School of Engineering and Applied Science (SEAS), Ahmedabad University

B.Tech(ICT) Semester IV: Probability and Random Processes (MAT 202)

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EBOLA DISEASE PREDICTION

1 Introduction

1.1 Background

- The Ebola virus disease(EVD) is an infectious zoonosis found in several mammals, including humans, bats and apes. A zoonosis is a disease that can be naturally transmitted from animals to humans. The virus spreads through direct contact with body fluids, such as blood from infected humans or other animals. Spread may also occur from contact with items recently contaminated with bodily fluids. Semen or breast milk of a person after recovery from EVD may carry the virus for several weeks to months. Fruit bats are believed to be the normal carrier in nature, able to spread the virus without being affected by it. EVD in humans is caused by four of five viruses of the genus Ebolavirus. The four are Bundibugyo virus (BDBV), Sudan virus (SUDV), Taï Forest virus (TAFV) and one simply called Ebola virus (EBOV, formerly Zaire Ebola virus). EBOV, species Zaire ebolavirus, is the most dangerous of the known EVD-causing viruses, and is responsible for the largest number of outbreaks. The fifth virus, Reston virus (RESTV), is not thought to cause disease in humans, but has caused disease in other primates.The first known spillover of EVD into the human population took place in Zaire(Now Democratic Republic of the Congo) near the Ebola River from which the disease took its name. Major EVD epidemics have taken place in Democratic Republic of the Congo, Gabon, Sudan, Uganda, and most recently in Guinea, Sierra Leone and Liberia. Symptoms usually begin with a sudden influenza-like stage characterised by feeling tired, fever, weakness, decreased appetite, muscular pain, joint pain, headache, and sore throat. The fever is usually higher than 38.3 C (101 F). This is often followed by nausea, vomiting, diarrhoea, abdominal pain, and sometimes hiccups. The combination of severe vomiting and diarrhoea often leads to severe dehydration. Next, shortness of breath and chest pain may occur, along with swelling, headaches, and confusion. In about half of the cases, the skin may develop a maculopapular rash, a flat red area covered with small bumps, five to seven days after symptoms begin.Depending on the viral strain, the disease may kill as much as 90% of an affected human population.

- Mathematical models and its associated literature have been well studied and researched for some time now. [1] Modelling and simulation of the pattern and spread of infectious disease in a particular location to provide vital and early information to public health experts and medical personnel has had sufficient grounds in recent times especially in Africa.[8] [2] A model is an appropriate representation of a given part of the material world, the study or understanding of whose part it facilitates, Oko, 1998.[9] [3] An epidemic model is a simplified means of describing the transmission of communicable disease through individuals.[10] [4] Generally any prediction model is modelled using basic SIR(Susceptible,Infected,Recovered) model. So ebola can also be modelled using SIR model. [11]
- There are also many other models like SIRS, SEIR etc.[12] [5] [13] But we have extended the SIR model and have modelled using SEIDHR(Susceptible,exposed,infected,deceased,
  hospitalized,recovered) model to make it more precise. [6] [14] [15]

1.2 Motivation

EVD, formerly known as Ebola haemorrhagic fever, is a severe, often fatal illness affecting humans and other primates. It is one of the most deadliest disease in the world. Its almost 40 years of Ebola disease around the world and new cases are still being reported in some countries of Africa. So as an initiative to eradicate it, a model with vaccination is introduced in our report.

1.3 Problem Statement/ Case Study

Ebola has a high transmission rate and also high mortality rate thus it has become an endemic. Vaccine should be provided in every part of the world where Ebola still persists.

2 Data Acquisition

- Is your Special Assignment Data Dependent?
  Yes our Special Assignment is data dependent.
- The data is collected from the data given by World Health Organization (WHO) . [7]

3 Probabilistic Model Used/ PRP Concept Used

A stochastic process is defined by the probabilities with which different events happen in a small time interval.In our model, there are two possible events for each population : production and death/removal.

For a large population size and a large number of infectious individuals, the deterministic threshold $R_o > 1$ provides a good prediction of a disease outbreak.

The compartmental model for EVD included here is an extension of exsisting SEIR(Susceptible,Exposed,Infected and Recovered) model. It has two additional classes : hospitalized and deceased thus SEIDHR model.

Assumptions :

1) Each person is equally susceptible to be infected and be cures/die of the disease.

2)The person cannot get the disease again.

3)There aren't any new people entering or exiting the sample space.

4)The individuals who die in the hospitalized class are immediately buried.

5) The death rate and birth rate are same preserving the number of population.

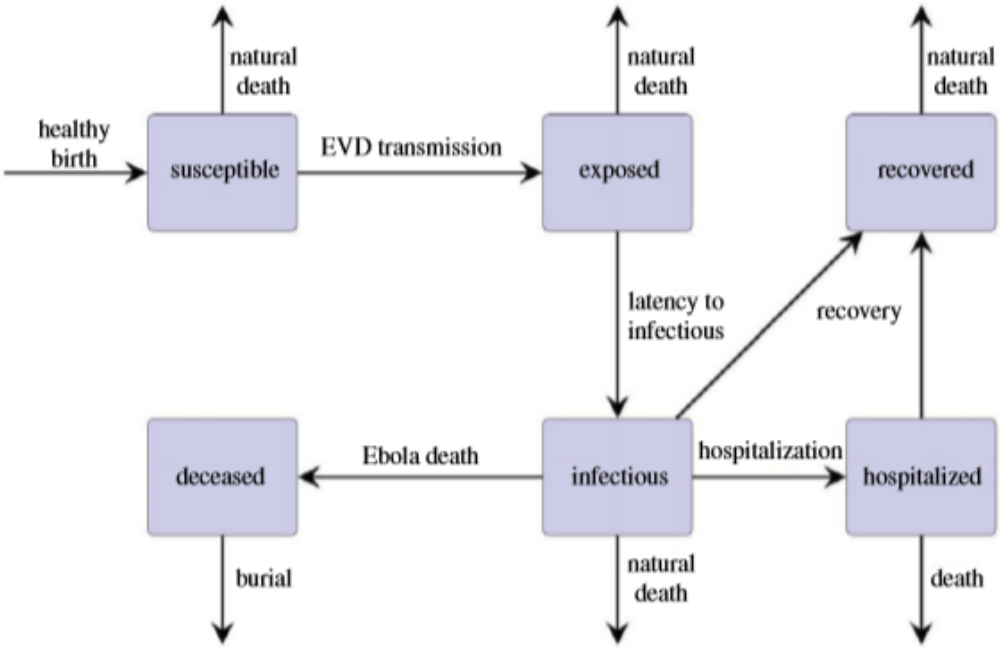


Figure 1: State diagram of EVD model

Figure 1 shows the division of the population into the following six classes:

(1) Susceptible class S consists of individuals who may become infected with EVD through contact with an infected individual , a hospitalized individual, or a deceased but unburied individual.

