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MATHEMATICAL MODEL AND ANALYSIS OF MEASLES DYNAMICS IN A POPULATION WITH LIMITED RESOURCES

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ABSTRACT. This study explores the mathematical modelling of measles transmission dynamics in Nigeria, with a specific focus on assessing the impact of a single-dose vaccination strategy. Given the resurgence of measles outbreaks, especially in regions with low vaccination coverage, this research aims to develop a robust model that can simulate disease transmission and evaluate vaccination strategies. The primary objective of the study is to understand how varying levels of vaccination coverage, vaccine efficacy, and immunity waning affect the disease dynamics. A modified SEIR (Susceptible-Exposed-Infectious-Recovered) model was used, incorporating additional compartments for individuals vaccinated with one dose, as well as a factor for immunity waning. Data from Nigeria's Measles Situation Report (April 2024) informed the parameter values, initial population distributions, and vaccination rates within the model, providing a real-world context. The study employed numerical simulations using MATLAB to analyse the effects of vaccination rates, immunity waning, and other epidemiological parameters on measles transmission. The results reveal that high vaccination coverage specifically, achieving coverage rates above 80% with the single-dose strategy significantly reduces the disease prevalence, indicating effective outbreak prevention. However, the simulations also show that immunity waning can increase susceptibility, suggesting a potential need for booster dose to sustain long-term immunity in the population. It recommends that public health authorities prioritize reaching at least 90% vaccination coverage with two doses and consider booster doses if immunity waning proves significant. These insights provide a foundation for enhancing measles control efforts, informing policy decisions, and guiding future research on infectious disease dynamics in Nigeria and similar settings.

1. INTRODUCTION

Each year, many individuals are affected by severe respiratory infectious diseases, including measles. A significant number of these individuals either die or experience serious illness and lifelong complications [39, 12, 29, 5, 1]. The World Health Organization (WHO) and UNICEF release annual reports on global measles cases and related deaths, based on data provided by member countries. According to WHO and UNICEF figures for 2017, there were 7,585,900 reported measles cases and 124,000 measles-related deaths. In

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2018, the numbers rose to approximately 9,769,400 cases and 142,300 deaths [34]. The highest case numbers were reported from Madagascar, Ukraine, Somalia, and Liberia. Additionally, some developed countries, such as the United Kingdom, Greece, Czechia, and Albania, lost their measles elimination status that year. The United States recorded its highest number of measles cases in 25 years in 2018. In 2019, measles caused 207,500 deaths worldwide, with 869,770 reported cases [13]. Madagascar, Ukraine, and Congo were among the countries with the highest case numbers, and outbreaks continued in Angola, Cameroon, Kazakhstan, Chad, Nigeria, Thailand, the Philippines, South Sudan, and Sudan. The continuous rise in cases presents a significant global health concern [35].

Measles is a highly contagious respiratory disease caused by the measles virus, which resides in the mucus of an infected person's nose and throat. It belongs to the paramyxovirus family and the morbillivirus genus, and is unique in that it only infects humans [39, 12]. The virus spreads through direct contact, such as coughing and sneezing. Common symptoms include high fever, runny nose, cough, conjunctivitis, rhinitis, small white spots in the mouth, and a rash. Measles is especially dangerous for children under five and adults over 20, leading to complications such as pneumonia, mouth ulcers, sinus and ear infections, diarrhea, malnutrition, blindness, and brain damage [29].

There is no specific cure for measles, but treatment typically involves bed rest, fluids, fever and pain management, and sometimes antibiotics to address complications [7]. The measles vaccine, which is effective and affordable, has greatly reduced the number of deaths caused by the disease. The measles, mumps, and rubella (MMR) vaccine has a 95% efficacy rate when the first dose is administered to children at 12 months [6]. Global health organizations like WHO, UNICEF, the American Red Cross, the Centres for Disease Control and Prevention (CDC), and the United Nations Foundation have made significant efforts to combat measles. In 2001, these groups launched the Measles and Rubella Initiative (MRI), aiming to reduce global measles deaths by 90% by 2010 [32]. They are currently working under the Measles and Rubella Strategic Framework 2021–2030 (MRSF 2021–2030), aiming for a world free of these diseases [36].

