



Modeling the transmission dynamics of meningococcal meningitis under hospitalization effects

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Abstract

Meningococcal meningitis remains a major public health concern owing to its rapid transmission, severe health consequences, and potential to cause recurrent outbreaks. In this study,

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a deterministic compartmental model is developed to investigate the transmission dynamics of meningococcal meningitis and assess the impact of hospitalization on disease spread. The total population is partitioned into five epidemiological classes: susceptible (S), exposed (E), infectious (I), hospitalized (H), and recovered (R). The proposed model is shown to be epidemiologically and mathematically well-posed based on the positivity and boundedness of its solutions. The disease-free and endemic equilibrium points are derived, and the basic reproduction number, $\mathcal{R}_0$, is obtained using the next-generation matrix method. It is proved that the disease-free equilibrium is locally and globally asymptotically stable whenever $\mathcal{R}_0 < 1$, whereas the unique endemic equilibrium is locally and globally asymptotically stable whenever $\mathcal{R}_0 > 1$. Furthermore, bifurcation analysis based on the center manifold theory of Castillo-Chavez and Song establishes that the model undergoes a forward (supercritical) bifurcation at the critical threshold $\mathcal{R}_0 = 1$, implying that reducing the basic reproduction number below unity is sufficient for disease eradication. Sensitivity analysis identified the transmission, hospitalization, and recovery rates as the most influential parameters governing disease transmission. Numerical simulations using MATLAB® supported the analytical results and demonstrated that timely hospitalization, improved recovery, and reduced transmission substantially decreased the number of infectious individuals and shortened the outbreak duration. These findings provide quantitative insights into meningococcal meningitis transmission and offer guidance for designing effective interventions and disease control strategies.

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1 Introduction

Meningococcal infection has been acknowledged as a severe issue for nearly 200 years. When an outbreak raged across Geneva in 1805, Vieusseux described meningococcal meningitis for the first time [23]. In 1887, *Neisseria meningitidis* was identified as the causative agent of meningococcal disease. Approximately 10% of people in the general population carry meningococcal germs in their nose and throat without experiencing any adverse consequences. Meningococci can occasionally evade the body's defenses, resulting in life-threatening illnesses. It causes fulminant meningococemia and meningococcal meningitis, two potentially fatal illnesses that frequently coexist. *Neisseria meningitidis* continues to be a major cause of meningitis and deadly sepsis, despite the availability of effective medicines and partially successful vaccinations. Meningitis is an inflammatory reaction to bacterial infection of the subarachnoid space and leptomeninges (pia-arachnoid). Meningococemia with petechiae can be caused by bacteria growing in the

bloodstream or seeding local sites such as the pericardium, joints, or meninges. In as many as 50% of instances, it can result in multi-organ failure, severe disability, or death [16, 27]. Adults with fever and maculopapular rash have a wide range of potential diagnoses. The differential diagnosis encompasses viral and bacterial infections and hypersensitivity reactions, such as those triggered by drugs. Some of these eruptions constitute medical emergencies, whereas others are benign [22].

One of the riskiest side effects of meningococcal meningitis is Waterhouse–Friderichsen syndrome. It is believed to be a coagulopathy that causes hemorrhagic necrosis of the adrenal glands, triggered by bacterial endotoxins. Petechial hemorrhages in the skin and mucous membranes are the defining features of this condition. Purpuric rashes can be caused by immune-mediated disorders, platelet dysfunction, vascular injury, or disseminated intravascular coagulation [13]. The fatality rate from meningococcal meningitis ranges from 5% to 15%, and if treatment is not received, it may increase to 50%. Nine percent of patients with invasive meningococcal illness have long-lasting sequelae, and more than one-third of patients survive but face significant problems [7, 8, 24].

Few studies have focused on creating mathematical models to examine the spread of meningococcal meningitis. They are all based on differential equations and have some limitations, such as not accounting for the unique characteristics of populations, not simulating the behavior of each individual, and not being suitable for computational implementation. Mathematical models of the dynamics of disease transmission are essential for better understanding disease behavior, optimizing the use of scarce resources, and suggesting control methods for infectious diseases. Decisions regarding intervention techniques to prevent and control the resurgence of meningitis and pneumonia are more successful when guided by models specifically designed for these diseases. Given the emergence and resurgence of diseases, mathematical modeling is an efficient and appealing field for estimating the effects of control measures and the underlying parameters of real-world phenomena that are difficult or costly to obtain through experimentation [11, 9, 20, 19].

Mathematical modeling serves as a vital framework for analyzing the spread and control of bacterial meningitis within a population, enabling researchers to understand transmission pathways and evaluate intervention measures such as vaccination and contact reduction [28, 1, 4, 25, 14, 12, 15, 35]. These models provide insights into the dynamics of *Neisseria meningitidis* infections, helping to predict epidemic patterns, assess the impact of immunization programs such as the MACV vaccine, and inform public health decision-making [34, 21]. Furthermore, optimal control and sensitivity analyses within these models support the identification of effective strategies to minimize infection rates and mitigate the disease burden in the affected regions [2, 32]. In this study, Anderson and May [3] analyzed the relationship between infection and human

populations using epidemiological data and mathematical models to support the implementation of public-health interventions. Karachaliou et al. [17] developed a mathematical model of MenA transmission, predicting effective short-term control after the introduction of MenAfriVac but a possible resurgence without booster vaccinations. In addition, Keeling and Rohani [18] presented comprehensive infectious disease models to predict, understand, and manage outbreaks in humans and animals using actual data and simulation tools.

Furthermore, Trotter [29] reported that the predominance of *Neisseria meningitidis* serogroups varies geographically, with conjugate vaccines, especially for serogroup C, significantly reducing the disease incidence in developed nations. They further suggested that global control could be achieved through serogroup B vaccines in developed countries and serogroup A conjugates in Africa. Vereen [31] developed a meningitis model incorporating a carrier class into the SIR framework to assess vaccination effects in epidemic-prone regions. Their analysis demonstrated that higher transmission rates require proportionally higher vaccination rates, as supported by MATLAB simulations using WHO parameters. In addition, the WHO [33] highlighted that *Neisseria meningitidis* serogroups A, B, and C cause most meningococcal cases, with increasing outbreaks of Y and W135. Vaccination using conjugate or polysaccharide vaccines targeting specific serogroups remains the most effective approach for preventing and controlling these epidemics. Recently, [30] identified four major demographic trends shaping the world: population growth, aging, urbanization, and international migration. The report projects that the global population will likely peak before 2100 owing to declining birth rates, emphasizing how these demographic shifts affect sustainability, gender equality, and poverty reduction, and offering policy recommendations to help nations prepare for future population changes.

If not treated promptly, meningococcal meningitis, an acute bacterial infection affecting the meninges, can rapidly lead to severe complications, hospitalization, and death. The disease spreads mainly through close contact and respiratory droplets, with both symptomatic and pre-symptomatic individuals contributing to the transmission. In this study, we develop a novel mathematical model to characterize the transmission dynamics of meningococcal meningitis. The remainder of this paper is organized as follows: Section 2 presents the model formulation, description, and corresponding system of ODEs. Section 3 discusses the basic qualitative analysis of the proposed model. Section 4 focuses on the key theoretical aspects, including the computation of the basic reproduction number ($\mathcal{R}_0$) and identification of disease-free and endemic equilibrium points. Section 5 examines the stability analysis and backward bifurcation, and Section 6 explores the sensitivity analysis of the proposed model. Section 7 provides the numerical simulations and discussion of the results, and Section 8 concludes the study with future research directions.

2 Formulation of the meningococcal meningitis model with hospitalization

In this section, a deterministic compartmental model is formulated to describe the transmission dynamics of meningococcal meningitis in the presence of hospitalization. The model consists of a system of nonlinear ordinary differential equations that captures the progression of individuals through different epidemiological stages and examines the effects of hospitalization, recovery, disease-induced mortality, and waning immunity on disease transmission.

The total living population at time t , denoted by $N(t)$, is partitioned into five mutually exclusive epidemiological classes: susceptible $S(t)$, exposed $E(t)$, infectious $I(t)$, hospitalized $H(t)$, and recovered $R(t)$. Disease-induced deaths are incorporated through the mortality rates of infectious and hospitalized individuals and were therefore not represented as a separate compartment.

The model is developed under the following assumptions:

- The susceptible population is replenished through logistic recruitment with intrinsic recruitment rate b and carrying capacity K .
- Disease transmission occurs through effective contact between susceptible individuals and both exposed and infectious individuals. The exposed class contributes to disease transmission at a reduced level, measured by the modification parameter σ_1 .
- Exposed individuals progress to the infectious class at a constant rate θ .
- Infectious individuals may be hospitalized at rate α , recover naturally at rate γ_1 , or die due to disease-induced complications at rate δ_1 .
- Hospitalized individuals either recover following treatment at rate γ_2 or die due to disease-induced complications at rate δ_2 .
- Recovered individuals lose temporary immunity at rate ψ and return to the susceptible class, thereby allowing reinfection.
- All living compartments experience natural mortality at the constant rate μ .

2.1 Description of the model

The susceptible population increases through logistic recruitment represented by

$$bN \left(1 - \frac{N}{K}\right),$$

where b denotes the intrinsic recruitment rate, and K is the carrying capacity of the population. Susceptible individuals acquire infection through effective contact with infectious and exposed individuals at the force of infection

$$\lambda = \beta \frac{I + \sigma_1 E}{N},$$

where β denotes the effective transmission coefficient, and σ_1 measures the relative infectiousness of exposed individuals compared to infectious individuals. Following infection, exposed individuals progress to the infectious class at the rate θ . Infectious individuals may be hospitalized at rate α , recover naturally at rate γ_1 , or die due to disease-induced complications at rate δ_1 . Hospitalized individuals recover under medical care at a rate γ_2 or die from disease-induced complications at a rate δ_2 . Individuals in the recovered class lose temporary immunity at the rate ψ and re-enter the susceptible class, thereby contributing to reinfection. In addition, all living compartments are subject to natural mortality at a constant rate μ . The interactions among the epidemiological classes are illustrated in the compartmental flow diagram in Figure 1.

