NUMERICAL SIMULATIONS AND COST-EFFECTIVE ANALYSIS OF OPTIMAL CONTROL STRATEGIES FOR PNEUMONIA-HIV CO-INFECTION

Abstract

This paper presents the numerical simulation and cost-effectiveness analysis of an optimal control model for Pneumonia-HIV co-infection, building upon a previously formulated deterministic model. The optimal control strategies incorporate Pneumonia treatment (u_1) and HIV Antiretroviral Therapy (ART, u_2) to minimize both disease prevalence and associated costs. Using Pontryagin's Maximum Principle, the optimality system was derived and solved numerically in MATLAB. Three control strategies were evaluated: Strategy 1 (Pneumonia treatment only), Strategy 2 (HIV treatment only), and Strategy 3 (combined treatment). The results demonstrate that the combined strategy (Strategy 3) is the most effective in reducing co-infection prevalence. A cost-effectiveness analysis using Incremental Cost-Effectiveness Ratios (ICER) and Infections Averted Ratios (IAR) further confirmed that Strategy 3 is the most cost-effective, achieving the highest number of infections averted at a lower cost per unit compared to other strategies. These findings provide critical insights for public health policymakers in allocating limited resources for managing Pneumonia-HIV co-infection.

Keywords: Optimal Control, Pneumonia-HIV Co-infection, Numerical Simulation, Cost-Effectiveness Analysis, Pontryagin's Maximum Principle, Incremental Cost-Effectiveness Ratio (ICER)

1. Introduction

The co-infection of Pneumonia and Human Immunodeficiency Virus (HIV) represents a significant public health challenge, particularly in resource-limited settings. Individuals with compromised immune systems due to HIV are highly susceptible to opportunistic infections like Pneumonia, leading to increased morbidity and mortality [4, 7]. While mathematical models have been developed to understand the dynamics of this co-infection [2, 3], few have integrated optimal control theory to determine the most efficient and cost-effective intervention strategies.

In our previous work [6], we had formulated a deterministic model for Pneumonia-HIV co-infection which was described by the following flow diagram in Figure 1 and described mathematically by the system (1) as;

$$\begin{aligned}
\dot{S(t)} &= \Lambda + \delta R_p - (\lambda_p + \lambda_h + \mu)S \\
\dot{I_p(t)} &= \lambda_p S - (\epsilon_1 + \mu + d_p + \delta_1 \lambda_h) I_p \\
\dot{R_p(t)} &= \epsilon_1 I_p - (\mu + \delta + \lambda_h) R_p \\
\dot{I_h(t)} &= \lambda_h R_p + \epsilon_2 I_{ph} + \lambda_h S - (\mu + \gamma_1 + \delta_2 \lambda_p) I_h
\end{aligned}$$

$$
\begin{aligned}
\dot{I_{ph}}(t) &= \delta_1\lambda_h I_p + \delta_2\lambda_p I_h - (\gamma_2 + \epsilon_2 + \mu + d_p)I_{ph} \\
\dot{A_{ph}}(t) &= \gamma_2 I_{ph} + \delta_3\lambda_p A_h - (\epsilon_3 + \mu + d_{Ap})A_{ph} \\
\dot{A_h}(t) &= \gamma_1 I_h + \epsilon_3 A_{ph} - (\delta_3\lambda_p + \mu + d_A)A_h
\end{aligned} \tag{1}
$$

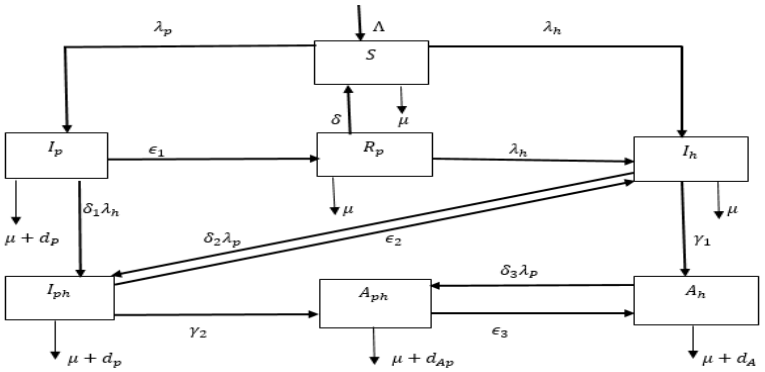

Figure 1: Flow chart diagram

where ϕ_1 is the effective transmission for Pneumonia, $\iota_2 > \iota_1$ are modification parameters which accounts for relative increased infectiousness with Pneumonia and ϕ_2 is the effective transmission rate for HIV. β_1 is a modification parameter for increased relative infectiousness of individuals with HIV. The state variables descriptions are given in the table below

