

## Mathematical Modeling of the Transmission Dynamics of Measles under the Effect of Vaccination.

Mutuguta John Wanjau\*, Rotich Titus<sup>†</sup>, Chepkwony Isaac<sup>‡</sup>

\*Murang'a University of Technology, Department of Mathematics and Actuarial Science

<sup>†</sup>Moi University, Department of Mathematics and Physics

<sup>‡</sup>Kenyatta University, Department of Mathematics

**Abstract:** Measles virus is a member paramyxoviridae within the genus of morbillivirus. Its genome consist of approximately 16,000 bases of non-segmented single stranded negative sense RNA. This means that the virus is transcribed immediately upon entry into the cell. The virus spreads from person to person through the release of the aerosol droplets. In this paper, we investigate the transmission of measles virus using the five compartments of susceptible, vaccinated, exposed, infectious and recovered individuals with demographic factors. We give the mathematical model describing the transmission of the measles virus. The results of the model analysis showed that the model has a unique disease free equilibrium (DFE) which is locally asymptotically stable when  $R_0 < 1$  and unstable when  $R_0 > 1$ . We further carried out numerical simulation of the model to investigate the effect of vaccination on the transmission dynamics of the virus. The results showed that there exist a minimum value of the vaccine efficacy below which herd immunity cannot be achieved. We further observed that increasing the vaccine efficacy above this critical value will lower the herd immunity of the population.

**Keywords;** Measles virus, basic reproduction number, vaccine efficacy.

Mathematics subject classification: 97M60, 00A71, 46N70

Date of Submission: 15-07-2019

Date of acceptance: 31-07-2019

### Notations and Symbols

MMR	Measles, Mumps and Rubella
$R_0$	Basic reproduction number.
$r$	Birthrate
$\mu$	perca-pita deathrate
$\gamma$	recoveryrate.
$\alpha$	vaccineefficacy.
$\omega$	Vaccinationrate
$\beta$	Transmissionprobability.
$\sigma$	rateofprogressionfromexposedtoinfectiousclass.
$d$	per ca-pita disease mortalityrate

### I. Introduction

In this paper we consider the transmission dynamics of Measles. Measles is a communicable disease which spreads through person - to - person transmission mode. It is a highly contagious disease with an attack rate of over 90% among the susceptible individuals. Measles is a severe illness with high attack rate especially in malnourished children and mainly those with vitamin A deficiency or those whose immune system have been weakened by other infections such as HIV/AIDS. It produces a red rash and may lead to serious complications such as pneumonia, diarrhoea and encephalitis. Children infected with measles virus do not normally die from the disease itself but may die from complications such as diarrhoea and pneumonia which are more common to children under the age of five years. Some children recovering from the disease suffer deafness, encephalitis, impaired vision or even blindness in some cases. It is a viral respiratory infectious disease which attacks the immune system and is transmitted through the respiratory droplets or contact with throat or nasal secretions of an infectious individual. Measles is an immunizing disease thus individuals recovering from the disease acquires lifelong immunity from further attacks. It poses a great challenge to human population and its prevention or control is an important milestone to the human population. The efficient intervention of the disease depends on the understanding of its transmission and persistence. Measles dynamics has a long history of data analysis and modeling. Considering only the local dynamics, the extinction of the virus results when the local chain of

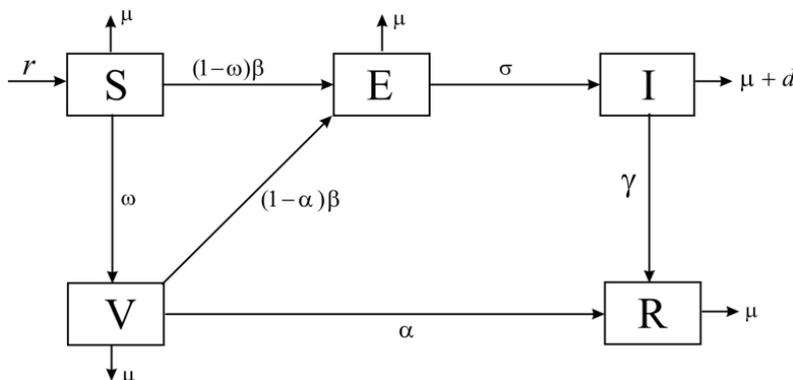
transmission is broken. In many instances this will happen in small host populations when the epidemic tends to diminish the susceptible individuals in the population which in turn reduces the force of infection leading to breakdown in the transmission chain.

