

A Novel S-C-I-R Model for Meningococcal Meningitis: Formulation, Analysis, and Implications for the African Meningitis Belt

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Date of Submission: 23-01-2026

Date of Acceptance: 05-02-2026

Abstract

Meningococcal meningitis remains a significant public health challenge in the African Meningitis Belt, characterized by seasonal epidemics driven by complex transmission dynamics. A critical feature of its epidemiology is the presence of asymptomatic carriers, who colonize the bacterium *Neisseria meningitidis* and facilitate silent transmission. This study formulates and rigorously analyzes a deterministic mathematical model for the spread of meningococcal meningitis, explicitly incorporating a Carrier (C) compartment into the classic SIR framework. The resulting S-C-I-R model captures the transition from susceptible (S) to carrier to infectious (I) and finally to recovered (R) states. We derive the basic reproduction number (R_0) as a threshold parameter for disease invasion and perform equilibrium and stability analyses. Analytical results reveal that R_0 decomposes into contributions from both carrier and symptomatic transmission, providing theoretical evidence that interventions targeting the carriage state are paramount for epidemic control. This work provides a foundational mathematical framework for understanding meningitis dynamics and offers strategic insights for public health policy in endemic regions like Nigeria.

Keywords: Mathematical Modeling, Infectious Disease, Meningococcal Meningitis, Carrier State, Basic Reproduction Number, Stability Analysis, African Meningitis Belt

1. Introduction

Meningococcal meningitis, caused by the bacterium *Neisseria meningitidis*, is a severe infection of the membranes surrounding the brain and spinal cord. It poses a persistent threat in the African Meningitis Belt, a region stretching from Senegal to Ethiopia, where large-scale epidemics occur periodically, particularly during the dry season [7]. Nigeria, situated within this belt, experiences significant annual morbidity and mortality from the disease, placing a heavy burden on its healthcare system [5]. The epidemiology of meningococcal meningitis is notably distinct from many other directly transmitted infections due to the pivotal role of asymptomatic carriers. A substantial proportion of individuals (often 5-20% in endemic areas) harbor the bacteria in their nasopharynx without developing clinical illness, yet remain capable of transmitting the pathogen to others [2].

This biological reality challenges the sufficiency of the classic Susceptible-Infectious-Recovered (SIR) model, which implicitly assumes all infected individuals are symptomatic and equally identifiable. Models that fail to account for the carrier state may significantly underestimate the true force of infection and the potential for silent community spread, leading to suboptimal intervention strategies. While compartmental models have been extensively applied to diseases like malaria and COVID-19 in Africa [1?], and some studies have incorporated carriers for meningitis in other contexts [4], there remains a need for a dedicated, analytically explored S-C-I-R model tailored to the transmission dynamics within the African Meningitis Belt.

The primary objective of this paper is to bridge this gap by developing a novel deterministic compartmental model that explicitly includes a Carrier (C) class. We aim to: (i)

formulate the S-C-I-R model with biologically realistic assumptions; (ii) perform a complete equilibrium and stability analysis; (iii) derive and interpret the basic reproduction number R_0 ; and (iv) discuss the public health implications of our analytical findings, with a specific focus on control strategies relevant to Nigeria and the wider region. This theoretical work establishes a rigorous foundation upon which future studies incorporating numerical simulation and local data can be built.

2 Model Formulation

2.1 Model Structure and Assumptions

The total human population at time t , denoted $N(t)$, is subdivided into four mutually exclusive compartments:

- **Susceptible ($S(t)$):** Individuals who are not colonized by *N. meningitidis* and are at risk of infection.
- **Carriers ($C(t)$):** Individuals who are asymptotically colonized in the nasopharynx. They are capable of transmitting the bacteria to susceptible individuals but do not exhibit symptoms of invasive meningococcal disease.
- **Infectious ($I(t)$):** Individuals who have developed symptomatic, clinical meningitis and are capable of transmitting the infection.
- **Recovered ($R(t)$):** Individuals who have cleared the infection (from either the C or I state) and have gained temporary or permanent immunity against reinfection.

The model is governed by the following core assumptions:

1. The population mixes homogeneously.
2. The birth and natural death rates are equal, denoted by μ , yielding a constant total population size $N = S + C + I + R$.
3. Newborns enter the susceptible compartment at rate μN .
4. Carriers are infectious. The force of infection λ is given by:

$$\lambda = \frac{\beta_C C + \beta_I I}{N}$$

where β_C and β_I are the effective contact rates for transmission from carriers and symptomatic individuals, respectively.

5. Upon effective contact, susceptible individuals enter the Carrier compartment.
6. Carriers progress to the symptomatic Infectious compartment at a constant rate θ .
7. Carriers may also clear colonization and move directly to the Recovered compartment at rate γ_C .
8. Infectious individuals recover at rate γ_I .
9. Disease-induced mortality is considered negligible relative to the epidemic timescale, an assumption supported by the availability of treatment in managed outbreaks.