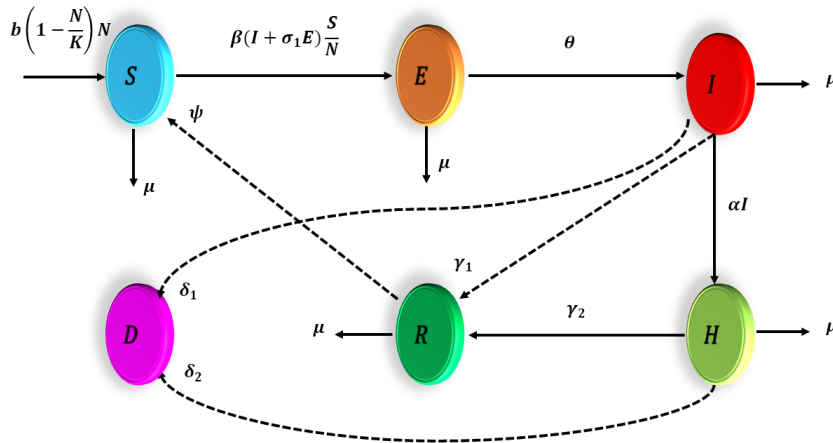


Figure 1: Transmission dynamics of the meningococcal meningitis model

2.2 Model equations

Given these assumptions, the mathematical formulation of the meningococcal meningitis model with hospitalization is presented below:

$$\begin{aligned}
\frac{dS}{dt} &= bN \left(1 - \frac{N}{K} \right) - \beta(I + \sigma_1 E) \frac{S}{N} - \mu S + \psi R, \\
\frac{dE}{dt} &= \beta(I + \sigma_1 E) \frac{S}{N} - (\theta + \mu) E, \\
\frac{dI}{dt} &= \theta E - (\delta_1 + \mu + \gamma_1 + \alpha) I, \\
\frac{dH}{dt} &= \alpha I - (\mu + \delta_2 + \gamma_2) H, \\
\frac{dR}{dt} &= \gamma_1 I + \gamma_2 H - (\mu + \psi) R, \\
\frac{dD}{dt} &= \delta_1 I + \delta_2 H,
\end{aligned} \tag{1}$$

where $N(t) = S(t) + E(t) + I(t) + H(t) + R(t)$ denotes the total population.

The corresponding initial conditions are

$$S(0) > 0, \quad E(0) \geq 0, \quad I(0) \geq 0, \quad H(0) \geq 0, \quad R(0) \geq 0, \quad D(0) \geq 0.$$

Table 1 summarizes the compartments utilized in the proposed model (1), and Table 2 lists the model parameters, along with their corresponding descriptions and numerical values.

Table 1: Meningococcal meningitis model variables

Variable	Description
$S(t)$	Population of susceptible individuals
$E(t)$	Population of exposed individuals
$I(t)$	Population of infectious individuals
$H(t)$	Population of hospitalized individuals
$R(t)$	Population of recovered individuals
$D(t)$	Population of deceased individuals

Table 2: Proposed model parameters, descriptions, and their numerical values

Parameter	Description	Numerical Value	Source
b	Intrinsic per capita recruitment rate	0.02–0.05	[30]
K	Carrying capacity of the system	Normalized to 1	Assumed
μ	Natural death rate	0.000039	[29]
β	Transmission rate of the disease	0.3–1.5	[31]
σ_1	Meningitis exposed coefficient	0.2–0.85	[4]
θ	Rate of progression from exposed to infectious	0.1–0.52	[29]
γ_1	Recovery rate of infected individuals	0.05–0.2	[3]
γ_2	Recovery rate of hospitalized individuals	0.03–0.1	[18]
δ_1	Induced death rate of infected individuals	0.005–0.05	[33]
δ_2	Induced death rate of hospitalized individuals	0.01–0.10	[33]
α	Hospitalization or isolation rate	0–1	Assumed
ψ	Rate of loss of immunity ($R \rightarrow S$)	0.04–2	[17]

3 Qualitative analysis

3.1 Nonnegativity of solutions

Theorem 1. Let

$$(S(0), E(0), I(0), H(0), R(0), D(0)) \in \mathbb{R}_+^6,$$

where $\mathbb{R}_+^6 = \{(S, E, I, H, R, D) : S, E, I, H, R, D \geq 0\}$. Then, the solutions of system (1) remain nonnegative for all $t \geq 0$. In other words, the nonnegative orthant $\mathbb{R}_+^6$ is positively invariant under the flow generated by system (1).

Proof. To establish positivity, it suffices to show that the vector field associated with system (1) points inward on each boundary hyperplane of the nonnegative orthant.

For $S = 0$, we have

$$\left. \frac{dS}{dt} \right|_{S=0} = bN \left(1 - \frac{N}{K} \right) + \psi R \geq 0.$$

Similarly, for $E = 0, I = 0, H = 0, R = 0, D = 0$, it follows that

$$\begin{aligned}
\left. \frac{dE}{dt} \right|_{E=0} &= \beta \frac{SI}{N} \geq 0, \\
\left. \frac{dI}{dt} \right|_{I=0} &= \theta E \geq 0, \\
\left. \frac{dH}{dt} \right|_{H=0} &= \alpha I \geq 0, \\
\left. \frac{dR}{dt} \right|_{R=0} &= \gamma_1 I + \gamma_2 H \geq 0, \\
\left. \frac{dD}{dt} \right|_{D=0} &= \delta_1 I + \delta_2 H \geq 0.
\end{aligned}$$

Thus, on every boundary component of $\mathbb{R}_+^6$, the corresponding derivative is nonnegative. Hence, the vector field does not point outward across any coordinate hyperplane. By the forward invariance theorem (also known as Nagumo's Theorem), the nonnegative orthant $\mathbb{R}_+^6$ is positively invariant. Therefore, any solution of system (1) that starts in $\mathbb{R}_+^6$ remains in $\mathbb{R}_+^6$ for all $t \geq 0$. Consequently, $S(t), E(t), I(t), H(t), R(t), D(t) \geq 0$, for $t \geq 0$. $\square$

3.2 Boundedness

Theorem 2. The solutions of system (1) are bounded in the feasible region

$$\Omega = \left\{ (S, E, I, H, R, D) \in \mathbb{R}_+^6 : N(t) \leq \frac{K(b - \mu)}{b} \right\}.$$

Proof. Let $N(t) = S(t) + E(t) + I(t) + H(t) + R(t)$ denotes the total living population. Summing the first five equations of system (1) yields

$$\frac{dN}{dt} = bN \left(1 - \frac{N}{K} \right) - \mu N - \delta_1 I - \delta_2 H.$$

Since $\delta_1 I + \delta_2 H \geq 0$, it follows that

$$\frac{dN}{dt} \leq bN \left(1 - \frac{N}{K} \right) - \mu N.$$

Hence

$$\frac{dN}{dt} \leq (b - \mu)N - \frac{b}{K}N^2.$$

Consider the auxiliary logistic equation

$$\frac{dy}{dt} = (b - \mu)y - \frac{b}{K}y^2, \quad y(0) = N(0).$$

Its positive equilibrium is $y^* = \frac{K(b-\mu)}{b}$. By the comparison theorem, $N(t) \leq y(t)$, $t \geq 0$. Since every solution of the logistic equation is bounded above by its carrying capacity y^* , we obtain

$$N(t) \leq \frac{K(b-\mu)}{b}, \quad t \rightarrow \infty.$$

Consequently,

$$0 \leq N(t) \leq \max \left\{ N(0), \frac{K(b-\mu)}{b} \right\}, \quad t \geq 0.$$

Therefore, all state variables $S(t), E(t), I(t), H(t), R(t)$ are bounded for all $t \geq 0$, and the region

$$\Omega = \left\{ (S, E, I, H, R, D) \in \mathbb{R}_+^6 : N(t) \leq \frac{K(b-\mu)}{b} \right\},$$

is positively invariant. Hence, the solutions of system (1) remain bounded for all times. $\square$

4 Threshold analysis of the model

In this section, the threshold dynamics of the proposed model are investigated by determining the disease-free equilibrium (DFE), basic reproduction number, and endemic equilibrium. The conditions for the existence and stability of these equilibria are subsequently established.

4.1 Disease-free equilibrium

The DFE of system (1) corresponds to the steady state at which meningococcal meningitis is absent from the population. Setting $E = I = H = R = 0$ in system (1), the susceptible equation becomes

$$0 = bN \left(1 - \frac{N}{K} \right) - \mu S.$$

Since $N = S$ at the DFE, we have

$$0 = bS \left(1 - \frac{S}{K} \right) - \mu S.$$

Factoring out S yields

$$S \left[b \left(1 - \frac{S}{K} \right) - \mu \right] = 0.$$

The trivial solution $S = 0$ is thus biologically irrelevant. Hence, the biologically feasible DFE is given by

$$S^* = K \left(1 - \frac{\mu}{b} \right), \quad b > \mu.$$

Therefore, the DFE of system (1) is

$$\mathcal{E}_0 = \left(K \left(1 - \frac{\mu}{b} \right), 0, 0, 0, 0 \right).$$

4.2 Basic reproduction number

The basic reproduction number, denoted by $\mathcal{R}_0$, is defined as the average number of secondary infections generated by a single infectious individual introduced into a completely susceptible population. Following the next-generation matrix approach of [26, 10, 5], we consider the infected-state vector

$$\mathbf{x} = (E, I)^T.$$

The vectors of new infection and transition terms are

$$F(\mathbf{x}) = \begin{pmatrix} \beta(\sigma_1 E + I) \\ 0 \end{pmatrix}, \quad V(\mathbf{x}) = \begin{pmatrix} (\theta + \mu)E \\ -\theta E + dI \end{pmatrix},$$

where $d = \alpha + \delta_1 + \gamma_1 + \mu$. The corresponding Jacobian matrices evaluated at the DFE are

$$F = \begin{pmatrix} \beta\sigma_1 & \beta \\ 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \theta + \mu & 0 \\ -\theta & d \end{pmatrix}.$$

The inverse of V is $V^{-1} = \frac{1}{(\theta + \mu)d} \begin{pmatrix} d & 0 \\ \theta & \theta + \mu \end{pmatrix}$. Hence,

$$FV^{-1} = \frac{1}{(\theta + \mu)d} \begin{pmatrix} \beta(\sigma_1 d + \theta) & \beta(\theta + \mu) \\ 0 & 0 \end{pmatrix}.$$

Therefore, the basic reproduction number is the spectral radius of the next-generation matrix, that is,

$$\begin{aligned}\mathcal{R}_0 &= \rho(FV^{-1}) \\ &= \frac{\beta [\theta + \sigma_1(\alpha + \delta_1 + \gamma_1 + \mu)]}{(\theta + \mu)(\alpha + \delta_1 + \gamma_1 + \mu)}.\end{aligned}\quad (2)$$

The threshold quantity $\mathcal{R}_0$ determines the disease transmission dynamics. In particular, meningococcal meningitis can invade the population if $\mathcal{R}_0 > 1$, whereas the disease eventually dies out when $\mathcal{R}_0 < 1$.

