

## Research Article

# Appraisal and Simulation on Codynamics of Pneumonia and Meningitis with Vaccination Intervention: From a Mathematical Model Perspective

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The membranes that encompass the brain and spinal cord become inflamed by the potentially fatal infectious disease called pneumococcal meningitis. Pneumonia and meningitis “coinfection” refers to the presence of both conditions in a single host. In this work, we accounted for the dynamics of pneumonia and meningitis coinfection in communities by erroneously using a compartment model to analyze and suggest management techniques to stakeholders. We have used the next generation matrix approach and derived the effective reproduction numbers. When the reproduction number is less than one, the constructed model yields a locally asymptotically stable disease-free equilibrium point. Additionally, we conducted a sensitivity analysis to determine how different factors affected the incidence and transmission rate, which revealed that both the pneumonia and meningitis transmission rates are extremely sensitive. The performance of our numerical simulation demonstrates that the endemic equilibrium point of the pneumonia and meningitis coinfection model is locally asymptotically stable when  $\max \{ \mathcal{R}_1, \mathcal{R}_2 \} > 1$ . Finally, as preventative and control measures for the coinfection of pneumonia and meningitis illness, the stakeholders must concentrate on reducing the transmission rates, reducing vaccination wane rates, and boosting the portion of vaccination rates for both pneumonia and meningitis.

## 1. Introduction

The word “epidemiology” is derived from the Greek term “demos,” which means “people,” and “logos,” which means “the study of”. In other words, the term “epidemiology” has its roots in the study of the experiences of a population. Despite the fact that many definitions have been provided, the one that best encapsulates the fundamental ideas and public health perspective of epidemiology is: “Epidemiology is the study of the prevalence and causes of health-related conditions or incidents in particular populations, as well as the application of this information to the prevention or treatment of health issues” [1, 2]. In epidemiology, the fre-

quency and distribution of health events in a population are studied.

By the term “frequency,” we not only mean the number of health events, such as the number of cases of meningitis or diabetes in a population, but also the correlation between that number and the size of the population [2, 3].

The underlying premise of epidemiology is that disease does not develop in a community at random but rather develops only when an individual has the proper confluence of risk factors or determinants. Individuals are the “patients” of clinicians, whereas communities are the “patients” of epidemiologists. In light of this, while dealing with a patient who is unwell, the clinician and the epidemiologist have

separate duties. When a patient with diarrheal illness first shows up, for instance, both parties are concerned with making the right diagnosis [3–5].

A potentially fatal infection called pneumococcal meningitis inflames the membranes that cover the brain and spinal cord. The meninges, which are these layers, serve to shield the brain from damage and infection [5]. Millions of people have died as a result of pneumonia, an airborne disease that is caused by breathing harmful organisms, primarily *Streptococcus pneumoniae*. Other illnesses, including meningitis, ear infections, and sinus infections, are also brought on by these bacteria [5–7]. Moreover, these illnesses can afflict people of all ages, from infants to the elderly. Pneumonia is particularly hazardous when the immune system is weakened, as in infants or the elderly, or when it is concomitant with another illness like meningitis [7, 8]. Pneumonia is a common coinfection that occurs at the time of admission in cases of bacterial meningitis and is independently linked to a poor prognosis and death [9].

The most frequent pneumococcal infection in children are caused by 13 different varieties of pneumococcal bacteria. There are vaccines named PCV13, which can protect against these types, and PPSV23, which can protect against 23 other types. For the sake of this investigation, we have studied prior work by other researchers who used mathematical modeling to explain the transmission and spread of coinfections with pneumonia and meningitis, such as [7, 10–13]. The majority of these investigations were carried out to identify community-level infectious disease control methods. To our knowledge, no one has created and examined the three kinds of vaccine independently in a mathematical model of meningitis and pneumonia coinfection in a specific community. As a result, this recently proposed study considers the dynamics of meningitis and pneumonia in communities, utilizing a deterministic compartmental model to analyze and recommend appropriate management techniques to actors. Therefore, we are driven and intrigued to investigate the three vaccine kinds for meningitis and pneumonia coinfection in this work by developing a mathematical model of meningitis and pneumonia coinfection combined with regulating techniques. We have laid up the basic framework for this investigation as follows: in Section 2, we outline and develop the compartmental mathematical model of coinfection with meningitis and pneumonia. The model analysis includes the equilibrium points, fundamental reproduction numbers, and stability analysis of the submodels and the main model also presented in Section 2. Numerical simulations and sensitivity analysis are presented in Section 3. The study's discussion and conclusion were then finalized.

## 2. Mathematical Model Formulations and Its Qualitative Analysis

In this section we have proposed a mathematical model which depend upon the assumption and present the qualitative properties of the constructed model.

**2.1. Baseline Model Formulation and Assumptions.** We have developed the new model by expanding the model which was developed previously by another researcher based on the following hypotheses. Under this study, we consider homogenous population and that the factors such as sex, social status, and race do not affect the probability of being infected. The model subdivides the human total population  $N(t)$  into nine mutually-exclusive compartments, namely, susceptible population  $S(t)$ , pneumonia-only infectious  $P_I(t)$ , meningitis-only infectious  $M_I(t)$ , meningitis and pneumonia coinfectious  $MP_I(t)$ , treated class  $T(t)$ , meningitis vaccinated  $M_V(t)$ , pneumonia vaccinated  $P_V(t)$ , class of people who take both vaccines of pneumonia and meningitis (PCV13 Pneumococcal conjugate vaccine) group  $MP_V(t)$ , and recovered class (R). The recovery from natural immunity and the effects of vertical transmission to pneumonia and meningitis were assumed to be insignificant in this study. Epidemiologically, individuals in the removed/recovered compartment  $R(t)$  do not attain permanent immunity so that we are assigned such case by the parameter  $Y$ .

In this study the mass action-incidence rate of new infections are used, and the modification parameters  $\omega_1$  and  $\omega_2$  are the factors that describe the fact of how infectiousness level of pneumonia increases the susceptibility level to meningitis disease and vice versa, respectively. The meningitis disease is assumed to be transmitted after effective contact between the susceptible and meningitis infectious classes with effective contact rate  $a$ , where  $a$  is a composite parameter that measures the meningitis contact rate  $\kappa$  and the probability of transmission upon contact  $q$ . Additionally, pneumonia disease is assumed to be transmitted after effective contact between the susceptible and pneumonia infectious classes with effective contact rate  $b$ , where  $b$  is a composite parameter which measure the pneumonia infectious contact rate  $c$  and the probability of transmission upon contact  $p$ . Individuals can get meningitis by contact rate  $a$  from a meningitis-only infected or coinfecting person with force of infection of meningitis  $\alpha_1 = a(M_I + e_1 MP_I)$  and join  $M_I$  compartment where  $e_1$  is the modification parameter.

An individual can get pneumonia with contact rate of  $b$  from a pneumonia-only infected or coinfecting person with force of infection of pneumonia  $\alpha_2 = b(P_I + e_2 MP_I)$  and join the  $P_I$  compartment with modification parameter  $e_2$ . Pneumonia-only infected individuals also can get an additional meningitis infection with force of infection and modification parameter  $\omega_1 \alpha_1$  and join coinfecting compartment  $MP_I$ . The coinfecting compartment increases because of individuals that come from meningitis-only infected compartment are infected by pneumonia with force of infection and modification parameter  $\omega_2 \alpha_2$ . Since the coinfecting individuals are aware of the disease, they remain there in the treated compartment even if they are free from either pneumonia or meningitis until they are free from all the diseases. The parameters used in the model are described in Table 1.

Using the above assumptions and parameters, we have constructed the following schematic diagram that is given in Figure 1.

From the diagram given in Figure 1, the corresponding dynamical systems will be as follows from the assumptions of the model and using the above basic model assumption we have the following flow chart.

$$\left. \begin{aligned} \frac{dS}{dt} &= (1 - \pi)\lambda + \phi P_V + \varphi M_V + YR - (\alpha_1 + \alpha_2 + \mu)S, \\ \frac{dM_V}{dt} &= (1 - \rho)\pi\lambda - (\mu + \varphi + \varepsilon_1)M_V, \\ \frac{dP_V}{dt} &= \rho\pi\lambda - (\mu + \phi + \varepsilon_2)P_V, \\ \frac{dMP_V}{dt} &= \varepsilon_1 M_V + \varepsilon_2 P_V - \mu MP_V, \\ \frac{dM_I}{dt} &= \alpha_1 S - (\omega_2 \alpha_2 + \tau_1 + \delta_1 + \mu)M_I, \\ \frac{dP_I}{dt} &= \alpha_2 S - (\omega_1 \alpha_1 + \tau_2 + \delta_2 + \mu)P_I, \\ \frac{dMP_I}{dt} &= \omega_1 \alpha_1 P_I + \omega_2 \alpha_2 M_I - (\tau_3 + \delta_3 + \mu)MP_I, \\ \frac{dT}{dt} &= \tau_1 M_I + \tau_2 P_I + \tau_3 MP_I - (\beta + \mu)T, \\ \frac{dR}{dt} &= \beta T - (Y + \mu)R. \end{aligned} \right\} \quad (1)$$

This system of differential equation is the mathematical representation of full meningitis and pneumonia model which is the combination of the two diseases. In the next section, we have studied the qualitative behavior of the constructed model. For simplification of our work, we split the full meningitis-pneumonia coinfection model into submodels, which are meningitis-only and pneumonia-only models. First, we will study the qualitative behavior of the submodel and then qualitative behavior of the full model is followed.

**2.2. Positivity of Solutions and Invariant Region of the Only Pneumonia-Infected Model.** In this subsection, we have considered the model of pneumonia only in the absence of meningitis disease. This procedure will help us to summarize and conclude some properties of the full coinfecting model depending on the properties of sub models. To gate this submodel from the full model, we set,  $M_I = MP_I = 0$ , and we have the following dynamical system.

The corresponding dynamical systems are as follow.

$$\left. \begin{aligned} \frac{dS}{dt} &= (1 - \pi)\lambda + \phi P_V + YR - (\alpha_2 + \mu)S, \\ \frac{dP_I}{dt} &= \alpha_2 S - (\tau_2 + \delta_2 + \mu)P_I, \\ \frac{dP_V}{dt} &= \pi\lambda - (\mu + \phi)P_V, \\ \frac{dT}{dt} &= \tau_2 P_I - (\beta + \mu)T, \\ \frac{dR}{dt} &= \beta T - (Y + \mu)R. \end{aligned} \right\} \quad (2)$$

TABLE 1: Descriptions of parameters of the model.

Parameter	Description
$a$	Meningitis effective contact rate
$b$	Pneumonia effective contact rate
$\tau_1$	The rate at which meningitis-infected individuals are treated enter to and treated class
$\tau_2$	The rate at which pneumonia-infected individuals are treated and enter to treated class
$\tau_3$	The rate at which meningitis and pneumonia coinfecting individuals are treated and enter to treated class
$\beta$	The rate at which treated class recovered
$\mu$	Natural death rate
$\delta_1$	Meningitis-only caused death rate
$\delta_2$	Pneumonia-only caused death rate
$\delta_3$	Meningitis and pneumonia coinfection caused death rate.
$\omega_1$	Modification parameter and $\omega_1 \geq 1$
$\omega_2$	The modification parameter and $\omega_2 \geq 1$
$Y$	Rate of loss of immunity
$\pi$	The portion of vaccinated new born
$\lambda$	Recruitment rate
$\varepsilon_2$	The rate at which a pneumonia vaccinated individual takes meningitis vaccination
$\varepsilon_1$	The rate at which a meningitis vaccinated individual takes pneumonia vaccination
$\phi$	Pneumonia vaccine wanes rate
$\varphi$	Meningitis vaccine wanes rate
$\rho$	The portion in which pneumonia vaccine is given

For the dynamical systems to be epidemiologically meaningful as well as well-posed, we need to prove that all the state variables of dynamical systems are nonnegative.

**Theorem 1.** *All the populations of the system with positive initial conditions are positive.*

*Proof:* assume  $S(0) > 0, P_I(0) > 0, P_V(0) > 0, T(0) > 0$  and  $R(0) > 0$  are positive for time  $t > 0$  and for all nonnegative parameters.

First, let us take  $T = \sup \{t > 0 \text{ such that } S(t') > 0, P_I(t') > 0, P_V(t') > 0, T(t') > 0 \text{ and } R(t') > 0, t' \in [0, t]\}$ .

From the first equation of system (2), we do have  $dS/dt = (1 - \pi)\lambda + \phi P_V + YR - (\alpha_2 + \mu)S \Rightarrow S(t) = S(0)e^{-\int_0^t (\alpha_2 + \mu)dt'} + e^{-\int_0^t (\alpha_2 + \mu)dt'} [\int_0^t e^{\int_0^{t'} (\alpha_2 + \mu)dt''} [(1 - \pi)\lambda + YR + \phi P_V] dt' > 0 \Rightarrow S(t) > 0$ .

There,  $S(t)$  is positive. Following the same procedure, all the remaining state variables are nonnegative. Therefore, from proof, we can conclude that whenever the initial values of the systems are all nonnegative, then all the solutions of our dynamical system are positive.

