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Mathematical model of measles transmission dynamics using real data from Nigeria

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ABSTRACT

Measles is a highly contagious and life-threatening disease caused by a virus called *morbillivirus*, despite the availability of a safe and cost-effective vaccine, it remains a leading cause of death, especially in children. Measles spreads easily from person to person via infected people's coughs and sneezes. It can also be transmitted through direct contact with the mouth or contaminated surfaces. To have a better knowledge of measles epidemiology in Nigeria, we develop a deterministic mathematical model to study the transmission dynamics of the disease in the population. The boundary of the model solution is performed, both equilibrium points are calculated, and the basic reproduction number $\mathcal{R}_0$ is determined. We have proved that when $\mathcal{R}_0 < 1$, the disease-free equilibrium point is both locally and globally stable. When $\mathcal{R}_0 > 1$, the endemic equilibrium point exists and is stable if it satisfies Routh–Hurwitz criteria. We demonstrate the model's effectiveness by using a real-life application of the disease spread in Nigeria. We fit the proposed model using available data from Nigeria Center for Disease Control (NCDC) from January to December 2020 to obtain the best fit, this help us to determine the accuracy of the proposed model's representation to the real-world data. We investigate the impact of vaccination rate and hospitalization of infected individuals on the dynamics of measles in the population. The result shows that the combined control strategies reduce the peak of infection faster than the single control strategy.

1. Introduction

Measles remains a vital universal public health issue, particularly in developing nations. Measles (also known as rubeola or morbilli) represents one of the very highly transmissible diseases triggered by the genus *Morbillivirus* within the family Paramyxoviridae [18,19]. Although effective vaccines against measles infection are readily available, yet measles

affects the mortality of children below five years of age [33], infecting ailing children in tens of millions yearly and resulting in deaths of about a million in number due primarily to intricate conditions that are coexistent with the disease examples of which are poor nourishment, diarrhea plus pneumonia [37]. Measles is transmitted by coughing and sneezing, contact with nasal or aerosol secretions, or near personal contact. It continues to remain highly infectious in the air or on the surface for up to two hours. Early symptoms include high-grade sore throat, cough, runny nose, blurry vision, and tiny white spots in the mouth; generally, 10–12 days after the infection appears. A later rash emerges, spreading downwards from the nose. The cycle of greatest infectiousness (meaning virus shedding) appears four days before the onset of rash and 4 days after the onset of rash. The average incubation period is 14 days, varying from 7 to 18 days [30]. In the real sense, some individuals who are vaccinated could still be vulnerable when the vaccination failed or their immunity caused by the vaccine waned. Although vaccination has reduced significantly global measles deaths by a 73% decrease between 2000 and 2018. Worldwide, measles is still prevalent in many developing countries, especially parts of Africa and Asia. Over 140,000 people died of measles in 2018. Between 2000 and 2018, global measles vaccination resulted in an 85 percent reduction in measles mortality, [15,24]. According to the World Health Organization (WHO), about 110,000 people died from measles especially children below the age of 6, despite the availability of a safe and efficient vaccine in 2017 [9]. Vaccination is one of the most effective public health interventions for lowering mortality rates and disease outbreaks; in Nigeria, it has been shown to save over three million lives each year. Vaccines work with the body's immune system to develop natural immunity against disease, lowering the chance of infection [36]. Measles is a vaccine-preventable disease that can be prevented with the MMR vaccine. The MMR vaccine protects children and adults against measles and is extremely safe. Two doses of the MMR vaccine are around 95% effective in preventing measles, while a single dose is approximately 92% effective. Mumps and rubella are also protected by MMR immunization. The MMR vaccine is also effective against is mumps and rubella [11] In Nigeria, measles is an endemic illness with repeated outbreaks at frequent intervals. Measles is spread throughout Nigeria at all times of the year, however, it is most prevalent during the dry season. According to a study conducted in Nigeria, measles accounted for up to 3% of hospital admissions, the majority of which resulted in measles complications. Nigeria is one of the few countries worldwide with a measles vaccine coverage of less than 40%. This is due to the country's low vaccine coverage [20]. There is no clear treatment for infection with measles. Although measles cases have a higher mortality rate that necessitates hospitalization, little is known about measles hospitalization and complications in the past few years. Measles can be dangerous in people of all ages, although children under the age of five and individuals over the age of 20 are more prone to develop complications. Ear infections and diarrhea are two common side effects. Pneumonia and encephalitis are serious complications. Measles can lead to a serious sickness that necessitates hospitalization. Even though there is no medication or cure for measles, the majority of people who contract the disease survive it. However, the majority of measles patients will feel severely ill for about a week, and up to 30% of those who have the disease will develop complications that may require hospitalization [12].

Measles cases have a high morbidity rate, necessitating hospitalization. Hence, further studies are needed to improve our understanding of the transmission dynamics, prevention, and management of measles. In the United States, one out of every five unvaccinated

people who contract measles is admitted to the hospital. One to three children in every 1000 who contract measles will die as a result of respiratory and neurologic disorders [10,14]. For several countries worldwide, measles remains prone to both economic and health issues despite the availability of the measles vaccine. Thus, a deeper and better understanding of measles transmission remains essential. The disease models help to understand the dynamics of the disease spread and prevention strategies.

Ashir et al. [3] conducted a systematic review of 124 children hospitalized with measles in Maiduguri Teaching Hospital, Nigeria, to determine the incidence of measles utilizing the length of hospital stay as a result of complications in hospitalized children with measles. The study found that treating measles and its sequelae might be a huge strain on medical resources in this part of Nigeria, especially unvaccinated young children and those from poor backgrounds. Other studies on measles hospitalization in Nigeria can be found in [8,16].