4.3 Existence of the endemic equilibrium

Theorem 3. System (1) admits a unique endemic equilibrium

$$\mathcal{E}^{**} = (S^{**}, E^{**}, I^{**}, H^{**}, R^{**}),$$

if and only if $\mathcal{R}_0 > 1$.

Proof. Let $\mathcal{E}^{**} = (S^{**}, E^{**}, I^{**}, H^{**}, R^{**})$ denote an arbitrary endemic equilibrium of system (1), where $N^{**} = S^{**} + E^{**} + I^{**} + H^{**} + R^{**}$.

Define the equilibrium force of infection by

$$\lambda^{**} = \frac{\beta(I^{**} + \sigma_1 E^{**})}{N^{**}}. \quad (3)$$

Setting the right-hand side of system (1) equal to zero and solving successively yields

$$\begin{aligned}E^{**} &= \frac{\lambda^{**} S^{**}}{k_1}, \\ I^{**} &= \frac{\theta \lambda^{**} S^{**}}{k_1 d}, \\ H^{**} &= \frac{\alpha \theta \lambda^{**} S^{**}}{k_1 d k_3}, \\ R^{**} &= \left(\frac{\gamma_1 \theta}{k_1 d k_4} + \frac{\alpha \theta \gamma_2}{k_1 d k_3 k_4} \right) \lambda^{**} S^{**},\end{aligned}\quad (4)$$

where $k_1 = \theta + \mu$, $d = \alpha + \delta_1 + \gamma_1 + \mu$, $k_3 = \delta_2 + \gamma_2 + \mu$, $k_4 = \mu + \psi$. Substituting (4) into $N^{**} = S^{**} + E^{**} + I^{**} + H^{**} + R^{**}$, gives

$$N^{**} = S^{**} (1 + L_2 \lambda^{**}), \quad (5)$$

where $L_2 = \frac{1}{k_1} + \frac{\theta}{k_1 d} + \frac{\alpha \theta}{k_1 d k_3} + \frac{\gamma_1 \theta}{k_1 d k_4} + \frac{\alpha \theta \gamma_2}{k_1 d k_3 k_4}$. Substituting (4) and (5) into the force of infection (3) yields

$$\lambda^{**} = \frac{\beta(\theta + d\sigma_1)}{k_1 d} \times \frac{\lambda^{**}}{1 + L_2 \lambda^{**}}. \quad (6)$$

Since $\lambda^{**} > 0$, (6) becomes

$$1 + L_2 \lambda^{**} = \frac{\beta(\theta + d\sigma_1)}{k_1 d} = \mathcal{R}_0. \quad (7)$$

Hence,

$$\lambda^{**} = \frac{\mathcal{R}_0 - 1}{L_2}. \quad (8)$$

Because every parameter of the model is positive, it follows that $L_2 > 0$. Therefore,

$$\lambda^{**} > 0 \iff \mathcal{R}_0 > 1.$$

Thus, system (1) possesses a unique biologically meaningful endemic equilibrium if and only if $\mathcal{R}_0 > 1$. $\square$

Remark 1. The above theorem establishes the existence and uniqueness of the endemic equilibrium. However, it does not determine the nature of the bifurcation occurring at the threshold $\mathcal{R}_0 = 1$. This is investigated in the subsequent section using the center manifold theory of Castillo-Chavez and Song.

5 Stability analysis

In this section, the local and global stability properties of the proposed model's disease-free and endemic equilibria are investigated.

5.1 Local stability of the disease-free equilibrium

Theorem 4. The DFE $\mathcal{E}_0$ of system (1) is locally asymptotically stable whenever $\mathcal{R}_0 < 1$ and unstable whenever $\mathcal{R}_0 > 1$.

Proof. The Jacobian matrix of system (1) evaluated at the DFE $\mathcal{E}_0$ is

$$J(\mathcal{E}_0) = \begin{pmatrix} \mu - b & -\beta\sigma_1 & -\beta & 0 & \psi \\ 0 & \beta\sigma_1 - k_1 & \beta & 0 & 0 \\ 0 & \theta & -d & 0 & 0 \\ 0 & 0 & \alpha & -k_3 & 0 \\ 0 & 0 & \gamma_1 & \gamma_2 & -k_4 \end{pmatrix},$$

where $k_1 = \theta + \mu$, $k_3 = \mu + \delta_2 + \gamma_2$, $k_4 = \mu + \psi$, $d = \alpha + \delta_1 + \gamma_1 + \mu$. The characteristic equation is obtained from $\det(\lambda I - J(\mathcal{E}_0)) = 0$, that is,

$$\begin{vmatrix} \lambda + b - \mu & \beta\sigma_1 & \beta & 0 & -\psi \\ 0 & \lambda - \beta\sigma_1 + k_1 & -\beta & 0 & 0 \\ 0 & -\theta & \lambda + d & 0 & 0 \\ 0 & 0 & -\alpha & \lambda + k_3 & 0 \\ 0 & 0 & -\gamma_1 & -\gamma_2 & \lambda + k_4 \end{vmatrix} = 0.$$

Since the first column contains only one nonzero entry, expanding along the first column gives

$$(\lambda + b - \mu) \begin{vmatrix} \lambda - \beta\sigma_1 + k_1 & -\beta & 0 & 0 \\ -\theta & \lambda + d & 0 & 0 \\ 0 & -\alpha & \lambda + k_3 & 0 \\ 0 & -\gamma_1 & -\gamma_2 & \lambda + k_4 \end{vmatrix} = 0.$$

The remaining determinant is a block lower triangular. Hence,

$$(\lambda + b - \mu) \begin{vmatrix} \lambda - \beta\sigma_1 + k_1 & -\beta \\ -\theta & \lambda + d \end{vmatrix} \begin{vmatrix} \lambda + k_3 & 0 \\ -\gamma_2 & \lambda + k_4 \end{vmatrix} = 0.$$

Therefore, $(\lambda + b - \mu)(\lambda + k_3)(\lambda + k_4)[(\lambda - \beta\sigma_1 + k_1)(\lambda + d) - \beta\theta] = 0$. Consequently, three eigenvalues are obtained immediately as

$$\lambda_1 = \mu - b, \quad \lambda_2 = -k_3, \quad \lambda_3 = -k_4.$$

Since $b > \mu$, $k_3 > 0$, $k_4 > 0$, it follows that $\lambda_1, \lambda_2, \lambda_3 < 0$. The remaining two eigenvalues satisfy

$$(\lambda - \beta\sigma_1 + k_1)(\lambda + d) - \beta\theta = 0,$$

or equivalently,

$$\lambda^2 + (k_1 + d - \beta\sigma_1)\lambda + (k_1 - \beta\sigma_1)d - \beta\theta = 0.$$

Using the definition of basic reproduction number $\mathcal{R}_0$, the constant term becomes $(k_1 - \beta\sigma_1)d - \beta\theta = k_1d(1 - \mathcal{R}_0)$. Hence, the quadratic equation reduces to

$$\lambda^2 + (k_1 + d - \beta\sigma_1)\lambda + k_1d(1 - \mathcal{R}_0) = 0.$$

Since $k_1 + d - \beta\sigma_1 > 0$ and $k_1d(1 - \mathcal{R}_0) > 0$, whenever $\mathcal{R}_0 < 1$, the Routh–Hurwitz criterion guarantees that both roots have negative real parts. Therefore, all eigenvalues of the Jacobian have negative real parts for $\mathcal{R}_0 < 1$. Therefore, the DFE is locally asymptotically stable.

Conversely, if $\mathcal{R}_0 > 1$, then the constant term of the quadratic becomes negative, implying that one eigenvalue is positive. Hence, the DFE is unstable. This completes the proof. $\square$

Theorem 5. The DFE of system (1) is globally asymptotically stable whenever $\mathcal{R}_0 < 1$.

Proof. Consider the Lyapunov function

$$V(E, I) = E + \frac{\beta}{d}I, \quad (9)$$

which is positive definite in the feasible region and vanishes only at the DFE.

Differentiating V along the solutions of system (1) gives

$$\begin{aligned} \dot{V} &= \dot{E} + \frac{\beta}{d}\dot{I} \\ &= \left[\beta(I + \sigma_1 E) \frac{S}{N} - (\theta + \mu)E \right] + \frac{\beta}{d} [\theta E - (\alpha + \delta_1 + \gamma_1 + \mu)I]. \end{aligned}$$

Since $d = \alpha + \delta_1 + \gamma_1 + \mu$, we obtain

$$\dot{V} = \beta(I + \sigma_1 E) \frac{S}{N} - (\theta + \mu)E + \frac{\beta\theta}{d}E - \beta I.$$

Using the fact that $S \leq N$, it follows that

$$\beta(I + \sigma_1 E) \frac{S}{N} \leq \beta(I + \sigma_1 E),$$

and therefore

$$\begin{aligned} \dot{V} &\leq \beta(I + \sigma_1 E) - (\theta + \mu)E + \frac{\beta\theta}{d}E - \beta I \\ &= \left(\beta\sigma_1 + \frac{\beta\theta}{d} - (\theta + \mu) \right) E. \end{aligned}$$

Since $\mathcal{R}_0 = \frac{\beta(\theta + \sigma_1 d)}{(\theta + \mu)d}$, we have

$$\beta\sigma_1 + \frac{\beta\theta}{d} = (\theta + \mu)\mathcal{R}_0.$$

Hence,

$$\dot{V} \leq (\theta + \mu)(\mathcal{R}_0 - 1)E.$$

Therefore, $\dot{V} \leq 0$ whenever $\mathcal{R}_0 < 1$, with equality if and only if $E = 0$. Since $\dot{I} = \theta E - dI$, it follows that $E = 0$ implies $I = 0$. Consequently, the largest positively invariant subset of $\{\dot{V} = 0\}$ is the DFE. According to LaSalle's invariance principle, every solution originating in the feasible region converges to the DFE. Hence, the DFE is globally asymptotically stable whenever $\mathcal{R}_0 < 1$. $\square$

Theorem 6 (Uniform Persistence). Assume that the feasible region Ω is positively invariant and that all solutions of the system are uniformly bounded. If $R_0 > 1$, the disease persists uniformly in the population. Consequently, there exists a positive constant $\Pi > 0$ such that every solution of the model with positive initial conditions satisfies

$$\liminf_{t \rightarrow \infty} S(t) \geq \Pi,$$

$$\liminf_{t \rightarrow \infty} E(t) \geq \Pi,$$

$$\liminf_{t \rightarrow \infty} I(t) \geq \Pi,$$

$$\liminf_{t \rightarrow \infty} H(t) \geq \Pi,$$

and

$$\liminf_{t \rightarrow \infty} R(t) \geq \Pi.$$

Proof. Consider the Lyapunov function

$$\begin{aligned} V = & q_1 \left(S - S^{**} - S^{**} \ln \frac{S}{S^{**}} \right) + q_2 \left(E - E^{**} - E^{**} \ln \frac{E}{E^{**}} \right) \\ & + q_3 \left(I - I^{**} - I^{**} \ln \frac{I}{I^{**}} \right) + q_4 \left(H - H^{**} - H^{**} \ln \frac{H}{H^{**}} \right) \\ & + q_5 \left(R - R^{**} - R^{**} \ln \frac{R}{R^{**}} \right), \end{aligned} \quad (10)$$

where $q_i > 0$, $i = 1, 2, \dots, 5$. Since

$$x - a - a \ln \left(\frac{x}{a} \right) \geq 0, \quad x > 0, \quad a > 0,$$

the Lyapunov function satisfies $V(S, E, I, H, R) \geq 0$, with equality if and only if $(S, E, I, H, R) = (S^{**}, E^{**}, I^{**}, H^{**}, R^{**})$. This is precisely the endemic equilibrium.

