




Mathematical model for the dynamics of bacterial meningitis (*Meningococcal meningitis*): a case study of Yobe State Specialist Hospital, Damaturu, Nigeria

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Abstract	Article History
<p>A model for bacterial meningitis was created by adding a class of transporters to the basic Susceptible Carrier Infected and Recovered (SCIR) model, since vaccination and treatment are the best methods of controlling the transmission of most overpowering sicknesses. Immunization assists helpless people with building either a drawn out invulnerability or transient resistance while treatment decreases the quantity of sickness actuated passing and the quantity of irresistible people locally or country. This study comprises of a mathematical model for bacterial meningitis dynamics that can be used to a wide range of mathematical modeling problems. In this exploration, a nonlinear deterministic model with time reliance controls has been proposed to depict the elements of bacterial meningitis in a populace. We discovered that the (EEP) and (DFE) are locally asymptotically stable in our study. We now advise the researcher to determine whether it is globally asymptotically stable in order to achieve optimal disease control. The presence of an endemic harmony and the calculation of the reproduction number R_0. The mathematical arrangement shows that vaccinating vulnerable people will prompt disposal of the infection in the public and furthermore it will lessen the weight on wellbeing suppliers. Numerical simulations were presented to explain the parameters in the end path in the model were carried out by using MAPLE software. Most likeable of this research work shows that the rate at which treatment and vaccination rate increases to the higher value the recovered compartment increases to the peak point. This means treatment and vaccination has an impact on reducing the case of bacterial meningitis in a population. Now we conclude that a high infection transmission rate requires a high vaccine and treatment rate on the effect of vaccination against meningitis.</p>	<p>Received: 18/04/2022 Accepted: 11/08/2023 Published: 16/08/2023</p> <p>Keywords Bacterial meningitis; Damaturu; Disease free equilibrium (DFE); Endemic equilibrium point (EEP); <i>Meningococcal meningitis</i>; Reproduction number; SCIR model</p> <p>License: CC BY 4.0*</p>  <p>Open Access Article</p>
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1.0 Introduction

Meningitis is derived from the Greek word “Meninx” which means membrane and the medical suffix “-itis” which implies inflammation stated Thus, *Meningococcal meningitis* is a bacterial form of Meningitis causing the inflammation of the thin lining surrounding the brain and the spinal cord. It could results into severe brain damage and death in about 50% of untreated cases, which was discussed

comprehensively by Asamoah *et al.* (2018). Meningitis is the inflammation of the meninges. This disease can be caused by different organisms. Bacteria and viruses are the most common causes of meningitis by Baba *et al.* (2020). When these organisms are in the cerebrospinal fluid, everything in this immediate area will become inflamed. The introduction of bacteria in the meninges will almost surely cause meningitis. Sometimes the presence of this bacterium is the result

of bacteria traveling from an infection in some other part of the body, this was mentioned by Asamoah *et al.* (2020). Bacterial meningitis occurs more often than viral. In fact more than 80% of all meningitis cases are caused by three distinct types of bacteria. The types are *Neisseria meningitides*, *haemophilus influenza*, and *Streptococcus pneumonia*. *Neisseria meningitides* is the most responsible and most frequent cause of *Meningococcal meningitis* occurrences of the three. Bacterial meningitis is the most serious type of meningitis. It can lead to death or permanent disability. It is a medical emergency. Meningitis affects the meninges, the membranes that surround the brain and spinal cord and protect the central nervous system (CNS), together with the cerebrospinal fluid according to Musa *et al.* (2020). Buonomo *et al.* (2020) discussed extensively in the work that the case of meningitis is of great public health importance because it has a high morbidity and fatality rate. Various researchers performed work on meningitis and found out that the effective vaccine against meningitis is the solution for the eradication to the disease. Despite the fact that we now wish to perform a new research work to critically look in to the transmission dynamics and control of the disease, so as to come up with best feasible solution in controlling meningitis disease. Authors like Novak *et al.* (2019) explain extensively with apprehended justification in their contribution towards future directions for meningitis surveillance and vaccine evaluation in the meningitis belt of sub-Saharan Africa. Likewise, Saha (2020), discussed on the unearthing the unknown causes of meningitis and Irving *et al.* (2012), proposed a mathematical modelling *Meningococcal meningitis* in the African meningitis belt of the continent and part of Africa as well as Elmojtaba and Adam (2017), plays a very important role on their work on a mathematical model for meningitis disease in the west Africa (Nigeria), explaining how effective and epidemic among children with major shortcomings and economic setbacks in medical strategy.

Blyuss (2016), further stated in his work that epidemic meningitis is often caused by *Neisseria meningitides* sero-group A, B and C (*Meningococcus meningitis*). Novak *et al.* (2019) also lamented clearly that the so-called meningitis is common in sub-saharan Africa. Countries like Benin, Burkina Faso, Chad, Niger, Nigeria and Mali make up the meningitis belt where large scale epidemic occur after every few years. In Nigeria, cases can occur all through the year and increase during the dry season. An epidemic threshold

is used to differentiate epidemic emergence from simple seasonal rise in incidence. Epidemic meningitis diseases caused by the *Meningococcal bacterium*, which is common in Nigeria. In 1996, over 3,386 people died of meningitis in Nigeria (Saha, 2020). The main aim of this research work is to formulate a mathematical model for the transmission of *Meningococcal meningitis* and analyze the impact of vaccination program and the impact of treatment control of the infectious individuals in a population.

2.0 Materials and Methods

In this section a deterministic mathematical model of Susceptible Carrier Infected and Recovered (*SCIR*) was developed to investigate the dynamic of bacterial meningitis. A system of ordinary differential is equation to be use to investigate the behavior and dynamic of bacterial meningitis transmission in a population.

2.1 Model Description and Formulation

Before developing a model for meningitis it was essential to first consider the different classes S-susceptible, C-carrier, I-infected, R-recovered. A proportion of the susceptible class will come in to contact with carriers. Individual in the carrier class are able to infect others without suffering from the disease themselves, they will then become infected. Individual in the infected class come directly from the carrier class. The recovered class consists of those in the infected class that have recovered from the disease or died. The change in the susceptible class is given by the recruitment rate Λ , whining rate of vaccination ω , minus the force of infection $\frac{\beta IS}{N}$, natural death μ , and the rate of vaccination ρ . The change in the carrier class is given by the force of infection $\frac{\beta IS}{N}$, minus natural death rate μ , and those individual leaving the carrier class to infected class by the disease progression rate γ . The change in the infected class is given by the disease progression rate γ , and those individual leaving the infected class due to natural death rate μ , or by the disease induce death rate σ (that is death by infection) and those individual that have received treatment and recovered by the rate τ , And the change in the recovered class is given by the rate of vaccination ρ , the rate of treatment from the infected class τ , and those individual leaving the recovered class by whining rate of vaccination ω , to susceptible class and the natural death rate μ (Paireau *et al.*, 2016).

