NIPES

Journal of Science and Technology Research

Journal homepage: www.nipesjournals.org.ng

Mathematical Modelling on Transmission Dynamics of Measles

[1]James Ibrahim Galadima, [2]Danjuma Abubakar, [3]Ayodele Abraham Agboluaje*

[1,2,3]Department of Mathematical Sciences, Faculty of Natural Sciences,
Ibrahim Badamasi Babangida University, Lapai, Postcode: 911001, Nigeria
*****Corresponding Author: E-mail: ayo_dele1960@yahoo.com Mobile phone number: +2348060533502

ARTICLE INFORMATION

Article history:
Received 04 March 2019
Revised 10 March 2019
Accepted 20 March 2019
Available online 5 April 2019

Keywords:
Disease free equilibrium Herd immunity level Immunized population ODEs MSEIR

ABSTRACT

In this work, deterministic model has been used to construct a transmission dynamics of measles using five compartmental models namely; MSEIR. The Disease Free Equilibrium (DFE) point effective reproduction number and the basic reproduction number for the model were obtained. The simulations of the ordinary differential equation (ODEs) and the reproduction number were established. Simulations of different variables of the model have been performed. The sensitivity analysis of different embedded parameters revealed that the proportion of the immunized population exceeded the herd immunity level of measles. Therefore, the disease cannot persist in the population.

1. Introduction

Over the past one hundred years, mathematics has been used to understand and predict the spread of diseases and relating important public health questions to the basic transmission parameters. From prehistory to the present day, diseases have been a source of fear and superstition. A comprehensive picture of diseases dynamics requires varieties of mathematical tools, from model creation to solving differential equations to statistical analysis [1]. Although, mathematics has been so far quite well in dealing with epidemiology, there is no denying of the fact that there are certain factors which still lack proper mathematics. Infection diseases pose a great challenge to both humans and animals worldwide according to [2]. Control and prevention are therefore important tasks both from humane and economic point of views. Efficient intervention hinges on complete understanding of disease transmission and persistence. Measles is an infectious disease highly contagious through person- to - person transmission mode, with greater than 90% secondary attack rate among susceptible persons as declared by [1]. It is the first and worst eruptive fever, occurring during childhood. Measles, also known as Rubeola that is highly infectious illness are caused by the rubeola virus. Measles is an endemic disease; meaning it is continually present in a community and many people develop resistance. If measles enters an area where the people have never been exposed to it, the result can be devastating. Measles symptoms invariably include fever, cough, coryza and conjunctivitis. Symptoms usually appear about nine to eleven days the after infection.

Measles disease is caused by infection with the rubeola virus and is transmitted by the respiratory route; it is a highly contagious virus that lives in the nose and throat mucus of an infected person [3]. It is transmitted by coughing, sneezing or by direct contact with contaminated respiratory secretions. After an incubation time of almost two weeks, disease starts with a prodromal phase of fever, cough, and coryza. A few days later a generalized maculopopular skin rash appears, often in

combination with conjunctivitis. Measles is associated with transient but profound immuno suppression, resulting in an increased susceptibility to opportunistic infections. This often leads to complications like pneumonia, diarrhea, and otitis media, which are the most important determinants of measles morbidity and mortality. The disease is also associated with the induction of strong measles virus specific humeral and cellular immune responses, resulting in lifelong immunity. Measles virus can live for up to two hours in an airspace where the infected person coughed or sneezed. If other people breathe the contaminated air or touch the infected surface and then touch their eyes, noses, or mouths, they can become infected too [3].

Measles is so contagious that if one has it, ninety percent of people close to that person who are not immuned will also become infected. The infected person is contagious for four days before the rash appears, and continues for about four to five days afterwards. It often confers lifelong immunity from further attacks. Measles virus infects the intestinal tracts, respiratory tracts multiple organ systems and targets epithelial and white blood cells, including monocytes, macrophages and tlymphocytes. Many infected children may subsequently suffer blindness, deafness or impaired vision. It may cause pregnant woman to give birth prematurely, or have a low-birth-weight baby. Measles is an infectious disease highly contagious through person-to- person transmission mode, with more than ninety percent secondary attack rates among susceptible persons. It is the first and worst eruptive fever occurring during childhood. However, person who had measles before does not become infected again [3]. Worldwide, measles vaccination has been very effective, preventing an estimated eighty million cases and more than four million deaths annually. Although global incidence has been significantly reduced through vaccination, measles remains an important public health problem. Since vaccination coverage is not uniformly high worldwide, measles stands as the leading vaccine-preventable killer of children worldwide. Measles is estimated to have caused six hundred and fourteen thousand global deaths annually in 2002, with more than half of measles deaths occurring in sub-Sahara Africa [4]. Measles occurs every year in some part of Nigeria, but the outbreak is more severe in alternate years. The epidemic begins in the middle of the dry season, and declines with the onset of the wet season. In this problem of measles transmission, a deterministic compartmental mathematical model is used to describe the transmission dynamics.

One of the earliest written description of measles as a disease was provided by an Arab physician in the 9th century who described differences between measles and smallpox in his medical notes. A Scottish physician Francis Home demonstrated in 1757 that measles was caused by an infectious agent present in the blood of patient. In 1954 the virus that causes measles was isolated in Boston, Massachusetts by John F. Enders and Thomas C. Peebles. Before measles vaccine discovery, nearly all children got measles by the time they were about 15 years of age [3]. Considered mathematical modelling on the control of measles by vaccination using S: susceptible E: exposed I: infected T: treated and R: recovered (SEIR) model and used it to show the control of measles by vaccination according to [2]. The study recommended that introduction of mass vaccination program can be used for improvement in early detection of measles cases to minimize transmission [5, 6]. Considering the recurrent infection and vaccination for the transmission of measles disease using the mathematical model introduced by [7]. They presented the disease free equilibrium with its stability. The discussion of the relation to the basic reproduction number and vaccination reproduction number was enumerated. The vaccination is able to prevent the disease from spreading from numerical examples. $Rv>1$ implies that the disease will persist.

