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Propagation Dynamics of Meningitis Disease Based on Complex Network Modeling

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Abstract In this study, we develop a mathematical model to explore the impact of awareness and lack thereof on the spread of meningitis within human populations, framed within a complex network context. The model's equilibrium solutions are used to ascertain its asymptotic stability, revealing that a meningitis-free equilibrium is locally and globally asymptotically stable when the basic reproduction number, R_{mg}, is below one. Conversely, a meningitis-endemic equilibrium is stable when R_{mg} exceeds one. To verify the model, we utilize parameters derived from existing literature on meningitis prevalence in Nigeria for simulation and prediction purposes. A sensitivity analysis of these parameters demonstrates that contact rates related to meningitis transmission affect R_{mg}, subsequently increasing infection rates within the population. Our simulations also indicate that the absence of awareness contributes to elevated meningitis transmission levels, suggesting that additional control measures are necessary to curb the spread of the disease.

Keywords Basic Reproduction Number R_{mg}, Stability Analysis, Sensitivity Analysis, Complex Network

1 Introduction

Meningitis, an inflammation of the meninges, is a global health concern causing morbidity, death, and economic loss, particularly within the meningitis belt of sub-Saharan Africa. Bacteria such as Streptococcus pneumoniae, Haemophilus influenzae, and Neisseria meningitidis cause the disease, with Neisseria meningitidis responsible for the largest epidemic outbreaks. Meningitis affects adults but is most common in babies and children. The disease, which incubates for 2-10 days, is transmitted directly between humans through nasal discharge, throat secretions, sneezing, and coughing. Symptoms include high fever, stiff neck, headache, brain damage, hearing loss,

disability, and death. Diagnostic tests such as agglutination tests and Polymerase Chain Reaction (PCR) are used to detect the disease, which can be treated with antibiotics and prevented through timely vaccinations [24 - 26]. Community awareness is crucial for rapid recognition of symptoms, improved understanding of risks, and prompt reporting of infections to public health facilities [7, 9, 11].

Mathematical models are valuable tools for describing and predicting disease transmission. Various mathematical and stochastic models have been formulated for meningitis transmission, incorporating factors such as time-dependent control strategies, hypothetical explanations for the spread in Africa, and differences between low and high risk susceptible populations. Research has also investigated the temporary immunity of humans in Africa and the impact of vaccinations on disease prevalence. Nigeria, a country heavily impacted by meningitis, has been the focus of several models [1, 2, 10 14, 17]. Moreover, [6] formulated deterministic models with different strains of meningitis. Their results show that the natural immunogen against asymptomatic and symptomatic meningococcal infection was consistent with field data and the introduction of vaccines into the population changes the prevalence of the disease. Also, [21] studied the dynamic models of meningococcal carriers of the disease, and the impact of serogroup C conjugate vaccination, while [22], formulated a mathematical model of cerebrospinal meningitis epidemic transmission using Jirapa District, Ghana as case study. Nigeria has been the epicenter of meningitis disease, which has ravaged the nation and caused many deaths [3, 16, 19].

Epidemic models can be represented as graphs or networks, with nodes representing individuals and links connecting pairs of nodes to indicate interactions. Compartmental models group nodes on the network by disease state and subdivide them according to their degrees [4, 5, 8, 12, 13, 15]. Works on the propagation of diseases based on complex network can be seen in the works of [18, 20, 23, 27].

Although many studies have examined deterministic and stochastic models of meningitis and their qualitative behavior, along with the effects of treatment and vaccination, little research has been conducted on the impact of awareness and unawareness of meningitis transmission within a Susceptible-Infected-Susceptible (SIS) complex network model. This paper addresses this gap by formulating a deterministic model of meningitis awareness on a complex network using estimated parameters of meningitis prevalence in Nigeria, a country within the meningitis belt. The paper is organized into sections, with Section 2 covering model formulation and qualitative analysis, Section 3 focusing on numerical simulation, and Section 4 providing the conclusion.

2 Mathematical Model Formulation and Qualitative Analysis

After the repeated outbreak of meningitis, humans become more conscious and responsive to the disease, but after some time, human individuals lose some degree of awareness due to certain reasons. The densities of the human population are classified into four sub-populations according to different physical and conscious states of awareness, which are; Susceptible meningitis-unaware humans $S^u_{m_k}$; Infected meningitis-unaware humans $I^u_{m_k}$; Susceptible meningitis-aware humans $S^a_{m_k}$; Infected meningitis-aware humans $I^a_{m_k}$. At time $t > 0$ the nodes on the network have a fluctuating degree k for $1 \leq k \leq n$, where n is the largest degree, and the total population $N = \sum_{k=1}^{n} N_k$, where $N_k = N_{u_k} + N_{a_k}$ is divided into n classes, that is, the total human population of unaware and aware yields $N_{u_k}(t) = S^u_{m_k}(t) + I^u_{m_k}(t)$ and $N_{a_k}(t) = S^a_{m_k}(t) + I^a_{m_k}(t)$ respectively satisfy the normalization condition for all k classes. Furthermore, the meningitis transmission rate between unaware nodes is larger than the meningitis infection rate between aware nodes. Based on these assumptions, the model on the networks yields