However, the World Health Organization (WHO) estimates that measles continues to be a leading cause of death among young children worldwide, despite the availability of a safe and effective vaccine [37]. To achieve herd immunity, the WHO recommends a vaccination coverage rate of at least 95% [38], but Nigeria's vaccination rate remains well below this target [16]. The country has one of the highest measles incidence rates globally and has faced recurring outbreaks in recent years [25, 30]. Currently, there is an alarming increase in measles cases across Nigeria. In 2023, the Nigerian Centre for Disease Control and Prevention (NCDC) reported 184 outbreaks, with 11,433 confirmed cases [23]. As of March 2024, the infection rate in Nigeria is 64.9 cases per 1 million children, which is far above the WHO target of fewer than 1 case per million. Measles cases have been reported across the country, with particularly high numbers in Borno state [24]. Notably, there were two major measles outbreaks in the northwest and northeast regions between 2012 and 2021 [30]. In 2019, measles outbreaks were reported in thirty-five states and the Federal Capital Territory (FCT) [17].

Measles research has become a critical field of study, with numerous mathematical [27, 4], theoretical, and experimental models developed to improve measles control strategies across different regions, including London, Afghanistan, Kenya, Madagascar, Ontario, Cape Coast, Italy, Senegal, Taiwan, and China [19, 9, 10, 3, 14, 26, 20, 8, 15, 40]. Additionally, Momoh et al. [22] explored an SEIR deterministic epidemic model to analyze the

impact of asymptomatic individuals during the latent period on measles dynamics. Adewale et al. [2] developed a mathematical model to assess the effect of distance between infected and non-infected individuals on controlling measles transmission. Their findings showed that increasing the distance between infected and susceptible individuals leads to a decrease in the number of infections. Furthermore, two studies emphasized the effectiveness of vaccination in controlling and preventing measles transmission [31, 28]. Garba et al. [11] also examined a compartmental mathematical model to evaluate the influence of vaccination and treatment on measles dynamics. Beay [5] proposed an SIQR epidemic model and conducted a numerical analysis to investigate the impact of treatment and quarantine on measles dynamics. The study concluded that the combined use of quarantine and treatment is more effective in controlling and preventing measles, noting that measles transmission decreases as a result of treating and quarantining infected individuals.

This study presents a novel compartmental model to simulate measles prevalence in Nigeria, using the next-generation matrix method to determine the basic reproduction number, a key factor in disease dynamics. Numerical method is also employed to solve the model equations and analyse epidemic trends under varying conditions. Local stability analyses of both disease-free and endemic equilibria are conducted using the trace and determinant methods. Additionally, we perform a sensitivity analysis to identify the parameters most influencing measles prevalence and examine the effects of progression rates, transmission rates, and single-dose vaccination on measles dynamics.

The organisation of this paper is as follows. The model is formulated in Section 2. The analyses of the formulated model and its numerical simulation are carried out in Section 3, while the results of the model analyses and simulations are discussed in Section 4. The main conclusions from this study are summarized in Section 5.

2. Model Formulation

This section develops a mathematical model for measles dynamics using deterministic ordinary differential equations. The model consists of five compartments or subpopulations:

i Susceptible compartment (first equation): This compartment increases due to the influx rate (Π) and vaccine inefficacy rate, while it decreases due to transmission, vaccination, and natural death rates.
ii Vaccinated compartment (second equation): This compartment increases with the vaccination rate and decreases due to vaccine inefficacy, immunity loss, and natural death rates.
iii Exposed compartment (third equation): This compartment increases with the disease transmission rate and decreases due to disease progression and natural death rates.
iv Infected compartment (fourth equation): This compartment increases with the disease progression rate and decreases due to recovery, disease-induced death, and natural death rates.
v Recovered compartment (fifth equation): This compartment increases with recovery and immunity rates and decreases due to the natural death rate.

2.1. **Assumptions of the model equations.**

For proper understanding and formulation of system of equations in this study, we present the following assumptions:

(H_1) A single-dose vaccination is administered due to limited resources

(H_2) Some vaccinated individuals became susceptible to measles due to vaccine's inefficacy

(H_3) Some of the vaccinated individuals developed permanent immunity to measles due to vaccine's efficacy and joined recovered individuals or compartment

(H_4) Some infected individuals died due to measles infected while some of the infected individuals recovered naturally and then developed permanent immunity to the disease.

2.2. **Model variables and parameters.** The variables and the parameters of the model are given as Table 1 below.

