International Journal of Scientific Research in Mathematical and Statistical Sciences **Research Paper**
Vol.6, Issue.2, pp.01-08, April (2019) **E-ISSN:** 2348-4519
DOI: https://doi.org/10.26438/ijsrmss/v6i2.18

A Mathematical Model for the Transmission of Measles with Passive Immunity

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Available online at: www.isroset.org

Received: 22/Feb/2019, Accepted: 14/Mar/2019, Online: 30/Apr/2019

Abstract- A mathematical model of the transmission dynamics of measles incorporating passively immune class is developed. The dynamics of the disease are expressed with the help of a set of ordinary differential equations. The model is analysed qualitatively and quantitatively. Equilibrium points of the determined and their stability analysed. From the study it has been shown that for a stable disease-free equilibrium the reproduction number is less than **1** and more than **1** for an unstable disease-free equilibrium. The spread of the disease in the population is dependent on the level of between the susceptible individuals and the infected individuals. The rate at which passive immunity in an infant is lost also has a great impact on the spread of the disease.

Keywords— *Equilibrium points, disease free equilibrium, measles, basic reproduction number, stability.*
AMS Subject classification: *Primary 92D30, 91B74, Secondary 37M05, 34A34, 34D99.*

I. INTRODUCTION

Measles is an infectious and highly contagious viral respiratory disease. It spreads through coughing and/or sneezing, close personal contact of the susceptible individual with an infected person or direct contact of the susceptible individuals with nasal or throat secretions from an infected individual. The infectious secretions remain in the air or on infected surfaces for up to two hours after an infected person sneezes or coughs. Worldwide, measles is the fifth leading cause of death among 'under-five' children with 109,638 reported deaths in 2017 [1]. There is no known specific treatment for measles. However, infection and subsequent recovery confers permanent and lifelong immunity on an individual [2, 3]. Because of the health burden and the high death rates the disease causes, it is important to develop effective control strategies.

Getting vaccinated is the best way to prevent measles [4]. Although the vaccines, which are available in two doses (MCV1 & MCV2), have an effectiveness of between 90 and 95%, only a small percentage of children in the Kenya and the sub-Saharan Africa receive MCV2 [4, 5]. Some of the challenges facing vaccination efforts include nomadic lifestyles [6], terrain, religious beliefs, accessibility to health facilities [7], conflicts and other logistical challenges. A mathematical model is a powerful tool in the analysis of measles transmission dynamics. Models can also be used to simulate possible scenarios in case of an epidemic. Mathematical models help in explaining a system, showing how different components affect the model and making important predictions about the systems behavior.

This study incorporates the passive immunity compartment in the transmission dynamics in order to give a proper insight into the measles dynamics.

The rest of the paper is organized as follows. In section 2, we formulate the model and illustrate some of its basic properties. In section 3, we demonstrate positivity, determine the points of equilibria and perform stability analysis of the system. In section 4, numerical results of the analysis are presented. Section 5 concludes the paper.

II. FORMULATION OF THE MATHEMATICAL MODEL OF MEASLES

In this section, we describe and develop the model of measles dynamics based on the several assumptions.

2.1. MODEL ASSUMPTIONS

In developing the model, the following assumptions were made;

1. Individuals get into the system by birth only.

2. All new born infants acquire passive immunity from their mothers hence they are disease free.
3. All the variables and parameters used in the model are non-negative.
4. All recovered individuals acquire permanent immunity.
5. There is free interaction within the population.