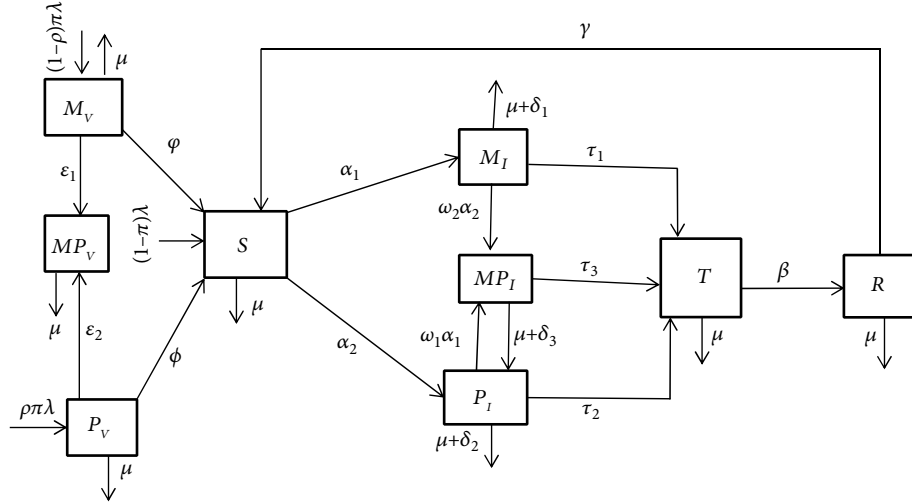


FIGURE 1: Schematic diagram of full pneumonia and meningitis codynamics.

**Theorem 2.** *The total human population of the dynamical system (2) is positively closed in the closed invariant set  $\Omega_1 = \{(S, P_I, P_V, T, R) \in \mathbb{R}_+^4 : N_1 \leq (\lambda/\mu)\}$ .*

*Furthermore, the system's nonnegative solutions are all constrained, and it may exhibit the persistence property under any nonnegative initial concentration conditions [14].*

*Proof:* assume the total population of the model is  $N_1$ . To get an invariant region, which shows boundedness of solution, it can be obtained as follows.

$$\begin{aligned} N_1 = S + P_I + P_V + T + R &\Rightarrow \frac{dN_1}{dt} \\ &= \lambda - \mu S - \delta_2 P_I - \mu P_I - \mu P_V - \mu T - \mu R, \end{aligned} \quad (3)$$

$$\frac{dN_1}{dt} = \lambda - \mu N_1 - \delta_2 P_I \Rightarrow \frac{dN_1}{dt} + \mu N_1 \leq \lambda, \quad (4)$$

$$N_1(t) \leq N_0 e^{-\mu t} + \frac{\lambda}{\mu} \Rightarrow \lim_{t \rightarrow \infty} N_1(t) \leq \frac{\lambda}{\mu}, \quad (5)$$

$$\Rightarrow 0 \leq N_1 \leq \lambda/\mu.$$

Therefore, the dynamical system that we do have is bounded.

**2.3. Existence and Stability of Disease-Free Equilibrium Point.** The disease-free equilibrium point is obtained by making all the equations equal to zero, provided that  $P_I = 0$  and the obtained disease-free equilibrium point is given by

$$\begin{aligned} E_p^0 &= (S^0, P_V^0, P_I^0, T^0, R^0) \\ &= \left( \frac{\lambda}{\mu} \left[ \frac{(1-\pi)(\mu+\phi) + \pi\phi}{(\mu+\phi)} \right], \frac{\pi\lambda}{(\mu+\phi)}, 0, 0, 0 \right). \end{aligned} \quad (6)$$

**2.3.1. Effective Reproduction Number.** The reproduction number is the number of secondary cases produced by one infectious individual joining in a completely susceptible population during its infectious period [15–17].

Using the next generation matrix method, we have obtained the effective reproduction number of pneumonia-infected-only submodel, which is  $\mathcal{R}_{ef(p)} = (\lambda b / \mu) ((1-\pi)(\mu+\phi) + \pi\phi / (\mu+\phi) (\tau_2 + \delta_2 + \mu))$ .

**Theorem 3.** *The disease-free equilibrium point  $E_p^0$  of the model in system (2) is locally asymptotically stable if the effective reproduction number  $\mathcal{R}_{ef(p)} < 1$  and is unstable if  $\mathcal{R}_{ef(p)} > 1$ .*

*Proof:* from the Jacobean matrix  $J(E_p^0)$  of the model (2), with respect to  $(S, P_V, P_I, T, R)$  at the disease-free equilibrium point, we have the following characteristics equation.

$$\begin{aligned} &(-\mu - \lambda_1)((r_1 - r_2) - \lambda_2) (-(\mu + \phi) - \lambda_3) \\ &\cdot (-(\beta + \mu) - \lambda_4) (-(\mu + \phi) - \lambda_5) = 0 \end{aligned} \quad (7)$$

$$\text{Where } r_1 = \mathcal{R}_{ef(p)}, r_2 \text{ and } r_2 = (\tau_2 + \delta_2 + \mu),$$

$$\Rightarrow \lambda_1 = -\mu, \lambda_3 = -(\mu + \phi), \lambda_4 = -(\beta + \mu), \lambda_5 = -(\mu + \phi). \quad (8)$$

Hence, all the parameters are nonnegative, and all the eigenvalues of the corresponding Jacobean matrix are negative. But  $\lambda_2 = (r_1 - r_2) = \mathcal{R}_{ef(p)} r_2 - r_2 = r_2 (\mathcal{R}_{ef(p)} - 1)$ ,

$$\Rightarrow \lambda_2 = r_2 (\mathcal{R}_{ef(p)} - 1), \quad (9)$$

$\Rightarrow \lambda_2 < 0$  if  $\mathcal{R}_{ef(p)} < 1$ . Therefore, the disease-free equilibrium point is locally asymptotically stable if and only if  $\mathcal{R}_{ef(p)} < 1$ , otherwise it is unstable, that is, if  $\mathcal{R}_{ef(p)} > 1$ .

**2.3.2. Global Stability of Disease-Free Equilibrium Point of the Model.** To verify the global stability of the disease-free equilibrium point of the pneumonia monoinfection model, we have used an adopted method of Castillo-Chavez et al. used by others scholar such as [18, 19].

**Lemma 4.** *If the pneumonia monoinfection model can be written as*

$$\begin{aligned}\frac{dY}{dt} &= G(Y, W), \\ \frac{dZ}{dt} &= H(Y, W), H(Y, 0) = 0,\end{aligned}\quad (10)$$

where  $Y \in \mathbb{R}^m$  be the components of noninfected individuals and  $W \in \mathbb{R}^n$  be the components of infected individuals including treated class and  $E_p^0 = (Y^*, 0)$  denotes the disease-free equilibrium point of dynamical system (2).

Assume

- (i) For  $(dY/dt) = G(Y, 0)$ ,  $Y^*$  is globally asymptotically stable (GAS)
- (ii)  $H(Y, W) = BW - H^*(Y, W)$ ,  $H^*(Y, W) \geq 0$  for  $(Y, W) \in \Omega_1$  where  $B = D_W H(Y^*, 0)$  is an M-matrix, i.e., the off diagonal elements of  $B$  are nonnegative and  $\Omega_1$  is the region in which the system makes biological sense. Then the fixed point  $E_p^0 = (Y^*, 0)$  is globally asymptotically stable equilibrium point of the system (2) whenever  $R_{ef(p)} < 1$ .

**Lemma 5.** *The disease-free equilibrium point  $E_p^0$  of the pneumonia monoinfection model (2) is globally asymptotically stable if  $R_{ef(p)} < 1$  and the two sufficient conditions given in Lemma 4 are satisfied.*

*Proof:* here we are applying Lemma 5 on the pneumonia monoinfection model (2) and we have gotten the following matrices.

$$\begin{aligned}\frac{dY}{dt} &= G(Y, W) = \begin{bmatrix} (1-\pi)\lambda + \phi P_V + YR - (\alpha_2 + \mu)S \\ \pi\lambda - (\mu + \phi)P_V \end{bmatrix}, \\ \frac{dW}{dt} &= H(Y, W) = \begin{bmatrix} \alpha_2 S - (\tau_2 + \delta_2 + \mu)P_I \\ \tau_2 P_I - (\beta + \mu)T \\ \beta T - (Y + \mu)R \end{bmatrix}, \\ G(Y, 0) &= \begin{bmatrix} (1-\pi)\lambda + \phi P_V - \mu S \\ \pi\lambda - (\mu + \phi)P_V \end{bmatrix},\end{aligned}\quad (11)$$

and

$$\begin{aligned}H^*(Y, W) &= \begin{bmatrix} H^*_1(Y, W) \\ H^*_2(Y, W) \\ H^*_3(Y, W) \end{bmatrix} = \begin{bmatrix} \left(\frac{b\lambda}{\mu} \left(\frac{(1-\pi)(\mu + \phi) + \pi\phi}{\mu + \phi}\right)\right) P_I - \alpha_2 S \\ 0 \\ 0 \end{bmatrix}, \\ H^*(Y, W) &= \begin{bmatrix} H^*_1(Y, W) \\ H^*_2(Y, W) \\ H^*_3(Y, W) \end{bmatrix} = \begin{bmatrix} (S_0 - S)bP_I \\ 0 \\ 0 \end{bmatrix}.\end{aligned}\quad (12)$$

Since  $S \leq S_0$ , we have  $H^*_1(Y, W) \geq 0$ , thus, the disease-free equilibrium point  $E_p^0$  is globally asymptotically stable if  $R_{ef(p)} < 1$ . Biologically, whenever  $R_{ef(p)} < 1$ , the only pneumonia infection disease dies out while the total population increases [18].

#### 2.4. The Existence and Stability of Endemic Equilibrium Point.

The endemic equilibrium point of the dynamical system of (2) is obtained by making the right side of the system equal to zero, providing that  $P_I \neq 0$ . We have supposed that the endemic equilibrium point of the model is denoted by  $E_p^* = (S^*, P_I^*, P_V^*, T^*, R^*)$  and the corresponding force of infection is  $\alpha_2(t) = b(P_I^*(t))$ .

For simplification of algebraic manipulation, we have assumed the parameters in the model by another variable as follows,  $k_1 = (1 - \pi)\lambda$ ,  $k_2 = \tau_2 + \mu$ ,  $k_3 = \tau_2 + \delta_2 + \mu$ ,  $k_4 = \pi\lambda$ ,  $k_5 = \phi + \mu$ ,

$k_6 = \beta + \mu$ ,  $k_7 = Y + \mu$ ,  $k_8 = \gamma\beta\tau_2$ ,  $k_9 = k_3 k_6 k_7$ ,  $k_{10} = k_1 + (\phi k_4 / k_5)$ ,  $k_{11} = k_9 k_{10}$ ,  $k_{12} = k_2 k_9$

$k_{13} = k_3 k_{12}$  and  $k_{14} = k_8 k_3$ . Now the equation of force of infection can be rearranged as

$$\alpha_2^* (k_{14} \alpha_2^* + b k_{11} - k_{13}) = 0 \Rightarrow \alpha_2^* = 0 \text{ or } k_{14} \alpha_2^* + b k_{11} - k_{13} = 0, \quad (13)$$

$\Rightarrow \alpha_2^* = k_{13} - b k_{11} / k_{14}$  but  $\alpha_2^* = k_{13} - b k_{11} / k_{14} = (\beta + \mu)(Y + \mu)((\tau_2 + \delta_2 + \mu)(\tau_2 + \mu)(\phi + \mu) - b\lambda((\phi + \mu)(1 - \pi) - \phi\pi) / \gamma\beta\tau_2(\phi + \mu)) = (\beta + \mu)(Y + \mu)((\tau_2 + \delta_2 + \mu)(\tau_2 + \mu)(\phi + \mu) / \gamma\beta\tau_2(\phi + \mu) - \lambda b((\phi + \mu)(1 - \pi) + \phi\pi) / \gamma\beta\tau_2(\phi + \mu))$ ,  $\alpha_2^* = (\beta + \mu)(Y + \mu)((\tau_2 + \delta_2 + \mu)(\tau_2 + \mu)(\phi + \mu) / \gamma\beta\tau_2)((\lambda b / \mu)((1 - \pi)(\mu + \phi) + \pi\phi / (\mu + \phi)(\tau_2 + \delta_2 + \mu)) - 1) \Rightarrow \alpha_2^* = (\beta + \mu)(Y + \mu)((\tau_2 + \delta_2 + \mu)(\tau_2 + \mu)(\phi + \mu) / \gamma\beta\tau_2)(R_p - 1) \Rightarrow \alpha_2^* > 0$  if  $R_{ef(p)} > 1$ .

Therefore, there is a unique endemic equilibrium point for pneumonia monoinfected model as given by  $E_p^* = (S^*, P_I^*, P_V^*, T^*, R^*)$  where

$$\begin{aligned}S^* &= \frac{((\tau_2 + \delta_2 + \mu)(\beta + \mu)(Y + \mu)(\phi + \mu) - (\phi + \mu)(1 - \pi)\lambda - \phi\pi\lambda)}{(\phi + \mu)((\tau_2 + \mu)((\tau_2 + \delta_2 + \mu)(\beta + \mu)(Y + \mu)) - \gamma\beta\tau_2\alpha_2^*)}, \\ P_I^* &= \frac{((\tau_2 + \delta_2 + \mu)(\beta + \mu)(Y + \mu)(\phi + \mu) - (\phi + \mu)(1 - \pi)\lambda - \phi\pi\lambda)}{(\phi + \mu)((\tau_2 + \mu)((\tau_2 + \delta_2 + \mu)(\beta + \mu)(Y + \mu)) - \gamma\beta\tau_2\alpha_2^*)} \left(\frac{\alpha_2^*}{\tau_2 + \delta_2 + \mu}\right), P_V^* = \frac{\pi\lambda}{\phi + \mu}, \\ T^* &= \frac{\alpha_2^* \tau_2}{(\tau_2 + \delta_2 + \mu)(\beta + \mu)} \frac{((\tau_2 + \delta_2 + \mu)(\beta + \mu)(Y + \mu)(\phi + \mu) - (\phi + \mu)(1 - \pi)\lambda - \phi\pi\lambda)}{(\phi + \mu)((\tau_2 + \mu)((\tau_2 + \delta_2 + \mu)(\beta + \mu)(Y + \mu)) - \gamma\beta\tau_2\alpha_2^*)}, \\ R^* &= \frac{((\tau_2 + \delta_2 + \mu)(\beta + \mu)(Y + \mu)(\phi + \mu) - (\phi + \mu)(1 - \pi)\lambda - \phi\pi\lambda)}{(\phi + \mu)((\tau_2 + \mu)((\tau_2 + \delta_2 + \mu)(\beta + \mu)(Y + \mu)) - \gamma\beta\tau_2\alpha_2^*)} \left(\frac{\beta\tau_2\alpha_2^*}{(\tau_2 + \delta_2 + \mu)(\beta + \mu)(Y + \mu)}\right).\end{aligned}\quad (14)$$

**Theorem 6.** *The endemic equilibrium point of system (2)  $E_p^* = (S^*, P_I^*, P_V^*, T^*, R^*)$  is locally asymptotically stable for the reproduction number  $R_{eff(p)} > 1$ .*

*Proof:* to show that the local stability of the endemic equilibrium point, we have used the method of the Jacobian matrix and the Routh Hurwitz stability criteria.

