A MODIFIED SUSCEPTIBLE-EXPOSED-INFECTED-RECOVERED MODEL APPROACH FOR MODELLING THE TRANSMISSION DYNAMICS OF INFECTIOUS DISEASES

BY

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ABSTRACT

Emergence of infectious diseases has renewed research interests in disease transmission modelling. Susceptible-Exposed-Infected-Recovered (S-E-I-R) model has been used for such studies. A major assumption of infectious disease modelling using S-E-I-R is that the population is closed to migration. However, this was violated with the outbreak of Ebola virus of 2014 that spread across national boundaries. This study was therefore designed to formulate a modified S-E-I-R model which incorporates migration and to ascertain its effects on control of the spread of infectious diseases during outbreaks.

Migration rate was introduced into the susceptible population of S-E-I-R non linear differential equations to model disease transmission. The equilibrium points of the modified model and basic reproduction number were investigated using the next generation matrix. The local stability was analysed and global stability of disease free equilibrium was conducted by applying the Lyapunov function. Furthermore, the sensitivity analysis of the parameters was studied to determine their sensitivity to reproduction number by finding the derivative of each parameter with respect to reproduction number. Consequently, optimal control of the model was considered using Pontryagin maximum principle to determine the best control strategy to stem out the effect of the disease. Effect of environmental noise in the model was also studied by applying the stochastic differential equation. The current and the modifiedS-E-I-R(with migration) models were both demonstrated on numerical simulation and on 2014 Ebola Virus outbreak data in West Africa retrieved from the WHO website to analyse the effect of migration on the disease transmission.

The equilibrium points of the model were $E=0$ and $S=\frac{(\mu+\text{ь}+\delta)(\text{ь}+e+\mu)}{a\delta}$, while the basic reproduction number was, $R_0=\frac{a\Lambda\delta}{\mu(\mu+\text{ь}+\delta)(\text{ь}+e+\mu)}$ where S = susceptible, E= Exposed, μ = natural death rate , Λ= migration rate, a = transmission rate, ь= recovery rate, e = disease induced death rate and δ = progression into infected. The parameter estimates gave a = 0.000025, b = 0.48941, c = 1.963907, δ = 0.0498, μ = 0.002165, Λ =1034, e = 0.05019. The system was asymptotically stable with $R_0 < 1$. Migration and disease transmission rates were most sensitive. The reproduction number (2. 027),revealed persistence of the disease over model without migration (1.88). The 95% confidence interval of $1.9399 \leq R_0 \leq 2.0346$ accommodated the value of R_0.

The formulated model predicted persistence of the infectious diseases as a result of migration into susceptible population. In view of this development, stringent migration controls should be deployed during infectious diseases outbreaks to enable early containment.

Keywords: Susceptible population, Disease transmission dynamics, Stochastic model, Persistence infectious disease, Ebola virus

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DEDICATION

This thesis is dedicated to : Akorfa, Mawunyo, Edem and Ewoenam my sources of inspiration

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Adekunle, 2019

CERTIFICATION

I certify that this work was carried out by Mr. Joseph Adekunle Akinyemi (Matric No: 124538) in the Department of Statistics, University of Ibadan.

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CHAPTER ONE

INTRODUCTION

1.0 BACKGROUND OF THE STUDY

The emergence and continuing re-emergence of infectious diseases has renewed curiousity research in infectious diseases modelling. Infectious diseases such as Ebola cause change in the population of any community, region or country. Infectious diseases remain the foremost reason of morbidity and deaths in the world today, with diseases like HIV, Tuberculosis expected to cause about 10 per cent of the deaths witnessed every year. The just witnessed 2014 Ebola virus outbreaks in some parts of West Africa led to a significant record number of cases and deaths within a short time. Just like in the year 2003 SARS epidemic in which new pathogens were formed and in the Swine flu pandemic of 2009 and also in 2013 where the world witnessed the MERS-CoV epidemic. Mathematical models are known to be necessary and appropriate instruments in examining the extent and how to control the spread of infectious diseases. A careful study of the transmission characteristics of these diseases in the world and most especially in the developing countries will lead to a better knowledge in decreasing the spread and the passing of these contagious diseases. However,mathematical models are continuously in use to investigate the diffusion patterns of infections and to ascertain the would-be effect of control strategies in controlling the various havocs of morbidity and mortality caused by these diseases. Applications of mathematical models include determining optimal control strategies in

opposition to sprouting infections, such as Lassa fever, Swine flu or Ebola, Tuberculosis or alongside HIV, Malaria, and Dengue fever and predicting the effect of vaccination strategies in opposition to these infections. Modelling of the spread of infectious diseases was used in the United Kingdom elaborately during the outbreak of swine flu pandemic to monitor the degree of the passage into individuals and the potential impact of management measures such as the closures of schools and also the use of vaccination. The formulation of models sheds light on the assumptions, parameters and variables, furthermore, models give theoretical results like thresholds number, reproduction and contact numbers, stability analysis, and replacement numbers. Mathematical model and computer numerical simulation are important investigational tools to build, test and examine these theories, assess quantitative speculations, answer specific questions, determine how parameters change in values due to their sensitivities, and estimate main parameters from data used. Mathematical models such as S-E-I-R are also used to compare, plan, implement, evaluate, analyse and optimize detection,therapy, prevention, and control of various programs to curb the effect of the spread. In the last few years, several efforts have been made to formulate real life mathematical models for the infectious diseases. Infectious diseases are the root cause of death in most of the developing countries. These models have important roles to play in illustrating the forceful evolution of infectious diseases into mankind lives. It enables the effective study of the spread and growth of infectious diseases possible. The spread of epidemics can be modelled by using deterministic compartmental models where the population amongst where the disease is sprouting can be divided into several classes of individuals such as susceptible (S), exposed (E), infected (I) and removed (R). In studying the spread of infectious diseases, two approaches readily come to mind:

deterministic approach and stochastic approach. Many works have been done on deterministic approach of studying the spread of infectious diseases. Another addition is the modelling of transmission dynamics of the diseases in a stochastic manner. This can shed more lights into the transmission of new infections under a random nature. In nature,various systems show stochasticity in themselves or they are subjected to random perturbation.Time to time there may be a need to inculcate such randomness into the modelling. However,in a stochastic approach, a better solution is to present the stochastic models based on deterministic one.The unconventional nature of the spread and growth of epidemic is interestingly random because of the unexpected mode of person-to-person contact and population involved is subject to an incessant variety of commotion. Because of the uncertainty in the spread of the disease, emerging infectious diseases are a global problem. At the inception of the disease outbreak, those that will be infected will be small in number, and due to the random variation which alone can make an outbreak of epidemic to cease, it is imperative to incorporate this variation in the study and analysing models for emerging infectious diseases. Stochastic epidemic models are concerned or take care of the randomness in infectious contacts occurring in the latent and infectious periods. A model that is stochastic in nature is formulated in terms of its random variables whose probabilistic dynamics depend on solution of differential or difference equation. It is used to model the inherent variability present in the process due to demography or the environment. Stochastic models are particularly important when the variability is large. Various methods or approaches have been used towards introducing stochastic implications of nature into epidemiology models. Some of the approaches are the Continuous Time Markov Chain (CTMC), Discrete Time Markov Chain (DTMC) and

Stochastic Differential Equation (SDE). Models of stochastic differential equation are referred to as SDE epidemic models in which significant uncertainty is present. Stochastic epidemic model is the one that allows some random fluctuation affecting the spread of the diseases. Due to environmental noise, the deterministic models have some constraints in the mathematical modelling of the spread of a deadly contagious disease, as a result, many authors like Cai et al (2013), Britton and Giardina (2014), Rachah and Torres (2015), Hu and Shen (2015) have started to think about the consequences of environmental noise in epidemic disease models, which entails perturbation of the parameters and also perturbation around the endemic equilibrium of the epidemic models or perturbation on the state variables.

A population is a group of individuals of the same species who live together in the same habitat. Also, it is an association of species of living things among whose members interbreeding occurs. A population is aggregation of all the organisms, of identical group, who live in the same environmental area, and have the tendency of inter breeding. United States Census Bureau estimated the world population in the year 2012 to be 7.185 billion which is expected to surpass 10 billion in the year 2055. In the future, the world's population is expected to reach its maximum after which it will decrease due to economic reasons, fitness concerns, land exhaustion and ecological hazards. However, the population keeps on changing due to the dynamics. Population dynamics is the study of short and long terms in the sizes and age composition of population in a community and the biological and environmental processes affecting these changes. These population dynamics include birth and death. One of the causes of death in human population is disease (National vital statistics reports 2007). The Oxford English Dictionary defines a disease as "a condition of

some parts or whole body, in which its activities are bothered." It is a gloomy corporeal condition and a departure from its present state of sound health, which is caused by structural physical changes. This definition encompasses a wide range of ailments from Aids to Arthritis, from Ebola to Dengue fever, from the common cold to cancer. Diseases can either be infectious or non-infectious. Infectious diseases as Ebola, HIV can be passed between individuals, whereas non-infectious diseases such as Arthritis develop over an individual's life span. Infectious diseases are those diseases that are passed from one person to another person by direct or indirect or unprotected contact. It is an illness caused by infectious agents. It is a disease caused by the entrance into the body of persons (as bacteria or viruses) which develop and multiply. Although, such a description of dynamical behaviour of the disease allows us to understand the behaviour of infection within an individual and may even show some lights on its potential spread and transmission.

1.1 JUSTIFICATION AND MOTIVATION FOR THE STUDY

Due to the extensive analysis on the SIR and SIRS models, some diseases like Ebola, however, spend some time inside the host before the hosts start manifesting the infection. In view of this, epidemic models that will incorporate the activities or role of incubation periods in the spread of the diseases that are more than the SIR and SIRS models need to be looked into to examine this phenomenon.

Following the recent outbreak of Ebola virus in Liberia, Sierra- Leonne and Guinea which extended to Nigeria through migration, we deemed it fit to review the existing models of Ebola virus disease and to evolve another model that will take into account of the

exposed individuals and migration into the susceptible population as well as extending the deterministic model to stochastic model by using the stochastic differential equation to see how the exposed individuals will behave in the presence of environmental fluctuation since our primary concern is the fate of the exposed individuals in the face of an epidemic taking into consideration of those that will recover upon treatment.

Also, we want to look at the introduction of treatment and if possible vaccines and other measures which we believe will curtail the diseases to the barest minimum.

1.2 STATEMENT OF THE PROBLEM

Zach (2012) model did not consider those who are exposed to the disease on the assumption that once an individual is infected, the individual automatically becomes infected. In this model, those that are latently infected to the disease, get infected but are not yet infectious are considered. Because, it is possible for an individual to be infected with disease and recovers from the impact of the disease. In addition, he considered a constant population but this model considered migration when the population is not constant which will be of great importance to the developing countries with less tight security at their borders, in order to put control measures in place.

1.3 AIM AND OBJECTIVES OF THE STUDY

General objective

The aim and general objective of this study is to formulate and analyse an epidemic model for infectious diseases.

Specific objectives

The specific objectives of this study are to:

- Formulate a deterministic model for the outbreak of the diseases and determine the equilibrium points of the model.
- Compute the Basic Reproduction Number, R_o of the model, and perform the stability analysis of the model.
- Determine the existence of bifurcation in the model and perform the sensitivity analysis of the parameters.
- Perform the optimal control system of the model.
- Extend the deterministic model to stochastic one.
- Perturb the exposed individuals in the stochastic model.
- Construct the conditions for the existence of the model .

1.4 DEFINITION OF TERMS

1.4.1 Epidemic

This is defined as the occurrence of infectious disease in a community that spreads rapidly and affecting a large number of individuals, then dies out. Mosby's dental dictionary (2008), defines epidemic as spreading widely and rapidly of infectious diseases among individuals in a single location or region. Illnesses termed epidemic are those that occur beyond expectations which can be traced to a single source. It can also be related to tragic events of large proportion such as the outbreak of Ebola in Liberia, Guinea and Sierra-Leonne which resulted in death of thousands of people.

The dynamics of an epidemic is as follows: initially, there are a few infectious individuals that are affected in a large susceptible population. If one of these affected

individuals passes the disease successfully to other individuals, the disease is likely to take off.

1.4.2 Endemic

This can be defined as the presence of an infectious disease in a region, community, or country over a long period of time, though definite, at a low infectious level. That is, it is a situation in which a disease is always present in a region. If an infectious disease remains in a population for a period of time, it is likely to stabilise fluctuation around the equilibrium. This equilibrium is referred to as the Endemic level of infection.

1.4.3 Epidemic model

An epidemic model is defined as a model that describes the transmission of infectious disease through individuals.

The modelling of these infectious diseases is an important tool to examine the process or how the disease spreads,transmits and to assess the expected cause of an outbreak in the future and to examine important ways or methods to control the outbreak (Daley and Gam, 2005).

There are two types of epidemic model namely: Deterministic model and Stochastic model.

1.4.4 Deterministic epidemic model

According to Brauver and Chavez (2001), deterministic or compartmental models are used when we are dealing with huge population. The people in the populace are assigned to diverse sub groups or compartments or class each in place of an identified stage of the epidemic. The rates at which individuals move from one subgroup to the other are articulated mathematically as derivatives, hence the model can be formulated by using

differential equations. Now, it is presumed that the size of the population in a partition can be obtained by finding the derivative with respect to time and that the process of the epidemic is deterministic in nature.

1.4.5 Stochastic epidemic model

It is a mechanism to describe the probability distribution of potential results of the epidemic to play a part in the chance variation in one or more inputs over time. These stochastic models rely on the likelihood variation in the risk of disease exposure and other illness dynamics (Trotter and Phillippe, 2001).

