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Modeling the Dynamics of HIV-Pneumonia Coinfections in Nigeria Using Delay Differential Equations (DDE)

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ABSTRACT
In nigeria, which has one of the world's highest hiv burdens, pneumonia is a leading cause of mortality among immune-compromised individuals. This bidirectional relationship where hiv increases susceptibility to pneumonia and pneumonia accelerates hiv progression is poorly captured by traditional models that ignore real world time delays. We develop a delay differential equation (dde) model incorporating critical delays: pneumonia incubation, treatment initiation, and immune recovery. The basic reproduction number ($\mathcal{R}_0$) is derived, and the disease-free equilibrium is shown to be locally and globally stable when $\mathcal{R}_0 < 1$. Model solutions are proven to be positive and bounded. Sensitivity analysis reveals that transmission rates are the most influential parameters. Numerical simulations demonstrate that time delays significantly elevate infection peaks and prolong epidemic duration. Longer diagnostic and treatment delays increase hiv and pneumonia prevalence by 15% and 25-30%, respectively, while combined delays result in the highest overall burden and slowest convergence to equilibrium. Our findings underscore that reducing delays in diagnosis and treatment is critical for outbreak control. This delay based model provides a more realistic framework for understanding hiv-pneumonia coinfection dynamics and offers validated insights for guiding public health intervention strategies in Nigeria.

Keywords:
Coinfections,
Modeling,
Pneumonia,
Transmission,
Treatment,
Immune

INTRODUCTION

Human Immunodeficiency Virus (HIV) continues to be one of the major public health concerns across the world. According to UNAIDS (2025), more than 38 million people are currently living with HIV, with sub-Saharan Africa accounting for over two-thirds of these cases. Nigeria remains one of the most affected countries, with about 1.9 million people living with HIV and an adult prevalence rate of around 1.3% (Onovo et al., 2023; Lawal et al., 2024). Although the introduction of antiretroviral therapy (ART) has greatly reduced HIV related deaths worldwide (Bassey et al., 2023), the infection continues to cause significant illness and death, especially in Nigeria where late diagnosis and poor treatment adherence are still common (Omololu et al., 2024).

Pneumonia is one of the most frequent secondary infections found in people living with HIV. This includes community acquired pneumonia (CAP), hospital acquired pneumonia, and *Pneumocystis jirovecii* pneumonia (PJP).

Many studies have shown that people with HIV are more likely to get pneumonia, and when they do, it tends to be more severe than in those without HIV. The risk of hospital admission and death rises sharply as CD4 (helper cells) counts drop (Benito et al., 2012; Crothers et al., 2011; Feldman et al., 2022). In Nigeria, pneumonia remains a major cause of respiratory illness and death among HIV positive patients, especially in hospitals where diagnosis and treatment are often delayed (Onyedum et al., 2011; Omololu et al., 2024).

The connection between HIV and pneumonia works in both directions. HIV weakens the immune system by affecting white blood cells that help fight infections, making the body more open to pneumonia causing germs (Benito et al., 2012; NIH, 2024). At the same time, repeated pneumonia infections can make HIV progress faster by increasing inflammation and putting more stress on the immune system (Crothers et al., 2011). This creates a cycle where a weak immune system leads to more infections, and more infections make the immune system even weaker.

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Understanding this relationship is important for improving treatment and prevention strategies.

Mathematical models have long helped scientists understand how diseases spread and behave. These models, often written as ordinary differential equations (ODEs), help estimate key values such as the basic reproduction number ($\mathcal{R}_0$), predict outbreak patterns, and test the impact of control measures (Diekmann et al., 1990; van den Driessche&Watmough, 2002). However, ODE models assume that changes between stages happen instantly, which is not realistic for HIV–pneumonia cases where there are delays in incubation, diagnosis, treatment, and immune recovery (Hale &VerduynLunel, 1993; McCluskey, 2010). Research shows that such delays can affect the stability of the disease system and may lead to waves or recurring infections (Beretta &Kuang, 2002; Zhou, 2009). In Nigeria, where diagnosis and treatment often take time, these delays play a key role in how the disease behaves.

HIV remains one of the most serious global health problems. It weakens the immune system, making people more likely to develop other infections. Among these, lung infections such as pneumonia are among the most common. In Nigeria, HIV continues to affect millions of people, many of whom have limited access to consistent treatment. Respiratory diseases remain major causes of hospital admissions, particularly among individuals with weakened immunity. Although many studies have examined HIV and tuberculosis together, research that focuses on HIV and pneumonia coinfection remains limited.

Although this study focuses on pneumonia, the literature on HIV and tuberculosis provides useful insight because both affect the lungs and share similar health system challenges. A meta-analysis by Reward, Ike, Muo, Soga-Oke, and Mbaawuaga (2020) found that about 25.8% of people with tuberculosis in Nigeria were also living with HIV. Regional differences were observed, with the North Central zone recording the highest rate (34.3%) and the Southeast the lowest (19.3%). Similarly, a five year retrospective study in Enugu State by Nwoga, Igweagu, and Umeh (2024) reported a coinfection rate of 29.0% among tuberculosis patients. Occupation and place of residence were found to influence infection rates, with urban residents showing lower prevalence. An earlier hospital based study by Iliyasu and Babashani (2009) also recorded a 10.5% coinfection rate among HIV patients with active tuberculosis.

