A Novel Model for Transmission Dynamics of Bacterial Meningitis Incorporating Vaccination and Treatment Using Counterfeit and Non-Counterfeit Drugs.

Abstract: The protective membranes around the brain and spinal cord become inflamed or swollen when meningitis occurs. The original mode of transmission for bacterial meningitis was from animal to human, but it can now spread from person to person through contaminated surfaces, saliva, respiratory secretions, and aerosol droplets. This study anaylse bacterial meningitis using counterfeit and non-counterfeit drugs. Numerous mathematical representations regarding bacterial meningitis transmission formerly been put up and examined, finding out what happen when you subject infected individuals to medication with counterfeit and non-counterfeit drugs is our objectives. According to the model analysis, the endemic equilibrium is only locally asymptotically stable when the basic reproduction number is smaller than unity, while the disease-free equilibrium is asymptotically stable both locally and globally. Using counterfeit drugs on infected individuals may have devastating effect on these individuals as most may never recover leading to high mortality case. Areas with weaker systems and less regulatory control may face challenges in ensuring that all patients receive genuine medications. MATLAB software is utilized to do numerical simulations that illustrate the influence of counterfeit and non-counterfeit drugs resulting to severe illness on areas prone to counterfeit drugs. A coordinated effort among governments, healthcare providers, pharmaceutical companies, and the public. Each stakeholder has a role to play in ensuring the integrity of the drug supply chain.

Key Words: Bacterial meningitis, Counterfeit drugs, Non-counterfeit drugs, Vaccination, Exposed

1. Introduction

The inflammation (swelling) of the membranes that envelop the brain and spinal cord is known as meningitis. [1].Bacterial, viral, or protozoan infections are the causes [2]. Bacterial meningitis is a prevalent illness in children and young adults. Listeria monocytogens, Haemophilus influenza, Group B Streptococcus, Neisseria menengitidis, and Streptococcus pneumonia are some of the bacteria that cause bacterial meningitis [3]. Initially spread by animal to human contact, bacterial meningitis can now spread from person to person through contaminated surfaces, saliva, respiratory secretions, and contaminated air droplets. When an infected individual interacts with others closely or over an extended period of time, the virus can spread quickly. A disease outbreak can be caused by living in cramped quarters, going to sporting or cultural activities, sharing cutlery, coughing, sneezing, and kissing [4]. The symptoms for the bacterial meningitis are fever, intense headache, vomiting, and sensitivity to light and stiff neck which results to convulsion, delirium and even death [2]. Bacterial meningitis is managed through preventive by use of vaccine or curative by use of antibiotic approaches.

Vaccination is an effective way of protecting children from bacterial meningitis. The majority of patients make a full recovery following rapid therapy; yet, some patients continue to have serious health issues after prompt treatment. Loss of limb function, neurological abnormalities, and hearing impairments are some of these problems. However, no amount of treatment can save meningitis-related death if the disease is discovered too late. Approximately 5 to 10% of patients will pass away within 24 to 48 hours of the onset of symptoms, even with early identification [5].

It is clear that studying the epidemiological behavior of meningitis has made mathematical modeling essential. Moreover, mathematical modeling aids in determining illness risk factors and explains why different people do not have the same virus [6]. [7] have created a SIQR model that incorporates persons who are quarantined, potentially lowering the risk of secondary infection and influencing the dynamics of the transmission process. The dynamics of the SVEIR model were created and investigated by [8], its functions include a saturated pneumonia vaccine and a saturated infection incidence force [9] investigated the meningitis and influenza mathematical modeling while under quarantine [10] spent a great deal of work creating a mathematical model for the co-dynamics of the bacterial diseases listeriosis and meningitis [4] created a model called susceptible-vaccinated-carrier-infected-recovered-susceptible to study the dynamics of meningitis. Based on their model, they distinguished between those who recovered and those who did not, with the idea that a high vaccination rate could aid in

keeping the disease under control.

Given the models that have been presented none have analyzed on modeling meningitis taking critical role of healthcare infrastructure, regulatory oversight, and public awareness in ensuring the availability and administration of genuine medications as these may affect the rate of disease mortality and its transmission ensuring there well flow of non-counterfeit drugs in health facilities and ensuring that the public is well informed on the existence of wrong provider of these counterfeit drugs will greatly improve on curbing the spread and mortality rate due to meningitis. In this research, bacterial meningitis with vaccination and non-counterfeit and counterfeit medicine treatment is presented. The model is numerically simulated and subjected to qualitative analysis to help guide policy decisions about disease control.

1.0.1. Research question ?

What is the impact of counterfeit and non-counterfeit drugs during the outbreak of bacterial meningitis. How will it affect the general public.

2. Model formulation

We use a set of ordinary differential equations to create a mathematical model for this one. The dynamics of bacterial meningitis were investigated using mathematical techniques to find threshold parameters for nonlinear ordinary differential equations. To ascertain their impact on the model's outcomes, sensitivity analysis of the model parameters as well as quantitative and qualitative analyses of the model were examined. Our model incorporates the dynamics of bacterial meningitis with vaccination and treatment using both non-counterfeit and counterfeit medications.

We provide a mathematical compartmental model in which the host population is split up into various classes; susceptible S (Healthy individuals who are at risk of a disease), infected with or without symptoms I (people who have contracted the illness and become unwell); Vaccinated V (well individuals immunized against the illness); Exposed E (healthy people who are more subjected to or is in direct contact with a particular risk or harmful condition than others); Treated (Individuals who are subjected to treatment using either counterfeit T_C or non-counterfeit T_n drugs); Recovered R (individuals who have been treated and acquiring temporal immunity or who are free of bacteria).

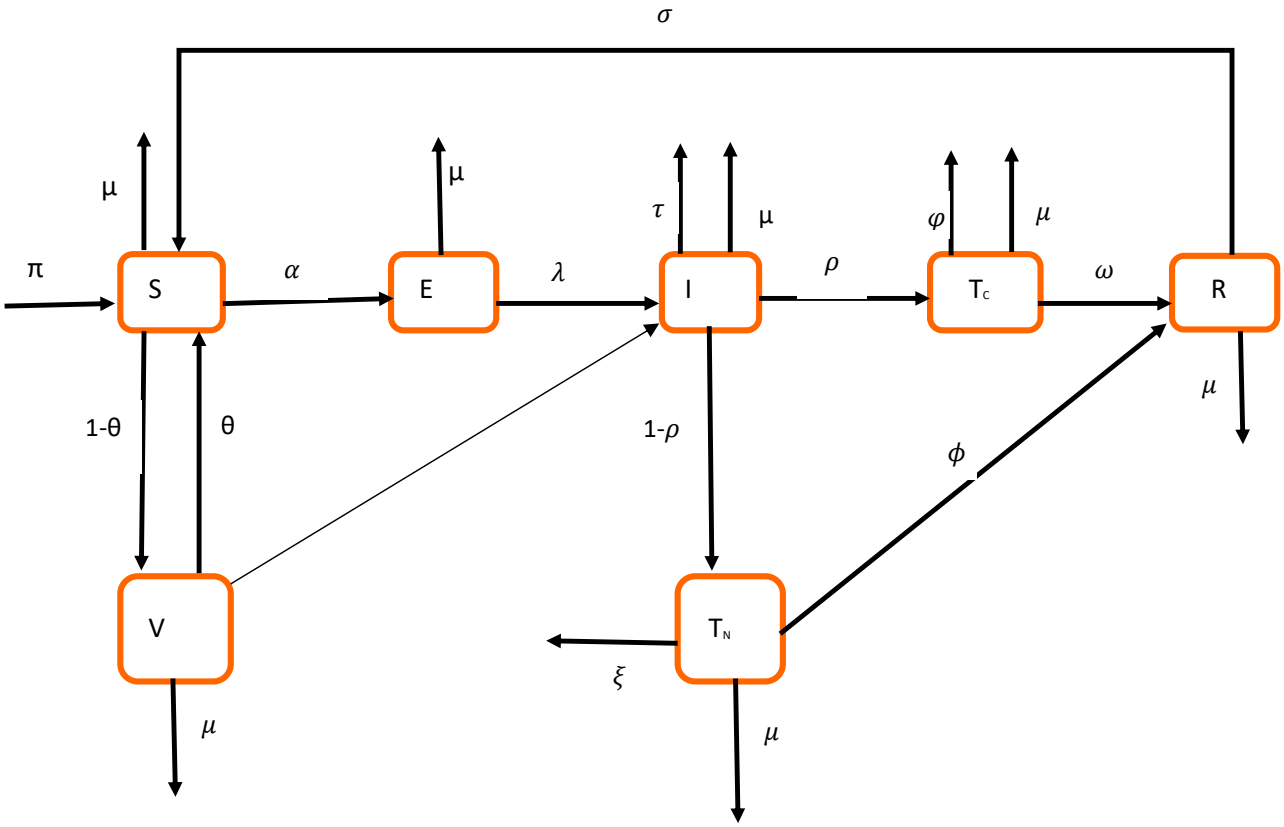
The infectious status is acquired immediately after the infection. As the period of time passed from the moment of the infection to the moment of the recovery we assume that the total population is constant (that is, no births or immigration are considered).

Parameter	definitions
π	rate of recruitment
μ	natural death rate
α	rate at which susceptible individuals get exposed
λ	force of infection
θ	vaccination rate
ϵ	rate at which the vaccinated individuals get exposed
τ	death rate due to infection
φ	death rate at treatment (T_C)
ξ	death rate at treatment (T_n)
ρ	rate at which infected receive either counterfeit or non-counterfeit drugs
ϕ	recovery rate for (T_n)
ω	recovery rate for (T_C)
σ	rate at which recovered gain temporal immunity and return to susceptible
β	infection rate
S	susceptible individuals
V	vaccinated individuals
E	exposed individuals
I	infected individuals
T_C	treated under counterfeit drugs
T_n	treatment under non-counterfeit drugs
R	recovered individuals

Table 1. *Parameter Values for the Model*

2.1. Model Flow Chart

Figure 1. *Flow chart for bacterial meningitis incorporating counterfeit and non-counterfeit drugs.*



2.2. Model equations

$$\frac{dS}{dt} = \pi + \sigma R + (1-\theta)V - \theta S - \alpha S - \mu S \tag{1}$$
$$\frac{dV}{dt} = \theta S - (1-\theta)V - \epsilon V - \mu V \tag{2}$$
$$\frac{dE}{dt} = \alpha S + \epsilon V - \lambda E - \mu E \tag{3}$$
$$\frac{dI}{dt} = \lambda E - \tau I - \rho I - (1-\rho)I - \mu I \tag{4}$$
$$\frac{dT_C}{dt} = \rho I - \phi T_C - \omega T_C - \mu T_C \tag{5}$$
$$\frac{dT_n}{dt} = (1-\rho)I - \xi T_n - \phi T_n - \mu T_n \tag{6}$$
$$\frac{dR}{dt} = \omega T_C + \phi T_n - \sigma R - \mu R \tag{7}$$

The force of infection is given by;

$$\lambda = \frac{\beta(I + \eta_1 T_C + \eta_2 T_n)}{N}$$

where λ is the force of infection, β is the infection rate and N=S+V+E+I+$T_C + T_N$+R

3. Model analysis

3.1. Bacterial meningitis without treatment and recovery

The model with meningitis was obtained by setting $T_C = T_n = R = 0$ and obtain (8-11) . In this case we consider individuals with meningitis and those the vaccinated and setting those at treatment (T_C, T_n) and recovered to zero.

$$\frac{dS}{dt} = \pi + \sigma R + (1-\theta)V - \theta S - \alpha S - \mu S \tag{8}$$
$$\frac{dV}{dt} = \theta S - (1-\theta)V - \epsilon V - \mu V \tag{9}$$
$$\frac{dE}{dt} = \alpha S + \epsilon V - \lambda E - \mu E \tag{10}$$
$$\frac{dI}{dt} = \lambda E - \tau I - \rho I - (1-\rho)I - \mu I \tag{11}$$

equation (8-11) can be considered in Ω. The region Ω=(S,V,E,I) $\in \mathbb{R}^4_+ : N \leq \frac{\pi}{\mu}$, is positively invariant. In this region, the results for system (8-11)'s existence, uniqueness, and continuation hold true, and all of system solutions that started in Ω remain in Ω for all time $t > 0$, supporting the model figure 1 is mathematically well posed and its dynamics can be considered in Ω.

