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STABILITY ANALYSIS OF DELAYED CO-INFECTION MODEL OF PNEUMONIA AND MENINGITIS

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Abstract. This study develops and analyzes a delay differential model to investigate the co-infection dynamics of pneumonia and meningitis in a human population. The model incorporates biologically motivated delay factors-such as isolation, social distancing, and public awareness represented through an exponential survival term. These delays capture the influence of behavioral and treatment-related interventions on disease transmission. The model's dynamical behavior is examined through the analysis of positivity, boundedness, and equilibrium states. Both

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the disease-free equilibrium (DFE) and disease-existing equilibrium (DEE) are shown to be locally and globally asymptotically stable under appropriate threshold conditions. Sensitivity analysis highlights the influence of key parameters, including delay-related terms, on the basic reproduction numbers of pneumonia, meningitis, and their co-infection subsystem. Numerical simulations support the analytical results and illustrate how increasing delay-based interventions can effectively suppress disease spread. The findings provide valuable insights for public health strategies, suggesting that isolation, social distancing, and awareness campaigns can substantially mitigate co-infection risks in affected communities.

Keywords: co-infection; delay; equilibrium; reproduction number; stability.

2020 AMS Subject Classification: 92D30.

1. INTRODUCTION

When a pathogenic microorganism, such as a bacterium, virus, protozoan, or toxin, exists and may propagate from one host to another through cuddling, air, tiny droplets of water, food, or disease-carrying mother to toddler, the illness is categorized as an infectious disease [1, 2]. A prevalent infectious microbiological agent that causes pneumococcal diseases like pneumonia, meningitis, and sepsis is the streptococcus pneumonia bacterium [3].

The fluid and tissues surrounding the brain and spinal cord get infected when someone has spinal meningitis. Once an infection begins, it can quickly spread throughout the body. Meningococcus, or Neisseria meningitides, is the causative agent of meningococcal meningitis, a contagious and serious bacterial disease. It can be transmitted by close, prolonged contact with an infected individual who is releasing respiratory secretions into the air or by sharing personal items contaminated with these secretions. The bloodstream or meninges can become infected with Neisseria meningitides, which can produce a variety of symptoms. Elevated temperature, headache, feeling queasy, rash, stiff neck. The bacterial meningitis remain a life threatening condition, with reported case-fatality rates ranging from approximately $5-10\%$, generally within 48 to 72 hours [4]. In addition, asymptotic nasopharyngeal carriage of Neisseria meningitides has been reported in approximately $11-25\%$ of individuals in certain populations, playing a critical role in disease transmission [5].

Pneumonia is characterized by inflammation of the alveoli in one or both lungs. This inflammation often leads to the accumulation of fluid or purulent material within the air sacs, resulting in clinical manifestations such as productive cough with sputum, fever, chills, and respiratory

distress. The condition is etiologically diverse, triggered by various infectious agents such as bacteria, viruses, and fungi. Pneumonia remains a highly transmissible respiratory infection, posing significant health risks to both adolescents and adults. South Asia and Sub-Saharan Africa were among the worst hit, according to WHO. Pneumonia claimed the lives of 15% of the children. Under the age of five, 0.88 million children lost their lives in 2017 [3].

The concurrent presence of multiple pathogen strains within a population manifests when a host harbors simultaneous infections by two or more antigenically distinct pathogens or divergent strains of a single pathogen species [6]. In the modern era, it is frequent for one person to have two or more diseases co-infected with them [7]. People get different types of co-infection, like COVID-19 and tuberculosis, HIV/AIDS-pneumonia, HIV/AIDS-COVID-19. Although the portion of pneumonia cases that progress to meningitis is clinically low, typically reported at approximately $1-3\%$, the severe outcomes and high mortality associated with bacterial meningitis justify explicit modeling of co-infection dynamics at population level.

To understand the dynamics of infectious disease transmission, mathematical modeling techniques have proven essential in providing fundamental frameworks. Mathematical approaches for the analysis of infectious diseases were first applied in 1760 by the mathematician Daniel Bernoulli. Utilizing three classes-susceptible, infected, and recovered-Kermack and McKendrick developed the mathematical model for the spread of infectious diseases in 1927 [8]. Subsequently, other researchers developed models for various infectious diseases with the objective to investigate the models' stability and figure out the most effective steps for halting an epidemic [9, 10, 11, 12].

In 2008 Sharomi developed a mathematical model developed to examine HIV/AIDS-TB co-infection dynamics in the context of therapeutic measures [13]. Similarly, Kotola, Belela Samuel analyzes the numerical solution of meningitis and pneumonia with intervention [14].A key component of mathematical modeling is played by delayed dynamical models. The dynamics of disease are discussed extensively by employing delay tactics. Under certain delayed conditions, one can witness the behavior of an epidemic-disease. Simple to analyze the methods used to suppress or spread the disease. In 1979, Cooke studied the stability analysis of a vector disease model using the delay factor τ. The fundamental premise was that, in illnesses spread by

vectors like mosquitoes, when an infectious human infects a susceptible vector, the infectious agent develops inside the vector throughout a specific amount of time, denoted as τ. After this duration has passed, the vector might proceed to spread the infection to a susceptible human. The model's compatibility with actual phenomena is improved by the delay factor's inclusion. Despite notable improvements in delay-related outcomes Differential Equations DDEs over the past fifty years, a comprehensive theory for DDEs is still lacking, unlike Ordinary Differential Equations ODEs [15].

Naveed et al. used well-established mathematical assumptions to study the delay modeling of various infectious illnesses [3, 16]. However, to date, few studies have incorporated delay mechanisms into co-infection models involving two interacting diseases. This research distinguishes itself by focusing on the co-infection dynamics of pneumonia and meningitis-two pathogens that share overlapping clinical symptoms and affect the respiratory and central nervous systems. Immunosuppressive treatment in pneumonia patients may increase susceptibility to meningitis through reinfection or secondary infection, posing significant clinical challenges [17].

In this study, we specifically focus on bacterial pneumonia and bacterial meningitis, which remain among the most sever and preventable infectious disease worldwide. In particular, pneumococcal pneumonia caused by streptococcus pneumonia and meningococcal meningitis caused by Neisseria meningitides are antibiotic based treatment protocols. Viral and fungal form of pneumonia and meningitis while clinically important, are not included due to their fundamentally different transmission dynamics, therapeutic regimens and immune response.

clinically bacterial meningitis may arise as a severe complication of pneumonia, particularly when caused by shared pathogens such as streptococcus pneumonia and Hemophilus influenzae type b. Severe pneumonia can facilitate bacterial invasion into the blood stream, weaken host immune defenses, and increase permeability of the blood-brain barrier, thereby elevating the risk of secondary meningitis. Motivated by these mechanisms, the present study models pneumonia-meningitis interaction through co-infection pathways rather than strict stage wise disease progression.

Classical ODEs-based epidemic models assume that intervention such as isolation, social distancing, and treatment exert instantaneous effect on disease transmission.nHowever, in practice, these measure are subjected to inherent delays arising from behavioral adaption, logistical constraints, and policy implementation. To account of this temporal gap, we incorporate an delay in to transmission process, enabling the model to explicitly represent the time lagged impact of control measure.

Motivated by all these above interactions, we develop a delayed deterministic model that captures the transmission dynamics of meningitis–pneumonia co-infection. The model introduces an delay term $e^{-\mu\tau}, \tau \geq 0$, representing survival probability over time, to evaluate the impact of isolation, social distancing, and public awareness on disease transmission. This study is restricted to delay differential equations (DDEs) analysis within a deterministic framework.

The other part of this work is fragmented into sections as follows: Section 2 presents the formulation of the model; the analysis of the model is given in Section 3; the discussion of the results follows in Section 4; and the conclusion is in Section 5.

2. Mathematical Model

In this section, we develop a delayed mathematical model for meningitis-pneumonia co-infection, predicated on the assumption of closed population size and the exclusion of demographic factors such as gender, socioeconomic status, and ethnicity from influencing susceptibility to infection.All the infection, treatment, recovery, and vaccination parameters in the model correspond to bacterial pneumonia and bacterial meningitis. The vaccinated compartment represents immunization against bacterial strain for which licensed vaccines are available,such as pneumococcal and meningococcal vaccines. The model systematically partitions the human population $N(t)$ into epidemiological compartments reflecting varying disease states into seven mutually exclusive compartments, named as susceptible $S(t)$, pneumonia infected only $I_P(t)$, meningitis infected only $I_M(t)$, both co-infected class $I_{PM}(t)$, co-infection treated class $T_{PM}(t)$, vaccinated class $V_{PM}(t)$, and recovered $R(t)$. For those who have contracted meningitis or pneumonia, recuperating from natural immunity is significant before they can enter the recovered compartment. The natural recovery rate is denoted by d_M and d_P, respectively. Epidemiologically speaking, those in the recovered compartment lack lifelong immunity. The interaction

between pneumonia and meningitis is incorporated via cross-infection terms governed by modification parameter ω and θ, which represent increased susceptibility to secondary infection due to immune compromise or system bacterial dissemination.Individuals infected with pneumonia may acquire meningitis and enter the co-infected class, and similarly meningitis-infected individuals may develop pneumonia. This approach captures population-level interaction effected without assuming that necessarily evolves deterministically form pneumonia.