(2) Exposed class E consists of individuals w2ho are infected with EVD but not yet infectious.

(3) Infectious class I consists of individuals who are capable of transmitting EVD to a susceptible individual.

(4) Recovered class R consists of individuals who have recovered from EVD.

(5) Deceased class D consists of deceased and unburied individuals who are capable of transmitting EVD to a susceptible individual.

(6) Hospitalized class H consists of individuals who have been hospitalized and are capable of transmitting EVD to a susceptible individual.

Deterministic Model:

This model represents time dependent discrete random variables . Here the maximum size of the population is N.

Although infectious diseases generally spread in a stochastic fashion, deterministic models are commonly used as tools for studying epidemic behaviour.These deterministic models have been very useful in understanding the dynamics of infectious disease, estimating important epidemiologic parameters(e.g. basic reproduction number R_o), and determining targets for disease control(e.g. critical proportions of the population to immunise.).

The deterministic mean-field dynamics are given by the following equations :

$\frac{dx_S}{dt} = \mu - \beta_i x_I x_S - \beta_d x_D x_S - \beta_h x_H x_S - \mu x_S - \kappa x_S,$

$\frac{dx_E}{dt} = \beta_i x_I x_S + \beta_d x_D x_S + \beta_h x_H x_S - (\mu + \sigma) x_E - \kappa x_S,$

$\frac{dx_I}{dt} = \sigma x_E - (\gamma_{ir} + \mu_e + \tau + \mu) x_I,$

$\frac{dx_D}{dt} = \mu_e x_I - (\delta + \mu)x_D,$

$\frac{dx_H}{dt} = \tau x_I - (\gamma_{hr} + \mu_e + \mu)x_H,$

$\frac{dx_R}{dt} = \gamma_{ir} x_I + \gamma_{hr} x_H - \mu x_R$

$\frac{dx_S}{dt} \rightarrow$ Susceptible humans (who are at risk of getting infected) over a period of time.
$\frac{dx_I}{dt} \rightarrow$ Infected humans over a period of time.
$\frac{dx_D}{dt} \rightarrow$ Deceased humans over a period of time.
$\frac{dx_H}{dt} \rightarrow$ Hospitalized humans over a period of time.
$\frac{dx_R}{dt} \rightarrow$ Recovered humans over a period of time.
$\mu \rightarrow$ Host life span.
$\beta_i \rightarrow$ contact rate for infectious.
$\beta_d \rightarrow$ contact rate for deceased.
$\beta_h \rightarrow$ contact rate for hospitalized.
$\sigma \rightarrow 1/$Latency period.
$\gamma_{ir} \rightarrow 1/$recovery period(no hospital).
$\mu_e \rightarrow$ Death from EVD.
$\tau \rightarrow 1/$mean time to hospitalization.
$\delta \rightarrow 1/$burial time.
$\gamma hr \rightarrow 1/$recovery period (hospital).
$\kappa \rightarrow$ reservoir transmission.

A common metric for infectivity of a disease is the *basic reproduction number* , R_o , which is defined as the average number of infections that a single infectious individual will trigger in a fully susceptible population. R_o can be described more roughly as the ratio between the inflow of infected individuals and the outflow of dead or recovered individuals in a fully susceptible population. If $R_o > 1$, the disease will spread. And if $R_o < 1$, then the disease will go extinct. The reproductive number for EVD is estimated to be approximately $R_o \leq 2$.

The standard approach to analyse the dynamics of a stochastic system such as this to start with the mean-field equations. This is done by assuming a value of zero for the conjugate momentum variables in Hamilton's equations. This reduced system captures the deterministic dynamics, which is the limit of the stochastic dynamics in the large population limit. Two steady states of the mean-field equations can be derived analytically. If we assume no reservoir transmission ($\kappa = 0$), the first steady state can be described as the disease-free equilibrium (DFE), for which the entire population is sus-

ceptible and no infection is present. This state is stable if $R_o < 1$, implying that the disease cannot persist in the population and all solutions will limit to the DFE. If $R_o > 1$, the DFE is unstable into a system having $R_o > 1$, solutions will tend to an endemic steady state, implying non-zero values for the infected and infectious classes.

By introducing reservoir transmission $\kappa > 0$, the traditional DFE no longer exists as a small number of exposed individuals are introduced to the system. Therefore, in addition to the traditional endemic state, there is another steady state that we call the invasion state that has a small number of non-susceptible individuals. While this deterministic system cannot exhibit periods of disease fade-out and re-invasion, its steady states provide guidance in understanding the dynamics of the full stochastic system.
After solving the first generation matrix and Hamiltonian Equation, and assuming no reservoir transmission($\kappa = 0$), we use the DFE to analytically determine R_o as

$$R_o = \frac{\sigma(\frac{\beta_i+\beta_d\mu_e}{(\delta+\mu)} + \frac{\beta_h\tau}{\gamma_{hr}+\mu_e+\mu})}{(\gamma_{ir}+\tau+\mu e+\mu)(\mu+\sigma)}$$

Stochastic Model:

Real-world data for EVD suggest that the basic reproduction rate is greater than one, but the disease dynamics cannot be captured by deterministic models with solutions that simply limit to as steady state. The stochastic EVD model allows behaviour beyond the dynamics predicted by the mean-field equations. Examples include solutions that switch steady states, resembling disease outbreak and fade-out events.

The six classes for stochastic EVD model are represented by the following variables : S = Susceptible, E = Exposed, I = Infectious, R = Recovered, H = Hospitalized and D = Deceased.