Infectious diseases have been of great concern to humanity for centuries with the threat of biological weapon whose research is lately concerned about microorganism and infectious diseases. We have great motivation to understand the spread and control of this disease and their

infectious characteristics. Therefore models are used in this research work to prevent and control the spread of one of the disease called measles using the five compartments known as M: immunized S: susceptible E: exposed I: infected T: treated and R: recovered (MSEIR) model.

2. Materials and Method

2.1. Mathematical Model for the Transmission Dynamics of Measles

2.1.1. Model Formation and Diagram

From Figure 1, the total population is divided into a non-intersecting compartments consisting of the M: immunized S: susceptible E: exposed I: infected T: treated and R: recovered, denoted by M, S, E, I, R respectively.

The immunized (M) are the people who are being immunized, the susceptible (S) are people that have never come into contact with measles, the exposed (E) are people who have come into contact with the disease but are not yet fully infectious, that is they can infect but with a lower probability when compared with infected, The infected (I) are people who have become fully infectious and are able to transmit the disease from any contact with susceptible, and the recovered
(R) are people who have recovered from the disease.

The corresponding mathematical equation of the formulation of the model schematic diagram below can be described by a system of ordinary differential equations given as in Equations system (1-5).

$$\frac{dM}{dt} = b\theta - (\mu + \varphi)\mathrm{M} \tag{1}$$

$$\frac{dS}{dt} = \pi + b(1-\theta) + \varphi M + \emptyset R - \beta_1(1-\rho)IS - \beta_1\rho IS - \mu S \tag{2}$$

$$\frac{dE}{dt} = \beta_1(1-\rho)IS + \gamma_2 I - (\mu + \gamma_1 + \sigma)E \tag{3}$$

$$\frac{dI}{dt} = \beta_1\rho IS + \gamma_1 E - (\mu + \delta + \alpha + \gamma_2)I \tag{4}$$

$$\frac{dR}{dt} = \sigma E + \alpha I - (\emptyset + \mu)R \tag{5}$$

2.1.2. Basic assumptions of the model

The following assumptions were taken into account of the model construction:
(1) Only susceptible migrants are recruited into the population
(2) The force of infection for fast and slow progression of measles are given as $\beta_1\rho IS$ and $\beta_1(1-\rho)IS$ respectively.
(3) The human population is homogeneous and dependant on time.
(4) Natural death rate is assumed to be equal for all subpopulation.

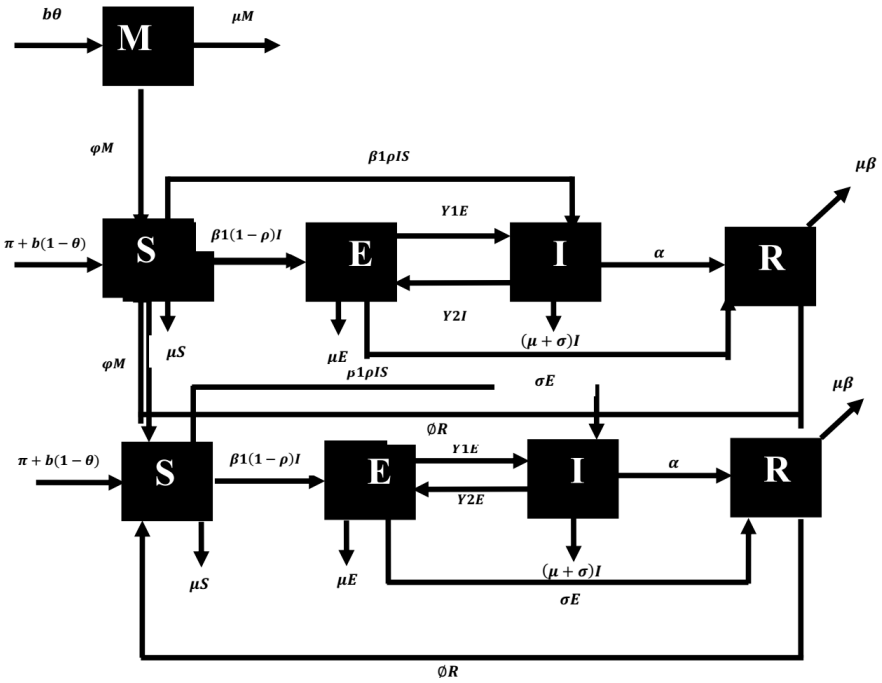

Figure 1: Formulation of the model

The definitions of parameters used in the model in Figure 1 are presented in Table 1.

Table 1: Definition of parameters used in the model

Parameters	Interpretation
b	Per capita birth rate on new-borns
θ	Rate of immunizing new-borns
φ	Warning rate of vaccine
π	Rate of recruiting susceptible migrants
β_1	Per capita contact rate
ρ	Rate of fast progression by new infective
μ	Natural death rate
γ_1	Progression rate from E to I
γ_2	Rate of effective chemoprophylaxis
α	Progression rate from I to R
δ	Measles induced death rate
σ	Progression rate from E to R
$\emptyset$	Rate of loss immunity

Thus, we have;

$$\left.\begin{aligned} \vartheta_1 &= b(1-\theta) \\ \vartheta_2 &= 1-\rho \\ k_1 &= \emptyset + \mu \\ k_2 &= \mu + \gamma_1 + \sigma \\ k_3 &= \mu + \delta + \alpha + \gamma_2 \\ k_4 &= \emptyset + \mu \end{aligned}\right\} \quad (6)$$

