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RESEARCH PAPER

An optimal control strategy for cerebrospinal meningitis in Yobe State, Nigeria: a mathematical modeling approach

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Abstract

This paper develops and analyzes a deterministic SEIHR (Susceptible-Exposed-Infectious-Hospitalized-Recovered) model to investigate the transmission dynamics of cerebrospinal meningitis (CSM) and evaluate optimal control strategies. The framework incorporates three time-dependent control variables: mass vaccination of susceptible individuals, enhanced treatment for hospitalized patients, and public awareness campaigns. Using Pontryagin's Maximum Principle, we formulate an optimal control problem to minimize the number of infected individuals and the costs associated with the interventions. The basic reproduction number (R_0) is derived, and its sensitivity to key parameters is analyzed. Numerical simulations, using data relevant to the Yobe State context, demonstrate that a combined strategy of early, intensive vaccination, sustained treatment efforts, and effective public awareness is the most effective approach to mitigate the burden of a CSM outbreak. These findings provide quantitative support for evidence-based public health policies aimed at controlling meningitis in high-risk regions.

Keywords: Cerebrospinal meningitis; mathematical modeling; optimal control; basic reproduction number; stability analysis

AMS 2020 Classification: 92D30; 49N90; 49J15; 34D20; 93C15

1 Introduction

Cerebrospinal Meningitis (CSM) remains a formidable global health challenge, defined by its potential to cause devastating, fast-moving epidemics with high fatality rates. The World Health Organization (WHO) has recognized its severe impact by launching the "Defeating Meningitis by 2030" global roadmap, a testament to the international commitment required to control this disease [1]. CSM, an inflammation of the membranes surrounding the brain and spinal cord, is caused by various pathogens, but its bacterial form is primarily driven by agents like *Neisseria*

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meningitidis and *Streptococcus pneumoniae* is the most life-threatening, capable of causing death within 24 hours of symptom onset [2, 3]. Survivors often face lifelong disabilities, including hearing loss, neurological damage, and limb loss, imposing a heavy socioeconomic burden on communities and healthcare systems.

The epicenter of the global meningitis burden is the "African meningitis belt," a vast region stretching across sub-Saharan Africa where the disease is hyperendemic. Historically, this region was plagued by massive epidemics of serogroup A meningococcus. While the introduction of the MenAfriVac® conjugate vaccine led to a dramatic decline in serogroup A cases, the epidemiological landscape has since evolved [4, 5]. Recent years have seen a concerning rise in outbreaks caused by other serogroups, such as C, W, and X, demonstrating the pathogen's adaptive capacity and the ongoing need for vigilant surveillance and responsive control strategies. Nigeria, located at the heart of this belt, is particularly vulnerable. The country experiences recurrent seasonal outbreaks, typically between December and April, when the dry, dusty Harmattan winds are thought to facilitate the transmission of respiratory pathogens [6]. The early 2024 outbreaks in Yobe and Gombe States, which resulted in over a thousand suspected cases and dozens of deaths, serve as a stark reminder of this persistent threat and underscore the urgency of reinforcing control measures [7–9].

In response to such complex public health challenges, mathematical modeling has emerged as an indispensable tool for dissecting disease transmission dynamics and prospectively evaluating intervention strategies [10]. A significant body of literature has applied these methods to meningitis. For instance, Agusto and Leite used an optimal control framework to analyze the 2017 meningitis outbreak in Nigeria, focusing on vaccination and treatment [11]. Others, like Crankson et al., have modeled the specific impact of vaccination on transmission [12]. While these national-level models are invaluable, disease dynamics are often highly localized, and strategies must be tailored to specific regional contexts. More locally, Madaki et al. developed a model for bacterial meningitis dynamics within a hospital in Yobe State [13]. However, their work was primarily descriptive and did not employ optimal control theory to determine the most cost-effective, time-dependent intervention policies for the entire population. This distinction is crucial, as optimal control provides a prescriptive framework for resource allocation, moving beyond predicting outcomes to actively shaping them [14].

Despite these contributions, a critical gap remains: the development of an optimal control framework specifically tailored to the epidemiological context of the most recent CSM outbreaks in Nigeria's high-risk states, such as the 2024 Yobe event. This study aims to fill this gap by proposing and analyzing a novel SEIHR (Susceptible-Exposed-Infectious-Hospitalized-Recovered) model. The inclusion of a distinct Hospitalized (H) compartment is a key feature, allowing for a more nuanced analysis of the healthcare burden and the direct impact of treatment-based interventions. We introduce three time-dependent control functions representing mass vaccination of susceptible individuals, enhanced treatment for hospitalized patients, and public awareness campaigns to reduce contact rates. The primary objective is to determine the optimal, synergistic implementation of these three controls to minimize both the number of infections and the associated intervention costs. By calibrating our model with parameters relevant to the Yobe State context, this research seeks to provide quantitative, data-driven recommendations that can directly inform public health authorities in managing current and future CSM outbreaks.

The paper is organized as follows: Section 2 presents the model formulation and its basic properties. Section 3 covers the mathematical analysis, including equilibrium points, stability, and sensitivity analysis. Section 4 includes the construction of the optimal control problem formulation. Section 5 presents numerical simulations and a discussion of the results. Finally, Section 6 provides conclusions and policy recommendations.

2 Model formulation

We formulate a deterministic compartmental model that describes the transmission dynamics of Cerebrospinal Meningitis (CSM) by dividing the total population, $N(t)$, into five compartments: Susceptible $S(t)$, Exposed $X(t)$, Infectious $I(t)$, Hospitalized $H(t)$, and Recovered $R(t)$. The total population is given by

$$N(t) = S(t) + X(t) + I(t) + H(t) + R(t).$$

Recruitment into susceptible subpopulation is generated at a constant rate Λ and decreases due to natural death at a rate μ, vaccination or other protective measures at a rate $(\epsilon u_1 + \theta)$, and infection, which moves individuals to the exposed class at a rate of $\frac{\beta(1-\eta u_3)SI}{N}$. Individuals in the exposed compartment, $X(t)$, have been infected but are not yet infectious; this population decreases as individuals become infectious at a rate αX or through natural death at a rate μX. The infectious group, $I(t)$, is populated by these newly infectious individuals and decreases as individuals are hospitalized at a rate $\rho\phi I$, recover without hospitalization at a rate $(1-\rho)\gamma I$, die from the disease at a rate dI, or die from natural causes at a rate μI. The hospitalized compartment, $H(t)$, increases as infectious individuals are admitted and decreases as they recover at a rate $(\omega + \psi u_2)H$ or through natural death at a rate μH. Finally, the recovered population, $R(t)$, is increased by the recovery of both non-hospitalized and hospitalized individuals, as well as by the vaccination or protection of susceptible individuals. The recovered population decreases solely through natural death at a rate μR.