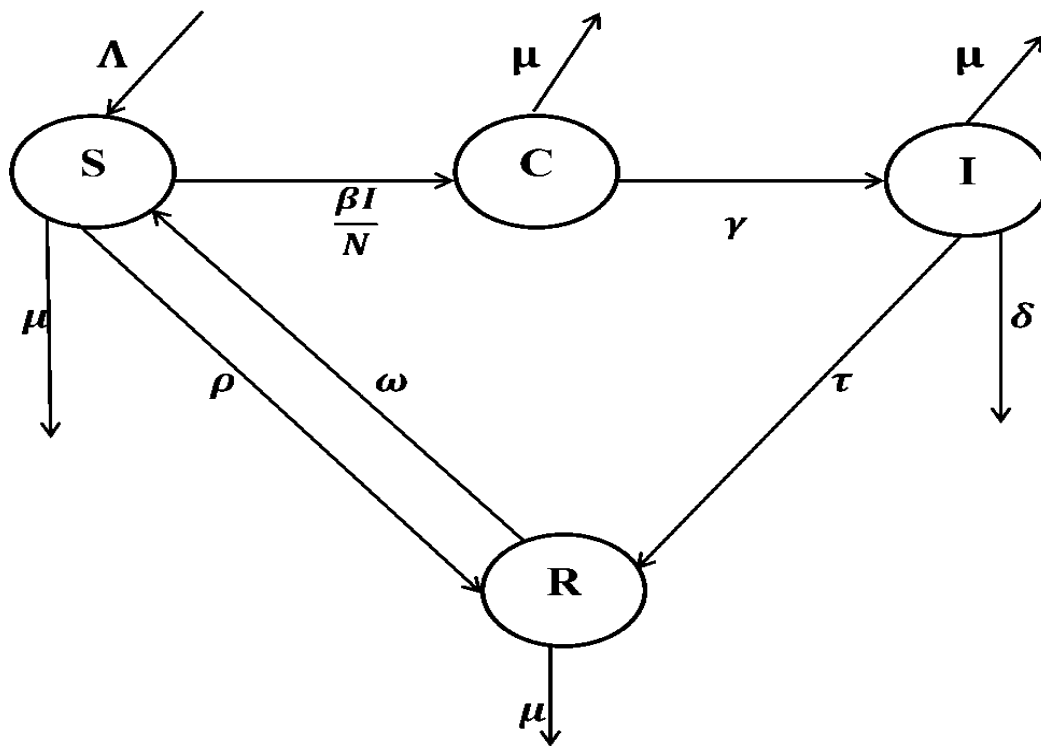


Figure 1: Schematic Diagram of The Model

These are the equations represented by the model

$$\frac{dS}{dt} = \Lambda - \frac{\beta IS}{N} + \omega R - \mu S - \rho S \tag{1}$$

$$\frac{dC}{dt} = \frac{\beta IS}{N} - \gamma C - \mu C \tag{2}$$

$$\frac{dI}{dt} = \gamma C - \tau I - \delta I - \mu I \tag{3}$$

$$\frac{dR}{dt} = \tau I + \rho S - \omega R - \mu R \tag{4}$$

2.2 Definition of Parameters and Compartment

S: Susceptible class.

C: Carrier class.

I: Infected class.

R: Recovered class.

Λ : Rate of recruitment.

β : Rate of transmission.

γ : Rate of progression.

σ : Disease induce rate.

τ : Rate of treatment.

ρ : Vaccination rate.

ω : Whining rate.

μ : Natural death rate.

N: Total population.

Simplifying the above equations, we have,

The equation now becomes

2.3 Invariant Region

Consider the region

Now let

$$K_1 = \mu + \rho \text{ (i)}$$

$$k_2 = \gamma + \mu \text{ (ii)}$$

$$k_3 = \tau + \delta + \mu \text{ (iii)}$$

$$k_4 = \omega + \mu \quad (iv)$$

$$\frac{dS}{dt} = \Lambda - \frac{\beta IS}{N} + \omega R - K_1 S \quad (5)$$

$$\frac{dC}{dt} = \frac{\beta IS}{N} - k_2 C \quad (6)$$

$$\frac{dI}{dt} = \gamma C - k_3 I \quad (7)$$

$$\frac{dR}{dt} = \tau I + \rho S - k_4 R \quad (8)$$

$$D = \left\{ (S, C, I, R) \in \mathbb{R}_+^4 : N \leq \frac{\Lambda}{\mu} \right\}$$

It is positively invariant and attracts all positive solutions of the model.

Theorem: The region D is positively invariant for the model.

Proof: Let $N = S + C + I + R$

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dC}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \quad (9)$$

$$\frac{dN}{dt} = \Lambda - \mu S - \mu C - \delta I - \mu I - \mu R \quad (10)$$

$$\frac{dN}{dt} = \Lambda - (S + C + I + R)\mu - \delta I \quad (11)$$

Since $N = S + C + I + R$ Now we have,

$$\frac{dN}{dt} = \Lambda - N\mu - \delta I \quad (12)$$

$$\frac{dN}{dt} = \Lambda - N\mu \quad (13)$$

By using method of integrating factor

$$IF = e^{\int \mu dt} \leq e^{\mu t}$$

$$\frac{d}{dt}(N \cdot IF) \leq \Lambda \cdot IF \quad (14)$$

$$d(N \cdot IF) \leq (\Lambda \cdot IF)dt \quad (15)$$

$$\int d(N \cdot IF) \leq \int (\Lambda \cdot IF) dt \quad (16)$$

$$N \cdot IF \leq \int (\Lambda \cdot IF) dt \quad (17)$$

$$[Ne^{\mu t}]_0^t \leq \int (\Lambda e^{\mu t}) dt \quad (18)$$

$$[Ne^{\mu t}]_0^t \leq \left[\frac{\Lambda}{\mu} e^{\mu t} \right]_0^t \quad (19)$$

$$N(t)e^{\mu t} - N(0)e^0 \leq \frac{\Lambda}{\mu} e^{\mu t} - \frac{\Lambda}{\mu} e^0 \quad (20)$$

$$N(t)e^{\mu t} - N(0) \leq \frac{\Lambda}{\mu} e^{\mu t} - \frac{\Lambda}{\mu} \quad (21)$$

$$N(t)e^{\mu t} \leq N(0) + \frac{\Lambda}{\mu} e^{\mu t} - \frac{\Lambda}{\mu} \quad (22)$$

Multiplying through by $e^{-\mu t}$

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu} - \frac{\Lambda}{\mu} e^{-\mu t} \quad (23)$$

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu} (1 - e^{-\mu t}) \text{ at } t > 0 \quad (24)$$

$$N(t) \leq 0 + \frac{\Lambda}{\mu} (1 - 0) \quad (25)$$

$$N(t) \leq \frac{\Lambda}{\mu} \quad (26)$$

If

$$N(0) \leq \frac{\Lambda}{\mu}, \text{ then } N(t) \leq \frac{\Lambda}{\mu}.$$

Hence the region D is positively invariant and attracts all the solution in \mathbb{R}_+^4 so that no solution path leaves through any boundary D.

2.4 Basic Reproduction Number (\mathfrak{R}_0)

The basic reproduction number is the number of secondary infections cause by the average infectious individual. Also the reproduction number is the threshold parameter that governs the spread of a disease. To obtain the basic reproduction number we let the variable

$$\chi = \left(\frac{dS}{dt} \frac{dC}{dt} \frac{dI}{dt} \frac{dR}{dt} \right).$$

Let $F_i(x)$ represent the rate at which new infections appear. While $v_i^+(x)$ and $v_i^-(x)$ are the rate at which individuals enter and leave each class.

$$F_i(x) = \begin{pmatrix} N \\ 0 \end{pmatrix} \tag{27}$$

$$V_i(x) = \begin{pmatrix} K_2 C \\ -\gamma C + k_3 I \end{pmatrix} \tag{28}$$

$$F_i(x) = \begin{pmatrix} \frac{\partial F_1}{\partial C} & \frac{\partial F_1}{\partial I} \\ \frac{\partial F_2}{\partial C} & \frac{\partial F_2}{\partial I} \end{pmatrix}$$

$$F = \begin{pmatrix} 0 & \frac{\beta s}{N} \\ 0 & 0 \end{pmatrix} \tag{29}$$

$$V = \begin{pmatrix} K_2 & 0 \\ -\gamma & k_3 \end{pmatrix} \tag{30}$$

$$V_i(x) = \begin{pmatrix} \frac{\partial V_1}{\partial C} & \frac{\partial V_1}{\partial I} \\ \frac{\partial V_2}{\partial C} & \frac{\partial V_2}{\partial I} \end{pmatrix}$$

Our reproduction number is represented by $R_o = \rho(FV^{-1})$

$$V^{-1} = \frac{1}{\det V} V^T \text{ Where } V^T = \begin{bmatrix} k_3 & 0 \\ \gamma & K_2 \end{bmatrix} \tag{31}$$

$$V^{-1} = \frac{1}{K_2 k_3} \begin{pmatrix} k_3 & 0 \\ \gamma & K_2 \end{pmatrix} = \begin{pmatrix} \frac{k_3}{K_2 k_3} & 0 \\ \frac{\gamma}{K_2 k_3} & \frac{K_2}{K_2 k_3} \end{pmatrix} \tag{32}$$

$$\det V = K_2 k_3 \tag{33}$$

$$(FV^{-1}) = \begin{bmatrix} 0 & \frac{\beta s}{N} \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{K_2} & 0 \\ \frac{\gamma}{K_2 k_3} & \frac{1}{k_3} \end{bmatrix} \tag{34}$$

$$(FV^{-1}) = \begin{bmatrix} \frac{\beta s}{NK_2 k_3} \\ 0 \end{bmatrix} \tag{35}$$