In their work titled *A Stochastic Modeling of Recurrent Measles Epidemics*, Kassem and Ndam [6] developed a simple stochastic Mathematical model for the dynamics of Measles with multidimensional diffusion process. When developing their model, they considered and partitioned the population into susceptible, exposed, infected and removed classes and assumed among other things that stochastic effects arise in the process of infection of susceptible individuals. The results of their simulation seemed to agree with the historical pattern of measles in Nigeria. [1] developed a model that divided the total population (N) into four classes of Susceptible, Exposed, infected and Recovered individuals they further incorporated testing and measles therapy into the dynamics at the exposed period to investigate the control of measles epidemiology at latent period. They assumed that the individuals recovering from exposed class as a result of measles therapy and those that recovered naturally from the infectious class became permanently immune. They developed a mathematical model of non-linear first order ordinary differential equation. The result of their stability analysis showed that the system was asymptotically stable. [2] developed a mathematical Model for the dynamics of measles by incorporating vaccination of susceptible individuals. When developing their model they considered a population with variable size and that vaccination conferred lifelong immunity to the vaccinated individuals and therefore the vaccinated individuals moved to the recovered class. Their model consisted of a set of Ordinary Differential Equations and Partial Differential Equations and they carried out numerical and qualitative analyses of the model by varying the values of the state variables. The results of their study showed that the model had the disease-free equilibrium (D.F.E.) which was locally asymptotically stable for  $R_0 < 1$  and unstable for  $R_0 > 1$ . [5] adopted compartmental modeling approach by partitioning the population into Susceptible, Vaccinated, Exposed, Infected and Recovered sub-populations. The result of the model analysis showed that the model had a unique disease free equilibrium which was found to be locally asymptotically stable whenever the basic reproduction number is less than one and unstable otherwise. The authors further carried out numerical experiment using the data of [1], the results of the numerical experiments revealed that eradicating measles would be more efficient if susceptible individuals were vaccinated and followed by drug therapy to screened infected individuals in the exposed class.

From available literature most authors assumed that vaccinated individuals acquire permanent immunity. However vaccines are not 100% efficient and some vaccinated individuals may still contract the disease. In this paper, we study the effect of vaccination of susceptible individuals by incorporating loss of immunity of vaccinated individuals due to drop in efficacy of vaccine.

## II. ModelFormulation

We formulate a deterministic compartmental model to describe the transmission dynamics of the disease. We divide the population into five compartments of susceptible individuals (S), vaccinated individuals (V), exposed individuals (E), infectious individuals (I) and recovered individuals (R). The flow chart for the model is given below



**Figure 1:** Measles model with vaccination

The class S of susceptible is increased by birth or immigration at a rate  $r$  and decreased by infection following contact with infected individuals at a rate  $\beta$ , natural death at a rate  $\mu$  and vaccination at a rate  $\omega$ . The class E of exposed individuals is generated through contact of susceptible individuals with infected individuals at rate  $\beta$  it is decreased by progression into infected class at a rate  $\sigma$  and natural death at a rate  $\mu$ . The class I of infected individuals is generated through progression of exposed individuals at a rate  $\sigma$  and diminished by

natural death at a rate  $\mu$ , disease mortality at a rate  $d$  and recovery from infection at a rate  $\gamma$ . The model assumes that recovered individuals become permanently immune to the disease. This generates a class  $R$  of individuals who have complete protection against the disease. The class  $R$  of recovered individuals is diminished by natural death at a rate  $\mu$ . The class  $V$  of vaccinated individuals is generated through vaccination of susceptible individuals at a rate  $\omega$ , it is diminished by natural mortality at a rate  $\mu$  and loss of immunity due to waning of the vaccine at a rate  $1 - \alpha$ . A proportion  $\alpha$  of vaccinated individuals acquires complete protection from the disease and enter the recovered class  $R$ .