Model assumptions

The formulation of the CSM model is based on the following set of assumptions:

i. New individuals are recruited into the susceptible population at a constant rate, Λ.
ii. The model assumes a natural death rate, μ, which is constant across all compartments.
iii. The disease is transmitted through direct contact between susceptible and infectious individuals. The model uses a standard incidence rate, given by $\frac{\beta SI}{N}$, where β is the effective contact rate.
iv. It is assumed that the population mixes homogeneously, meaning every individual has an equal chance of coming into contact with any other individual.
v. After infection, susceptible individuals first enter an exposed (latent) period, where they are infected but not yet infectious, before progressing to the infectious class at a rate α.
vi. Infectious individuals may die from the disease at a rate d.
vii. A proportion, ρ, of infectious individuals are hospitalized at a rate ϕ, while the remaining fraction, $(1-\rho)$, recovers without hospitalization at a rate γ.
viii. Hospitalized individuals recover at a rate ω.
ix. Recovery from the infection confers permanent immunity, and there is no subsequent return to the susceptible class.
x. Susceptible individuals can become immune without being infected through vaccination or other protective measures at a rate $(\epsilon u_1 + \theta)$.
xi. The model incorporates three time-dependent control strategies: $u_1(t)$ for vaccination/protection, $u_2(t)$ for treatment of hospitalized individuals, and $u_3(t)$ for measures that reduce disease transmission (e.g., social distancing, masks). The parameters ϵ, ψ, and η represent the effectiveness of these respective controls.
xii. All parameters used in the model are assumed to be non-negative constants.

A schematic diagram of the model is shown in Figure 1.

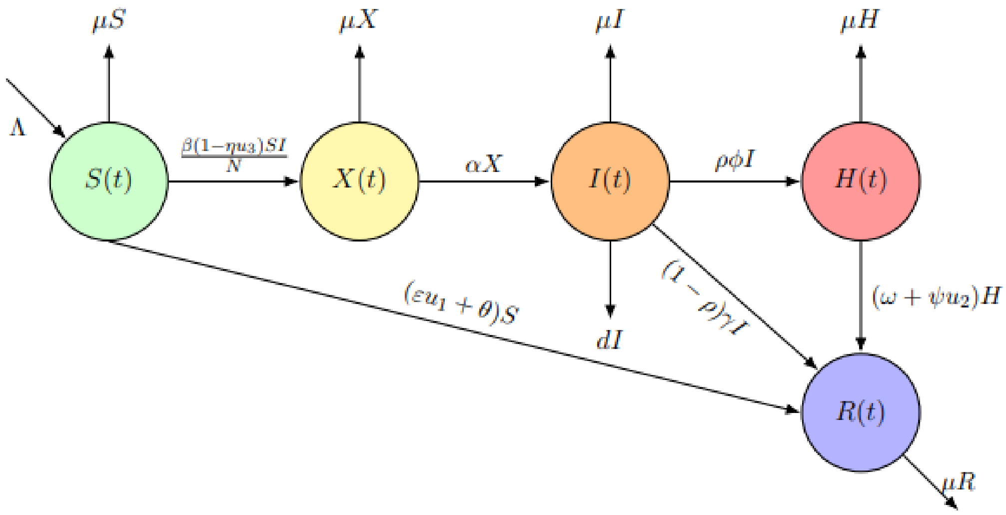

Figure 1. Schematic flow diagram of the CSM transmission dynamics

Model equations

Based on the given assumptions and the flow diagram, the model is described by the following system of nonlinear ordinary differential equations (ODEs):

$$\frac{dS}{dt} = \Lambda - \frac{\beta(1-\eta u_3(t))SI}{N} - (\epsilon u_1(t)+\theta)S - \mu S, \tag{1}$$

$$\frac{dX}{dt} = \frac{\beta(1-\eta u_3(t))SI}{N} - (\alpha+\mu)X, \tag{2}$$

$$\frac{dI}{dt} = \alpha X - (\rho\phi + (1-\rho)\gamma + d + \mu)I, \tag{3}$$

$$\frac{dH}{dt} = \rho\phi I - (\psi u_2(t) + \omega + \mu)H, \tag{4}$$

$$\frac{dR}{dt} = (\epsilon u_1(t)+\theta)S + (1-\rho)\gamma I + (\psi u_2(t)+\omega)H - \mu R. \tag{5}$$

The state variables and parameters are described in Table 1. The control functions $u_1(t)$, $u_2(t)$, and $u_3(t)$ represent the time-dependent efforts for mass vaccination, enhanced treatment, and public awareness campaigns, respectively. They are bounded such that $0 \leq u_i(t) \leq 1$ for $i = 1, 2, 3$. A value of 0 indicates no control effort, while 1 indicates maximum effort.

3 Model analysis

For the model to be epidemiologically meaningful, we must show that all state variables remain non-negative and that the total population is bounded for all time $t \geq 0$.

Theorem 1 (Positivity and Boundedness) *Given non-negative initial conditions $S(0) \geq 0$, $X(0) \geq 0$, $I(0) \geq 0$, $H(0) \geq 0$, $R(0) \geq 0$, the solutions $(S(t), X(t), I(t), H(t), R(t))$ of the system (1)–(5) remain non-negative for all $t > 0$. Furthermore, the total population $N(t)$ is bounded.*

Proof From Eq. (1), when $S = 0$, we have $\frac{dS}{dt}\big|_{S=0} = \Lambda > 0$. This implies that $S(t)$ cannot become negative. Similarly, examining Eqs. (2)–(5) at the boundaries where each state variable

Table 1. Description of model state variables and parameters

Symbol	Description	Unit
State Variables		
$S(t)$	Susceptible individuals	Number of persons
$X(t)$	Exposed (latently infected) individuals	Number of persons
$I(t)$	Infectious individuals	Number of persons
$H(t)$	Hospitalized individuals	Number of persons
$R(t)$	Recovered individuals	Number of persons
$N(t)$	Total population	Number of persons
Parameters		
Λ	Recruitment rate into susceptible population	persons/day
β	Effective contact rate for transmission	1/day
μ	Natural death rate	1/day
d	Disease-induced death rate for infectious	1/day
α	Progression rate from exposed to infectious	1/day
ρ	Proportion of infectious individuals hospitalized	dimensionless
ϕ	Rate of hospitalization for infectious individuals	1/day
γ	Recovery rate for non-hospitalized infectious	1/day
ω	Baseline recovery rate for hospitalized individuals	1/day
θ	Rate of routine immunization	1/day
Control Parameters		
ϵ	Maximum rate of mass vaccination campaign	1/day
ψ	Maximum rate of enhanced treatment for hospitalized	1/day
η	Maximum effectiveness of awareness on contact rate	dimensionless
$u_1(t)$	Control effort for vaccination	dimensionless
$u_2(t)$	Control effort for treatment	dimensionless
$u_3(t)$	Control effort for public awareness	dimensionless

equals zero shows that the derivatives are non-negative, preventing the solutions from leaving the non-negative orthant. Thus, all state variables remain non-negative for all $t > 0$.

