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MATHEMATICAL MODEL ON THE TRANSMISSION DYNAMICS OF MENINGOCOCCAL MENINGITIS WITH VACCINATION

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Abstract

This paper presents a mathematical model for the transmission dynamics of meningococcal meningitis within a population. The model incorporates the key epidemiological and biological features of meningococcal meningitis, such as the vaccine efficacy and also investigate the impacts of mass vaccination with waning immunity in the population. The model is governed by six (6) system of differential equations namely: Susceptible individuals, vaccinated individuals, carrier individuals, infectious individuals, recovery without disability and recovery with disability. The disease free equilibrium point, endemic equilibrium point, basic reproduction number, R_0***, local stability of the disease free equilibrium using Routh-Hurwitz criterion for stability analysis and global stability of the disease free equilibrium using Castillo-Chavez criterion were obtained. It was observed that the DFE is locally asymptotically stable when*** $R_0 < 1$ ***and globally asymptotically stable when*** $R_0 \leq 1$. ***The analytical results were supported by numerical simulations, which further suggested that the control of the transmission dynamics of meningococcal meningitis passed through an impact of large vaccination coverage and the production of vaccine with a high level of efficacy.***

Keywords: Vaccination, meningococcal meningitis, waning immunity, disability

1.0 Introduction

Meningitis is an inflammation of the protective membrane covering the brain and spinal cord known as the meninges. The inflammation is usually activated by infections of the fluid surrounding the brain and spinal cords. The purpose of the meninges and the cerebrospinal fluid is to protect and provide nourishment to the central nervous system [1]. The most common organisms that causes meningitis are bacterial, viral and fungal. Bacterial organism that enter the brain also occur when bacterial directly invade the meninges causes ear infection, skull fracture. Several strains of bacteria can causes acute meninges, it includes; neisseria meningitis serogroup A, B and C (meningococcus) haemophilus influenzae (Haemophitus) and listeria mono cytogeneses (Listeria) [2]. Viral organism is usually mild and often clears on its own. Fungal is relatively and can cause chronic meningitis. It may mimic acute bacterial meningitis. Fungal meningitis is not contagious from person to person. cryptococcus is a common fungal form of the disease that affect people with immune deficiencies such as AIDS, it life threatening if not treated with an antifungal medications [3]. Meningococcal meningitis is a leading cause of bacterial meningitis and other serious infection worldwide. Meningococcal bacterial are the most common causes of meningococcal diseases in Europe, America, Australia, New Zealand [4]. Africa cases meningococcal meningitis tend to occur more frequently in winter and spring. The highest rates of disease incidence are recorded in the meningitis belt of sub- Sahara Africa [5, 6]. In the year 2009 epidemics season, 14 African countries reported 78,416 suspected cases and 4053 deaths, this is the largest reported number since the 1996 epidemics [5]. More than 85% of these cases were reported to one epidemic foci, encompassing northern Nigeria and Niger and were characterized by the predominance of meningococcal [6]. One of the most recent epidemics of meningococcal disease occurred in Burkina Faso, Meningitis had spread to 22 district in the country by march 20, 2007, one million people has been vaccinated and it was planned that 3 million more would be vaccinated in the next few days. At that time almost 600 of the 7000 infected died. This particular epidemics was also reported to have reached the democratic republic of Congo (DRC) with 7000 cases, Sudan with 700 cases and Uganda 3000

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cases [7]. One hundred and six local government in Nigeria experience cases of meningitis in April 2017. Those states affected are summed up to nineteen include the FCT with 3959 suspected cases, and 438 deaths recoded, which is about 11.1%. Reactive vaccinations campaign started on 15/4/2017 in Zamfara state with massive turnout in all the targeted communities. A joint teams of NCDC, and private partners were supporting the reactive vaccination campaigns and outbreak response in the state. Meningitis outbreak occurred reported in 19 states with 5 states mostly affected; zamfara, Katsina, Kebbi and Sokoto in the north- west zone and Niger in the central zone of Nigeria. As at 5th April 2017, a total of 3959 cases with 438 deaths have been reported 181 laboratories confirmed case [8].

Bacterial meningitis occurs more often than viral meningitis .In fact more than 80% of all meningitis cases are caused by these three distinct types of bacterial, Neisseria meningitis, haemophilus influenza and streptococcus pneumonia. Neisseria meningitis which causes meningococcus occurs the most frequently of three. About 10% of the population carry the games for days, weeks, or even months without becoming ills, meningococcus carriage is most prevalent in teenagers and young adults, where up to 60% of the carries often recorded [9]. To protect against meningococcal and childhood pneumococcal infections, have your child vaccinated promptly starting at the age of two month, parents should not wait until the child ready to enter school before injection for immunization.. For adolescents, a single dose of meningococcal (conjugate vaccine) mcv is recommended started at age of 11- 12 years [10]. Pregnant women should talk to their doctors or midwife about getting tested for group B. streptococcus, women receives the test when they are 35 to 37 weeks pregnant. Doctors gives antibiotic (during labor) to women who test positive in order to prevent passing meningitis to their newborns. Women should avoid certain food during pregnancy and safely prepare others [11].Once the bacteria are inside the blood, they have the ability to defeat the body's defense mechanism that would normally fight off infections [12]

This research will deal with bacterial meningitis caused by meningococcus, this is a very serious form of meningitis, and it has a high mortality rate if not treated. It has been reported that the fatality rate in developed countries was about 70 -80% before successful treatment were discovered such as antibiotics which causes the rate to drop to 25 % [13]. Transmission of meningococcal meningitis passes through several stages. First, an individual has to be susceptible to the disease when the three main bacterial causes meningococcal meningitis are present in the environment. When a susceptible individual come in to contact with any of these bacteria or another infected individual they can then experience carriage of the bacterium, once they are carrier they can then developed the disease. Carriage meningococcal meningitis does not necessary lead to the development of the disease [13].