The mathematical model for the system is of the form;

$$\begin{aligned} \frac{dS}{dt} &= r - (\mu + \omega)S - (1 - \omega)\beta SI \\ \frac{dV}{dt} &= \omega S - \mu V - (1 - \alpha)\beta VI - \alpha V \\ \frac{dE}{dt} &= (1 - \omega)\beta SI + (1 - \alpha)\beta VI - (\mu + \sigma)E \\ \frac{dI}{dt} &= \sigma E - (\mu + d + \gamma)I \\ \frac{dR}{dt} &= \alpha V + \gamma I - \mu R \end{aligned} \tag{1}$$

### 2.1 Equilibrium Analysis

At equilibrium points  $\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$  thus we get the system of equations;

$$r - (\mu + \omega)S - (1 - \omega)\beta SI = 0 \tag{2}$$

$$\omega S - \mu V - (1 - \alpha)\beta VI - \alpha V = 0 \tag{3}$$

$$(1 - \omega)\beta SI + (1 - \alpha)\beta VI - (\mu + \sigma)E = 0 \tag{4}$$

$$\sigma E - (\mu + d + \gamma)I = 0 \tag{5}$$

$$\alpha V + \gamma I - \mu R = 0 \tag{6}$$

From equation [5](#)

$$E = \frac{(\mu + d + \gamma)I}{\sigma} \tag{7}$$

Substituting this into equation [4](#) and simplifying, we get

$$\left( (1 - \omega)\beta S + (1 - \alpha)\beta V - \frac{(\mu + \sigma)(\mu + d + \gamma)}{\sigma} \right) I = 0 \tag{8}$$

thus either  $I = 0$  or

$$(1 - \omega)\beta S + (1 - \alpha)\beta V - \frac{(\mu + \sigma)(\mu + d + \gamma)}{\sigma} = 0$$

Substituting  $I = 0$  into equations 2, 3, 6 and 7 we get the disease free equilibrium given by,

$$\{S^0, V^0, E^0, I^0, R^0\} = \left\{ \frac{r}{\mu + \omega}, \frac{\omega r}{(\mu + \omega)(\mu + \alpha)}, 0, 0, \frac{\alpha \omega r}{\mu(\mu + \omega)(\mu + \alpha)} \right\} \quad (9)$$

### 2.2 Basic Reproduction Number

Basic reproduction number is one of the most important quantity in infectious disease modeling. Its value provides insights when designing control interventions for established infections and in emerging disease outbreaks, it is one of the most urgently estimated quantity to provide insights on the suitable control methods. Basic reproduction number is usually denoted by  $R_0$  and defined as the average number of infections resulting from an index case in an otherwise susceptible population. In general if  $R_0 > 1$  then the index case will on average infect more than one individual during its entire infectious period thus the pathogen will invade the population. If on the other hand  $R_0 < 1$  then each index case will on average infect less than one other individual in the population during its entire infectious period which implies that the infectious disease will die out. In this case the pathogen is not able to invade the population. It has been shown that the basic reproduction number is mathematically characterised by regarding infections as a demographic process where producing offspring is not seen as giving birth in the normal demographic sense but as causing a new infection through transmission. This process is termed epidemiological birth. In natural way this leads to viewing the infection process in terms of consecutive generations of infected individuals [4].

In epidemic modeling, generations are the waves of secondary infections that flow from each previous infection. Thus the first generation of an epidemic comprise of all the secondary infections that result from the infectious contact with the index case who is regarded as generation zero. Therefore if  $R_i$  denotes the reproduction number of the  $i^{th}$  generation, then  $R_0$  will denote the number of infections generated by the index case [3]. These numbers are small and are subject to sampling errors, we however determine the mean value hence we find an average over a large number of epidemics. The first step when determining this number is to construct the next generation matrix (NGM) of the epidemic. In the construction of the next generation matrix, one begins by identifying those equations in the system that describe the production of new infections and the changes in state among infected individuals. This set of equations is referred to as the infected subsystem. We then linearise the system over the disease free equilibrium (DFE), the linearization indicates that  $R_0$  characterises the potential for the initial spread of the infectious agent when it is introduced into a fully susceptible population and therefore we assume that the change in the susceptible population is negligible during the initial spread.