The general form of the master equation is :

$$\frac{d\rho(\mathbf{X})}{dt} = \mu N(\rho(S-1,E,I,D,H,R) - \rho(S,E,I,D,H,R))$$
$$+ \frac{\beta_i I}{N}((S+1)\rho(S+1,E-1,I,D,H,R) - S\rho(S,E,I,D,H,R))$$
$$+ \frac{\beta_d D}{N}((S+1)\rho(S+1,E-1,I,D,H,R) - S\rho(S,E,I,D,H,R))$$
$$+ \frac{\beta_h H}{N}((S+1)\rho(S+1,E-1,I,D,H,R) - S\rho(S,E,I,D,H,R))$$
$$+ \kappa((S+1)\rho(S+1,E-1,I,D,H,R) - S\rho(S,E,I,D,H,R))$$
$$+ \mu((S+1)\rho(S+1,E,I,D,H,R) - S\rho(S,E,I,D,H,R))$$
$$+ \mu((E+1)\rho(S,E+1,I,D,H,R) - E\rho(S,E,I,D,H,R))$$
$$+ \sigma((E+1)\rho(S,E+1,I-1,D,H,R) - E\rho(S,E,I,D,H,R))$$
$$+ \mu((I+1)\rho(S,E,I+1,D,H,R) - I\rho(S,E,I,D,H,R))$$
$$+ \tau((I+1)\rho(S,E,I+1,D,H-1,R) - I\rho(S,E,I,D,H,R))$$
$$+ \mu_e((I+1)\rho(S,E,I+1,D-1,H,R) - I\rho(S,E,I,D,H,R))$$
$$+ \gamma_{ir}((I+1)\rho(S,E,I+1,D,H,R-1) - I\rho(S,E,I,D,H,R))$$
$$+ (\mu+\delta)((D+1)\rho(S,E,I,D+1,H,R) - D\rho(S,E,I,D,H,R))$$
$$+ (\mu+\mu_e)((H+1)\rho(S,E,I,D,H+1,R) - H\rho(S,E,I,D,H,R))$$
$$+ \gamma_{hr}((H+1)\rho(S,E,I,D,H+1,R-1) - H\rho(S,E,I,D,H,R))$$
$$+ \mu((R+1)\rho(S,E,I,D,H,R+1) - R\rho(S,E,I,D,H,R))$$

where $\mathbf{X} = [S,E,I,D,H,R]^T$.

In probability theory, the Gillespie algorithm (or occasionally the Doob-Gillespie algorithm) generates a statistically correct trajectory (possible solution) of a stochastic equation. So in our model we have used Gillespie algorithm. Mathematically, it is a variant of a dynamic Monte Carlo method and similar to the kinetic Monte Carlo methods. It is used heavily in computational systems biology.Gillespie developed two different, but equivalent formulations; the direct method and the first reaction method. In a simulation, we follow one possible set of reactive events and the consequent changes in populations. In the simulation, at any time t, we have a specific composition and therefore specific values of the propensities. The propensities therefore become transition probabilities per unit time.

Each propensity is the probability per unit time that a specific person shifts from one state to other state. The probability per unit time that any person shifts from one state to other state is just the sum of the propensities. $a_0 = \sum a_n$. The probability per unit time of a transition occurring is constant until a reaction occurs. A constant probability per unit time implies exponential decay of the probability that any transition has not occurred yet

$P_{withoutTransition} = e^{-a_0(t-t_{ref})}$

where t_{ref} is some reference time The cumulative distribution for the probability of reaction is therefore $P_{Transition}$ = 1 - $e^{-a_0(t-t_{ref})}$

The distribution of transition between states is therefore
p(t) = $\frac{dP_{Transition}}{dt} = a_0 e^{-a_0(t-t_{ref})}$

Suppose that we had just two states. If state 1 has probability per unit time (propensity) of a1 and state 2 has probability per unit time of a2, then the probability that the next state to occur is state 1 is a1/(a1 + a2). In general, the probability that the next reaction is state n is $\frac{a_n}{a_0}$.

4 Pseudo Code/ Algorithm

In the stochastic version of the SEIDHR model, the continuous variables are replaced by discrete numbers, and the process rates are replaced by process probabilities. We denote the process probability of the ith process by ai . There are sixteen such processes in our stochastic SEIDHR models which are listed below.

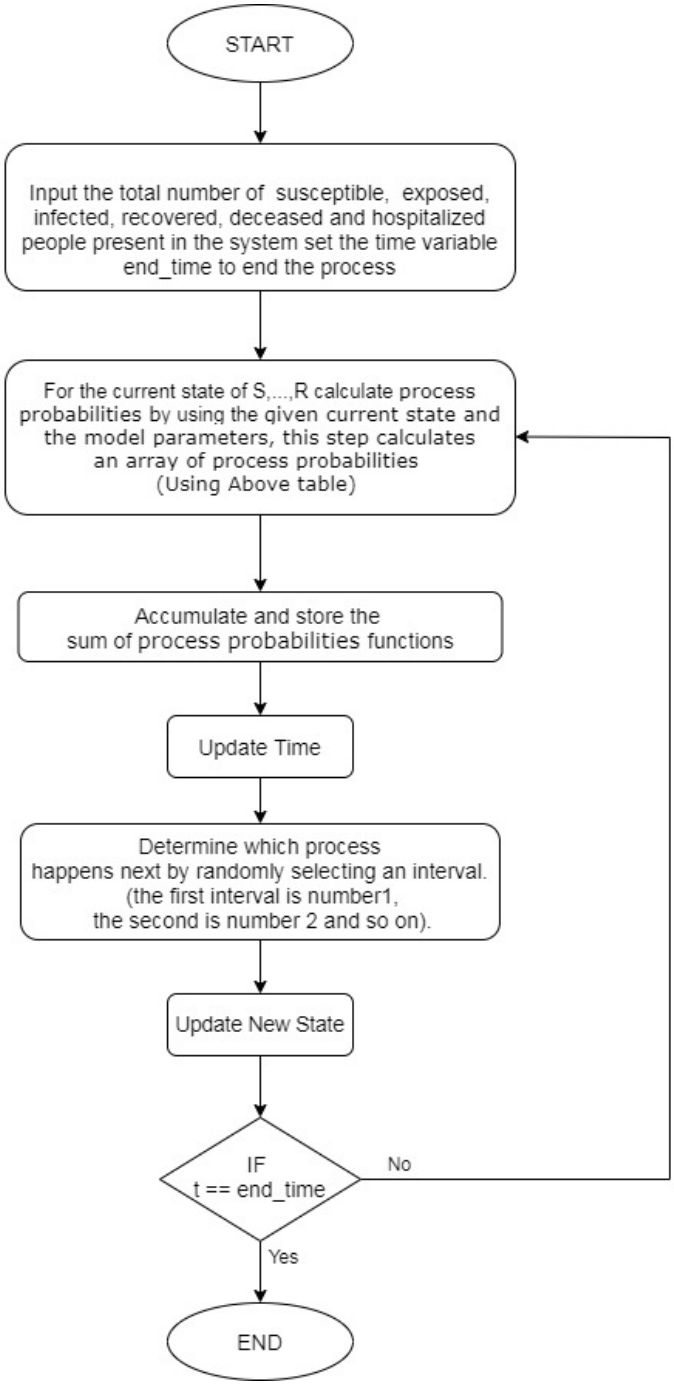
PROCESS	**TRANSITION**	**PROBABILITY**
Contact rate for Infectious	S $\Longrightarrow$ E	a1 = $\beta_i IS$
Contact rate for Deceased	S $\Longrightarrow$ E	a2 = $\beta_d DS$
Contact rate for Hospitalized	S $\Longrightarrow$ E	a3 = $\beta_h HS$
Death of Susceptible Host	S $\Longrightarrow$ 0	a4 = μS
Death of Exposed Host	E $\Longrightarrow$ 0	a5 = μE
Latency to infectious	E $\Longrightarrow$ I	a6 = σE
EVD Death	I $\Longrightarrow$ D	a7 = $\mu_e I$
Recovery	I $\Longrightarrow$ R	a8 = $\gamma_{ir} I$
Hospitalization	I $\Longrightarrow$ H	a9 = τI
Death of Infected Host	I $\Longrightarrow$ 0	a10 = μI
Burial	D $\Longrightarrow$ 0	a11 = δD
Death	D $\Longrightarrow$ 0	a12 = μD
Recovery from Hospital	H $\Longrightarrow$ R	a13 = $\gamma hr H$
Death from Hospital	H $\Longrightarrow$ 0	a14 = $\mu_e H$
Death of Hospitalized Host	H $\Longrightarrow$ 0	a15 = μH
Death of Recovered Host	R $\Longrightarrow$ 0	a16 = μH

