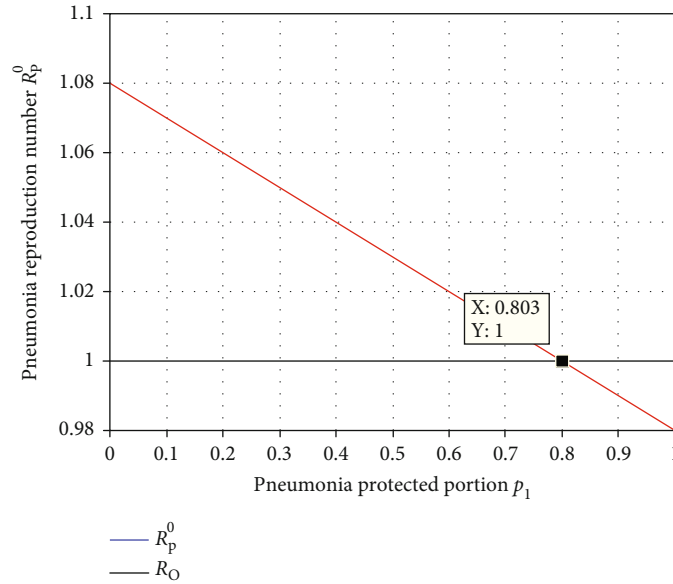


FIGURE 14: Effect of portion p_1 of pneumonia protection on \mathcal{R}_p^0 .

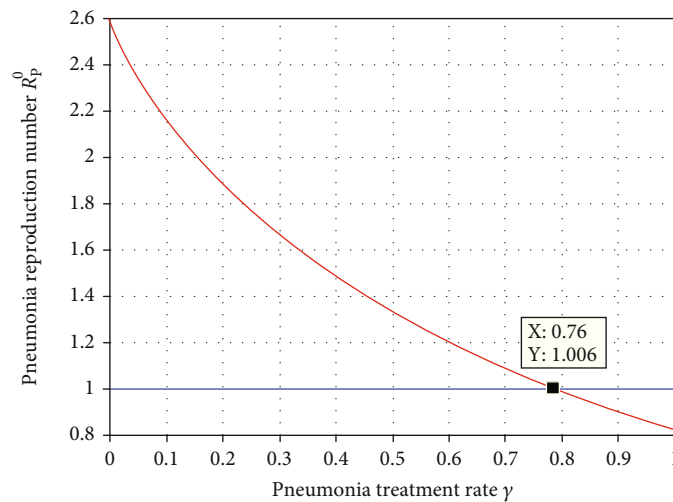


FIGURE 15: Effect of pneumonia treatment rate γ on \mathcal{R}_p^0 .

for health stakeholders to exert their optimal effort to maximize treatment rate γ more than the value 0.76 and making the value of \mathcal{R}_p^0 less than unity.

5. Conclusions

This study presented the HIV/AIDS and pneumonia coinfection dynamical system analysis to investigate protection and treatment intervention mechanisms' impacts on the coinfection spreading dynamics. Using parameter values adopted from published literatures, we have determined some basic results from the HIV/AIDS and pneumonia coinfection dynamical system qualitative and numerical analysis stated as follows: the proposed coinfection model has six equilibrium points; the HIV/AIDS submodel disease-free and endemic equilibrium points that are both

globally and locally asymptotically stable whenever its effective reproduction number is less than one which indicates that the HIV/AIDS submodel do not exhibits the phenomenon of backward bifurcation; the pneumonia only disease-free and endemic equilibrium points that are both globally and locally asymptotically stable whenever its effective reproduction number is less than one which indicates that the pneumonia submodel do not exhibits the phenomenon of backward bifurcation; and the HIV/AIDS and pneumonia coinfection model disease-free and endemic equilibrium points that are both globally and locally asymptotically stable whenever its effective reproduction number is less than one which indicates that the HIV/AIDS submodel do not exhibits the phenomenon of backward bifurcation. The qualitative and quantitative sensitivity analyses reveal that the disease-spreading rates, protection rates, and treatment

rates are the most sensitive parameters at which the stakeholders should give emphasis on these parameters and exert their maximum effort to control the transmission of the diseases in the community by applying suitable intervention measures. The coinfection model numerical simulation performed verified the qualitative results by investigating the impacts of some model parameters on the models associated with effective reproduction, the model state variables, and the behavior of the coinfection model solutions regarding convergence to the model equilibrium points. From the result, we recommend to the health stakeholders to minimize the disease-spreading rates and to maximize the protection and treatment rates for reducing the effective reproduction numbers below one. Finally, since the model formulation in this study is not exhaustive, any potential researcher can modify this study in various ways, such as by incorporating optimal control strategies, stochastic method, fractional order approach, environment effects, age structure, or validating models by collecting real data.

Data Availability

Data used to support the findings of this study are included in the article

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Authors' Contributions

The author has read and approved the final manuscript.

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