The Spread of Meningitis: A Compartmental Mathematical Model

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Abstract. Meningitis has been among the infectious diseases affecting the African continent especially countries within the meningitis belt (East and West Africa). In this report, we formulated a model to investigate the spread dynamics of meningitis, the model divided the total population into four mutually disjoint compartments, Susceptible (S), Carrier (C), Infectious (I), and Removed (R). The model was solved both analytically and numerically, and also the expression for basic reproduction number ($\Re_0$) was derived. In the model analysis some parameter values were altered and the effects of the changes on the basic reproduction number ($\Re_0$) were observed. The model analysis discovered that immediate intervention is a strong factor in the control of meningitis.

Keywords: Meningitis, Mathematical Model, Numerical Simulation

Introduction

Meningitis is inflammation of the meninges (the covering of the brain and spinal cord). It is most often caused by infection (bacterial, viral, or fungal), but can also be produced by chemical irritation, subarachnoid hemorrhage, cancer and other conditions (Martínez *et al.*, 2013).

Meningococcal meningitis is a major public health problem threatening large area of the Sub-Saharan Africa known as the meningitis belt. Meningitis incidence increases every dry season, before dying out with the first rains of the year. Large epidemics, which can kill tens of thousands of people, occur frequently but unpredictably every 6–14 years. It has been suggested that these patterns may be attributable to complex interactions between the bacteria, human hosts and the environment (Irving *et al.*, 2012). Several mathematical models have been formulated for studying the epidemic of diseases (Abubakar *et al.*, 2017; Driessche & Watmough, 2002; Fleming & Rishel, 1975; Nguyen & Rohani, 2008; Hethcote, 2000; Ireland, Mestel, & Norman, 2017; Mugisha, 2017; Nigeria Centre for Disease Control, 2018; Zhang & Teng, 2008).

However, meningitis was also studied using mathematical models by many researchers Blyuss, 2016; Elmojtaba & Adam, 2017; Greenwood, 1999; Kiddy *et al.*, 2018; Lawi *et al.*, 2011; Mueller & Gessner, 2010; Trotter & Greenwood, 2007; Vareen, 2008; Yaesoubi *et al.*, 2018; Yusuf & Benyah, 2012; Yusuf, 2018; Yusuf & Olayinka, 2019). Martcheva and Crispino-O'Connell (2003) formulated an age-structured mathematical model to ease the understanding of the dynamics of meningitis. The model analyses conditions for the stability of the disease-free steady state (which imply extinction of the disease) and the existence of an endemic state (which leads to persistence of the disease in the population). The study applied the results of the model to identify contribution of the carriers to the spread of the disease. Wiah and Adetunde (2010) developed a mathematical model that governs the epidemiology of cerebrospinal meningitis in Jirapa, Ghana. The study reports on the dynamics of cerebrospinal meningitis caused by bacterial infection and gave suggestions as how they can be controlled. The study also performed numerical simulation on the model for Jirapa District in Upper West Region of Ghana.

Recently, Yusuf and Olayinka (2019) considered a deterministic model for the transmission dynamics of the disease which incorporates vaccination of the susceptible

members and timely treatment of the infective members as control measures is considered. This report formulated an SCIR model to study the dynamics of meningitis in Africa.

Model Formulation

The model formulated in this study consists of four (4) compartments as shown in Figure 1 below. Initially, individuals belong to Susceptible (S) compartment and the infected ones within latency period are members of Carrier (C) compartment, the Infectious (I) compartment accommodates all individuals that are capable of spreading the meningitis disease while people who recovered with or without disability as well as those killed by the disease occupy the Removed (R) compartment. The model is based upon the assumptions that: the population is not constant i.e. the model considered demographic factors; carriers can recover and regain susceptibility and the disease does not confer immunity on recovery. Lastly, Table 1 shows the model parameters used and their denotations.