Variable	Interpretation
$S(t)$	Susceptible human population at time t
$V(t)$	vaccinated human population at time t
$E(t)$	Exposed human population at time t
$I(t)$	Infected human population at time t
$R(t)$	Recovered human population with immunity at time t
Parameter	**Interpretation**
μ	Natural death rate
δ	Disease induced death rate
γ	Natural recovery rate of the infected individuals
ρ	Vaccine inefficacy
ω	Rate at which vaccinated individuals developed immunity
α	Rate at which exposed individuals become infected
β	Disease transmission rate
η	Vaccination rate of susceptible individuals
Π	Recruitment rate of susceptible individuals

TABLE 1. Description of the model variables and parameters

2.3. **Model schematic diagram.** The model schematic diagram is given below as Figure 1.

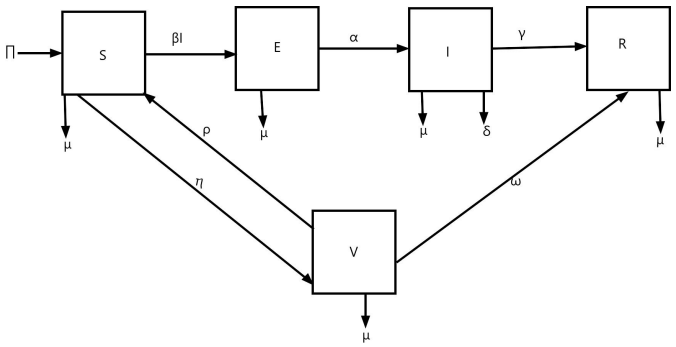

FIGURE 1. Model Schematic Diagram

2.4. **Descriptions of the model equations.** The model

2.5. **Model equations.** Considering the above assumptions, variables and parameters descriptions, and the flow diagram, the model equations is given by

$$\begin{aligned}
\frac{dS}{dt} &= \Pi - \beta SI - \eta S - \mu S + \rho V,\\
\frac{dV}{dt} &= \eta S - (\rho + \omega + \mu)V,\\
\frac{dE}{dt} &= \beta SI - (\alpha + \mu)E,\\
\frac{dI}{dt} &= \alpha E - (\gamma + \delta + \mu)I,\\
\frac{dR}{dt} &= \gamma I + \omega V - \mu R.
\end{aligned} \tag{2.1}$$

The non-negativity conditions of system (2.1) is given by

$$S > 0, V \geq 0, E \geq 0, I \geq 0, R \geq 0. \tag{2.2}$$

3. Model Analysis

This section considers some basic analytical analyses that are useful and paramount to understanding the dynamics of measles disease.

3.1. **Fixed point analysis.** In order to study the model behaviour explicitly, the model fixed point analysis is divided into the disease-free equilibrium (DFE), and the disease persistent equilibrium (DPE). The two equilibria can be obtained from system (2.1) by rewritten system (2.1) as a steady state system as follows.

$$\begin{aligned}
\Pi - \beta SI - \eta S - \mu S + \rho V &= 0,\\
\eta S - \rho V - \omega V - muV &= 0,\\
\beta SI - \alpha E - \mu E &= 0,\\
\alpha E - \gamma I - \delta I - \mu I &= 0,\\
\gamma I + \omega V - \mu R &= 0.
\end{aligned} \tag{3.1}$$

Solving system (3.1) simultaneously, the disease-free equilibrium (DFE), and the disease persistent equilibrium (DPE) are given as E_1 and E_2 respectively.

$$E_1 = (S^*, V^*, E^*, I^*, R^*),$$

where

$$S^* = \frac{\pi(\mu + \rho + \omega)}{\alpha\eta\mu + \alpha\mu^2 + \mu\rho + \eta\omega + \mu\omega}, V^* = \frac{\eta\pi}{\eta\mu + \mu^2 + \mu\rho + \eta\omega + \mu\omega},$$
$$E^* = 0, I^* = 0, R^* = \frac{\eta\pi\omega}{\mu(\eta\mu + \mu^2 + \mu\rho + \eta\omega + \mu\omega)}.$$