1.4.6 Markov process

It is a development in which its future behavior cannot be accurately predicted from its past behaviour and which involves random chance. The progress of an epidemic is an illustration of Markov process. The next state of the epidemic depends only on the existing state and does not rely on the sequence of past behaviour that came before it. This process will be characterized by a state space, and transition matrix which relates to the probability of a particular transition with an initial state of the state space.

1.4.7 Spatial epidemiology

This describes the depiction and the way geographic variations play important role in disease spread in the areas of demographic environment,behavioural, socio-economic analysis,infectious and genetic risk factors. It is a sub field of health geography which examines the study of the space distribution of health implications. Specifically, spatial epidemiology can be described by the examination of disease spread with the geographic variations.

1.4.8 Equilibrium points of the model

Equilibrium points are the points when there is no change in the system of equation. That is, when the equations in the subgroup are equal. They are also set of points in the system to decide whether there is a presence of disease in the system or not. If there is no disease in the system, it is called disease-free equilibrium, if there is a presence of disease then the points are endemic points.

1.4.9 Basic reproduction number

It is known or described as the anticipated cases of newly created or fresh cases of infections from one individual that is infectious in a population that is wholly susceptible through the entire length of the infection period denoted by, R_0. It defines the dynamical behaviour of the model,whether the disease dies out or it persists in the system. If $R_0 < 1$, the infection in one individual cannot reinstate itself so the pathogen dies out (stable disease free population). If $R_0 > 1$, the number of infectious persons increases and the disease persists and if $R_0 = 1$, there is an equilibrium: the endemic and the disease-free being equal.

1.4.10 Stability analysis of the model

The stability of the model is in two ways: Local and global stability. The stability nature of the system depends on the nature of the eigenvalues.

1.4.11 Local stability of the disease free equilibrium

For a given equilibrium point, the local stability can be instituted by analysing the Jacobian matrix alongside its eigenvalues. If all the eigenvalues at the equilibrium point have the real-part being negative values then the system is referred to be asymptotically stable locally.

1.4.12 Global stability of the disease free equilibrium

For an equilibrium point to be asymptotically stable globally, the first derivative of Lyapunov function L(E,I) of the model must be negative at the equilibrium.

1.4.13 Bifurcation

It is the point at which at least one point of the eigenvalues is zero. For forward bifurcation to occur the basic reproduction number has to be less than 1, while in the neighbourhood of 1, coexistence is possible with the equilibra of the endemic and the disease free, before the reproduction number is greater than 1 and this will make the backward bifurcation to exist.It shows how small dynamical change in an output of a parameter can cause changes in the behaviour of the whole system.

1.5 SOURCES OF DATA

Simulated data and real live data of Ebola 2014 are used.

1.6 VARIOUS MODELS OF AN INFECTIOUS DISEASE

Model is a way of representing the behaviour of a situation to facilitate us deduces what is the best to do about the system. Models are hence tools for representing a situation to understand it and for interpretation about it.

Mathematical model is an overt mathematical description of the basic dynamics of a system. These models are supportive in the control strategies and prevention mechanism of sprouting infectious diseases like Ebola, SARS,Influenza, HIV/AIDS.They are also functional in the careful study of the progress and spread of these drug resistant diseases.

THE S - I MODEL(Susceptible - Infected). In this model, the prone population (Susceptible) is infected and the infected population remain infected until they die for example, plant infection, HIV.

THE S - I -S MODEL (Susceptible - Infected - Susceptible) In this model, the susceptible population are infected with the disease and the infected population return to the susceptible class on recovery because the infected individuals have no permanent immunity against reinfection for example, sexually transmitted diseases like Gonorrhea, Syphilis.

THE S - I - R MODEL (Susceptible - Infected - Recovered) This is a model that describes individuals that are born into the Susceptible population and they are able to be infected after which the infected population move into the infected group and the infected population move into the recovered group and are assumed to be immune for life.

THE S -E - I - R MODEL (Susceptible - Exposed - Infected - Recovered) In this model, the Susceptible individuals move into the exposed class, but not yet infectious after which they become infected and are treated, the recovered individuals move into the recovered group.

THE S-E-I-R-S MODEL (Susceptible - Exposed - Infected - Recovered - Susceptible) In this model, the Susceptible individuals are exposed to the infection, has a latent period of being infected, those that are infected proceed to the Infected group, after the infected individuals are recovered either dead or alive, they later proceed to the Recovered group and the living has no permanent immunity to the diseases and they progress into the susceptible class again.

1.7 FACTORS THAT CAN LEAD TO EMERGENCE OF INFECTIOUS DISEASES

Here are a number of factors that can lead to the sprouting of infectious diseases: these are Population growth, global climate change, increased use of antibiotic for humans

and animals, industrial agriculture, human-animal contact, war and social disruption, relocation of animals, increase in number of day care and so on.

1.8 MODES OF TRANSMISSION OF INFECTIOUS DISEASES

These are some of the modes by which infectious diseases are being transmitted:

(a) Through Contact: This is sub-divided into four namely : direct, indirect, fomites and body secretion as in blood, urine, saliva.Diseases spread by contact are sexually transmitted diseases for example, Syphilis, Gonorrhea, AIDS.

(b) Through Airbone : This is in some particle aerosol as in tuberculosis, measles and so on.

(c) Through Food and water: This is as in contamination e.g Cholera, histeriosisand so on.

(d) Through Vector: This is as in Yellow fever, Dengue fever, Trypanosomiasis.

Reservoirs of Infectious diseases.

These are some known reservoirs:

(i) Humans: these are AIDS, Syphilis, Gonorrhea, Typhoid.

(ii) Animals: these are in Ebola, Rabies, Plague.

(iii) Soil: these are in Tetanus, Histoplasmosis.

(iv) Water: these are in Pseudomonous infection.

CHAPTER TWO

LITERATURE REVIEW

Infectious diseases are diseases that cause misery, sickness in the body of humans and death in humans and animals worldwide. Effective study of the spread, prevention and control are therefore important tasks both from human and an economic point of view.

2.1 REVIEW OF LITERATURE ON DETERMINISTIC MODEL

Lajmonovich and Yorke (1976) ascertained the occurrence of the stability of the unique equilibrium that is endemic using a class of deterministic SIS models and a complete analysis of the global dynamics of the disease by giving the global Lyapunov function.

Hethcote and Tudor(1980) studied endemic infectious diseases using a system of Volterra integration equations that are non linear and of convolution type. They looked at the models of the parameters with the introduction of vital dynamics, immunization and infectious period. After determining the threshold criteria and the asymptotic behaviour, they reached a conclusion that with a delay in the model the thresholds variable and the asymptotic behaviour of the model do not change.

Anderson and May (1991) did a study on the dynamic modeling of infectious diseases as an improvement on the Hethcote and Tudor (1980) and concluded that they occur in two different temporal patterns: Epidemic and Endemic.

Greenhalgh (1992) painstakingly examined models of SEIR type that could

incorporate death rate with density dependence, while Cooke and Drisssche (1996) initiated a research of SEIRS models with two delays.

Herwaarden and Grasmen(1995) specified that, in a deterministic system, there was an endemic situation which can disappear due to the stochastic fluctuations in the corresponding stochastic model.

Astacio*et al (*1996) used the S-E-I-R model which was a modification to the S - I - R to model the 1976 outbreak of Ebola in Yambuktu and 1995 outbreak in Kikwit, Zaire. They assumed a constant population and did not consider the idea of quarantine of the infected individuals.

In the works of Greenhalgh(1997), he introduced Hopf bifurcation in the model which is SEIRS in nature with a density dependent contact rate and death rate.

Li and Muldoney (1995) and Li *et al* (1999), both studied global scenario of the dynamics of SEIR models with an incidence rate that is non- linear and with an incidence rate that is standard respectively. Hal *et al* (2001) worked on SEIR model to find the global dynamics of the disease having a vertical transmission and a bilinear incidence rate.

Alum (2001) studied the effects of the period of infection within SIR models and he discovered that less spread distribution wave seem to have two fundamental epidemiological consequences. It was discovered that the disease reduced in its persistence and showed an unstable behaviour in the model that has a finite population.

Grenfell and Hutscher (2003), in their lecture notes on modeling the dynamics of infectious diseases used three areas namely (i) childhood disease dynamics and vaccination, (ii) Spatio-temporal disease dynamics and (iii) evolution of diseases with multiple strains. They used mathematical techniques to analyse the bifurcation theory of

Ordinary Differential Equations (ODEs), wavelet analysis and stochastic simulation.

Hisashi(2003) used a simple S-I model to check the presence of bifurcation in the model. It showed a backward one for a disease caused by the transmission of a vector: protozoan parasite Trypanosonacruiz. He introduced the birth rate into the host population in the model and showed that there was an occurrence of backward bifurcation with an endemic state that was steady depending on the parameters, and that existence of death induced by the disease formed an essential role for the occurrence of a backward bifurcation.

Chowell*et al*(2005) used epidemic modeling and data of Ebola outbreak to ascertain the average number of newly created infections produced by an indicator index when there was no control management intervention put in place. The SEIR epidemic model was used to model the cause of the outbreak and also to look at the rate of transmission of the disease after control interventions were put in place. They performed uncertainty analysis of the effective reproductive number R_0 to see how sensitive the model is to other disease-related parameters. They discovered that the control measures put in place reduced the final size of the epidemic by a factor of 2 relative to the final size with a delay of two weeks with the implementation,however the exposed stage did not consider those who recovered at this stage.

Hohle*et al* (2005) introduced the use of spatial study by using a multi group epidemic in the SEIR model by extending the works on Monte Carlo Markov Chain(MCMC) estimation of parameters by O'Neill and Roberts (1999). They extended the deterministic SEIR epidemic model to stochastic SEIR model to reflect the transmission experiments and for the estimation of the parameters by maximum likelihood

and Bayesian inference.

Bubniakova (2007) used the deterministic modelling approach to study the dynamical behaviour of infectious diseases. He modified three (3) models S-I, S-I-R and S-E-I-R

Legrand*et al* (2007) modified the S-E-I-R model and developed a S-E-I-H-F-R model to study the spread and transmission of Ebola virus to include those at hospital and funeral but assumed homogeneous population which according to them was too simple which may not be effective in countries where the structure of the community favours infection in households and recovered individuals were not returned in to the population

Zhang *et al (2007)* also studied and introduced a saturating contact rate in the SEIR model while considering its global dynamics.

Also, in the book of Allen(2008),the three approaches DTMC, CTMC and SDE were compared using SIR epidemic model.

Jiang and Yang(2009) studied the dynamics of an SIS epidemic model with the introduction of birth pulses and a varying population. They conducted a research about the presence and stability of two periodic solutions namely the endemic and the infection-free using discrete maps,the bifurcation theorem and the center manifold theorem and discovered that the numerical results and bifurcation diagrams agreed with the theoretical analysis.

Neilan (2009) presented three different models with optimal control problem which describe population dynamics of diseases using systems of differential equation. She introduced quadratic growth function in the first model and in the second model, three control strategies viz: sanitation, antibiotic treatment and vaccination were proposed. The

third model showed a control function that represents vaccination in a three parabolic partial differential equations.

Smieszek(2009) examined the configuration and the quality of contacts between hosts shape. He evaluated the importance of characteristics of contact for constructing mathematical models for the spread of the disease to ascertain the effectiveness of interventions on the epidemics.

Bunomo and Lacitignola (2011) modified S-I model to include vaccinated group with a non linear incidence rate and an inadequate vaccine that serves as a preventive measure which was given to susceptible individuals to study the backward bifurcation of the model. They introduced a recruitment parameter rate of the susceptible and got the conditions for the backward bifurcation.

Wang *et al*(2011) introduced a function which is a treatment function that is saturated into the epidemic S-I-R model and a bi-linear prevalence rate with density-dependent demographics. They performed global qualitative and bifurcation analysis, they found that the system showed that the moment basic reproduction number is under the turning point values, the disease can be removed and also, existence of backward bifurcation in the model was established.

Zach (2012) instituted,developed and examined a mathematical model to analyse the spread of Ebola virus disease from S-I-R model, assumed constant population and did not consider exposed stage and failed to include those that may be treated as in the case of 2014.

Evans (2012) applied social network and analysis together with the data mining method to showcase a model on negative social response (NSR) in a community which

demonstrates a strain that is associated with a disease. He developed a meta-model that showed the relationship between the spread and NSR to an outbreak of a disease over a network. He used a S-I-R model and a social influence model.

Althaus (2014) modified S-I-R model for the outbreak of Ebola virus in 2014 in West Africa to study the spread of infection in the countries that were affected by the virus when there were no control measures in place using an SEIR model. The study showed actual time estimates of the disease transmission parameters during the outbreak and assumed closed population and those that were not affected by the disease at the exposed stage were not considered. He however estimated the reproduction number for the outbreak of 2014 Ebola virus in some parts of West Africa as between 1.5 and 2.5

Fasina*et al* (2014) analysed epidemiological data of 2014 Ebola virus disease outbreak in Nigeria. Their model divided the population into five categories: susceptible, exposed, infectious and symptomatic individuals, hospitalised individuals and those individuals separated from isolation centre after recovery or those that died as a result of the disease. The model developed was S-E-I-H-P from S-E-I-R

Jianjun*et al* (2015) used S-I-R model for two regions which can be connected by transportation to study the effect of vertical transmission, impulsive display on the spread of the disease. The model showed the evolvement of the disease. In the study, it was concluded that the approach of controlling the activities of the infected individuals using transportation provides an important basis of preventing the spread and control of the disease and did not consider those who may be exposed to the infection as a result of the transportation.

Njankou (2015) did a work on six compartmental mathematical models

wereformulated to gain an insight into the role of media campaign on Ebola transmission as a mean of controlling the spread using two different approaches with a conclusion that media campaigns should be spaced out for them to be effective.

In the work of Andrea (2016), the optimal treatment of infectious disease was investigated using the SIS epidemic model. The model was transformed to SEIV incorporating the cost of treatment of the entire population. Five different cost functions were considered.