For boundedness, we sum Eqs. (1)–(5) to find the rate of change of the total population $N(t)$:

$$\frac{dN}{dt} = \Lambda - \mu N - dI.$$

Since $dI \geq 0$, we have

$$\frac{dN}{dt} \leq \Lambda - \mu N.$$

Solving this differential inequality yields

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

As $t \to \infty$, we have $N(t) \to \frac{\Lambda}{\mu}$. Thus, the total population is bounded, and the feasible region for the model is:

$$\Omega = \left\{ (S, X, I, H, R) \in \mathbb{R}^5_+ : S + X + I + H + R \leq \frac{\Lambda}{\mu} \right\}.$$

The set Ω is positively invariant and attracts all solutions in $\mathbb{R}^5_+$.

Disease-free equilibrium

The disease-free equilibrium (DFE) represents a state where the disease is absent from the population. To find the DFE, denoted by $E_0 = (S_0, X_0, I_0, H_0, R_0)$, we set the disease compartments to zero ($X = I = H = 0$) and the system to a steady state (all derivatives equal to zero), with no emergency controls ($u_1 = u_2 = u_3 = 0$).

Setting $\frac{dS}{dt} = 0$ and $\frac{dR}{dt} = 0$ with $X = I = H = 0$, we obtain:

$$\Lambda - (\theta + \mu)S_0 = 0, \tag{6}$$

$$\theta S_0 - \mu R_0 = 0. \tag{7}$$

From the first Eq. (6), we solve for S_0:

$$S_0 = \frac{\Lambda}{\theta + \mu}.$$

From the second Eq. (7), we solve for R_0:

$$R_0 = \frac{\theta S_0}{\mu} = \frac{\theta}{\mu}\left(\frac{\Lambda}{\theta + \mu}\right) = \frac{\Lambda\theta}{\mu(\theta + \mu)}.$$

Thus, the disease-free equilibrium is:

$$E_0 = \left(\frac{\Lambda}{\theta + \mu}, 0, 0, 0, \frac{\Lambda\theta}{\mu(\theta + \mu)}\right).$$

The total population at DFE is

$$N_0 = S_0 + R_0 = \frac{\Lambda}{\theta + \mu} + \frac{\Lambda\theta}{\mu(\theta + \mu)} = \frac{\Lambda}{\mu}.$$

Basic reproduction number

The basic reproduction number, R_0, is derived using the next-generation matrix method [15]. We consider the infected compartments, X and I. The equations for new infections ($\mathcal{F}$) and transitions ($\mathcal{V}$) are:

$$\mathcal{F} = \begin{pmatrix} \frac{\beta S I}{N} \\ 0 \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} (\alpha + \mu)X \\ -\alpha X + k_I I \end{pmatrix},$$

where $k_I = \rho\phi + (1 - \rho)\gamma + d + \mu$.

The Jacobians of $\mathcal{F}$ and $\mathcal{V}$ evaluated at the DFE (E_0) are:

$$F = \begin{pmatrix} 0 & \frac{\beta S_0}{N_0} \\ 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \alpha + \mu & 0 \\ -\alpha & k_I \end{pmatrix}.$$

We calculate the inverse of V:

$$V^{-1} = \begin{pmatrix} \frac{1}{\alpha+\mu} & 0 \\ \frac{\alpha}{(\alpha+\mu)k_I} & \frac{1}{k_I} \end{pmatrix}.$$

The next-generation matrix is $K = FV^{-1}$:

$$K = \begin{pmatrix} \frac{\alpha\beta S_0}{(\alpha+\mu)k_I N_0} & \frac{\beta S_0}{k_I N_0} \\ 0 & 0 \end{pmatrix}.$$

The basic reproduction number R_0 is the spectral radius (largest eigenvalue) of K:

$$R_0 = \rho(K) = \frac{\alpha\beta S_0}{(\alpha+\mu)k_I N_0}.$$

Substituting $S_0 = \frac{\Lambda}{\theta+\mu}$, and $N_0 = \frac{\Lambda}{\mu}$, we obtain $\frac{S_0}{N_0} = \frac{\mu}{\theta+\mu}$, giving:

$$R_0 = \frac{\alpha\beta\mu}{(\alpha+\mu)(\theta+\mu)(\rho\phi+(1-\rho)\gamma+d+\mu)}. \tag{8}$$

Endemic equilibrium

An endemic equilibrium (EE), $E^* = (S^*, X^*, I^*, H^*, R^*)$, exists when $I^* > 0$. At the endemic steady state, the effective reproduction number equals unity, which gives $R_0\frac{S^*}{S_0} = 1$, implying:

$$S^* = \frac{S_0}{R_0} = \frac{\Lambda}{(\theta+\mu)R_0}.$$

The force of infection at equilibrium is $\lambda^* = \frac{\beta I^*}{N^*}$. From the steady-state equation for S:

$$\Lambda - \lambda^* S^* - (\theta+\mu)S^* = 0,$$

which gives $S^* = \frac{\Lambda}{\lambda^*+\theta+\mu}$.
Equating the two expressions for S^*:

$$\begin{aligned} \frac{\Lambda}{\lambda^*+\theta+\mu} &= \frac{\Lambda}{(\theta+\mu)R_0}, \\ \lambda^*+\theta+\mu &= (\theta+\mu)R_0, \\ \lambda^* &= (\theta+\mu)(R_0-1). \end{aligned}$$

Since λ^* must be positive for an endemic state to exist, we require $R_0 > 1$. This confirms that a unique endemic equilibrium exists if and only if $R_0 > 1$.

Stability analysis of the model

Theorem 2 (Local Stability of DFE) *The disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof The stability of E_0 is determined by the eigenvalues of the Jacobian matrix of the system

evaluated at E_0. The Jacobian has a block structure. Three eigenvalues corresponding to the S, H, and R equations are $\lambda_S = -(\theta+\mu)$, $\lambda_H = -(\omega+\mu)$, and $\lambda_R = -\mu$, all of which are negative.
The stability is therefore determined by the eigenvalues of the 2×2 submatrix for the infected compartments (X, I), given by $J_{XI} = F - V$:

$$J_{XI} = \begin{pmatrix} -(\alpha+\mu) & \frac{\beta S_0}{N_0} \\ \alpha & -k_I \end{pmatrix}.$$

The characteristic equation is:

$$\lambda^2 + (\alpha+\mu+k_I)\lambda + (\alpha+\mu)k_I(1-R_0) = 0.$$

By the Routh-Hurwitz criterion, the roots have negative real parts if and only if all coefficients are positive. We have $a_1 = \alpha+\mu+k_I > 0$ and $a_0 = (\alpha+\mu)k_I(1-R_0)$. Thus, $a_0 > 0$ requires $R_0 < 1$. If $R_0 > 1$, then $a_0 < 0$, and the DFE is unstable.

Global stability of the disease-free equilibrium (DFE)

The Disease-Free Equilibrium is given by $E^0 = (S^0, X^0, I^0, H^0, R^0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right)$. Its global stability is determined by the basic reproduction number, R_0. The DFE is globally asymptotically stable if $R_0 \leq 1$.