Differentiating V along the solutions of the system gives

$$\begin{aligned} \frac{dV}{dt} = & q_1 \left(1 - \frac{S^{**}}{S} \right) \frac{dS}{dt} + q_2 \left(1 - \frac{E^{**}}{E} \right) \frac{dE}{dt} \\ & + q_3 \left(1 - \frac{I^{**}}{I} \right) \frac{dI}{dt} + q_4 \left(1 - \frac{H^{**}}{H} \right) \frac{dH}{dt} \\ & + q_5 \left(1 - \frac{R^{**}}{R} \right) \frac{dR}{dt}. \end{aligned} \quad (11)$$

Substituting the governing equations into the above expression yields

$$\begin{aligned} \frac{dV}{dt} = & q_1 \left(1 - \frac{S^{**}}{S} \right) \left[bN \left(1 - \frac{N}{K} \right) - \frac{\beta(I + \sigma_1 E)S}{N} - \mu S + \psi R \right] \\ & + q_2 \left(1 - \frac{E^{**}}{E} \right) \left[\frac{\beta(I + \sigma_1 E)S}{N} - (\theta + \mu)E \right] \\ & + q_3 \left(1 - \frac{I^{**}}{I} \right) [\theta E - (\delta_1 + \mu + \gamma_1 + \alpha)I] \\ & + q_4 \left(1 - \frac{H^{**}}{H} \right) [\alpha I - (\mu + \delta_2 + \gamma_2)H] \\ & + q_5 \left(1 - \frac{R^{**}}{R} \right) [\gamma_1 I + \gamma_2 H - (\mu + \psi)R]. \end{aligned} \quad (12)$$

For convenience, we decompose the derivative into five components,

$$\frac{dV}{dt} = L_1 + L_2 + L_3 + L_4 + L_5,$$

where

$$\begin{aligned}
L_1 &= q_1 \left(1 - \frac{S^{**}}{S}\right) bN \left(1 - \frac{N}{K}\right), \\
L_2 &= -q_1 \left(1 - \frac{S^{**}}{S}\right) \frac{\beta(I + \sigma_1 E)S}{N} + q_2 \left(1 - \frac{E^{**}}{E}\right) \frac{\beta(I + \sigma_1 E)S}{N}, \\
L_3 &= -q_1 \mu \left(1 - \frac{S^{**}}{S}\right) S - q_2 (\theta + \mu) \left(1 - \frac{E^{**}}{E}\right) E \\
&\quad - q_3 (\delta_1 + \mu + \gamma_1 + \alpha) \left(1 - \frac{I^{**}}{I}\right) I - q_4 (\mu + \delta_2 + \gamma_2) \left(1 - \frac{H^{**}}{H}\right) H \\
&\quad - q_5 (\mu + \psi) \left(1 - \frac{R^{**}}{R}\right) R, \\
L_4 &= q_3 \theta \left(1 - \frac{I^{**}}{I}\right) E + q_4 \alpha \left(1 - \frac{H^{**}}{H}\right) I, \\
L_5 &= q_1 \psi \left(1 - \frac{S^{**}}{S}\right) R + q_5 \left(1 - \frac{R^{**}}{R}\right) (\gamma_1 I + \gamma_2 H).
\end{aligned}$$

Hence,

$$\frac{dV}{dt} = L_1 + L_2 + L_3 + L_4 + L_5.$$

Because the feasible region Ω is positively invariant and bounded, every solution of the system remains positive and uniformly bounded. Consequently, there exists a positive constant M such that

$$0 < S(t), E(t), I(t), H(t), R(t), N(t) \leq M, \quad t \geq 0. \quad (13)$$

Furthermore, all model parameters are positive constants, and the incidence function is continuously differentiable on Ω . Hence, every component of the vector field is continuous in the compact invariant region Ω .

Recall that $\frac{dV}{dt} = L_1 + L_2 + L_3 + L_4 + L_5$. Because each state variable remains bounded in the compact invariant region Ω , every function L_i , $i = 1, \dots, 5$, is continuous on Ω . Therefore, by the Extreme Value Theorem, there exist positive constants C_1, C_2, C_3 and C_4 such that

$$L_1 \leq C_1, \quad L_2 \leq C_2, \quad L_4 \leq C_3, \quad L_5 \leq C_4.$$

Moreover,

$$\left(1 - \frac{x^{**}}{x}\right) x = x - x^{**},$$

so that

$$\begin{aligned}
L_3 = & -q_1\mu(S - S^{**}) - q_2(\theta + \mu)(E - E^{**}) \\
& - q_3(\delta_1 + \mu + \gamma_1 + \alpha)(I - I^{**}) \\
& - q_4(\mu + \delta_2 + \gamma_2)(H - H^{**}) \\
& - q_5(\mu + \psi)(R - R^{**}).
\end{aligned}$$

Let

$$\eta = \min \left\{ q_1\mu, q_2(\theta + \mu), q_3(\delta_1 + \mu + \gamma_1 + \alpha), \right. \\
\left. q_4(\mu + \delta_2 + \gamma_2), q_5(\mu + \psi) \right\} > 0.$$

Then,

$$L_3 \leq -\eta [(S - S^{**}) + (E - E^{**}) + (I - I^{**}) + (H - H^{**}) + (R - R^{**})]. \quad (14)$$

Combining the above estimates yields

$$\frac{dV}{dt} \leq C - \eta [(S - S^{**}) + (E - E^{**}) + (I - I^{**}) + (H - H^{**}) + (R - R^{**})], \quad (15)$$

where $C = C_1 + C_2 + C_3 + C_4$. Equation (15) shows that the derivative of the Lyapunov function is bounded above by a negative linear function of the deviation of the solution from the endemic equilibrium outside a compact neighborhood of the endemic equilibrium. $\square$

5.2 Bifurcation analysis

The local stability result established that the DFE is locally asymptotically stable whenever $\mathcal{R}_0 < 1$. However, stability analysis alone does not describe the qualitative behavior of the model in a neighborhood of the critical threshold $\mathcal{R}_0 = 1$. In particular, it does not indicate whether the model undergoes a forward or backward bifurcation as the basic reproduction number passes through unity. To investigate the local dynamics near the critical threshold, the center manifold theory developed by Castillo-Chavez and Song [6] is employed. The transmission coefficient β was selected as the bifurcation parameter. The following theorem provides a criterion for determining the direction of the bifurcation.

Theorem 7 (Castillo-Chavez and Song). Suppose that the Jacobian matrix of system (1) evaluated at the DFE has a simple zero eigenvalue when $\mathcal{R}_0 = 1$, and all the remaining eigenvalues have negative real parts. Let

$$a = \sum_{k,i,j=1}^5 v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\mathcal{E}_0, \beta^*), \quad (16)$$

and

$$b = \sum_{k,i=1}^5 v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta}(\mathcal{E}_0, \beta^*), \quad (17)$$

denote the center manifold coefficients, where $w = (w_1, \dots, w_5)^T$ and $v = (v_1, \dots, v_5)$ are the right and left eigenvectors associated with the simple zero eigenvalue, respectively. Then

1. if $a < 0$ and $b > 0$, the system undergoes a forward (supercritical transcritical) bifurcation;
2. if $a > 0$ and $b > 0$, the system undergoes a backward (subcritical) bifurcation.

Proof. The basic reproduction number obtained in section 4 is

$$\mathcal{R}_0 = \frac{\beta(\theta + \sigma_1 d)}{(\theta + \mu)d}, \quad (18)$$

where $d = \alpha + \delta_1 + \gamma_1 + \mu$. Substituting the bifurcation point $\mathcal{R}_0 = 1$ into (18) gives the critical transmission coefficient

$$\beta^* = \frac{(\theta + \mu)d}{\theta + \sigma_1 d}. \quad (19)$$

Hence, the DFE loses stability as the transmission coefficient passes through the critical value $\beta = \beta^*$. To facilitate the center manifold analysis, introduce the new variables

$$x_1 = S, \quad x_2 = E, \quad x_3 = I, \quad x_4 = H, \quad x_5 = R.$$

Then system (1) can be written in vector form as

$$\frac{dX}{dt} = F(X, \beta),$$

where $X = (x_1, x_2, x_3, x_4, x_5)^T$ and $F(X, \beta) = (f_1, f_2, f_3, f_4, f_5)^T$, with

$$f_1 = bN \left(1 - \frac{N}{K}\right) - \beta(x_3 + \sigma_1 x_2) \frac{x_1}{N} - \mu x_1 + \psi x_5,$$

$$f_2 = \beta(x_3 + \sigma_1 x_2) \frac{x_1}{N} - (\theta + \mu)x_2,$$

$$f_3 = \theta x_2 - dx_3,$$

$$\begin{aligned} f_4 &= \alpha x_3 - k_3 x_4, \\ f_5 &= \gamma_1 x_3 + \gamma_2 x_4 - k_4 x_5, \end{aligned} \tag{20}$$

where $k_3 = \mu + \delta_2 + \gamma_2$, $k_4 = \mu + \psi$. The DFE is

$$E_0 = \left(K \left(1 - \frac{\mu}{b} \right), 0, 0, 0, 0 \right),$$

which satisfies $N = S^* = K \left(1 - \frac{\mu}{b} \right)$. Consequently, $\frac{S^*}{N^*} = 1$. The Jacobian matrix of system (20) evaluated at the DFE and at the critical transmission coefficient $\beta = \beta^*$ is

$$J(E_0, \beta^*) = \begin{pmatrix} \mu - b & -\beta^* \sigma_1 & -\beta^* & 0 & \psi \\ 0 & \beta^* \sigma_1 - k_1 & \beta^* & 0 & 0 \\ 0 & \theta & -d & 0 & 0 \\ 0 & 0 & \alpha & -k_3 & 0 \\ 0 & 0 & \gamma_1 & \gamma_2 & -k_4 \end{pmatrix}, \tag{21}$$

where $k_1 = \theta + \mu$, $d = \alpha + \delta_1 + \gamma_1 + \mu$, $k_3 = \mu + \delta_2 + \gamma_2$, $k_4 = \mu + \psi$. The characteristic polynomial of the Jacobian matrix is obtained from

$$\chi(\lambda) = \det(\lambda I - J(E_0, \beta^*)).$$

Since the Jacobian matrix possesses a block lower triangular structure, its determinant can be factorized as

$$\chi(\lambda) = (\lambda + b - \mu)(\lambda + k_3)(\lambda + k_4) \det(M), \tag{22}$$

where

$$M = \begin{pmatrix} \lambda - \beta^* \sigma_1 + k_1 & -\beta^* \\ -\theta & \lambda + d \end{pmatrix}.$$