Now for Stochastic Simulation we use Gillespie's Algorithm to plot the trajectory of individual random variable.
Suppose the current time is t. The time t + at which something happens next is an exponentially distributed

random number scaled by the sum of all process rates,$\sum$i ai.

Then, the Gillespie algorithm determines what happens next by drawing a process randomly from all possible processes according to their respective probabilities.

Algorithm used :



The result data is collected and Running averages are calculated from the result data. To do this, unique times and the corresponding data are extracted with the MATLAB function unique
Lastly, the mean values of the susceptible, exposed, infected, deceased, hospitalized and recovered individuals are plotted against time.

5 Coding and Simulation

5.1 Simulation Framework

We have six random variables that are S, E, I, D, H, R.
Initial values of these random variables are
S[0] = 100000
E[0] = 500
I[0] = 100
R[0] = 0
D[0] = 2
H[0] = 5

Also different transition rates used are

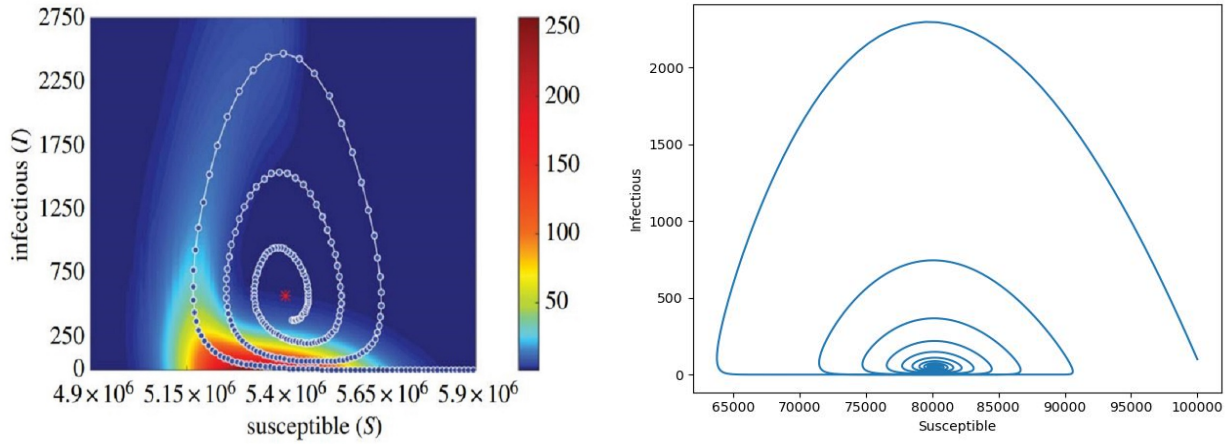
1/host life span (and birth rate)	μ	0.00005 d^{-1}
contact rate for infectious	β_i	0.5 d^{-1}
contact rate for deceased	β_d	0.6 d^{-1}
contact rate for hospitalized	β_h	0.00016 d^{-1}
1/latency period	σ	0.1 d^{-1}
1/recovery period (no hospital)	γ_{ir}	0.07 d^{-1}
death rate from EVD	μ_e	0.12 d^{-1}
1/mean time to hospitalization	τ	0.2 d^{-1}
1/burial time	δ	0.33 d^{-1}
1/recovery period (hospital)	γ_{hr}	0.10 d^{-1}
reservoir transmission	κ	5×10^{-9} d^{-1}

Figure 2: Transition Rates used

5.2 Reproduced Figures

- Used Tools (MATLAB and Python)

- Reproduced Figure-1

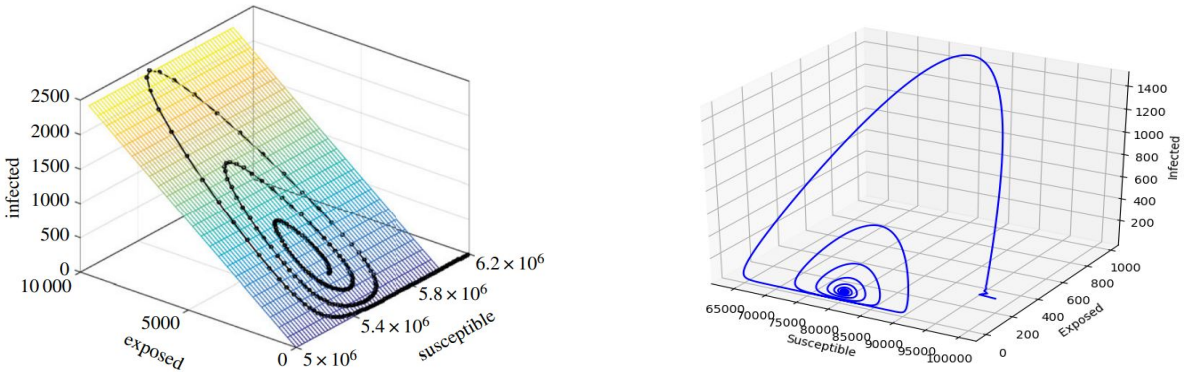


(a) 1B (b) 1R

Here the above plots are susceptible vs infectious population.

