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Modelling Measles Reoccurrence in Vaccinated Infants

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Abstract

Measles is a highly contagious viral disease caused by the *morbillivirus*, marked by symptoms including fever, cough, runny nose, conjunctivitis, and a characteristic widespread rash. In severe cases, especially among young children and pregnant women, it can lead to complications such as ear infections, pneumonia, encephalitis, and death. This study develops a six-compartment deterministic mathematical model, expressed as a system of ordinary differential equations, to investigate the transmission dynamics of measles in human populations. The model was demonstrated to be both mathematically and epidemiologically well-posed. The basic reproduction number (R_0) was derived, and the stability analysis of the disease-free equilibrium showed it to be locally and globally asymptotically stable when $R_0 < 1$, and unstable when $R_0 > 1$. Sensitivity analysis using normalized forward sensitivity indices revealed the impact of various parameters on R_0. Specifically, parameters with negative indices, such as the vaccination rate and treatment rate reduce R_0 when increased, while those with positive indices, such as the effective contact rate increase R_0 when increased. These findings underscore the importance of increasing vaccination coverage, enhancing treatment efforts, and isolating infected individuals to control and prevent measles outbreaks. The model provides a theoretical framework for designing

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effective public health strategies to minimize the disease burden in the population.

Keywords: Modeling; Measles; Transmission Dynamics; Vaccination; Public Health Control Strategies

Introduction

Measles virus is a paramyxovirus disease of genius morbillivirus. It is highly contagious, serious airborne disease caused by a virus that can lead to severe complications and death. It is an infectious disease highly contagious respiratory disease through person – to-person transmission mode, with over 93% transmission rates among susceptible persons. It is the worst eruptive fever during childhood. It also shows characteristics of reddish rash, fever, and leads to serious and fatal complications including, diarrheal, pneumonia, and encephalitis [1]. It can affect anyone but is most common among children. The mortality rate and the incidence rate of different infectious disease such as measles, cholera, and tuberculosis is a major public health concern in the developing countries. Most infectious diseases are caused by micro-organisms such as virus, parasites, bacteria, fungi. There modes of transmission is through respiratory droplets such as coughing, sneezing or contact with an infected person. Transmission spread of these diseases to human population depends on various factors, such as number of susceptible, infective, exposed, modes of transmission (social, ecological, geographical) conditions. These diseases are spread by direct contact between infective and susceptible from droplet of an infected individual by talking, sneezing, coughing, drinking, kissing, or body contact. Diseases such as measles are mostly spread by respiration, while others are spread by vectors or bacteria [2]. Over ten million two hundred thousand (10.2 million) deaths annually attributed to infectious diseases and most of these diseases occur in developing countries [3].

Measles can lead to serious complications such as ear infections pneumonia and even death in young children, pregnant women and people with weakened immune system. Many infected children suffer blindness, impaired vision or deafness. Measles confers a lifelong immunity from further attacks. Measles vaccination have been very effective, and it's been prevented by MMR (Measles Mumps Rubella) vaccine. Before the vaccination program an estimated value of five million to six million (5million -6 million) people are

yearly infected and 6000-7000 deaths are confirmed, also 52,000 are hospitalized and develops a chronic disability from measles encephalitis. However, the global vaccination as helped in reducing the global incidence through the vaccination, but measles remains a public health problem in most part of developing countries. For instance, vaccination coverage is not uniformly high in Nigeria, measles stands as the leading vaccine-preventable killer of many children in Nigeria. As of January 2022, 254 cases of measles were confirmed in Nigeria. At the end of 2021, there were over 10,000 such cases in the country [4]. There have been 11 outbreaks (defined as more related cases) reported in 2024, and 67% of cases (101 of 151) are outbreak- associated. 4 outbreaks were reported during 2023 and 48% of cases (28 of 58) were outbreak-associated [5]. In 2018, about 140,000 people died from measles globally [4]. The overall case fatality rate for children below 5 years was 12.6%, for unvaccinated children below 5, 16.2% and among children below 9 months, 24%.

Measles can be transmitted from an infected person to another through a contagious respiratory disease or through body contact with an infected person. Measles can be contacted through kissing, hugging, exchange of sweat from an infected person or close respiratory contact with an infected individual. Measles cannot be contacted through handshaking, dishes, door knobs or drinking glasses. An infected person can spread the virus at any stage of the infection. Some people develop the measles symptoms shortly after been infected, but these symptoms quickly show in the children between the ages of 2-8 years. Early detention of the virus can help to reduce complications by using medications and vaccinations.

Some researchers have worked on the model of measles. Few among them are:

[1] developed a mathematical analysis of effect of measles. This paper presents a robust compartmental mathematical model of (SVEIR). It shows model has a disease-free equilibrium which is globally asymptotically stable (GAS). There also exist a unique endemic equilibrium point which is locally stable whenever the association threshold quantity (R_0) $R_0>1$. Range – Kutta of order (4) was used to solve the model numerically. [6] developed a mathematical model for control of measles epidemiology. They used SEIR model to determine the impact of exposed individuals at latent period through the stability analysis and numerical simulation. [7] based his work on the dynamical analysis of a new model for measles infection. His study used SEIR model modified by adding vaccinated

compartment. His model determined the required vaccination coverage and dosage that will guarantee eradication of measles within a population. [8] also proposed a mathematical model of measles disease dynamics with vaccination by considering the total number of recovered individuals either from natural recovery or recovery due to vaccination. Numerical simulation of the model shows that vaccination is capable of reducing the number of exposed and infectious population. [9] performed a study on predicting and preventing measles epidemics in New Zealand with application of mathematical model. In their study they used a deterministic SIR to model the dynamics of measles under varying immunization strategies in a population with size and age structure. The model successfully predicted an epidemic in 1997 and was instrumental in the decision to carry out an intensive MMR (Measles, Mumps and Rubella) immunization campaign in that year in New Zealand.

However, the aim of this research is to carry out a mathematical model of measles reoccurrence in vaccinated infants and isolate an infected individual for comprehensive treatment. Extant studies on vaccination of measles as infectious disease have shown that the introduction of vaccines has led to a tremendous reduction in measles cases. However, recent observations in the public health literature have shown that there are cases and instances of reoccurrence of measles among vaccinated infants. It is therefore against this background that this study seeks to provide an explanation on the reasons for reoccurrence of measles in Nigeria. A gap filled by this study.

Model Formulation

A mathematical model for measles was formulated in other to study and analyze the transmission dynamics of measles in human population. The mathematical model was governed by a system of ordinary differential equations which was subdivided into six mutually exclusive classes, namely; Susceptible human $S_h(t)$, exposed human $E_h(t)$, isolated human $J_h(t)$, infectious human $I_h(t)$, vaccinated human $V_h(t)$and recovered human $R_h(t)$, respectively. In this research, A population size of $N(t)$ was partitioned into 6 subclasses of individual with sizes denoted by $S+V+E+I+J$ and $R(t)$, respectively such that $N = S+V+E+I+J+R$ as shown in figure 1 below

