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Mathematical modeling and analysis of Meningococcal Meningitis transmission dynamics

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Abstract

Meningococcal meningitis (MCM) is one of the serious public health threats in the tropical and sub-tropical regions. In this paper, we propose an epidemic model to study the transmission dynamics of MCM with high- and low-risk susceptible populations. The model considers two different groups of susceptible individuals depending on the availability of medical resources (MR, including hospitals, health workers, etc), which varies the infection risk. We find that the model exhibits the phenomenon of backward bifurcation (BB), which increases the difficulty of MCM control since the dynamics are not merely relying on the basic reproduction number, $\mathcal{R}_0$. This study explores the effects of MR on the MCM epidemics by mathematical analysis and shows the existence of BB on MCM disease. Our findings suggest that providing adequate MR in a community is crucial in mitigating MCM incidences and deaths, especially in the MCM endemic regions.

Keywords: Meningococcal meningitis, mathematical modeling, bifurcation analysis, stability, medical resource

1 Introduction

Meningococcal meningitis (MCM), a thin lining infection that surrounds the brain and the spinal cord (mostly affects children and young adults), is a bacterial disease caused by the bacterium *Neisseria meningitidis* (i.e., *N. meningococcus*). MCM is transmitted from person-to-person through droplets of respiratory or throat secretions from infected individuals [2, 13, 38, 40, 55]. The transmission of MCM is highly fueled by close and prolonged contact (e.g., kissing or sneezing), mass gathering, seasonality, and smoking [2, 38, 55]. Meningitis disease have twelve different serogroups with MCM mostly prevalent [7, 33, 55]. The incubation period of the MCM disease varies between 2 to 10 days. Since every infected individual will pass through the exposure stage (E), before becoming infectious [12, 17]. This stage of the sub-clinical disease, or sometimes called the latency period for chronic diseases, is a period from exposure to onset of disease symptoms (or infectious) which may be as brief as seconds for some diseases and may be very long for some other diseases. During the exposure stage, the MCM disease is said to be asymptomatic (no symptoms) or inapparent [5, 12, 17, 31]. About 8% to 15% of infected individuals within 1 to 2 days after the onset of symptoms even with early treatment, and is higher of up to 30.9% if left untreated. After recovery MCM may result in brain failure, loss of hearing or disability in 10% to 20% of individuals who survives [10, 12, 14, 15, 55].

The transmission of MCM is largely from carriers (asymptomatically infected individuals, i.e., individuals who are infected and can infect others yet did not show any clinical symptoms of the MCM disease) [55]. Therefore, individuals in the carrier stage have a high chance of spreading the MCM infection than symptomatic individuals (i.e., infected individuals with clinical symptoms of the MCM disease) who will sometimes be bound to bed during the acute phase of the infection. Thus, carriers are likely to have more contacts with susceptible individuals than symptomatically infected individuals who will typically be inactive during the acute phase of the MCM infection. Also, when carriers become more in a highly-populated setting (e.g., Nigeria) there is a high tendency of the MCM infection spreads more rapidly, which may result an increase in the number of symptomatic individuals [5, 13, 55]. Some of the common symptoms of the MCM are a stiff neck, headaches, high fever, confusion, sensitivity to light and vomiting. In severe cases, MCM may cause meningococcal septicaemia, which is less common abut more severe that is distinguished by a haemorrhagic rash and rapid circulatory collapse and result in brain failure [55].

The safe and cost-effective vaccines against meningococcal disease exist for more than 40 years [47, 55], and it was initiated in 2010 in Burkina Faso, some parts of Niger and Mali, and targeting children and young adults (from 1 to 29 years age group) [49]. Since November 2017, more than 280 million individuals have been vaccinated in 21 African countries, and many lives were saved. To continue with such kind of tremendous success, there is a need to have a long-term immunization strategy in order to retain the protection of the population more especially in areas with limited medical resources mostly in the underdeveloped or developing countries [7, 27, 55].

MCM has been endemic in many parts of the world for many years, the disease continues to be a major public health concern in affected areas, notably the sub-Saharan "meningitis belt", which spans across Africa from Senegal to Ethiopia [36, 41, 55, 63]. The MCM accounts for more than 1.2 million cases and 135,000 deaths every year globally [6]. In Nigeria, MCM accounts for 14,473 cases with 1156 deaths cases in 2017 of which over a half were from children under the age of 16 [13]. Most of the places with MCM as an endemic disease are underdeveloped, where the medical resources are non-uniformly-

distributed (among different groups of the population) and limited. Several mathematical modeling studies have been designed and used to gain qualitative insights into the transmission dynamics of emerging and re-emerging infectious diseases in a community, dating back to the pioneering works [3, 11, 24, 29, 30, 43, 59]. These studies have been extended over the years to include different aspects related to the diseases transmission dynamics, such as the impact of limited medical resources on some diseases dynamics [1, 23, 44, 45, 52, 60], role of public health awareness programs [16, 26, 28, 33, 35, 58, 64].

In this study, we consider the impact of limited medical resources for the transmission dynamics of MCM disease with the aim of assessing its impact in a community where the MCM cases are high (e.g., northern part of Nigeria) so as to lower the disease transmission and spreads. The model considers two different groups of populations as follows: the population with sufficient/adequate medical resources (such as urban areas, where there are the availability of hospitals, primary health cares, pharmacies etc.); and the population with insufficient/inadequate medical resources (i.e, limited or no hospitals, limited health cares, limited pharmacies etc.) in order to highlight some of the significance and needs to provide sufficient medical resources in a community with high MCM cases (e.g., rural areas of the northern part of Nigeria). This is to notify policymakers and government to put down more emphasis in providing sufficient medical resources in such a population, so as to reduce disease transmission and spreads. Further, the model exhibits a phenomenon of backward bifurcation (a situation where a stable disease-free equilibrium co-exists with a stable endemic equilibrium even if the basic reproduction number is less than unity) which makes the MCM control more difficult as the MCM disease eradication is no longer merely relying on the basic reproduction number.

To the authors' best knowledge, there is no mathematical modeling study which has been designed to consider and access (examine) the impact of limited medical resources for the MCM transmission dynamics, though there are (biological) evidence which shows that lack of sufficient medical resources can affects and courses significant increase for the MCM transmission and spreads, especially in a rural areas where the disease is epidemic and have high MCM cases and deaths [55]. Hence, it will be imperative to examine the impact of medical resources on the dynamics of MCM disease, using a mathematical modeling approach. Also, to the best of the author's knowledge, our model is the first to show the existence of backward bifurcation for the MCM transmission dynamics. This paper is organized as follows. The epidemic model is formulated and analyzed in section 2 and 3. Numerical simulations and sensitivity analysis are performed in section 4.