2. Mathematical Formulation

In this paper, we divide the populations $N(t)$ in to six compartments: susceptible individuals $S(t)$ those who are susceptible to infection, vaccinated individuals $V(t)$ those who exhibits temporal immunity from the vaccination, carrier individuals $C(t)$ those who carry the infection and can transmit the disease but they have no sign of the infection, infectious individual $I(t)$ those who have infections and exhibits the symptoms and still infect and can transmit, recovered individuals $R_1(t)$ those who have been recovered without disability after treatment and recovered individuals $R_2(t)$ those who have been recovered with disability after treatment.

Individuals are recruited into the susceptible populations $S(t)$ by birth at a constant rate b and decrease by natural death μS . Since the vaccine does not coffer a permanent immunity to all vaccine recipients, vaccinated individuals who lose their immunity with waning of vaccines at a constant rate ωV return to the susceptible populations.

Furthermore, when the susceptible populations come in to contact with either carrier infection class $C(t)$ or infectious class $I(t)$, they will now increase by proportions of susceptible carrier infection $\psi\beta S\frac{(C+I)}{N}$ or by susceptible infectious individuals at a constant rate $(1-\psi)\beta S\frac{(C+I)}{N}$ respectively, where ψ is the proportions of susceptible individuals having carrier infections, β is the rate of transmission and the two populations diminished by natural death μC and μI respectively. Disease induced mortality rate γI only takes place in the infectious populations $I(t)$

Similarly, when the vaccinated individuals interact with either carrier infections or infectious populations, the carrier infections class now will be increase by the proportions of vaccinated individuals $\tau(1-\upsilon)\beta V\frac{(C+I)}{N}$ and diminished by those that developed an invasive disease at a constant rate aC , where a is the carrier infections and natural death μV , while the remaining vaccinated individuals move to the infectious populations at a constant rate $(1-\tau)(1-\upsilon)\beta V\frac{(C+I)}{N}$, where τ is the proportion of vaccinated individuals and υ is the vaccine efficacy, when $\upsilon=1,$ the vaccine is perfectly done and when $\upsilon=0,$ the vaccine has no effect at all. In the infectious class after maybe treatment, some infectious individuals will now

recovered without disability $R_1(t)$ with proportion of recovery without disability $\kappa\pi I$ while some will recover with disability $R_2(t)$ at a constant rate $\kappa(1-\pi)I$, where κ is the rate of specified recovery and π is the proportions of recovery without disability and the two populations will diminish by natural death μR_1 and μR_2 respectively. Finally, infected individuals after recovery may recover with disability.The model diagram for the effect of vaccination on the transmission dynamics of meningococcal meningitis is presented in Figure 2.1.

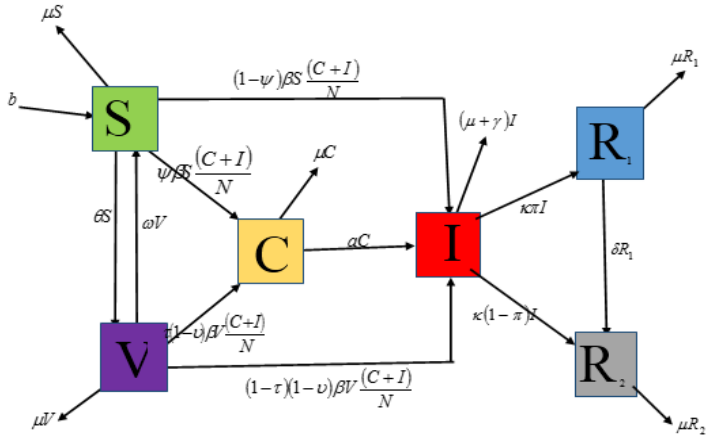

Figure 2.1. Epidemiological diagram for model with vaccination and two recovered classes