And

$$E_2 = (S^{**}, V^{**}, E^{**}, I^{**}, R^{**}),$$

where

$$S^{**} = \frac{(\gamma + \delta + \mu)(\alpha + \mu)}{\alpha\beta},$$
$$V^{**} = \frac{\eta(\gamma + \delta + \mu)(\alpha + \mu)}{\alpha\beta(\rho + \omega + \mu)},$$

$$E^{**} = -\frac{\alpha\left(-\beta\mu\Pi - \beta\Pi\rho - \beta\Pi\omega + \gamma\eta(\mu+\omega) + \gamma\mu(\mu+\rho+\omega) + \delta\eta(\mu+\omega) + \delta\mu(\mu+\rho+\omega) + \eta\mu^2 + \eta\mu\omega + \mu^3 + \mu^2\rho + \mu^2\omega\right) + \mu(\gamma+\delta+\mu)(\eta(\mu+\omega) + \mu(\mu+\rho+\omega))}{\alpha\beta(\alpha+\mu)(\mu+\rho+\omega)},$$
$$I^{**} = \frac{\alpha\left(-\beta\mu\Pi - \beta\Pi\rho - \beta\Pi\omega + \gamma\eta(\mu+\omega) + \gamma\mu(\mu+\rho+\omega) + \delta\eta(\mu+\omega) + \delta\mu(\mu+\rho+\omega) + \eta\mu^2 + \eta\mu\omega + \mu^3 + \mu^2\rho + \mu^2\omega\right) + \mu(\gamma+\delta+\mu)(\eta(\mu+\omega) + \mu(\mu+\rho+\omega))}{\beta(\alpha+\mu)(\gamma+\delta+\mu)(\mu+\rho+\omega)},$$

$R^{**} = \frac{-\left(\alpha^2\left(\gamma\left(\left(\mu^2-\beta\Pi\right)(\mu+\rho+\omega)+\delta\eta(\mu-\omega)+\delta\mu(\mu+\rho+\omega)+\eta\mu(\mu-\omega)\right)+\gamma^2\mu(\eta+\mu+\rho+\omega)-\eta\omega(\delta+\mu)^2\right)\right)-\alpha\mu(\gamma+\delta+\mu)+\Delta_1}{\alpha\beta\mu(\alpha+\mu)(\gamma+\delta+\mu)(\mu+\rho+\omega)}$,
with $\Delta_1 = (\gamma\eta(\mu-\omega)+\gamma\mu(\mu+\rho+\omega)-2\eta\omega(\delta+\mu))+\eta\mu^2\omega(\gamma+\delta+\mu)^2$
The equilibrium points E_1 and E_2 represent measles free population and measles persistent population respectively.

3.2. **Basic reproduction number computation.** To calculate the basic reproduction number, system (2.1) is divided into appearance of infection and transfer of infection as matrix $F_i(x)$ and $M_i(x)$ where $F_i(x)$ be the rate of appearance of new infections in compartment i and $M_i(x)$ be the difference between the transfer rate of individuals out of compartment i by all other means. Let x_0 be the DFE of model equations. Thus, we have the following partitioned derivatives, thus, the $F_i(x)$ and $M_i(x)$ of model equations is shown below:

$$F = \begin{pmatrix} \beta SI \\ 0 \end{pmatrix} = \frac{\partial F}{\partial X}(E_1) = \begin{pmatrix} 0 & \beta S \\ 0 & 0 \end{pmatrix}$$

and

$$M = \begin{pmatrix} (\alpha+\mu)E \\ -\alpha E + (\gamma+\delta+\mu)I \end{pmatrix} = \frac{\partial M_i}{\partial x_j(E_1)} = \begin{pmatrix} \alpha+\mu & 0 \\ -\alpha & \delta+\gamma+\mu \end{pmatrix}$$

The basic reproduction number is therefore computed to be

$$R_0 = \rho_1(FM^{-1}) = \frac{\alpha\beta\pi(\mu+\rho+\omega)}{(\alpha+\mu)(\gamma+\delta+\mu)(\eta\mu+\mu^2+\mu\rho+\eta\omega+\mu\omega)}$$

Where ρ_1 is the spectral radius of FM^{-1}(the spectral radius implies the maximum eigenvalue of FM^{-1})

Theorem 1. Consider the disease transmission model with $f(x)$ satisfying the stability conditions if x_0 is a DFE of the model,the x_o is locally asymptotically stable if $R_0 < 1$, but unstable if $R_0 > 1$.

3.3. **Stability analysis.** In this section, stability analysis of the equilibrium points E_1 and E_2 are carried out.