The contribution of co-infected individuals to the force of infection is scaled by modification parameters that accounts for increased infectiousness due to elevated bacterial loads. These parameters allow co-infection individuals to exerts a higher transmission potential that singly infected individuals, thereby capturing differential transmissibility in parsimonious manner. Pneumonia spread is modeled by the bilinear term, λ_P, formally expressed as:

$$\lambda_P(t) = (I_P(t) + I_{PM}(t)). \tag{1}$$

Additionally, individuals who are susceptible acquire meningitis infection after effective contact with individuals of the virus at a force of infection λ_M, expressed as:

$$\lambda_M(t) = (I_M(t) + I_{PM}(t)). \tag{2}$$

α_P and α_M are the effectual contact rates of pneumonia and meningitis transmission, respectively. Furthermore, we assumed pneumonia-only infected individuals can also develop an additional meningitis infection with force of infection and modification parameters $\omega\alpha_M\lambda_M$ and join the co-infection class I_{PM}. Due to those who only enter the co-infected compartment after contracting pneumonia with a prior infection, the number of co-infected compartments has increased, the modification parameter $\Theta\alpha_P\lambda_P$. A key aspect of this study is the inclusion of an artificial delay term $e^{-\mu\tau}$ where $\tau > 0$ represents the delay associated with disease progression, treatment, or intervention strategies such as isolation and medical response. This exponential term serves as a survival factor, representing the proportion of individuals who remain infectious after the delay period, considering natural mortality at rate μ. The incorporation of delay enhances the biological realism of the model by accounting for the time lag between exposure and infection onset or between infection and isolation etc. using the above basic model assumption and parameters described in Table (1), we have the following dynamical system:

TABLE 1. Description of parameters.

S. no	Parameter	Description
1	d_P	The natural recovery rate of individuals infected with pneumonia.
2	d_M	The intrinsic rate at which meningitis-infected individuals undergo spontaneous recovery.
3	d_{PM}	The rate at which co-infected individuals receive treatment and move into the treated category.
4	β	The recovery rate of co-infected individuals from both diseases.
5	μ	Natural death.
6	δ_P	The mortality rate attributed solely to pneumonia.
7	δ_M	The mortality rate due to meningitis alone.
8	δ_{PM}	The mortality rate resulting from co-infection with both diseases.
9	ω, Θ	Modification parameters, where $\omega \geq 1$ and $\Theta \geq 1$.
10	ρ	The rate at which immunity is lost.
11	λ	Recruitment rate.
12	ϕ	Vaccine waning rate.
13	π	The proportion of newborns who have been vaccinated.
14	α_P	Pneumonia contact rate.
15	α_M	Meningitis contact rate.

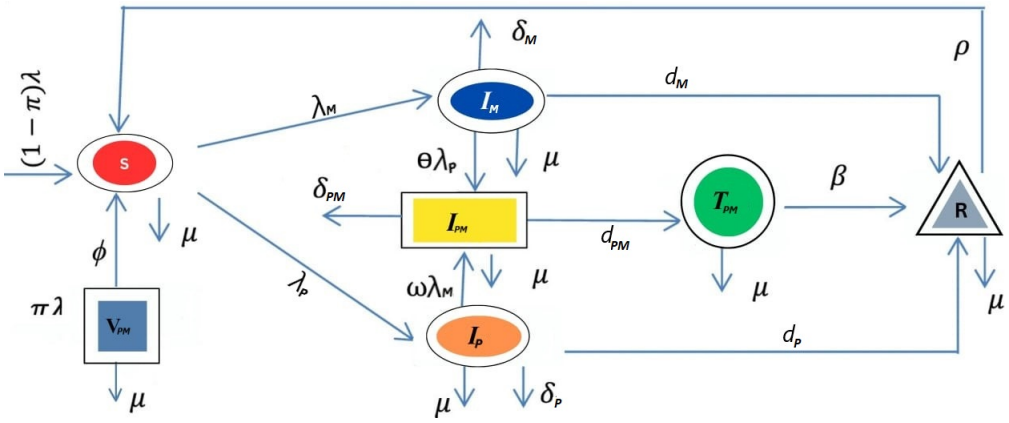

FIGURE 1. Flow chart of model.

$$
\begin{aligned}
\frac{dS}{dt} &= (1-\pi)\lambda + \rho R(t) + \phi V_{PM}(t) - \alpha_P \lambda_P(t-\tau)S(t-\tau)e^{-\mu\tau} \\
&\quad - \alpha_M \lambda_M(t-\tau)S(t-\tau)e^{-\mu\tau} - \mu S(t), \\
\frac{dI_P}{dt} &= \alpha_P \lambda_P(t-\tau)S(t-\tau)e^{-\mu\tau} - (\omega\alpha_M\lambda_M(t) + d_P + \delta_P + \mu)I_P(t), \\
\frac{dI_M}{dt} &= \alpha_M \lambda_M(t-\tau)S(t-\tau)e^{-\mu\tau} - (\Theta\alpha_P\lambda_P(t) + d_M + \delta_M + \mu)I_M(t), \\
\frac{dI_{PM}}{dt} &= \Theta\alpha_P\lambda_P(t)I_M(t) + \omega\alpha_M\lambda_M(t)I_P(t) - (d_{PM} + \delta_{PM} + \mu)I_{PM}(t), \\
\frac{dT_{PM}}{dt} &= d_{PM}I_{PM}(t) - (\beta + \mu)T_{PM}(t), \\
\frac{dV_{PM}}{dt} &= \pi\lambda - (\phi + \mu)V_{PM}(t), \\
\frac{dR}{dt} &= \beta T_{PM}(t) + d_M I_M(t) + d_P I_P(t) - (\rho + \mu)R(t).
\end{aligned} \tag{3}
$$

Where $\lambda_P(t)$ and $\lambda_M(t)$ are given in equations (1) and (2), and with initial conditions $S(0) > 0, I_P(0) \geq 0, I_M(0) \geq 0, I_{PM} \geq 0, T_{PM} \geq 0, V_{PM}(0) \geq 0$ and $R(0) \geq 0$.

2.1. Positivity and boundedness of solutions. Given the host population framework, it is imperative that all solutions maintain biological plausibility and mathematical rigor. To formalize this, we present the ensuing theorem ensuring the feasibility and positivity of solutions.

Theorem 1. *The solutions of system* (3) *exhibit positivity, uniqueness, and boundedness within the specified invariant region.*

$$\Omega = \{(S, I_P, I_M, I_{PM}, T_{PM}, V_{PM}, R) \in \mathbb{R}^7_+ : 0 \leq N(t) \leq \frac{\lambda}{\mu}\}$$

Proof. The vector field $F(x,t)$ given by the right-hand side of system (3) is continuously differentiable in x on $\mathbb{R}^7_+$. Therefore F is locally Lipschitz in x and by the Picard–Lindelöf theorem the system (3) admits a unique solution for any initial value $x(t_0) \in \mathbb{R}^7_+$.

To show positivity, assume $x(t)$ has nonnegative components. For each coordinate x_j evaluate the derivative on the corresponding boundary face where $x_j = 0$ while other coordinates remain ≥ 0. We obtain

$$\left.\frac{dS}{dt}\right|_{S=0} = (1-\pi)\lambda \geq 0, \quad \left.\frac{dI_P}{dt}\right|_{I_P=0} = \alpha_P\lambda_P S \geq 0, \quad \left.\frac{dI_M}{dt}\right|_{I_M=0} = \alpha_M\lambda_M S \geq 0,$$

$$\left.\frac{dI_{PM}}{dt}\right|_{I_{PM}=0} = \Theta\alpha_P\lambda_P(t)I_M + \omega\alpha_M\lambda_M(t)I_P \geq 0, \quad \left.\frac{dT_{PM}}{dt}\right|_{T_{PM}=0} = d_{PM}I_{PM} \geq 0,$$
$$\left.\frac{dV_{PM}}{dt}\right|_{V_{PM}=0} = \pi\lambda \geq 0, \quad \left.\frac{dR}{dt}\right|_{R=0} = \beta T_{PM} + d_M I_M + d_P I_P \geq 0.$$

Hence the vector field points inward on the boundary of $\mathbb{R}^7_+$, which implies that if $x(t_0) \in \mathbb{R}^7_+$ then $x(t)\mathbb{R}^7_+$ for all $t \geq t_0$.

Define the total population

$$N(t) = S + I_P + I_M + I_{PM} + T_{PM} + V_{PM} + R.$$

From (3) we deduce

$$\frac{dN}{dt} = \lambda - \delta_P I_P - \delta_M I_M - \delta_{PM} I_{PM} - \mu N \leq \lambda - \mu N,$$

since the terms $-\alpha_P I_P, -\alpha_M I_M, -d_{PM} I_{PM}$ are non-positive. Consider the scalar linear ODE

$$\frac{dY}{dt} = \lambda - \mu Y, \qquad Y(0) = N(0), \qquad Y(t) = N(0)e^{-\mu t} + \frac{\lambda}{\mu}\left(1 - e^{-\mu t}\right).$$

By comparison, $N(t) \leq Y(t)$ for all $t \geq 0$, and therefore

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

Thus the system (3) admits a unique solution which is positive and uniformly bounded in the biologically feasible region Ω. □

3. Model Analysis

We begin by determining the equilibrium points of the proposed model and subsequently analyze the system's dynamics in the vicinity of these steady states to gain deeper insight into the model's qualitative behavior. A comprehensive examination is conducted on the local and global stability of the equilibrium points corresponding to the meningitis-only, pneumonia-only, and co-infection sub-models, thereby elucidating the infection dynamics and inter-pathogen interactions near these critical thresholds.