2.1 Model equations

$$\left.\begin{aligned}
\frac{dS}{dt} &= b+\omega V-\psi\beta S\frac{(C+I)}{N}-(1-\psi)\beta S\frac{(C+I)}{N}-(\mu+\theta)S\\
\frac{dV}{dt} &= \theta S-(1-\tau)(1-\upsilon)\beta V\frac{(C+I)}{N}-\tau(1-\upsilon)\beta V\frac{(C+I)}{N}-(\mu+\omega)V\\
\frac{dC}{dt} &= \tau(1-\upsilon)\beta V\frac{(C+I)}{N}+\psi\beta S\frac{(C+I)}{N}-(\mu+a)C\\
\frac{dI}{dt} &= (1-\tau)(1-\upsilon)\beta V\frac{(C+I)}{N}+(1-\psi)\beta S\frac{(C+I)}{N}-\kappa\pi I-\kappa(1-\pi)I+aC-(\mu+\gamma)I\\
\frac{dR_1}{dt} &= \kappa\pi I-(\delta+\mu)R_1\\
\frac{dR_2}{dt} &= \kappa(1-\pi)I+\delta R_1-\mu R_2
\end{aligned}\right\} \quad (1)$$

where initial conditions are:

$S(0)=S_0,\ V(0)=V_0,\ C(0)=C_0,\ I(0)=I_0,\ R_1(0)=R_{10},\ R_2(0)=R_{20}$

$N(t)=S(t)+V(t)+C(t)+I(t)+R_t(t)++R_2(t)$

Table 2.1 Variable and parameters of the meningococcal meningitismodel

Symbol	Description
$S(t)$	Susceptible individual who are susceptible to the disease at time t
$C(t)$	Carrier-healthy individuals who carry meningococcal meningitis bacterial and are infected at time t.
$I(t)$	Infectious- infected individuals who shows the symptoms of infection at time t
$R(t)$	Recovered individuals from the disease after treatment and has no specification of recovering time t.
$R_1(t)$	Recovered individuals without disability, these who are recovered after treatment at time t.
$R_2(t)$	Recovered individuals with disability, those who are recovered after treatment time t.

$V(t)$	Vaccinated individual, healthy individual who have been vaccinated acquiring temporal immunity at time t
b	Rate of recruitment in to the susceptible population by birth
β	Rate of transmission
μ	Natural Death
γ	Diseases induce mostly rate
a	Carrier infection of the invasive disease
τ	Proportions of vaccinated individuals that becomes Carrier as a result of vaccine failure
υ	Vaccine efficacy.
π	Proportions of recovery without disability
θ	Rate of vaccine coverage
ω	Rate of warning of vaccine induced immunity
δ	Rate of having disability
κ	Rate of recovery from infectious class
ψ	Proportion of susceptible individuals that becomes carrier

3. Model Analysis

3.1 Positivity of solutions

It is necessary to prove that all solutions of system (1) with positive initial conditions will remain positive for all time $t > 0$.

Theorem 1.

Let $S(0) > 0, V(0) \geq 0, C(0) \geq 0, I(0) \geq 0, R_1(0) \geq 0, R_2 \geq 0$ Then the solutions (S, V, C, I, R_1, R_2) of the system (1) are positive $\forall t \geq 0$.

Proof From the first equation of the system (1), we obtain the inequality expression $N(t) = S(t) + V(t) + C(t) + I(t) + R_1(t) + R_2(t)$

$N(t) \leq \frac{b}{\mu}$

The system will be studied in the following region

$$D_i = \left\{ (S, V, C, I, R_1, R_2) \in R_+^{\,6} : N(t) \leq \frac{b}{\mu} \right\}$$

3.2 Disease free equilibrium

Setting $\frac{dS}{dt} = 0, \frac{dV}{dt} = 0, \frac{dC}{dt} = 0, \frac{dI}{dt} = 0, \frac{dR_1}{dt} = 0, \frac{dR_2}{dt} = 0,$ the disease free equilibrium state (DFE) denoted by E_0 of system (1) is given by

$$E_0 = S(t), V(t), C(t), I(t), R_1(t), R_2(t) = \left(\frac{b(\mu+\omega)}{\mu(\mu+\theta+\omega)}, \frac{\theta b}{\mu(\mu+\theta+\omega)}, 0, 0, 0, 0 \right)$$

3.3 The endemic equilibrium

The endemic equilibrium state for system (1) denoted by $E^* = S^*(t), V^*(t), C^*(t), I^*(t), R_1^*(t), R_2^*$ is

$$\left.\begin{aligned}
S^* &= \frac{N^* b\left\{\beta\left(C^* + I^*\right)(1-\upsilon) + N^*(\mu+\omega)\right\}}{\left\{\beta\left(C^* + I^*\right)(1-\upsilon) + N^*(\mu+\omega)\right\} + \left\{\beta\left(C^* + I^*\right) + N^*(\mu+\theta)\right\} - N^{*2}\theta\omega} \\
V^* &= \frac{N^{*2}\theta b}{\left\{\beta\left(C^* + I^*\right) + N^*(\mu+\theta)\right\}\left\{\beta\left(C^* + I^*\right)(1-\upsilon) + N^*(\mu+\omega)\right\} - N^{*2}\theta\omega} \\
C^* &= \frac{\tau(1-\upsilon)\left(V^* + \psi S^*\right)\beta I^*}{N^*(\mu+a) - \tau(1-\upsilon)\left(V^* - \psi S^*\right)\beta} \\
I^* &= \frac{\left\{(1-\tau)(1-\upsilon)\beta V^* + (1-\psi)\beta S^* + Na\right\}C^*}{N^*\kappa + N^*(\mu+\gamma) - (1-\tau)(1-\upsilon)\beta V^* - (1-\psi)\beta S^*} \\
R_1^* &= \frac{\kappa\pi I^*}{(\delta+\mu)} \\
R_2^* &= \frac{\kappa\left\{\delta + \mu(1-\pi)\right\}I^*}{\mu(\delta+\mu)}
\end{aligned}\right\} \qquad (2)$$