2 Mathematical model

2.1 Model formulation

Let N denote the total human population size. The population is separated into sub-populations of high risk susceptible individuals (with limited MR) (S_n), low risk susceptible individuals (with adequate or sufficient MR) (S_a), individuals exposed to meningitis (E), carriers (asymptomatic infections) (C), infectious individuals (I) and recovered humans (R).

The susceptible individuals are recruited at a rate π (by birth). A fraction ω of total newly recruited individuals join the S_a class and the remaining fraction $(1-\omega)$ join the S_n class. Individuals in the S_n and S_a are assumed to join the exposed class following effective contact with an infected

Table 1: Summary table of the parameters description in model (2.1)

Parameter	Description/Interpretation
π	Recruitment rate
β	Effective contact rate (transmission rate)
γ	Waning immunity rate
μ	Natural death rate
δ	Disease-induced death rate
τ	Rate of loss of carriage
θ	Recovery rate from the I class
σ	Recovery rate from the C class
k	Fraction of E moving to I without first passing through C
ω	Fraction of newly recruited individuals to S_a
α	Fraction of S_a moving to E
ρ	Fraction of R moving to S_a

individuals at a rates λ and $\alpha\lambda$ respectively (noting that $0 \leq \alpha \leq 1$: shows that the individuals in the S_n class are assumed to contract the disease at a higher rate than the individuals in the S_a class, this is due to the poor/insufficient medical resources which brings about high disease prevalence). The exposed individuals (E) are assumed to move to the infected class at a rate ϕ, a fraction k join the infected class I, while the remaining fraction join the carrier class. The individuals in the C and I classes can recover at a rate σ and θ respectively. A waning of immunity can occur in the recovered class R of which a fraction γ will return to S_a class at a rate ρ, while the remaining fraction will move to the S_n class. The parameter δ represent disease induced death by MCM infected individuals from the I class, and the parameter β is the transmission probability, while μ is the natural death rate of humans which is assumed to be constant for all the compartments. We assumed that there is no movement of individuals from S_n to S_a and vice versa in order to have more insights when there are inadequate medical resources in a community.

The nonlinear systems of differential equations below describes the transmission dynamics of MCM disease. All the variables and parameters used in the model are summarized in Table 1.

$$\begin{aligned}
\frac{dS_a}{dt} &= \omega\pi + \rho\gamma R - (\alpha\lambda + \mu)S_a, \\
\frac{dS_n}{dt} &= (1-\omega)\pi + (1-\rho)\gamma R - (\lambda + \mu)S_n, \\
\frac{dE}{dt} &= \lambda(S_n + \alpha S_a) - (\phi + \mu)E, \\
\frac{dC}{dt} &= (1-k)\phi E - (\tau + \sigma + \mu)C, \\
\frac{dI}{dt} &= k\phi E + \tau C - (\theta + \delta + \mu)I, \\
\frac{dR}{dt} &= \sigma C + \theta I - (\gamma + \mu)R.
\end{aligned} \tag{2.1}$$

Here, the force of infection is given by $\lambda = \frac{\beta(C+I)}{N}$.

2.2 Basic properties

The basic properties of the model (2.1) will now be explored. Consider the following equations for the rate of change of the total human $N'(t)$ population (where the prime represents differentiation with respect to time):

$$\frac{dN}{dt} = \pi - \delta I - \mu N \leq \pi - \mu N. \tag{2.2}$$

Furthermore, consider the region $\Omega = \left\{(S_a, S_n, E, C, I, R) \in \mathbb{R}^6_+ : N \leq \frac{\pi}{\mu}\right\}$. By solving N in equation (2.2), it can be shown that all solutions of the system starting in Ω remain in Ω for all $t \geq 0$. Thus, Ω is positive-invariant, and it is sufficient to consider solutions restricted in Ω. In this region, the usual existence, uniqueness and continuation results hold for the system (2.1) [25, 37, 50].

2.3 Model fitting

The model (2.1) is fitted to the yearly MCM cases in Nigeria from 2010-2017. The MCM cases time series are obtained from the World Health Organization (WHO) [56]. The demographic time series are obtained from the World Bank [54]. The demographic parameters, e.g., π and μ, are calculated as follows; the average number of population in Nigeria is 174,387,515, and the average life expectancy is 52.34 years [53]. Therefore, the term $\mu^{-1} = 52.34$ years, and then, it follows that $\frac{\pi}{\mu} = 174,387,515$, this implies that $\pi = 9128$ per day. Note that all other parameters are fixed as in the Table 2. Fig. 1 shows the fitting results of model (2.1).

3 Mathematical analysis

3.1 Disease-free equilibrium (DFE)

In the absence of the disease (i.e., $E = C = I = R = 0$), the DFE of the model (2.1) obtained at steady state is given by

$$E_1^0 = (S_a^0, S_n^0, E^0, C^0, I^0, R^0) = \left(\frac{\omega\pi}{\mu}, \frac{(1-\omega)\pi}{\mu}, 0, 0, 0, 0\right).$$

Using the next generation matrix method technique [51], we obtained the associated reproduction number of the model (2.1) (denoted by $\mathcal{R}_0 = \rho(FV^{-1})$, (where ρ is the spectral radius of the next generation matrix, FV^{-1})) is given in equation (2.1) below. The matrices F (for the new infection terms) and V (for the remaining transition terms), associated with the model (2.1) , are given, respectively, by:

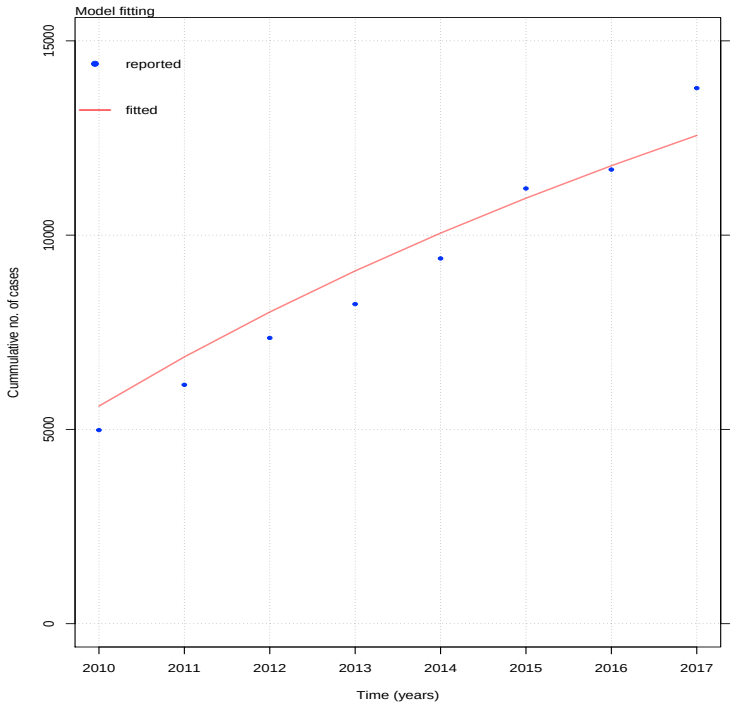

Figure 1: The fitting results from the model (2.1) using the parameter values from Table 2 and the following initial conditions: $S_a = 10^8$, $S_n = 6 \times 10^7$, $E = 4 \times 10^5$, $C = 5600$, $I = 4983$ and $R = 2500$. The vertical axes indicate the cumulative number of MCM cases in Nigeria from 2010 to 2017.