2.2. MODEL EQUATIONS

The model starts with the birth of infants who enter the passive immune class, M, at a rate b of the total population, N. Compartment M diminishes by φM, bpN and μM due to immunity loss, successful vaccination and natural deaths respectively at the appropriate rates φ, bp and μ respectively. Children who lose passive immunity, enters the susceptible class, S. This is the class of individuals who can get infected when there is sufficient contact with an infected individual or secretion. The number of individuals in S diminishes by βS and μS due to Measles exposure and natural deaths, at the rates β and μ respectively and increases by φM due to loss of passive immunity at a rate φ. The susceptible individuals enter the class of exposed individuals, E. In this class, are the people who are not infectious but are in latent period. The compartment E is increased by βS as a result of susceptible individual(s) coming into contact with infectious individual(s) at a rate β. This population is decreased by αE and μE due to individuals entering the infective class, I, and dying naturally at rates α and μ respectively. The compartment I of the infected individuals increases by αE as a result of infection at rate α, and diminishes by γI, μI and σI due to natural recovery, natural deaths and infection-induced deaths, at rates γ, μ and σ respectively. Finally, individuals enter the recovered class, R when they recover from the disease. The number of individuals in compartment R increases by γI and bpN at rates γ and bp respectively. This is because of recovery from the disease and successful vaccination. The population in R reduces by μR as a result of natural deaths. The total population with respect to all the compartments is given by:

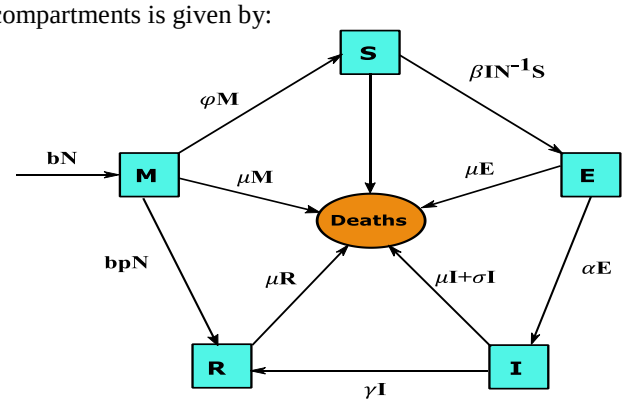


Figure 1: Schematics depicting transitions between different compartments, transmission and mortalities rates (Authors 2019)

The mathematical model is described by the following system of equations

$$\begin{aligned} dM/dt &= bN - bpN - (\varphi + \mu)M \\ dS/dt &= \varphi M - \beta IS/N - \mu S \\ dE/dt &= \beta IS/N - (\mu + \alpha)E \\ dI/dt &= \alpha E - (\mu + \sigma + \gamma)I \\ dR/dt &= bpN + \gamma I - \mu R \end{aligned} \qquad (1)$$

$dN/dt = (b - \mu)N - \sigma I$ describe the rate of change in the total population.

The initial conditions of model (1) are: $M(0) = M_0 > 0$, $S(0) = S_0 \geq 0$, $E(0) = E_0 \geq 0$, $I(0) = I_0 \geq 0$, $R(0) = R_0 \geq 0$.

The population in each compartment can be scaled by the total population N to get the fraction of the respective population. The scaling is done using the following transformations $m = M/N$, $s = S/N$, $i = I/N$, $e = E/N$ and $r = R/N$.

This scaling transforms the system of equations in (1) to:

$$\begin{aligned} dm/dt &= b - bp - \varphi m - bm + \sigma im \\ ds/dt &= \varphi m - \beta si - bs + \sigma is \\ de/dt &= \beta si - \alpha e - be + \sigma ie \\ di/dt &= \alpha e - \sigma i - \gamma i - bi + \sigma i^2 \\ dr/dt &= bp - \gamma i - br + \sigma ir \end{aligned} \qquad (3)$$

III. MODEL ANALYSIS

In this section, the scaled model (3) is analysed qualitatively to help us study the dynamics of the overall system.

3.1. POSITIVITY AND INVARIANT REGION

The system of equations in model (3) are solved to obtain $m(t) \geq m(0)e^{-(\varphi+b-\sigma i)t} \geq 0$, $s(t) \geq s(0)e^{-(\beta i+b-\sigma i)t} \geq 0$, $e(t) \geq e(0)e^{-(\alpha+b-\sigma i)t} \geq 0$ and $i(t) \geq i(0)e^{-(\sigma+\gamma+b-\sigma i)t} \geq 0$. From these solutions, all the variables are non-negative which is consistent with our expectation since the variables represent human beings.

