

Modeling the Spread of Measles with the Role of Vaccination and Treatment

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Abstract—Protection of children from vaccine-preventable diseases, such as measles, is among primary goal for health workers. Since vaccination and treatment has turned out to be the most effective method against childhood diseases, developing a framework that would predict an optimal vaccine coverage level needed to control the spread of these diseases is important and essential, with inclusion of treatment to those who have contracted the disease but not yet infectious. In this project, the use of a population with variable size to provide this framework was adopted. It majorly relied on a compartmental model expressed by a set of ordinary and partial differential equations based on the dynamics of measles infections. The numerical and qualitative analyses of the model were performed and different state variables were determined. Qualitative results show that the model has the disease-free equilibrium which is locally asymptotically stable when a certain threshold quantity is less than one and unstable when the quantity is more than one. Simulation of different epidemiological classes revealed that most of the individuals undergoing treatment join the recovered class.

Keywords—Basic Reproduction Ratio; Compartmental Model; Disease-Free Equilibrium; Herd Immunity; Mathematical Modeling; Measles; Second Opportunity; Therapeutic Treatment; Vaccination.

Abbreviations—Centre for Disease Control (CDC); Measles Mumps Rubella (MMR); Millennium Development Goal 4 (MDG4); Susceptible Exposed Infectives Removed (SEIR).

I. INTRODUCTION

THE Measles virus is a paramyxovirus, genus Morbillivirus. Measles is an infectious disease highly contagious respiratory disease through person-to-person transmission mode, with > 95% secondary attack rates among susceptible persons. It is the first and worst eruptive fever occurring during childhood. It produces also a characteristic red rash and can lead to serious and fatal complications including pneumonia, diarrheal and encephalitis. Many infected children subsequently suffer blindness, deafness or impaired vision. Measles confer lifelong immunity from further attacks [Murray, 10; Dhanabhakya et al., 17].

Measles is one of the leading causes of death among young children even though a safe and cost-effective vaccine is available [Anderson & May, 1; Mose et al., 9]. In 2016, there were 89 780 measles deaths globally – marking the first year measles deaths have fallen below 100 000 per year. Measles vaccination resulted in a 84% drop in measles deaths between 2000 and 2016 worldwide [Rost, 12; Trottier &

Philippe, 13]. In 2016, about 85% of the world's children received one dose of measles vaccine by their first birthday through routine health services – up from 72% in 2000. During 2000-2016, measles vaccination prevented an estimated 20.4 million deaths making measles vaccine one of the best buys in public health. CDC, (2017)

Worldwide; measles vaccination has been very effective, prevented by MMR (Measles, Mumps and Rubella) vaccine. In the last decade before the vaccine program an estimated 3-4 million people were infected yearly, of who 4000-5000 died, 48000 hospitalized and 1000 developed chronic disability from measles encephalitis. Although global incidence has been significantly reduced through vaccination and supplemental treatment, measles remains a public health problem, [Andrei, 2; Chamuchi et al., 3]. Since vaccination coverage is not uniformly high in Kenya, measles stands as the leading vaccine-preventable killer of many children in Africa; measles is estimated to have caused 614 000 global deaths annually in 2002, with more than half of measles deaths occur in sub-Saharan Africa, as given by WHO (2006), and Grais et al., [4].

The objectives of this research are to find threshold conditions that determine whether an infectious disease will spread or will die out into a population remains one of the fundamental questions of epidemiological modeling. For this purpose, there exists a key epidemiological quantity R_0 , the basic reproductive ratio. R_0 is the number of secondary cases that result from a single infectious individual in an entirely susceptible population. Introduced by Ross in 1909 and Hethcote [7], the current usage of R_0 is the following: if $R_0 < 1$, the modeled disease dies out, and if $R_0 > 1$, the disease spreads in the population. Reproductive ratios turned out to be an important factor in determining targets for vaccination coverage. In mathematical models, the reproductive number R_0 is determined by the dominant eigenvalue of the Jacobian matrix at the infection-free equilibrium for models in a finite-dimensional space.

The contributions of this manuscript are:

- a) To show that the spread of a disease largely depend on the contact rate, therefore, the National Measles Control Programme should emphasize on the improvement in early detection and treatment of measles cases so that the disease transmission can be minimized.
- b) To attain herd immunity level of 94.75% for the disease, mass vaccination exercise with supplemental treatment should be encouraged to cover the majority of the population whenever there is an outbreak of the disease in Kenya.
- c) To provide treatment to the exposed class so that it reduces the recovery period (shorten the infectious time) by providing the necessary drugs and required supportive care.