The right hand side of the system is split into the transmission matrix (T) and the transition matrix (V) where T is a non-negative matrix and V in a non-singular M-matrix. The basic reproduction number is then obtained as the spectral radius of the next generation matrix ( $TV^{-1}$ ) i.e  $R_0 = \rho(TV^{-1})$  [4].

For the system 1 above the infected subsystem is given by;

$$\frac{dE}{dt} = (1 - \omega)\beta SI + (1 - \alpha)\beta VI - (\mu + \sigma)E$$

$$\frac{dI}{dt} = \sigma E - (\mu + d + \gamma)I$$

or

$$\begin{pmatrix} \frac{dE}{dt} \\ \frac{dI}{dt} \end{pmatrix} = F - M$$

where

$$F = \begin{pmatrix} (1 - \omega)\beta SI + (1 - \alpha)\beta VI \\ 0 \end{pmatrix}$$

and

$$M = \begin{pmatrix} (\mu + \sigma)E \\ -\sigma E + (\mu + d + \gamma)I \end{pmatrix}$$

The next generation matrix  $K$  of the system is given by

$$K = TV^{-1}$$

where

$$T = DF = \begin{pmatrix} 0 & (1 - \omega)\beta S + (1 - \alpha)\beta V \\ 0 & 0 \end{pmatrix}$$

and

$$V = DM = \begin{pmatrix} \mu + \sigma & 0 \\ -\sigma & \mu + d + \gamma \end{pmatrix}$$

Thus the NGM is given by;

$$K = \begin{pmatrix} \frac{\sigma((1 - \omega)\beta S^* + (1 - \alpha)\beta V^*)}{(\mu + \sigma)(\mu + d + \gamma)} & \frac{(1 - \omega)\beta S^* + (1 - \alpha)\beta V^*}{\mu + d + \gamma} \\ 0 & 0 \end{pmatrix}$$

at DFE  $S^* = S^0$  and  $V^* = V^0$  thus we get

$$K = \begin{pmatrix} \frac{\sigma((1 - \omega)\beta S^0 + (1 - \alpha)\beta V^0)}{(\mu + \sigma)(\mu + d + \gamma)} & \frac{(1 - \omega)\beta S^0 + (1 - \alpha)\beta V^0}{\mu + d + \gamma} \\ 0 & 0 \end{pmatrix} \tag{10}$$

and the basic reproduction number is given by

$$\begin{aligned} R_0 &= \rho(TV^{-1}) \\ &= \frac{\sigma((1 - \omega)\beta S^0 + (1 - \alpha)\beta V^0)}{(\mu + \sigma)(\mu + d + \gamma)} \\ &= \frac{r\beta\sigma}{(\mu + \omega)(\mu + \sigma)(\mu + d + \gamma)} \left( 1 - \omega \left( 1 - \left( \frac{1 - \alpha}{\mu + \alpha} \right) \right) \right) \end{aligned} \tag{11}$$

Differentiating  $R_0$  with respect to  $\omega$  we get

$$\begin{aligned} \frac{\partial R_0}{\partial \omega} &= \frac{-r\beta\sigma}{(\mu + \omega)^2(\mu + \sigma)(\mu + d + \gamma)} \left( 1 - \omega \left( 1 - \left( \frac{1 - \alpha}{\mu + \alpha} \right) \right) \right) \\ &\quad - \frac{r\beta\sigma}{(\mu + \omega)(\mu + \sigma)(\mu + d + \gamma)} \left( 1 - \left( \frac{1 - \alpha}{\mu + \alpha} \right) \right) \end{aligned} \tag{12}$$

Thus  $R_0$  decreases with increasing vaccination rate provided that  $1 - \left( \frac{1 - \alpha}{\mu + \alpha} \right) > 0$  or  $\alpha > \frac{1}{2}(1 - \mu)$ . From the above analysis, we observe that the vaccine should not be administered unless its efficacy is greater than  $\frac{1}{2}(1 - \mu)$ .

**2.3 Stability Analysis**

In this section, we investigate the stability of the disease free equilibrium. To determine its stability criteria, we first construct the Jacobian matrix and linearise it at disease free equilibrium.