There has been an increasing interest in the use of deterministic compartmental models in recent decades to study the dynamics of measles and find measures to control and prevent the outbreak [6]. Bauch examined the implication of vaccination with regards to the effect of reaching herd immunity [23]. Mossong and Muller carried out a study on the modelling of measles re-emergence attributable to weakened immunity of vaccinated populations [38]. Zhang et al. examined the degree of the epidemic against the policy of discretional vaccination on Erdos-Renyi random graphs and Barabasi-Albert scale-free networks [22]. Momoh et al. designed a mathematical model to limit the spread of measles [17]. Fred et al. carried out a study on mathematical modelling on how vaccination curbs measles. In [1], the authors found that a wider gap between measles-infected and non-infected individuals is effective to control the spread of the disease. The impacts of the role of vaccination in controlling the spread of measles dynamics were investigated in [31,35] . Other notable contributions are in [2,4,5,7,21,26,27,29,34]. Many mathematical models have been introduced in recent years by many researchers to study the transmission dynamics of measles by considering different scenarios in the measles models, we observed that none investigated the effects of hospitalization of the infected individuals. The goal of this study is to investigate the impact of vaccination rate and hospitalization of infected individuals on the dynamics of measles in the population. The remaining part of the paper is organized as follows: Section 2 deals with the model's descriptions based on the epidemiology of individuals, in Section 3, we carried out the analysis of the measles model which includes, boundedness of the solutions, the equilibrium states, the basic reproduction number, and stability analysis. Section 4 deals with data fitting and numerical simulation, results of the findings and discussion are presented in Section 5, Section 6 is the conclusion.

2. Formulation of the model

In this section, we formulate a deterministic mathematical model on the transmission dynamics of measles. Based on the epidemiological status of individuals, the model subdivides the human population into six classes which are susceptible class $S(t)$, vaccinated class $V(t)$, exposed class $E(t)$, infected class $I(t)$, hospitalized class $H(t)$ and recovered class $R(t)$. Daily recruitment into the susceptible class is at a rate ϕ. Individuals in the susceptible class receive a vaccination at a rate τ and loss immunity at a vaccine wane rate ω. The transmission rate of susceptible individuals is α and the force of infection term is
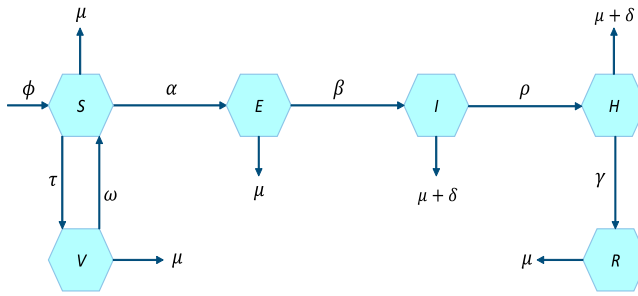
Figure 1. Flow chart of the measles model.

given as αSI, the progression from the exposed class to infected class is at a rate β. Infected individuals visit the hospital due to complications from measles at a rate ρ and recovered from complications from measles infection upon treatment at a rate γ. Natural mortality occurs in all the classes at a rate μ and the mortality caused by measles is denoted by δ. We have not considered the natural recovery rate from measles in this study. The model's flowchart and description of parameters are illustrated in Figure 1 and Table 1, respectively. The description above can be written in the form of a system of ordinary differential equations as

$$\left.\begin{aligned}
\frac{dS}{dt} &= \phi - \alpha SI + \omega V - (\tau + \mu)S, \\[1em]
\frac{dV}{dt} &= \tau S - (\mu + \omega)V, \\[1em]
\frac{dE}{dt} &= \alpha SI - (\mu + \beta)E, \\[1em]
\frac{dI}{dt} &= \beta E - (\mu + \delta + \rho)I, \\[1em]
\frac{dH}{dt} &= \rho I - (\gamma + \delta + \mu)H, \\[1em]
\frac{dR}{dt} &= \gamma H - \mu R.
\end{aligned}\right\} \tag{1}$$

3. Analysis of the model

3.1. Boundedness of the solution

Let the total population be $N = S(t) + V(t) + E(t) + I(t) + H(t) + R(t)$ then

$$\begin{aligned}
\frac{dN}{dt} &= \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dH}{dt} + \frac{dR}{dt} \\
&= \phi - \mu(S + V + E + I + H + R) - \delta I - \delta H. \tag{2}
\end{aligned}$$

Table 1. Description of the model variables and parameters.

Variable	Description
$S(t)$	Susceptible class
$V(t)$	Vaccinated class
$E(t)$	Exposed class
$I(t)$	Infected class
$H(t)$	Hospitalized class
$R(t)$	Recovered class
Parameter	**Description**
ϕ	Recruitment rate into susceptible class
μ	Natural death rate
δ	Measles death rate
τ	Rate of vaccinating susceptible class
ω	Vaccine wane rate
α	Transmission rate of susceptible individuals
β	Rate of progression from the exposed to infected class
ρ	Rate at which infected individuals visit hospital due to complications from measles
γ	Recovery rate

From (2), we have

$$\frac{dN}{dt} \leq \phi - \mu N \tag{3}$$

Integrating both sides of (3) yields

$$\int_0^t \frac{dN}{\phi - \mu N} \leq \int_0^t dt$$
$$-\frac{1}{\mu} \ln(\phi - \mu N) \mid_0^t \leq t. \tag{4}$$