Hence (1) to (5) becomes

$$\frac{dM}{dt} = b\theta - k_1 M \quad (7)$$

$$\frac{dS}{dt} = \pi + \vartheta_1 + \varphi M + \emptyset R - \beta_1 \vartheta_2 IS - \beta_1 \rho IS - \mu S \quad (8)$$

$$\frac{dE}{dt} = \beta v_2 IS + \gamma_2 I - k_2 E \quad (9)$$

$$\frac{dI}{dt} = \beta_1 \rho IS + \gamma_1 E - k_3 I \quad (10)$$

$$\frac{dR}{dt} = \sigma E + \alpha I - k_4 R \quad (11)$$

2.1.3. Existence and uniqueness solution of the model

To check the validity and instability of any mathematical model, we have to confirm that the system of equation has a solution, if it has then is the solution unique? This subsection is concerned with finding whether the system of equations has a solution and if the solution to the system is unique. Consider the following theorems

- **Theorem 1**:

let D denotes the region, then we have that

$$|t - t_0| \leq a, \|x - x\| \leq b, \text{then } x = (x_1, x_2 \ldots . x_n) \text{ and } x_0 = x_{10}, x_{20} \ldots . x_{n0}) \quad (12)$$

And suppose that $f(t, x_1)$ satisfies the lipschitz condition that is

$$\|f(t, x_1) - f(t, x_2)\| \leq k\|x_1 - x_2\| \quad (13)$$

Whenever the pair (t, x_1) and (t, x_2) belong to D, where k is a positive constant. Then there is a constant $\delta > 0$ such that there exist a unique continuous vector solution $x(t)$ of the system in the interval $t - t_0 < \delta$. It is important that the condition (1) - (5) is satisfied by the requirement that

$\frac{\partial f_i}{\partial x_j}$, $i, j = 1, 2, \ldots \ldots$ is continuous and bounded in D.

Now return to the model (1) - (5), the interest is in the region:

$0 \leq \epsilon \leq R$. Look for abounded solution in this region and whose partial derivatives satisfies $0 \leq R < \infty$ where ϵ and δ are positive constants.

- **Theorem 2:**

Let D denotes the region $0 \leq \epsilon \leq R$, then the system (1- 5) has a unique solution is continuous and bounded in D. To show that $\frac{\partial f_i}{\partial x_j}, i, j = 1,2, \ldots \ldots$ are continuous and bounded in D.

Equation system (1-5) is the proof of the theorems stated above;
Proof:
Let,

$$f_1 = b\theta - (\mu + \varphi)M \quad (14)$$
$$f_2 = \pi + b(1-\theta) + \varphi M + \emptyset R - \beta_1(1-\rho)IS - \beta_1\rho IS - \mu S \quad (15)$$
$$f_3 = \beta_1(1-\rho)IS + \gamma_2 I - (\mu + \gamma_1 + \sigma)E \quad (16)$$
$$f_4 = \beta_1\rho IS + \gamma_1 E - (\mu + \delta + \alpha + \gamma_2)I \quad (17)$$
$$f_5 = \sigma E + \alpha I - (\emptyset + \mu)R \quad (18)$$

From Equation (14), we have the partial derivatives below:

$$\left.\begin{aligned} \left|\frac{\partial f_1}{\partial M}\right| &= |-(\mu+\varphi)| < \infty \\ \left|\frac{\partial f_1}{\partial S}\right| &= 0 < \infty \\ \left|\frac{\partial f_1}{\partial E}\right| &= 0 < \infty \\ \left|\frac{\partial f_1}{\partial I}\right| &= 0 < \infty \\ \left|\frac{\partial f_1}{\partial R}\right| &= 0 < \infty \end{aligned}\right\} \quad (19)$$

These partial derivatives of Equation (14) exist continuously and are bounded. Similarly from Equation (15), the partial derivative is as follows:

$$\left.\begin{aligned} \left|\frac{\partial f_2}{\partial M}\right| &= |\varphi| < \infty \\ \left|\frac{\partial f_2}{\partial S}\right| &= |-\beta_1(1-\rho)I - \beta_1\rho I - \mu| < \infty \\ \left|\frac{\partial f_2}{\partial E}\right| &= 0 < \infty \\ \left|\frac{\partial f_2}{\partial I}\right| &= |-\beta_1(1-\rho)S - \beta_1\rho S| < \infty \\ \left|\frac{\partial f_2}{\partial R}\right| &= |\varphi| \end{aligned}\right\} \quad (20)$$

Similarly, from Equation (16) the partial derivatives are as follows:

$$\left.\begin{aligned} \left|\frac{\partial f_3}{\partial M}\right| &= 0 < \infty \\ \left|\frac{\partial f_3}{\partial S}\right| &= |\beta_1(1-\rho)I| < \infty \\ \left|\frac{\partial f_3}{\partial E}\right| &= |-(\mu+\gamma_1+\sigma)| < \infty \\ \left|\frac{\partial f_3}{\partial I}\right| &= |\beta_1(1-\rho)S+\gamma_2| < \infty \\ \left|\frac{\partial f_3}{\partial R}\right| &= 0 < \infty \end{aligned}\right\} \quad (21)$$

Similarly, from Equation (17) the partial derivatives are as follow:

$$\left.\begin{aligned} \left|\frac{\partial f_4}{\partial M}\right| &= 0 < \infty \\ \left|\frac{\partial f_4}{\partial S}\right| &= |\beta_1\rho| < \infty \\ \left|\frac{\partial f_4}{\partial E}\right| &= |\gamma_1| < \infty \\ \left|\frac{\partial f_4}{\partial I}\right| &= |\beta_1\rho S-(\mu+\delta+\alpha+\gamma_2)| < \infty \\ \left|\frac{\partial f_4}{\partial R}\right| &= 0 < \infty \end{aligned}\right\} \quad (22)$$

Lastly, from Equation (18), the partial derivatives thus are:

$$\left.\begin{aligned} \left|\frac{\partial f_5}{\partial M}\right| &= 0 < \infty \\ \left|\frac{\partial f_5}{\partial S}\right| &= 0 < \infty \\ \left|\frac{\partial f_5}{\partial E}\right| &= |\sigma| < \infty \\ \left|\frac{\partial f_5}{\partial I}\right| &= |\alpha| < \infty \\ \left|\frac{\partial f_5}{\partial R}\right| &= |-(\emptyset+\mu)| < \infty \end{aligned}\right\} \quad (23)$$

As clearly shown above, the partial derivatives of the whole system (14) to (18) exist, they are finite and bounded as shown in Equations (19) – (23). Hence, by theorem 1, the model system (1) to (5) has a unique solution.

