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MATHEMATICAL MODELING AND STABILITY ANALYSES ON THE TRANSMISSION DYNAMICS OF BACTERIAL MENINGITIS

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Abstract. Bacterial meningitis has posed a serious threat to lives and livelihood of people, especially those in the meningitis belt. This study presents a deterministic compartmental model of the disease based on Susceptible-Vaccinated-Carrier-Infected-Treated-Recovered (SVCITR). This transmission process is made up of seven mutually exclusive epidemiological compartments for the transmission dynamics of the disease. The invariant region, positivity of the solutions and stability of the equilibrium points were examined using quantitative analysis. The basic reproduction number, \mathcal{R}_0 was computed using the next generation matrix approach and this was used as a threshold to establish the local and global stabilities of the model. The simulation results from the numerical simulation of the model demonstrate the effects of the model parameters on each compartment. The results show that getting people vaccinated is crucial to the control of the disease. Furthermore, the sensitivity analysis of \mathcal{R}_0 was performed in order to determine the effect of each of the model parameters in controlling the disease. Hence, reducing the values of the parameters with negative sensitivity index will help to curtail the spread of the disease.

Keywords: bacterial meningitis; vaccination; treatment; stability analysis; sensitivity analysis.

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1. INTRODUCTION

Meningitis, a disease of the Central Nervous System is an acute inflammation of the three protective membranes covering the brain and spinal cord called the meninges. It is an epidemic-prone disease affecting a substantial proportion of the world's population. The causative agents of meningitis are viruses, bacteria, parasites and fungi but the global burden of disease is seen with bacterial meningitis, WHO [19]. Both viral and bacterial meningitis may have similar symptoms at the onset of infection, but bacteria meningitis usually presents more severe symptoms as time goes on. This is largely due to the infection of the fluid surrounding the meninges. Injuries, cancer, drugs and other infections can also lead to meningitis. It is therefore pertinent to know the particular cause of meningitis to aid in effective treatment. About 4 out of every 5 cases of bacteria meningitis are caused by *Streptococcus pneumoniae*, *Haemophilus influenzae* type B and *Neisseria meningitidis* (Nm), Amidu *et al.* [1].

Bacterial meningitis is one of the most dangerous infections due to repeated occurrence of the infection and the sequelae of debilitating effects among survivors even after treatment. Regional outbreaks can occur at any time, though the Meningitis belt stands at a higher risk. The Meningitis belt spans from the Atlantic Ocean to the Red Sea - a semi-arid area of sub-Saharan Africa, Woods *et al.* [21]. The proximity and inflammation of the protective membranes to the brain and spinal cord can make bacterial meningitis very fatal. It can lead to permanent disability, coma, swelling of the brain and even death if not treated immediately. Therefore, the condition is considered as a medical emergency, Martinez *et al.* [14]. Case fatality rates which is often between 1 to 2 days after the onset of symptoms may be as high as 50-80% when not treated and about 8-15% when treated. Also, about 10-20% of survivors have serious permanent health problems like epilepsy, hearing impairment or mental retardation. In totality, about 10% of all bacterial meningitis results in death, Letsa *et al.* [12]. The average incubation period for bacterial meningitis is four (4) days, but symptoms may develop over several hours after exposure to the bacteria usually between 2 to 10 days, Asamoah *et al.* [3].

Most people have a good recovery from bacterial meningitis; however many recover from the acute phase of the disease only to experience some difficulties while trying to get back to their everyday routine, Kaburi *et al.* [10]. Bacterial meningitis can result in severe health complications such as headaches, decreased appetite, paralysis, irritability, memory problems, stroke, hearing loss, brain damage, kidney failure, seizures and septicemia (body-wide infection and shock). These complications are often permanent. The longer one has the infection without treatment, the greater the risk of these complications. With prompt treatment, even patients with severe meningitis can have good recovery, Nuoh *et al.* [16].

Bacterial meningitis is preventable due to the availability of effective vaccines against most of the disease causing agents - *S. pneumonia*, *H. influenza* type b and *N. meningitidis* serogroups: A, B, C, W135 and Y. These vaccines are used for prevention, that is routine immunization and in prompt reactive vaccination during outbreaks, McCarthy, Sharyan and Sheikhi Moghaddam [15]. There is also treatment with antibiotics such as benzyl penicillin, ampicillin, ceftriaxone and chloramphenicol, Trestioreanu *et al.* [17].

Wiah and Adetunde [20] investigated the dynamics of cerebrospinal meningitis (CSM) in Jirapa district in the Upper West region of Ghana. Their paper presented the dynamics of cerebrospinal meningitis and suggested ways on how to control the disease. The existence of the solution of the model was established and the stability of equilibria was examined. The numerical simulation showed that early treatment, implementation of cerebrospinal meningitis protocols and cooperation with medical personnel and traditional healers could help control the disease.

Martcheva and Crispino-O'Connell [13] used an age-structured mathematical model to study the transmission dynamics of meningococcal infection. The conditions that give rise to the stability of the disease-free steady state and the existence of an endemic state were examined. The contribution of the carrier class to the transmission of the disease was established from the

numerical simulation.

The pattern of the transmission dynamics of meningococcal meningitis was investigated using deterministic compartmental models. The results from the numerical simulation of the model showed that seasonal vibration and temporary immunity were due to the irregular epidemics which often occur in the meningitis belt, Irving, Blyuss, Colijn and Trotter [9]. Martinez *et al.* [14] presented a novel mathematical model for the transmission of meningococcal meningitis using cellular automata. Their results established that both the individual and global behaviours of the disease could be determined. This result agreed favourably with the empirical predictions.

The dynamics of bacterial meningitis in a given population was presented using time-dependent controls, nonlinear deterministic model. The results indicate that effective contact rate and infectious carriers have a great effect in transmitting the disease. The model was extended as an optimal control problem in order to determine the best strategies for the control of the disease. The solution of the optimal control problem showed that the best strategies for controlling bacterial meningitis is the combination of vaccination of susceptible population with other interventions, Asamoah *et al.* [3].

Other researchers such as Karachaliou, Conlan, Preziosi and Trotter [11], Blyuss [4], Yusuf [22], Dukić *et al.* [6], Agosto and Leite [2] have also presented works on meningitis. Most of the mathematical models developed represent the different types of Bacterial Meningitis. As an extension of the available models with a broader focus on Bacterial meningitis, a new mathematical model based on the Susceptible-Vaccinated-Carrier-Infected-Treated-Recovered (SVCITR) is developed with new model parameters to have a more realistic model which is closer to what is obtainable in the real life situation.

2. MODEL FORMULATION

2.1. Model Description. The total population at time t , denoted by $N(t)$, is divided into seven (7) mutually exclusive epidemiological classes, namely, the Susceptible Class, $S(t)$ Vaccinated Class, $V(t)$ Carrier Class, $C(t)$ Infected Class, $I(t)$ Treated Class, $T(t)$ and two Recovered

Classes, $R_1(t)$ and $R_2(t)$. The Susceptible Class is made up of the individuals who are not yet infected and have also not been vaccinated against the disease. This is generated by the recruitment of individuals at a rate α and by loss of immunity from previous vaccination. The Susceptible class is reduced by natural death, vaccination or by infection through effective contact with infected individuals at the rate

$$(1) \quad \lambda(t) = \frac{\beta [\eta_1 C(t) + I(t)]}{N(t)}$$

The parameter β is the effective transmission probability per contact and the parameter $\eta_1 \leq 1$ is a modification parameter, Augusto and Leite [2].

The Vaccinated Class are the individuals who have taken the vaccine as a form of protection from the disease. This population is increased by vaccination of susceptible individuals. Often, individuals develop immunity within two (2) weeks after taking the Meningitis vaccines and should protect one for three (3) to five (5) years. Since the vaccines confer varying degrees of immunity to its recipients, the vaccinated individuals may become infected, but at a lower rate than the unvaccinated. The vaccinated class is therefore decreased by been exposed to the disease or by vaccine waning and by natural death.