The Jacobian matrix  $J$  of the system 1 is given by;

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial V} & \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial V} & \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial R} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial V} & \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial R} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial V} & \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial R} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial V} & \frac{\partial f_5}{\partial E} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial R} \end{pmatrix} \tag{13}$$

where

$$\begin{aligned} f_1 &= r - (\mu + \omega)S - (1 - \omega)\beta SI \\ f_2 &= \omega S - \mu V - (1 - \alpha)\beta VI - \alpha V \\ f_3 &= (1 - \omega)\beta SI + (1 - \alpha)\beta VI - (\mu + \sigma)E \\ f_4 &= \sigma E - (\mu + d + \gamma)I \\ f_5 &= \alpha V + \gamma I - \mu R \end{aligned}$$

substituting, we get

$$J = \begin{pmatrix} -(\mu + \omega) - (1 - \omega)\beta I & 0 & 0 & -(1 - \omega)\beta S & 0 \\ \omega & -\mu - \alpha - (1 - \alpha)\beta I & 0 & -(1 - \alpha)\beta V & 0 \\ (1 - \omega)\beta I & (1 - \alpha)\beta I & -(\mu + \sigma) & (1 - \omega)\beta S + (1 - \alpha)\beta V & 0 \\ 0 & 0 & \sigma & -(\mu + d + \gamma) & 0 \\ 0 & \alpha & 0 & \gamma & -\mu \end{pmatrix}$$

At DFE,  $I = E = 0$ ,  $S = S^0$  and  $V = V^0$ , substituting these values we get

$$J = \begin{pmatrix} -(\mu + \omega) & 0 & 0 & -(1 - \omega)\beta S^0 & 0 \\ \omega & -\mu - \alpha & 0 & -(1 - \alpha)\beta V^0 & 0 \\ 0 & 0 & -(\mu + \sigma) & (1 - \omega)\beta S^0 + (1 - \alpha)\beta V^0 & 0 \\ 0 & 0 & \sigma & -(\mu + d + \gamma) & 0 \\ 0 & \alpha & 0 & \gamma & -\mu \end{pmatrix}$$

The characteristic equation is given by;  $|J - \lambda I| = 0$

$$\begin{vmatrix} -(\mu + \omega) - \lambda & 0 & 0 & -(1 - \omega)\beta S^0 & 0 \\ \omega & -\mu - \alpha - \lambda & 0 & -(1 - \alpha)\beta V^0 & 0 \\ 0 & 0 & -(\mu + \sigma) - \lambda & (1 - \omega)\beta S^0 + (1 - \alpha)\beta V^0 & 0 \\ 0 & 0 & \sigma & -(\mu + d + \gamma) - \lambda & 0 \\ 0 & \alpha & 0 & \gamma & -\mu - \lambda \end{vmatrix} = 0$$

Solving, we get the Eigen values

$$\begin{aligned} \lambda_1 &= -\mu \\ \lambda_2 &= -(\mu + \omega) \\ \lambda_3 &= -(\mu + \alpha) \\ \lambda_4 &= \frac{1}{2}(B - \sqrt{B^2 - 4C}) \\ \lambda_5 &= \frac{1}{2}(B + \sqrt{B^2 - 4C}) \end{aligned}$$

where

$$B = -(2\mu + \sigma + d + \gamma) \text{ and}$$

$$C = (\mu + \sigma)(\mu + d + \gamma) - \sigma[(1 - \omega)\beta S^0 + (1 - \alpha)\beta V^0]$$

We note that  $Re\{\lambda_{1,2,3,4}\} < 0$  and  $Re\{\lambda_5\} < 0$  when  $C > 0$  thus the stability criteria for DFE may be stated as

$$(\mu + \sigma)(\mu + d + \gamma) - \sigma[(1 - \omega)\beta S^0 + (1 - \alpha)\beta V^0] > 0 \tag{14}$$

but  $S^0 = \frac{r}{\mu + \omega}$  and

$$V^0 = \frac{\omega r}{(\mu + \omega)(\mu + \alpha)}$$

substituting these values into equation [14](#) and simplifying, we get

$$\frac{\beta \sigma r [(\mu + \alpha)(1 - \omega) + \omega(1 - \alpha)]}{(\mu + \omega)(\mu + \alpha)(\mu + \sigma)(\mu + d + \gamma)} < 1 \tag{15}$$

but

$$\frac{\beta \sigma r [(\mu + \alpha)(1 - \omega) + \omega(1 - \alpha)]}{(\mu + \omega)(\mu + \alpha)(\mu + \sigma)(\mu + d + \gamma)} = R_0$$

Thus the DFE is locally asymptotically stable whenever  $R_0 < 1$  and unstable when  $R_0 > 1$ .