Table 1: Symbols and Description of Parameter Values

Parameter	**Description**
$I_p(t)$	The number of individuals infected with Pneumonia. They are reduced by recoveries from Pneumonia at the rate ϵ_1 while others get infected with HIV and progress to I_{ph} class at the rate $\delta_1\lambda_h$. Mortality induced by Pneumonia under I_p class occurs at the rate d_p.
$R_p(t)$	The number of individuals who have recovered from Pneumonia infection. These individuals loose their temporary immunity and they get to susceptible class at the rate δ while others get infection from HIV at the rate λ_h and progress to I_h class.
$I_h(t)$	The number of individuals infected with HIV with no clinical symptoms of HIV and AIDS who progress to the class A_h by developing clinical symptoms of HIV and AIDS at the rate γ_1 while others get infected with Pneumonia and progress to I_{ph} class at the rate $\delta_2\lambda_p$.
$I_{ph}(t)$	The number of individuals co-infected with both Pneumonia and HIV with no clinical symptoms of HIV and AIDS. These individuals recover from Pneumonia at the rate ϵ_2 and move to I_h class, others develop HIV and AIDS symptoms and progress to A_{ph} class at the rate γ_2 while others die from Pneumonia induced mortality rate d_p.
$A_h(t)$	The number of individuals infected with HIV showing clinical symptoms of HIV and AIDS. These individuals seek medication from Pneumonia and they progress to A_h class at a recovery rate ϵ_3 and are increased from A_h class who get infection from Pneumonia at the rate $\delta_3\lambda_p$ where $\delta_3 > \delta_2$. They incur induced death from both Pneumonia and AIDS at mortality rate d_{Ap}.
$A_{ph}(t)$	The number of individuals co-infected with both Pneumonia and HIV with clinical symptoms of HIV and AIDS. They incur AIDS induced death at the rate d_A. All classes are reduced by deaths from natural causes at the rate μ.

The qualitative analysis of the model system (1) were performed and the model extended into optimal control problem by introduction of controls u_1(treatment for Pneumonia using antibiotics) and u_2 (treatment for HIV using Antiretroviral Therapy - ART) by [6]. The proposed objective function J given by:

$$J = \int_{t_0}^{T} \left(k_1 I_p + k_2 I_h + k_3 I_{ph} + k_4 A_{ph} + k_5 A_h + \frac{1}{2} w_1 {u_1}^2 + \frac{1}{2} w_2 {u_2}^2 \right) dt \tag{2}$$

where $k_1, k_2, k_3, k_4, k_5, w_1$ and w_2 are positive balancing coefficients (weights)which regularize the optimal control. [6] obtained the optimal controls $({u_1}^*, {u_2}^*)$ such that:

$$J({u_1}^*, {u_2}^*) = min\{J(u_1(t), u_2(t)) : u_1, u_2 \in U\} \tag{3}$$

subject to the optimal control problem (4) and the control set $U = \{(u_1(t), u_2(t)) : 0 \leq u_1 < 1, 0 \leq u_2 < 1, 0 \leq t \leq T\}$.

$$\begin{aligned}
\dot{S(t)} &= \Lambda + \delta R_p - (\lambda_p + \lambda_h + \mu)S \\
\dot{I_p(t)} &= \lambda_p S - (\epsilon_1 + u_1 + \mu + d_p + \delta_1\lambda_h)I_p \\
\dot{R_p(t)} &= (\epsilon_1 + u_1)I_p - (\mu + \delta + \lambda_h)R_p \\
\dot{I_h(t)} &= \lambda_h R_p + (\epsilon_2 + u_1)I_{ph} + \lambda S - (\mu + \gamma_1(1-u_2) + \delta_2\lambda_p)I_h \\
\dot{I_{ph}(t)} &= \delta_1\lambda_h I_p + \delta_2\lambda_p I_h - (\gamma_2(1-u_2) + (\epsilon_2 + u_1) + \mu + d_p)I_{ph} \\
\dot{A_{ph}(t)} &= \gamma_2(1-u_2)I_{ph} + \delta_3\lambda_p A_h - ((\epsilon_3 + u_1) + \mu + d_{Ap})A_{ph} \\
\dot{A_h(t)} &= \gamma_1(1-u_2)I_h + (\epsilon_3 + u_1)A_{ph} - (\delta_3\lambda_p + \mu + d_A)A_h
\end{aligned} \tag{4}$$