$$F = \begin{bmatrix} 0 & \frac{\beta(S_n^0+\alpha S_a^0)}{N^0} & \frac{\beta(S_n^0+\alpha S_a^0)}{N^0} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \; V = \begin{bmatrix} J_1 & 0 & 0 \\ -\phi a_3 & J_2 & 0 \\ -k\phi & -\tau & J_3 \end{bmatrix}, \tag{3.1}$$

where, $N^0 = \frac{\pi}{\mu}$, $S_a^0 = \frac{\omega\pi}{\mu}$, $S_n^0 = \frac{(1-\omega)\pi}{\mu}$, $d_1 = \frac{\beta(\alpha S_a^0+S_n^0)}{N^0}$, $a_1 = 1-\omega$, $a_2 = 1-\rho$, $a_3 = 1-k$, $J_1 = \phi+\mu$, $J_2 = \tau+\sigma+\mu$, $J_3 = \theta+\delta+\mu$, and $J_4 = \gamma+\mu$. Therefore, the basic reproduction number, $\mathcal{R}_0$, is given by

$$\mathcal{R}_0 = \frac{d_1\phi[kJ_2 + d_2(J_3+\tau)]}{J_1J_2J_3} = \frac{\phi\beta(\alpha\omega+a_1)[kJ_2+a_3(J_3+\tau)]}{(\phi+\mu)(\tau+\sigma+\mu)(\theta+\delta+\mu)}. \tag{3.2}$$

The basic reproduction number, $\mathcal{R}_0$, is defined as the average number generated by a single MCM infected human if placed in a completely susceptible population [23, 51]. The result below follows from Theorem 2 of [51].

Theorem 3.1. *The DFE, E_1^0, of the model (2.1), is locally-asymptotically stable (LAS) in Ω if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.*

The epidemiological interpretation of the $\mathcal{R}_0$ is that if $\mathcal{R}_0 < 1$, the disease will dies out in time

provided the initial sizes of the infected sub populations of the system (2.1) are sufficiently small so that the initial state of the system is in basin of attraction of the DFE. Note that this result includes the possible situation of the coexistence of DFE with a stable EE.

3.2 Endemic equilibrium (EE)

The EE of the model (2.1) are the steady states where the disease may persist in the population, that is when at least one of the infected class is non-empty.

Let $E_2^2* = (S_a^*, S_n^*, E^*, C^*, I^*, R^*)$ be the EE for the system (2.1), in terms of the force of infection λ^* is given by

$$\begin{aligned} S_a^* &= \frac{J_2J_3J_4\omega\pi + \rho\gamma[a_3J_3\phi\sigma + \theta(k\phi J_2 + a_3\tau\phi)]E^*}{J_2J_3J_4(\alpha\lambda^* + \mu)} \\ S_n^* &= \frac{J_2J_3J_4a_1\pi + a_2\gamma[a_3J_3\phi\sigma + \theta(k\phi J_2 + a_3\tau\phi)]E^*}{J_2J_3J_4(\lambda^* + \mu)} \\ C^* &= \frac{a_3\phi E^*}{J_2} \\ I^* &= \frac{[k\phi J_2 + a_3\tau\phi]E^*}{J_2J_3} \\ R^* &= \frac{a_3J_3\phi\sigma + \theta(k\phi J_2 + a_3\tau\phi)]E^*}{J_2J_3J_4}. \end{aligned} \tag{3.3}$$

Here,

$$\lambda^* = \frac{\beta(C^* + I^*)}{N^*}, \tag{3.4}$$

and

$$N^* = S_a^* + S_n^* + E^* + C^* + I^* + R^*. \tag{3.5}$$

Eqn (3.4) can now be written as

$$S_a^* + S_n^* + E^* + (1 - \frac{\beta}{\lambda^*})C^* + (1 - \frac{\beta}{\lambda^*})I^* + R^* = 0. \tag{3.6}$$

Hence, by substituting equation (3.3) into equation (3.6), we obtained the EE of the model (2.1) which correspond to positive solutions of the equation (3.6).

3.3 Backward bifurcation analysis

The backward bifurcation (BB) phenomenon has been shown to exist in several modeling studies [4, 17, 21, 39, 58]. The existence of BB in the current study reveal that the classical epidemiological requirement of having $\mathcal{R}_0 < 1$ is although necessary, but no longer sufficient for effective control of the MCM infections. The analysis of BB for the model (2.1) will be explored in the next sub-section.

Table 2: Values and ranges of the parameters of the model (2.1)

Parameter	Value [Range]	Units/Remarks	Sources
γ	0.00023 $[0.0001, 0.00035]$	Day^{-1}	[27]
η	0.000296 $[0.0001, 0.0003523]$	Day^{-1}	[23]
υ	0.0002 $[0, 1]$	Day^{-1}	[23]
ξ	0.002 $[0.001, 0.0035]$	Day^{-1}	[58]
β	0.343 $[0.137, 0.548]$	Day^{-1}	[31]
μ	5.5×10^{-5} $[1 \times 10^{-5}, 1 \times 10^{-4}]$	Day^{-1}	[31]
δ	0.0142 $[0.01, 0.018]$	Day^{-1}	[31]
τ	0.071 $[0.000274, 0.142]$	Day^{-1}	[31]
θ	0.142 $[0.12, 0.23]$	Day^{-1}	[31]
k	0.45 $[0.001, 0.9]$	Nil	[31]
π	540 $[510, 570]$	$\text{person}\cdot\text{day}^{-1}$	[39]
σ	0.724 $[0.00274, 0.142]$	Day^{-1}	[5, 31]
ω	0.5 $[0, 1]$	Nil	Assumed
α	0.5 $[0, 1]$	Nil	Assumed
ρ	0.45 $[0.001, 0.9]$	Day^{-1}	Assumed
ϕ	0.0085 $[0.0045, 0.012]$	Day^{-1}	Assumed