2.1.4. Existence of Equilibrium Point $(\in)$

The long term behaviour of the solutions of the ODEs (1) – (5) above can be examined at equilibrium states since the solution is independent of time. At equilibrium state, the rate of change of each variable is equal to zero.

$$\text{i.e.}\frac{dM}{dt}=\frac{dS}{dt}=\frac{dE}{dt}=\frac{dI}{dt}=\frac{dR}{dt}=0 \quad (23)$$

At any equilibrium state, let

$$\begin{bmatrix} M \\ S \\ E \\ I \\ R \end{bmatrix}=\begin{bmatrix} M^* \\ S^* \\ E^* \\ I^* \\ R^* \end{bmatrix} \quad (24)$$

Thus gives from system of Equations (7) – (11)

$$b\theta - k_1 M^* = 0 \quad (25)$$

$$\pi + \vartheta_1 + \varphi M^* + \emptyset R^* - \beta_1 \vartheta_2 S^* I^* - \beta_1 \rho I^* S^* - \mu S^* = 0 \quad (26)$$
$$\beta v_2 I^* S^* + \gamma_2 I^* - k_2 E^* = 0 \quad (27)$$
$$\beta_1 \rho I^* S^* + \gamma_1 E^* - k_3 I^* = 0 \quad (28)$$
$$\sigma E^* + \alpha I^* - k_4 R^* = 0 \quad (29)$$

From Equation (25)

$$M^* = \frac{b\theta}{k_1} \quad (30)$$

From Equation (29)

$$R^* = \frac{\sigma E^* + \alpha I^*}{k_4} \quad (31)$$

$$E^* \quad \frac{\beta_1 \vartheta_2 S^* I^* + \gamma_2 I^*}{k_2}$$

Substituting (32) into (28) gives

$$\beta_1 \rho I^* S^* + \frac{\beta_1 \vartheta_2 S^* I^* \gamma_1 + \gamma_1 \gamma_2 I^*}{k_2} - k_3 I^* = 0$$
$$\left[\frac{k_2 \beta_1 \rho S^* + \beta_1 \vartheta_2 \gamma_1 S^* + \gamma_1 \gamma_2 - k_2 k_3}{k_2}\right] I^* = 0 \quad (33)$$

This means that either

$$I^* = 0 \quad (34)$$

Or

$$\left[\frac{k_2 \beta_1 \rho S^* + \beta_1 \vartheta_2 \gamma_1 S^* + \gamma_1 \gamma_2 - k_2 k_3}{k_2}\right] = 0 \quad (35)$$

Equation (35) will be greater than zero if

$$k_2 \beta_1 \rho S^* > k_2 k_3 - \gamma_1 \gamma_2$$
$$\frac{k_2 \beta_1 \rho S^*}{\gamma_1 \gamma_2} > 1 \quad (36)$$

which resulted into an equilibrium state where each of the sub-population is greater than zero. Therefore, the system (1)– (5) has two different equilibrium states, namely: the disease free equilibrium in which all the infected compartments are zero and the endemic equilibrium in which all the compartments are greater than zero.

2.1.5. Linearization

Linearization of the system (1) gives the Jacobian matrix

$$J = \begin{bmatrix} -k_1 & 0 & 0 & 0 \\ \varphi & -\beta_1 \vartheta_2 I^* - \beta_1 \rho I^* - \mu & 0 & -\beta_1 \vartheta_2 S^* - \beta_1 \rho S^* & 0 \\ 0 & \beta_1 \vartheta_2 I^* & -k_2 & \beta_1 \vartheta_2 S^* + \gamma_2 & \emptyset \\ 0 & -\beta \beta_1 \rho I^* & \gamma_1 & \beta_1 \rho S^* - k_3 & 0 \\ 0 & 0 & \sigma & \alpha & -k_4 \end{bmatrix} \quad (37)$$

At the disease free equilibrium point ($\in^*$) the jacobian matrix becomes

$$J(\in^*) = \begin{bmatrix} -k_1 & 0 & 0 & 0 & 0 \\ \varphi & -\mu & 0 & -(\vartheta_2 + \rho)\beta_1 S^0 & \emptyset \\ 0 & 0 & -k_2 & \beta_1 \vartheta_2 S^0 + \gamma_2 & 0 \\ 0 & 0 & \gamma_1 & \beta_1 \rho S^0 - k_3 & 0 \\ 0 & 0 & \sigma & \alpha & -k_4 \end{bmatrix} \quad (38)$$

The jacobian matrix (38) shall be used in the local stability analysis of both the disease free and endemic equilibria.

2.1.6. Basic reproduction number (R_0)

The basic reproduction number, R_0 is a measure of the number of infections produced on average, by an infected individual in the early stages of an epidemic, when virtually all contacts are susceptible when R_0<1, then on average, an infected individual produces less than one newly infected individual over the course of its infection period, and hence the infection may die out in the long run. If R_0>1, each infected individual produces on average more than one new infection, the infection will be able to spread in a population thus becoming endemic. To find the R_0 of the system $(1)-(5)$ we use the next generation approach i.e.

$$R_0=\rho(FV^{-1}).$$

Where ρ *is the* spectral radius, is the matrix of new infection terms and is the matrix of transmission terms.

