https://doi.org/10.53656/nat2025-1.04

MATHEMATICAL MODELLING OF THE TRANSMISSION DYNAMICS OF PNEUMONIA AND MENINGITIS COINFECTION WITH VACCINATION

Deborah O. Daniel, Sefiu A. Onitilo, Omolade B. Benjamin, Ayoola A. Olasunkanmi
Olabisi Onabanjo University Ago Iwoye – Ogun State, Nigeria

Abstract. Pneumonia and meningitis pose substantial threats to global public health due to their high morbidity and mortality rates. This study investigates the dynamics of these diseases with a focus on coinfection and evaluates the effectiveness of vaccination as a control measure. Using a mathematical model, the transmission dynamics are explored and the basic reproduction number is derived to identify conditions for disease-free and endemic states. Numerical simulations analyze the impact of varying vaccination compliance levels, demonstrating that higher compliance significantly reduces the number of susceptible and infected individuals while increasing the vaccinated population. The study emphasizes the need for integrated public health strategies combining vaccination campaigns, efficient vaccine distribution, and supportive medical care to mitigate the burden of pneumonia and meningitis coinfections. These findings provide a framework for designing effective interventions aimed at reducing disease prevalence and improving public health outcomes.

Keywords: Pneumonia; Meningitis; Coinfection; Mathematical Modelling; Transmission Dynamics, Vaccination; Sensitivity Analysis

1. Introduction

Infectious diseases remain a persistent challenge for public health systems worldwide, causing substantial illness and death. Among these, pneumonia and meningitis are particularly concerning due to their prevalence and severe consequences. Pneumonia, primarily caused by pathogens such as *Streptococcus pneumoniae*, is a major cause of death, particularly in young children and older adults (McLuckie 2009). Meningitis, commonly caused by bacteria like *Neisseria meningitidis* and *Haemophilus influenzae*, is a life-threatening condition that affects the brain and spinal cord, often resulting in significant fatalities or long-term disabilities (Fresnadillo Martínez et al. 2013; Afolabi et al. 2021). Addressing these diseases is a key priority for reducing their burden on global health.

Pneumonia affects the respiratory system and is transmitted through airborne particles. It is responsible for millions of deaths annually, with *Streptococcus pneumoniae* being a leading cause (World Health Organization, 1991). This bacterium is also linked to other serious infections, including meningitis and sinusitis (Opatowski et al. 2013; Kotola et al. 2022). Meningitis primarily targets the central nervous system and is associated with high mortality and long-term neurological complications (Zunt et al. 2018; Tilahun 2019). When pneumonia and meningitis occur together, they pose significant challenges to clinical management and public health efforts. This combination increases the severity of illness and places additional strain on healthcare systems (Tabatabaei et al. 2022; Chukwu et al. 2020). Despite these challenges, the mechanisms of coinfection remain insufficiently studied, leaving gaps in our understanding of effective treatment and prevention strategies.

Coinfection, where two or more pathogens infect a person simultaneously, has significant implications for disease progression, transmission, and treatment outcomes (Kehr & Engelmann, 2015). The combined occurrence of pneumonia and meningitis is particularly concerning, as it may worsen patient outcomes and complicate medical interventions (Obi et al. 2010; Kotola & Mekonnen 2022). Understanding the interplay between these infections is critical for developing effective public health strategies and improving clinical outcomes.

Mathematical modeling plays a pivotal role in understanding and addressing infectious diseases by offering frameworks to simulate disease dynamics and assess intervention strategies (Tilahun et al. 2018; Asamoah et al. 2018). According to Bailey (1975), the primary goal of mathematical modeling in epidemiology is to support informed decision-making. Models help evaluate the most cost-effective approaches to minimize the adverse impacts of diseases (Di Liddo 2016; Kizito & Tumwiine 2018). Researchers have developed models for pneumonia and meningitis individually, enhancing our understanding of their spread and informing control measures (Blyuss 2016; Joseph 2012; Musa et al. 2020). Pneumonia, for instance, has been the subject of extensive modeling efforts due to its significant global burden (McLuckie 2009; Joseph 2012). Tilahun (2019) utilized the SIR model to explore the co-dynamics of pneumonia and meningitis, contributing valuable insights into their interactions.

The application of mathematical models extends to evaluating vaccination and treatment strategies. For example, Tilahun et al. (2017) developed an SVCIR model to investigate cost-effective control strategies for pneumonia, concluding that a combination of prevention and treatment yields the most significant impact. Similarly, Onyinge et al. (2016) formulated models to analyze pneumonia coinfection with HIV/AIDS, highlighting the importance of combined intervention strategies. These efforts underscore the utility of mathematical frameworks in optimizing resource allocation for disease management.

In the case of meningitis, mathematical modeling has been instrumental in understanding its epidemiology and guiding control efforts. Meningitis remains a

severe global health threat, particularly in resource-limited settings (Zunt et al. 2018; Ghia & Rambhad 2021; Abdullahi Baba et al. 2020). Outbreaks of bacterial meningitis, often caused by pathogens like *Streptococcus pneumoniae* and *Neisseria meningitidis*, demand urgent interventions (Van De Beek et al. 2010; Scarborough & Thwaites, 2008; Türkün et al. 2023). Models have been used to predict outbreak dynamics and assess the impact of vaccination programs (Jayaraman et al. 2018; Oordt-Speets et al. 2018; Kotola et al. 2022). For instance, the introduction of vaccines targeting *Haemophilus influenzae* type b and *Streptococcus pneumoniae* has significantly reduced the incidence of bacterial meningitis in high-income countries, though the burden persists in low- and middle-income regions (McIntyre et al. 2012; Peter et al. 2021).

Despite these advances, the combined dynamics of pneumonia and meningitis remain underexplored, particularly in the context of vaccination. Few studies have addressed their coinfection using mathematical models. Tilahun (2019) developed a model incorporating both diseases and emphasized the need for integrated approaches to improve intervention outcomes. Such efforts are essential for addressing the complexities of coinfection, where interactions between diseases may amplify severity and complicate treatment (Kotola & Mekonnen 2022; Chukwu et al. 2020). Still, studies on their combined dynamics remain limited, and existing models often fail to capture the combined effects of these diseases or explore the role of vaccination in reducing their impact. This highlights the need for a comprehensive approach to modeling pneumonia-meningitis coinfection, particularly in resource-constrained settings. This study seeks to address these gaps by constructing a mathematical model to examine the dynamics of pneumonia-meningitis coinfection considering the impact of vaccination.