Figure 1B and 1R shows the resulting solution, starting at the endemic state and spiralling out to the disease-free state.The result is verified by comparing it with the probability density of extinction prehistory in the susceptible–infectious plane.

- Reproduced Figure-2



(a) 2B (b) 2R

Here the above plots are susceptible vs exposed vs infected population.

The Additional verification of the optimal path to extinction is achieved by projecting the optimal path.

- Produced Figure-3

Various initial values of random variables are :

S[0] = 100

E[0] = 10

I[0] = 8

R[0] = 1

D[0] = 2

H[0] = 5

Rates are taken same as mentioned above

$\implies$ A) DETERMINISTIC MODEL

We have derived equations of $\frac{dx_S}{dt}$, $\frac{dx_E}{dt}$, $\frac{dx_I}{dt}$, $\frac{dx_D}{dt}$, $\frac{dx_H}{dt}$, $\frac{dx_R}{dt}$ above.

Our MATLAB code simulates a deterministic SIR model using a differential equation solver ode45

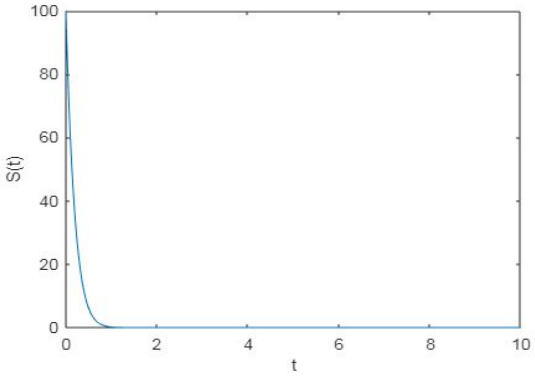
The solver takes a function of two variables as the first argument. This function is called derivative and calculates the derivatives of the SIR model. Its output is a list with the derivatives $\frac{dx_S}{dt}$, $\frac{dx_E}{dt}$, $\frac{dx_I}{dt}$, $\frac{dx_D}{dt}$, $\frac{dx_H}{dt}$, $\frac{dx_R}{dt}$ at time t.

The second argument is a row vector that specifies the integration time interval.

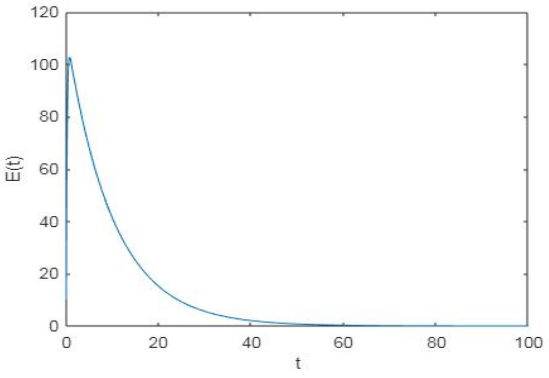
The third argument is a column vector with the initial values for the three functions S, E, I, D, H and R.