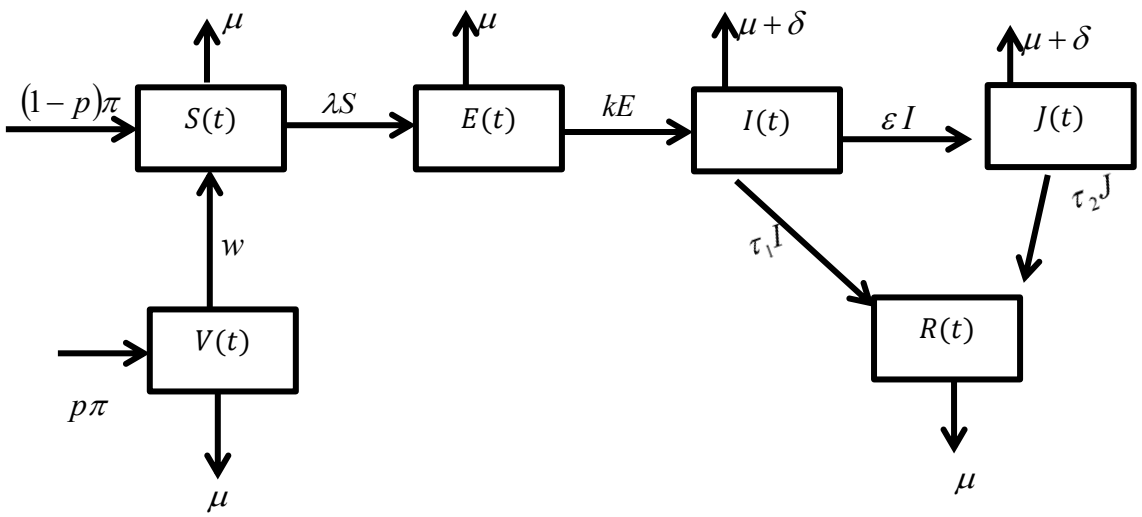


Figure 1: Schematic Diagram of the Model

The following system of ordinary differential equation of the proposed model is therefore considered:

$$\frac{dS}{dt} = (1-p)\pi - \left(\frac{\lambda S}{A}\right) - \mu S + \omega V + \sigma R$$

$$\frac{dE}{dt} = \frac{\lambda S}{A} - (k+\mu)E$$

$$\frac{dI}{dt} = kE - (\varepsilon + \tau_1 + \mu + \delta)I \qquad (1)$$

$$\frac{dJ}{dt} = \varepsilon I - (\tau_2 + \mu + \delta)J$$

$$\frac{dV}{dt} = P\pi - \omega V\ (\omega + \mu)V$$

$$\frac{dR}{dt} = \tau_1 I + \tau_2 J - (\delta + \mu)R$$

where $\lambda = \beta\eta_d I$

The model parameters are defined as follows

Table 1: Variables and Definitions of Sub-population used as parameters

Variable	Definition
S (t)	Number of Susceptible at times (t)
E (t)	Number of Exposed at times (t)
I (t)	Number of infected at times (t)
J (t)	Number of Isolated Individuals at times (t)
V (t)	Number of Vaccinated at times (t)
R (t)	Number of Recovered at times (t)
P	Vaccine Rate
π	Birth Rate
β	Contact Rate
μ	Natural death rate
ω	Vaccine waning rate
σ	Loss of immunity
$\tau_1\tau_2$	Treatment for infected and isolated individuals
ε	Rate of Isolation
k	Progression rate to infectious class
δ	Disease induced death rate
η_d	Modification parameter based on Area

The Invariant Region

Lemma 1: The feasible region of the measles model given by

$$D = \left\{ (S,V,E,I,J,R) \in R_+^6 : S+V+E+I+J+R \le \frac{\pi}{\mu} \right\} \tag{2}$$

is positively invariant and attracting

Proof: Let the total human of the model be denoted by N(t). Adding all the parameters of the model (1) together, then, the rate of change of total human population gives

$$\frac{dN}{dt} = \pi - \mu N - \alpha(I+J) \tag{3}$$

So that,

$$\frac{dN}{dt} \leq \pi - \mu N \tag{4}$$

Then, using method of integrating factor,

$$\frac{dN}{dt} - \mu N \leq \pi$$

Solving this gives

$$N(t) \leq N(0)e^{-\mu t} + \frac{\pi}{\mu} - \frac{\pi}{\mu}e^{-\mu t}$$
$$N(t) \leq N(0)e^{-\mu t} + \frac{\pi}{\mu}(1 - e^{-\mu t}) \tag{5}$$

Since $N(t) \leq \frac{\pi}{\mu}$ wherever $N(0) \leq \frac{\pi}{\mu}$, then the region D is positively invariant, Further, if $N(0) > \frac{\pi}{\mu}$, either the solution enters D in finite time or the total population tends to the limit $\frac{\pi}{\mu}$ and the infected classes tends to zero. Therefore, the feasible region D is attracting, which implies that all solutions initiated in R_+^6 eventually enter D. Therefore, it is sufficed to study the dynamics of measles in the feasible region D, where the model is considered to be mathematically and epidemiologically well-posed.

Positivity and Boundedness of solution of the model.

Lemma 2: The solution set $\{S(t), V(t), E(t), I(t), J(t), R(t)\}$ of the measles model (1) with positive initial data in D, remains positive in D for all time $t > 0$.

Proof:

The first compartment of the model:

$$\frac{ds}{dt} = (1-p)\pi + \sigma R - \frac{\lambda S}{A} - \omega v - \mu S$$

$$\frac{ds}{dt} + \left(\frac{\lambda}{A} + \mu\right)S(t) \geq 0$$

Using the integrating factor method gives

$$I.F = e^{\int_o^t \left(\frac{\lambda}{A} + \mu\right)dt}$$

simplifying further yields

$$S(t) = S_0 \geq e^{\int_o^t \left(\frac{\lambda}{A}+\mu\right)dt} \geq 0$$

Thence

$S(t) \geq 0$ for all $t \geq 0$

The remain variables can be solved following same procedure to be positive.Therefore, all the state variables are non-negative.

Mathematical Analysis of the model

Disease Free equilibrium

At DFE, it is assumed that there is no infection, i.e E=I=J=0 but the DFE of the model be denoted by $\left(\varepsilon^0\right)$ such that, at critical points, $\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dJ}{dt} = \frac{dR}{dt} = 0$.Then the model equation (3.1.2) becomes:-

Disease –free Equilibrium at disease free, E=I=J =R =0

$$0 = (1-P)\pi - \mu S + \omega V$$

$$0 = P\mu - (\omega + \mu)V$$

Solving for S and V simultaneously, we have our disease-free equilibrium points as

$$\therefore \varepsilon^0 = \left(S^0, V^0, E^0, I^0, J^0, R^0\right)$$

$$= \left(\frac{1}{\mu}\left[(1-p)\pi + \frac{\omega p\pi}{w+\mu}\right], \frac{p\pi}{\omega+\mu}, 0,0, 0,0\right) \quad (6)$$

Endemic Equilibrium Point

At endemic equilibrium, disease is present but the endemic equilibrium point be denoted by $\left(\varepsilon^{**}\right)$, then at steady states,

$$\frac{dS^{**}}{dt} \neq 0, \frac{dV^{**}}{dt} \neq 0, \frac{dE^{**}}{dt} \neq 0, \frac{dI^{**}}{dt} \neq 0, \frac{dJ^{**}}{dt} \neq 0, \frac{dR^{**}}{dt} \neq 0.$$

since $\varepsilon^{**} = (S,V,E,I,J,R) = \left(S^{**}, V^{**}, E^{**}, I^{**}, J^{**}, R^{**}\right)$

hen, solvingsimultaneously, the endemic equilibrium for model (1) is obtained as thus:

$$V^{**} = \frac{p\pi}{w+\mu}; I^{**} = \frac{kE^{**}}{\varepsilon+\tau_1+\mu+\delta}; J^{**} = \frac{k\,\varepsilon\,E^{**}}{(\varepsilon+\tau_1+\mu+\delta)(\tau_2+\mu+\delta)},$$

$$R^{**} = \frac{E^{**}}{\sigma+\mu}\left[\frac{\tau_1 k}{\varepsilon+\tau_1+\mu+\delta} + \frac{\tau_2 k\varepsilon}{(\varepsilon+\tau_1+\mu+\delta)(\tau_2+\mu+\delta)}\right];$$

$$S^{**} = (1-p)\pi + \frac{\omega p\pi}{w+\mu} + \frac{\sigma+w}{\sigma+w}\left[\frac{\tau_1 k}{\varepsilon+\tau_1+\mu+\delta} + \frac{\tau_2\, k\,\varepsilon}{(\varepsilon+\tau_1+\mu+\delta)(\tau_2+\mu+\delta)}\right];$$

$$E^{**} = \frac{\mu\sigma A(\varepsilon+\tau_1+\mu+\delta)^2(\tau_2+\mu+\delta)(\sigma+w)(k+\mu)(R_0-1)}{\beta\eta_d k\sigma\{(\sigma+w)(k+w)(\varepsilon+\tau_1+\mu+\delta)(\tau_2+\mu+\delta)-\sigma k[\tau_1(\tau_2+\mu+\delta)+\tau_2\varepsilon]\}}. \quad (7)$$

The endemic exists only if $R_0 > 1$

If $R_0 < 1$, no endemic equilibrium existsand also coincides with the DFE

Basic Reproduction Number

The basic reproduction number (R_0) is a fundamental concept in epidemiology that represents the average number of secondary cases generated by a single infectious person in a completely susceptible population, during the early stages of an outbreak [10].

