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Analysis of a Mathematical Vaccination Model of an Infectious Measles Disease

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Abstract

The analysis of a mathematical vaccination model of an infectious Measles disease was carried out in this paper. In the SVEIR model, epidemic of the disease was investigated in which the incidence rate and epidemic were considered. After the model was formulated, the analytical application of the formulated model showed that the model has two equilibrium points (disease free equilibrium (DFE) and endemic equilibrium (EE)). Using our model, we proved the positivity of solutions and then obtained the basic reproduction number for determining whether the disease dies out completely or not. The local stability of disease-free equilibrium was proved, and determined with the help of the basic reproduction number. Using Lyapunov function method, it was proved that the DFE and EE were globally asymptotically stable. Furthermore, numerical simulations were carried out on the model to make the analytical studies easy to understand. Simulation results show that the number of susceptible, infected and vaccinated individuals is consistent with theoretical analysis (as could be seen in the attached graphs) and the results are presented and discussed quantitatively to illustrate the solution.

Keywords: Deterministic Model, Disease free equilibrium, SVEIR, Basic reproduction number, Global Stability

1. Introduction

A lot of relational factors has made the spread of infectious diseases a complex phenomenon with many interacting factors, e.g., the type of environment in which the pathogen and hosts are situated, and the intra and inter-dynamics of the population it is exposed to. The role of mathematical model is to model the beginning and spread of diseases. A common method of doing these is to use the idea of dividing the model into compartments under certain assumptions, which represent their status of the health with respect to the pathogen in the system. Most of the models in mathematical epidemiology are compartmental models, with the population being divided into compartments and assumptions about the nature and time rate of transfer from one compartment to another.

Many researchers have worked on the modeling and analysis of infectious disease. In particular, a deterministic and stochastic model of the dynamics of drug Resistant

Tuberculosis was discussed by Umana, *et al* (2016). The assumption that exposed individuals will develop active tuberculosis due to endogenous reactivation and exogenous re-infection was made. Ochoche *et al* (2014) also discussed an SVEIR mathematical model of measles with vaccination and two phases of infectiousness (that is, asymptomatic infectives and symptomatic infectives). Even though vaccination was considered in their model, but global stability was not shown. A Mathematical Model (VSEIR) for control and elimination of the transmission dynamics of Measles was discussed by Stephen *et al* (2015). They focused on the mathematical model for control and removal of transmission dynamics of Measles. Also, Safi *et al* (2012) worked on an SEIR model which is a deterministic model for the transmission dynamics of a communicable disease. It was developed and rigorously analyzed. In their model, Lyapunov functions were constructed to prove the asymptotic global stability of disease free equilibrium and endemic equilibrium point.

Momoh *et al* (2013) developed a mathematical model for control of measles epidemiology. They used SEIR model to determine the impact of exposed individuals at latent period through the stability analysis and numerical simulation. Panjeti *et al* (2011) discussed a mathematical model for rabies. The model was based on a simple susceptible-infectious-removed (SIR) compartment models of fox rabies emergence and spread.
The model incorporated dynamics across heterogeneous landscapes, host demographic variation, and environmental stochastic nature.

The spread of carrier dependent infectious diseases with environmental effects using variable carrier population was studied by Ghosh *et al* (2004). The density observed that the carrier population increases as the human population density increases. With increase in human population density, the effects of human population related factors like discharge of household wastes, open sewage drainage, and industrial efficient in residential areas, open water storage tanks and ponds etc. leads to further growth of carrier population density.

Mossong *et al* (2003) studied the re-emergence of Measles as a result of waning of immunity in vaccinated individuals. Their model was based on age structure. One of the major understanding from their model is that waning of immunity and subsequent mild subclinical infection in vaccines would not necessarily result in a rapid re-emergence of measles.
Also, Singh *et al* (2003) Studied the spread of malaria by putting into account mosquito population density governed by a generalized logistic model. They showed that the outbreak of these infectious diseases cause death of millions of individuals as well as expenditure of huge amount of money in health care and disease control.

Siabouh *et al* (2013) studied an SEIR model for the transmission dynamics of measles in Cape Coast Metropolis, Ghana. Their study showed that the disease will die out if part of the population that is immune is more than the herd immunity level for the disease.

More recently, Nudee *et al* (2019) developed a deterministic model for the transmission dynamics of measles, incorporating logistic growth rate and vaccination. Other researchers on measles include Omame and Inyama (2014), Bai and Liu (2015), Ntirampeba *et al* (2017) and many others.

This model seeks to rigorously assess the impact of vaccination on the control and dynamics of measles disease.

2. Model Formulation

2.1. Assumptions of the Model

The assumptions of the model are stated below:

1. We assume the standard incidence rate $\frac{SI}{N}$
2. People in the Vaccinated compartment can be infected.
3. People can be get the disease only through contacts with infectious people except those who are immune.
4. Recovered individuals are automatically immune.
5. The population is homogeneously mixed (A population that interacts with one another to the same degree and Fixed)

2.2. Flow Diagram of the Model

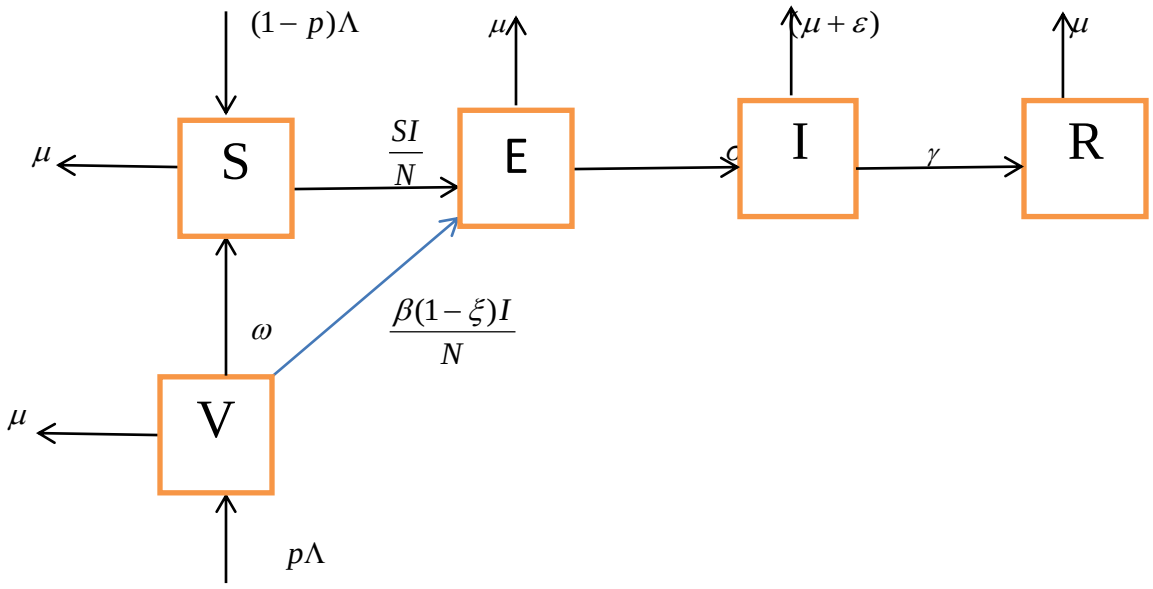

Fig. 2.1: Flow Diagram of the Model

2.3. Symbols and Parameter of the Model

Below are the symbols and parameters of the proposed model

Symbols and Parameters	Description
S	Population of the susceptible Individuals at time t
V	Population of Vaccinated Individuals at time t
E	The number of exposed individuals at time t
I	The number of infectious individuals at time t
R	The number of recovered individuals at time t
μ	Natural death rate
β	Contact rate
$\Re_0$	Basic reproduction number
ξ	Effectiveness of vaccine
N	Total population
P	Fraction of recruited individuals
σ	The rate at which exposed individuals become

	infectious
γ	The rate at which infected individuals recover.
ω	The rate at which vaccines wane
Λ	Recruitment rate into the susceptible compartment
$\lambda = \frac{\beta I}{N}$	Standard force of infection