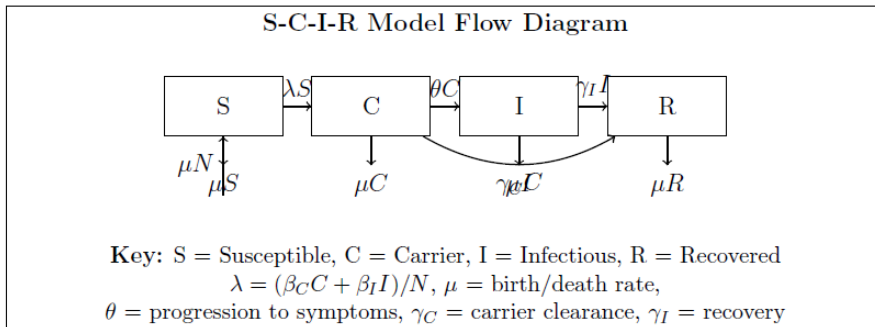


Figure 1: Flow diagram of the S-C-I-R model for meningococcal meningitis.

2.2 Governing Equations

Based on the structure and assumptions above, the dynamics of meningococcal meningitis are described by the following system of ordinary differential equations:

$$\frac{dS}{dt} = \mu N - \lambda S - \mu S, \quad (1)$$

$$\frac{dC}{dt} = \lambda S - (\theta + \gamma_C + \mu)C, \quad (2)$$

$$\frac{dI}{dt} = \theta C - (\gamma_I + \mu)I, \quad (3)$$

$$\frac{dR}{dt} = \gamma_C C + \gamma_I I - \mu R, \quad (4)$$

with the force of infection $\lambda = (\beta_C C + \beta_I I)/N$.

Since the equation for R is decoupled from the others, we can analyze the reduced system for S , C , and I with $N = S + C + I + R$ held constant.

3 Model Analysis

3.1 Disease-Free Equilibrium (DFE) and Basic Reproduction Number (R_0)

The system has a disease-free equilibrium (DFE), where the population comprises only susceptible and recovered individuals who were never infected, given by:

$$\mathcal{E}_0 = (S^0, C^0, I^0, R^0) = (N, 0, 0, 0).$$

We compute the basic reproduction number R_0 using the next-generation matrix method [6]. The infected compartments are C and I . The matrices for new infections (\mathbf{F}) and transitions between compartments (\mathbf{V}), evaluated at the DFE, are:

$$\mathbf{F} = \begin{pmatrix} \beta_C & \beta_I \\ 0 & 0 \end{pmatrix}, \quad \mathbf{V} = \begin{pmatrix} \theta + \gamma_C + \mu & 0 \\ -\theta & \gamma_I + \mu \end{pmatrix}.$$

The basic reproduction number is the spectral radius of the next-generation matrix \mathbf{FV}^{-1} :

$$R_0 = \rho(\mathbf{FV}^{-1}) = \frac{\beta_C}{\theta + \gamma_C + \mu} + \frac{\beta_I \theta}{(\theta + \gamma_C + \mu)(\gamma_I + \mu)}. \quad (5)$$

3.2 Biological Interpretation of R_0

The expression for R_0 (5) has a clear biological interpretation as the sum of two contributions:

- **Carrier-driven transmission:** The first term, $R_0^C = \frac{\beta_C}{\theta + \gamma_C + \mu}$, represents the average number of secondary infections produced by an individual who enters the Carrier compartment during their entire infectious period as a carrier. The denominator $(\theta + \gamma_C + \mu)$ is the average duration of the carrier state.
- **Symptomatic-driven transmission:** The second term, $R_0^I = \frac{\beta_I \theta}{(\theta + \gamma_C + \mu)(\gamma_I + \mu)}$, represents the number of secondary infections produced via the symptomatic pathway.

This is the product of:

1. The probability that a carrier progresses to disease before clearing the infection or dying: $\frac{\theta}{\theta + \gamma_C + \mu}$.
2. The average number of secondary cases generated by a symptomatic individual during their infectious period: $\frac{\beta_I}{\gamma_I + \mu}$.

This decomposition is a central result, highlighting that total transmission is a compound process dominated by the parameters governing the carrier state.