The necessary conditions for optimality were established using Pontryagin's Maximum Principle in [6], through characterization of the optimal controls using the Hamiltonian function H (which is considered as the total energy) given as:

$$\begin{aligned}
H(S, I_p, R_p, I_h, I_{ph}, A_{ph}, A_h, u_1, u_2, \zeta_1, \zeta_2, \zeta_3, \zeta_4, \zeta_5, \zeta_6, \zeta_7) = k_1 I_p + k_2 I_h \\
+k_3 I_{ph} + k_4 A_{ph} + k_5 A_h + \frac{1}{2}w_1 {u_1}^2 + \frac{1}{2}w_2 {u_2}^2 + \sum_{i=1}^{7}\zeta_i l_i
\end{aligned} \tag{5}$$

where l_i are the ith state variable equations on the right hand side of equation (1) and $\zeta_1, ..., \zeta_7$ are the adjoint variables satisfying:

$$\begin{aligned}
\frac{d\zeta_1}{dt} &= \zeta_1(\lambda_{p_1} + \lambda_{h_1} + \mu) - \zeta_2\lambda_{p_1} - \zeta_4\lambda_{h_1} \\
\frac{d\zeta_2}{dt} &= -k_1 + \zeta_1\frac{\phi_1 S}{N} - \zeta_2\frac{\phi_1 S}{N} - \zeta_4\frac{\delta_1\phi_1 I_h}{N} - \zeta_5\frac{\delta_2\phi_1 I_h}{N} - \zeta_6\frac{\delta_3\phi_1 A_h}{N} + \\
&\quad \zeta_7\frac{\delta_3\phi_1 A_h}{N} + \zeta_2(\epsilon_1 + u_1 + \mu + d_p + \delta_1\lambda_{h_2}) - \zeta_3(\epsilon_1 + u_1) - \zeta_5\delta_1\lambda_{h_2} \\
\frac{d\zeta_3}{dt} &= -\zeta_1\delta + \zeta_3(\mu + \delta + \lambda_{h_2}) - \zeta_4\lambda_{h_2} \\
\frac{d\zeta_4}{dt} &= -k_2 + \zeta_1\frac{\phi_2 S}{N} + \zeta_2\frac{\phi_2 I_p}{N} + \zeta_3\frac{\phi_2 R_p}{N} - \zeta_4\frac{\phi_2 R_p}{N} - \zeta_4\frac{\phi_2 S}{N} - \zeta_5\frac{\delta_1\phi_2 I_p}{N} + \\
&\quad \zeta_4(\mu + \gamma_1(1-u_2) + \delta_2\frac{\phi_1(I_p + \iota_1 I_{ph})}{N}) - \zeta_5\delta_2\frac{\phi_1(I_p + \iota_1 I_{ph})}{N} - \zeta_7\gamma_1(1-u_2) \\
\frac{d\zeta_5}{dt} &= -k_3 + \zeta_2\frac{\delta_1\phi_2\beta_1 I_p}{N} + \zeta_4\frac{\delta_2\phi_1\iota_1 I_h}{N} - \zeta_5\frac{\delta_1\phi_2\beta_1 I_p}{N} - \zeta_5\frac{\delta_2\phi_1\iota_1 I_h}{N} - \\
&\quad \zeta_4(\epsilon_2 + u_1) + \zeta_5(\gamma_2(1-u_2) + \epsilon_2 + u_1 + \mu + d_p) - \zeta_6\gamma_2(1-u_2) \\
\frac{d\zeta_6}{dt} &= -k_4 - \zeta_6\frac{\delta_3\phi_1\iota_2 A_h}{N} + \zeta_7\frac{\delta_3\phi_1\iota_2 A_h}{N} + \zeta_6(\epsilon_3 + u_1 + \mu + d_{Ap}) - \zeta_7(\epsilon_3 + u_1) \\
\frac{d\zeta_7}{dt} &= -k_5 + \zeta_7(\delta_3\lambda_{p_3} + \mu + d_A) - \zeta_6\delta_3\lambda_{p_3}
\end{aligned} \tag{6}$$