Note,

$$N(t) = S(t) + V(t) + E(t) + I(t) + R(t)$$

Using the Assumptions, flow diagram, the symbols and parameters stated above, we write the proposed model as:

$$\left.\begin{aligned}
\frac{dS}{dt} &= (1-p)\Lambda + \omega\Lambda - \frac{\beta I}{N}S - \mu S \\
\frac{dV}{dt} &= p\Lambda - \frac{\beta(1-\xi)VI}{N} - (\mu+\omega)V \\
\frac{dE}{dt} &= \frac{\beta IS}{N} + \frac{\beta(1-\xi)VI}{N} - (\mu+\sigma)E \\
\frac{dI}{dt} &= \sigma E - (\mu+\varepsilon+\gamma)I \\
\frac{dR}{dt} &= \gamma I - \mu R \\
where\ \lambda &= \frac{\beta I}{N}\ which\ is\ the\ standard\ \ force\ of\ \ infection
\end{aligned}\right\} \qquad (2.1)$$

3. Analysis of the Model

3.1. Basic Properties of the Model

3.1.1. Boundedness of Solutions

The model will be analyzed in a biologically feasible region as follows. We first show that the system is bounded (that is, all feasible solutions are uniformly-bounded) in a proper subset $D \subset \Re_+^5$.

Theorem 3.1: Consider the closed set, $D = \left\{(S,V,E,I,R) \varepsilon \Re_+^5 : S+V+E+I+R \le \frac{\Lambda}{\mu}\right\}$,

D is positively invariant.

Proof: Note that $N(t) = S(t) + V(t) + E(t) + I(t) + R(t)$ and therefore

$$\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dV(t)}{dt} + \frac{dE(t)}{dt} + \frac{dI(t)}{dt} + \frac{dR(t)}{dt}$$

$$\Rightarrow \frac{dN(t)}{dt} = (1-p)\Lambda + \omega\Lambda - \frac{\beta I}{N}S - \mu S + p\Lambda - \frac{\beta(1-\xi)VI}{N} - (\mu+\omega)V + \frac{\beta IS}{N} + \frac{\beta(1-\xi)VI}{N} - (\mu+\sigma)E +$$

$$\sigma E - (\mu+\varepsilon+\gamma)I + \gamma I - \mu R$$

$$\frac{dN(t)}{dt} = \Lambda - \mu S - \mu V - \mu E - \mu I - \varepsilon I - \mu R = \Lambda - \mu(S+V+E+I+R) + \varepsilon I$$

$$\frac{dN(t)}{dt} = \Lambda - \mu N - \varepsilon I$$

In the absence of the disease, $\varepsilon = 0$, hence,

$$\frac{dN(t)}{dt} \le \Lambda - \mu N \Rightarrow \frac{dN(t)}{dt} + \mu N \le \Lambda$$

Solving the above we have

$$d[Ne^{\mu t}) \le \Lambda e^{\mu t} dt \Rightarrow Ne^{\mu t} \le \frac{\Lambda}{\mu} e^{\mu t} + c$$

$$\Rightarrow N(t) \le \frac{\Lambda}{\mu} + ce^{-\mu t}$$

$$At\ t = 0,\ N(0) = N_0$$

$$\Rightarrow N(0) \le \frac{\Lambda}{\mu} + c \Rightarrow c > N_0 - \frac{\Lambda}{\mu}$$

$$\therefore\ N(t) \le e^{-\mu t}N_0 + \frac{\Lambda}{\mu}(1-e^{-\mu t})$$

As $t \to \infty,\ N(t) \le \frac{\Lambda}{\mu}$

Hence, $N(t) \le \frac{\Lambda}{\mu}\ if\ N(0) \le \frac{\Lambda}{\mu}$

Thus, the region D is positively invariant. Also, if $N(0) > \frac{\Lambda}{\mu}$, then either the solutions of the model (2.1) enters D at finite time or N(t) approaches $\frac{\Lambda}{\mu}$ asymptotically. Hence, the region D attracts all solutions in $\Re_+^5$

Thus the region D is positively invariant, it is sufficient to consider the dynamics of the flow diagram that generated the model (2.1) in D, where the usual existence, uniqueness, continuation results hold for the system.

3.1.2. Positivity of Solutions

For the model equation to be epidemiologically meaningful, it is important to show that all its state variables are non-negative for all time. In other words, solutions of the model system with positive initial data will remain positive for all time t > 0.

Theorem 3.2: Let the initial data be $S(0),V(0),E(0),I(0),R(0)$. Then the set of solutions $\Omega=\{S(t),V(t),E(t),I(t),R(t)\}$ of

the model are positive for all time $t>0$

Proof: Let $t_1 = Sup\{S(t)>0,V(t)>0,E(t)>0,I(t)>0,R(t>0)\}$

Thus,

$t_1>0$. It follows, from the second equation of (2.1) given as,

$$\frac{dV}{dt}=p\Lambda-\frac{\beta(1-\xi)VI}{N}-(\mu+\omega)V$$

Which becomes,

$$\frac{dV}{dt}=p\Lambda-\lambda(1-\xi)V-(\mu+\omega)V \Rightarrow \frac{dV}{dt}+\left[\lambda(1-\xi)+(\mu+\omega)\right]V=p\Lambda \qquad (3.1)$$

(3.1) is a linear differential equation, hence we shall solve by finding the integrating factor which is given as;

$$\text{Integrating factor}=\exp\left[\int_0^t\left(\lambda(1-\xi)+(\mu+\omega)dt\right]=\exp\left[\lambda(1-\xi)+(\mu+\omega)\right]\int_0^t\lambda(\tau)d\tau$$

$$\therefore \frac{d}{dt}\left\{V(t)\exp\left[(\mu+\omega)+\lambda(1-\xi)\int_0^t\lambda(\tau)d\tau\right]\right\}=p\Lambda\exp\left[(\mu+\omega)+(1-\xi)\int_0^t\lambda(\tau)d\tau\right] \qquad (3.2)$$

Now, if we integrate both sides of (3.2) from $_o$ to t1, we shall obtain:

$$V(t)\exp\left[(\mu+\omega)+(1-\xi)\int_0^t\lambda(\tau)d\tau\right]=\int_0^t p\Lambda\exp\left[(\mu+\omega+(1-\xi)\int_0^t\lambda(\tau)d\tau\right]$$

$$\therefore V(t)\exp\left[(\mu+\omega)t+(1-\xi)\int_0^t\lambda(\tau)d\tau\right]=p\Lambda\left[\frac{\exp\left[(\mu+\omega)t_1+(1-\xi)\int_0^{t_1}\lambda(t)dt_1\right]}{(\mu+\omega)+\frac{d}{dt}(1-\xi\int_0^{t_1}\lambda(t_1)dt_1)}\right]+V(0)$$

$$\therefore \quad V(t)=p\Lambda\left[\frac{\exp\left[(\mu+\omega)t_1+(1-\xi)\int_0^{t_1}\lambda(t)dt_1\right]}{(\mu+\omega)+\frac{d}{dt}(1-\xi\int_0^{t_1}\lambda(t_1)dt_1)}\right]\bullet\exp\left\{-\left[(\mu+\omega)t+(1-\xi)\int_0^t\lambda(\tau)d\tau\right]\right\}+V(0)\bullet\exp\left\{-\left[(\mu+\omega)t+(1-\xi)\int_0^t\lambda(\tau)d\tau\right]\right\}$$

$$V(t)=\frac{p\Lambda}{(\mu+\omega)+\frac{d}{dt}(1-\xi\int_0^{t_1}\lambda(t_1)dt_1)}+V(0)\bullet\exp\left\{-\left[(\mu+\omega)t+(1-\xi)\int_0^t\lambda(\tau)d\tau\right]\right\}$$

Recall that recall $\lambda=\frac{\beta I}{N}$

Hence, we shall observe that at t1 = $_o$, V($_o$) is always positive since exponential of a number is always positive.