3.1. Pneumonia only Sub-model. By systematically eliminating the meningitis-related compartments and transmission pathways from the full meningitis-pneumonia co-infection framework, we derive a reduced sub-model that encapsulates the standalone transmission dynamics of pneumonia. This decoupled system facilitates focused analysis of pneumonia progression in isolation from co-infection effects:

$$\begin{aligned}
\frac{dS}{dt} &= (1-\pi)\lambda + \rho R(t) + \phi V_P(t) - \alpha_P I_P(t-\tau)S(t-\tau)e^{-\mu\tau} - \mu S(t),\\
\frac{dI_P}{dt} &= \alpha_P I_P(t-\tau)S(t-\tau)e^{-\mu\tau} - (d_P+\delta_P+\mu)I_P(t),\\
\frac{dV_P}{dt} &= \pi\lambda - (\phi+\mu)V_P(t),\\
\frac{dR}{dt} &= d_P I_P(t) - (\rho+\mu)R(t).
\end{aligned} \tag{4}$$

3.1.1. *Local stability analysis of equilibrium points.* The right-hand side system (4) must be resolved in order to acquire all of the steady state solutions for the pneumonia sub-model. One of the DFE E_0^P is obtained by putting $I_P = 0$. As a result, the DFE are

$$E_0^P = \left(S^{''}, I_P^{''}, V^{''}, R^{''}\right) = \left(\frac{\lambda(\phi+(1-\pi)\mu)}{\mu(\phi+\mu)}, 0, \frac{\pi\lambda}{(\phi+\mu)}, 0\right)$$

A fundamental threshold parameter in epidemiological modeling is the basic reproduction number, denoted R_0, It denotes the projected number of new infections generated by one infectious individual upon entry into a completely susceptible population, serving as a critical threshold parameter in epidemiological modeling. It serves as a critical indicator for assessing the potential for disease invasion or elimination. We calculate R_0, denoted by $\left(R_0^P\right)$, of the pneumonia sub-model using the next generation method. Following the result given in [21] and system 4, we have:

$$\begin{bmatrix} I_P' \\ V_P' \\ R' \end{bmatrix} = \begin{bmatrix} \alpha_P \frac{(\phi+(1-\pi)\mu)}{\mu(\phi+\mu)} e^{-\mu\tau} & 0 & 0 \\ 0 & \pi\lambda & 0 \\ d_P & 0 & 0 \end{bmatrix} \begin{bmatrix} I_P \\ V_P \\ R \end{bmatrix} - \begin{bmatrix} (d_P+\delta_P+\mu) & 0 & 0 \\ 0 & (\mu+\phi) & 0 \\ 0 & 0 & (\rho+\mu) \end{bmatrix} \begin{bmatrix} I_P \\ V_P \\ R \end{bmatrix}$$

where

$$F = \begin{bmatrix} \alpha_P \frac{\lambda(\phi+(1-\pi)\mu)}{\mu(\phi+\mu)} e^{-\mu\tau} & 0 & 0 \\ 0 & \pi\lambda & 0 \\ d_P & 0 & 0 \end{bmatrix} \qquad V = \begin{bmatrix} (d_P+\delta_P+\mu) & 0 & 0 \\ 0 & (\phi+\mu) & 0 \\ 0 & 0 & (\rho+\mu) \end{bmatrix}$$

$$V^{-1} = \begin{bmatrix} \frac{1}{(d_P+\delta_P+\mu)} & 0 & 0 \\ 0 & \frac{1}{(\phi+\mu)} & 0 \\ 0 & 0 & \frac{1}{(\rho+\mu)} \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} \alpha_P \left(\frac{\lambda(\phi+(1-\pi)\mu)}{\mu(\phi+\mu)(d_P+\delta_P+\mu)} \right) e^{-\mu\tau} & 0 & 0 \\ 0 & \frac{\pi\lambda}{(\phi+\mu)} & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

The spectral radius of FV^{-1} is denoted by

$$R_0^P = \frac{\alpha_P e^{-\mu\tau}}{d_P+\delta_P+\mu} \cdot \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu\left(\phi+\mu\right)}.$$

Theorem 2. *The DFE point of pneumonia only sub-model is locally asymptotically stable LAS if* $R_0^P < 1$ *other wise unstable.*

Proof. The local stability of the equilibrium states is examined via linearization, wherein the nonlinear dynamical system is approximated in the neighborhood of each steady state by its first-order Taylor expansion. Specifically, we compute the Jacobian matrix evaluated at the equilibrium point and assess the sign of the real parts of its eigenvalues. Stability is ensured if all eigenvalues possess strictly negative real parts, indicating that perturbations around the equilibrium decay over time. The Jacobean matrix of sub-model system (4) at DFE point is

(5)

$$J(S,I_P,V_P,R) = \begin{pmatrix} -\mu & -\alpha_P \frac{\lambda(\phi+(1-\pi)\mu)}{\mu(\phi+\mu)} e^{-\mu\tau} & \phi & \rho \\ 0 & \alpha_P \frac{\lambda(\phi+(1-\pi)\mu)}{\mu(\phi+\mu)} e^{-\mu\tau} - (d_P+\delta_P+\mu) & 0 & 0 \\ 0 & 0 & -(\phi+\mu) & 0 \\ 0 & d_P & 0 & -(\mu+\rho) \end{pmatrix}$$

Two of eigenvalues for the matrix (5) are $\lambda_1 = -\mu$, and $\lambda_2 = -(\mu+\rho)$, the other two are obtained from reduced matrix

$$J_0 = \begin{pmatrix} \alpha_P \dfrac{\lambda(\phi+(1-\pi)\mu)}{\mu(\phi+\mu)} e^{-\mu\tau} - (d_P+\delta_P+\mu) & 0 \\ 0 & -(\phi+\mu) \end{pmatrix}$$

As $\lambda_1, \lambda_2 < 0$, the other two eigenvalues are negative if the trace of reduced matrix J_0 is negative and its determinant is positive. Thus the trace of reduced matrix is

$$trJ_0 = \alpha_P \frac{\lambda(\phi+(1-\pi)\mu)}{\mu(\phi+\mu)} e^{-\mu\tau} - (d_P+\delta_P+\mu) - (\phi+\mu)$$

and the determinant of J_0 is

$$detJ_0 = \left(-\alpha_P \left(\frac{\lambda(\phi+(1-\pi)\mu)}{\mu(\phi+\mu)} \right) e^{-\mu\tau} + (d_P+\delta_P+\mu) \right) (\phi+\mu))$$

The sign of determinant is positive if

$$\alpha_P \left(\frac{\lambda(\phi+(1-\pi)\mu)}{\mu(\phi+\mu)(d_P+\delta_P+\mu)} \right) e^{-\mu\tau} < 1$$

that shows that $detJ_0 > 0$ if $R_0^P < 1$ and $detJ_0 < 0$ if $R_0^P > 1$

As a result, the DFE points (E_0^P) of the sub-model is stable if $R_0^P < 1$ and unstable if $R_0^P > 1$. □

Let $c_1 = d_P + \delta_P + \mu$ and assume $I_P^* > 0$. Solving the steady-state equations of system (4) of the pneumonia-only subsystem yields

$$\alpha_P S^* e^{-\mu\tau} = c_1 \quad \Rightarrow \quad S^* = \frac{c_1}{\alpha_P e^{-\mu\tau}}, \qquad V_P^* = \frac{\pi\lambda}{\phi+\mu}, \qquad R^* = \frac{d_P I_P^*}{\rho+\mu}.$$

Writing $Y_P^* = \alpha_P I_P^*$ and substituting into the susceptible balance gives the linear relation

$$Y_P^* = \frac{\mu\, c_1(\rho+\mu)}{e^{-\mu\tau}\big(c_1(\rho+\mu) - d_P\rho\big)} (R_0^P - 1),$$

where R_0^P is the reproduction number of the pneumonia subsystem. Hence

$$I_P^* = \frac{Y_P^*}{\alpha_P} = \frac{\mu\, c_1(\rho+\mu)}{\alpha_P e^{-\mu\tau}\big(c_1(\rho+\mu) - d_P\rho\big)} (R_0^P - 1).$$

An endemic equilibrium with $I_P^* > 0$ exists if and only if $R_0^P > 1$.

Theorem 3. *For $R_0^P > 1$ the pneumonia sub-model* (4) *has unique DEE points.*

3.1.2. *Global Stability.* In order to rigorously assess the global asymptotic stability GAS of both the DFE and DEE states of system (4). we will prove following results:

Theorem 4. *Let $\tau > 0$ and assume all parameters are positive. Consider the pneumonia-only sub-model system* (4) *on the biologically feasible region $\mathbb{R}^4_+$ is GAS if $R_0^P < 1$ and unstable if $R_0^P > 1$.*

Proof. From the system (4) when $I_P = 0$. We define the Lyapunov function

$$\mathscr{H}(t) = S(t) - S'' - S'' \ln \frac{S(t)}{S''} + I_P(t) + \frac{(\rho+\mu)}{d_P} R(t) + \frac{\phi+\mu}{\pi\lambda} V_P(t).$$

Differentiating $\mathscr{H}$ along solutions of (4) and using the equilibrium relations, we obtain:

$$\dot{\mathscr{H}} = \left(1 - \frac{S''}{S}\right)\left[(1-\pi)\lambda + \rho R + \phi V_P - \alpha_P I_P S e^{-\mu\tau} - \mu S\right] + \alpha_P I_P S e^{-\mu\tau} - (d_P + \delta_P + \mu) I_P$$

$$+ \frac{\rho+\mu}{d_P}\left[d_P I_P - (\rho+\mu)R\right] + \frac{\phi+\mu}{\pi\lambda}\left[\pi\lambda - (\phi+\mu)V_P\right].$$

After substituting S'', V_P'' and R'' from E_0^P and simplifying, we get:

$$\dot{\mathscr{H}} = -\mu \frac{(S-S'')^2}{S} - (d_P + \delta_P + \mu)\left(1 - \frac{S''}{S} R_0^P\right) I_P,$$

where

$$R_0^P = \frac{\alpha_P e^{-\mu\tau}}{d_P + \delta_P + \mu} \cdot \frac{\lambda\left(\phi + \mu(1-\pi)\right)}{\mu(\phi+\mu)}.$$

If $R_0^P < 1$, then $\dot{\mathscr{H}} \leq 0$ with equality if and only if $S = S''$, $I_P = 0$, $V_P = V_P''$, and $R = R''$. By LaSalle's invariance principle, all solutions starting in $\mathbb{R}^4_0$ approach E_0^P as $t \to \infty$. Hence, E_0^P is GAS.