3.3.1 Existence of endemic equilibria (EE)

After substituting equation (3.3) into equation (3.6) and simplifying, we obtained the following quadratic equation in terms of the λ^*,

$$b_1\lambda^{*2} + b_2\lambda^* + b_3 = 0, \tag{3.7}$$

where

$$\begin{aligned}
b_1 =& [(((J_3+\tau)\,a_3 + kJ_2)\,J_4 + (\sigma\,J_3 + \theta\,\tau)\,a_3 + \theta\,kJ_2)\,\phi + J_2J_4J_3]\,\alpha(a_1+\omega),\\
b_2 =& ((((-\beta(J_3+\tau)J_4 - \rho\gamma(\sigma J_3 + \theta\tau))a_1 + \omega((J_3+\tau)(-\beta+\mu)J_4 + (\sigma J_3+\theta\tau)(\mu+a_2\gamma)))\\
& a_3 + ((-\gamma\rho\theta - \beta J_4)a_1 + \omega((-\beta+\mu)J_4 + \theta(\mu+a_2\gamma)))kJ_2)\phi + J_2(J_1a_1 + \omega\mu)J_3J_4)\alpha +\\
& (((\mu(J_3+\tau)J_4 + (\sigma J_3+\theta\tau)(\rho\gamma+\mu))a_1 - \gamma a_2\omega(\sigma J_3+\theta\tau))a_3 +\\
& ((J_4\mu + \theta(\rho\gamma+\mu))a_1 - \gamma a_2\omega\theta)kJ_2)\phi + J_2(\omega J_1 + \mu a_1)J_3J_4, \quad \text{and}\\
b_3 =& \mu J_4[J_1J_2J_3 - \phi\beta((J_3+\tau) + kJ_2)(\alpha\omega + a_1)] = \mu J_1J_2J_3J_4[1-\mathcal{R}_0].
\end{aligned}$$

Thus, the following theorem 3.2 is established.

Theorem 3.2. *The model (2.1) has*

i) a unique endemic equilibrium (EE), if $b_3 < 0$;

ii) a unique EE, if $b_2 < 0$ and $b_3 = 0$;

iii) two EEs, if $b_2 < 0$, $b_3 > 0$ *and* $\Delta > 0$*; and*

iv) no EE otherwise.

If $\alpha > 0$, the proof is straightforward to see from the properties of the roots of a quadratic equation. For $\alpha = 0$, it is easy to verify the statements by inspection. Thus, b_1 is always positive and b_3 is positive or negative depending on the value of $\mathcal{R}_0$ less than, greater than or equal to one. It is clear from Case (i) of Theorem 3.2 that the model (2.1) has a unique EE whenever $\mathcal{R}_0 > 1$. Additionally, the possibility of BB, where a stable DFE coexists with a stable EE when $\mathcal{R}_0 < 1$, in model (2.1) is highlighted by Case (iii) of Theorem 3.2 [4, 9, 17, 18, 21, 22, 34]). To check this phenomenon in model (2.1), the discriminant Δ of equation (3.6), is set to be zero, and solved for the critical value of $\mathcal{R}_0$, denoted by $\mathcal{R}_{02}^c$, given by

$$\mathcal{R}_{02}^c = 1 - \frac{b_2^2}{4b_1\mu J_1 J_2 J_3 J_4}. \tag{3.8}$$

Thus, the BB would occurs for the values of $\mathcal{R}_{02}^c$ such that $\mathcal{R}_{02}^c < \mathcal{R}_0 < 1$. This is illustrated in Fig.1 by simulating the model with the following set of parameter values. Please note that the choice of parameters are only for the demonstration purpose, which may not necessarily be practical reasonable: $\pi = 540$, $\beta = 0.241$, $\gamma = 0.00023$, $\mu = 0.00001$, $\delta = 0.0142$, $\tau = 0.071$, $\theta = 0.142$, $\sigma = 0.724$, $k = 0.81$, $\omega = 0.98$, $\alpha = 0.56$, $\rho = 0.45$ and $\phi = 0.085$ (see Table 2 for the units of the parameters). So that, $b_1 = 0.00001281532644$, $b_2 = -6.10753290 \times 10^{-10}$, $b_3 = 1.681514704 \times 10^{-15}$, $\mathcal{R}_{02}^c = 0.8426220063$ and $\mathcal{R}_0 = 0.9636333993$ (that is, $\mathcal{R}_{02}^c < \mathcal{R}_0 < 1$). The associated BB diagram is shown in Fig. 2.

Thus, we have the following lemma.

Lemma 3.3. *The model* (2.1) *undergoes backward bifurcation (BB) when Case (iii) of Theorem 3.2 holds and* $\mathcal{R}_{02}^c < \mathcal{R}_0 < 1$.

The epidemiological importance of the existence of BB in MCM is that the classical requirement of having $\mathcal{R}_0 < 1$ is, although necessary, no longer sufficient for the disease control of MCM. In such a case, disease eradication would depend on the initial sizes on the sub-populations of the model (2.1). In addition, the BB phenomenon has been shown to exists in several modeling studies, see, for instance [4, 17, 21, 39, 58]. To the author's knowledge, the BB phenomenon is the first time shown to exist in the MCM transmission dynamics. Furthermore, the causes of the existence of BB phenomenon in the model (2.1) are explored below.

3.3.2 Non-existence of backward bifurcation: special cases

The following results highlight the impossibility of backward bifurcation in the model (2.1), since no endemic equilibrium exists in each case when the basic reproduction number is less than or equal to one (since for BB to occur there must be at least two endemic equilibria when basic reproduction number is less than or equal to one). A global stability results is established for the DFE in each case(to rule out the occurrence of the BB completely), and is given below.