$\Rightarrow V(t)>V(t_1)>V(0)$ Since $\sup V(0)>0$

Hence $V(t)>0\ always$

Similarly, the same proof shows that: $S(t)>0, E(t)>0,\ I(t)>0, R(t)>0:$ which concludes that S(t); V (t);E(t); I(t);R(t)) of the model are all positive for all time t > $_o$.

3.2. Steady State of the Model

Steady state solutions are the solutions of the system of equations (2.1) when the right-hand side of a nonlinear system is set to zero. At the steady state, the model equation (2.1) is set to zero that is:

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

This implies,

$$\left.\begin{aligned}&(1-p)\Lambda+\omega\Lambda-\frac{\beta I}{N}S-\mu S=0\\&p\Lambda-\frac{\beta(1-\xi)VI}{N}-(\mu+\omega)V=0\\&\frac{\beta IS}{N}+\frac{\beta(1-\xi)VI}{N}-(\mu+\sigma)E=0\\&\sigma E-(\mu+\varepsilon+\gamma)I=0\\&\gamma I-\mu R=0\end{aligned}\right\}\qquad(3.3)$$

Solving (3.3) we have

$$(1-p)\Lambda+\omega\Lambda-\frac{\beta I}{N}S-\mu S=0\Rightarrow(1-p)\Lambda+\omega\Lambda-\lambda S-\mu S=0\ \sin ce\ \lambda=\frac{\beta I}{N}$$

$$\therefore S^*=\frac{(1-p+\omega)\Lambda}{(\lambda+\mu)}$$

$$p\Lambda-\lambda(1-\xi)V-(\mu+\omega)V=0\Rightarrow p\Lambda=\left[\lambda(1-\xi)-(\mu+\omega)\right]V$$

$$V^*=\frac{\Lambda p}{\left[\lambda(1-\xi)-(\mu+\omega)\right]}$$

$$\lambda S+\lambda(1-\xi)V-(\mu+\sigma)E=0\Rightarrow\lambda S+\lambda(1-\xi)V=(\mu+\sigma)E$$

$$E^*=\frac{\lambda S^*+\lambda(1-\xi)V^*}{(\mu+\sigma)}$$

$$\sigma E - (\mu + \varepsilon + \gamma)I = 0 \Rightarrow \sigma E = (\mu + \varepsilon + \gamma)I$$

$$I^* = \frac{\sigma E^*}{(\mu + \varepsilon + \gamma)}$$

$$\gamma I - \mu R = 0 \Rightarrow \gamma I = \mu R$$

$$R^* = \frac{\gamma I^*}{\mu}$$

Hence, the steady state of our model is

$$\left.\begin{aligned} S^* &= \frac{(1 - p + \omega)\Lambda}{(\lambda + \mu)} \\ V^* &= \frac{\Lambda p}{\left[\lambda(1 - \xi) - (\mu + \omega)\right]} \\ E^* &= \frac{\lambda S^* + \lambda(1 - \xi)V^*}{(\mu + \sigma)} \\ I^* &= \frac{\sigma E^*}{(\mu + \varepsilon + \gamma)} \\ R^* &= \frac{\gamma I^*}{\mu} \end{aligned}\right\} \qquad (3.4)$$

3.3. Disease Free Equilibrium (DFE) ξ_0

To determine the stability of the model, we first evaluate the equilibrium points or steady states of the ordinary differential equations (2.1). The equilibrium point under consideration in this model is the Disease-Free (E=I=0) Equilibrium point. At the disease free equilibrium, the disease compartments $E^* = I^* = 0$ .Then equation (3.4) is at steady state and the disease-free (DF) equilibrium is now:

$$\xi_0(S^*, V^*, 0, 0, 0) = \xi_0\left(\frac{(1 - p + \omega)\Lambda}{(\lambda + \mu)}, \frac{\Lambda p}{\left[\lambda(1 - \xi) - (\mu + \omega)\right]}, 0, 0, 0\right) \qquad (3.5)$$

3.4. Basic Reproduction Number R_o

The basic reproduction R_o number is the average number of secondary infection that occurs if a single infected individual is introduced into an entirely susceptible population. The basic reproduction number R_o can also be defined as the effective number of secondary infections caused by an infected individual during his/her entire period of infectiousness (Van den Driesshe and Watmough (2002)). This definition is given for the models that represent spread of infection in a population. It is obtained by taking the largest (dominant) eigenvalue or spectral radius.

Where $F_{ij}\left[\frac{\partial F(\xi_0)}{\partial X_j}\right]$ *and* $V_i = \left[\frac{\partial \nu_i(\xi_0)}{\partial X_j}\right]$

Also if we let $X = [E \quad T]^T$ (3.7)

$$\frac{dX}{dt} = F - V \tag{3.8}$$

Where $\nu(X) = \nu^{-1} - \nu^{+}$ (3.9)

given that

$F_j =$ The rate of appearance of new infection in compartment I

$V_i^- =$ The transfer of individuals out of the disease compartments

$V_i^+ =$ The rate of transfer of individuals into the disease compartments

$\xi_0 =$ The disease free equilibrium

By linearization approach, the associated matrices at disease free equilibrium are F_i and V_i respectively.

The infected compartments are E and I, we shall first solve for the rate of appearance of new infection, that is,

$$F = \begin{bmatrix} \lambda S + (1-\xi)V \\ 0 \end{bmatrix}$$

Hence, we shall find the Jacobian of F to get our F

$$F = \begin{bmatrix} 0 & \frac{\beta S_0 + (1-\xi)}{N} \\ 0 & 0 \end{bmatrix} \tag{3.10}$$

Also, the rate of transfer of individuals out of the disease compartments E and I is given as;

$$V_i^- = \begin{bmatrix} (\sigma + \mu)E \\ (\gamma + \mu + \varepsilon)I \end{bmatrix}$$

and the rate of transfer of individuals into the disease compartments E and I is given as;

$$V_i^+ = \begin{bmatrix} 0 \\ \sigma E \end{bmatrix}$$

From (3.9), $\nu(X) = \nu^{-1} - \nu^{+}$

Hence,

$$V(X) = \begin{bmatrix} (\sigma+\mu)E \\ (\gamma+\mu+\varepsilon)I - \sigma E \end{bmatrix}$$

Also the Jacobian of V is obtained as:

$$V(X) = \begin{bmatrix} (\sigma+\mu) & 0 \\ -\sigma & (\gamma+\mu+\varepsilon) \end{bmatrix} \tag{3.11}$$

The inverse of V is obtained as;

$$V^{-1} = \begin{bmatrix} \frac{1}{(\sigma+\mu)} & 0 \\ \frac{\sigma}{(\sigma+\mu)(\gamma+\mu+\varepsilon)} & \frac{1}{(\gamma+\mu+\varepsilon)} \end{bmatrix}$$

This implies that ,

$$FV^{-1} = \begin{bmatrix} 0 & \frac{\beta S_0 + (1-\xi)}{N} \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{(\sigma+\mu)} & 0 \\ \frac{\sigma}{(\sigma+\mu)(\gamma+\mu+\varepsilon)} & \frac{1}{(\gamma+\mu+\varepsilon)} \end{bmatrix} \tag{3.12}$$