$$\left.\begin{aligned}
\dot{S^u_{m_k}} &= \Theta_a - k(\Delta_1 I^u_{m_k} + \Delta_2 I^a_{m_k})S^u_{m_k} + \\
&\alpha S^a_{m_k} - d_1 S^u_{m_k} + \mu S^a_{m_k}, \\
\dot{I^u_{m_k}} &= k(\Delta_1 S^u_{m_k} + \Delta_3 S^a_{m_k})I^u_{m_k} + \alpha I^a_{m_k} \\
&- (d_1 + \delta_1)I^u_{m_k} + \mu I^a_{m_k}, \\
\dot{S^a_{m_k}} &= \Theta_b - k(\Delta_3 I^u_{m_k} + \Delta_4 I^a_{m_k})S^a_{m_k} \\
&- (\alpha + d_1)S^a_{m_k} - \mu S^a_{m_k}, \\
\dot{I^a_{m_k}} &= k(\Delta_2 S^u_{m_k} + \Delta_4 S^a_{m_k})I^a_{m_k} \\
&- (\alpha + d_1 + \delta_1)I^a_{m_k} - \mu I^a_{m_k}.
\end{aligned}\right\} \quad (1)$$

Subject to the initial start $S^u_{m_k} \geq 0, I^u_{m_k} \geq 0, S^a_{m_k} \geq 0, I^a_{m_k} \geq 0$. Table 1 presents the definitions and estimated values of parameters describing the formulation of model (1) and Figure 1 illustrates the meningitis interaction according the status of unawareness and awareness in human host population.

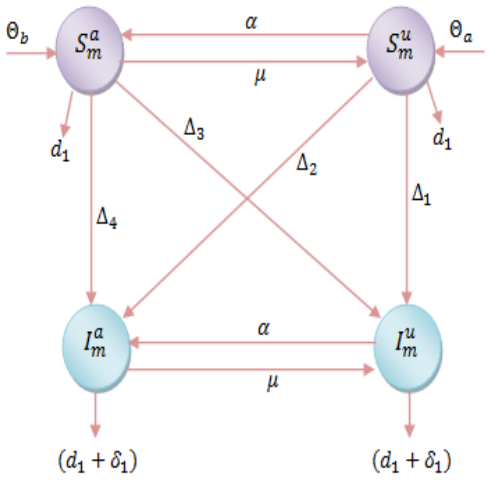

Figure 1. Diagram describing the meningitis disease transmission network in the human host population.

2.1 Basic Model Analysis

Theorem 1. *Given the initial value* $S^u_{m_k} \geq 0, I^u_{m_k} \geq 0, S^a_{m_k} \geq 0, I^a_{m_k} \geq 0$, *the solutions of model (1) are positive for all time* $t > 0$.

Proof. Let $t_* = sup(t > 0 | S^u_{m_k} > 0, l^u_{m_k} > 0, S^a_{m_k} > 0, l^a_{m_k} > 0)$. From the first equation in (1), given by

$$\dot{S^u_{m_k}} = \Theta_a - k(\Delta_1 I^u_{m_k} + \Delta_2 I^a_{m_k}) S^u_{m_k} + \alpha S^a_{m_k} - d_1 S^u_{m_k} + \mu S^a_{m_k}. \tag{2}$$

The integrating factor for (2) is given by

$$exp\Big(\int_0^{t_*} k((\Delta_1 l^u_{m_k}(s) + \Delta_2 l^a_{m_k}(s))ds + d_1 \Big) t_*. \tag{3}$$

Multiplying (2) by (3), one obtains

$$\begin{aligned} &\frac{d}{dt}\Big(S^u_{m_k} exp\Big(\int_0^{t_*} (\Delta_1 l^u_{m_k}(s) + \Delta_2 l^a_{m_k}(s))ds + d_1 \Big) t \Big) = \\ &\Theta_a + (\alpha + \mu) S^a_{m_k} \Big(exp\Big(\int_0^{t_*} (\Delta_1 l^u_{m_k}(s) \\ &+ \Delta_2 I^a_{m_k}(s) \Big) ds + d_1) t \Big). \end{aligned} \tag{4}$$