Case 1: $(\omega = \alpha = 1)$

In this case, the basic reproduction number $(\mathcal{R}_0)$ can now be written as

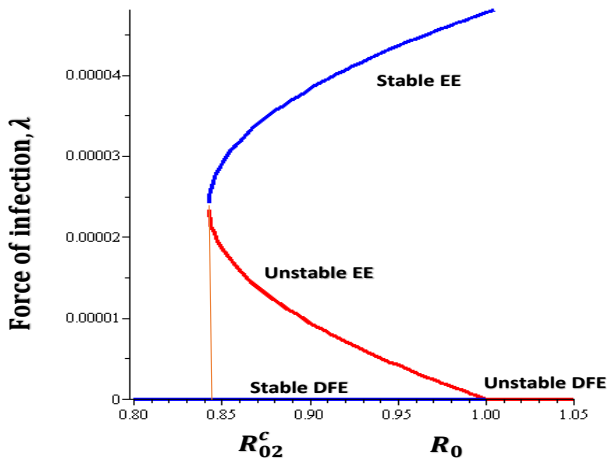

Figure 2: Bifurcation diagram of the model (2.1). The parameters values of the model (2.1) can be found in Table 2 with $\beta = 0.241$, $\mu = 0.00001$, $k = 0.81$, $\omega = 0.98$ and $\alpha = 0.56$ (so that, $b_1 = 0.00001281532644$, $b_2 = -6.10753290\times10^{-10}$, $b_3 = 1.681514704\times10^{-15}$, and $\mathcal{R}_{02}^c = 0.8426220063 < \mathcal{R}_{02} = 0.9636333993 < 1$).

$$\mathcal{R}_0^1 = \frac{\phi\beta[kJ_2 + a_3(J_3+\tau)]}{J_1J_2J_3}, \tag{3.9}$$

and the coefficients b_1, b_2 and b_3 are now given by

$$b_1 = [(((\tau+J_3)a_3 + kJ_2)J_4 + (J_3\sigma + \theta\tau)a_3 + \theta kJ_2)\phi + J_2J_3J_4],$$
$$b_2 = [((kJ_2 + a_3(J_3+\tau))J_4 + (J_3\sigma+\theta\tau)a_3 + \theta kJ_2)\phi + J_2J_3J_4)\mu] + [J_1J_2J_3J_4(1-\mathcal{R}_0^1)],$$
$$b_3 = \mu J_1J_2J_3J_4(1-\mathcal{R}_0^1).$$

Thus, following Theorem 3.2, there is no EE when $\mathcal{R}_0^1 \leq 1$. Therefore, the following Theorem is established, and the proof is given in Appendix A1.

Theorem 3.4. *The DFE of the model (2.1) is GAS on Ω whenever $\mathcal{R}_0^1 \leq 1$ provided that*

$$(1-\frac{\lambda}{\lambda^*})(1-\frac{I\lambda^*}{I^*\lambda}) \geq 0. \tag{3.10}$$

Case 2: $(\alpha = \omega = 0)$

In this case, the basic reproduction number $(\mathcal{R}_0)$ can now be written as

$$\mathcal{R}_0^2 = \frac{\phi\beta[kJ_2 + a_3(J_3+\tau)]}{J_1J_2J_3} \tag{3.11}$$

where the coefficients b_1, b_2 and b_3 are now given by
$b_1 = 0$, $b_2 = [(((kJ_2 + a_3(J_3 + \tau))J_4 + (J_3\sigma + \theta\tau)a_3 + \theta k J_2)\phi + J_2J_3J_4)] + [((\theta\tau + \sigma J_3)a_3 + \theta k J_2)\phi\rho\gamma]$,
$b_3 = \mu J_1J_2J_3J_4(1 - \mathcal{R}_0^2)$.

Thus, following Theorem 3.2, there is no EE when $\mathcal{R}_0^2 \leq 1$. Hence, a similar results to Theorem 3.4 can also be established, and the proof is similar. Therefore, the analysis shows that the two parameters ω and α are the causes of the BB, which are related to the lack of sufficient medical resources in a community.

4 Numerical results and sensitivity analysis

4.1 Numerical simulation

In this section, we give a numerical example to demonstrate our results for the model (A-1) which is a special case of the model (2.1) with parameters $\omega = \alpha = 1$, all other parameters have the same biological meaning as in the Table (1) .

In Fig. 3, we consider the case when $\mathcal{R}_0^1 = 0.9659358382 < 1$, the parameter values are the same with Table 2, for different initial conditions. The dynamics of the model (A-1) with $\mathcal{R}_0^1 < 1$ is presented in Fig. (3) (a-d) which shows that the system (A-1) has a DFE and it is globally asymptotically stable whenever $\mathcal{R}_0^1 < 1$ which supports the result stated in Theorem 3.4.

In Fig 4, we consider the case when $\mathcal{R}_0^1 = 1.900120600 > 1$, the parameter values are $\beta = 0.6343$, $\phi = 0.060085$ and the rest are the same with Table (2), for different initial conditions. The dynamics of the model (A-1) with $\mathcal{R}_0^1 > 1$ is presented in Fig. 4 (a-d) which shows that the system (A-1) has a DFE which is unstable whenever $\mathcal{R}_0^1 > 1$.

4.2 Sensitivity analysis

Following previous studies [19, 48, 57, 61, 62], we adopted the partial rank correlation coefficient (PRCC) for sensitivity analysis. The PRCCs of the basic reproduction number and (infection) attack rate of the model (2.1) presented with estimated parameters in Fig. 5.

5 Discussion

We proposed a model to explore the impact of a medical resource (MR) on the MCM transmission dynamics. The model (2.1) considers two different groups of population (i.e., with and without adequate MR). Rigorous analyses were carried out to gain qualitative insights into the MCM dynamical behaviors. The main epidemiological and theoretical findings of this paper are discussed below.
(i) The basic reproduction number, $\mathcal{R}_0$ of the model were given in Eqns. (3.2), which highlighted that the asymptotic behaviors of the model (2.1) are determined by its reproductive number.
(ii) The model (2.1) has two kinds of equilibria, i.e., the disease-free equilibrium (DFE), where the disease is not present in the community; and the endemic equilibrium (EE), where the disease spread and persist in the populations.
(iii) The locally asymptotically stable (LAS) DFE exists for the model (2.1) if $\mathcal{R}_0 < 1$; and the