3.3 Local Stability of the Disease-Free Equilibrium

Theorem 1. *The disease-free equilibrium \mathcal{E}_0 of the system is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof. The stability is governed by the eigenvalues of the Jacobian matrix of the system (S, C, I) evaluated at \mathcal{E}_0 . The characteristic equation factors, yielding eigenvalues $-\mu$ (from the S -direction) and the roots of a quadratic equation derived from the infected

subsystem. Using the Routh-Hurwitz criteria, it can be shown that all eigenvalues have negative real parts if and only if $R_0 < 1$. The condition $R_0 > 1$ implies a positive eigenvalue, rendering the DFE unstable. This establishes R_0 as a sharp threshold for disease invasion. \square

3.4 Existence and Uniqueness of the Endemic Equilibrium

An endemic equilibrium (EE) $\mathcal{E}^* = (S^*, C^*, I^*, R^*)$ with $C^* > 0$ and $I^* > 0$ exists when $R_0 > 1$. Solving the equilibrium equations yields:

$$S^* = \frac{N}{R_0}, \tag{6}$$

$$C^* = \frac{\mu N(R_0 - 1)}{\beta_C + \frac{\beta_I \theta}{\gamma_I + \mu} - \mu}, \tag{7}$$

$$I^* = \frac{\theta}{\gamma_I + \mu} C^*. \tag{8}$$

The expression for S^* is classic: at equilibrium, the susceptible population is depleted to the inverse of the reproduction number. The positivity of C^* and I^* is contingent upon $R_0 > 1$, confirming the threshold property.

4 Discussion and Public Health Implications

The analytical results of this S-C-I-R model provide several critical insights for understanding and controlling meningococcal meningitis in endemic regions.

Firstly, the structure of R_0 underscores the paramount importance of the asymptomatic carrier population in sustaining transmission. In many realistic scenarios, especially in the African Meningitis Belt where carriage prevalence is high, the term R_0^C is likely to dominate R_0 . This is because the carrier state is typically longer-lasting than the symptomatic illness ($\gamma_C \ll \gamma_I$), and carriers, being asymptomatic, may have wider and more frequent social contacts. This theoretical finding strongly argues that surveillance and intervention strategies focused solely on symptomatic cases will be inadequate for epidemic control.

Secondly, the model identifies the most sensitive parameters for intervention. The reproduction number R_0 is most sensitive to the carrier transmission rate β_C and the duration of the carrier state $1/(\theta + \gamma_C + \mu)$. Therefore, the most effective public health strategies should aim to:

- 1. Reduce β_C (Carrier Transmissibility):** This can be achieved through vaccination with conjugate vaccines (e.g., MenAfriVac), which are known to reduce nasopharyngeal carriage and induce herd protection [3]. Our model provides a mathematical basis for the observed success of such mass vaccination campaigns in the Meningitis Belt.
- 2. Reduce the Duration of Carriage ($1/\gamma_C$):** This is the target of antibiotic chemoprophylaxis used for outbreak response and in close contacts of cases. The model suggests that even partially effective chemoprophylaxis that shortens carriage can significantly reduce R_0 .
- 3. Increase the Rate of Leaving the Carrier State ($\theta + \gamma_C$):** While not directly controllable, understanding factors that promote natural clearance can inform research.

The threshold condition $R_0 < 1$ for disease elimination provides a quantitative goal for vaccination programs. The critical vaccination coverage p_c needed to achieve herd immunity, assuming a perfect vaccine that confers lifelong immunity and blocks carriage, is given by $p_c > 1 - 1/R_0$. For a disease with an R_0 estimated between 2 and 5 in epidemic settings, this implies a coverage target of 50% to 80%, aligning with the objectives of real-world vaccination initiatives in the region.

5 Conclusion

In this study, we have developed and analyzed a novel S-C-I-R compartmental model for meningococcal meningitis that explicitly accounts for the asymptomatic carrier state. The model's formulation is grounded in the biological and epidemiological reality of the

disease, particularly in high-burden areas like the African Meningitis Belt. Through rigorous mathematical analysis, we derived the basic reproduction number R_0 , established the local stability of the disease-free equilibrium, and demonstrated the existence of a unique endemic equilibrium when $R_0 > 1$.

The key theoretical contribution is the decomposition of R_0 into carrier-driven and symptomatic-driven components. This structure irrefutably demonstrates that controlling meningococcal meningitis epidemics necessitates interventions that target the silent reservoir of carriers. Our work thus provides a robust mathematical framework that justifies and guides current cornerstone public health strategies—specifically, mass vaccination with conjugate vaccines and targeted chemoprophylaxis.

Future work will involve refining this model by incorporating age structure (as carriage prevalence is highest in adolescents), seasonal forcing in transmission parameters to mimic the dry season epidemic peak, and multiple bacterial serogroups. Furthermore, numerical simulation and fitting of the model to epidemiological data from Nigeria will be essential to validate its predictive power and to estimate context-specific parameters for guiding national and sub-national control policies.

Acknowledgments

This work was originally developed as an undergraduate research project in 2017 at Enugu State University of Science and Technology. The author acknowledges the guidance of project supervisors and the Department of Industrial Mathematics and Applied Statistics.

Conflict of Interest

The author declares no conflict of interest.

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