with transversality conditions; $\zeta_1(T) = \zeta_2(T) = \zeta_3(T) = \zeta_4(T) = \zeta_5(T) = \zeta_6(T) = \zeta_7(T) = 0$ in which $\zeta_1, \ldots, \zeta_7$ are adjoint variables.

The optimal controls that were determined and were given as:

$$\begin{aligned}
{u_1}^* &= max\{0, min(1, \frac{(\zeta_2 - \zeta_3)I_p + (\zeta_4 - \zeta_5)I_{ph} + (\zeta_6 - \zeta_7)A_{ph}}{w_1})\} \\
{u_2}^* &= max\{0, min(1, \frac{(\zeta_4 - \zeta_7)I_h + (\zeta_5 - \zeta_6)I_{ph}}{w_2})\}
\end{aligned} \tag{7}$$

This resulted into a complex optimality system of differential equations of the form:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda + \delta R_p - (\lambda_{p_1} + \lambda_{h_1} + \mu)S \\
\frac{dI_p}{dt} &= \lambda_p S - (\epsilon_1 + {u_1}^* + \mu + d_p + \delta_1\lambda_{h_2})I_p \\
\frac{dR_p}{dt} &= (\epsilon_1 + {u_1}^*))I_p - (\mu + \delta + \lambda_{h_1})R_p \\
\frac{dI_h}{dt} &= \lambda_{h_1} R_p + (\epsilon_2 + {u_1}^*))I_{ph} + \lambda_{h_1} S - (\mu + \gamma_1(1 - {u_2}^*)) + \delta_2\lambda_{p_2})I_h \\
\frac{dI_{ph}}{dt} &= \delta_1\lambda_{h_2} I_p + \delta_2\lambda_{p_2} I_h - (\gamma_2(1 - {u_2}^*)) + (\epsilon_2 + {u_1}^*)) + \mu + d_p)I_{ph} \\
\frac{dA_{ph}}{dt} &= \gamma_2(1 - {u_2}^*))I_{ph} + \delta_3\lambda_{p_3} A_h - ((\epsilon_3 + {u_1}^*)) + \mu + d_{Ap})A_{ph} \\
\frac{dA_h}{dt} &= \gamma_1(1 - {u_2}^*))I_h + (\epsilon_3 + {u_1}^*))A_{ph} - (\delta_3\lambda_{p_3} + \mu + d_A)A_h
\end{aligned} \tag{8}$$