If $R_0^P > 1$, the linearized system about E_0^P has a positive eigenvalue, so the DFE is unstable.

□

Theorem 5. *Let $\tau > 0$ and assume all parameters are positive. Consider the pneumonia-only sub-model system* (4) *on the biologically feasible region*

$$\Sigma := \{(S, I_P, V_P, R) \in \mathbb{R}^4 : N = S + I_P + V_P + R \leq \tfrac{(1-\pi)\lambda}{\mu}\}.$$

Assume:

(H1) $R_0^P > 1$ and $c_1(\rho+\mu) > d_P\rho$ so the positive endemic equilibrium E_P^* exists and is unique.

(H2) The semi-flow generated by system (4) *on* Σ *is point-dissipative.*

(H3) The delayed vector field is cooperative and irreducible on the interior of Σ.

(H4) The system is uniformly persistent: there exists $\eta > 0$ *such that every solution with initial data in the interior of* Σ *satisfies* $\liminf_{t\to\infty} I_P(t) \geq \eta$.

Then the unique endemic equilibrium

$$E_P^* = (S^*, I_P^*, V_P^*, R^*) \in \operatorname{int}\Sigma$$

is GAS in the interior of Σ*:*

$$\lim_{t\to\infty}(S(t), I_P(t), V_P(t), R(t)) = E_P^*.$$

Proof. Under (H1) we already derived the DEE in closed form:

$$S^* = \frac{c_1}{\alpha_P E}, \quad V_P^* = \frac{\pi\lambda}{\phi+\mu}, \quad Y_P^* = \frac{\mu c_1(\rho+\mu)}{E(c_1(\rho+\mu)-d_P\rho)}(R_0^P - 1), \quad I_P^* = \frac{Y_P^*}{\alpha_P}, \quad R^* = \frac{d_P}{\rho+\mu}I_P^*.$$

The stated inequality $c_1(\rho+\mu) > d_P\rho$ guarantees the denominator in Y_P^* is positive, and hence $I_P^* > 0$ exactly when $R_0^P > 1$. The system (4) has constant recruitment and linear natural death.

$$0 \leq N(t) \leq N_{\max} := \max\{N(0), \tfrac{(1-\pi)\lambda}{\mu}\},$$

so solutions that start in Σ remain in Σ for all forward time. This proves (H2). Writing the system in the retarded functional-differential form

$$\dot{x}(t) = F\big(x(t), x(t-\tau)\big), \qquad x = \big(S, I_P, V_P, R\big), \tag{6}$$

Take

$$x(t) = \begin{pmatrix} S(t) \\ I_P(t) \\ V_P(t) \\ R(t) \end{pmatrix},$$

The system (4) can be written in retarded functional-differential form as

$$\dot{x}(t) = F\big(x(t), x(t-\tau)\big),$$

where

$$F\big(x(t),x(t-\tau)\big) = \begin{pmatrix} (1-\pi)\lambda + \rho R(t) + \phi V_P(t) - \alpha_P S(t-\tau) I_P(t-\tau)\, e^{-mu\tau} - \mu S(t) \\ \alpha_P S(t-\tau) I_P(t-\tau)\, e^{-mu\tau} - c_1 I_P(t) \\ \pi\lambda - (\phi+\mu) V_P(t) \\ d_P I_P(t) - (\rho+\mu) R(t) \end{pmatrix},$$

$$c_1 := d_P + \delta_P + \mu.$$

We now compute the two Jacobian-like matrices

$$A_0\big(x(t),x(t-\tau)\big) := \frac{\partial F}{\partial x}\big(x(t),x(t-\tau)\big), \qquad A_1\big(x(t),x(t-\tau)\big) := \frac{\partial F}{\partial y}\big(x(t),y\big)\Big|_{y=x(t-\tau)},$$

so that the linearization of the system takes the form

$$\dot{z}(t) \approx A_0\, z(t) + A_1\, z(t-\tau).$$

From the definition of F above, we obtain

$$A_0 = \begin{pmatrix} -\mu & 0 & \phi & \rho \\ 0 & -c_1 & 0 & 0 \\ 0 & 0 & -(\phi+\mu) & 0 \\ 0 & d_P & 0 & -(\rho+\mu) \end{pmatrix},$$

and

$$A_1 = \begin{pmatrix} -\alpha_P I_P(t-\tau)e^{-\mu\tau} & -\alpha_P S(t-\tau)e^{-\mu\tau} & 0 & 0 \\ \alpha_P I_P(t-\tau)e^{-\mu\tau} & \alpha_P S(t-\tau)e^{-\mu\tau} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}.$$

Here, A_0 contains the partial derivatives with respect to the current-state variables $x(t)$, and A_1 contains the partial derivatives with respect to the delayed variables $x(t-\tau)$. Equivalently, A_0 is a Metzler matrix. The infection terms enter bilinearly as $-\alpha_P S(t-\tau)I_P(t-\tau)e^{-\mu\tau}$ in $\dot{S}$ and $+\alpha_P S(t-\tau)I_P(t-\tau)e^{-\mu\tau}$ in $\dot{I}_P$; these yield nonnegative partial derivatives of $\dot{I}_P$ w.r.t. $I_P(t-\tau)$ and of $\dot{S}$ w.r.t. $I_P(t-\tau)$ in the cooperative sign pattern. All other off-diagonal entries

are nonnegative or zero. Under the irreducibility condition in (H3), the semi-flow generated by the retarded system (6) is *strongly monotone* on the interior of Σ [18]. This ensures strict ordering is eventually improved by the flow.

For $R_0^P > 1$, there is a uniform lower bound $\eta > 0$ with $\liminf_{t\to\infty} I_P(t) \geq \eta$ for all initial histories in the interior of Σ. [19, 20] Thus the assumption (H4) verified. With (H2)–(H4) established, the semiflow on the compact attractor in the interior of Σ is strongly monotone and point-dissipative with a unique DEE. Because the interior equilibrium is unique, every such trajectory must converge to E_P^*. Thus E_P^* is GAS in the interior of Σ. □

3.1.3. *Sensitivity Analysis of Parameter.* We addressed the study of the pneumonia sub-model's sensitive parameters in this subsection. The sensitivity parameter q, which refers to a parameter q, is established by;

$$S_P = \frac{\partial R_P}{\partial q} . \frac{q}{R_P}$$

Where

$$R_0^P = \frac{\alpha_P e^{-\mu\tau}}{d_P + \delta_P + \mu} \cdot \frac{\lambda\left(\phi + \mu(1-\pi)\right)}{\mu\left(\phi + \mu\right)} \qquad c_1 = d_P + \delta_P + \mu.$$

TABLE 2. Sensitivity indices for the pneumonia-only sub-model based on parameter values.

Parameter	Description	Sensitivity Index
ϕ	Vaccine waning rate	+0.0432
α_P	Pneumonia contact rate	+1
λ	Recruitment rate	+0.9941
π	Portion of vaccinated newborns	-0.0812
δ_P	Pneumonia-induced death rate	-0.1965
μ	Natural death rate	-0.4168
d_P	Natural recovery rate of pneumonia cases	-0.9939

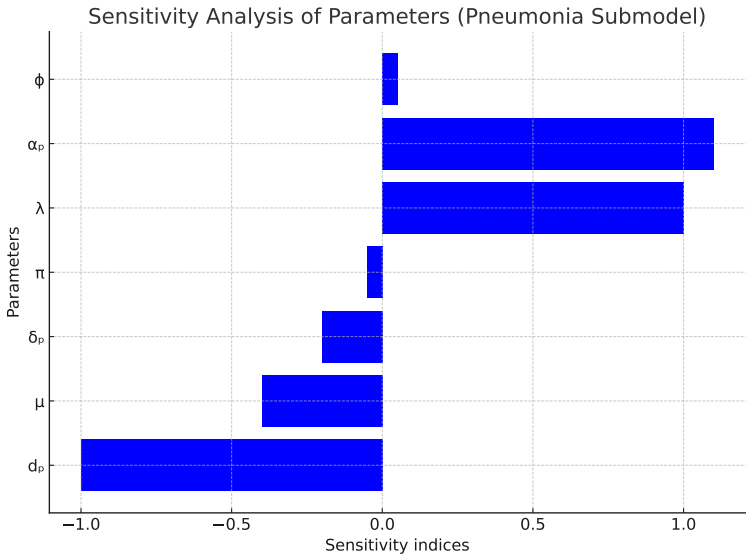

FIGURE 2. Sensitivity indices.

