Asian Research Journal of Mathematics

12(3): 1-11, 2019; Article no.ARJOM.43960
ISSN: 2456-477X

Simple MSEIR Model for Measles Transmission

Mojeeb Al-Rahman EL-Nor Osman[1,2*], Appiagyei Ebenezer[1,3] and Isaac Kwasi Adu[1,3]

[1]School of Mathematics and Statistics, Central China Normal University, Wuhan 430079, P.R. China.
[2]Department of Mathematics and Computer Science, International University of Africa, P.O.Box 2469, Khartoum, Sudan.
[3]Department of Mathematics, Valley View University, Techiman Campus, P.O.Box 183 B/A, Ghana.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/ARJOM/2019/v12i330090
Editor(s):
(1) Dr. Hari Mohan Srivastava, Professor, Department of Mathematics and Statistics, University of Victoria, Canada.
Reviewers:
(1) Grienggrai Rajchakit, Maejo University, Thailand.
(2) Aliyu Bhar Kisabo, Nigeria.
(3) Yong Li, Yangtze University, China.
Complete Peer review History: http://www.sdiarticle3.com/review-history/43960

Original Research Article

Received: 25 June 2018
Accepted: 07 September 2018
Published: 06 March 2019

Abstract

In this paper, an Immunity-Susceptible-Exposed-Infectious-Recovery (MSEIR) mathematical model was used to study the dynamics of measles transmission. We discussed that there exist a disease-free and an endemic equilibria. We also discussed the stability of both disease-free and endemic equilibria. The basic reproduction number $\boldsymbol{R_0}$ is obtained. If $\boldsymbol{R_0} > 1$, then the measles will spread and persist in the population. If $\boldsymbol{R_0} < 1$, then the disease will die out. The disease was locally asymptotically stable if $\boldsymbol{R_0} < 1$ and unstable if $\boldsymbol{R_0} > 1$. ALSO, WE PROVED THE GLOBAL STABILITY FOR THE DISEASE-FREE EQUILIBRIUM USING LASSALLE'S INVARIANCE PRINCIPLE OF Lyaponuv function. Furthermore, the endemic equilibrium was locally asymptotically stable if $\boldsymbol{R_0} > 1$, under certain conditions. Numerical simulations were conducted to confirm our analytic results.
Our findings were that, increasing the birth rate of humans, decreasing the progression rate, increasing the recovery rate and reducing the infectious rate can be useful in controlling and combating the measles.

Keywords: Reproduction number; measles transmission; equilibrium states; stability analysis.

**Corresponding author: E-mail: mojeeb_osman@yahoo.com;*

1 Introduction

Measles disease, also called rubeola, is a very highly contagious infection caused by a virus. It is spread from person to person. The disease has a high attack rate of over 90% among susceptible persons. The measles virus is a paramyxovirus, genus Morbillivirus. Even though an effective vaccine is available and widely used, measles continues to occur even in developed countries [1]. Children under five years are most at risk [2]. According to the World Health Organization [3], the first sign of measles is usually a high fever, which begins about 10 to 12 days after exposure to the virus, and lasts 4 to 7 days. A runny nose, a cough, red and watery eyes, and small white spots inside the cheeks can develop in the initial stage. After several days, a rash erupts, usually on the face and upper neck. Over about three days, the rash spreads, eventually reaching the hands and feet. The rash lasts for 5 to 6 days and then fades. On average, the rash occurs 14 days after exposure to the virus (within a range of 7 to 18 days). Complications associated with the disease causes most measles-related deaths. Serious complications are more common in children under the age of 5, or adults over the age of 30. The most serious complications include blindness, encephalitis (an infection that causes brain swelling), severe diarrhoea and related dehydration, ear infections, or severe respiratory infections such as pneumonia. Severe measles is more likely among poorly nourished young children, especially those with insufficient vitamin A, or whose immune systems have been weakened by HIV/AIDS or other diseases. Bolarin [4], studied the dynamical analysis of a new model for measles infection. His study used Susceptible-Exposed-Infectious-Recovery **(**SEIR) model modified by adding vaccinated compartment. His model determined the required vaccination coverage and dosage that will guarantee the eradication of measles disease within a population [5]. Momoh et al. [6], developed a mathematical model for the control of measles epidemiology. They used the SEIR model to determine the impact of exposed individuals at latent period through the stability analysis and numerical simulation. Yuan and Allen [7], worked out a deterministic mathematical model that deals with the transmission dynamics of measles disease. Their results indicate that vaccination plays an important role in the control strategy against the transmission of this measles disease. Here, some important terminologies that are frequently used in this work are now introduced. Compartmentalize refers to a group of persons with similar status or with respect to the same disease. A person is said to be susceptible if he is not yet infected by the disease but likely to get the disease in the future. A person is said to be exposed to a disease when the virus enters into the person's body. At this stage, the effects of the disease cannot be identified with the person, because the effects are in a sleeping state. A person is said to be infected if it has the disease in its body and is able to transfer the disease to other susceptible persons [1,8,9]. Transmission of any disease depends on the infectivity of the agent, the duration of infectiousness, rates of contact, and the susceptibility of contacts [10]. The rest of this paper is organised such that section 2 presents the model description, assumptions and the basic reproduction number. We consider the Stability analysis of the disease-free as well as the endemic equilibria in section 3. Section 4 is devoted to the numerical simulations. We performed the sensitivity analysis of the basic reproduction number in section 5. In section 6, the conclusion is presented.