2. Mathematical Formulation

In this study, we present a deterministic mathematical model designed to capture the transmission dynamics of pneumonia and meningitis coinfection within a population. The model divides the population into seven distinct compartments. These compartments include susceptible individuals (denoted as S), who are healthy but can be infected by pneumonia, meningitis, or both; vaccinated individuals (V), who have received protection through vaccination; pneumonia-infected individuals (Ip), who are infected solely with pneumonia and can transmit the disease to others; meningitis-infected individuals (Im), who are infected solely with meningitis; coinfected individuals (Ipm), who are infected by both pneumonia and meningitis; and recovered individuals (R), representing those who have recovered from pneumonia, meningitis, or both diseases, or those who have removed themselves from the transmission cycle through vaccination.

The total population at any time t is the sum of all these compartments, given by $N(t) = S(t) + I_p(t) + I_m(t) + I_{pm}(t) + R(t) + V(t)$.

The dynamics of each compartment are governed by a system of differential equations that describe how individuals move between compartments due to factors such as infection, vaccination, recovery, and death. The rate of change for each compartment is determined by various transmission, recovery, and death rates.

The susceptible population, denoted by *S(t)*, is influenced by recruitment at a rate Λ , and it is impacted by the force of infection due to contact with infected individuals, which is represented by the transmission rates σ_1, σ_2 and σ_3 for pneumonia, meningitis, and coinfection, respectively. The susceptible compartment also decreases due to natural deaths at a rate μ, as well as due to recovery from either pneumonia or meningitis. Furthermore, there is a transfer from the vaccinated group to the susceptible group, modeled by the rate χ, as vaccination immunity wanes. This is balanced by a natural recruitment rate of new susceptible individuals, with vaccination and recovery contributing to the pool of new individuals who might be susceptible again at rate θ.

The vaccinated individuals, represented by *V(t)*, are recruited from the susceptible population through vaccination, but they are also susceptible to infection at a reduced rate due to the effectiveness of the vaccine. The vaccination rate is affected by the contact with infected individuals as well as the natural death rate. Additionally, some vaccinated individuals may become susceptible again if immunity wanes, which is modeled by the transfer rate χ .

The compartment for pneumonia-infected individuals, $I_p(t)$, represents those infected only with pneumonia. These individuals become infected through contact with susceptible individuals or those vaccinated but not fully protected. The rate of change of this group is influenced by the transmission rate for pneumonia, σ_1, as well as the rate of recovery, denoted by γ_1, and the death rate due to pneumonia complications, represented by μ. These individuals either recover or die from pneumonia, and they may transition to the recovered state at a rate δ_1, which reflects the rate of recovery from pneumonia.

Similarly, the meningitis-infected individuals, $I_m(t)$, represent those infected solely with meningitis. The rate of change of this compartment depends on the transmission rate σ_2 and the interaction between susceptible and vaccinated individuals. These individuals also recover from meningitis at a rate γ_2 and may transition to the recovered state at a rate δ_2 , while some individuals may die from meningitis at a rate μ.

The coinfected individuals, $I_{pm}(t)$, represent those who are infected with both pneumonia and meningitis. The dynamics of this group are influenced by the transmission rates of both pneumonia and meningitis. The rate of change in this compartment depends on the interaction between the pneumonia and meningitis infected individuals, and the recovery rate from coinfection is denoted by γ_3. Coinfected individuals also transition to the recovered state at a rate defined by δ_1 and δ_2, and some may die due to the complications of having both infections.

The recovered individuals, *R(t)*, represent those who have recovered from pneumonia, meningitis, or coinfection. Their rate of recovery is governed by the recovery rates γ_1, γ_2 and γ_3, corresponding to pneumonia, meningitis, and coinfection recovery, respectively. However, these individuals can be removed from the recovered group due to natural deaths or loss of immunity, as modeled by the natural death rate μ and the immunity loss rate θ.

The system of differential equations describing these dynamics is given by:

$$\begin{aligned}
\frac{dS}{dt} &= (1-\kappa)\Lambda - (\sigma_1 I_p + \sigma_2 I_m + \sigma_3 I_{pm})S + \theta R - (\zeta + \mu)S + \chi V,\\
\frac{dV}{dt} &= \kappa\Lambda + \zeta S - \phi(\sigma_1 I_p + \sigma_2 I_m + \sigma_3 I_{pm})V - (\chi + \mu)V,\\
\frac{dI_p}{dt} &= (\sigma_1 I_p + \sigma_1 I_{pm})(S + \phi V) - (\sigma_4 + \gamma_1 + \delta_1 + \mu)I_p,\\
\frac{dI_m}{dt} &= (\sigma_2 I_m + \sigma_2 I_{pm})(S + \phi V) - (\sigma_5 + \gamma_2 + \delta_2 + \mu)I_m,\\
\frac{dI_{pm}}{dt} &= \sigma_4 I_p + \sigma_5 I_m - (\delta_1 + \delta_2 + \gamma_3 + \mu)I_{pm},\\
\frac{dR}{dt} &= \gamma_1 I_p + \gamma_2 I_m + \gamma_3 I_{pm} - (\theta + \mu)R.
\end{aligned} \tag{1}$$

where the parameters are defined as follows:

$(\sigma_3 = \sigma_1 + \sigma_2, \sigma_4 = \sigma_2 (I_m + I_{pm}), \sigma_5 = \sigma_1 (I_p + I_{pm})$

The mathematical model proposed in this study aims to capture the transmission dynamics of pneumonia and meningitis coinfection, with a particular focus on the role of vaccination as a public health intervention. To keep the model tractable while still maintaining its ability to reflect real-world scenarios, several assumptions have been made which include the following:

The population is homogeneous, with equal contact rates between individuals.

Vaccination is modeled as a constant rate, affecting the overall population without individual-level variation.

No cross-protection is assumed between pneumonia and meningitis.

Vaccine effectiveness is constant across the entire population.

These assumptions balance the need for simplicity and computational feasibility with the necessity of accurately representing the underlying epidemiological processes.

2.1. Qualitative Analysis

2.1.1. Invariant Region

The invariant region defines the domain where the solutions of the coinfection model are both biologically and mathematically valid. It is essential to demonstrate that the region Ω, where the model is feasible, remains positively invariant for all $t > 0$. This ensures that the system described by equation (1) is well-posed.

Theorem 1. *The region* $\Omega = \{(S, V, I_p, I_m, I_{pm}, R) \in \mathbb{R}^6_+ : \mathcal{N}(t) \leq \frac{\Lambda}{\mu}\}$ *is positively invariant.*

Proof. The total population at any time t is defined as:

$$\mathcal{N}(t) = S(t) + V(t) + I_p(t) + I_m(t) + I_{pm}(t) + R(t).$$

The rate of change of the total population is given by:

$$\frac{d\mathcal{N}}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dI_p}{dt} + \frac{dI_m}{dt} + \frac{dI_{pm}}{dt} + \frac{dR}{dt}.$$

Substituting from equation (1), we get:

$$\frac{d\mathcal{N}}{dt} = \Lambda - \mu\mathcal{N}.$$

Integrating this differential equation yields:

$$\mathcal{N}(t) \leq \frac{\Lambda}{\mu} + Ce^{-\mu t},$$

where C is a constant of integration.