The Sensitivity analysis for each parameter of the system (4) with respect to R_0^P is given as:

$$S_\lambda = \frac{\partial R_0^P}{\partial \lambda} = 1,$$

$$S_{\alpha_P} = \frac{\partial R_0^P}{\partial \alpha_P} = 1,$$

$$S_{d_P} = \frac{\partial R_0^P}{\partial d_P} = -\frac{d_P}{c_1} < 0, \qquad S_{\delta_P} = -\frac{\delta_P}{c_1} < 0,$$

$$S_\pi = \frac{\partial R_0^P}{\partial \pi} = -\frac{\mu\pi}{\mu(1-\pi)+\phi} < 0,$$

$$S_\phi = \frac{\partial R_0^P}{\partial \phi} = \frac{\mu\phi\pi}{\big(\mu(1-\pi)+\phi\big)\big(\mu+\phi\big)} = \frac{\mu\phi\pi}{(\phi+\mu(1-\pi))(\phi+\mu)} > 0,$$

$$S_\mu = \mu\frac{\partial R_0^P}{\partial \mu} = -\mu\tau - \frac{\mu}{c_1} + \frac{\mu(1-\pi)}{\phi+\mu(1-\pi)} - 1 - \frac{\mu}{\phi+\mu} < 0.$$

3.2. Meningitis only Sub-model. Eliminating the pneumonia-related compartments and transmission pathways from system (3) yields a reduced sub-model that characterizes the standalone dynamics of meningitis as follows:

$$
\begin{aligned}
\frac{dS}{dt} &= (1-\pi)\lambda + \rho R(t) + \phi V_M(t) - \alpha_M I_M(t-\tau)S(t-\tau)e^{-\mu\tau} - \mu S(t),\\
\frac{dI_M}{dt} &= \alpha_M I_M(t-\tau)S(t-\tau)e^{-\mu\tau} - (d_M+\delta_M+\mu)I_M(t),\\
\frac{dV_P}{dt} &= \pi\lambda - (\phi+\mu)V_M(t),\\
\frac{dR}{dt} &= d_M I_M(t) - (\rho+\mu)R(t).
\end{aligned} \tag{7}
$$

3.2.1. *Local stability analysis of equilibrium points.* In order to obtain all of the stable state solutions for the Meningitis sub-model, the RHS system (7) requires to be solve as one of DFE point E_0^M is obtained by putting $I_M = 0$.

$$E_0^M = \left(S^{"}, I_M^{"}, V_M{"}, R^{"}\right) = \left(\frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)}, 0, \frac{\pi\lambda}{(\mu+\phi)}, 0\right)$$

Using next-generation technique, we compute the meningitis sub-model's R_0, represented as $\left(R_0^M\right)$. By system (7) and the result from [21], we obtain:

$$R_0^M = \frac{\alpha_M\,\lambda\left(\phi+\mu(1-\pi)\right)}{\mu\left(\phi+\mu\right)\left(d_M+\delta_M+\mu\right)}\,e^{-\mu\tau}.$$

Theorem 6. *The DFE point of Meningitis only sub model is LAS if $R_0^M < 1$ other wise unstable.*

Proof. The Jacobean matrix of system (7) using DFE point is

$$
G(S,I_M,V_M,R) = \begin{pmatrix}
-\mu & -\alpha_M\frac{\lambda(\phi+\mu(1-\pi))}{\mu(\phi+\mu)}e^{-\mu\tau} & \phi & \rho\\
0 & \alpha_M\frac{\lambda(\phi+\mu(1-\pi))}{\mu(\phi+\mu)}e^{-\mu\tau} - (d_M+\delta_M+\mu) & 0 & 0\\
0 & 0 & -(\mu+\phi) & 0\\
0 & d_M & 0 & -(\mu+\rho)
\end{pmatrix} \tag{8}
$$

Two of eigenvalues for the matrix (8) are $\lambda_1 = -\mu$ and $\lambda_2 = -(\mu+\rho)$ the other two are obtained from reduced matrix

$$G_0 = \begin{pmatrix}
\alpha_M\frac{\lambda(\phi+\mu(1-\pi))}{\mu(\phi+\mu)}e^{-\mu\tau} - (d_M+\delta_M+\mu) & 0\\
0 & -(\mu+\phi)
\end{pmatrix}$$

As $\lambda_{1,2} = -\mu < 0$, the other two eigenvalues are obtained by

$$\lambda^2 - \left(\operatorname{tr} G_0\right)\lambda + \det(G_0) = 0$$

$$trG_0 = \alpha_M \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)} e^{-\mu\tau} - (d_M+\delta_M+\mu) - (\mu+\phi)$$
$$detG_0 = \left(-\alpha_M \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)} e^{-\mu\tau} + (d_M+\delta_M+\mu)\right)(\mu+\phi)$$

The sign of determinant is positive if

$$\frac{\alpha_M \lambda\left(\phi+\mu(1-\pi)\right)}{\mu\left(\phi+\mu\right)\left(d_M+\delta_M+\mu\right)} e^{-\mu\tau} < 1$$

that shows that $detG_0 > 0$ if $R_0^M < 1$ and $detG_0 < 0$ if $R_0^M > 1$. As a result, the DFE point E_0^M of the sub-model is stable if $R_0^M < 1$ and unstable if $R_0^M > 1$. □

The DEE point of the meningitis only sub-model is obtained by:

$$\begin{aligned}
0 &= (1-\pi)\lambda + \rho R(t) + \phi V_M(t) - Y_M(t-\tau)S(t-\tau)e^{-\mu\tau} - \mu S(t) \\
0 &= Y_M(t-\tau)S(t-\tau)e^{-\mu\tau} - c_2 I_M(t) \\
0 &= \pi\lambda - (\phi+\mu)V_M(t) \\
0 &= d_M I_M(t) - (\rho+\mu)R(t)
\end{aligned} \tag{9}$$

Let $E_M^0 = (S^*, I_M^*, V_M^*, R^*)$ be the DEE point and $c_2 = (d_M+\delta_M+\mu)$. we solve the system of equation in term of force of infection $Y_M^* = \alpha_M I_M^*$. After solving the system (9), we have following expression:

$$V_M^* = \frac{\pi\lambda}{\phi+\mu}, \qquad R^* = \frac{d_M I_M^*}{\rho+\mu}, \qquad I_M^* = \frac{Y_M^* S^* e^{-\mu\tau}}{c_2}..$$

Substituting V_M^*, R^*, I_M^* into the first equation of system (9) and solving for S^* yields

$$S^* = \frac{(1-\pi)\lambda + \dfrac{\phi\pi\lambda}{\phi+\mu}}{\mu + Y_M^* e^{-\mu\tau}\dfrac{c_2(\rho+\mu)-\rho d_M}{c_2(\rho+\mu)}}.$$

Equivalently, using $Y_M^* = \alpha_M I_M^*$ together with $I_M^* = \dfrac{Y_M^* S^* e^{-\mu\tau}}{c_2}$ we obtain the identity

$$Y_M^*\left(1 - \frac{\alpha_M S^* e^{-\mu\tau}}{c_2}\right) = 0.$$

Therefore at steady-state either

$$Y_M^* = 0 \quad \text{(disease-free equilibrium)}$$

or the nontrivial endemic condition

$$\frac{\alpha_M S^* e^{-\mu\tau}}{c_2} = 1\,.$$

Evaluating the threshold at the DFE ($S = S^{"}$) gives the basic reproduction number

$$R_0^M \;=\; \frac{\alpha_M \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)} e^{-\mu\tau}}{c_2}.$$

A nonzero endemic equilibrium requires solving the nonlinear scalar equation obtained by substituting the expression for $S^*(Y_M^*)$ above into the endemic condition.

$$\frac{\alpha_M S^*(Y_M^*) e^{-\mu\tau}}{c_2} = 1,$$

Hence $Y_M^* > 0$ when $R_0^M > 1$.

Theorem 7. *For $R_0^M > 1$ the meningitis sub-model system* (7) *has unique DEE point.*

3.2.2. *Global Stability.* To illustrate the globally asymptotically stable GAS at both equilibria of the system (7). we will prove following results:

Theorem 8. *Let $\tau > 0$ and assume all parameters are positive. Consider the system* (7) *on the biologically feasible region $\Sigma_1 \subset \mathbb{R}^4_+$ then E_0^M is GAS in Σ_1. If $R_0^M < 1$.*

Proof. We use a Volterra-type Lyapunov function adapted to the presence of a vaccinated class with nonzero equilibrium. Define

$$X(S, I_M, V_M, R) = \left(S - S^{"} - S^{"} \ln \frac{S}{S^{"}}\right) + \left(V_M - V_M^{"} - V_M^{"} \ln \frac{V_M}{V_M^{"}}\right) + I_M + R.$$

denote the delayed infection term by

$$\mathscr{T}(t) := \alpha_M I_M(t-\tau) S(t-\tau) e^{-\mu\tau}.$$

Differentiate X along solutions of system (7).

$$\dot{X} = \left(1 - \frac{S^{"}}{S}\right)\dot{S} + \left(1 - \frac{V_M^{"}}{V_M}\right)\dot{V}_M + \dot{I}_M + \dot{R}. \tag{10}$$

Now substitute the right-hand sides of system (7) into (10) and grouping terms gives

$$\begin{aligned}\dot{X} &= \Big(1-\frac{S^{"}}{S}\Big)\big((1-\pi)\lambda+\phi V_M-\mu S\big)+\Big(1-\frac{S^{"}}{S}\Big)\rho R\\ &+\Big(1-\frac{V_M^{"}}{V_M}\Big)\big(\pi\lambda-(\phi+\mu)V_M\big)\\ &+\Big(1-\frac{S^{"}}{S}\Big)\big(-\mathscr{T}(t)\big)+\mathscr{T}(t)-c_2I_M+d_MI_M-(\rho+\mu)R.\end{aligned}\tag{11}$$

Use the DFE point substitute terms in (11).

$$\begin{aligned}\dot{X} &= -\mu\frac{(S-S^{"})^2}{S}-\phi\frac{(S-S^{"})(V_M-V_M^{"})}{S}-(\phi+\mu)\frac{(V_M-V_M^{"})^2}{V_M}\\ &+\Big(1-\frac{S^{"}}{S}\Big)\rho R+\Big(1-\frac{S^{"}}{S}\Big)\big(-\mathscr{T}(t)\big)+\mathscr{T}(t)-c_2I_M+d_MI_M-(\rho+\mu)R.\end{aligned}\tag{12}$$

The two terms involving $\mathscr{T}(t)$ read

$$\Big(1-\frac{S^{"}}{S}\Big)\big(-\mathscr{T}(t)\big)+\mathscr{T}(t)=\mathscr{T}(t)\frac{S^{"}}{S}.$$

$$\mathscr{T}(t)\frac{S^{"}}{S}-c_2I_M+d_MI_M=\mathscr{T}(t)\frac{S^{"}}{S}-(c_2-d_M)I_M-d_MI_M=\mathscr{T}(t)\frac{S^{"}}{S}-c_2I_M.$$