Bohm $et\ al$ (2016) incorporated individual specific immunity in a heterogenous population and exploring the best optimal control system viz : vaccination, tretament and chemoprophylaxis intervention on a SIS models. They concluded that the continuation of chemoprophylaxis and treatment provided the strongest effect.

Bonjakjian(2016) used SEIR model to explore the potential impact of vaccination and quarantine on the spread of Ebola in West Africa. However, in the model, birth rate variable was not included in the model.

In the works of Akinyemi*et al* (2018), they discussed about the stability analysis of infectious diseases by introducing a migration rate into a population that is dynamic, the model exhibited two equilibria and it was discovered that the system is locally and globally stable when the reproduction number is less than 1.

2.2 REVIEW OF LITERATURE ON STOCHASTIC MODEL

Mckendrick (1926) was the first person to propose the study of stochastic epidemic in 1926 in Kermacck and Mckendrick (1927) which in 1928 and 1931, Reed and Frostr and Greenwood proposed the use of discrete time stochastic models to solve generations of

infective respectively.

Jacquez and O'Neill(1991) compared the deterministic threshold results and the stochastic threshold results of a population that is divided into two namely: susceptible population and infected population. They used S-I model with recruitment,also with death due to the disease and discovered that basic reproduction number,R_0,has an important role to play for both versions though, the threshold results were different. For the deterministic model, no epidemic occurred when $R_0 < 1$ and epidemic occurred when $R_0 > 1$. In the study, the stochastic model showed that, when $R_0 < 1$, no epidemic occurred and when $R_0 > 1$, a probability of finite value that is less than 1 that an epidemic will occur was discovered and eventuated in an endemic quasi-equilibrium. Also, Jacquez and O'Neill (1991) compared the reproduction number in deterministic and stochastic models and found out that the reproduction number plays much the same role in defining thresholds for epidemic take off in both models.

Mao *et al*(2002) showed that with the introduction of a sufficiently small noise explosion in the population dynamics, the impact of the disease could be reduced.

To better understand the stochastic models, Nasell (2002) extended the stochastic models to infer that stochastic models have a better and a reasonable approach in describing the spread of the epidemics for a sufficiently large array of reasonable values of the parameter when compared with their counterpart deterministic models.

Imhof and Walcher (2005)analysed a variant deterministic chemostat model and compared it with a stochastic chemostat model. By studying the two models, they proved that the stochastic model went into extinction while there was a persistence showing with the deterministic model.

Frank (2007) considered problems associated with modeling of stochastic energy infections. He proposed solution to the problem that an epidemic died out when no asymptomatic cases observed. He looked at the effect on the behaviour of a household model and the delay length between discovering time of infection in a household and implementation of an intervention. He also developed a model for an emerging strain of influenza in humans to look for the risk that the disease poses.

Dalal*et al* (2007) discovered that stochastic models had solutions that are positive in nature.They did a survey on the asymptotic behaviour of the models by analysing the stability of the models. In the works of Tornatore*et al* (2005), the nature of the stability of a disease free equilibrium of a stochastic SIR model was studied while in the works of Beretta *et al*(1998), stochastic perturbation around the endemic equilibrium that is positive was considered.

Britton(2009) used a stochastic epidemic model to study the effects of vaccination on a small and large community. He made use of S-I-R model. He did a survey paper on stochastic epidemic models. He assumed that the population is homogeneous and closed.

Ji*et al* (2011) accounted for the effect and nature of environment that is randomly fluctuating by doing a study on the various group of epidemic model that are of SIR type using stochastic perturbation. The states variables were perturbed.

Mitchell (2011) developed a semi -markov discrete time multi-state models that can be used to study HPV persistence and proposed a maximum likelihood estimator of HPV persistence using a semi-markov two -state discrete-time model for incident infectious. Anderson and Britton (2001) did a Maximum Likelihood Estimation (MLE) process and Monte-Carlo Markov Chain(MCMC) methods to study the spread of the

disease.

Yuan and Allen (2012) considered the problem arising from multi group SEIR model of Guo and Li (2008) and SIR model of Guo*et al* (2006) by using stochastic perturbation around their endemic equilibrium.

O'Neill and Wen (2012) studied the application of the different criteria and computational methods inside the epidemic models by setting up effective scheme to identify a feasible criterion for specific epidemic data sets.

In their study of infectious disease,Cai*et al*(2013) did a careful study of the dynamics of an epidemic model globally which is of SIRS type and introduced a dependent ratio of incidence rate into the population with a resultant stochastic differential equation (SDE) version. The study showed that, the reproduction number R_0 of the disease indicates whether or not an endemic outbreak would occur. When $R_0 \leq 1$ it revealed that the disease free dynamic occurred and the endemic steady state that was globally stable occurred when $R_0 > 1$. Random fluctuations were introduced in the stochastic model to reduce the outbreak of the disease which provided good control strategies on the disease dynamics.

Ndanguza*et al* (2013) used onset and death data of Ebola outbreak in the Democratic Republic of Congo DRC in 1995, to analyse the spread of the disease. They used Markov Chain Monte Carlo (MCMC) algorithm and Least squares estimation to analyse the two sets of the data. They discovered that the model fitted well in to the onset data of Ebola virus at 99.95% and that of the death data at 98.6% confidence interval.

In the papers of Hamidreza*et al* (2013), they considered diffusion model in social network in stochastic information. They used discrete time markov model.

Cai*et al* (2013) investigated how environment fluctuations affect the dynamics of the disease by using a stochastic SIRS epidemic model and discovered that fluctuation introduced in the model can reduce the outbreak of the disease which provided useful way of controlling and regulating the disease transmission by perturbing the state variables.

Fabio *et al* (2013) used two different approaches to SIS epidemiological model namely: one with a differential equation and the other one on discrete time markov chain and compared the two scenarios and found out that the stochastic model and the ODE model are at variance. While, Rachah and Torres (2015) used SIR epidemic model to model the 2014 Ebola outbreak in West Africa and introduced vaccination to the susceptible as part of the control system and concluded that vaccination was a very efficient factor in reducing the number of infected individuals.

Britton andGiardina (2014) used stochastic epidemic S-I-R model for the spread of infectious diseases and performed parameter estimation for the stochastic epidemic models. They assumed a closed population and no immune individuals.

Hu and Shen (2015) extended the deterministic SIS classical model to a stochastic SIS discussed by Gray *et al* (2011), by introducing two random perturbation namely: the cure and transmission parameters and discovered that the stochastic SIS is weaker than the deterministic which allows infectious I(t) go extinct and in the deterministic SIS model, I(t) did not go extinct.

Chuang (2015) studied the global dynamics of a stochastic SIS model by using stochastic differential equation. He discovered that the stochastic prevalence of disease is bigger than that of deterministic disease prevalence that is noise may increase severity of disease.

In the paper of Xu (2015) also presented the stochastic SIS epidemic model developed by Gray et al (2011) and found the stochastic reproduction number R_0^s. He discussed that the disease was recurrent when $R_0^s \geq 1$.

Britton (2009) studied the effect of vaccination on the stochastic epidemic model by using SIR epidemic model and Swishchuk*et al* (2016) studied different types of stochastic stability for the deterministic epidemic models while Britton and Giardina (2016) analysed the general stochastic epidemic model using SIR model and found the reproduction number (R_0) by introducing the critical vaccination coverage under a closed population with homogeneous mixing in continuation of the work of Britton (2009).

Kitengeso (2016), introduced the development of a stochastic model to capture and minimize the transmission dynamics of measles in order to show the strength of stochastic methods in the analysis vis-a-viz deterministic methods and also to show the importance of vaccination in the control of the spread of measles. He used SDE on SEIR with control strategy. The study shows the effectiveness of stochastic analysis.

Meng $etal$ (2016) proposed a stochastic model in studying the dynamics in heterogeneous population of infectious disease using temporal spatial surveillance data. They perturbated the infected group and used stochastic model to quantify the significant role of the heterogeneity in the disease spread dynamics analysis.

Rachah and Torres (2017) used a continuous time model to study the effect of Ebola spread in the SEIR model. They dealt with classical derivatives and integer order system. They introduced vaccination as a control system with vital dynamics effects on the population.

CHAPTER THREE

METHODOLOGY

The objective of this work is to formulate a model that will best describe the recent outbreak. Mathematical models can be employed to project and explain how these infectious diseases spread and progress to showcase the expected outcome of an outbreak of the epidemic and to help in the area of public health intervention. We are going to make use of the last model reviewed by Zach (2012) to develop our model.

The model from Zach (2012).

$$\frac{dS(t)}{dt} = -aS(t)I(t) + cR(T) \quad (1)$$

$$\frac{dI(t)}{dt} = aS(t)I(t) - ط I(t) - eI(t) \quad (2)$$

$$\frac{dR(t)}{dt} = ط I(t) - cR(T) \quad (3)$$

$$\frac{dD(t)}{dt} = eI(t) \quad (4)$$

where

a = the rate of infection

ط = the rate of recovery

c = the rate of susceptibility

e = the rate of death.

3.1 DISCUSSION OF THE MODEL

The model we are considering used epidemic model SIR. The model divides the population into the groups of Susceptible, Infected and the Recovered. The Susceptible group is expressed by S(t) , Infected by I(t) and the Recovered by R(t).

Zach (2012) assumed a closed population which we are not going to assume. We assume that the population is dynamic. The infection is assumed to spread through contact between the susceptible and the infected.The model assumed the population of susceptible and that of the infected are distributed randomly over an area. But with what happened in the outbreak of Ebola virus in 2014, which violated the closed population assumption which made the virus extended to Nigeria, migration plays important role in the transmission of infectious diseases. The exposed population will be introduced into the model.

3.2 THE GENERAL S-E-I-R MODEL WITHOUT THE DYNAMICS

The general S-E-I-R model without incorporating migration and other vital dynamics is given below:

$$\frac{dS_*(t)}{dt} = -aS(t)I(t) + cR(t)$$

$$\frac{dE_*(t)}{dt} = aS(t)I(t) - \delta E(t)$$

$$\frac{dI_*(t)}{dt} = \delta E(t) - \text{ط}I(t)$$

$$\frac{dR_*(t)}{dt} = \text{ط}I(t) - cR(t)$$

which will be regarded as system 1.

In the course of the epidemic, the susceptible (S)comes in contact with the infected(I)at a constant rate of "a" which will reduce the susceptible population, which

signifies that there is a negative constant 'a' which expresses the product of susceptible (S) and the infected (I) in the two populations and defines the rate at which susceptible become infected. There will be a similar relationship between the infected and the recovered too.

3.3 THE GENERAL S-E-I-R MODEL WITH THE VITAL DYNAMICS

With the vital dynamics to be introduced into the S-E-I-R model which will accommodate the migration into the susceptible population, and the disease induced and natural death rates will be studied via the diagram in Figure 1. The schematic diagram showing the flow of the virus in an individual where infection occurs at a(S,I),latency to infectious at δE and the recovered from infected group move to recovered group at طI.

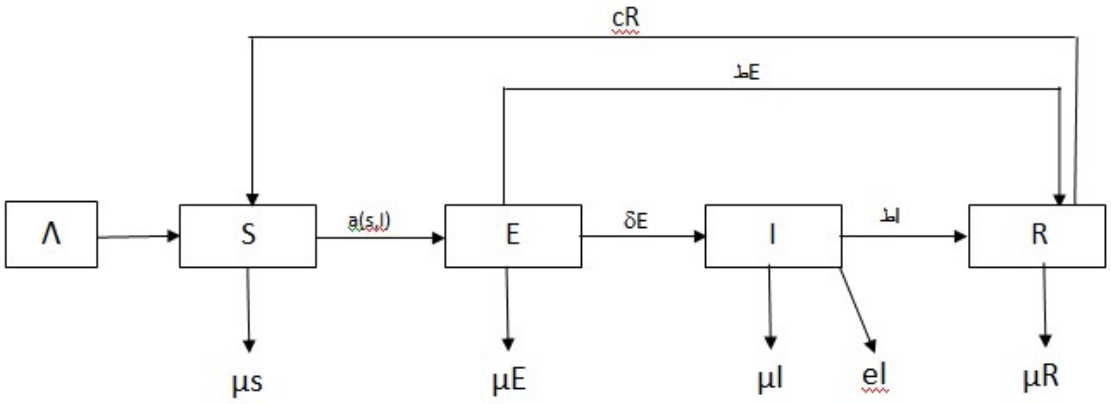


Figure 1 The schematic diagram of the flow of the virus.

We will build the model for infectious diseases based on the Zach Yarus model by slightly modifying the model.

To create a robust equation that will describe the susceptible population in relation to time, we introduced a migration rate into the susceptible class which is denoted by Λ, that is the population is assumed dynamic. Also, the susceptible becomes infected at the rate of "a" and the population of susceptible is reduced as infected come into contact with the susceptible.

We also looked at those that will die natural death in the susceptible group not as a result of the epidemic but other natural death which also reduce the susceptible group. This will be expressed as

$$\frac{dS_*(t)}{dt} = \Lambda - aS(t)I(t) - \mu S(t) + cR(t).$$

We looked at the susceptible before they are infected, they will also be exposed to the infection but not yet infectious so we introduced the exposed group into the model which will be that the susceptible individuals will be reduced by the natural death of the susceptible. Those that will be exposed to the infection and recover will exit the group and move to the recovered group. This can be written as

$$\frac{dE_*(t)}{dt} = aS(t)I(t) - \mu E(t) - ط E(t) - \delta E(t).$$

The equation that describes the infected population is adding what was removed from the exposed group. Those that eventually recovered from this group upon treatment or after the control measures have been put in place will exit the group and be moved into recovered group. Also, those that died naturally and those that died as a result of the infection will leave the group. Thus, the equation becomes

$$\frac{dI_*(t)}{dt} = \delta E(t) - ط I(t) - eI(t) - \mu I(t).$$

The population of the infected group is reduced in three ways; those that recovered , those that died naturally and those that are killed by the disease.

