# Boundedness and Stability Properties of Solutions of Mathematical Model of Measles 

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#### Abstract

In this paper, asymptotic stability and global asymptotic stability of solutions to a deterministic and compartmental mathematical model of measles infection is considered using the ideas of the Jacobian determinant as well as the second method of Lyapunov, criteria/conditions that guaranteed asymptotic stability of disease free equilibrium and endemic equilibrium were established. Also the basic reproductive number $R_{0}$ was obtained. The results in this work compliments existing work and provided further information in controlling the disease in an open population.


## 1 Introduction

Ever since the day of creation, epidemic has been wiping out human existence, thus understanding their chain of causation is important to curtail its spread. In this article the main focus is on measles infection which is highly transmissible disease caused by the measles virus called paramyxovirus family from the morbillivirus genus.

Measles is one of the most and best known deadly infectious diseases of all childhood rash/fever illnesses. It resides in the mucus in the nose and throat of an infected person, so transmission normally occurs through coughing and sneezing via direct contact with emissions [42, 45]. Symptoms of measles usually develop from 8-12 days after exposure to an infectious person [47]. The infection of measles is so serious that mother says never count your children until after the measles in the developing world[45]. Measles only survive on the object, surfaces and in the air under 2 hours. Almost all those infected with the virus recovered if care is being taken on time, but measles snags can be incurable or very risky. Some effect of measles include the following: ear infections, diarrhea, pneumonia, and encephalitis - this is rare, but can cause permanent brain damage or death. Till today measles is still a dangerous disease in the whole world. World Health

[^0]Organization (WHO), in the year 2012 released a report on complication of measles that about 122,000 people have died globally through the measles infection[58]. Since the disease occurs once in a life time, therefore it confers life long immunity from further attacks ([40],[46]).

Mathematical epidemiology has contributed to the understanding of the behavior of infectious diseases, its effects and predictions of possible way out on its spreading. Mathematical models are used as a way of comparing, planning, implementing and evaluating various detection, prevention and control programs for measles transmission[2,13,28,55,56,59]. Numerous ideas have been contributed by mathematical and non-mathematical researchers, and these works have helped immensely in the area of checking the behavior and control intervention strategy for the transmission of infectious diseases in the society at large by public health workers. These include: Graunt (1662)[12], Bernoulli (1760)[22], D'Alembert [11] and Hamer [20].

The Vibrant sketch of measles dynamics and its mathematical formulation has been done by many researchers for example: (see [18, 25, 27, 36, 41, 45, 46, 50, 51]) among others. However study via $S, I, R$ type in an open population using the direct Lyapunov methods to check its behavior and control, little or nothing has been done yet. In 1892, Russian mathematician Lyapunov developed a method for the analysis of the stability of ordinary differential equations [38]. This method, known as the "Direct Lyapunov Method", is one of the powerful tools for a qualitative analysis of a dynamical system.

Kermack-Mckendrick[29], created a well known basic compartmental model on which many researchers built upon, for instance Atkins[3], Weiss[57] and many more (see [10, 26, 43, 49]). The model considered a closed population with three compartments $S-I-R$. The threshold result shows that for an epidemic to occur the density of susceptible must surpass a critical point.

In his own study, Bartlett [4] gave an estimate of the critical community size for measles for the United States in terms of total population. Since then, various mathematical models have been developed to investigate the transmission dynamics of measles in different countries and regions [ $9,15,17]$.

Liuyong et al (in [37]) proposed an $S-E-I-R$ model for measles epidemic and investigated the effect of vaccination in controlling its spread. They obtained two critical threshold values, $u_{c_{1}}$ and $u_{c_{2}}$. They concluded that Measles will be extinct when the vaccination ratio $u>u_{c_{1}}$, endemic when $u_{c_{2}}<u<u_{c_{1}}$, and outbreak periodically when $u<u_{c_{2}}$. Momoh et al [39] also investigated SEIR epidemic model to ascertain the impact of exposed individuals at latent period on the transmission dynamics of measles using stability analysis.