II. RELATED WORKS/LITERATURE SURVEY

Preparedness for, and measures to prevent outbreaks of measles (measles outbreak in Daadab camps) and diarrhoea diseases should be a priority, particularly if these poorly immunized populations are housed in overcrowded settings with limited water, sanitation and hygiene resources. According to public health risk assessment and interventions; the horn of Africa: Drought and famine crisis: July 2011.

In 2017, Researchers from CDC reported that the global goal to reduce measles deaths by 75% by 2015, compared with 2009 had been achieved. Firstly, Building on this accomplishment, in 2008 the World Health Organization summit on measles endorsed a target of 90% reduction in measles mortality by 2010, compared with 2000. Endemic transmission of measles virus was interrupted in the Kenyans in 2002, and four of the remaining five WHO regions (all except Southeast Asia) have set target dates for measles elimination by 2020 or earlier. Secondly, the establishment of a global measles eradication goal has been extensively discussed by the World Health Assembly and advisory committees to WHO and now hinges on progress towards regional elimination outside Africa. Monitoring measles mortality is relevant for all partners involved in child

survival. The fourth Millennium Development Goal (MDG4) aims to reduce deaths of children by two thirds by 2015 compared with 1990. The proportion of children vaccinated against measles was adopted as an indicator to measure progress towards MDG4; a rebound in measles deaths would pose a substantial threat to achieving this goal [15]. The rapid progress in measles control from 2000 to 2007 was based on implementation of recommended measles mortality reduction strategies, including increasing routine immunisation coverage, periodic Supplemental Immunisation Activities (SIAs—i.e., mass vaccination campaigns aimed at immunising 100 percent of a predefined population within several days or weeks), laboratory-supported surveillance, and appropriate management of measles cases as by WHO. Progress in Global measles control and mortality reduction 2008 Countries that have fully implemented and sustained these strategies have experienced reductions in measles cases of greater than 90 percent. However, not all countries have managed to do so, and several of the largest recorded outbreaks of the past decade were during 2009–10, WHO measles outbreaks (2011). Because most measles deaths are in countries where vital registration systems cannot provide reliable information on cause-specific mortality, WHO has relied on mathematical models to estimate the global burden of measles.

Previous models have not objectively incorporated measles surveillance data and instead relied on vaccination coverage data as the primary indicator of local disease burden. Consequently, these models could neither consistently capture the effects of large outbreaks on measles mortality where high vaccination coverage was not reported, nor show periods of low mortality between outbreaks when low vaccination coverage was reported. To assess progress towards the 2010 global measles mortality reduction goal, we developed a new model that, unlike previous models, uses surveillance data objectively to estimate both incidence and the age distribution of cases, accounts for herd immunity, and uses robust statistical methods to estimate uncertainty.

By 2011, all 194 WHO Member States had introduced or begun the process of introducing a two-dose measles vaccination strategy delivered through routine immunization services and/or SIAs. According to WHO and UNICEF estimates, global routine coverage with a first dose of measles vaccine (MCV1) increased from 72% in 2000 to 85% in 2010. WHO/UNICEF measles elimination, (2010). During this same period, coverage increased from 58% to 78% in the 47 countries with the highest burden of measles. By the end of 2010, the routine immunization schedules of 139 countries included two doses of measles-containing vaccine (MCV), and in 2011, GAVI supported 11 more countries to introduce a routine second dose of measles (MCV2). The timing of MCV2 serves as an important contact between the child and the Expanded Programme on Immunization (EPI) because it provides an opportunity to catch up on any missed vaccinations and deliver boosters, e.g. Diphtheria-Tetanus-Pertussis (DTP) vaccine to older age groups.

Overwhelming evidence demonstrates the benefit of providing universal access to measles and rubella-containing vaccines. Globally, an estimated 535 000 children died of measles in 2000. By 2010, the global push to improve vaccine coverage resulted in a 74% reduction in deaths. These efforts, supported by the Measles and Rubella Initiative, contributed 23% of the overall decline in under-five deaths between 1990 and 2008 and are driving progress towards meeting Millennium Development Goal 4 (MDG4) Measles and Rubella strategic plan 2012-2020 [Malfait et al., 11].

