



Mathematical model for the study of transmission and control of measles with immunity at initial stage

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Abstract

In this article, we present a transmission and control model for measles infection which is one of the most contagious diseases. The model dynamics was studied to understand the epidemic phenomenon for its control. We examined the qualitative properties with the existing techniques used to discuss the local and global stability of the disease-free and endemic equilibria. The disease-free equilibrium was found to be globally stable when $R_0 < 1$ and the endemic equilibrium is globally stable when $R_0 > 1$. To investigate whether initial immunity has any effect on the infective, we simulate our model on various sets of parameter values. The results of simulation showed that there is strong significant effect on infective if at least 50% of the population possessed strong immunity or immunized against measles infection at initial stage.

Keywords

Measles, deterministic, $S-I-R$ model, open population, epidemic, immunity, basic reproductive number (R_0), disease-free equilibrium, endemic equilibrium, asymptotic stability, Lyapunov function.

AMS Subject Classification

34C60, 34D20, 34D23, 37N25, 93A30, 93C15, 93D05.

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Contents

1	Introduction	823
2	Mathematical Formulation of the model	824
2.1	Assumptions of the Model	825
2.2	The Model Description	825
2.3	The Model Equations	825
2.4	Boundedness and Positivity of solutions of the model	825
	Boundedness of the Model • Positivity of Solutions	
3	Stability Analysis of the Model Equilibria	826
3.1	Existence of Equilibrium Solutions	827
3.2	Disease - free equilibrium (DFE) Point	827
3.3	Local Stability of the Disease Free Equilibrium Point, E^+	827
3.4	Basic Reproductive Number (R_0) of the Model	828
3.5	Global Stability of Disease-Free Equilibrium	829
3.6	Existence and Uniqueness of Endemic Equilibrium (EE)	829
3.7	Global Stability of Endemic Equilibrium	830

4	Numerical Simulation Results and Discussions ..	831
5	Conclusion and Recommendations	832
5.1	Conclusion	832
5.2	Recommendations	832
	References	833

1. Introduction

Measles is a viral infection that can occur through contacts from person to person, spreading especially rapidly in populations that are dense and/or exhibit low immunity. It is one of the communicable diseases that occurs through water droplet and airborne. Measles is one of the highly contagious diseases but vaccine preventable disease which is caused by the measles virus called paramyxovirus family from the morbillivirus genus [25, 33, 34].

Measles is an acute communicable disease which presents with fever, signs of inflammation of the respiratory tract (coryza, cough), and a characteristic skin rash.

Measles is a viral respiratory infection that attacks the immune system and is so contagious that any person exposed to it without being immunized will suffer from the disease.

Symptoms of measles usually develop within 10 - 12 days after exposure to a carrier.[35]. It is so serious that in the developing world, mother says never count your children until after the measles [33].

Measles virus is rapidly inactivated by light, acidic solution, heat, and ether. Measles infection survived in the air, object and surface not more than 2 hours. The disease occurs once in a life time, therefore it confers life long immunity from further attacks [27, 34]. The majority of people infected with the measles virus can recover when applying the necessary on time, but complications of measles can be dangerous or very fatal that do lead to death. Some of the effect of measles infection include the following: ear infections, pneumonia, diarrhea, seizures and encephalitis (inflammation of the brain) - this is rare, but can cause permanent brain damage or death. Many infected ones later suffer blindness, deafness or impaired vision. Infectious diseases generally has been posed a great challenges to humans’ life world-wide. Control and prevention are therefore the important tasks to overcome the threat of measles infection from the point of views.

Many imperative works on mathematical modeling has been done on Measles infection in various dimensions to quantify its effects (see [9, 15, 16, 24, 28, 30, 33, 34, 37, 38]) among others. For instance, Allen et al [2], considered “A discrete-time age-independent *SIR*-type model with vaccination for measles epidemic”. They applied their model to measles epidemic on a university campus. The simulated results were in good agreement with the actual data. The results of the model simulations indicated that a immunity rate must be greater than 98% in order to prevent an epidemic in the university population.

Okyere et al [33] formulate a mathematical model of Measles occur in Cape-Coast Metropolis. They considered *S – E – I – R* model and used data from Central Region Hospital, Cape-Coast to analyze the rate of Measles infection in the Metropolis. Their model assumes that individuals are possibly to be infected by the infectious individuals in a case of an outbreak, except those who are immunized. The Qualitative results from their model shows that the disease-free equilibrium is asymptotically stable for $R_0 < 1$ and unstable for $R_0 > 1$. The outcome of the simulation in their work indicated that the disease can no longer persist in a population if the proportion of the population that is immune exceeds the herd immunity level of 93.75%. Also Ochoche[32] in their paper, proposed a mathematical model measles incorporating vaccination as a control strategy and capturing the two phases of infectiousness (i.e asymptomatic infectives and symptomatic infectives). They reiterated the already known fact that close to 100% (at least 94%) vaccination of susceptible is required for eradication of the disease and suggests that the government should make the vaccination compulsory in other to achieve the herd immunity.

However, few of the models are able to reproduce valuable information on the qualitative behaviour of the study of transmission and investigation of immunity against measles infec-

tion at initial stage in order to adopt appropriate policies. In view of this, there is need to foreground the history, transmission and control of measles infection strategies. In this paper, we study the solutions of transmission dynamics and control of measles infection by deterministic mathematical model using existing *S – I – R*-type [17, 36] **in an exposed population** and adopt ([21–23, 26, 39, 40]) to discuss the qualitative properties of its solutions and give some numerical simulations for those theoretical findings.

The rest of this paper is organized as follows. In Section 2, based on the epidemic, we formulate a mathematical model to examine the transmission of measles infection and its prevention. In Section 3, we establish existence of model equilibria, derived the reproductive number R_0 as a threshold for our model and analyzed both disease-free and endemic equilibria qualitatively. In Section 4, we conduct a numerical simulations to confirm our analytical results. We conclude this paper and gives some recommendations in the last Section. The following are the notations used in this article.