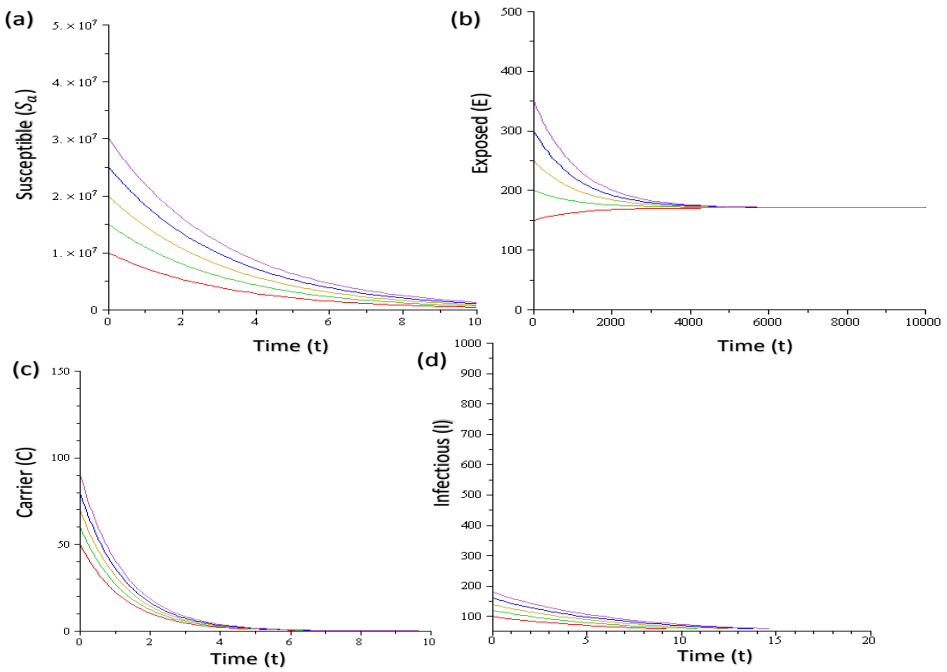

Figure 3: Time series plot of the model (A-1) with different initial conditions (represented by the different colours). The parameters values are given in Table 2 with $\mathcal{R}_0^1 = 0.9659358382 < 1$; (a) the number of susceptible, (b) the number of exposed, (c) the number of carrier, (d) the number of infectious.

DFE becomes unstable whenever $\mathcal{R}_{0>}1$. Further, the backward bifurcation phenomenon exists for the model (2.1), a situation where the stable DFE coexists with the stable EE even if the $\mathcal{R}_0 < 0$. By implication, in backward bifurcation settings, if $\mathcal{R}_0 < 1$, the disease control depends on the initial sizes (i.e., numbers) of individuals in each compartment. In addition, the parameters ω and α plays a crucial role to guarantee the existence of the phenomenon of backward bifurcation for the model (2.1). The results from section 3.3.2 highlights that the backward bifurcation can be taken away if $\mathcal{R}_0 < \mathcal{R}_{02}^c < 1$, or $\omega = \alpha = 1$ or $\omega = \alpha = 0$. Therefore, there is a need to prevent the occurrence of backward bifurcation phenomenon, otherwise, the disease may persist even if $\mathcal{R}_0 < 1$. This could be achieved by providing adequate MR in the community, especially in the area where the MCM is endemic (e.g., northern Nigeria). In addition, if the BB is inevitable, then the $\mathcal{R}_0$ needs to be sufficiently decreased to be smaller than the sub-threshold quantity (i.e., the critical reproduction ratio), $\mathcal{R}_{02}^c$. This could also be realized by providing sufficient MR in the endemic areas, especially during the MCM epidemics.

(v) We have shown that, under the special scenario (where $\omega = \alpha = 1$ 0r $\omega = \alpha = 0$), the DFE of the model (2.1) is GAS whenever $\mathcal{R}_0^1 \leq 1$ (or $\mathcal{R}_0^2 \leq 1$). This further shows that the MCM disease always dies out as in line with Theorem 3.4. Numerical simulations also show that (see Figs. 3 and 4) the changing dynamics converge to the steady-state.

(vi) The sensitivity analyses also reveal that the parameters β, k, and α are remarkably sensitive for the model (2.1). This, further, suggests that the aforementioned parameters (which are the transmission

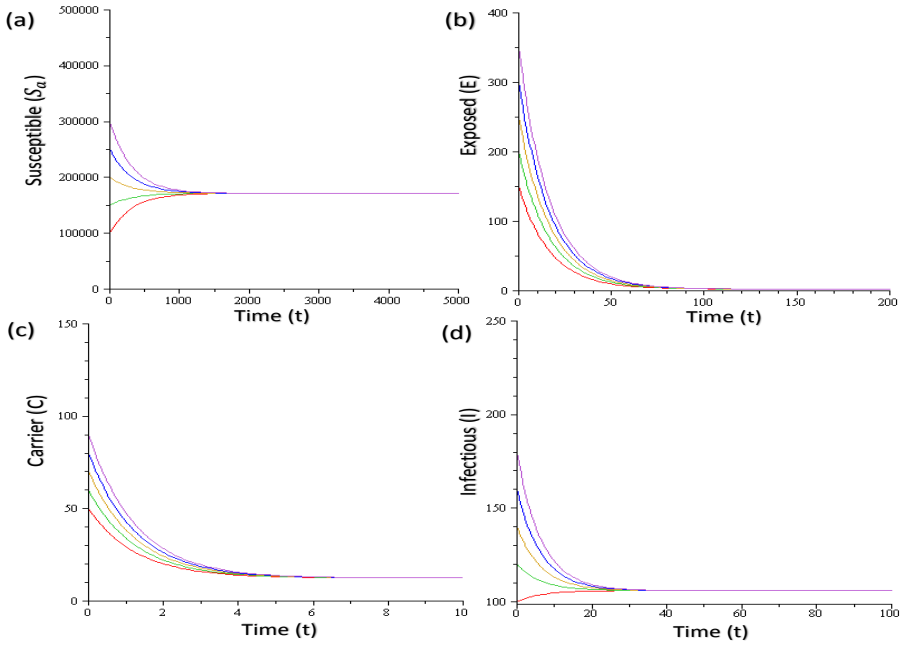

Figure 4: Time series plot of the model (A-1) with different initial conditions (represented by the different colours). The parameters values are given in Table 2 with $\beta = 0.6343$ and $\phi = 0.060085$ so that $\mathcal{R}_0^1 = 1.900120600 > 1$; (a) the number of susceptible, (b) the number of exposed, (c) the number of carrier, (d) the number of infectious.

parameters) are highly dependent on the availability of MR, which should be given adequate emphasis in controlling the MCM in a community.

In summary, our analysis shows that the model (2.1) exhibits the phenomenon of backward bifurcation (BB) (a situation where a stable DFE coexist with a stable EE even if the $\mathcal{R}_0 < 1$), and this phenomenon of BB can be removed using one of the following methods. Firstly, the BB can be removed by reducing the basic reproduction ratio ($\mathcal{R}_0$) below the sub-threshold quantity ($\mathcal{R}_{02}^c$) to prevent the critical range (i.e., $\mathcal{R}_{02}^c < \mathcal{R}_0 < 1$). Secondly, the BB can also be removed by making the model parameters $\omega = \alpha = 1$ (or $= 0$) as guaranteed by Theorem 3.4. This second condition (or method) could be achieved by the availability of sufficient MR in the community, especially in the region where the MCM is endemic. Finally, our model can be extended to incorporate seasonality which may show additional dynamical features, this could be achieved when there are sufficient seasonality data available. To incorporate the active particles approach to population dynamics is one of the options, see [20], which provides appropriate tools to account for heterogeneity and is an essential feature of biological systems; migration and diffusion models over networks can describe key features of space dynamics, and also the Darwinian mutations and selection approaches which have a central role in the evolution of chronic disease like cancer [8, 20].