Now by $\mathscr{T}(t)$ and $I_M(t)$:

$$\mathscr{T}(t)=\alpha_MI_M(t-\tau)S(t-\tau)E.$$

A standard bounding argument for nonnegative solutions of system (7) gives [20]

$$\mathscr{T}(t)\frac{S^{"}}{S}\le\alpha_MS^{"}E\,I_M(t),$$

because for nonnegative solutions and $S(t)>0$ we have $I_M(t-\tau)S(t-\tau)\le I_M(t)S^{\sup}$ in the invariant region. Using this estimate we obtain from (12) the inequality

$$\dot{X}\le-\mu\frac{(S-S^{"})^2}{S}-(\phi+\mu)\frac{(V_M-V_M^{"})^2}{V_M}-(\rho+\mu)R-\big(c_2-\alpha_MS^{"}e^{-\mu\tau}\big)I_M.$$

Replace $\alpha_MS^{"}e^{-\mu\tau}=c_2R_0^M$

$$\dot{X}\le-\mu\frac{(S-S^{"})^2}{S}-(\phi+\mu)\frac{(V_M-V_M^{"})^2}{V_M}-(\rho+\mu)R-c_2\big(1-R_0^M\big)I_M.$$

Clearly if $\mathscr{R}_0^M<1$ then every term on the right-hand side is non-positive and

$$\dot{X}\le0\quad\text{on }\Sigma_1,$$

with $\dot{X} = 0$ only when

$$S = S^{"}, \qquad V_M = V_M^{"}, \qquad R = 0, \qquad I_M = 0,$$

i.e. only at the DFE E_0^M. By LaSalle's invariance principle E_0^M is globally asymptotically stable in Σ_1. If $R_0^M > 1$ then DFE point has a positive eigenvalue then is unstable. □

Theorem 9. *Assume $\tau > 0$ and all parameters of system (7) are positive. Suppose system (7) admits a positive DEE*

$$C_4 = (S^*, I_M^*, V_M^*, R^*) \in \Sigma_1, \qquad I_M^* > 0,$$

and that C_4 is unique in Σ_1. Then for $R_0^M > 1$ $\dot{\mathscr{V}} \leq -\mathscr{W}$ for a positive definite function $\mathscr{W}$ vanishing only at C_4, and consequently C_4 is GAS in Σ_1.

Proof. Let $K_1, K_2, K_3, K_4, \beta > 0$ be positive constants such that the following collection of inequalities holds for some positive constants C_1, C_2, C_3:

$$\begin{aligned} & K_1\mu \ \geq \ C_2, \\ & K_3(\phi+\mu) \ \geq \ C_0, \qquad K_4(\rho+\mu) \ \geq \ C_0, \\ & K_2c_2 - (C_1 + C_3 + \tfrac{\beta}{2}) \ \geq \ 0, \end{aligned} \tag{13}$$

where $C_0, C_1, C_2, C_3 > 0$ are suitably chosen constants [22]. Define the Lyapunov–Krasovskii functional centered at the endemic C_4:

$$\mathscr{V}(t) = \sum_{i=1}^{4} K_i\Big(x_i(t) - x_i^* - x_i^* \ln\frac{x_i(t)}{x_i^*}\Big) + \beta \int_{t-\tau}^{t} \big(I_M(s) - I_M^*\big)^2 ds,$$

where $(x_1, x_2, x_3, x_4) = (S, I_M, V_M, R)$ and $K_i, \beta > 0$ are chosen constant. Differentiate $\mathscr{V}$ with system (7).

$$\dot{\mathscr{V}} = \sum_{i=1}^{4} K_i\Big(1 - \frac{x_i^*}{x_i}\Big)\dot{x}_i + \beta\big(I_M(t) - I_M^*\big)^2 - \beta\big(I_M(t-\tau) - I_M^*\big)^2.$$

$$\mathscr{T}(t) := \alpha_M I_M(t-\tau) S(t-\tau) e^{-\mu\tau}.$$

After substitution grouped the terms into three categories:

- negative definite quadratic forms arising from the Volterra terms:

$$\mu K_1 \frac{(S-S^*)^2}{S}, \quad K_2 c_2 \frac{(I_M - I_M^*)^2}{I_M}, \quad (\phi+\mu)K_3 \frac{(V_M - V_M^*)^2}{V_M}, \quad (\rho+\mu)K_4 \frac{(R-R^*)^2}{R};$$

- a linear term involving $\alpha_M S^* e^{-\mu\tau} - c_2$:

$$K_2\big(\alpha_M S^* E - c_2\big)(I_M - I_M^*),$$

- delay remainder terms:

$$\Theta_{\text{delay}} = K_2\Big(1 - \frac{I_M^*}{I_M}\Big)\alpha_M\big(I_M(t-\tau)S(t-\tau) - I_M^* S^*\big)E + \beta\big(I_M - I_M^*\big)^2 - \beta\big(I_M(t-\tau) - I_M^*\big)^2.$$

Use the identity

$$I_M(t-\tau)S(t-\tau) - I_M^* S^* = S^*\big(I_M(t-\tau) - I_M^*\big) + I_M(t-\tau)\big(S(t-\tau) - S^*\big),$$

and apply Young/Cauchy inequalities to the two resulting pieces [23].

$$K_2\alpha_M E S^*\Big(1 - \frac{I_M^*}{I_M}\Big)\big(I_M(t-\tau) - I_M^*\big) \le \frac{\beta}{2}\big(I_M(t-\tau) - I_M^*\big)^2 + C_1\big(I_M - I_M^*\big)^2,$$
$$K_2\alpha_M E\Big(1 - \frac{I_M^*}{I_M}\Big) I_M(t-\tau)\big(S(t-\tau) - S^*\big) \le C_2\frac{(S-S^*)^2}{S} + C_3\big(I_M - I_M^*\big)^2.$$

Using these bounds and the last two terms of $\dot{\mathscr{V}}$, we obtain an inequality of the form

$$\begin{aligned}\dot{\mathscr{V}} \le &-\Big(\mu K_1 - C_2\Big)\frac{(S-S^*)^2}{S} - \Big(K_2 c_2 - (C_1 + C_3 + \tfrac{\beta}{2})\Big)(I_M - I_M^*)^2 \\ &- (\phi+\mu)K_3\frac{(V_M - V_M^*)^2}{V_M} - (\rho+\mu)K_4\frac{(R-R^*)^2}{R}.\end{aligned}$$

Thus, if the weights K_i, β are chosen so that the inequalities (13) hold, then the right-hand side is a negative semi-definite function which is strictly negative except at the equilibrium C_4. Equivalently there exists a positive definite function $\mathscr{W}$ vanishing only at C_4 with

$$\dot{\mathscr{V}} \le -\mathscr{W}.$$

By LaSalle's invariance principle [20, 24], therefore all trajectories in Σ_1 approach C_4 as $t \to \infty$. So C_4 is GAS. □

3.2.3. *Sensitivity Analysis of Meningitis only.*

$$S_M = \frac{\partial R_M}{\partial q_M} . \frac{q_M}{R_M}$$

where

$$R_0^M = \frac{\alpha_M \lambda \left(\phi + \mu(1-\pi)\right)}{\mu\left(\phi+\mu\right)\left(d_M + \delta_M + \mu\right)} e^{-\mu\tau}.$$

TABLE 3. Sensitivity indices for the Meningitis-only sub-model.

Parameter	**Description**	**Sensitivity indices**
ϕ	vaccine wanes rate	$+0.0432$
α_M	Meningitis contact rate	$+1$
λ	Recruitment rate	$+0.9843$
π	The portion of vaccinated new born	-0.2315
δ_M	meningitis only caused death rate	-0.1989
μ	Natural death rate	-0.4768
d_M	The rate at which meningitis infected individuals are recovered naturally	-0.6951

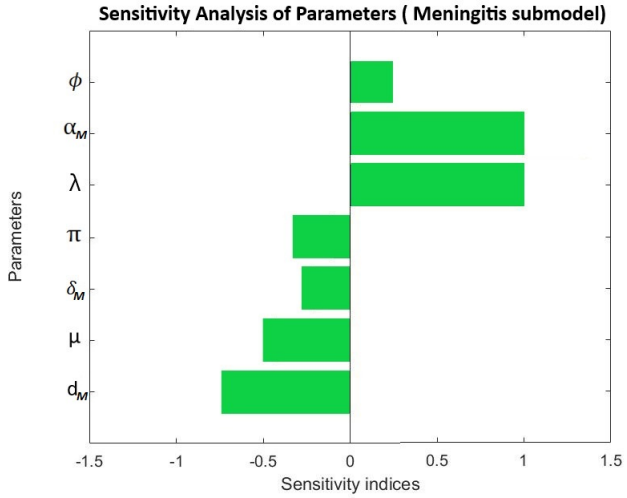

FIGURE 3. Sensitivity indices.

The Sensitivity analysis for each parameter of the system (7) with respect to R_0^M is given as:

$$
\begin{aligned}
S_\lambda &= 1 > 0,\\
S_{\alpha_M} &= 1 > 0,\\
S_\pi &= -\frac{\pi\mu}{\phi+\mu(1-\pi)} < 0,\\
S_\phi &= \frac{\phi\,\mu\,\pi}{(\phi+\mu)\,(\phi+\mu(1-\pi))} > 0,\\
S_\mu &= -\mu\left(\frac{1-\pi}{\phi+\mu(1-\pi)} - \frac{1}{\mu} - \frac{1}{\phi+\mu} - \frac{1}{d_M+\delta_M+\mu} - \tau\right) < 0,\\
S_{d_M} &= -\frac{d_M}{d_M+\delta_M+\mu} < 0,\\
S_{\delta_M} &= -\frac{\delta_M}{d_M+\delta_M+\mu} < 0.
\end{aligned}
$$

3.3. Analysis of Co-infection model. We next derive the equilibrium configurations of the full co-infection model by solving the system (3) under steady-state assumptions, thereby characterizing the long-term dynamical behavior of the interacting disease compartments. The DFE point

$$
E_0^{PM} = (S^{\cdot\cdot}, I_{\dot{P}}^{\cdot\cdot}, I_{\dot{M}}^{\cdot\cdot}, I_{\dot{P}\dot{M}}^{\cdot\cdot}, T_{\dot{P}\dot{M}}^{\cdot\cdot}, V_{\dot{P}\dot{M}}^{\cdot\cdot}, R^{\cdot\cdot}) = \left(\frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)},\, 0,\, 0,\, 0,\, 0,\, \frac{\pi\lambda}{\phi+\mu},\, 0\right).
$$

3.3.1. *local stability.* It is vital to differentiate newly infectious from all other host population changes in order to calculate the R_0.