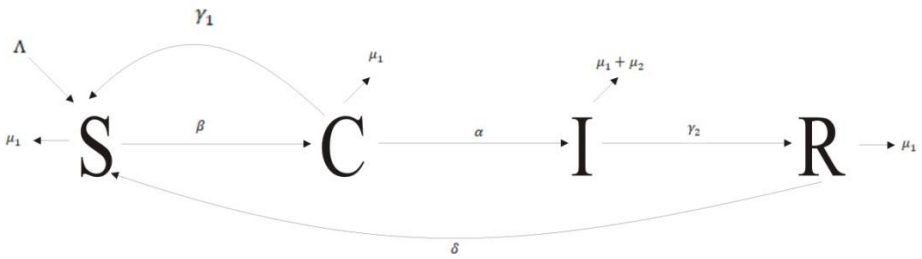


Figure 1. Schematic representation of the compartmental flow

Model Equations

$$S' = \Lambda - \frac{\beta SC}{N} + \gamma_1 C - \mu_1 C + \delta R$$

$$C' = \frac{\beta SC}{N} - \gamma_1 C - \alpha C - \mu_1 C$$

$$I' = \alpha C - \gamma_1 I - \mu_2 I - \mu_1 I$$

$$R' = \gamma_2 I + \mu_2 I - \delta R - \mu_1 R$$

Table 1. Description of model parameters

Parameter	Description
Λ	Recruitment rate
β	Transmission rate
γ_1	Rate at which carrier become susceptible
γ_2	Rate at which infected recovered
μ_1	Natural death rate
μ_2	Death rate due to infection
α	Rate at which carrier become infected
δ	Rate at which removed become susceptible

Model Analysis

Local stability of the disease-free equilibrium $\boldsymbol{E_0}$

The disease-free equilibrium (DFE) of the above system is given by:

$$E_0 = (S, C, I. R) = (\frac{\Lambda}{\mu}, 0,0,0).$$

In order to study the local stability of the DFE we need to find the basic reproduction number $\Re_0$. It is the average number of secondary infections caused by the average infectious individuals.

$$F = \begin{pmatrix} \frac{\beta S}{N} & 0 \\ 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} \gamma_1 + \alpha + \mu_1 & 0 \\ -\alpha & \gamma_2 + \mu_2 + \mu_1 \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{\gamma_1 + \alpha + \mu_1} & 0 \\ \frac{\alpha}{(\gamma_1 + \alpha + \mu_1)(\gamma_2 + \mu_2 + \mu_1)} & \frac{1}{(\gamma_2 + \mu_2 + \mu_1)} \end{pmatrix}$$

$$G = FV^{-1} = \begin{pmatrix} \frac{\beta S}{N(\gamma_1 + \alpha + \mu_1)} & 0 \\ 0 & 0 \end{pmatrix}$$

The Eigen values of G are $\lambda_1 = 0$ and $\lambda_2 - \frac{\beta S}{N(\gamma_1 + \alpha + \mu_1)}$

Thus, $\Re_0 = p(G) = \frac{\beta S}{N(\gamma_1 + \alpha + \mu_1)}$

To investigate the stability of the epidemic the study employed the following Theorem by Weah and Adetunde (2010).

Theorem 1: If $\Re_0 < 1$ then E_0 is stable, if $\Re_0 > 1$ then E_0 is unstable (Weah & Adetunde, 2010).

Existence of Endemic Equilibrium

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

$S^* = \frac{N(\gamma_1 + \alpha + \mu_1)}{\beta}$

$C^* = (\gamma_2 + \mu_1 + \mu_2)(\mu_1 + \delta)$

$I^* = \lambda(\mu_1 + \delta)$

$R^* = \lambda(\gamma_1 + \mu_2\mu)$

Where $\lambda = \frac{\Lambda}{(\alpha+2\mu_1)(\gamma_2+\mu_1+\mu_2)(\mu_1+\delta)-\delta\alpha(\gamma_2+\mu_2)}$

Therefore, the endemic equilibrium exists if $\lambda > 0$. This shows that in the absence of a control measure there can be prevalence of the disease which may lead to a wide spread outbreak.

Numerical Simulation and Discussion

In this study the estimated parameters are; the transmission rate from susceptible to carrier, the rate at which carrier becomes susceptible and the rate at which removed becomes susceptible. All other parameters are from references. The initial conditions were S=460, C=12, I=5, and R=0.