Further simplification of (4) result in

$$N_t \leq \frac{\phi}{\mu} - \left[\frac{\phi - \mu N_0}{\mu} \right] e^{-\mu t}, \tag{5}$$

by taking $t \longrightarrow \infty$, then we obtain that $N_t \leq \frac{\phi}{\mu}$. This implies that the model in (1) can be studied in the feasible region

$$\Gamma = \left\{ (S, V, E, I, H, R) \in R^6 : N \leq \frac{\phi}{\mu} \right\}. \tag{6}$$

3.2. Disease-free equilibrium (DFE)

This happens when no infection occurs. Thus, we set E, I and H to zero in Equation (1), in the absence of infection and the resulting solution gives the disease-free equilibrium states that are given as

$$\mathcal{E}_0 = (S_0, V_0, E_0, I_0, H_0, R_0) = \left(\frac{(\mu + \omega)\phi}{(\mu + \omega + \tau)\mu}, \frac{\phi\tau}{(\mu + \omega + \tau)\mu}, 0, 0, 0, 0 \right). \tag{7}$$

3.3. Endemic equilibrium

This happens in the presence of infection that is, $\Gamma_{EE} = (S^*, V^*, C^*, I^*, H^*, R^*)$ population appears to have the infection. We set the left hand side of Equation (1) to zero for obtaining it. Therefore,

$$S^* = \frac{k_3 k_4}{\alpha \beta}, \tag{8}$$

$$V^* = \frac{\tau k_3 k_4}{\alpha \beta k_2}, \tag{9}$$

$$E^* = \frac{\alpha \beta k_2 \phi - k_1 k_2 k_3 k_4 + k_3 k_4 \omega \tau}{\alpha \beta k_2 k_3}, \tag{10}$$

$$I^* = \frac{\alpha \beta k_2 \phi - k_1 k_2 k_3 k_4 + k_3 k_4 \omega \tau}{\alpha k_2 k_3 k_4}, \tag{11}$$

$$H^* = \frac{\rho(\alpha \beta k_2 \phi + k_3 k_4 \omega \tau - k_1 k_2 k_3 k_4)}{\alpha k_2 k_3 k_4 k_5}, \tag{12}$$

$$R^* = \frac{\gamma(\alpha \beta k_2 \phi - k_1 k_2 k_3 k_4 + k_3 k_4 \omega \tau)}{\alpha \mu k_2 k_3 k_4}, \tag{13}$$

where $k_1 = \tau + \mu, k_2 = \mu + \omega, k_3 = \mu + \beta, k_4 = \mu + \delta + \rho$, and $k_5 = \gamma + \delta + \mu$.

3.4. The basic reproduction number

The basic reproductive number of a single infected person is a threshold that indicates the total number of potential diseases that have been developed into a completely susceptible population during its transmission period. F and V are the matrices created for new infection and transition terms respectively. Following the same method as in [31], the basic reproduction number is calculated as follows.

The new infection components are $E(t), I(t)$ and $H(t)$

$$\frac{dE}{dt} = \alpha SI - (\mu + \beta)E,$$
$$\frac{dI}{dt} = \beta E - (\rho + \mu + \delta)I,$$
$$\frac{dH}{dt} = \rho I - (\gamma + \delta + \mu)H, \tag{14}$$
$$f = \begin{pmatrix} \alpha SI \\ 0 \\ 0 \end{pmatrix}, \quad v = \begin{pmatrix} (\beta + \mu)E \\ (\rho + \mu + \delta)I - \beta E \\ (\gamma + \delta + \mu)H - \rho I \end{pmatrix}.$$

F and V are the Jacobian matrices which shall be computed at the DFE such that

$$F = \begin{pmatrix} 0 & \alpha S & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \mu + \beta & 0 & 0 \\ -\beta & \mu + \delta + \rho & 0 \\ 0 & -\rho & \gamma + \delta + \mu \end{pmatrix}$$

$$F(\mathcal{E}_0) = \begin{pmatrix} 0 & \alpha S_0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

$$V^{-1} = \begin{bmatrix} (\mu + \beta)^{-1} & 0 \\ \dfrac{\beta}{(\mu + \beta)(\mu + \delta + \rho)} & (\mu + \delta + \rho)^{-1} \\ \dfrac{\beta \rho}{(\mu + \beta)(\mu + \delta + \rho)(\gamma + \delta + \mu)} & \dfrac{\rho}{(\mu + \delta + \rho)(\gamma + \delta + \mu)} \end{bmatrix}$$

$$\begin{matrix} 0 \\ 0 \\ (\gamma + \delta + \mu)^{-1} \end{matrix}\Bigg]$$

$$F(\mathcal{E}_0)V^{-1} = \begin{pmatrix} \dfrac{\beta \alpha S_0}{(\mu + \beta)(\mu + \delta + \rho)} & \dfrac{\alpha S_0}{\mu + \delta + \rho} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

The basic reproduction number $\mathcal{R}_0$ is obtained by the spectral radius of the matrix $F(\mathcal{E}_0)V^{-1}$, given as

$$\mathcal{R}_0 = \frac{\beta \alpha S_0}{(\mu + \beta)(\mu + \delta + \rho)}, \tag{15}$$

where

$$S_0 = \frac{\phi(\mu + \omega)}{(\mu + \omega + \tau)\mu}.$$

3.5. Stability analysis of the model

Theorem 3.1: *The disease-free equilibrium point $(\mathcal{E}_0)$ is locally asymptotically stable if $\mathcal{R}_0 < 1$ otherwise it is unstable.*

Proof:

$$J(S, V, E, I, H, R)$$

$$= \begin{bmatrix} -\alpha I - \tau - \mu & \omega & 0 & -\alpha S & 0 & 0 \\ \tau & -\mu - \omega & 0 & 0 & 0 & 0 \\ \alpha I & 0 & -\mu - \beta & \alpha S & 0 & 0 \\ 0 & 0 & \beta & -\mu - \delta - \rho & 0 & 0 \\ 0 & 0 & 0 & \rho & -\gamma - \delta - \mu & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\mu \end{bmatrix}.$$