Note that $N = S + V + E + I + R$ which means that using (3.4) we obtain

$$N = \frac{\omega[(1-p)\Lambda + \omega V_0] + p\Lambda\mu}{\mu(\omega+\mu)} \tag{3.13}$$

Putting (3.13) in (3.12) we have

$$FV^{-1} = \begin{bmatrix} \frac{\sigma\beta[S_0] + (1-\xi)V_0}{[(1-p)\Lambda + \omega V_0](\sigma+\mu)(\gamma+\mu+\varepsilon)} & \frac{\beta[S_0] + (1-\xi)V}{(\gamma+\mu+\varepsilon)} \\ 0 & 0 \end{bmatrix} \tag{3.14}$$

The basic reproduction number R_o is given by taking the highest eigen-value of the spectral radius

FV^{-1} (Van den Driesshe and Watmough (2$_{oo}$2))

That is,

$$R_0 = \left| FV^{-1} - \lambda I \right| = 0$$

Which gives us,

$$R_0 = \frac{\sigma\beta[S_0] + (1-\xi)V_0}{N(\sigma+\mu)(\gamma+\mu)} \tag{3.15}$$

3.5. Local Stability of the disease free equilibrium

The local stability of DFE (ξ_0) is established by using the next generation operator method on the system.

Theorem 3.3: The disease-free equilibrium or steady state of the model (2.1) is locally asymptotically stable.

Proof:
Using Theorem (2) in van den Driessche and Watmough (2002), we establish that for the model equation (2.1), the disease free equilibrium is locally asymptotically stable if the basic reproduction number is less than unity, $(R_0 < 1)$, and unstable if the reproduction number is greater than unity, $(R_0 > 1)$.

3.6. Global Asymptotic Stability (GAS) of the Disease-free Equilibrium (DFE) ξ_0 .

Theorem 3.4: Consider the model equation (2.1) with the DFE (3.5) given by ξ_0. The DFE ξ_0 is

Globally Asymptotically table in D whenever $R_0 < 1$.

Proof: We shall first construct a Lyapunov function which shall enable us to show the global stability

of the DFE. From equation (3.15), the basic Reproduction number Ro is given as;

$$R_0 = \frac{\alpha\beta A}{N^0 K_1 K_2}$$

Where $K_1 = \sigma + \mu,\ K_2 = (\gamma + \mu + \varepsilon)$

If $\omega = (\omega_1 \quad \omega_2)$, hence, $\omega V^{-1} F = R_0 \omega$

Given that

$$V = \begin{bmatrix} K_1 & 0 \\ -\sigma & K_2 \end{bmatrix} \quad and \quad V^{-1} = \begin{bmatrix} \frac{1}{K_1} & 0 \\ \frac{\sigma}{K_2 K_1} & \frac{1}{K_2} \end{bmatrix}, \quad F = \begin{bmatrix} 0 & \frac{\beta A}{N} \\ 0 & 0 \end{bmatrix} \ where \ A = S + (1-\xi)V$$

Hence,

$$V^{-1}F = \begin{bmatrix} \frac{1}{K_1} & 0 \\ \frac{\sigma}{K_2 K_1} & \frac{1}{K_2} \end{bmatrix} \begin{bmatrix} 0 & \frac{\beta A}{N} \\ 0 & 0 \end{bmatrix} = \begin{bmatrix} 0 & \frac{\beta A}{N K_1} \\ 0 & \frac{\sigma\beta A}{K_2 K_1} \end{bmatrix}$$

$$\Rightarrow \begin{bmatrix} \omega_1 & \omega_2 \end{bmatrix} \begin{bmatrix} 0 & \frac{\beta A}{N K_1} \\ 0 & \frac{\sigma\beta A}{K_2 K_1} \end{bmatrix} = \frac{\sigma\beta A}{N^0 K_2 K_1} \begin{bmatrix} \omega_1 & \omega_2 \end{bmatrix} \Rightarrow \begin{bmatrix} 0 & \omega_1 \frac{\beta A}{N K_1} + \omega_2 \frac{\sigma\beta A}{K_2 K_1} \end{bmatrix} = \begin{bmatrix} \omega_1 \frac{\beta A}{N K_1} & \omega_2 \frac{\sigma\beta A}{K_2 K_1} \end{bmatrix}$$

Equating and solving, we have

$$0 = \omega_1 \frac{\beta A}{N K_1} \Rightarrow \omega_1 = 0 \quad and \quad \omega_1 \frac{\beta A}{N K_1} + \omega_2 \frac{\sigma\beta A}{K_2 K_1} = \omega_2 \frac{\sigma\beta A}{K_2 K_1} \Rightarrow \omega_2 = 0$$

Hence,

$$\omega = [\omega_1 \quad \omega_2] = [0 \quad 1]$$

We shall now multiply $[\omega_1 \quad \omega_2]$ by V^{-1}

$$\Rightarrow [0 \quad 1] \begin{bmatrix} \frac{1}{K_1} & 0 \\ \frac{\sigma}{K_2 K_1} & \frac{1}{K_2} \end{bmatrix} = \begin{bmatrix} \frac{\sigma}{K_2 K_1} & \frac{1}{K_2} \end{bmatrix}$$

Consider the Lyapunov function

$$L = C_1 E + C_2 I$$

$$C_1 = \frac{\sigma}{K_1 K_2} \text{ and } C_2 = \frac{1}{K_2}$$

$$\therefore L = \frac{\sigma}{K_1 K_2} E + \frac{1}{K_2} I$$

The Lyapunov derivative is

$$\dot{L} = \frac{\sigma}{K_1 K_2} \dot{E} + \frac{1}{K_2} \dot{I}$$

$$\dot{L} = \frac{\sigma}{K_1 K_2} \left[S + (1-\xi) V \frac{\beta I}{N} - K_1 \right] + \frac{1}{K_2} [\sigma E - K_2 I]$$

$$\dot{L} = \frac{\sigma\beta[S + (1-\xi)V]I}{N K_1 K_2} - \frac{\sigma E}{K_2} + \frac{\sigma E}{K_2} - I \leq \frac{\sigma\beta A I}{K_1 K_2 N^0} - I = R_0 I - I < [R_0 - 1] I$$

Since all the model parameters and variables are non-negative, it follows that $\dot{L} \leq 0$ for $R_0 \leq 1$ with $\dot{L} = 0$ if and only if E = I = $_o$. Hence, L is a Lyapunov function on D. Thus, using the Lassalle Invariance Principle (La Salle and Lefschetz (1976)), $E \to 0, I \to 0$. If we substitute $E \to 0, I \to 0$ in (2.1), it shows that $S(t) \to 0$ *and* $V(t) \to 0$ *as* $t \to \infty$. Thus, every solution to the equations of the model (2.1) with $\xi_0 = 0, \ \xi = 1$ with initial conditions in D approaches the DFE E_o as $t \to 0$ whenever $R_0 \leq 1$.

3.7. Global Asymptotic Stability of Endemic Equilibrium (EE)

Here, the global asymptotic stability property of the endemic equilibrium of the model equation
(2.1) is given for special case where the rate at which the vaccine wanes is zero, that is, $\omega = 0$.
This results to a new model equation as shown below;

$$\left.\begin{aligned}
&\frac{dS}{dt} = (1-p)\Lambda - \frac{\beta I}{N}S - \mu S \\
&\frac{dV}{dt} = p\Lambda - \frac{\beta(1-\xi)VI}{N} - (\mu+\omega)V \\
&\frac{dE}{dt} = \frac{\beta IS}{N} + \frac{\beta(1-\xi)VI}{N} - (\mu+\sigma)E \\
&\frac{dI}{dt} = \sigma E - (\mu+\varepsilon+\gamma)I \\
&\frac{dR}{dt} = \gamma I - \mu R \\
&where\ \lambda = \frac{\beta I}{N}\ which\ is\ the\ standard\ \ force\ of\ \ \text{inf}\ ection
\end{aligned}\right\} \qquad (3.16)$$

Theorem 3.5: The endemic equilibrium of the reduced model equation, given in equation (3.16), is

Globally asymptotic stable GAS in D if $R_o > 1$; thus it is unique.