On the other hand, if we let the set $\Gamma = \{(m, s, e, i,) \in \mathbb{R}_+^4 : 0 \leq m + s + e + i \leq 1\}$ and $(m, s, e, i, r)(0) \geq 0 \in \Gamma$, then the solution set $(m, s, e, i, r)(t)$ of equations is positive for all $t > 0$ which makes biological sense.

3.2. BASIC REPRODUCTION NUMBER (R_0)

Basic reproduction number, R_0, is a significant threshold in determining whether the disease dies out or persists in the population. It is a measure of the speed with which a disease spreads through a population. In this study, we compute R_0,

using the next generation matrix method as formulated by [8] in which we determine the dominant eigenvalue of the steady state Jacobian matrix of the model after linearization. This is done by taking the column matrix of new infections getting in compartment E and I from S and denoting it as X, that is;

$$X = (\beta si \quad 0)^T$$

then taking the column matrix of transfer of individuals into compartment E and I by any other means and denoting it as Y , that is;

$$Y = \begin{pmatrix} (\alpha + b - \sigma i)e \\ -\alpha e + (\sigma + \gamma + b - \sigma i)i \end{pmatrix}$$

The partial derivatives of X and Y with respect to e and i gives the square matrix F and V respectively. Thus,

$F = \begin{pmatrix} 0 & \beta s \\ 0 & 0 \end{pmatrix}$ and $V = \begin{pmatrix} \alpha + b - \sigma i & -\sigma e \\ -\alpha & \sigma + \gamma + b - 2\sigma i \end{pmatrix}$

At the disease-free equilibrium (DFE), $V = \begin{pmatrix} \alpha + b & 0 \\ -\alpha & \sigma + \gamma + b \end{pmatrix}$ and $F = \begin{pmatrix} 0 & \frac{\beta\varphi(1-p)}{(\varphi+b)} \\ 0 & 0 \end{pmatrix}$

Evaluating F and V at disease free equilibrium and then computing the dominant eigenvalue of FV^{-1} we obtain R_0 as

$$R_0 = \frac{\alpha\beta\varphi(1-p)}{(\alpha+b)(\sigma+\gamma+b)(\varphi+b)} \qquad (4)$$

3.3. EXISTENCE AND STABILITY OF DISEASE-FREE EQUILIBRIUM, DFE

The disease-free equilibrium E_0^* is obtained by equating the system of equations (3) to zero and solving it in the absence of infection i.e. $e = 0$ and $i = 0$. The disease free equilibrium of our model is given by; $E_0^*(m^*, s^*, e^*, i^*) =$ $(b(1-p)/(\varphi+b), \varphi(1-p)/(\varphi+b), 0, 0)$ where m^*, s^*, e^*, i^* and r^* are proportions of passively immunes, susceptible, exposed, infected and recovered in absence of the disease.

The Jacobian obtained from equation (3) at the DFE is

$$J_0 = \begin{pmatrix} -(\varphi+b) & 0 & 0 & \sigma\varphi(1-p)/(\varphi+b) \\ \varphi & -b & 0 & \{\beta b(1-p) + b(\varphi+b) + \sigma\varphi(1-p)\}/(\varphi+b) \\ 0 & 0 & -(\alpha+b) & \beta\varphi(1-p)/(\varphi+b) \\ 0 & 0 & \alpha & -(\sigma+\gamma+b) \end{pmatrix}$$

To simplify the analysis of the Jacobian, we use the transformations $x = (\varphi + b)$, $y = (\alpha + b)$, $z = (\sigma + \gamma + b)$, the expression for R_0 which yields the eigenvalues $\lambda_1 = -x$, $\lambda_2 = -b$, $\lambda_3 = \frac{-(yx+zx)-\sqrt{(yx+zx)^2-4x^2yz(1-R_0)}}{2x}$ and $\lambda_4 = \frac{-(yx+zx)+\sqrt{(yx+zx)^2-4x^2yz(1-R_0)}}{2x}$. While λ_1, λ_2 and λ_3 have negative real parts, λ_4 will be negative if and only if $-4x^2yz(1-R_0) < 0$. Therefore, the model is locally stable whenever $R_0 < 1$ and unstable if $R_0 > 1$.