Solving (4), yields

$$\begin{aligned} &S^u_{m_k}(t_*) exp\Big(\int_0^{t_*} k(\Delta_1 I^u_{m_k}(s) + \Delta_2 I^a_{m_k}(s))ds + d_1) t \Big) \\ &- S^u_{m_k}(0) = \int_0^{t_*} (\Theta_a + (\alpha + \mu) S^a_{m_k}(v) \times \\ &exp\Big(\int_0^{v} k(\Delta_1 I^u_{m_k}(s) + \Delta_2 I^a_{m_k}(s))ds \\ &+ (d_1)v \Big) dv. \end{aligned} \tag{5}$$

Therefore,

$$\left.\begin{aligned} &S^u_{m_k}(t_*) = S^u_{m_k}(0) exp\Big(- \int_0^{t_*} \Big((\Delta_1 I^u_{m_k}(s) + \\ &\Delta_2 I^a_{m_k}(s))ds + d_1 \Big) t_* \Big) + \\ &exp\Big(- \int_0^{t_*} (\Delta_1 I^u_{m_k}(s) + \Delta_2 I^a_{m_k}(s))ds + d_1) t \Big) \\ &\times \int_0^{t_*} \Theta_a + (\alpha + \mu) S^a_{m_k}(v) \\ &\times exp\Big(\int_0^{v} k(\Delta_1 I^u_{m_k}(s) + \Delta_2 I^a_{m_k}(s))ds + d_1 v \Big) dv > 0. \end{aligned}\right\} \tag{6}$$

Hence, (2)-(6) proves the positivity of model (1), which can also be verified for $I^u_{m_k} > 0, S^a_{m_k} > 0$, and $I^a_{m_k} > 0$.

Theorem 2. *All the solutions $(S^u_{m_k}(t), I^u_{m_k}(t), S^a_{m_k}(t), I^a_{m_k}(t))$ of model (1) together with initial conditions are bounded and positively invariant in the feasible region $\Omega = \Omega_x \times \Omega_y$.*

Proof. The total human host population of the meningitis-unaware population yields

$$\frac{N_{u_k}}{dt} = \Theta_a - d_1 N_{u_k} \leq \Theta_a - d_1 N_{u_k}. \tag{7}$$

Since $S^u_{m_k} + I^u_{m_k} = N_{u_k}$, solving (7) yields

$$\lim_{t \to \infty} sup\ N_{u_k} \leq \frac{\Theta_a}{d_1}. \tag{8}$$

Similarly, for the meningitis-aware population, one obtains

$$\frac{N_{a_k}}{dt} = \Theta_b - d_1 N_{a_k} \leq \Theta_b - d_1 N_{a_k}, \tag{9}$$

so that

$$\lim_{t \to \infty} \sup\ N_{a_k} \leq \frac{\Theta_b}{d_1} \tag{10}$$

Therefore, $\Omega_x = \Big\{ \Big((S^u_{m_k}, I^u_{m_k}) \in \Re^2 | N_{u_k} \leq \frac{\Theta_a}{d_1} \Big) \Big\}$ and $\Omega_y = \Big\{ \Big((S^a_{m_k}, I^a_{m_k}) \in \Re^2 | N_{a_k} \leq \frac{\Theta_b}{d_1} \Big) \Big\}$. Hence, the model solution stays in the set $\Omega = \Omega_x \times \Omega_y$, which shows that Ω is a positively invariant region. Thus, model (1) is well-posed and valid in the sense of the meningitis–disease transmission.

2.2 Stability Analysis of the Model

In order to determine the asymptotic stability of the model, the meningitis-free and meningitis-endemic equilibrium solutions are obtained. Fixing the right hand side of model (1) to zero, the meningitis-free equilibrium solution is given by

$$M^o_g = (S^{uo}_{m_k}, I^{uo}_{m_k}, S^{ao}_{m_k}, I^{ao}_{m_k}) = \Big(\frac{\Theta_a}{d_1}, 0, \frac{\Theta_b}{d_1}, 0 \Big), \tag{11}$$

while the meningitis-endemic equilibrium solution yields

$$\left.\begin{aligned} &M^*_g = (S^{u*}_{m_k}, I^{u*}_{m_k}, S^{a*}_{m_k}, I^{a*}_{m_k}) = \\ &S^{u*}_{m_k} = \frac{\Theta_a + \alpha S^{a*}_{m_k} + \mu S^{a*}_{m_k}}{(\Delta_1 I^{u*}_{m_k} + \Delta_2 I^{a*}_{m_k}) - d_1}, \\ &I^{u*}_{m_k} = \frac{\alpha I^{a*}_{m_k} + \mu I^{a*}_{m_k}}{(\Delta_1 S^{u*}_{m_k} + \Delta_3 S^{a*}_{m_k}) - (d_1 + \delta_1)}, \\ &S^{a*}_{m_k} = \frac{\Theta_b}{(\Delta_3 I^{u*}_{m_k} + \Delta_4 I^{a*}_{m_k}) - (\alpha + d_1 + \delta_1)}, \\ &I^{a*}_{m_k} = \frac{\alpha I^{a*}_{m_k} + \mu I^{a*}_{m_k}}{(\Delta_2 S^{u*}_{m_k} + \Delta_4 S^{a*}_{m_k}) - (\alpha + d_1 + \delta_1 + \mu)}. \end{aligned}\right\} \tag{12}$$