and

$$\begin{aligned}
\frac{d\zeta_1}{dt} &= \zeta_1(\lambda_{p_1} + \lambda_{h_1} + \mu) - \zeta_2\lambda_{p_1} - \zeta_4\lambda_{h_1} \\
\frac{d\zeta_2}{dt} &= -k_1 + \zeta_1\frac{\phi_1 S}{N} - \zeta_2\frac{\phi_1 S}{N} - \zeta_4\frac{\delta_1\phi_1 I_h}{N} - \zeta_5\frac{\delta_2\phi_1 I_h}{N} - \zeta_6\frac{\delta_3\phi_1 A_h}{N} + \\
&\quad \zeta_7\frac{\delta_3\phi_1 A_h}{N} + \zeta_2(\epsilon_1 + {u_1}^* + \mu + d_p + \delta_1\lambda_{h_2}) - \zeta_3(\epsilon_1 + u_1) - \zeta_5\delta_1\lambda_{h_2} \\
\frac{d\zeta_3}{dt} &= -\zeta_1\delta + \zeta_3(\mu + \delta + \lambda_{h_2}) - \zeta_4\lambda_{h_2} \\
\frac{d\zeta_4}{dt} &= -k_2 + \zeta_1\frac{\phi_2 S}{N} + \zeta_2\frac{\phi_2 I_p}{N} + \zeta_3\frac{\phi_2 R_p}{N} - \zeta_4\frac{\phi_2 R_p}{N} - \zeta_4\frac{\phi_2 S}{N} - \zeta_5\frac{\delta_1\phi_2 I_p}{N} + \\
&\quad \zeta_4(\mu + \gamma_1(1 - u_2) + \delta_2\frac{\phi_1(I_p + \iota_1 I_{ph})}{N}) - \zeta_5\delta_2\frac{\phi_1(I_p + \iota_1 I_{ph})}{N} - \zeta_7\gamma_1(1 - u_2) \\
\frac{d\zeta_5}{dt} &= -k_3 + \zeta_2\frac{\delta_1\phi_2\beta_1 I_p}{N} + \zeta_4\frac{\delta_2\phi_1\iota_1 I_h}{N} - \zeta_5\frac{\delta_1\phi_2\beta_1 I_p}{N} - \zeta_5\frac{\delta_2\phi_1\iota_1 I_h}{N} - \\
&\quad \zeta_4(\epsilon_2 + u_1) + \zeta_5(\gamma_2(1 - u_2) + \epsilon_2 + {u_1}^* + \mu + d_p) - \zeta_6\gamma_2(1 - u_2) \\
\frac{d\zeta_6}{dt} &= -k_4 - \zeta_6\frac{\delta_3\phi_1\iota_2 A_h}{N} + \zeta_7\frac{\delta_3\phi_1\iota_2 A_h}{N} + \zeta_6(\epsilon_3 + u_1 + \mu + d_{Ap}) - \zeta_7(\epsilon_3 + u_1) \\
\frac{d\zeta_7}{dt} &= -k_5 + \zeta_7(\delta_3\lambda_{p_3} + \mu + d_A) - \zeta_6\delta_3\lambda_{p_3}
\end{aligned} \tag{9}$$

with transversality conditions; $\zeta_1(T) = \zeta_2(T) = \zeta_3(T) = \zeta_4(T) = \zeta_5(T) = \zeta_6(T) = \zeta_7(T) = 0$ in which $\zeta_1, \ldots, \zeta_7$ are adjoint variables.

The theoretical derivation of optimal controls, while crucial, must be complemented with numerical validation to assess the practical implications of different intervention strategies. Furthermore, in a world of finite health resources, determining not just the most effective, but the most cost-effective strategy is paramount for policy implementation. Therefore, this paper serves focuses on determining:

(i) The numerical solution of the optimality system.

(ii) The simulation and comparison of different control strategies.

(iii) A rigorous cost-effectiveness analysis to determine the best strategy for practical implementation.

2. Numerical Methods and Simulation Setup

The optimality system comprising state system (8)and adjoint equations system (9) was solved numerically using MATLAB's forward-backward sweep method with a fourth-order Runge-Kutta scheme. This approach ensured accurate, convergent solutions through iterative multi-step computation across discretized time intervals [5], with results visualized graphically. The approach was given by the following method:

(i) Approximate the solutions of the optimal control state system with initial guess values on the control variables and the given into conditions of the state variable using the Runge-Kutta forward sweep mathod.

(ii) Approximate the solutions for the adjoint system with final time conditions for adjoints and the given values in step (i) using Runge-Kutta backwad sweep method.

(iii) Update value of the control variables by taking the average of the previous values and knnew value from the control characterization.

(iv) Repeat the process above in (i) by using the new values of the sate variables and the control values obtained in (iii), and in (ii) by using the new values of the adjoint variables until we get sufficiently close values of state, adjoints and controls are attained (converge).

In the numerical simulations, the estimated initial values of the state variables were $S(0) = 1,000, I_p(0) = 550, R_p = 250, I_h(0) = 550, I_{ph}(0) = 400, A_{ph}(0) = 300, A_h(0) = 250$ while for the adjoint system we used the terminal conditions at final time. The transversality conditions for the adjoint variables were $\zeta_1(T) = \zeta_2(T) = \zeta_3(T) = \zeta_4(T) = \zeta_5(T) = \zeta_6(T) = \zeta_7(T) = 0$, where $T = 100$ days. A set of parameter/variable values as shown in Table 2 whose sources were from literature and others were estimated were also used. The coefficients to the state variables under the objective functional were estimated to be $k_1 = 1, k_2 = 1, k_3 = 5, k_4 = 1, k_5 = 1, w_1 = 80$ and $w_2 = 100$.