The recovery group comprises of those that recovered after being infected and those that are recovered upon exposure.However, this group will be reduced by those that died naturally from the recovered group,and this will be written as

$$\frac{dR_*(t)}{dt} = ط I(t) + ط E(t) - cR(t) - \mu R(t).$$

The assumptions are:

(i) There are births and deaths, immigration and emigration during the period of epidemic.

(ii) Only susceptible individuals can get exposed.

(iii) Exposed individuals get infected but not yet infectious for some time.

(iv) The recovered individuals that are not killed by the disease move into the susceptible class again.

(v) It is a dynamic population.

So, from Zach (2012) model, our own model will now be

$$\frac{dS_*(t)}{dt} = \Lambda - aS(t)I(t) - \mu S(t) + cR(t) \quad (5)$$

$$\frac{dE_*(t)}{dt} = aS(t)I(t) - \mu E(t) - ط E(t) - \delta E(t) \quad (6)$$

$$\frac{dI_*(t)}{dt} = \delta E(t) - ط I(t) - eI(t) - \mu I(t) \quad (7)$$

$$\frac{dR_*(t)}{dt} = ط I(t) + ط E(t) - cR(t) - \mu R(t) \quad (8)$$

$$\frac{dD}{dt} = eI(t) \quad (9)$$

where

Λ= the migration rate at which the susceptible class is being populated

a= the rate at which the infection spreads

ط= the rate of recovery from the infection

c= the rate at which recovered humans progress back to the susceptible class

e= rate of death caused by the disease

μ= death rate caused by natural phenomenon

δ= progress rate of the exposed class into the infected compartment.

Equations 5-9 will be regarded as system 2. Since the ninth differential equation is independent of equations 5-8 then it suffices to consider equations 5-8 which will be regarded as system 3. So our system 3 will now be

$$\frac{dS_*(t)}{dt} = \Lambda - aS(t)I(t) - \mu S(t) + cR(t) \quad (5)$$

$$\frac{dE_*(t)}{dt} = aS(t)I(t) - \mu E(t) - ط E(t) - \delta E(t) \quad (6)$$

$$\frac{dI_*(t)}{dt} \delta E(t) - ط I(t) - eI(t) - \mu I(t) \quad (7)$$

$$\frac{dR_*(t)}{dt} = ط I(t) + ط E(t) - cR(t) - \mu R(t) \quad (8).$$

In system 3,

N=S+E+I+R

of those that are living.

Taking the differential equation with respect to time

$$\frac{dN(t)}{dt} = \frac{dS_*(t)}{dt} + \frac{dE_*(t)}{dt} + \frac{dI_*(t)}{dt} + \frac{dR_*(t)}{dt}$$

substituting,

$$\frac{dN(t)}{dt} = \Lambda - aSI(t) - \mu S(t) + ط E(t) + cR(t) + aSI(t) - \mu E(t) -$$

$ط E(t) - \delta E(t) + \delta E(t) - ط I(t) - eI(t)$

$$-\mu I(t) + ط I(t) + ط E(t) - cR(t) - \mu R(t)$$

This will be reduced to

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

$$= \Lambda - \mu N - eI$$

if e $\approx$ 0, that is, in the long run if control measures and intervention are put in place and the disease induced death is reduced to the minimum, the population is assumed to be

$$\frac{dN}{dt} = \Lambda - \mu N$$

i.e

$$\frac{dN}{dt} + \mu N = \Lambda$$

solving the ODE becomes,

$$e^{\mu t}\frac{dN}{dt} + Ne^{\mu t} = \Lambda e^{\mu t}$$

$$I.F = e^{\int \mu dt} = e^{\mu t}$$

$$\frac{d(e^{\mu t}N)}{dt} = \Lambda e^{\mu t}$$

$$e^{\mu t}N = \Lambda \int e^{\mu t}$$

$$e^{\mu t}N = \frac{\Lambda}{\mu}e^{\mu t} + C_1$$

where C_1 is a constant of integration

$$N(t) = \frac{\Lambda}{\mu} + c_1 e^{-\mu t}$$

as t→ ∞, the total population in that community reaches a constant value.

$$N(t) = \frac{\Lambda}{\mu}$$

Thus, the size of the population may vary in time, but without the disease over time, the size of the population returns to the steady state of $\frac{\Lambda}{\mu}$.

3.4 EQUILIBRIUM POINTS OF THE SYSTEM(MODEL)

At equilibrium, we set

$$\frac{dS_*}{dt} = \frac{dE_*}{dt} = \frac{dI_*}{dt} = \frac{dR_*}{dt} = 0$$

that is, when there is no change in the number of susceptible class, exposed class, infected class and the recovered class over time then,

$$\frac{dS_*}{dt} = \frac{dE_*}{dt} = \frac{dI_*}{dt} = \frac{dR_*}{dt} = 0$$

If the rate of change is zero that means the system is at equilibrium. The work is to find out, at what points the system 3is at equilibrium?

$$\frac{dS_*}{dt} = \Lambda - aSI - \mu S + cR = 0$$

$$\frac{dE_*}{dt} = aSI - (\mu + ط + \delta)E = 0$$

$$\frac{dI_*}{dt} = \delta E - (ط + e + \mu)I = 0$$

$$\frac{dR_*}{dt} = ط I + ط E - (c + \mu)R = 0$$

from equation 7,

$$\delta E - (ط + e + \mu)I = 0$$

therefore,

$$I = \frac{\delta E}{(b+e+\mu)} \tag{10}$$

substitute I in equation 6

$$aS\left(\frac{\delta E}{b+e+\mu}\right) - (\mu + ط + \delta)E = 0$$

$$E\left(\frac{a\delta S}{b+e+\mu} - (\mu + ط + \delta)\right) = 0$$

therefore,

$$E = 0$$

or

$$\frac{a\delta S}{b+e+\mu} - (\mu + ط + \delta) = 0$$

$$E = 0$$

or

$$S = \frac{(\mu+b+\delta)(ط+e+\mu)}{a\delta}$$

These are the stationary points.

Now, when E=0, I=0 substituting I=0, E=0 in equation (8)

$$ط I + ط E - (c + \mu)R = 0$$

therefore,

$$-(c + \mu)R = 0$$

R=0 substituting R=0, I=0 in equation(5)

$$\Lambda - aSI - \mu S + cR = 0$$

$$\Lambda - \mu S = 0$$

it implies

$$S = \frac{\Lambda}{\mu}$$

Now, we have values for S, E, I, R when E=0

Let

$$P_*^0 = (S^0,\ E^0,\ I^0,\ R^0) = (\frac{\Lambda}{S}, 0,\ 0,\ 0)$$

This is the disease-free point because at this point, there is no infection because I=0

Let

$$P^* = (S^*, E^*, I^*, R^*)$$

be the values for S,E,I,R when

$$S = \frac{(\mu + ط + \delta)(ط + e + \mu)}{a\delta}$$

substituting S in equation(6)

$$aSI - (\mu + ط + \delta)E = 0$$

$$a\left(\frac{(\mu+ь+\delta)(ь+e+\mu)}{a\delta}\right)I-(\mu+ь+\delta)E=0$$

$$(\mu+ь+\delta)(ь+e+\mu)I=\delta(\mu+ь+\delta)E$$

$$(ь+e+\mu)I=\delta E$$

therefore,

$$E=\frac{(ь+e+\mu)I}{\delta} \tag{11}$$

substituting E in equation (8) we have

$$ьI+ьE-(c+\mu)R=0$$

$$ьI+ь\left(\frac{ь+e+\mu}{\delta}\right)I-(c+\mu)R=0$$

$$(c+\mu)R=ь\left(1+\frac{(ь+e+\mu)}{\delta}\right)I$$

$$R=ь\left(\frac{\delta+(ь+e+\mu)}{\delta(c+\mu)}\right)I \tag{12}$$

substitute equation (12) and the value of S into equation (5)

$$\Lambda-aSI-\mu S+cR=0$$

$$\Lambda-\left(\frac{(\mu+ь+\delta)(ь+e+\mu)}{\delta}\right)I-\mu\left(\frac{(\mu+ь+\delta)(ь+e+\mu)}{a\delta}\right)+bc\left(\frac{\delta+(ь+e+\mu)}{\delta(c+\mu)}\right)I=0$$

$$I\left(bc\frac{\delta+(ь+e+\mu)}{\delta(c+\mu)}\right)-\left(\frac{(\mu+ь+\delta)(ь+e+\mu)}{\delta}\right)=\mu\left(\frac{(\mu+ь+\delta)(ь+e+\mu)}{a\delta}-\Lambda\right)$$

$$I\left(\frac{ьc(\delta+ь+e+\mu)-(c+\mu)(\mu+ь+\delta)(ь+e+\mu)}{\delta(c+\mu)}\right)=\frac{\mu(\mu+ь+\delta)(ь+e+\mu)-a\Lambda\delta}{a\delta}$$

therefore,

$$I=\frac{(c+\mu)(\mu(\mu+ь+\delta)(ь+e+\mu)-a\Lambda\delta}{aьc((\delta+ь+e+\mu)-(c+\mu)(\mu+ь+\delta)(ь+e+\mu)} \tag{13}$$

Since $I\neq 0,$ it shows there is a presence of infection in the system. It is at endemic point.

At the disease-free point, there is no need of using drug because there is no

infection at this point. The migration rate into the susceptible class should be more than the natural death rate that is, $\Lambda > \mu$.

3.5 COMPUTATION OF THE BASIC REPRODUCTION NUMBER, R_0

By the use of next generation matrix,

$$G = FV^{-1}$$

where F is the matrix of the newly created infection, V is the matrix of transferred infection.

V^{-1} is the inverse of matrix V

so,

$$F_i = \begin{pmatrix} aSI \\ 0 \\ 0 \end{pmatrix} i = 1,2,3$$

$$V_i = \begin{pmatrix} (\mu + ط + \delta)E \\ (ط + e + \mu)I - \delta E \\ (c + \mu)R - طI - طE \end{pmatrix} i = 1,2,3$$

$$F = \begin{pmatrix} \frac{\partial f_1}{\delta E}\Big|_{P^o} & \frac{\partial f_1}{\delta I}\Big|_{P^o} & \frac{\partial f_1}{\delta R}\Big|_{P^o} \\ \frac{\partial f_2}{\delta E}\Big|_{P^o} & \frac{\partial f_2}{\delta I}\Big|_{P^o} & \frac{\partial f_2}{\delta R}\Big|_{P^o} \\ \frac{\partial f_3}{\delta E}\Big|_{P^o} & \frac{\partial f_3}{\delta I}\Big|_{P^o} & \frac{\partial f_3}{\delta R}\Big|_{P^o} \end{pmatrix} = \begin{pmatrix} 0 & \frac{a\Lambda}{\mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

$$f_1 = asI, f_2 = f_3 = 0$$

Obtaining the derivative of V with respect to E, I and R respectively, we have

$$V = \begin{pmatrix} (\mu + ط + \delta) & 0 & 0 \\ -\delta & (ط + e + \mu) & 0 \\ -ط & -ط & (c + \mu) \end{pmatrix},$$

$$|V| = (\mu + ط + \delta)(ط + e + \mu)(c + \mu)$$

Let V_{cf} be the cofactor,

That is,

$$V_{cf} = \begin{pmatrix} g_{11} & g_{12} & g_{13} \\ g_{21} & g_{22} & g_{23} \\ g_{31} & g_{32} & g_{33} \end{pmatrix}$$

so,

$$\mathrm{V_{cf}} g_{11} = \begin{vmatrix} (ط + e + \mu) & 0 \\ -ط & (c + \mu) \end{vmatrix} = (ط + e + \mu)(c + \mu) \tag{14}$$

$$\mathrm{V_{cf}} g_{12} = -\begin{vmatrix} -\delta & 0 \\ -ط & (c + \mu) \end{vmatrix} = \delta(c + \mu) \tag{15}$$

$$\mathrm{V_{cf}} g_{13} = \begin{vmatrix} -\delta & (ط + e + \mu) \\ -ط & -ط \end{vmatrix} = ط(\delta + (ط + e + \mu)) \tag{16}$$

$$\mathrm{V_{cf}} g_{21} = -\begin{vmatrix} 0 & 0 \\ -ط & (c + \mu) \end{vmatrix} = 0 \tag{17}$$

$$\mathrm{V_{cf}}g_{22} = \begin{vmatrix} (\mu + ط + \delta) & 0 \\ -ط & (c + \mu) \end{vmatrix} = (\mu + ط + \delta)(c + \mu) \quad (18)$$

$$\mathrm{V_{cf}}g_{23} = -\begin{vmatrix} (\mu + ط + \delta) & 0 \\ -ط & -ط \end{vmatrix} = ط(\mu + ط + \delta \quad (19)$$

$$\mathrm{V_{cf}}g_{31} = \begin{vmatrix} 0 & 0 \\ (ط + e + \mu) & 0 \end{vmatrix} = 0 \quad (20)$$

$$\mathrm{V_{cf}}g_{32} = -\begin{vmatrix} (\mu + ط + \delta) & 0 \\ -\delta & 0 \end{vmatrix} = 0 \quad (21)$$

$$\mathrm{V_{cf}}g_{33} = \begin{vmatrix} (\mu + ط + \delta) & 0 \\ -\delta & (ط + e + \mu) \end{vmatrix} = (\mu + ط + \delta)(ط + e + \mu) \quad (22)$$

therefore,

$$V_{cf} = \begin{pmatrix} m_1 & m_2 & m_3 \\ m_4 & m_5 & m_6 \\ m_7 & m_8 & m_9 \end{pmatrix}$$

$$AdjV_{cf} = \begin{pmatrix} m_1 & m_4 & m_7 \\ m_2 & m_5 & m_8 \\ m_3 & m_6 & m_9 \end{pmatrix}$$

$$\mathrm{V}^{-1} = \frac{Adj(V_{cf})}{|V|}$$

$$= \begin{pmatrix} \frac{1}{\mu+ъ+\delta} & 0 & 0 \\ \frac{\delta}{(\mu+ъ+\delta)(ъ+e+\mu)} & \frac{1}{(ъ+e+\mu)} & 0 \\ \frac{ъ(\delta+(ъ+e+\mu))}{(\mu+ъ+\delta)(ъ+e+\mu)(c+\mu)} & \frac{ъ}{(ъ+e+\mu)(c+\mu)} & \frac{1}{c+\mu} \end{pmatrix}$$

$$G = FV^{-1}$$

$$= \begin{pmatrix} 0 & \frac{a\Lambda}{\mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{\mu+ъ+\delta} & 0 & 0 \\ \frac{\delta(c+\mu)}{(\mu+ъ+\delta)(b+e+\mu)} & \frac{1}{(ъ+e+\mu)} & 0 \\ \frac{ъ+(\delta+(ъ+e+\mu))}{(\mu+ъ+\delta)(ъ+e+\mu)} & \frac{ъ}{(ъ+e+\mu)(c+\mu)} & \frac{1}{(c+\mu)} \end{pmatrix}$$

$$G = \begin{pmatrix} \frac{a\Lambda\delta(c+\mu)}{\mu(\mu+ъ+\delta)(b+e+\mu)} & \frac{a\Lambda}{\mu(ъ+e+\mu)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