### III. Results And Discussion

In the previous section we have derived the condition under which the disease free equilibrium is asymptotically stable. In this section, we present the results for the numerical simulation of the model. We choose the parameter values  $\beta = 0.95$ ,  $\gamma = 0.09$ ,  $\sigma = 0.25$ ,  $d = 0.01$ ,  $r = 0.025$  per year and  $\mu = \frac{1}{70}$  per year. We vary the parameters  $\omega$  and  $\alpha$  in order to establish the effect of the vaccination to the disease transmission. Further we choose the following initial conditions  $S_0 = 0.93$ ,  $E_0 = 0.07$  and  $V_0 = I_0 = R_0 = 0$ . The results of the model show that increasing the vaccine efficacy lowers the herd immunity and vice versa but there exist a critical vaccine efficacy ( $\alpha_c$ ) below which the herd immunity cannot be achieved. We found that this critical vaccine efficacy  $\alpha_c = 71.4\%$  and if the vaccine efficacy is below this value then increasing the vaccination rate will lower the transmission rate but will not prevent the epidemic. Figures [2a](#) and [2b](#) show the propagation of the measles virus disease in the population in the absence of vaccination. As can be seen in figure [2b](#) in the absence of vaccination, the virus will invade the

population and infect more than 60% of the total susceptible population.

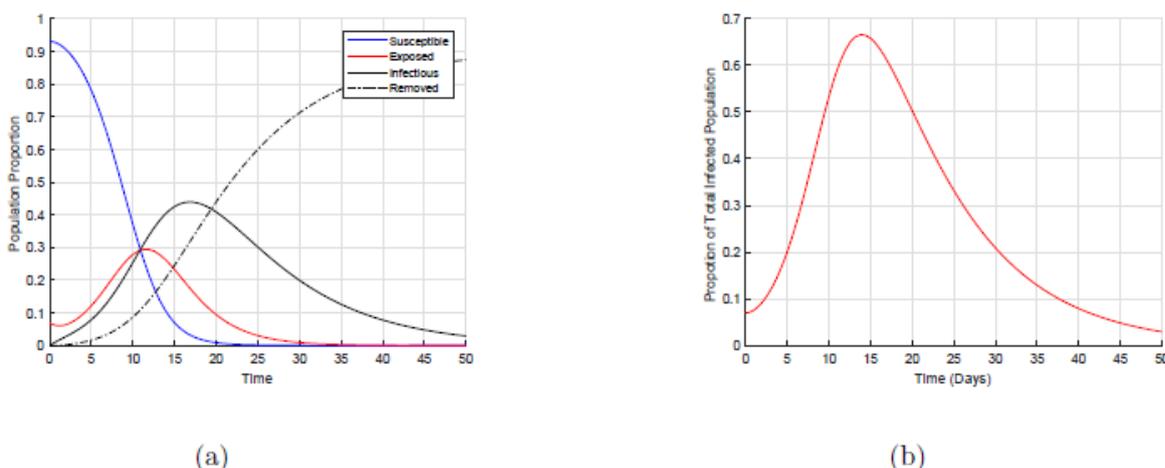


Figure 2: Transmission dynamics of Measles virus in the absence of vaccination

In figure 3a below vaccination is introduced to the population at a lower rate of 30% and with a vaccine whose efficacy is 60%. The results show that although the vaccination rate is low, there is significant decrease in the proportion of the population infected by the disease. In figure 3b, vaccination rate is increased to 90%. It is noted that increasing the vaccination rate greatly reduced the proportion of the infected population but was not sufficient to prevent the epidemic.