Now we the eigenvalues $|(FV^{-1}) - \lambda I| = 0$ we have,

$$\begin{bmatrix} 0 - \lambda & \frac{\beta s}{NK_2 k_3} \\ 0 & 0 \end{bmatrix} = 0 \tag{36}$$

$$\left(\frac{\beta s \gamma}{NK_2 k_3} - \lambda \right) (0 - \lambda) = 0 \tag{37}$$

$$-\frac{\beta s \gamma \lambda}{NK_2 k_3} + \lambda^2 = 0 \tag{38}$$

$$\lambda \left(\lambda - \frac{\beta s \gamma}{NK_2 k_3} \right) = 0 \tag{39}$$

$$\Rightarrow \lambda_1 = 0 \text{ And } \lambda_2 = \frac{\beta s \gamma}{NK_2 k_3} \tag{40}$$

Therefore the value $\frac{\beta s \gamma}{NK_2 k_3}$ is our \mathfrak{R}_0 and is the effective reproduction number since there is the presence of control strategies.

2.5 Existence of Endemic Equilibrium Point (EEP)

Let $\lambda = \frac{\beta I}{N}$ and

Setting system (5), (6), (7) and (8) to zero, the system becomes

$$\Lambda - \lambda S + \omega R - K_1 S = 0 \tag{41}$$

$$\lambda S - k_2 C = 0 \tag{42}$$

$$\gamma C - k_3 I = 0 \tag{43}$$

$$\tau I + \rho S - k_4 R = 0 \tag{44}$$

From system (41), (42) and (43) we have

$$C = \frac{\lambda S}{K_2} \tag{45}$$

$$I = \frac{\gamma C}{K_3} \tag{46}$$

$$R = \frac{\tau I + \rho S}{K_4} \tag{47}$$

Substitute system (45) in (46) we have

$$I = \frac{\gamma \left(\frac{\lambda S}{K_2} \right)}{K_3} = \frac{\gamma \lambda S}{K_2 K_3} \tag{48}$$

Substitute system (48) in (47) we have

$$R = \frac{\tau \left(\frac{\gamma \lambda S}{K_2 K_3} \right) + \rho S}{K_4} = \frac{\tau \gamma \lambda S + K_2 K_3 \rho S}{K_4 K_2 K_3} = \frac{(\tau \gamma \lambda + K_2 K_3 \rho) S}{K_2 K_3 K_4}$$

$$R = \frac{(\tau \gamma \lambda + K_2 K_3 \rho) S}{K_2 K_3 K_4} \tag{49}$$

From system (41) we can find S,

$$\Lambda + \omega R - (\lambda + K_1) S = 0$$

$$S = \frac{\Lambda + \omega R}{(\lambda + K_1)} \tag{50}$$

Substitute system (49) in (50) to solve for S

$$S = \frac{\Lambda + \omega \left(\frac{(\tau \gamma \lambda + K_2 K_3 \rho) S}{K_2 K_3 K_4} \right)}{(\lambda + K_1)} \tag{51}$$

$$(\lambda + K_1) S = \Lambda + \left(\frac{\tau \gamma \lambda + K_2 K_3 \rho}{K_2 K_3 K_4} \right) \omega S \tag{52}$$

$$\left((\lambda + K_1) - \left(\frac{\tau \gamma \lambda + K_2 K_3 \rho}{K_2 K_3 K_4} \right) \omega \right) S = \Lambda \tag{53}$$

$$S = \frac{\Lambda}{\frac{\lambda K_2 K_3 K_4 + K_1 K_2 K_3 K_4 - \tau \gamma \lambda \omega - K_2 K_3 \rho \omega}{K_2 K_3 K_4}} \tag{54}$$

$$S = \frac{\Lambda K_2 K_3 K_4}{\lambda K_2 K_3 K_4 + K_1 K_2 K_3 K_4 - \tau \gamma \lambda \omega - K_2 K_3 \rho \omega} \tag{55}$$

$$S = \frac{\Lambda K_2 K_3 K_4}{K_2 K_3 (\lambda K_4 + K_1 K_4 - \rho \omega) - \tau \gamma \lambda \omega} \tag{56}$$

Substitute system (56) in (45) to solve for C

$$C = \frac{\lambda S}{K_2} \tag{57}$$

$$C = \frac{\lambda}{K_2} \left(\frac{\Lambda K_2 K_3 K_4}{K_2 K_3 (\lambda K_4 + K_1 K_4 - \rho \omega) - \tau \gamma \lambda \omega} \right) \tag{58}$$

$$C = \left(\frac{\Lambda \lambda K_3 K_4}{K_2 K_3 (\lambda K_4 + K_1 K_4 - \rho \omega) - \tau \gamma \lambda \omega} \right) \tag{59}$$

Substitute system (59) in (46) to solve for I

$$I = \frac{\gamma C}{K_3} \tag{60}$$

$$I = \frac{\gamma}{K_3} \left(\frac{\Lambda \lambda K_3 K_4}{K_2 K_3 (\lambda K_4 + K_1 K_4 - \rho \omega) - \tau \gamma \lambda \omega} \right) \tag{61}$$

$$I = \left(\frac{\Lambda \lambda \gamma K_4}{K_2 K_3 (\lambda K_4 + K_1 K_4 - \rho \omega) - \tau \gamma \lambda \omega} \right) \tag{62}$$

Substitute system (56), in (49) to solve for R

$$R = \frac{(\tau \gamma \lambda + K_2 K_3 \rho) S}{K_2 K_3 K_4} \tag{49}$$

$$R = \frac{(\tau \gamma \lambda + K_2 K_3 \rho)}{K_2 K_3 K_4} \left(\frac{\Lambda K_2 K_3 K_4}{K_2 K_3 (\lambda K_4 + K_1 K_4 - \rho \omega) - \tau \gamma \lambda \omega} \right) \tag{63}$$

$$R = \frac{\Lambda (\tau \gamma \lambda + K_2 K_3 \rho)}{K_2 K_3 (\lambda K_4 + K_1 K_4 - \rho \omega) - \tau \gamma \lambda \omega} \tag{64}$$

$$\begin{pmatrix} S \\ C \\ I \\ R \end{pmatrix} = \begin{bmatrix} \frac{\Lambda K_2 K_3 K_4}{K_2 K_3 (\lambda K_4 + K_1 K_4 - \rho \omega) - \tau \gamma \lambda \omega} \\ \frac{\Lambda \lambda K_3 K_4}{K_2 K_3 (\lambda K_4 + K_1 K_4 - \rho \omega) - \tau \gamma \lambda \omega} \\ \frac{\Lambda \lambda \gamma K_4}{K_2 K_3 (\lambda K_4 + K_1 K_4 - \rho \omega) - \tau \gamma \lambda \omega} \\ \frac{\Lambda (\tau \gamma \lambda + K_2 K_3 \rho)}{K_2 K_3 (\lambda K_4 + K_1 K_4 - \rho \omega) - \tau \gamma \lambda \omega} \end{bmatrix} \tag{65}$$

The above system (95) is the required endemic equilibrium point (EEP).