The dominant eigenvalue of G is the basic reproduction number denoted by R_0. i.e $|G - \lambda I| = 0$ where I is the identity matrix.

$$\begin{vmatrix} (\frac{a\Lambda\delta}{\mu(\mu+ъ+\delta)(ъ+e+\mu)} - \lambda) & \frac{a\Lambda}{\mu(ъ+e+\mu)} & 0 \\ 0 & -\lambda & 0 \\ 0 & 0 & -\lambda \end{vmatrix} = 0$$

$$\left(\frac{a\Lambda\delta}{\mu(\mu+ъ+\delta)(ъ+e+\mu)} - \lambda\right)(-\lambda)(-\lambda) = 0$$

therefore,

$$\lambda_1 = \frac{a\Lambda\delta}{\mu(\mu+ط+\delta)(ط+e+\mu)} \quad `\lambda_2 = \lambda_3 = 0$$

The dominant eigenvalue is

$$\lambda_1 = R_0 = \qquad \frac{a\Lambda\delta}{\mu(\mu+ط+\delta)(ط+e+\mu)}$$

Therefore, the basic reproduction number of the model is given as

$$R_0^* = \frac{a\Lambda\delta}{\mu(\mu+ط+\delta)(ط+e+\mu)} \qquad (23).$$

3.6 THE STABILITY ANALYSIS OF THE MODEL

The stability of the model is investigated in two ways: Locally and globally.

3.6.1 Local stability of disease free equilibrium:

Theorem:The stability of the disease-free equilibrium of system (3) is asymptotically local if $R_0^* < 1$ otherwise it is not stable.

Proof: The matrix of the Jacobian of system (3) at P_*^0

$$J = \begin{pmatrix} -aI-\mu & 0 & -as & c \\ aI & -(\mu+ط+\delta) & as & 0 \\ 0 & \delta & -(ط+e+\mu) & 0 \\ 0 & ط & ط & -(c+\mu) \end{pmatrix}$$

at $P_*^0 = (S^0, E^0, I^0, R^0) = (\frac{\Lambda}{\mu}, 0,0,0)$

therefore,

$$J(P_*^0) = \begin{pmatrix} -\mu & 0 & \frac{-a\Lambda}{\mu} & c \\ 0 & -(\mu + ط + \delta) & \frac{a\Lambda}{\mu} & 0 \\ 0 & \delta & -(ط + e + \mu) & 0 \\ 0 & ط & ط & -(c + \mu) \end{pmatrix} \quad (24)$$

The characteristic equation of equation (24) is

$$|J(P_*^0) - \lambda I| = 0$$

$$\begin{vmatrix} -(\mu + \lambda) & 0 & \frac{-a\Lambda}{\mu} & c \\ 0 & (-(\mu + ط + \delta) - \lambda) & \frac{a\Lambda}{\mu} & 0 \\ 0 & \delta & (-(ط + e + \mu) - \lambda) & 0 \\ 0 & ط & ط & (-(c + \mu) - \lambda) \end{vmatrix} = 0$$

$$(-\mu - \lambda)(-(c + \mu) - \lambda)\left((-(\mu + ط + \delta) - \lambda)(-(ط + e + \mu) - \lambda) - \frac{a\Lambda\delta}{\mu}\right) = 0$$

$$\lambda_1 = -\mu, \qquad \lambda_2 = -(c + \mu),$$

$$\left(\lambda^2 + (ط + e + \mu + \mu + ط + \delta)\lambda + (\mu + ط + \delta)(ط + e + \mu) - \frac{a\Lambda\delta}{\mu}\right) = 0$$

$$\left(\lambda^2 + (2ط + e + 2\mu + \delta)\lambda + (\mu + ط + \delta)(ط + e + \mu)(1 - \frac{a\Lambda\delta}{\mu})\right) = 0$$

$$\lambda^2 + (2ط + e + 2\mu + \delta)\lambda + (\mu + ط + \delta)(ط + e + \mu)(1 - R_0^*) = 0 \quad (25)$$

If $R_0 < 1$, then by Descarte's rule of signs, there is no sign change, hence, there are no positive roots of equation (25).

Furthermore, if λ is replaced by $-\lambda$ in equation (25)

$$\lambda^2 - (2ط + e + 2\mu + \delta)\lambda + (\mu + ط + \delta)(ط + e + \mu)(1 - R_0) = 0 \quad (26)$$

If $R_0^* < 1$, then equation (26) has two signs change, hence there are exactly two negative roots of equation (26) Therefore, P_*^0 is asymptotically stable locally if $R_0^* < 1$. The result follows immediately that P^0 is unstable if $R_0^* > 1$.

3.6.2 Global stability of the disease-free equilibrium

We make use of Lyapunov function which says

$$L_*(E, I) = (\mu + ط + \delta)I + \delta E \quad (27)$$

Obtain the derivative of equation (27) along the solutions of equations (6) and (7)

$$L_*' = (\mu + ط + \delta)I' + \delta E'$$

$$= (\mu + ط + \delta)\big(\delta E - (ط + e + \mu)I\big) + \delta\big(aSI - (\mu + ط + \delta)E\big)$$

$$= (\mu + ط + \delta)\delta E - (\mu + ط + \delta)(ط + e + \mu)I + \delta aSI - (\mu + ط + \delta)\delta E$$

$$= \delta aSI - (\mu + ط + \delta)(ط + e + \mu)I$$

$$= \big(\delta aS - (\mu + ط + \delta)(ط + e + \mu)\big)I$$

$$= (\mu + ط + \delta)(ط + e + \mu)\left(\frac{\delta aS}{(\mu+ط+\delta)(ط+e+\mu)} - 1\right)I$$

At the disease free,

$$S = S^0 = \frac{\Lambda}{\mu}$$

$$= (\mu + ط + \delta)(ط + e + \mu)\left(\frac{\delta a\Lambda}{\mu(\mu + ط + \delta)(ط + e + \mu)} - 1\right)I$$

therefore, $L_*' = (\mu + ط + \delta)(ط + e + \mu)(R_0^* - 1)I$, $L_*' < 0$ whenever $R_0^* < 1$ and $I > 0$ furthermore, $L_*' = 0$ whenever $R_0^* = 1$ and or $I \geq 0$, $L_*' \leq 0$ if $R_0^* \leq 1$ and $I \geq 0$.

3.7 THE EXISTENCE OF BIFURCATION IN THE MODEL

3.7.1 The local stability of the endemic equilibrium

We made use of bifurcation and the center manifold theorem in Buonomo and Lacitignola (2011) to show whether or not the stability of the endemic equilibrium of system 3 exists.

Let a = a* be the bifurcation parameter

Let us consider a system of ordinary differential equation with parameter a^*

$$\frac{df^*}{dx} = f^*(x, a^*), f^*: R^n \times R \to R^n, f^* \in C^2(R^n \times R). \qquad (P)$$

Without any loss of generality, we assume that $x_*' = 0$ is an equilibrium point for P

So from Buonomo and Lacitignola (2011),

Theorem A: Assume:

(1) $A_1^* = D_x f^*(0,0)$is the linearization matrix of system (P) around the equilibrium $x_*' = 0$ with a^* evaluated at 0. Zero is a simple eigenvalue of A_1^* and all other eigenvalues of A_1^* are of negative parts that are real;

(2) Matrix A_1^* with a (non negative) right eigenvector ω and a left eigenvector v which is equivalent to eigenvalue of value zero. Let f_k denotes the kth component of f, and

$$A_1^* = \sum_{k,i,j=1}^{4} V_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$$
$$B_1^* = \sum_{k,i,=1}^{4} V_k w_i \frac{\partial^2 f_k}{\partial x_i \partial x_a}(0,0)$$

Where $\omega_{i,,}$ ω_j are the right eigenvectors and $V_k$the left eigenvector. Then the local dynamics of system (P) around $x_*' = 0$ can be found totally by A^*_1 and B^*_1.

(i) If $A_1^* > 0, B_1^* > 0.$, when $a^* < 0$, with $|a^*| < 1$, then the point at which $x_*' = 0$ is asymptotically stable locally and it shows a positive equilibrium that is not stable; when $0 < a^* < 1, x_*' = 0$ is unstable it shows a local equilibrium that is asymptotically stable which is negative.

(ii) If $A_1^* < 0, B_1^* < 0$, when $a^* < 0$, with $|a^*| < 1$ then the point at which $x_*' = 0$ is not stable ; when $0 < a^* < 1, x_*' = 0$ is asymptotically stable locally and it shows a positive equilibrium that is not stable.

(iii) If $A_1^* > 0, B_1^* < 0$, when $a^* < 0$, with $|a^*| < 1$, then the point at which $x_*' = 0$ is not stable and it shows an asymptotically locally stable negative equilibrium when $0 < a^* < 1$, then the point at which $x_*' = 0$ is stable and there appears a positive equilibrium.

(iv) If $A_1^* < 0, B_1^* > 0$, when a^* moves from negative point to positive point, then the point at which $x_*{}' = 0$ loses its stability from stable point to unstable. Consequently, a negative equilibrium that is unstable becomes a positive one and asymptotically stable equilibrium locally.

Proof

Let a^* be the bifurcation parameter.

The bifurcation nature around the disease free equilibrium is now investigated as follows, if $a < a^*$ then stability is achievedand the stability is lost if $a > a^*$.

Recall that,

$$R_0^* = \frac{a\Lambda\delta}{\mu(\mu+ط+\delta)(ط+e+\mu)}$$

By considering the case

$$R_0^* = 1$$

$$\frac{a\Lambda\delta}{\mu(\mu+ط+\delta)(ط+e+\mu)} = 1$$

therefore,

$$a = a^* = \frac{\mu(\mu+ط+\delta)(ط+e+\mu)}{\Lambda\delta}$$

It is now shown that the system of equations in (5) to (8) has a simple zero eigenvalue. By taking the Jacobian of equations (5) to (8) at the disease free equilibrium, we have that

$$J = \begin{pmatrix} -aI-\mu & 0 & -aS & c \\ aI & -(\mu+ط+\delta) & aS & 0 \\ 0 & \delta & -(ط+e+\mu) & 0 \\ 0 & ط & ط & -(c+\mu) \end{pmatrix}$$

at the disease free P_*^0

$$J(P_*^0) = \begin{pmatrix} -\mu & 0 & \frac{-a^*\Lambda}{\mu} & c \\ 0 & -(\mu + ط + \delta) & \frac{a^*\Lambda}{\mu} & 0 \\ 0 & \delta & -(ط + e + \mu) & 0 \\ 0 & ط & ط & -(c + \mu) \end{pmatrix} \quad (28)$$

The characteristic polynomial of equation (28)

$$|J(p_*^0) - \lambda I| = 0$$

$$\begin{vmatrix} -\mu - \lambda & 0 & \frac{-a^*\Lambda}{\mu} & c \\ 0 & (-(\mu + ط + \delta) - \lambda) & \frac{a^*\Lambda}{\mu} & 0 \\ 0 & \delta & (-(ط + e + \mu) - \lambda) & 0 \\ 0 & ط & ط & (-(c + \mu) - \lambda) \end{vmatrix} = 0$$

Evaluating using the first column gives

$$(-\mu - \lambda)(-(c + \mu) - \lambda)\left[(1 - (\mu + ط + \delta) - \lambda)(-(ط + e + \mu) - \lambda) - \frac{a^*\Lambda\delta}{\mu}\right] = 0$$

$$\lambda_1 = -\mu, \qquad \lambda_2 = -(c + \mu)$$

$$\left[((\mu + ط + \delta) + \lambda)((ط + e + \mu) + \lambda) - \frac{a^*\Lambda\delta}{\mu}\right] = 0$$

$$\lambda^2 + [\mu + ط + \delta + ط + e + \mu]\lambda + (\mu + ط + \delta)(ط + e + \mu)$$

$$- \frac{\mu(\mu + ط + \delta)(ط + e + \mu)\Lambda\delta}{\Lambda\delta\mu} = 0$$

$$\lambda^2 + (2\mu + 2ط + \delta + e)\lambda = 0$$

$$\lambda(\lambda + (2\mu + 2ط + \delta + e)) = 0$$

$$\lambda_3 = 0, \quad `\lambda_4 = -(2\mu + 2ط + \delta + e)$$

so the eigenvalues are

$$\lambda_1 = -\mu. \quad \lambda_2 = -(c + \mu), \quad \lambda_3 = 0 \quad \lambda_4 = -(2\mu + 2ط + \delta + e)$$

Hence we have $\lambda_3 = 0$ to be simple zero eigenvalue while other eigenvalues are negative. Hence, when $a = a^*$ (when R_0^* = 1), the disease free equilibrium in the assumption of theorem A is then verified.