Parameter Values

Parameter	Parameter Value
Λ	0.2
β	0.9
γ_1	0.33
γ_2	0.3
μ_1	0.01
μ_2	0.1
α	0.1
δ	0.5

In Figure 2a, 2b and 2c graph models for the simulation using Euler's method are illustrated based on the consideration of different levels of intervention.

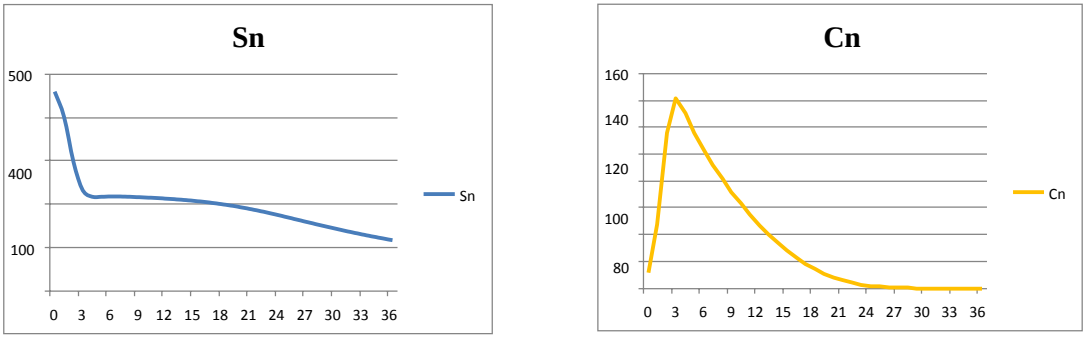
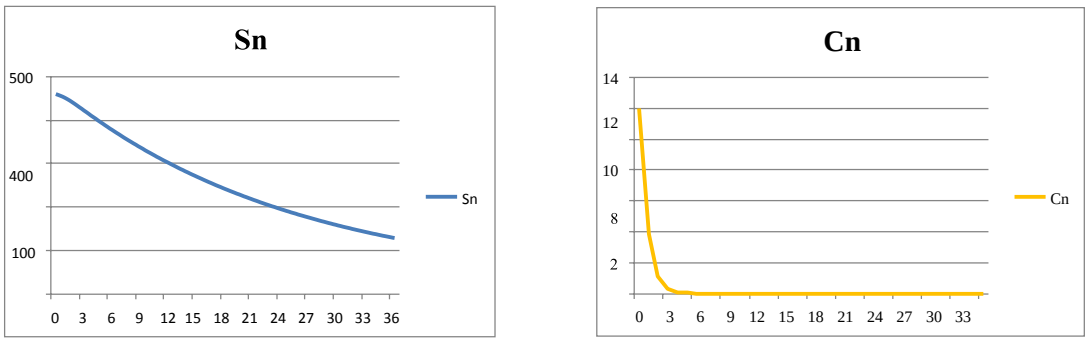
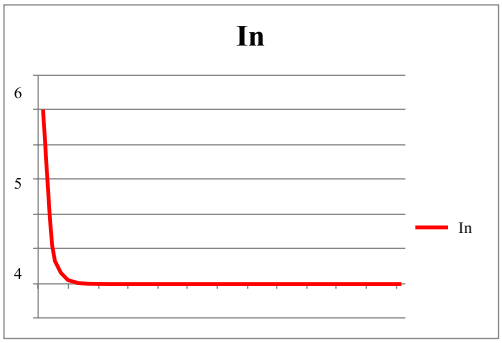


Figure 2a. Simulation of model when β = 0.9, γ_1 = 0.33, *and* α = 0.1, $\Re_0$ = 0.36