3.2. Positivity of the system

We prove positivity by stating and proving the theorem below.
Theorem 3.1. For time $t > 0$, all of the system of equations (1-7) solutions with positive initial data remained positive.

Proof. We take the system of differential equations (1-7) and it follows directly from the first equation that;

$$\frac{dS}{dt} = \pi + \sigma R + (1-\theta)V - \theta S - \alpha S - \mu S \tag{12}$$

considering the negative term only;

$$\frac{dS}{dt} = -(\theta + \alpha + \mu)S \tag{13}$$

by separation of variables and integration, we have;

$$\int \frac{dS}{S} = \int -(\theta + \alpha + \mu)dt \tag{14}$$

$$lnS(t) = \int_0^t -(\theta + \alpha + \mu)ds \tag{15}$$

$$S \geq e^{\int_0^t -(\theta+\alpha+\mu)+C} \tag{16}$$

$$S(t) \geq S(0) \int_0^t -(\theta + \alpha + \mu)dt > 0. \tag{17}$$

$$\tag{18}$$

is non-negative function of t, thus S(t) stays positive. Similarly, by integration and applying the initial conditions, the positivity of V (t), E (t), T_C (t), T_n (t) and R (t) are proved along the same way as S (t) accordingly, from the system of equations (1-7), it can be shown that,

$$V(t) \geq V(0) \int_0^t -[(1-\theta) + \epsilon + \mu]V > 0. \tag{19}$$

$$E(t) \geq E(0) \int_0^t -(\lambda + \mu)E > 0. \tag{20}$$

$$I(t) \geq I(0) \int_0^t -(\tau + \rho + (1-\rho) + \mu)I > 0. \tag{21}$$

$$T_C(t) \geq T_C(0) \int_0^t -(\Phi + \omega + \mu)T_C > 0. \tag{22}$$

$$T_n(t) \geq T_n(0) \int_0^t -(\xi + \Phi + \mu)T_n > 0. \tag{23}$$

$$R(t) \geq R(0) \int_0^t -(\sigma + \mu)R > 0. \tag{24}$$

$$\tag{25}$$

3.3. Boundedness of the system

The boundedness of the system was verified by stating and proving the theorem below

Theorem 3.2. Let the feasible region be defined by;
$\Omega = S(t), V(t), E(t), I(t), T_C, T_n$, R(t) with the initial conditions $S(0) \geq 0, V(0) \geq 0, E(t) \geq 0, I(0) \geq 0, T_C \geq 0, T_n \geq 0, R(t) \geq 0$. The region Ω is positively invariant and attracting with respect to the system of equations (1-7) for all $t > 0$

Proof.

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dT_C}{dt} + \frac{dT_n}{dt} + \frac{dR}{dt}. \tag{26}$$

Recovery and differential equation solution via variable separation do not exist in the absence of illness, integrating the differential equation to obtain;

$$\frac{\pi}{\mu} - N \geq \frac{\pi - \mu N_0}{\mu} e^{-\mu t}, \tag{27}$$

As $t \to \infty$, the number of people in $N \to \frac{\pi}{\mu}$ and this suggests that;
$0 \leq N < \frac{\pi}{\mu}$ and $N \leq \frac{\pi}{\mu}$, therefore $\Omega = \{S(t), V(t), E(t), I(t), T_C(t), T_n(t), R(t) \in R_+^7; N \leq \frac{\pi}{\mu}$.

This demonstrates that the solutions are bounded inside Ω. It means that for all $t > 0$, all of our system (1-7) solutions will start in Ω and remain in Ω. Therefore, the proof can be concluded by taking into account the way our system behaves in Ω.

3.4. Disease free equilibrium

The disease free equilibrium point of the system of equations (1-7) is obtained by setting all the vaccinated class,exposed class, the treated class , the infectious class and recovered class to zero.

$$\pi - \mu S^0 = 0, \tag{28}$$

$$S^0 = \frac{\pi}{\mu.} \tag{29}$$

Therefore, the disease free equilibrium point for our system is given by,
$\zeta^0 = (S^0, V^0, E^0, I^0, T_C^0, T_n^0, R^0) = (\frac{\pi}{\mu}, 0, 0, 0, 0, 0, 0)$ as seen in figure 2.

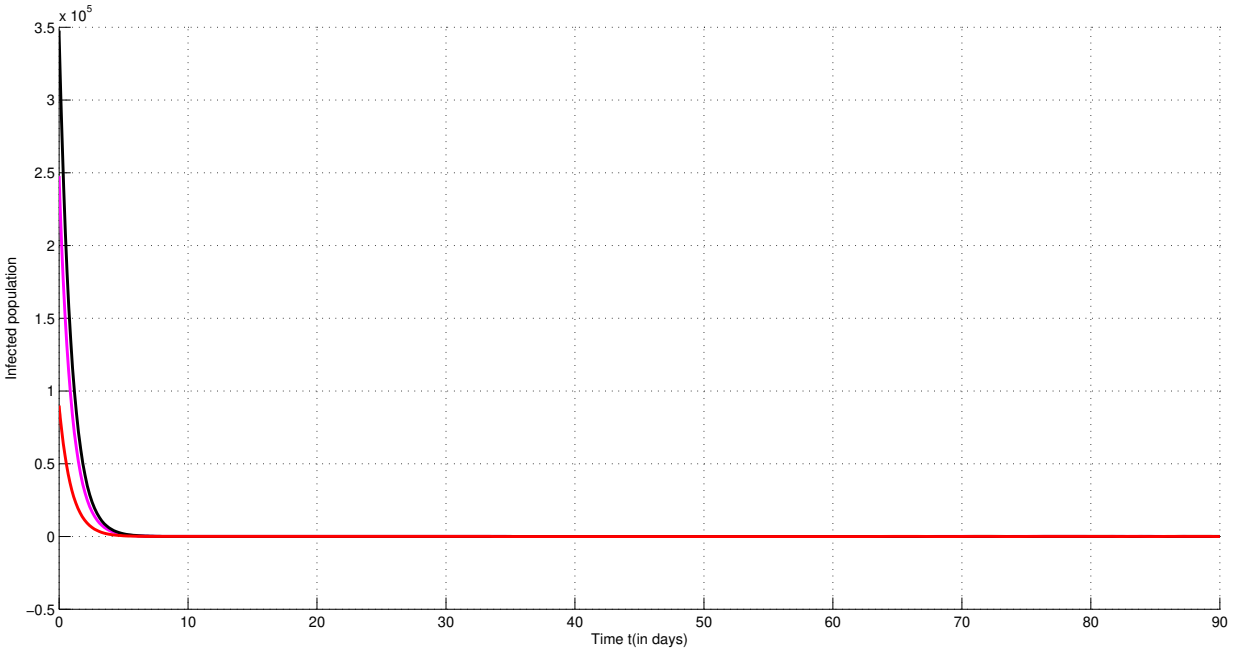


Figure 2. *Over time, the population of infectious cholera cases in the absence of bacterial meningitis disease*

3.5. Effective Reproduction number

Using the next generation matrix method [11, 12], the effective reproduction number of the bacterial meningitis model is obtained as follows;

$$f = \begin{pmatrix} \lambda E \\ 0 \\ 0 \\ 0 \end{pmatrix}, \tag{30}$$

(31)

$$v = \begin{pmatrix} (\tau + \rho + (1-\rho) + \mu)I \\ -\rho I + (\phi + \omega + \mu)T_C \\ -(1-\rho)I + (\xi + \phi + \mu)T_n \end{pmatrix} \tag{32}$$

The matrices F and V are obtained by determining the jacobian matrices of F and V to obtain the eigenvalues at D.F.E FV^{-1} we obtain;
$Y_1 = 0$
$Y_2 = 0$
$Y_3 = \frac{\zeta^0 \beta(1+\frac{\rho\eta_1}{\mu+\phi+\omega} - \frac{-1+\rho\eta_2}{\mu+\xi+\phi)}}{1+\tau+\mu}$

Theorem 3.3. If $R_0 < 1$ the D.F.E of bacterial meningitis was locally asymptotically stable and unstable otherwise.

Proof. Calculating the following Jacobian matrix for our equations (1-7)

$$
\begin{aligned}
f_1 =& \pi + \sigma R + (1-\theta)V - \theta S - \alpha S - \mu S,\\
f_2 =& \theta S - (1-\theta)V - \epsilon V - \mu V,\\
f_3 =& \alpha S + \epsilon V - \lambda E - \mu E,\\
f_4 =& \lambda E - \tau I - \rho I - (1-\rho) I - \mu I,\\
f_5 =& \rho I - \phi T_C - \omega T_C - \mu T_C,\\
f_6 =& (1-\rho) I - \xi T_n - \phi T_n - \mu T_n,\\
f_7 =& \omega T_C + \phi T_n - \sigma R - \mu R.
\end{aligned}
$$

The force of infection is given by $\lambda = \frac{\beta(I+\eta_1 T_C + \eta_2 T_n)}{N}$

Determining the Jacobian matrix at D.F.E we obtain;

$$
\begin{bmatrix}
-\theta-\alpha-\mu & 1-\theta & 0 & 0 & 0 & 0 & \sigma \\
\theta & -1-\epsilon+\theta-\mu & 0 & 0 & 0 & 0 & 0 \\
\alpha & \epsilon & -\mu & -\beta & -\beta\eta_1 & -\beta\eta_2 & 0 \\
0 & 0 & 0 & \beta-\tau-1-\mu & \beta\eta_1 & \beta\eta_2 & 0 \\
0 & 0 & 0 & \rho & -\phi-\omega-\mu & 0 & 0 \\
0 & 0 & 0 & 1-\rho & 0 & -\xi-\phi-\mu & 0 \\
0 & 0 & 0 & 0 & \omega & \phi & -\sigma-\mu
\end{bmatrix}
$$

the sidetermine signs of the eigen values using Routh-Hurwitz criterion. The characteristic function $|A - X_i I| = 0$ with i=1,2,3,4,5,6,7.
by Routh-Harwitz criterion for determining the negatives real signs of the eigen values of the cubic polynomial are;

$\lambda^7 + a_1\lambda^6 + a_2\lambda_5 + a_3\lambda_4 + a_4\lambda_3 + a_5\lambda_2 + a_6\lambda_1 + a_7$ with conditions:

$a_1 > 0, a_1a_2a_3a_4a_5a_6 > a_7 > 0$

from the characteristic polynomial the values of $a_1, a_2, a_3, a_4, a_5, a_6$ and a_7 expressed in terms of R_0^* are;
$a_1 = -1$
$a_2 = -2-\alpha+\frac{1+\mu+\tau}{S(1+\frac{\rho\eta_1}{\mu+\phi+\omega}-\frac{(-1+\rho)\eta_2}{\mu+\xi+\phi)}} - \epsilon - 7\mu - \xi - \sigma - \tau - 2\phi - \omega$ $a_3 = -1-2\alpha+\frac{1+\mu+\tau}{S(1+\frac{\rho\eta_1}{\mu+\phi+\omega}-\frac{(-1+\rho)\eta_2}{\mu+\xi+\phi)}} + \alpha\frac{1+\mu+\tau}{S(1+\frac{\rho\eta_1}{\mu+\phi+\omega}-\frac{(-1+\rho)\eta_2}{\mu+\xi+\phi)}} - \epsilon - \alpha\epsilon + \frac{1+\mu+\tau}{S(1+\frac{\rho\eta_1}{\mu+\phi+\omega}-\frac{(-1+\rho)\eta_2}{\mu+\xi+\phi)}}\epsilon + \alpha\theta - \epsilon\theta - 12\mu - 6\alpha\mu + 6\frac{1+\mu+\tau}{S(1+\frac{\rho\eta_1}{\mu+\phi+\omega}-\frac{(-1+\rho)\eta_2}{\mu+\xi+\phi)}}\mu - 6\epsilon\mu - 21\mu^2 - 2\xi - \alpha\xi + \frac{1+\mu+\tau}{S(1+\frac{\rho\eta_1}{\mu+\phi+\omega}-\frac{(-1+\rho)\eta_2}{\mu+\xi+\phi)}}\xi - \epsilon\xi - 6\mu\xi - 2\rho - \alpha\rho + \frac{1+\mu+\tau}{S(1+\frac{\rho\eta_1}{\mu+\phi+\omega}-\frac{(-1+\rho)\eta_2}{\mu+\xi+\phi)}}\rho - \epsilon\rho - 6\mu\rho - \xi\rho - \tau - \alpha\tau - \epsilon\tau - 6\mu\tau - \xi\tau - \rho\tau - 4\phi - 2\alpha\phi + 2\frac{1+\mu+\tau}{S(1+\frac{\rho\eta_1}{\mu+\phi+\omega}-\frac{(-1+\rho)\eta_2}{\mu+\xi+\phi)}}\phi - 2\epsilon\phi - 12\mu\phi - \xi\phi - 2\rho\phi - 2\tau\phi - \phi^2 - 2\omega - \alpha\omega + \frac{1+\mu+\tau}{S(1+\frac{\rho\eta_1}{\mu+\phi+\omega}-\frac{(-1+\rho)\eta_2}{\mu+\xi+\phi)}}\omega - \epsilon\omega - 6\mu\omega - \xi\omega - \rho\omega - \tau\omega - \phi\omega + \frac{1+\mu+\tau}{S(1+\frac{\rho\eta_1}{\mu+\phi+\omega}-\frac{(-1+\rho)\eta_2}{\mu+\xi+\phi)}}\rho\eta_1 + \frac{1+\mu+\tau}{S(1+\frac{\rho\eta_1}{\mu+\phi+\omega}-\frac{(-1+\rho)\eta_2}{\mu+\xi+\phi)}}\eta_2 - \frac{1+\mu+\tau}{S(1+\frac{\rho\eta_1}{\mu+\phi+\omega}-\frac{(-1+\rho)\eta_2}{\mu+\xi+\phi)}}\rho\eta_2$. The values for $a_3, a_4, a_5 a_6$ and a_7 are given in the supplementary materials provided. This made the evidence complete. This outcome indicates believed bacterial meningitis would vanish at any time provided $R < 1$, if the original prerequisites of the disease dynamics start in the vicinity of D.F.E.

3.6. Global stability of the D.F.E

The system of equation (1-7) was proved to lie in the positive region using theorems in section (40-41). The global stability of disease free equilibrium was investigated using Metzler matrix stability method proposed by [13].

$$\frac{dX}{dt} = F(X, Z) \tag{33}$$

$$\frac{dZ}{dt} = G(X, Z), G(X, 0) = 0 \tag{34}$$

where:$X = (S, V, E, R) \in \mathbb{R}^4_+$ denotes non-infectious bacterial meningitis compartment and $Z = (I, T_C, T_n) \in \mathbb{R}^3_+$ denotes the infectious bacterial meningitis compartment $\zeta_0 = (X^*, 0)$ represents the disease free equilibrium of the system if this point satisfies following condition:
i.$\frac{dX}{dt} = F(X, 0)$, where X^* is globally asymptotically stable.
ii.$\frac{dZ}{dt} = D_Z G(X, 0)Z - G(X, Z) \geq 0$ for all $(X, Z) \in \Omega$, then we can conclude that ζ_0 is locally asymptotically stable if the following theorems holds:

Theorem 3.4. The equilibrium point $\zeta_0(X^*, 0)$ of the system (1-7) is globally asymptotically stable if $\mathbb{R}^*_0 \leq 1$ and the conditions (i) and (ii) are satisfied, otherwise unstable.

Proof. Let X=(S,V,E,R) and Z=(I, T_C, T_n), be the new variables and the sub-systems of the system model (1-7). From equation (1-7) two vector functions G(X, Z) and F(X, Z) are obtained, we consider reduced systems,

F(X, 0)= $\begin{pmatrix} \pi - \mu S, \\ 0, \\ 0, \\ 0. \end{pmatrix}$ It is noted that this is an asymptomatic dynamics system independent of the initial condition in Ω; therefore, the convergence of the solutions of the reduced system (1-7) is global in Ω by computing:
$\hat{G}(XZ) = D_Z G(X^*, 0)Z - G(XZ)$
$\hat{G}(X, Z) \geq 0$. Now let A=$D_Z G(X^*.0)$, which is the jacobian of $\hat{G}(X, Z)$ taken in (I, T_C, T_n) and evaluated at $(X^*, 0)$, such that the matrix A is given by;

A= $\begin{bmatrix} \beta - \tau - 1 - \mu & \beta\eta_1 & \beta\eta_2 \\ \rho & -\phi - \omega - \mu & 0 \\ 1 - \rho & 0 & -\xi - \phi - \mu \end{bmatrix}$

AZ= $\begin{bmatrix} (\beta - \tau - 1 - \mu)I & \beta\eta_1 T_C & \beta\eta_2 T_n \\ \rho I & (-\phi - \omega - \mu)I_C & \\ (1 - \rho)I & (-\xi - \phi - \mu)I_n & \end{bmatrix}$

$\hat{G}(X, Z) = \begin{bmatrix} (1 - \frac{S}{N})\beta(I + T_C\eta_1 + \eta_2 T_n) \\ 0 \\ 0 \\ 0 \end{bmatrix}$. Therefore if $\hat{G}(X, Z) \geq 0$, then the disease free equilibrium, ζ_0 is globally asymptotically stable otherwise its unstable. Since $S \leq N$, $\frac{S}{N} \leq 1$, thus $D(X, Z) \geq 0$ for all $X, Z \in \mathbb{R}^3_+$, then, the D.F.E will be globally asymptotically stable. Its clear that matrix A is an M-matrix since the off-diagonal elements of A are non-negative. Therefore, this proves that G.D.F.E is globally asymptotically stable. This complete the proof. This result show that cholera would die out whenever$R^*_0 < 1$ irrespective of the initial conditions.

3.7. Bifurcation

One can investigate the bifurcation analysis by employing center manifold theorems [14]. For the sake of straightforwardness, the variables are changed first. Let S=y_1, V= y_2, $E = y_3$, $I = y_4$, $T_C = y_5$, $T_n = y_6$, $R = y_7$. Further, by using vector notation, y=$(y_1, y_2, y_3, y_4, y_5, y_6, y_7)$, the meningitis model (42-48) is composed in the format $\frac{dy}{dt} = F(y)$, with F=$(p_1, p_2, p_3, p_4, p_5, p_6)$ as follows:

$$\dot{y_1} = p_1 = \pi + \sigma y_7 + (1-\theta)y_2 - \theta y_1 - \alpha y_1 - \mu y_1, \tag{35}$$
$$\dot{y_2} = p_2 = \theta y_1 - (1-\theta)y_2 - \epsilon y_2 - \mu y_2, \tag{36}$$
$$\dot{y_3} = p_3 = \alpha y_1 + \epsilon y_2 - \frac{\beta(y_4 + \eta_1 y_5 + \eta_2 y_6)}{N} y_3 - \mu y_3, \tag{37}$$
$$\dot{y_4} = p_4 = \frac{\beta(y_4 + \eta_1 y_5 + \eta_2 y_6)}{N} y_3 - \tau y_4 - \rho y_4 - (1-\rho)y_4 - \mu y_4, \tag{38}$$
$$\dot{y_5} = p_5 = \rho y_4 - \phi y_5 - \omega y_5 - \mu y_5, \tag{39}$$
$$\dot{y_6} = p_6 = (1-\rho)y_4 - \xi y_6 - \phi y_6 - \mu y_6, \tag{40}$$
$$\dot{y_7} = p_7 = \omega y_5 + \phi y_6 - \sigma y_7 - \mu y_7. \tag{41}$$

where, $\lambda = \beta(I + \eta_1 T_C + \eta_2 T_n)$ The process comprises assessing the system (42-48)'s Jacobian at D.F.E $\zeta_*^0(S_*^0, V_*^0, E_*^0, {I_*}^0, T_{C*}^0, T_{n*}^0, R_*^0) = (\frac{\theta\pi}{\mu}, 0, 0, 0, 0, 0)$, indicated by $J(\zeta_*^0)$, we obtain

$$JE_*^0 = \begin{bmatrix} -\theta-\alpha-\mu & 1-\theta & 0 & 0 & 0 & 0 & \sigma \\ \theta & -1-\epsilon+\theta-\mu & 0 & 0 & 0 & 0 & 0 \\ \alpha & \epsilon & -\mu & -\beta & -\beta\eta_1 & -\beta\eta_2 & 0 \\ 0 & 0 & 0 & \beta-\tau-1-\mu & \beta\eta_1 & \beta\eta_2 & 0 \\ 0 & 0 & 0 & \rho & -\phi-\omega-\mu & 0 & 0 \\ 0 & 0 & 0 & 1-\rho & 0 & -\xi-\phi-\mu & 0 \\ 0 & 0 & 0 & 0 & \omega & \phi & -\sigma-\mu \end{bmatrix} \tag{42}$$

(43)

we consider the case where $R_0^* = 1$. If $\beta = \beta^*$ is selected as the bifurcation parameter, then β^* can be found by solving for $R_C^* = 1$:
$\beta^* = \frac{1+\mu+\tau}{S(1+\frac{\rho\eta_1}{\mu+\phi+\omega} - \frac{(-1+\rho)\eta_2}{\mu+\xi+\phi)}}$ The theorem by [15], investigated the following broad system of β^*-parameterized ordinary differential equations

$\frac{dy}{dx} = f(y, \beta^*), f : R^n \times R \longrightarrow R^n$ and $f \in C^2(R^2)$ in which the system's equilibrium point is 0(which is, $f(y, \beta^*) \equiv 0$ and

1) $A = D_Y f(0,0) = (\frac{\delta P_i}{\delta y_j(0,0)})$, is the system's linearization matrix for the region surrounding its equilibrium with 0 β^* evaluated at 0;

2) The only simple eigenvalue of A is zero, while all other eigenvalues of A contain negative real components.

3) The zero eigenvalue is represented by the right eigenvector u and the left eigenvector v of matrix A. Suppose that p_k is the kth component of p.

$$a = \sum_{k,ij=1}^{n} v_k u_i u_j \frac{\delta^2 p_k}{\delta y_i \delta \beta^*}(0,0)$$
$$b = \sum_{k,ij=1}^{n} v_k u_i \frac{\delta^2 p_k}{\delta y_i \delta \beta^*}(0,0) \tag{44}$$

then the local dynamics of the system around the equilibrium point (0,0) is totally determined by the signs of a and b. Particularly when:

i. $a > 0$ and $b > 0$, when $\beta^* < 0$ with $|\beta^*| << 1, (0,0)$, is locally asymptotically stable and there exists a positive unstable equilibrium; $0 < \beta^* << 1, (0,0)$ is unstable and there exists a negative and locally asymptotically stable equilibrium.
ii. $a < 0$ and $b < 0$ when $\beta^* < 0$ with $|\beta| << 1, (0,0)$, is unstable; when $0 < \beta << 1, (0,0)$ is asymptotically stable and there exists a positive unstable equilibrium.
iii. $a < 0$ and $b > 0$, when $\beta^* < 0$ is unstable, and there exists a negative and locally asymptotically stable equilibrium; when $0 < \beta << 1, (0,0)$ is stable and there exists a positive unstable equilibrium.

iv $a < 0$ and $b > 0$, when β^* changes from negative to positive, (0,0) changes its stability from stable to unstable. Correspondingly a negative equilibrium becomes positive and locally asymptotically stable.