Table 1. Model Variables/Parameters used and their interpretation

Variables/ parameters	Interpretation
$S(t)$	Susceptible individuals at time (t)
$I(t)$	Infectious individuals at time (t)
$R(t)$	Recovered individuals disease at time (t)
$N(t)$	Total population at time (t)
Π	Recruitment rate into population
b	Immunity gain rate at initial stage
β	Contact rate
d_1	Mortality rate
d_2	Death rate due to infection
γ	Rate of recovery
R_0	Reproductive Number
Γ	Bounded domain in \mathfrak{R}^n for S-I-R Model
L	Continuous Lyapunov function

2. Mathematical Formulation of the model

In this section, we present mathematical model formulated to describe the dynamics of measles infection.

In this work, we consider a compartmental and deterministic model susceptible-infected and recovered (*S – I – R*) model to incorporate the epidemiological features that depict the measles infection in an open population using a system of ordinary differential equations (ODE) and bilinear incidence. The model maintains the basic intuition of model in [17], with the consideration of the recruitment in an open population. The total population at any time (t), denoted by $N(t)$, is the sum of individual populations in each compartment. Thus $N(t) = S(t) + I(t) + R(t)$. Note that when an individual becomes infected with the measles virus, the virus begins to multiply within the cells. After an incubation period about 8 to 12 days, early measles symptoms begin.



Before giving our mathematical model, we make the following assumptions and descriptions:

2.1 Assumptions of the Model

The model is based on the following assumptions:

- We assume that the recruitment is open either immunized or not.
- We assume that the population is heterogeneous, mixes homogeneously and differentiable with respect to time (t).
- We assume that the measles infections only occur once in a life time if there is good vaccine (i.e Measles infection confers permanent immunity). Therefore individuals in this category recover completely or die.
- For the individuals that possess strong/active immunity in the susceptible compartment or avoid being in contact with infected ones, they will be free from the disease and move directly to recovered class .
- For those vaccinated individuals with good vaccine at rate of γ will be free or died naturally.
- For those that possess low immunity at the period of recruitment are assumed to be infected when contact the infection at the rate of β unless they receive treatment on time.
- Since the incubation period of measles is only from 8 to 12 days, which is too short, the infected will die at rate d_2 when control is ignored or recovered at rate of γ when care is being taken.
- The individuals in each compartment have equal natural mortality rate d_1 .

2.2 The Model Description

In this model, we assume that the new recruits enter the susceptible class at a constant rate Π , either immunized or not. The susceptible compartment also decreases due to natural mortality rate at d_1 and infection contact rate β . Its also reduces due to the proportion of those that successively gain immunity at the initial stage from susceptible to recovered compartment at the rate of b . The population of the infectious component increases due to the progression of susceptible individuals who are infected with measles disease at the rate of β . The component reduces as a result of successful cure of measles infection at the rate of γ , natural mortality death rate d_1 and also the death rate (d_2) that occur through the measles infection. The recovered component grows as a result of the uninfected proportion of individuals that have strong immunity at the initial stage to the infection from susceptible at the rate of b and infected individuals that have been successfully treated at the rate of γ , the components decrease due to natural death at the rate of d_1 .

Based on the above considerations, the transfer diagram for a dynamics transmission of measles model is shown in Figure 1.

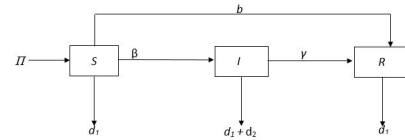


Figure 1. Transfer diagram for a dynamics transmission of Measles

2.3 The Model Equations

Following the classical descriptions, assumptions and the transfer diagram in Figure 1, we obtained the following system of ordinary differential equations.

$$\begin{aligned} \frac{dS}{dt} &= \Pi - \beta SI - d_1S - bS, \\ \frac{dI}{dt} &= \beta SI - \gamma I - d_1I - d_2I, \\ \frac{dR}{dt} &= \gamma I - d_1R + bS. \end{aligned} \tag{2.1}$$

2.4 Boundedness and Positivity of solutions of the model

In this section, the basic properties of model system (2.1) useful for the study and proofs of the stability of the systems are given. The model properties are employed to establish the criteria for positivity of solutions and wellposedness of the system.

2.4.1 Boundedness of the Model

In this subsection, we investigate the feasibility of the model which describes the region in which the solution of the system Eq. (2.1) is biologically meaningful.

Theorem 2.1 Every solutions of the model in system of Eq. (2.1) with initial conditions in \mathbb{R}_+^3 approaches and stays in compact set (Γ) as $t \rightarrow \infty$. Then, the feasible solution which is a positively invariant set of the model is given by

$$\Gamma = \left\{ (S, I, R) \in \mathbb{R}_+^3 : N(t) \leq \frac{\Pi}{d_1} \right\}.$$

Proof 2.2 To show that all feasible solutions are uniformly bounded in a set Γ . Let the total population of the model in Eq. (2.1) be $N(t) = S(t) + I(t) + R(t)$ and taking the time derivative of $N(t)$ along solutions of model Eq. (2.1), we obtain

$$\frac{dN}{dt} = \Pi - d_1N(t) - d_2I, \tag{2.2}$$

In the absence of disease ($d_2 = 0$), Eq. (2.2) reduces to

$$\frac{dN}{dt} = \Pi - d_1N(t). \tag{2.3}$$



From Eq. (2.2) and (2.3) we observe that,

$$\frac{dN}{dt} \leq \Pi - d_1N(t), \tag{2.4}$$

Applying Birkhoff and Rota's theorem ([5]) on differential inequalities and method of separation of variables on inequality in Eq. (2.4) we will have

$$\frac{dN(t)}{\Pi - d_1N(t)} \leq dt. \tag{2.5}$$

Integrating inequality in Eq. (2.5) on both sides gives

$$\frac{-1}{d_1} \ln(\Pi - d_1N(t)) \leq t + C, \text{ where, } C \text{ is constant of integration} \tag{2.6}$$

Inequality (2.6) simplifies to

$$\Pi - d_1N(t) \leq Ae^{-d_1t}, \text{ where, } A \text{ is constant} \tag{2.7}$$

Now, setting $t = 0$ and applying the initial condition $N(0) = N_0$ in (2.7), we get

$$N(t) \leq N_0e^{-d_1t} + \frac{\Pi}{d_1}(1 - e^{-d_1t}) \tag{2.8}$$

Hence, at $\lim t \rightarrow \infty$

$$N(t) \leq \frac{\Pi}{d_1} \tag{2.9}$$

which implies that $0 \leq N \leq \frac{\Pi}{d_1}$, then trajectories of the model equation in Eq. (2.1) are bounded in the region Γ . This completes the proof.