The effective basic reproduction number R_{mg} is interpreted as the average number of effective contacts multiplied by the initial susceptible population and their average infectious period, such that if $R_{mg} < 1$, the meningitis disease propagation becomes ineffective, but if $R_{mg} > 1$, the disease propagation persists and becomes endemic in the system. Using the next generation matrix method [7, 9, 11], the two effective basic reproduction numbers obtained for model (1), are given by $R_1 = \frac{1}{k} \frac{(\Delta_1 S^u_{m_k} + \Delta_3 S^a_{m_k})}{(d_1 + \delta_1)}$ and $R_2 = \frac{1}{k} \frac{(\Delta_2 S^u_{m_k} + \Delta_4 S^a_{m_k})}{(\alpha + d_1 + \delta_1 - \mu)}$, where, $R_{mg} = R_1 \times R_2$ so that

$$R_{mg}(R_1 \times R_2) = \frac{1}{k^2} \frac{(\Delta_1 S^u_{m_k} + \Delta_3 S^a_{m_k})}{(d_1 + \delta_1)} \cdot \frac{(\Delta_2 S^u_{m_k} + \Delta_4 S^a_{m_k})}{(\alpha + d_1 + \delta_1) - \mu)}. \tag{13}$$

Theorem 3. *The meningitis-free equilibrium solution (11) of model (1) is locally asymptotically stable if $R_{mg} < 1$.*

Proof. Linearizing model (1) using the meningitis-free equilibrium solution (11) yields two negative eigenvalues given by $-d_1$ and $-(\alpha_1 + d_1 + \mu)$, while the remaining two positive

eigenvalues are $(\Delta_2 S^a_{m_k} + \Delta_4 S^a_{m_k} - (\alpha + d_1 + \delta_1 + \mu)$ and $(\Delta_1 S^u_{m_k} + \Delta_3 S^a_{m_k} - (d_1 + \delta_1)$, so that

$$\frac{(\Delta_2 S^a_{m_k} + \Delta_4 S^a_{m_k})}{(\alpha + d_1 + \delta_1 + \mu)} - \frac{(\alpha + d_1 + \delta_1 + \mu)}{(\alpha + d_1 + \delta_1 + \mu)} > 0 \tag{14}$$

and

$$\frac{(\Delta_1 S^u_{m_k} + \Delta_3 S^a_{m_k})}{(d_1 + \delta_1)} - \frac{(d_1 + \delta_1)}{(d_1 + \delta_1)} > 0. \tag{15}$$

From (14) and (15), $(R_1 - 1) > 0$ and $(R_2 - 1) > 0$, which implies that $-R_1 > -1 \Leftrightarrow R_1 < 1$ and $-R_2 > -1 \Leftrightarrow R_2 < 1$. Since $R_{mg} = R_1 \cdot R_2$, then $R_{mg} < 1$ implies that the meningitis-free equilibrium solution (11) of model (1) is locally asymptotically stable.

Theorem 4. *The meningitis-free fixed equilibrium solution (11) of model (1) is globally asymptotically stable if $R_{mg} < 1$.*

Proof. Consider the Lyapunov $(L_p = L_{p1} \cdot L_{p2})$ function given by $L_{p1}(S^u_{m_k}, I^u_{m_k}) = I^u_{m_k}$ and $L_{p2}(S^a_{m_k}, I^a_{m_k}) = I^a_{m_k}$ such that

$$\begin{aligned} &\frac{dL_{p1}}{dt} = \frac{dI^u_{m_k}}{dt} = [(\Delta_1 S^u_{m_k} + \Delta_3 S^a_{m_k}) - (d_1 + \delta_1)] \\ &I^u_{m_k} \leq \frac{1}{(d_1 + \delta_1)}[R_1 - (d_1 + \delta_1)] I^u_{m_k} \leq 0, \end{aligned} \tag{16}$$

and

$$\begin{aligned} &\frac{dL_{p2}}{dt} = \frac{dI^a_{m_k}}{dt} = [(\Delta_2 S^a_{m_k} + \Delta_4 S^a_{m_k}) - (\alpha + d_1 + \delta_1) - \mu] \\ &I^u_{m_k} \leq \frac{1}{(\alpha + d_1 + \delta_1)} - \mu)[R_2 - (\alpha + d_1 + \delta_1) - \mu] I^u_{m_k} \leq 0. \end{aligned} \tag{17}$$

From (16) and (17), if $R_{mg} < 1$, then $\dot{L_{p1}} = \dot{L_{p2}} = 0 \Leftrightarrow I^u_{m_k} = I^a_{m_k} = 0$. By the La-Salle Lyapunov principle [11], M^o_g is globally asymptotically stable.