3. Numerical Results and Discussion

In this section, the numerical results of our optimal control were presented by considering the implications of three control strategies. The graphs were plotted to show the effects of the control measures when implemented under different combination options. The three strategies were given as:

Strategy 1: Control measure against Pneumonia is implemented ($u_1 \neq 0, u_2 = 0$)

Strategy 2: Control measure against HIV is implemented ($u_1 = 0, u_2 \neq 0$)

Strategy 3: All control measures are implemented ($u_1, u_2 \neq 0$)

Table 2: Parameter/Variable Values and Sources

Parameters	**Values Per Day**	**Source**
Λ	8.7×10^{-3}	[3]
μ	7.0×10^{-3}	[3]
ϵ_1	9.0×10^{-1}	[5]
δ	0.003-0.1	[5]
δ_1	1.8×10^{-1}	[2]
γ_1	1.25×10^{-1}	[3]
ϕ_1	8.8×10^{-3}	Calculated
ϕ_2	8.8×10^{-1}	Calculated
δ_2	1.2	Estimated
ϵ_2	8.0×10^{-1}	Estimated
γ_2	1.2	Estimated
ϵ_3	7.5×10^{-1}	Estimated
δ_3	1.25	Estimated
d_p	3.4×10^{-2}	[3]
d_A	2.3×10^{-4}	[3]
β_1	0.4	Estimated
ι_2, ι_1	0.6, 0.1	Estimated
d_{Ap}	4.0×10^{-3}	[2]

3.1 Dynamics without Controls (Baseline Scenario) $(u_1 = 0, u_2 = 0)$

Figure 2 presents the baseline scenario with no controls. Over 100 days, the susceptible population sharply declined as individuals contracted both HIV and Pneumonia, causing a temporary spike in HIV infections. This was followed by a gradual decrease in infections due to disease-induced mortality. Meanwhile, the sub-population showing symptoms of HIV and AIDS decreased initially but then rose steadily, ultimately becoming the dominant group. By day 100, the number of uninfected individuals (susceptible and recovered) was surpassed by those infected with and showing symptoms of HIV and AIDS, with natural and disease-induced death rates driving the decline of all other groups.

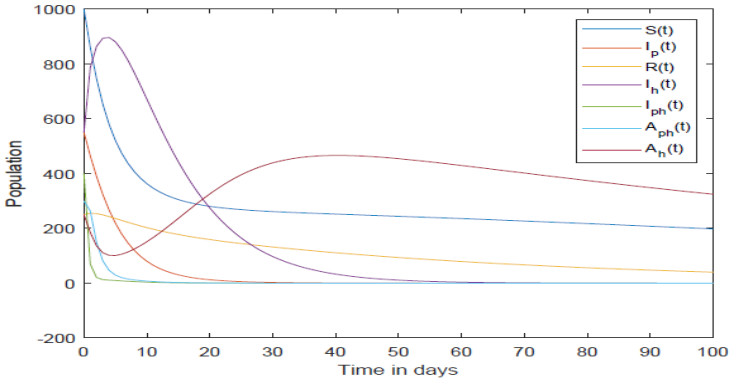

Figure 2: The Dynamics of Population with no controls

3.2 Dynamics for Strategy 1: Control measure against Pneumonia is implemented $(u_1 \neq 0, u_2 = 0)$

Under this strategy, only the control measure against pneumonia was optimized. As shown in Figure 3, this involved intensive administration of pneumonia treatment at the optimal rate of 0.98 throughout the 100-day period. The objective was to minimize the number of infected individuals relative to the susceptible population. The model results show that, apart from the susceptible, those recovered from pneumonia, and those with symptomatic HIV, all other groups eventually declined and died out due to combined natural and disease-induced death rates.