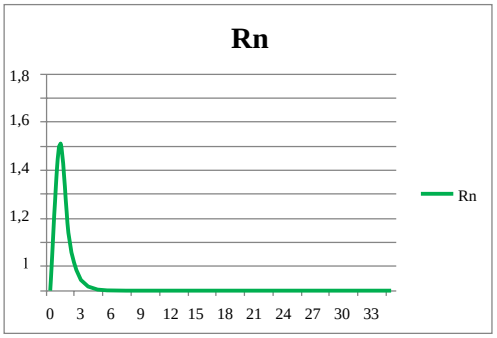
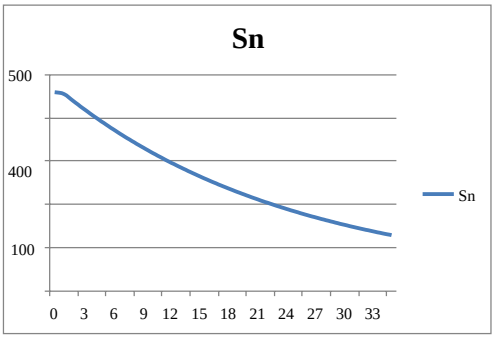
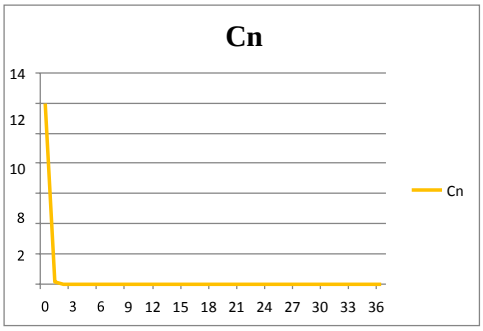
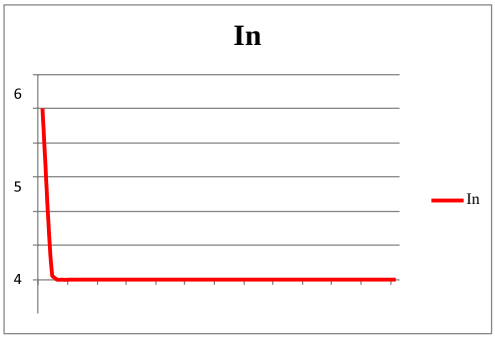


Figure 2b. Simulation of model when β = 0.5, γ_1 = 0.66 and α = 0.01, $\Re_0$ = 0.15







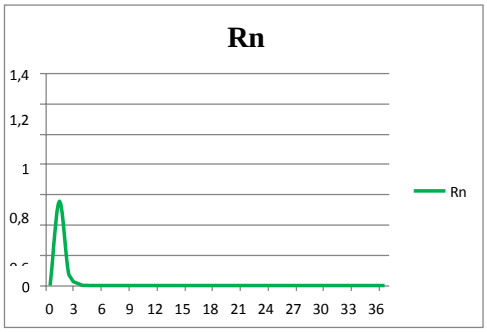


Figure 2c. Simulation of model when β = 0.1, γ_1 = 0.99 and α = 0.001, $\Re_0$ = 0.03

Results

Figure 2a shows that the number of susceptible individuals which decreased during the first three (3) months caused an increase in the number of carrier, infected and removed. Then after the initial period of five (5) months the number of susceptible individuals dropped and remained constant for the rest of the period while the number of carrier, infected and susceptible also decreased to a point near the disease-free equilibrium.

Figure 2b shows that the number of susceptible individuals decreased continuously for the whole duration. The number of carrier and infected individuals dwindle within the period of first five (5) months, the population of the removed experienced a hike initially during the first two (2) months and then decreased until it diminishes.

Figure 2c shows that the number of susceptible individuals decreased continuously for the whole duration. The number of carrier and infected individuals dwindle within the period of first two (2) months, the population of the removed experienced a hike initially during the first month and then decreased to the lowest ebb.

Conclusion

The basic reproduction number ($\Re_0$) is very important parameter that reveals the on average number of secondary cases produced by the existing primary ones. Reducing the rate of transmission can cause increase in the rate at which carrier individuals become susceptible as well as the infectious rate of carrier individuals, thereby lowering the basic reproduction number ($\Re_0$). *Figure 2a* shows that when $\beta = 0.9$, $\gamma_1 = 0.33$, and $\alpha = 0.1$, it will take an infectious person approximately three (3) days to produce a new case, when $\beta = 0.5$, $\gamma_1 = 0.66$ and $\alpha = 0.01$, an infectious individual needs approximately seven (7) days to produce a new case, more so, the primary index will take approximately thirty five (35) days to infect someone when $\beta = 0.1$, $\gamma_1 = 0.99$ and $\alpha = 0.001$. Finally, we recognized and recommend rapid intervention as a burly strategy to dwindle the spread of meningitis.

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