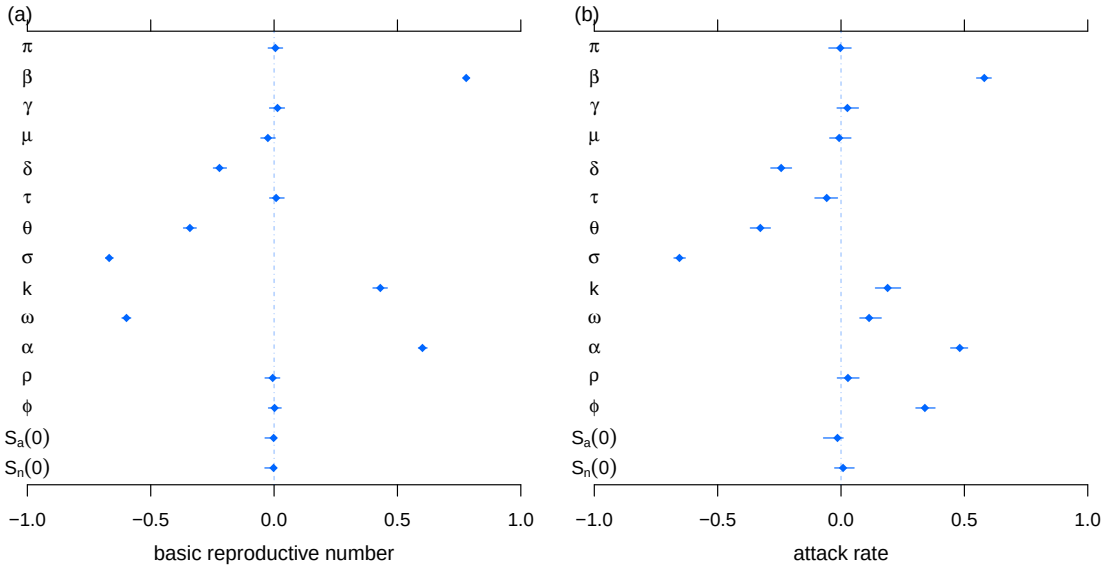

Figure 5: The partial rank correlation coefficient (PRCC) of the model (2.1). The dots are the PRCC estimation; and the bars are the 95% confidence intervals (CI). The values and ranges of the model parameters are summarized in Table 2.

Declarations

Ethics approval and consent to participate Since no personal data was collected, the ethical approval or individual consent was not applicable.

Availability The epidemic time series data were obtained from the World Health Organization [56], which is online for free-access.

Consent for publication Not applicable.

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Conflict of Interests The authors declare that they have no competing interests.

Authors' Contributions SSM and DH conceived and carried out the study. SSM conducted the analyses, discussed the results and drafted the first manuscript. SSM, SZ, NH, AGH and DH revised the manuscript. All authors gave final approval for publication.

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Appendices

A1 The Proof of theorem 3.4

Proof. Since $\omega = \alpha = 1$ which means that there are no new recruits from the S_n class, then $S_n \to 0$ as $t \to \infty$. Thus, the model (2.1) reduced to the following;

$$\begin{aligned}
\frac{dS_a}{dt} &= \pi + \gamma R - \lambda S_a - \mu S_a, \\
\frac{dE}{dt} &= \lambda S_a - J_1 E, \\
\frac{dC}{dt} &= a_3 \phi E - J_2 C, \\
\frac{dI}{dt} &= k\phi E + \tau C - J_3 I, \\
\frac{dR}{dt} &= \sigma C + \theta I - J_4 R.
\end{aligned} \tag{A-1}$$

We define a Lyapunov function as follows:

$$\begin{aligned}
V(t) =& g_1(S_a - S_a^* - S_a^* \ln \frac{S_a}{S_a^*}) + g_2(E - E^* - E^* \ln \frac{E}{E^*}) + g_3(C - C^* - C^* \ln \frac{C}{C^*}) + \\
& g_4(I - I^* - I^* \ln \frac{I}{I^*}),
\end{aligned} \tag{A-2}$$

where $g_1 = g_2 = g_4 = 1$, $g_3 = \frac{\tau C^*}{a_3 \phi E^*}$.

Thus, the Lyapunov derivative computed along solutions of the systems (A-1) is given by

$$\dot{V}(t) = g_1(1 - \frac{S_a^*}{S_a})\dot{S}_a + g_2(1 - \frac{E^*}{E})\dot{E} + g_3(1 - \frac{C^*}{C})\dot{C} + g_4(1 - \frac{I^*}{I})\dot{I}. \tag{A-3}$$

Following the previous studies [42, 46, 58]. For the function $v(x) = 1 - x + \ln x$, then, if $x > 0$ it leads to $v(x) \leq 0$. And if $x = 1$, then $v(x) = 0$. So that $x - 1 \geq \ln(x)$ for any $x > 0$. Then, we have that,

$$\begin{aligned}
(1 - \frac{S_a^*}{S_a})\dot{S}_a &= (1 - \frac{S_a^*}{S_a})[\pi + \gamma R - \lambda S_a - \mu S_a] \\
&= (1 - \frac{S_a^*}{S_a})[\lambda^* S_a^* + \mu S_a^* - \lambda S_a - \mu S_a] \\
&\leq \lambda^* S_a^* (1 - \frac{S_a^*}{S_a})(1 - \frac{\lambda S_a}{\lambda^* S_a^*}) \\
&= \lambda^* S_a^* [1 - \frac{\lambda S_a}{\lambda^* S_a^*} - \frac{S_a^*}{S_a} + \frac{\lambda}{\lambda^*}],
\end{aligned} \tag{A-4}$$

and

$$
\begin{aligned}
(1-\frac{E^*}{E})\dot{E} &= (1-\frac{E^*}{E})[\lambda S_a - J_1 E] \\
&= (1-\frac{E^*}{E})[\lambda S - \lambda^* S^* \frac{E}{E^*}] \\
&= \lambda^* S_a^* (1-\frac{E^*}{E})(\frac{\lambda S_a}{\lambda^* S_a^*} - \frac{E}{E^*}) \\
&= \lambda^* S_a^* [\frac{\lambda S_a}{\lambda^* S_a^*} - \frac{E}{E^*} - \frac{\lambda S_a E^*}{\lambda^* S_a^* E} + 1],
\end{aligned}
\tag{A-5}
$$