As $t \to \infty$, the total population $\mathcal{N}(t)$ approaches:

$$\mathcal{N}(t) \leq \frac{\Lambda}{\mu}.$$

Hence, the feasible region for the solution of the system is within Ω, making the region positively invariant and ensuring that the model is biologically meaningful. □

2.1.2. Positivity of the Solutions

The positivity of the solutions ensures that all model variables remain non-negative over time. Since the coinfection model represents human populations, it is assumed that all variables and parameters are positive for $t \geq 0$.

Theorem 2. *Given initial conditions* $S(0), V(0), I_p(0), I_m(0), I_{pm}(0), R(0) > 0$*, the solutions of the system (1) remain positive for all* $t \geq 0$.

Proof. Starting with the first equation in the system (1):

$$\frac{dS}{dt} = (1-\kappa)\Lambda - (\sigma_1 I_p + \sigma_2 I_m + \sigma_3 I_{pm})S + \theta R - (\zeta + \mu)S.$$

Rewriting, we have:

$$\frac{dS}{dt} \geq -(\sigma_1 I_p + \sigma_2 I_m + \sigma_3 I_{pm})S - (\zeta + \mu)S.$$

Using separation of variables and integrating:

$$S(t) = S(0)e^{-(\sigma_1 I_p + \sigma_2 I_m + \sigma_3 I_{pm} + \zeta + \mu)t}.$$

Since $S(0) > 0$, it follows that $S(t) > 0$ for all $t \geq 0$.

A similar approach can be used for $V(t)$, $I_p(t)$, $I_m(t)$, $I_{pm}(t)$, and $R(t)$, showing that all state variables remain positive over time.

Thus, the solutions of the system are positive for all $t \geq 0$. □

2.2. Disease-Free Equilibrium

The disease-free equilibrium (DFE) of a coinfection model represents the steady-state solutions when no infections are present in the population. Let E_0 denote the disease-free equilibrium. Setting $I_p(t)$, $I_m(t)$, and $I_{pm}(t)$, to zero in equation (1) and solving for $S(t)$, we obtain:

$$E_0 = (S_0, V_0, 0,0,0,0),$$

where

$$S_0 = \frac{(1-\kappa)\Lambda}{\zeta + \mu}, V_0 = \frac{(\kappa\Lambda + \zeta S_0)}{\chi + \mu}.$$

2.2.1. The Effective Reproduction Number

The effective reproduction number, R_E, is a critical threshold that determines whether an infection can invade and persist in a population in the presence of intervention. For this coinfection model, R_E is calculated as the dominant eigenvalue of the next-generation matrix, defined as:

$$R_E = \rho(FV^{-1}),$$

where ρ is the spectral radius of the matrix product FV^{-1}. The matrices F and V are defined as follows:

$$F = \begin{pmatrix} \sigma_1(S_0 + \phi V_0) & 0 & \sigma_1(S_0 + \phi V_0) \\ 0 & \sigma_2(S_0 + V_0) & \sigma_2(S_0 + V_0) \\ 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} (\gamma_1 + \delta_1 + \mu) & 0 & 0 \\ 0 & (\gamma_2 + \delta_2 + \mu) & 0 \\ 0 & 0 & (\delta_1 + \delta_2 + \gamma_3 + \mu) \end{pmatrix}.$$

The next-generation matrix is given by:

$$FV^{-1} = \begin{pmatrix} \frac{\sigma_1(S_0+\phi V_0)}{\gamma_1+\delta_1+\mu} & 0 & \frac{\sigma_1(S_0+\phi V_0)}{\delta_1+\delta_2+\gamma_3+\mu} \\ 0 & \frac{\sigma_2(S_0+V_0)}{\gamma_2+\delta_2+\mu} & \frac{\sigma_2(S_0+V_0)}{\delta_1+\delta_2+\gamma_3+\mu} \\ 0 & 0 & 0 \end{pmatrix}.$$

The effective reproduction numbers for pneumonia and meningitis are:

$$R_p = \frac{\sigma_1((\kappa\mu+\zeta)\Lambda\phi + (1-\kappa)(\chi+\mu)\Lambda)}{(\zeta+\mu)(\chi+\mu)(\gamma_1+\delta_1+\mu)},$$

$$R_m = \frac{\sigma_2((\kappa\mu+\zeta)\Lambda\phi + (1-\kappa)(\chi+\mu)\Lambda)}{(\zeta+\mu)(\chi+\mu)(\gamma_2+\delta_2+\mu)}.$$

Thus, the overall reproduction number is:
$R_E = \max\{R_p, R_m\}$.

2.2.2. Local Stability of the Disease-Free Equilibrium

Theorem 3. *The disease-free equilibrium (DFE) of the system (1) is locally asymptotically stable if* $R_E < 1$ *and unstable if* $R_E > 1$.

Proof. The Jacobian matrix at the DFE is given by:

$$J(S,V,I_p,I_m,I_{pm},R) = \begin{pmatrix} -(\zeta+\mu) & \chi & \sigma_1 S_0 & \sigma_2 S_0 & \sigma_3 S_0 & \theta \\ \zeta & -(\chi+\mu) & \phi\sigma_1 V_0 & \phi\sigma_2 V_0 & \phi\sigma_3 V_0 & 0 \\ 0 & 0 & -(\gamma_1+\delta_1+\mu) & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\gamma_2+\delta_2+\mu) & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\delta_1+\delta_2+\gamma_3+\mu) & 0 \\ 0 & 0 & \gamma_1 & \gamma_2 & \gamma_3 & -(\theta+\mu) \end{pmatrix}.$$

The eigenvalues of this matrix include:

$$-(\gamma_1+\delta_1+\mu), -(\gamma_2+\delta_2+\mu), -(\delta_1+\delta_2+\gamma_3+\mu), -(\theta+\mu).$$

The remaining eigenvalues are determined from the submatrix:

$$\begin{vmatrix} -(\zeta+\mu)-\lambda & \chi \\ \zeta & -(\chi+\mu)-\lambda \end{vmatrix} = 0.$$

Solving, we find:

$$\lambda^2 + \lambda(\zeta+\chi+2\mu) + (\zeta+\mu)(\chi+\mu) - \zeta\chi = 0.$$

Since all coefficients are positive, the roots of the characteristic equation have negative real parts, ensuring local stability when $R_E < 1$.□

2.2.3. Global Stability of the Disease-Free Equilibrium

Theorem 4. *The disease-free equilibrium point is globally asymptotically stable*

if $R_E < 1$. Otherwise unstable.