Then the corresponding characteristic equation is obtained from the determinant of

$$\begin{vmatrix} A - \lambda & B & C & 0 & D \\ E & F - \lambda & 0 & 0 & 0 \\ 0 & 0 & G - \lambda & 0 & 0 \\ 0 & H & 0 & I - \lambda & 0 \\ 0 & 0 & 0 & J & K - \lambda \end{vmatrix} = 0, \quad (15)$$

where  $A = -(\alpha_2 I_p^* + \mu)$ ,  $B = -\alpha_2 S^*$ ,  $C = \phi$ ,  $D = Y$ ,  $E = \alpha_2 I_p^*$ ,  $F = \alpha_2 S^* - (\tau_2 + \delta_2 + \mu)$ ,

$G = -(\mu + \phi)$ ,  $H = \tau_2$ ,  $I = -(\beta + \mu)$ ,  $J = \beta$  and  $K = -(Y + \mu)$ ,

$$\Rightarrow a_0 \lambda^5 + a_1 \lambda^4 + a_2 \lambda^3 + a_3 \lambda^2 + a_4 \lambda + a_5 = 0, \quad (16)$$

where  $a_0 = 1$ ,  $-(I + A + F + G + K) = a_1$ ,  $(IA + IF + AF + IG + AG + FG + IK + AK + FK + GK) = a_2$ ,  $-(-B + IAF + IAG + IFG + AFG + IAK + IFK + AFK + IGK + AGK + FGK) = a_3$ ,  $-(IB - IAFG + DEHJ + BK - IAFK - IAGK - IFGK - AFGK) = a_4$ ,  $a_5 = DEGHJ + IBK - IAFGK$ .

To apply the Routh-Hurwitz stability criteria, it is obligatory to check if the necessary condition of all the coefficients have the same sign or not. Since  $a_0 = 1$  is positive in sign, all  $a_1, a_2, a_3, a_4$ , and  $a_5$  should be positives in sign. All the coefficients of the characteristic's polynomial are positives whenever  $\mathcal{R}_{eff(p)} > 1$ . We have observed that the first column of the Routh Hurwitz array has no sign change, thus the root of the characteristics equation of the dynamical system are negative. Hence, the endemic equilibrium point of the dynamical system is locally asymptotically stable.

**2.5. Positivity of Solutions and Invariant Region of the Only Meningitis-Infected Model.** We have made  $P_I = MP_I = 0$  from the full pneumonia and meningitis coinfection model to obtain this submeningitis-only model, and got the following dynamical system.

$$\left. \begin{aligned} \frac{dS}{dt} &= (1 - \pi)\lambda + \phi M_V + YR - (\alpha_1 + \mu)S, \\ \frac{dM_I}{dt} &= \alpha_1 S - (\tau_1 + \delta_1 + \mu)M_I, \\ \frac{dM_V}{dt} &= \pi\lambda - (\mu + \phi)M_V, \\ \frac{dT}{dt} &= \tau_1 M_I - (\beta + \mu)T, \\ \frac{dR}{dt} &= \beta T - (Y + \mu)R. \end{aligned} \right\} \quad (17)$$

The above dynamical systems are needed to be epidemiologically meaningful as well as well-posed. To prove that, we have intimated that all the state variables of dynamical systems are nonnegative.

**Theorem 7.** All the populations of the system with positive initial conditions are positive.

*Proof:* assume  $S(0) > 0, M_I(0) > 0, M_V(0) > 0, T(0) > 0$  and  $R(0) > 0$  are positive for time  $t > 0$  and for all nonnegative. First let us take  $T = \sup \{t > 0 \text{ such that } S(t') > 0, M_I(t') > 0, M_V(t') > 0, T(t') > 0 \text{ and } R(t') > 0, t' \in [0, t]\}$ .

From the first equation of system (17), we do have

$$\begin{aligned} \frac{dS}{dt} &= (1 - \pi)\lambda + \phi M_V + YR - (\alpha_1 + \mu)S, \\ S(t) &= S(0)e^{-\int_0^t (\alpha_1 + \mu)dt'} + e^{-\int_0^t (\alpha_1 + \mu)dt'} \\ &\quad \cdot \left[ \int_0^t e^{\int_0^t (\alpha_1 + \mu)dt'} [(1 - \pi)\lambda + YR + \phi M_V dt] > 0, \right. \\ &\quad \left. \Rightarrow S(t) > 0. \right. \end{aligned} \quad (18)$$

Therefore,  $S(t)$  is positive. Subsequent to the same procedure, the remaining state variables are nonnegative. Therefore, from the stated proof, we can conclude that whenever the initial values of the systems are all nonnegative, then all the solutions of our dynamical system are positive.

**Theorem 8.** All the populations of the system with positive initial conditions are nonnegative

The total human population of the dynamical system (17) is positively closed in the closed invariant set  $\Omega_2 = \{(S, M_I, M_V, T, R) \in \mathbb{R}_+^5 : N_2 \leq \lambda/\mu\}$ . Furthermore, the system's nonnegative solutions are all constrained, and it may exhibit the persistence property under any nonnegative initial concentration conditions [14].

*Proof:* to get an invariant region, boundedness of solution is obtained as follow.

$$\begin{aligned} N_2 &= S + M_I + M_V + T + R, \\ \frac{dN_2}{dt} &= \lambda - \mu N_2 - \delta_1 M_I \Rightarrow 0 \leq N_2 \leq \frac{\lambda}{\mu}. \end{aligned} \quad (19)$$

Therefore, the dynamical system that we do have is bounded.

**2.6. Existence and Stability of Disease-Free Equilibrium Point.** The disease-free equilibrium point is obtained by making all the equations in the system equal to zero, provided that providing that  $M_I = 0$ . Therefore, the disease-free equilibrium point is

$$\begin{aligned} E_p^0 &= (S^0, M_V^0, M_I^0, T^0, R^0) \\ &= \left( \frac{\lambda}{\mu} \left[ \frac{(1 - \pi)(\mu + \phi) + \pi\phi}{(\mu + \phi)} \right], \frac{\pi\lambda}{(\mu + \phi)}, 0, 0, 0 \right). \end{aligned} \quad (20)$$

**2.6.1. Effective Reproduction Number.** The reproduction number can be defined as a number of secondary cases produced by one infectious individual joining in a completely susceptible population during its infectious period [16, 17, 20].

To compute the reproduction number, first distinguishing the new infected from all other changes in the host population is mandatory as follows.

Let  $\mathcal{F}_i(x)$ : be the rate of appearance of new infected in compartment  $i$ ,

$\mathcal{V}_i^+(x)$ : be the rate of transfer of individuals in to compartment  $i$ ,

$\mathcal{V}_i^-(x)$ : be the rate of transfer of individuals out of compartment  $i$ .

And then  $\mathcal{V}_i(x) = \mathcal{V}_i^-(x) - \mathcal{V}_i^+(x)$  but  $F = [(\partial \mathcal{F}_i / \partial X_j)(X_0)]$  and  $V = [(\partial \mathcal{V}_i / \partial X_j)(X_0)]$ , where  $F$  and  $V$  are  $m \times m$  matrix with  $m$  is number of infected compartment.  $FV^{-1}$  is the next generation matrix, and the spectral radius of next generation matrix is needed for the reproduction number we are seeking for.

Thus  $\partial \mathcal{F}_i(X) / \partial X_j = aS$  and  $\partial \mathcal{V}_i(X) / \partial X_j$  and  $FV^{-1} = (\lambda a / \mu) ((1 - \pi)(\mu + \varphi) + \pi \varphi) / ((\mu + \varphi)(\tau_1 + \delta_1 + \mu))$

Therefore, the effective reproduction number of meningitis monoinfected submodel is

$$\mathcal{R}_{ef(m)} = \frac{\lambda a}{\mu} \left( \frac{(1 - \pi)(\mu + \varphi) + \pi \varphi}{(\mu + \varphi)(\tau_1 + \delta_1 + \mu)} \right). \quad (21)$$

**Theorem 9.** The disease-free equilibrium point  $E_m^0$  of the model in system (17) is locally asymptotically stable if the effective reproduction number  $\mathcal{R}_{ef(m)} < 1$ , and it is unstable if  $\mathcal{R}_{ef(m)} > 1$ .

*Proof:*

Using the Jacobean matrix  $J(E_p^0)$  of the model (17) with respect to  $(S, M_V, M_I, T, R)$  at the disease-free equilibrium point, we have the characteristic equation

$$\begin{aligned} &\Rightarrow (-\mu - \lambda_1)((t_1 - t_2) - \lambda_2)(-\mu + \varphi - \lambda_3)(-\beta + \mu - \lambda_4) \\ &\quad \cdot (-(Y + \mu) - \lambda_5) = 0 \text{ where } t_1 = \mathcal{R}_{ef(m)}, t_2 = (\tau_1 + \delta_1 + \mu), \\ &\Rightarrow \lambda_1 = -\mu, \lambda_3 = -(\mu + \varphi), \lambda_4 = -(\beta + \mu), \lambda_5 = -(Y + \mu). \end{aligned} \quad (22)$$

Hence, all the parameters are nonnegative, all the eigenvalues of the corresponding Jacobean matrix are negative other than  $\lambda_2$ .

$$\text{For } \lambda_2, \lambda_2 = t_2(\mathcal{R}_{ef(m)} - 1) \Rightarrow \lambda_2 = t_2(\mathcal{R}_{ef(m)} - 1),$$

$$\Rightarrow \lambda_2 < 0 \text{ iff } \mathcal{R}_{ef(m)} < 1. \quad (23)$$

Therefore, the disease-free equilibrium point of the meningitis monoinfected model is locally asymptotically stable if the effective reproduction number  $\mathcal{R}_{ef(m)} < 1$  and is unstable if  $\mathcal{R}_{ef(m)} > 1$ .

**2.6.2. Global Stability of Disease-Free Equilibrium Point.** We utilized the approach developed by Castillo-Chavez et al. and used it to confirm the overall stability of the disease-free equilibrium point of the meningitis monoinfection model [18, 19].

**Lemma 10.** If the pneumonia monoinfection model can be written as

$$\begin{aligned} \frac{dY}{dt} &= G(Y, W), \\ \frac{dZ}{dt} &= H(Y, W), H(Y, 0) = 0, \end{aligned} \quad (24)$$

where  $Y \in \mathbb{R}^m$  be the components of noninfected individuals and  $W \in \mathbb{R}^n$  be the components of infected individuals including the treated class, and  $E_m^0 = (Y^*, 0)$  denotes the disease-free equilibrium point of the dynamical system (3).

Assume

- (i) For  $(dY/dt) = G(Y, 0)$ ,  $Y^*$  is globally asymptotically stable (GAS)
- (ii)  $H(Y, W) = BW - \check{H}(Y, W)$ ,  $\check{H}(Y, W) \geq 0$  for  $(Y, W) \in \Omega_1$  where  $B = D_W H(Y^*, 0)$  is an  $M$ -matrix, i.e., the off diagonal elements of  $B$  are nonnegative and  $\Omega_2$  is the region in which the system makes biological sense

Then the fixed point  $E_m^0 = (Y^*, 0)$  is globally asymptotically stable equilibrium point of the system (17) whenever  $\mathcal{R}_{ef(m)} < 1$ .

**Lemma 11.** The disease-free equilibrium point  $E_m^0$  of the pneumonia monoinfection model (17) is globally asymptotically stable if  $\mathcal{R}_{ef(m)} < 1$  and the two sufficient conditions given in Lemma 10 are satisfied.

*Proof:* here we are applying Lemma 11 on the meningitis monoinfection model (17) and we have gotten the following

$$\text{matrices } dY/dt = G(Y, W) = \begin{bmatrix} (1 - \pi)\lambda + \varphi M_V + YR - (\alpha_1 + \mu)S \\ \pi\lambda - (\mu + \varphi)M_V \end{bmatrix},$$

$$\begin{aligned} \frac{dW}{dt} &= H(Y, W) \\ &= \begin{bmatrix} \frac{a\lambda}{\mu} \left( \frac{(1 - \pi)(\mu + \varphi) + \pi\varphi}{(\mu + \varphi)} \right) - (\tau_1 + \delta_1 + \mu) & 0 & 0 \\ \tau_1 & -(\beta + \mu) & 0 \\ 0 & \beta & -(Y + \mu) \end{bmatrix}, \end{aligned}$$

$$G(Y, 0) = \begin{bmatrix} (1 - \pi)\lambda + \varphi M_V - \mu S \\ \pi\lambda - (\mu + \varphi)M_V \end{bmatrix}. \quad (25)$$

Here after some steps of calculations, we have determined that

$$\check{H}(Y, W) = \begin{bmatrix} \check{H}_1(Y, W) \\ \check{H}_2(Y, W) \\ \check{H}_3(Y, W) \end{bmatrix} = \begin{bmatrix} (S_0 - S)aM_I \\ 0 \\ 0 \end{bmatrix}. \quad (26)$$

Since  $S \leq S_0$ , we have  $\check{H}_1(Y, W) \geq 0$ , thus, the disease-free equilibrium point  $E_m^0$  of model (17) is globally asymptotically stable if  $\mathcal{R}_{ef(m)} < 1$ . Biologically, whenever  $\mathcal{R}_{ef(m)} < 1$ , the

meningitis monoinfection disease dies out while the total population increases [18].