Theorem 5. *The meningitis-endemic endemic equilibrium solution (12) of model (1) is locally asymptotically stable if $R_{mg} > 1$.*

Proof. The Jacobian matrix (J_c) of model (1) is evaluated at the meningitis-endemic equilibrium solution (12) of model (1) is given by

$$J_c = \begin{pmatrix} q_o & \alpha + \mu \\ \Delta_4 I^{a*}_{m_k} & q_1 \end{pmatrix}, \tag{18}$$

where $q_o = \Delta_1 S^{u*}_{m_k} + \Delta_3 S^{a*}_{m_k} - (d_1 + \delta_1))$ and $q_1 = (\Delta_2 S^{u*}_{m_k} + \Delta_4 S^{u*}_{m_k} - ((\alpha + d_1 + \delta_1) + \mu))$ The characteristic polynomial of (18) yields

$$\lambda^2 + z_1 \lambda + z_2, \tag{19}$$

where

$$\left.\begin{aligned} &z_1 = ((\Delta_1 + \Delta_2) S^{u*}_{m_k} + (\Delta_3 + \Delta_4) S^{a*}_{m_k} \\ &- (\alpha + \mu) - 2(d_1 + \delta_1)), \\ &z_2 = ((\Delta_2 S^{u*}_{m_k} + \Delta_4 S^{a*}_{m_k} - (\alpha + d_1 + \delta_1 + \mu) S^{u*}_{m_k} + \\ &(\Delta_1 S^{u*}_{m_k} + \Delta_3 S^{a*}_{m_k}) S^{a*}_{m_k} - (d_1 + \delta_1 + \mu)(R_{mg} - 1). \end{aligned}\right\} \tag{20}$$

Since $z_1 > 0$, and $z_2 > 0$ then the meningitis–endemic equilibrium solution (12) is locally asymptotically stable when $R_{mg} > 1$.

Theorem 6. *The meningitis-endemic equilibrium solution (12) of model (1) is globally asymptotically stable if $R_{mg} > 1$.*

Proof. Using the Lyapunov direct method, a Lyapunov function V_p is constructed given by

$$\begin{aligned} V_p = &\Big(S^u_{m_k} - S^{u*}_{m_k} - S^{u*}_{m_k} \ln \frac{S^{u*}_{m_k}}{S^u_{m_k}}\Big) + \Big(I^u_{m_k} - I^{u*}_{m_k} - I^{u*}_{m_k} \\ &\ln \frac{l^{u*}_{m_k}}{l^u_{m_k}}\Big) + \Big(S^a_{m_k} - S^{a*}_{m_k} - S^{a*}_{m_k} \ln \frac{S^{a*}_{m_k}}{S^a_{m_k}}\Big) + \\ &\Big(l^a_{m_k} - l^{a*}_{m_k} - l^{a*}_{m_k} \ln \frac{l^{a*}_{m_k}}{l^a_{m_k}}\Big), \end{aligned} \tag{21}$$

where the Lyapunov derivative of (21) becomes

$$\left.\begin{aligned} \dot{V_p} = &\Big(1 - \frac{S^{u*}_{m_k}}{S^u_{m_k}}\Big) \dot{S^u_{m_k}} + \Big(1 - \frac{I^{u*}_{m_k}}{I^u_{m_k}}\Big) \dot{I^u_{m_k}} \\ &\Big(1 - \frac{S^{a*}_{m_k}}{S^a_{m_k}}\Big) \dot{S^a_{m_k}} + \Big(1 - \frac{I^{a*}_{m_k}}{I^a_{m_k}}\Big) \dot{I^a_{m_k}}. \end{aligned}\right\} \tag{22}$$

Let

$$\begin{aligned} &\Theta_a = (\Delta_1 I^{u*}_{m_k} + \Delta_2 I^{a*}_{m_k}) S^u_{m_k} + \alpha S^{a*}_{m_k} - d_1 S^{u*}_{m_k} + \mu S^{a*}_{m_k}, \\ &(\Delta_1 S^{u*}_{m_k} + \Delta_3 S^{a*}_{m_k}) I^u_{m_k} = (d_1 + \delta_1) I^{u*}_{m_k} + (\mu + \alpha) I^{a*}_{m_k}, \\ &\Theta_b = (\Delta_3 I^{u*}_{m_k} + \Delta_4 I^{a*}_{m_k}) S^{a*}_{m_k} - (\alpha + d_1) S^{a*}_{m_k} - \mu S^{a*}_{m_k}, \\ &(\Delta_2 S^{u*}_{m_k} + \Delta_4 S^{a*}_{m_k}) I^{a*}_{m_k} = ((\alpha + d_1 + \delta_1) - \mu) I^{a*}_{m_k}. \end{aligned} \tag{23}$$