3.4. EXISTENCE AND STABILITY OF ENDEMIC EQUILIBRIUM, EE

The EE point E_1^*, is a steady state solution that shows that the disease does not die in the population instead it persists. From equations (3) the EE point is given by setting the respective derivatives to zero. The endemic equilibrium point of the model $E_1^* = (m^*, s^*, e^*, i^*)$ where,

$$m^* = \frac{b(1-p)}{(\varphi+b-\sigma)}$$

$$s^* = \frac{(\gamma+b)(\alpha+b-\sigma)}{\beta\alpha}$$

$$e^* = \frac{b(1-p)}{(\alpha+b-\sigma)} - \frac{b(1-p)(b+\sigma)}{(\varphi+b-\sigma)(\alpha+b-\sigma)} + \frac{(\sigma-b)(\gamma+b)}{\beta\alpha}$$

$$i^* = \frac{b\alpha(1-p)}{(\gamma+b)(\alpha+b-\sigma)} - \frac{b\alpha(1-p)(b+\sigma)}{(\varphi+b-\sigma)(\gamma+b)(\alpha+b-\sigma)} + \frac{(\sigma-b)}{\beta}$$

The Jacobian obtained from equation (3) at the EE is;

$$J_{(E_1^*)} = \begin{pmatrix} -J_{11} & 0 & 0 & J_{14} \\ J_{21} & -J_{22} & 0 & -J_{24} \\ 0 & J_{32} & -J_{33} & J_{34} \\ 0 & 0 & J_{43} & -J_{44} \end{pmatrix}$$

where

$$J_{11=}(\varphi+b)+\sigma\left[\frac{b\alpha(1-p)}{(\gamma+b)(\alpha+b-\sigma)}-\frac{b\alpha(1-p)(b+\sigma)}{(\varphi+b-\sigma)(\gamma+b)(\alpha+b-\sigma)}+\frac{(\sigma-b)}{\beta}\right]$$

$$J_{22}=(\beta\left[\frac{b\alpha(1-p)}{(\gamma+b)(\alpha+b-\sigma)}-\frac{b\alpha(1-p)(b+\sigma)}{(\varphi+b-\sigma)(\gamma+b)(\alpha+b-\sigma)}+\frac{(\sigma-b)}{\beta}\right]+b)$$
$$+\sigma\left[\frac{b\alpha(1-p)}{(\gamma+b)(\alpha+b-\sigma)}-\frac{b\alpha(1-p)(b+\sigma)}{(\varphi+b-\sigma)(\gamma+b)(\alpha+b-\sigma)}+\frac{(\sigma-b)}{\beta}\right]$$

$$J_{32}=\beta\left[\frac{b\alpha(1-p)}{(\gamma+b)(\alpha+b-\sigma)}-\frac{b\alpha(1-p)(b+\sigma)}{(\varphi+b-\sigma)(\gamma+b)(\alpha+b-\sigma)}+\frac{(\sigma-b)}{\beta}\right]$$

$$J_{33}=(\alpha+b)+\sigma\left[\frac{b\alpha(1-p)}{(\gamma+b)(\alpha+b-\sigma)}-\frac{b\alpha(1-p)(b+\sigma)}{(\varphi+b-\sigma)(\gamma+b)(\alpha+b-\sigma)}+\frac{(\sigma-b)}{\beta}\right]$$