Therefore,

$$\det(M) = (\lambda - \beta^* \sigma_1 + k_1)(\lambda + d) - \beta^* \theta. \tag{23}$$

Expanding (23) gives

$$\det(M) = \lambda^2 + (d + k_1 - \beta^* \sigma_1) \lambda + d(k_1 - \beta^* \sigma_1) - \beta^* \theta.$$

Consequently,

$$\begin{aligned}\chi(\lambda) &= (\lambda + b - \mu)(\lambda + k_3)(\lambda + k_4) \\ &\times [\lambda^2 + (d + k_1 - \beta^* \sigma_1)\lambda + d(k_1 - \beta^* \sigma_1) - \beta^* \theta].\end{aligned}\quad (24)$$

To verify the existence of a simple zero eigenvalue, let $\lambda = 0$, then $\det(M) = d(k_1 - \beta^* \sigma_1) - \beta^* \theta$. Using $\beta^* = \frac{k_1 d}{\theta + \sigma_1 d}$, we obtain

$$\begin{aligned}d(k_1 - \beta^* \sigma_1) - \beta^* \theta &= dk_1 - \frac{k_1 d^2 \sigma_1}{\theta + \sigma_1 d} - \frac{k_1 d \theta}{\theta + \sigma_1 d} \\ &= dk_1 - \frac{k_1 d(\sigma_1 d + \theta)}{\theta + \sigma_1 d} \\ &= dk_1 - dk_1 \\ &= 0.\end{aligned}$$

Hence, $\lambda = 0$ is the eigenvalue of the Jacobian matrix. Next, $\mu - b < 0$, since the DFE exists whenever $b > \mu$. Similarly, $-k_3 < 0$, $-k_4 < 0$, because every parameter is positive. The remaining eigenvalue associated with the quadratic factor satisfies

$$\lambda = -(d + k_1 - \beta^* \sigma_1),$$

which is negative because $d + k_1 - \beta^* \sigma_1 = d + \theta + \mu - \beta^* \sigma_1 > 0$. Therefore, the Jacobian matrix possesses exactly one simple zero eigenvalue, whereas all the remaining eigenvalues have strictly negative real parts. Hence, all hypotheses of the Castillo–Chavez and Song theorem are satisfied. $\square$

Computation of the right eigenvector

Let $w = (w_1, w_2, w_3, w_4, w_5)^T$ be the right eigenvector associated with the simple zero eigenvalue of Jacobian matrix, then $J(\mathcal{E}_0, \beta^*)w = 0$. From (21), we obtain the following system of linear equations

$$(\mu - b)w_1 - \beta^* \sigma_1 w_2 - \beta^* w_3 + \psi w_5 = 0, \quad (25)$$

$$(\beta^* \sigma_1 - k_1)w_2 + \beta^* w_3 = 0, \quad (26)$$

$$\theta w_2 - d w_3 = 0, \quad (27)$$

$$\alpha w_3 - k_3 w_4 = 0, \quad (28)$$

$$\gamma_1 w_3 + \gamma_2 w_4 - k_4 w_5 = 0. \quad (29)$$

Equation (27) immediately gives

$$w_3 = \frac{\theta}{d} w_2. \quad (30)$$

Substituting (30) into (28) yields

$$w_4 = \frac{\alpha \theta}{d k_3} w_2. \quad (31)$$

Similarly, substituting (30) and (31) into (29) gives

$$w_5 = \frac{\theta}{d k_4} \left(\gamma_1 + \frac{\alpha \gamma_2}{k_3} \right) w_2. \quad (32)$$

Finally, substituting (30) and (32) into (25) gives

$$(\mu - b)w_1 = \beta^* \sigma_1 w_2 + \frac{\beta^* \theta}{d} w_2 - \frac{\psi \theta}{d k_4} \left(\gamma_1 + \frac{\alpha \gamma_2}{k_3} \right) w_2.$$

Hence,

$$w_1 = \frac{\beta^* \left(\sigma_1 + \frac{\theta}{d} \right) - \frac{\psi \theta}{d k_4} \left(\gamma_1 + \frac{\alpha \gamma_2}{k_3} \right)}{\mu - b} w_2. \quad (33)$$

Therefore, the right eigenvector corresponding to the simple zero eigenvalue is

$$w = \begin{pmatrix} \frac{\beta^* \left(\sigma_1 + \frac{\theta}{d} \right) - \frac{\psi \theta}{d k_4} \left(\gamma_1 + \frac{\alpha \gamma_2}{k_3} \right)}{\mu - b} w_2 \\ w_2 \\ \frac{\theta}{d} w_2 \\ \frac{\alpha \theta}{d k_3} w_2 \\ \frac{\theta}{d k_4} \left(\gamma_1 + \frac{\alpha \gamma_2}{k_3} \right) w_2 \end{pmatrix},$$

where w_2 is arbitrary.

Computation of the left eigenvector

Let $v = (v_1, v_2, v_3, v_4, v_5)$ be the left eigenvector associated with the simple zero eigenvalue, then $vJ(\mathcal{E}_0, \beta^*) = 0$, or equivalently, $J(\mathcal{E}_0, \beta^*)^T v = 0$. This gives

$$(\mu - b)v_1 = 0, \quad (34)$$

$$-\beta^* \sigma_1 v_1 + (\beta^* \sigma_1 - k_1)v_2 + \theta v_3 = 0, \quad (35)$$

$$-\beta^* v_1 + \beta^* v_2 - dv_3 + \alpha v_4 + \gamma_1 v_5 = 0, \quad (36)$$

$$-k_3 v_4 + \gamma_2 v_5 = 0, \quad (37)$$

$$\psi v_1 - k_4 v_5 = 0. \quad (38)$$

Since $\mu - b \neq 0$, (34) gives $v_1 = 0$. Substituting into (38) yields $v_5 = 0$. Equation (37) therefore becomes $v_4 = 0$.

Substituting these results into (36) gives $\beta^* v_2 - dv_3 = 0$, from which

$$v_3 = \frac{\beta^*}{d} v_2. \quad (39)$$

Finally, substituting (39) into (35) gives

$$\left(\beta^* \sigma_1 - k_1 + \frac{\beta^* \theta}{d} \right) v_2 = 0.$$

Since $\beta^* = \frac{k_1 d}{\theta + \sigma_1 d}$, we have

$$\beta^* \sigma_1 - k_1 + \frac{\beta^* \theta}{d} = 0.$$

Hence, Equation (35) is satisfied identically, implying that v_2 is arbitrary. Therefore, the left eigenvector associated with the simple zero eigenvalue is

$$v = \left(0, v_2, \frac{\beta^*}{d} v_2, 0, 0 \right),$$

where v_2 is arbitrary.

Second-order partial derivatives

Because the functions f_3 , f_4 and f_5 are linear, all their second-order partial derivatives vanish identically. Consequently, only the nonlinear functions f_1 and f_2 contribute to the computation of the center-manifold coefficients.

The nonlinear incidence term is

$$\Phi(S, E, I, H, R) = \beta \frac{S(I + \sigma_1 E)}{N},$$

where $N = S + E + I + H + R$. The required second-order partial derivatives are evaluated at the DFE

$$\mathcal{E}_0 = (S^*, 0, 0, 0, 0),$$

where $S^* = N^*$. The second-order partial derivatives of the vector field are required to compute the center manifold coefficients. Because the functions f_3 , f_4 and f_5 are linear, all their second-order partial derivatives vanish identically. Therefore, only the nonlinear functions f_1 and f_2 contribute to the bifurcation analysis. The nonlinear incidence term is $\Phi(x) = \beta \frac{x_1(x_3 + \sigma_1 x_2)}{N}$, where $N = x_1 + x_2 + x_3 + x_4 + x_5$. The required derivatives are evaluated at the DFE

$$\mathcal{E}_0 = (S^*, 0, 0, 0, 0), \quad S^* = N^* = K \left(1 - \frac{\mu}{b}\right).$$

Since $f_2 = \Phi - k_1 x_2$, The linear term contributes no second-order derivatives. Differentiating the incidence function and evaluating at the DFE yields

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_2}(\mathcal{E}_0) = 0, \tag{40}$$

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_3}(\mathcal{E}_0) = 0, \tag{41}$$

$$\frac{\partial^2 f_2}{\partial x_2^2}(\mathcal{E}_0) = -\frac{2\beta^* \sigma_1}{S^*}, \tag{42}$$

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_3}(\mathcal{E}_0) = -\frac{\beta^*(1 + \sigma_1)}{S^*}, \tag{43}$$

$$\frac{\partial^2 f_2}{\partial x_3^2}(\mathcal{E}_0) = -\frac{2\beta^*}{S^*}. \tag{44}$$

Since $f_1 = bN \left(1 - \frac{N}{K}\right) - \Phi - \mu x_1 + \psi x_5$, The nonlinear infection term in f_1 is the negative of that appearing in f_2 . Consequently,

$$\frac{\partial^2 f_1}{\partial x_1 \partial x_2}(\mathcal{E}_0) = 0, \tag{45}$$

$$\frac{\partial^2 f_1}{\partial x_1 \partial x_3}(\mathcal{E}_0) = 0, \tag{46}$$

$$\frac{\partial^2 f_1}{\partial x_2^2}(\mathcal{E}_0) = \frac{2\beta^* \sigma_1}{S^*}, \tag{47}$$

$$\frac{\partial^2 f_1}{\partial x_2 \partial x_3}(\mathcal{E}_0) = \frac{\beta^*(1 + \sigma_1)}{S^*}, \quad (48)$$

$$\frac{\partial^2 f_1}{\partial x_3^2}(\mathcal{E}_0) = \frac{2\beta^*}{S^*}. \quad (49)$$

Moreover,

$$\frac{\partial^2 f_k}{\partial x_i \partial x_j}(\mathcal{E}_0) = 0, \quad k = 3, 4, 5,$$

for all $i, j = 1, \dots, 5$. Hence, the nonzero second-order partial derivatives required in the computation of the Castillo–Chavez coefficients are given by (42)–(44) and (47)–(49).