Considering the infection related compartment.

$$\frac{dE}{dt} = \frac{\lambda S}{A} - (K+\mu)E$$
$$\frac{dI}{dt} = KE - (\varepsilon+\tau_1+\mu+\delta)I$$
$$\frac{dJ}{dt} = \varepsilon I - (\tau_2+\mu+\delta)J$$
$$\frac{dR}{dt} = \tau_1 I + \tau_2 J - (\sigma+\mu)R$$

where

$$K_1 = K + \mu$$

$$K_2 = \varepsilon + \tau_1 + \mu + \delta$$
$$K_3 = \tau_2 + \mu + \delta$$
$$K_4 = \sigma + \mu$$

$$F=\begin{pmatrix}\frac{\beta n_d I S_0}{A}\\0\\0\end{pmatrix},V=\begin{pmatrix}K_1E\\K_2I-KE\\K_3J-\varepsilon_1 I\end{pmatrix} \tag{8}$$

$$F=\begin{pmatrix}0&\frac{\beta n_d S_0}{A}&0\\0&0&0\\0&0&0\end{pmatrix},\ V=\begin{pmatrix}K_1&0&0\\-K_1&K_2&0\\0&-\varepsilon&K_3\end{pmatrix}$$

$$FV^{-1}=\begin{pmatrix}0&\frac{\beta\eta_d S_0}{A}&0\\0&0&0\\0&0&0\end{pmatrix}\begin{pmatrix}\frac{1}{K_1}&0&0\\\frac{K}{K_1K_2}&\frac{1}{k_2}&0\\\frac{k\varepsilon}{K_1K_2K_3}&\frac{\varepsilon}{K_2K_3}&\frac{1}{K_3}\end{pmatrix}$$

$$=\begin{pmatrix}\frac{\beta\eta_d KS_0}{Ak_1k_2}&\frac{\beta\eta_d S_0}{Ak_2}&0\\0&0&0\\0&0&0\end{pmatrix}$$

$\left|FV^{-1}-\lambda I\right|=0,$ where I is a 3x3 identify matrix

$$\begin{vmatrix}\frac{\beta\eta_d KS_0}{AK_1K_2}-\lambda&\frac{\beta\eta_d S_0}{AK_2}&0\\0&-\lambda&0\\0&0&-\lambda\end{vmatrix}=0 \tag{9}$$

$$\Rightarrow\left(\frac{\beta\eta_d KS_0}{AK_1K_2}-\lambda\right)\lambda^2=0$$

$$\Rightarrow\left(\frac{\beta\eta_d kS_0}{Ak_1k_2}-\lambda\right)\lambda^2=o$$

$$\Rightarrow \lambda^2 = 0 \quad or \quad \frac{\beta \eta_d K S_0}{A K_1 K_2} - \lambda = 0$$

$$\Rightarrow \lambda = 0 \quad (twice) \quad or \quad \lambda = \frac{\beta \eta_d K S_0}{A K_1 K_2} = R_0$$

The basic reproduction number $R_0 = \rho\left(FV^{-1}\right)$, is the spectral radius of the dominant eigenvalue of (9).

Hence, $R_0 = \frac{\beta \eta_d K}{\mu A(K+\mu)(\varepsilon+\tau_1+\mu+\delta)}\left[(1-p)\pi + \frac{\omega p \pi}{(\omega+\mu)}\right]$ (10)

Stability Analysis of the DFE

Local Stability

Theorem 1: If $R_0 < 1$, then the disease-free equilibrium is locally asymptotically stable, and unstableif otherwise

Proof: using the Jacobian matrix method

$$J(\varepsilon^0) = \begin{pmatrix} -\mu & 0 & \frac{\beta \eta_d S_0}{A} & 0 & w & \sigma \\ 0 & -(K+\mu) & \frac{\beta \eta_d S_0}{A} & 0 & 0 & 0 \\ 0 & K & -(\varepsilon+\tau_1+\mu+\delta) & 0 & 0 & 0 \\ 0 & 0 & \varepsilon & -(\tau_2+\mu+\delta) & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\omega+\mu) & 0 \\ 0 & 0 & \tau_1 & \tau_2 & 0 & -(\sigma+\mu) \end{pmatrix} \quad (11)$$

To final the eigen values, we solving the characteristics equation, we obtained the eigen values as, $\lambda_1 = -\mu,\ \lambda_2 = (\tau_2+\mu+\delta+\lambda), \lambda_3 = -(\omega+\mu), \lambda_4 = -(\sigma+\mu)$

and the roots of

$$\lambda^2 + \left[(K+\mu)+(\varepsilon+\tau_1+\mu+\delta)\right]\lambda + (K+\mu)(\varepsilon+\tau_1+\mu+\delta) - \frac{K\beta\eta_d S_0}{A} = 0.$$

$$\lambda^2+\left[\left(K+\mu\right)+\left(\varepsilon+\tau_1+\mu+\delta\right)\right]\lambda+\left(K+\mu\right)\left(\varepsilon+\tau_1+\mu+\delta\right)\left[\frac{K\beta\eta_d S_0}{\left(K+\mu\right)\left(\varepsilon+\tau_1+\mu+\delta\right)A}\right]=0$$

$$\lambda^2+\left[\left(K+\mu\right)+\left(\varepsilon+\tau_1+\mu+\delta\right)\right]\lambda+\left(K+\mu\right)\left(\varepsilon+\tau_1+\mu+\delta\right)\left\{1-\frac{K\beta\eta_d}{\mu A\left(K+\mu\right)\left(\varepsilon+\delta+\mu+\delta\right)}\left[\left(1-P\right)\pi+\frac{\omega p\pi}{\omega+\mu}\right]\right\}=0$$

$$\lambda^2+\left[\left(K+\mu\right)+\left(\varepsilon+\tau_1+\mu+\delta\right)\right]\lambda+\left(K+\mu\right)\left(\varepsilon+\tau_1+\mu+\delta\right)\left(1-R_0\right)=0 \quad (12)$$

By desecrates rule of sign, the polynomial equation (12) has no sign change. Therefore, the disease-free equilibrium is locally asymptotically stable, whenever $R_o<1$ and unstable otherwise.

Global Stability

Theorem 2: If $R_0 \le 1$, then the disease-free equilibrium is globally asymptotically stable, and unstable otherwise.

Proof: Let F be a candidate Lyapunov function for the model, such that:

$$F=\left(S-S^*-S^*\,In\frac{S}{S^*}\right)+\frac{KE}{\left(K+\mu\right)\left(\varepsilon+\tau_1+\mu+\delta\right)}+\frac{I}{\varepsilon+\tau_1+\mu+\delta} \quad (13)$$

where $S^*=\frac{1}{\mu}\left[\left(1-P\right)\pi+\frac{\omega p\pi}{w+\mu}\right]$ is the value of S(t) at DFE. Obviously the second and third terms on the RHS of (13) are positive. For the first term, $S^* \le S$ (since S^* is an equilibrium point of S). Then $S-S^*-S^*\,In\frac{S}{S}$ is also positive.