The Carrier Class is made up of the individuals who have the infection but do not show any signs/symptoms even though they are infectious. The Infected Class are the individuals with the fully blown infection and showing signs/symptoms. This population is said to have survived the average incubation period of four (4) days.

The Treated Class are the individuals undergoing treatment as a result of an infection. Since the after-effects of meningitis aren't always pleasant, the recovered class is divided into two. The first Recovered class $R_1(t)$ are the individuals who have either undergone treatment and have fully recovered from the infection or have recovered by their own natural immunity. The second Recovered class $R_2(t)$ is made up of the individuals who have undergone treatment and have recovered with complications. The recovered classes are decreased due to natural death.

We note that all the model parameters are assumed to be non-negative.

TABLE 1. Description of the Model State Variables

| Variables | Description |
|-----------|------------------------------|
| $S(t)$ | Susceptible Population |
| $V(t)$ | Vaccinated Population |
| $C(t)$ | Carrier Population |
| $I(t)$ | Infected Population |
| $T(t)$ | Treated Population |
| $R_1(t)$ | Fully Recovered Population |
| $R_2(t)$ | Recovered with Complications |

TABLE 2. Description of Model Parameters

| Parameters | Description |
|---------------|--|
| α | Recruitment rate into Susceptible population |
| β | Transmission probability |
| δ | Disease-induced death |
| μ | Natural death rate |
| σ | Progression rate from Carrier to Infected population |
| γ | Recovery rate |
| r | Natural recovery rate |
| θ | Vaccine uptake rate |
| τ | Vaccine efficacy |
| ω | Vaccine waning |
| κ | Treatment rate |
| \wedge | Treatment efficacy |
| η_1 | Modification parameter of infectiousness of the carrier population |
| η_2 | Modification parameter of disease death rate of treated population |
| ε | Complication rate after a period of time |

2.2. Model Assumptions.

- (1) Every individual in the studied population who has not been infected is susceptible to the disease.
- (2) A vaccinated individual who loses immunity becomes susceptible with no vaccine protection.
- (3) The vaccine is only administered to the susceptible population.
- (4) There is a natural death rate from each compartment.
- (5) Recovered individuals cannot be re-infected.

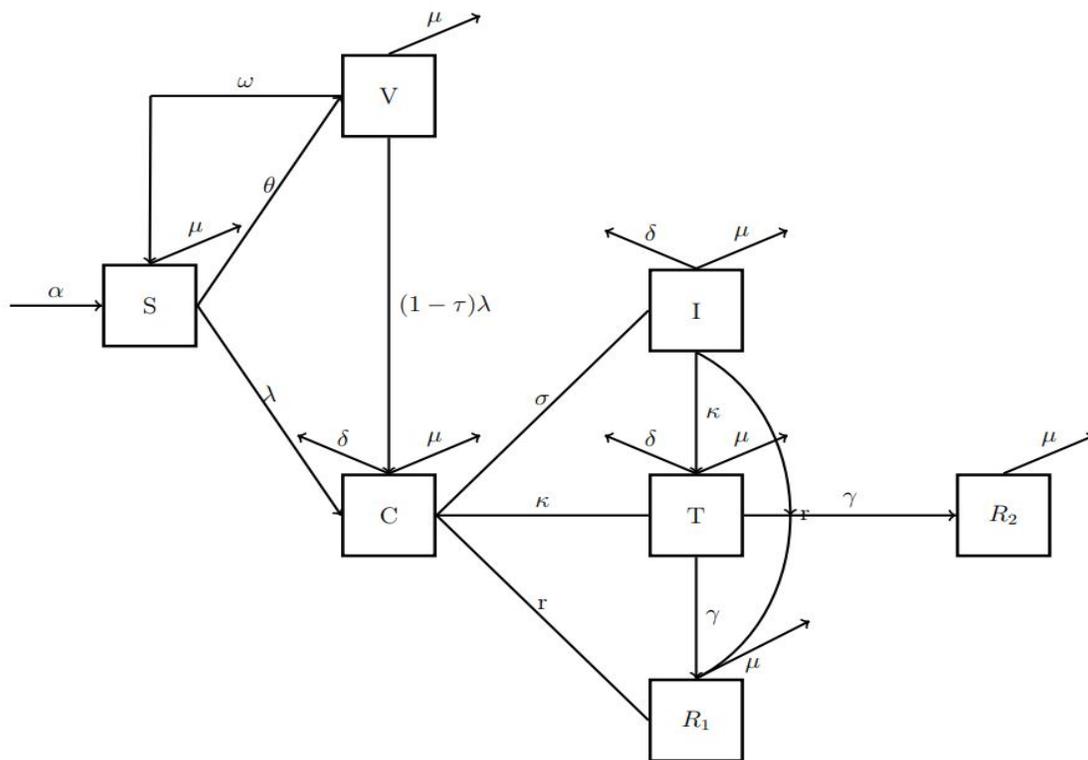


FIGURE 1. Schematic Flow Diagram of the Transmission of Bacterial Meningitis

2.3. Model Equations.

$$(2) \quad \left\{ \begin{array}{l} \frac{dS}{dt} = \alpha + \omega V - (\lambda + \theta + \mu)S \\ \frac{dV}{dt} = \theta S - (1 - \tau)\lambda V - (\omega + \mu)V \\ \frac{dC}{dt} = \lambda S + (1 - \tau)\lambda V - (\sigma + \kappa + r + \delta + \mu)C \\ \frac{dI}{dt} = \sigma C - (r + \kappa + \delta + \mu)I \\ \frac{dT}{dt} = \kappa C + \kappa I - (1 - \eta_2)\delta T - (1 - \wedge)T - (\gamma + \gamma r + \mu)T \\ \frac{dR_1}{dt} = rC + rI + \gamma r T - (\varepsilon + \mu)R_1 \\ \frac{dR_2}{dt} = \gamma T + (1 - \wedge)T + \varepsilon R_1 - \mu R_2 \end{array} \right.$$

3. THE MODEL ANALYSES

3.1. The Invariant Region.

Definition 3.1. A region within which the solutions to the model are uniformly bounded is defined as $\Omega \in \mathfrak{R}_+^7$.

The total population is given as

$$(3) \quad N(t) = S(t) + V(t) + C(t) + I(t) + T(t) + R_1(t) + R_2(t)$$

Therefore

$$(4) \quad \frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dV(t)}{dt} + \frac{dC(t)}{dt} + \frac{dI(t)}{dt} + \frac{dT(t)}{dt} + \frac{dR_1(t)}{dt} + \frac{dR_2(t)}{dt}$$

Substituting (2) into (4) yields

$$(5) \quad \frac{dN(t)}{dt} = \alpha - \mu N - \delta C - \delta I - (1 - \eta_2)\delta T$$

$$\frac{dN(t)}{dt} \leq \alpha - \mu N(t)$$

Integrating both sides, we have

$$-\frac{1}{\mu} \int \frac{\mu}{\alpha - \mu N} \leq \int dt$$

which gives

$$-\frac{1}{\mu} \ln(\alpha - \mu N) \leq t + c$$

where c is the constant of integration.

$$\ln(\alpha - \mu N) \geq -(\mu t + c)$$

$$(\alpha - \mu N) \geq e^{-(\mu t + c)}$$

$$(6) \quad (\alpha - \mu N) \geq ke^{-\mu t}$$

where k is e^c .

Let $N(0) = N_0$. This implies

$$(7) \quad (\alpha - \mu N_0) \geq k$$

From Equations (6) and (7), we get

$$(\alpha - \mu N) \geq (\alpha - \mu N_0)e^{-\mu t}$$

$$\mu N \leq \alpha - (\alpha - \mu N_0)e^{-\mu t}$$

$$N(t) \leq \frac{\alpha}{\mu} - \frac{(\alpha - \mu N_0)}{\mu} e^{-\mu t}$$

$$(8) \quad \Rightarrow N(t) \rightarrow \frac{\alpha}{\mu} \text{ as } t \rightarrow \infty$$

This implies $N(t) \in \left[0, \frac{\alpha}{\mu}\right]$.