$a < 0$ and $b > 0$, then a backward bifurcation occurs at $\beta^* = 0$[16]. Jacobian $[J(E^0_*)]$ at $\beta = \beta^*$ (denoted by J^*_β) has a right eigen vector given by u= $[u_1, u_2, u_3, u_4, u_5, u_6, u_7]^T$, let $u_3 = u_3 > 0$, then

$$u_1 = \frac{(1-\theta)v_2 + \rho v_7}{\theta + \alpha + \mu} \tag{45}$$

$$u_2 = \frac{-\theta v_1}{-1 - \epsilon + \theta - \mu} \tag{46}$$

$$u_3 = \frac{\alpha v_1 + \epsilon v_2 - \beta v_4 - \beta\eta_1 v_5 - \beta\eta_2 v_6}{\mu} \tag{47}$$

$$u_4 = \frac{-\beta\eta_1 v_5 - \beta\eta_2 v_6}{\beta - \tau - 1 - \mu} \tag{48}$$

$$u_5 = \frac{\rho v_4}{\phi + \omega + \mu} \tag{49}$$

$$u_6 = \frac{(1-\rho)v_4}{\xi + \phi + \mu} \tag{50}$$

$$u_7 = \frac{\omega v_5 + \phi v_6}{\rho + \mu} \tag{51}$$

$$\tag{52}$$

Further, J^*_β has a left eigenvectors v=$[v_1, v_2, v_3, v_4, v_5, v_6, v_7]$, let $v_1 = v_1 > 0$

$$v_1 = \frac{(1-\theta)v_2 + \rho v_7}{\theta + \alpha + \mu} \tag{53}$$

$$v_2 = \frac{-\theta v_1}{-1 - \epsilon + \theta - \mu} \tag{54}$$

$$v_3 = \frac{\alpha v_1 + \epsilon v_2 - \beta v_4 - \beta\eta_1 v_5 - \beta\eta_2 v_6}{\mu} \tag{55}$$

$$v_4 = \frac{-\beta\eta_1 v_5 - \beta\eta_2 v_6}{\beta - \tau - 1 - \mu} \tag{56}$$

$$v_5 = \frac{\rho v_4}{\phi + \omega + \mu} \tag{57}$$

$$v_6 = \frac{(1-\rho)v_4}{\xi + \phi + \mu} \tag{58}$$

$$v_7 = \frac{\omega v_5 + \phi v_6}{\rho + \mu} \tag{59}$$

$$\tag{60}$$

$$v_1 = \frac{\theta u_2 + \alpha u_3}{\theta + \alpha + \mu} \tag{61}$$

$$v_2 = \frac{-(1-\theta)u_1 - \xi u_3}{-1 - \xi + \theta + \mu} \tag{62}$$

$$v_3 = 0 \tag{63}$$

$$v_4 = \frac{-\rho u_5 - (1-\rho)u_6}{\beta - \tau - 1 - \mu} \tag{64}$$

$$v_5 = \frac{\beta\eta_1 u_4 + \omega u_7}{\theta + \omega + \mu} \tag{65}$$

$$v_6 = \frac{\beta\eta_2 u_4 + \phi u_7}{\xi + \phi + \mu} \tag{66}$$

$$v_7 = \frac{\sigma u_1}{\sigma + \mu} \tag{67}$$

$$\frac{dp_3}{dy_3} = -\mu - \beta(\frac{y_4 + \eta_1 y_5 + \eta_2 y_6}{N}) \tag{68}$$

$$\frac{d^2p_3}{dy_3 dy_4} = -\frac{\beta}{N} \tag{69}$$

$$\frac{d^2p_3}{dy_3 dy_5} = -\beta\frac{\eta_1}{N} \tag{70}$$

$$\frac{d^2p_3}{dy_3 dy_6} = -\beta\frac{\eta_2}{N} \tag{71}$$

$$\frac{dp_4}{dy_3} = \beta(\frac{y_4 + \eta_1 y_5 + \eta_2 y_6}{N}) \tag{72}$$

$$\frac{d^2p_4}{dy_3 dy_4} = \frac{\beta}{N} \tag{73}$$

$$\frac{d^2p_4}{dy_3 dy_6} = \frac{\beta\eta_3}{N} \tag{74}$$

$$a = v_3[-2u_3u_4\frac{\beta}{N} - 2u_3u_5\frac{\beta\eta_1}{N} - 2u_3u_6\frac{\beta\eta_2}{N}] + v_4[2u_3u_4\frac{\beta}{N} + 2u_3u_5\frac{\beta\eta_1}{N} + 2u_3u_6\frac{\beta\eta_2}{N}] \tag{75}$$

$$\frac{dp_3}{dy_3} = -\mu - \beta(\frac{y_4 + \eta_1 y_5 + \eta_2 y_6}{N}) \tag{76}$$

$$\frac{d^2p_3}{dy_3 d\beta^*} = -(\frac{y_4 + \eta_1 y_5 + \eta_2 y_6}{N}) \tag{77}$$

$$at D.F.E(0,0), y_4 = y_5 = y_6 = 0 \tag{78}$$

$$\frac{dp_3}{dy_4} = -\frac{\beta y_3}{N} \tag{79}$$

$$\frac{d^2p_3}{dy_4 d\beta^*} = -\frac{y_3}{N} \tag{80}$$

$$\frac{dp_3}{dy_5} = -\frac{\eta_1\beta y_3}{N} \tag{81}$$

$$\frac{d^2p_3}{dy_5\beta^*} = -\frac{\eta_1 y_3}{N} \tag{82}$$

$$\frac{dp_3}{dy_6} = -\frac{\beta y_3\eta_2}{N} \tag{83}$$

$$\frac{d^2p_3}{dy_6 d\beta^*} = -\frac{\eta_2 y_3}{N} \tag{84}$$

$$\frac{dp_4}{dy_3} = \frac{\beta(y_4 + \eta_1 y_5 + \eta_2 y_6)}{N} \tag{85}$$

$$\frac{d^2p_4}{dy_3 d\beta^*} = \frac{(y_4 + \eta_1 y_5 + \eta_2 y_6)}{N} \tag{86}$$

$$at D.F.E(0,0), y_4 = y_5 = y_6 = 0 \tag{87}$$

$$\frac{dp_4}{dy_4} = \frac{\beta}{N} - \tau - 1 - \mu \tag{88}$$

$$\frac{d^2p_4}{dy_4 d\beta^*} = \frac{1}{N} \tag{89}$$

$$\frac{dp_4}{dy_5} = \frac{\beta\eta_1}{N} \tag{90}$$

$$\frac{d^2p_4}{dy_5 d\beta^*} = \frac{\eta_1}{N} \tag{91}$$

$$\frac{dp_4}{dy_6} = \frac{\beta\eta_2}{N} \tag{92}$$

$$\frac{d^2p_4}{dy_6 d\beta^*} = \frac{\eta_2}{N} \tag{93}$$

$$b = v_3[-u_4\frac{y_3}{N} - u_5\frac{\eta_1 y_3}{N} - u_6\frac{\eta_2}{N}] + v_4[\frac{u_4}{N} + \frac{\eta_2 u_5}{N} + \frac{u_6\eta_2}{N}] \tag{94}$$

but $v_1 > 0$, $b > 0$. Hence $a < 0$ and $b > 0$, when $\beta^* < 0$ with $|\beta^*| <<, (0,0)$ is unstable and there exists a negative and locally asymptotically stable equilibrium; when $0 < \beta^* << 1, (0,0)$ is stable and there exists a positive unstable equilibrium. The direction of the bifurcation of system (1-7) at $R_0 > 1$ is forward. Since the bifurcation parameter changes from negative to positive and the disease free equilibrium changes its stability from negative to positive. Therefore, When $R_0 < 1$, there is no backward bifurcation, demonstrating that bacterial meningitis can be wiped out.

3.8. Existence of endemic equilibrium point

A point at which the infection cannot be completely eliminated but endures within the population is known as the point of endemic equilibrium. The vulnerable class, the transmissible class, and the therapy class must not equal zero at equilibrium state for the infection to continue in the population. Thus, the condition required and sufficient for the existence of an endemic equilibrium point is determined in this section. The contagious classes at EEP;

$$\lambda E - \Omega_1 I, \rho I - \Omega_2 T_C, (1-\rho) I - \Omega_3 T_n \tag{95}$$

our new equations reduces on solving for T_n. We obtain;

$$T_n = \frac{I(-1+\rho)}{\Omega_3} \tag{96}$$

Solving for I we obtain;

$$I = \frac{T_C \Omega_2}{\rho} \tag{97}$$

solving for T_n and replacing in equation (95-96) we obtain;

$$\frac{-E\beta(-1+\rho)\eta_2\Omega_2 + (E\beta\rho\eta_1 + (E\beta - \Omega_1)\Omega_2)\Omega_3}{\rho\Omega_3} \tag{98}$$

Which correspond back to the fundamental reproduction number, which demonstrate the disease's persistence; therefore, the existence of the endemic equilibrium point (E.E.P) completes the proof as seen in figure 3.

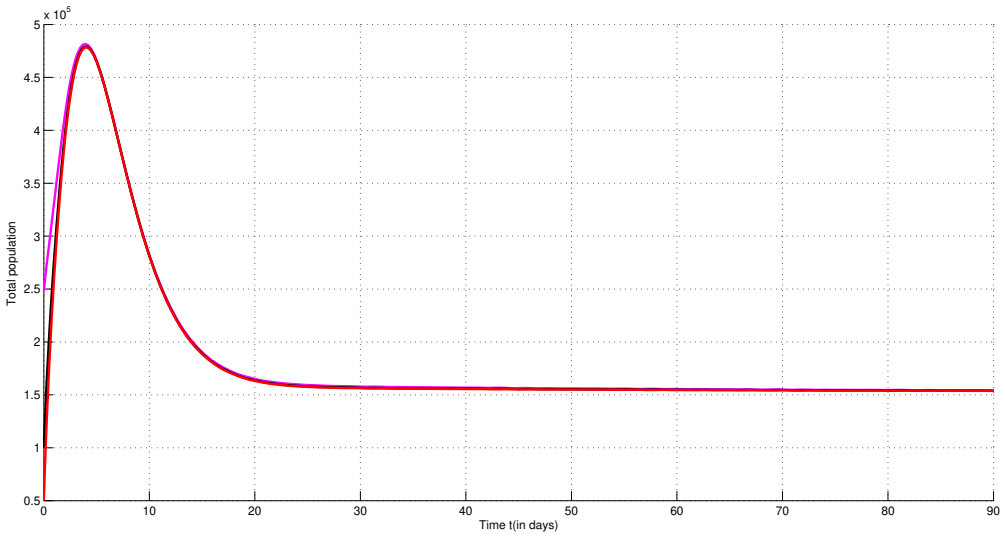


Figure 3. *With time and the existence of meningitis illness, the population of infectious bacterial meningitis*

3.9. Global stability of EEP

The requirement for the stability of the endemic equilibrium point was ascertained using a Lyapunov stability criterion. We found requirements that must be met in order to guarantee global asymptotic stability for the derivative of the Lyapunov function to be negative definite. For the system of reduced equations;

3.10. Model equations

$$\frac{dS}{dt} =\pi + \sigma R + (1-\theta)V - \theta S - \alpha S - \mu S, \tag{99}$$
$$\frac{dV}{dt} =\theta S - (1-\theta)V - \epsilon V - \mu V, \tag{100}$$
$$\frac{dE}{dt} =\alpha S + \epsilon V - \lambda E - \mu E, \tag{101}$$
$$\frac{dI}{dt} =\lambda E - \Omega_1 I, \tag{102}$$
$$\frac{dT_C}{dt} =\rho I - \Omega_2 T_C, \tag{103}$$
$$\frac{dT_n}{dt} =(1-\rho) I - \Omega_3 T_n, \tag{104}$$
$$\frac{dR}{dt} =\omega T_C + \phi T_n - \sigma R - \mu R. \tag{105}$$