3.4 Model reproduction number

We apply the next generation matrix technique by Diekmann et al. [14] obtain the basic reproduction number, R_0 by considering the infected compartment of the system (1).Let F_i be the rate of appearance of new infections in the i compartment and V_i be the rate of transfer of individuals out of i, given the disease free equilibrium, then R_0 is the spectral radius (the largest eigenvalues) of the next generation matrix denoted by

$$R_0 = \rho FV^{-1}$$

$$R_0 = \frac{\{\tau(1-\upsilon)\beta\theta + \psi\beta(\mu+\omega)\}\{(\kappa+\mu+\gamma)+a\} + (\mu+a)\{(1-\tau)(1-\upsilon)\beta\theta + (1-\psi)\beta(\mu+\omega)\}}{(\mu+\theta+\omega)(\mu+a)(\kappa+\mu+\gamma)}$$

3.5 Local stability of the disease free equilibrium

Theorem 2.

The disease free equilibrium point E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof

Let

$$\left.\begin{aligned}
F_1 &= b + \omega V - \psi\beta S\frac{(C+I)}{N} - (1-\psi)\beta S\frac{(C+I)}{N} - (\mu+\theta)S \\
F_2 &= \theta S - (1-\tau)(1-\upsilon)\beta V\frac{(C+I)}{N} - \tau(1-\upsilon)\beta V\frac{(C+I)}{N} - (\mu+\omega)V \\
F_3 &= \tau(1-\upsilon)\beta V\frac{(C+I)}{N} + \psi\beta S\frac{(C+I)}{N} - (\mu+a)C \\
F_4 &= (1-\tau)(1-\upsilon)\beta V\frac{(C+I)}{N} + (1-\psi)\beta S\frac{(C+I)}{N} - \kappa\pi I - \kappa(1-\pi)I + aC - (\mu+\gamma)I \\
F_5 &= \kappa\pi I - (\delta+\mu)R_1 \\
F_6 &= \kappa(1-\pi)I + \delta R_1 - \mu R_2
\end{aligned}\right\} \quad (3)$$

Given

$$|J(E_O) - \lambda I| = 0 \quad (4)$$

Evaluating the Jacobean of (3) and substituting into (4) we get

$$\begin{vmatrix}
J_{11}-\lambda & \omega & \frac{-\beta(\mu+\omega)}{(\mu+\omega)} & \frac{-\beta(\mu+\omega)}{(\mu+\omega)} & 0 & 0 \\
\theta & J_{22}-\lambda & \frac{(\upsilon-1)\beta\theta}{(\mu+\theta+\omega)} & \frac{(\upsilon-1)\beta\theta}{(\mu+\theta+\omega)} & 0 & 0 \\
0 & 0 & J_{33}-\lambda & \frac{\{\tau(1-\upsilon)\theta+\psi(\mu+\omega)\}\beta}{(\mu+\theta+\omega)} & 0 & 0 \\
0 & 0 & \frac{\{(1-\tau)(1-\upsilon)\theta+(1-\psi)(\mu+\omega)\}}{(\mu+\theta+\omega)}+a & J_{44}-\lambda & 0 & 0 \\
0 & 0 & 0 & \kappa\pi & J_{55}-\lambda & 0 \\
0 & 0 & 0 & \kappa(1-\pi) & \partial & J_{66}-\lambda
\end{vmatrix} = 0 \quad (5)$$

From equation (5) we observed that $\lambda_1 = -\mu$ and $\lambda_2 = -(\delta+\mu)$, thus equation (5) reduces to

$$\begin{vmatrix}
-J_{11}-\lambda & \omega & \frac{-\beta(\mu+\omega)}{(\mu+\theta+\omega)} & \frac{-\beta(\mu+\omega)}{(\mu+\theta+\omega)} \\
\theta & -J_{22}-\lambda & \frac{(\upsilon-1)\beta\theta}{(\mu+\theta+\omega)} & \frac{(\upsilon-1)\beta\theta}{(\mu+\theta+\omega)} \\
0 & 0 & -J_{33}-\lambda & \frac{\{\tau(1-\upsilon)\theta+\psi(\mu+\omega)\}\beta}{(\mu+\theta+\omega)} \\
0 & 0 & \frac{\{(1-\tau)(1-\upsilon)\theta+(1-\psi)(\mu+\omega)\}}{(\mu+\theta+\omega)} & -J_{44}-\lambda
\end{vmatrix} = 0 \quad (6)$$

Equation (6) can be transformed into

$$\begin{vmatrix} -J_{11}-\lambda & J_{12} & J_{13} & J_{14} \\ J_{21} & -J_{22}-\lambda & J_{23} & J_{24} \\ 0 & 0 & -J_{33}-\lambda & J_{34} \\ 0 & 0 & J_{43} & -J_{44}-\lambda \end{vmatrix} = 0 \qquad (7)$$