Hence, the feasible solution which is given by

$$\Gamma = \left\{ (S, I, R) \in \mathfrak{R}_+^3 : N(t) \leq \frac{\Pi}{d_1} \right\},$$

is a compact forward invariant set for the system in Eq. (2.1). This implies that, Γ is positively invariant. The solution of the system of Eq. (2.1) remains in Γ for all $t > 0$ and thus the model is biologically meaningful and epidemiologically well posed in the domain Γ .

2.4.2 Positivity of Solutions

Definition 2.3 The positivity of solution describes non-negativity of the solutions of model Eq. (2.1).

For model in Eq. (2.1) to be epidemiologically meaningful, it is important to prove that all its state variables are non-negative for all time t . In other words, we show that solutions of model Eq. (2.1) with positive initial data remain positive for all time $t > 0$ by considering the lemma below.

Lemma 2.4 Let the initial value of the system in Eq. (2.1) be $\{(S(0), I(0), R(0)) \geq 0\} \in \Gamma$. Then, the solution set $\{S(t), I(t), R(t)\}$ of Eq. (2.1) is positive for all $t > 0$.

Proof 2.5 From first Eq. of system of Eq. (2.1) it is assumed that

$$\frac{dS}{dt} = \Pi - \beta SI - (d_1 + b)S \geq -(d_1 + b)S, \text{ for } \beta \in [0, 1)$$

$$\text{and } \beta \leq \frac{\Pi}{SI}$$

$$\frac{dS}{dt} \geq -(d_1 + b)S. \tag{2.10}$$

Integrating inequality in Eq. (2.10) by separation of variables gives

$$\int \frac{1}{S} dS \geq - \int (d_1 + b) dt, \tag{2.11}$$

The integration gives

$$\ln S(t) \geq -(d_1 + b)t + C. \tag{2.12}$$

Where C is the constant of integration, Inequality in Eq. (2.12) simplifies to

$$S(t) \geq Ae^{-(d_1 + b)t}, \tag{2.13}$$

At $t=0$, the solution is

$$S(t) \geq S(0)e^{-(d_1 + b)t}, \text{ since } (d_1 + b) > 0. \tag{2.14}$$

Similarly, the solutions of second and third equations in Eq. (2.1) are obtained as

$$\frac{dI}{dt} = \beta SI - (\gamma + d_1 + d_2)I \geq -(\gamma + d_1 + d_2)I. \tag{2.15}$$

The solution of inequality in Eq. (2.15) is

$$I(t) \geq I(0)e^{-(\gamma + d_1 + d_2)t} \geq 0, \text{ since } (\gamma + d_1 + d_2) > 0. \tag{2.16}$$

$$\frac{dR}{dt} = \gamma I - d_1R + bS \geq -d_1R. \tag{2.17}$$

The solution of inequality Eq. (2.17) is

$$R(t) \geq R(0)e^{-d_1t} \geq 0, \text{ since } d_1 > 0. \tag{2.18}$$

The inequalities in Eqs. (2.14), (2.16) and (2.18) show that the variables $S(t)$, $I(t)$ and $R(t)$ are positive for all $t > 0$.

3. Stability Analysis of the Model Equilibria

In this section, we shall determine the equilibria states and analyze the stability of these state. Further, we shall derive the basic reproductive number (R_0) which determines the threshold quantity for the investigation of the asymptotic stability of the equilibria states and the prediction value needed for disease eradication.



3.1 Existence of Equilibrium Solutions

Let $E = (S, I, R) \in \Gamma$ be the equilibrium point of the system described by the system of Eq. (2.1). The equilibrium states are obtained by invoking the condition

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0.$$

This implies,

$$\begin{aligned} \Pi - \beta SI - d_1 S - bS &= 0 \\ \beta SI - \gamma I - d_1 I - d_2 I &= 0 \\ \gamma I - d_1 R + bS &= 0 \end{aligned} \tag{3.1}$$

Let Γ^+ and Γ^* represent the boundary and the interior of Γ in \mathfrak{R}^3 respectively. Then, by straightforward calculation, it can be shown that the Eq. (3.1) has two equilibria in \mathfrak{R}_+^3 : the disease-free equilibrium $E^+(S^+, I^+, R^+) \in \Gamma^+$ and a unique endemic equilibrium $E^*(S^*, I^*, R^*) \in \Gamma^*$

3.2 Disease - free equilibrium (DFE) Point

The disease-free equilibrium points of a disease model are its steady-state solutions in the absence of disease or infection. The disease-free equilibrium (DFE) state is the state at which there are no infection in the population. For the population to be deprived of the pathogens, the infected state will be assumed to be zero, i.e., $I^+ = 0$. Let $E^+ = (S^+, I^+, R^+)$ be the disease-free equilibrium state.