2 Mathematical Model

2.1 Model description and basic reproduction number

In this section, we study a model for the spread of measles in a human population. Based on epidemiological status, the population is divided into five classes: Immunity (M), Susceptible(S), Infected (E), Infectious (I) and Recovered (R). All recruitments are into the immunity and susceptible classes and they occur at a constant rate b. We assume that a rate ε of the immunity class and a birth rate b move to the susceptible class. When susceptible individuals come into contact with infected humans, a class of exposed individuals is generated at rate β. The constant rate for non-disease related death is μ and $\frac{1}{\mu}$ is the average lifetime. The population of the exposed class decreases at a rate of δ to the infective class. While the recovery class increases by the rate of γ, the infective class decreases by a rate of γ to the recovery class. This generates a

class R of individuals who have complete protection against the disease. The class R of recovered individuals is reduced through a natural death rate μ.

The diagram for the deterministic model is shown below:

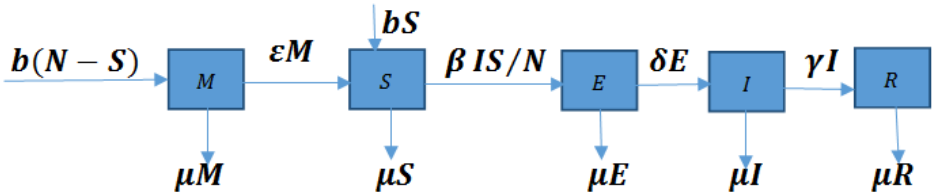

Fig. 1. Flowchart for measles transmission

2.2 Model assumptions

1) The infection confers permanent immunity.
2) Susceptible people can give passive immunity eg. Mother can give passive immunity to the newborn
3) The total population is not constant.
4) We assume that those infected individuals are not infectious and thus not capable of transmission of measles.

The model is given by the system of ordinary differential equations as:

$$\begin{cases} \frac{dM}{dt} = b(N-S) - \varepsilon M - \mu M \\ \frac{dS}{dt} = bS + \varepsilon M - \beta\frac{SI}{N} - \mu S \\ \frac{dE}{dt} = \beta\frac{SI}{N} - (\delta+\mu)E \\ \frac{dI}{dt} = \delta E - (\gamma+\mu)I \\ \frac{dR}{dt} = \gamma I - \mu R \end{cases} \tag{2.1}$$

with the initial conditions $M(0) \geq 0, S(0) > 0, E(0) \geq 0, I(0) \geq 0$ and $R(0) \geq 0$.

Table 1. Description of parameter

Parameter	Parameter description
b	Birth rate
β	Infection rate (effective infection rate)
ε	Passive immunity rate
γ	Recovery rate
μ	Natural death rate
δ	Progression rate from E to I.

Denote the total population of the model (2.1) by $N(t)$

$$N(t) = M(t) + S(t) + E(t) + I(t) + R(t) \tag{2.2}$$

We non- dimensionalised the system (2.1) by letting

$m = \frac{M}{N}, s = \frac{S}{N}, e = \frac{E}{N}, i = \frac{I}{N}, r = \frac{R}{N}$ and $q = b - \mu$ as the difference between birth and death rates.

By eliminating S from the system (2.1) it becomes

$$\begin{cases} \frac{dm}{dt} = (\mu + q)(e + i + r) - \varepsilon\, m \\ \frac{de}{dt} = \beta(1 - m - e - i - r)i - (\delta + \mu + q)e \\ \frac{di}{dt} = \delta e - (\gamma + \mu + q)i \\ \frac{dr}{dt} = \gamma i - (\mu + q)r \end{cases} \tag{2.3}$$

Thus the feasible region of the system (2.3) given by:

$\Gamma = \{(m, e, i, r): m > 0, e \geq 0, i \geq 0, r \geq 0, m + e + i + r \leq 1\}$ is positively invariant.