Proof. Let F and V denote the Jacobian matrices for the new infection rates F_i and net transition rates $V_i = V_i^- - V_i^+$, evaluated at the disease-free equilibrium x_0:

$$F = \left(\frac{\partial F_i(x_0)}{\partial x_j}\right), V = \left(\frac{\partial V_i(x_0)}{\partial x_j}\right), 1 \le i, j \le n.$$

The dynamics of the infected compartments (I_p, I_m, I_{pm}) are bounded by:

$$\frac{d}{dt}\begin{pmatrix} I_p \\ I_m \\ I_{pm} \end{pmatrix} \le (F - V)\begin{pmatrix} I_p \\ I_m \\ I_{pm} \end{pmatrix},$$

where F captures new infections and V transitions between compartments, with:

$$F = \begin{pmatrix} \frac{\beta_1(1-\rho)\Gamma[\psi + d + \alpha(\rho+\epsilon)]}{(\epsilon+d)(\psi+d)} & 0 & \frac{\beta_1(1-\rho)\Gamma[\psi + d + \alpha(\rho d+\epsilon)]}{(\epsilon+d)(\psi+d)} \\ 0 & \beta_2(S_0 + \alpha V_0) & \frac{\beta_2(1-\rho)\Gamma[\psi + d + \alpha(\rho d+\epsilon)]}{(\epsilon+d)(\psi+d)} \\ 0 & 0 & 0 \end{pmatrix}, V$$

$$= \text{diag}(\eta_1 + k_1 + d, \eta_2 + k_2 + d, \eta_3 + k_3 + d).$$

When the reproduction numbers R_p and R_m are below unity, all eigenvalues of $(F - V)$ have negative real parts. By the comparison principle:

$$(I_p, I_m, I_{pm}) \to (0,0,0) \text{ as } t \to \infty.$$

Consequently, the susceptible population $S(t)$ approaches the steady state:

$$S(t) \to \frac{(1-\rho)\Gamma}{\epsilon + d}.$$

Thus, the disease-free equilibrium E_0 is globally asymptotically stable when $R_p < 1$ and $R_m < 1$.□

2.3. The Endemic Equilibria

The endemic equilibria represent the steady states where one or both infections persist in the population. We examine three cases: the pneumonia endemic equilibrium, the meningitis endemic equilibrium, and the coexistence equilibrium.

2.3.1. Pneumonia Endemic Equilibrium

The pneumonia endemic equilibrium occurs when pneumonia persists in the population ($I_p \neq 0$), but meningitis infection is absent ($I_m = 0$). Substituting $I_m = 0$ into system (1) and solving, the system reduces to:

$$(1-\kappa)\Lambda - \sigma_1 I_p S + \theta R - (\zeta+\mu)S + \chi V = 0,$$
$$\kappa\Lambda + \zeta S - \phi\sigma_1 I_p V - (\chi+\mu)V = 0,$$
$$\sigma_1 I_p (S + \phi V) - (\gamma_1 + \delta_1 + \mu) I_p = 0,$$
$$\gamma_1 I_p - (\theta+\mu)R = 0.$$

The pneumonia endemic equilibrium is given by $E_p = (S^p, V^p, I_p^p, 0, 0, R^p)$, where:

$$S^p = \frac{(1-\kappa)\Lambda + \theta R^p}{\sigma_1 I_p^p + \zeta + \mu},$$

$$V^p = \frac{\kappa\Lambda + \zeta S^p}{\phi\sigma_1 I_p^p + \chi + \mu},$$

$$R^p = \frac{\gamma_1 I_p^p}{\theta + \mu}.$$

Here, I_p^p is obtained numerically or analytically by solving:

$$\sigma_1 I_p^p (S^p + \phi V^p) = (\gamma_1 + \delta_1 + \mu) I_p^p.$$

2.3.2. Meningitis Endemic Equilibrium

The meningitis endemic equilibrium occurs when meningitis persists in the population ($I_m \neq 0$), but pneumonia infection is absent ($I_p = 0$). Substituting $I_p = 0$ into system (1), the system reduces to:

$$(1-\kappa)\Lambda - \sigma_2 I_m S + \theta R - (\zeta+\mu)S + \chi V = 0,$$
$$\kappa\Lambda + \zeta S - \phi\sigma_2 I_m V - (\chi+\mu)V = 0,$$
$$\sigma_2 I_m (S + \phi V) - (\gamma_2 + \delta_2 + \mu) I_m = 0,$$
$$\gamma_2 I_m - (\theta+\mu)R = 0.$$

The meningitis endemic equilibrium is given by $E_m = (S^m, V^m, 0, I_m^m, 0, R^m)$, where:

$$S^m = \frac{(1-\kappa)\Lambda + \theta R^m}{\sigma_2 I_m^m + \zeta + \mu},$$

$$V^m = \frac{\kappa\Lambda + \zeta S^m}{\phi\sigma_2 I_m^m + \chi + \mu},$$

$$R^m = \frac{\gamma_2 I_m^m}{\theta + \mu}.$$

Here, I_m^m is obtained numerically or analytically by solving:

$$\sigma_2 I_m^m (S^m + \phi V^m) = (\gamma_2 + \delta_2 + \mu) I_m^m.$$

2.3.3. Coexistence Equilibrium

The coexistence equilibrium occurs when both pneumonia and meningitis persist in the population ($I_p \neq 0$, $I_m \neq 0$). Solving system (1) with all compartments non-zero, the equilibrium is given by $E_{mp} = (S^*, V^*, I_p^*, I_m^*, I_{pm}^*, R^*)$, where:

$$S^* = \frac{\Lambda - (\sigma_1 I_p^* + \sigma_2 I_m^* + \sigma_3 I_{pm}^*) S^* + \theta R^*}{\zeta + \mu},$$

$$V^* = \frac{\kappa\Lambda + \zeta S^* - \phi(\sigma_1 I_p^* + \sigma_2 I_m^* + \sigma_3 I_{pm}^*) V^*}{\chi + \mu},$$

$$R^* = \frac{\gamma_1 I_p^* + \gamma_2 I_m^* + \gamma_3 I_{pm}^*}{\theta + \mu}.$$

Here, I_p^*, I_m^*, and I_{pm}^* are obtained by solving the nonlinear system numerically or analytically, incorporating interaction terms between pneumonia and meningitis.

2.4. Sensitivity Analysis

In the study of epidemiology, it is critical to identify the factors that significantly contribute to disease transmission and prevalence. Sensitivity analysis serves as an essential method for assessing how variations in parameters influence the dynamics of a disease, enabling the development of targeted strategies to reduce both morbidity and mortality.

To evaluate the impact of parameters on the outcomes of the model, sensitivity indices are employed. These indices provide a measure of how a relative change in a parameter translates into a relative change in a state variable. The normalized forward sensitivity index is particularly useful in this context, as it quantifies the proportional effect of parameter variations on a given variable.