### 2.7. Existence and Stability of Endemic Equilibrium Point.

The endemic equilibrium point of the dynamical system of (3) is obtained by making the right side of the system equal to zero, providing that  $M_I \neq 0$ . We have supposed that the endemic equilibrium point of the model is denoted by  $E_m^* = (S^*, M_I^*, M_v^*, T^*, R^*)$  and the corresponding force of infection is  $\alpha_1(t) = b(M_I^*(t))$ . For simplification of algebraic manipulation, we have assumed the parameters in the model by another variable as follows.

$$\begin{aligned} n_1 &= (1 - \pi)\lambda, n_2 = \tau_1 + \mu, n_3 = (\tau_1 + \delta_1 + \mu), \\ n_4 &= \pi\lambda, n_5 = (\mu + \varphi), \\ n_6 &= \beta + \mu, n_7 = Y + \mu, n_8 = \gamma\beta\tau_1, n_9 = n_3n_6n_7, \\ n_{10} &= n_1 + \varphi n_4/n_5, n_{11} = n_9n_{10}, n_{12} = n_2n_9 \end{aligned} \quad (27)$$

$n_{13} = n_3n_{12}$  and  $n_{14} = n_8n_3$ . Now the equation of force of infection can be rearranged as

$$\begin{aligned} \alpha_1^*(n_{14}\alpha_1^* + an_{11} - n_{13}) &= 0 \Rightarrow \alpha_1^* = 0 \text{ or } n_{14}\alpha_1^* + an_{11} - n_{13} = 0 \\ \Rightarrow \alpha_1^* &= \frac{n_{13} - an_{11}}{n_{14}}, \text{ but } \alpha_1^* = \frac{n_{13} - an_{11}}{n_{14}} \\ &= (\beta + \mu)(Y + \mu) \left( \frac{(\tau_1 + \delta_1 + \mu)(\tau_1 + \mu)(\varphi + \mu) - a\lambda((\varphi + \mu)(1 - \pi) - \varphi\pi)}{\gamma\beta\tau_1(\varphi + \mu)} \right) \\ &= (\beta + \mu)(Y + \mu) \left( \frac{(\tau_1 + \delta_1 + \mu)(\tau_1 + \mu)(\varphi + \mu)}{\gamma\beta\tau_2(\varphi + \mu)} - \frac{\lambda a((\varphi + \mu)(1 - \pi) + \varphi\pi)}{\gamma\beta\tau_1(\varphi + \mu)} \right), \\ \alpha_1^* &= (\beta + \mu)(Y + \mu) \left( \frac{(\tau_1 + \delta_1 + \mu)(\tau_1 + \mu)(\varphi + \mu)}{\gamma\beta\tau_1} \right) \left( \frac{\lambda a}{\mu} \left( \frac{(1 - \pi)(\varphi + \mu) + \pi\varphi}{(\varphi + \mu)(\tau_1 + \delta_1 + \mu)} \right) - 1 \right), \\ \Rightarrow \alpha_1^* &= (\beta + \mu)(Y + \mu) \left( \frac{(\tau_1 + \delta_1 + \mu)(\tau_1 + \mu)(\varphi + \mu)}{\gamma\beta\tau_1} \right) (\mathcal{R}_{eff(m)} - 1) \Rightarrow \alpha_1^* > 0 \text{ if } \mathcal{R}_{eff(m)} > 1. \end{aligned} \quad (28)$$

Therefore, the unique endemic equilibrium point for the meningitis monoinfected model is given by  $E_m^* = (S^*, M_I^*, M_v^*, T^*, R^*)$ , where

$$\begin{aligned} S^* &= \frac{((\tau_1 + \delta_1 + \mu)(\beta + \mu)(Y + \mu)(\varphi + \mu) - (\varphi + \mu)(1 - \pi)\lambda - \varphi\pi\lambda)}{(\varphi + \mu)((\tau_1 + \mu)((\tau_1 + \delta_1 + \mu)(\beta + \mu)(Y + \mu)) - \gamma\beta\tau_1\alpha_1^*)}, \\ M_I^* &= \frac{((\tau_1 + \delta_1 + \mu)(\beta + \mu)(Y + \mu)(\varphi + \mu) - (\varphi + \mu)(1 - \pi)\lambda - \varphi\pi\lambda)}{(\varphi + \mu)((\tau_1 + \mu)((\tau_1 + \delta_1 + \mu)(\beta + \mu)(Y + \mu)) - \gamma\beta\tau_1\alpha_1^*)} \\ &\quad \cdot \left( \frac{\alpha_1^*}{(\tau_1 + \delta_1 + \mu)} \right), \\ M_v^* &= \frac{\pi\lambda}{(\varphi + \mu)}, \\ T^* &= \left( \frac{\alpha_1^*\tau_1}{(\tau_1 + \delta_1 + \mu)(\beta + \mu)} \right) \frac{((\tau_1 + \delta_1 + \mu)(\beta + \mu)(Y + \mu)(\varphi + \mu) - (\varphi + \mu)(1 - \pi)\lambda - \varphi\pi\lambda)}{(\varphi + \mu)((\tau_1 + \mu)((\tau_1 + \delta_1 + \mu)(\beta + \mu)(Y + \mu)) - \gamma\beta\tau_1\alpha_1^*)}, \\ R^* &= \frac{((\tau_1 + \delta_1 + \mu)(\beta + \mu)(Y + \mu)(\varphi + \mu) - (\varphi + \mu)(1 - \pi)\lambda - \varphi\pi\lambda)}{(\varphi + \mu)((\tau_2 + \mu)((\tau_1 + \delta_1 + \mu)(\beta + \mu)(Y + \mu)) - \gamma\beta\tau_2\alpha_2^*)} \\ &\quad \cdot \left( \frac{\beta\tau_2\alpha_2^*}{(\tau_1 + \delta_1 + \mu)(\beta + \mu)(Y + \mu)} \right). \end{aligned} \quad (29)$$

**Theorem 12.** *The endemic equilibrium point of system (17)  $E_m^* = (S^*, M_I^*, M_v^*, T^*, R^*)$  is locally asymptotically stable for the reproduction number  $\mathcal{R}_{eff(m)} > 1$ .*

*Proof:* To show the local stability of the endemic equilibrium point we have used the method of Jacobian matrix and Routh Hurwitz stability criteria.

From Jacobian matrix, we have obtained the following characteristic equation

$$(a - \lambda)(f - \lambda)(g - \lambda)(i - \lambda)(k - \lambda) - b(i - \lambda)(k - \lambda) - de(g - \lambda)hj = 0 \quad (30)$$

Where  $a = -(\alpha_1 I_m^* + \mu)$ ,  $b = -\alpha_1 S^*$ ,  $c = \varphi$ ,  $d = Y$ ,  $e = \alpha_1 I_m^*$ ,  $f = \alpha_1 S^* - (\tau_1 + \delta_1 + \mu)$ ,

$$\begin{aligned} g &= -(\mu + \varphi), h = \tau_1, i = -(\beta + \mu), j = \beta \text{ and } k = -(Y + \mu). \\ \Rightarrow a_0\lambda^5 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5 &= 0 \end{aligned} \quad (31)$$

Where  $a_0 = 1$ ,  $-(i + a + f + g + k) = a_1$ ,  $(ai + if + af + ig + ag + fg + ik + ak + fk + g) = a_2$ ,  $(-b + aif + aig + ifg + afg + aik + ifk + afk + gik + agk + f gk) = a_3$ ,  $(bi - aifg + dehj + bk - aifk - aigk - ifgk - afgk) = a_4$   
 $a_5 = aeghj + bik - aifgk$ .

To apply Routh-Hurwitz stability criteria, it is the must to check the necessary condition of all the coefficients have the same sign or not. Since  $a_0 = 1$  is positive in sign, all  $a_1, a_2, a_3, a_4$  and  $a_5$  should be positives in sign. All the coefficients of the characteristic's polynomial are positives whenever  $\mathcal{R}_{eff(p)} > 1$ .

We have observed that the first column of the Routh Hurwitz array has no sign change, thus the root of the characteristics equation of the dynamical system  $a_0\lambda^5 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5 = 0$  are negative. Hence, the endemic equilibrium point of the dynamical system is locally asymptotically stable.

**2.8. Positivity and Boundedness of Full Pneumonia and Meningitis Coinfected Model.** The corresponding dynamical system of the full pneumonia and meningitis coinfection model is given in Equation (1).

The constructed model is expected to be meaningful epidemiologically as well as well-posed. We need to prove that all the state variables of the dynamical system are positive.

**Theorem 13.** *All the population of the system with positive initial conditions are nonnegative*

*Proof:* Assume  $S(0) > 0, M_V(0) > 0, P_V(0) > 0, MP_V(0) > 0, M_I(0) > 0, P_I(0) > 0, MP_I(0) > 0, T(0) > 0$ , and  $R(0) > 0$  are positive for time  $t > 0$  and for all nonnegative parameters. Let us define  $\tau = \sup \{t > 0 \text{ such that } S(t) > 0, M_V(t) > 0, P_V(t) > 0, MP_V(t) > 0, M_I(t) > 0, P_I(t) > 0, MP_I(t) > 0, T(t) > 0, R(t) > 0 \text{ and } t \in [0, \tau]\}$ .

Since all  $S(t), M_V(t), P_V(t), MP_V(t), M_I(t), P_I(t), MP_I(t), T(t)$  and  $R(t)$  are continuous, we can say for  $\tau > 0$ . If  $\tau = +\infty$ , then positivity holds.

Nevertheless, if  $0 < \tau < +\infty$ , then all the state variables are zeros.



From the first equation of system (1) we do have

$$\begin{aligned} \frac{dS}{dt} &= (1 - \pi)\lambda + \phi P_V + \varphi M_V + YR - (\alpha_1 + \alpha_2 + \mu)SS(t) \\ &= S(0)e^{-\int_0^t (\alpha_1 + \alpha_2 + \mu)dt} + e^{-\int_0^t (\alpha_1 + \alpha_2 + \mu)dt} \\ &\quad \cdot \left[ \int_0^t e^{\int_0^s (\alpha_1 + \alpha_2 + \mu)dt} ((1 - \pi)\lambda + \phi P_V + \varphi M_V + YR)dt \right] \\ &> 0 \Rightarrow S(t) > 0 \end{aligned} \quad (32)$$

Following same procedure, all the remaining state variables are nonnegative.

Therefore, from proof, we can conclude that whenever the initial values of the systems are all nonnegative, then all the solutions of our dynamical system are positive.

**Theorem 14.** *The total human population is assumed to be  $N$  and the dynamical system (1) is positively invariant in the closed invariant set  $\Omega = \{(S, M_V, P_V, MP_V, M_I, P_I, MP_I, T, R) \in \mathbb{R}_+^9 : N \leq \lambda/\mu\}$ . Furthermore, the system's nonnegative solutions are all constrained, and it may exhibit the persistence property under any nonnegative initial concentration conditions [24]. Proof: to get an invariant region, which shows boundedness of solution, is obtained as follow.*

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dM_V}{dt} + \frac{dP_V}{dt} + \frac{dMP_V}{dt} + \frac{dM_I}{dt} + \frac{dP_I}{dt} + \frac{dMP_I}{dt} + \frac{dT}{dt} + \frac{dR}{dt}, \quad (33)$$

$$\Rightarrow \frac{dN}{dt} = \lambda - \mu N - \delta_2 P_I - \delta_1 M_I - \delta_3 MP_I, \quad (34)$$

$$\Rightarrow N(t) \leq N_0 e^{-\mu t} + \frac{\lambda}{\mu} \Rightarrow 0 \leq N \leq \frac{\lambda}{\mu}. \quad (35)$$

Therefore, the dynamical system that we have constructed is bounded.

**2.9. Disease-Free Equilibrium Point and Its Stability.** The disease-free equilibrium point of full pneumonia and meningitis coinfection model  $E_{pm}^0$  is obtained by making all the right-hand-side of equation in system (1), providing that all the infectious classes are equal to zero.

$$\begin{aligned} &(S^0, M_V^0, P_V^0, MP_V^0, M_I^0, P_I^0, MP_I^0, T^0, R^0) \\ &= \left( \frac{(1 - \pi)\lambda}{(\alpha_1 + \alpha_2 + \mu)} + \left( \frac{\pi\lambda}{(\alpha_1 + \alpha_2 + \mu)} \right) \left( \frac{\phi\rho}{(\mu + \phi + \varepsilon_2)} + \frac{(1 - \rho)\varphi}{(\mu + \varphi + \varepsilon_1)} \right), \right. \\ &\quad \frac{(1 - \rho)\pi\lambda}{(\mu + \varphi + \varepsilon_1)}, \frac{\rho\pi\lambda}{(\mu + \phi + \varepsilon_2)}, \frac{(1 - \rho)\pi\lambda\varepsilon_1}{\mu(\mu + \varphi + \varepsilon_1)} \\ &\quad \left. + \frac{\rho\pi\lambda\varepsilon_2}{\mu(\mu + \phi + \varepsilon_2)}, 0, 0, 0, 0, 0 \right) \end{aligned} \quad (36)$$

**2.9.1. Effective Reproduction Number.** The reproduction number is the average number of people that become infected because of the entry of one infectious person into

a completely susceptible population in the absence of intervention. Moreover, reproduction number is utilized to determine the effect of the control measures and to understand the capability of the spread of the infection to disseminate in the entire community when the control strategies are applied [15, 17, 21].