Therefore, the feasible set of solution of the model equations enter and remain in the region:

$$(9) \quad \Omega = \{(S, V, C, I, T, R_1, R_2) \in \mathfrak{R}_+^7 : N(t) \leq \frac{\alpha}{\mu}\}$$

We note that $\frac{\alpha}{\mu}$ is the upper bound of $N(t)$. However, if $N > \frac{\alpha}{\mu}$ then $N(t)$ will decrease to $\frac{\alpha}{\mu}$ and the solutions $(S, V, C, I, T, R_1, R_2)$ will enter Ω or approach it asymptotically, as such, the region will attract all solutions in \mathfrak{R}_+^7 . Hence, the model is well posed mathematically and epidemiologically since the region Ω is positively invariant and attracting.

3.2. Positivity of the Solution.

Theorem 3.2. (*The Positivity Theorem*) Let $\Omega = \{(S, V, C, I, T, R_1, R_2) \in \mathfrak{R}_+^7 : S_0 > 0, V_0 > 0, C_0 > 0, I_0 > 0, T_0 > 0, R_{10} > 0, R_{20} > 0\}$, then the solution of $(S, V, C, I, T, R_1, R_2)$ are positive for $t \geq 0$.

Proof.

Considering the first equation of the model

$$\frac{dS}{dt} = \alpha + \omega V - (\lambda + \theta + \mu)S$$

$$\frac{dS}{dt} \geq -(\lambda + \theta + \mu)S$$

$$\int \frac{dS}{S} \geq - \int (\lambda + \theta + \mu) dt$$

$$\ln S(t) \geq -f(t) + c$$

where $f(t) = \int (\lambda + \theta + \mu) dt$ and c is the constant of integration.

$$S(t) \geq e^{(-f(t)+c)}$$

$$S(t) \geq e^{-f(t)} \cdot e^c$$

$$(10) \quad S(t) \geq A_1 e^{-f(t)}$$

where $A_1 = e^c$. From the theorem, at $t = 0$, $S_0 > 0$ which implies $A_1 = e^c \geq 0$ since $S(0) \geq A_1$.

Consequently, $S(t) \geq S_0 e^{-f(t)} \geq 0 \forall t \geq 0$.

Similarly, considering the second equation of the model

$$\frac{dV}{dt} = \theta S - (1 - \tau)\lambda V - (\omega + \mu)V$$

$$\frac{dV}{dt} \geq -[(1 - \tau)\lambda + \omega + \mu]V$$

$$\int \frac{dV}{V} \geq - \int [(1 - \tau)\lambda + \omega + \mu] dt$$

$$\ln V(t) \geq -g(t) + c$$

where $g(t) = \int[(1 - \tau)\lambda + \omega + \mu]dt$ and c is the constant of integration.

$$(11) \quad V(t) \geq A_2 e^{-g(t)}$$

where $A_2 = e^c$. At $t = 0$, $V_0 > 0$ which implies $A_2 = e^c \geq 0$.

Consequently, $V(t) \geq V_0 e^{-g(t)} \geq 0 \quad \forall t \geq 0$.

Applying the same technique to the remaining equations of the system, the third equation yields

$$(12) \quad C(t) \geq C_0 e^{-bt} \geq 0 \quad \forall t \geq 0$$

where $b = (\sigma + \kappa + r + \delta + \mu)$.

The fourth equation yields

$$(13) \quad I(t) \geq I_0 e^{-dt} \geq 0 \quad \forall t \geq 0$$

where $d = (r + \kappa + \delta + \mu)$.

The fifth equation yields

$$(14) \quad T(t) \geq T_0 e^{-gt} \geq 0 \quad \forall t \geq 0$$

where $g = [(1 - \eta_2)\delta + (1 - \lambda) + \gamma + \gamma r + \mu]$.

The sixth equation yields

$$(15) \quad R_1(t) \geq R_{10} e^{-ht} \geq 0 \quad \forall t \geq 0$$

where $h = (\varepsilon + \mu)$.

Lastly, the seventh equation yields

$$(16) \quad R_2(t) \geq R_{20} e^{-\mu t} \geq 0 \quad \forall t \geq 0$$

This completes the proof of the theorem.

4. EXISTENCE OF EQUILIBRIA AND BASIC REPRODUCTION NUMBER

For the developed model, the disease free and endemic equilibrium points are obtained. A disease free equilibrium is a state solution to the model in which the studied population remains in the absence of the disease. An endemic equilibrium point of a disease is defined as a positive steady state solution when the disease persists in the studied population.

4.1. The Disease Free Equilibrium Point. The DFE of the model is defined as

$$(S^*(t), V^*(t), 0, 0, 0, 0, 0) \text{ satisfying } \frac{dS(t)}{dt} = \frac{dV(t)}{dt} = \frac{dC(t)}{dt} = \frac{dI(t)}{dt} = \frac{dT(t)}{dt} = \frac{dR_1(t)}{dt} = \frac{dR_2(t)}{dt} = 0.$$

Equating the system of equations in (2) to 0 and substituting $C = I = T = R_1 = R_2 = 0$, we obtain the system

$$(17) \quad \begin{cases} \omega V - (\theta + \mu) S = -\alpha \\ \theta S - (\omega + \mu) V = 0 \end{cases}$$

Solving the system simultaneously, the DFE is obtained as:

$$(18) \quad \left(\frac{\alpha b_1}{\mu (b_1 + \theta)}, \frac{\alpha \theta}{\mu (b_1 + \theta)}, 0, 0, 0, 0, 0 \right)$$

where, $b_1 = \omega + \mu$.

4.2. Endemic Equilibrium Point. The EEP of the model is defined as

$$(S^*(t), V^*(t), C^*(t), I^*(t), T^*(t), R_1^*(t), R_2^*(t)) \text{ satisfying } \frac{dS(t)}{dt} = \frac{dV(t)}{dt} = \frac{dC(t)}{dt} = \frac{dI(t)}{dt} = \frac{dT(t)}{dt} = \frac{dR_1(t)}{dt} = \frac{dR_2(t)}{dt} = 0.$$

This yields the system of equations

$$(19) \quad \left\{ \begin{array}{l} \alpha + \omega V - (\lambda + \theta + \mu) S = 0 \\ \theta S - (1 - \tau) \lambda V - (\omega + \mu) V = 0 \\ \lambda S + (1 - \tau) \lambda V - (\sigma + \kappa + r + \delta + \mu) C = 0 \\ \sigma C - (\kappa + r + \delta + \mu) I = 0 \\ \kappa C + \kappa I - (1 - \eta_2) \delta T - (1 - \Lambda) T - (\gamma + \gamma r + \mu) T = 0 \\ rC + rI + \gamma r T - (\varepsilon + \mu) R_1 = 0 \\ \gamma T + (1 - \Lambda) T + \varepsilon R_1 - \mu R_2 = 0 \end{array} \right.$$

which results in,

$$\begin{aligned} S^* &= \frac{\alpha [(1 - \tau) \lambda + b_1]}{(1 - \tau) \lambda^2 + G_1 \lambda + G_2}, & V^* &= \frac{\alpha \theta}{(1 - \tau) \lambda^2 + G_1 \lambda + G_2}, \\ C^* &= \frac{\alpha \lambda [(1 - \tau) (\lambda + \theta) + b_1]}{(\sigma + b_2) [(1 - \tau) \lambda^2 + G_1 \lambda + G_2]}, & I^* &= \frac{\alpha \lambda \sigma [(1 - \tau) (\lambda + \theta) + b_1]}{b_2 (\sigma + b_2) [(1 - \tau) \lambda^2 + G_1 \lambda + G_2]}, \\ T^* &= \frac{\kappa (C^* + I^*)}{b_3}, & R_1^* &= \frac{r (C^* + I^* + \gamma T^*)}{\varepsilon + \mu} \\ R_2^* &= \frac{(\varepsilon + \mu) (r + 1 - \Lambda) T^* + r \varepsilon (C^* + I^* + \gamma T^*)}{\mu (\varepsilon + \mu)} \end{aligned}$$