Proof: Given that the basic reproduction number R_o is:

$$R_0 = \frac{\sigma\beta A}{NK_1K_2},\ where\ A = S_0 + (1-\xi),\ M_1 = \sigma+\mu,\ M_2 = (\gamma+\mu+\varepsilon)$$

From Lyapunov stability theory, once we have a system of ordinary differential equations, and if we construct a Lyapunov function positive definite (L > $_o$ for $t \neq 0$ and negative semi definite $L \leq 0$ for $t \neq 0$, then the trivial solution is globally asymptotically stable.

$$L = S - S^{**} - S^{**}\ln\frac{S}{S^{**}} + V - V^{**} - V^{**}\ln\frac{V}{V^{**}} + E - E^{**} - E^{**}\ln\frac{E}{E^{**}} + I - I^{**} - I^{**}\ln\frac{I}{I^{**}} + R - R^{**} - R^{**}\ln\frac{R}{R^{**}} \qquad (3.17)$$

Differentiating (3.17) with respect to t we have

$$\dot{L} = \left(1-\frac{S^{**}}{S}\right)\dot{S} + \left(1-\frac{V^{**}}{V}\right)\dot{V} + \left(1-\frac{E^{**}}{E}\right)\dot{E} + \left(1-\frac{I^{**}}{I}\right)\dot{I} + \left(1-\frac{R^{**}}{R}\right)\dot{R} \qquad (3.18)$$

Now substituting the model equation (2.1) into the (3.18) gives,

$$\dot{L} = \left(1-\frac{S^{**}}{S}\right)\left[(1-p\Lambda) - (\lambda+\mu)S\right] + \left(1-\frac{V^{**}}{V}\right)\left[p\Lambda - [(1-\xi)\lambda+\mu]V\right] + \left(1-\frac{E^{**}}{E}\right)\left[(1-\xi)\lambda V + \lambda S - K_1E\right] +$$
$$K_1\left(1-\frac{I^{**}}{I}\right)\left(\sigma E + K_2 I\right)\ \ where\ \lambda = \frac{\beta I}{N},\ N = \frac{\Lambda}{\mu} \Rightarrow \lambda = \frac{\beta\mu I}{\Lambda}\ but\ \frac{\mu}{\Lambda} = 1 \Rightarrow \lambda = \beta I \qquad (3.19)$$

At steady state,

$$\left.\begin{aligned}
&(1-p)\Lambda = \beta I^{**}S^{**} + \mu S^{**} \\
&\frac{dV}{dt} = p\Lambda = (1-\xi)I^{**}\beta^{**}V^{**} + \mu V^{**} \\
&K_1E = \beta(1-\xi)I^{**}S^{**} \\
&\sigma E^{**} = K_2{}^{**}I
\end{aligned}\right\} \qquad (3.20)$$

Substituting (3.2_o) in (3.19) we have

$$\dot{L}=\left(1-\frac{S^{**}}{S}\right)\left[\beta I^{**}S^{**}+\mu S^{**}-\beta IS-\mu S\right]+\left(1-\frac{V^{**}}{V}\right)\left[(1-\xi)\beta I^{**}V^{**}+\mu V^{**}-[(1-\xi)\lambda+\mu]V\right]+\left(1-\frac{E^{**}}{E}\right)\left[(1-\xi)\lambda V+\lambda S-K_1E\right]+K_1\left(1-\frac{I^{**}}{I}\right)\left(\sigma E-K_2I\right) \quad (3.21)$$

Expanding (3.21) we have

$$\dot{L}=\beta I^{**}S^{**}+\mu S^{**}-\beta IS-\mu S-\frac{\beta I^{**}S^{**2}}{S}-\frac{\mu S^{**2}}{S}+\beta IS^{**}+\mu S^{**}+1-\xi)\beta I^{**}V^{**}+\mu V^{**}-[(1-\xi)\lambda+\mu]V-(1-\xi)\frac{\beta I^{**}V^{**2}}{V}-\frac{\mu V^{**2}}{V}$$
$$+[(1-\xi)\lambda+\mu]V^{**}+(1-\xi)\beta IV+\lambda S-K_1E-\frac{(1-\xi)\beta IVE^{**}}{E}-\frac{\beta ISE^{**}}{E}+K_1E^{**}+\left(\sigma E-K_2I\right)K_1-\frac{K_1\sigma E}{I}+K_1K_2I^{**} \quad (3.22)$$

Collecting the coefficients of μ we have

$$\dot{L}=\mu S^{**}-\mu S-\frac{\mu S^{**2}}{S}+\mu S^{**}+\mu V^{**}-(1-\xi)\lambda-\mu V-\frac{\mu V^{**2}}{V}-\frac{\beta I^{**}S^{**2}}{S}+(1-\xi)\beta I^{**}V^{**}-(1-\xi)\frac{\beta I^{**}V^{**2}}{V}+-(1-\xi)\frac{\beta I^{**}V^{**2}}{V}$$
$$\beta I^{**}S^{**}-\beta IS+\beta IS^{**}+[(1-\xi)\lambda+\mu]V^{**}+(1-\xi)\beta IV+\lambda S-K_1E-\frac{(1-\xi)\beta IVE^{**}}{E}-\frac{\beta ISE^{**}}{E}+K_1E^{**}+\left(\sigma E-K_2I\right)K_1$$
$$-\frac{K_1\sigma E}{I}+K_1K_2I^{**} \quad (3.23)$$

Collecting the terms without ** in the infected classes and equate to zero so that we can solve for the coefficient K1

$$\beta IS^{**}+(1-\xi)\beta IV^{**}-K_1M_2I=0$$
$$I\left[\beta S^{**}+(1-\xi)\beta V^{**}-K_1M_2\right]=0$$

Since $I\neq 0$, $\beta S^{**}+(1-\xi)\beta V^{**}-K_1M_2=0$

$$\Rightarrow \beta S^{**}+(1-\xi)\beta V^{**}=K_1M_2\Rightarrow K_1=\frac{\beta S^{**}+(1-\xi)\beta V^{**}}{M_2}$$

Substituting K1 in (3.22) we shall obtain,

$$\dot{L}=\mu S^{**}\left(2-\frac{S}{S^{**}}-\frac{S^{**}}{S}\right)+\mu V^{**}\left(2-\frac{V}{V^{**}}-\frac{V^{**}}{V}\right)+\beta I^{**}S^{**}\left(3-\frac{S^{**}}{S}-\frac{ISE^{**}}{EI^{**}S^{**}}-\frac{I^{**}E}{IE^{**}}\right)-\beta(1-\xi)V^{**}I^{**}\left(3-\frac{EI^{**}}{IE^{**}}-\frac{V^{**}}{V}\frac{IVE^{**}}{EV^{**}I^{**}}\right)$$

Finally, since arithmetic mean is greater that geometric mean, the following inequalities hold:

$$\left(2-\frac{S}{S^{**}}-\frac{S^{**}}{S}\right)\leq 0$$

$$\left(2-\frac{V}{V^{**}}-\frac{V^{**}}{V}\right)\leq 0$$

$$\left(3-\frac{S^{**}}{S}-\frac{ISE^{**}}{EI^{**}S^{**}}-\frac{I^{**}E}{IE^{**}}\right)\leq 0$$

$$\left(3-\frac{EI^{**}}{IE^{**}}-\frac{V^{**}}{V}\frac{IVE^{**}}{EV^{**}I^{**}}\right)\leq 0$$

Thus, $\dot{L}\leq 0$ for R > 1

This shows that L is a Lyapunov function in D and it follows from the LaSalle's Invariance principle (LaSalle, 1976), that every solution to the equations of the model equation (3.16) with the initial conditions in D approaches the associated unique endemic equilibrium ξ_0 , of the model as $t\to\infty$ for R > 1. This approach was also used by Omame *et al* (2018).