There are fewer published studies that examine pneumonia or other lung infections among people living with HIV in Nigeria. A review of respiratory admissions at the Federal Medical Centre, Owo, showed that pneumonia accounted for 21.1% of respiratory cases. Additionally, 17.5% of pulmonary tuberculosis cases were HIV positive (Annals of Medical & Health Sciences Research, 2023). This pattern highlights that lung infections, including pneumonia, contribute significantly to the disease burden among HIV infected individuals.

Mathematical modeling helps to understand how diseases spread and to evaluate possible control measures. Several models have been developed in Nigeria to study HIV transmission and related diseases. Musa and Udoaka (2024) developed a model to examine HIV spread and control strategies in different regions of Nigeria, finding that wider access to treatment and preventive measures reduced infection levels. Another study, 'Understanding the Transmission Dynamics and Control of HIV Infection: A Mathematical Model Approach' (2024), emphasized that delays in treatment significantly affect control outcomes. Chibuisi et al. (2020) demonstrated how delay differential equations can be used to capture the real life time delays in disease spread and recovery processes.

MATERIALS AND METHODS

Research Design

This study uses a deterministic mathematical approach to study how HIV and pneumonia interact within the Nigerian population. Instead of focusing only on the present state of infection, the model also considers the effect of time delay**s** for example, the time between infection, showing symptoms, getting diagnosed, starting treatment, and recovering.

These delays are included using delay differential equations (DDEs) because, in real life, diseases like HIV and pneumonia do not progress instantly. It takes time before symptoms appear or treatment begins.

A deterministic design is suitable because it helps identify general trends, such as when the disease will die out or persist, without focusing on random variations.

Model Formulation

The model divides the population into five groups susceptible, HIV only, pneumonia only, coinfected, and recovered. People move between these groups according to infection, recovery, and death rates.

These delays make the system more realistic and help explain why infections sometimes rise and fall in waves instead of settling quickly.

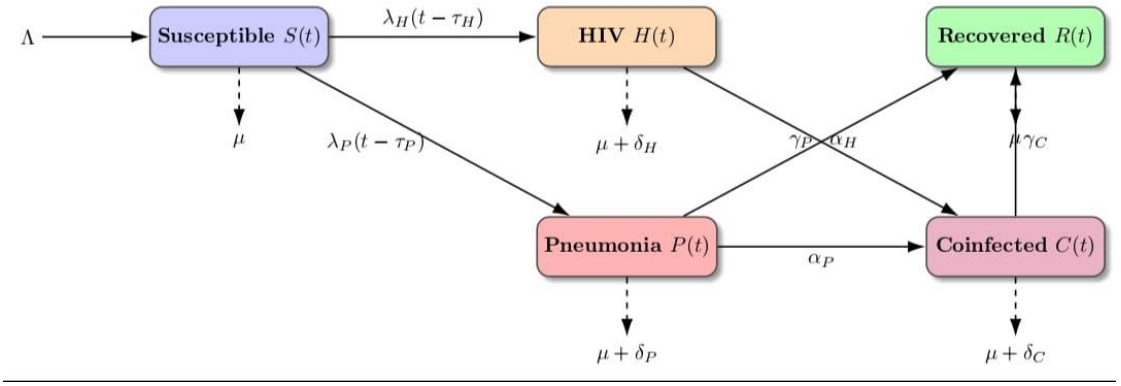

Figure 1 *Schematic Diagram for HIV-Pneumonia Coinfection*

$$\frac{dS}{dt} = \lambda - \lambda_H(t)S(t) - S(t)\lambda_p(t-\tau_p) - \mu S(t) \quad (1)$$

$$\frac{dH}{dt} = \lambda_H(t)S(t) - \alpha_P H(t)\lambda_p(t-\tau_p) - (\mu+\delta_H)H(t) \quad (2)$$

$$\frac{dP}{dt} = S(t)\lambda_p(t-\tau_p) - \alpha_H P(t)\lambda_H(t) - (\gamma_p + \mu + \delta_p)P(t) \quad (3)$$

$$\frac{dC}{dt} = \alpha_P H(t)\lambda_p(t-\tau_p) + \alpha_H P(t)\lambda_H(t) - (\gamma_C(t) + \mu + \delta_C)C(t) \quad (4)$$

$$\frac{dR}{dt} = \gamma_p P(t-\tau_d) + \gamma_C C(t-\tau_d) - \mu R(t) - \chi\lambda_p(t-\tau_r)R(t) \quad (5)$$

2.2.1 Forces of infection (with interaction modifiers ϕ,η ∈ [0,1]

$$\lambda_H(t) = \beta_H \frac{H(t)+\phi C(t)}{N(t)}, \quad (6)$$

$$\lambda_p(t) = \beta_P \frac{P(t)+\eta C(t)}{N(t)} \quad (7)$$

Notations

Table 1: Parameter descriptions

Parameters	Descriptions
S(t)	Susceptible individuals.
H(t)	Infected individuals with HIV
P(t)	Infected individuals with pneumonia
C(t)	Coinfected individuals
R(t)	Recovered individuals from pneumonia/coinfection
N(t)	Total population size
$\lambda_H(t)$	The force of infection for acquiring HIV