where,

$$b_2 = \kappa + r + \delta + \mu, \quad b_3 = \mu + \gamma(r + 1) + \delta(1 - \eta_2) + 1 - \Lambda$$

$$G_1 = (1 - \tau) (\mu + \theta) + b_1, \quad G_2 = \mu (b_1 + \theta)$$

From the force of infection in Equation (1),

$$\lambda^* = \frac{\beta (\eta_1 C^* + I^*)}{N^*}$$

which can be written as

$$(20) \quad \lambda^* N^* - \beta (\eta_1 C^* + I^*) = 0$$

Substituting all the state solutions into Equation (20) and simplifying leads to the equation

$$(21) \quad K_1 (\lambda^*)^2 + K_2 \lambda^* + K_3 = 0$$

where,

$$K_1 = (\sigma + b_2) (1 - \tau) [b_3 (\mu + r + k) - k\delta (1 - \eta_2)]$$

$$K_2 = \mu (1 - \tau) b_3 b_2^2 + [(b_2 - \delta) ((1 - \tau) \theta + b_1) - \mu (1 - \tau) (\beta \eta_1 - \sigma)] b_2 b_3 + \\ [(b_2 - \delta) ((1 - \tau) \theta + b_1) - \mu \beta (1 - \tau)] \sigma b_3 - \delta (\sigma + b_2) (1 - \eta_2) ((1 - \tau) \theta + b_1) \kappa$$

$$K_3 = \mu b_3 [(\theta + b_1) b_2^2 + (\theta + b_1) \sigma b_2 + \beta (\theta \tau - \theta - b_1) (\eta_1 b_2 + \sigma)] \\ = \mu b_3 [b_2 (\theta + b_1) (b_2 + \sigma) - \beta ((1 - \tau) \theta + b_1) (\eta_1 b_2 + \sigma)] \\ = \mu b_3 b_2 (\theta + b_1) (b_2 + \sigma) \left[1 - \frac{\beta ((1 - \tau) \theta + b_1) (\eta_1 b_2 + \sigma)}{b_2 (\theta + b_1) (b_2 + \sigma)} \right] \\ = \mu b_2 b_3 (\theta + b_1) (\sigma + b_2) (1 - \mathcal{R}_0)$$

4.3. The Basic Reproduction Number. The basic reproduction number is a fundamental threshold in mathematical study of epidemiology. It helps to forecast the transmission potential of a disease. The basic reproduction number associated with (2) is given as:

$$\begin{pmatrix} \frac{dC}{dt} \\ \frac{dI}{dt} \end{pmatrix} = f_i - v_i$$

where,

$$f_i = \begin{pmatrix} \frac{\beta (\eta_1 C + I) S}{N} + \frac{(1 - \tau) \beta (\eta_1 C + I) V}{N} \\ 0 \end{pmatrix}$$

and

$$v_i = \begin{pmatrix} (\sigma + b_2)C \\ -\sigma C + b_2 I \end{pmatrix}$$

f_i is the rate at which new infections appear in compartment i and v_i represents the movement of individuals into compartment i , with $i \in [1, 2]$.

The matrices \mathcal{F} and \mathcal{V} are obtained as follows:

$$(22) \quad \mathcal{F} = \begin{pmatrix} \frac{\partial f_1}{\partial C} & \frac{\partial f_1}{\partial I} \\ \frac{\partial f_2}{\partial C} & \frac{\partial f_2}{\partial I} \end{pmatrix} = \begin{pmatrix} \frac{\beta \eta_1 (\theta(1-\tau) + b_1)}{b_1 + \theta} & \frac{\beta (\theta(1-\tau) + b_1)}{b_1 + \theta} \\ 0 & 0 \end{pmatrix}$$

and

$$(23) \quad \mathcal{V} = \begin{pmatrix} \frac{\partial v_1}{\partial C} & \frac{\partial v_1}{\partial I} \\ \frac{\partial v_2}{\partial C} & \frac{\partial v_2}{\partial I} \end{pmatrix} = \begin{pmatrix} \sigma + b_2 & 0 \\ -\sigma & b_2 \end{pmatrix}$$

$$(24) \quad \mathcal{V}^{-1} = \begin{pmatrix} \frac{1}{\sigma + b_2} & 0 \\ \frac{\sigma}{b_2(\sigma + b_2)} & \frac{1}{b_2} \end{pmatrix}$$

Thus, the next generation matrix:

$$(25) \quad G = \mathcal{F} \mathcal{V}^{-1} = \begin{pmatrix} \frac{(\eta_1 b_2 + \sigma)((1-\tau)\theta + b_1)\beta}{b_2(b_1 + \theta)(\sigma + b_2)} & \frac{\beta(\theta(1-\tau) + b_1)}{b_2(b_1 + \theta)} \\ 0 & 0 \end{pmatrix}$$

The eigenvalues of the matrix, G are

$$\begin{pmatrix} 0 \\ \frac{\beta(\eta_1 b_2 + \sigma)(\theta(1-\tau) + b_1)}{b_2(b_1 + \theta)(\sigma + b_2)} \end{pmatrix}$$

Consequently, the Basic Reproduction Number, which is the spectral radius of G is given as

$$(26) \quad \mathcal{R}_0 = \frac{\beta(\eta_1 b_2 + \sigma)[(1-\tau)\theta + b_1]}{b_2(b_1 + \theta)(\sigma + b_2)}$$

\mathcal{R}_0 provides the expected number of newly infected individuals that would arise from introduction of a single case of bacterial meningitis into a completely susceptible population.

5. STABILITY ANALYSIS

5.1. Local Stability of the Disease-free Equilibrium.

Theorem 5.1. *The DFE is Locally Asymptotically Stable (LAS) if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Using Theorem 5.1, the result in Lemma 5.2 follows immediately based on the expressions of \mathcal{R}_0 .

Lemma 5.2. *The DFE of the bacterial meningitis model in (2) is Locally Asymptotically Stable (LAS) if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof.

The Jacobian matrix, J evaluated at E_0 is given as

$$(27) \quad J = \begin{pmatrix} -(\theta + \mu) & \omega & -\frac{\beta \eta_1 b_1}{b_1 + \theta} & -\frac{\beta b_1}{b_1 + \theta} & 0 & 0 & 0 \\ \theta & -b_1 & -\frac{\beta \eta_1 (1-\tau)\theta}{b_1 + \theta} & -\frac{\beta (1-\tau)\theta}{b_1 + \theta} & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta \eta_1 b_1 (1-\tau)\theta}{b_1 + \theta} - (\sigma + b_2) & \frac{\beta b_1 (1-\tau)\theta}{b_1 + \theta} & 0 & 0 & 0 \\ 0 & 0 & \sigma & -b_2 & 0 & 0 & 0 \\ 0 & 0 & \kappa & \kappa & -b_3 & 0 & 0 \\ 0 & 0 & r & r & \gamma r & -(\varepsilon + \mu) & 0 \\ 0 & 0 & 0 & 0 & \gamma + (1 - \wedge) & \varepsilon & -\mu \end{pmatrix}$$