In the year 2000, the World Health Organization (WHO) estimated that 535 000 children died of measles, the majority in developing countries, and this burden accounted for 5% of all under five mortality, levels & trends in child mortality report 2011 [14]. In some developing countries, case-fatality rates for measles among young children may still reach 5–6%. In industrialized countries, approximately 10–30% of measles cases require hospitalization, and one in a thousand of these cases among children results in death from measles complications, as per [15, 16].

III. MEDICATION AND PREVENTION

3.1. Treatment and Drugs

These are measures taken to protect vulnerable individuals who have been exposed to the virus. They include

1. Post-exposure vaccination for non-immunized groups to be given measles vaccination within 72 hours of exposure to the measles virus (exposed) if the illness develops it lasts for shorter time
2. Immune serum globulin – pregnant women, infants and people with weakened immune system who are exposed to the virus may receive an injection of protein (antibodies) that can fight off the infection called immune serum globulin, which when given within six days of exposure to the virus, these antibodies can prevent measles or make symptoms less severe

3.2. Medication

1. Analgesia: these are over-the-counter medications such as acetaminophen (Tylenol) ibuprofen (advil, motrin etc) or naproven (aleve) to help relieve the fever that accompanies measles.
2. Antibiotics: if a bacterial infection such as pneumonia or an ear infection develops only while the child has measles consult the doctor for prescription.
3. Isolation: because measles is a highly contagious from about four days before to four days after the rash breaks out, people with measles should be isolated to avoid more infections.
4. Oral vitamin A – give two doses of it to the child.
5. For hospitalized cases give supportive care.

IV. METHODS/DISCUSSION

In this problem a deterministic, compartmental, mathematical model is used to describe the transmission dynamics of measles as by Hethcote & Waltman [5, 6, 7]. The population is homogeneously mixing and reflects the demography of a typical developing country like Kenya, as it experiments an exponential increasing dynamics. Compartments with labels such as S , E , I , and R are often used for the epidemiological classes. These four equations represent four different groups of people: the Susceptibles, the Exposed, the Infectives, and the Recovered. The Susceptibles (" 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 infectives, the Infectives (" I ") are people who have become infected with measles and are able to transmit the disease, and the Recovered, R are people who have recovered from the disease. Note that $S+E+I+R=N$, where N being the total population is constant.

In all, the assumption is that the population is a value somewhere between zero and N , with N meaning the population is at full capacity. It is further assumed that all individuals are equally likely to be infected by the infectious individuals in a case of contact except those who are immune. The undetected or late detected infectious individuals are the ones contributing to disease transmission and spread. Those detected are isolated to the hospital for immediate treatment and education. It is further assumed that those recovered become immune and they get some form of education about the transmission of the measles. The transmission of the measles within the compartment is negligible. It's further assumed that there is no treatment failure in the compartment, therefore a patient will either recover or dies.

Table 1: Variables and Definitions of Sub-Populations used as Variables

Variable	Definition
$S(t)$	The number of susceptibles at time, t
$E(t)$	The number of Exposed at time, t
$I(t)$	The number of Infected at time, t
$R(t)$	The number of Removed at time, t

Table 2: Parameter and their Definitions

Parameter	Definition
B	Birth rate
μ	Mortality rate
	Contact rate
–	Average latent rate
–	Average infectious period
	Proportion of those successively vaccinated at birth
	Differential mortality due to measles
	Proportion of those successively treated after being infected

4.1. SEIR Model without Vaccination and Treatment

The differential equations for this model are;

$$\frac{dS}{dt} = bN(t) - \beta S(t)I(t) - \mu S(t) \quad (1)$$

$$\frac{dE}{dt} = \beta S(t)I(t) - (\sigma + \mu)E(t) \quad (2)$$

$$\frac{dI}{dt} = \sigma E(t) - (\gamma + \delta)I(t) \quad (3)$$

$$\frac{dR}{dt} = \gamma I(t) - \mu R(t) \quad (4)$$

$$N = S + E + I + R, \text{ and } \frac{dN}{dt} = 0, \text{ and } N \text{ is thus constant.} \quad (5)$$

4.1.1. Properties of the SEIR Model Equations

The basic properties of the of the model equations 1-4 are feasible solutions and positivity of solutions. The feasible solution shows the region in which the solution of the equations are biologically meaningful and the positivity of the solutions describes the non-negativity of the solutions of the equations 1-4.