Thus we obtained plot will be S(t), E(t), I(t), D(t), H(t), R(t) vs Time

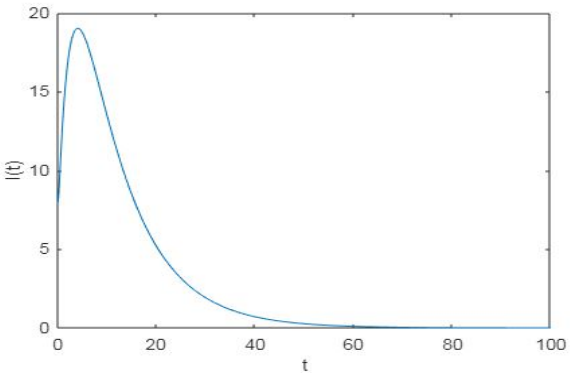
Generated Graphs



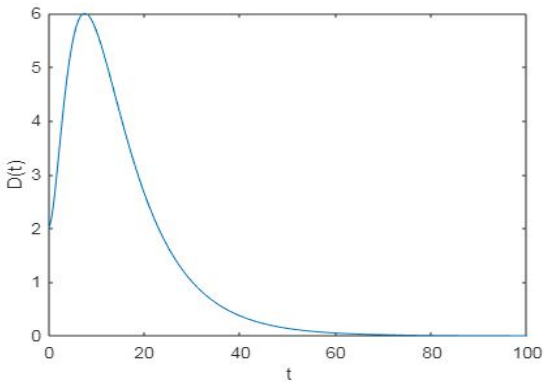
(a) Susceptible



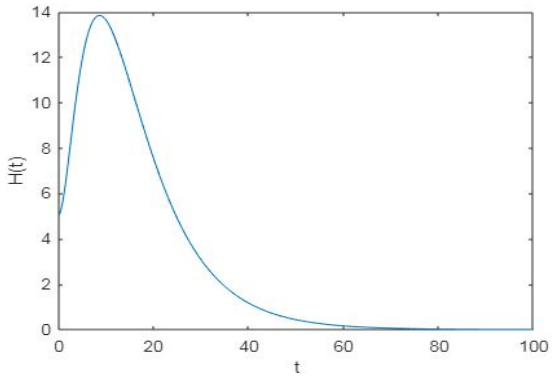
(b) Exposed



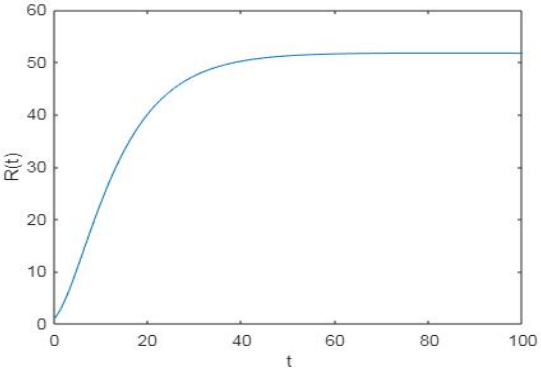
(a) Infected



(b) Deceased



(a) Hospitalized



(b) Recovered

Combined S E I D H R deterministic plot

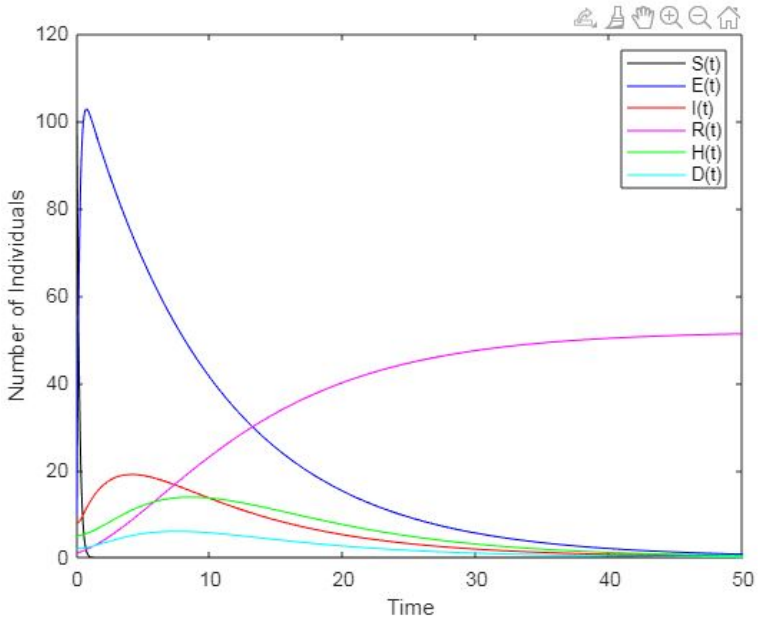


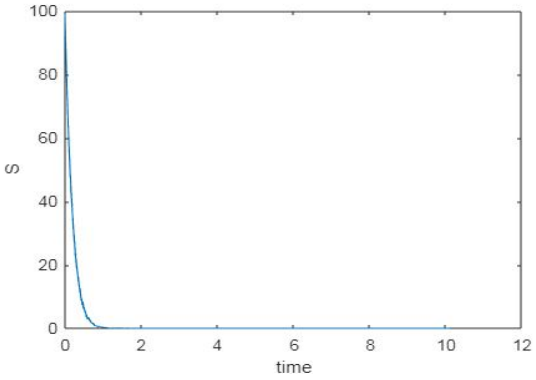
Figure 8: Deterministic Simulation.

$\Longrightarrow$ B) STOCHASTIC MODEL

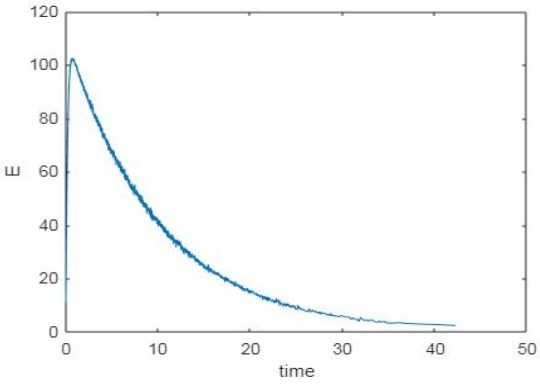
In the stochastic version of the SEIDHR model, the continuous variables are replaced by discrete numbers, and the process rates are replaced by process probabilities. Let us denote the process probability of the ith process by ai. There are sixteen such processes in our stochastic SEIDHR models which are listed above.

For the Simulation of the stochastic SEIDHR model we have used Gillespie algorithm.