$\lambda_P(t)$	The force of infection for acquiring pneumonia
Λ	Recruitment rate into susceptible
μ	Natural mortality rate
β_H	Contact rate for HIV transmission.
β_p	Contact rate for pneumonia transmission.
Φ	Modification factor for the infectiousness of Coinfected individuals C relative to HIV only.
χ	Modification factor for the infectiousness of coinfected individuals 'C' relative to pneumonia only.
α_H	Enhancement factor for a pneumonia only individual P to acquire HIV.
α_p	Enhancement factor for an HIV only individual H to acquire pneumonia.
γ_P	Recovery rate from pneumonia only infection.
γ_C	Recovery rate from pneumonia for coinfected individuals.
δ_H	Disease induced mortality rate due to HIV.
δ_p	Disease induced mortality rate due to pneumonia.
δ_c	Excess mortality rate due to coinfection
X	Reduction factor for susceptibility to pneumonia after recovery.
τ_P	Pneumonia Incubation Delay.
τ_d	Diagnosis/Treatment Delay.
τ_r	Immune Recovery Delay.

Positivity and Boundedness

Theorem: Positivity and Boundedness of the Solutions

Let $N(t) = S(t) + H(t) + P(t) + C(t) + R(t)$ be the total population. Then the solution trajectories are uniformly positive and bounded in the region

Proof: Sum all equations of the system:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dH}{dt} + \frac{dP}{dt} + \frac{dC}{dt} + \frac{dR}{dt}$$

$$\frac{dN}{dt} = \lambda - \mu N - \delta_H H(t) - \delta_P P(t) - \delta_C C(t) \quad (8)$$

$since\ \delta_H, \delta_P, \delta_C \geq 0$, the inequality holds:

$$-\delta_H - \delta_P - \delta_C \leq 0$$

Thus, we can write

$$\frac{dN}{dt} \leq \lambda - \mu N(t) \quad (9)$$

This inequality tells us that the growth rate is at most $\lambda - \mu N(t)$ $\frac{dN}{dt} + \mu N(t) \leq \lambda$

$$\frac{dN}{dt} + \mu N(t) = \lambda \quad (10)$$

Using integrating factor method $IF = e^{\int \mu dt} = e^{\mu t}$, multiply both sides of (10) by the IF

$$e^{\mu t}\left(\frac{dN}{dt} + \mu N(t)\right) = \lambda e^{\mu t} e^{\mu t}\frac{dN}{dt} + \mu e^{\mu t} N(t) = \lambda e^{\mu t}$$

$$\frac{d}{dt}(N(t)e^{\mu t}) \leq \lambda e^{\mu t} \quad (11)$$

Integrating both side from 0 to t:

$$N(t) - N(0) \leq \int_0^t \lambda e^{\mu s}\, ds \quad (12)$$

$$\int_0^t \lambda e^{\mu s}\, ds = \lambda \int_0^t e^{\mu s}\, ds$$

$$\int_0^t e^{\mu s}\, ds = \lambda[\frac{1}{\mu} e^{\mu s}]_0^t$$

$$= \lambda(\frac{1}{\mu} e^{\mu t} - \frac{1}{\mu} e^{\mu * 0})$$

$$= \frac{\lambda}{\mu}(e^{\mu t} - 1)$$

$$N(t)e^{\mu t} - N(0) \leq \int_0^t \lambda e^{\mu s}\, ds = \frac{\lambda}{\mu}(e^{\mu t} - 1) \quad (13)$$

Solve for N(t):

$$N(t) \leq N(0)e^{\mu t} + \frac{\lambda}{\mu} + (1 - e^{-\mu t})$$

$$N(t) \leq \frac{\lambda}{\mu} + (N(0) - \frac{\lambda}{\mu})\, e^{-\mu t}$$

$$N(0) \leq \frac{\lambda}{\mu} \quad (14)$$

$$(N(0) - \frac{\lambda}{\mu})\, e^{-\mu t}$$

$$N(t) \leq \frac{\lambda}{\mu}$$

$$N(t) > \frac{\lambda}{\mu}$$

$$(N(0) - \frac{\lambda}{\mu})\, e^{-\mu t} \quad (16)$$

is positive but decreases exponentially to 0 as $t \to \infty. thus\ N(t)$ decays towards $\frac{\lambda}{\mu}$ from above.

In both cases, N(t) is bounded for all $t \geq 0$ and approaches $\frac{\lambda}{\mu}$ as $t \to \infty$.

Hence the system is positive and bounded above by $\frac{\lambda}{\mu}$

Disease free Equilibrium

Theorem: Disease-Free Equilibrium (DFE)

The disease free equilibrium (DFE) of the given system exists and is given by:

$$E^* = S^*, H^*, P^*, C^*, R^* = (\frac{\lambda}{\mu}, 0,0,0,0) \quad (17)$$

Provided that the forces of infection λ_H^*, λ_P^* vanish when all infected compartments H, P, C is zero.