4.1.2. Feasible Solution

The feasible solution set which is positively invariant set of the model is given by,

$$\{(S, E, I, R) \in \mathbb{R}_+^4\}$$

From the Model Equations 1-4 it will be shown that the region is positively invariant. Considering the steps below from the Model equations, the total population of individuals is given by

Therefore adding the differential equations 1-4, the results becomes

$$\frac{dN}{dt} = (b - \mu)N$$

4.2. SEIR Model with Vaccination and Treatment

In Kenya, vaccination against measles consists of one dose of standard titer Schwarz vaccine given to infants after early age. Nevertheless, during epidemics an early two-dose strategy is implemented: at different times, as given by Kaninda et al., [8] and Grais et al., [4]. Before these times, suppose that children gain protection from the maternal antibodies. Taking into account this schedule of p vaccination, the differential equations for this deterministic model are as follows from figure 1:

$$\frac{dS}{dt} = b(1 - p)N(t) - \beta S(t)I(t) - \mu S(t) \quad (5)$$

$$\frac{dE}{dt} = \beta S(t)I(t) - (\sigma + \omega)E(t) \quad (6)$$

$$\frac{dI}{dt} = \sigma E(t) - (\gamma + \delta)I(t) \quad (7)$$

$$\frac{dR}{dt} = \gamma I(t) - \mu R(t) \quad (8)$$

$$\frac{dN}{dt} = 0, \text{ and } N = S + E + I + R \text{ is thus constant.} \quad (8)$$

From the Model equations, the total population of individuals is given by

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V. METHOD OF SOLUTION

A first-order linear differential equation of the form

$$\frac{dN}{dt} = (b - \mu)N$$

$$\text{Thus } N(t) = Ce^{(b-\mu)t} \text{ at } t = 0, N(0)$$

Hence the solution of the linear differential equation then becomes

$$N(t) = N(0)e^{(b-\mu)t}$$

Therefore, $\emptyset$ is positively invariant.

5.1. Positivity of Solutions

It can be proved that all the variables in the model equations 1-4 are non-negative.

Let the initial data set be $(S, E, I, R)(0) \geq 0 \in \emptyset$, then the solution set $(S, E, I, R)(t)$

Of the equations 1-4 is positive for all $t > 0$.

5.1.1. Proof

From equation 1 we have that, $S(t) = S(0)e^{-(\mu - \beta I(t))t} \geq 0$, since $(\mu - \beta I(t)) \geq 0$

From equation 2, $E(t) = E(0)e^{-(\sigma + \mu)t} \geq 0$, since $(\mu + \sigma) > 0$.

From equation 3, $I(t) = I(0)e^{-(\gamma + \delta)t} \geq 0$ since $(\gamma + \delta) > 0$.

From equation 4, is obtained $R(t) = R(0)e^{-\mu t} \geq 0$ since $\mu > 0$.

5.2. Existence of Steady States of the System

The equilibrium points of the system can be obtained by equating the rate of changes to zero.

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

5.3. Global Asymptotic Stability of the Model

In proving the global stability of the SEIR Model, there is need to find the equilibrium points of the system 5-8.

Assuming that the birth rate, b is equal to death rate, μ i.e $\mu = b$.

$$\frac{b(1 - p)N - (\sigma + \omega)E}{\mu(1 - p)N - (\sigma + \mu + \omega)E} = 0 \quad (9)$$

If the system is set to zero, will give

$$\frac{(\gamma + \delta)I}{\mu(1 - p)N - (\sigma + \mu + \omega)E} = 0 \quad (10)$$

$$\frac{\mu(1 - p)N - (\sigma + \mu + \omega)E}{(\gamma + \delta)I} = 0 \quad (11)$$

Since the birth rate is equal to death rate i.e.