The reproduction number of pneumonia and meningitis coinfection model denoted by  $\mathcal{R}_{eff}$ , which is manipulated by the Van den Driessche, Pauline, and James Warmouth next generation matrix approach [20], is the largest eigenvalue of the next generation matrix  $FV^{-1} = [\partial\mathcal{F}_i(E_r^0)/\partial x_j] [\partial v_i(E_r^0)/\partial x_j]^{-1}$ , where  $\mathcal{F}_i$  is the rate of appearance of new infection in compartment  $i$ ,  $v_i$  is the transfer of infections from one compartment  $i$  to another, and  $E_{pm}^0$  is the disease-free equilibrium point.

The

$$\mathcal{F}_i(\mathbf{x}) = \begin{bmatrix} \alpha_1 S^0 \\ \alpha_2 S^0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \quad v_i = \begin{bmatrix} (\pi - 1)\lambda - \phi P_V^0 - \varphi M_V^0 - YR^0 + \mu S^0 \\ (\mu + \varphi + \varepsilon_1)M_V^0 - (1 - \rho)\pi\lambda \\ (\mu + \phi + \varepsilon_2)P_V^0 - \rho\pi\lambda \\ \mu MP_V^0 - \varepsilon_1 M_V^0 - \varepsilon_2 P_V^0 \\ (\omega_2 \alpha_2 + \tau_1 + \delta_1 + \mu)M_I^0 \\ (\omega_1 \alpha_1 + \tau_2 + \delta_2 + \mu)P_I^0 \\ (\tau_3 + \mu)MP_I^0 - \omega_1 \alpha_1 P_I - \omega_2 \alpha_2 M_I^0 \\ (\beta + \mu)T - \tau_1 M_I^0 - \tau_2 P_I - \tau_3 MP_I^0 \\ (Y + \mu)R^0 - \beta T^0 \end{bmatrix}. \quad (37)$$

Then

$$F = \left[ \frac{(1 - \pi)\lambda}{\mu} + \left( \frac{\pi\lambda}{\mu} \right) \left( \frac{\phi\rho}{(\mu + \phi + \varepsilon_2)} + \frac{(1 - \rho)\varphi}{(\mu + \varphi + \varepsilon_1)} \right) \right] \cdot \begin{bmatrix} a & 0 & a \\ 0 & b & b \\ 0 & 0 & 0 \end{bmatrix}, \quad (38)$$

and

$$V = \begin{bmatrix} (\tau_1 + \delta_1 + \mu) & 0 & 0 \\ 0 & (\tau_2 + \delta_2 + \mu) & 0 \\ 0 & 0 & (\tau_3 + \mu) \end{bmatrix}$$

$$\Rightarrow FV^{-1} = \begin{bmatrix} \frac{a}{(\tau_1 + \delta_1 + \mu)} & 0 & \frac{a}{(\tau_3 + \mu)} \\ 0 & \frac{b}{(\tau_2 + \delta_2 + \mu)} & \frac{b}{(\tau_3 + \mu)} \\ 0 & 0 & 0 \end{bmatrix}. \quad (39)$$

The eigenvalues of the next generation matrix  $F \cdot V^{-1}$  are

$$\left\{ 0, \frac{a\lambda(1-\pi)}{(\tau_1 + \delta_1 + \mu)\mu} + \frac{\phi\rho(\mu + \varphi + \varepsilon_1) + \varphi(1-\rho)(\mu + \phi + \varepsilon_2)}{(\mu + \phi + \varepsilon_2)\mu} \cdot \left( \frac{\pi\lambda a}{\tau_1 + \delta_1 + \mu} \right), \frac{b\lambda(1-\pi)}{(\tau_2 + \delta_2 + \mu)\mu} + \frac{\phi\rho(\mu + \varphi + \varepsilon_1) + \varphi(1-\rho)(\mu + \phi + \varepsilon_2)}{(\mu + \phi + \varepsilon_2)\mu} \left( \frac{\pi\lambda b}{\tau_2 + \delta_2 + \mu} \right) \right\}. \quad (40)$$

Therefore, the effective reproduction number of full meningitis and pneumonia model is

$$\mathcal{R}_{eff} = \max \left\{ \frac{a\lambda(1-\pi)}{(\tau_1 + \delta_1 + \mu)\mu} + \frac{\phi\rho(\mu + \varphi + \varepsilon_1) + \varphi(1-\rho)(\mu + \phi + \varepsilon_2)}{(\mu + \phi + \varepsilon_2)\mu} \cdot \left( \frac{\pi\lambda a}{\tau_1 + \delta_1 + \mu} \right), \frac{b\lambda(1-\pi)}{(\tau_2 + \delta_2 + \mu)\mu} + \frac{\phi\rho(\mu + \varphi + \varepsilon_1) + \varphi(1-\rho)(\mu + \phi + \varepsilon_2)}{(\mu + \phi + \varepsilon_2)\mu} \left( \frac{\pi\lambda b}{\tau_2 + \delta_2 + \mu} \right) \right\}, \quad (41)$$

$\mathcal{R}_{eff} = \max \{R_1, R_2\}$  where  $R_1 = a\lambda(1-\pi)/(\tau_1 + \delta_1 + \mu)\mu + (\phi\rho(\mu + \varphi + \varepsilon_1) + \varphi(1-\rho)(\mu + \phi + \varepsilon_2))/(\mu + \phi + \varepsilon_2)\mu(\pi\lambda a/\tau_1 + \delta_1 + \mu)$ , and  $R_2 = b\lambda(1-\pi)/(\tau_2 + \delta_2 + \mu)\mu + (\phi\rho(\mu + \varphi + \varepsilon_1) + \varphi(1-\rho)(\mu + \phi + \varepsilon_2))/(\mu + \phi + \varepsilon_2)\mu(\pi\lambda b/(\tau_2 + \delta_2 + \mu))$ .

**Theorem 15.** The disease-free equilibrium point  $E_{pm}^0$  of the model in system (17) is locally asymptotically stable if the effective reproduction number  $\mathcal{R}_{eff} < 1$  and is unstable if  $\mathcal{R}_{eff} > 1$ .

*Proof:*

The Jacobean matrix  $J(E_{pm}^0)$  of the model (1) with respect to  $(S, M_V, P_V, MP_V, M_I, P_I, MP_I, T, R)$  at the disease-free equilibrium point is the following:

$$J(E_{pm}^0) = \begin{bmatrix} -\mu & \varphi & \phi & 0 & r_1 & r_2 & r_3 & 0 & Y \\ 0 & r_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & r_5 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \varepsilon_1 & \varepsilon_2 & -\mu & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & r_6 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & r_7 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & r_8 & 0 & 0 \\ 0 & 0 & 0 & 0 & \tau_1 & \tau_2 & \tau_3 & r_9 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \beta & r_{10} \end{bmatrix}, \quad (42)$$

where

$$\begin{aligned} r_1 &= -a(((1-\pi)\lambda/\mu) + (\pi\lambda/\mu)((\phi\rho/(\mu + \phi + \varepsilon_2)) \\ &\quad + ((1-\rho)\varphi/(\mu + \varphi + \varepsilon_1))))), r_2 = -b((1-\pi)\lambda/(\alpha_1 + \alpha_2 + \mu) \\ &\quad + (\pi\lambda/\mu)((\phi\rho/(\mu + \phi + \varepsilon_2)) + ((1-\rho)\varphi/(\mu + \varphi + \varepsilon_1))), \\ r_3 &= -(ae_1 + be_2)((1-\pi)\lambda/\mu) + (\pi\lambda/\mu)((\phi\rho/(\mu + \phi + \varepsilon_2)) \\ &\quad + ((1-\rho)\varphi/(\mu + \varphi + \varepsilon_1))), r_4 = -(\mu + \varphi + \varepsilon_1), r_5 = -(\mu + \phi + \varepsilon_2), \\ r_6 &= a\left(\frac{(1-\pi)\lambda}{\mu} + \left(\frac{\pi\lambda}{\mu}\right)\left(\frac{\phi\rho}{(\mu + \phi + \varepsilon_2)} + \frac{(1-\rho)\varphi}{(\mu + \varphi + \varepsilon_1)}\right)\right) - (\tau_1 + \delta_1 + \mu), \\ r_7 &= b\left(\frac{(1-\pi)\lambda}{\mu} + \left(\frac{\pi\lambda}{\mu}\right)\left(\frac{\phi\rho}{(\mu + \phi + \varepsilon_2)} + \frac{(1-\rho)\varphi}{(\mu + \varphi + \varepsilon_1)}\right)\right) - (\tau_2 + \delta_2 + \mu), \\ r_8 &= -(\tau_3 + \mu), r_9 = -(\beta + \mu) \text{ and } r_{10} = -(Y + \mu) \Rightarrow (\lambda_1 + \mu)(\lambda_4 + \mu) \\ &\quad \cdot (-\lambda_2 + r_4)(-\lambda_3 + r_5)(-\lambda_5 + r_6)(-\lambda_6 + r_7)(-\lambda_7 + r_8)(-\lambda_8 + r_9) \\ &\quad \cdot (-\lambda_9 + r_{10}) \Rightarrow \lambda_1 = -\mu, \lambda_4 = -\mu, \lambda_2 = r_4 = -(\mu + \varphi + \varepsilon_1), \\ &\quad \lambda_3 = r_5 = -(\mu + \phi + \varepsilon_2), \lambda_7 = r_8 = -(\tau_3 + \mu), \lambda_8 = r_9 = -(\beta + \mu), \end{aligned}$$

$$\begin{aligned} \lambda_5 &= (\tau_1 + \delta_1 + \mu) \left[ \left( \frac{a\lambda(\mu + \varphi)}{\mu(1-\pi)(\mu + \varphi) + \pi\varphi} \right) \cdot \left( \frac{(\mu + \phi + \varepsilon_2) + \pi\phi\rho}{(\mu + \phi + \varepsilon_2)} + \frac{(1-\rho)\pi\varphi}{(\mu + \varphi + \varepsilon_1)} \right) \cdot \left( \left( \frac{a\lambda(1-\pi)(\mu + \varphi) + \pi\varphi}{\mu(\mu + \varphi)(\tau_1 + \delta_1 + \mu)} \right) - 1 \right) \right] \\ &\Rightarrow \lambda_5 = (\tau_1 + \delta_1 + \mu) \left[ \left( \frac{a\lambda(\mu + \varphi)}{\mu(1-\pi)(\mu + \varphi) + \pi\varphi} \right) \cdot \left( \frac{(\mu + \phi + \varepsilon_2) + \pi\phi\rho}{(\mu + \phi + \varepsilon_2)} + \frac{(1-\rho)\pi\varphi}{(\mu + \varphi + \varepsilon_1)} \right) (R_{eff(m)} - 1) \right]. \\ \lambda_6 &= (\tau_2 + \delta_2 + \mu) \left[ \left( \frac{b\lambda(\mu + \phi)}{\mu(1-\pi)(\mu + \phi) + \pi\varphi} \right) \cdot \left( \frac{(\mu + \phi + \varepsilon_2) + \pi\phi\rho}{(\mu + \phi + \varepsilon_2)} + \frac{(1-\rho)\pi\varphi}{(\mu + \varphi + \varepsilon_1)} \right) \cdot \left( \frac{\lambda b \left( \frac{(1-\pi)(\mu + \phi) + \pi\varphi}{(\mu + \phi)(\tau_2 + \delta_2 + \mu)} \right)}{\mu} \right) - 1 \right] \\ &\Rightarrow \lambda_6 = (\tau_2 + \delta_2 + \mu) \left[ \left( \frac{b\lambda(\mu + \phi)}{\mu(1-\pi)(\mu + \phi) + \pi\varphi} \right) \cdot \left( \frac{(\mu + \phi + \varepsilon_2) + \pi\phi\rho}{(\mu + \phi + \varepsilon_2)} + \frac{(1-\rho)\pi\varphi}{(\mu + \varphi + \varepsilon_1)} \right) (R_{eff(p)} - 1) \right] \text{ and } \lambda_9 = r_{10} = -(Y + \mu). \end{aligned} \quad (43)$$

Hence, all the parameters are nonnegative as well as all the eigenvalues of the corresponding Jacobean matrix are negative except  $\lambda_5$  and  $\lambda_6$ .

The sign of eigenvalues  $\lambda_5$  and  $\lambda_6$  depends on the values of  $R_{eff(m)}$  and  $R_{eff(p)}$ , respectively. Moreover,  $\lambda_5 < 0$  whenever  $R_{eff(m)} < 1$  and  $\lambda_6 < 0$  if and only if  $R_{eff(p)} < 1$ .

Therefore, the disease-free equilibrium point of the full meningitis and pneumonia coinfection model is local asymptotically stable only if  $R_{eff(m)} < 1$  and  $R_{eff(p)} < 1$ , otherwise it is unstable.

**2.9.2. Global Stability of Disease-Free Equilibrium Point.** The disease-free equilibriums of the meningitis monoinfection model and the pneumonia monoinfection model are both globally asymptotically stable whenever their corresponding effective reproduction numbers values are smaller than unity, as demonstrated in Section 3 Figure 2 and 3, respectively. In light of this conclusion, the disease-free equilibrium point of the pneumonia and meningitis coinfection model (1) is globally asymptotically stable if  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \} < 1$ .