The eigenvalues of the Jacobian matrix, J are $-\mu$ (of multiplicity 2), $-(b_1 + \theta)$, $-(\varepsilon + \mu)$, $-b_3$ and $\frac{-\beta \tau \theta \eta_1 + (-\beta \eta_1 + \sigma + 2b_2)(b_1 + \theta) - \sqrt{(\beta \tau \theta \eta_1 - (\beta \eta_1 - \sigma)(b_1 + \theta))^2 + 4(\theta(1-\tau) + b_1)(b_1 + \theta)\sigma\beta}}{2b_1 + 2\theta}$ (of multiplicity 2). Clearly, all the eigenvalues of the Jacobian matrix are strictly negative provided

$$\frac{-\beta \tau \theta \eta_1 + (-\beta \eta_1 + \sigma + 2b_2)(b_1 + \theta) - \sqrt{(\beta \tau \theta \eta_1 - (\beta \eta_1 - \sigma)(b_1 + \theta))^2 + 4(\theta(1-\tau) + b_1)(b_1 + \theta)\sigma\beta}}{2b_1 + 2\theta} < 0$$

Thus for stability, the negativity condition imposed yields

$$\beta \tau \theta \eta_1 + (-\beta \eta_1 + \sigma + 2b_2)(b_1 + \theta) - \sqrt{(\beta \tau \theta \eta_1 - (\beta \eta_1 - \sigma)(b_1 + \theta))^2 + 4(\theta(1-\tau) + b_1)(b_1 + \theta)\sigma\beta} > 0$$

$$\begin{aligned}
& [\beta \tau \theta \eta_1 + (-\beta \eta_1 + \sigma + 2b_2)(b_1 + \theta)]^2 > \left[\sqrt{(\beta \tau \theta \eta_1 - (\beta \eta_1 - \sigma)(b_1 + \theta))^2 + 4(\theta(1 - \tau) + b_1)(b_1 + \theta)\sigma\beta} \right]^2 \\
& [\beta \tau \theta \eta_1 + (-\beta \eta_1 + \sigma + 2b_2)(b_1 + \theta)]^2 > (\beta \tau \theta \eta_1 - (\beta \eta_1 - \sigma)(b_1 + \theta))^2 + 4(\theta(1 - \tau) + b_1)(b_1 + \theta)\sigma\beta \\
& (\beta \tau \theta \eta_1 + (-\beta \eta_1 + \sigma + 2b_2)(b_1 + \theta))^2 - (\beta \tau \theta \eta_1 - (\beta \eta_1 - \sigma)(b_1 + \theta))^2 - 4(\theta(1 - \tau) + b_1)(b_1 + \theta)\sigma\beta > 0 \\
& ((b_1 + \theta)b_2^2 + ((\beta(-1 + \tau)\eta_1 + \sigma)\theta - (\beta\eta_1 - \sigma)b_1)b_2 + \sigma(\theta(-1 + \tau) - b_1)\beta)(b_1 + \theta) > 0 \\
& \beta\theta(b_2\eta_1 + \sigma)(\theta\tau - \theta - b_1) + \beta b_1(b_2\eta_1 + \sigma)(\theta\tau - \theta - b_1) + b_2(b_1 + \theta)^2(\sigma + b_2) > 0 \\
& (\theta(-1 + \tau) - b_1)\theta(b_2\eta_1 + \sigma)\beta + b_1(\theta(-1 + \tau) - b_1)(b_2\eta_1 + \sigma)\beta + b_2(b_1 + \theta)^2(\sigma + b_2) > 0 \\
& \beta(b_2\eta_1 + \sigma)(\theta\tau - \theta - b_1)(b_1 + \theta) + b_2(b_1 + \theta)^2(\sigma + b_2) > 0 \\
& b_2(b_1 + \theta)(\sigma + b_2) - \beta(b_2\eta_1 + \sigma)(\theta(1 - \tau) + b_1) > 0 \\
& (b_1 + \theta)b_2(\sigma + b_2) \left(1 - \frac{\beta(b_2\eta_1 + \sigma)(\theta(1 - \tau) + b_1)}{b_2(b_1 + \theta)(\sigma + b_2)} \right) > 0 \\
(28) \qquad \qquad \qquad & (b_1 + \theta)b_2(\sigma + b_2)(1 - \mathcal{R}_0) > 0
\end{aligned}$$

Therefore, for Equation (28) to be valid, \mathcal{R}_0 must be less than 1. Hence the DFE is LAS.

5.2. Global Stability of the Disease-free Equilibrium. The global asymptotic stability of the model in (2) is investigated by following Castillo-Chavez, Feng and Huang [5]. The model is denoted by:

$$(29) \qquad \qquad \qquad \begin{cases} \frac{dX}{dt} = F(X, Y) \\ \frac{dY}{dt} = G(X, Y) \end{cases}$$

where $X = (S, V, R_1, R_2)$ denotes the uninfected population and $Y = (C, I, T)$ denotes the infected population.

Theorem 5.3. *The Disease-Free Equilibrium is said to be globally asymptotically stable in Ω if $\mathcal{R}_0 < 1$ and the following two conditions hold:*

C1: For $\frac{dX}{dt} = F(X, 0)$, E_0 is globally asymptotically stable.

C2: $G(X, Y) = J[G(X^*, 0)]Y - \hat{G}(X, Y)$, $\hat{G}(X, Y) \geq 0$, $\forall (X, Y) \in \Omega$

where $(X^*, 0) = E_0 = \left(\frac{\alpha b_1}{\mu(b_1 + \theta)}, \frac{\alpha \theta}{\mu(b_1 + \theta)}, 0, 0, 0, 0, 0\right)$, $J[G(X^*, 0)]$ is the Jacobian of $G(X, Y)$ obtained with respect to (C, I, T) and evaluated at $(X^*, 0)$.

Proof.

C1: From the model, it follows that:

$$(30) \quad F(X, 0) = \begin{pmatrix} \alpha + \omega V - (\theta + \mu)S \\ \theta S - b_1 V \\ -(\varepsilon + \mu)R_1 \\ \varepsilon R_1 - \mu R_2 \end{pmatrix}$$

From Equation (30), it is clear that

$$E_0 = (S, V, C, I, T, R_1, R_2) = \left(\frac{\alpha b_1}{\mu(b_1 + \theta)}, \frac{\alpha \theta}{\mu(b_1 + \theta)}, 0, 0, 0, 0, 0\right)$$

This can be verified using the method of integrating factors. From Equation (30), we have:

$$(31) \quad \frac{dV}{dt} = \theta S - b_1 V$$

which can be written in standard form as

$$(32) \quad \frac{dV}{dt} + b_1 V = \theta S$$

The integrating factor is given as $I.F. = e^{\int b_1 dt} = e^{b_1 t}$.

Multiplying Equation (32) through by the integrating factor yields

$$(33) \quad e^{b_1 t} \left(\frac{dV}{dt} + b_1 V\right) = \theta S e^{b_1 t}$$

$$(34) \quad \int \frac{d}{dt} \left(V e^{b_1 t}\right) dt = \theta \int S e^{b_1 t} dt$$

Let $I = \int S e^{b_1 t} dt$. Integrating by parts, we have

$$u = S \implies du = S' dt, \text{ and } dv = e^{b_1 t} \implies v = \frac{e^{b_1 t}}{b_1}$$

So,

$$(35) \quad I = \frac{Se^{b_1 t}}{b_1} - \frac{1}{b_1} \int S' e^{b_1 t} dt$$

$$(36) \quad \Rightarrow Ve^{b_1 t} = \theta \left[\frac{Se^{b_1 t}}{b_1} - \frac{1}{b_1} \int S' e^{b_1 t} dt \right]$$

$$(37) \quad = \frac{\theta S}{b_1} e^{b_1 t} - \frac{\theta}{b_1} \int S' e^{b_1 t} dt$$

Therefore,

$$(38) \quad V = \frac{\theta S}{b_1} - \frac{\theta}{b_1 e^{b_1 t}} \int S' e^{b_1 t} dt$$

From Equation (38), $V \rightarrow \frac{\theta S}{b_1}$ as $t \rightarrow \infty$.