Thus

$$\frac{\mu(1-p)}{(\sigma+\mu+\omega)} \quad \frac{N(\gamma-\delta)}{(\gamma-\delta)(\sigma+\omega)} \quad (12)$$

Thus the disease free equilibrium Z_0 is $Z_0 = (1, 0, 0, 0)$

Consider S from equation 9 and E from equation 10 i.e.

$$\frac{b(1-p)N - (\sigma + \mu + \omega)E}{(\gamma - \delta)(\sigma + \omega)}$$

$$\frac{\mu(1-p)}{(\sigma + \mu + \omega)} \quad \frac{N(\gamma - \delta)}{(\gamma - \delta)(\sigma + \omega)}$$

Using

$$\frac{(\gamma - \delta)(\sigma + \omega)}{(\gamma - \delta)(\sigma + \omega)}$$

Therefore, — and $\frac{(\gamma - \delta)(\sigma + \omega)}{(\gamma - \delta)(\sigma + \omega)} \left[1 - \right]$

And $\left[1 - \right]$ and $\frac{(\gamma - \delta)(\sigma + \omega)}{(\gamma - \delta)(\sigma + \omega)} \left[1 - \right]$

This corresponds to an endemic steady state with constant number of people in the population being infected with the disease.

This is biologically reasonable when $S^* < N$, that is when

$$\frac{(\gamma + \mu + \delta)(\sigma + \mu + \omega)}{(\gamma + \mu + \delta)(\sigma + \mu + \omega)}$$

where R_0 is the basic reproduction ratio of the infection.

The local stability may be determined from eigenvalues of the Jacobian matrix of the model equations (5) - (8). The Jacobian of Equations (5-8) at the equilibrium point (S^*, E^*, I^*, R^*) is

$$\begin{pmatrix} b(1-p) - \frac{(\gamma + \mu + \delta)(\sigma + \mu + \omega)}{(\gamma + \mu + \delta)(\sigma + \mu + \omega)} & -\frac{(\gamma + \mu + \delta)(\sigma + \mu + \omega)}{(\gamma + \mu + \delta)(\sigma + \mu + \omega)} \\ b(1-p) & -(\sigma + \mu + \omega) \\ b(1-p) - \frac{(\gamma + \mu + \delta)(\sigma + \mu + \omega)}{(\gamma + \mu + \delta)(\sigma + \mu + \omega)} & -\frac{(\gamma + \mu + \delta)(\sigma + \mu + \omega)}{(\gamma + \mu + \delta)(\sigma + \mu + \omega)} \\ b(1-p) & 0 \end{pmatrix} \quad (13)$$

5.4. Initial Conditions

The mathematical formulation of the epidemic is completed given the initial conditions:

- (i). $S(0) = S_0 > 0$ (ii). $E(0) = E_0 > 0$
- (iii). $I(0) = I_0 > 0$ (iv). $R(0) = R_0 > 0$ for all $t > 0$.

In absence of infection $E^* = I^* = 0$, the Jacobian of (5-8) at the disease-free equilibrium

$\varepsilon_0 = (S^*, 0, 0, R^*)$ is

$$\begin{pmatrix} b(1-p) - \frac{(\gamma + \mu + \delta)(\sigma + \mu + \omega)}{(\gamma + \mu + \delta)(\sigma + \mu + \omega)} & -\frac{(\gamma + \mu + \delta)(\sigma + \mu + \omega)}{(\gamma + \mu + \delta)(\sigma + \mu + \omega)} \\ b(1-p) & -(\sigma + \mu + \omega) \\ b(1-p) - \frac{\beta b(1-p)}{\beta} \frac{S^*}{0} & -(\gamma + \mu + \delta) \\ b(1-p) & 0 \end{pmatrix} \quad (14)$$

Its eigenvalues are $\lambda_1 = -\mu$, $\lambda_2 = -(b - \mu)$ and the roots of

$$+ (2 \quad \frac{\delta X}{\beta b(1-p)} \quad \frac{(\sigma - \mu)(\gamma - \delta)}{(\gamma - \delta)(\sigma + \omega)}) \quad (15)$$

The disease-free equilibrium ε_0 is locally stable if $R_p < 1$ and unstable if $R_p > 1$ where

$$(1-p) \frac{(\gamma - \delta)(\sigma + \omega)}{\mu(\sigma + \omega)(\gamma - \delta)} \quad (16)$$

The eigenvalues at the disease „free equilibrium“ are given by $\{-\mu, -(\mu + \lambda), -(\mu + \alpha), -\mu\}$. All the eigenvalues being negative means that the disease-free equilibrium is asymptotically stable. The basic reproductive number R_0 can be computed by

$R_0 \times S^* = 1$, Therefore, $R_0 = \frac{1}{S^*}$, where $(\mu + \alpha)(\mu + \lambda) \neq 0$