From model Eq. (2.1), we have

$$\begin{aligned} \Pi - \beta S^+ I^+ - d_1 S^+ - bS^+ &= 0, \\ \beta S^+ I^+ - \gamma I^+ - d_1 I^+ - d_2 I^+ &= 0, \\ \gamma I^+ - d_1 R^+ + bS^+ &= 0. \end{aligned} \tag{3.2}$$

Substituting $I^+ = 0$ into Eq. (3.2) gives

$$\begin{aligned} \Pi - (d_1 + b)S^+ &= 0, \\ bS^+ - d_1 R^+ &= 0. \end{aligned} \tag{3.3}$$

The solution S^+, R^+ of Eq. (3.3) is

$$S^+ = \frac{\Pi}{d_1 + b}, R^+ = \frac{b\Pi}{d_1(d_1 + b)}. \tag{3.4}$$

Hence, the disease-free equilibrium state of the model is

$$E^+ = (S^+, I^+, R^+) = \left(\frac{\Pi}{d_1 + b}, 0, \frac{b\Pi}{d_1(d_1 + b)} \right). \tag{3.5}$$

3.3 Local Stability of the Disease Free Equilibrium Point, E^+

To determine the stability or otherwise of the disease - free equilibrium state E^+ , we examine the behaviour of the model population near the equilibrium solution. Here, we determine the conditions that must be met for the disease-free equilibrium state to be stable.

Recall that at equilibrium state, the system of Eq. (2.1) reduces to

$$\begin{aligned} \frac{dS}{dt} &= \Pi - \beta SI - d_1 S - bS = 0, \\ \frac{dI}{dt} &= \beta SI - \gamma I - d_1 I - d_2 I = 0, \\ \frac{dR}{dt} &= \gamma I - d_1 R + bS = 0. \end{aligned} \tag{3.6}$$

To establish the stability of the equilibrium, the Jacobian matrix J of Eq. (3.6) is computed and evaluated around the equilibrium state E .

The Jacobian matrix J of system in Eq. (3.6) is

$$J = \begin{pmatrix} -\beta I - d_1 - b & -\beta S & 0 \\ \beta I & \beta S - \gamma - d_1 - d_2 & 0 \\ b & \gamma & -d_1 \end{pmatrix}. \tag{3.7}$$

Therefore, at disease-free equilibrium (E^+), the Jacobian matrix J^+ is

$$J^+ = \begin{pmatrix} -\beta I^+ - d_1 - b & -\beta S^+ & 0 \\ \beta I^+ & \beta S^+ - (\gamma + d_1 + d_2) & 0 \\ b & \gamma & -d_1 \end{pmatrix} \tag{3.8}$$

Substituting $S^+ = \frac{\Pi}{d_1 + b}$ and $I^+ = 0$ into Eq. (3.8) gives

$$J^+ = \begin{pmatrix} -d_1 - b & \frac{-\beta\Pi}{d_1 + b} & 0 \\ 0 & \frac{\beta\Pi}{d_1 + b} - \gamma - d_1 - d_2 & 0 \\ b & \gamma & -d_1 \end{pmatrix}. \tag{3.9}$$

The determinant of the matrix in Eq. (3.9) is

$$|J^+ - I\lambda| = \begin{vmatrix} -d_1 - b - \lambda & \frac{-\beta\Pi}{d_1 + b} & 0 \\ 0 & \frac{\beta\Pi}{d_1 + b} - \gamma - d_1 - d_2 - \lambda & 0 \\ b & \gamma & -d_1 - \lambda \end{vmatrix}, \tag{3.10}$$

The solution of $|J^+ - I\lambda| = 0$ in Eq. (3.10) i.e its eigenvalues are

$$\begin{aligned} \lambda_1 &= -d_1, \lambda_2 = -(d_1 + b), \text{ and} \\ \lambda_3 &= \frac{\beta\Pi - (d_1 + b)(\gamma + d_1 + d_2)}{d_1 + b}. \end{aligned} \tag{3.11}$$

Lemma 3.1 *The disease-free equilibrium point (E^+) in Eq. (2.1) is asymptotically stable if $\lambda_1, \lambda_2, \lambda_3 < 0$ and unstable if at least one of $\lambda_1, \lambda_2, \lambda_3$ is greater than zero for all $\beta, d_1, \gamma, d_2, b$ and Π are positive.*

Proof 3.2 *The disease-free equilibrium point (E^+) is asymptotically stable if all the eigenvalues $\lambda_i, i = 1, 2, 3$ of $J^+(E^+)$ satisfy Routh-Hurwitz criterion [10]:*

Applying the Routh-Hurwitz theorem, from Eq. (3.11), we see that the first two eigenvalues λ_1 and λ_2 have negative real parts. We now establish the necessary and sufficient



condition for the λ_3 to have negative real part in order for the disease-free equilibrium to be stable and as well to be asymptotically stable. From λ_3 we obtain

$$-\frac{[(d_1 + b)(\gamma + d_1 + d_2) - \beta\Pi]}{d_1 + b} < 0, \tag{3.12}$$

inequality in Eq. (3.12) becomes

$$(d_1 + b)(\gamma + d_1 + d_2) > \beta\Pi, \tag{3.13}$$

or

$$\beta\Pi < (d_1 + b)(\gamma + d_1 + d_2). \tag{3.14}$$

Dividing Eq. (3.14) by $(d_1 + b)$ we obtain

$$\frac{\beta\Pi}{(d_1 + b)} < (\gamma + d_1 + d_2). \tag{3.15}$$

The inequality in Eqs. (3.14) and (3.15) gives the necessary and sufficient condition for the disease-free equilibrium state E^+ of the model to be asymptotically stable.

The product of total contraction and total breakdown of infectious class given by $(\beta\Pi)$ must be less than the total removal rate from both susceptible and infectious classes given by $(d_1 + b)(\gamma + d_1 + d_2)$.

3.4 Basic Reproductive Number (R_0) of the Model

The basic Reproductive Number (R_0) is the average number of secondary infections caused by a single infectious individual during their entire infectious lifetime.

Heesterbeek and Dietz [11] stated that “ R_0 is one of the foremost and most valuable ideas that mathematical thinking has brought to epidemic theory”. It helps us to set the threshold in the study of the disease both for predicting its outbreak and for evaluating its control strategies.

Thus, the reproduction number, R_0 , simply enables us to know whether a disease has died out or is persistent in a community.

In this work, we derived the threshold quantity known as reproductive number (R_0) from the largest eigenvalue of Jacobian matrix corresponding to equilibrium state.