$$J_{14}=\sigma\frac{b(1-p)}{(\varphi+b-\sigma)},\ J_{24}=\left[\beta\frac{(\gamma+b)(\alpha+b-\sigma)}{\beta\alpha}+b\right]-\sigma\frac{(\gamma+b)(\alpha+b-\sigma)}{\beta\alpha}$$

$$J_{34}=\beta\frac{(\gamma+b)(\alpha+b-\sigma)}{\beta\alpha}+\sigma\frac{b(1-p)}{(\alpha+b-\sigma)}-\frac{b(1-p)(b+\sigma)}{(\varphi+b-\sigma)(\alpha+b-\sigma)}+\frac{(\sigma-b)(\gamma+b)}{\beta\alpha}$$

$$J_{44}=(\sigma+\gamma+b)+2\sigma\frac{b\alpha(1-p)}{(\gamma+b)(\alpha+b-\sigma)}-\frac{b\alpha(1-p)(b+\sigma)}{(\varphi+b-\sigma)(\gamma+b)(\alpha+b-\sigma)}+\frac{(\sigma-b)}{\beta}$$

$$J_{21}=\varphi,\ J_{43}=\alpha$$

The eigenvalues were obtained by solving the matrix $J_{(E_1^*)}$ with a characteristic equation

$$A_4\lambda^4+A_3\lambda^3+A_2\lambda^2+A_1\lambda+A_0=0 \qquad (5)$$

Where

$$A_4=1,\ \ A_3=J_{11}+J_{22}+J_{33}+J_{44}>0,\ A_2=J_{11}J_{22}+J_{11}J_{44}+J_{11}J_{33}+J_{22}J_{44}+J_{22}J_{33}+J_{33}J_{44}-J_{43}J_{34}$$
$$A_1=J_{11}J_{22}J_{44}+J_{11}J_{22}J_{33}+J_{11}J_{33}J_{44}+J_{22}J_{44}J_{33}+J_{32}J_{43}J_{24}-J_{11}J_{34}J_{43}-J_{22}J_{34}J_{43}$$
$$A_0=J_{11}J_{22}J_{33}J_{44}+J_{43}J_{11}J_{33}J_{24}-J_{21}J_{32}J_{43}J_{14}-J_{43}J_{11}J_{22}J_{34}$$

To determine the signs and roots of equation (5), we use Routh-Hurwitz criterion and Descartes rule i.e. given the polynomial $P(\lambda)=A_4\lambda^4+A_3\lambda^3+A_2\lambda^2+A_1\lambda+A_0$, where the coefficients of A_i are real constants, $i=1,2,3,4$; we define the n Hurwitz matrices equal to the number of sign changes A_i of the characteristic polynomial as;

$$F_0=1,\ F_1=A_1,\ F_2=\begin{vmatrix}A_1 & 1\\ 0 & A_2\end{vmatrix},\ F_3=\begin{vmatrix}A_1 & 1 & 0\\ A_3 & A_2 & A_1\\ 0 & 0 & A_3\end{vmatrix},F_4=\begin{vmatrix}A_1 & 1 & 0 & 0\\ A_3 & A_2 & A_1 & 1\\ 0 & A_4 & A_3 & A_2\end{vmatrix}$$