This means the transmission rate, i.e., the rate at which exposed become infected and the contact rate, that is the average number of effective contacts with other (susceptible) individuals per infective per unit time relative to the rate at which an infectious individuals recovered per unit time play an important role in determining whether or not an epidemic will occur. Hence the disease free equilibrium $(1, 0, 0, 0)$ is locally asymptotically stable provided that $R_0 < 1$, that is, $\lambda \beta < (\mu + \alpha + \omega)(\mu + \lambda)$. Where as if $R_0 > 1$, then the disease free equilibrium is unstable, that is the system is said to be uniformly persistent, in other words the disease is endemic. Thus, R_0 is a threshold parameter for the model. As λ_1 and λ_2 are negative, it remains to prove that λ_3 and λ_4 , the roots of the quadratic part of that characteristic polynomial of J^* are both negative. Using Routh-Hurwitz theorem, it is the case when

and (17)

As $-(2 \quad \frac{\delta X}{\beta b(1-p)} \quad \frac{(\sigma - \mu)(\gamma - \delta)}{(\gamma - \delta)(\sigma + \omega)})$ is true (18)

From $(\sigma + \omega)(\gamma - \delta) > \beta b(1-p)\mu$ (19)

Remark: • R_p is the effective reproduction number in presence of vaccination.

• If $p = 0$, we have the basic reproductive number (20)

$$\frac{(\gamma - \delta)(\sigma + \omega)}{\mu(\sigma + \omega)(\gamma - \delta)}$$

VI. RESULTS/PROBLEMS AND SOLUTIONS

This part gives an illustration of the analytical results of the model by carrying out stability analysis and numerical simulations of the model using the parameter values pertinent to Kenya in Kisii County in 2016. These parameters were obtained from different sources in the medical field literature (KEMRI 2013; Kisii level 6 hospital Immunization Action Programme, 2013; Ministry of health-Kisii level 6 2015).

Parameter Symbol	Parameter Value	Literature Source
B	0.02755 per year	KEMRI 2016, Kisii level 6 hospital
	0.00875 per year	KEMRI 2016, Kisii level 6 hospital
	0.09091 per day	IMMUNIZATION PROGRAMME-KISII COUNTY-2016
	0.125 per day	Ministry of health-Kisii level 6 2015
	0.14286 per day	Ministry of health-Kisii level 6 2015
	0.1175 per day	Ministry of health-Kisii level 6 2015

6.1. Stability Analysis of the Model - Endemic Model

$$\begin{array}{c} \text{---} \quad (\mu \quad \beta i)s \\ \text{---} \quad (\mu \quad \omega)e \\ \text{---} \quad (\mu \quad \delta)i \\ \text{---} \quad (\gamma + \delta)i \end{array}$$

Linearising the system of the differential equations, the Jacobian matrix is given as

$$J(s \quad r) = \begin{bmatrix} \text{---} & \text{---} \\ \text{---} & \text{---} \end{bmatrix}$$

For the infection free equilibrium $(s \quad i) = (1 \quad 0)$, the Jacobian matrix then becomes

$$J(1 \quad 0) = \begin{bmatrix} \text{---} & \text{---} \\ \text{---} & \text{---} \end{bmatrix}$$

The important sub-matrix is the second 2x2 matrix. From this, the trace $(T) < 0$, but if $R_0 < 1$, then the determinant $(D) > 0$ and if $R_0 > 1$ then $(D) < 0$ for all parameters.

Routh-Hurwitz stability condition for T and D is given as follows:

$$\begin{array}{c} \text{---}(2 \quad \alpha) \\ (\mu + \alpha)(\mu + \sigma + \omega)(1 - R_0) \end{array}$$

Hence the disease free study state when $\alpha = 0$, and unstable when $\alpha > 0$. The eigenvalues at the disease free equilibrium are given by $\{- (\mu + \sigma + \omega), -(\mu + \alpha)\}$. All the eigenvalues are negative meaning the disease free equilibrium $(1, 0, 0, 0)$ is asymptotically stable.