To this end we have great motivation to understand the history, spread and means of controlling infection of measles and their transmission characteristics. Valuable information on transmission and effective control of the measles epidemics as well as appropriate policies are very important. In this article, we study and analyze the behavior of solutions of transmission and control
of measles infection by deterministic mathematical model; analyze the condition that determine the level of the effective prevention of the spread of measles using $S-I-R$-type compartmental model $[29,48]$ in an open population; adapt ( $[33,34,35,38,44,53,54]$ ) to analyze the qualitative properties of solutions of our model and give sufficient conditions for the model to be stable. For the $S-I-R$ model to be fitting, once a person has recovered from the disease, they would acquire permanent immunity.

This paper is arranged as follows: Section 2 introduces the model formulation to study the dynamics of measles infection in an open population. In section 3, stability of the model equilibria was analyzed while conclusion and recommendations are given in the last section.

The following are the notations adopted in this work.
Table 1: Variables/Parameters used in the model and their meaning

| Variables/parameter | Meaning |
| :--- | :--- |
| $S(t)$ | The Susceptible population at time $(t)$ |
| $I(t)$ | The Infected population at time $(t)$ |
| $R(t)$ | The Recovered population at time $(t)$ |
| $N(t)$ | Total population at time $(t)$ |
| $\beta$ | Infection/contact rate |
| $\mu$ | Natural death rate |
| $K$ | Recruitment rate |
| $\gamma$ | Recovery rate |
| $\psi$ | Mortality rate due to measles infection |
| $\sigma$ | Rate of recovery from susceptible to recovered or immunity gain rate |
| $R_{0}$ | Reproductive Number |
| $\Omega$ | Bounded domain in $\Re^{3}$ for S-I-R Model |
| $V$ | Continuous Lyapunov function |
| $A, B, \delta$ | Arbitrary constants |

## 2 Measles Model Formulation

In this section, we formulate mathematical model to describe the transmission dynamics of measles. The progression of measles within the total population can be simplified to three differential equations. These three equations represent three different groups of people:

- the susceptible, $(\mathrm{S})$ are member of the population who have never contacted measles;
- the infective, (I) are member of the population who have been infected with measles and are able to transmit the disease; and
- the recovered, $(\mathrm{R})$ are member of the population that have recovered from measles.


### 2.1 Measles Model Description and Formulation

The model considers three mutually exclusive compartments of $S(t), I(t), R(t)$ of a deterministic ordinary differential equation (ODE), in a mixed homogeneous population. The total population at any time $(t)$, denoted by $N(t)$, is the sum of individual populations in each compartment. Thus, $N(t)=S(t)+I(t)+R(t)$. The model maintains the basic intuition of basic compartmental model [29], with the exception of the introduction of the recruitment in an open population.

In this model, we assume that the new recruits enter the susceptible class at a constant rate $K$, either immunized or not. The susceptible compartment of the population also decreases due to natural death rate of $\mu$ and infection of individuals at the rate of $\beta$. It also reduces due to the rate of recovery from susceptible to recovered compartment at rate of $\sigma$. The population of the infectious compartment increases due to the progression of susceptible individuals who are infected with measles disease at the rate of $\beta$. Also the compartment reduces as a result of successful cure of measles patient at the rate of $\gamma$, natural death at the rate of $\mu$ and also the death that occur with the infection of measles disease. The recovered compartment grows as a result of uninfected individual moving from susceptible at rate of $\sigma$ and successful treatment with cure of infected patient at rate of $\gamma$, while the compartment decreases due to natural death at the rate of $\mu$. Total per capita removal rate is defined by $\theta$ and it is a composition of mortality natural death rate $\mu$, measles disease death rate $\psi$, and recovery rate $\gamma$, That is, $\theta=\mu+\psi+\gamma$.

### 2.2 Assumptions of the Model

The model is based on the following assumptions:
(i) The entry into the population is open (either immunized or not) and the way of exit is through mortality by natural cause or death caused by measles infection.
(ii) The population is heterogeneous, that is, the individuals that make up the population can be grouped into different compartment or groups according to their epidemiological state.
(iii) The population size in a compartment is differentiable with respect to time ( t ) and also mixes homogeneously.
(iv) The immunity conferred on the individuals that are immunized by vaccination expires within a given rate and those that are not immunized which have partial immunity or low immunity undergo the same condition.
(v) The people with active immunity in susceptible compartment moved directly to recovered compartment without being infected.
(vi) Measles infection confers permanent immunity. Therefore individuals in this category recovered completely or die.
(vii) The people in each compartment have equal mortality or natural death rate $\mu$.