3.8. Numerical (Simulation) Solution

S/N	Parameter	Symbol	Hypothetical values	Reference
	Natural death rate	μ	0.002	Estimated
	Recruitment Rate	Λ	0.00007	Song *et al*
	Waning Rate of Vaccine	ω	0—1	Estimated
	Infection Rate	β	0.11	Robert and Tobias (1998)
	The rate at which the exposed is infected	σ	9.52	Mclyn (1994)
	Rate of recovery of infectious	γ	0.1	Robert and Tobias(1998)
	Death rate due to disease	β	0.03	Mohamed *et al* (2010)
	Effectiveness of vaccine	ξ	0--1	Estimated
	Fraction of recruited individuals	p	0.9	Estimated

In this section, we shall present computer simulation results for our model equation (3.17) by using MATLAB 7.9.

We shall consider the following values for the parameters involved in the model.

A graph of Individuals with time

The Simulation Graphs

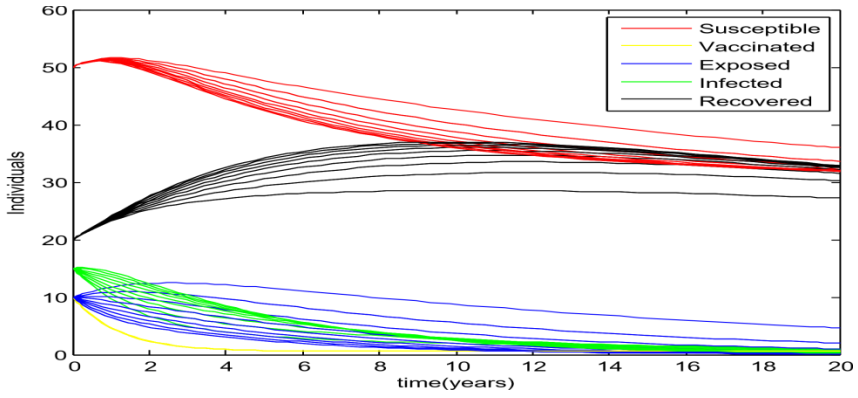

Figure 4.1: A graph of individuals with time when beta (contact rate) is 0.5

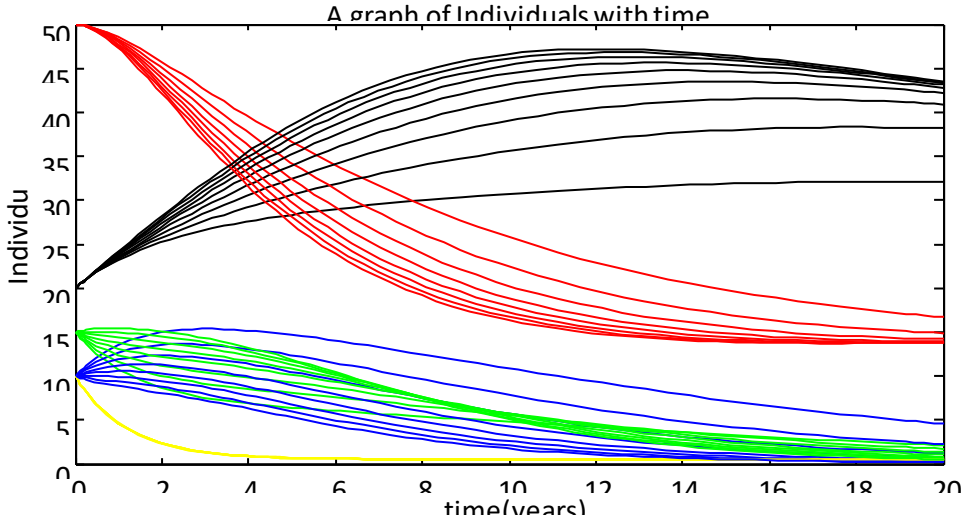

Figure 4.2: A graph of individuals with time when the contact rate (beta) is Increased to 1.0

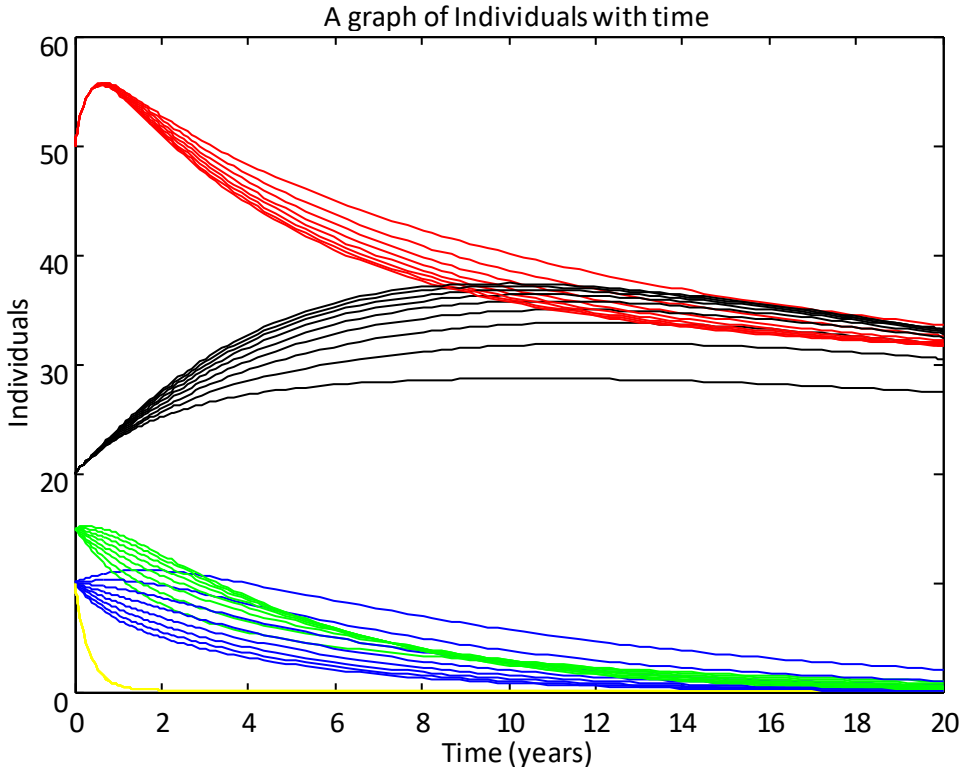

Figure 4.3: A graph of the individuals (when the rate at which vaccine wanes is Increased) with time