We now obtain the right eigenvector denoted by ω

associated with the $\lambda_3 = 0$. Thus,

$$\omega = (\omega_1, \omega_2, \omega_3, \omega_4)^T$$

$$\begin{pmatrix} -\mu & 0 & \frac{-a^*\Lambda}{\mu} & c \\ 0 & -(\mu + ط + \delta) & \frac{a^*\Lambda}{\mu} & 0 \\ 0 & \delta & -(ط + e + \mu) & 0 \\ 0 & ط & ط & -(c + \mu) \end{pmatrix} \begin{pmatrix} \omega_1 \\ \omega_2 \\ \omega_3 \\ \omega_4 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

we have,

$$-\mu\omega_1 - \frac{a^*\Lambda}{\mu}\omega_3 + c\omega_4 = 0$$

$$-(\mu + ط + \delta)\omega_2 + \frac{a^*\Lambda}{\mu}\omega_3 = 0$$

$$\delta\omega_2 - (ط + e + \mu)\omega_3 = 0$$

$$ط\omega_2 + ط\omega_3 - (c + \mu)\omega_4 = 0$$

therefore,

$$\omega_3 = \frac{\mu(\mu+ط+\delta)\omega_2}{a^*\Lambda}$$

then from

$$ط\omega_2 + ط\omega_3 - (c + \mu)\omega_4 = 0,$$

$$ط\omega_2 + \frac{ط\mu(\mu+ط+\delta)\omega_2}{a^*\Lambda} - (c + \mu)\omega_4 = 0$$

then,

$$\omega_4 = \frac{ط(a^*\Lambda+\mu(\mu+ط+\delta))}{a^*\Lambda(c+\mu)}\omega_2$$

and from

$$-\mu\omega_1 - \frac{a^*\Lambda}{\mu}\omega_3 + c\omega_4 = 0,$$

then,

$$\omega_1 = \frac{c}{\mu}\omega_4 - \frac{(\mu+ط+\delta)}{\mu}\omega_2$$

substituting

$$\omega_2 \; and \; \omega_4$$

we have,

$$\omega_1 = \frac{ъc}{\mu}\left(\frac{a^*\Lambda+\mu(\mu+ъ+\delta)}{a^*\Lambda(c+\mu)} - \frac{(\mu+ъ+\delta)}{\mu}\right)\omega_2$$

so that,

$$\omega = (\omega_1, \omega_2, \omega_3, \omega_4)^T$$

will now be,

$$\omega = \left(\frac{ъc}{\mu}\left(\frac{a^*\Lambda+\mu(\mu+ъ+\delta)}{a^*\Lambda(c+\mu)}\right) - \frac{(\mu+ъ+\delta)}{\mu}\right)\omega_2,\ \omega_2,\ \frac{\mu(\mu+ъ+\delta)}{a^*\Lambda}\omega_2,$$

$$\left(\frac{ъ(a^*\Lambda+\mu(\mu+ъ+\delta))}{a^*\Lambda(c+\mu)}\omega_2\right)^T$$

where $\omega_2 > 0$ is a free right eigenvector.

Similarly,left eigenvector denoted by V defined thus:

$$V = (V_1, V_2, V_3, V_4)$$

$$(V_1, V_2, V_3, V_4)\begin{pmatrix} -\mu & 0 & \frac{-a^*\Lambda}{\mu} & c \\ 0 & -(\mu+ъ+\delta) & \frac{a^*\Lambda}{\mu} & 0 \\ 0 & \delta & -(ъ+e+\mu) & 0 \\ 0 & ъ & ъ & -(c+\mu) \end{pmatrix} = (0,0,0,0)$$

solving this, we have, $V_1 = 0$, and $V_4 = 0$ then,

$$-(\mu+ъ+\delta)V_2 + \delta V_3 + bV_4 = 0$$

therefore,

$$V_3 = \frac{(\mu+ъ+\delta)}{\delta}V_2$$

let V_2 be a positive free variable, then,

$$V = (V_1, V_2, V_3, V_4)$$

will now be,

$$V = \left(0, V_2, \frac{(\mu+ط+\delta)}{\delta} V_2, 0\right)$$

where, $V_2 > 0$ is a free left eigenvector.

To solve for the coefficients A_1^* and B_1^* in the theorem A,

$$A_1^* = \sum_{k,i,j=1}^{4} V_k w_i w_j \frac{\partial^2 f_x}{\partial x_i \partial x_j}(E_0, a^*)$$
$$B_1^* = \sum_{k,i,=1}^{4} V_k w_i w_j \frac{\partial^2 f_x}{\partial x_i \partial x_p}(E_0, a^*)$$

We now consider the non-zero components of V in the system 3

$$\frac{dS_*(t)}{dt} = \Lambda - aS(t)I(t) - \mu S(t) + cR(t) = f_1 \quad (29)$$

$$\frac{dE_*(t)}{dt} = aS(t)I(t) - \mu E(t) - ط E(t) - \delta E(t) = f_2 \quad (30)$$

$$\frac{dI_*(t)}{dt} = \delta E(t) - ط I(t) - eI(t) - \mu I(t) = f_3 \quad (31)$$

$$\frac{dR_*(t)}{dt} = ط I(t) + ط E(t) - cR(t) - \mu R(t) = f_4 \quad (32)$$

Let S = x_1, E = x_2, I = x_3, R = x_4

$$X = (x_1, x_2, x_3, x_4)^T$$

$$\frac{dX}{dt} = F(x), F = (f_1, f_2, f_3, f_4)^T$$

so,

$$\frac{dx_1}{dt} = f_1 = \Lambda - ax_1x_3 - \mu x_1 + cx_4$$

$$\frac{dx_2}{dt} = f_2 = ax_1x_3 - (\mu + \text{ط} + \delta)x_2$$

$$\frac{dx_3}{dt} = f_3 = \delta x_2 - (\text{ط} + e + \mu)x_3$$

$$\frac{dx_4}{dt} = f_4 = \text{ط}x_3 + \text{ط}x_2 - (c + \mu)x_4$$

when k = 2, for i = 1,2,3,4 and j = 1,2,3,4 and when k = 3, for i = 1,2,3,4 and j = 1,2,3,4 the sum

$$A = \sum_{k,i,j=1}^{4} V_k w_i w_j \frac{\partial^2 f_x}{\partial x_i \partial x_j}(E_0, a^*)$$

may be computed explicitly as

$$A_1^* = V_2\omega_1^2\frac{\partial^2 f_2}{\partial x_1^2} + V_2\omega_1\omega_2\frac{\partial^2 f_2}{\partial x_1 x_2} + V_2\omega_1\omega_3\frac{\partial^2 f_2}{\partial x_1 x_3} + V_2\omega_1\omega_4\frac{\partial^2 f_2}{\partial x_1 x_4} + V_2\omega_2\omega_1\frac{\partial^2 f_2}{\partial x_2 x_1}$$

$$+ V_2\omega_2^2\frac{\partial^2 f_2}{\partial x_2^2} + V_2\omega_2\omega_3\frac{\partial^2 f_2}{\partial x_2 x_3} + V_2\omega_2\omega_4\frac{\partial^2 f_2}{\partial x_2 x_4} + V_2\omega_3\omega_1\frac{\partial^2 f_2}{\partial x_3 x_1}$$

$$+ V_2\omega_3\omega_2\frac{\partial^2 f_2}{\partial x_3 x_2} + V_2\omega_3^2\frac{\partial^2 f_2}{\partial x_3^2} + V_2\omega_3\omega_4\frac{\partial^2 f_2}{\partial x_3 x_4}$$

$$+V_2\omega_4\omega_1\frac{\partial^2 f_2}{\partial x_4 x_1} + V_2\omega_4\omega_2\frac{\partial^2 f_2}{\partial x_4 x_2} + V_2\omega_4\omega_3\frac{\partial^2 f_2}{\partial x_4 x_3} + V_2\omega_4^2\frac{\partial^2 f_2}{\partial x_4^2} +$$

$$V_3\omega_1^2\frac{\partial^2 f_3}{\partial x_1^2} + V_3\omega_1\omega_2\frac{\partial\ f_3}{\partial x_1 x_2} + V_3\omega_1\omega_3\frac{\partial^2 f_3}{\partial x_1 x_3} + V_3\omega_1\omega_4\frac{\partial^2 f_3}{\partial x_1 x_4} + V_3\omega_2\omega_1\frac{\partial^2 f_3}{\partial x_2 x_1}$$

$$+ V_3\omega_2^2\frac{\partial^2 f_3}{\partial x_2^2}$$

$$+V_3\omega_2\omega_3\frac{\partial^2 f_3}{\partial x_2 x_3}+V_3\omega_2\omega_4\frac{\partial^2 f_3}{\partial x_2 x_4}+V_3\omega_3\omega_1\frac{\partial^2 f_3}{\partial x_3 x_1}+V_3\omega_3\omega_2\frac{\partial^2 f_3}{\partial x_3 x_2}+V_3\omega_3^2\frac{\partial^2 f_3}{\partial x_3^2}$$

$$+V_3\omega_3\omega_4\frac{\partial^2 f_3}{\partial x_3 x_4}$$

$$+V_3\omega_4\omega_1\frac{\partial^2 f_3}{\partial x_4 x_1}+V_3\omega_4\omega_2\frac{\partial^2 f_3}{\partial x_4 x_2}+V_3\omega_4\omega_3\frac{\partial^2 f_3}{\partial x_4 x_3}+V_3\omega_4^2\frac{\partial^2 f_3}{\partial x_4^2}$$

therefore,

$$A_1^* = V_2\omega_1^2\frac{\partial^2 f_2}{\partial x_1^2}+2V_2\omega_1\omega_2\frac{\partial^2 f_2}{\partial x_1\,\partial x_2}+2V_2\omega_1\omega_3\frac{\partial^2 f_2}{\partial x_1\,\partial x_3}+2V_2\omega_1\omega_4\frac{\partial^2 f_2}{\partial x_1\,\partial x_4}$$

$$+V_2\omega_2^2\frac{\partial^2 f_2}{\partial x_2^2}+2V_2\omega_2\omega_3\frac{\partial^2 f_2}{\partial x_2\,\partial x_3}+2V_2\omega_2\omega_4\frac{\partial^2 f_2}{\partial x_2\,\partial x_4}+V_2\omega_3^2\frac{\partial^2 f_2}{\partial x_3^2}$$

$$+2V_2\omega_3\omega_4\frac{\partial^2 f_2}{\partial x_3\,\partial x_4}+V_2\omega_4^2\frac{\partial^2 f_2}{\partial x_4^2}+V_3\omega_1^2\frac{\partial^2 f_3}{\partial x_1^2}+2V_3\omega_1\omega_2\frac{\partial^2 f_3}{\partial x_1\,\partial x_2}$$

$$+2V_3\omega_1\omega_3\frac{\partial^2 f_3}{\partial x_1\,\partial x_3}+2V_3\omega_1\omega_4\frac{\partial^2 f_3}{\partial x_1\,\partial x_4}+V_3\omega_2^2\frac{\partial^2 f_3}{\partial x_2^2}$$

$$+2V_3\omega_2\omega_3\frac{\partial^2 f_3}{\partial x_2\,\partial x_3}+2V_3\omega_2\omega_4\frac{\partial^2 f_3}{\partial x_2\,\partial x_4}+V_3\omega_3^2\frac{\partial^2 f_3}{\partial x_3^2}$$

$$+2V_3\omega_3\omega_4\frac{\partial^2 f_3}{\partial x_3\,\partial x_4}+V_3\omega_4^2\frac{\partial^2 f_3}{\partial x_4^2}$$

But,

$$\frac{\partial^2 f_2}{\partial x_1^2}=\frac{\partial^2 f_2}{\partial x_1\,\partial x_2}=\frac{\partial^2 f_2}{\partial x_1\,\partial x_4}=\frac{\partial^2 f_2}{\partial x_2^2}=\frac{\partial^2 f_2}{\partial x_2\,\partial x_3}=\frac{\partial^2 f_2}{\partial x_2\,\partial x_4}=\frac{\partial^2 f_2}{\partial x_3^2}=\frac{\partial^2 f_2}{\partial x_3\,\partial x_4}=\frac{\partial^2 f_2}{\partial x_4^2}$$

$$= \frac{\partial^2 f_3}{\partial x_1^2} = \frac{\partial^2 f_3}{\partial x_1 \partial x_2} = \frac{\partial^2 f_3}{\partial x_1 \partial x_3} = \frac{\partial^2 f_3}{\partial x_1 \partial x_4} = \frac{\partial^2 f_3}{\partial x_2^2} = \frac{\partial^2 f_3}{\partial x_2 \partial x_3} = \frac{\partial^2 f_3}{\partial x_2 \partial x_4} = \frac{\partial^2 f_3}{\partial x_3^2}$$

$$= \frac{\partial^2 f_3}{\partial x_3 \partial x_4} = \frac{\partial^2 f_3}{\partial x_4^2} = 0$$

and

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_3} = a$$

so,

$$A_1^* = 2V_2\omega_1\omega_3 \frac{\partial^2 f_2}{\partial x_1 \partial x_3}$$

$$= 2V_2\omega_1\omega_3 a$$

$$= \frac{2}{\mu}\left(\frac{\mu(\mu+ط+\delta)}{a^*\Lambda}\right)\omega_2\left(\frac{ط c(a^*\Lambda+\mu(\mu+ط+\delta))}{a^*\Lambda+(c+\mu)} - (\mu + ط + \delta)\right)\omega_2 V_2 a$$

$$= \frac{2(\mu+ط+\delta)^2}{a^*\Lambda}\omega_2^2 V_2 a\left(\frac{ط c(a^*\Lambda+\mu(\mu+ط+\delta))}{(a^*\Lambda+(c+\mu))(\mu+ط+\delta)} - 1\right)$$

Let,

$$A^* = \frac{ط c(a^*\Lambda+\mu(\mu+ط+\delta))}{(a^*\Lambda+(c+\mu))(\mu+ط+\delta)}$$

therefore,

$$A_1^* = \frac{2(\mu+ط+\delta)^2}{a^*\Lambda}\omega_2^2 V_2 a(A^* - 1)$$

The sign of coefficient A_1^* is completely determined by A^*, thus If

(i) $A^* > 1$, then A_1^* is positive.