Then, we find the eigenvalues of the above Jacobian matrix at $\mathcal{E}_0$

$$det(J(\mathcal{E}_0) - \lambda I) = \begin{vmatrix} -\tau - \mu - \lambda & \omega & 0 & -\alpha S_0 \\ \tau & -\mu - \omega - \lambda & 0 & 0 \\ 0 & 0 & -\mu - \beta - \lambda & \alpha S_0 \\ 0 & 0 & \beta & -\mu - \delta - \rho - \lambda \\ 0 & 0 & 0 & \rho \\ 0 & 0 & 0 & 0 \end{vmatrix}$$

$$\begin{matrix} 0 & 0 \\ 0 & 0 \\ 0 & 0 \\ 0 & 0 \\ -\gamma - \delta - \mu - \lambda & 0 \\ \gamma & -\mu - \lambda \end{matrix} = 0.$$

We have that $\lambda_1 = -\mu < 0$, $\lambda_2 = -\gamma - \delta - \mu < 0$ and

$$(-\beta) \begin{vmatrix} -\tau - \mu - \lambda & \omega & -\alpha S_0 \\ \tau & -\mu - \omega - \lambda & 0 \\ 0 & 0 & \alpha S_0 \end{vmatrix} + (-\mu - \delta - \rho - \lambda)$$

$$\times \begin{vmatrix} -\tau - \mu - \lambda & \omega & 0 \\ \tau & -\mu - \omega - \lambda & 0 \\ 0 & 0 & -\mu - \beta - \lambda \end{vmatrix} = 0,$$

$$(-\beta\alpha S_0)[(-\tau - \mu - \lambda)(-\mu - \omega - \lambda) - \tau\omega] + (-\mu - \delta - \rho - \lambda)(-\mu - \beta - \lambda)$$

$$\times [(-\tau - \mu - \lambda)(-\mu - \omega - \lambda) - \tau\omega] = 0.$$

Then,

$$[(\mu + \delta + \rho + \lambda)(\mu + \beta + \lambda) - \beta\alpha S_0][(\tau + \mu + \lambda)(\mu + \omega + \lambda) - \tau\omega] = 0.$$

First consider the latter term, we have $\lambda^2 + (2\mu + \tau + \omega)\lambda + \mu(\tau + \mu + \omega) = 0$.

By the Routh–Hurwitz criteria, we then have $a_1 = 2\mu + \tau + \omega > 0$ and $a_2 = \mu(\tau + \mu + \omega) > 0$, which meet the criteria as required.

Next, consider the first term, we have $\lambda^2 + (2\mu + \delta + \rho + \beta)\lambda + (\mu + \delta + \rho)(\mu + \beta) - \beta\alpha S_0 = 0$, similarly, we have $a_1 = 2\mu + \delta + \rho + \beta > 0$ and

$$a_2 = (\mu + \delta + \rho)(\mu + \beta)\left[1 - \frac{\beta\alpha S_0}{(\mu + \delta + \rho)(\mu + \beta)}\right]$$

$$= (\mu + \delta + \rho)(\mu + \beta)[1 - \mathcal{R}_0].$$

Thus, $a_2 > 0$ when $\mathcal{R}_0 < 1$. Therefore, $\mathcal{E}_0$ is locally asymptotically stable when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$. ∎

Theorem 3.2: *The disease-free equilibrium point ($\mathcal{E}_0$) is globally asymptotically stable when $\mathcal{R}_0 < 1$.*

Proof: We begin the proof by using the boundary condition from (6), we have,

$$S + V_0 \le \frac{\phi}{\mu},$$

$$S \le \frac{\phi}{\mu} - \frac{\phi\tau}{\mu(\mu + \omega + \tau)}$$

$$= \frac{\phi(\mu + \omega + \tau) - \phi\tau}{\mu(\mu + \omega + \tau)}$$

$$= \frac{\phi(\mu + \omega)}{\mu(\mu + \omega + \tau)} = S_0.$$

Thus, $S(t) \le S_0$.

Next we define the Lyapunov function as follows:

$$L = \beta E + (\mu + \beta)I,$$

$$\frac{dL}{dt} = \beta(\alpha SI - (\mu + \beta)E) + (\mu + \beta)(\beta E - (\mu + \delta + \rho)I)$$

$$= \beta\alpha SI - (\mu + \beta)(\mu + \delta + \rho)I$$

$$= (\mu + \beta)(\mu + \delta + \rho)\left[\frac{\beta\alpha S}{(\mu + \beta)(\mu + \delta + \rho)} - 1\right]I$$

$$\le (\mu + \beta)(\mu + \delta + \rho)\left[\frac{\beta\alpha S_0}{(\mu + \beta)(\mu + \delta + \rho)} - 1\right]I$$

$$= (\mu + \beta)(\mu + \delta + \rho)[\mathcal{R}_0 - 1]I.$$

Then, $\frac{dL}{dt} = 0$ when $I = 0$ and $\frac{dL}{dt} < 0$ when $\mathcal{R}_0 < 1$. Therefore, $\mathcal{E}_0$ is globally asymptotically stable when $\mathcal{R}_0 < 1$. ∎

Theorem 3.3: *When $\mathcal{R}_0 > 1$, the endemic equilibrium point (Γ_{EE}) is locally stable.*