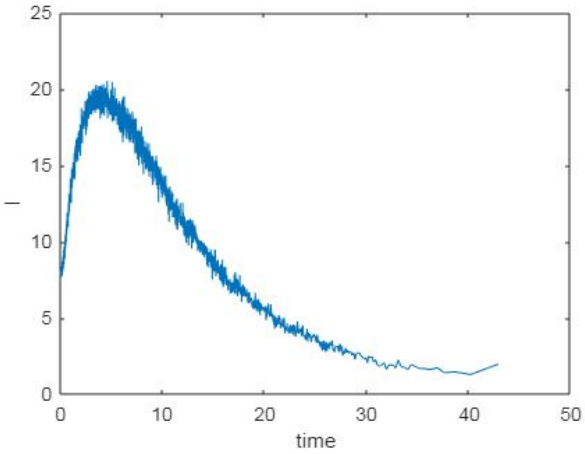
Generated Graphs



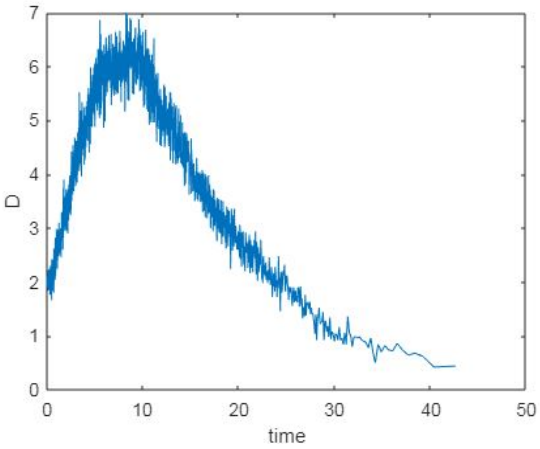
(a) Susceptible



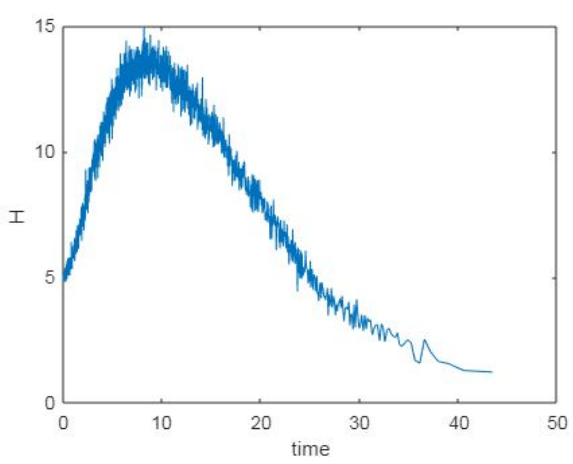
(b) Exposed



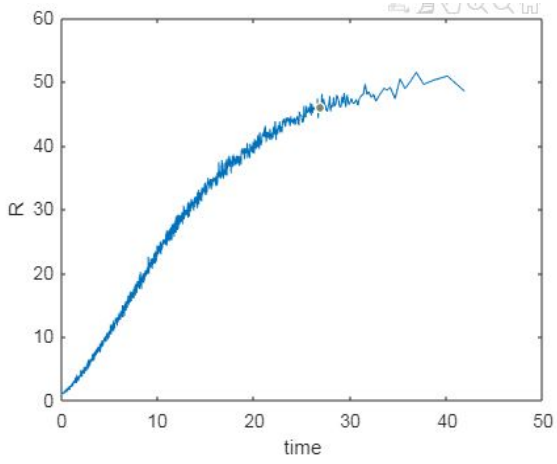
(a) Infected



(b) Deceased



(a) Hospitalized



(b) Recovered

Combined S E I D H R stochastic plot

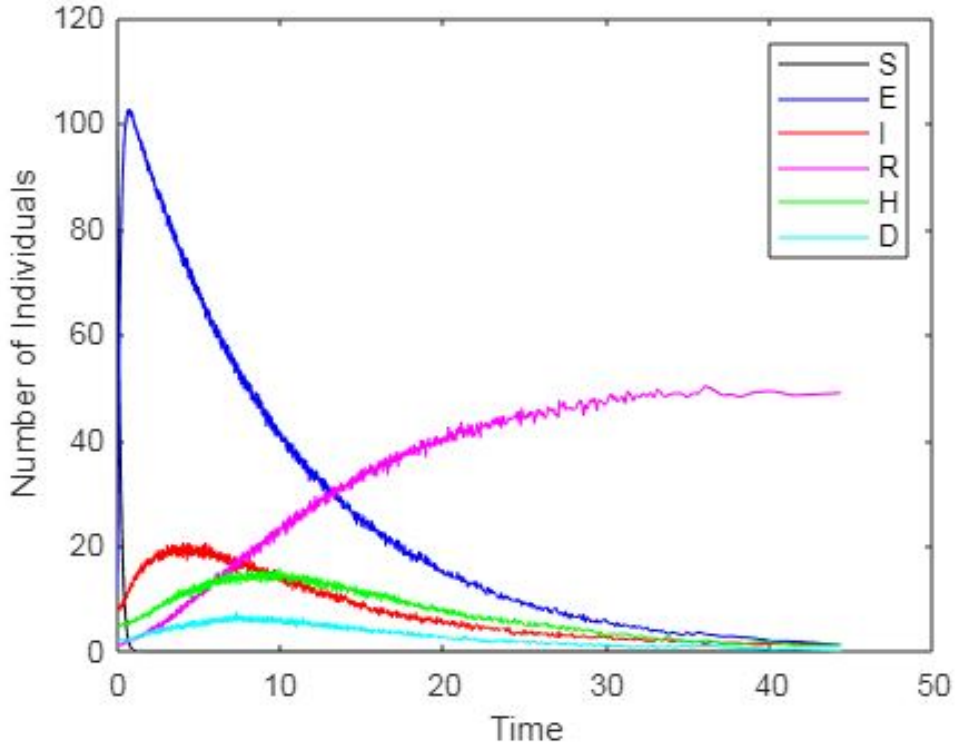


Figure 12: Stochastic Stimulation.

5.3 New Work Done

5.3.1 New Analysis

We have introduced **Vaccination** factor in the above Disease Dynamics and again calculated the master equation

For that we have added a new rate called the Vaccination rate (v). We have taken three different vaccination rates which are 0, 0.01 and 0.05 and tried to plot the difference in susceptible, infected, exposed, deceased, hospitalized and recovered population.

So the new deterministic mean-field dynamics are given by the following equations :

$$\frac{dx_S}{dt} = \mu - \beta_i x_I x_S - \beta_d x_D x_S - \beta_h x_H x_S - \mu x_S - \kappa x_S - v x_S$$

$$\frac{dx_E}{dt} = \beta_i x_I x_S + \beta_d x_D x_S + \beta_h x_H x_S - (\mu + \sigma) x_E - \kappa x_S - v x_E,$$

$$\frac{dx_I}{dt} = \sigma x_E - (\gamma_{ir} + \mu_e + \tau + \mu) x_I,$$

$$\frac{dx_D}{dt} = \mu_e x_I - (\delta + \mu) x_D,$$

$$\frac{dx_H}{dt} = \tau x_I - (\gamma_{hr} + \mu_e + \mu) x_H,$$

$$\frac{dx_R}{dt} = \gamma_{ir} x_I + \gamma_{hr} x_H - \mu x_R + v x_S + v x_E$$