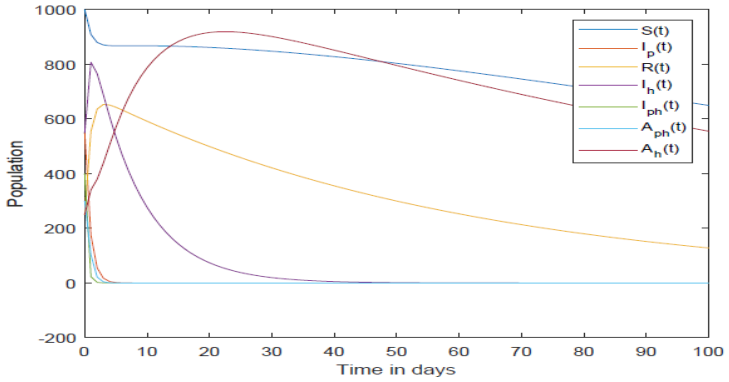

Figure 3: The Dynamics of Population with u_1 only

3.2 Dynamics for Strategy 2: Control measure against HIV is implemented $(u_1 = 0, u_2 \neq 0)$

Under this strategy, only control measure against pneumonia infection was used for optimization of the objective functional and the dynamics of the population was as shown in Figure 4. From Figure 4, the study noted that all individuals apart from recoveries from Pneumonia, susceptible and infected with HIV and showing symptoms of HIV decreased and died out due to induced death rates and natural death rates at the end of 100 days. To keep the number of individuals living with the infections as low as possible (as compared to susceptible individuals), HIV treatment was administered at the optimal rate of 0.12 throughout for 100 days.

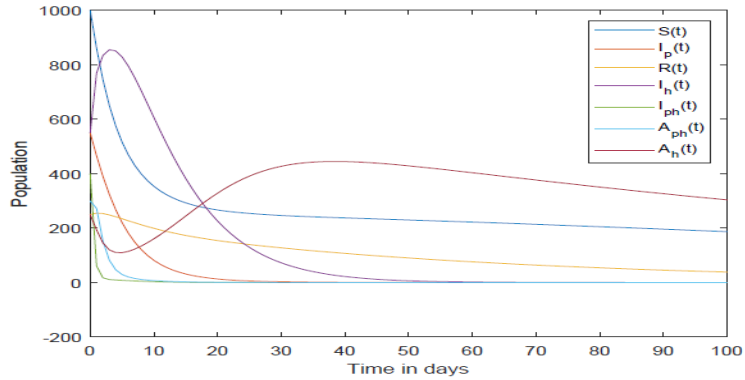

Figure 4: The Dynamics of Population with u_2 only

3.3 Dynamics for Strategy 3: All control measures are implemented $(u_1, u_2 \neq 0)$

This strategy involved the optimized use of all controls against HIV and Pneumonia co-

infections. The resulting population dynamics are shown in Figure 5. To minimize the number of infected individuals, the model administered HIV treatment at an optimal rate of 0.41 and Pneumonia treatment at a rate of 0.98 throughout the 100-day period. The simulation revealed the following trends: the susceptible population decreased, individuals infected with HIV (but without symptoms) saw a sharp initial increase before declining to lower levels, and the symptomatic HIV group increased slowly before eventually decreasing. By the end of the simulation, all sub-populations except the susceptible, the pneumonia-recovered, and the HIV infected (with and without symptoms) had been eliminated due to natural and disease-induced death rates.

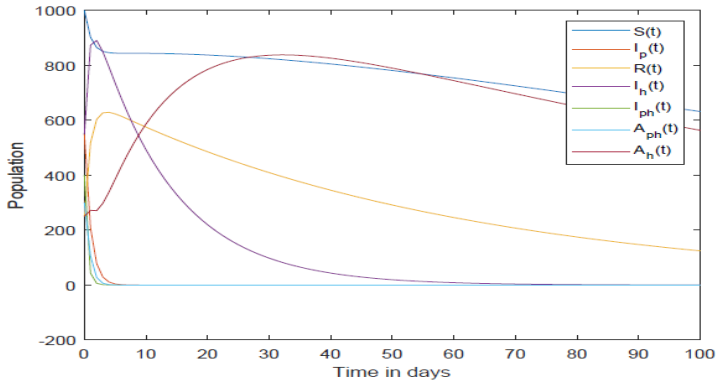

Figure 5: The Dynamics of Population with u_1 and u_2

Generally, analysis of Figures 2 through 5 revealed that the control strategies successfully kept the susceptible population above the infected populations in all cases except Figure 4. This indicates that the interventions had a positive impact, effectively reducing the overall number of infections. However, the strategy of applying only HIV treatment (Figure 4) was less effective. In this scenario, infected individuals outnumbered the susceptible population. The analysis showed that HIV treatment alone only reduced the number of symptomatic HIV and AIDS cases by a small margin-approximately 20 individuals.