2.6 Disease Free Equilibrium (DFE)

In the equilibrium state we let

$$\frac{dS}{dt} = \frac{dC}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

Which is the same as system (8), (9),(20) and (21)

$$\Lambda - \frac{\beta IS}{N} + \omega R - K_1 S = 0$$

$$\frac{\beta IS}{N} - k_2 C = 0$$

$$\gamma C - k_3 I = 0$$

$$\tau I + \rho S - k_4 R = 0$$

From system (9) we have

$$\frac{\beta IS - Nk_2 C}{N} = 0 \Rightarrow \beta IS - Nk_2 C = 0 \tag{66}$$

$$\beta IS = Nk_2 C \Rightarrow S = \frac{Nk_2 C}{\beta I} \tag{67}$$

From system (20) we have

$$\gamma C - k_3 I = 0 \Rightarrow k_3 I = \gamma C \tag{68}$$

$$I = \frac{\gamma C}{k_3} \tag{69}$$

Now substitute system (69) in (67) we have

$$S = \frac{Nk_2 C}{\beta \left(\frac{\gamma C}{k_3}\right)} = \frac{Nk_2 C}{\beta \gamma C k_3} \tag{70}$$

$$S = \frac{Nk_2}{\beta \gamma k_3} \tag{71}$$

Substitute system (69) and (71) in (9) to find C

$$\frac{\beta IS}{N} - k_2 C = 0 \tag{72}$$

$$\frac{\beta \left(\frac{\gamma C}{k_3}\right) \left(\frac{Nk_2}{\beta \gamma k_3}\right)}{N} - k_2 C = 0 \tag{73}$$

$$\frac{\beta \left(\frac{\gamma C}{k_3}\right) \left(\frac{Nk_2}{\beta \gamma k_3}\right) - Nk_2 C}{N} = 0 \Rightarrow \frac{N\gamma C k_2}{\gamma k_3^2} - Nk_2 C = 0 \tag{74}$$

$$\frac{N\gamma C k_2 - \gamma k_3^2 Nk_2 C}{\gamma k_3^2} = 0 \tag{75}$$

$$N\gamma C k_2 - \gamma k_3^2 Nk_2 C = 0 \tag{76}$$

$$C(N\gamma k_2 - \gamma k_3^2 Nk_2) = 0 \tag{77}$$

By dividing through by $N\gamma k_2 - \gamma k_3^2 Nk_2$ we have

$$C = 0 \tag{78}$$

From system (69) we substitute $C = 0$ to find I

$$I = \frac{\gamma C}{k_3}$$

We have that $I = 0$

$$I = 0 \tag{79}$$

From system (9) we can find R by substituting system (71) and (9)

$$\tau I + \rho S - k_4 R = 0 \tag{80}$$

$$R = \frac{\tau I + \rho S}{k_4} \tag{81}$$

$$R = \frac{0 + \rho \left(\frac{Nk_2}{\beta \gamma k_3}\right)}{k_4} \tag{82}$$

$$R = \frac{\rho Nk_2}{\beta \gamma k_3 k_4} \tag{83}$$

$$R = \frac{\rho Nk_2 k_4}{\beta \gamma k_3} \tag{84}$$

$$\begin{pmatrix} S^* \\ C^* \\ I^* \\ R^* \end{pmatrix} = \begin{bmatrix} \frac{Nk_2}{\beta \gamma k_3} \\ 0 \\ 0 \\ \frac{\rho Nk_2 k_4}{\beta \gamma k_3} \end{bmatrix} \tag{85}$$

The above system (85) is the required disease free equilibrium point (DFE).

2.7 Local Stability Analysis of Disease Free Equilibrium (DFE)

Theorem: the disease free equilibrium point is locally asymptotically stable if $\mathfrak{R}_0 < 1$ and unstable if $\mathfrak{R}_0 > 1$.

Proof: to proof this theorem we first obtain the Jacobean matrix of the model equations at the disease free equilibrium.

Let

$$\frac{dS}{dt} = \Lambda - \frac{\beta IS}{N} + \omega R - K_1 S = f_1 \tag{86}$$

$$\frac{dC}{dt} = \frac{\beta IS}{N} - k_2 C = f_2 \tag{87}$$

$$\frac{dI}{dt} = \gamma C - k_3 I = f_3 \tag{88}$$

$$\frac{dR}{dt} = \tau I + \rho S - k_4 R = f_4 \tag{89}$$

The Jacobean matrix for the model equation is given by

$$J(S, C, I, R) = \begin{bmatrix} -\frac{\beta I}{N} - K_1 & 0 & 0 & \omega \\ \frac{\beta I}{N} & -K_2 & 0 & 0 \\ 0 & \gamma & -K_3 & 0 \\ \rho & 0 & \tau & -K_4 \end{bmatrix} \tag{90}$$

At DFE we have

$$\begin{bmatrix} -K_1 & 0 & 0 & \omega \\ 0 & -K_2 & 0 & 0 \\ 0 & \gamma & -K_3 & 0 \\ \rho & 0 & \tau & -K_4 \end{bmatrix} \tag{91}$$

To find the eigenvalues we have $|J - \lambda I| = 0$

$$\begin{bmatrix} -K_1 - \lambda & 0 & 0 & \omega \\ 0 & -K_2 - \lambda & 0 & 0 \\ 0 & \gamma & -K_3 - \lambda & 0 \\ \rho & 0 & \tau & -K_4 - \lambda \end{bmatrix} \tag{92}$$

$$(-K_1 - \lambda) \begin{vmatrix} -K_2 - \lambda & 0 & 0 \\ \gamma & -K_3 - \lambda & 0 \\ 0 & \tau & -K_4 - \lambda \end{vmatrix} - 0 + 0 + (\omega) \begin{vmatrix} 0 & -K_2 - \lambda & 0 \\ \rho & 0 & -K_3 - \lambda \end{vmatrix} = 0 \tag{93}$$

$$(-K_1 - \lambda)[(-K_2 - \lambda)((-K_3 - \lambda)(-K_4 - \lambda) - 0)] - (\omega)[(-K_2 - \lambda)((0 - \rho(-K_3 - \lambda))] = 0 \tag{94}$$

$$(-K_1 - \lambda)[(-K_2 - \lambda)(-K_3 - \lambda)(-K_4 - \lambda)] + (\omega\rho)[(-K_2 - \lambda)(-K_3 - \lambda)] = 0 \tag{95}$$

$$(-K_2 - \lambda)(-K_3 - \lambda)[(-K_1 - \lambda)(-K_4 - \lambda) + \omega\rho] = 0 \tag{96}$$

$$\Rightarrow \lambda_1 = -K_2 < 0, \lambda_2 = -K_3 < 0 \tag{97}$$

The remaining eigenvalues can be resolved from:

$$K_1 K_4 + K_1 \lambda + K_4 \lambda + \lambda^2 + \omega\rho = 0 \tag{98}$$

$$\lambda^2 + (K_1 + K_4)\lambda + K_1 K_4 + \omega\rho = 0 \tag{99}$$

$$\rho(\lambda) = \lambda^2 + (K_1 + K_4)\lambda + K_1 K_4 + \omega\rho = 0 \tag{100}$$

Using Routh-Horwitz criterion,

$$\rho(\lambda) = a_0 \lambda^2 + a_1 \lambda + a_2 \tag{101}$$

$$a_0 > 0, a_1 > 0, a_2 > 0 \tag{102}$$

Compare with system (100) we have,

$$a_0 = 1 > 0, \tag{103}$$

$$a_1 = K_1 + K_4 > 0, \tag{104}$$

$$a_2 = K_1 K_4 + \omega\rho > 0 \tag{105}$$

From Routh-Horwitz criterion for $\rho(\lambda)$ to have negative root all the coefficients must be greater than zero.

$$a_0 > 0, a_1 > 0, a_2 > 0$$

Therefore $\lambda_3 < 0$ and $\lambda_4 < 0$.

Now we conclude that the DFE is locally asymptotically stable, since

$$\begin{aligned} \lambda_1 &< 0, \\ \lambda_2 &< 0, \\ \lambda_3 &< 0 \\ \lambda_4 &< 0. \end{aligned}$$

Hence, the disease free equilibrium (DFE) is locally asymptotically stable.

2.8 Numerical Simulations

The model equations (1) to (4) were numerically simulated using the defined parameters presented in the table of variables and parameters (table 1.0) we will vary the key parameters to investigate the impact

of varying infection rate on the number of infected individuals by different rate of treatment and impact of susceptible individuals by different rate of vaccination to study the transmission dynamics of bacterial meningitis inspired by Elmojtaba and Adam (2017).