Using scientific workplace 5.5, we obtained the characteristics equation of (7) as

$$A_4\lambda^4 + A_3\lambda^3 + A_2\lambda^2 + A_1\lambda + A_0 = 0 \qquad (8)$$

where

$$\left.\begin{aligned} A_4 &= 1 \\ A_3 &= J_{11} + J_{22} + J_{33} + J_{44} \\ A_2 &= J_{11}\left(J_{22}+J_{33}\right) + J_{44}\left(J_{11}+J_{33}\right) + J_{33}\left(J_{11}+J_{12}\right) - \left(J_{12}J_{21} + J_{34}J_{43}\right) \\ A_1 &= J_{11}J_{22}\left(J_{33}+J_{44}\right) + J_{33}J_{44}\left(J_{11}+J_{22}\right) - \left\{J_{12}J_{21}\left(J_{33}+J_{44}\right) + J_{34}J_{43}\left(J_{11}+J_{22}\right)\right\} \\ A_0 &= J_{11}J_{22}J_{33}J_{44} + J_{12}J_{21}J_{34}J_{43} - \left(J_{11}J_{22}J_{34}J_{43} + J_{12}J_{21}J_{33}J_{44}\right) \end{aligned}\right\} \qquad (9)$$

We apply Routh-Hurwitz criterion which states that all roots of the polynomial have negative real part if and only if the coefficient A_I are positive and the determinant of the matrices $H_I > 0$ for $i = 0,1,2,3,4,5$. Thus

$$H_4 = \begin{bmatrix} A_3 & A_1 & 0 & 0 \\ 1 & A_2 & A_0 & 0 \\ 0 & A_3 & A_1 & 0 \\ 0 & 1 & A_2 & A_0 \end{bmatrix} \quad H_3 = \begin{bmatrix} A_3 & A_1 & 0 \\ 1 & A_2 & A_0 \\ 0 & A_3 & A_1 \end{bmatrix} \quad H_2 = \begin{bmatrix} A_3 & A_1 \\ 1 & A_2 \end{bmatrix} \quad H_1 = A_3 > 0$$

Therefore

$$H_2 = \begin{bmatrix} A_3 & A_1 \\ 1 & A_2 \end{bmatrix} = A_3A_2 - A_1 > 0 \text{ iff } A_2A_3 > A_1$$

$$H_3 = \begin{bmatrix} A_3 & A_1 & 0 \\ 1 & A_2 & A_0 \\ 0 & A_3 & A_1 \end{bmatrix}$$

$$A_1A_2A_3 - \left(A_0A_3^{\,2} + A_1^{\,2}\right) > 0 \text{ iff } A_1A_2A_3 > A_0A_3 + A_1^{\,2}$$

$$H_4 = \begin{bmatrix} A_3 & A_1 & 0 & 0 \\ 1 & A_2 & A_0 & 0 \\ 0 & A_3 & A_1 & 0 \\ 0 & 1 & A_2 & A_0 \end{bmatrix}$$

$$A_0A_1A_2A_3 - \left(A_0A_1^{\,2} + A_0^{\,2}A_3^{\,2}\right) > 0 \text{ iff } A_0A_1A_2A_3 > \left(A_0A_1^{\,2} + A_0^{\,2}A_3^{\,2}\right)$$

Therefore, all the eigenvalues of the polynomials have negative real parts, implying that $\lambda_3 < 0, \lambda_4 < 0, \lambda_5 < 0$ since all the values of $\lambda_i < o,$ for all $i = 1,2,3,4,5$ if $R_0 < 1.$

We conclude that the disease free equilibrium point is locally asymptotically stable.

3.6 Global stability

The two conditions that are sufficient to guarantee the global stability of the point following Castillo-Chavez and Feng [15] (1998) criterion are

$$\left.\begin{aligned} \frac{dX}{dt} &= F(X, Z) \\ \frac{dZ}{dt} &= G(X, Z) \\ G(X, 0) &= 0 \end{aligned}\right\} \qquad (10)$$

Where the component of the column-vector $X_1 \in \mathbb{R}^4$ denote the number of uninfected individuals and the component of the vector $Z \in \mathbb{R}^2$ denote the number of infected individuals, $E_0 = (X^*, 0)$ denote the disease free equilibrium of the system (1). The fixed point $E_0 = (X^*, 0)$ is globally asymptotically stable equilibrium provided that $R_0 < 1$ and the following two conditions are satisfied

(Y_1): for $\frac{dX}{dt} = F(X^*, 0)$, X^* is a globally asymptotically stable (11)

(Y_2): $G(X,Z) = AZ - \hat{G}(X,Z)$, $\hat{G}(X,Z) \geq 0$, for $(X,Z) \in D$ and $A = D_Z G(X^*, 0)$ (12)

Is an M-matrix (the off-diagonal elements of A are negative) and D is the region where the model makes biological meaning. Therefore we rewrite the system (1) in the form

$$\left.\begin{aligned} \frac{dX}{dt} &= F(X,Z) \\ \frac{dZ}{dt} &= G(X,Z),\ G(X,0) = 0 \end{aligned}\right] \quad (10)$$