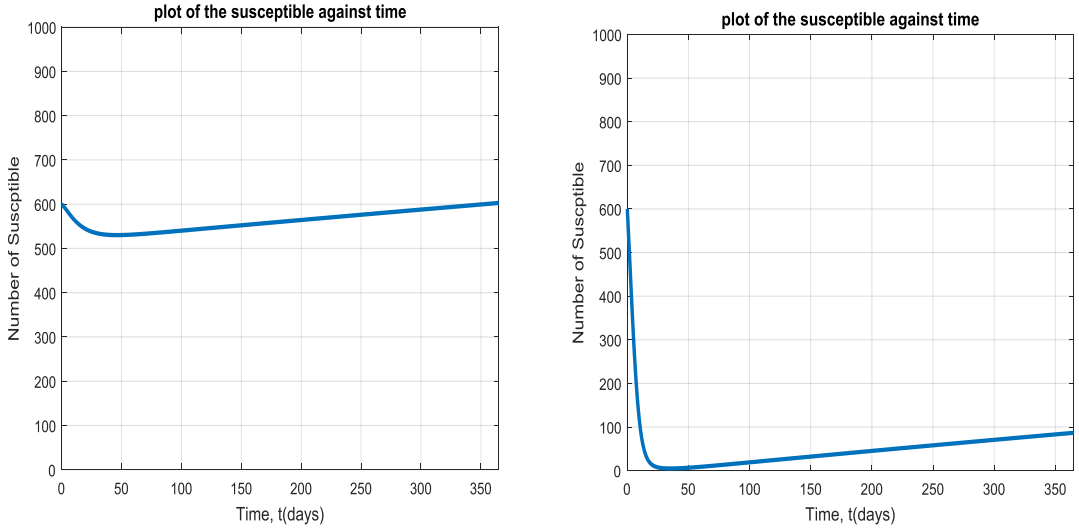
Where $A_i=0$ if $i>n$. All the roots of the polynomial are negative or have negative real parts if and only if the determinants of all the Hurwitz matrices are positive. From our equation 5, It is clear that A_4, A_3, A_2, A_1 and A_0 are all positive, $\det(F_1)=A_1>0$, $\det(F_2)=A_1A_2>0$, $\det(F_3=A_1A_2A_3-A_3^2)$ and $\det(F_4)=A_1A_2A_3-A_1^2A_4-A_3^2$.

Since all the determinants are positive it implies that all the eigenvalues of the Jacobian matrix have negative real part. Hence the EE is stable when $R_0>1$.

IV. NUMERICAL RESULTS AND SIMULATIONS

In this section, we employ numerical techniques and MATLAB's built-in $ode45$ solver function to compute and simulate the formulated mathematical model. The

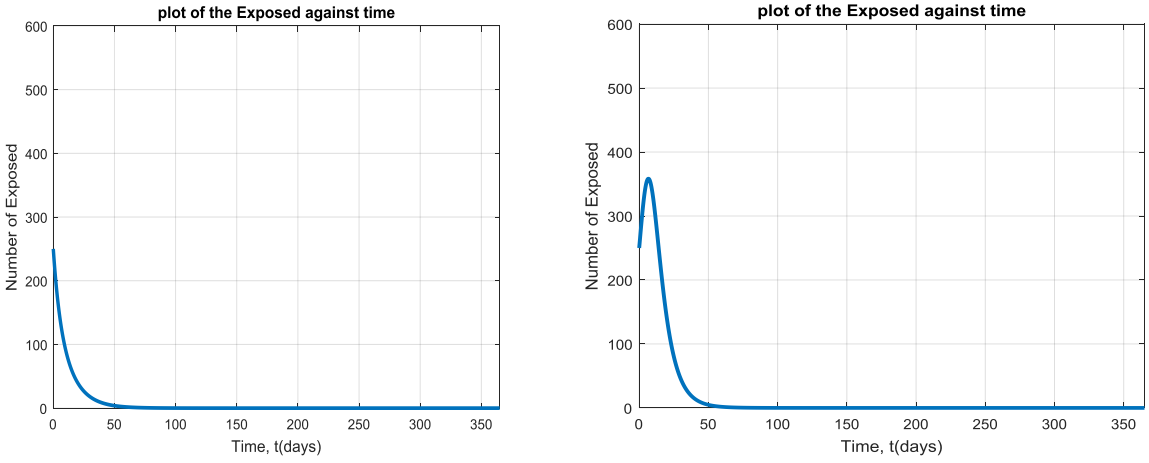
$ode45$ solver evaluates the system of differential equations using the fourth-order Runge-Kutta method for ODEs. The initial population sizes and conditions are $M = 800$, $S = 600$, $E = 250$, $I = 100$ and $R = 50$ while the parameter values used in the simulation are indicated in Appendix A. The results from the numerical simulations are shown in Figures $5.1 - 5.4$.