Therefore $F=F\left(S,E,I\right)$ is positive definite.

Now for the time derivative of F along the solutions of the model equation (13), we have

$$\frac{dF}{dt}=\left(1-\frac{S^*}{S}\right)\frac{dS}{dt}+\frac{K}{\left(K+\mu\right)\left(\varepsilon+\tau_1+\mu+\delta\right)}\frac{dE}{dt}+\frac{1}{\varepsilon+\tau_1+\mu+\delta}\frac{dI}{dt} \quad (14)$$

Substituting $\frac{dS}{dt},\frac{dE}{dt}$ *and* $\frac{dI}{dt}$ from (1) gives

$$\frac{dF}{dt}\left(1-\frac{S^*}{S}\right)\left[(1-P)\pi-\frac{\beta\eta_d I S}{A}-\mu S+wV+\sigma R\right]$$

$$+\frac{K}{(K+\mu)(\varepsilon+\tau_1+\mu+\delta)}\left[\frac{\beta\eta_d IS}{A}-(K+\mu)E\right]+\frac{1}{\varepsilon+\tau_1+\mu+\delta}\left[KE-(\varepsilon+\tau_1+\mu+\delta)I\right]$$

$$\frac{dF}{dt}=\left(1-\frac{S^*}{S}\right)\left[\left(\frac{\beta\eta_d I^*S^*}{A}-\frac{\beta\eta_d IS}{A}\right)+\mu(S^*-S)+\omega(V-V^*)+\sigma(R-R^*)\right]$$

$$+\frac{K}{(K+\mu)(\varepsilon+\tau_1+\mu+\delta)}\left[\frac{\beta\eta_d IS}{A}-(K+\mu).\frac{\beta\eta_d I^*S^*}{A(K+\mu)}\right]+\frac{1}{\varepsilon+\tau_1+\mu+\delta}\left[\varepsilon+\tau_1+\mu+\delta(I^*I)\right]$$

At DFE, $I^*=R^*=0,$ so $\frac{dF}{dt}$ becomes

$$\frac{dF}{dt}=-\frac{\beta\eta_d I S}{A}\left(1-\frac{S^*}{S}\right)-\mu(S-S^*)-\omega(V-V^*)+\sigma R$$

$$+\left[\frac{K\beta\eta_d S}{A(K+\mu)(\varepsilon+\tau_1+\mu+\delta)}-1\right]I$$

At disease –free equilibrium,

$$\varepsilon^0=(S^0,V^0,E^{0*},I^0,J^0,R^0)=\left(\frac{1}{\mu}\left[(1-P)\pi+\frac{\omega P\pi}{w+\mu}\right],\frac{P\pi}{w+\mu},0,0,0,0\right), \quad (15)$$

Therefore,

$$\frac{dF}{dt}=-\frac{\beta\eta_d I S}{A}\left(\frac{S-S^*}{S}\right)-\mu(S-S^*)-\omega(V-V^*)+\left\{\frac{\beta\eta_d k}{\mu A(K+\mu)(\varepsilon+\tau_1+\mu+\delta)}\left[(1-P)\pi+\frac{\omega P\pi}{\omega+\mu}\right]-1\right\}I$$

$$\frac{dF}{dt}=-\frac{\beta\eta_d I S}{A}\left(\frac{S-S^*}{S}\right)-\mu(S-S^*)-\omega(V-V^*)+(R_0-1)I \quad (16)$$

Obviously from (16), $\frac{dF}{dt}<0$ *if* $R_0\le 1$

$\frac{dF}{dt}=0$ *iff* $S=S^*, V=V^*$ and $I=0$

$$\text{Thus } (S,V,E,I,J,R) \to \left(\frac{1}{\mu}\left[(1-P)\pi + \frac{\omega P\pi}{\omega+\mu} \right], \frac{P\pi}{\omega+\mu}, 0,0,0,0 \right) \tag{17}$$

as $t \to \infty$, and the target compact invariant set is the singleton $\{\varepsilon^*\}$. So, by Lassalle's invariance principle (Lasalle, 1976), every solution of the model system (1) with initial conditions in D approaches ε^* as $t \to \infty$ whenever $R_0 \le 1$. Hence, the disease-free equilibrium is globally asymptotically stable whenever $R_0 \le 1$, and unstable otherwise.

Sensitivity Analysis

Using approach of [11], the normalized forward sensitivity index of a variable "P" that depends differentiable on a parameter "q" is defined as

$$X_v^{R_0} := \frac{\partial R_0}{\partial v} * \frac{v}{R_0} \tag{18}$$

As we have an explicit formula for R_0 in equation (9), we derive an analytical expression and the associated numeric values for the sensitivity of R_0, as $X_v^{R_0} := \frac{\partial R_0}{\partial v} * \frac{v}{R_0}$ with respect to each of the parameters involved in R_0 in table 2 which is depicted by the bar chart in figure 2:

Table 2. Parameters Values and Sensitivity Indices of R_0

Parameter	Sensitivity Expression	Sensitivity index value
β	1	1
A	-1	-1
π	1	1
δ	$\frac{\delta}{k_2}$	-0.07438
τ	$-\frac{\tau}{k_2}$	-0.661157
η_d	1	1
μ	$\frac{-\mu(k_1+k_2+k_1k_2(1+\omega\rho\pi))}{\mu k_1k_2(\omega+\mu)^2}$	-0.92288
P	$\frac{-p\mu}{\mu(1-p)+\omega}$	0.05263
K	$\frac{\mu}{k+\mu}$	0.06250

Parameter	Sensitivity Expression	Sensitivity index value
ω	$\frac{\omega\rho\mu}{\omega+\mu)(\mu(1-p)+\omega)}$	0.04386
ε	$-\frac{\varepsilon}{k_2}$	-0.24793

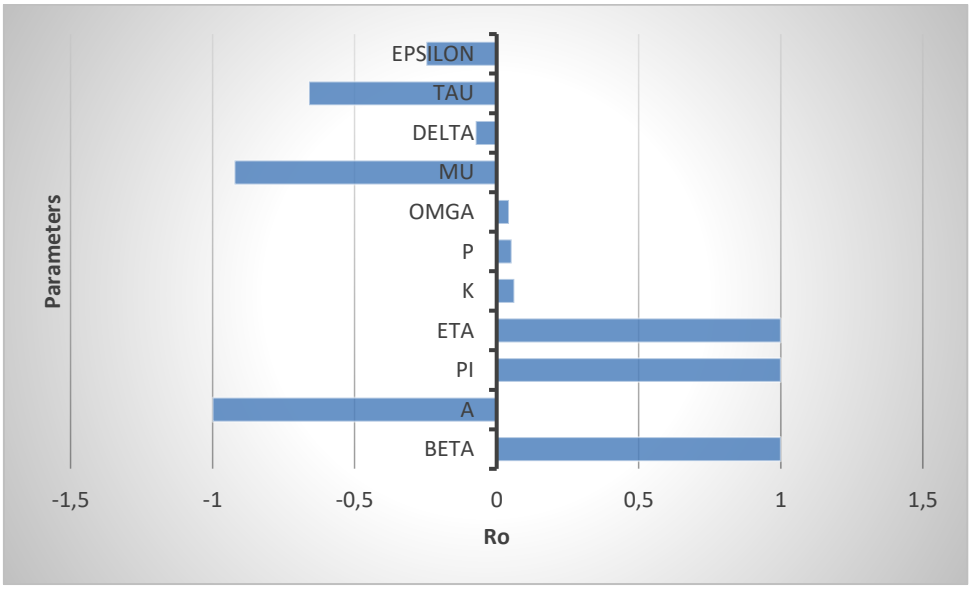


Figure 2. Sensitivity indices value chart

Interpretation of Sensitivity Indices

The Table 2 and figure 2 represents the sensitivity index for the base line parameter values and it shows that when the parameters with positive sensitivity index value increase while the other parameters remain constant, the value of R_0 will also increase implying that they increase the endemicity of the disease as they have positive indices and should be targeted by intervention strategies.