Recall that

$$\lambda_1 = -d_1, \quad \lambda_2 = -(d_1 + b), \quad \text{and} \quad \lambda_3 = \frac{\beta\Pi - (d_1 + b)(\gamma + d_1 + d_2)}{d_1 + b}, \forall \beta, d_1, b, d_2, \gamma, \text{ and } \Pi \text{ are all positive}$$

If we considered λ_3 to be the largest eigenvalue since λ_1 and λ_2 already contain negative real part.

To derive our threshold quantity R_0 we let $\lambda_3 < 0$ then, we have

$$\frac{\beta\Pi - (d_1 + b)(\gamma + d_1 + d_2)}{d_1 + b} < 0, \tag{3.16}$$

Simplifying inequality in Eq. (3.16), we obtain

$$\frac{\beta\Pi}{(d_1 + b)(\gamma + d_1 + d_2)} < 1. \tag{3.17}$$

Hence, Eq. (3.17) allows the definition of R_0 for the model as

$$R_0 = \frac{\beta\Pi}{(d_1 + b)(\gamma + d_1 + d_2)}, \tag{3.18}$$

where $(d_1 + b)(\gamma + d_1 + d_2) \neq 0$

Remark 3.3 The threshold quantity R_0 , defined in Eq. (3.18) is the basic reproduction ratio of infection for the nonlinear autonomous ordinary differential equations in Eq. (2.1) ([6–8, 12]).

Remark 3.4 Epidemiologically,

- (i) if $R_0 < 1$, the occurrence of the disease will decrease and the disease will eventually be eliminated.
- (ii) if $R_0 = 1$, the disease occurrence will be constant.
- (iii) if $R_0 > 1$ the occurrence of the disease will increase. Disease persist.

Thus, we have also establish the following result.

Theorem 3.5 The disease-free equilibrium E^+ of the system in Eq. (2.1) is locally asymptotically stable in Γ if $R_0 < 1$ and unstable if $R_0 > 1$ for $\Pi, \beta, \gamma, d_1, b$ and d_2 are all positive.

Proof 3.6 From Lemma 3.1 we see that, $\lambda_1, \lambda_2 < 0$, then the disease-free equilibrium points E^+ is locally asymptotically stable if $\lambda_3 < 0$.

By definition

$$R_0 = \frac{\beta\Pi}{(d_1 + b)(d_1 + \gamma + d_2)}.$$

Using the inequality in Eq. (3.17)

$$R_0 < 1,$$

Noting that $\lambda_3 < 0$ if and only if $R_0 < 1$. Therefore, disease-free equilibrium E^+ of (2.1) is locally asymptotically stable. Otherwise, if

$$R_0 > 1,$$

λ_3 is positive. Therefore, disease-free equilibrium point E^+ of (2.1) becomes locally asymptotically unstable, the Theorem (3.5) is proven.

In view of remark (3.4) to our model, we have

Remark 3.7

- (i) if $\beta\Pi < (d_1 + b)(d_1 + \gamma + d_2)$, the occurrence of the measles infection will decrease and the disease will eventually be eliminated. i.e no epidemic.
- (ii) if $\beta\Pi = (d_1 + b)(d_1 + \gamma + d_2)$, the occurrence of measles infection will be constant.



(iii) if $\beta\Pi > (d_1 + b)(d_1 + \gamma + d_2)$ the occurrence of the measles infection will increase. Infection persists, each individual will produce more than one new infected.

In the context of epidemiological modelling according to Anderson and May [3], it is generally known that if $R_0 < 1$, then the disease-free equilibrium is locally asymptotically stable (and the disease will be eradicated from the community if the initial sizes of the three state variables are within the vicinity of E^+).

If the equilibrium E^+ is globally asymptotically stable, then the disease will be eradicated from the population irrespective of the initial sizes of the three state variables. Therefore, in the event of an epidemic, the theoretical determination of conditions that can make R_0 less than unity is of great public health interest.

3.5 Global Stability of Disease-Free Equilibrium

In this subsection, we prove the global stability of the disease-free equilibrium (E^+) when the basic reproductive number is less than or equal to unity. The result is obtained by means of Lyapunov function constructed of the combinations of composite quadratic, common quadratic and linear functions (see, [39, 40]) using the LaSalle's Invariance Principle [13].

Theorem 3.8 *The disease-free equilibrium E^+ , of Eq. (3.2) is globally asymptotically stable in Γ . If $R_0 \leq 1$.*

Proof 3.9 *Consider the following Lyapunov function candidate*

$$V(S, I, R) = \frac{1}{2}[(S - S^+) + I + R]^2 + \frac{(d_2 + 2d_1)}{\beta}I + \frac{(d_2 + 2d_1)}{2\gamma}R^2. \tag{3.19}$$

Then it is obvious that $V(t)$ is defined and continuous for all $t \geq 0, S(t), I(t), R(t) > 0$, and $V(t) \geq 0$ for all $t \geq 0$ with $V(t) = 0$ only at E^+ . The time derivative of $V(t)$ along the solution of (2.1) is given by

$$\begin{aligned} \dot{V} = & [(S - S^+) + I + R](\Pi - d_1(S + I + R) - d_2I) \\ & + \frac{(d_2 + 2d_1)}{\beta}(\beta SI - (\gamma + d_1 + d_2)I) \\ & + \frac{(d_2 + 2d_1)}{\gamma}R(\gamma I - d_1R + bS). \end{aligned} \tag{3.20}$$

Using $\Pi = (d_1 + b)S^+$, we get

$$\begin{aligned} \dot{V} = & [(S - S^+) + I + R]((d_1 + b)S^+ - d_1(S + I + R) \\ & - d_2I) + \frac{(d_2 + 2d_1)}{\beta}(\beta SI - (\gamma + d_1 + d_2)I) \\ & + \frac{(d_2 + 2d_1)}{\gamma}R(\gamma I - d_1R + bS), \end{aligned} \tag{3.21}$$

After simplification, we obtain

$$\begin{aligned} \dot{V} = & -d_1[(S - S^+) + R]^2 - (d_1 + d_2)I^2 \\ & - \frac{d_1(d_2 + 2d_1)}{\gamma}[d_1R - bS]R - bS^+[S^+ - S - R - I] \\ & - (d_2 + 2d_1)\left[\frac{(\gamma + d_1 + d_2)}{\beta} - S^+\right]I. \end{aligned} \tag{3.22}$$