Substituting the expression in (23) into (22) and solving part by part yields

$$\left.\begin{aligned} \dot{V_p} = &\Big(1 - \frac{S^{u*}_{m_k}}{S^u_{m_k}}\Big)(\Delta_1 I^{u*}_{m_k} + \Delta_2 I^{a*}_{m_k}) \\ &S^u_{m_k} + \alpha S^{a*}_{m_k} - d_1 S^{u*}_{m_k} + \mu S^{a*}_{m_k} - (\Delta_1 I^u_{m_k} + \Delta_2 I^a_{m_k}) \\ &S^u_{m_k} + \alpha S^{a*}_{m_k} - d_1 S^{u*}_{m_k} + \mu S^{a*}_{m_k} = (\Delta_1 I^{u*}_{m_k} + \Delta_2 I^{a*}_{m_k}) - \\ &d_1 \Big(2 - \frac{S^{u*}_{m_k}}{S^u_{m_k}} - \frac{S^u_{m_k}}{S^{u*}_{m_k}}\Big) + (\alpha - \mu)(S^{a*}_{m_k} - S^a_{m_k}), \end{aligned}\right\} \tag{24}$$

$$\left.\begin{aligned} &\Big(1 - \frac{I^{u*}_{m_k}}{I^u_{m_k}}\Big)((d_1 + \delta_1) I^{u*}_{m_k} + (\mu + \alpha) I^{a*}_{m_k} - \\ &(d_1 + \delta_1) I^u_{m_k} + (\mu + \alpha) I^a_{m_k}) = (d_1 + \delta_1) I^{u*}_{m_k} \Big(2 - \frac{I^{u*}_{m_k}}{I^u_{m_k}} \\ &- \frac{I^u_{m_k}}{I^{u*}_{m_k}}\Big) + (\alpha + \mu)\Big(1 - \frac{I^{u*}_{m_k}}{I^u_{m_k}} - \frac{I^u_{m_k}}{I^{u*}_{m_k}} \\ &+ \frac{I^u_{m_k} I^{u*}_{m_k}}{I^{u*}_{m_k} I^u_{m_k}}\Big), \end{aligned}\right\} \tag{25}$$

$$\left.\begin{aligned} &(\alpha + \mu) S^{a*}_{m_k} \Big(1 - \frac{S^{u*}_{m_k}}{S^u_{m_k}} - \frac{S^a_{m_k}}{S^{a*}_{m_k}} \\ &+ \frac{S^{a*}_{m_k} S^a_{m_k}}{S^a_{m_k} S^{a*}_{m_k}}\Big) - d_1 S^{u*}_{m_k} \Big(2 - \frac{S^{u*}_{m_k}}{S^u_{m_k}} \\ &- \frac{S^u_{m_k}}{S^{u*}_{m_k}}\Big) - (\Delta_1 I^{u*}_{m_k} + \Delta_2 I^{a*}_{m_k}) S^{u*}_{m_k} \\ &\Big(2 - \frac{S^{u*}_{m_k}}{S^u_{m_k}} - \frac{S^u_{m_k}}{S^{u*}_{m_k}}\Big), \end{aligned}\right\} \tag{26}$$

and

$$((\alpha + d_1 + \delta_1) - \mu) I^{a*}_{m_k} \left(1 - \frac{I^{a*}_{m_k}}{I^{a}_{m_k}} - \frac{I^{a}_{m_k}}{I^{a*}_{m_k}}\right). \quad (27)$$

Since the arithmetic mean exceeds the geometric mean, it follows from (24)-(27)

$$\left.\begin{aligned} &\left(1 - \frac{I^{a*}_{m_k}}{I^{a}_{m_k}} - \frac{I^{a}_{m_k}}{I^{a*}_{m_k}}\right) \leq 0, \\ &\left(\frac{I^{u*}_{m_k}}{I^{u}_{m_k}} - \frac{I^{u}_{m_k}}{I^{u*}_{m_k}} + \frac{I^{u}_{m_k} I^{u*}_{m_k}}{I^{u*}_{m_k} I^{u}_{m_k}}\right) \leq 0, \\ &\left(2 - \frac{S^{u*}_{m_k}}{S^{u}_{m_k}} - \frac{S^{u}_{m_k}}{S^{u*}_{m_k}}\right) \leq 0 \end{aligned}\right\} \quad (28)$$

It is observed from (28) that if $S^{u}_{m_k} = S^{u*}_{m_k}, I^{u}_{m_k} = I^{u*}_{m_k}, S^{a}_{m_k} = S^{a*}_{m_k}$ and $I^{a}_{m_k} = I^{a*}_{m_k}$, then $\dot{V}_p \leq 0$ for all $(S^{u}_{m_k}(t), I^{u}_{m_k}(t), S^{a}_{m_k}(t), I^{a}_{m_k}(t)) > 0$. Then the meningitis-endemic equilibrium solution M^{o}_{g} (12) is the only largest invariant set. By the La-Salle invariance principle [11], the meningitis-endemic equilibrium of model (1) is globally asymptotically stable when $R_{mg} > 1$.