Where $X = (S, V, R_1, R_2)$, $Z = (C, I)$

Considering the uninfected classes of equations (1) which are

$$\left.\begin{aligned} \frac{dS}{dt} &= b + \omega V - \psi\beta S\frac{(C+I)}{N} - (1-\psi)\beta S\frac{(C+I)}{N} - (\mu+\theta)S \\ \frac{dV}{dt} &= \theta S - (1-\tau)(1-\upsilon)\beta V\frac{(C+I)}{N} - \tau(1-\upsilon)\beta V\frac{(C+I)}{N} - (\mu+\omega)V \\ \frac{dR_1}{dt} &= \kappa\pi I - (\delta+\mu)R_1 \\ \frac{dR_2}{dt} &= \kappa(1-\pi)I + \delta R_1 - \mu R_2 \end{aligned}\right] \quad (11)$$

From equation (11) we find that $\frac{dX}{dt} = F(X^*, 0)$ hence

$$\frac{dX}{dt} = F(X^*, 0) = \begin{bmatrix} \frac{b(\mu+\omega)}{\mu(\mu+\theta+\omega)} \\ \frac{\theta b}{\mu(\mu+\theta+\omega)} \\ 0 \\ 0 \end{bmatrix} \quad (12)$$

Therefore condition Y_1 is satisfied.

(ii) Considering the infected classes of equations (1) which are

$$\left.\begin{aligned} \frac{dC}{dt} &= \tau(1-\upsilon)\beta V\frac{(C+I)}{N} + \psi\beta S\frac{(C+I)}{N} - (\mu+a)C \\ \frac{dI}{dt} &= (1-\tau)(1-\upsilon)\beta V\frac{(C+I)}{N} + (1-\psi)\beta S\frac{(C+I)}{N} - \kappa\pi I - \kappa(1-\pi)I + aC - (\mu+\gamma)I \end{aligned}\right] \quad (13)$$

From equation (13) we find that $A = D_Z G(X^*, 0)$ hence

$$G(X,Z) = 0 \quad (14)$$

We have the condition Y_2 satisfies, since

Hence, we conclude that E_0 is globally asymptotically stable for $R_0 < 1$.

4. Numerical Simulation In this section, we carry out some numerical simulations using ode function from MATLABR2016a.

Table 4.1 Parameters and variables values used for numerical simulation results

Variables/ Parameter	Values	Reference
$S(t)$	100000	Assumed values
$C(t)$	50	Assumed values
$I(t)$	30	Assumed values
$R_1(t)$	10	Assumed values.
$R_2(t)$	5	Assumed values
$V(t)$	2000	Assumed values
b	0.2	[16]
β	0.4	[16]
μ	0.05	[17]
γ	0.1	[17]
a	0.2	[16]
τ	0.1	[17]
υ	0.5	[16]
π	0.85	[16]
θ	0.01	[16]
ω	0.5	[16]
δ	0.15	[16]
κ	0.3	[16]
ψ	0.3	[16]

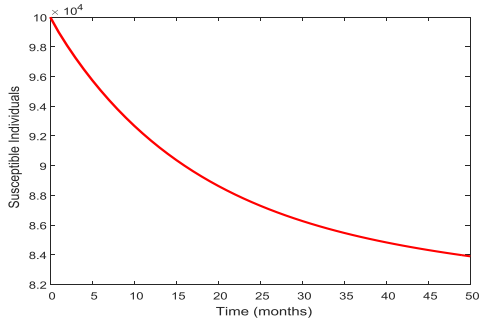

Figure 4.1 Numerical simulations for susceptible individuals. Values used in generating the graph presented in Table 4.1.

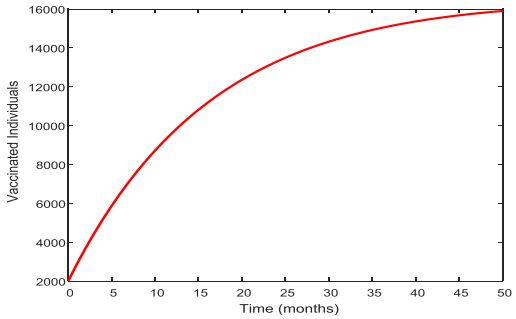

Figure 4.2 Numerical simulations for vaccinated individuals. Valued used in generating are presented in Table 4.1.

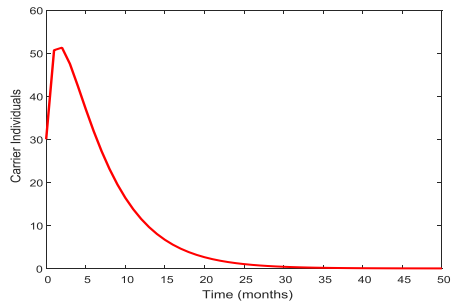

Figure 4.3 Numerical simulations for carrier individuals. The graph is generated using values presented in Table 4.1.