More so, when the parameters with negative sensitivity index values increase while keeping other parameters constant, the value of R_0 will decrease, implying that they decrease the endemicity of the disease as they have negative indices and should equally be targeted by intervention strategies. For instance, $X_{\beta}^{R_0}$ = +1.0 means that increasing or decreasing β_1 by 100% increases or (decreases) R_0 by 100% while $X_{\tau_1}^{R_0}$ = - 0.661157 means that increasing or (decreasing) τ_1 by 100% decreases (or increases) R_0 by 66.1157%. we can easily calculate for other parameters following similar procedure.

Numerical Simulations and Discussion

This section presents the numerical simulation results of modeling measles reoccurrence in vaccinatedinfants.

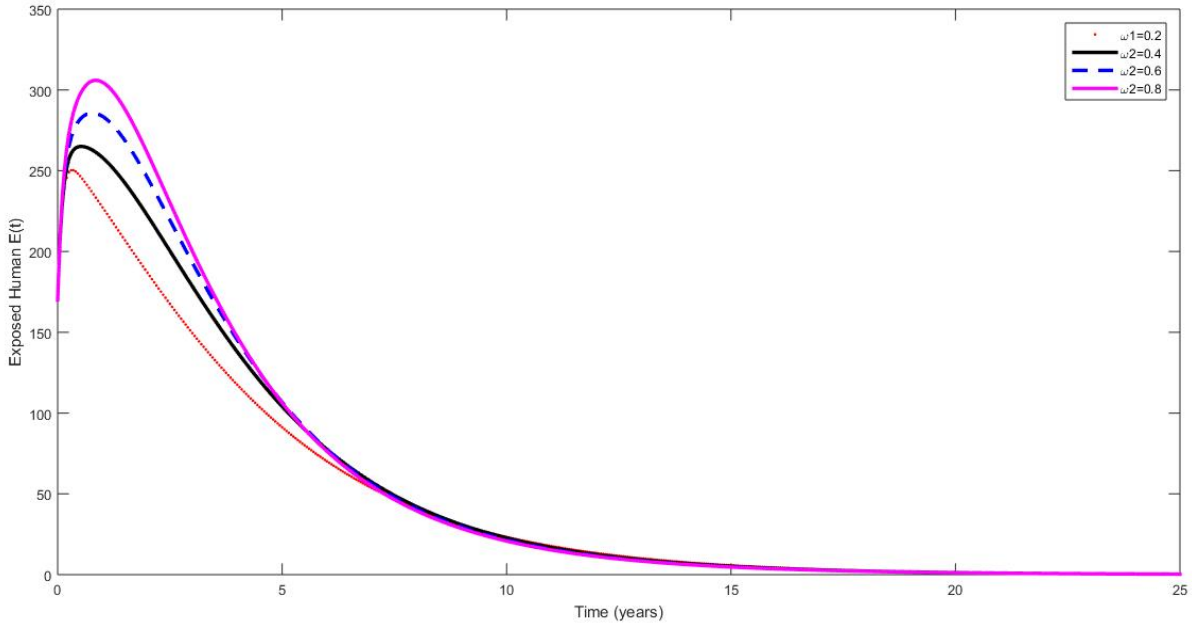