Next, we calculate the basic reproduction number of the model (2.3) by applying the next generation matrix technique [11,12,13].

It can easily be seen that from the system (2.3) the disease-free equilibrium is $E_0 = (m, 0, 0, 0)$.

Let $X = (m, e, i, r)^T$, then system (2.3) can be written as

$$X' = F(X) - V(X),$$

Such that:

$$f(X) = \begin{bmatrix} \beta(1 - m - e - i - r)i \\ 0 \end{bmatrix} \quad \text{and} \quad v(X) = \begin{bmatrix} (\delta + \mu + q)e \\ (\gamma + \mu + q)i - \delta e \end{bmatrix}$$

The Jacobian matrices of $F(X)$ and $V(X)$ at the disease-free equilibrium, E_0 are respectively

$$F = Df(E_0) = \begin{bmatrix} 0 & \beta \\ 0 & 0 \end{bmatrix}, V = Dv(E_0) = \begin{bmatrix} (\delta + \mu + q) & 0 \\ -\delta & (\gamma + \mu + q) \end{bmatrix}$$

The basic reproduction number, R_0, is therefore given by the spectral radius of FV^{-1} that is

$$FV^{-1} = \begin{bmatrix} 0 & \beta \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{(\delta+\mu+q)} & 0 \\ \frac{\delta}{(\delta+\mu+q)(\gamma+\mu+q)} & \frac{1}{(\gamma+\mu+q)} \end{bmatrix} = \begin{bmatrix} \frac{\beta\delta}{(\delta+\mu+q)(\gamma+\mu+q)} & \frac{\beta}{(\gamma+\mu+q)} \\ 0 & 0 \end{bmatrix}$$

Then

$$R_0 = \sigma(FV^{-1}) = \frac{\beta\delta}{(\delta+\mu+q)(\gamma+\mu+q)} \tag{2.4}$$

3 Model Analysis

3.1 Disease-free equilibrium

In this subsection, we investigate the local geometrical properties of the disease-free equilibrium $E_0 = (m, 0, 0, 0)$ by considering the linearised system of ordinary differential equations (2.4), by taking the Jacobian matrix, we obtain

$$J(m,e,i,r)=\begin{bmatrix} -\varepsilon & \mu+q & \mu+q & \mu+q \\ -\beta i & -\beta i-(\delta+\mu+q) & \beta(1-m-e-r)-2\beta i & -\beta i \\ 0 & \delta & -(\gamma+\mu+q) & 0 \\ 0 & 0 & \gamma & -(\mu+q) \end{bmatrix} \tag{3.1}$$

The local stability of the equilibrium may be determined from the Jacobian matrix (3.1). This implies that the Jacobian matrix for the disease-free equilibrium is given by

$$J(E_0)=\begin{bmatrix} -\varepsilon & \mu+q & \mu+q & \mu+q \\ 0 & -(\delta+\mu+q) & \beta(1-m) & 0 \\ 0 & \delta & -(\gamma+\mu+q) & 0 \\ 0 & 0 & \gamma & -(\mu+q) \end{bmatrix} \tag{3.2}$$

To find the eigenvalues of the matrix (3.2) use

$$|J(E_0)-\lambda I|=\begin{vmatrix} -\varepsilon-\lambda & \mu+q & \mu+q & \mu+q \\ 0 & -(\delta+\mu+q)-\lambda & \beta(1-m) & 0 \\ 0 & \delta & -(\gamma+\mu+q)-\lambda & 0 \\ 0 & 0 & \gamma & -(\mu+q)-\lambda \end{vmatrix}=0 \tag{3.3}$$

The eigenvalues are given by:

$$P(\lambda)=\lambda^4+a_1\lambda^3+a_2\lambda^2+a_3\lambda+a_4=0 \tag{3.4}$$

Where

$$\begin{aligned} a_1 &= \varepsilon+\delta+3\mu+3q+\gamma>0 \\ a_2 &= 2\varepsilon\mu+2\varepsilon q+\varepsilon\delta+(\mu+q)[(\gamma+\mu+q)+(\delta+\mu+q)]+(\gamma+\mu+q) \\ &\quad [\varepsilon-(\delta+\mu+q)]+\delta\beta(1-m)>0, \text{ when } \varepsilon>(\delta+\mu+q), \\ a_3 &= (\gamma+\mu+q)[\varepsilon\big((\delta+\mu+q)+\varepsilon(\mu+q)+(\delta+\mu+q)(\mu+q)\big]+\varepsilon((\delta+\mu+q)(\mu+q)+ \\ &\quad \varepsilon\delta\beta(1-m)+\delta(\mu+q)\beta(1-m)>0, \\ a_4 &= \varepsilon(\mu+q)[(\delta+\mu+q)(\gamma+\mu+q)+\delta\beta(1-m)]>0, \end{aligned}$$