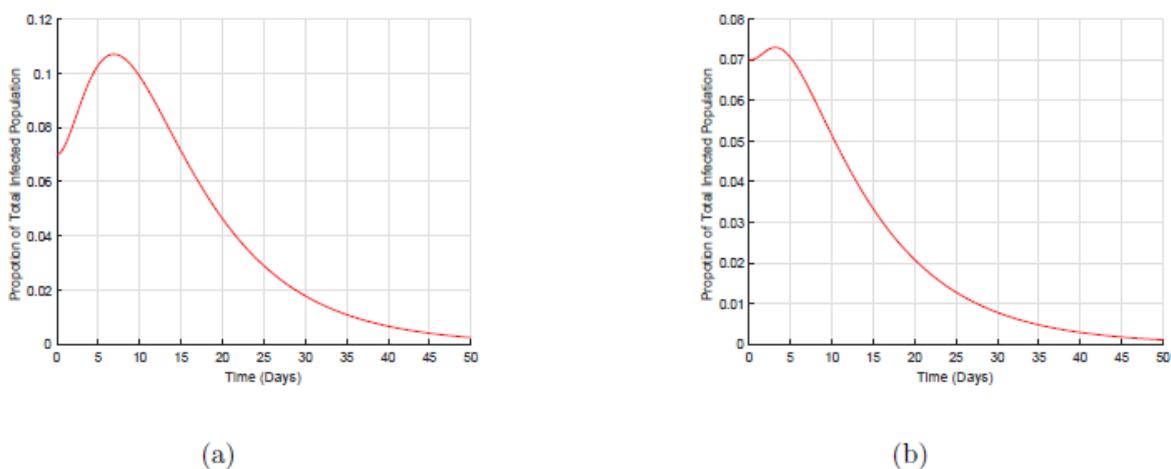


Figure 3: Transmission dynamics of Measles virus in the presence of vaccination with a vaccine whose efficacy is 60%

By increasing the vaccine efficacy to 85 % the herd immunity was achieved with a vaccination rate of 90%. This is demonstrated by figure 4.

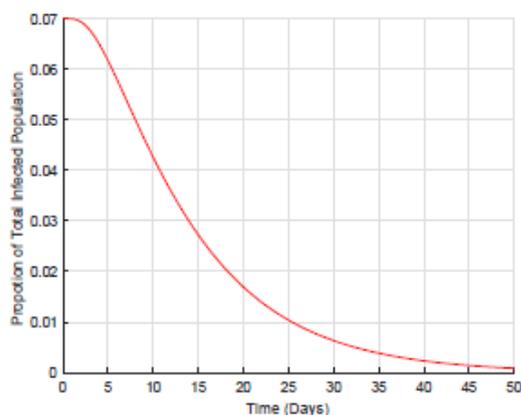
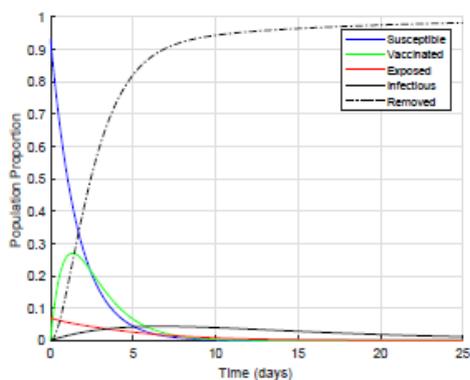
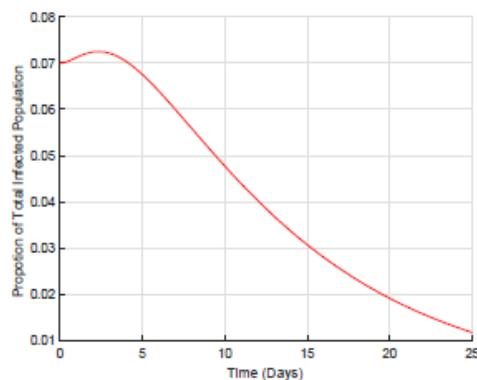


Figure 4: Transmission propagation for the virus when  $\omega = 0.90$  and  $\alpha = 0.85$

Further improvement of the vaccine efficacy to 93 % which is equivalent to the MMR (Single-dose) vaccine reduced the herd immunity to 89%. Figures 5a and 5b show the transmission dynamics of the disease when the vaccine is administered at a rate of 60%. From figure 5b it is observed that when the infectious individuals are introduced into the population, the number of infected individuals will first rise which indicates the outbreak of the disease. Figure 6a on the other hand shows the transmission dynamics of the disease when the vaccine is administered at an increased rate of 90%. The result show that the infected population decays as a function of time for all time thus there is no occurrence of the epidemic.