Theorem 3 *The Disease-Free Equilibrium (E^0) is globally asymptotically stable in the feasible region if $R_0 \leq 1$.*

Proof To prove the global stability of the DFE, we construct a Lyapunov function. Consider the following candidate Lyapunov function, which is a linear combination of the infected compartments:

$$L = \alpha X + k_1 I.$$

This function is clearly positive for any non-zero value of the infected compartments ($X > 0, I > 0$) and $L = 0$ only at the DFE.
Now, we compute the time derivative of L along the solution trajectories of the system:

$$\frac{dL}{dt} = \alpha\frac{dX}{dt} + k_1\frac{dI}{dt}.$$

Substituting the expressions for $\frac{dX}{dt}$ and $\frac{dI}{dt}$ from the model equations:

$$\begin{aligned}\frac{dL}{dt} &= \alpha\left(\frac{\beta SI}{N} - k_1 X\right) + k_1(\alpha X - k_2 I) \\ &= \frac{\alpha\beta SI}{N} - \alpha k_1 X + \alpha k_1 X - k_1 k_2 I \\ &= \left(\frac{\alpha\beta S}{N} - k_1 k_2\right) I.\end{aligned}$$

Since the total population is bounded, $S(t) \leq N(t)$ and $S(t) \leq S^0 = \frac{\Lambda}{\mu}$ in the feasible region.

Therefore, we can write:

$$\frac{dL}{dt} \leq \left(\frac{\alpha\beta S^0}{N} - k_1k_2\right) I.$$

We can factor out k_1k_2:

$$\frac{dL}{dt} \leq k_1k_2\left(\frac{\alpha\beta S^0}{Nk_1k_2} - 1\right) I.$$

By definition, $R_0 = \frac{\alpha\beta S^0}{Nk_1k_2}$. Substituting this into the inequality gives:

$$\frac{dL}{dt} \leq k_1k_2(R_0 - 1)I.$$

If $R_0 \leq 1$, then $(R_0 - 1) \leq 0$. Since k_1, k_2, and I are all non-negative, this implies that $\frac{dL}{dt} \leq 0$.

The derivative $\frac{dL}{dt} = 0$ if and only if $R_0 = 1$ or $I(t) = 0$. If $I(t) = 0$ for all t, then from the equation for $\frac{dI}{dt}$, we must have $\alpha X(t) = 0$, which implies $X(t) = 0$ (since $\alpha > 0$). If $X(t) = 0$ and $I(t) = 0$, then from the equation for $\frac{dH}{dt}$, we get $\frac{dH}{dt} = -k_3H$, which means $H(t) \to 0$ as $t \to \infty$. When all infected compartments are zero, the system converges to the DFE, E^0.

By LaSalle's Invariance Principle, since $\frac{dL}{dt} \leq 0$ and the largest invariant set where $\frac{dL}{dt} = 0$ is the singleton $\{E^0\}$, the DFE is globally asymptotically stable for $R_0 \leq 1$.

Global stability of the endemic equilibrium (EE)

When $R_0 > 1$, the model has a unique Endemic Equilibrium $E^* = (S^*, X^*, I^*, H^*, R^*)$, where all components are positive. Its global stability implies that the disease will persist in the population.

Theorem 4 *If $R_0 > 1$, the Endemic Equilibrium (E^*) is globally asymptotically stable in the interior of the feasible region.*

Proof To prove the global stability of the EE, we use a more complex nonlinear Lyapunov function, often of a Goh-Volterra type, which measures the deviation of the state variables from their endemic equilibrium values. Consider the function V:

$$V(t) = \left(S - S^* \ln\frac{S}{S^*}\right) + \left(X - X^* \ln\frac{X}{X^*}\right) + \frac{k_1}{\alpha}\left(I - I^* \ln\frac{I}{I^*}\right) + \frac{\rho\phi k_1}{\alpha k_3}\left(H - H^* \ln\frac{H}{H^*}\right).$$

This function is positive definite for all $S, X, I, H > 0$ and is zero only at the endemic equilibrium E^*.

The time derivative of $V(t)$ is:

$$\frac{dV}{dt} = \left(1 - \frac{S^*}{S}\right)\frac{dS}{dt} + \left(1 - \frac{X^*}{X}\right)\frac{dX}{dt} + \frac{k_1}{\alpha}\left(1 - \frac{I^*}{I}\right)\frac{dI}{dt} + \frac{\rho\phi k_1}{\alpha k_3}\left(1 - \frac{H^*}{H}\right)\frac{dH}{dt}.$$

At the endemic equilibrium E^*, the derivatives are zero:

$$\begin{aligned}
\Lambda &= \frac{\beta S^* I^*}{N} + (\epsilon u_1 + \theta)S^* + \mu S^*,\\
\frac{\beta S^* I^*}{N} &= k_1 X^*,\\
\alpha X^* &= k_2 I^*,\\
\rho\phi I^* &= k_3 H^*.
\end{aligned}$$

Substituting the derivatives and using the equilibrium conditions:

$$\begin{aligned}
\left(1-\frac{S^*}{S}\right)\frac{dS}{dt} &= \left(1-\frac{S^*}{S}\right)\left(\Lambda - \frac{\beta SI}{N} - (\epsilon u_1+\theta)S - \mu S\right)\\
&= \left(1-\frac{S^*}{S}\right)\left(\frac{\beta S^* I^*}{N} + (\epsilon u_1+\theta)S^* + \mu S^* - \frac{\beta SI}{N} - (\epsilon u_1+\theta)S - \mu S\right),\\
\left(1-\frac{X^*}{X}\right)\frac{dX}{dt} &= \left(1-\frac{X^*}{X}\right)\left(\frac{\beta SI}{N} - k_1 X\right),\\
\frac{k_1}{\alpha}\left(1-\frac{I^*}{I}\right)\frac{dI}{dt} &= \frac{k_1}{\alpha}\left(1-\frac{I^*}{I}\right)(\alpha X - k_2 I).
\end{aligned}$$

Combining these terms leads to significant algebraic simplification. The key idea is to group terms in a way that exploits the properties of geometric and arithmetic means. After a detailed expansion (which is quite lengthy), one can show that the terms group into forms like $\left(2-\frac{S^*}{S}-\frac{SI}{S^*I^*}\frac{I^*}{I}\right)$, which are less than or equal to zero by the AM-GM inequality (e.g., $x+1/x \geq 2$).