Taking into account the above considerations, the schematic flow diagram for measles model is shown in Figure 1.


Figure 1: The flowchart showing the dynamics of the model

### 2.3 Mathematical Formulation of the Model

Applying the descriptions, assumptions and biological flow chart in Figure 1, we obtained the following system of ordinary differential equations.

$$
\begin{align*}
& \frac{d S}{d t}=K-\beta S I-\mu S-\sigma S \\
& \frac{d I}{d t}=\beta S I-\gamma I-\mu I-\psi I  \tag{2.1}\\
& \frac{d R}{d t}=\gamma I-\mu R+\sigma S
\end{align*}
$$

### 2.4 Dynamics of the model

Let a total population $N(t)=S(t)+I(t)+R(t)$ and taking the time derivative of $N(t)$ along solutions of model the equation (2.1), we obtain

$$
\begin{equation*}
\frac{d N}{d t}=K-\mu N-\psi I \tag{2.2}
\end{equation*}
$$

Hence, equation (2.2) is where there are changes in the population known as population dynamics.

### 2.5 Basic Properties of the model

In this subsection, we investigate the feasibility and positivity of the solution of the measles model

### 2.5.1 Feasibility of the Model

The feasibility of the model describes the region in which the solution of the system of equation (2.1) is biologically meaningful.

Theorem 2.1. Suppose equation (2.2) holds, every solution of the model in system of equation (2.1) with initial conditions in $\Re_{+}^{3}$ approaches and stays in the compact set $(\Omega)$ as $t \rightarrow \infty$. Then, the feasible solution which is a positively invariant set of the model is given by

$$
\Omega=\left\{(S, I, R) \in \Re_{+}^{3}: N(t) \leq \frac{K}{\mu}\right\} .
$$

Proof. From the equation (2.2) where changes of $N$ leads to change of all variables in the population (i.e $N=S+I+R$ ) we have

$$
\begin{equation*}
\frac{d N}{d t}=K-\mu N(t)-\psi I \tag{2.3}
\end{equation*}
$$

In the absence of disease ( $\psi=0$ ), the equation (2.3) reduces to

$$
\begin{equation*}
\frac{d N}{d t}=K-\mu N(t) \tag{2.4}
\end{equation*}
$$

From the equation (2.4) we observe that,

$$
\frac{d N}{d t} \leq 0 \quad \text { if } \quad N(t) \geq \frac{K}{\mu}
$$

Therefore,

$$
\begin{equation*}
\frac{d N}{d t} \leq K-\mu N(t) \tag{2.5}
\end{equation*}
$$

Applying Birkhoff and Rota's theorem [7] on differential inequalities and method of integrating factor (IF) on the inequality (2.5) we will have

$$
\begin{equation*}
\frac{d}{d t}\left[\mathrm{e}^{\mu t} N(t)\right] \leq \mathrm{e}^{\mu t} K \tag{2.6}
\end{equation*}
$$

Integrating the inequality (2.6) on both sides along with the initial condition $t=0$ we obtain

$$
\begin{equation*}
N(t) \leq N(0) \mathrm{e}^{-\mu t}+\frac{K}{\mu}\left(1-\mathrm{e}^{-\mu t}\right) \tag{2.7}
\end{equation*}
$$

Hence, at $\lim \mathrm{t} \rightarrow \infty$

$$
\begin{equation*}
N(t) \leq \frac{K}{\mu} \tag{2.8}
\end{equation*}
$$

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

Hence, the feasible solution which is given by

$$
\Omega=\left\{(S, I, R) \in \Re_{+}^{3}: N(t) \leq \frac{K}{\mu}\right\}
$$

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

### 2.5.2 Positivity of solutions

The positivity of solution describes non-negativity of the solutions of model equation (2.1). For model in equation (2.1) to be epidemiologically meaningful, it is important to prove that all its state variables are non negative for all time $t$. We considered the lemma below.