(ii) $A^* < 1$, then A_1^* is negative.

Similarly, we compute the sum for B in the theorem explicitly. Recall that,

$$B_1^* = \sum_{k,i,=1}^{4} V_k w_i \frac{\partial^2 f_k}{\partial x_i \, \partial a}(E_0, a^*)$$

for k=2, when i = 1,2,3,4 and j =1,2,3,4 and for k = 3, when i = 1,2,3,4

Remember $(x_1, x_2, x_3, x_4) = (\frac{\Lambda}{\mu}, 0,0,0)$

$$B = V_2\omega_1 \frac{\partial^2 f_2}{\partial x_1 \, \partial_a} + V_2\omega_2 \frac{\partial^2 f_2}{\partial x_2 \, \partial_a} + V_2\omega_3 \frac{\partial^2 f_2}{\partial x_3 \, \partial_a} + V_2\omega_4 \frac{\partial^2 f_2}{\partial x_4 \, \partial_a}$$

$$+V_3\omega_1 \frac{\partial^2 f_3}{\partial x_1 \, \partial_a} + V_3\omega_2 \frac{\partial^2 f_3}{\partial x_2 \, \partial_a} + V_3\omega_3 \frac{\partial^2 f_3}{\partial x_3 \, \partial_a} + V_3\omega_4 \frac{\partial^2 f_3}{\partial x_4 \, \partial_a}$$

But,

$$\frac{\partial^2 f_2}{\partial x_1 \, \partial_a} = \frac{\partial^2 f_2}{\partial x_2 \, \partial_a} = \frac{\partial^2 f_2}{\partial x_4 \, \partial_a} = \frac{\partial^2 f_3}{\partial x_1 \, \partial_a} = \frac{\partial^2 f_3}{\partial x_2 \, \partial_a} = \frac{\partial^2 f_3}{\partial x_3 \, \partial_a} = \frac{\partial^2 f_3}{\partial x_4 \, \partial_a} = 0$$

and

$$\frac{\partial^2 f_2}{\partial x_3 \, \partial_a} = \frac{\Lambda}{\mu}$$

therefore,

$$B_1^* = V_2\omega_3 \frac{\partial^2 f_2}{\partial x_3 \, \partial_a}$$

$$= V_2\omega_3 \frac{\Lambda}{\mu}$$

$$= \frac{\mu(\mu+\text{ь}+\delta)}{a^*\Lambda} \omega_2 V_2 \frac{\Lambda}{\mu}$$

This implies,

$$B_1^* = \frac{(\mu+\text{ь}+\delta)\omega_2 V_2}{a^*} > 0$$

Which is always positive.

Then from the theorem A,

(i) If $A_1^* > 0$ and $B_1^* > 0$, there exists a backward bifurcation in the system.

(ii) If $A_1^* < 0$ and $B_1^* > 0$, there exits a forward bifurcation in the system.

3.8 SENSITIVITY ANALYSIS OF THE MODEL

"Sensitivity analysis is often used to study how the variation in the output of a model can be apportioned, qualitatively or quantitatively, to different services of variation, and of how the given model depends on the information feeds into it"(Saltelli et al, 2008). In this section, we shall take a critical look at the parameters to know which one will make our model to be in an endemic state. The parameters that keep the model in a stable disease free will be left alone but the ones that make the reproduction number greater than one will be the one the policy makers have to be concerned with and to find the necessary control measures that will minimize the spread of the disease. The parameters with negative value will reduce the reproduction and make it to be less than 1, but, the ones with positive values will increase the reproduction number. So we look for the most positive parameters which will be the most sensitive ones that the policy makers need to check and control in order to bring the reproduction number to be less than 1.

To check the sensitivity of the parameters as given by Arriola and Hyman (2005), we make use of the reproduction number

$$R_0^* = \frac{\alpha\Lambda\delta}{\mu(\mu+\text{ somethingb}+\delta)(\text{somethingb}+e+\mu)}$$

and each parameter will be tested. Let p represent each parameter,

$$R_p = \frac{p}{R_0^*}.\frac{\partial R_0^*}{\partial p}$$

So for parameter Λ,

$$R_\Lambda = \frac{\Lambda}{R_0^*}.\frac{\partial R_0^*}{\partial \Lambda} = \frac{\Lambda}{R_0^*}.\frac{R_0^*}{\Lambda} = 1.$$

For parameter a,

$$R_a = \frac{a}{R_0^*}.\frac{\partial R_0^*}{\partial a} = \frac{a}{R_0}.\frac{\Lambda\delta}{\mu(\mu+ь+\delta)(ь+e+\mu)} = \frac{a}{R_0^*}.\frac{1}{a}R_0^* = 1.$$

For parameter $ь$,

$$R_b = \frac{ь}{R_0^*}.\frac{\partial R_0^*}{\partial b} = -\frac{ьb\mu(\mu+ь+\delta)(ь+e+\mu)a\Lambda\delta}{a\Lambda\delta\mu(\mu+ь+\delta)(ь+e+\mu)}\left(\frac{1}{(\mu+ь+\delta)} + \frac{1}{(ь+e+\mu)}\right)$$

$$= -\left(\frac{b}{(\mu+ь+\delta)} + \frac{b}{(ь+e+\mu)}\right) < 1.$$

For parameter c,

$$R_c = \frac{c}{R_0^*}.\frac{\partial R_0^*}{\partial c} = 0.$$

For parameter δ,

$$R_\delta = \frac{\delta}{R_0^*}.\frac{\partial R_0^*}{\partial \delta} = \frac{\mu(\mu+ь+\delta)(ь+e+\mu)a\Lambda}{a\Lambda\mu(\mu+ь+\delta)(ь+e+\mu)}\left(1 - \frac{\delta}{(\mu+ь+\delta)}\right) = \frac{(\mu+ь+\delta-\delta)}{\mu+ь+\delta} = \frac{\mu+ь}{\mu+ь+\delta} < 1.$$

For parameter μ,

$$R_\mu = \frac{\mu}{R_0^*}.\frac{\partial R_0^*}{\partial \mu} = -\frac{\mu(\mu+ь+\delta)(ь+e+\mu)a\Lambda\delta}{a\Lambda\delta\mu(\mu+ь+\delta)(ь+e+\mu)}\left(\frac{1}{\mu} + \frac{1}{(\mu+ь+\delta)} + \frac{1}{(ь+e+\mu)}\right)$$

$$= -\left(1 + \frac{\mu}{\mu+ь+\delta)} + \frac{\mu}{ь+e+\mu}\right) < 1.$$

For parameter e,

$$R_e = \frac{e}{R_0^*}.\frac{\partial R_0^*}{\partial e} = -\frac{e}{(ь+e+\mu)} < 1.$$

From the sensitivity analysis, the most sensitive parameters are Λ and awhich are the rate at which the susceptible population is being populated and the transmission rate respectively.

3.9 OPTIMAL CONTROL OF THE MODEL

This section is to seek an optimal control strategy U throughout the length of $0 \leq t \leq t_f$where t_f is the final time for the infection to exit, such that the number of infected individual I and exposed individual E are minimized while minimising the cost of control U. Let U_1 be the cost of the treatment of infected and exposed individuals and U_2 be the cost of the vaccine for them. From the system 3, the model now becomes

$$\frac{dS_*(t)}{dt} = \Lambda - (1 - U_1 - U_2)aS(t)I(t) - \mu S(t) + cR(t)$$

$$\frac{dE_*(t)}{dt} = (1 - U_1 - U_2)aS(t)I(t) - U_1E(t) - U_2E(t) - \mu E(t) - \delta E(t)$$

$$\frac{dI_*(t)}{dt} = \delta E(t) - U_1I(t) - eI(t) - \mu I(t)$$

$$\frac{dR_*(t)}{dt} = U_1I(t) + U_1E(t) + U_2E(t) - cR(t) - \mu R(t)$$

The objective function now is

$$J(U_1, U_2) = \int_0^{t_f} (m_1I(t) + m_2E(t) - m_3U_1^2 - m_4U_2^2)dt \qquad (33)$$

Where m_i, i= 1...4 are the weights to balance the effects of the control measures. thus, the optimal control of the model is hereby sought as follows

$$U^* = (U_1^*, U_2^*)$$

such that $J(U_1^*, U_2^*) = \min_{u_1,u_2}\left[J(U_1, U_2)|_{u_1,u_2} \varepsilon\, U\right]$

$U = \left[(u_1, u_2|_{u_1,u_2}: [0, t_f] \rightarrow (0,1)\right]$ is Lebesque measurable.

3.9.1 Analysis of the Optimal control problem of infectious diseases.

Let H be the Hamiltonia function and using maximum principle given by Pontryagin as cited in Fleming and Rishell (1975) to derive necessary conditions for the optimal control problem,

$$H = m_1 I(t) + m_2 E(t) - m_3 U_1^2 - m_4 U_2^2 + \lambda_1[\Lambda - (1 - U_1 - U_2)aS(t)I(t) - \mu S + cR(t)] + \lambda_2[(1 - U_1 - U_2)aS(t)I(t) - (U_1 + U_2 + \mu + \delta)E(t)] + \lambda_3[\delta E(t) - (U_1 + e + \mu)I(t)] + \lambda_4[U_1 I(t) + U_1 E(t) + U_2 E(t) - (c + \mu)R(t)]$$

Whereλ_i , i= 1...4 are the adjoints or the co-state variables.

3.9.2 The Adjoint conditions

The adjoint conditions for the system 3 are given thus

$$\frac{d\lambda_1}{dt} = -\frac{dH}{dS_*} = (\lambda_1 - \lambda_2)(1 - U_1 - U_2)aI(t) + \mu\lambda_1 \quad (34)$$

$$\frac{d\lambda_2}{dt} = -\frac{dH}{dE_*} = \lambda_2(U_1 + U_2 + \mu + \delta) - \lambda_4(U_1 + U_2) - \lambda_3\delta - m_2 \quad (35)$$

$$\frac{d\lambda_3}{dt} = -\frac{dH}{dI_*} = (\lambda_3(U_1 + e + \mu) - \lambda_4 U_1 - m_1 \ + (\lambda_1 - \lambda_2)(1 - U_1 - U_2)\text{as}) \quad (36)$$

$$\frac{d\lambda_4}{dt} = -\frac{dH}{dR_*} = \lambda_4(c + \mu) - \lambda_1 c \quad (37)$$

with the boundary conditions at the final time

$$t_f: \lambda_1(t_f) = 0, \lambda_2(t_f) = 0, \lambda_3(t_f) = 0, \lambda_4(t_f) = 0 \quad (38).$$

3.9.3 The Optimality conditions

$$\frac{\partial H}{\partial U_1} = -2m_3U_1 + \lambda_1 aS(t)I(t) - \lambda_2 aS(t)I(t) - \lambda_2 E(t) - \lambda_3 I(t) + \lambda_4 I(t) + \lambda_4 \text{ E(t)} \quad (39).$$

At the absolute minimum, the slope of the function is zero. Then,

$$u_1^* = \frac{(\lambda_2 - \lambda_1)aS(t)I(t) + (\lambda_2 - \lambda_4)E(t) + (\lambda_3 - \lambda_4 I(t)}{-2m_3}$$

$$\frac{\partial H}{\partial U_2} = -2m_4U_2 + \lambda_1 aS(t)I(t) - \lambda_2 aS(t)I(t) - \lambda_2 E(t) + \lambda_4 E(t)$$

at $\frac{\partial H}{\partial U_2} = 0$,

$$U_2^* = \frac{(\lambda_2 - \lambda_1)aS(t)I(t) + (\lambda_2 - \lambda_4)E(t)}{-2m_4}$$

$$U_1^* = min\frac{[1, max(0, (\lambda_2 - \lambda_1)aS(t)I(t) + (\lambda_2 - \lambda_4)E(t) + (\lambda_3 - \lambda_4)I(t))]}{-2m_3} \quad (40)$$

$$U_2^* = min\frac{[1, max(0, (\lambda_2 - \lambda_1)aS(t)I(t) + (\lambda_2 - \lambda_4)E(t))]}{-2m_3} \quad (41)$$

So for the control system, the conditions have been set for U_1 and U_2.

3.10 THE STOCHASTICITY OF THE MODEL

Allen (2008) introduced the different methods of formulating stochastic epidemic models that relate to their deterministic models directly. Three main methods were introduced:

(1) Discrete Time Markov Chain (DTMC)

(2) Continuous Time Markov chain (CTMC)

(3) Stochastic Differential Equation (SDE).

For DTMC, its underlying assumption is that the time and the state variable are discrete. For CTMC, the time is continuous but the state variable is discrete while, SDE is based on diffusion process and both the time and the state variables are continuous.