Furthermore, from Equation (30), we have,

$$(39) \quad \frac{dS}{dt} = \alpha + \omega V - (\theta + \mu)S$$

Since $V \rightarrow \frac{\theta S}{b_1}$, Equation (39) is rewritten as

$$(40) \quad \frac{dS}{dt} = \alpha + \frac{\omega \theta S}{b_1} - (\theta + \mu)S$$

$$(41) \quad = \alpha - \frac{\mu(b_1 + \theta)}{b_1} S$$

Therefore, Equation (41) can be put in standard form as

$$(42) \quad \frac{dS}{dt} + \frac{\mu(b_1 + \theta)}{b_1} S = \alpha$$

The integrating factor is given as $I.F. = e^{\int \frac{\mu(b_1 + \theta)}{b_1} dt} = e^{\frac{\mu(b_1 + \theta)}{b_1} t}$.

Multiplying Equation (42) through by the integrating factor gives

$$(43) \quad e^{\frac{\mu(b_1 + \theta)}{b_1} t} \left(\frac{dS}{dt} + \frac{\mu(b_1 + \theta)}{b_1} S \right) = \alpha e^{\frac{\mu(b_1 + \theta)}{b_1} t}$$

$$(44) \quad \int \frac{d}{dt} \left(S e^{\frac{\mu(b_1 + \theta)}{b_1} t} \right) dt = \int \alpha e^{\frac{\mu(b_1 + \theta)}{b_1} t} dt$$

$$(45) \quad S e^{\frac{\mu(b_1 + \theta)}{b_1} t} = \frac{\alpha b_1}{\mu(b_1 + \theta)} e^{\frac{\mu(b_1 + \theta)}{b_1} t} + c$$

where c is the constant of integration. Therefore,

$$(46) \quad S = \frac{\alpha b_1}{\mu(b_1 + \theta)} + Ce^{-\frac{\mu(b_1 + \theta)}{b_1}t}$$

From Equation (46), $S \rightarrow \frac{\alpha b_1}{\mu(b_1 + \theta)}$ as $t \rightarrow \infty$; and this implies the global convergence of Equation (30) in Ω .

C2: $G(X, Y)$ is given as

$$(47) \quad G(X, Y) = \begin{bmatrix} \lambda S + (1 - \tau)\lambda V - (\sigma + b_2)C \\ \sigma C - b_2 I \\ \kappa C + \kappa I - b_3 T \end{bmatrix}$$

where λ is the force of infection defined in Equation (1).

The Jacobian matrix of $G(X, Y)$, $J[G(X^*, 0)]$ is given as

$$(48) \quad \begin{pmatrix} \frac{\beta \eta_1 [S^* + (1 - \tau)V^*]}{N^*} - \sigma - b_2 & \frac{\beta [S^* + (1 - \tau)V^*]}{N^*} & 0 \\ \sigma & -b_2 & 0 \\ \kappa & \kappa & -b_3 \end{pmatrix}$$

By the condition in C2 with Equations (47) and (48), $\hat{G}(X, Y)$ is given by

$$(49) \quad \begin{pmatrix} \frac{\beta(\eta_1 C + I)[(1 - \tau)V^* + S^*]}{N^*} \left(1 - \frac{V(1 - \tau) + S}{N} \frac{N^*}{(1 - \tau)V^* + S^*} \right) \\ 0 \\ 0 \end{pmatrix}$$

Since

$$S^* = \frac{\alpha b_1}{\mu(b_1 + \theta)}, \quad V^* = \frac{\alpha \theta}{\mu(b_1 + \theta)} \quad \text{and} \quad N^* = \frac{\alpha}{\mu}$$

we have that $S \leq S^*$, and $V \leq V^*$. Thus, it follows that $S \leq N$, and $V \leq N$ in Ω . Therefore, if the total population is at equilibrium level, we have $\left(1 - \frac{V(1 - \tau) + S}{N} \frac{N^*}{(1 - \tau)V^* + S^*} \right) > 0$; thus, $\hat{G}(X, Y) \geq 0$. Hence it follows from Theorem (5.3) that the DFE, $E_0 = (X^*, 0)$ is globally asymptotically stable.

6. MODEL PARAMETER ESTIMATION

6.1. Initial Conditions. Ghana's demographic data for the year 2017 is adopted for our simulation. Since the disease is endemic in the northern part of Ghana, the total population of the northern part as at 2017 was 4953293, GSS [8], as such the initial total population, $N(0) = 4953293$. It is known that 10 – 20% of every population is carrier of Meningitis, WHO [18], so the average which is 15% is adopted as the case in Ghana. This gives a carrier population of about 742993.95. 10% of the population is assumed to be vaccinated against the disease. In addition, it is assumed that the population in each of the infected and treated class is about one-third of those in carrier class, which is 247664.65. The two recovered classes is assumed to be zero. Thus, the model variables' initial conditions are: $S(0) = 3219640$, $V(0) = 495329.3$, $C(0) = 742993.95$, $I(0) = T(0) = 247664.65$, $R_1(0) = 0$ and $R_2(0) = 0$.

6.2. Model Parameter Values.

- (1) Natural death rate (μ): The average life span in Ghana is 64.17 years, therefore $\mu = \frac{1}{64.17 \times 365} = 4.269 \times 10^{-5}$ per day.
- (2) Birth or recruitment rate (α): In the absence of the disease, the limiting total human population is assumed to be $\frac{\alpha}{\mu} = 4953293$, so $\alpha = 211$ per day.
- (3) Disease-induced death rate (δ): The mortality rate due to bacterial meningitis disease in Ghana is 36 – 50%. Taking the average to be 43% gives $\delta = 0.43$.
- (4) Progression rate (σ): The average incubation period is 4 days. Thus, $\sigma = \frac{1}{4} = 0.25$
- (5) Vaccine waning rate (ω): It takes an average of 4 years for the available vaccines to wane. Therefore, $\omega = \frac{1}{4 \times 365} = 6.8 \times 10^{-4}$ per day
- (6) Recovery rate (γ): The period of infection of the disease is 1-2 weeks with hospitalization and right treatment, so taking the average, we have 8 days. Therefore, $\gamma_1 = \frac{1}{8} = 0.125$.
- (7) Complication rate (ε): Even with appropriate treatment, 10 – 20% of survivors have serious complications or long-term sequelae. Therefore, $\varepsilon = \frac{15}{100} = 0.15$.

TABLE 3. Model Parameter Values

| Parameter | Value | Source |
|---------------|---------------|---------------------------|
| α | 211 | Estimated |
| μ | 0.000043 | Estimated |
| ω | 0.00068 | Estimated |
| β | 0.88 | Asamoah <i>et al.</i> [3] |
| γ | 0.125 | Estimated |
| r | 0.13 | Asamoah <i>et al.</i> [3] |
| η_1 | 0.75 | Assumed |
| η_2 | 0.75 | Assumed |
| δ | 0.43 | Estimated |
| ε | 0.15 | Estimated |
| σ | 0.25 | Estimated |
| τ | 0.85 | Elmojtaba and Adam [7] |
| κ | 0.6 [0,1] | Assumed |
| θ | 0.6 [0,1] | Assumed |
| \wedge | 0.6 [0.1-0.9] | Elmojtaba and Adam [7] |

We note that the set of parameter values in Table (3) yields a basic reproduction number less than unity ($\mathcal{R}_0 = 0.091$) which implies that with effective vaccination and treatment, this disease which is considered to be endemic could be eradicated.

7. SENSITIVITY ANALYSIS

In modeling infectious diseases, it is pertinent to ascertain the major model parameters influencing the disease's transmission. Sensitivity analysis is therefore performed to determine the model's robustness predictions to parameter values.

Definition 7.1. The normalized forward sensitivity index of \mathcal{R}_0 , that depends differentially on a parameter ψ , is defined by

$$(50) \quad S_{\psi}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \psi} \times \frac{\psi}{\mathcal{R}_0}$$

In particular, the sensitivity index is a local estimate to establishing an efficient way of reducing \mathcal{R}_0 .