3.3.1. *Stability of the disease-free equilibrium point.* The characteristic equation is given by

$$J(E_0) = \begin{pmatrix} -\phi_1 & \rho & 0 & \beta S & 0 \\ \eta & -\phi_2 & 0 & 0 & 0 \\ 0 & 0 & -\phi_3 & \beta S & 0 \\ 0 & 0 & \alpha & -\phi_4 & 0 \\ 0 & \omega & 0 & \gamma & -\phi_5 \end{pmatrix}$$

$-\lambda^4(-A-B-C-D-\mu)+ABCD\mu-\lambda^3(-AB-AC-AD-A\mu-BC-BD-B\mu-CD-C\mu-D\mu+\eta\rho+\alpha\beta S)-\lambda^2(-ABC-ABD-AB\mu-ACD-AC\mu-AD\mu+\alpha A\beta S-BCD-BC\mu-BD\mu+\alpha\beta BS-CD\mu+C\eta\rho+D\eta\rho+\eta\mu\rho+\alpha\beta\mu S)-\lambda(-ABCD-ABC\mu-ABD\mu+\alpha A\beta BS-ACD\mu+\alpha A\beta\mu S-BCD\mu+\alpha\beta B\mu S+CD\eta\rho+C\eta\mu\rho+D\eta\mu\rho-\alpha\beta\eta\rho S)-\alpha A\beta B\mu S-CD\eta\mu\rho+\lambda^5+\alpha\beta\eta\mu\rho S=0$

$Tr(J(E_0)) = -(\Phi_1+\Phi_2+\Phi_3+\Phi_4+\Phi_5)$

$Det(J(E_0)) = S\alpha\beta\Phi_1\Phi_2\Phi_5+\eta\rho\Phi_3\Phi_4\Phi_5-(\Phi_1\Phi_2\Phi_3\Phi_4\Phi+S\alpha\beta\eta\rho\Phi_5)$

The eigenvalues corresponding to $J(E_0)$ is given by

$\frac{1}{2}\left(-\sqrt{4\eta\rho+\phi_1^2-2\phi_2\phi_1+\phi_2^2}-\phi_1-\phi_2\right), \frac{1}{2}\left(\sqrt{4\eta\rho+\phi_1^2-2\phi_2\phi_1+\phi_2^2}-\phi_1-\phi_2\right),$
$\frac{1}{2}\left(-\sqrt{4\alpha\beta S+\phi_3^2-2\phi_4\phi_3+\phi_4^2}-\phi_3-\phi_4\right), \frac{1}{2}\left(\sqrt{4\alpha\beta S+\phi_3^2-2\phi_4\phi_3+\phi_4^2}-\phi_3-\phi_4\right), -\phi_5$

3.4. **Stability of the disease persistent equilibrium point.** The stability of the disease persistent equilibrium point is given by

$$J(E_1)=\begin{pmatrix} -\eta+\beta-\mu I^{**} & \rho & 0 & \beta S & 0 \\ \eta & -\mu-\rho-\omega & 0 & 0 & 0 \\ \beta I** & 0 & -\alpha-\mu & \beta S & 0 \\ 0 & 0 & \alpha & -\gamma-\delta-\mu & 0 \\ 0 & \omega & 0 & \gamma & -\mu \end{pmatrix}$$

$Det(J(E_1)) = I^{**}\beta\mu(2S\alpha\beta-(\alpha+\mu)(\gamma\ +\delta+\mu))(\mu+\rho+\omega)+\mu(S\alpha\ \beta-(\alpha+\mu)(\gamma+\delta+\mu))(\eta(\ \mu+\omega)+\mu(\mu+\rho+\omega))$

$Tr(J(E_1)) = -(\alpha+\beta I^{**}+\gamma+\delta+\eta+5\mu+\rho+\omega)$

3.5. **Global stability analysis.**

Theorem 2. The endemic equilibrium of system (2.1) is globally asymptotically stable (GAS) whenever $R_0>1$.