3.0 Results and Discussion

Table 1: Variables and Parameter Values

PARAMETERS	DIFINITION	VALUES	REFERENCE
Λ	Rate of recruitment	100	Assumed
μ	Natural death	0.02	C.L. Trotter 2010
β	Rate of transmission	0.88	K.Vereen 2008
γ	Rate of progression	0.52	C.L. Trotter 2012
τ	Rate of treatment	0.9	Assumed
δ	Disease induced rate	0.5	WHO 2010
ρ	R vaccination	0.85	M. J. F. Martinez 2013.
ω	Whining rate	0.04	C.L. Trotter, 2012
S	Susceptible class	700	Assumed
C	Carriers class	250	Assumed
I	Infected class	40	Assumed
R	Recovered class	10	Assumed

Table 2: Time rate and Susceptible class

T	0	2	4	6	8	10
S	0	12	25	50	200	700

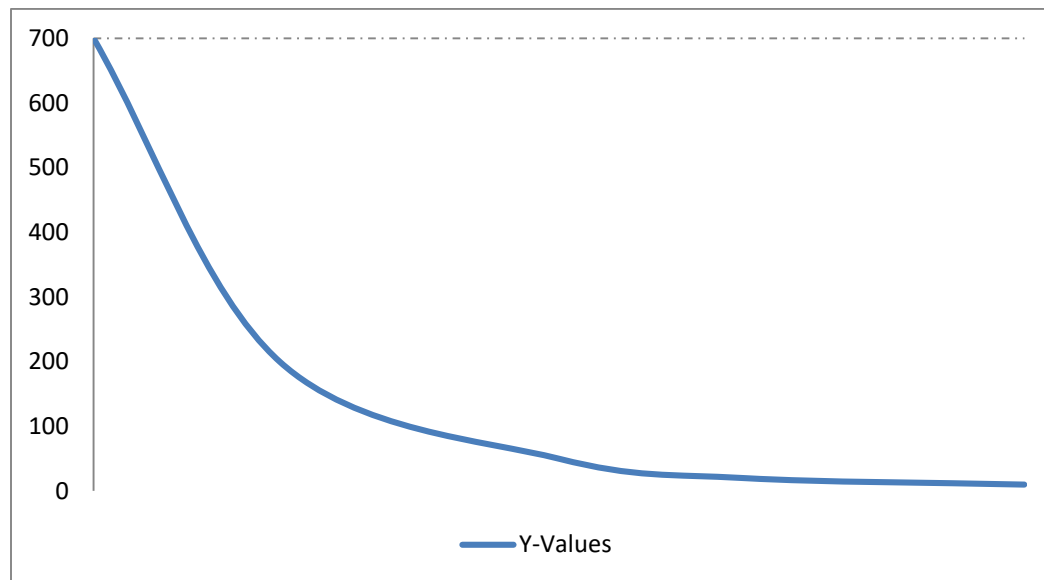


Figure 1: Susceptible class against time

This figure above show that the susceptible individuals reduce due to vaccination and the awareness of the disease to the population.

From model 1, we now solve for the susceptible individuals for the period of ten years to get another results.

From equation 1

$$\frac{ds}{dt} = \Lambda - \frac{\beta IS}{N} + \omega R - (\mu + \rho)S$$

Integrating we get

$$\int ds = \int \left(\Lambda - \frac{\beta IS}{N} + \omega R - (\mu + \rho)S \right) dt$$

$$S(t) = \left[\Lambda - \frac{\beta IS}{N} + \omega R - (\mu + \rho)S \right] t$$

t=0

$$s(0) = \left[100 - \frac{0.88(40)(0)}{1000} + 0.04(0) - (0.02 + 0.85)0 \right] * 0$$

$$= \left[100 - \frac{0}{1000} + 0 - (0.87)0 \right] * 0$$

$$= 0$$

When t=2

$$s(2) = \left[100 - \frac{0.88(100)(12)}{1000} + 0.04(60) - (0.02 + 0.85)12 \right] * 2$$

$$= \left[100 - \frac{105.6}{1000} + 2.4 - (0.87)12 \right] * 12$$

$$= [100 - 0.1056 + 2.4 - 10.44] * 12$$

$$= 1102.2528$$

$$s(4) = \left[100 - \frac{0.88(8)(25)}{1000} + 0.04(80) - (0.02 + 0.85)25 \right] * 4$$

$$= \left[100 - \frac{176}{1000} + 3.2 - (0.87)25 \right] * 4$$

$$= [100 - 0.176 + 3.2 - 21.75] * 4$$

$$= 325.096$$

$$s(6) = \left[100 - \frac{0.88(6)(50)}{1000} + 0.04(100) - (0.02 + 0.85)50 \right] * 6$$

$$= [100 - 0.264 + 4 - 43] * 6$$

$$= 364.416$$

$$s(8) = \left[100 - \frac{0.88(4)(200)}{1000} + 0.04(120) - (0.02 + 0.85)200 \right] * 8$$

$$= [100 - 0.704 + 4.8 - 170] * 8$$

$$= -1255.904$$

$$s(10) = \left[100 - \frac{0.88(2)(700)}{1000} + 0.04(140) - (0.02 + 0.85)700 \right] * 10$$

$$= [100 - 1.232 + 5.6 - (0.87)700] * 10$$

$$= [-504.632] * 10$$

$$= -5046.32$$

Based on the values we got from the above solution, we can see that at the initial years of treatment that is from (0, 2, 4, 6) is positive values to shows that the treatment of the susceptible individuals is not stable, and also after two years that is (8, 10,..) is negative values is also to shows that the susceptible individuals are responding to treatment at a stable state. (Although there are chances of reinfection).

Table 3: Population Carriers

T	0	2	4	6	8	10
C	240	220	200	180	160	140

The figure below shows that the population of carriers class individual decreases with respect to time.

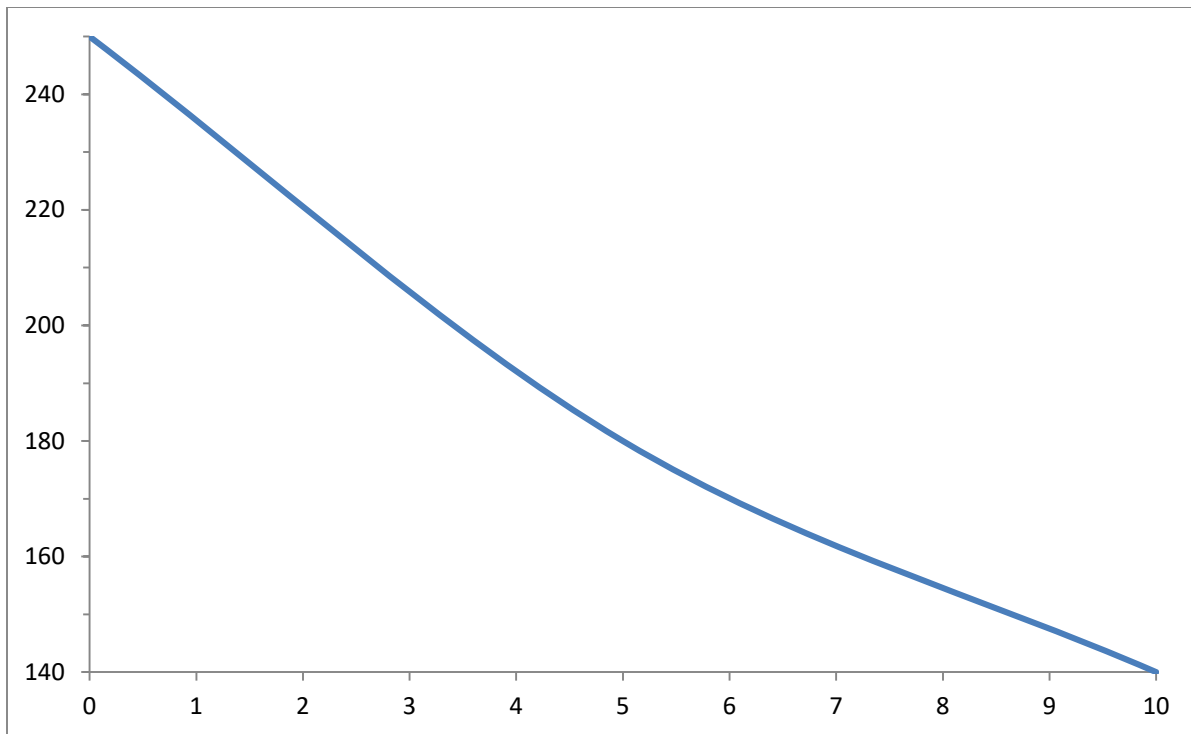


Figure 2: Carriers' class against time

$$\begin{aligned} \frac{dc}{dt} &= \frac{\beta IS}{N} - (\gamma + \mu)c \\ \int \frac{dc}{dt} &= \int \left[\frac{\beta IS}{N} - (\gamma + \mu)c \right] dt \\ \int dc &= \int \left[\frac{\beta IS}{N} - (\gamma + \mu)c \right] dt \\ c(t) &= \left[\frac{\beta IS}{N} - (\gamma + \mu)c \right] dt \\ c(t) &= \left[\frac{\beta IS}{N} - (\gamma + \mu)c \right] dt \end{aligned}$$