**2.10. Endemic Equilibrium Point and Its Stability.** The endemic equilibrium point of the dynamical system (1) is obtained by making the right side of the system equal to zero providing that  $M_I \neq 0$ ,  $P_I \neq 0$  and  $MP_I \neq 0$ . We have supposed the endemic equilibrium point of the model is denoted by  $E_{pm}^* = (S^*, M_v^*, P_v^*, MP_v^*, M_I^*, P_I^*, MP_I^*, T^*, R^*)$  and the corresponding forces of infection are

$$\alpha_1^* = a(M_I^* + e_1 MP_I^*) \text{ and } \alpha_2^* = b(P_I^* + e_2 MP_I^*),$$

$$S^* = \frac{1}{(\alpha_1^* + \alpha_2^* + \mu)} \cdot \left( (1-\pi)\lambda + \frac{\phi\rho\pi\lambda}{(\mu + \phi + \varepsilon_2)} + \frac{(1-\rho)\varphi\pi\lambda}{(\mu + \varphi + \varepsilon_1)} + Y \frac{\beta\Gamma^*}{(Y + \mu)} \right),$$

$$M_v^* = \frac{(1-\rho)\pi\lambda}{(\mu + \varphi + \varepsilon_1)}, P_v^* = \frac{\rho\pi\lambda}{(\mu + \phi + \varepsilon_2)},$$

$$MP_v^* = \left( \frac{\varepsilon_1}{\mu} \right) \left( \frac{(1-\rho)\pi\lambda}{(\mu + \varphi + \varepsilon_1)} \right) + \left( \frac{\varepsilon_2}{\mu} \right) \left( \frac{\rho\pi\lambda}{(\mu + \phi + \varepsilon_2)} \right),$$

$$M_I^* = \left( \frac{\alpha_1}{(\omega_2\alpha_2^* + \tau_1 + \delta_1 + \mu)} \right) \left( \frac{1}{(\alpha_1^* + \alpha_2^* + \mu)} \cdot \left( (1-\pi)\lambda + \frac{\phi\rho\pi\lambda}{(\mu + \phi + \varepsilon_2)} + \frac{(1-\rho)\varphi\pi\lambda}{(\mu + \varphi + \varepsilon_1)} + Y \frac{\beta\Gamma^*}{(Y + \mu)} \right) \right),$$

$$P_I^* = \left( \frac{\alpha_2}{(\omega_1\alpha_1^* + \tau_2 + \delta_2 + \mu)} \right) \left( \frac{1}{(\alpha_1^* + \alpha_2^* + \mu)} \cdot \left( (1-\pi)\lambda + \frac{\phi\rho\pi\lambda}{(\mu + \phi + \varepsilon_2)} + \frac{(1-\rho)\varphi\pi\lambda}{(\mu + \varphi + \varepsilon_1)} + Y \frac{\beta\Gamma^*}{(Y + \mu)} \right) \right),$$

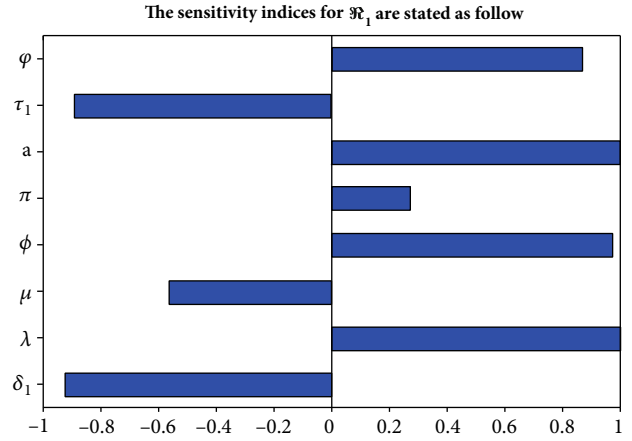


FIGURE 2: Sensitivity indices for  $\mathcal{R}_2$ .

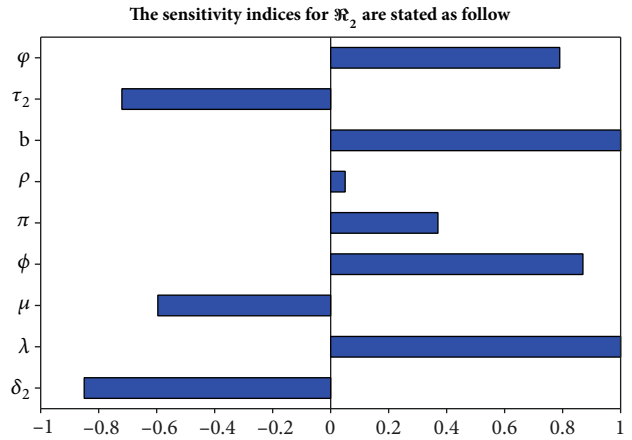


FIGURE 3: Stability of endemic equilibrium when  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \} > 1$ .

$$MP_I^* = \left( \frac{1}{(\tau_3 + \mu)} \right) (\omega_1\alpha_1^*) \left( \frac{\alpha_2}{(\omega_1\alpha_1^* + \tau_2 + \delta_2 + \mu)} \right) \cdot \left( \frac{1}{(\alpha_1^* + \alpha_2^* + \mu)} \left( (1-\pi)\lambda + \frac{\phi\rho\pi\lambda}{(\mu + \phi + \varepsilon_2)} + \frac{(1-\rho)\varphi\pi\lambda}{(\mu + \varphi + \varepsilon_1)} + Y \frac{\beta\Gamma^*}{(Y + \mu)} \right) \right) + \left( \left( \frac{\alpha_1\omega_2\alpha_2^*}{(\omega_2\alpha_1^* + \tau_1 + \delta_1 + \mu)(\alpha_1^* + \alpha_2^* + \mu)(\tau_3 + \mu)} \right) \cdot \left( (1-\pi)\lambda + \frac{\phi\rho\pi\lambda}{(\mu + \phi + \varepsilon_2)} + \frac{(1-\rho)\varphi\pi\lambda}{(\mu + \varphi + \varepsilon_1)} + Y \frac{\beta\Gamma^*}{(Y + \mu)} \right) \right),$$

$$T^* = \frac{\tau_1 M_I^* + \tau_2 P_I^* + \tau_3 MP_I^*}{(\beta + \mu)}, R^* = \frac{\beta\Gamma^*}{(Y + \mu)}. \quad (44)$$

**Theorem 16.** The endemic equilibrium point  $E_{pm}^*$  is locally asymptotically stable if the  $\mathcal{R}_{eff} > 1$ , otherwise it is unstable.

*Proof:* the local stability of the endemic equilibrium point of the full model is verified by using a numerical simulation in Section 4 Figure 3.

### 3. Sensitivity Analysis and Numerical Simulations

In this section, we have carried out the sensitivity analysis to find the possible sensitive parameters having important implications to prevent and control the meningitis and pneumonia coinfection spread and the numerical simulations of model parameters and model solutions to approve the analytical results that we have done in Section 3.

In the numerical simulation of the meningitis and pneumonia coinfection model, we assessed the possible impact of controlling strategies on the dynamics of the disease.

**3.1. Sensitivity Analysis. Definition.** the normalized forward sensitivity index of a variable meningitis and pneumonia reproduction number denoted by the symbol  $\mathcal{R}_{eff}$  that depends differentially on a parameter  $\xi$  is defined as  $SI(p) = \partial\mathcal{R}_{eff}/\partial\xi * \xi/\mathcal{R}_{eff}$  [2, 17].

Conducting sensitivity analysis provides a number of benefits for decision-makers. First, it acts as an in-depth study of all the variables. Secondly, it allows decision-makers to identify where they can make improvements in the future. In our case, it helps us to determine the relative and importance of different parameters in meningitis and pneumonia incidence and prevalence. The most sensitive parameter has the magnitude of the sensitivity index larger than that of all other parameters. We have manipulated the sensitivity index in terms of

$$\begin{aligned} \mathcal{R}_{eff} = \max & \left\{ \frac{a\lambda(1-\pi)}{(\tau_1 + \delta_1 + \mu)\mu} \right. \\ & + \frac{\phi\rho(\mu + \varphi + \varepsilon_1) + \varphi(1-\rho)(\mu + \phi + \varepsilon_2)}{(\mu + \phi + \varepsilon_2)\mu} \\ & \cdot \left( \frac{\pi\lambda a}{\tau_1 + \delta_1 + \mu} \right), \frac{b\lambda(1-\pi)}{(\tau_2 + \delta_2 + \mu)\mu} \\ & + \frac{\phi\rho(\mu + \varphi + \varepsilon_1) + \varphi(1-\rho)(\mu + \phi + \varepsilon_2)}{(\mu + \phi + \varepsilon_2)\mu} \\ & \cdot \left. \left( \frac{\pi\lambda b}{\tau_2 + \delta_2 + \mu} \right) \right\}. \end{aligned} \quad (45)$$

We could have manipulated the sensitivity index in terms of  $\mathcal{R}_1$  and  $\mathcal{R}_2$  since  $\mathcal{R}_{eff} = \max \{\mathcal{R}_1, \mathcal{R}_2\}$ .

Sensitivity analysis results and the numerical simulation are given in this section with parameter values given in Table 2 with the initial population of the full meningitis and pneumonia coinfection model.

**3.1.1. The Sensitivity Indices for  $\mathcal{R}_1$ .** In this section, we have stated the sensitivity indices for  $\mathcal{R}_1$ . Using the values of the parameters in Table 2, the sensitivity indices for  $\mathcal{R}_1$  is calculated in the following:

$$(1) SI(a) = \partial\mathcal{R}_1/\partial a * (a/\mathcal{R}_1)(\partial\mathcal{R}_1/\partial a) = \lambda[1-\pi]/\mu(\mu + \delta_1 + \tau_1) + (\pi\lambda(\phi\rho[1+\mu+\varphi] + (1+\mu+\phi)\varphi[1-\rho]))/\mu(1+\mu+\phi)(\mu + \delta_1 + \tau_1) \Rightarrow SI(a) = 1$$

TABLE 2: Parameter values used for the coinfection model simulation.

Parameter	Values	Unit	Source
$a$	0.9	size <sup>-1</sup> * Time <sup>-1</sup>	[8]
$b$	0.007-0.6	size <sup>-1</sup> * Time <sup>-1</sup>	[8]
$\tau_1$	0.02	Time <sup>-1</sup>	[8]
$\tau_2$	0.012	Time <sup>-1</sup>	[8]
$\tau_3$	0.31	Time <sup>-1</sup>	[8]
$\beta$	0.06 -0.13	Time <sup>-1</sup>	[16, 17]
$\mu$	0.01	Time <sup>-1</sup>	[8]
$\delta_1$	0.002-0.2	Time <sup>-1</sup>	[8]
$\delta_2$	0.006-0.05	Time <sup>-1</sup>	[8]
$\delta_3$	0.008-0.7	Time <sup>-1</sup>	[8]
$\omega_1$	1	Time <sup>-1</sup>	Assumed
$\omega_2$	1	Time <sup>-1</sup>	Assumed
$Y$	<b>0.007-0.4</b>	Time <sup>-1</sup>	[8]
$\pi$	0.105	Time <sup>-1</sup>	[8]
$\lambda$	<b>0.0413</b> * $N_0$	Size * Time <sup>-1</sup>	[8]
$\varepsilon_2$	0.007	Time <sup>-1</sup>	Assumed
$\varepsilon_1$	0.025	Time <sup>-1</sup>	Assumed
$\phi$	0.0115	Time <sup>-1</sup>	[22]
$\varphi$	0.5	Time <sup>-1</sup>	[23]
$\rho$	0.05	Time <sup>-1</sup>	[21]

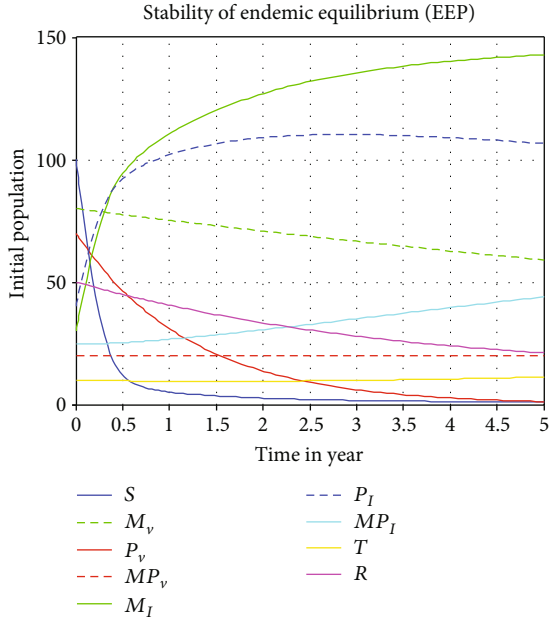
$$\begin{aligned} (2) SI(\delta_1) &= \partial\mathcal{R}_1/\partial\delta_1 * \delta_1/\mathcal{R}_1 \\ &\Rightarrow - \\ &\partial\mathcal{R}_1/\partial\delta_1 = -a\lambda[1-\pi]/\mu(\mu + \delta_1 + \tau_1)^2 - (a\pi\lambda(\phi\rho[1 \\ &+ \mu + \varphi] + (1 + \mu + \phi)\varphi[1 - \rho]))/\mu(1 + \mu + \phi) \\ &(\mu + \delta_1 + \tau_1)^2 \Rightarrow SI(\delta_1) = -\delta_1/(\mu + \delta_1 + \tau_1) \end{aligned}$$

Following the procedures we have generalized and stated the sensitivity parameters as follows:

In this section, the obtained figure was explored with parameter values given in Table 2, and we have gotten  $\mathcal{R}_1 = 12.33$  at  $a = 0.9$ , which implies meningitis has been expanded throughout the considered community, and additionally, we have manipulated the sensitivity indices for other parameters as show in Figure 4. The obtained sensitivity analysis shows that the recruitment rate  $\lambda$  and meningitis effective contact rate  $a$  have the highest impact on  $\mathcal{R}_1$ .