Computation of the coefficient a

Following Castillo–Chavez and Song [6], the coefficient a is given by

$$a = \sum_{k,i,j=1}^5 v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\mathcal{E}_0, \beta^*). \quad (50)$$

Since $v_1 = v_4 = v_5 = 0$, and the functions f_3 , f_4 and f_5 are linear, their second-order partial derivatives vanish identically. Hence, only the nonlinear infection equation f_2 contributes to the coefficient a . Therefore,

$$a = v_2 \sum_{i,j=1}^5 w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j}(\mathcal{E}_0, \beta^*). \quad (51)$$

From the Hessian matrix evaluated at the DFE, the only nonzero second-order partial derivatives are

$$\frac{\partial^2 f_2}{\partial x_2^2} = -\frac{2\beta^* \sigma_1}{S^*},$$

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_3} = \frac{\partial^2 f_2}{\partial x_3 \partial x_2} = -\frac{\beta^*(1 + \sigma_1)}{S^*},$$

and

$$\frac{\partial^2 f_2}{\partial x_3^2} = -\frac{2\beta^*}{S^*}.$$

Consequently,

$$a = v_2 \left[w_2^2 \frac{\partial^2 f_2}{\partial x_2^2} + 2w_2 w_3 \frac{\partial^2 f_2}{\partial x_2 \partial x_3} + w_3^2 \frac{\partial^2 f_2}{\partial x_3^2} \right]. \quad (52)$$

Since $w_3 = \frac{\theta}{d} w_2$, it follows that

$$a = v_2 w_2^2 \left[\frac{\partial^2 f_2}{\partial x_2^2} + 2 \frac{\theta}{d} \frac{\partial^2 f_2}{\partial x_2 \partial x_3} + \frac{\theta^2}{d^2} \frac{\partial^2 f_2}{\partial x_3^2} \right]. \quad (53)$$

Substituting the corresponding second-order partial derivatives gives

$$\begin{aligned} a &= v_2 w_2^2 \left[-\frac{2\beta^* \sigma_1}{S^*} - 2 \frac{\theta}{d} \left(\frac{\beta^* (1 + \sigma_1)}{S^*} \right) - \frac{\theta^2}{d^2} \left(\frac{2\beta^*}{S^*} \right) \right] \\ &= -\frac{2\beta^* v_2 w_2^2}{S^*} \left[\sigma_1 + \frac{\theta(1 + \sigma_1)}{d} + \frac{\theta^2}{d^2} \right]. \end{aligned} \quad (54)$$

Since all model parameters are positive,

$$\beta^* > 0, \quad S^* > 0, \quad d > 0, \quad v_2 > 0, \quad w_2 > 0,$$

and

$$\sigma_1 + \frac{\theta(1 + \sigma_1)}{d} + \frac{\theta^2}{d^2} > 0,$$

it follows immediately that $a < 0$. The coefficient b is defined by

$$b = \sum_{k,i=1}^5 v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta}(\mathcal{E}_0, \beta^*). \quad (55)$$

Since $v_1 = v_4 = v_5 = 0$ and the functions f_3 , f_4 , and f_5 do not depend explicitly on the bifurcation parameter β , only the infected equation f_2 contributes to the above equation. Hence,

$$b = v_2 \sum_{i=1}^5 w_i \frac{\partial^2 f_2}{\partial x_i \partial \beta}(\mathcal{E}_0, \beta^*). \quad (56)$$

The infection equation is

$$f_2 = \beta \frac{x_1(x_3 + \sigma_1 x_2)}{N} - k_1 x_2.$$

Differentiating with respect to β gives

$$\frac{\partial f_2}{\partial \beta} = \frac{x_1(x_3 + \sigma_1 x_2)}{N}.$$

Differentiating with respect to the state variables and evaluating at the DFE yields

$$\frac{\partial^2 f_2}{\partial x_1 \partial \beta}(\mathcal{E}_0, \beta^*) = 0,$$

$$\frac{\partial^2 f_2}{\partial x_2 \partial \beta}(\mathcal{E}_0, \beta^*) = \sigma_1,$$

$$\frac{\partial^2 f_2}{\partial x_3 \partial \beta}(\mathcal{E}_0, \beta^*) = 1,$$

while

$$\frac{\partial^2 f_2}{\partial x_4 \partial \beta} = \frac{\partial^2 f_2}{\partial x_5 \partial \beta} = 0.$$

Therefore,

$$b = v_2 (\sigma_1 w_2 + w_3). \quad (57)$$

Using the right eigenvector relation $w_3 = \frac{\theta}{d} w_2$, we obtain

$$b = v_2 w_2 \left(\sigma_1 + \frac{\theta}{d} \right). \quad (58)$$

Since

$$v_2 > 0, \quad w_2 > 0, \quad \sigma_1 > 0, \quad \theta > 0, \quad d > 0,$$

it follows immediately that $b > 0$. Therefore, $a < 0$, $b > 0$. Hence, by the Castillo–Chavez and Song theorem, the proposed epidemic model undergoes a *forward (supercritical transcritical) bifurcation* at the critical threshold $\mathcal{R}_0 = 1$. Consequently, the DFE is locally asymptotically stable whenever $\mathcal{R}_0 < 1$, whereas a unique locally asymptotically stable endemic equilibrium emerges for $\mathcal{R}_0 > 1$.

6 Sensitivity analysis

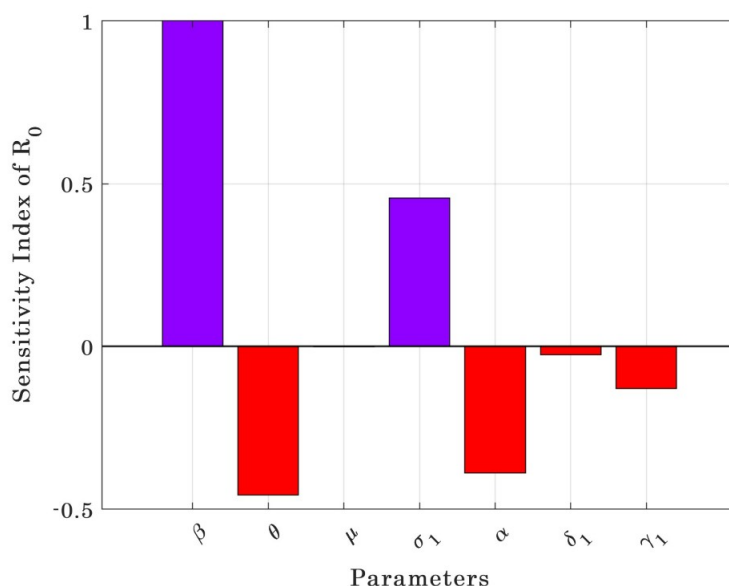
Sensitivity analysis plays a vital role in determining which parameters should be prioritized when designing intervention strategies to reduce and control the spread of meningococcal meningitis. This analysis measures how variations in the basic reproduction number ($\mathcal{R}_0$) correspond to proportional changes in individual parameters. In this study, the influence of each model parameter on ($\mathcal{R}_0$) was examined at its given value. The analytical expression used to evaluate the sensitivity of ($\mathcal{R}_0$) with respect to any parameter (τ) is given as:

$$\Upsilon_{\tau}^{\mathcal{R}_0} = \frac{\tau}{\mathcal{R}_0} \times \frac{\partial \mathcal{R}_0}{\partial \tau}.$$

Here, (τ) represents any arbitrary parameter that influences ($\mathcal{R}_0$). By substituting the baseline parameter values listed in Table 2, the numerical sensitivity indices of model (1) were computed using ($\mathcal{R}_0$) as the response variable. The outcomes of the sensitivity analysis are shown in Figure 2 and Table 3.

Table 3: Sensitivity indices of the model parameters

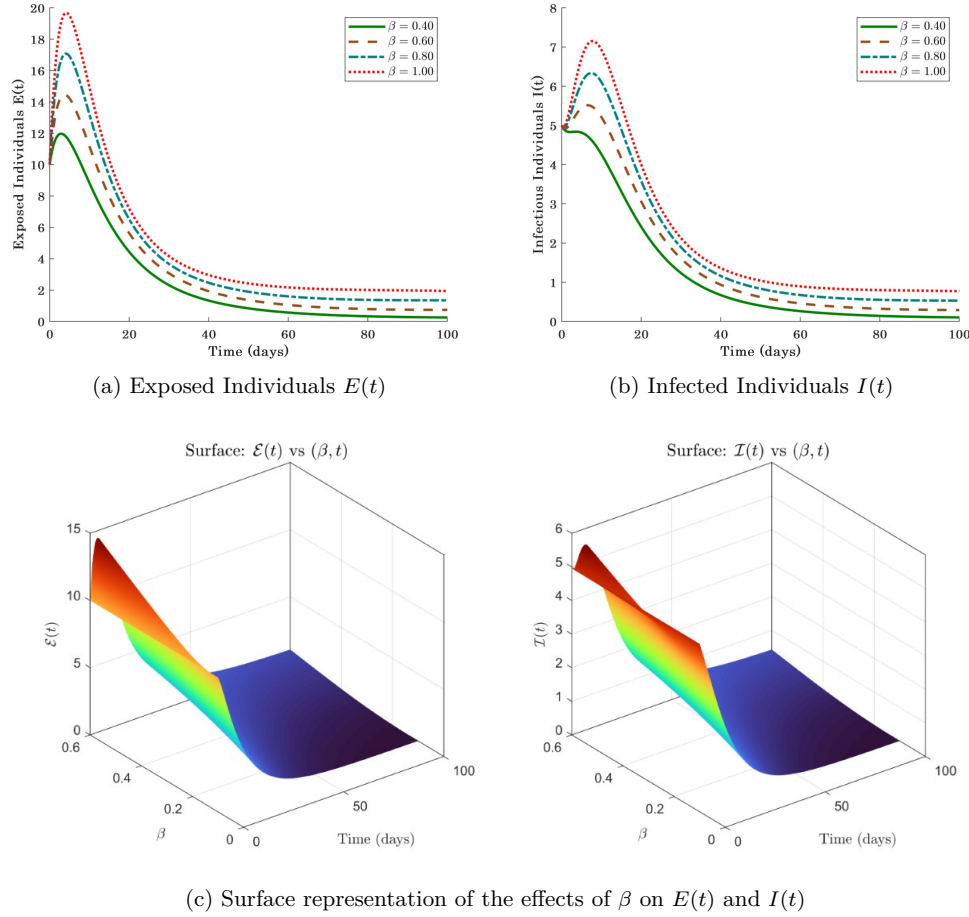
Parameter	Sensitivity Index
β	1
θ	-0.45635
μ	-0.000245421
σ_1	0.456545
α	-0.388146
δ_1	-0.0258764
γ_1	-0.129382