Rewritten \dot{V} in term of basic reproductive number, we have

$$\begin{aligned} \dot{V} = & -d_1[(S - S^+) + R]^2 - (d_1 + d_2)I^2 \\ & - \frac{d_1(d_2 + 2d_1)}{\gamma}[d_1R - bS]R - b[S^+ - S - R - I]S^+ \\ & - \frac{(d_2 + 2d_1)(\gamma + d_1 + d_2)}{\beta}(1 - R_0)I, \end{aligned} \tag{3.23}$$

$$\begin{aligned} \dot{V} \leq & -\{d_1[(S - S^+) + R]^2 + (d_1 + d_2)I^2 \\ & + \frac{d_1(d_2 + 2d_1)}{\gamma}[d_1R - bS]R + b[S^+ - S - R - I]S^+ \\ & + \frac{(d_2 + 2d_1)(\gamma + d_1 + d_2)}{\beta}(1 - R_0)I\}. \end{aligned} \tag{3.24}$$

Hence, $\dot{V}(S, I, R) < 0$, if $R_0 \leq 1$. Also, $\dot{V}(S, I, R) = 0$ if and only if $S = S^+, I = 0$ and $R = 0$.

Therefore, the largest compact invariant set in $\{(S, I, R) \in \Gamma : \dot{V}(S, I, R) = 0\}$ is the singleton E^+ , where E^+ is the disease-free equilibrium. Therefore, by LaSalle's Lyapunov theorem ([13, 21–23, 31, 39, 40]), every solution that starts in Γ approaches E^+ as $t \rightarrow \infty$. This shows that E^+ is globally asymptotically stable in Γ .

3.6 Existence and Uniqueness of Endemic Equilibrium (EE)

Endemic equilibrium state is the state where the disease cannot be totally eradicated but persists in the population. Let the endemic equilibrium be $E^* = (S^*, I^*, R^*)$. Then, the susceptible class S , the infectious class, I and the recovered class, R , must not be zero at equilibrium state i.e. $E^* = (S^*, I^*, R^*) \neq (0, 0, 0)$.

In order to obtain the endemic equilibrium state, one solves Eq. (2.1)

$$\begin{aligned} \Pi - (\beta I^* + d_1 + b)S^* &= 0, \\ \beta S^* I^* - (\gamma + d_1 + d_2)I^* &= 0, \\ \gamma I^* - d_1 R^* + bS^* &= 0. \end{aligned} \tag{3.25}$$

The solution $E^* = (S^*, I^*, R^*) \neq (0, 0, 0)$ of the above equation is

$$\begin{aligned} S^* &= \frac{(\gamma + d_1 + d_2)}{\beta}, I^* = \frac{\beta\Pi - (d_1 + b)(d_1 + \gamma + d_2)}{\beta(\gamma + d_1 + d_2)}, \\ R^* &= \frac{\beta\Pi\gamma - (d_1 + \gamma + d_2)(\gamma d_1 - b d_1 - b d_2)}{\beta d_1(\gamma + d_1 + d_2)}. \end{aligned} \tag{3.26}$$



The vector representation of solution in Eq.(3.26) is

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

$$= \left(\frac{(\gamma + d_1 + d_2)}{\beta}, \frac{\beta \Pi - (d_1 + b)(d_1 + \gamma + d_2)}{\beta(\gamma + d_1 + d_2)}, \frac{\beta \Pi \gamma - (d_1 + \gamma + d_2)(\gamma d_1 - b d_1 - b d_2)}{\beta d_1(\gamma + d_1 + d_2)} \right), \quad (3.27)$$

Now, we proceed to express the endemic equilibrium state in term of reproductive number (R_0)

$$S^* = \frac{(\gamma + d_1 + d_2)}{\beta}. \quad (3.28)$$

It can be rewritten in terms of R_0 as

$$S^* = \frac{S^+}{R_0}, \quad (3.29)$$

where

$$S^+ = \frac{\Pi}{(d_1 + b)}, \text{ and } R_0 = \frac{\beta \Pi}{(d_1 + b)(\gamma + d_1 + d_2)}.$$

Therefore,

$$S^* = \frac{\Pi}{(d_1 + b)R_0}. \quad (3.30)$$

Substituting Eq. (3.30) into first Eq. of system of Eq. (3.25) yields

$$I^* = \frac{(d_1 + b)(R_0 - 1)}{\beta}. \quad (3.31)$$

Substituting Eqs. (3.30) and (3.31) into third Eq. of Eq. (3.25) gives

$$R^* = \frac{\gamma R_0 (d_1 + b)^2 (R_0 - 1) + \beta b \Pi}{\beta d_1 R_0 (d_1 + b)}. \quad (3.32)$$

Thus, if $R_0 > 1$, it shows that $I^* > 0$ and model equation in Eq. (2.1) has a unique endemic equilibrium given by $E^* = (S^*, I^*, R^*)$ where in the presence of infection ($I \neq 0$),

$$E^*(S^*, I^*, R^*) = \begin{cases} S^* = \frac{\Pi}{(d_1 + b)R_0} \\ I^* = \frac{(d_1 + b)(R_0 - 1)}{\beta} \\ R^* = \frac{\gamma R_0 (d_1 + b)^2 (R_0 - 1) + \beta b \Pi}{\beta d_1 R_0 (d_1 + b)} \end{cases} \quad (3.33)$$

Clearly, it is evident from the above three equations that if $R_0 < 1$, then the model has no positive endemic equilibrium (since I^* will assume negative values which are biologically unrealistic).

Therefore, to ensure the existence of a positive endemic equilibrium, we require $R_0 > 1$. Since $S^*, I^*, R^* > 0$ (when $R_0 > 1$), the endemic equilibrium E^* is positive and $I^* > 0$. This is the condition for the existence and uniqueness of the endemic equilibrium for the system of Eq. (2.1).