**3.1.2. The Sensitivity Indices for  $\mathcal{R}_2$ .** In this section we have presented the sensitivity indices for  $\mathcal{R}_2$ . Using the values of parameters in Table 2, the sensitivity indices for  $\mathcal{R}_2$  is calculated in following chart.

$$(1) SI(b) = \partial\mathcal{R}_2/\partial b * (b/\mathcal{R}_2)(\partial\mathcal{R}_2/\partial b) = (\pi\lambda\phi\rho[1 + \mu + \varphi] + (1 + \mu + \phi)(\lambda[1 - \pi] + \pi\lambda\varphi[1 - \rho]))/\mu(1 + \mu + \phi)(\mu + \delta_2 + \tau_2) \Rightarrow SI(b) = 1$$


 FIGURE 4: Sensitivity indices for  $\mathcal{R}_1$ .

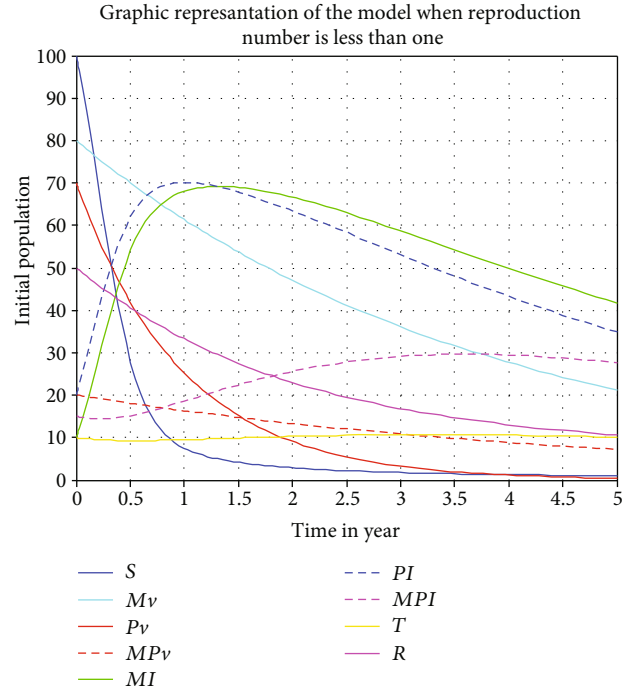
$$(2) SI(\delta_2) = \partial \mathcal{R}_2 / \partial \delta_2 * \delta_2 / \mathcal{R}_2 \Rightarrow \partial \mathcal{R}_2 / \partial \delta_2 = (b(\pi\lambda((1 + \mu)\rho(\phi - \varphi) + (1 + \mu + \phi)\varphi) + (1 + \mu + \phi)\lambda[1 - \pi])) / \mu(1 + \mu + \phi)(\mu + \delta_2 + \tau_2)$$

Following the procedures we have generalized and stated the sensitivity parameters as follow

In this section, the obtained figure was explored with parameters values given in Table 2 and we have got  $\mathcal{R}_2 = 15.1$  at  $b = 0.85$  which implies pneumonia has been expanded throughout the considered community and additionally we have manipulated the sensitivity indices for another parameters as show in Figure 2 above. The obtained sensitivity analysis shows that the recruitment rate  $\lambda$  and pneumonia effective contact rate  $b$  have the highest impact on  $\mathcal{R}_2$ .

**3.2. Numerical Simulations.** MATLAB software is used to ensure the accuracy of the mathematical terminology descriptions and the validity of the analytical solution. To show the verification of the analytical solution we acquired in the previous part, we utilized the MATLAB code ODE 45. Additionally, we have shown and investigated the effects of a number of traits (parameters) that are related to meningitis and pneumonia coinfection illness.

**3.2.1. Local Stability of the Endemic Equilibrium Point.** The parameters from Table 2 were used to run a numerical simulation using the ODE 45, which produced the results shown in Figure 3. From Figure 3, we can observe that after a year, the solutions of the meningitis and pneumonia coinfection dynamical system (1) will be approaching to the endemic equilibrium point of the meningitis and pneumonia coinfection depends on the value of  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \}$ . More specifically, Figure 3 shows that after a time, the solutions


 FIGURE 5: model representation when  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \} < 1$ 

of the meningitis and pneumonia infection transmission dynamics will be converging to its endemic equilibrium point, i.e., the endemic equilibrium point is locally asymptotically stable whenever  $a = 0.9$  and  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \} = 15.1 > 1$ . This mathematical conclusion illustrates the physical phenomena known as the proliferation and spread of meningitis and pneumonia coinfection regularly happening throughout the population which is confined to a certain location.

Figure 5 demonstrates that while the  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \} < 1$ , the solution of the system is converged to the disease-free equilibrium point.

**3.2.2. The Impact of Meningitis Treatment Rate on Meningitis and Pneumonia Coinfected Populations.** We looked at how the coinfecting class was affected by  $\tau_3$  in this part. We can see from Figure 6 that the prevalence of meningitis and pneumonia coinfection decreases as values of  $\tau_3$  rise. To manage the disease in the community, public authorities must focus on increasing the values of the treatment rate  $\tau_3$ .

**3.2.3. The Impact of Meningitis Treatment Rate on Coinfected Class.** The endeavor we conducted about the impact of  $\tau_1$  on the coinfecting class is illustrated in Figure 7. The figure shows that the population with coinfections of meningitis and pneumonia is decreasing as the values of the meningitis treatment rate, indicated by  $\tau_1$ , rise. The most crucial thing to remember is that treating just meningitis-infected people can significantly reduce the co-occurrence of meningitis and pneumonia infections in communities, which is one of the study's main findings.

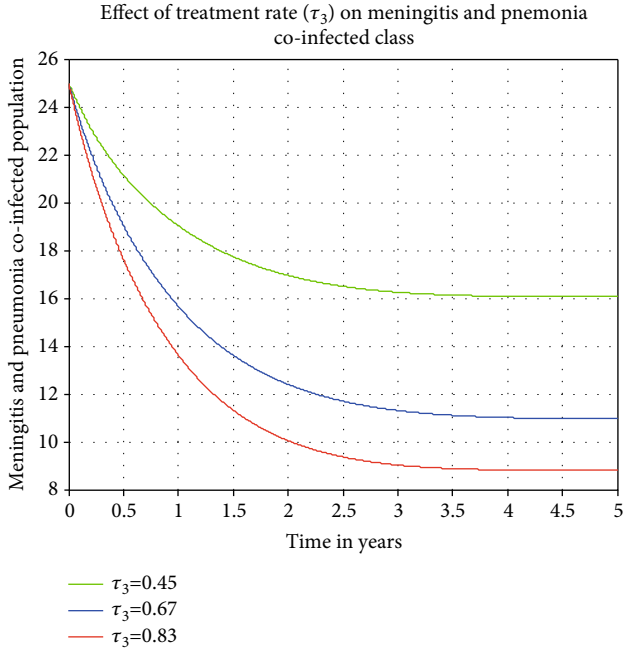


FIGURE 6: The effect of treatment rate on the coinfected population.

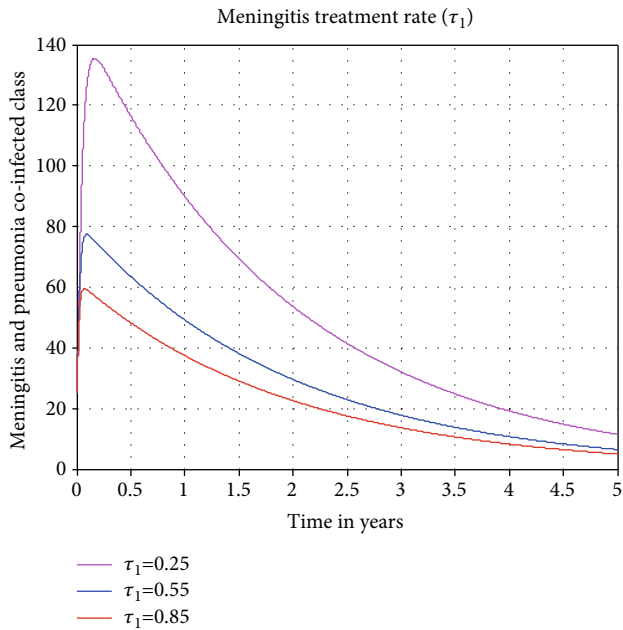


FIGURE 7: Plot effect of meningitis treatment rate on the coinfected groups.

As a result, we advise the stakeholders to maximize the meningitis treatment rate in order to confine and stop the spread of the meningitis and pneumonia coinfection illness in the community.

3.2.4. *The Impact of Pneumonia Treatment Rate on Coinfected Class.* The influence of pneumonia treatment rate ( $\tau_2$ ) on the coinfected class is shown in Figure 8.

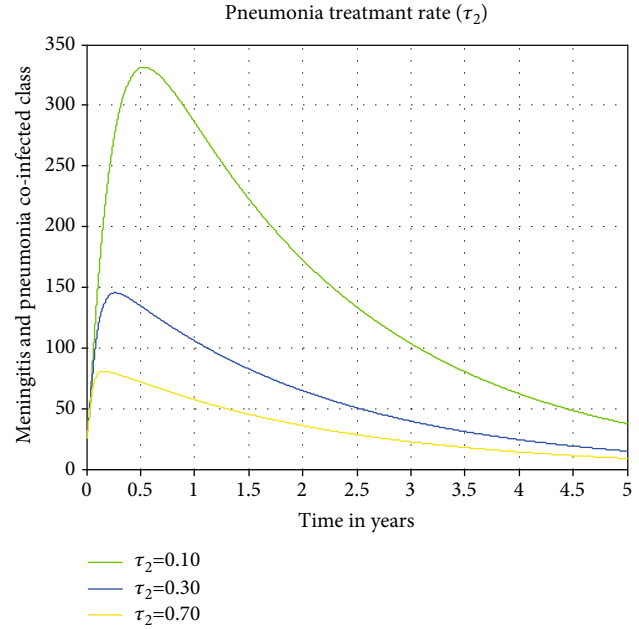


FIGURE 8: Plot effect of pneumonia treatment rate on the coinfected class.

The graph shows that when the incidence of pneumonia treatment rises, the population with coinfections of meningitis and pneumonia declines. This study's other key observation is that treating pneumonia-infected people only has a substantial impact on reducing the cooccurrence of meningitis and pneumonia infections in communities, which is perhaps most relevant. In order to prevent and slow the spread of the meningitis and pneumonia coinfection illness in the community, we advise the stakeholders to maximize the values of the pneumonia treatment rate of  $\tau_2$ .

3.2.5. *The Impact of Parameters on  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \}$ .* In this subsection, as we see in Figure 9, we have investigated the effect of vaccination ( $\varepsilon_2$ ) and the effective reproduction number  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \}$ . The figure reflects that when the value of  $\varepsilon_2$  increases, both  $\mathcal{R}_1$  and  $\mathcal{R}_2$  decrease, which implies that the maximum of them also declines.

Moreover, the value  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \}$  becomes smaller than one when the value of  $\varepsilon_2 > 0.39$ .

In this subsection, as we can see in Figure 10, we have investigated the effect of vaccination ( $\varepsilon_1$ ) and the effective reproduction number  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \}$ . The figure reflects that when the value of  $\varepsilon_1$  increases, both  $\mathcal{R}_1$  and  $\mathcal{R}_2$  decrease, which implies that the maximum of them also declines.

Moreover, the value  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \}$  becomes smaller than one when the value of  $\varepsilon_1 > 0.258$ .

Figure 11 elaborates the investigation of effect contact rates  $a$  and  $b$  on  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \}$ . The figure represents that as the values of  $a$  and  $b$  increase, both  $\mathcal{R}_1$  and  $\mathcal{R}_2$  increase. To have the minimum values of  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \}$ , the value of  $a$  and  $b$  should be less than 0.19. As a result, we urge all relevant parties to pay close attention

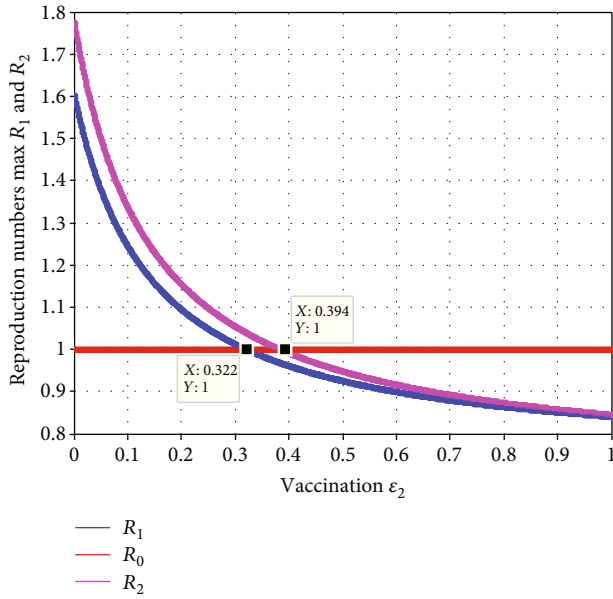


FIGURE 9: Effect of vaccination ( $\epsilon_2$ ) on reproduction numbers.