Proof:

Define the DFE Condition:

A disease-free equilibrium is a steady state where no disease is present. Thus:

$$H^* = 0, P^* = 0, C^* = 0$$

From the definitions:
$\lambda_H(t) = \beta_H \frac{H(t) + \phi\, C(t)}{N(t)}$, $\lambda_p(t) = \beta_P \frac{P(t) + \eta\, C(t)}{N(t)}$

Substituting
$$H^* = 0, P^* = 0, C^* = 0 \lambda_H^* = 0, \lambda_P^* = 0$$

At equilibrium, all derivatives are zero. Substituting

$\lambda_H^* = 0$ and $\lambda_P^* = 0$.

Equation for S:

$$S^* = \lambda - \lambda_H^*(t)S(t) - S(t)\lambda_P^*(t-\tau_p) - \mu S(t) = 0 \quad (18)$$

$$\lambda = \mu S(t)$$

$$\mu S(t) = \lambda$$

$$S^* = \frac{\lambda}{\mu} \quad (19)$$

Equation for H

$$H^* = \lambda_H^*(t)S^* - \alpha_P H^*(t)\lambda_P^*(t-\tau_p) -$$

$$(\mu + \delta_H)H^* = 0 \quad (20)$$

$$H^* = 0. \quad (21)$$

Equation for P:

$$S^*\lambda_p^*(t-\tau_p) - \alpha_H P^*(t)\lambda_P^*(t) -$$

$$(\gamma_p + \mu + \delta_p)P^* = 0 \quad (22)$$

$$P^* = 0 \quad (23)$$

Equation for C:

$$C^* = \alpha_P H^*(t)\lambda_P^*(t-\tau_p) + \alpha_H P^*(t)\lambda_H^* -$$

$$(\gamma_C(t) + \mu + \delta_C)C^* = 0 \quad (24)$$

$$C^* = 0 \quad (25)$$

Equation for R:

$$R^* = \gamma_p P^*(t-\tau_d) + \gamma_C C^*(t-\tau_d) - \mu R^*(t) - \chi\lambda_P^*(t-\tau_r)R^*(t) = 0 \quad (26)$$

$$\Rightarrow R^* = 0 \quad (27)$$

$E^* = S^*, H^*, P^*, C^*, R^* = (\frac{\lambda}{\mu}, 0,0,0,0)$

Satisfies all equilibrium equations when $\lambda_H^* = 0$ and $\lambda_P^* = 0$. Hence, it is a disease free equilibrium.

Basic reproduction number via Next Generation Matrix

Basic reproduction number is the process of determining the secondary infection, that is either the disease will die out or it will spread nationwide or even globally.

At DFE only susceptible individuals are present that is

$$S^* = \frac{\lambda}{\mu}, H^* = 0, P^* = 0, C^* = 0, R^* = 0$$

Also, the total population is:

$$N^* = S^* = \frac{\lambda}{\mu}$$

Next generation matrix is the product of F and V^{-1}

$$FV^{-1} = \begin{pmatrix} \beta_H & 0 & \beta_H\phi \\ 0 & \beta_P & \beta_P\eta \\ 0 & 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{\mu+\delta_H} & 0 & 0 \\ 0 & \frac{1}{\gamma_p+\mu+\delta_p} & 0 \\ 0 & 0 & \frac{1}{\gamma_C+\mu+\delta_C} \end{pmatrix} \quad (28)$$

$$K = FV^{-1} = \begin{pmatrix} \frac{\beta_H}{\mu+\delta_H} & 0 & \frac{\beta_H\phi}{\gamma_C+\mu+\delta_C} \\ 0 & \frac{\beta_P}{\gamma_p+\mu+\delta_p} & \frac{\beta_P\eta}{\gamma_C+\mu+\delta_C} \\ 0 & 0 & 0 \end{pmatrix} \quad (29)$$

Basic Reproduction number $\mathcal{R}_0$

Since K is an upper triangular, its eigenvalues are the diagonal entries:

$$\lambda_1 = \mathcal{R}_{0H} = \frac{\beta_H}{\mu+\delta_H}, \lambda_2 = \mathcal{R}_{0P} = \frac{\beta_P}{\gamma_p+\mu+\delta_p}, \lambda_3 = 0 \quad (30)$$

Thus, the basic reproduction number is

$$\mathcal{R}_0 = \max\{\mathcal{R}_{0H} = \frac{\beta_H}{\mu+\delta_H}, \mathcal{R}_{0P} = \frac{\beta_P}{\gamma_p+\mu+\delta_p}\} \quad (31)$$

Local Stability around DFE Using Jacobian Method

To establish local stability, we analyze the system without delays (that is $\tau_p = \tau_d = \tau_r = 0$)

The local stability condition at the DFE requires that all the eigenvalues must have negative real parts to be asymptotically stable.

- $\lambda_1 = -\mu$ (32)
- $\lambda_2 = -\mu$ (33)
- $\lambda_3 = \beta_H - (\mu + \delta_H)$we have to ascertain to be negative that is $\beta_H - (\mu + \delta_H) < 0 \Rightarrow \beta_H < (\mu + \delta_H)$ thus its negative. since
- $\mathcal{R}_{0H} = \frac{\beta_H}{\mu+\delta_H} < 1$ (34)
- $\lambda_4 = \beta_P - (\gamma_P + \mu + \delta_P) < 0 \Rightarrow \beta_P < (\gamma_P + \mu + \delta_P)$ its negative since
- $\mathcal{R}_{0P} = \frac{\beta_P}{\gamma_p+\mu+\delta_p} < 1$ (35)
- $\lambda_5 = -(\gamma_C + \mu + \delta_C)$ (36)
  Hence the disease free equilibrium (DFE) is locally asymptotically stable.