Proof: First, consider $\det(J(\Gamma_{EE}) - \lambda I) = 0$

$$\det(J(\Gamma_{EE}) - \lambda I) = \begin{vmatrix} -\alpha I^* - \tau - \mu - \lambda & \omega & 0 & -\alpha S^* & 0 & 0 \\ \tau & -\mu - \omega - \lambda & 0 & 0 & 0 & 0 \\ \alpha I^* & 0 & -\mu - \beta - \lambda & \alpha S^* & 0 & 0 \\ 0 & 0 & \beta & -\mu - \delta - \rho - \lambda & 0 & 0 \\ 0 & 0 & 0 & \rho & -\gamma - \delta - \mu - \lambda & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\mu - \lambda \end{vmatrix} = 0.$$

Then, we have

$$(-\mu - \lambda)(-\gamma - \delta - \mu - \lambda)$$

$$\times \begin{vmatrix} -\alpha I^* - \tau - \mu - \lambda & \omega & 0 & -\alpha S^* \\ \tau & -\mu - \omega - \lambda & 0 & 0 \\ \alpha I^* & 0 & -\mu - \beta - \lambda & \alpha S^* \\ 0 & 0 & \beta & -\mu - \delta - \rho - \lambda \end{vmatrix} = 0.$$

Here, $\lambda_1 = -\mu < 0$ and $\lambda_2 = -\gamma - \delta - \mu < 0$.

The rest of the term is

$$(-\beta) \begin{vmatrix} -\alpha I^* - \tau - \mu - \lambda & \omega & -\alpha S^* \\ \tau & -\mu - \omega - \lambda & 0 \\ \alpha I^* & 0 & \alpha S^* \end{vmatrix} + (-\mu - \delta - \rho - \lambda)$$

$$\times \begin{vmatrix} -\alpha I^* - \tau - \mu - \lambda & \omega & 0 \\ \tau & -\mu - \omega - \lambda & 0 \\ \alpha I^* & 0 & \alpha S^* \end{vmatrix} = 0,$$

$$(-\beta) \left[(\alpha I^*)(-\alpha S^*)(\mu + \omega + \lambda) + (\alpha S^*) \begin{vmatrix} -\alpha I^* - \tau - \mu - \lambda & \omega \\ \tau & -\mu - \omega - \lambda \end{vmatrix} \right]$$

$$- (\mu + \delta + \rho + \lambda)\alpha S^* \begin{vmatrix} -\alpha I^* - \tau - \mu - \lambda & \omega \\ \tau & -\mu - \omega - \lambda \end{vmatrix} = 0,$$

$$\beta \alpha^2 S^* I^* (\mu + \omega + \lambda) - \beta \alpha S^* \begin{vmatrix} -\alpha I^* - \tau - \mu - \lambda & \omega \\ \tau & -\mu - \omega - \lambda \end{vmatrix}$$

$$- (\mu + \delta + \rho)(\alpha S^*) \begin{vmatrix} -\alpha I^* - \tau - \mu - \lambda & \omega \\ \tau & -\mu - \omega - \lambda \end{vmatrix} = 0,$$

$$\beta \alpha^2 S^* I^* (\mu + \omega + \lambda) - \alpha S^* (\mu + \delta + \rho + \lambda + \beta)$$
$$\times [(\alpha I^* + \tau + \mu + \lambda)(\mu + \omega + \lambda) - \tau \omega] = 0,$$
$$\beta \alpha^2 S^* I^* (\mu + \omega) + \beta \alpha^2 S^* I^* \lambda - \alpha S^* (\mu + \delta + \rho + \lambda + \beta)$$
$$\times [\lambda^2 + (\alpha I^* + \tau + \omega + 2\mu)\lambda + (\alpha I^* + \tau + \mu)(\mu + \omega) - \tau \omega] = 0,$$
$$\beta \alpha^2 S^* I^* (\mu + \omega) + \beta \alpha^2 S^* I^* \lambda - \alpha S^* (\mu + \delta + \rho + \beta)\lambda^2$$
$$- \alpha S^* (\mu + \delta + \rho + \beta)(\alpha I^* + \tau + \omega + 2\mu)\lambda$$
$$- \alpha S^* (\mu + \delta + \rho + \beta)[(\alpha I^* + \tau + \mu)(\mu + \omega) - \tau \omega]$$
$$- \alpha S^* \lambda^3 - \alpha S^* (\alpha I^* + \tau + \omega + 2\mu)\lambda^2$$
$$- \alpha S^* [(\alpha I^* + \tau + \mu)(\mu + \omega) - \tau \omega]\lambda = 0.$$

Then, we have that

$$\lambda^3 + (\alpha I^* + \tau + \omega + \delta + \rho + \beta + 3\mu)\lambda^2 + ((\mu + \delta + \rho + \beta)(\alpha I^* + \tau + \omega + 2\mu)$$
$$+ ((\alpha I^* + \tau + \mu)(\mu + \omega) - \tau \omega) - \beta \alpha I^*)\lambda$$
$$+ ((\mu + \delta + \rho + \beta)[(\alpha I^* + \tau + \mu)(\mu + \omega) - \tau \omega] - \beta \alpha I^* (\mu + \omega)) = 0.$$