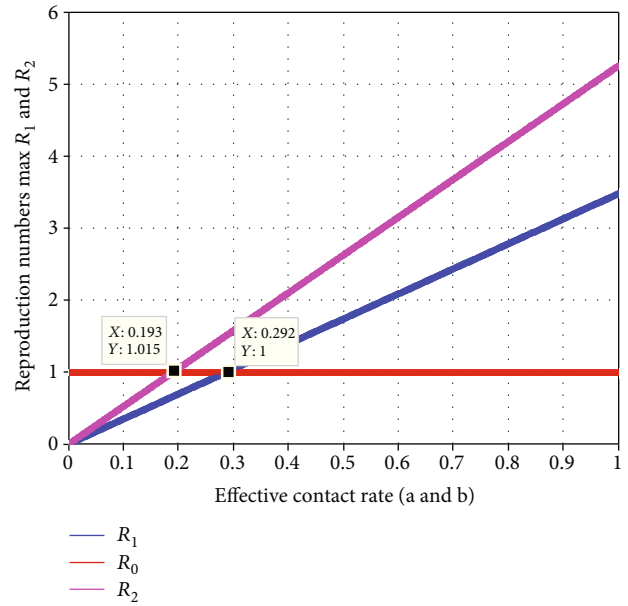


FIGURE 11: Effect of effective contact rate on reproduction numbers.

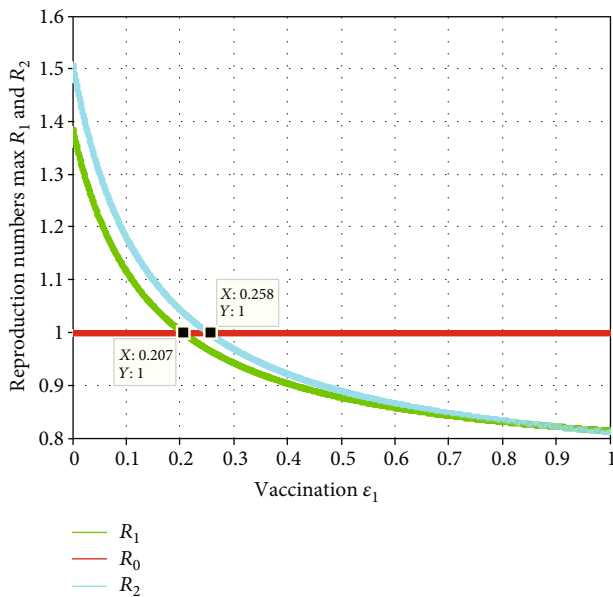


FIGURE 10: Effect of vaccination ( $\epsilon_1$ ) on reproduction numbers.

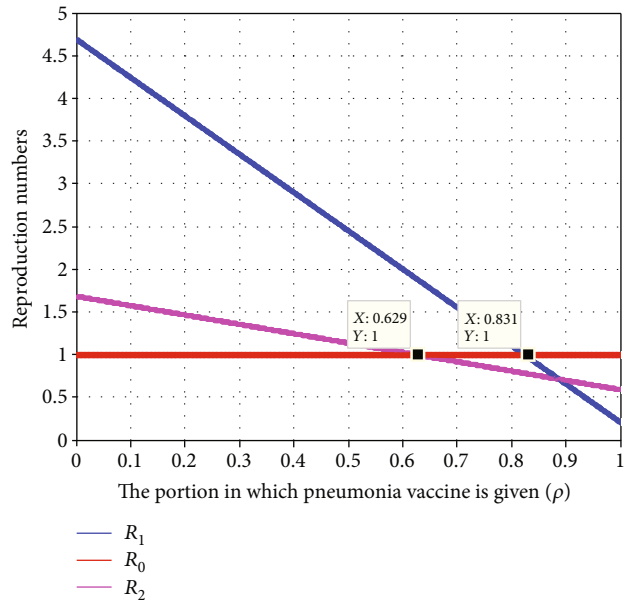


FIGURE 12: Effect of a portion of pneumonia vaccination on reproduction numbers.

to the effectiveness of the pneumonia vaccine in preventing the coinfection of meningitis and pneumonia in society.

In this subsection, as we see in Figure 12, we have investigated the effect of a portion of vaccination of pneumonia protection portion  $\rho$  on the effective reproduction number  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \}$ . The figure reflects that when the value of  $\rho$  increases, both  $\mathcal{R}_1$  and  $\mathcal{R}_2$  decrease, which implies that the maximum of them also declines. Moreover, when the value of  $\rho > 0.629$ , the value of  $\mathcal{R}_2$  is less than one; when the value of  $\rho > 0.831$ , the value of  $\mathcal{R}_1$  is less than one.

This suggests that if and only if the percentage of pneumonia vaccination rate is higher than 0.831, then  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \} < 1$ . Therefore, to avoid and regulate the

dynamic transmission of meningitis and pneumonia coinfection, public authorities must look to increase the gains of the pneumonia vaccination portion.

Figure 13 elaborates the investigation of the effect of pneumonia vaccination wanes ( $\phi$ ) on  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \}$ . The figure represents as the value of  $\phi$  increases, both  $\mathcal{R}_1$  and  $\mathcal{R}_2$  increase. To have the minimum values of  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \}$ , the value  $\phi$  should be less than 0.4. As a result, we urge all relevant parties to pay close attention to the effectiveness of the pneumonia vaccine in preventing the coinfection of meningitis and pneumonia in society.

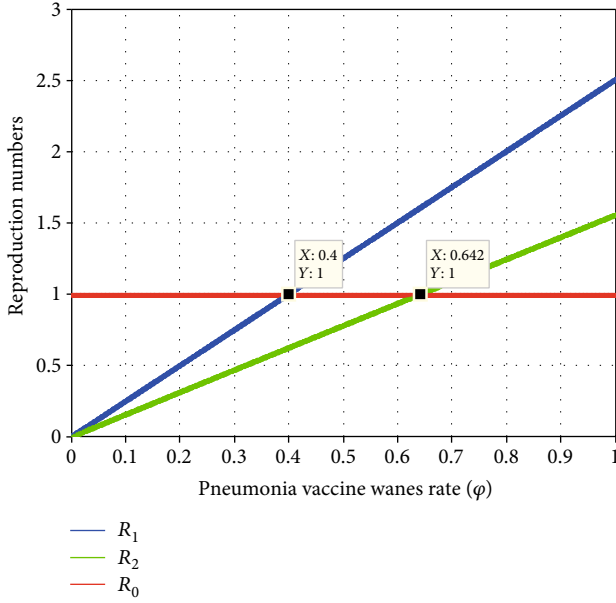


FIGURE 13: Effect of pneumonia vaccination wanes rate on reproduction numbers.

Figure 14 explains the investigation of the effect of meningitis vaccination wanes ( $\varphi$ ) on  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \}$ . The graph illustrates how both  $\mathcal{R}_1$  and  $\mathcal{R}_2$  grow when the value of  $\varphi$  rises. Our conclusion from this finding is that when the value of  $\varphi$  is smaller than 0.396, the minimal value of  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1, \mathcal{R}_2 \}$  is reached. The effectiveness of the meningitis vaccination in avoiding the coinfection of meningitis and pneumonia in society should thus be closely monitored, thus we encourage all pertinent parties to pay attention for it.

Analytic solution of some differential equations is complicated and tough. In such case, we need a numerical simulation of the system. In our case we have used a numerical simulation for stability analysis of steady state and obtained the result; after a year, the solutions of the meningitis and pneumonia coinfection dynamical system will tend to the endemic equilibrium point of the model. This expression of the plot illustrates the biological meaning of the proliferation and spread of meningitis and pneumonia coinfection which is regularly happening throughout the population of which is confined to a certain location. Additionally, we have used a numerical simulation to investigate the effects of a number of traits (parameters) that are related to the meningitis and pneumonia coinfection illness, such as, treatment rate, meningitis treatment rate, pneumonia treatment rate, effect of vaccination, and effective contact rate and vaccination wanes rate. As a result, we have shown that increasing availability to meningitis and pneumonia prevention, treatment, and meningitis and pneumonia coinfection vaccine has a significant influence on the frequency of meningitis and pneumonia in a particular population.

#### 4. Discussion

We addressed the epidemiology and historical context of meningitis and pneumonia in Section 1.

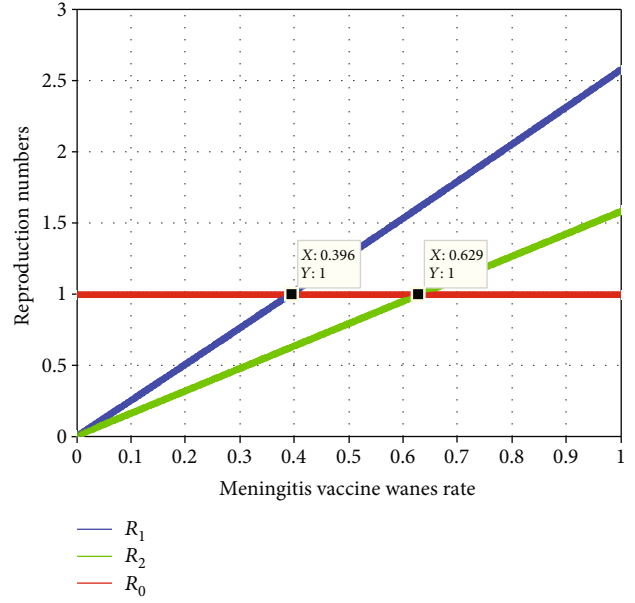


FIGURE 14: Effect of meningitis vaccination wanes rate on reproduction numbers.

In Section 2, the deterministic meningitis and pneumonia coinfection dynamical system was built by dividing the entire human population into nine groups based on the severity of the infection. Moreover, we looked at the model's descriptive phenomena, including the model's prospective solutions being positive, the dynamical system being bounded, the existence of a disease-free equilibrium point, the existence of an effective reproduction number using the next generation matrix technique, the existence of endemic equilibria, and the stability analysis of the disease-free equilibrium point using the Routh-Hurwitz criteria in this section. Additionally, the Routh-Hurwitz criteria were used to demonstrate the local stability of endemic equilibrium points of submodels, and we were able to conclude that these points are locally asymptotically stable if the reproduction number is less than one. Nevertheless, the endemic equilibrium point of the combined meningitis and pneumonia coinfection model is revealed by numerical simulation presented in Section 3 by Figure 3, i.e., the endemic equilibrium point is locally asymptotically stable just when  $a = 0.9$ ,  $b = 0.85$ , and  $\mathcal{R}_{eff} = \max \{ \mathcal{R}_1 = 12.33, \mathcal{R}_2 = 15.1 \} > 1$ .

Through the use of numerical simulation, we have examined the effects of several parameters on the effective reproduction rate as well as the prevention and control of the development of meningitis and pneumonia infections in Section 3. The results show that increasing availability to meningitis and pneumonia prevention, treatment, and meningitis and pneumonia coinfection vaccine has a significant influence on the frequency of meningitis and pneumonia in a particular population. The optimal control analysis of pneumonia and meningitis coinfection [8] having solely evaluated prevention for both meningitis and pneumonia, we found that preventative regulation had a significant influence on reducing the spread of meningitis, pneumonia, and their coinfection within the given time frame. Efforts to



avoid pneumonia and treat meningitis are two more techniques addressed in this study. They discovered that each of the stated measures is successful in reducing the growth of the infectious populations that only have meningitis, pneumonia, or both throughout the allotted time frame.

A mathematical model of seven nonlinear differential equations for the pneumonia and meningitis coinfection with PCV vaccination for a newly born population and treatment for coinfecting class has also been developed and entitled as “mathematical model analysis and numerical simulation for codynamics of meningitis and pneumonia infection with intervention” [5]. They have taken into account the PCV13 (pneumococcal conjugate vaccine), which is a vaccination given to infants and protects against a variety of pneumococcal bacteria that can cause the most severe forms of pneumococcal illness, such as pneumonia and meningitis. Additionally, researchers [7] have demonstrated that the availability of hospital beds, medicines, and other treatment options all contribute to a decline in the number of cases of bacterial meningitis. However, they observe that an increased recruitment rate causes a backward split in a town with few hospital beds.

Finally, they advise using vaccination when there is a large influx of new people. Numerous studies have demonstrated that meningitis and pneumonia infections may be wiped out in the community when treatment and immunization efforts are combined. Despite treatment interventions, these diseases and their combined forms continue to exist in the entire population. Three different vaccine combinations with similar treatments were not taken into account in any model across all studies. In contrast to the previous studies described above, the model developed in this study took into consideration all three vaccines, namely the meningitis vaccine, the pneumonia vaccine, and people who had received both the vaccine and therapy at the same time.

## 5. Conclusion

The study is aimed at building and examining a compartmental deterministic mathematical model of the dynamics of the spread of the coinfection of meningitis and pneumonia. In the population under the study, this model considered how vaccination and therapy of single infection of meningitis or pneumonia may reduce the risk of meningitis and pneumonia coinfection. When the effective reproduction number is smaller than unity, the model possesses a disease-free equilibrium point that is locally asymptotically stable. The outcome of the numerical simulation demonstrates that when  $\mathcal{R}_{eff} > 1$ , the meningitis and pneumonia coinfection model's endemic equilibrium point is locally asymptotically stable.

Besides obtaining the model's matching effective reproduction number  $\mathcal{R}_{eff}$ , we were able to identify the effects of changing specific parameter values and provide future guidance for the public's partners. Our findings have significant public health implications because they affect whether a disease is eradicated or persists in the community under investigation.

As a result of our numerical findings which show that  $\mathcal{R}_{eff} = 15.1$  at  $b = 0.85$  and  $a = 0.9$ , we notify public authorities to focus on raising or maximizing the values of the involved individuals' meningitis vaccination, pneumonia vaccination, and meningitis and pneumonia coinfection vaccination as well as treatment rates in order to reduce and eradicate the disease from the study's target community. Finally, other key results of this study are meningitis contact rates, pneumonia contact rates, and vaccination deficits of corresponding diseases that have contributed immensely to the spread of meningitis and pneumonia coinfection in the community.

## Data Availability

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

## Conflicts of Interest

The authors declare that they have no conflicts of interest

## Authors' Contributions

All authors have read and approved the final manuscript

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