Global Stability around DFE (using Lyapunov functional)

To analyze global stability using Lyapunov functional, the $\mathcal{R}_0$ must be strictly < 1 and assume a constant total population. It is for the purpose of analytical tractability in the global stability proof.

To maintain a constant population let $N(t) = S(t) + H(t) + P(t) + C(t) = 1, \lambda = \mu$ (37)

Lyapunov function: $V = P(t) + H(t) + c(t) + \beta_p \int_{t-\tau_p}^{t} [P(\theta) + \eta C(\theta)] d\theta$ (38)

This account for the delay τ_p in $\lambda_p(t - \tau_p)$.

$$\frac{dV}{dt} = \frac{dH}{dt} + \frac{dP}{dt} + \frac{dC}{dt} + \beta_p[P(t) + \eta C(t) - \left(P(t - \tau_p) + \eta C(t - \tau_p)\right)] \quad (39)$$

Substitute the equations for $\frac{dH}{dt}, \frac{dP}{dt}, \frac{dC}{dt}$:

Thus, the term becomes: $\beta_p \left[P(t) + \eta C(t) - \left(P(t - \tau_p) + \eta C(t - \tau_p)\right)\right] = \beta_p[P(t) + \eta C(t)] - \lambda_p(t - \tau_p)$

Substitute back: $\frac{dV}{dt} = \lambda_H S + \lambda_p S(t - \tau_p) - (\mu + \delta_H)H - (\gamma_p + \mu + \delta_p)P - (\gamma_p(t) + \mu + \delta_C)C + +\beta_p[P(t) + \eta C(t)] - \lambda_p(t - \tau_p)$ (40)

Thus:

$$\frac{dV}{dt} \leq \beta_H(H + \phi C) - (\mu + \delta_H)H - (\gamma_p + \mu + \delta_p)P - (\gamma_p(t) + \mu + \delta_C)C + \beta_p[P(t) + \eta C(t)] \quad (41)$$

Rearrange:

$$\frac{dV}{dt} \leq H(\beta_H - (\mu + \delta_H)) + P(\beta_p - (\gamma_p + \mu + \delta_p)) \quad (42)$$

Global stability condition

For $\frac{dV}{dt} \leq 0$, the coefficients must be negative:

$$\beta_H < \mu + \delta_H \quad (43)$$

$$\beta_p < \gamma_p + \mu + \delta_p \quad (44)$$

$$\beta_H \phi + \beta_P < \mu + \delta_C + \gamma_C \quad (45)$$

If these hold,$\frac{dV}{dt} \leq 0$ with $H = P = C = 0$.By LaSalle's invariance principle, the infected compartment approach zero. The system reduces to:

$$\frac{dV}{dt} = \mu - \mu S, \ \frac{dR}{dt} = -\mu R, \quad (46)$$

Which converge $S = 1$ and $R = 0$. thus the DFE globally, asymptotically stable.

Sensitivity analysis

Consider the HIV-Pneumonia model whose disease free equilibrium yields the component reproduction numbers $\mathcal{R}_{0H} = \frac{\beta_H}{\mu+\delta_H}, \mathcal{R}_{0P} = \frac{\beta_P}{\gamma_p+\mu+\delta_p}$

Proof

The normalized sensitivity index of a quantity Q(θ) with respect to a parameter θ is

For $\mathcal{R}_{0H} = \frac{\beta_H}{\mu+\delta_H}$:

$$\Gamma_{\beta_H}^{\mathcal{R}_{0H}} = \frac{\partial \mathcal{R}_{0H}}{\partial \beta_H} = \frac{\beta_H}{\mu+\delta_H} * \frac{\theta}{Q(\theta)} = \frac{1}{\mu+\delta_H} * \frac{\beta_H}{\frac{\beta_H}{\mu+\delta_H}} = 1 \quad (47)$$

$$\Gamma_{\mu}^{\mathcal{R}_{0H}} = \frac{\partial \mathcal{R}_{0H}}{\partial \mu} * \frac{\theta}{Q(\theta)} = -\frac{\beta_H}{(\mu+\delta_H)^2} * \frac{\mu}{\frac{\beta_H}{\mu+\delta_H}} = -\frac{\mu}{\mu+\delta_H} \quad (48)$$

$$\Gamma_{\delta_H}^{\mathcal{R}_{0H}} = \frac{\partial \mathcal{R}_{0H}}{\partial \delta_H} * \frac{\theta}{Q(\theta)} = -\frac{\beta_H}{(\mu+\delta_H)^2} * \frac{\delta_H}{\frac{\beta_H}{\mu+\delta_H}} = -\frac{\delta_H}{\mu+\delta_H} \quad (49)$$