5.3.2 Graphs Plotted:

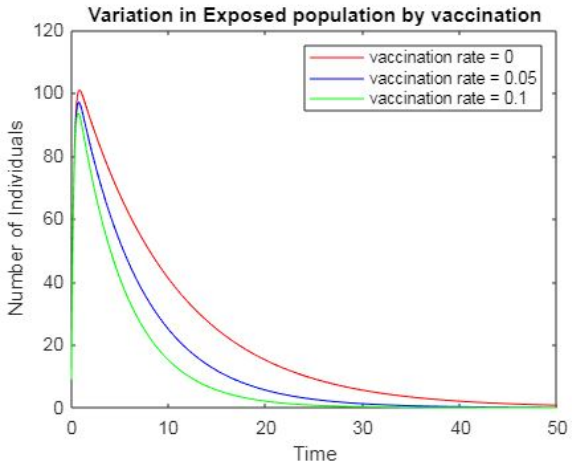


Figure 13: Exposed

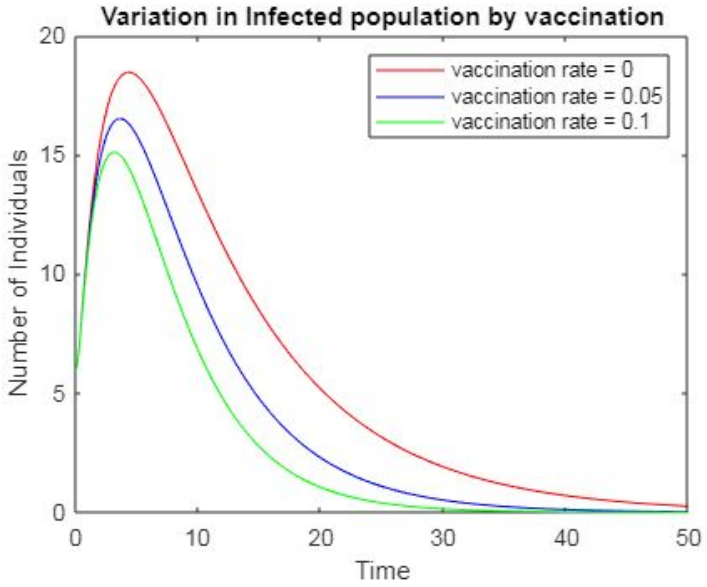


Figure 14: Infected

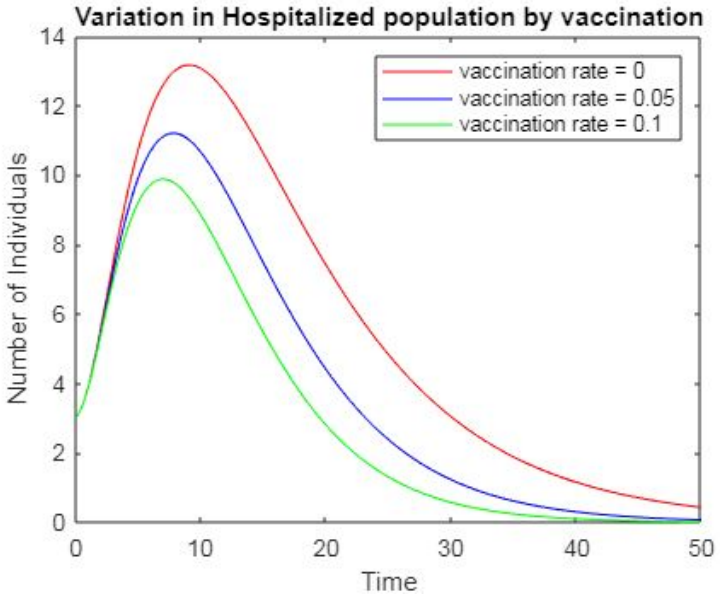


Figure 15: Hospitalized

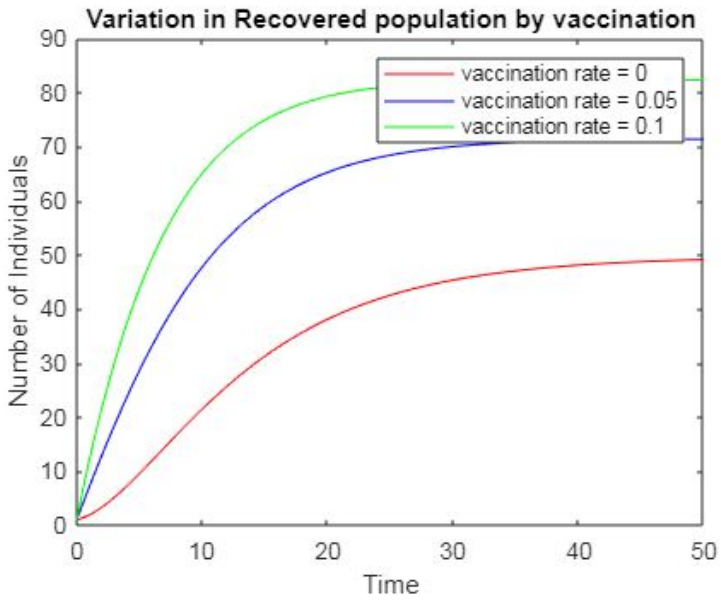


Figure 16: Recovered

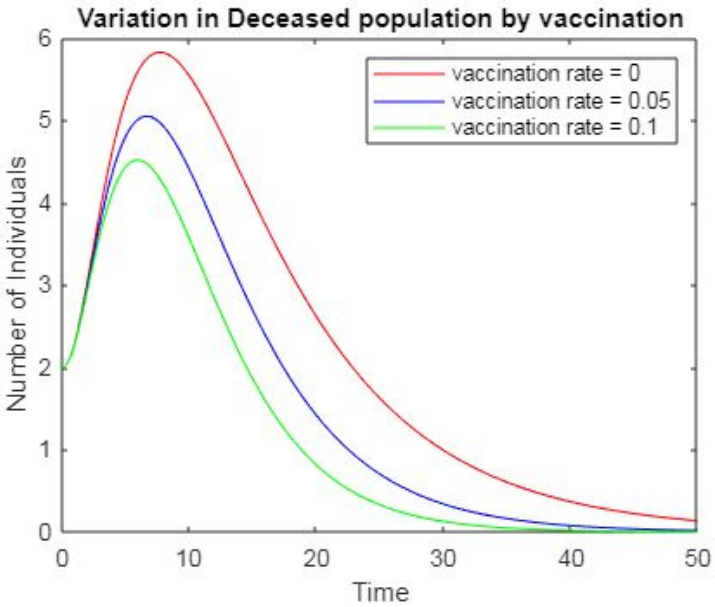


Figure 17: Deceased

5.3.3 New Inferences

- Here from the graphs above we can clearly point that due to Vaccination introduction in EVD disease dynamics there has been decrease in the number of Exposed, Infected, Hospitalized and Deceased population while number of Recovered individuals have increased in same time interval compared to zero vaccination rate. Hence due to control interventions like Vaccination disease extinction would occur at an early stage.

6 Inference Analysis/ Comparison

- Our analytical and numerical results showed that both deterministic and stochastic models predict disease extinction when $R_o < 1$. However, the predictions by these models are different when $R_o > 1$. In this case, deterministic model predicts with certainty disease outbreak while the stochastic model has a probability of disease extinction at the beginning of an infection. Hence, with stochastic models, it is possible to attain a disease-free equilibrium even when $R_o > 1$. Also, we noticed that initial conditions do not affect the deterministic threshold while the stochastic thresholds are affected. Thus, the dynamics of the stochastic model are highly dependent on the initial conditions and should not be ignored.

- The model has shown success in attempting to predict the causes of Ebola transmission within a population. The model strongly indicated that the spread of a disease largely depends on the contact rates with infected individuals within a population.

  The model also pointed out that early detection has a positive impact on the reduction of ebola transmission; that is there is a need to detect new cases as early as possible so as to provide early treatment for the disease.

- It is also realized that if the proportion of the population that is immune exceeds the susceptible population, 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.

7 Contribution of team members

7.1 Technical contribution of all team members

Tasks	Team member 1	Team member 2
Simulation of deterministic figures	Yashvi Pipaliya	Yashvi Gandhi
Simulation of stochastic figures	Yashvi Pipaliya	Yashvi Gandhi
Simulation of figures with optimal path	Yashvi Pipaliya	Yashvi Gandhi
Simulation of new figures with vaccination	Yashvi Pipaliya	Yashvi Gandhi

7.2 Non-Technical contribution of all team members

Tasks	Team member 1	Team member 2
Concept map-1	Yashvi Pipaliya	Yashvi Gandhi
Concept map-2	Yashvi Pipaliya	Yashvi Gandhi
Report writing	Yashvi Pipaliya	Yashvi Gandhi

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