The reproducibility number of control (R_o^*), the intensity of the illness (λ^*), D.F.E $\zeta^0 = (S^0, I^0, T^0, R^0, S_0^0, I_0^0, T_0^0, R_0^0) = (\frac{\pi}{\mu}, 0, 0, 0, \frac{(1-\theta)\pi}{N}, 0, 0, 0)$ and E.E.P $E^* = (S^*, I^*, T^*, R^*, S_0^*, I_0^*, T_0^*, R_0^*)$ of system (106-112) are provided by the Lyapunov function that follows
$K(S, V, E, I, T_C, T_n, R) = S - S^* - S^* Ln\frac{S}{S^*} + y_1(V - V^* - V^* Ln\frac{V}{V^*}) + y_2(E - E^* - E^* Ln\frac{E}{E^*}) + y_3(I - I^* - I^* Ln\frac{I}{I^*}) + T_C - T_C^* - T_C^* Ln\frac{T_C}{T_C^*} + y_5(T_n - T_n^* - T_n^* Ln\frac{T_n}{T_n^*}) + y_6(R - R^* - R^* Ln\frac{R}{R^*})$,
where y_1, y_2, y_3, y_5, y_6 are to be determined as positive constants. The function of Lyapunov $K(S, V, E, I, T_C, T_n, R)$ satisfies the conditions $K(S^*, V^*, E^*, I^*, T_C^*, T_n^*, R_0^*) = 0$ and $K(S, V, E, I, T_C, T_n, R) > 0$, it is therefore positive definite for;

$$\frac{dk(S, V, E, I, T_C, T_n, R)}{dt}. \tag{106}$$

It must meet in order to be negative categorical,

$$\frac{dk(S^*, V^*, I^*, T_C^*, T_n^*, R_0^*)}{dt} = 0, \tag{107}$$

and

$$\frac{dk(S^*, V^*, E^*, T_C^*, T_n^*, R^*)}{dt} < 0, \tag{108}$$

the E.E.P $E^* = (S^*, V^*, E^*, I^*, T_C^*, T_n^*, R^*)$ for the system satisfies,

$$\pi = \mu S^{**} + \alpha S^{**} + \theta S^{**} - (1-\theta)V^{**} - \rho R^{**} \tag{109}$$
$$\theta S^{*}* = \alpha S^{*} * + EV^{*} * + \mu V^{*} * \tag{110}$$
$$\lambda^{**} E^{**} = \alpha S^{**} + EV^{**} - \mu E^{**} \tag{111}$$
$$\lambda^{*} * E^{**} = \Omega_1 I^{**} \tag{112}$$
$$\Omega_2 T_C^{**} = ^{**} \tag{113}$$
$$\Omega_3 T_n^{**} = (1-\rho)I \tag{114}$$
$$\Omega_4 R^{**} = \omega T_C^{**} + \phi T_n^{**} \tag{115}$$

$dK(S,V,E,I,T_C,T_n,R) = (1-\frac{S^{**}}{S})\frac{dS}{dt} + y_1(1-\frac{V^{**}}{V})\frac{dV}{dt} + y_2(1-\frac{E^{**}}{E})\frac{dE}{dt} + y_3(1-\frac{dI}{dt})\frac{dI}{dt} + (1-\frac{T_C^{**}}{T_C})\frac{dT_C}{dt} + y_4(1-\frac{T_n^{**}}{T_n})\frac{dT_n}{dt} + (1-\frac{dR_0^{**}}{R_0})\frac{dR_0}{dt}$
substituting for $\frac{dS}{dt}$, $\frac{dI}{dt}$, $\frac{dT}{dt}$, $\frac{dR}{dt}$, $\frac{dS_0}{dt}$, $\frac{dI_0}{dt}$, $\frac{dT_0}{dt}$, $\frac{dR}{dt}$ in the equation we to obtain
$dK(S,V,E,I,T_C,T_n,R) = (1-\frac{S^{**}}{S})\frac{dS}{dt} + y_1(1-\frac{V^{**}}{V})\frac{dV}{dt} + y_2(1-\frac{E^{**}}{E})\frac{dE}{dt} + y_3(1-\frac{I^{**}}{I})\frac{dI}{dt} + y_4(1-\frac{T_C^{**}}{T_C})\frac{dT_C}{dt} + y_5(1-\frac{T_n^{**}}{T_n})\frac{dT_n}{dt} + y_6(1-\frac{R^{**}}{R})\frac{dR}{dt}$ substituting for $\frac{dS}{dt}$, $\frac{dV}{dt}$, $\frac{dE}{dt}$, $\frac{dI}{dt}$, $\frac{dT_C}{dt}$, $\frac{dT_n}{dt}$, $\frac{dR}{dt}$ in the equation we to obtain
$dk(S,V,E,I,T_C,T_n,R) = (1-\frac{S^{**}}{S})(\pi + \rho R + (1-\theta)V - \theta S - \alpha S - \mu S - \rho R - (1-\theta)V + \theta S + \alpha S + \alpha S + \mu S) + y_1(1-\frac{V^{**}}{V})(\theta S - (1-\theta)V - \epsilon V - \mu V) + y_2(1-\frac{E^{**}}{E}(\alpha S + \epsilon V - \lambda E - \mu E) +_3 (1-\frac{I^{**}}{I})(\lambda E - \Omega_1 I) + y_4(1-\frac{T_C^{**}}{T_C})(\rho I - \Omega_2 T_C) + y_5(1-\frac{T_n^{**}}{T_n})(1-\rho)I - \Omega_3 T_n) + y_6(1-\frac{R^{**}}{R})(\omega T_C + \phi T_n - \Omega_4 R)$
$dk(S,V,E,I,T_C,T_n,R) = \mu S^{**} + \alpha S^{**} + \theta S^{**} - (1-\theta)V6** - \rho R^{**} + \rho R + (1-\theta)V - \theta S - \alpha S - \mu S - \frac{S^{**}}{S}\mu S^{**} - \frac{S^{**}}{S}\alpha S^{**} - \frac{S^{**}}{S}\theta S^{**} + \frac{S^{**}}{S}(1-\theta)V^{**} + \frac{S^{**}}{S}\rho R^{**} - \frac{S^{**}}{S}\rho R - (1-\theta)V\frac{S^{**}}{S} + \theta S\frac{S^{**}}{S} + \frac{S^{**}}{S}\alpha S + \frac{S^{**}}{S}\mu S + \theta y_1 S - (1-\theta)Vy_1 - \epsilon V y_1 - \frac{V^{**}}{V}y_1\theta S + \frac{V^{**}}{V}y_1(1-\theta)V + \frac{V^{**}}{V}\epsilon y_1 V + \frac{V^{**}}{V}\mu V y_1 + \alpha y_2 S + \epsilon y_2 V - \lambda y_2 E - \mu y_2 E - \frac{E^{**}}{E}\alpha y_2 S - \frac{E^{**}}{E}E y_2 V + \frac{E^{**}}{E}\lambda y_2 E + \frac{E^{**}}{E}\mu y_2 E + \lambda y_3 E - \Omega_1 y_3 I - \frac{I^{**}}{I}y_3\Omega_1 I + \rho y_4 I - \Omega_2 y_4 T_C - \frac{T_C^{**}}{T_C}\rho y_4 I + \frac{T_C^{**}}{T_C}\Omega_2 y_4 T_C + (1-\rho)y_5 I - \Omega_3 y_5 T_n - \frac{T_n^{**}}{T_n}(1-\rho)y_5 I + \frac{T_n^{**}}{T_n}\Omega_3 y_5 T_n +_6 T_C + \phi y_6 T_n - \Omega_4 y_6 R - \frac{R^{**}}{R}y_6\phi y_6 T_n + \frac{R^{**}}{R}\Omega_4 R$
$dk(S,V,E,I,T_C,T_n,R) = \mu S^{**} + \alpha S^{**} + \theta S^{**} - (1-\theta)V^{**} - \rho R^{**} + \rho R + (1-\theta)V - \theta S - \alpha S - \mu S - \frac{S^{**}}{S}\mu S^{**} - \frac{S^{**}}{S}\alpha S^{**} - \frac{S^{**}}{S}\theta S^{**} + \frac{S^{**}}{S}(1-\theta)V^{**} + \frac{S^{**}}{S}\rho R^{**} - \frac{S^{**}}{S}\rho R - (1-\theta)V\frac{S^{**}}{S} + \theta S\frac{S^{**}}{S} + \frac{S^{**}}{S}\alpha S + \frac{S^{**}}{S}\mu S + \theta y_1 S - (1-\theta)Vy_1 - \epsilon V y_1 - \mu V y_1 - \frac{V^{**}}{V}y_1\theta S + \frac{V^{**}}{V}y_1(1-\theta)V + \frac{V^{**}}{V}\epsilon y_1 V + \frac{V^{**}}{V}\mu V y_1 + \alpha y_2 S + \epsilon y_2 V - \lambda y_2 E - \mu y_2 E - \frac{E^{**}}{E}\alpha y_2 S - \frac{V^{**}}{V}\epsilon y_2 V + \frac{V^{**}}{V}\lambda y_2 E + \frac{V^{**}}{V}\mu y_2 E + \lambda y_3 E - \Omega_1 y_3 I - \frac{I^{**}}{I}\lambda y_3 E + \frac{V^{**}}{V}y_3\Omega_1 I + \rho y_4 I - \Omega_2 y_4 T_C - \frac{T_C^{**}}{T_C}\rho y_4 I + \frac{V^{**}}{V}\Omega_2 y_4 T_c + (1-\rho)y_5 I - \Omega_3 y_5 T_n - \frac{T_n^{**}}{T_n}(1-\rho)y_5 I + \frac{T_n^{**}}{T_n}\Omega_3 y_5 T_n + \omega y_6 T_C + \phi y_6 T_n - \Omega_4 y_6 R - \frac{R^{**}}{R}y_6\omega T_C - \frac{R^{**}}{R}\phi y_6 T_n + \frac{R^{**}}{R}\Omega_4 R$
$\lambda = \beta\frac{(I+\eta_1 T_C + \eta_2 T_n)}{N}$,
$I(s) = -\frac{\beta I}{N} + \frac{y_1\beta I}{N}$,
$T_C = -\frac{\beta\eta_1 T_C}{N} + \frac{y_1\beta T_C}{N}$,
$T_n = -\frac{\beta\eta_2 T_n}{N} + \frac{y_1\beta\eta_2 T_n}{N}$,

$dk(S,V,E,I,T_C,T_n,R) = \mu S^{**} + \alpha S^{**} + \theta S^{**} - (1-\theta)V^{**} - \rho R^{**} + \rho R + (1-\theta)V - \theta S - \alpha S - \mu S - \frac{S^{**}}{S}\mu S^{**} - \frac{S^{**}}{S}\alpha S^{**} - \frac{S^{**}}{S}\theta S^{**} + \frac{S^{**}}{S}(1-\theta)V^{**} + \frac{S^{**}}{S}\rho R^{**} - \frac{S^{**}}{S}\rho R - (1-\theta)V\frac{S^{**}}{S} + \theta S\frac{S^{**}}{S} + \frac{S^{**}}{S}\alpha S + \frac{S^{**}}{S}\mu S + \theta y_1 S - (1-\theta)Vy_1 - \epsilon V y_1 - \mu V y_1 - \frac{V^{**}}{V}y_1\theta S + \frac{V^{**}}{V}y_1(1-\theta)V + \frac{V^{**}}{V}\epsilon y_1 V + \frac{V^{**}}{V}\mu V y_1 + \alpha y_2 S + \epsilon y_2 V - [-\frac{\beta I}{N} + \frac{y_1\beta I}{N}I + -\frac{\beta I}{N} + \frac{y_1\beta I}{N}T_C + -\frac{\beta\eta_2 T_n}{N} + \frac{y_1\beta\eta_2 T_n}{N}T_n]y_2 E - \mu y_2 E - \frac{E^{**}}{E}\alpha y_2 S - \frac{V^{**}}{V}\epsilon y_2 V + \frac{V^{**}}{V}\lambda y_2 E + \frac{V^{**}}{V}\mu y_2 E + \lambda y_3 E - \Omega_1 y_3 I - \frac{I^{**}}{I}\lambda y_3 E + \frac{V^{**}}{V}y_3\Omega_1 I + \rho y_4 I - \Omega_2 y_4 T_C - \frac{T_C^{**}}{T_C}\rho y_4 I + \frac{V^{**}}{V}\Omega_2 y_4 T_c + (1-\rho)y_5 I - \Omega_3 y_5 T_n - \frac{T_n^{**}}{T_n}(1-\rho)y_5 I + \frac{T_n^{**}}{T_n}\Omega_3 y_5 T_n + \omega y_6 T_C + \phi y_6 T_n - \Omega_4 y_6 R - \frac{R^{**}}{R}y_6\omega T_C - \frac{R^{**}}{R}\phi y_6 T_n + \frac{R^{**}}{R}\Omega_4 R$
$I\frac{\beta}{N}(y_1 - 1) = 0$,