Therefore, all the partially differentiable model parameters with respect to \mathcal{R}_0 , their values and sensitivity indices are given in Table 4.

TABLE 4. Sensitivity Index of Each Model Parameter on \mathcal{R}_0

| Parameter | Parameter Value | Sensitivity Index |
|-----------|-----------------|------------------------|
| ω | 0.00068 | $+6.36 \times 10^{-3}$ |
| β | 0.88 | +1 |
| η_1 | 0.75 | +0.7768 |
| σ | 0.25 | +0.0459 |
| δ | 0.43 | -0.3877 |
| μ | 0.000043 | -3.64×10^{-4} |
| κ | 0.6 | -0.5410 |
| τ | 0.85 | -5.6215 |
| r | 0.13 | -0.1172 |
| θ | 0.6 | -6.77×10^{-3} |

A positive sensitivity index suggests that the parameter is directly proportional to the value of \mathcal{R}_0 . Thus, an increase in any of the values of ω , β , η_1 and σ by some percentage will increase the value of \mathcal{R}_0 , thereby increasing the spread of the disease, and vice versa. However, the parameters with a negative sensitivity index means that these parameters are inversely proportional to the value of \mathcal{R}_0 . Therefore, when the value of any of these parameters, δ , μ , κ , τ , r , θ is increased while holding all other parameters constant, it will reduce the value of \mathcal{R}_0 and, hence, contribute to the eradication of the disease and vice versa. For instance, increasing the

modification parameter of the infectiousness of the carrier, η_1 by 10% will lead to a 7.768% increase on \mathcal{R}_0 while increasing the treatment rate, κ by 10% will result in a reduction of 5.410% on \mathcal{R}_0 .

8. NUMERICAL RESULTS AND DISCUSSIONS

The numerical solutions of the model (2) is obtained by using MATLAB ODE45 Algorithm for solving non-stiff system of ordinary differential equations with the initial conditions and parameter values estimated.

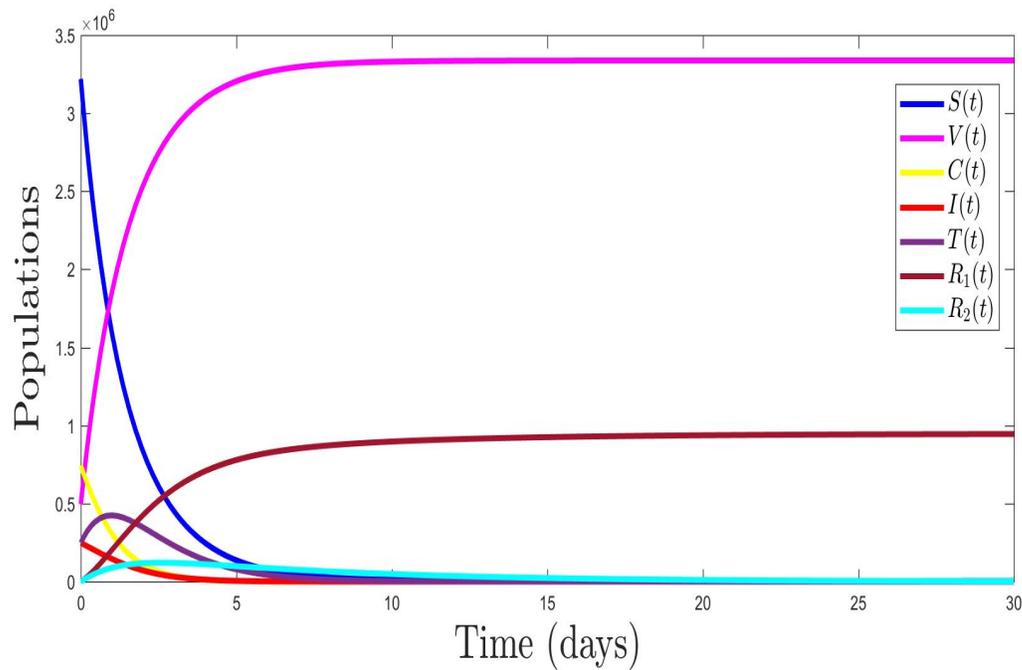


FIGURE 2. Evolution of each subpopulation with Time

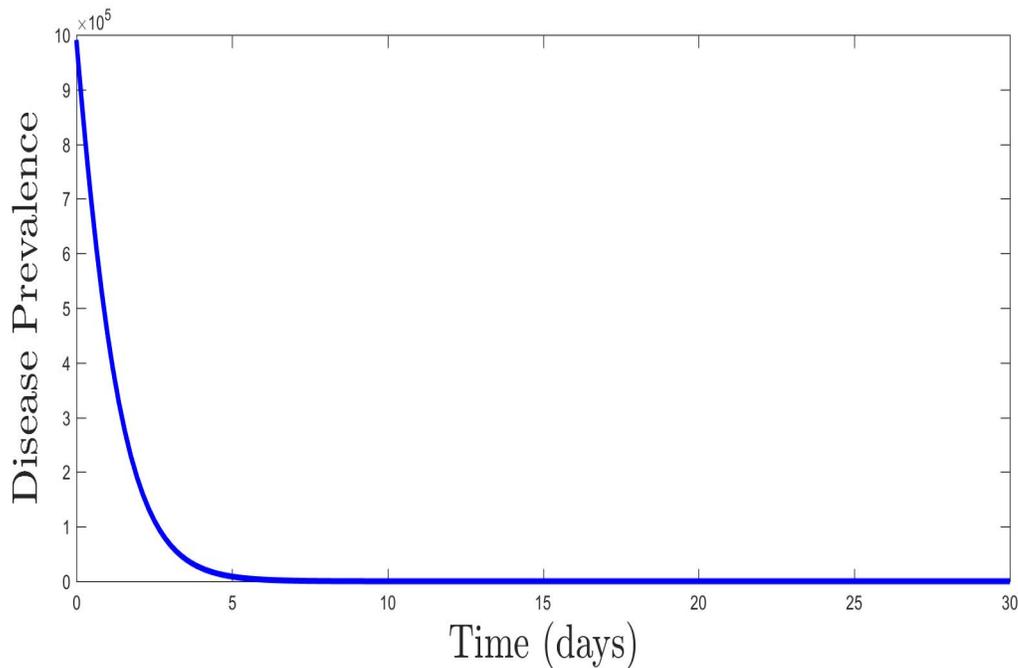
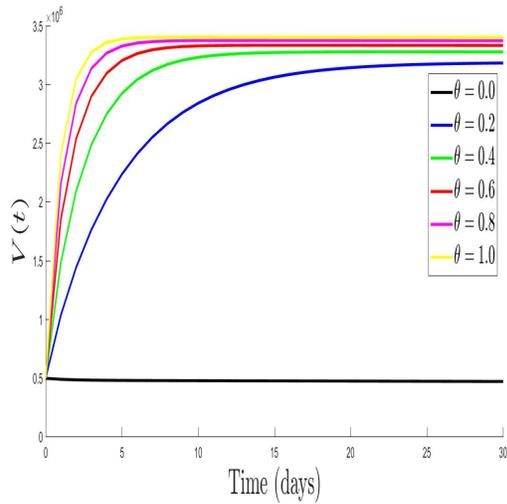
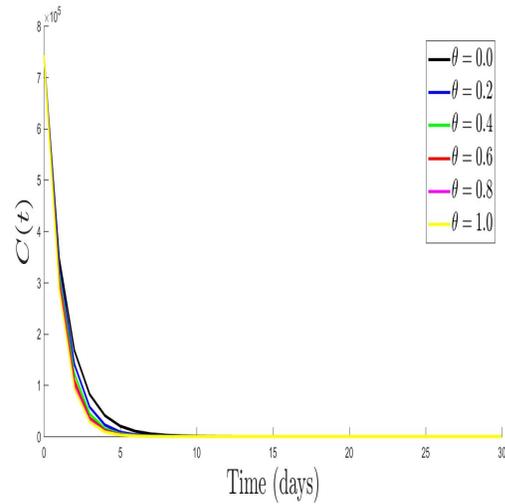
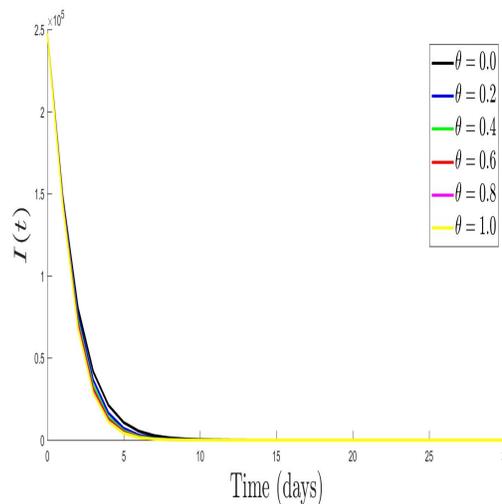