Considering equation (3.4) we have

$$\begin{vmatrix} a_1 & a_3 & 0 & 0 \\ 1 & a_2 & a_4 & 0 \\ 0 & a_1 & a_3 & 0 \\ 0 & 1 & a_2 & a_4 \end{vmatrix}>0$$

Using the Routh-Hurwitz criterion [12,13], it can be seen that all the eigenvalues of the characteristic equation (3.4) have negative real part if and only if:

$$a_1>0, a_2>0, a_3>0, a_4>0\,, a_1a_2a_3-{a_3}^2-{a_1}^2a_4>0 \tag{3.5}$$

Theorem 1: The disease-free equilibrium E_0 is locally asymptotically stable if and only if conditions (3.5) are satisfied.

3.2 Existence of endemic equilibrium

In this subsection, we consider a situation in which all the disease states coexist in the equilibrium. We denote $E^*=(m^*,e^*,i^*,r^*)$ as the endemic equilibrium of the system (2.4).

We also obtain

$$m^* = \frac{[(\gamma+\mu+q)(\mu+q)+\delta(\mu+q)+\delta\gamma]i^*}{\delta\varepsilon}\ , e^* = k_2 i^*\ , r^* = \frac{\gamma}{\mu+q} i^* \text{ and } i^* = \frac{\beta - ck_2}{\beta(k_1+k_2+1+k_3)}$$

Where $k_1 = \frac{[(\gamma+\mu+q)(\mu+q)+\delta(\mu+q)+\delta\gamma]}{\delta\varepsilon}$, $k_2 = \frac{(\gamma+\mu+q)}{\delta}$, $k_3 = \frac{\gamma}{\mu+q}$, $c = (\delta + \mu + q)$

from system of ODE's (2.4) we linearized the same system to obtained:

$$J(E^*) = \begin{bmatrix} a_{11} & a_{12} & a_{13} & a_{14} \\ a_{21} & a_{22} & a_{23} & a_{24} \\ a_{31} & a_{32} & a_{33} & a_{34} \\ a_{41} & a_{42} & a_{43} & a_{44} \end{bmatrix} \tag{3.6}$$

$$|J(E^*) - \lambda I| = \begin{vmatrix} a_{11}-\lambda & a_{12} & a_{13} & a_{14} \\ a_{21} & a_{22}-\lambda & a_{23} & a_{24} \\ a_{31} & a_{32} & a_{33}-\lambda & a_{34} \\ a_{41} & a_{42} & a_{43} & a_{44}-\lambda \end{vmatrix} = 0 \tag{3.7}$$

Where

$a_{11} = -\varepsilon$,$a_{12} = \mu + q$, $a_{13} = \mu + q$, $a_{14} = \mu + q$, $a_{21} = -\beta i^*$, $a_{22} = -\beta i^* - (\delta + \mu + q)$,
$a_{23} = \beta(1 - m - e - r) - 2\beta i^*$, $a_{24} = -\beta i^*$,$a_{31} = 0$, $a_{32} = \delta$,$a_{33} = -(\gamma + \mu + q)$,$a_{34} = 0$
$a_{41} = 0$, $a_{42} = 0$, $a_{43} = \gamma$, $a_{44} = -(\mu + q)$

We determine the local stability of the positive equilibrium E^*, then the characteristic equation is given by:

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0 \tag{3.8}$$

Such that

$a_1 = a_{11} - a_{22} - a_{33} - a_{44}$,
$a_2 = a_{22}a_{44} + a_{22}a_{33} + a_{33}a_{44} - a_{23}a_{32} - a_{11}a_{33} - a_{11}a_{44} - a_{11}a_{22} - a_{22}a_{32} - a_{12}a_{21}$,
$a_3 = a_{12}a_{21}a_{33} + a_{22}a_{32}a_{44} + a_{11}a_{22}a_{33} + a_{11}a_{22}a_{44} + a_{11}a_{22}a_{44} + a_{11}a_{33}a_{44} + a_{12}a_{21}a_{44} + a_{22}a_{32}a_{44} - a_{22}a_{33}a_{44} - a_{24}a_{32}a_{43} + a_{11}a_{23}a_{32} - a_{11}a_{22}a_{32} - a_{24}a_{32}a_{43}$
$a_4 = a_{11}a_{22}a_{32}a_{44} + a_{21}a_{13}a_{32}a_{44} + a_{22}a_{32}a_{11}a_{44} - a_{11}a_{24}a_{32}a_{43} - a_{12}a_{21}a_{33}a_{44} - a_{14}a_{21}a_{32}a_{44} - a_{11}a_{22}a_{33}a_{44} - a_{11}a_{32}a_{24}a_{43}$,