When $t = 0$

$$c(0) = \left[\frac{0.88(40)(0)}{100} - (0.52 + 0.02)240 \right] * 0$$

$$\begin{aligned} &= [0 - 129.6] * 0 \\ &= 0 \end{aligned}$$

When $t = 2$

$$c(2) = \left[\frac{0.88(10)(12)}{1000} - (0.52 - 0.02)220 \right] * 2$$

$$\begin{aligned} &= [0.1056 - 118.8] * 2 \\ &= 235.7888 \end{aligned}$$

When $t = 4$

$$c(4) = \left[\frac{0.88(8)(25)}{1000} - (0.52 + 0.02)200 \right] * 4$$

$$\begin{aligned} &= [0.176 - 108] * 4 \\ &= -31.296 \end{aligned}$$

When $t = 6$

$$\begin{aligned}
 c(6) &= \left[\frac{0.88(6)(50)}{1000} - (0.52 + 0.02)180 \right] * 6 \\
 &= [0.264 - 97.2] * 6 \\
 &= -581.616 \\
 \text{When } t &= 8 \\
 c(8) &= \left[\frac{0.88(4)(200)}{1000} - (0.52 + 0.02)(160) \right] * 8 \\
 &= [0.704 - 86.4] * 8 \\
 &= -685.568 \\
 \text{When } t &= 10 \\
 c(10) &= \left[\frac{0.88(2)(700)}{1000} - (0.52 + 0.02)140 \right] * 10 \\
 &= [1.232 - 75.6] * 10 \\
 &= -743.68
 \end{aligned}$$

The results we obtained from the carrier group has a unique character which increases from the years (0 - 2), but later starts to decrease with the time due to treatment

This figure show that the population of infected individuals decays with respect to time due to treatment which results of progression into recovered compartment.

Table 3: Population of infected individuals

T	0	2	4	6	8	10
I	40	10	8	6	4	2

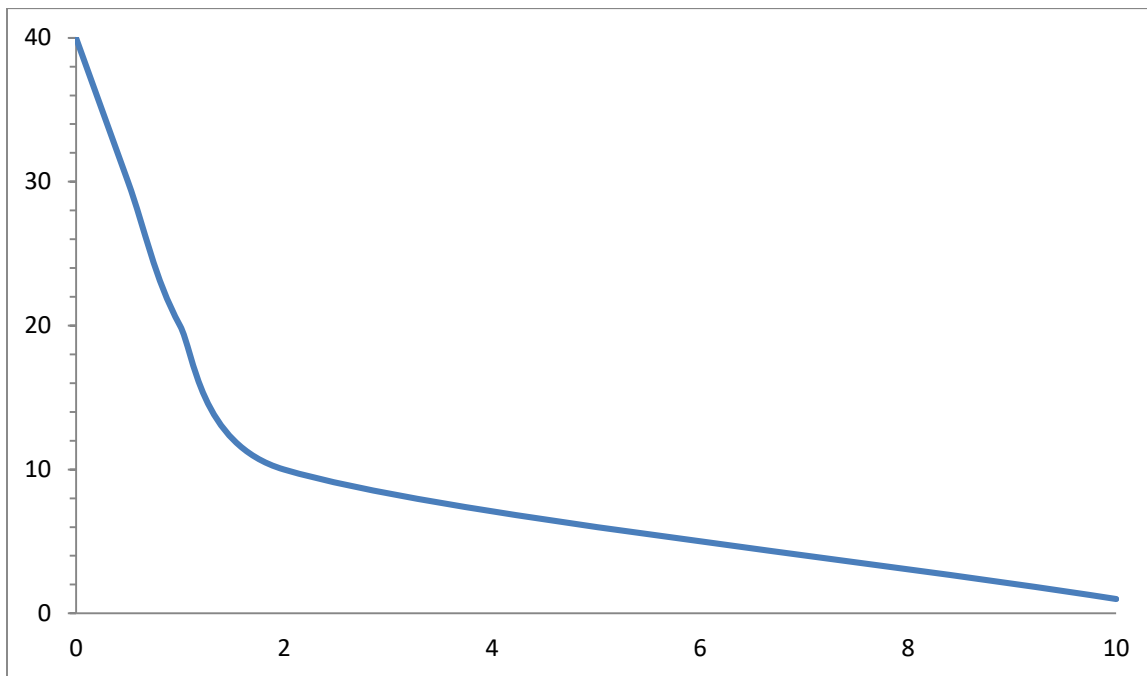


Figure 3: Infected class against time

From the above model we the values. We obtain to shows the effect of varying treatment rate on the population infected and their progression to recovered compartment.

$$\begin{aligned}
 \frac{dI}{dt} &= \gamma c - (\tau + \delta + \mu)I \\
 \int \frac{dI}{dt} &= \int [\gamma c - (\tau + \delta + \mu)] dt
 \end{aligned}$$

$$\int dt = \int [\gamma c - (\tau + \delta + \mu)I] dt$$

$$I(t) = [rc - (\tau + \delta + \mu)I]t$$

When $t = 0$

$$I(t) = [\gamma c - (\tau + \delta + \mu)I]t$$

$$I(0) = [0.52(240) - (0.9 + 0.5 + 0.02)40] * 0$$

$$= [124.8 - 56.8] * 0$$

$$= 68 * 0$$

= 0

When $t = 2$

$$I(2) = [0.52(220) - (0.9 + 0.5 + 0.02)10] * 2$$

$$= [114.4 - 1.6] * 2$$

$$= 225.6$$

When $t = 4$

$$I(4) = [0.52(200) - (0.9 + 0.5 + 0.02)8] * 4$$

$$= [104 - 11.36] * 4$$

$$= 370.56$$

When $t = 6$

$$I(6) = [0.52(180) - (0.9 + 0.5 + 0.02)6] * 6$$

$$= [93.6 - 8.52] * 6$$

$$= 510.48$$

When $t = 8$

$$I(8) = [0.52(160) - (0.9 + 0.5 + 0.02)4] * 8$$

$$= [83.2 - 5.68] * 8$$

$$= 620.16$$

When $t = 10$

$$I(10) = [0.52(140) - (0.9 + 0.5 + 0.02)2] * 10$$

$$= [72.8 - 2.84] * 10$$

$$= 699.6$$

From the above model we the values. We obtain to shows the effect of varying treatment rate on the population infected and their progression to recovered compartment.

This figure shows that the population of recovered individuals increase with time as we varies treatment and vaccination rate.