3.7 Global Stability of Endemic Equilibrium

Herein, we study the global behaviour of the endemic equilibrium E^* for the model Eq. (2.1), where we use the same Lyapunov functions used in [1, 4, 6, 14, 18–20], to demonstrate the global stability of the endemic equilibrium of our model. We have the following results.

Theorem 3.10 *If $R_0 > 1$, the unique endemic equilibrium E^* is globally asymptotically stable on Γ .*

Proof 3.11 *Consider the following Lyapunov function candidate*

$$V(S, I, R) = S - S^* \ln S + A(I - I^* \ln I) + B(R - R^* \ln R) \quad (3.34)$$

defined and continuous for all $S, I, R > 0$ and satisfies

$$\frac{dV}{dt} = \frac{\partial V}{\partial S} \frac{dS}{dt} + \frac{\partial V}{\partial I} \frac{dI}{dt} + \frac{\partial V}{\partial R} \frac{dR}{dt},$$

which becomes

$$\dot{V}(S, I, R) = \left(1 - \frac{S^*}{S}\right) \dot{S} + A \left(1 - \frac{I^*}{I}\right) \dot{I} + B \left(1 - \frac{R^*}{R}\right) \dot{R}. \quad (3.35)$$

$$= \left(1 - \frac{S^*}{S}\right) (\Pi - (\beta SI + d_1 S + bS)) + A \left(1 - \frac{I^*}{I}\right) (\beta SI - (\gamma I + d_1 I + d_2 I)) + B \left(1 - \frac{R^*}{R}\right) (\gamma I - d_1 R + bS). \quad (3.36)$$

By considering Eq. (2.1) at endemic equilibrium where first Eq. is

$$\Pi - (\beta S^* I^* + d_1 S^* + bS^*) = 0,$$

which becomes

$$\Pi = \beta S^* I^* + d_1 S^* + bS^*.$$

Also from second Eq. of system of Eq. (2.1) we have

$$\beta S^* = \gamma + d_1 + d_2.$$

Then from third Eq. of system of Eq. (2.1) where

$$\gamma I^* - d_1 R^* + bS^* = 0,$$

$$d_1 = \frac{\gamma I^* + bS^*}{R^*}.$$

Substituting the values of Π , βS^ and d_1 into Eq. (3.36) we obtain*

$$\dot{V}(S, I, R) \leq \left(1 - \frac{S^*}{S}\right) (\beta S^* I^* + d_1 S^* + bS^* - (\beta SI + d_1 S + bS)) + B \left(1 - \frac{R^*}{R}\right) \left(\gamma I - R \frac{(\gamma I^* + bS^*)}{R^*} + bS\right).$$



After simplification, we have

$$\begin{aligned} \dot{V} \leq & - \left(\frac{d_1 + b}{S} (S - S^*)^2 + \beta \left(1 - \frac{S^*}{S} \right) (SI - S^*I^*) \right. \\ & + B(\gamma I^* + bS^*) \left(\frac{R}{R^*} - 1 \right) + B(\gamma I + bS) \left(\frac{R^*}{R} - 1 \right) \\ & \left. + A\beta(I^* - I)(S - S^*) \right). \end{aligned} \tag{3.37}$$

Hence, $\dot{V} < 0$ for $A, B > 0$. Note that, $\dot{V} = 0$ if and only if $S = S^*, I = I^*$ and $R = R^*$. Therefore the largest compact invariant set in $(S, I, R) \in \Gamma : \dot{V} = 0$ is the singleton E^* , where E^* is the endemic equilibrium, LaSalle's invariant principle then implies that E^* is globally asymptotically stable in the interior of Γ .

4. Numerical Simulation Results and Discussions

In this section, we present a numerical simulation of solutions of the model Eq. (2.1). From practical point of view, we realized that numerical solutions are very important beside analytical study. For this work, we compare the parameters used with some existing work as indicated in the table 2 and 3.

Table 2. Values of parameters used for simulation

parameter	Values	Source
Π	1000	Assumed
γ	0.2	[29]
β	0.00025	Assumed
d_1	0.05	Assumed
d_2	0.075	[32]
b	0.00 - 1.00	Assumed

Table 3. Various control used and the outcome of Reproduction number (R_0) in the simulation of the model

Immunity gain (b)	(S)	(I)	(R)	R_0
0.00	9900	100	0	15.38461538
0.25	9650	100	250	2.564102564
0.50	9400	100	500	1.398601398
0.75	9150	100	750	0.9615384615
1.00	8900	100	1000	0.7326007326

We implement the system (2.1) in Maple-13 using a fourth order Runge-Kutta method for solving system of ODEs. For our simulations, we choose the initial populations to be $S(0) = 9900, I(0) = 100, R(0) = 0$ for $b = 0.00$ at initial stage. The variables S, I, R changed along with the immunity gain rate (b) see table 2 and 3. We have investigated the stability of the disease-free and the endemic equilibria based on R_0 using linearization approach and Lyapunov functions for both the local and global stability respectively. We described

in figure 2 - 6, the effect of immunity gain at initial stage on the infective; thus reducing the number of infections in the population.

The infectivity rate, β and immunity gain rate, b are used to govern the value of the basic reproduction number. For this system, if $b \leq 0.50, R_0 > 1$ and if $b > 0.50, R_0 \leq 1$ (β is constant) this is shown in figure 7. The simulations have been done for various values of b as a key parameter and are shown in the figures below.

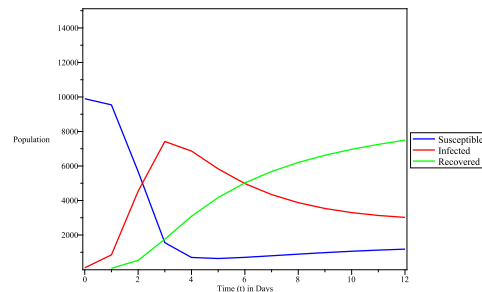


Figure 2. The plot shows the simulation of the population with time at initial immunity level: $b = 0\%$. $R_0 = 15.38461538 > 1$

Figure 2 shows that if at initial points, none of the new recruits is having immunity against measles infection that is $b = 0\%$, the graph shows that infected will increase drastically but at later hour if attention is being taken on time with higher recovery rate the graph shows that the recovered class will now increase and infected class will reduce from the entire population.