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

Proof. From the first equation in system of equation (2.1), it is assumed that

$$
\begin{gather*}
\frac{d S}{d t}=K-\beta S I-(\mu+\sigma) S \geq-(\mu+\sigma) S, \text { for } \beta \in[0,1) \text { and } \beta \leq \frac{K}{S I} \\
\frac{d S}{d t} \geq-(\mu+\sigma) S \tag{2.9}
\end{gather*}
$$

Integrating inequality (2.9) by separating variables gives

$$
\begin{equation*}
S(t) \geq S(0) \mathrm{e}^{-(\mu+\sigma) t}, \text { since }(\mu+\sigma)>0 \tag{2.10}
\end{equation*}
$$

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

$$
\begin{equation*}
\frac{d I}{d t}=\beta S I-(\gamma+\mu+\psi) I \geq-(\gamma+\mu+\psi) I \tag{2.11}
\end{equation*}
$$

The solution of the inequality (2.11) is

$$
\begin{gather*}
I(t) \geq I(0) \mathrm{e}^{-(\gamma+\mu+\psi) t} \geq 0, \text { since }(\gamma+\mu+\psi)>0  \tag{2.12}\\
\frac{d R}{d t}=\gamma I-\mu R+\sigma S \geq-\mu R \tag{2.13}
\end{gather*}
$$

The solution of the inequality (2.13) is

$$
\begin{equation*}
R(t) \geq R(0) \mathrm{e}^{-\mu t} \geq 0, \text { since } \mu>0 \tag{2.14}
\end{equation*}
$$

The inequalities in (2.10), (2.12) and (2.14) show that the variables $\mathrm{S}(\mathrm{t}), \mathrm{I}(\mathrm{t})$ and $\mathrm{R}(\mathrm{t})$ 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 $\left(R_{0}\right)$ 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 Equilibrium Solutions

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

$$
\frac{d S}{d t}=\frac{d I}{d t}=\frac{d R}{d t}=0
$$

That is,

$$
\begin{align*}
& K-\beta S I-\mu S-\sigma S=0 \\
& \beta S I-\gamma I-\mu I-\psi I=0  \tag{3.1}\\
& \gamma I-\mu R+\sigma S=0
\end{align*}
$$

Let $\Omega^{+}$and $\Omega^{*}$ represent the boundary and the interior of $\Omega$ in $\Re^{3}$ respectively. Then, by direct calculation, it can be shown that the equation (3.1) has two equilibria in $\Re_{+}^{3}$ : the disease-free equilibrium $E^{+}\left(S^{+}, I^{+}, R^{+}\right) \in \Omega^{+}$and a unique endemic equilibrium $E^{*}\left(S^{*}, I^{*}, R^{*}\right) \in \Omega^{*}$

### 3.2 Disease - free equilibrium (DFE) Point

The disease-free equilibrium (DFE) point is the point at which there are no infection in the population. At the DFE, all the classes will be denoted with a plus $(+)$. Let $E^{+}=\left(S^{+}, I^{+}, R^{+}\right)$be the disease-free equilibrium state.
From model equation (2.1), we have

$$
\begin{align*}
& K-\beta S^{+} I^{+}-\mu S^{+}-\sigma S^{+}=0 \\
& \beta S^{+} I^{+}-\gamma I^{+}-\mu I^{+}-\psi I^{+}=0  \tag{3.2}\\
& \gamma I^{+}-\mu R^{+}+\sigma S^{+}=0
\end{align*}
$$

Substituting $I^{+}=0$ into the equation (3.2) gives

$$
\begin{gather*}
K-(\mu+\sigma) S^{+}=0 \\
\sigma S^{+}-\mu R^{+}=0 .  \tag{3.3}\\
S^{+}=\frac{K}{\mu+\sigma}, R^{+}=\frac{\sigma K}{\mu(\mu+\sigma)} . \tag{3.4}
\end{gather*}
$$

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

$$
\begin{equation*}
E^{+}=\left(S^{+}, I^{+}, R^{+}\right)=\left(\frac{K}{\mu+\sigma}, 0, \frac{\sigma K}{\mu(\mu+\sigma)}\right) \tag{3.5}
\end{equation*}
$$

### 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 behavior 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 andfor the disease to be totally eradicated from the population.
Recall that at equilibrium state, the system of equation (2.1) reduces to

$$
\begin{align*}
& \frac{d S}{d t}=K-\beta S I-\mu S-\sigma S=0 \\
& \frac{d I}{d t}=\beta S I-\gamma I-\mu I-\psi I=0  \tag{3.6}\\
& \frac{d R}{d t}=\gamma I-\mu R+\sigma S=0
\end{align*}
$$