$\Psi_(i)(x)$ the incidence rate of new infections in the infectious compartment i.

$\Upsilon_i^+(x)$ is the transition rate of individuals into the compartment i.

$\Upsilon_i^-(x)$ is the transition rate of individuals out of the compartment i.

Then $\Upsilon_i(x) = \Upsilon_i^-(x) - \Upsilon_i^+(x)$ also $\Psi = \dfrac{\partial\Psi(X_0)}{\partial X_j}$ and $\Upsilon = \dfrac{\partial\upsilon_i(X_0)}{\partial X_j}$

where Ψ and Υ are $m \times m$ as m is number of compartment.

$$\Psi_i(x) = \begin{bmatrix} 0 \\ \alpha_P \lambda_P S e^{-\mu\tau} \\ \alpha_M \lambda_M S e^{-\mu\tau} \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \Upsilon_i(x) = \begin{bmatrix} (\lambda_p + \lambda_M) S e^{-\mu\tau} + \mu S - (1-\pi)\lambda - \rho R - \phi V_{PM} \\ (\omega \alpha_M \lambda_M + d_P + \delta_P + \mu) I_P \\ (\Theta \alpha_P \lambda_P + d_M + \delta_M + \mu) I_M \\ -(\delta_P + \delta_P M + \mu) I_{PM} - \omega \alpha_P \lambda_M - \Theta \alpha_M \lambda_P \\ (\mu + \phi) V_{PM} - \pi\lambda \\ (\beta + \mu) T_{PM} - d_{PMI_{PM}} \\ (\rho + \mu) R - \beta T_{PM} - d_M I_M - d_P I_P \end{bmatrix} \tag{14}$$

The Reduce matrix

$$\Psi = \begin{bmatrix} \alpha_P \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)} e^{-\mu\tau} & 0 & \alpha_p \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)} e^{-\mu\tau} \\ 0 & \alpha_M \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)} e^{-\mu\tau} & \alpha_M \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)} e^{-\mu\tau} \\ 0 & 0 & 0 \end{bmatrix}$$

$$\Upsilon = \begin{bmatrix} d_P + \delta_P + \mu & 0 & 0 \\ 0 & d_M + \delta_M + \mu & 0 \\ 0 & 0 & d_{PM} + \mu + \delta_{PM} \end{bmatrix}$$

$$\Upsilon^{-1} = \begin{bmatrix} \frac{1}{d_P + \delta_P + \mu} & 0 & 0 \\ 0 & \frac{1}{d_M + \delta_M + \mu} & 0 \\ 0 & 0 & \frac{1}{d_{PM} + \mu + \delta_{PM}} \end{bmatrix}$$

Now calculate $\Psi\Upsilon^{-1}$

$$\Psi\Upsilon^{-1} = \begin{bmatrix} \alpha_P \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)(d_P+\delta_P+\mu)} e^{-\mu\tau} & 0 & \alpha_P \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)(d_{PM}+\mu+\delta_{PM})} e^{-\mu\tau} \\ 0 & \alpha_M \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)(d_M+\mu+\delta_M)} e^{-\mu\tau} & \alpha_M \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)(\delta_{PM}+d_{PM}+\mu)} e^{-\mu\tau} \\ 0 & 0 & 0 \end{bmatrix}$$

the eigenvalues are

$$R_0^P = \alpha_P \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)(d_P+\delta_P+\mu)} e^{-\mu\tau}$$

and

$$R_0^M = \alpha_M \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)(d_M+\mu+\delta_M)} e^{-\mu\tau}$$

Hence the $R_0^{PM} = max\{R_0^P, R_0^M\}$

Now for the local stability we use linearization method. The Jacobean matrix of the system (3) is given by firstly for easy calculation we assume

$$r_1 = \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)}e^{-\mu\tau}, r_2 = d_P+\delta_P+\mu, r_3 = d_M+\delta_M+\mu, r_4 = d_{PM}+\mu+\delta_{PM}$$

Now

$$G(E_0^{PM}) = \begin{pmatrix} -\mu & \phi & -\alpha_1 r_1 & \alpha_P r_1 & -(\alpha_M+\alpha_P)r_1 & 0 & \rho \\ 0 & -(\mu+\phi) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_M r_1 - r_2 & 0 & \alpha_M r_1 & 0 & 0 \\ 0 & 0 & 0 & \alpha_M r_1 - r_3 & \alpha_P r_1 & 0 & 0 \\ 0 & 0 & 0 & 0 & -r_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & d_{PM} & -(\beta+\mu) & 0 \\ 0 & 0 & d_{PM} & d_M & 0 & \beta & -(\rho+\mu) \end{pmatrix}$$

After expanding the characteristic equation $|\lambda I_7 - G(E_0^{PM})| = 0$ with its first and seventh columns, the sixth row we obtained three eigenvalues $\lambda_1 = -\mu, \lambda_2 = -(\rho+\mu)$ and $-(\beta+\mu)$. we will then calculate the remaining four eigenvalues from reduced matrix

$$(G_4 - \lambda I_4) = \begin{pmatrix} -(\mu+\phi)-\lambda & 0 & 0 & 0 \\ 0 & \alpha_M r_1 - r_2 - \lambda & 0 & \alpha_M r_1 \\ 0 & 0 & \alpha_M r_1 - r_3 - \lambda & \alpha_P r_1 \\ 0 & 0 & 0 & -r_4 - \lambda \end{pmatrix}$$

$$\Xi_4(\lambda) = (-(\mu+\phi)-\lambda)(\alpha_M r_1 - r_2 - \lambda)(\alpha_M r_1 - r_3 - \lambda + r_4 + \lambda)$$

By solving the above equation we get $\lambda_4 = r_2(R_P - 1), \lambda_5 = r_3(R_M - 1), \lambda_6 = -r_4$ and $\lambda_7 = -(\mu+\phi)$

Hence using Routh-Hurwitz stability condition, the roots λ_4 and λ_5 are negative if and only if $R_0^P < 1$ and $R_0^M < 1$. Therefore DFE are locally asymptotically stable if and only if $R_0^{PM} = \max\{R_0^P, R_0^M\} < 1$.

Theorem 10. *The DFE is GAS if* $R_0^{PM} = \max\{R_0^P, R_0^M\} < 1$.

Proof. We used the Laypounov function method to prove **GAS** by assuming the coefficient of co-infected compartment is zero as $U_{PM} = wI_M + sI_P$ where $w = \dfrac{1}{d_M+\delta_M+\mu}$ and $s =$

$\frac{1}{d_P+\delta_P+\mu}$.

$$\frac{dU}{dt} = \frac{\alpha_P I_M S^{\cdot\cdot} e^{-\mu\tau}}{w} + \frac{\alpha_M I_P S^{\cdot\cdot} e^{-\mu\tau}}{s} - I_M - I_P$$

$$\frac{dU}{dt} = \left[\frac{\alpha_P \lambda(\phi+\mu(1-\pi))e^{-\mu\tau}}{w\mu(\phi+\mu)} - 1\right] I_M + \left[\frac{\alpha_M \lambda(\phi+\mu(1-\pi))e^{-\mu\tau}}{s\mu(\phi+\mu)} - 1\right] I_P$$

$$\frac{dU}{dt} = (R_0^M - 1)I_M + (R_0^P - 1)I_P$$

So $\frac{dU}{dt} \leq 0$ if $R_0^{PM} < 1$ and furthermore $\frac{dU}{dt} = 0$ if $I_M = 0$ and $I_P = 0$. Therefore by the principle DFE is GAS if $R_0^{PM} < 1$. □

3.4. Impact of Pneumonia on Meningitis. To investigate the modulatory effect of pneumonia on the transmission dynamics of meningitis, we derive the basic reproduction number of the meningitis-only sub-model, denoted R_0^M, as an explicit function of the pneumonia reproduction number, R_0^P. This dependency enables a deeper analytical understanding of how the presence and progression of pneumonia may influence, or potentially amplify, the spread of meningitis within a co-infected population.

The approach begins by assuming a shared constant recruitment rate, λ, into the population, which serves as a unifying parameter across both sub-models. Through this framework, we capture the interdependence of the two infections, wherein the epidemiological parameters governing pneumonia indirectly affect the transmission threshold of meningitis via co-infection dynamics. Accordingly, we define the reproduction number for pneumonia in terms of its constituent parameters and use it as a basis to reformulate R_0^M, thereby allowing us to analytically trace how shifts in pneumonia dynamics (e.g., transmission rate, recovery, or co-infection enhancement factors) contribute to the escalation or suppression of meningitis outbreaks. The explicit formulation is given as

$$R_0^P = \alpha_P \frac{\lambda\left(\phi+\mu(1-\pi)\right)}{\mu(\phi+\mu)(d_P+\delta_P+\mu)} e^{-\mu\tau}$$

$$\lambda = \frac{(\mu(\phi+\mu)(d_P+\delta_P+\mu))e^{\mu\tau}R_P}{\alpha_p(\phi+(1-\pi)\mu)}$$

Now putting the value of λ in R_0^M, we get

$$R_0^M = \frac{\alpha_P(d_P + \delta_P + \mu)R_P}{\alpha_M(d_M + \delta_M + \mu)}$$

Now by taking partial derivative with respect to R_0^P

$$\frac{\partial R_0^M}{\partial R_0^P} = \frac{\alpha_P(d_P + \delta_P + \mu)}{\alpha_M(d_M + \delta_M + \mu)}$$

Here we analyze that partial derivative of R_0^M with respect to R_0^P is positive. The results indicate that the transmission of pneumonia within the population exerts a positive influence on the propagation dynamics of meningitis.