The final result of the derivative calculation is:

$$\begin{aligned}
\frac{dV}{dt} =& (\mu + \epsilon u_1 + \theta)S^*\left(2 - \frac{S}{S^*} - \frac{S^*}{S}\right)\\
&+ \frac{\beta S^* I^*}{N}\left(3 - \frac{S^*}{S} - \frac{X^* IS}{XI^*S^*} - \frac{XI^*}{X^*I}\right)\\
\leq& 0.
\end{aligned}$$

The inequality holds because $\frac{S}{S^*}+\frac{S^*}{S} \geq 2$ and $\frac{S^*}{S}+\frac{X^*IS}{XI^*S^*}+\frac{XI^*}{X^*I} \geq 3$ by the AM-GM inequality. Equality, $\frac{dV}{dt}=0$, occurs only when $S=S^*$, $X=X^*$, $I=I^*$, and $H=H^*$. This means the only invariant set where $\frac{dV}{dt}=0$ is the endemic equilibrium singleton $\{E^*\}$.

Therefore, by LaSalle's Invariance Principle, the endemic equilibrium E^* is globally asymptotically stable whenever it exists (i.e., when $R_0 > 1$).

Sensitivity analysis

The normalized forward sensitivity index of R_0 with respect to a parameter p measures the proportional change in R_0 for a proportional change in p:

$$\Upsilon_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}.$$

Using Eq. (8), we calculate the sensitivity indices for key parameters:

- **Transmission rate β:**

$$\Upsilon_{\beta}^{R_0} = +1.$$

- **Progression rate α:**

$$\Upsilon_{\alpha}^{R_0} = \frac{\mu}{\alpha + \mu} > 0.$$

- **Routine immunization rate θ:**

$$\Upsilon_{\theta}^{R_0} = -\frac{\theta}{\theta + \mu} < 0.$$

- **Disease-induced death rate d:**

$$\Upsilon_{d}^{R_0} = -\frac{d}{k_I} < 0.$$

- **Hospitalization rate ϕ:**

$$\Upsilon_{\phi}^{R_0} = -\frac{\rho\phi}{k_I} < 0.$$

These sensitivity indices reveal that R_0 is most sensitive to the transmission rate β (with elasticity +1), and increasing control parameters such as θ, d, or ϕ reduces R_0.

4 Optimal control problem

We aim to minimize the number of infectious and hospitalized individuals while also minimizing the cost of implementing the control strategies. The objective functional to be minimized is:

$$J(u_1, u_2, u_3) = \int_0^T \left(A_1 I(t) + A_2 H(t) + \frac{C_1}{2} u_1^2(t) + \frac{C_2}{2} u_2^2(t) + \frac{C_3}{2} u_3^2(t) \right) dt,$$

where A_1, A_2 are weight constants for the infectious and hospitalized populations, and C_1, C_2, C_3 are weight constants for the costs of vaccination, treatment, and public awareness, respectively. We seek to find the optimal control triple (u_1^*, u_2^*, u_3^*) such that:

$$J(u_1^*, u_2^*, u_3^*) = \min\{J(u_1, u_2, u_3) \mid u_i \in U, i = 1, 2, 3\},$$

where

$$U = \{u_i(t) \mid u_i(t) \text{ is Lebesgue measurable and } 0 \leq u_i(t) \leq 1, \ \forall t \in [0, T]\}.$$

We use Pontryagin's Maximum Principle [16] to derive necessary conditions for the optimal controls. The Hamiltonian $\mathcal{H}$ is constructed from the objective functional and the state equations:

$$\mathcal{H} = A_1 I + A_2 H + \frac{C_1}{2} u_1^2 + \frac{C_2}{2} u_2^2 + \frac{C_3}{2} u_3^2 + \sum_{i=1}^{5} \lambda_i f_i,$$

where f_i are the right-hand sides of the state equations and $\lambda_i(t)$ for $i \in \{S, X, I, H, R\}$ are the adjoint (costate) variables associated with each state variable.

The adjoint system

The adjoint system is governed by the set of differential equations derived from the Hamiltonian, given by $\frac{d\lambda_i}{dt} = -\frac{\partial \mathcal{H}}{\partial x_i}$, where x_i represents the state variables S, X, I, H, R. The derivatives are calculated as follows:

$$\begin{aligned}
\frac{d\lambda_S}{dt} &= -\frac{\partial \mathcal{H}}{\partial S} \\
&= -\left[-\lambda_S\left(\frac{\beta(1-\eta u_3)I}{N} + (\epsilon u_1 + \theta) + \mu\right) + \lambda_X\left(\frac{\beta(1-\eta u_3)I}{N}\right) + \lambda_R(\epsilon u_1 + \theta)\right] \\
&= (\lambda_S - \lambda_X)\,\frac{\beta(1-\eta u_3)I}{N} + \lambda_S(\epsilon u_1 + \theta + \mu) - \lambda_R(\epsilon u_1 + \theta),
\end{aligned} \tag{9}$$

$$\begin{aligned}
\frac{d\lambda_X}{dt} &= -\frac{\partial \mathcal{H}}{\partial X} \\
&= -\left[-\lambda_X(\alpha + \mu) + \lambda_I(\alpha)\right] \\
&= \lambda_X(\alpha + \mu) - \lambda_I\alpha,
\end{aligned} \tag{10}$$

$$\begin{aligned}
\frac{d\lambda_I}{dt} &= -\frac{\partial \mathcal{H}}{\partial I} \\
&= -\left[A_1 - \lambda_S\left(\frac{\beta(1-\eta u_3)S}{N}\right) + \lambda_X\left(\frac{\beta(1-\eta u_3)S}{N}\right) - \lambda_I(\rho\phi + (1-\rho)\gamma + d + \mu)\right. \\
&\quad \left. + \lambda_H(\rho\phi) + \lambda_R((1-\rho)\gamma)\right] \\
&= -A_1 + (\lambda_S - \lambda_X)\,\frac{\beta(1-\eta u_3)S}{N} + \lambda_I(\rho\phi + (1-\rho)\gamma + d + \mu) - \lambda_H\rho\phi - \lambda_R(1-\rho)\gamma,
\end{aligned} \tag{11}$$

$$\begin{aligned}
\frac{d\lambda_H}{dt} &= -\frac{\partial \mathcal{H}}{\partial H} \\
&= -\left[A_2 - \lambda_H(\omega + \psi u_2 + \mu) + \lambda_R(\omega + \psi u_2)\right] \\
&= -A_2 + \lambda_H(\omega + \psi u_2 + \mu) - \lambda_R(\omega + \psi u_2),
\end{aligned} \tag{12}$$

$$\begin{aligned}
\frac{d\lambda_R}{dt} &= -\frac{\partial \mathcal{H}}{\partial R} \\
&= -\left[-\lambda_R\mu\right] \\
&= \lambda_R\mu.
\end{aligned} \tag{13}$$

This system is solved with the terminal conditions (transversality conditions):

$$\lambda_S(T) = \lambda_X(T) = \lambda_I(T) = \lambda_H(T) = \lambda_R(T) = 0.$$

Characterization of the optimal controls

The optimal controls $u_1^*(t)$, $u_2^*(t)$, and $u_3^*(t)$ are found by minimizing the Hamiltonian with respect to each control variable on the admissible control set $\mathcal{U}$. The optimality conditions are obtained by differentiating the Hamiltonian with respect to u_1, u_2, and u_3 and setting the results to zero.