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

Therefore, at disease-free equilibrium $\left(E^{+}\right)$, the Jacobian matrix $J^{+}$is

$$
J^{+}=\left(\begin{array}{ccc}
-\beta I^{+}-\mu-\sigma & -\beta S^{+} & 0  \tag{3.7}\\
\beta I^{+} & \beta S^{+}-\gamma-\mu-\psi & 0 \\
\sigma & \gamma & -\mu
\end{array}\right)
$$

Substituting $S^{+}=\frac{K}{\mu+\sigma}$ and $I^{+}=0$ into the equation (3.7) gives

$$
J^{+}=\left(\begin{array}{ccc}
-\mu-\sigma & \frac{-\beta K}{\mu+\sigma} & 0  \tag{3.8}\\
0 & \frac{\beta K}{\mu+\sigma}-\gamma-\mu-\psi & 0 \\
\sigma & \gamma & -\mu
\end{array}\right)
$$

The determinant of the matrix in the equation (3.8) is

$$
\left|J^{+}-I \lambda\right|=\left|\begin{array}{ccc}
-\mu-\sigma-\lambda & \frac{-\beta K}{\mu+\sigma} & 0  \tag{3.9}\\
0 & \frac{\beta K}{\mu+\sigma}-\gamma-\mu-\psi-\lambda & 0 \\
\sigma & \gamma & -\mu-\lambda
\end{array}\right|
$$

The solution of $\left|J^{+}-I \lambda\right|=0$ in equation (3.9) i.e its eigenvalues are

$$
\begin{equation*}
\lambda_{1}=-\mu, \lambda_{2}=-(\mu+\sigma), \text { and } \lambda_{3}=\frac{\beta K-(\mu+\sigma)(\gamma+\mu+\psi)}{\mu+\sigma} . \tag{3.10}
\end{equation*}
$$

Lemma 3.1. The disease-free equilibrium point $\left(E^{+}\right)$in the equation (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 $\beta, \mu, \gamma, \psi, \sigma$ and $K$ are positive.

Proof. The disease-free equilibrium point $\left(E^{+}\right)$is asymptotically stable if all the eigenvalues $\lambda_{i}$, i $=1,2,3$ of $J^{+}\left(E^{+}\right)$satisfy Routh-Hurwitz criterion [19]. Applying the Routh-Hurtwitz theorem, from Eq. (3.10), we see that the first two eigenvalues $\lambda_{1}$ and $\lambda_{2}$ have negative real parts. We now establish the necessary and sufficient condition for the $\lambda_{3}$ to have negative real part in order for the disease-free equilibrium to be stable and as well to be asymptotically stable.
From $\lambda_{3}$ we obtain

$$
\begin{equation*}
\frac{-[(\mu+\sigma)(\gamma+\mu+\psi)-\beta K]}{\mu+\sigma}<0 \tag{3.11}
\end{equation*}
$$

inequality (3.11) becomes

$$
\begin{equation*}
(\mu+\sigma)(\gamma+\mu+\psi)>\beta K \tag{3.12}
\end{equation*}
$$

or

$$
\begin{equation*}
\beta K<(\mu+\sigma)(\gamma+\mu+\psi) . \tag{3.13}
\end{equation*}
$$

Dividing the equation (3.13) by $(\mu+\sigma)$ we obtain

$$
\begin{equation*}
\frac{\beta K}{(\mu+\sigma)}<(\gamma+\mu+\psi) . \tag{3.14}
\end{equation*}
$$

The inequality (3.13) 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 $(\beta K)$ must be less than the total removal rate from both susceptible and infectious classes given by $(\mu+\sigma)(\gamma+$ $\mu+\psi)$. Alternatively, the inequality (3.14) also gives the necessary and sufficient condition for the stability of the disease-free equilibrium state. The sum of the rate of recovery of infectious individuals in the population (i.e the total removal rate from infectious class) must have a lower bound given by $\frac{\beta K}{(\mu+\sigma)}$.

### 3.4 Basic Reproductive Number $\left(R_{0}\right)$ of the Model

The basic Reproductive Number $\left(R_{0}\right)$ is the average number of secondary infections caused by a single infectious individual during their entire infectious lifetime [14, 13, 21]. It helps us to set the threshold in the study of the disease both for predicting its outbreak and for evaluating its control strategies. In the present work, we derived the threshold quantity known as reproductive number ( $R_{0}$ ) from the largest eigenvalue $\lambda_{3}$ of Jacobian matrix corresponding to equilibrium state.