For $\mathcal{R}_{0P} = \frac{\beta_P}{\gamma_p+\mu+\delta_p}$:

$$\Gamma_{\beta_H}^{\mathcal{R}_{0P}} = \frac{\partial \mathcal{R}_{0H}}{\partial \beta_P} * \frac{\theta}{Q(\theta)} = \frac{1}{\gamma_p+\mu+\delta_p} * \frac{\beta_P}{\frac{\beta_P}{\gamma_p+\mu+\delta_p}} = 1 \quad (50)$$

$$\Gamma_{(\gamma_p}^{\mathcal{R}_{0P}} = \frac{\partial \mathcal{R}_{0H}}{\partial \gamma_p} * \frac{\theta}{Q(\theta)} = -\frac{\beta_P}{(\gamma_p+\mu+\delta_p)^2} * \frac{\gamma_p}{\frac{\beta_P}{\gamma_p+\mu+\delta_p}} = -\frac{\gamma_p}{\gamma_p+\mu+\delta_p} \quad (51)$$

$$\Gamma_{\mu}^{\mathcal{R}_{0P}} = \frac{\partial \mathcal{R}_{0H}}{\partial \mu} * \frac{\theta}{Q(\theta)} = -\frac{\mu}{(\gamma_p+\mu+\delta_p)^2} * \frac{\mu}{\frac{\beta_P}{\gamma_p+\mu+\delta_p}} = -\frac{\mu}{\gamma_p+\mu+\delta_p} \quad (52)$$

$$\Gamma_{\delta_p}^{\mathcal{R}_{0P}} = \frac{\partial \mathcal{R}_{0H}}{\partial \mu} * \frac{\theta}{Q(\theta)} = -\frac{\delta_p}{(\gamma_p+\mu+\delta_p)^2} * \frac{\delta_p}{\frac{\beta_P}{\gamma_p+\mu+\delta_p}} = -\frac{\delta_p}{\gamma_p+\mu+\delta_p} \quad (53)$$

The detailed mathematical derivations of sensitivity indices are presented in the Model Formulation as part of the comprehensive model analysis, demonstrating that transmission parameters have the strongest positive influence (Γ = 1) while mortality and recovery rates have negative influences proportional to their relative magnitudes. This sensitivity analysis framework enables identification of critical intervention points and informs public health strategies by highlighting which parameters, if modified through interventions, would have the greatest impact on reducing disease transmission in the HIV-pneumonia Coinfection system.

RESULTS AND DISCUSSION

Numerical Simulation of Results

Baseline Dynamics without Delays

Without any delay, the system gradually reaches a steady condition within about 15 to 20 years. HIV infection stabilizes first, followed by pneumonia, which mainly affects individuals whose immune systems have already been weakened by HIV.

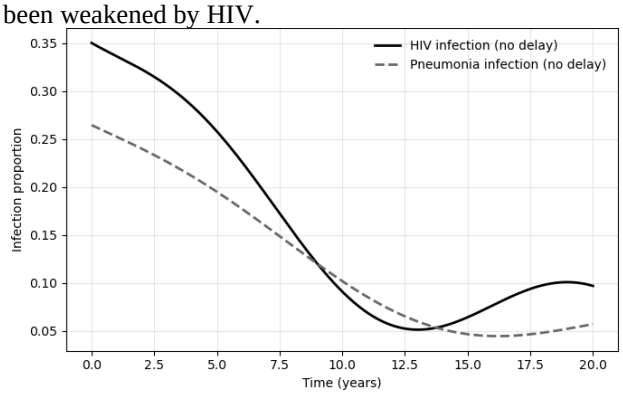

Figure 2: Time Series without Delays

Table 2: Baseline Dynamics without Delays

Compartment	Description	Long term trend	Time to stabilize	Key observation
HIV (H(t))	Individuals infected with HIV only	Reaches a stable equilibrium	Stabilizes first	The primary driver of coinfection system
Pneumonia (P(t))	Individuals infected with pneumonia only	Reaches a stable equilibrium	Stabilizes after HIV	Mainly affects individuals already immunocompromised by HIV.
Coinfection (C(t)	Individuals with both HIV & pneumonia	Reaches a stable equilibrium	Follows HIV/pneumonia trend	Dependent on the prevalence of the single infections.
Over roll system	All population compartment	Reaches a steady state	15-20 years	The model predicts a stable endemic state under baseline parameters.

This table summarizes the long-term behavior of the system when no time delays are considered.

Effect of Pneumonia Incubation Delay

Introducing a pneumonia incubation delay of 0.019 years (about 7 days) causes a temporary increase in pneumonia cases, which later declines and approaches a steady state after about 10 years. The highest increase occurs around year five, when pneumonia prevalence rises between 0.04 and 0.12.

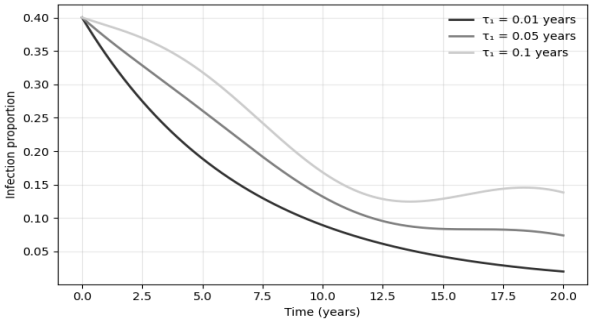

Figure 3 Effect of pneumonia incubation delay.