The endemic equilibrium

$$(s \quad *) = \left(\text{---} \frac{\mu(R \quad 1)}{(\mu \quad \omega)} \frac{\mu(R \quad 1)}{(\mu \quad \omega)} \right)$$

The Jacobian matrix for the endemic equilibrium is given as

$$\begin{pmatrix} \mu(R \quad 1) & \text{---}(\mu \quad \omega) & \text{---}(\mu \quad \alpha)(\mu \quad \omega) \\ \mu(R \quad 1) & \text{---}(\mu \quad \omega) & \text{---}(\mu \quad \alpha)(\mu \quad \omega) \\ \text{---}(\mu \quad \alpha) & \text{---}(\mu \quad \alpha) & \text{---}(\mu \quad \alpha) \end{pmatrix}$$

Characteristic equation is given as $X^3 + a_1X^2 + a_2X + a_3 = 0$, where

$$\begin{array}{l} a_1 = (2 + R_0)\mu, \\ a_2 = (2\mu + \sigma + \alpha), \\ a_3 = \mu(R_0 - 1)[\mu 2 - \mu(\sigma + \alpha) + \sigma\alpha] \end{array}$$

If $a_1 > 0$, $a_2 > 0$ and $a_1a_2 - a_3 > 0$ are true then from the Routh-Hurwitz criteria for stability, all the roots of the Characteristic equation have negative real part which means stable equilibrium.

6.2. Numerical Simulations of the Model Equations

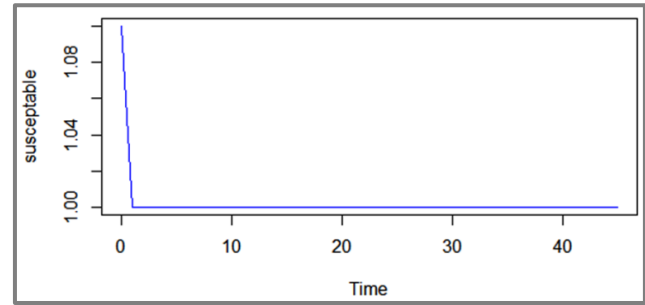


Figure 2: Simulation of the Susceptible Population

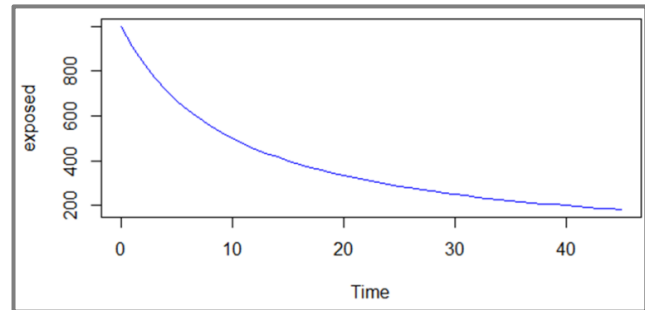


Figure 3: Simulation of the Exposed Population

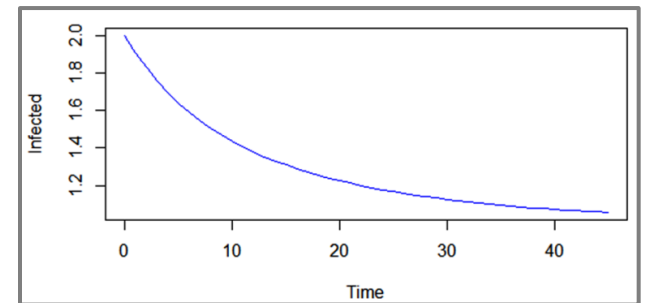


Figure 4: Simulation of the Infected Population

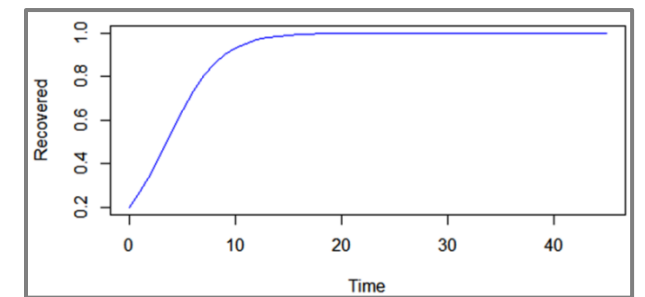


Figure 5: Simulation of the Recovered Population

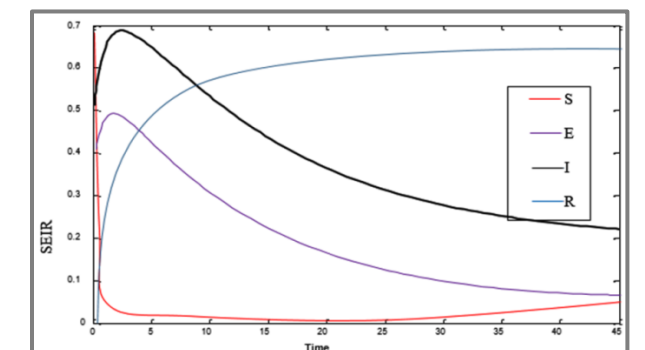


Figure 6: Simulation of the Combined Susceptible, Exposed, Infected and Recovered Populations