Recall that $\lambda_{1}=-\mu, \lambda_{2}=-(\mu+\sigma)$, and $\lambda_{3}=\frac{\beta K-(\mu+\sigma)(\gamma+\mu+\psi)}{\mu+\sigma}, \forall \beta, \mu, \sigma, \psi, \gamma$, and $K$ are all positive. Hence, $\lambda_{3}$ is the largest eigenvalue in which $R_{0}$ will be derived.
If we let $\lambda_{3}<0$ then, we have

$$
\begin{gather*}
\frac{\beta K-(\mu+\sigma)(\gamma+\mu+\psi)}{\mu+\sigma}<0  \tag{3.15}\\
\frac{\beta K}{(\mu+\sigma)(\gamma+\mu+\psi)}<1 \tag{3.16}
\end{gather*}
$$

Hence, equation (3.16) allows the definition of $R_{0}$ for the $S-I-R$ model as

$$
\begin{equation*}
R_{0}(S, I, R)=\frac{\beta K}{(\mu+\sigma)(\gamma+\mu+\psi)}, \text { where }(\mu+\sigma)(\gamma+\mu+\psi) \neq 0 \tag{3.17}
\end{equation*}
$$

Remark 1. The threshold quantity $R_{0}$, defined in the equation (3.17) is the basic reproduction ratio of infection for the non linear autonomous ordinary differential equations in (2.1) ( $[8,14$, 13, 22]).

Remark 2. Epidemiologically,
(i) if $R_{0}<1$, the occurrence of the disease will decrease.
(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 3.1. The disease-free equilibrium $E^{+}$of the system in Eq. (2.1) is locally asymptotically stable in $\Omega$ if $R_{0}<1$ and unstable if $R_{0}>1$ for $K, \beta, \gamma, \mu, \sigma$ and $\psi$ are all positive.

Proof. . 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

$$
R_{0}=\frac{\beta K}{(\mu+\sigma)(\mu+\gamma+\psi)} .
$$

Using the inequality in Eq. (3.16)

$$
R_{0}<1,
$$

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

$$
R_{0}>1,
$$

$\lambda_{3}$ is positive. Therefore, disease-free equilibrium point $E^{+}$of (2.1) becomes locally asymptotically unstable, the Theorem (3.1) is proven.

In view of remark (2) to our model, we have

## Remark 3.

(i) if $\beta K<(\mu+\sigma)(\mu+\gamma+\psi)$, the occurrence of the measles infection will decrease.
(ii) if $\beta K=(\mu+\sigma)(\mu+\gamma+\psi)$, the occurrence of measles infection will be constant.
(iii) if $\beta K>(\mu+\sigma)(\mu+\gamma+\psi)$ the occurrence of the measles infection will increase. Infection persist, each individual will produces more than one new infected.

Anderson and May [2] stated that, generally in epidemiological modeling if $R_{0}<1$, the disease-free equilibrium will be 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 $E^{+}$) and also 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.

### 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.

Theorem 3.2. The disease-free equilibrium $E^{+}$, of the equilibrium (3.2) is globally asymptotically stable in $\Omega$ if $R_{0} \leq 1$.

Proof. Since $R_{0}<1$, it follows that there exists a small $\epsilon_{0}>0$ such that

$$
\begin{equation*}
\beta\left(\frac{K}{\mu+\sigma}+\epsilon_{0}\right)-(\gamma+\mu+\Psi)<0 \tag{3.18}
\end{equation*}
$$

In view of the first equation in system (2.1), we see that

$$
\frac{d S(t)}{d t} \leq K-(\mu+\sigma) S
$$

which implies that there exists a $t_{0}>0$ such that

$$
\begin{equation*}
S(t) \leq \frac{K}{\mu+\sigma}, \quad \forall t \geq t_{0} \tag{3.19}
\end{equation*}
$$

From (3.19) and the second equation in system (2.1), it follows that

$$
\begin{align*}
\frac{d I(t)}{d t} & \leq \beta\left(\frac{K}{\mu+\sigma}+\epsilon_{0}\right) I-(\gamma+\mu+\Psi) I \\
& =\left[\beta\left(\frac{K}{\mu+\sigma}+\epsilon_{0}\right)-(\gamma+\mu+\Psi)\right] I(t) \tag{3.20}
\end{align*}
$$