$a)\ \beta = 0.09091$ $b)\ \beta = 1.6667$

Figure 5.1: Simulations of susceptible individuals.

In figure 5.1 the number of the susceptible decreases for around the first twenty days and then it starts increasing. This decrease could be due to increase in recovery due to vaccination, since when individuals are vaccinated, they acquire permanent immunity upon recovery. It could also be attributed to those susceptible entering into the exposed class. After around thirty days, the number of susceptibles starts increasing due to the number of individuals from the partial immune class entering to the susceptible class who are not vaccinated increasing.

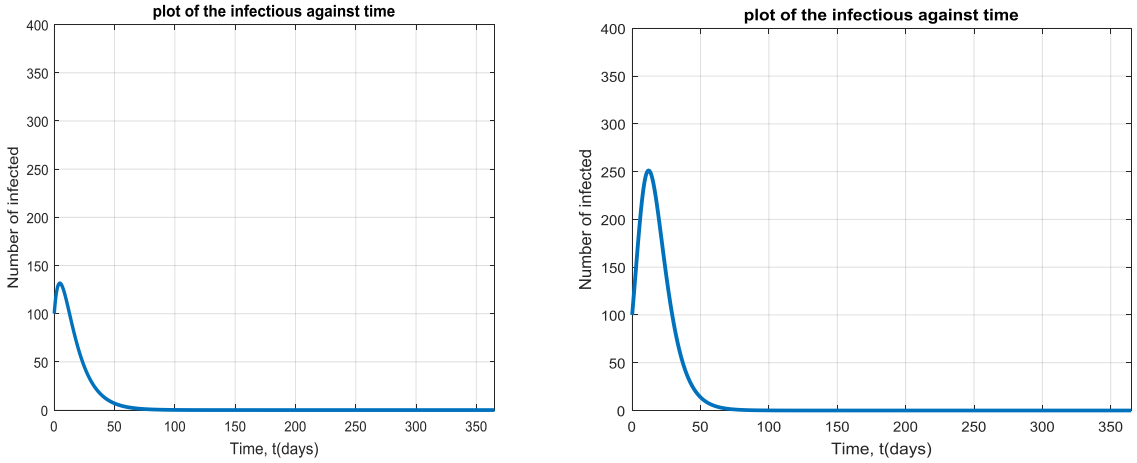


$a)\ \beta = 0.09091$ $b)\ \beta = 1.6667$

Figure 5.2: Simulations of exposed individuals

In figure 5.2, there is a rapid decrease in the population of the exposed individuals in the first few days. This rapid decrease might be as a result of treatment of early detected cases as well as transition from exposed to the infectious states. However, in figure 5.2 (b) the number of infected individual increases slightly then

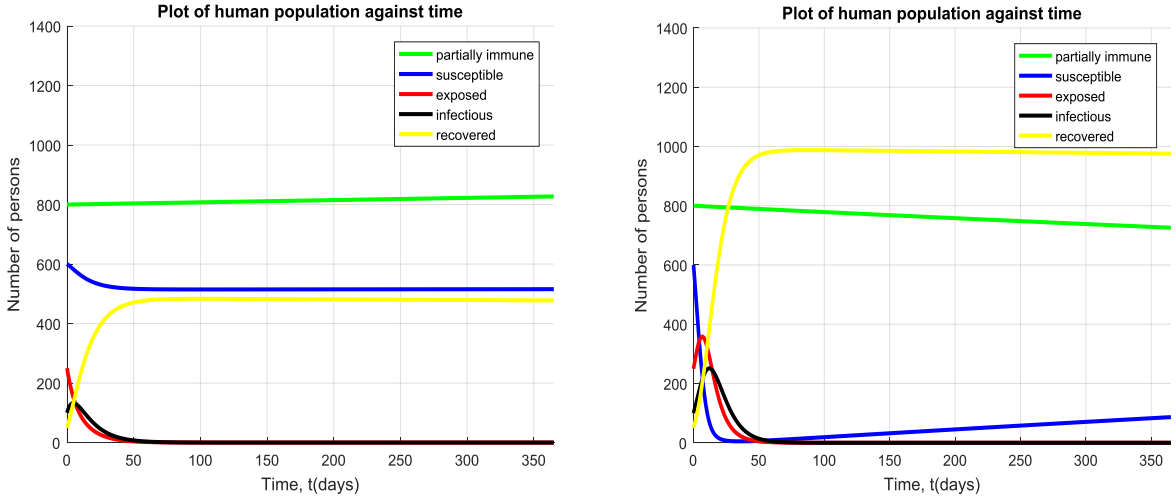
there is a significant drop between the tenth and fortieth day. The increase in the infected individuals could have been due to more susceptible individuals becomes exposed due to high contact rate with those infected. The decrease might be as a result of treatment of early detected cases as well as transition from exposed to the infectious states.