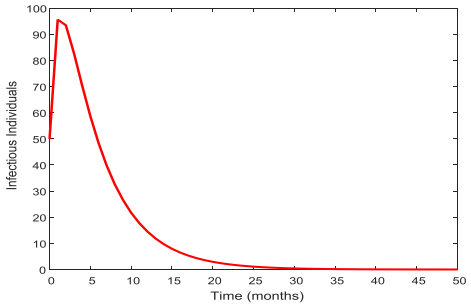

Figure 4.4 Numerical simulations for infectious individuals. The graph is generated using values presented in Table 4.1.

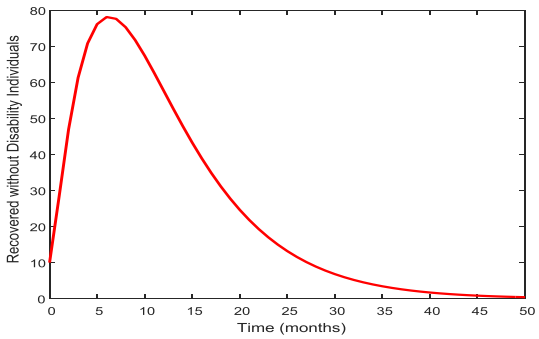

Figure 4.5 Numerical simulations for recovered without disability individuals. The graph is generated using values presented in Table 4.1.

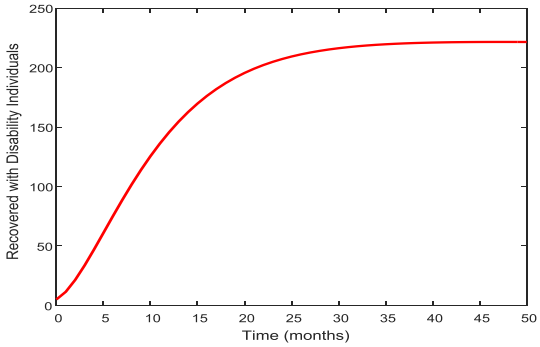

Figure 4.6 Numerical simulations for recovered with disability individuals. The graph is generated using values presented in Table 4.1.

InFigure 4.1, the susceptible population drops exponentially with time due to the vaccine coverage. In Figure 4.2, the vaccinated individuals rise exponentially as those who received vaccine increase with time. Figure 4.3 illustrate the evolution of carrier population drops slowly from it stability point with respect to time. Figure 4.4 also illustrates how it drop slowly as the impact of vaccine efficacy is perfect over time and used the parameter values on Table 4.1 In Figure 4.5 those recovered without disability from it stability point begin to drop exponentially and as the result those individual population recovered with disability increase rapidly in number over time in Figure 4.6 with the use of parameter values in Table 4.1.

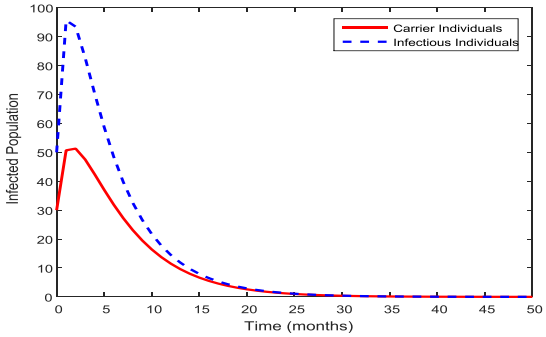

Figure 4.7 Numerical simulations for infected individuals. The graph is generated using values presented in Table 4.1.
In figure 4.7 the population reached their peak and drop to remain stable in some point near to disease free equilibrium and has indicate large differences between the number of those who has carrier infection with those who are in infectious class for the first 50 months which shows that carrier individuals and infectious individuals has been eradicated from the community for the 50 months. Which are generated using the parameter values presented in table 4.1

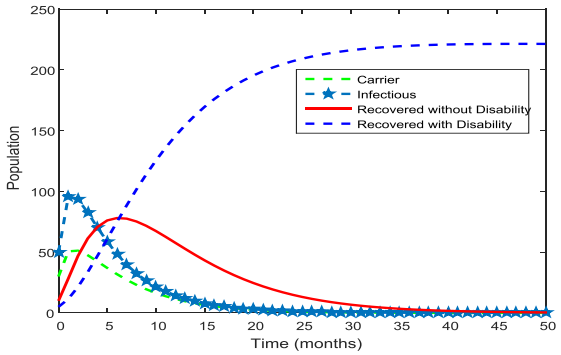

Figure 4.8 Numerical simulations for carrier, infectious and the two recovered individuals. All cases are generated using parameters values presented in Table 4.1.
In Figure 4.8 we decided to have numerical simulation of the whole populations on the same figure that is carrier individuals, infectious individuals, recovered without disability individuals and recovered with disability individuals. From the beginning,

there was few number of those recovered without disability individuals and those recovered with disability individuals and the number of infectious individuals are more in number than the carrier individuals. Where are at last those recovered without disability rises exponentially for some time before it begin to drop exponentially with time while those recovered with disability rises rapidly and exponentially to the highest level. Finally most of the populations met at a certain point with time and all generated using parameter values presented in Table 4.1