$I\frac{\beta}{N} \neq 0,$

$$y_1 - 1 = 0, \tag{116}$$
$$y_1 = 1, \tag{117}$$
$$y_2 = \frac{-\theta}{\theta} \tag{118}$$
$$y_3 = -\frac{\theta\mu}{\alpha\lambda} \tag{119}$$
$$y_4 = 0 \tag{120}$$
$$y_5 = -\frac{-\Omega_1\theta\mu}{\alpha\lambda} \tag{121}$$
$$y_6 = 0 \tag{122}$$

P=$\mu S^{**} + \alpha S^{**} + \rho R + (1-\theta)V + \frac{S^{**}}{S}(1-\theta)V^{**} + \frac{S^{**}}{S}\rho R^{**} + \theta S\frac{S^{**}}{S} + \alpha S\frac{S^{**}}{S} + \mu S\frac{S^{**}}{S} + \theta S - (1-\theta)V - \epsilon V - \mu V - \frac{V^{**}}{V}\theta S + \frac{V^{**}}{V}(1-\theta)V + \frac{V^{**}}{V}\epsilon V + \frac{V^{**}}{V}\mu V + \frac{\mu\theta}{\alpha}E + \frac{E^{**}}{E}\theta S + \frac{E^{**}\epsilon\theta}{E\alpha}V + \frac{\Omega_1\theta\mu}{\alpha\lambda} + \frac{I^{**}\theta\mu}{I\alpha}$

Q=$-\theta S - \frac{\epsilon\theta}{\alpha}V - \frac{E^{**}}{E}\lambda\frac{\mu\theta}{\alpha}E - \frac{E^{**}}{E}\frac{\mu\theta}{\alpha}E - \frac{\theta\mu}{\alpha} - \frac{\Omega_1\theta\mu}{\alpha\lambda} - \frac{I^{**}}{I}\frac{\theta\mu}{\lambda\alpha}\Omega_1 I$ Then $\frac{dK}{dt} = 0$, holds only when $(S = S^{**}, V = V^{**}, I = I^{**}, T_C = T_C^{**})$,$T_n = T_n^{**}$,$R = R^{**}$ Consequently, the largest compact unchanging set in $(S; E; I) \in \sqcap$: $\frac{dv}{dt} = 0$ is the singleton E_*^{**} using Lasalle's invariance principal, $\frac{dL(S,I,A,R)}{dt} < 0$ if and only if $P > Q$ [17]. This finding indicates that bacterial meningitis would continue regardless of the starting circumstances anytime $P > Q$.

4. Model parameters used for simulation

In order to examine the dynamic Runge-Kutta analysis of the framework's state variables' dynamics when the model specifications are present technique is applied to the model equations in this section and then utilized to do numerical simulations using the fourth order Runge-Kutta method in MatlabR2015a. An ordinary differential equation's initial value problem can be solved numerically using the Runge-Kutta method. The initial conditions and parameters values listed in table 2 are used to carry out the numerical simulations and a graphical presentation of the numerical results is made.

Parameter	Value	Source
π	100-100000	[18]
μ	0.02	[19]
λ	0.00005	assumed
α	0.02	assumed
θ	0.5	[20]
ϵ	0.6	assumed
τ	0.05-0.5	[2]
φ	0.6	assumed
ξ	0.05	assumed
ϕ	0.5	assumed
ω	0.045	assumed
ρ	0.6	assumed
σ	0.851	[21]
β	0.88	[2]
S	3219640	assumed
V	495329	[22]
E	742993	assumed
I	247664	assumed
T_C	33682	assumed
T_n	213982	assumed
R	181885	[23]

Table 2. *Parameter values and initial conditions for the Model*

4.1. Sensitivity study of fundamental reproduction numbers normalized

Differential calculus is used to do parameter sensitivity analysis. The investigation entails looking at the parameter that has the biggest impact on the fundamental reproduction number as in table 3. It is commonly used to determine the robustness of model predictions to parameter values since there are errors in collecting data or pre-assumed parameter values. It is used to discover parameters that have high impact on R_0 and should be targeted by intervention strategy. Sensitivity indices allow us to measure the relative change in a variable when a parameter changes. The normalized forward sensitivity index of a variable with respect to a parameter is the ratio of the relative change in the parameter. Since errors can occur in data collection or pre-assumed parameter values, it is frequently used to assess how resistant model predictions are to parameter values.

Parameters	Sensitivity indices
μ	-0.000682545
ϕ	-0.203802
φ	0.110635
ω	0.226211
ρ	-45.2403
η_1	0.00557279
η_2	1.0021
τ	-0.0475964

Table 3. *Sensitivity index of the model*

4.2. The impact of bacterial meningitis disease on susceptible population.

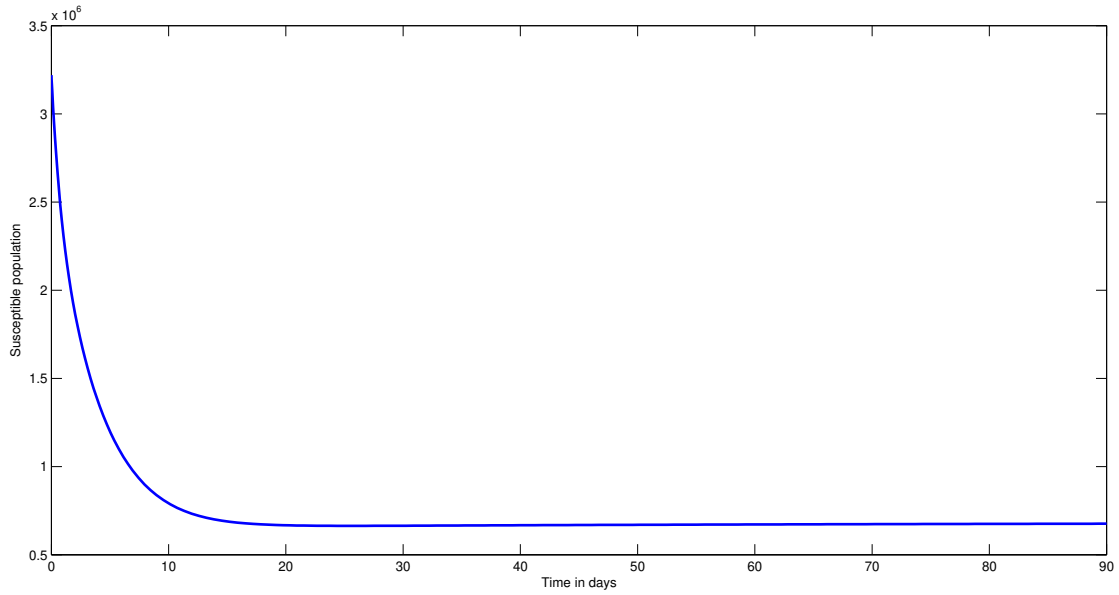


Figure 4. *Number of vulnerable individuals across period*

From literature, it's known that without an outbreak of any disease the population of any given place is supposed to increase exponentially as we only have rates of natural births and mortality. In case of an outbreak like bacterial meningitis has negative effect on the general public population as it tends to grow slowly, as some will be infected and others will be vaccinated hence leaving the susceptible class, the susceptible population decrease with time until a certain point where it remain constant, if appropriate and early intervention measures are not implemented, more individuals will contract bacterial meningitis. The transmissibility of bacterial meningitis also increases as the volume of bacteria in the environment increases, as observed in figure 4.

4.3. The impact of bacterial meningitis disease on vaccinated population.

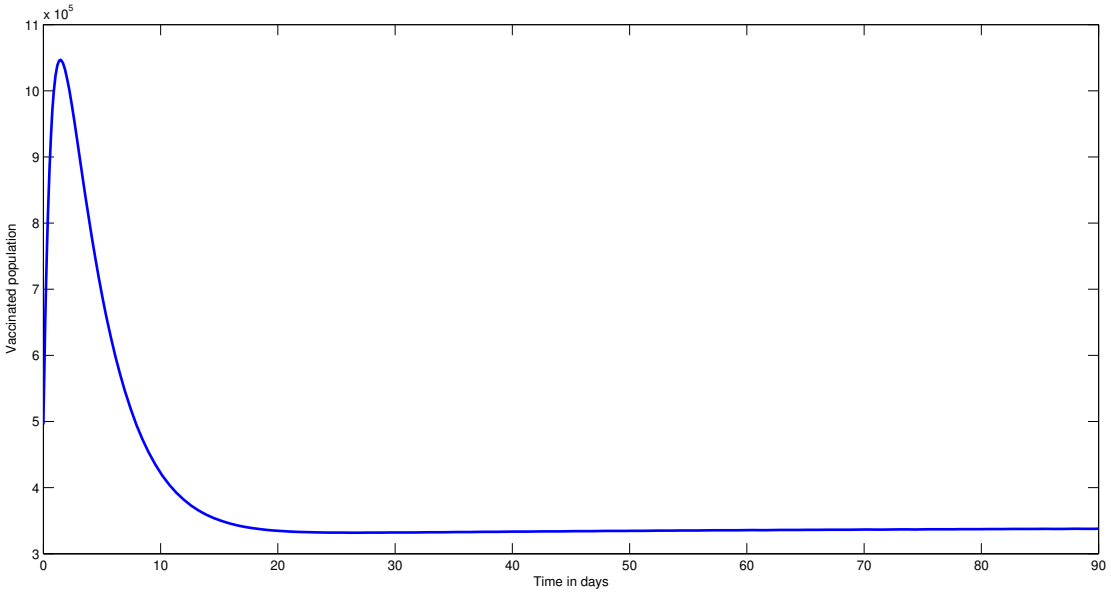


Figure 5. *Number of vaccinated individuals across period*

Vaccines reduce risks of getting a disease by working with your body's natural defenses to build protection. When you get a vaccine, your immune system responds. As individual gets vaccinated the population increases to a maximum point then start decreasing as some don't respond well to the vaccines and others may get exposed to bacterial meningitis. Vaccination is key to primary health care, an indisputable human right, and one of the best health investments money can buy. Vaccines are also critical to the prevention and control of bacterial meningitis outbreaks. They underpin global health security and are a vital tool in the battle against antimicrobial resistance as observed in figure 5.

4.4. The impact of bacterial meningitis disease on exposed population.

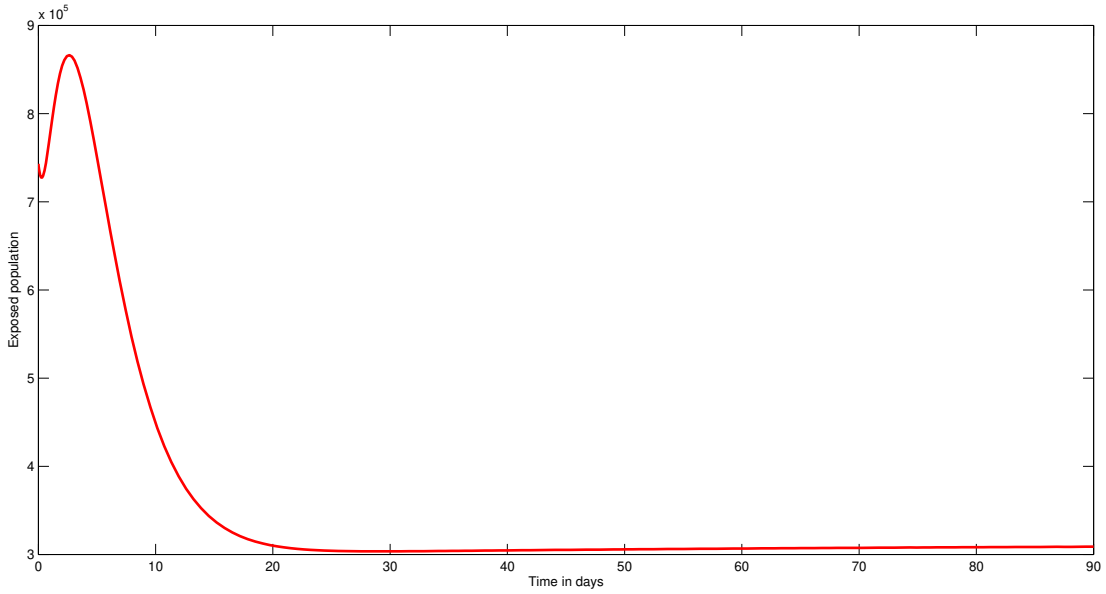


Figure 6. *Number of exposed individuals across period*

During bacteria meningitis outbreaks most individuals gets exposed to the disease either knowingly or unknowingly and become at risk of infections, higher risk is seen when people are living in close proximity, for example at mass gatherings, in refugee camps, in overcrowded households or in student, military and other occupational settings. The exposed population in figure 6 increases to a maximum point then start decreasing as most them will get screened and get early diagnosis against the disease.