4. NUMERICAL SIMULATIONS

In this section, we conduct numerical simulations to explore the dynamic behavior of the proposed co-infection model system (3) and its associated sub-models. The simulations are performed using the **dde23** solver in MATLAB, which is specifically designed to handle systems of DDEs. This numerical approach allows for accurate approximation of the system's temporal evolution, particularly in the presence of delay effects that influence disease progression.

All simulations are initialized using biologically plausible initial conditions and calibrated parameter values drawn from Table 4. These values are selected to reflect realistic epidemiological scenarios, thereby ensuring the model's relevance to public health dynamics. Through these simulations, we examine the transient and asymptotic behavior of key state variables and assess the impact of delay parameters and co-infection on the stability and persistence of disease within the population.

TABLE 4. State variable values with sources

S. no	Parameter	Value	Source	S. no	Parameter	Value	Source
1	d_P	0.02	[14]	2	ω, Θ	1	Assumed
3	d_M	0.015	[14]	4	ρ	0.352	[14]
5	d_{PM}	0.3102	[25]	6	λ	$0.0413 * N$	WHO 2019
7	β	0.2	[26]	8	ϕ	0.263	[29]
9	μ	0.01	[27]	10	π	0.1052	[29]
11	δ_P	0.003	[28]	12	α_P	0.009	[25]
13	δ_M	0.009	[14]	14	α_M	0.9	[30]
15	δ_{PM}	0.008	[28]	-	-	-	-

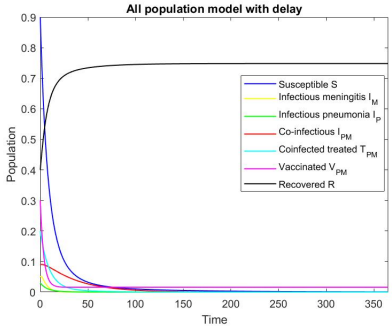

FIGURE 4. The behavior of all population model with delay at any time t.

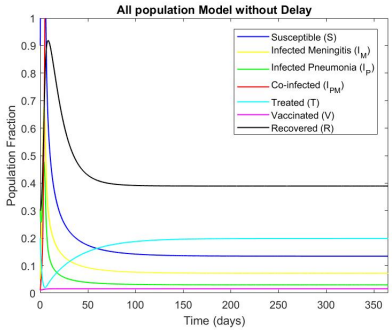

FIGURE 5. The behavior of all population model without delay at any time t.

In above Figures 4, and 5 we have plotted all population models with and without delay using the different non-negative initial conditions. $S(0) = 0.99, I_P(0) = 0.05, I_M(0) = 0.03, I_{PM}(0) = 0.1, V_{PM}(0) = 0.3, T_{PM}(0) = 0.2, R(0) = 0.4$, and parametric values table 4. The simulation demonstrates that with delay and proper intervention (treatment, recovery, and vaccination), both single and co-infections can be controlled, leading the population toward a DFE point.

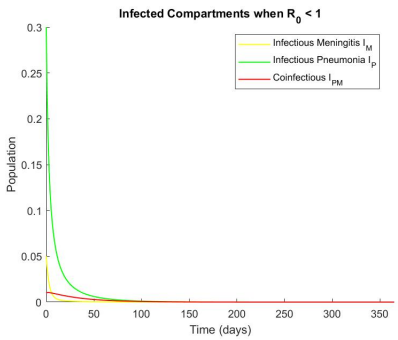

FIGURE 6. The behavior of infected compartments when $\max(R_P, R_M) < 1$.

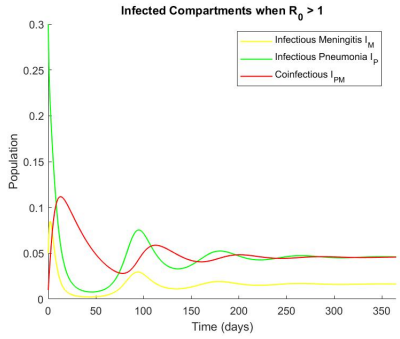

FIGURE 7. The behavior of infected compartment when $\max(R_P, R_M) > 1$.

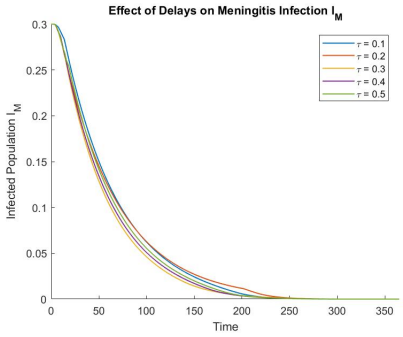

FIGURE 8. Effect of delay on meningitis infected compartment.

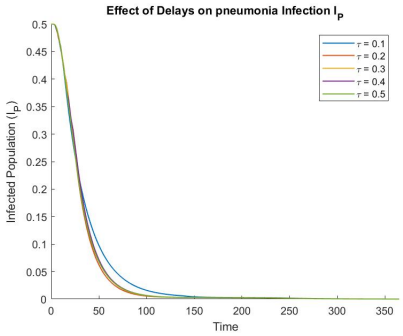

FIGURE 9. Effect of delay on Pneumonia infected compartment.

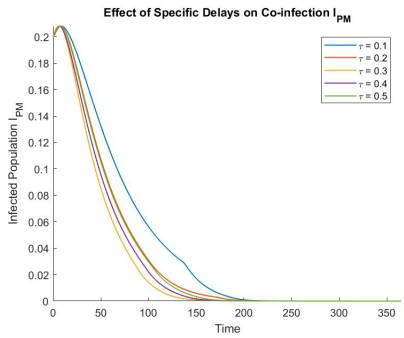

FIGURE 10. Effect of delay on Co-infected compartment.

In Figures 6 and 7 show the infected compartments for reproduction number $max(R_0^P, R_0^M) < 1$ and $max(R_0^P, R_0^M) > 1$. The Figures 8, 9, and 10 illustrate the impact of delay τ on infected compartments. These numerical outcomes robustly validate the analytical insights obtained from the sensitivity analysis of the respective sub-models. The numerical simulations demonstrate that artificial delays (traveling restriction, Social distancing) influence the dynamics of pneumonia and meningitis co-infection. All infection classes ultimately decline to negligible levels, leading the system toward a stable DFE. This highlight that delays tactics reduces disease burden, effective interventions and natural recovery ensure long-term disease eradication. The results emphasize the critical importance of delays tactics in treatment and intervention to reduce the infection peak and overall disease impact. Effective vaccination and timely healthcare responses remain vital strategies to control and eventually eliminate co-infections within the population.

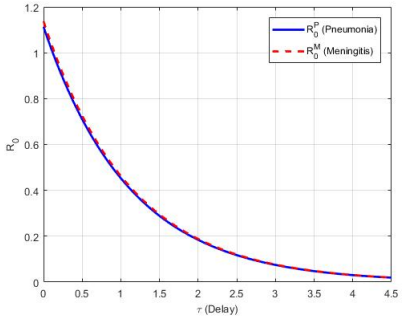

FIGURE 11. comparison of delay term and reproduction numbers.

In Figure 11, the comparative analysis of the artificial delay parameter and the basic reproduction numbers shows that at $\tau = 0.391$, the reproduction number is 0.985. Increasing the delay can induce a bifurcation from the endemic equilibrium (DEE) to the disease-free equilibrium (DFE). Specifically, by applying artificial delay tactics for approximately 142 days, the disease can be overcome. This finding underscores the critical role of artificial delay in shaping epidemic outcomes.

5. CONCLUSIONS

The concept of artificial delay provides a powerful framework for understanding and controlling the spread of infectious diseases such as pneumonia and meningitis. By intentionally introducing lags through interventions like isolation, social distancing, quarantine, diagnostic procedures, and behavioral modifications, disease transmission can be slowed sufficiently to reduce the basic reproduction number below unity. This not only suppresses epidemic persistence but can also drive the system toward a disease-free equilibrium. Our findings demonstrate that even modest artificial delays can critically alter epidemic trajectories, highlighting their potential as effective, low-cost, and adaptable public health strategies. Recognizing and leveraging artificial delays therefore offers valuable insights for designing integrated, data-driven policies that enhance preparedness and improve disease control outcomes in co-infection contexts.

Unlike fractional or stochastic modeling approaches, which capture memory effect or random fluctuation, the proposed delay-based framework offer a direct and interpretable representation of intervention-including times lags. this allows for precise threshold analysis and provides actionable insights into how delayed responses influence disease persistence or eradication.

Explicitly modeling distinct transmission coefficient for co-infection individuals could further enhance biological details; however the present formulation balance realism with analytical tractability and parameter identifiability.

Future research can extend this framework in several directions.Although the present model is restricted to bacterial forms of pneumonia and meningitis, the proposed delay-based framework is sufficiently general and may be extended in future work to viral or fungal infections by redefining transmission, recovery, and intervention parameters accordingly. A stochastic

version of the model could capture random fluctuations and uncertainties in transmission dynamics, which are particularly relevant in small populations or during early outbreak phases. A fuzzy logic–based model may account for vagueness and imprecision in epidemiological parameters, such as incubation periods or contact rates, providing a more flexible representation of real-world conditions. Additionally, fractional-order differential equation models could be employed to incorporate memory and hereditary properties of disease dynamics, offering deeper insights into long-term effects of artificial delay interventions. Together, these approaches can enrich the mathematical understanding of co-infection systems and improve the design of robust, data-driven strategies for disease mitigation.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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