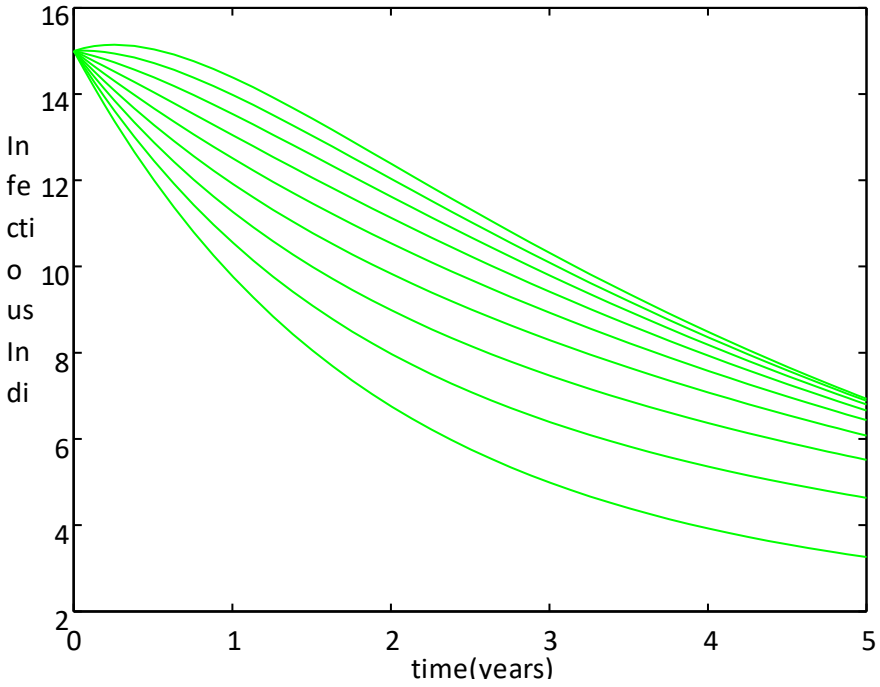

Figure 4.4: A graph of the Infectious individuals (beta=$_o$.5) with time

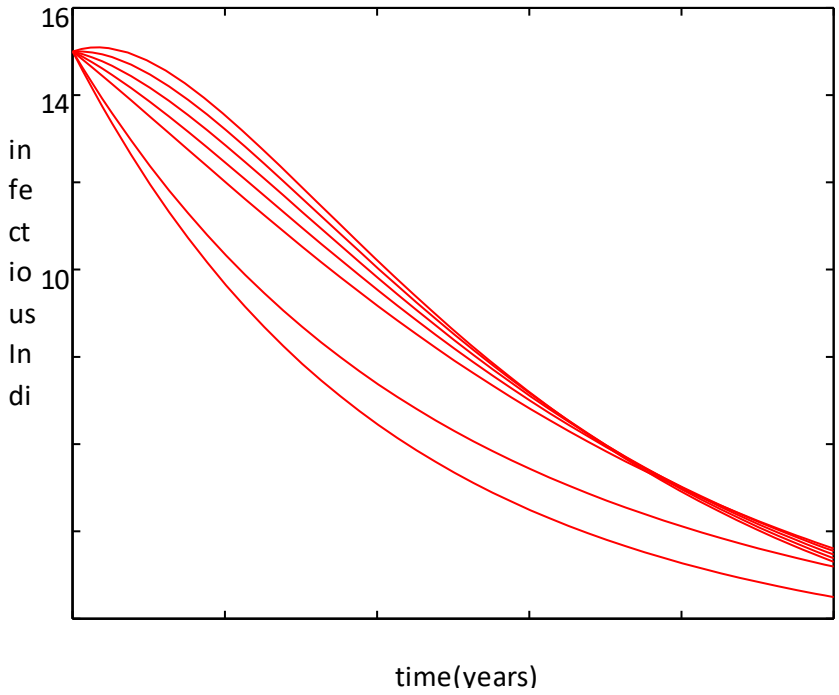

Figure 4.5: A graph of the infectious individuals (when beta is varied) with time