Then,

$$F=\begin{bmatrix}0 & \beta_1\vartheta_2 S^0\\ 0 & \beta_1\rho S^0\end{bmatrix} \tag{39}$$

And

$$V=\begin{bmatrix}K_2 & -\gamma_2\\ -\gamma_1 & K_2\end{bmatrix} \tag{40}$$

Remark: Both F and V are obtained from the jacobian matrix (37) of the linearized system of the disease free equilibrium.

In order to determine the matrix V^{-1}, recalls that

$$V^{-1}=\frac{c^T}{|V|} \tag{41}$$

$$V=\begin{vmatrix}K_2 & -\gamma_2\\ -\gamma_1 & K_3\end{vmatrix}=K_2K_3-\gamma_1\gamma_2 \tag{42}$$

$$V^{-1}=\frac{1}{K_2K_3-\gamma_1\gamma_2}\begin{bmatrix}K_3\gamma_2\\ \gamma_2 K_2\end{bmatrix} \tag{43}$$

$$V^{-1}=\begin{bmatrix}\frac{K_3}{K_2K_3-\gamma_1\gamma_2} & \frac{\gamma_2}{K_2K_3-\gamma_1\gamma_2}\\ \frac{\gamma_1}{K_2K_3-\gamma_1\gamma_2} & \frac{K_2}{K_2K_3-\gamma_1\gamma_2}\end{bmatrix} \tag{44}$$

$$FV^{-1}=\begin{bmatrix}0 & \beta_1\vartheta_2 S^0\\ 0 & \beta_1\rho S^0\end{bmatrix}\begin{bmatrix}\frac{K_3}{K_2K_3-\gamma_1\gamma_2} & \frac{\gamma_2}{K_2K_3-\gamma_1\gamma_2}\\ \frac{\gamma_1}{K_2K_3-\gamma_1\gamma_2} & \frac{K_2}{K_2K_3-\gamma_1\gamma_2}\end{bmatrix} \tag{45}$$

Now find the eigenvalues by $|FV^{-1}-\lambda I|=0$

$$FV^{-1}=\begin{bmatrix}\frac{\beta_1\vartheta_2\gamma_1 S^0}{K_2K_3-\gamma_1\gamma_2}-\lambda & \frac{K_2\beta_1\vartheta_2 S^0}{K_2K_3-\gamma_1\gamma_2}\\ \frac{\beta_1\rho\gamma_1 S^0}{K_2K_3-\gamma_1\gamma_2} & \frac{K_2\beta_1\rho S^0}{K_2K_3-\gamma_1\gamma_2}-\lambda\end{bmatrix} \tag{46}$$

$$\lambda_1=\frac{\beta_1\vartheta_2\gamma_1 S^0}{K_2K_3-\gamma_1\gamma_2} \tag{47}$$

$$\lambda_2=\frac{K_2\beta_1\rho S^0}{K_2K_3-\gamma_1\gamma_2} \tag{48}$$

$$R_0 = \frac{K_2\beta_1\rho S^0}{K_2K_3-\gamma_1\gamma_2} \quad (49)$$

3. Results and Discussion

The baseline values are presented in Table 1.

Table 1 Baseline values of the parameter for the model

Parameter	Description	Baseline Value
n_1	Per capital birth rate on new born	0.02755/year
$\emptyset$	Rate of immunizing new born	0.5(cell/mc)
θ	Warning rate of vaccine	0.7/year
φ	Rate of recruiting susceptible migrant	0.167/year
$\propto_1$	Per capital contact rate	0.09091/year
μ	Rate of fast progression by new infective	0.125/year
n_2	Natural death rate	0.00875/year
$\in$	Proportion of individuals who received a first vaccination	0.7/year
q	Proportion of individual who are vaccinated twice	0.5/year
P_1	Progression rate from E to I	0.125/year
P_2	Rate of effective chemoprophylaxis	0.096/year
β	Measles induced death rate	0.125/year
δ	Progression rate from E to R	0.14286/year
φ	Rate of loss immunity	0.25/year
b	Progression rate from I to R	0.14286/year

Simulation of the Mathematical model is presented as follows:

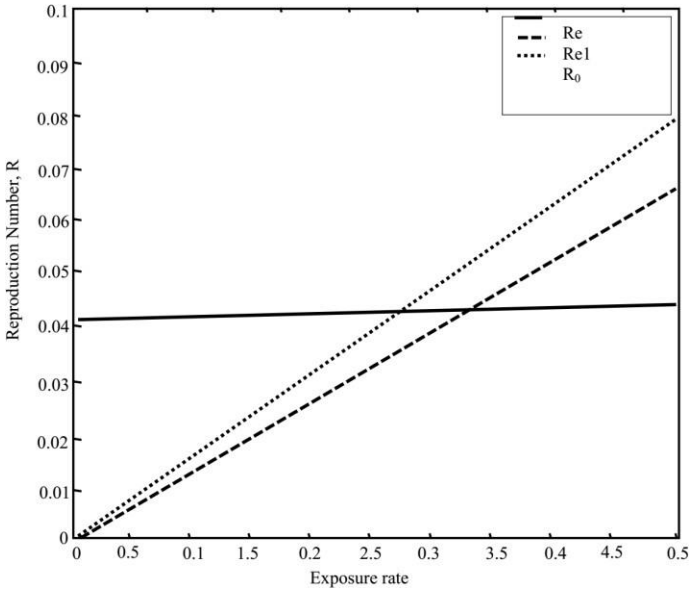

Figure 2: Variations in reproduction number with respect to exposure rate

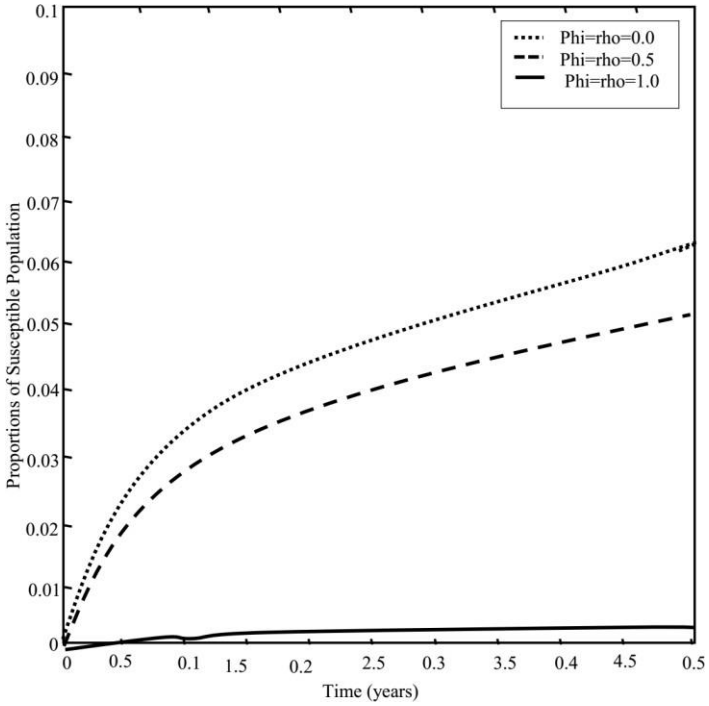

Figure 3: Susceptible population in an outbreak versus the proportion of immunized coverage of both the new-borns and immigrants (phi=rho= 0.0, 0.5, 1.0)

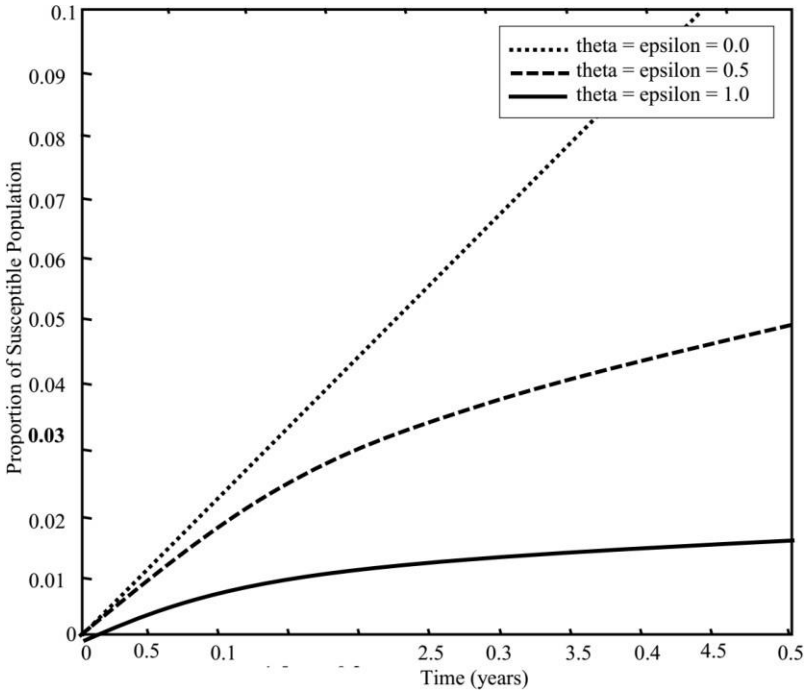

Figure 4: Susceptible population in an outbreak versus immunized population *(theta =epsilon=0.0, 0.5, 1.0)*

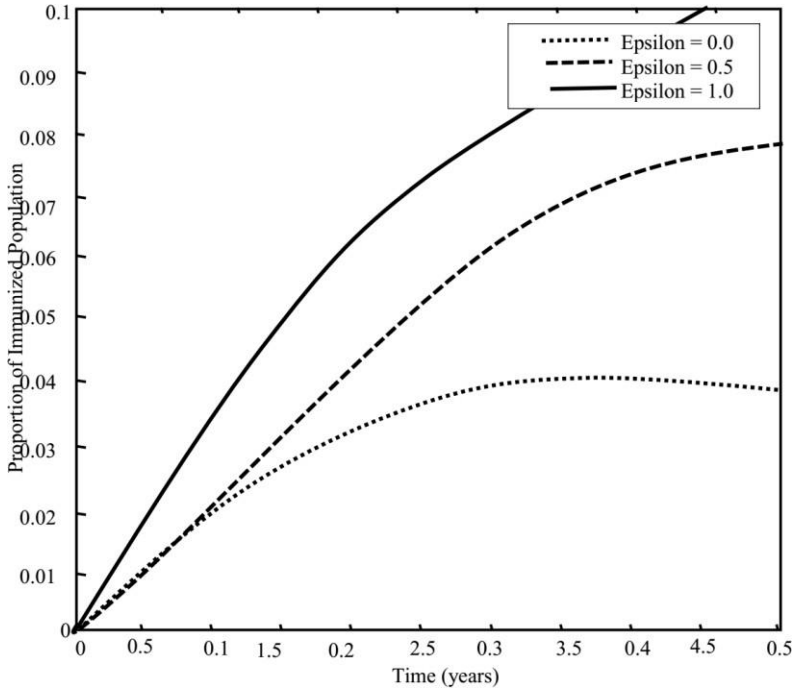

Figure 5: Immunized population in an outbreak versus the proportion of first dose vaccination (epsilon =0.0, 0.5, 1.0)