FIGURE 3. Disease Prevalence

Figure (2) gives the numerical simulation of the model compartments in a time span of 30 days. The susceptible population decreases rapidly within the first few days due to getting people vaccinated and the force of infection. However, after these few days, stationarity is achieved due to progression to the other compartments. The vaccinated population on the other hand increases rapidly within the first few days, and this can be attributed to the awareness and sensitivity of the government to get people vaccinated as soon as an infection strikes. The carrier population reduces drastically in size due to the intervention of early treatment given to people who have come into contact with an infected person and the progression of the carriers to the infected class since the period of incubation is very short. The infected population also decreases with time and this can be ascribed to the immediate treatment given to them since the disease is termed as a 'medical emergency'. There is a short increase in the treated class as a result of progression of the carrier and infected but later decreases with time. This decrease is due to the treated population moving to the recovered populations. The two recovered populations increase and remain stable after a period of time.

(A) Effects of Varying θ on $V(t)$ (B) Effects of Varying θ on $C(t)$ (C) Effects of Varying θ on $I(t)$ FIGURE 4. Effects of Varying θ on $V(t)$, $C(t)$ and $I(t)$ Compartments

From figure (4), as the vaccine uptake rate increases, the vaccinated population in figure (4(a)) increases and remains stable. There is also a sharp decrease in both the carrier population in figure (4(b)) and infected population in figure (4(c)) even with a small vaccine uptake rate. This shows that infection will be controlled if people continue to receive vaccination.

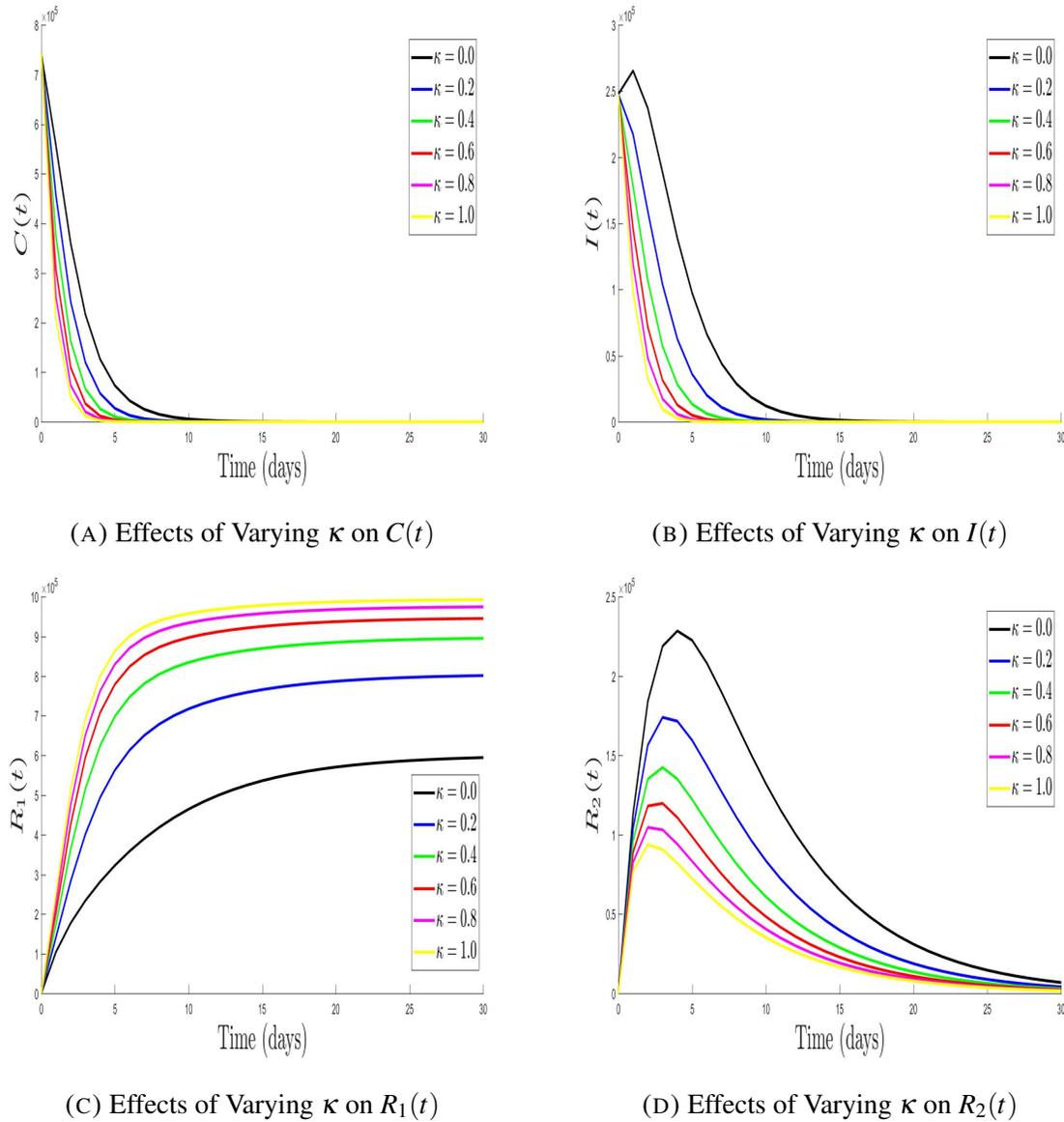


FIGURE 5. Effects of Varying κ on $C(t)$, $I(t)$, $R_1(t)$ and $R_2(t)$ Compartments

Figure (5) shows that as the treatment rate increases, there is a rapid decrease in both the carrier population in figure (5(a)) and infected population in figure (5(b)). Also, the higher the treatment rate, the more people get fully recovered in figure (5(c)) and the less people recover with complications as seen in figure (5(d)).

9. CONCLUSION

The transmission dynamics of bacterial meningitis with a focus on vaccination and effective treatment in curtailing the spread of the disease is presented. The basic reproduction number of the model is computed using the Next Generation matrix. The equilibrium solutions of the model are obtained and used to establish criteria for the model's stability. Using the basic reproduction number, \mathcal{R}_0 , as a threshold given $\mathcal{R}_0 < 1$, the disease-free equilibrium point is established to be both locally and globally asymptotically stable. This study relates to the fact that Bacterial meningitis is a vaccine preventable disease. This is because as the vaccine uptake rate increases, the vaccinated population increases and remains stable. The numerical simulations established that the disease can be eradicated with effective and efficient vaccination and treatment since that led the basic reproduction number below unity. The contributions of the model parameters on \mathcal{R}_0 using the normalized sensitivity index was examined. The results indicate that the transmission probability, β is an effective contributor to \mathcal{R}_0 , as such very essential in the spread and control of the disease. Therefore, control mechanisms that can reduce the transmission probability significantly will most definitely curtail the endemicity of the disease.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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