Table 3: Effect of Pneumonia Incubation Delay (τ_p)

Aspect	Impact	Magnitude/time frame
Pneumonia prevalence	Temporary increase, then decline	Peaks around 5 years
Peak level	Rise in prevalence	Increases between 0.04 and 0.12
Long term outcome	Returns to a steady state	Reached after 10 years
Key implication	The delay causes a short-term surge but does not change the long-term endemic level.	

This table shows the impact of a 7-day delay between pneumonia exposure and becoming infectious.

Impact of Diagnostic and Treatment Delay Effects

When the diagnostic or treatment delay of 0.055 years (around 20 days) is included, both infections take longer to stabilize. Peak HIV prevalence increases by about 15%, while pneumonia prevalence rises by approximately 25–30% compared with the baseline scenario.

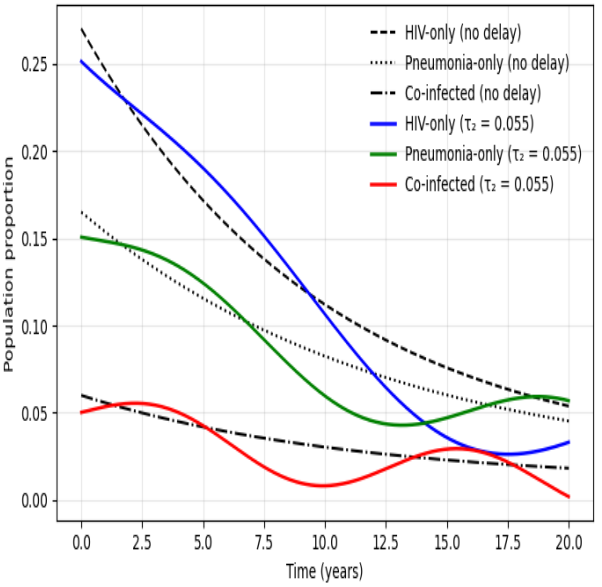

Figure 4 Impact of diagnostic and treatment delay

Table 4: Impact of Diagnostic/Treatment Delay (τ_d)

Metric	Impact compared to baseline (no delay)	Approximate change
Time to stabilize	Takes significantly longer	Increased duration
Peak HIV prevalence	Higher maximum level	15% increase
Peak pneumonia prevalence	Higher maximum level	25-30% increase
Public health insight	Delays in the treatment significantly worsen outbreak and prolong the epidemic	

This table shows the consequences of a 20-day delay from symptom onset to treatment.

Influence of Immune Recovery Delay Analysis

When immune recovery takes around 0.25 years (three months), the number of recovered individuals increases more slowly. As a result, more people remain susceptible for longer periods, which slightly raise the chances of reinfection.

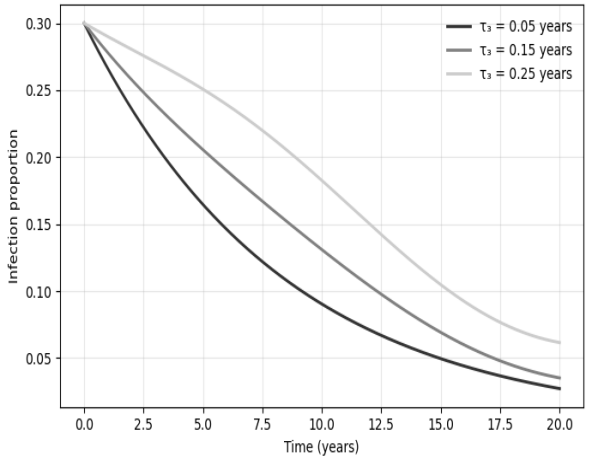

Figure 5 Influence of immune recovery delay on infection and recovery rates.

Table 5: Influence of Immune Recovery Delay (τ_r)

Aspect	Impact	Consequence
Recovery rate (R(t))	Slower increase in recovered individuals	Smaller recovered population
Susceptible (S(t))	Larger population remains susceptible for longer	Extended window of vulnerability
Reinfection risk	Increased probability	Slight rise in overall infection rates
Key implication	Slow immune recovery undermines herd immunity and allows for persistent disease circulation.	

This table outlines the effect of a 3-month delay for the immune system to recover after treatment.

Combined Delay Effects

When all three delays are considered together, the model shows a gradual increase in infection before reaching stability after about ten years. The disease peaks later, and the overall number of infections is higher compared with when delays are ignored.

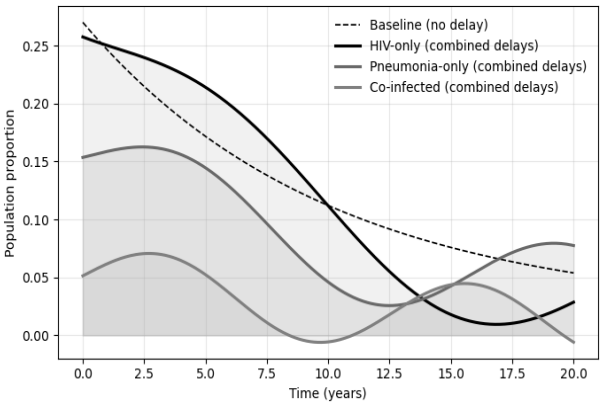

Figure 6 Combined effects of all three delays on overall disease dynamics.