Table 3: Population of recovered individuals

T	0	2	4	6	8	10
R	0	60	80	100	120	140

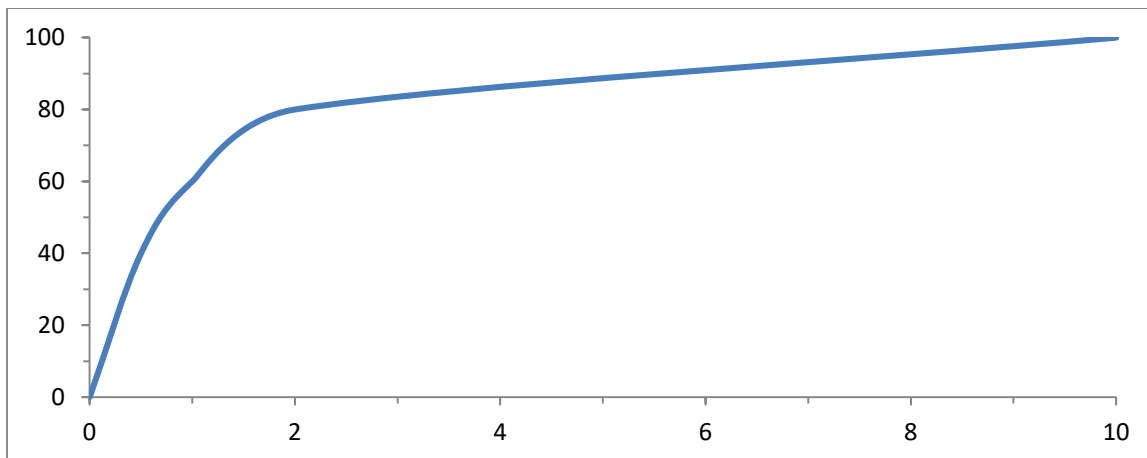


Figure 4: Recovered class against time

From the equation we have

$$\begin{aligned} \frac{dR}{dt} &= \tau I + \rho S - (\omega + \mu)R \\ \int \frac{dR}{dt} &= \int [\tau I + \rho S - (\omega + \mu)R] \mu \\ \int dR &= \int [\tau I + \rho S - (\omega + \mu)R] dt \\ R(t) &= [\tau I + \rho S - (\omega + \mu)R]t \end{aligned}$$

When $t = 0$

$$\begin{aligned} R(0) &= [0.9(10) + 0.85(0) - (0.04 + 0.02)0] * 0 \\ &= [36 + 0 - 0] * 0 \\ &= 0 \end{aligned}$$

When $t = 2$

$$\begin{aligned} R(2) &= [0.9(10) + 0.85(10) - (0.04 + 0.02)60] * 2 \\ &= [9 + 10.2 - 3.6] * 2 \\ &= [15.6] * 2 \\ &= 31.2 \end{aligned}$$

When $t = 4$

$$\begin{aligned} R(4) &= [0.9(8) + 0.85(25) - (0.04 + 0.02)80] * 4 \\ &= [7.2 + 21.25 - 4.8] * 4 \\ &= 94.6 \end{aligned}$$

When $t = 6$

$$\begin{aligned} R(6) &= [0.9(6) + 0.85(50) - (0.04 + 0.02)100] * 6 \\ &= [5.4 + 42.5 - 6] * 6 \\ &= 251.4 \end{aligned}$$

When $t = 8$

$$\begin{aligned} R(8) &= [0.9(4) + 0.85(200) - (0.04 + 0.02)100] * 8 \\ &= [3.6 + 170 - 7.2] * 8 \\ &= 1331.2 \end{aligned}$$

When $t = 10$

$$\begin{aligned} R(10) &= [0.9(2) + 0.85(700) - [0.04 + 0.02]140] * 10 \\ &= [1.8 + 595 - 8.4] * 10 \\ &= 5884 \end{aligned}$$

According to the values obtained in this model we can see that the population of infected individuals increase for a while and then later decrease after some time by undergoing treatment.

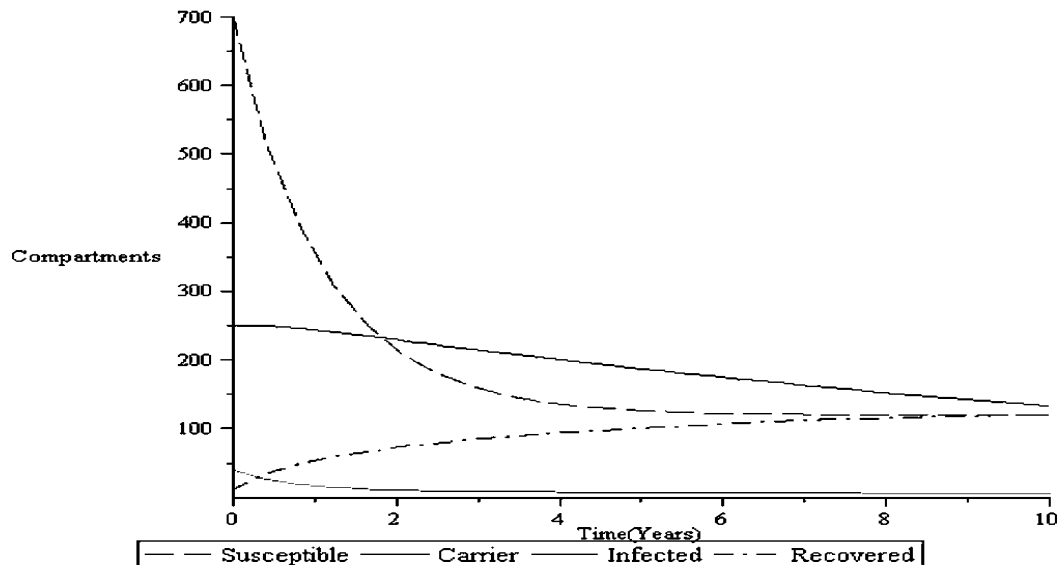


Figure 5: Four compartments against time

This figure show that the population of infected individuals decreases with time due to treatment, the susceptible population is reduce due to vaccination rate, the carriers population is also reduce, while the recovered population will increase higher due to treatment and vaccination strategies.

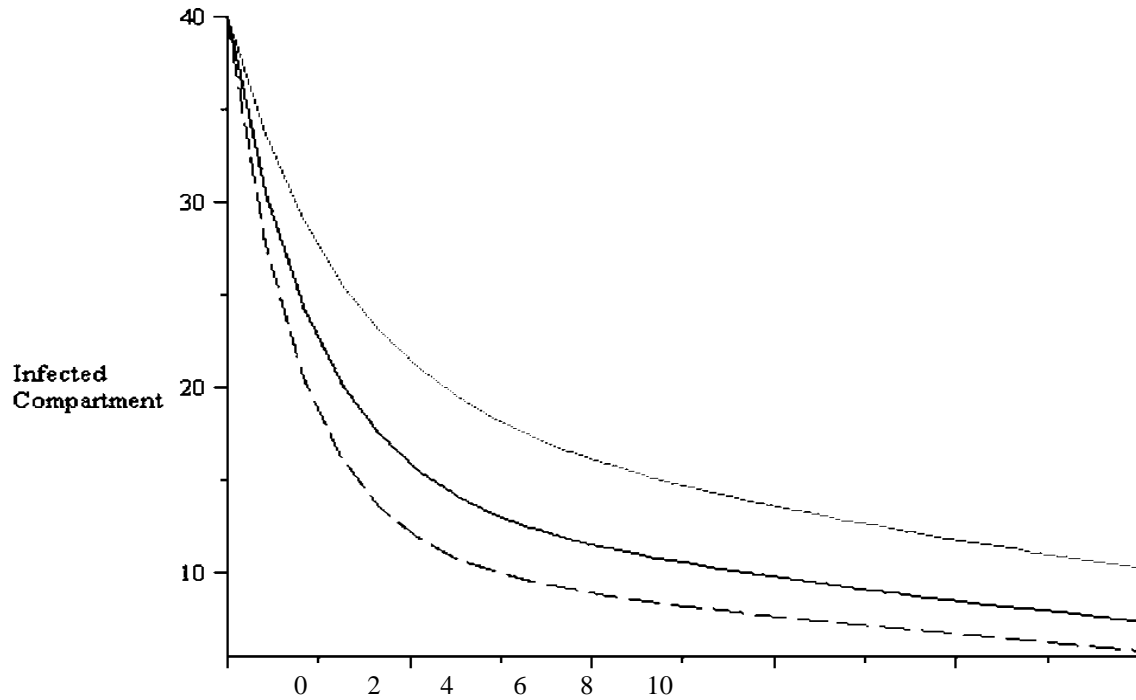


Figure 6: Effect of varying treatment rate on the infected compartment

The above figure shows that the higher the rate of treatment, the lower the number of infected individuals. This shows that treatment plays a vital role in reducing the disease burden in a population.