This can be written in the form $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$ where

$$a_1 = \alpha I^* + \tau + \omega + \delta + \rho + \beta + 3\mu > 0,$$

$$a_2 = ((\mu + \delta + \rho + \beta)(\alpha I^* + \tau + \omega + 2\mu)$$

$$+ ((\alpha I^* + \tau + \mu)(\mu + \omega) - \tau\omega) - \beta\alpha I^*)$$

$$= (\mu + \delta + \rho)(\alpha I^* + \tau + \omega + 2\mu) + \beta(\tau + \omega + 2\mu)$$

$$+ (\alpha I^* + \mu)(\mu + \omega) + \tau\mu > 0,$$

$$a_3 = ((\mu + \delta + \rho + \beta)[(\alpha I^* + \tau + \mu)(\mu + \omega) - \tau\omega] - \beta\alpha I^*(\mu + \omega))$$

$$= (\mu + \delta + \rho)[(\alpha I^* + \mu)(\mu + \omega) + \tau\mu] + \beta\mu(\mu + \omega + \tau) > 0,$$

$$a_1 a_2 - a_3 = (\alpha I^* + \tau + \omega + \delta + \rho + \beta + 3\mu)(\mu + \delta + \rho)(\alpha I^* + \tau + \omega + 2\mu)$$

$$+ (\alpha I^* + \tau + \omega + \delta + \rho + \beta + 2\mu)\beta(\tau + \omega + 2\mu)$$

$$+ \beta\mu^2 + (\alpha I^* + \tau + \omega + \beta + 2\mu)[(\alpha I^* + \mu)(\mu + \omega) + \tau\mu] > 0.$$

This leads to $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ and $a_1, a_2 > a_3$ which meets the Routh–Hurwitz criteria. Hence, the equilibrium point (Γ_{EE}) is stable. This completes the proof. ∎

3.6. Sensitivity analysis

We further explore the effects of each parameter on measles transmission dynamics in Nigeria. To accomplish this, we used the data in Table 3 to conduct a sensitivity analysis to examine the impact of each threshold quantity parameter. This will allow us to identify the parameters that have the most impact on the model's numerical simulation results. Sensitivity analysis reveals the importance of each parameter in disease transmission, allowing public health experts to prioritize a well-planned intervention approach for preventing and controlling disease spread in the population. The normalized forward sensitivity index, often known as elasticity, was employed, following the approach of [28,32]. The normalized forward sensitivity index of the basic reproduction number R_0 with respect to parameter q is expressed as

$$K_q^{R_\circ} = \frac{\partial R_\circ}{\partial q} \times \frac{q}{R_\circ}. \tag{16}$$

The numerical values for the elasticity index are calculated using the elasticity formula [32] and the baseline parameters listed in Table 3. Table 2 shows the results of estimating the elasticity index with respect to each parameter. The results of the sensitivity analysis shows that recruitment rate ϕ and the progression rate from exposed to infected class β have the highest positive index, hospitalization of the infected individuals is critical in reducing the spread of the disease since the increase in the value of hospitalization rate ρ should reduce the progression from exposure to infectious β. Also, the natural death rate μ and the rate at which infected individuals visit the hospital ρ have the highest negative index. The negative figure indicates that increasing natural death rate or the rate at which infected individuals visit the hospital by H% will reduce the reproduction number by H% and vice versa.

Table 2. Sensitivity indexes of R_0 to model parameters.

Parameter	Sensitivity Index
ϕ	1
μ	-1.0050
δ	-0.47983
τ	-0.00027809
ω	0.000023902
α	0
β	0.00061762
ρ	-0.51577

Table 3. The description of parameters and values.

Parameter	Description	Value	Source
ϕ	Recruitment rate into susceptible class	68,027	Estimated
μ	Natural death rate	0.000309	Estimated
δ	Measles death rate	0.033720	Estimated
τ	Vaccination rate	0.000001	Fitted
ω	Rate of loss of immunity	0.003286	Fitted
α	Transmission rate of susceptible individuals	1×10^{-9}	Fitted
β	Rate of progression from the exposed class to infected class	0.500000	Estimated
ρ	Hospitalization rate of infected individuals due to complications	0.036246	Fitted
γ	Recovery rate	0.062366	Fitted

4. Data fitting and numerical simulation

Mathematical models have been used by many researchers to effectively replicate the observed incidence and prevalence of several diseases. Furthermore, it has been used to describe the dynamics of disease epidemics from the present data to predict the future and particularly, to quantify the imprecision in these predictions. These are achieved by validating a formulated model with real data if available. This will provide a meaningful result to the model predictions. In this section, we fit the proposed model given with the real data from Nigeria, to describe the dynamics of the measles epidemic in the Nigerian population.

We used the measles reported cases from Nigeria for the period between the first week in January through the last week in December 2020, obtained through the Nigerian Centre for Disease Control (NCDC) database [25]. As presented in Table 3, we obtained our parameter values through parameter estimation and the data fitting method. Four out of the nine parameters of the proposed model were estimated, while the remaining unknown five parameter values were obtained through data fitting of the model. The natural death of a human is estimated as $\frac{1}{60.87 \times 53}$ per week, where 60.87 year is the average life expectancy of humans in Nigeria [13]. Furthermore, the recruitment rate is estimated by $\mu \times N$, where the total human population N is reported as 219,463,862 [13]. We used the reported confirmed cases and reported death cases due to measles denoted as (I, D) respectively to estimate the measles death rate δ (which is the death caused by measles). This is obtained by