Figure 2: Sensitivity index of the basic reproduction number $\mathcal{R}_0$

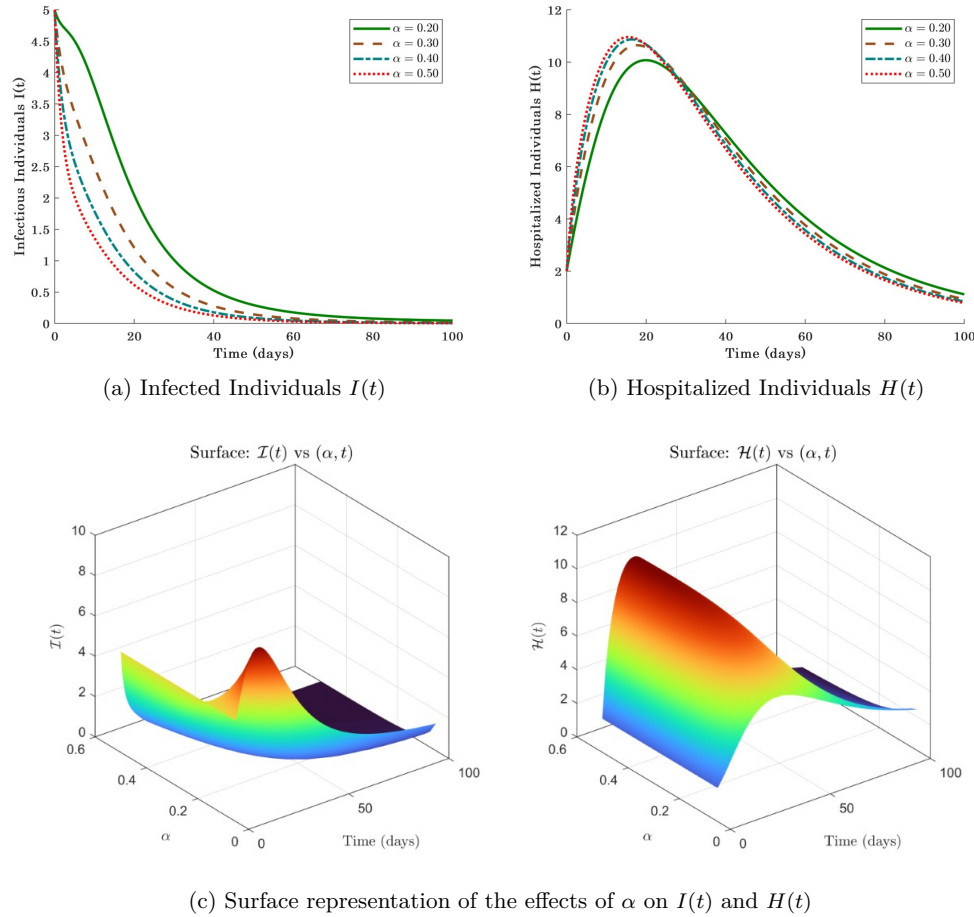
7 Numerical simulation

In this section, the proposed model (1) is numerically solved using the `ode45` solver in MATLAB®. The initial conditions are assumed as $S(0) = 1000$, $E(0) = 10$, $I(0) = 5$, $H(0) = 2$, $R(0) = 1$, $D(0) = 0$.

Figure 3 illustrates the impact of varying transmission rates (β) on the dynamics of exposed and infected human populations. The plot of the exposed group $E(t)$ for $\beta = 0.40, 0.60, 0.80$, and 1.00 is shown in Figure 3(a). It can be observed that as the transmission rate increases, the number of exposed individuals rises sharply, reaching a higher peak before gradually declining over time. This behavior indicates that higher transmission rates accelerate the spread of

Figure 3: Impact of the transmission rate β on exposed and infected individuals

meningococcal meningitis, leading to increased exposure during the early stages of an outbreak. However, with effective hospitalization and treatment measures, the exposed population eventually decreases, contributing to the stabilization of the epidemic. Similarly, Figure 3(b) shows that the number of infected individuals $I(t)$ increases as β increases. The trajectories reveal that higher values of β lead to earlier and larger infection peaks, followed by a steady decline. This trend implies that elevated transmission rates hasten the progression from exposure to infection and prolong the active infection period until recovery or intervention reduces the case count. Figure 3(c) shows a surface plot summarizing these scenarios. Overall, the results depicted in Figure 3 emphasize that the transmission rate β plays a crucial role in shaping both the intensity and duration of meningococcal meningitis outbreaks. While higher β values intensify and

Figure 4: Impact of the hospitalization rate α on infected and hospitalized individuals

accelerate disease spread, timely medical responses and treatment interventions help mitigate infections and restore system equilibrium over time.

Figure 4 illustrates the influence of varying hospitalization rates (α), which represent the cure rate of infected individuals ($\alpha > 0$), on the dynamics of the infected and hospitalized human populations. Simulations were conducted for $\alpha = 0.20, 0.30, 0.40$, and 0.50 . Figure 4(a) depicts the temporal evolution of the infected population $I(t)$. It is observed that as the hospitalization rate α increases, the number of infected individuals decreases. A higher value of α implies faster hospitalization and recovery, thereby reducing the proportion of actively infected individuals within the population. Consequently, the infection curves corresponding to larger α values decrease more sharply, indicating a faster containment of the disease. The temporal

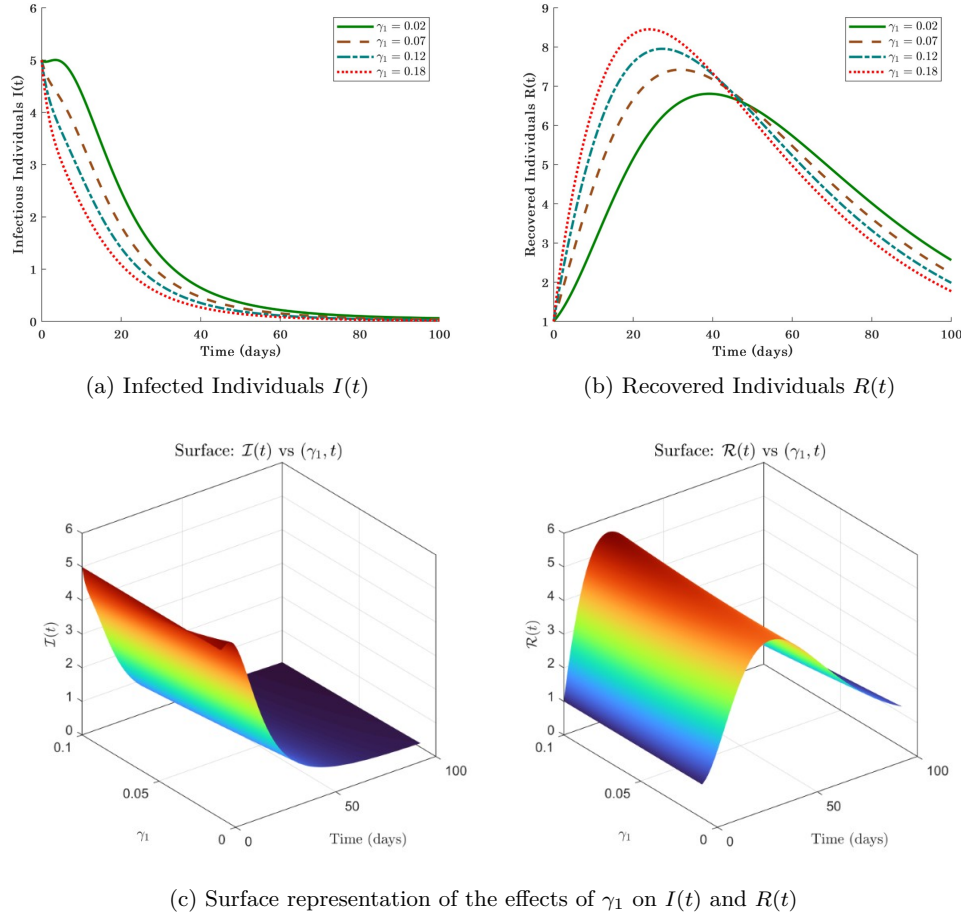


Figure 5: Impact of the recovery rate of infectious individuals γ_1 on infected and recovered individuals

behavior of hospitalized individuals $H(t)$ is shown in Figure 4(b). Initially, a higher α resulted in an earlier and slightly elevated peak in hospitalizations due to the faster transfer of infected individuals to treatment facilities. Over time, however, the hospitalized population diminishes as recovery rates improve with increasing α . Figure 4(c) shows a surface representation of these outcomes. Overall, the results in Figure 4 demonstrate that the hospitalization rate α plays a vital role in controlling the spread of meningococcal meningitis cases. A higher α accelerates the hospitalization and recovery of infected individuals and contributes to stabilizing the epidemic and reducing the overall disease prevalence in the long term.