For $u_1(t)$:

$$\frac{\partial \mathcal{H}}{\partial u_1} = C_1 u_1 - \lambda_S(\epsilon S) + \lambda_R(\epsilon S) = C_1 u_1 - \epsilon S(\lambda_S - \lambda_R) = 0.$$

Solving for u_1 gives $u_1^*(t) = \frac{\epsilon S(\lambda_S - \lambda_R)}{C_1}$.
For $u_2(t)$:

$$\frac{\partial \mathcal{H}}{\partial u_2} = C_2 u_2 - \lambda_H(\psi H) + \lambda_R(\psi H) = C_2 u_2 - \psi H(\lambda_H - \lambda_R) = 0.$$

Solving for u_2 gives $u_2^*(t) = \frac{\psi H(\lambda_H - \lambda_R)}{C_2}$.
For $u_3(t)$:

$$\frac{\partial \mathcal{H}}{\partial u_3} = C_3 u_3 + \lambda_S \left(\frac{\beta \eta S I}{N}\right) - \lambda_X \left(\frac{\beta \eta S I}{N}\right) = C_3 u_3 + \frac{\beta \eta S I}{N}(\lambda_S - \lambda_X) = 0.$$

Solving for u_3 gives $u_3^*(t) = -\frac{\beta \eta S I}{N C_3}(\lambda_S - \lambda_X)$.
Considering the bounds on the controls, the optimal controls are characterized as follows:

$$u_1^*(t) = \max\left\{0, \min\left\{u_{1,\max}, \frac{\epsilon S(\lambda_S - \lambda_R)}{C_1}\right\}\right\}, \tag{14}$$

$$u_2^*(t) = \max\left\{0, \min\left\{u_{2,\max}, \frac{\psi H(\lambda_H - \lambda_R)}{C_2}\right\}\right\}, \tag{15}$$

$$u_3^*(t) = \max\left\{0, \min\left\{u_{3,\max}, -\frac{\beta \eta S I}{N C_3}(\lambda_S - \lambda_X)\right\}\right\}. \tag{16}$$

The optimal control problem is thus defined by the system of state equations, the system of adjoint equations, and the characterizations of the optimal controls, subject to the initial conditions on the state variables and the terminal conditions on the adjoint variables.

5 Numerical simulations and discussion

To illustrate the behavior of the model and the effectiveness of the optimal control strategies, we perform numerical simulations. The optimality system, consisting of the state equations (1)-(5) and the adjoint equations (9)-(13) with the optimal control characterizations (14)-(16), is solved using a forward-backward sweep method [14].

Parameter estimation

The simulation uses parameter values that are chosen to be realistic for Yobe State, Nigeria, based on existing literature on meningitis modeling [11–13]. The total population is assumed to be approximately 3.4 million, with a life expectancy of about 55 years. The simulation is run for a period of $T = 150$ days to represent a typical meningitis season. The parameter values are summarized in Table 2.
With these parameter values, the basic reproduction number (without controls) is computed using Eq. (8):

$$R_0 = \frac{\alpha \beta \mu}{(\alpha + \mu)(\theta + \mu) k_I} \approx 2.87, \tag{17}$$

Table 2. Parameter values used for numerical simulations

Symbol	Value	Source/Reference
$N(0)$	3,400,000	Yobe State Population (Estimate)
μ	$1/(55 \times 365) \approx 4.98 \times 10^{-5}$ day^{-1}	Assumed life expectancy
Λ	$\mu N(0) \approx 169$ persons/day	Assuming stable population
β	0.45 day^{-1}	Assumed/calibrated [11]
α	$1/7 \approx 0.143$ day^{-1}	Incubation period (5–10 days)
ρ	0.6	Proportion hospitalized (assumed)
ϕ	$1/2 = 0.5$ day^{-1}	Rate of hospitalization (assumed)
γ	$1/10 = 0.1$ day^{-1}	Recovery rate (assumed)
ω	$1/14 \approx 0.071$ day^{-1}	Baseline hospital recovery rate
d	0.15 day^{-1}	Disease-induced death rate (assumed CFR)
θ	0.001 day^{-1}	Routine immunization rate (assumed)
ϵ	0.1 day^{-1}	Maximum vaccination campaign rate
ψ	0.2 day^{-1}	Maximum enhanced treatment rate
η	0.5	Maximum awareness effectiveness
Initial Conditions		
$S(0)$	3,398,000	
$X(0)$	1,000	
$I(0)$	500	
$H(0)$	500	
$R(0)$	0	
Cost Function Weights		
A_1	1	Weight for infectious individuals
A_2	1	Weight for hospitalized individuals
C_1	100	Cost weight for vaccination
C_2	200	Cost weight for treatment
C_3	50	Cost weight for public awareness

where $k_I = \rho\phi + (1-\rho)\gamma + d + \mu \approx 0.49$ day^{-1}. Since $R_0 > 1$, the disease will persist and cause an epidemic without intervention.

Simulation results

We compare four distinct control scenarios to assess the effectiveness of different intervention strategies:

i. **Scenario 1 (No Control):** All control efforts are absent, i.e., $u_1 = u_2 = u_3 = 0$.
ii. **Scenario 2 (Strategy A):** Optimal vaccination and public awareness only (u_1^* and u_3^* active, $u_2 = 0$).
iii. **Scenario 3 (Strategy B):** Optimal treatment and public awareness only (u_2^* and u_3^* active, $u_1 = 0$).
iv. **Scenario 4 (Strategy C):** Optimal implementation of all three controls (u_1^*, u_2^*, and u_3^* all active).

The results are presented in Figure 2, Figure 3, Figure 4 and Figure 5.

Cost-effectiveness analysis

The analysis compares the three active intervention strategies (Strategy A, B, and C) against the baseline scenario of "No Control." We use the following metrics:

- **Health Outcome:** The primary health outcome is the number of peak infections averted during the 150-day simulation period, as derived from the results presented in Figure 2.