3 Numerical Simulations

In this section, we perform numerical simulations of the model using the ODE45 solver, which is integrated within the Maple computational software. We utilize estimated parameters describing the spread of meningitis in Nigeria, as shown in Table 1. Additionally, we employ the normalized forward sensitivity index to conduct a sensitivity analysis of the model parameters related to R_{mg} (12).

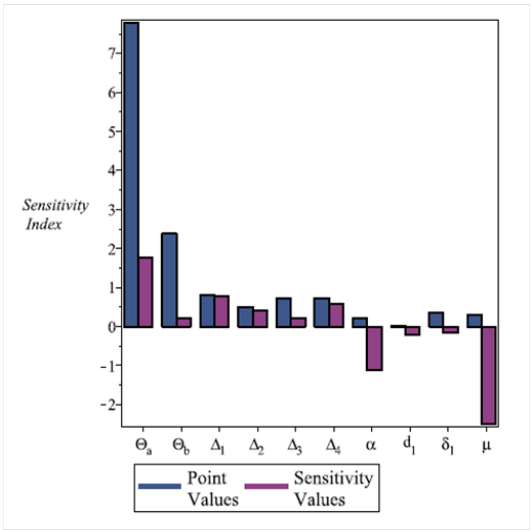

Figure 2. Bar plot of Table 2 using the point values in Table 1

The sensitivity bar plot in Figure 2, derived from Table 2, demonstrates that the recruitment rates (Θ_a and Θ_b) and meningitis transmission contact rates $\Delta_1, \Delta_2, \Delta_3$ and Δ_4 are the most sensitive parameters, directly affecting R_{mg}. This results in a steady increase in the value of R_{mg}, contributing to the prevalence of meningitis within human populations. Conversely, the least sensitive negative parameters cause a decrease in R_{mg}, but still remain detrimental due to the loss of awareness and fatalities associated with meningitis infection. From an epidemiological perspective, the findings in Table 2 and Figure 2 suggest that control measures aimed at increasing awareness among the recruited population, reducing the disease contact rates $\Delta_i (i = 1-4)$, and further enhancing disease awareness could effectively limit the transmission of meningitis within human communities.

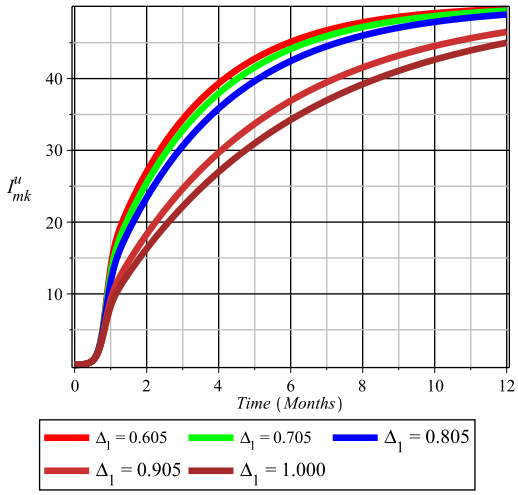

Figure 3. Effect of contact between susceptible and infected unaware humans

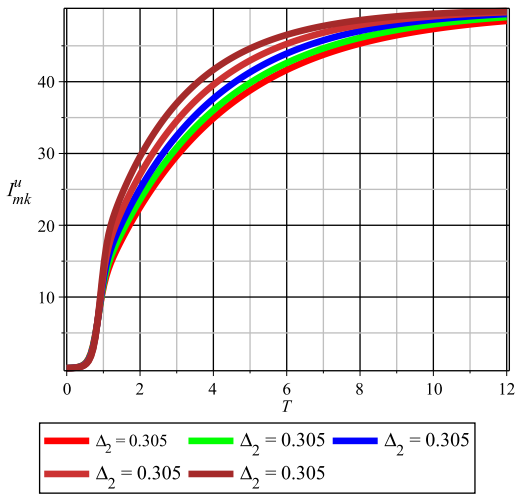

Figure 4. Effect of contact between unaware susceptible and infected aware humans

Figure 3 illustrates that meningitis infection surges rapidly when the level of disease contact Δ_1 increases among susceptible and infected individuals without awareness. Figure 4 reveals that, in the absence of awareness, meningitis infection accelerates more quickly when the level of disease contact Δ_2 is varied compared to Δ_1. Figure 5 depicts the behavior of infected individuals who are aware of meningitis infection; as the disease contact rate Δ_3 varies, the infection peaks but then