and

$$
\begin{aligned}
(1-\frac{C^*}{C})\dot{C} &= (1-\frac{C^*}{C})[a_3\phi E - J_2 C] \\
&= (1-\frac{C^*}{C})[a_3\phi E - a_3\phi E^* \frac{C}{C^*}] \\
&= a_3\phi E^* (1-\frac{C^*}{C})[\frac{E}{E^*} - \frac{C}{C^*}] \\
&= a_3\phi E^* [\frac{E}{E^*} - \frac{C}{C^*} - \frac{C^* E}{C E^*} + 1],
\end{aligned}
\tag{A-6}
$$

and

$$
\begin{aligned}
(1-\frac{I^*}{I})\dot{I} &= (1-\frac{I^*}{I})[k\phi E + \tau C - J_3 I] \\
&= (1-\frac{I^*}{I})[k\phi E + \tau C - (k\phi E^* + \tau C^*)\frac{I}{I^*}] \\
&= (1-\frac{I^*}{I})[k\phi E - k\phi E^* \frac{I}{I^*} + \tau C - \tau C^* \frac{I}{I^*}] \\
&= k\phi E^* (1-\frac{I^*}{I})[\frac{E}{E^*} - \frac{I}{I^*}] + \tau C^* (1-\frac{I^*}{I})[\frac{C}{C^*} - \frac{I}{I^*}] \\
&= k\phi E^* [\frac{E}{E^*} - \frac{I}{I^*} - \frac{I^* E}{I E^*} + 1] + \tau C^* [\frac{C}{C^*} - \frac{I}{I^*} - \frac{I^* C}{I C^*} + 1].
\end{aligned}
\tag{A-7}
$$

Substituting equations (A-4)–(A-7) into equation (A-3), we have

$$
\begin{aligned}
\dot{V}(t) \leq & g_1 \lambda^* S_a^* [2 - \frac{S_a^*}{S_a} - \frac{E}{E^*} - \frac{\lambda S_a E^*}{\lambda^* S_a^* E} + \frac{\lambda}{\lambda^*}] + g_2 a_3 \phi E^* [\frac{E}{E^*} - \frac{C}{C^*} - \frac{C^* E}{C E^*} + 1] + \\
& g_3 [k\phi E^* [\frac{E}{E^*} - \frac{I}{I^*} - \frac{I^* E}{I E^*} + 1] + \tau C^* [\frac{C}{C^*} - \frac{I}{I^*} - \frac{I^* C}{I C^*} + 1]].
\end{aligned}
\tag{A2-8}
$$

By direct calculation, we have that,

$$
\begin{aligned}
&[2-\frac{S_a^*}{S_a}-\frac{E}{E^*}-\frac{\lambda S_a E^*}{\lambda^* S_a^* E}+\frac{\lambda}{\lambda^*}]\\
&=[-(1-\frac{\lambda}{\lambda^*})(1-\frac{I\lambda^*}{I^*\lambda})+3-\frac{S_a^*}{S_a}-\frac{\lambda S_a E^*}{\lambda^* S_a^* E}-\frac{I\lambda^*}{I^*\lambda}-\frac{E}{E^*}+\frac{I}{I^*}]\\
&\leq[-(\frac{S_a^*}{S_a}-1)-(\frac{\lambda S_a E^*}{\lambda^* S_a^* E}-1)-(\frac{I\lambda^*}{I^*\lambda}-1)-\frac{E}{E^*}+\frac{I}{I^*}]\\
&\leq[-\ln(\frac{S_a^*}{S_a}\frac{\lambda S_a E^*}{\lambda^* S_a^* E}\frac{I\lambda^*}{I^*\lambda})-\frac{E}{E^*}+\frac{I}{I^*}]\\
&=[\frac{I}{I^*}-\ln(\frac{I}{I^*})+\ln(\frac{E}{E^*})-\frac{E}{E^*}].
\end{aligned}
\tag{A-9}
$$

It is easy to see that,

$$
\frac{E}{E^*}-\frac{C}{C^*}-\frac{C^*E}{CE^*}+1\leq\frac{E}{E^*}-\ln(\frac{E}{E^*})+\ln(\frac{C}{C^*})-\frac{C}{C^*},
\tag{A-10}
$$

similarly,

$$
\frac{E}{E^*}-\frac{I}{I^*}-\frac{I^*E}{IE^*}+1\leq\frac{E}{E^*}-\ln(\frac{E}{E^*})+\ln(\frac{I}{I^*})-\frac{I}{I^*},
\tag{A-11}
$$

and

$$
\frac{C}{C^*}-\frac{I}{I^*}-\frac{I^*C}{IC^*}+1\leq\frac{C}{C^*}-\ln(\frac{C}{C^*})+\ln(\frac{I}{I^*})-\frac{I}{I^*}.
\tag{A-12}
$$

Thus,

$$
\begin{aligned}
\dot{V}(t)\leq&(k\phi E^*+\tau C^*)\lambda^* S_a^*[\frac{I}{I^*}-\ln(\frac{I}{I^*})+\ln(\frac{E}{E^*})-\frac{E}{E^*}]+\\
&\tau C^*\lambda^* S_a^*[\frac{E}{E^*}-\ln(\frac{E}{E^*})+\ln(\frac{C}{C^*})-\frac{C}{C^*}]+\\
&k\phi E^*\lambda^* S_a^*[\frac{E}{E^*}-\ln(\frac{E}{E^*})+\ln(\frac{I}{I^*})-\frac{I}{I^*}]+\\
&\tau C^*\lambda^* S_a^*[\frac{C}{C^*}-\ln(\frac{C}{C^*})+\ln(\frac{I}{I^*})-\frac{I}{I^*}].
\end{aligned}
\tag{A-13}
$$

Equations (A-4)-(A-13) ensure that $\dot{V(t)}\leq 0$. Furthermore, the equality $\frac{dV}{dt}=0$ holds only if $S_a=S_a^*$, $E=E^*$, $C=C^*$, $I=I^*$, and $R=R^*$. Thus, the endemic equilibrium (EE) state, E^{**}, is the only positive invariant set to the system (3.9) contained entirely in $\{(S_a,E,C,I,R)\in\Omega: S_a=S_a^*,E=E^*,C=C^*,I=I^*,R=R^*\}$. Therefore, it follows from the LaSalle's invariance principle [32] that every solutions to the equations (A-1) with initial conditions in Ω converge to endemic equilibrium point, E^{**}, as $t\to\infty$. Hence, the positive endemic equilibrium is globally asymptotically stable.

A2 MCM reported cases time series in Nigeria

The numbers of MCM cases time series in Nigeria used to fit the model (2.1) are summarized in Table A1.

Table A1: The MCM reported cases time series in Nigeria from 2010 to 2017 [56]

Year	No. of cases	Cumulative sum
2010	4983	4983
2011	1165	6148
2012	1206	7354
2013	871	8225
2014	1175	9400
2015	1801	11201
2016	486	11687
2017	2097	13784