4. Discussions of Cost-effective Analysis

A cost-effectiveness analysis was performed to identify the optimal strategy by comparing control costs against health outcomes using the Infections Averted Ratio (IAR) and Incremental Cost-Effectiveness Ratio (ICER), which quantifies the additional cost per additional health benefit.

The ICER were evaluated by comparing any two competing interventions using their differences in the costs and health outcomes using the formula:

$$ICER = \frac{Difference\ \ in\ \ infection\ \ averted\ \ costs\ \ in\ \ stategy\ \ i\ \ and\ \ j}{Difference\ \ in\ \ total\ \ infections\ \ averted\ \ costs\ \ is\ \ stategy\ \ i\ \ and\ \ j} \tag{10}$$

The difference between the total number of infections among individuals with controls and those without controls was taken to give the infections averted. The infection averted ratio

(IAR) were given as;

$$IAR = \frac{Infections \quad averted}{Number \quad of \quad Recoveries} \tag{11}$$

The total control costs and IAR were calculated and estimated in ($) USD over 100 as shown in table 3;

Table 3: Total infections averted and total cost

Strategies	**Total infections averted**	**Total costs J** ($)
Strategy 1	1.9688	$59,741.6$
Strategy 2	0.4500	$32,472$
Strategy 3	2.02362	$59,700.5$

From the table 3, Strategy 3 was the most effective as compared to the other strategies (Strategies 1 and 2) since it had the highest IAR. We therefore embarked on determining the strategy which was cost effective.

The ICER was calculated by first ranking the control strategies in increasing order of their effectiveness which is based on the infection that is averted. Strategy 2 averts the least number of infections, followed by Strategy 1 and then Strategy 3.
The ICER were computed as follows:

$$ICER(2) = \frac{32,472}{0.45} = 72,160 \tag{12}$$

$$ICER(1) = \frac{59,741.6 - 32,472}{1.9688 - 0.45} = 17,954.3060 \tag{13}$$

$$ICER(3) = \frac{59,700.5 - 32,472}{2.02362 - 0.45} = 17,328.8341 \tag{14}$$

From the above calculations of ICER, Strategy 1 effectively controls Pneumonia but not HIV. Strategy 2 shows the least impact, as the modest ART rate (0.12) is insufficient to quickly alter HIV dynamics, and it does not address Pneumonia directly. But the ICER analysis revealed that Strategy 3 dominated Strategy 1, as it was both less costly and more effective. This resulted in a cost saving of $17,954.31$ for Strategy 3 over Strategy 1. Consequently, Strategy 1 was excluded from the set of viable options to preserve limited resources. Hence, Strategy 3 was selected as the most cost-effective strategy.

5.Conclusion and Recommendations

This study successfully implemented and numerically solved an optimal control model for Pneumonia-HIV co-infection. The simulations unequivocally demonstrate that a combined intervention strategy, simultaneously administering anti-Pneumonia drugs and ART, is the most effective for reducing disease prevalence. The cost-effectiveness analysis further solidifies this finding, identifying the combined strategy as the most efficient use of limited resources. The study recommends implementing Strategy 3, with Pneumonia treatment at

0.98 and HIV treatment at 0.41, as it simultaneously addresses both infections by boosting immunity against Pneumonia while prolonging life for HIV patients.

5.1 Policy Implications

Health policymakers and practitioners should prioritize integrated treatment programs for HIV and Pneumonia. Focusing solely on one disease, particularly HIV, without addressing the opportunistic Pneumonia infection, yields suboptimal outcomes. The model suggests that Pneumonia treatment has a more immediate and pronounced effect on reducing the overall disease burden, but its impact is sustained and enhanced when coupled with ART.

5.2 Limitations and Future Research

This research is deterministic and does not account for stochastic events or heterogeneous population mixing. Therefore future work should consider extending the model to include age-structure or spatial heterogeneity to better reflect real-world transmission dynamics. Despite these limitations, this study provides a robust mathematical framework and a clear, data-driven recommendation for managing Pneumonia-HIV co-infection, serving as a valuable tool for epidemiologists and public health strategists.

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