If the state variable is a differentiable function of the parameter, the sensitivity index can be calculated using partial derivatives. Specifically, the normalized forward sensitivity index of a variable η with respect to a parameter Ω is defined as:

$$\Upsilon_\Omega^\eta = \frac{\partial \eta}{\partial \Omega} \times \frac{\Omega}{\eta},$$

where Ω represents any of the fundamental parameters in the model. This formulation allows researchers to systematically determine the relative importance of different parameters, providing valuable insights into the dynamics and control of the disease.

3. Numerical Simulations

This section presents the numerical simulation of the pneumonia-meningitis co-infection model, utilizing the baseline parameter values outlined in Table 1.

Simulations were performed and visualized over time (in days) using MATLAB, with the resulting plots displayed in Figures 1 – 6.

The initial conditions for the pneumonia and meningitis coinfection model are obtained as follows: The initial number of susceptible individuals is taken to be $S(0) = 2000$. The initial number of vaccinated individuals is $V(0) = 250$. The initial number of individuals infected with pneumonia is $I_p(0) = 500$. The initial number of individuals infected with meningitis is $I_m(0) = 700$. The initial number of individuals coinfected with both pneumonia and meningitis is $I_{pm}(0) = 280$. The initial number of recovered individuals is $R(0) = 200$.

Table 1. Parameters Description and Values Used in the Simulation Model

Parameters	Description	Value	Source
ϕ	Proportion not covered by the vaccine	0.002	Tilahun *et al.* (2017)
θ	Rate of immunity loss among recovered individuals	0.0241/day	Kizito and Tumwiine (2018)
χ	Waning rate	0.0025/day	Tilahun *et al.* (2017)
σ_1	Contact rate: pneumonia-infected and susceptible	0.007 – 0.6	Konstatin (2016)
σ_2	Contact rate: meningitis-infected and susceptible	0.9	Fresnadillo *et al.* (2013)
δ_1	Disease-induced death rate: pneumonia	0.006 – 0.5	Tilahun (2019)
δ_2	Disease-induced death rate: meningitis	0.002 – 0.2	Tilahun (2019)
γ_1	Recovery rate: pneumonia	0.9	Tilahun (2019)
γ_2	Recovery rate: meningitis	0.8	Tilahun (2019)
γ_3	Recovery rate: coinfection	0.1	Tilahun *et al.* (2017)
κ	Proportion vaccinated against one or both diseases	0.02	Swai *et al.* (2021)
ζ	Vaccination rate	0.3	Swai *et al.* (2021)
μ	Natural death rate	4.566e-6	Tilahun *et al.* (2017)
Λ	Birth rate	150	Assumed

The numerical simulation of the effect of vaccination on the population dynamics is analysed using the baseline values given in Table 1. The numerical simulation are done and plotted against time (days) using MATLAB and the results are

shown in Figures 1 – 6. Figures 1 – 6 illustrates the impact of varying levels of vaccination effectiveness on the dynamics of a pneumonia-meningitis coinfection model over a period of 150 days. The figure compares scenarios with no vaccination, slightly effective vaccination (30% compliance), moderately effective vaccination (50% compliance), and highly effective vaccination (100% compliance). It demonstrates how these different levels of vaccination compliance affect the number of susceptible individuals (Figure 1), vaccinated individuals (Figure 2), pneumonia-infected individuals (Figure 3), meningitis-infected individuals (Figure 4), coinfected individuals (Figure 5), and recovered individuals (Figure 6). As shown in Figure 1, the number of susceptible individuals decreases over time across all levels of vaccination effectiveness. Without any vaccination (denoted by dots), the decline in susceptible individuals is the least steep. The highly effective strategy (100% compliance) demonstrates the steepest decline. However, slightly effective (30% compliance) and moderately effective (50% compliance) strategies also show significant reductions, indicating a gradient effect of vaccination effectiveness on reducing the number of susceptible individuals.

Figure 2 displays the increase in vaccinated individuals over time. The graph shows that with higher compliance to vaccination (30%, 50%, and 100% effectiveness), the number of vaccinated individuals increases significantly compared to no vaccination. The highly effective strategy (100% compliance) results in the highest number of vaccinated individuals. Interestingly, the slightly effective strategy shows a quicker initial increase compared to the moderately effective strategy. This might be due to a higher initial vaccination uptake in the slightly effective scenario, which could be attributed to increased public awareness or access to vaccines.

As displayed in Figure 3, the number of pneumonia-infected individuals decreases over time. The graph shows that with no vaccination (denoted by dots), the reduction is the slowest. Slightly effective (30% compliance) and moderately effective (50% compliance) vaccination strategies result in a more significant decrease, but the highly effective strategy (100% compliance) shows the most substantial reduction in pneumonia infections. The slightly effective strategy appears to reduce infections quicker than the moderately effective one. This could be due to initial variations in how the vaccine impacts different population subsets or logistical differences in vaccine distribution and uptake.

Figure 4 shows the trends for meningitis-infected individuals. Similar to pneumonia, the graph demonstrates a decrease in meningitis infections over time. The reduction is least significant without vaccination. The slightly effective strategy initially performs better than the moderately effective one, which might be due to similar reasons as discussed for pneumonia: initial vaccine impact variations and differences in vaccine distribution logistics. The highly effective strategy (100% compliance) leads to the most considerable decline in meningitis infections.

In Figure 5, the number of coinfected individuals (infected with both pneumonia and meningitis) increases over time across all strategies. The rate of increase is highest without any vaccination (denoted by dots) and progressively decreases with more effective vaccination strategies. The highly effective strategy (100% compliance) shows the slowest increase. Interestingly, the slightly effective strategy shows a lower rate of increase compared to the moderately effective strategy at certain points, potentially due to initial population dynamics and vaccine distribution efficiency.

Figure 6 depicts the number of recovered individuals over time. The number of recoveries is lowest with no vaccination and increases with the effectiveness of the vaccination strategy. However, the difference in the number of recoveries among the slightly effective, moderately effective, and highly effective strategies is minimal. This small difference indicates that while vaccination does contribute to recovery, its impact on this specific outcome is less pronounced compared to its effect on reducing the number of susceptible and infected individuals.