Proof. The endemic equilibrium exists iff $R_0>1$, and $N=\frac{\Pi}{\mu}$ as $t\to\infty$. Therefore, using $S=\frac{\Pi}{\mu}-E-I$ and substituting in system (2.1) gives

$$\begin{aligned} \frac{dE}{dt} &= \beta\left(\frac{\Pi}{\mu}-E-I)I-(\alpha+\mu\right)E, \\ \frac{dI}{dt} &= \alpha E-(\gamma+\delta+\mu)I. \end{aligned} \tag{3.2}$$

Introducing Dulac's multiplier $\frac{1}{EI}$, system (3.2) becomes

$$\begin{aligned} &\frac{\partial}{\partial E}\left[\frac{\beta(\frac{\Pi}{\mu}-E-I)}{E}\right]+\frac{\partial}{\partial I}\left[\frac{\alpha}{I}-\frac{(\gamma+\delta+\mu)}{E}\right], \\ &=-\left[\frac{\alpha}{I^2}+\frac{\beta}{E^2}\left(\frac{\Pi}{\mu}-I\right)\right]. \end{aligned} \tag{3.3}$$

$$N\le\frac{\Pi}{\mu}>I.$$

or

$$=\frac{(\beta\Pi^3\mu-\beta\Pi I^2-\alpha\mu E^2)(\alpha+\mu)(\gamma+\delta+\mu)(\eta\mu+\mu^2+\mu\rho+\eta\omega+\mu\eta)}{\mu I^2E^2[\alpha\beta\Pi(\mu+\beta+\omega)-(\alpha+\mu)(\gamma+\delta+\mu)(\eta\mu+\mu^2+\mu\rho+\eta\omega+\mu\eta)]}\left[1-R_0\right].$$

for $R_0>1$.

3.6. **Sensitivity Analysis.** In this section, we performed the sensitivity analysis in order to determine the relative importance of the model parameters on disease transmission. The analysis will enable us to find out parameters that have high impact on the basic reproduction number and which should be targeted for intervention strategies. We perform sensitivity analysis by calculating the sensitivity indices of the effective reproduction number R_0 since our major emphasis is on the measles disease dynamics can be controlled or not. These indices tell us how crucial each parameter is on the disease transmission. The

sensitivity index of R_0 to a parameter say τ, where τ is any of the parameters in Table 2 is given by

$$\Gamma_{\tau}^{R_0} = \frac{\partial R_0}{\partial \tau} \times \frac{\tau}{R_0},$$

where $\alpha = \frac{\mu}{\alpha+\mu} = +0.47245$
$\mu = \mu\left(-\frac{1}{\alpha+\mu} - \frac{1}{\gamma+\delta+\mu} - \frac{\eta+2\mu+\rho+\omega}{\eta(\mu+\omega)+\mu(\mu+\rho+\omega)} + \frac{1}{\mu+\rho+\omega}\right) = -0.535302$
$\rho = \frac{\eta\rho(\mu+\omega)}{(\mu+\rho+\omega)(\eta(\mu+\omega)+\mu(\mu+\rho+\omega))} = +0.41145$
$\omega = -\frac{\eta\rho\omega}{(\mu+\rho+\omega)(\eta(\mu+\omega)+\mu(\mu+\rho+\omega))} = -0.403323$
$\delta = -\frac{\delta}{\gamma+\delta+\mu} = -0.012818$
$\gamma = -\frac{\gamma}{\gamma+\delta+\mu} = -0.961354$
$\eta = -\frac{\eta(\mu+\omega)}{(\eta+\mu)(\mu+\rho+\omega)-\eta\rho} = -0.971103$
$\beta = +1.00000$

4. Numerical Simulation

In this section, we use the inbuilt MATLAB function ode 45 to solve the modified model equations above. The graphical user interface in the MATLAB version 7.5 was used for the solution method, simulation and visualization on graphs. We made use of the parameters of the model and their values from related literature and we assumed some parameters values that are not found in literature. The values used here are as contained in Table 2.

4.1. **Model variables and parameters.**

The parameter's values are given below in Table 2.

Parameters	Values	References
Π	612	Assumed
μ	0.016	[21]
δ	0.018	[21]
γ	0.6	[18]
ρ	$0.000 - 0.400$	Varied
ω	0.800	[18]
α	0.018	[18]
β	$6.3 \times 10^{-2} - 7.45 \times 10^{-7}$	[21]
η	0.960	[18]

TABLE 2. Description of the model parameters and values

5. Discussions and Conclusions

Figures 2, 3, 4, and 6 depict the impact of transmission rate, progression rate, and vaccine inefficacy rate respectively on measles prevalence. These figures reveal that an increase in transmission rate, progression rate, or vaccine inefficacy rate leads to a higher burden of measles prevalence, indicating a positive correlation between these parameters and the likelihood of a measles outbreak. In contrast, Figure 5 demonstrates that increasing the vaccination rate decreases measles prevalence, thereby reducing the risk of an outbreak.