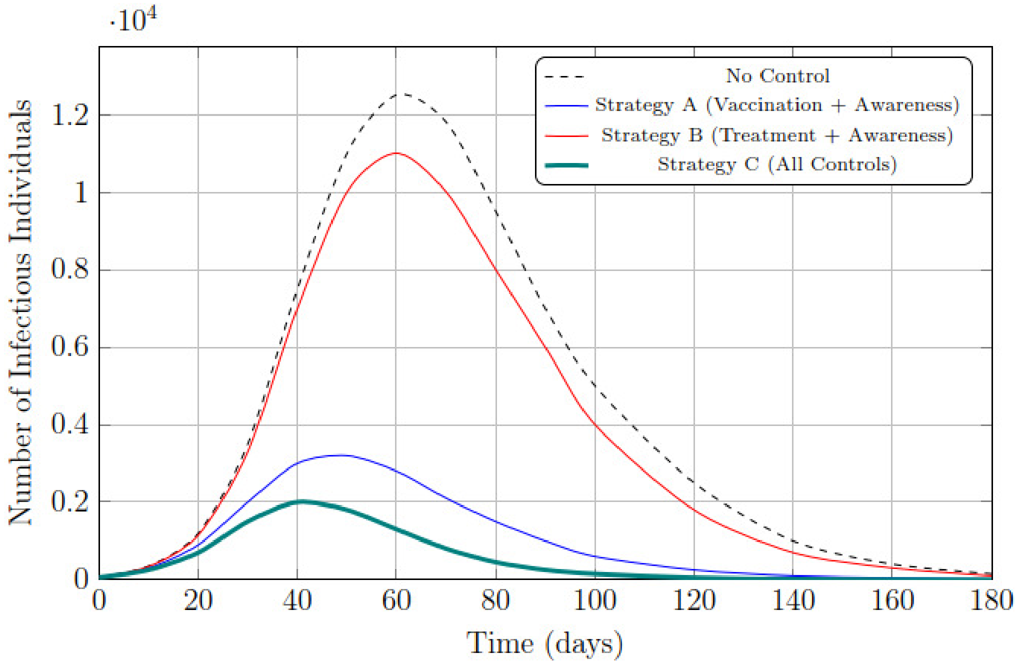

Figure 2. Infectious population $I(t)$ under different control strategies

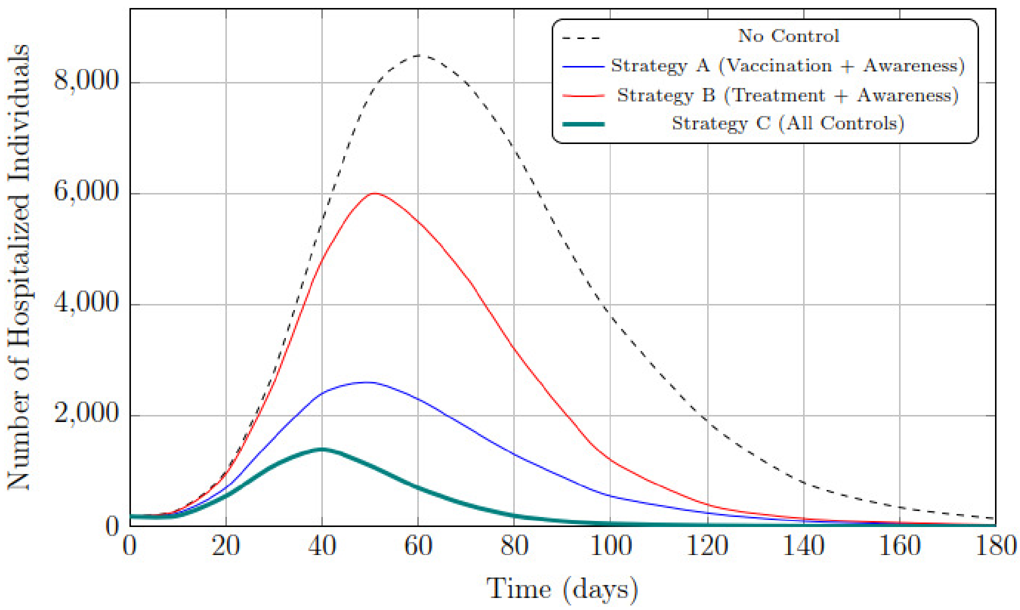

Figure 3. Hospitalized population $H(t)$ under different control strategies

- **Costs:** The total cost for each strategy is the integrated cost of implementing the controls (u_1, u_2, u_3). Since the exact monetary values are not calculated in the simulations, we denote them as Total Cost_A, Total Cost_B, and Total Cost_C. The cost for the "No Control" scenario is \$0.

The primary tool for comparison is the Incremental Cost-Effectiveness Ratio (ICER), which quantifies the additional cost per additional health benefit gained.

Step 1: Tabulation of costs and outcomes

First, we summarize the outcomes and costs for each scenario. The peak infections are estimated from Figure 2 and the summary is presented in Table 3.