The slightly effective strategy shows better initial performance in some graphs due to variations in how different population subsets respond to the vaccine. Some individuals might have a quicker immune response, leading to an initial rapid decline in infections or an increase in recoveries. Differences in how the vaccine is distributed and administered can impact initial results. Efficient distribution in the slightly effective scenario might lead to quicker initial results compared to a more evenly distributed but slower uptake in the moderately effective scenario. Higher public awareness or better access to vaccines in the slightly effective scenario might result in a quicker initial response, leading to better performance in the early stages. The small difference in the number of recovered individuals among the different vaccination strategies suggests that other factors, such as natural recovery rates and the effectiveness of medical treatments, play a significant role in recovery. To enhance the impact of vaccination on recovery rates, it might be necessary to combine vaccination with improved medical treatments and supportive care. Improving the logistics of vaccine distribution to ensure a more uniform and efficient rollout can help achieve better initial results across all effectiveness levels. By addressing any logistical challenges, we can ensure that vaccines reach the population more quickly and evenly. Implementing campaigns to raise awareness about the importance of vaccination can increase uptake and compliance, improving overall effectiveness. These campaigns can educate the public on the benefits of vaccination and encourage more people to get vaccinated. Regularly monitoring how different population subsets respond to the vaccine can help adjust strategies to maximize effectiveness. This monitoring can identify which groups are responding well and which may need additional support or different strategies. To enhance the impact of vaccination on recovery rates, it is important to also focus on improving medical treatments and supportive care for infected individuals. Combin-

ing vaccination with better medical care can ensure that those who contract the disease receive the best possible treatment, further reducing the overall impact of the infection. Despite some initial better performance in the slightly effective strategy, the highly effective vaccination strategy (100% compliance) remains the best approach in the long run. It results in the steepest decline in susceptible and infected individuals and the highest number of vaccinated individuals. While the impact on recovered individuals is less pronounced, achieving high compliance with vaccination significantly improves public health outcomes by reducing infection rates and increasing recovery in combination with effective medical care.

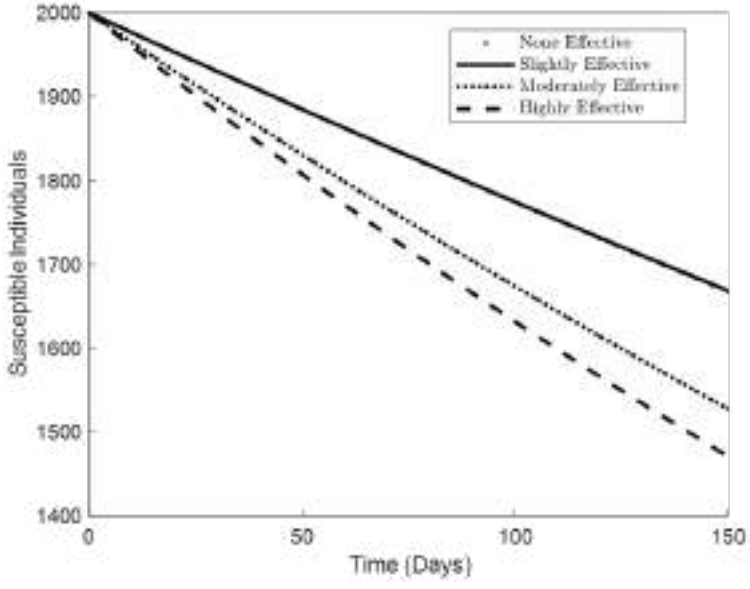

Figure 1. Simulation effect of vaccination on the susceptible individuals

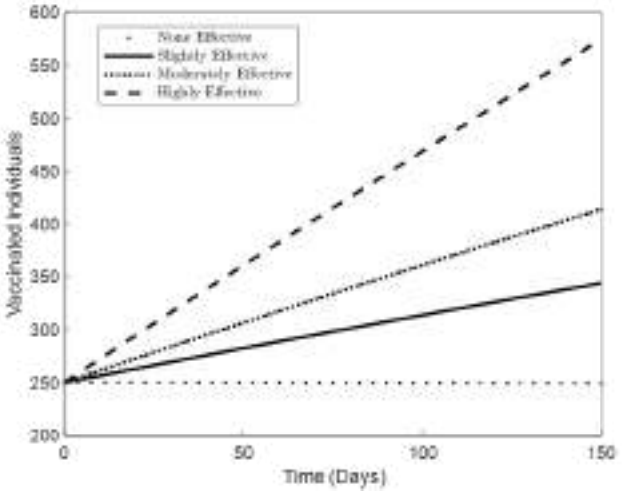

Figure 2. Simulation effect of vaccination on the vaccinated individuals

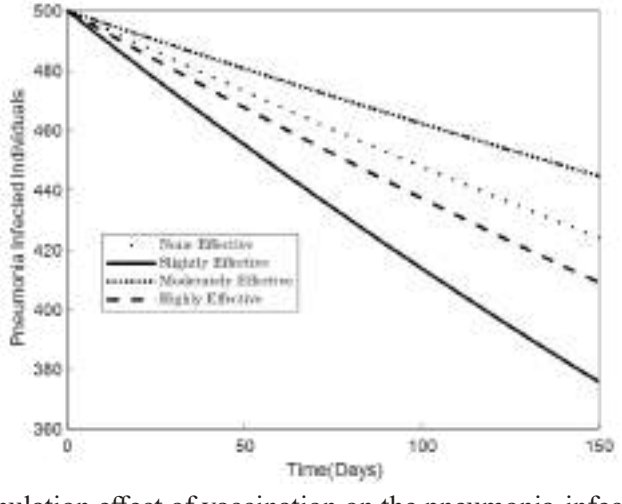

Figure 3. Simulation effect of vaccination on the pneumonia-infected individuals

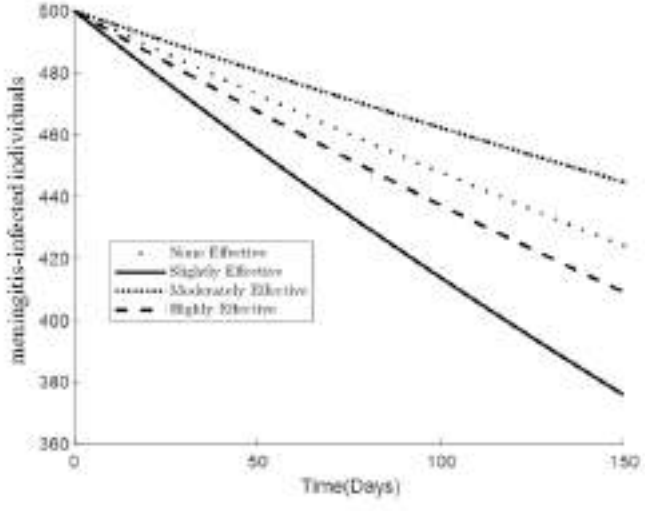

Figure 4. Simulation effect of vaccination on the meningitis-infected individuals