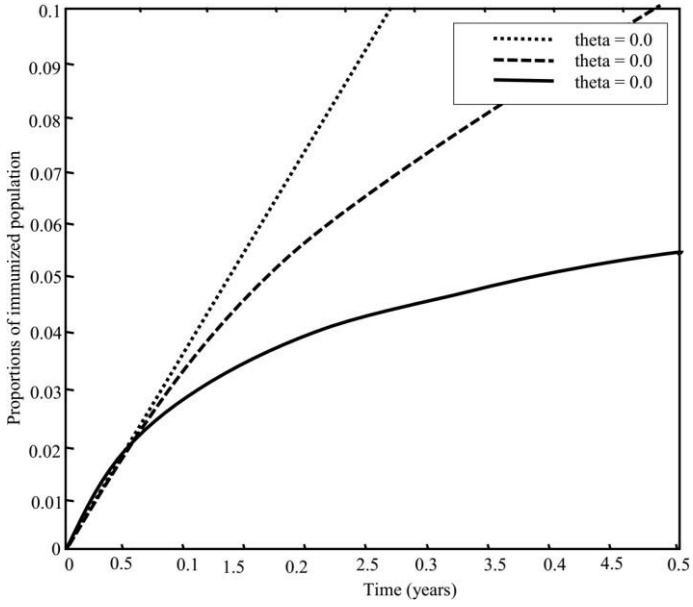

Figure 6: Immunized population in an outbreak versus the proportion of second vaccination (theta =0.0, 0.5, 1.0)

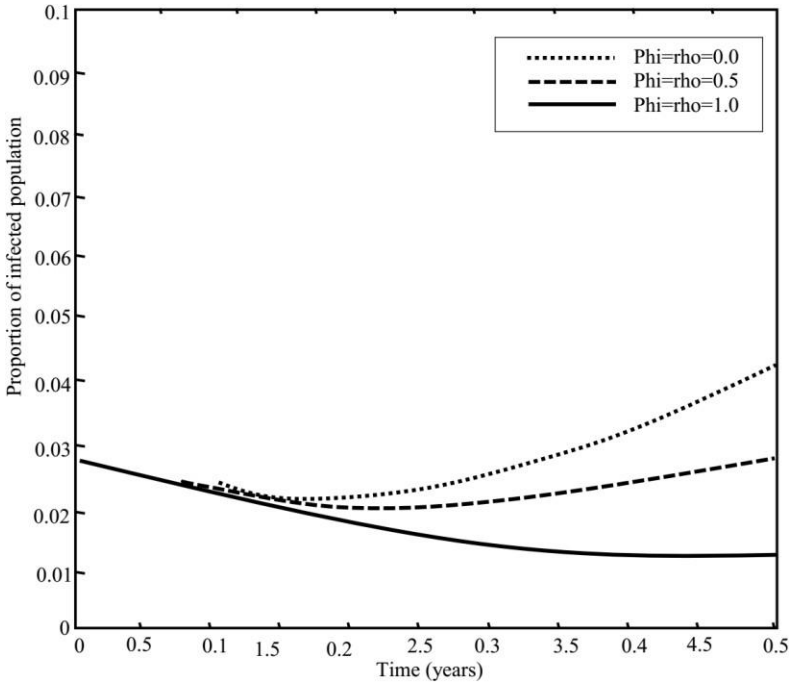

Figure 7: Infected population in an outbreak versus the proportion of new born and immunized immigrants (phi=rho=0.0, 0.5, 1.0)

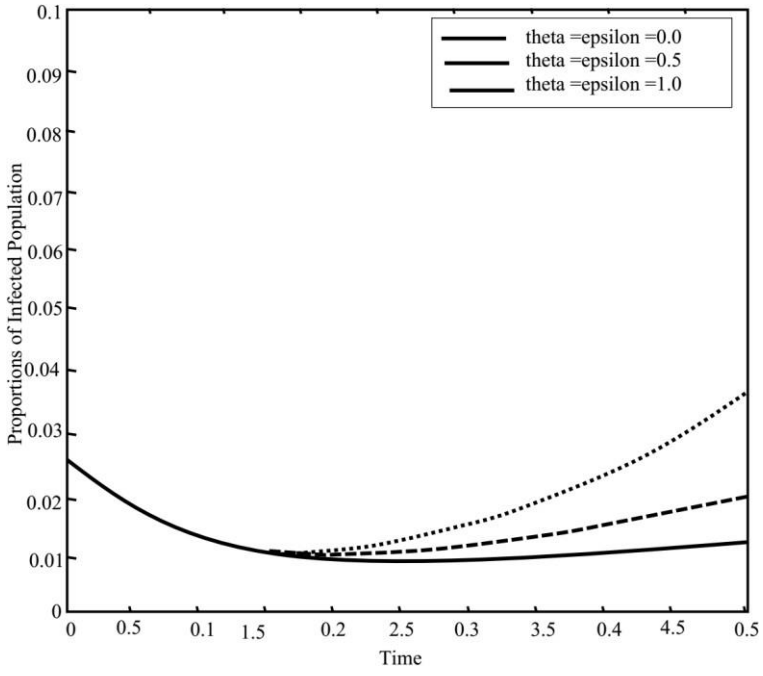

Figure 8: Infected population versus immunized coverage (theta = epsilon =0.0, 0.5, 1.0)

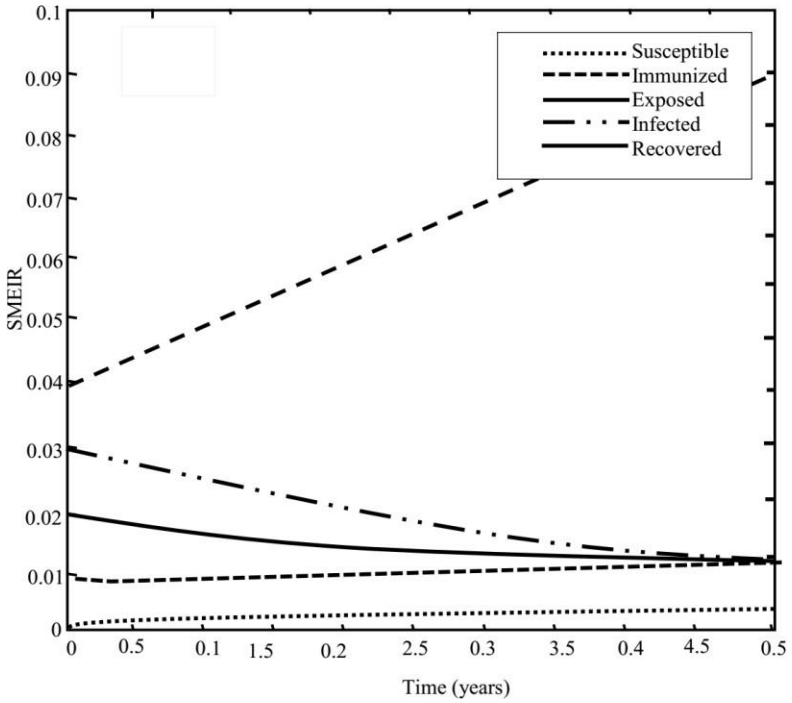

Figure 9: Combined Susceptible, Immunized, Exposed, Infectious and Recovered population