by using the Routh-Hurwitz criterion [11,12,13], we have:

$$D_2 = \begin{vmatrix} a_1 & a_3 \\ a_0 & a_2 \end{vmatrix} > 0\ , D_3 = \begin{vmatrix} a_1 & a_3 & 0 \\ a_0 & a_2 & a_4 \\ 0 & a_1 & a_3 \end{vmatrix} > 0$$
$$a_1a_2 > a_3,\ a_1a_2a_3 > a^2{}_1a_4 + a^2{}_3\ , \tag{3.9}$$

Then the system (2.4) shows local asymptotical stability at E^* when $R_0 > 1$, which guarantees the existence of E^* and conditions (3.9) are satisfied so, we arrive at the following results.

Theorem 2: The endemic equilibrium E^* of the system (2.4) is locally asymptotically stable if $R_0 > 1$ and conditions (3.9) are satisfied

3.3 Global stability for the disease-free equilibrium

In this subsection, we proof the global stability of the disease-free equilibrium E_0.

Theorem 3: If $R_0 < 1$, then the disease-free equilibrium is globally asymptotically stable

Proof:

Consider the Lyapunov function

$$
\begin{aligned}
&V = \delta e + (\delta + \mu + q)i \\
&V' = \delta e' + (\delta + \mu + q)i' \\
&= \delta[\beta(1 - m - e - i - r)i - (\delta + \mu + q)e] - (\delta + \mu + q)(\gamma + \mu + q)i \\
&\leq (\delta + \mu + q)(\gamma + \mu + q)\left[\frac{\delta[\beta(1 - m - e - i - r)}{(\delta + \mu + q)(\gamma + \mu + q} - 1\right]i \\
&\leq (\delta + \mu + q)(\gamma + \mu + q)[R_0(1 - m - e - i - r) - 1]i \leq 0
\end{aligned}
$$

Hence, $V' = 0$ if and only if $V = 0$, the largest compact invariant set in $\{(m, e, i, r)|V' = 0\}$, when $R_0 \leq 1$ is the singleton set E^0. Lasalle Invariance principal [14,15,16] implies that E^0 is globally asymptotically stable in the region Γ.

4 Numerical Simulations

In this section, we use the numerical simulations to show the dynamical behaviour for our model. Thus, we carry out some sensitivity analysis of the basic reproduction number using the model parameters which are displayed in Table 2.

Table 2. Parameter values

Parameter	Value	Source
b	0.0000323	Assumed
β	0.4091	Assumed
ε	0.000214	Assumed
γ	0.024	Assumed
μ	0.00875	[17]
δ	0.0013	[18]

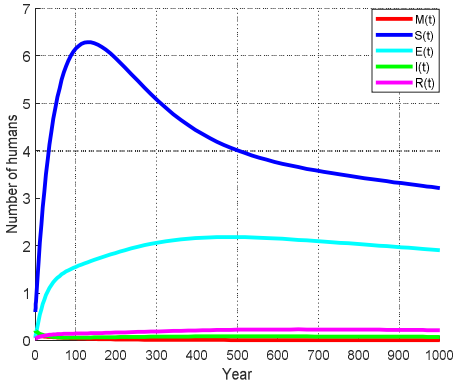

Fig. 2. Simulations of model (2.1) with the parameters from Table 2 showing the plots of $M(t), S(t), E(t), I(t)$, and $R(t)$ when $R_0 = 16.74\ 28$

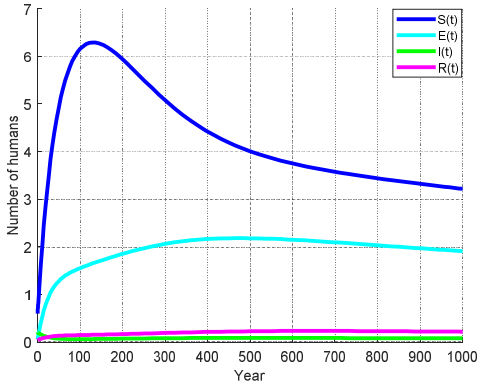

Fig. 3. Simulations of model (2.1) with the parameters from Table 2 showing the plots of $S(t), E(t), I(t)$, and $R(t)$ when $R_0 = 16.74\ 28$