Table 1. Meanings of parameters associated to meningitis transmission

Parameters	Descriptions	Range Values	Point Values	Sources
Θ_a	Recruitment rate of susceptible meningitis unaware humans	3-8	7.781	[2, 3, 19]
Θ_b	Recruitment rate of susceptible meningitis aware humans	1-3	2.382	[2, 3, 19]
Δ_1	Contact rate	0-1	0.805	[2, 6]
Δ_2	Contact rate	0-1	0.505	[2, 6, 17]
Δ_3	Contact rate	0-1	0.712	[2, 6, 17]
Δ_4	Contact rate	0-1	0.721	[2, 6, 17]
α	Rate of spread of awareness	0-1	0.220	[2, 6, 18]
d_1	Natural death rate	0-1	0.027	[2, 6, 18]
μ	Rate of loss of awareness	0-1	0.361	[2, 6, 18]
δ_1	Death due to meningitis	0-1	0.311	[2, 6, 18]

Table 2. Sensitivity Results

Parameters	Sensitivity Values
Θ_a	1.786928
Θ_b	0.213011
Δ_1	0.786928
Δ_2	0.411908
Δ_3	0.213071
Δ_4	0.588091
α	-1.1167512
d_1	-0.213071641
δ_1	-2.49879854
μ	-0.167512

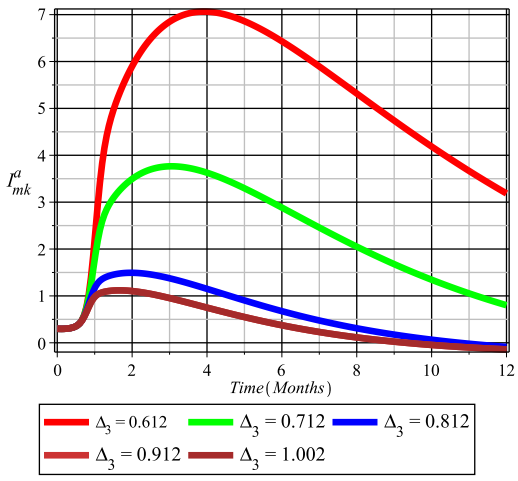

Figure 5. Effect of infectious contact between unaware susceptible and infected aware humans

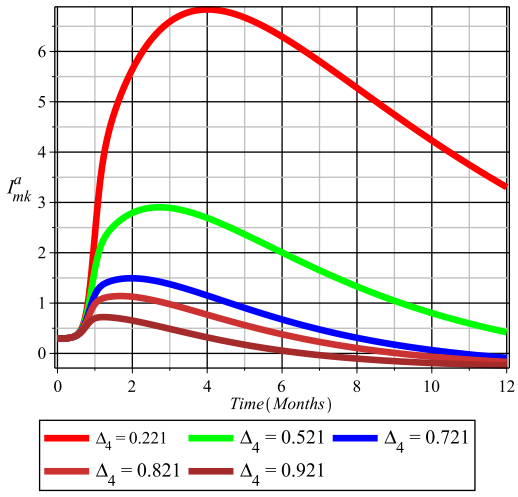

Figure 6. Effect of infectious contact between susceptible and infected aware humans

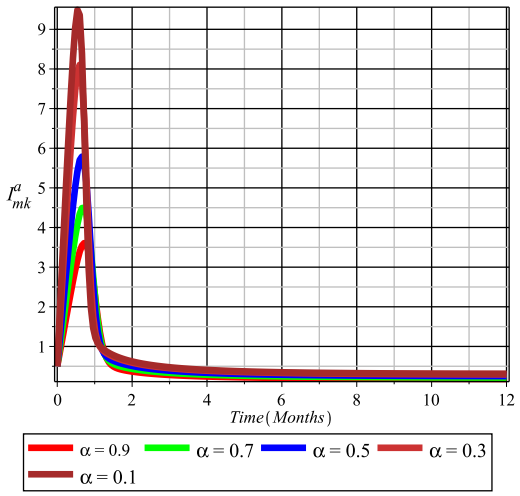

Figure 7. Effect of awareness about the disease spread in the human host community

gradually declines with the presence of awareness. Figure 6 demonstrates that the number of infected individuals reaches a peak in a short time but subsequently decreases as the contact rate Δ_4 is varied, given the presence of awareness. Figure 7 shows that when awareness decreases, infected humans who are aware about the disease increase due to loss of consciousness about the disease overtime.

4 Conclusion

Based on the results, we can conclude that the model describing meningitis infection transmission in a complex network is positive, well-posed, and locally and globally asymptotically stable when R_{mg} is less than or greater than one. Furthermore, we discovered that controlling sensitive parameters that contribute to the prevalence of meningitis is essential since even the least sensitive parameters can still be fatal. Additionally, our simulations revealed that awareness can reduce meningitis prevalence, but implementing further restriction strategies is necessary to prevent the disease from spreading further.

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