Figure 2 indicates that the basic reproduction number R_0 is worst case scenario, it occurred when there is no immunizing strategy to control the epidemic, in this situation individual recovered naturally. The basic reproduction number R_0 is at the peak, this implied that there was a high increase in reproduction number with respect to exposure rate and such increase results in the outbreak of measles in the community. The middle graph R_m, from the same Figure1 shows effect of immunizing immigrants and new-borns there by leaving away only the susceptible population. The best case scenario occurred at graph R_L from the same Figure1, where vaccination was offered to new-borns, immigrants and the susceptible adults, it was observed that R_L has the least value of increase in reproduction number with respect to exposure rate, which implied that measles can be eradicated from the community if immunization policies was seriously targeted to a large population. Figure 3 shows that the increase in immunized coverage to both the new-borns and immigrants leads to reduction in the susceptible population and therefore reducing the risk of an outbreak. Figure 4 Indicates that when immunization programmes were effectively implemented to the population, it may reach a stage that the disease fail to erupt since there are very few susceptible individuals to infect. Figure 5 shows that the number of immunized individuals increases by offering first dose of vaccine to susceptible individuals in the population and therefore reducing the number of susceptible adults and children in the population. Figure 6 shows that provision of second dose of vaccine increases the number of individuals who cannot be infected by the disease. Figure 7 showed that if more new born and immigrants are been immunized then, the probability of individuals to be infected with the disease becomes very small and this could lead to the disease to die out in a population. Figure 8 showed that the population of infected individuals decreases with an increase in immunized coverage. This is also attributed with the fact that less people will be susceptible as they will be immune to the disease. Figure 9 showed

that the model provided the illustration for control and elimination of the transmission dynamics of measles. It can be observed that recovered individual can be increased by increasing immunization and consequently reducing the susceptible and infectious individuals.

A model was constructed for the transmission dynamics of measles, individuals were categories into five compartments the immunized, susceptible, exposed, infectious and recovered. It was found that they are asymptotically stable since $R_0 < 1$, i.e the epidemiological implication of the system was that the disease can be controlled in the population if the initial sizes of the subpopulation of the model are in the basin of attraction of the disease free equilibrium. Since the production number is defined as the number of secondary cases generated by an infectious individual, Hence the model establish that if the rate of contact between the infectious and susceptible as well as some factors explained to have been the cause of spread of measles are maintained and carefully looked into, then asymptotical stability of the problem shall always be obtained otherwise there would be endemic (Persistent of the disease in a population).

Considering the mathematical modelling on the control of measles by vaccination using S: susceptible E: exposed I: infected T: treated and R: recovered (SEIR) model by [2]. Their study recommended the introduction of mass vaccination programme and improvement in early detection of measles cases to minimize transmission.While this study used the five compartments known as M: immunized S: susceptible E: exposed I: infected T: treated and R: recovered (MSEIR) model for prevention and control of the disease called measles and the disease cannot persist in the population. In addition, Figure 9 showed that the model provided the illustration for control and elimination of the transmission dynamics of measles. It can be observed that recovered individual can be increased by increasing immunization and consequently reducing the susceptible and infectious individuals.

4. Conclusion

It is observed that the model has shown significance of measles vaccination in controlling and preventing transmission within a population. The model pinpoint that the spread of a disease greatly depend on the contact rate with infected individuals within a population. It is also realized that the proportion of the population that is immune exceeded the herd immunity level of measles. Therefore the disease cannot persist in the population.

5. Acknowledgment

The authors wish to thank the reviewers who have taken their time to perfect this article.

6. Conflict of Interest

There is no conflict of interest associated with this work.

References

[1] Bakare, E. A., Adekunle, Y. A. and Kadiri, K. I. O. (2012). Modelling and Simulation of Dynamics of Transmission of the Measles. International Journal of Computer Trends and Technology. 3 Issue 1. pp. 174- 192

[2] Fred, M. O., Okello, J. A., Okwyo, J. M. and Kang'ethe, G. J. (2014). Mathematical Modelling on the Control of Measles by Vaccination: Case Study of KISII Country, Kenya. The SIJ Transmission on Computer Science Engineering and its Applications (CSEA) 2, pp. 61-69.

[3] WHO (2006). Measles Vaccines. In Weekly Epidemiology Record, World Health Organisation.

[4] Tessa, O. M. (2006). Mathematical model for control of measles by vaccination. In Proceedings of Mali Symposium on Applied Sciences pp. 31-36
[5] Momoh, A.A., Ibrahim, M.O., Uwanta, I.J. and Manga, S.B. (2013). Mathematical Model for Control of Measles Epidemiology. International Journal of Pure and Applied Mathematics.67, pp 707-718.
[6] Ebenezer, B., Asamoah, J. K. K., Khan, I., Kwesi, A.-A. S. and Khan, M. A. (2017). The multi-step homotopy analysis method for a modified epidemiological model for measles disease. Nonlinear Sci. Lett. A, 8(2), pp. 320-332.
[7] Peter, O. J., Afolabi, O. A., Victor, A.A., Akpan, C. E. and Oguntolu, F. A. (2018). Journal of Applied Science Environment and Management. 22 (4), pp. 571 – 576