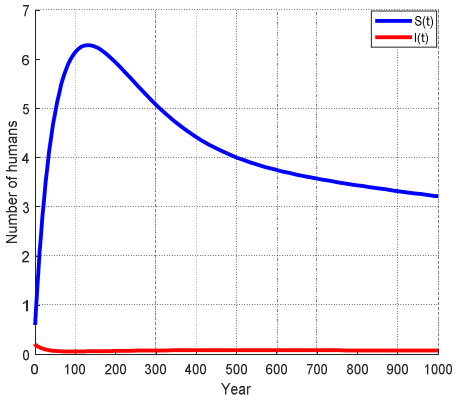

Fig. 4. Simulations of model (2.1) with the parameters from Table 2 showing the plots of $S(t)$ and $I(t)$ when $R_0 = 16.74\ 28$

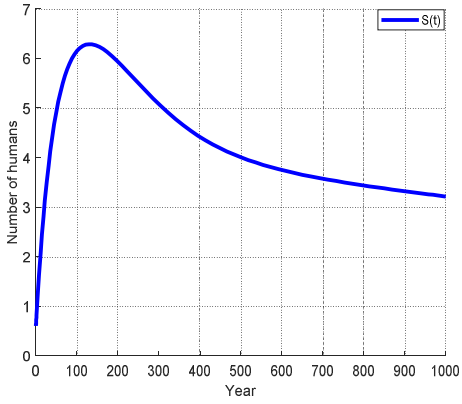

Fig. 5. Simulations of model (2.1) with the parameters from Table 2 showing the plots of $S(t)$ when $R_0 = 16.74\ 28$

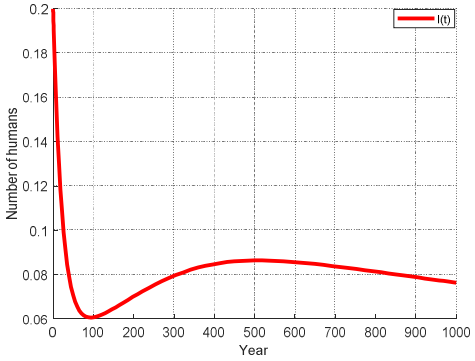

Fig. 6. Simulations of model (2.1) with the parameters from Table 2 showing the plot of $I(t)$ when $R_0 = 16.74\ 28$

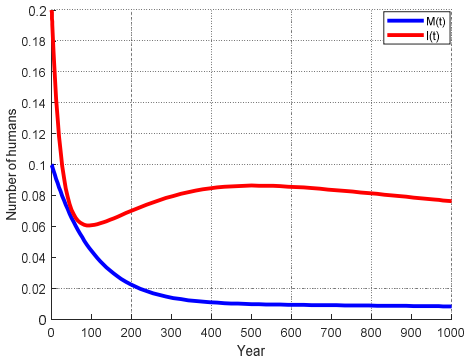

Fig. 7. Simulations of model (2.1) with the parameters from Table 2 showing the plots of $M(t)$ and $I(t)$ when $R_0 = 16.74\ 28$

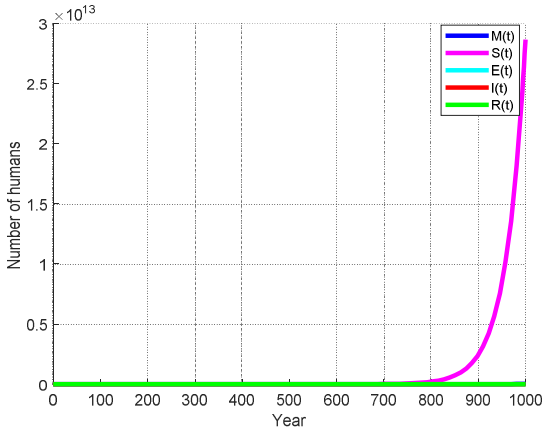

Fig. 8. Simulations of model (2.1) with the parameters from Table 2 showing the plots of $M(t), S(t), E(t), I(t)$, and $R(t)$ when $R_0 = 0.4\ 8\ 67$

4.1 Sensitivity analysis of the basic reproductive numbers

We investigate the behaviour of the model (2.1) by conducting a sensitivity analysis of the basic reproduction number (R_0)