$a)\ \beta = 0.09091$ $b)\ \beta = 1.6667$

Figure 5.3: Simulations of infectious individuals**.**

In figure 5.3, the number of infected individual increases slightly then there is a significant drop between the tenth and fortieth day. The increase in the infected individuals could have been due to the exposed individual becoming infectious. The decrease in infection could be due to the infected recovering from the disease as a result of treatment.



$a)\ \beta = 0.09091$ $b)\ \beta = 1.6667$

Figure 5.4: Simulation of the human population

The number of recovered individual increases steadily and then becomes constant as indicated in figure 5.4. This could be due to the number of individuals who were successively vaccinated after birth increasing as well as the disease being detected and treated early, leading to recovery. The number of individuals with passive immunity is increasing slightly in (a), while in (b), it is decreasing with time. The increase in (a) could be due to the increase in the number of new born healthy babies in the compartment M who are successively vaccinated and hence have a permanent immunity, while the decrease in (b) could be due to high number of the individuals becoming susceptible

after losing their partial immunity and yet they are in high contact rate with the infected.

V. DISCUSSION AND CONCLUDING REMARKS

In this study we developed a system of differential equations to model diverse dynamics of measles transmission. To accomplish this, a five compartmental model was developed. In our analysis, we derived R_0 mathematically and proved that $R_0 < 1$ for a stable equilibrium point; whereas is $R_0 > 1$ in the case of unstable equilibrium point. The numerical simulations showed that the spread of measles in the population depends on how the susceptible individuals come into contact with the infected people. From Fig. 5.4, it is evident that the rate at which passive immunity is lost is directly proportional to the contact rates. It has also been shown that the loss of the passive immunity the infants get from their mothers has an impact on the spread of the disease. In this study, measles vaccination and treatment of the infected individuals have proved to be very vital in its prevention and eradication.

ACKNOWLEDGMENT

The authors wish to sincerely thank the two anonymous reviewers for the invaluable comments and suggestions that improved our manuscript.

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APPENDIX: DATA FOR BASELINE PARAMETER VALUES

In this appendix we show the data and explanation for the parameter values of the model and the references for the values.

Table 1: Parameter values used in simulating the model for the transmission of measles with passive immunity

Parameter description	Symbol	Dimension	Value	Reference
Birth rate	b	$humans \times year^{-1}$	0.04015	[9]
Mortality rate	μ	$humans \times year^{-1}$	0.0165	[9]
Contact rate	β	day^{-1}	$0.09091, 1.6667$	[10,11]
Infectious rate	α	day^{-1}	0.125	[11]
Recovery rate due to treatment	γ	day^{-1}	0.14286	[11]
Rate of mortality due to measles	σ	$year^{-1}$	0.00246	[5]
Passive immunity loss rate	φ	$year^{-1}$	0.012045	[12]
Proportion of individuals successfully vaccinated at birth.	p	$year^{-1}$	0.028105	[5]