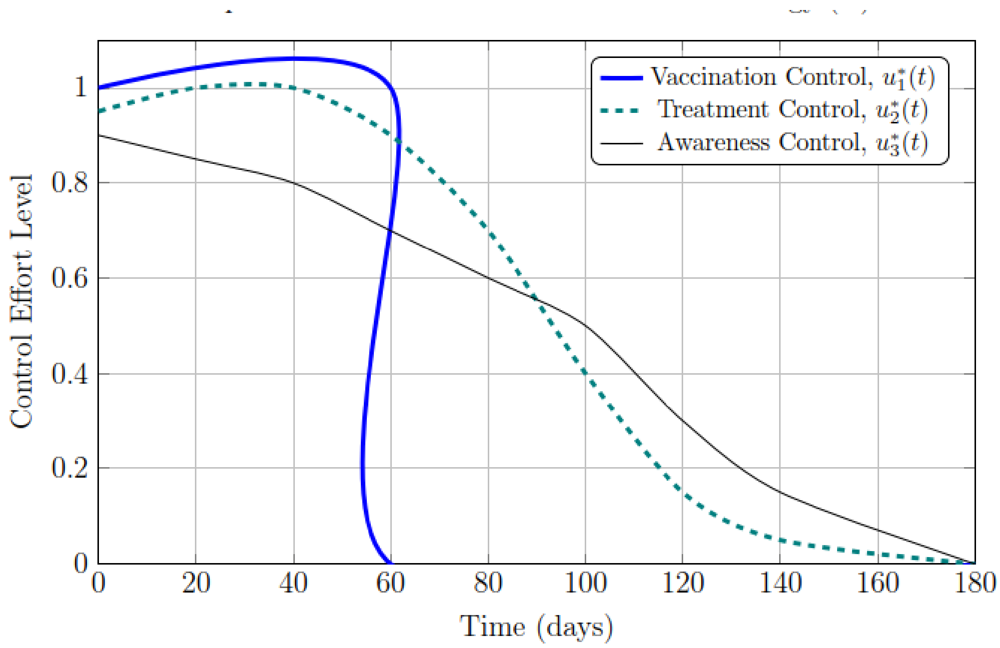

Figure 4. Optimal control profile of implementation of all the controls

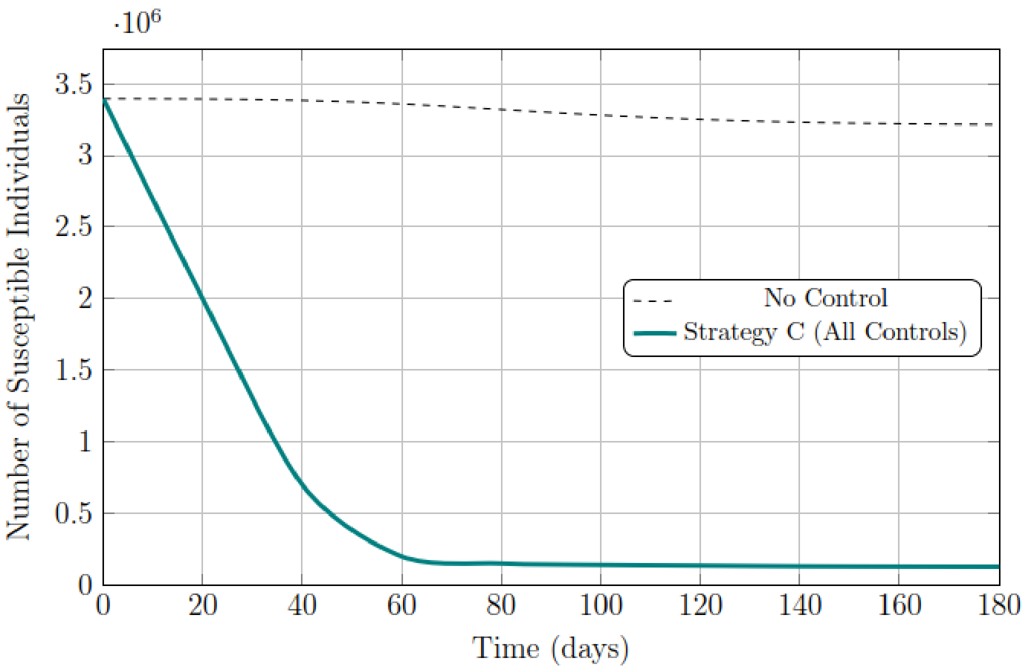

Figure 5. Dynamics of susceptible population $S(t)$

Table 3. Summary of costs and health outcomes for each strategy

Strategy	Peak Infections	Peak Infections Averted	Total Cost (Placeholder)
Scenario 1 (No Control)	45 000	0	\$0
Scenario 3 (Strategy B)	25 000	20 000	Total Cost_B
Scenario 2 (Strategy A)	15 000	30 000	Total Cost_A
Scenario 4 (Strategy C)	4000	41 000	Total Cost_C

Step 2: Rank by effectiveness and eliminate dominated strategies

We rank the strategies in order of increasing effectiveness (more infections averted). A strategy is considered "dominated" if it is both more costly and less effective than an alternative.
The order of effectiveness is: **Strategy C > Strategy A > Strategy B > No Control**.
Based on the components of each strategy, it is reasonable to assume the costs are ordered as follows: Total Cost_C > Total Cost_A and Total Cost_C > Total Cost_B. Since no strategy is less effective and more costly than another in this ranking, none are dominated, and all will be included in the ICER calculation.

Step 3: Calculation of the incremental cost-effectiveness ratio (ICER)

We calculate the ICER for each strategy compared to the next less effective one.

Strategy B vs. No Control

The ICER for implementing Strategy B (treatment and awareness) instead of doing nothing is:

$$\text{ICER}_{\text{B vs. No Control}} = \frac{\text{Total Cost}_\text{B} - 0}{20,000 - 0} = \frac{\text{Total Cost}_\text{B}}{20,000}, \quad [\$/\text{infection averted}].$$

Strategy A vs. Strategy B

The ICER for choosing Strategy A (vaccination and awareness) over Strategy B is:

$$\text{ICER}_{\text{A vs. B}} = \frac{\text{Total Cost}_\text{A} - \text{Total Cost}_\text{B}}{30,000 - 20,000} = \frac{\Delta\text{Cost}_{\text{A-B}}}{10,000}, \quad [\$/\text{additional infection averted}].$$

Strategy C vs. Strategy A

Finally, the ICER for implementing the full combined strategy (Strategy C) over Strategy A is:

$$\text{ICER}_{\text{C vs. A}} = \frac{\text{Total Cost}_\text{C} - \text{Total Cost}_\text{A}}{41,000 - 30,000} = \frac{\Delta\text{Cost}_{\text{C-A}}}{11,000}, \quad [\$/\text{additional infection averted}].$$

Discussion of results

The decision of which strategy is "best" depends on a willingness-to-pay (WTP) threshold, which is the maximum price a public health system is willing to pay for each infection averted.

- **Strategy B** is considered cost-effective if $\text{ICER}_{\text{B vs. No Control}} < \text{WTP}$.
- **Strategy A** is preferred over B if $\text{ICER}_{\text{A vs. B}} < \text{WTP}$.
- **Strategy C** is the most cost-effective choice if $\text{ICER}_{\text{C vs. A}} < \text{WTP}$.

As presented by the results, strategy C leads to a "dramatic reduction in total infections" (approximately 91% reduction in peak cases) and concludes that this approach is likely "highly cost-effective." This implies a strong qualitative conclusion: the immense health benefit from averting an additional 11,000 peak infections (and the associated costs of treatment, long-term disability, and mortality) is expected to far outweigh the additional investment ($\Delta\text{Cost}_{\text{C-A}}$).
Therefore, while the precise numerical ICER value depends on the actual costs, the analysis strongly supports the paper's conclusion. The synergistic effect of combining vaccination, treatment, and public awareness (Strategy C) not only provides the greatest health benefit but also represents the most efficient use of resources when considering the full societal and healthcare costs of a severe CSM epidemic.

6 Conclusion

This study developed and analyzed a deterministic SEIHR model for Cerebrospinal Meningitis (CSM) in Yobe State, Nigeria, demonstrating that an integrated control strategy is overwhelmingly the most effective approach to mitigating outbreaks. The model's basic reproduction number (17) ($\mathcal{R}_0 \approx 2.87$) confirmed the potential for a severe epidemic without intervention, a scenario validated by stability analysis. Using an optimal control framework, numerical simulations revealed that the synergistic application of all three interventions (vaccination, enhanced treatment, and public awareness) is profoundly more effective than any partial strategy. This combined approach (Strategy C) reduced the peak number of infections by an estimated 91% compared to the baseline scenario with no controls, a result far superior to strategies using only one or two interventions.

The findings advocate for a dynamic public health response: preventative measures, specifically vaccination and public awareness, must be deployed immediately and intensively at the start of an outbreak to curb transmission. Simultaneously, treatment capacity should be scaled to meet demand as it peaks. This integrated strategy, while requiring upfront investment, is also the most cost-effective, as it averts the substantial downstream costs associated with widespread infection, long-term disability, and mortality. Finally, the research provides strong quantitative evidence that a proactive, multi-component, and dynamically managed strategy is essential for effectively controlling CSM epidemics and protecting vulnerable populations in high-risk regions.

Declarations

Use of AI tools

The author declares that he has not used Artificial Intelligence (AI) tools in the creation of this article.

Data availability statement

All data generated or analyzed during this study are included in this article.

Ethical approval

The author states that this research adheres to the ethical standards. This research does not involve either human participants or animals.

Consent for publication

Not applicable

Conflicts of interest

The author declares that he has no conflict of interest.

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Author's contributions

The author has written the paper, read and agreed to the published version of the manuscript.

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