(a)



(b)

Figure 5: Transmission dynamics for the measles virus for  $\omega = 0.60$  and  $\alpha = 0.93$

When the vaccine efficacy was increased to 97% which is equivalent to a two-dose MMR vaccine then the herd immunity was attained at 87.9 %. These results are shown by figure 6b.

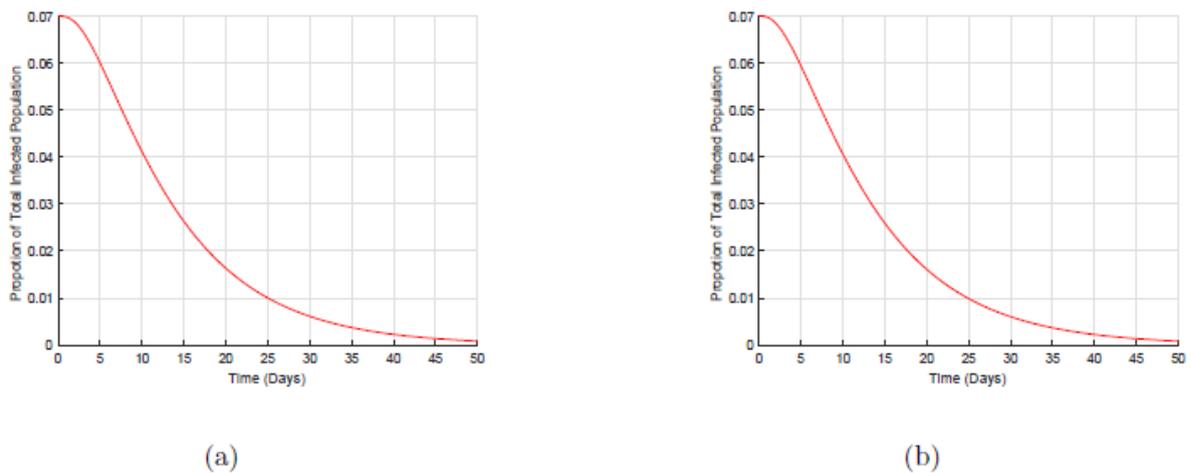


Figure 6: Transmission dynamics for the measles virus (a)  $\omega = 0.89$  and  $\alpha = 0.93$  and (b)  $\omega = 0.879$  and  $\alpha = 0.97$

### References

- [1]. Momoh A.A., Ibrahim M.O., Uwanta J.I., and Manga S.B. Mathematical model for control of measles epidemiology. *International Journal of Pure and Applied Mathematics*, 87(5):707–718, 2013.
- [2]. Mose Ongau Fred, Johana K. Sigey, Jeconiah A. Okello, M. Okwoyo James, and Giterere J. Kang’ethe. Mathematical modeling on the control of measles by vaccination: Case study of KISII County, Kenya. *The SIJ Transactions on Computer Science Engineering and its Applications (CSEA)*, 2(3), 2014.
- [3]. Diekmann O. and Heesterbeek J. A. P. *Mathematical epidemiology of infectious diseases: Model building, analysis and interpretation*. Chichester, UK: Wiley, 2000.
- [4]. Diekmann O., Heesterbeek J. A. P., and Roberts M. G. The construction of the next generation matrices for compartmental epidemic models. *Journal of Royal Society Interface*, 7:873–885, 2010.
- [5]. Christopher Obumneke, Ibrahim Isa Adamu, and Shamaki Timothy Ado. Mathematical model for the dynamics of measles under the combined effect of vaccination and measles therapy. *International Journal of Science and Technology*, 6(6):862–874, 2017.
- [6]. Kassem T.G. and Ndam J.N. A stochastic modeling of recurrent measles epidemics. *Science World Journal*, 3(4), 2010.