Figure 3. The Exposed Human against time

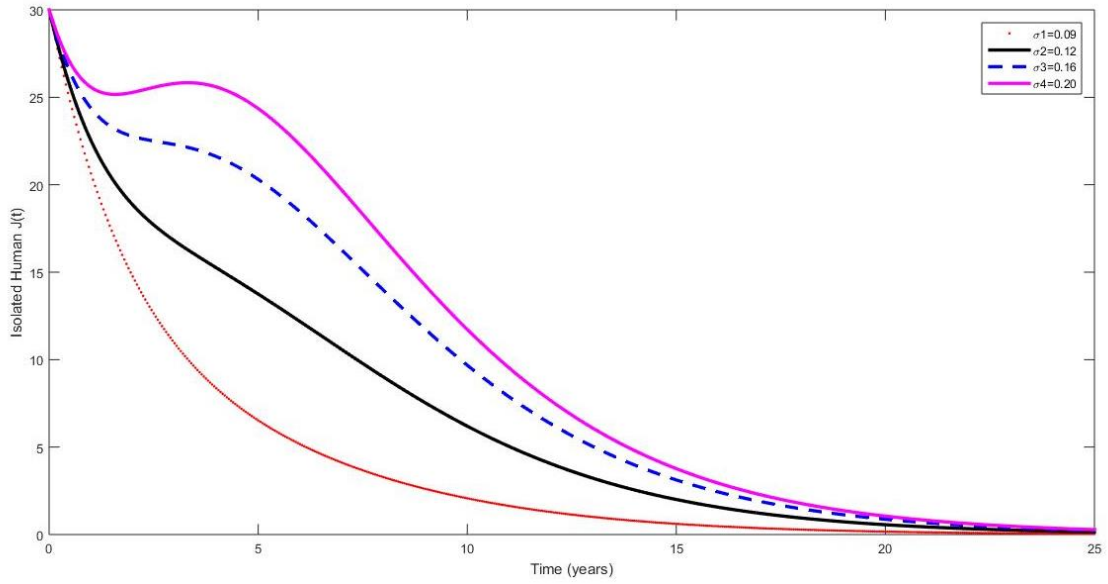


Figure 4. The Isolated Human against time

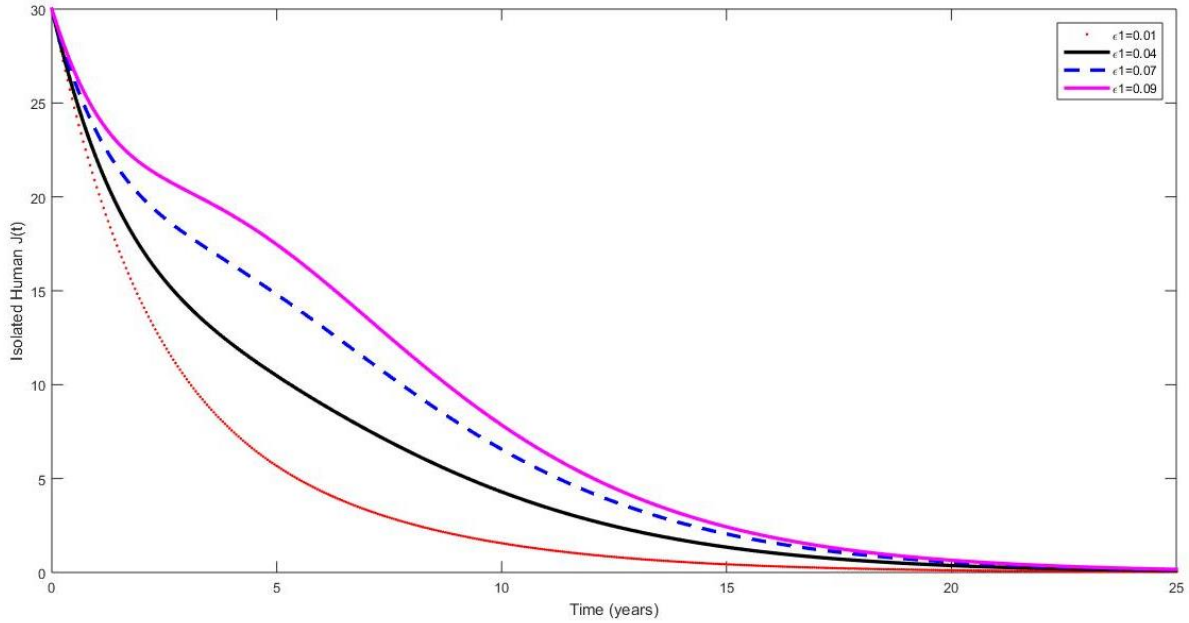


Figure 5. The Isolated Human against time

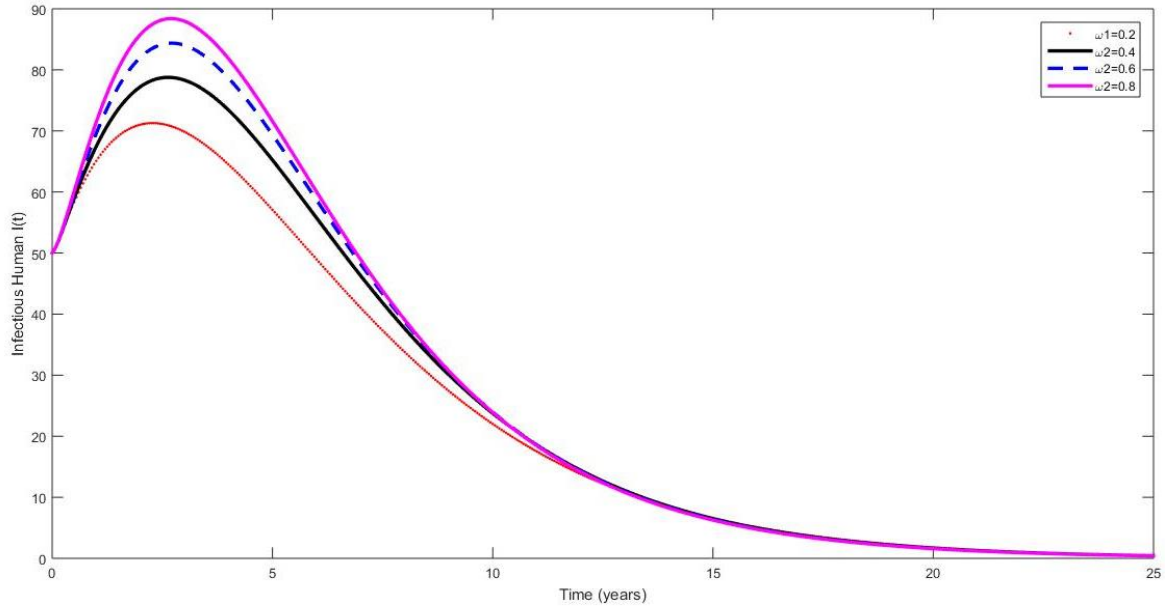


Figure 6. The Infectious Human against time

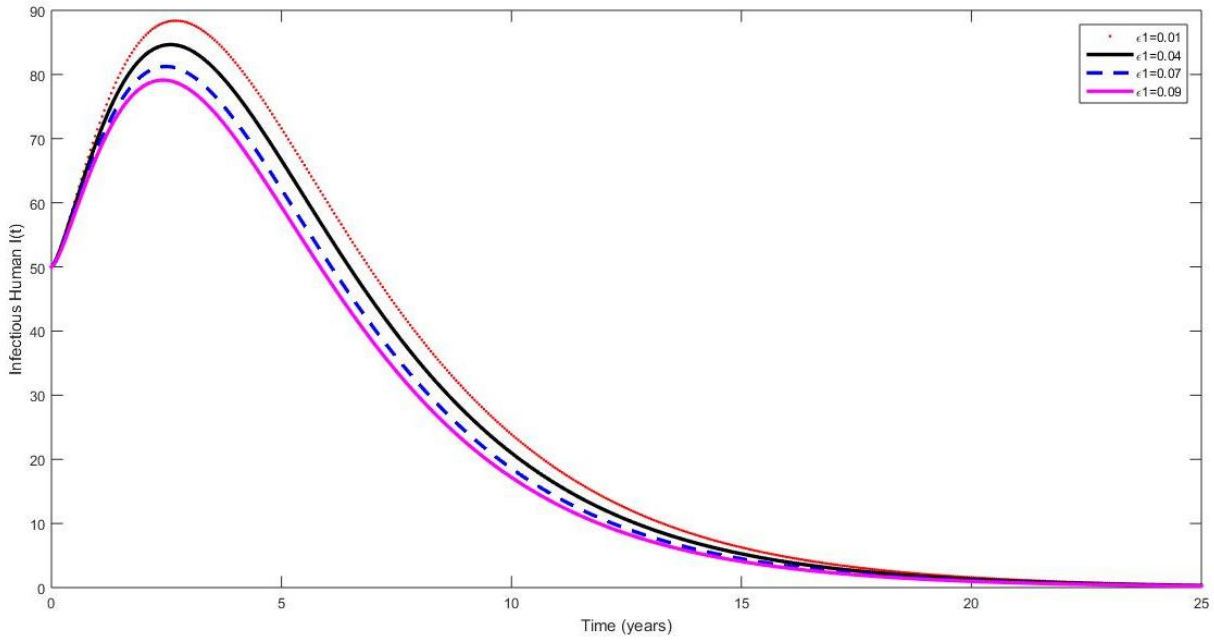


Figure 7. The Infectious Human against time

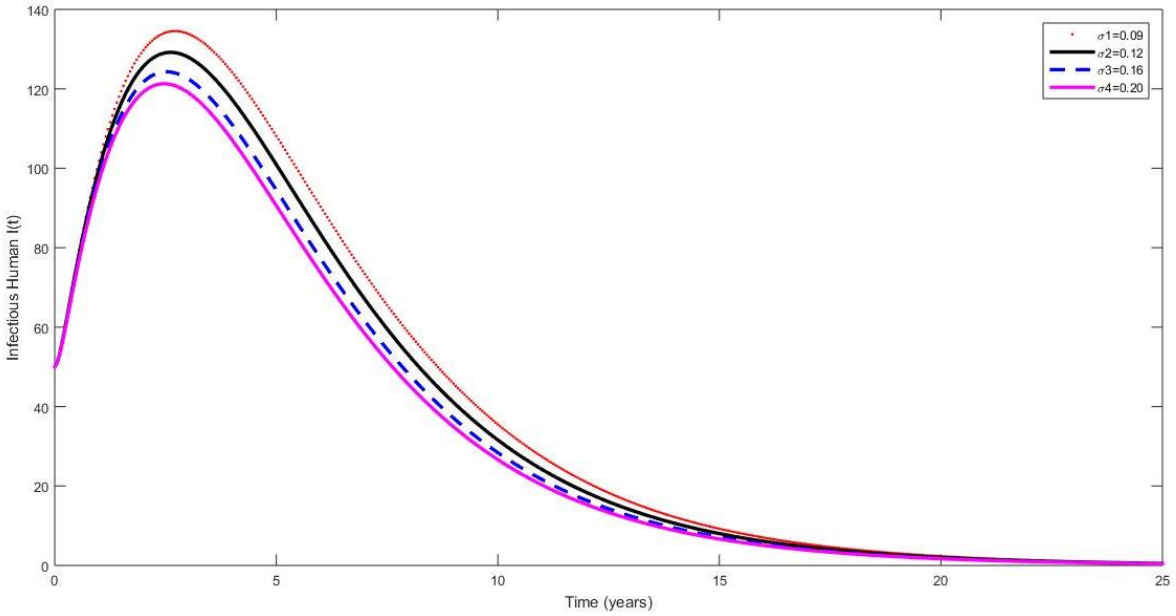


Figure 8. The Infectious Human against time

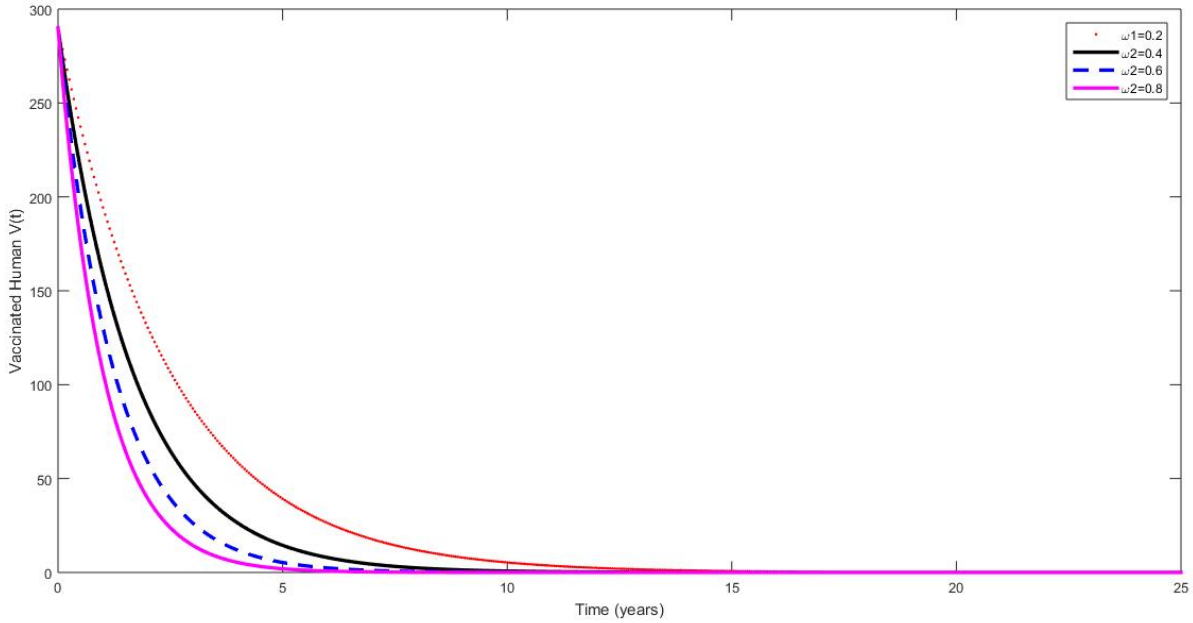


Figure 9. The Vaccinated Human against time

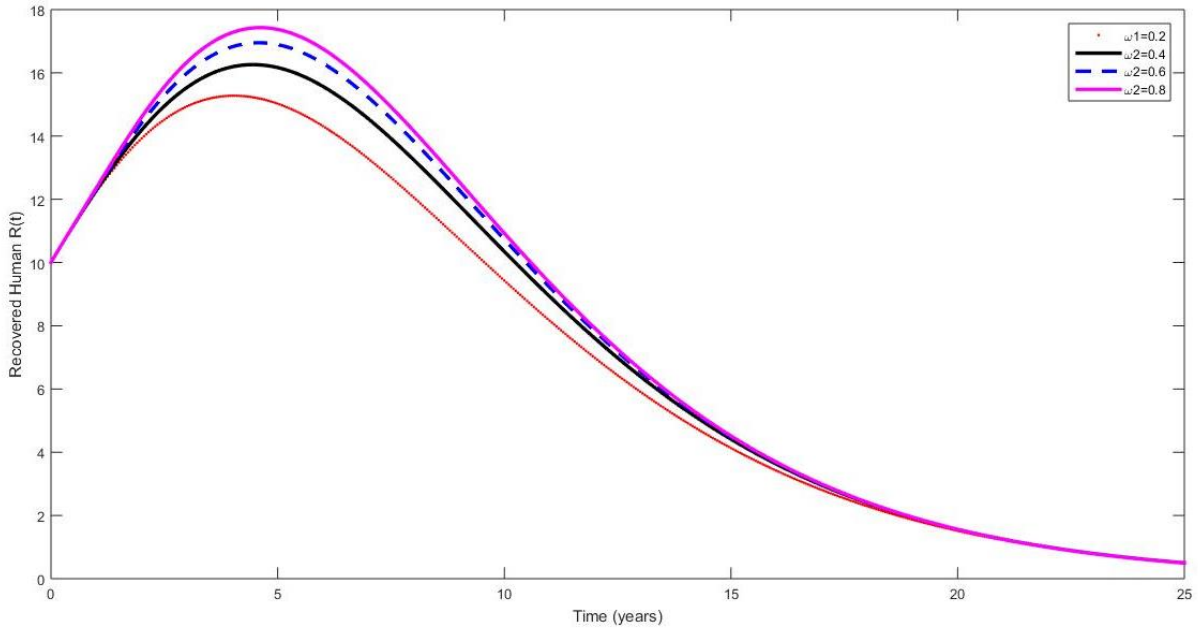


Figure 10. The Recovered Human against time

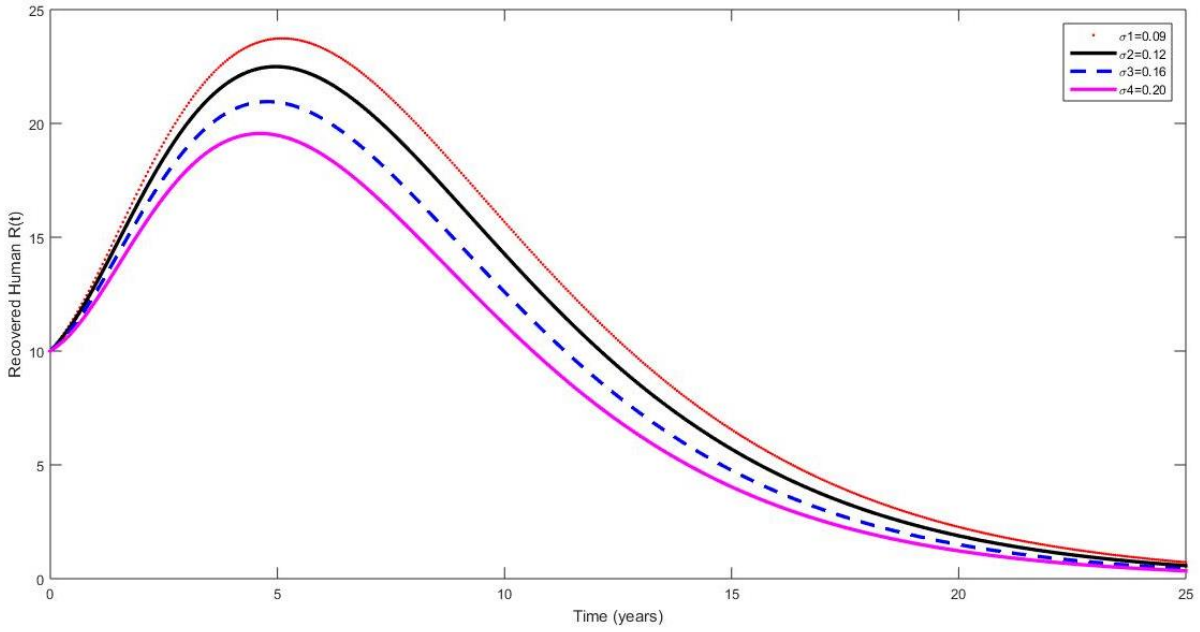


Figure 11. The Recovered Human against time

Discussion of Result

Fig 3 shows the graph of Exposed Human (E) against time (t). It represents the effect of $loss\ of\ imunnity\ (\sigma)$ on the population of the Exposed class. It was noted that at $\sigma_2 = 0.8$, there is an Outright increase in Human exposure population, the figure shows that there is high degree of exposurewithin the first three years and that the strength of exposure has greatly reduced at the end of the five years which further reduces till it vanishes at time (t) = 20 years, the figure indicate that there is no further rise in exposure rate as indicated from time (t) = 20 years to time (t) = 25 years. The figure shows that there is slight drop in the rate of exposure at $\sigma_2 = 0.6$, $\sigma_2 = 0.4$ and $\sigma_2 = 0.2$ respectively. It was indicated that the rate of exposure vanishes at the same point and there is no further rise in exposure just as when $\sigma_2 = 0.8$,