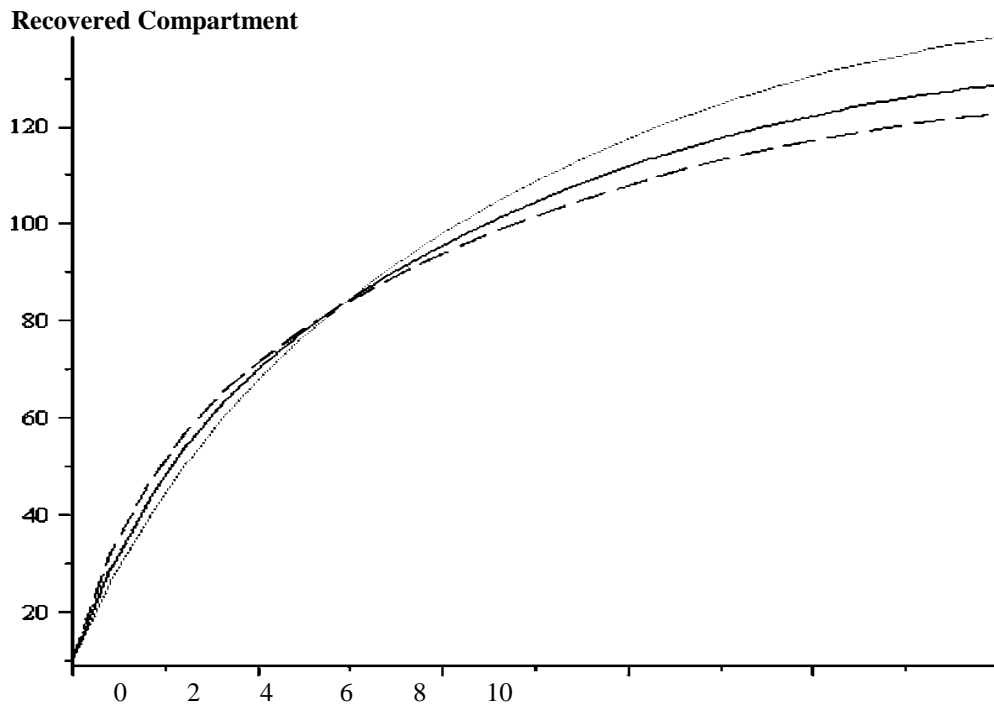


Figure 7: Effect of varying treatment rate on the recovered compartment

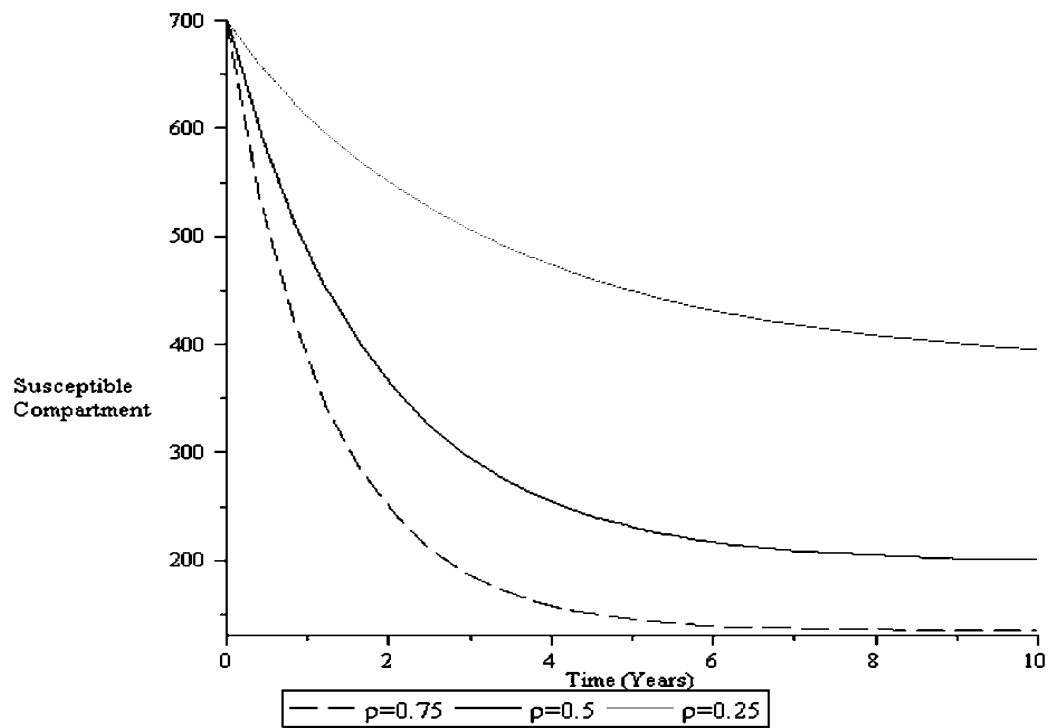


Figure 8: Effect of varying vaccination rate on susceptible compartment

The above figure shows that the higher the vaccination rate, the lower the number of susceptible individuals. This is because when susceptible individual progress to recovered compartment.

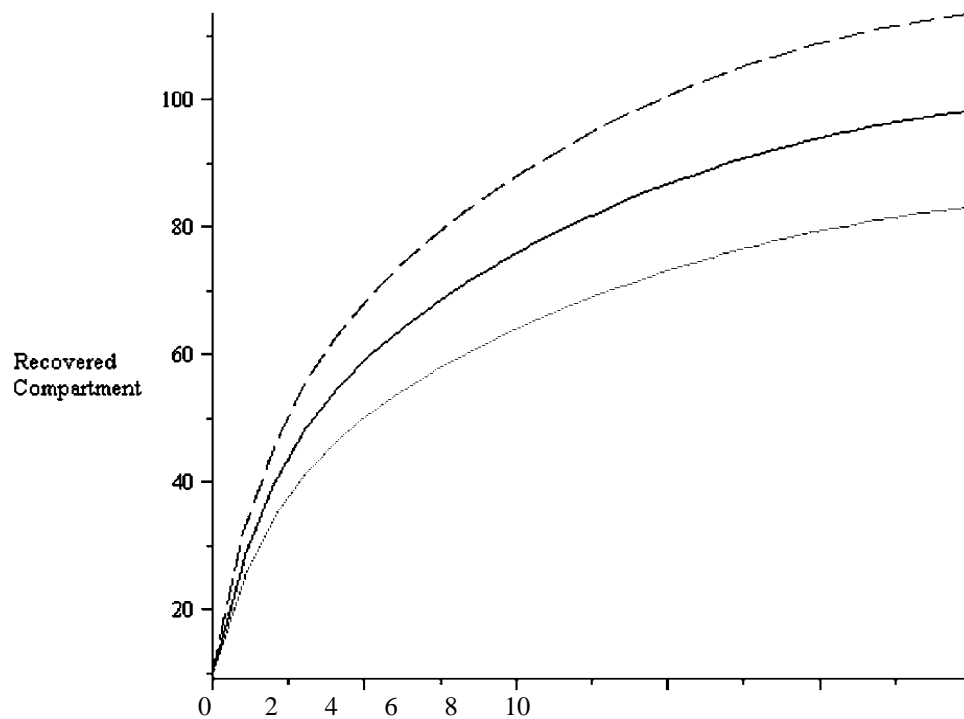


Figure 9: Effect of varying vaccination rate on recovered compartment

The above figure shows that the population of recovered class increases by the higher rate of vaccination. This shows that vaccination plays a vital role in the control and elimination of the disease in a population.

4.0 Conclusion

In this study, a mathematical model for the dynamics of bacterial meningitis was developed and designed to investigate the transmission dynamics of meningitis in a population. The invariant region, basic reproduction number, endemic equilibrium point (EEP), and local stability analysis of the disease free equilibrium (DFE) were analyzed. The basic reproduction number was obtained using the Jacobean matrix method. The analysis revealed that the disease free equilibrium is locally asymptotically stable for $R_0 < 1$. and the endemic equilibrium point is locally asymptotically stable for $R_0 > 1$. Most likeable of this research is that the research work shows that the rate at which treatment and vaccination rate increases to the higher value the recovered compartment increases to the peak point. This means treatment and vaccination has an impact on reducing the case of bacterial meningitis in a population. Now we conclude that a high infection transmission rate requires a high vaccine and treatment rate, which is similar to the findings of Vereen, (2008) on the effect of vaccination against meningitis.

Declarations

Ethics approval and consent to participate

Not Applicable

Consent for publication

All authors have read and consented to the submission of the manuscript.

Availability of data and material

Not Applicable.

Competing interests

All authors declare no competing interests.

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