$$\delta = \frac{\sum_{t=1}^{n} D_t}{\sum_{t=1}^{n} I_t},$$

where $t = 1, 2, \ldots, n$ is the time measured in weeks and $n = 53$ is the total number of weeks as reported in the data used. Lastly, since the average incubation period of measles
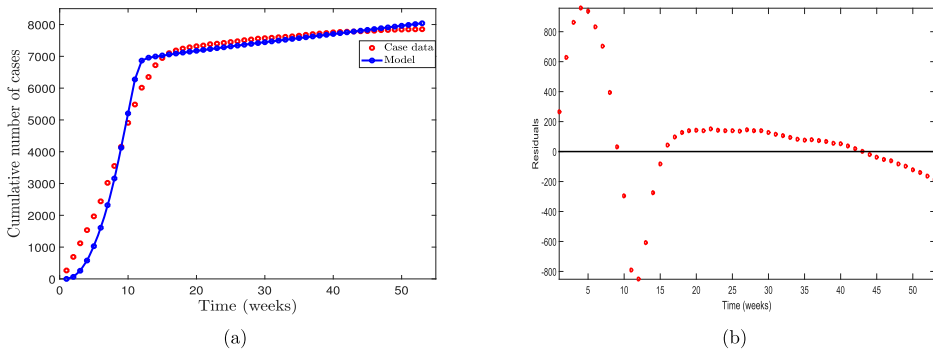
Figure 2. Best fitting of the proposed measles model with real statistical cases in Nigeria and the corresponding (b) residual plot.
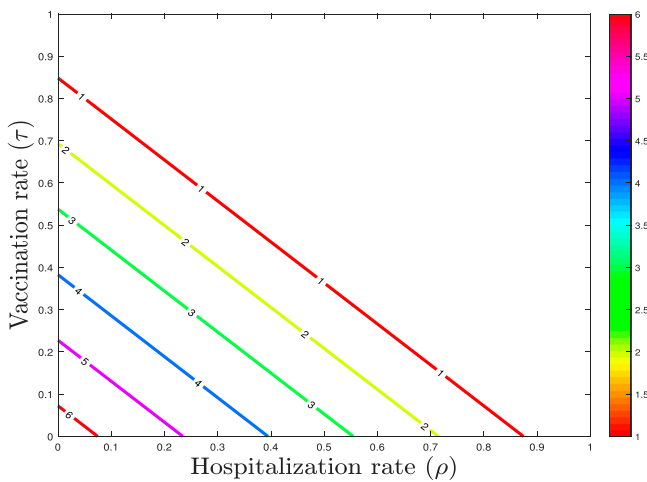

Figure 3. 2-D contour plot of the reproduction number $\mathcal{R}_0$ of measles model, varying vaccination rate τ with respect to the hospitalization rate ρ. Parameter values used are as given in Table 3.

is 14 days, varying from 7 to 18 days [30], the progression rate from the exposure to infected class β is estimated as 0.500 per week. Using the standard nonlinear least square method, we fit the cumulative confirmed reported cases to the measles model. We present all the parameter values estimated and fitted in Table 3, and the data fitting of the observed cumulative cases are depicted in Figure 2(a) wherein residuals have also been graphically depicted in Figure 2(b). It must be noted that all parameter values are presented in per week.

5. Results and discussion

Following the estimation of parameter values and data fitting, we simulate the measles model using the parameter values as presented in Table 3. It is imperative to note that the reproduction number of measles in Nigeria is estimated as 3.13, using the parameter values in Table 3. This implies that measles is expected to be endemic in Nigeria if there
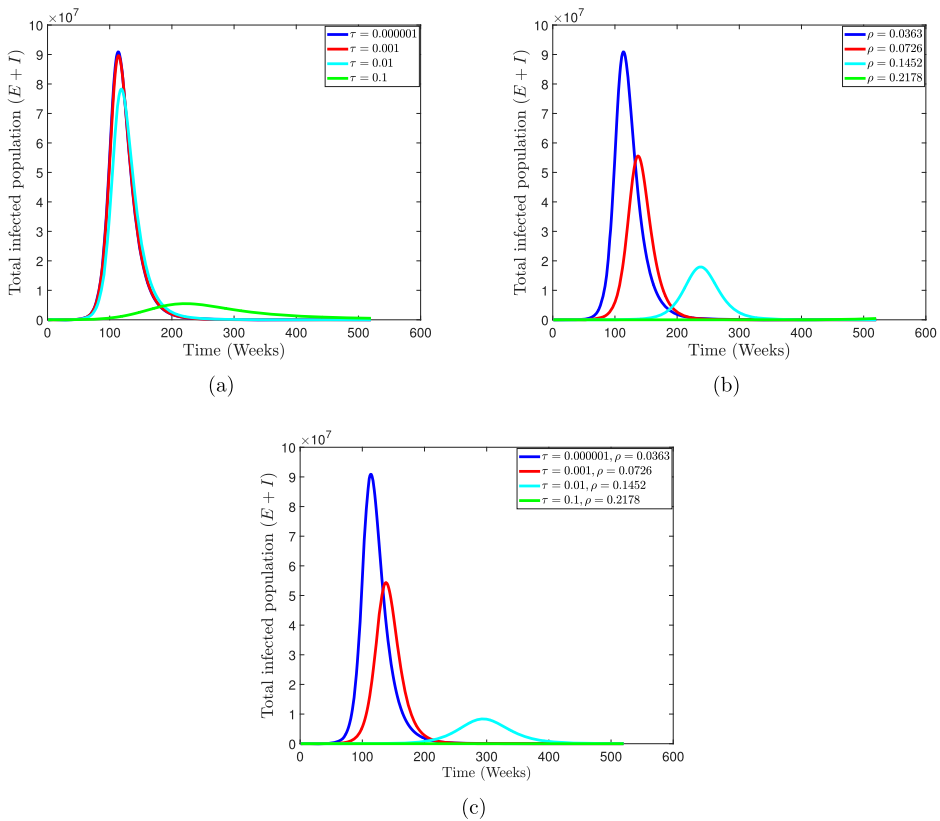
Figure 4. Simulations of the measles model with varying effects of parameters on total infected humans population: (a) vaccination rate $\tau = 0.000001(\mathcal{R}_0 = 3.13)$, $\tau = 0.001(\mathcal{R}_0 = 3.05)$, $\tau = 0.01(\mathcal{R}_0 = 2.45)$, and $\tau = 0.1(\mathcal{R}_0 = 0.83)$; (b) hospitalization rate $\rho = 0.0363(\mathcal{R}_0 = 3.13)$, $\rho = 0.0726(\mathcal{R}_0 = 2.07)$, $\rho = 0.1452(\mathcal{R}_0 = 1.23)$, and $\rho = 0.2178(\mathcal{R}_0 = 0.88)$; (c) vaccination rate and hospitalization rate $\tau = 0.000001, \rho = 0.0363(\mathcal{R}_0 = 3.13)$, $\tau = 0.001, \rho = 0.0726(\mathcal{R}_0 = 2.01)$, $\tau = 0.01, \rho = 0.1452(\mathcal{R}_0 = 0.96)$, and $\tau = 0.1, \rho = 0.2178(\mathcal{R}_0 = 0.23)$. Other parameter values used are given in Table 3.