This study developed and analyzed a transmission dynamics model for measles in a resource-limited population relying on single-dose vaccination. An analytical expression for the basic reproduction number (R_0) was derived using the next-generation matrix. The

analysis showed that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$, while measles persists in the community if $R_0 > 1$. Sensitivity analysis revealed that the transmission rate ($\beta = 1.0000$) significantly influences the spread of measles, emphasizing the need to minimize unnecessary contact with infected individuals. The study underscores the critical role of vaccination in controlling and preventing measles outbreaks in Nigerian communities (see Figure 7). Vaccination remains the most effective strategy for managing measles outbreaks. However, the inefficacy of vaccines was identified as a contributing factor to measles prevalence. Numerical analysis demonstrated that increased vaccination rates have a negative correlation with measles prevalence, indicating that enhancing vaccination coverage reduces the disease's spread. To achieve high herd immunity and prevent measles outbreaks in Nigeria, mass vaccination campaigns should be prioritized to cover a larger proportion of the population.

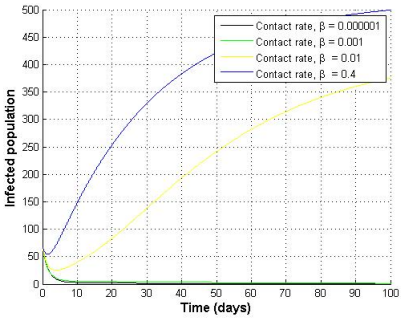

FIGURE 2. Impact of transmission rate (β) on measles prevalence

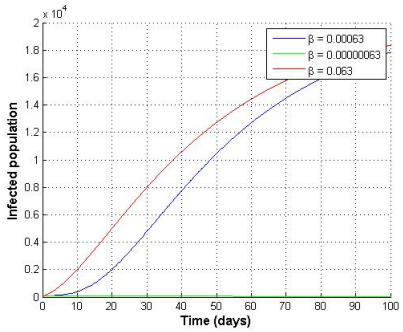

FIGURE 3. Impact of transmission rate (β) on measles prevalence

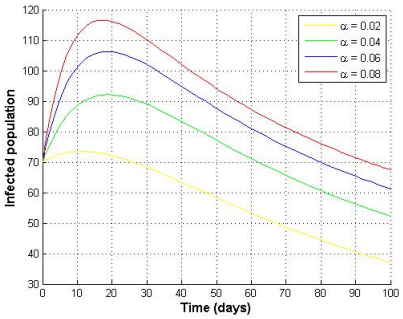

FIGURE 4. Impact of progression rate (α) on measles prevalence

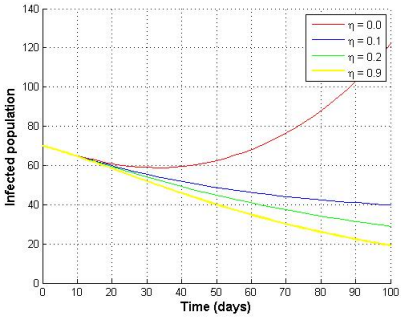

FIGURE 5. Impact of single dose vaccine(η) on measles prevalence

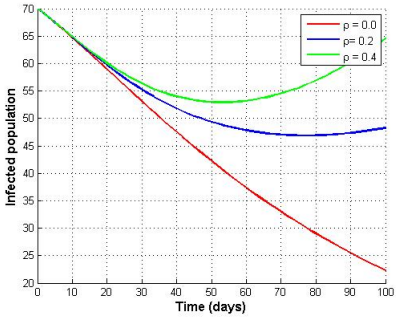

FIGURE 6. Impact of weaned vaccine(ρ) on measles prevalence

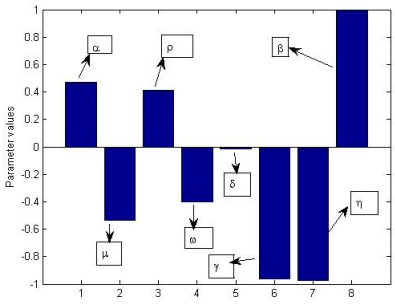

FIGURE 7. Sensitivity index of the basic reproduction number

6. ACKNOWLEDGEMENTS

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