4.5. The impact of bacterial meningitis disease on infected population.

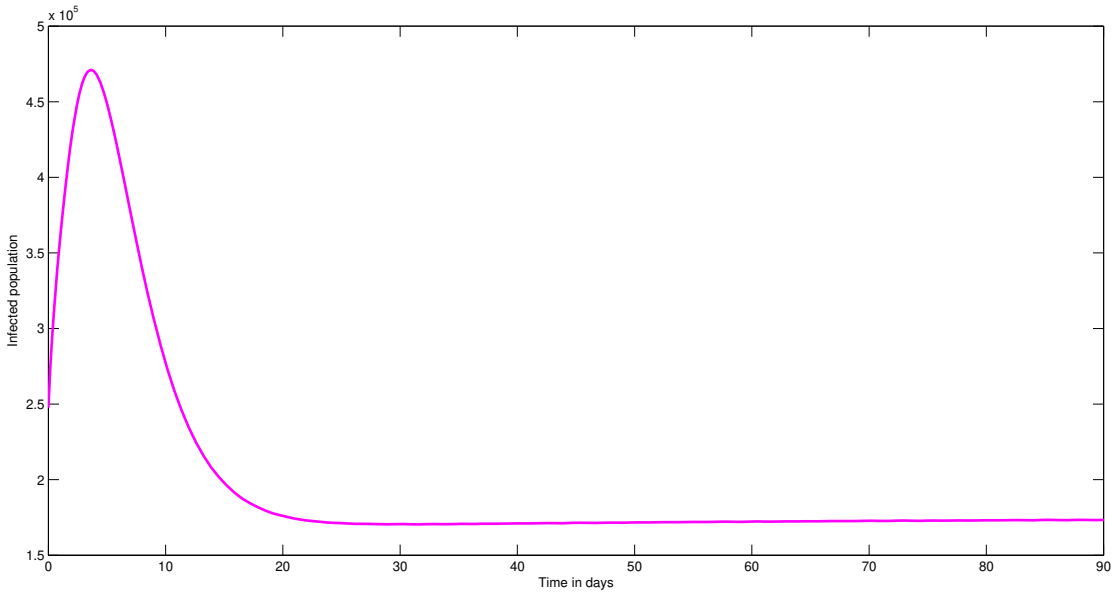


Figure 7. *Number of infected individuals across period*

If an individual is infected with bacterial meningitis either through direct contact with respiratory or throat secretions the population increases to a maximum point then decreases as diagnosis methods are applied. Without treatment, bacterial meningitis can lead to serious complications, such as brain damage, hearing loss, seizures, and in some cases, death. Even with treatment, some patients may experience long-term neurological problems or disabilities as seen in figure 7.

4.6. The impact of bacterial meningitis disease on treated with counterfeit drugs population.

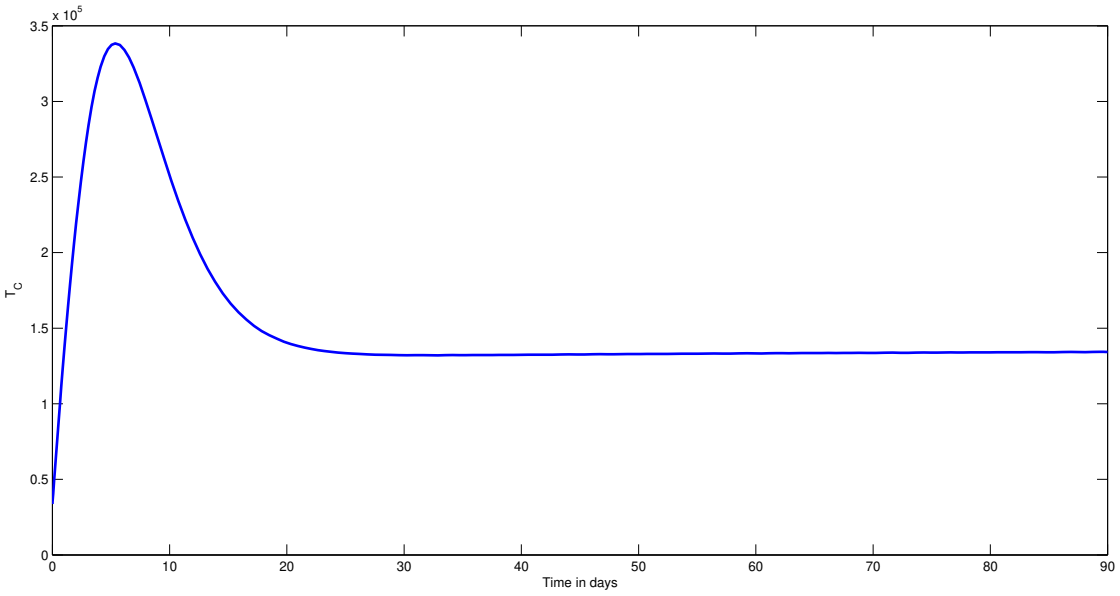


Figure 8. *Number of treated with counterfeit drugs individuals across period*

Treating individuals with counterfeit drugs can have serious consequences for their health and well-being. Counterfeit drugs are medications that are deliberately and fraudulently mislabeled with respect to their identity or source. As seen in fig 8 the population increases sharply then decrease and maintain at a level where more people remain infected with the disease. Some of the effect of counterfeit drugs include;lack of efficacy, toxicity and side effects, Public Health Concerns. To mitigate the risks associated with counterfeit drugs, regulatory agencies and healthcare providers play a crucial role in ensuring the integrity of the pharmaceutical supply chain. Patients should also be vigilant and obtain medications from reputable sources, such as licensed pharmacies and healthcare providers.

4.7. The impact of bacterial meningitis disease on non counterfeit drugs population.

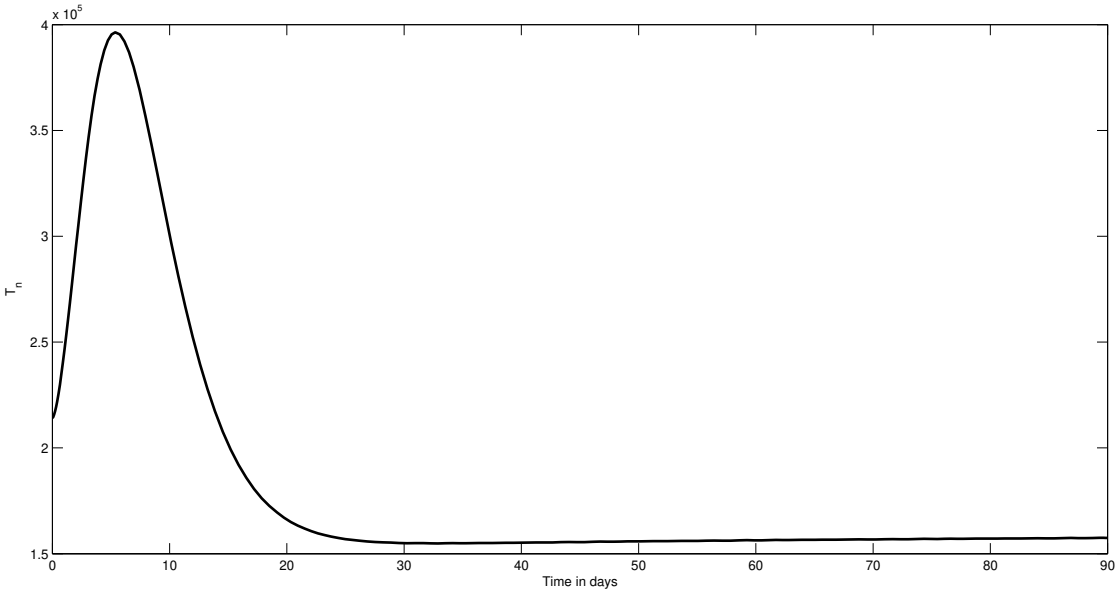


Figure 9. *Number of treated with non-counterfeit drugs individuals across period*

Treating bacterial meningitis with genuine, non-counterfeit drugs is crucial for effectively combating the infection and improving patient outcomes. As observed in figure 9 the population initially increases then decreases gradually until its maintained at base line level. using non-counterfeit, genuine drugs for treating bacterial meningitis is essential for effective therapy, reducing complications, preventing resistance, and ensuring patient safety and recovery. Always obtaining medications from reputable sources and following healthcare provider recommendations are crucial steps in ensuring effective treatment outcomes.

4.8. The impact of bacterial meningitis disease on recovered population.

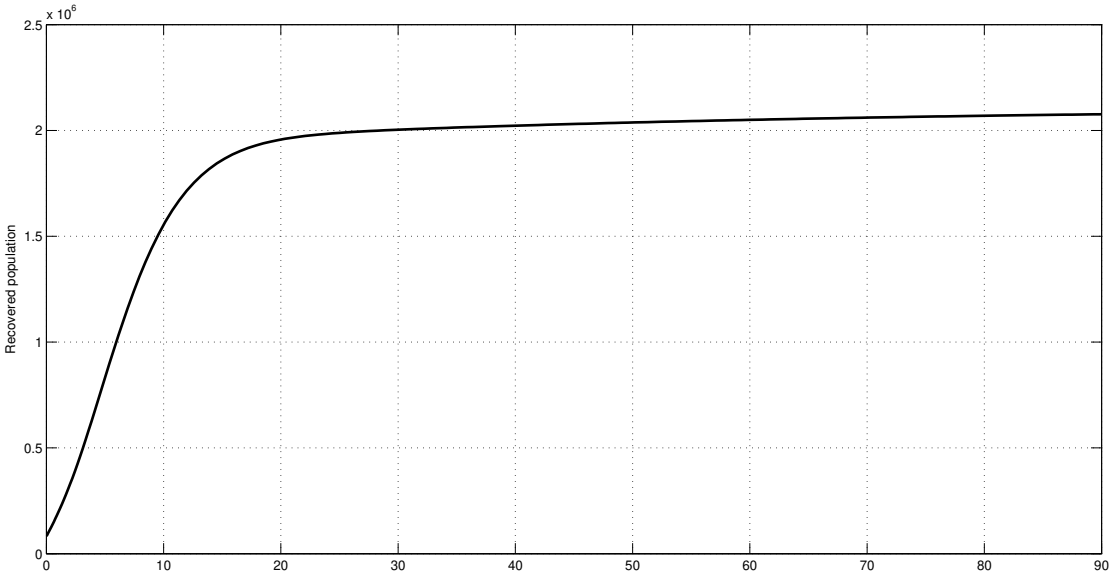


Figure 10. *Number of recovered individuals across period*

Recovery from bacterial meningitis is often a complex and ongoing process. While many individuals recover well, understanding and addressing the potential long-term effects is crucial for improving quality of life and ensuring continued support and care. The population increases as more are receiving medication as observed in figure 10. while recovery from bacterial meningitis is a significant achievement, the impact on individuals and the broader community can be extensive. Addressing the long-term effects, supporting survivors, and investing in preventive measures are crucial for mitigating these impacts.