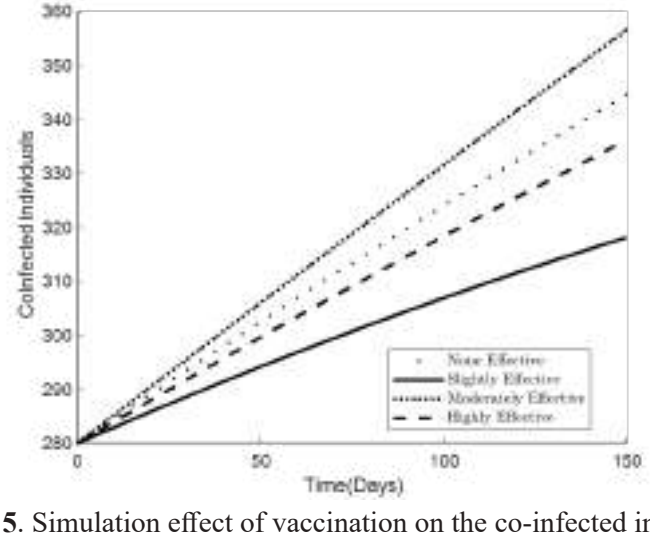

Figure 5. Simulation effect of vaccination on the co-infected individuals

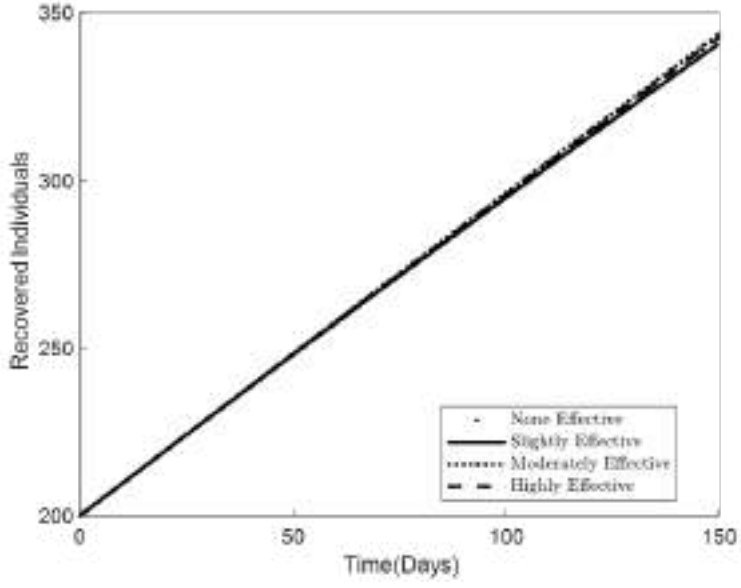

Figure 6. Simulation effect of vaccination on the recovered individuals

3.1. Sensitivity Index

The sensitivity indices of the basic reproduction numbers are summarized in Tables 2 and 3. Parameters with positive sensitivity indices in Tables 2 and 3 indicate that increasing their values, while keeping all other parameters constant, leads to an increase in R_{0p} and R_{0m}, respectively. These parameters play a significant role in driving the spread of pneumonia and meningitis infections.

On the other hand, parameters with negative sensitivity indices in Tables 2 and 3 suggest that increasing their values, while holding other parameters fixed, results in a decrease in R_{0p} and R_{0m}, respectively. These parameters contribute to mitigating the transmission of the diseases.

Thus, the key parameters for effective disease control include δ_1, δ_2, and ζ, which have negative sensitivity indices. Parameters such as σ_1, σ_2, and Λ exhibit the highest sensitivity with positive indices, emphasizing their importance in disease propagation. Although μ has a negative sensitivity index, biologically, increasing its value is recommended as it aids in disease control.

Table 2. Value of Sensitivity Indices of R_{0p}

Parameters	Sensitivity Index
σ_1	+1
μ	-0.10207
γ_1	$+4.5 \times 10^{-12}$
δ_1	-1
χ	-0.00168
ϕ	+0.0020103
Λ	+1
ζ	-0.89625
κ	-0.01996

Table 3. Value of Sensitivity Indices of R_{0m}

Parameters	Sensitivity Index
σ_2	+1
μ	-0.10207
γ_2	$+3.96 \times 10^{-12}$
δ_2	-1
χ	-0.00168
ϕ	+0.0020103
Λ	+1
ζ	-0.89625
κ	-0.01996

4. Conclusion

This study presents a compartmental deterministic mathematical model of the dynamics of pneumonia and meningitis coinfection. It highlights the important

role of vaccination in controlling the spread of both diseases within a population. Our analysis demonstrates that varying levels of vaccination compliance can notably reduce the number of susceptible and infected individuals, with higher vaccination compliance leading to the most considerable reductions. Although the effect of vaccination on recovery rates is less pronounced, the combination of high vaccination coverage and improved medical care offers significant public health benefits.

The findings emphasize the need to address logistical challenges, increase public awareness, and improve vaccine accessibility to maximize the effectiveness of vaccination programs. While slightly effective strategies may show initial success, the long-term impact is most pronounced with highly effective vaccination coverage. Therefore, to optimize public health outcomes, it is crucial to expand vaccination campaigns, enhance distribution logistics, and integrate vaccination with improved medical treatments. Regular monitoring and adjustments to vaccination strategies, based on population responses, will further enhance the effectiveness of disease control measures.

While the model does not explicitly capture variations in immune responses or the logistics of vaccine distribution, it simplifies the vaccine impact by assuming that vaccination reduces susceptibility in proportion to the coverage level. The vaccine's effectiveness is modeled as a constant factor in the transition dynamics between compartments, reflecting an overall reduction in susceptibility and infection. Further refinements to the model could include the influence of varying immune responses or more detailed vaccine administration strategies, but these aspects were not included in the current study.

REFERENCES

AFOLABI, M. A., ADEWOYE, K. S., FOLORUNSO, A. I., ET AL., 2021. A mathematical model on transmission dynamics of meningococcal meningitis. *Iconic Research and Engineering Journals*, vol. 4, no. 10, pp. 59 – 66.

ASAMOAH, J.K.K., NYABADZA, F., SEIDU, B. et al., 2018. Mathematical modelling of bacterial meningitis transmission dynamics with control measures. *Computational and Mathematical Methods in Medicine*. https://doi.org/10.1155/2018/2657461.

BAILEY, N. T. J., 1975. *The Mathematical Theory of Infectious Diseases and Its Applications* (2nd ed.). Charles Griffin & Company Ltd.

BLYUSS, K. B., 2016. Mathematical modelling of the dynamics of meningococcal meningitis in Africa. In: P. ASTON, A. MULHOLLAND, K. TANT (Eds.). *UK Success Stories in Industrial Mathematics*, pp. 221–226. Springer, Cham.

CHUKWU, C. W., MUSHANYU, J., JUGA, M. L., ET AL., 2020. A mathematical model for co-dynamics of listeriosis and bacterial meningitis diseases. *Communications in Mathematical Biology and Neuroscience*, pp. 1 – 25.