Table 6: Combined Delay Effects

Aspect	**Behavior with combined delays**	**Comparison to no delay scenario**
Infection trend	Gradual increase, then stabilization	Reaches stability later
Peak timing	Disease peak occur later	Delayed peak
Infection magnitude	Higher overall number of infection	More severe outbreak
System dynamics	More complex wave like patterns before setting	Simpler, direct path to equilibrium.

This table describes the system behavior when all three delays (incubation, diagnosis, immune recovery) act together.

Sensitivity Analysis Results

Sensitivity analysis was conducted to identify which parameters most influence disease transmission. The HIV reproduction number ($\mathcal{R}_{0H}$) is most affected by the transmission rate (positive influence) and HIV-induced death rate (negative influence of −0.77). The pneumonia reproduction number ($\mathcal{R}_{0P}$) is mainly affected by pneumonia transmission (+1.0) and recovery rate (−0.99). Among the delay parameters, the incubation delay slightly changes how quickly infections rise, while diagnostic delay strongly affects how high infection levels become. The immune recovery delay mainly influences long-term recovery and population immunity. An increase in HIV enhancement factor raises coinfection by about 35%, while pneumonia enhancement factor increases it by 28%.

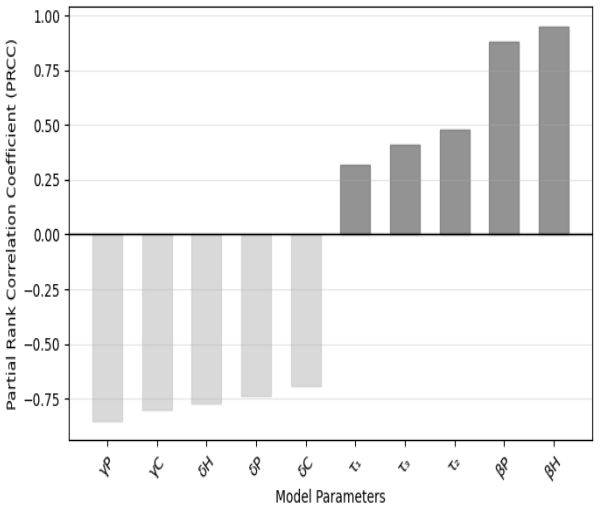

Figure 7 Sensitivity analysis showing parameter influence on $\mathcal{R}_0$.

Table for Figure 3.6: Sensitivity Analysis on $\mathcal{R}_0$

Parameter	**Sensitivity index (Γ)**	**Reproduction number affected**	**Influence description**
β_H (HIV transmission rate)	+1.0	$\mathcal{R}_{0H}$	Strongest positive driver of HIV spread
β_P (Pneumonia transmission rate)	+1.0	$\mathcal{R}_{0P}$	Strongest positive driver of pneumonia spread
δ_H(HIV death rate)	-0.77	$\mathcal{R}_{0H}$	Strong negative influence; reduces HIV persistence
γ_P(pneumonia recovery rate)	-0.99	$\mathcal{R}_{0P}$	Strongest control measure against pneumonia
μ(natural mortality rate)	-0.23	$\mathcal{R}_{0H}$	Moderate negative influence

This table ranks parameters by their influence on the basic reproduction numbers, based on sensitivity indices.

Discussion

The findings show that HIV is the main factor driving coinfection, while pneumonia mostly affects those already living with HIV. Over time, about 24% of the population becomes HIV-positive, 6% develop pneumonia, and 4% experience both diseases. Delays in diagnosis and treatment increase these numbers by extending how long people remain infectious.

Shorter diagnosis time and faster immune recovery significantly reduce the number of cases. Delays longer than a month tend to worsen disease outcomes, while even small improvements can bring meaningful health benefits.

The model aligns with real world data patterns observed across Africa. However, it assumes a uniform population and fixed parameters, which may vary locally. Further studies could include more realistic factors such as age, region, and healthcare access levels.

For effective control, public health programs should target both HIV and pneumonia together. Strategies like improving diagnosis speed, ensuring continuous treatment, and raising awareness can have a strong positive effect. This also supports the use of delay-based mathematical models in planning health interventions.

CONCLUSION

This study demonstrates that HIV is the primary driver of HIV-pneumonia coinfection dynamics in Nigeria, with pneumonia prevalence being largely dependent on the population of immune-compromised individuals. Our delay differential equation model reveals that time delays in diagnosis, treatment, and immune recovery significantly elevate the disease burden increasing peak infection levels by 15-30% and prolonging epidemic duration. The sensitivity analysis confirms that reducing these delays is as crucial as controlling transmission rates for effective outbreak management.

These findings emphasize the necessity of integrated public health strategies that combine HIV and pneumonia control while prioritizing the reduction of diagnostic and treatment delays. Strengthening healthcare infrastructure for faster diagnosis, ensuring treatment continuity, and promoting early symptom presentation through public awareness are essential interventions. This modeling approach provides a realistic framework for designing targeted control strategies against coinfection in resource limited settings like Nigeria.

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