From the equations initial value problems IVP equations (9)-(12) the solutions $S(t)$, $E(t)$, $I(t)$, $R(t)$ and the combined graph of both the four are displayed graphically in figures 2 to 6

6.3. Analysis of the Simulations

In figure 2, the susceptible population decreases as time increases. This decrease may be possibly because of the high rate of recovery due to mass vaccination, since individuals become permanently immune upon recovery. The contact rate also has large impact on the spread of a disease through a population. The higher the rates of contact, the more rapid the spread of the disease, it is also observed that as the contact rate decreases, the fraction of individuals infected decreases at a faster rate as would be expected logically. It can also be observed that as the rate increases, the population of exposed individuals shows some rapid decrease after the earlier intervals of rise. The decrease in the exposed population could be due to early detection and also possibly due to those who enter the infective class. This decrease could also be due to the education about the measles transmission, very few individuals are coming out as infected individuals. Also the dynamics of the exposed population depend on the contact number.

In figure 3, it is realized that the population of infected individuals at the very beginning rise sharply as the rate increases and then fall uniformly as time increases. This rapid decline of the infected individuals may be due to early detection of the measles and partly due to those who revert to the Exposed class. This graph also demonstrates that the contact rate has large impact on the spread of the disease through population. If the contact rate is observed to be high then the rate of infection of the disease will also be high as would be expected logically. However, there exists another parameter to consider as more individuals are infected with the disease and $I(t)$ grows, as some individuals are leaving the infected class by being cured and joining the recovered class.

In figure 4, it is realized that the number of people recovering increases steadily as rate increases.

This may be due to early detection of the disease as well as education about the measles transmission. It can also be observed that the population of the recovered individuals rise up steadily for some number of years and then drops and remains nearly a constant. This could be due to the greater number of infectious individuals who have been treated and also acquired education about the measles transmission.

VII. CONCLUSION AND FUTURE WORK

The conclusion and recommendations are offered to stakeholders, the Kenyan central government, public health agencies health care providers and the Kisii county government to enable them determine how best to allocate scarce resources for measles prevention and treatment in the country.

7.1. Conclusion

The model has shown success in attempting to predict the causes and reason for rapid spread of measles transmission within the Kisii County a population. The model strongly indicated that the spread of a disease largely depend on the contact rates with infected individuals within a population.

From the model the herd immunity level for measles in the county was found to be 94.75%. It is also realized that if the proportion of the population that is immune exceeds the herd immunity level for the disease, then the disease can no longer persist in the population. Thus if this level can be exceeded by mass vaccination, then the disease can be eliminated.

The model also pointed out that early detection and therapeutic treatment has a positive impact on the reduction of measles transmission; that is there is a need to detect new cases as early as possible so as to provide early treatment for the disease. More people should be educated in order create awareness to the disease so that the community will be aware of the deadly measles disease.

7.2. Recommendations

Eradication of contagious diseases such as measles has remained one of the biggest challenge facing developing countries like Kisii. It is realized that the herd immunity level for the disease which is 94.75% is high, and mostly when there is an outbreak of the disease, and there is an introduction of a mass vaccination programme which can cover about 97% of the population, not everybody will be immune because vaccine efficacy is usually not 100%. It therefore means that part of the population will be immune and others will be vaccinated but not immune. Therefore there is an urgent need for Health Ministry to come up with some new control strategies and more efficient ones to fight the spread of the disease in the country.

Therefore; from the outcome of the results,

- The model shows that the spread of a disease largely depend on the contact rate, therefore, the National Measles Control Programme should emphasize on the improvement in early detection of measles cases so that the disease transmission can be minimized.
- To attain herd immunity level of 94.75% for the disease, mass vaccination exercise should be encouraged to cover the majority of the population whenever there is an outbreak of the disease in Kisii county, Kenya.

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