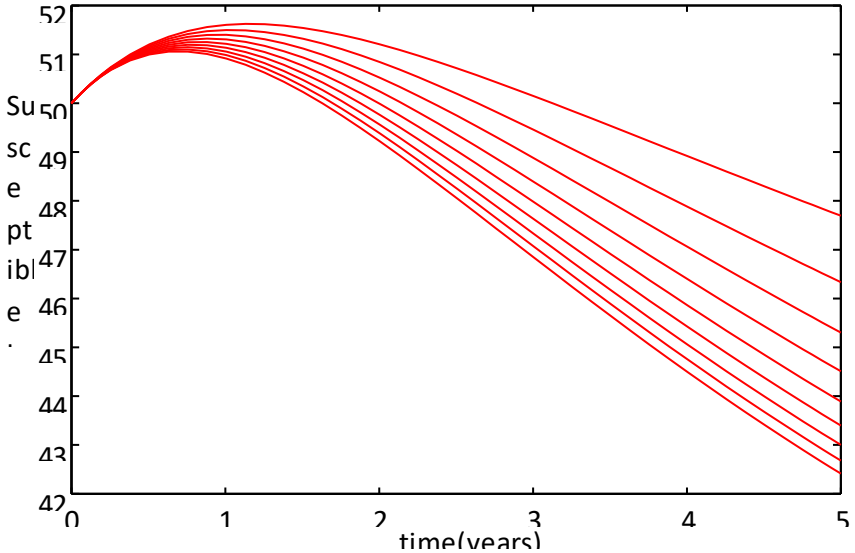

Figure 4.6: A graph of the susceptible individuals with time

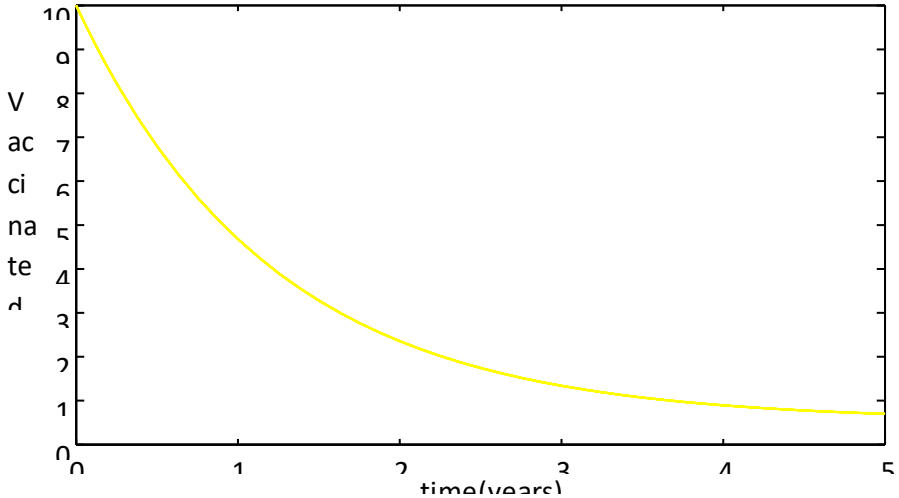

Figure 4.7: A graph of the Vaccinate individuals with time

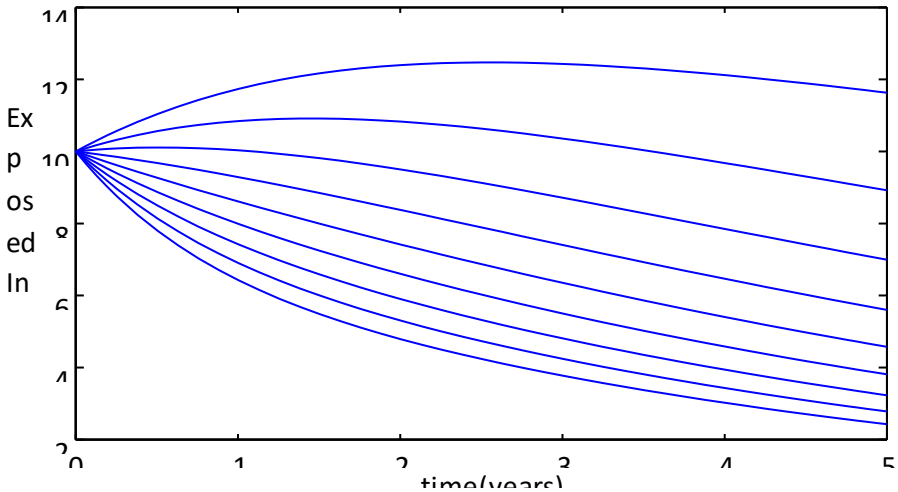

Figure 4.8: A graph of the Exposed individuals with time

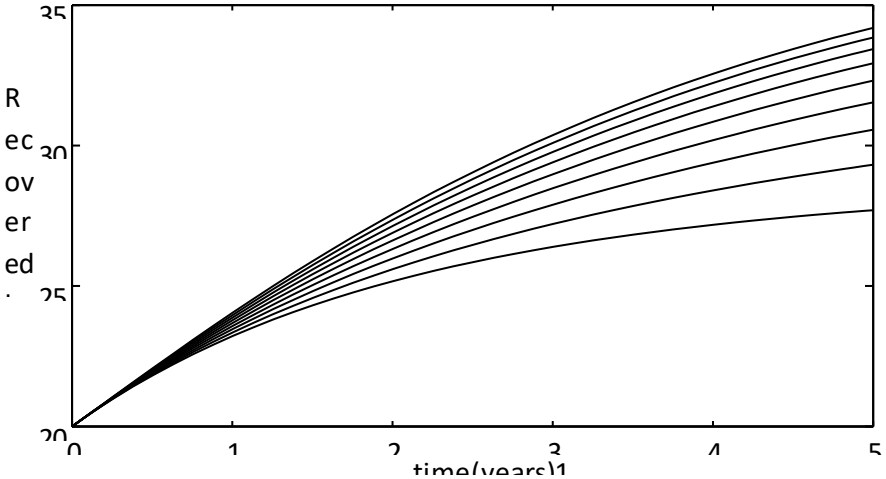

Figure 4.9: A graph of the Recovered individuals with time

4. Discussions

The Figures 4.4 and 4.5 above show the variations of the infected population against time. These show the distribution of the infected population over time when the contact rate β is varied. Here, the population rises to a certain level and then experiences a fall and remains steady again as time goes on. On the other hand, Figure 4.2 shows the variation of all the classes with time. When the contact rate β was increased, we can see a sharp fall in the susceptible and the vaccinated compartment whereby the vaccinated individuals later maintained an equilibrium point as time goes on. The infectious individuals reduced as a lot of individuals recovered from the disease. The population raises, drops and then maintains a stable increase. This is due to unawareness by the people. From the Figures, we could see that as far as the vaccination is effective and does not wane, the infected class kept on reducing until the disease is totally eradicated. The fact is that, at the onset of a disease, the people will not take any serious measures to guard themselves. It is when the number of infectious persons reaches a certain level that individuals will begin to take protective measures against the disease. Moreover, it is noted in the Figures that people become more infected once there is a contact between the susceptible and the infectious. Even the vaccinated individuals also become infected if there is a contact.

5. Conclusions

Our work proposed a mathematical model which incorporates vaccination of an infectious disease whereby we took measles as a special case of reference. The model was shown to be locally asymptotically stable when the basic reproduction number R_o is less than one and unstable otherwise. Various analyses were done during the course of our research such as the construction of Lyapunov function to prove the global asymptotic stability of the Disease free equilibrium and Endemic equilibrium point. Finally, simulations were equally carried out to support the analyses.

The model has shown importance of Vaccinating an infected individuals especially measles as a case study in stopping transmission within a group of individuals. The model shows that the spread of a disease depends on the rates of interactions with infected individuals. With the help of reproduction number, we proved the condition for stability of disease-free equilibrium. When the reproduction number is less than one, our model has only a disease

free equilibrium which is locally stable, which implies that the disease dies out. Otherwise, if the reproduction number is greater than one, the model has a unique endemic equilibrium which is globally stable, the implication is that the disease continues to spread in the population and tends to a steady state.

Based on the results of our project, we suggest the following control strategies.

1. Our model shows that the disease spread through contact rate, Hence action should be taken to reduce the interactions with individuals that are infected with measles, this will minimize the disaster of outbreak.
2. There should be mass vaccination to prevent the plague of disease.
3. The government should focus on the aim of discovering a more effective vaccine and making it available for proper vaccination.

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References

Ghosh, M. Chandra, P., Sinha, P. & Shuka, J. B. (2004). Modeling the spread of carrier dependent infectious diseases with Environmental effects; ELSERVIER: www.Elservier mathematics.com

LaSalle, J. P. (1976) Stability of non-autonomous systems; Nonlinear Analysis: Theory, Methods and Applications (Elsevier Journals). 1(1), 83-90

Safi M. A. & Garba S. M. (2012). Global Stability Analysis of SEIR Model with Holling Type II Incidence Function; Computational and Mathematical Methods in Medicine. 2012, 826052

Momoh A. A, Ibrahim M.O, Uwanta I.J. & Manga S.B. (2013) Mathematical Model for control of measles Epidemiology; International Journal of Pure and Applied Mathematics. 87(5), 707-718.

Mossong J. & Muller C.P. (2003) Modeling measles re-emergence as a result of waning of immunity in vaccinated populations; Pubmed Vaccine. 21(31), 459-603

Ochoche J.M. & Gweryina R. I. (2014) A Mathematical model of measles with vaccination and Two phases of infectiousness; IOSR Journal of mathematics(IOSR.JM). 10(1), 95-105.

Panjeti, V.G. & Real, L. A. (2011). A mathematical model for rabies. Review article Adv Virus Res 2011. 79, 377-95.

Siabouh S.O & Adetunde I.A (2013). A Mathematical model for the study measles in Cape Coast Metropolis; International Journal of Modern Biology and Medicine, 4(2), 110-133

Singh, N., Mishra, A. K. & Shukla, M. M. (2003). Forest Malaria in Chindwara, Madhya Pradesh Central India, A case study in a tribal community; *American Journal of Tropical Medicine Hygiene,* 68, 602-607

Stephen E., Kitengeso R., Kiria G. & Felician N. (2015). A Mathematical Model for control and Elimination of the Transmission Dynamics of Measles, *Science Publishing Group, Applied and Computational Mathematics,* 4(6), 396-408

Umana R. A., Omame, A. & Inyama, S.C. (2016) A deterministic and Stochastic models of the dynamics of drug Resistant Tuberculosis. *FUTO Journal Series (FUTOJNLS),* 2(2), 173-194

Van den Driessche P. & Watmough (2002) Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease, Mathematical Biosciences, 180, 29-48

Omame, A., Umana, R. A. Okuonghae, D. & Inyama, S. C. (2018). Mathematical analysis of a two-sex Human Papillomavirus (HPV) model, International Journal of Biomathematics, 11(7)

Bai, Z. & Liu, D. (2015) Modelling seasonal measles transmission in China. Commun Nonlinear Sci Numer Simul, 25, 19–26

Ntirampeba, D., Neema, I. & Kazembe, L. N. (2017) Modelling spatial patterns of misaligned disease data: An application on measles incidence in Namibia. Clin Epidemiol Glob Health, 5, 190–5.

Omame, A. & Inyama, S. C. (2014). Stochastic model and simulation of the prevalence of Measles, International J. of Math. Sci. & Eng. Appls. (IJMSEA). 8(1), 311-323

Nudee, K., Chinviriyasit, S. & Chinviriyasit, W. (2019), The effect of backward bifurcation in controlling measles transmission by vaccination Chaos, Solitons and Fractals 123, 400–412