4.9. A graph of variation of rate at which the vaccinated individuals get exposed on Vaccinated and exposed population.

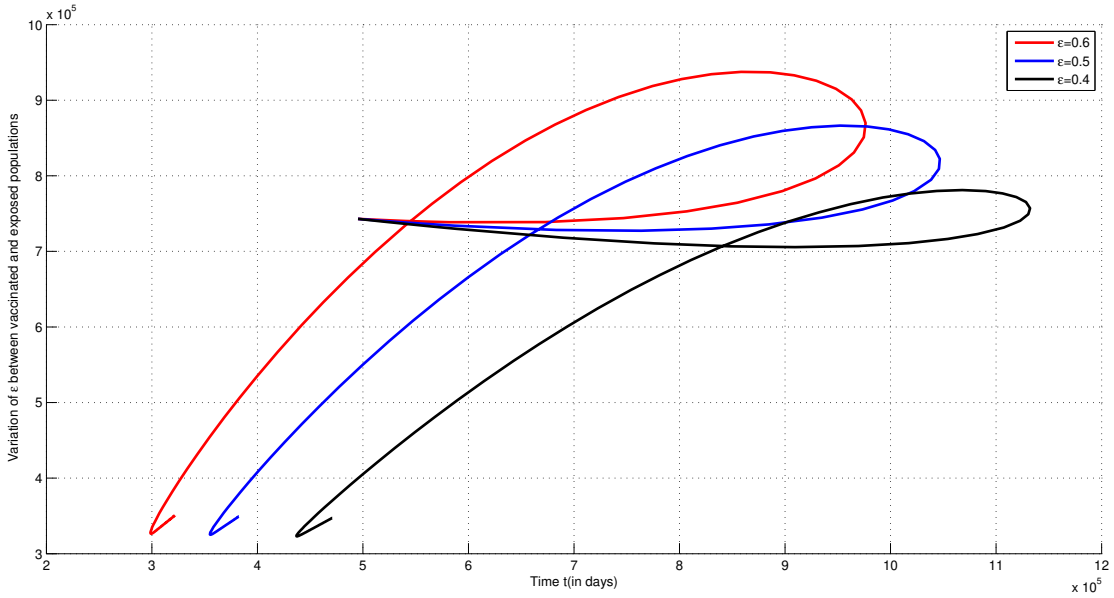


Figure 11. *Number of vulnerable individuals across period*

When considering bacterial meningitis, the differences between individuals who have been vaccinated and those who have been exposed to the disease are significant. From figure 11 it can be seen that if you increase the rate at which vaccinated get exposed it increases the vaccinated and exposed population and if you reduce the rate, it reduces the population of vaccinated and exposed. In summary, vaccination against bacterial meningitis provides significant protection against severe disease, reduces the risk of complications, and contributes to community-wide immunity. Natural exposure without vaccination, on the other hand, poses a higher risk of severe disease and complications.

4.10. A graph of variation of rate at which the exposed individuals get infected.

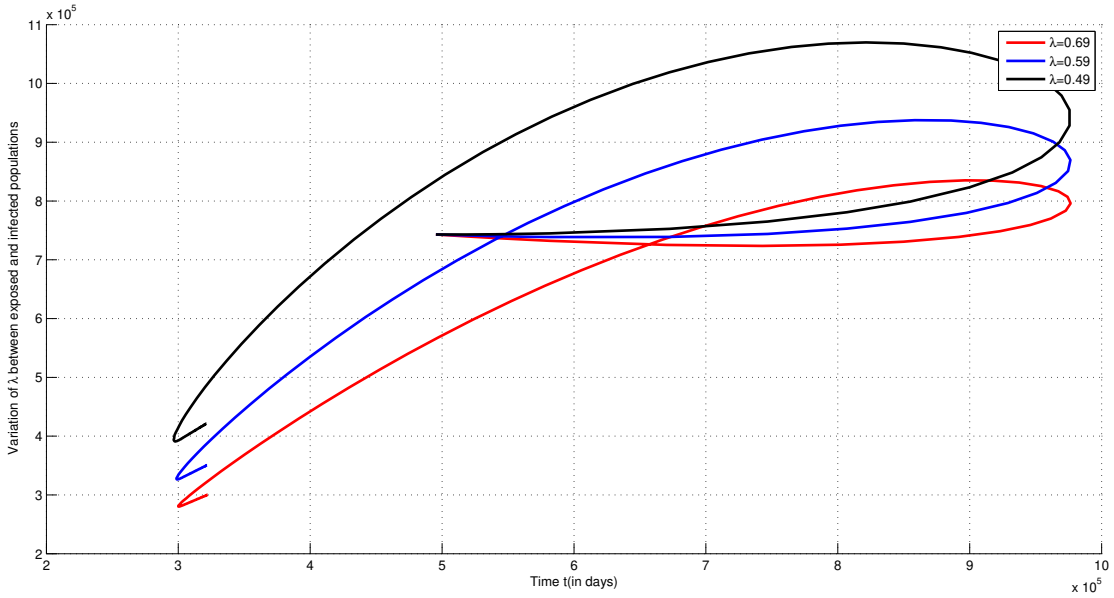


Figure 12. *Number of vulnerable individuals across period*

The primary difference between exposed and infected individuals is the presence of symptoms and clinical disease. Exposure means contact with the bacteria but does not necessarily lead to illness, while infection results in the actual disease with significant health implications. From figure 12 it can be observed that increasing the force of infection leads to decreasing the exposed and infected population and increasing the force of infection lead to decrease in exposed and infected population. Monitoring and preventive measures are important for those exposed, particularly in high-risk settings, to prevent the progression to full-blown bacterial meningitis.

4.11. A graph of variation of the rate at which the infected individuals get treated using counterfeit drugs.

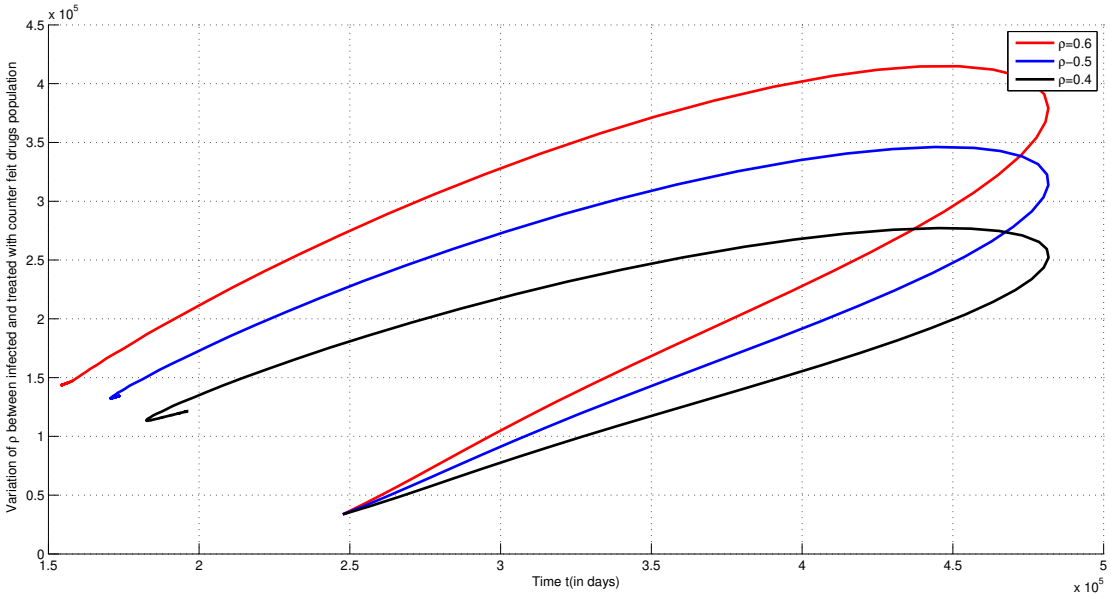


Figure 13. *Number of vulnerable individuals across period*

The rate at which infected individuals receive counterfeit drugs for bacterial meningitis is influenced by factors including geographic location, the strength of regulatory frameworks, the integrity of healthcare systems, public awareness, economic conditions, and healthcare provider practices. From figure 13 if you increase rate at which infected receive counterfeit drugs the population of infected and treated with counterfeit drugs increases and if you decrease rate at which infected receive counterfeit drugs the population of infected and treated with counterfeit drugs decreases. Regions with weak regulatory systems and healthcare infrastructure are more likely to see higher rates of counterfeit drug use, impacting the effectiveness of treatment and patient outcomes.

4.12. A graph of variation of the rate at which the infected individuals get treated using non counterfeit drugs.

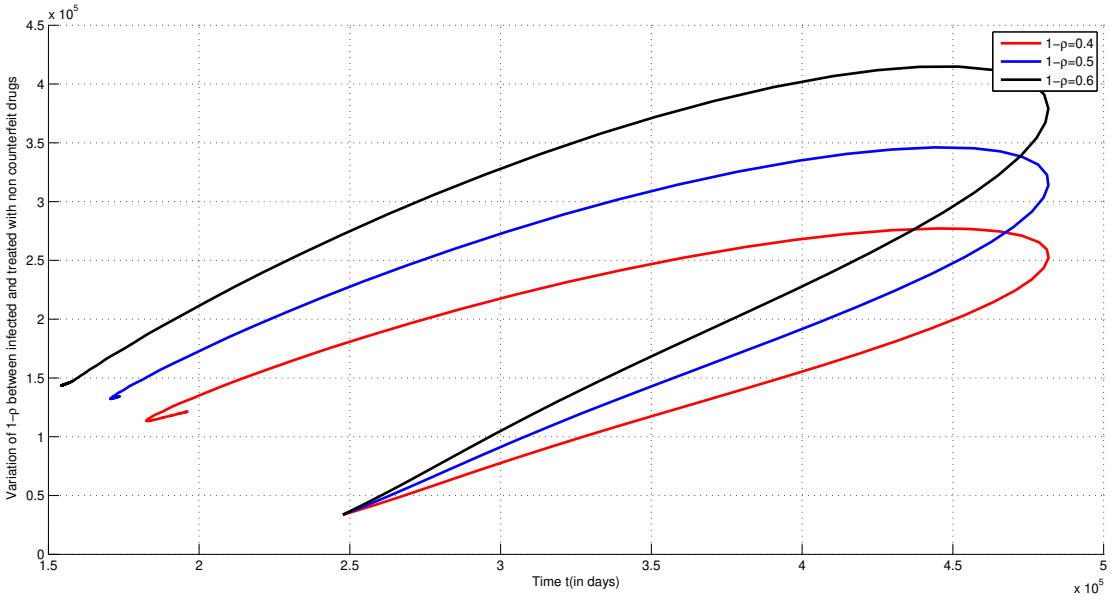


Figure 14. *Number of vulnerable individuals across period*

The rate at which infected individuals receive non-counterfeit drugs for bacterial meningitis is influenced by the quality of the healthcare system, regulatory oversight, supply chain integrity, economic conditions, public awareness, and healthcare provider practices. From figure 14 if you increase rate at which infected receive non- counterfeit drugs the population of infected decreases while the treated population increases and if you decrease rate at which infected receive non- counterfeit drugs the population of infected increases while the treated population decreases. Regions with strong healthcare infrastructure, effective regulations, and high public awareness tend to have higher rates of non-counterfeit drug administration. Conversely, areas with weaker systems and less regulatory control may face challenges in ensuring that all patients receive genuine medications.

4.13. Discussion and conclusion

The feasible region, positivity of the solution set, effective reproductive number, equilibria points, and their stability were all obtained during the model analysis in section 3. Numerical simulation was carried out using Matlab software to show the relationship between counterfeit and non counterfeit drugs. In those institution where there poor health system and limited awareness to the public it become difficult to treat bacterial meningitis due to counterfeit drugs in the health system.
The government or health provider should be equipped with machines or personnel to determine the effectiveness of the drugs administered to the people to minimize case of counterfeit drugs

4.14. Conflict of interest

The writers state that they have no personal or professional ties to individuals or groups that could improperly influence their work. There isn't any personal or professional relationship with any firm, product, or service that would be thought to have an impact on the evaluation or the opinion stated in the article.

4.16. Compliance with Ethical Standards

Informed consent is not required for this type of study.

4.17. Ethical Conduct

The required norms while carrying out research was adhered to.

4.18. Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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