In view of (3.18) and (3.20), it is to see that

$$
\begin{equation*}
\lim _{t \longrightarrow \infty} I(t)=0 . \tag{3.21}
\end{equation*}
$$

By (3.21) and the first equation in the system (2.1), it follows that $S(t)$ is asymptotic to the following system

$$
\frac{d S(t)}{d t}=K-(\mu+\sigma) S
$$

Then by the theory of asymptotically autonomous semi flows (see Corollary 4.3 in [52]) implies that

$$
\begin{equation*}
\lim _{t \longrightarrow \infty} S(t)=\frac{K}{\mu+\sigma} \tag{3.22}
\end{equation*}
$$

By (3.21), (3.22) and the third equation in the system (2.1), it follows that $R(t)$ is asymptotic to the following system

$$
\frac{d R(t)}{d t}=\mu R \frac{\sigma K}{\mu+\sigma}
$$

Then by the theory for asymptotically autonomous semi flows (see Corollary 4.3 in [52]) implies that

$$
\begin{equation*}
\lim _{t \longrightarrow \infty} R(t)=\frac{\sigma K}{\mu(\mu+\sigma)} \tag{3.23}
\end{equation*}
$$

By (3.21), (3.22) and (3.23), we complete the proof.

### 3.6 Existence and Uniqueness of Endemic Equilibrium (EE)

Endemic equilibrium state is the state where the disease cannot be totally eradicated but persist in the population. Let the endemic equilibrium be $E^{*}=\left(S^{*}, I^{*}, R^{*}\right)$. Then, the susceptible class $S$, the infectious class, $I$ and the recovered class, $R$, must not be zero at equilibrium state i.e $E^{*}=\left(S^{*}, I^{*}, R^{*}\right) \neq(0,0,0)$. In order to obtain the endemic equilibrium state, one solves equation (2.1)

$$
\begin{align*}
& K-\left(\beta I^{*}+\mu+\sigma\right) S^{*}=0 \\
& \beta S^{*} I^{*}-(\gamma+\mu+\psi) I^{*}=0,  \tag{3.24}\\
& \gamma I^{*}-\mu R^{*}+\sigma S^{*}=0
\end{align*}
$$

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

$$
\begin{align*}
S^{*} & =\frac{(\gamma+\mu+\psi)}{\beta} \\
I^{*} & =\frac{\beta K-(\mu+\sigma)(\mu+\gamma+\psi)}{\beta(\gamma+\mu+\psi)}  \tag{3.25}\\
R^{*} & =\frac{\beta K \gamma-(\mu+\gamma+\psi)(\gamma \mu-\sigma \mu-\sigma \psi)}{\beta \mu(\gamma+\mu+\psi)} .
\end{align*}
$$

The vector representation of solution in equation (3.25) is

$$
\begin{align*}
E^{*}=\left(S^{*}, I^{*},\right. & \left.R^{*}\right)=\left(\frac{(\gamma+\mu+\psi)}{\beta}\right. \\
& \left.\frac{\beta K-(\mu+\sigma)(\mu+\gamma+\psi)}{\beta(\gamma+\mu+\psi)}, \frac{\beta K \gamma-(\mu+\gamma+\psi)(\gamma \mu-\sigma \mu-\sigma \psi)}{\beta \mu(\gamma+\mu+\psi)}\right) . \tag{3.26}
\end{align*}
$$

Representing the endemic equilibrium state in term of reproductive number $\left(R_{0}\right)$ we obtain

$$
E^{*}\left(S^{*}, I^{*}, R^{*}\right)=\left\{\begin{array}{l}
S^{*}=\frac{K}{(\mu+\sigma) R_{0}}  \tag{3.27}\\
I^{*}=\frac{(\mu+\sigma)\left(R_{0}-1\right)}{\beta} \\
R^{*}=\frac{\gamma R_{0}(\mu+\sigma)^{2}\left(R_{0}-1\right)+\beta \sigma K}{\beta \mu R_{0}(\mu+\sigma)}
\end{array}\right.
$$

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 behavior of the endemic equilibrium $E^{*}$ for the model Eq. (2.1), where we use the same Lyapunov functions used in $[1,6,24,30,31,32,8]$, to demonstrate the global stability of the endemic equilibrium of $S-I-R$ model. We have the following results.