are no further strategies to mitigate the disease in the population. Since vaccination of susceptible individuals and treatment of infected individuals has been shown to reduce the burden of measles in the population, we simulate the effect of these two control strategies on the reproduction number in Figure 3. The result shows that increasing the vaccination rate decreases the reproduction number. For instance, if we fix the hospitalization rate of infected humans at $\rho = 0.5$, a vaccination rate at $\tau = 0.3$ will yield a reproduction number between the range of $(1, 2)$, while the vaccination rate at $\tau = 0.7$ reduces the reproduction number below unity. A similar result was observed when we fix the value of the vaccination rate and vary the hospitalization rate. The result shows that increasing the hospitalization rate of the infected individuals will reduce the reproduction number. Overall, the result from Figure 3 suggests that to reduce the reproduction number of measles below unity, increasing the vaccination rate and the hospitalization rate of infected individuals simultaneously will reduce the burden of measles in Nigeria.
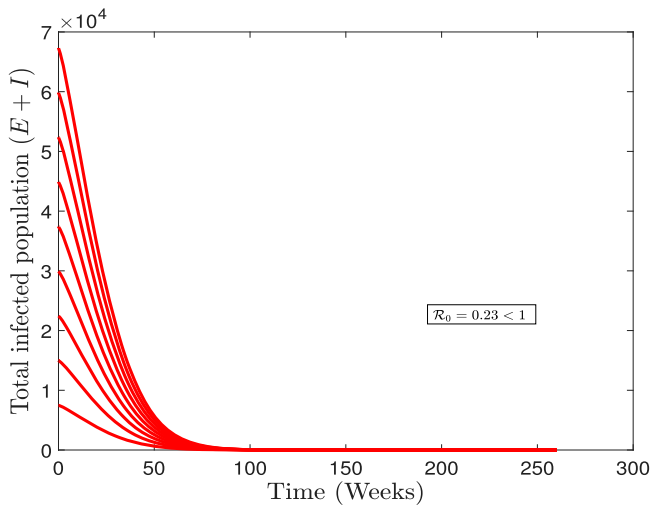
Figure 5. Convergence of solution trajectories for total infected population ($E + I$) with different initial sizes. Parameter values used are as provided in Table 3 except for $\tau = 0.1$ and $\rho = 0.2178$, so that $\mathcal{R}_0 = 0.23 < 1$.

To investigate the impact of vaccination rate and hospitalization of infected individuals on the dynamics of measles in the population, we examine the behaviour of the total infected human population under different scenarios. We note that since measles exposed individuals can transfer the infection, we defined the total infected human population as the sum of exposed humans and infected human population ($E + I$). We illustrate the effects of vaccination rate on the total infected population in Figure 4(a). The result shows that the total infected population reduces with increasing value of vaccination rate from 1×10^{-6} to 0.1. A similar result is observed in Figure 4(b); an increase in the hospitalization rate of infected individuals reduces the total infected population. Thus, an increase in any of the control strategies will reduce the burden of measles in the population. To investigate the dynamics of measles under the combination of vaccination and hospitalization of infected individuals on the population, we combined the two control strategies (vaccination and hospitalization of infected individuals) as depicted in Figure 4(c). The result shows that the combined control strategies reduce the peak of infection faster than the single control strategy. The overall result from Figure 4 emphasizes the importance of control strategies such as vaccination and hospitalization of measles-infected individuals in mitigating the spread of measles in the population. Figure 5 illustrates the convergence of solution trajectories for the infected population. The result shows that regardless of the changes in the initial sizes of the infected population, the infected population equilibrium will remain the same. This result validates the global stability result presented in Theorem 3.2.

6. Conclusion

The dynamics of measles have been examined using a mathematical model with six compartments: susceptible, vaccinated, exposed, infected, hospitalized, and recovered. The

boundary of solutions is proved, the basic reproduction number is calculated. Two equilibrium points are determined and their stability are analysed. The proposed model was validated by using real data of measles incidence obtained from NCDC, we obtained the best-fitted values for the model, this shows that the model will be useful for measles prediction, this will also help the health workers in decision making and control policy in eradicating the spread of measles in Nigeria. The study suggests that an increase in the hospitalization rate of infected individuals with serious complications reduces the total infected population. Also, without vaccination, the number of infections will increase and in most cases, a complication occurs in most infected individuals which will require hospitalization. Thus, an increase in any of the control strategies will reduce the burden of measles in the population. To investigate the dynamics of measles under the combination of vaccination and hospitalization of infected individuals, on the population, we combined the two control strategies (vaccination and hospitalization of infected individuals). The result shows that the combined control strategies reduce the peak of infection faster than the single control strategy. The result of our study recommends the importance of control strategies such as vaccination and hospitalization of measles-infected individuals in mitigating the spread of measles in the population.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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