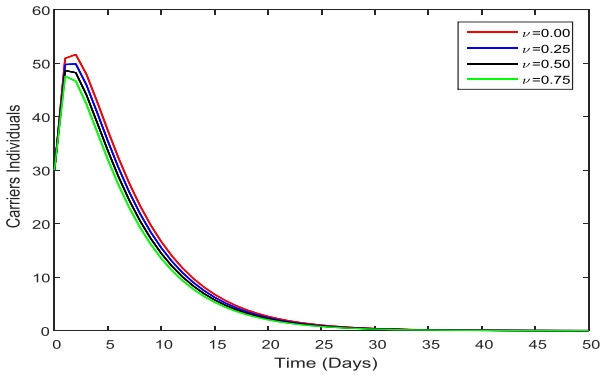

Figure 4.9 Showing the effects of vaccine efficacy on carrier individuals. The values of υ is varied from $(\upsilon: 0.00 - 0.75)$. All other parameter values are as presented in Table 4.1.

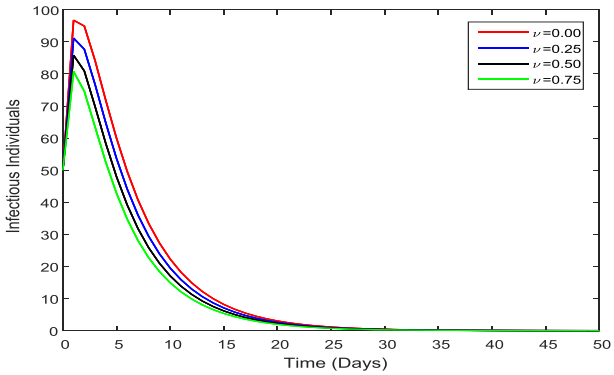

Figure 4.10 Showing the effects of vaccine efficacy on infectious individuals. The values of υ is varied from $(\upsilon: 0.00 - 0.75)$.All other parameter values are as presented in Table 4.1.

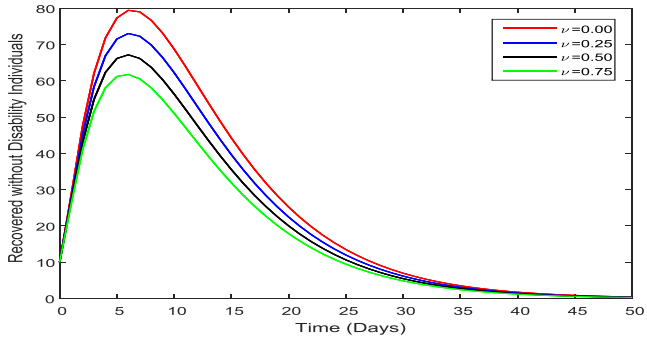

Figure 4.11 Showing the effects of vaccine efficacy on recovered without disability individuals. The values of υ is varied from $(\upsilon: 0.00 - 0.75)$. All other parameter values Are presented in Table 4.1.

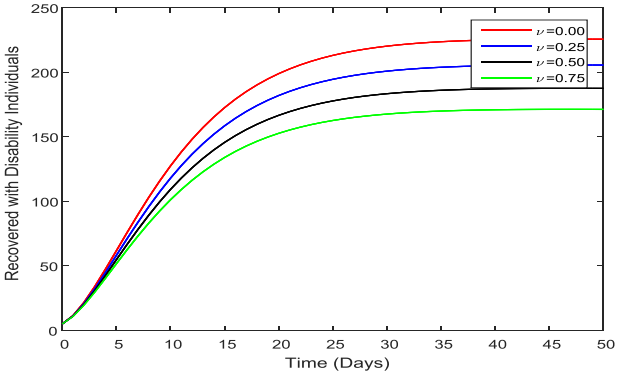

Figure 4.12 Showing the effects of vaccine efficacy on recovered with disability individuals. The values of υ is varied from $(\upsilon: 0.00 - 0.75)$. All other parameter values are as presented in Table 4.1.

In Figure 4.9, we varied υ to test the effects of vaccine efficacy on carrier individuals, infectious individuals, recovered without disability individuals and recovered with disability individuals. It was observed from Figures 4.9 - 4.12 that as the vaccine efficacy increase, the population of carrier individuals, infectious individuals, recovered with disability and recovered without disability individuals decreases.

5.0 Conclusion

The paper consists of six 6-dimensional system of ordinary differential equation. The disease free equilibrium point was established for the system of equations (1). The endemic equilibrium of the system was established and given by equations (2). We also established the basic reproduction number, R_0 using the next generation matrix. The local stability of the infection free equilibrium of the modified model was established using the Routh-Huritz condition for stability, we observed

that all the eigenvalues are negative, that is $\lambda_i < 0$, for $i = 1,2,3,4,5,6$, when $R_0 < 1$. This implies that the disease free equilibrium is locally asymptotically stable, indicating that meningococcal meningitis can be eradicated from the populations. We obtained the global stability of the disease free equilibrium of the model using Castillo- Chavez criterion for global stability. We observed that the model is globally asymptotically stable for (it is an M-matrix that is, the element of the off diagonal of the matrix are non-negative).The results of our numerical simulations show that that as the vaccine efficacy increase, the population of carrier individuals, infectious individuals, recovered with disability and recovered without disability individuals decreases.

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