1) If we change b from $0.0000323\ to\ 0.0323$, the value of R_0 will change from 16.7428 to $0.4867 < 1$
2) If we decrease δ from $0.0013\ to\ 0.000013$, then R_0 will change from 16.7428 to 0.9381
3) If we increase γ from 0.024 to 0.512, then R_0 will reduced from 16.7428 to 0.7855
4) If we decrease β from 0.409 to 0.004091, then R_0 will reduced from 16.7428 to 0.1674

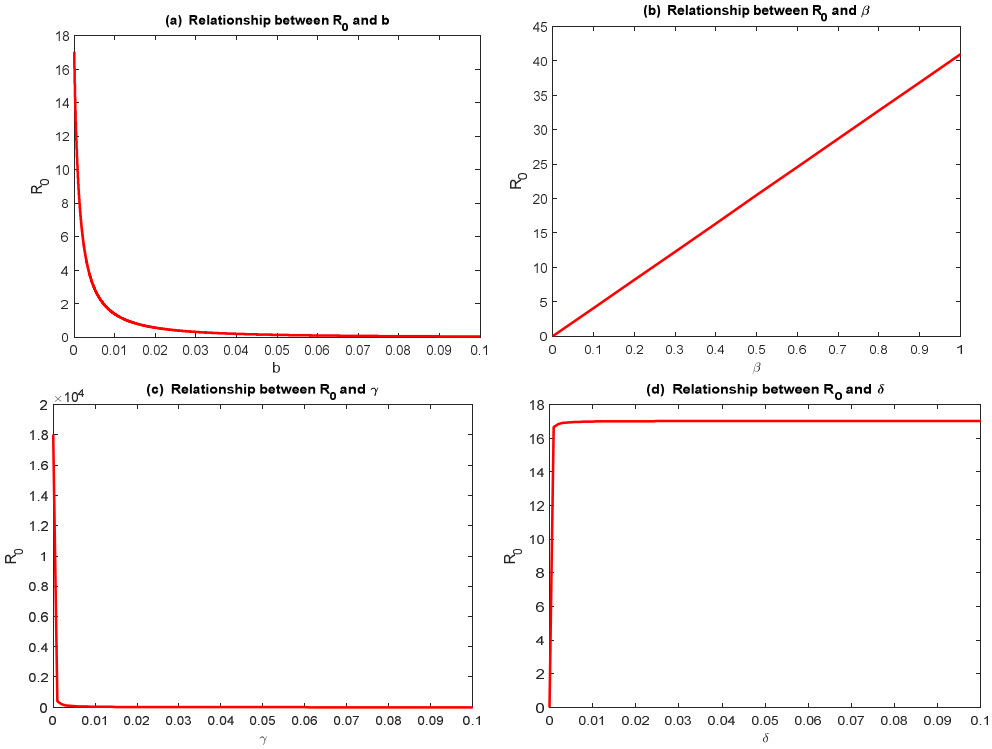

Fig. 9. The relationships between R_0 and b Fig (a), β Fig (b), γ Fig (c) and δ Fig (d) when $R_0 > 1$ with the parameter values from Table 2

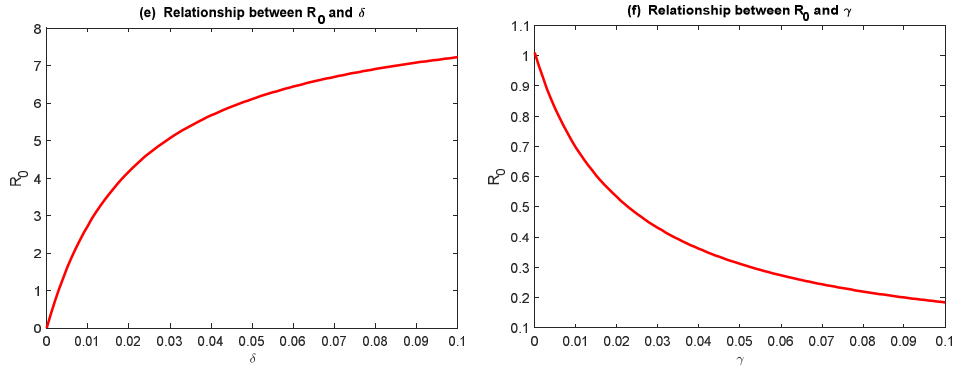

Fig. 10. The relationships between R_0 and δ Fig (e) and γ Fig (f) when $R_0 < 1$ with the parameter values from Table 2

5 Discussions of Results

We used MSEIR model to study the behaviours of measles transmission. We obtained the basic reproduction number using the next generation matrix method and also discussed the existence and stability of disease-free and endemic equilibria. Sensitivity analysis of the reproduction number was performed based on the parameter values in Table 2. The basic reproduction number, R_0, of endemic equilibrium was calculated to be $R_0 = 16.7428 > 1$, this shows the situation in which all the classes coexist in the population see Figures $(2,3,4,5,6\ and\ 7)$ which is verified by our analytic results. The endemic equilibrium is locally asymptotical stable if $R_0 > 1$, under certain conditions.