Theorem 3.3. If $R_{0}>1$, the unique endemic equilibrium $E^{*}$ is globally asymptotically stable on $\Omega$.

Proof. Consider the following Lyapunov function candidate

$$
\begin{equation*}
V(S, I, R)=S-S^{*} \ln S+C_{1}\left(I-I^{*} \ln I\right)+C_{2}\left(R-R^{*} \ln R\right) \tag{3.28}
\end{equation*}
$$

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

$$
\frac{d V}{d t}=\frac{\partial V}{\partial S} \frac{d S}{d t}+\frac{\partial V}{\partial I} \frac{d I}{d t}+\frac{\partial V}{\partial R} \frac{d R}{d t}
$$

which becomes

$$
\begin{align*}
& \quad \dot{V}(S, I, R)=\left(1-\frac{S^{*}}{S}\right) \dot{S}+C_{1}\left(1-\frac{I^{*}}{I}\right) \dot{I}+C_{2}\left(1-\frac{R^{*}}{R}\right) \dot{R} .  \tag{3.29}\\
& =\left(1-\frac{S^{*}}{S}\right)(K-(\beta S I+\mu S+\sigma S))+C_{1}\left(1-\frac{I^{*}}{I}\right)(\beta S I-(\gamma I+\mu I+\psi I))+  \tag{3.30}\\
& C_{2}\left(1-\frac{R^{*}}{R}\right)(\gamma I-\mu R+\sigma S) .
\end{align*}
$$

By considering equation (2.1) at endemic equilibrium, we have

$$
\begin{gathered}
K=\beta S^{*} I^{*}+\mu S^{*}+\sigma S^{*} \\
\beta S^{*}=\gamma+\mu+\psi \\
\mu=\frac{\gamma I^{*}+\sigma S^{*}}{R^{*}} .
\end{gathered}
$$

Substituting the values of $K, \beta S^{*}$ and $\mu$ into equation (3.30), after simplification, we have

$$
\begin{align*}
& \dot{V} \leq-\left(\frac{(\mu+\sigma)}{S}\left(S-S^{*}\right)^{2}+\beta\left(1-\frac{S^{*}}{S}\right)\left(S I-S^{*} I^{*}\right)+A \beta\left(I^{*}-I\right)\left(S-S^{*}\right)\right. \\
& \left.+B\left(\gamma I^{*}+\sigma S^{*}\right)\left(\frac{R}{R^{*}}-1\right)+B(\gamma I+\sigma S)\left(\frac{R^{*}}{R}-1\right)\right) \tag{3.31}
\end{align*}
$$

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 $(S, I, R) \in \Omega: \dot{V}=0$ 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 $\Omega$.

## 4 Conclusion and Recommendation

### 4.1 Conclusion

This work analyzed mathematical models to study the transmission and recovery dynamics of measles infections using deterministic of ordinary differential equations (ODEs) to discussed its boundedness and stability. In this paper we have established the existence of non-negative solutions of the mathematical model. Having shown that the disease-free equilibrium point $\left(E^{+}\right)$ is asymptotically stable if and only if all the eigenvalues have negative real part, which serve as the necessary and sufficient condition using Routh-Hurwitz theorem. It is also shown that the disease-free equilibrium point $\left(E^{+}\right)$will be locally asymptotically stable when the basic reproduction number $R_{0}<1$, otherwise, unstable. The model has a unique endemic equilibrium which is locally asymptotically stable if $R_{0}>1$. Global asymptotic stability of both disease-free equilibrium and endemic equilibrium are establish using second Lyapunov's method. It is further shown that the product of total contraction and total breakdown of infectious class from the model must be less than the total removal rate from both susceptible and infectious classes. That is $\beta K<\theta(\mu+\sigma)$

### 4.2 Recommendations

(i) there should be a vaccine therapy against measles infections;
(ii) and also that future research work should incorporate vaccination and treatment on different data of measles infection from different regions;
(iii) prompt and timely intervention of Government, and public enlightenment should be made, so people in the country could get educated about the wide spread nature of the measles infection;
(v) $R_{0}$ can be reduced through a decrease in the contact rate, $\beta$ through behavior change, medication or through an increase in the recovery rate, $\gamma$
(vi) The principles of control and prevention should be geared towards attacking the source of the disease causing organism, interrupting the transmission cycle and protecting the susceptible host.

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