Figure 5 demonstrates how variations in the recovery rate (γ_1) of infectious individuals influence the dynamics of both the infected and recovered populations. The simulations were

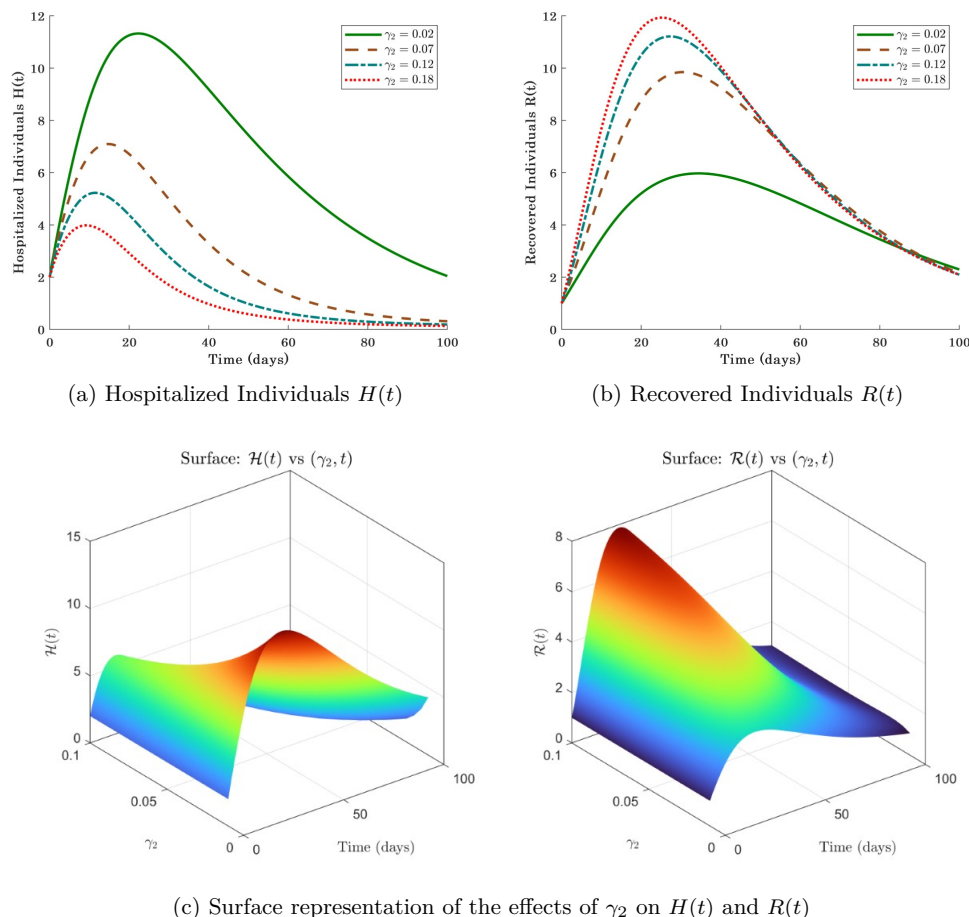
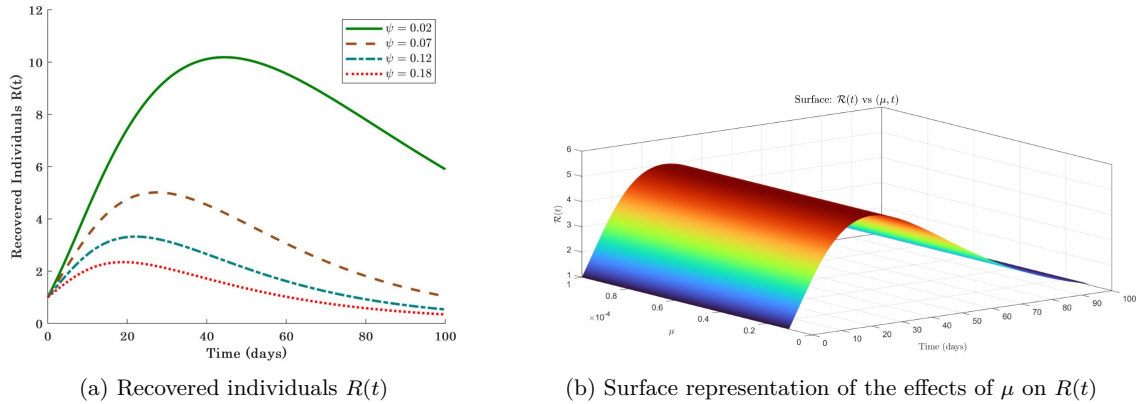


Figure 6: Impact of the recovery rate of hospitalized individuals γ_2 on hospitalized and recovered individuals

performed for $\gamma_1 = 0.02, 0.07, 0.12$, and 0.18 . Figure 5(a) shows the temporal evolution of the number of infected individuals $I(t)$ for various recovery rates. It is evident that as γ_1 increases, the number of infected individuals declines more rapidly. Lower γ_1 values corresponded to slower recovery, resulting in a prolonged infection period and a delayed reduction in cases. In contrast, higher recovery rates led to a faster decrease in infections, indicating that individuals recovered more quickly and the disease was brought under control sooner. These results highlight that enhancing the recovery rate significantly accelerates infection suppression and shortens the infectious period of the disease. Figure 5(b) illustrates the corresponding changes in the recovered population $R(t)$ for different values of γ_1 . As the recovery rate increased, the recovered population grew more steeply, reaching a higher peak in a shorter period. This pattern implies that a larger

Figure 7: Impact of the loss of immunity rate ψ on recovered individuals

fraction of infected individuals transitions into the recovered class earlier when recovery is more efficient. After peaking, the number of recovered individuals gradually stabilizes, signifying that the outbreak is under control as recovery surpasses new infection. Figure 5(c) presents a surface plot that visually summarizes these dynamics. Overall, the results in Figure 5 underscore that the recovery rate γ_1 is a crucial determinant of the progression and mitigation of meningococcal meningitis. A higher γ_1 not only reduces the number of active infections but also enhances the accumulation of recovered individuals, thereby promoting faster epidemic stabilization and improving the disease management.

Figure 6 depicts the influence of the recovery rate of hospitalized individuals (γ_2) on the variation in both hospitalized ($H(t)$) and recovered ($R(t)$) populations. Simulations were performed for different recovery rate values: $\gamma_2 = 0.02, 0.07, 0.12$, and 0.18 . Figure 6(a) illustrates the temporal behavior of hospitalized individuals $H(t)$ for the given recovery rates. It is observed that as γ_2 increases, the number of hospitalized individuals decreases noticeably. At lower recovery rates (e.g., $\gamma_2 = 0.02$), hospitalization levels reach a higher peak and decline slowly over time, reflecting prolonged hospital stays and delayed recovery. Conversely, at higher recovery rates (e.g., $\gamma_2 = 0.18$), hospitalization peaks are smaller and decline more rapidly, indicating a faster recovery and shorter hospitalization durations. These observations highlight that an increased recovery rate effectively reduces the overall hospitalization burden by facilitating quicker patient discharge. The dynamics of the recovered population $R(t)$ under the same recovery rate variation are shown in Figure 6(b). As γ_2 increases, the number of recovered individuals increases more sharply, attaining a higher peak in a shorter time frame. This indicates that higher recovery rates among hospitalized patients directly accelerate the accumulation of recoveries within the population. Following the peak, the recovered population stabilizes gradually, signifying that the

epidemic subsides as recovery becomes more dominant than new admissions. Figure 6(c) provides a surface representation that visually consolidates these observations. Overall, the results in Figure 6 emphasize the crucial role of the recovery rate γ_2 in shaping both hospitalization duration and recovery trends. A higher γ_2 not only shortens hospital stays and promotes faster patient recovery but also alleviates pressure on healthcare systems and contributes significantly to the effective control of meningococcal meningitis.

Figure 7 demonstrates the influence of the loss of immunity rate (ψ) on the dynamics of the recovered population ($R(t)$) in a human community. Simulations were conducted for different values of $\psi = 0.02, 0.07, 0.12$, and 0.18 . As shown in Figure 7(a), the number of recovered individuals decreases notably as the loss of immunity rate ψ increases. For lower ψ values (e.g., $\psi = 0.02$), the recovered population reaches a higher peak and remains elevated for a longer period, indicating prolonged immunity and fewer individuals reverting to the susceptible class. Conversely, at higher ψ values (e.g., $\psi = 0.18$), the recovered curve displays a smaller peak followed by a faster decline, signifying a quicker waning of immunity and a faster return of recovered individuals to being susceptible. These observations suggest that an elevated loss of immunity reduces the duration of post-recovery protection, thereby accelerating the transition of recovered individuals back into the susceptible group. This process diminishes the long-term resistance of the population to infections and increases the likelihood of disease resurgence. Figure 7(b) presents a surface plot that visually summarizes these relationships. In summary, the results in Figure 7 highlight that the loss of immunity rate ψ plays a critical role in determining the persistence and stability of recovery. Lower ψ values support sustained immunity and a larger recovered population over time, whereas higher ψ values weaken recovery durability, potentially leading to repeated meningococcal meningitis outbreaks.

8 Conclusion

This study investigated the transmission dynamics of meningococcal meningitis using a novel compartmental model that incorporates hospitalization and treatment effects. The theoretical behavior of the model was examined using the threshold dynamics. The model incorporates key epidemiological parameters, such as the intrinsic per capita recruitment rate (b), carrying capacity (K), natural death rate (μ), and transmission rate (β), which characterize the demographic and epidemiological features of the disease. The progression rate (θ) governs the transition from the exposed class to the infectious class, whereas the modification parameter (σ_1) accounts for the reduced infectiousness of exposed individuals. Disease progression and clinical outcomes are

further described by the hospitalization rate (α), recovery rates of infectious and hospitalized individuals (γ_1 and γ_2), and disease-induced death rates (δ_1 and δ_2).

Numerical simulations demonstrated that increasing the hospitalization and recovery rates substantially reduced the number of infectious individuals and shortened the duration of the epidemic. Furthermore, the sensitivity analysis identified the transmission, hospitalization, and recovery rates as the most influential parameters affecting the basic reproduction number ($\mathcal{R}_0$). These findings highlight the important role of timely hospitalization and effective clinical management in reducing disease transmission. Overall, the proposed framework provides valuable insights into the transmission dynamics of meningococcal meningitis and offers a useful tool for evaluating disease-control strategies. Future work may extend the present model by incorporating vaccination, delay differential equations to capture time-dependent epidemiological processes, stochastic effects, and optimal control strategies to design more effective intervention policies.

Author contributions

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N. Jeeva: Conceptualization, Writing-original draft, Software, Investigation, Validation, Supervision and Writing-review and Editing.

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All data used are available within the manuscript.

Ethics Statement

This study did not require ethical approval.

Conflicts of Interest

The authors declare no conflicts of interest.

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