DI LIDDO, A., 2016. Optimal control and treatment of infectious diseases: The case of huge treatment costs. *Mathematics*, vol. 4, no. 2, p. 21.

FRESNADILLO MARTÍNEZ, M.J., GARCÍA MERINO, E., GARCÍA SÁNCHEZ, E. ET AL., 2013. A mathematical model to study the meningococcal meningitis. *Procedia Computer Science*, vol. 18, pp. 2492 – 2495.

GHIA, C. J., & RAMBHAD, G. S., 2021. A systematic literature review on the prevalence and etiology of meningitis among critically ill and hospitalized patients in India. *Therapeutic Advances in Infectious Disease*, vol. 8. https://doi.org/10.1177/20499361211046453.

JAYARAMAN, Y., VEERARAGHAVAN, B., CHETHRAPILLY PURUSHOTHAMAN, G. K., ET AL., 2018. Burden of bacterial meningitis in India: Preliminary data from a hospital-based sentinel surveillance network. *PLoS One*, vol. 13, no. 5, p. e0197198.

JOSEPH, E., 2012. *Mathematical Analysis of Prevention and Control Strategies of Pneumonia in Adults and Children*. (Doctoral dissertation). University of Dar es Salaam.

KIZITO, D., & TUMWIINE, J., 2018. A mathematical model of treatment and vaccination interventions of pneumococcal pneumonia. *Journal of Applied Mathematics*. https://doi.org/10.1155/2018/2539465.

KOTOLA, B. S., & MEKONNEN, T. T., 2022. Mathematical model analysis and numerical simulation for codynamics of meningitis and pneumonia infection with intervention. *Scientific Reports*, vol. 12, pp. 1 – 18.

KOTOLA, B. S., GEBRU, D. M., & ALEMNEH H. T., 2022. Appraisal and simulation on codynamics of pneumonia and meningitis with vaccination intervention. *Computational and Mathematical Methods in Medicine*, pp. 1 – 20.

KEHR, J., & ENGELMANN, L., 2015. Double trouble: Towards an epistemology of co-infection. *Medicine, Anthropology, Theory*, vol. 2, no. 1, pp. 1 – 31.

MCINTYRE, P. B., O'BRIEN, K. L., GREENWOOD, B., & VAN DE BEEK, D., 2012. Effect of vaccines on bacterial meningitis worldwide. *The Lancet*, vol. 380, no. 9854, pp. 1703 – 1711.

MCLUCKIE, A., 2009. *Respiratory Disease and Its Management*. Springer Science & Business Media.

MUSA, S. S., ZHAO, S., H., NAFIU, H., ET AL., 2020. Mathematical modeling and analysis of meningococcal meningitis transmission dynamics. *International Journal of Biomathematics*, vol. 13, no. 1, p. 2050006.

ONYINGE, D., ONGATI, N., & ODUNDO, F., 2016. Mathematical model for co-infection of pneumonia and HIV/AIDS with treatment. *International Journal of Science, Engineering and Applied Sciences*, vol. 2, no. 1, pp. 106 – 111.

OORDT-SPEETS, A. M., BOLIJN, R., VAN HOORN, R. C., ET AL., 2018. Global etiology of bacterial meningitis: A systematic review and meta-analysis. *PLoS One*, vol. 13, no. 6, p. e0198772.

OPATOWSKI, L., VARON, E., DUPONT, C., ET AL., 2013. Assessing pneumococcal meningitis association with viral respiratory infections and antibiotics: insights from statistical and mathematical models. *Proceedings of the Royal Society B*, vol. 280, p. 20130519.

PETER, O.J., YUSUF, A., OSHINUBI, K. ET AL., 2021. Fractional order of pneumococcal pneumonia infection model with Caputo Fabrizio operator. Results in Physics, vol. 29, p. 104581.

SCARBOROUGH, M., & THWAITES, G. E., 2008. The diagnosis and management of acute bacterial meningitis in resource-poor settings. *The Lancet Neurology*, vol. 7, no. 7, pp. 637 – 648.

TABATABAEI, S. R., SHAMSHIRI, A., AZIMI, L., ET AL., 2022. Co-infection with dual *Streptococcus pneumoniae* serotypes as a cause of pediatric bacterial meningitis in Iran: A multi-center cross-sectional study. *BMC Infectious Diseases*, vol. 22, no. 1, p. 625.

TILAHUN, G. T., 2019. Modeling co-dynamics of pneumonia and meningitis diseases. *Advances in Difference Equations*, pp. 1 – 18.

TILAHUN, G. T., MAKINDE, O. D., & MALONZA, D., 2017. Modeling and optimal control of pneumonia disease with cost-effective strategies. *Journal of Biological Dynamics*, vol. 11, no. 2, pp. 400 – 426.

TÜRKÜN, C., GÖLGELI, M. & ATAY, F.M., 2023. A mathematical interpretation for outbreaks of bacterial meningitis under time-dependent transmission parameters. *Nonlinear Dynamics*, vol. 111, p. 14467 – 14484.

VAN DE BEEK, D., FARRAR, J. J., GANS, J., ET AL., 2010. Adjunctive dexamethasone in bacterial meningitis: A meta-analysis of individual patient data. *The Lancet Neurology*, vol. 9, no. 3, pp. 254 – 263.

WORLD HEALTH ORGANIZATION, 1991. *Technical Bases for the WHO Recommendations on the Management of Pneumonia in Children at First-Level Health Facilities*. World Health Organization.

ZUNT, J. R., KASSEBAUM, N. J., BLAKE, N., ET AL., 2018. Global, regional, and national burden of meningitis, 1990–2016: A systematic analysis for the global burden of disease study 2016. *The Lancet Neurology*, vol. 17, no. 12, pp. 1061 – 1082

✉ **Dr. Deborah O. Daniel**
ORCID iD: 0000-0003-4025-1448
Olabisi Onabanjo University
Ago-Iwoye, Ogun State, Nigeria
E-mail: oludeboradaniel@gmail.com

✉ **Sefiu A. Onitilo**
ORCID iD: 0000-0002-4418-197X
Olabisi Onabanjo University
Ago Iwoye, Ogun State, Nigeria
E-mail: onitilo.sefiu@oouagoiwoye.edu.ng

✉ **Omolade B. Benjamin**
ORCID iD: 0009-0004-0665-5558
Olabisi Onabanjo University
Ago Iwoye, Ogun State, Nigeria
E-mail: omoladebabajide342@gmali.com

✉ **Ayoola A. Olasunkanmi**
ORCID iD: 0009-0006-3530-5391
Olabisi Onabanjo University
Ago Iwoye, Ogun State, Nigeria
E-mail: ayoolaolasunkanmi243@mail.com