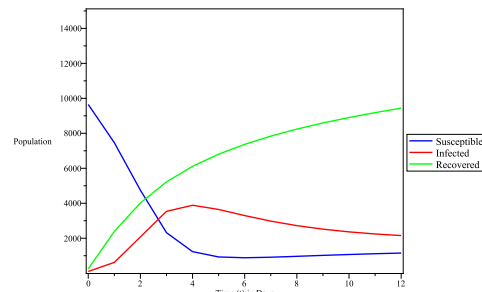


Figure 3. The plot shows the simulation of the population with time at initial immunity level: $b = 25\%$. $R_0 = 2.564102564 > 1$

Figure 3, indicates the graph of population against time (t). It is seen that the infected class has reduced drastically with time when 25% of people that are coming into the susceptible class are already immunized against measles infection, the susceptible class also reduced and Recovered class increased. But there is still infection due to the $R_0 > 1$ at this stage.



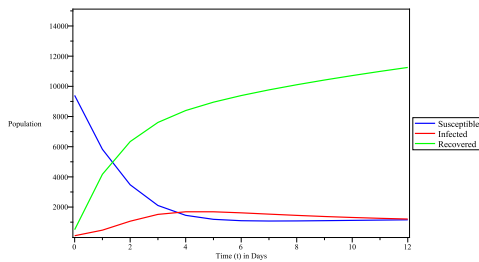


Figure 4. The plot shows the simulation of the population with time at initial immunity level: $b = 50\%$. $R_0 = 1.398601398 \geq 1$

Figure 4, also displays the graph of population against time (t). It is also seen that the infected class has reduced drastically with time when 50% of people that are coming into the susceptible class are already immunized against measles infection, the susceptible class also reduced and Recovered class increased more. But at this stage, infection is neither increased nor reduced. It remains constant in the population due to the $R_0 = 1$ at this stage.

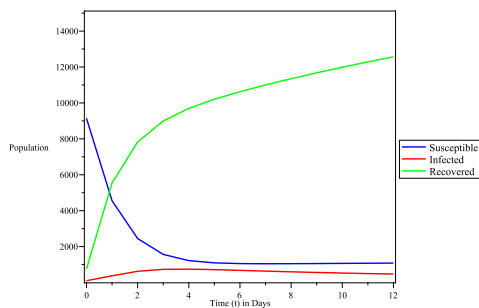


Figure 5. The plot shows the simulation of the population with time at initial immunity level: $b = 75\%$. $R_0 = 0.9615384615 < 1$

Figure 5, also shows the graph of population against time (t). It is as well seen that the infected class has reduced rapidly with time when 75% of people that are coming into the susceptible class are already immunized against measles infection, the susceptible class also reduced and and Recovered class increased more. At this stage, measles infection was reduced to some certain level in the population due to the $R_0 \leq 1$.

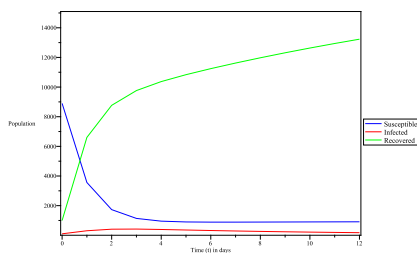


Figure 6. The plot shows the simulation of the population with time at initial immunity level: $b = 100\%$. $R_0 = 0.7326007326 < 1$

Figure 6, also shows the graph of population against time (t). It has been seen that when 100% of people that are coming into the susceptible class are already immunized against measles infection, the susceptible class reduced, the Recovered class increased more and the infected class has reduced extremely with time, which implies that the disease would die out as time goes on. At this stage the $R_0 < 1$

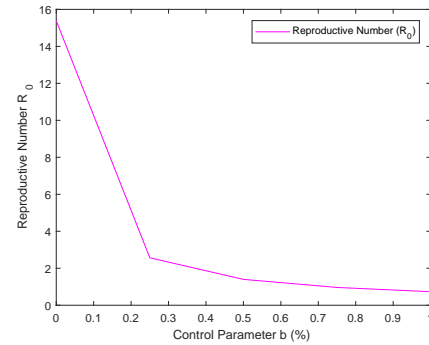


Figure 7. Reproductive Number R_0 against Control Parameter b

Figure 7 shows the graph of reproduction number against control parameters. It can be seen that the disease would die out with respect to time, if certain proportion of the population has been immunized or possessed strong immunity against the measles infection, which would make the population free of the measles disease.

5. Conclusion and Recommendations

5.1 Conclusion

As stated in the introduction, the purpose of this work is to study the behaviour and analyze the prevention of dynamics of measles infection in an open population and recommend strategies for its control. We have carried out both local and global qualitative analysis for this model where we discussed the local and global stability of disease-free and endemic equilibria. Analysis of the system was done by evaluating the basic reproduction number $R_0 = \frac{\beta \Pi}{(d_1 + b)(\gamma + d_1 + d_1)}$, our results shows that when $R_0 < 1$, the disease-free equilibrium is globally stable and when $R_0 > 1$, the endemic equilibrium is globally stable. And the model was simulated using the parameter evaluated. It was proved that as long as the value of R_0 is kept minimal and $\beta \Pi < (d_1 + b)(\gamma + d_1 + d_1)$, the disease can be eradicated from the population. The model shows that the higher the value of R_0 , the more likely the measles infection will spread at higher rates.

5.2 Recommendations

- R_0 can be kept low by employing various policies such as increasing knowledge of public in terms of prevention and treatment, increase hygiene conditions at all levels of their daily activities.



- For the population to be free from measles infection, we recommend that individuals need to be vaccinated before joining the population either during birth or through immigration and emigration.
- We would strongly recommend that the future work should integrate the vaccination and treatment.

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