Fig 4 shows the graph of Isolated Human (J) against time (t). It represents the effect of loss of immunity(σ) on the population of the isolated class. It was noted that at $\sigma_2 = 0.20$and$\sigma_2 = 0.16$, there is inconsistent reduction in the number of isolated individuals for the first five years. At $\sigma_2 = 0.09$ there is a great reduction in the number of

isolated humans as indicated by the curve in the figure. However, the figure shows that the rate of Isolated Human terminated at the same point at time (t) = 25 years.

Fig 5 shows the graph of Isolated Human (J) against time (t). It represents the effect of isolation(ε) on the population of the isolated class. It was noted that at $\in_1 = 0.09$and $\in_1 = 0.07$, there is inconsistent reduction in the number of isolated individuals for the first five years. At $\in_1 = 0.04$, $\in_1 = 0.01$ there is a great reduction in the number of isolated humans as indicated by the curve in the figure. However, the figure shows that the rate of Isolated Human terminated at the same point at time (t) = 25 years.

Fig 6 shows the graph of Infectious Human (I) against time (t). It represents the effect of waning of vaccine(ω) on the population of the Infectious class. It was noted that at $\omega_2 = 0.8$, there is an Outright increase in Infectious Human population, the figure shows that there is high degree of Infectious human recorded within the first four to five years and that the strength of the infection has greatly reduced at the end of the tenth year which further reduces till it vanishes at time (t) = 25 years. The figure shows that there is gradual drop in the rate of infectious human population rateat $\omega_2 = 0.6$, $\omega_2 = 0.4$ and $\omega_2 = 0.2$ respectively. It was indicated that the rate of infectious human vanishes at the same point at time (t)= 25 years

Fig 7 shows the graph of Infectious Human (I) against time (t). It represents the effect of Isolation ($\in_1$) on the population of the Infectious class. It was noted that at $\in_1 = 0.01$, there is an Outright increase in Infectious Human population, the figure shows that there is high degree of Infectious human recorded within the first four to five years and that the strength of the infection has greatly reduced at the end of the tenth year which further reduces till it vanishes at time (t) = 25 years. The figure shows that there is gradual drop in the rate of infectious human population rate at $\in_1 = 0.04$, $\in_1 = 0.07$ and $\in_1 = 0.09$ respectively. It was indicated that the rate of infectious human vanishes at the same point at time (t) = 25 years

Fig 8 shows the graph of Infectious Human (I) against time (t). It represents the effect of loss of immunity (σ_1) on the population of the Infectious class. It was noted that at $\sigma_1 = 0.09$, there is an Outright increase in Infectious Human population, the figure shows that there is high degree of Infectious human recorded within the first four to five years and that the strength of the infection has greatly reduced at the end of the tenth year

which further reduces till it vanishes at time (t) = 25 years. The figure shows that there is gradual drop in the rate of infectious human population rate at $\sigma_2 = 0.12$, $\sigma_3 = 0.16$ and $\sigma_4 = 0.20$ respectively. It was indicated that the rate of infectious human vanishes at the same point at time (t) = 25 years

Fig 9 shows the graph of Vaccinated Human (V) against time (t). the graph shows that (ω), the vaccination administration on the infectious human population at the beginning was very high. It was noted that at $\omega_2 = 0.8$ the vaccinated human reduced greatly for the next five (5) years from around 290 to about 40 and then gradually till it terminated at time t = 15 years. The graph further explained that there is no further rise in the vaccinated human indicating the vaccination is accurately administered and that can be achieved by the professionalism of the administering personnel and obedience of the infectious human by complying by the medication guide. The figure shows that there is gradual drop in the rate of infectious human population rate at $\omega_2 = 0.6$, $\omega_2 = 0.4$ and $\omega_2 = 0.2$ respectively but at $\omega_2 = 0.4$ the Vaccinated Human rate vanished at time (t) = 11 years, $\omega_2 = 0.6$ vanished at time (t) = 9 years and $\omega_2 = 0.8$ vanished at time (t) = 7 years. It was indicated that the rate of vaccinated human did not vanish at the same point, it means adequate vaccination produced a corresponding effective solution base on the degree of the disease.

Fig 10 shows the graph of Recovered Human (R) against time (t). It represents the effect of vaccine(ω) on the population of the Recovered class. It was noted that at $\omega_2 = 0.8$, there is an Outright increase in Recovered Human population, the figure shows that there is high degree of Recovered human recorded within the first ten years and that the human population at the recovery stage continue to increase until after ten (10) years when there is a gradual fall in the recovered population. The figure indicated that the recovery although minimal is still continue after the 25th year period thereby revealing that people will undergo further recovery care after the 25th year. This also apply for $\omega_2 = 0.6$, $\omega_2 = 0.4$ and $\omega_2 = 0.2$ respectively although there is a slight fall in their respective recovered rate. At time (t) = 20 the Recovered human population for $\omega_2 = 0.8$, $\omega_2 = 0.6$, $\omega_2 = 0.4$ and $\omega_2 = 0.2$ the recovered human population rate become equal

Fig 11 shows the graph of Recovered Human (R) against time (t). It represents the effect of loss of immunity(σ) on the population of the Recovered class. It was noted that

at $\sigma_1 = 0.09$, there is an Outright increase in Recovered Human population, the figure shows that there is high degree of Recovered human recorded within the first ten years and that the human population at the recovery stage continue to increase until after ten (10) years when there is a gradual fall in the recovered population. The figure indicated that the recovery although minimal is still continue after the 25th year period thereby revealing that people will undergo further recovery care after the 25th year. This also apply for$\sigma_2 = 0.12$, $\sigma_3 = 0.16$and $\sigma_4 = 0.20$ respectively although there is a slight fall in their respective recovered rate. The Recovered human population curve for$\sigma_1 = 0.09$ $\sigma_2 = 0.12$, $\sigma_3 = 0.16$ and $\sigma_4 = 0.20$ is neither equal nor cross over one another but rather continue to reduce till after 25 years.

Conclusion

This study was formulated and analyzed as a mathematical model for Measles in order to gain more insights and understanding in the epidemiological features of some parameters on thetransmission dynamics of Measles in human population. The mathematical model for Measles was shown to mathematically andepidemiologically well posed through the theory of positivity and boundedness of solution in D. The disease-free and endemic equilibrium points of the model were obtained. The basic reproduction number of the model wascalculated using next generation matrix method and the stability of the disease-freeequilibrium was investigated and shown to be locally asymptotically stable whenever the basic reproduction number $R_0<1$ and unstable if otherwise. The global asymptotic stability of the model was shown to be globally asymptotically stable whenever the associated threshold parameter $R_0<1$ and unstable if otherwise. The effect of some parameters of the model relative to the basic reproduction number was calculated using the normalized forward sensitivity indices and it was established that the parameters with negative indices will reduce the value of the basic reproduction number while those with positive indices will increases the value of the basic reproduction number.

Therefore, epidemiological features such as the vaccination rate, effective contact rate, progression rate and treatment rate should be given great attention in order to effectively control and prevent the dynamical spread of Measles infections in human population.

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