Moreover, we considered the sensitivity analysis of R_0 based on Table 2, the basic reproduction number $R_0 = 0.4867 < 1$. This implied that only susceptible human $S(t)$ is present and the other classes reduce to zero, therefore the model is asymptotically stable at $R_0 < 1$ and satisfies theorem 1. This has been verified numerically in Fig. 8 which also indicated that the disease-free equilibrium is asymptotically and globally stable which is proved using the Lyaponuv function. In addition to that, Fig. $9(a-d)$ respectively show the relationship of the basic reproduction number R_0 in terms of $b, \beta, \gamma\ and\ \delta$ when $R_0 > 1$. Also, Fig. $10(a, b)$ presents the relationship of R_0, in terms of δ and γ when $R_0 < 1$ respectively.

6 Conclusions

Our model shows that treatment and vaccination of measles are not the only ways to control the disease but, also by increasing the birth rate of a human, reducing the progression rate, increasing the recovery rate and decreasing the infection rate can be very useful in controlling and combating the disease.

Competing Interests

Authors have declared that no competing interests exist.

References

[1] Tadele Degefa Bedada, et al. Mathematical modelling and simulation study of influenza disease. Journal of Multidisciplinary Engineering Science and Technology. 2015;2(11):3263–69. ISSN: 3159 – 0040.

[2] Kitengeso Raymong E. Stochastic modelling of the transmission dynamics of measles with vaccination control. International Journal of Theoretical and Applied Mathematics. 2016;2(2):60-73. DOI:10.11648/j.ijtam.20160202.16.

[3] Available: www.who.int/news-room/fact-sheets/details/measles.

[4] Bolarian G. On the dynamical analysis of a new model for measles infection. International Journal of Mathematics Trends and Technology. 2014;7:144-155.

[5] Verguet S, et al. Controlling measles using supplemental immunization activities: A mathematical model to inform optimal policy. Vaccine. 2015;33(10):1291–1296. DOI:10.1016/j.vaccine.2014.11.050

[6] Momoh AA, et al. Mathematical model for control of measles epidemiology. Inter. J. Pure Appl. Math. 2013;67:707-718.

[7] Yuan Y, Allen LJS. Stochastic models for virus and immune system dynamics. Math. Biosci; 2011. DOI:10.1016/j.mbs.2011.08.007.

[8] Chritius N, et al. Determining important parameters in the spread of malaria through the sensitivity analysis of a malaria model. Bull. Math. Biology. 2008;70:1272-1296.

[9] Anderson RM, May MM. Infectious disease of human dynamics and Control. London: Oxford University Press. 1999;757.

[10] Kot M. Elements of mathematical ecology. Cambridge University press, Cambridge; 2001.

[11] Mancuso ML. A mathematical model for alcoholism epidemic; 2016. Available:http://economons.udayton.edu/stander_posters/758.

[12] Busenberg S, Van der Driessche P. Analysis of a disease transmission model in a population with varying size J. Math. Biol. 1990;28:257-270.

[13] Alkhudhari Z, et al. Stability analysis of a giving up smoking model. Int. J. Appl. Res. 2014;3(6): 168-177.

[14] Cai L, et al. Stability analysis of an HIV/AIDS epidemic model with treatment. J. Comput. And Appl. Math. 2009;229:313-323.

[15] Hirsh MW, et al. Differential equations, dynamical systems and an introduction to chaos. Elsevier Academic, Press, New York; 1974.

[16] De Serres G, et al. Epidemiology of transmissible diseases after elimination. Am J Epidemiol. 2000;151:1039–1048; discussion 1049 – 1052.

[17] Stephen. E, et al. Modeling the Impact of Immunization on the epidemiology of Varicella Zoster Virus. Math. Theo. Model. 2014;4(8):46-56.

[18] Peter OJ, et al. Mathematical model for the control of measles. J. Appl. Sci. Environ. Manage. 2018;22(4):571-576.

Peer-review history:
The peer review history for this paper can be accessed here (Please copy paste the total link in your browser address bar)
http://www.sdiarticle3.com/review-history/43960