When the epidemic breaks, it breaks in an infinitesimal time which is a continuous process and the process of epidemic is a dynamic process. Initially, the model is a random walk model which will be developed into stochastic differential equation model which is known as SDE model. Because it is a continuous process, it is Markovian that happens in an infinitesimal time. This aspect will be approached in the following manner:

(1) As a pure birth process.

(2) Formulation of the SEIR model.

(3) Extension of SEIR model to stochastic model.

(4) Perturbation process.

(5) Condition for uniqueness and persistence of the disease.

(6) Condition for extinction of the disease.

3.10.1 Pure birth process

To build a stochastic epidemic model, one must be conversant with some notations and versed with the ideas of stochastic compartmental models. At this time, we treat the compartment as one. After, the compartment models become disease states and their members. The movement of members within the compartments is defined on a vector-valued process. This process is a Markovian having a continuous time because the epidemic happens in an infinitesimal time.

For each time $t \geq 0, X_1^*(t), X_2^*(t), X_3^*(t), X_4^*(t)$,are the number of the individuals in the compartment respectively where the total number N(t) is the sum of the individuals in the compartment that is,

N(t) =$X_1^*(t)$+$X_2^*(t)$+$X_3^*(t)$+$X_4^*(t)$

Let us assume that there is just one compartment X_t^* which represents the number of individuals at time t and X_0^* is the initial value before the epidemic, for some $\lambda > 0$, where λ is the stochastic rate of the stochastic process and $o(\Delta_t)$ is a function that is negligible.

$$P_*(X^*(t+\Delta_t) - X(t) = 1) = \lambda\Delta_t + o(\Delta_t) \quad (42)$$

$$P_*(X^*(t+\Delta_t) - X(t) = 0) = 1 - \lambda\Delta_t + o(\Delta_t) \quad (43)$$

which can be described as a poisson process for $X^*(t), t \geq 0$.

The time interval between successful jumps or moves of the process can be described as exponentially distributed with the parameter λ so that, $\lambda = \lambda(t)$ depends on t

$$P_*(X^*(t+\Delta_t) - X(t) = 1) = aX^*(t)\Delta_t + o(\Delta_t) \quad (44)$$

where a$X^*(t)$ is the conditional instantaneous stochastic rate at time t of the process. This process is described as a pure birth process.

For more than one compartment, the times between jumps is also exponentially distributed with the parameters as the states of the compartment at the inception of the interval. Every component of the Markov jump process is described as a birth and death process with intensity rates of the stochastic process depending on all the compartments. A stochastic model is formulated as a stochastic process with a collection of random variables

3.10.2 Formulation of the SEIR model

Let us consider four (4) subgroup of individuals in the population namely, Susceptible (S), Exposed (E), Infected (I) and Recovered (R) and N= S+E+I+R. Within the time interval [t,t+Δ_t], the transition states of an infection will be $S \to S-1$, $E \to E+1$, $E \to E-1$, $I \to I+1$and for the probability for this to occur for the first stage is aSI$\Delta_t + o(\Delta_t)$ and for the second stage, let the movement rate of the exposed subgroup of individuals to the infected class be δ, then the probability for the second stage to occur is $\delta E\Delta_t + o(\Delta_t)$ and for the third stage, if the rate at which the infected recovered is assumed to be "ط" then the probability for the third stage will be ط$I\Delta_t + o(\Delta_t)$.

At a glance, the probabilities of the progression of the infection up to the recovery at time interval [t, t+Δ_t] are

$$P(S_{t+\Delta_t}, E_{t+\Delta_t}) - (S_t, E_t) = (-1,1) = aSI\Delta_t + o\Delta_t \quad (45)$$

$$P(E_{t+\Delta}\ , I_{t+\Delta_t}) - (E_t, I_t) = (-1,1) = \Delta E_t + o\Delta_t \quad (46)$$

$$P(I_{t+\Delta_t}, R_{t+\Delta_t}) - (I_t, R_t) = (-1,1) = ط I\Delta_t + o\Delta_t \quad (47)$$

with the complementary probability

$$P(S_{t+\Delta_t}, I_{t+\Delta_t}) - (S_t, I_t) = (0,0) = 1 - \left(aSI + \Delta E + \text{ط}I\right)\Delta_t + o\Delta_t \quad (48)$$

The increment of the stages $\Delta S = S_{t+\Delta_t} - S_t$,

$\Delta E = E_{t+\Delta_t} - E_t$, $\Delta I = I_{t+\Delta_t} - I_t$, are (-aSI)$\Delta_t$, (aSI-$\delta E$)$\Delta_t$ and ($\delta E - \text{ط}I$)Δ_t respectively, which is written as

$$\Delta S = (-asI)\Delta_t + \Delta\sigma_1$$

$$\Delta E = (asI - \delta E)\Delta_t - \Delta\sigma_1 + \Delta\sigma_2$$

$$\Delta I = (\delta E - \text{ط}I)\Delta_t - \Delta\sigma_2 + \Delta\sigma_3$$

where σ_i, i=1,2,3 are poisson increments with mean zero and conditional variances asIΔ_t, $\delta E\Delta_t$, ط IΔ_t.

Suppose we let $\Delta\sigma_i \to 0$ and $\Delta_t \to 0$ we have the resulting differential equation

$$\frac{dS(t)}{dt} = -aS(t)I(t)$$

$$\frac{dE(t)}{dt} = aS(t)I(t) - \delta E(t)$$

$$\frac{dI(t)}{dt} = \delta E(t) - \text{ط}I(t)$$

$$\frac{dR(t)}{dt} = \text{ط}I(t) + \text{ط}E(t) - cR(t)$$

which follows the deterministic model in system 3.

3.10.3 Stochastic SEIR with demography

Now with demography such as the birth, immigration/ migration and death whether natural or disease induced. Their inclusion in the model will make the model more realistic. The transition rates are given in the Table1.

Transition	Rate
$S \to S+1$	Λ
$S \to S-1$	$\mathrm{aSI}\Delta_t + \Lambda - \mu S(\Delta_t)$
$E \to E+1$	$\mathrm{aSI}\Delta_t$
$E \to E-1$	$(\ \mathrm{aSI}-\delta E)\Delta_t$-ط$\mathrm{E}\Delta_t$-μ$E\Delta_t$ = $\mathrm{aSI}\Delta_t$-(δ + ط +μ) Δ_t)
$I \to I+1$	$\delta E\Delta_t$
$I \to I-1$	$\delta E\Delta_t$ -(طIΔ_t- eIΔ_t-$\mu\Delta_t$) = $\delta E\Delta_t$-(ط +e+μ)I Δ_t)
$R \to R+1$	(طI+طE) Δ_t
$R \to R-1$	(طI +طE) Δ_t -(μ +c)RΔ_t

Table 1 The transition rates.

Where the migration rate= Λ, natural death rate = μ, disease induced rate = e and progress back to the susceptible class =c.

The corresponding probabilities are $P(S_t + \Delta_t, E_t + \Delta_t - (S_t, E_t = (1,0) = P(S_t + \Delta_t, E_t + \Delta_t) - (S_t, E_t = (-1,0) = \Lambda\Delta_t + o(\Delta_t)$ second stage also will be

$$P(S_t + \Delta_t, E_t + \Delta_t - (S_t, E_t = (-1,1) = asI\Delta_t + o(\Delta_t)$$

and for the remaining stages ,the probabilities were formulated and following the same procedures as in system 3 above, thus it results to

$$\frac{dS_*(t)}{dt} = \Lambda - aS(t)I(t) - \mu S(t) + cR(t)$$

$$\frac{dE_*(t)}{dt} = aS(t)I(t) - \mu E(t) - \text{ط}E(t) - \delta E(t)$$

$$\frac{dI_*(t)}{dt} = \delta E(t) - \text{ط}I(t) - eI(t) - \mu I(t)$$

$$\frac{dR_*(t)}{dt} = \text{ط}I(t) + \text{ط}E(t) - cR(t) - \mu R(t)$$

which is the same as the deterministic model and have the same deterministic rates.

3.10.4 Extension of the model to stochastic differential equation SEIR written as SDE SEIR

In the formulation of the stochastic differential equation, it takes various forms but the most prominent are to either perturb the states variables or the parameters or the transmission coefficient or the state variables using their deviation.To describe the

variability in the model, it is reasonable to add a noise term for example,

$\frac{dX^*t}{dt} = a(X^*(t), t) + \sigma(X^*(t), t)\omega(t)$ in which $\omega(t)$denotes the noise term and $\sigma(.)$ is a function representing the interaction between the noise and the present state. That is, the stochastic epidemic models via stochastic differential equation will make the parameters in deterministic models change randomly. Allowing environmental noise to play a part in the stochastic model may indicate the severity of the diseases. Adding stochastic to a model gives the model flexibility to fit the real data.

To extend the model to SDE model, let (Ω, F,$(F_t)_t \geq 0$,P) represents a complete probability space having a filtration $(F_t)_t \geq 0$ which satisfies increasing and right continuity condition and F_0 in P-null sets)and $B_i(t)$ to be an n-dimensional independent standard Brownian motion i.e. $B_i(t),\ 1 \leq i \leq n$.

If a parameter is to be estimated, it will be an average value of the parameter plus an error term. The parameters in the exposed class were perturbed only to see the behavioural change of the exposed individuals in the presence of environmental noise. That is,$\hat{a} = a + \sigma_1$,$\hat{\mu} = \mu + \sigma_2,\ \hat{b} = \text{ь} + \sigma_3,\ \hat{\delta} = \delta + \sigma_4$ so I =1,2,3,4.

The error term,$\sigma_i dt,\ 0 \leq i \leq 4$ is assumed to follow a normal distribution having a value of mean zero and variance $\sigma_i^2 dt$ that is,$\sigma_i d \approx \widehat{N}(0, \sigma_i^2 dt)$

These errors are presented as N-dimensional noise. $Bt = (B_1(t), \ldots, B_N(t))$

$$\sigma_i dt = \Sigma_{j=1}^{4} \sigma_{ij} dB_j(t), 0 \leq i \leq 4$$

Where$dB_j(t) = B_j(t + dt) - B_j(t),\ \sigma_{ij}$ are all real numbers such that

$\sigma_i^2 = \Sigma_{j=1}^{N} \sigma_i^2 j,\ 0 \leq i \leq 4\sigma^2 = \Sigma_{i=1}^{4} \sigma_i^2$.

In this model, since we are interested in the behaviour of the exposed, we make

random perturbation of the parameters of the exposed class other than the disease induced death rate. The parameters are $a, \mu, ط \; and \; \delta$.

The system 3 will now be

$$dS_*(t) = (\Lambda - aS(t)I(t) - \mu S(t) + cR(t))dt - \sigma_1 aS(t)I(t)dB_1 - \sigma_2 \mu S(t)dB_2(t) \quad (49)$$

$$dE_*(t) = (aS(t)I(t) - \mu E(t) - ط E(t) - \delta E(t))dt + \sigma_1 aS(t)I(t)dB_1(t) - \sigma_2 \mu S(t)dB_2(t) - \sigma_3 ط E(t)dB_3(t) - \sigma_4 \delta E(t)dB_4(t) \quad (50)$$

$$dI_*(t) = (\delta E(t) - ط I(t) - eI(t) - \mu I(t))dt + \sigma_4 \delta E(t)dB_4 - \sigma_3 ط I(t)dB_3(t) - \sigma_2 \mu I(t)dB_2 \quad (51)$$

$$dR_*(t) = (ط I(t) + ط E(t) - \mu R(t) - cR(t))dt + \sigma_3 ط E(t)dB_3(t) + \sigma_3 ط I(t)dB_3 t - \sigma_2 \mu R(t)db_2(t) \quad (52)$$

where σ_i, i =(1,2,3,4), the intensity of the white noise and B_i, i =1,2,3,4 are the standard independent Brownian motion.

3.10.5 Uniqueness of the solution to the stochastic SEIR model

We present the following theorem, which gives the uniqueness of the system 3 which is motivated by Yang and Mao (2010), Cai *et al (*2013), Rao (2014), Yang (2016), Miao *et al* (2017) and Wang *et al* (2017) and adapted for stochastic SEIR model of system 3.

Theorem: $Let (S'_*(0), E'_*(0), I'_*(0), and \; R'_*(0))^T \varepsilon \; R_+^4$ be the stochastic differential equation if and only if $P\big((S'_*(t), E'_*(t), I'_*(t), R'_*(t))^T \varepsilon \; R_+^4 \; \forall \; t \geq 0\big) = 1$.

We can prove the uniqueness of the solution to stochastic 3 as thus:

Proof

Let the values of the system U satisfy local Lipschitz continuity condition and let there be a unique solution$[0, T_e']$ where T_e' is the explosion time.

Assume $k_0' \geq 0$ is large enough so that $S'(0)$, $E'(0)$, $I'(0)$,and $R'(0)$ lie in the interval $[\frac{1}{k_0'}, k_0']$ for every $k' \geq k_0'$ is the stopping time

Say, $T_k' = if\left(t\ \varepsilon\ [0, T_e']: min\left(S_*'(t), E_*'(t), I_*'(t), R_*'(t) \leq \frac{1}{k'}\right)\right)$

or max $\left(\left(S_*'(t),\ E_*'(t),\ I_*'(t),\ R_*'(t)\right) \geq k'\right)$ with the assumption that $k_0' \geq 0$ such that $[\frac{1}{k_0'}, k_0']$ for every integer k $\geq k_0$ as the stopping time. Let inf $\phi = \infty$, T_k be increasing and let $T_\infty = \lim_{k\to\infty} T_k$, $0 \leq T_\infty \leq T_e$ almost everywhere (a.e)

If $T_\infty = \infty\ a.e\ then,\ T_e = \infty$ and the solution remains in R_+^4 for all $t \geq 0$

Suppose $T_\infty \neq \infty\ a.e$ then there are constants $T > o$ and e ε (0, 1) such that $P(T_\infty \leq T) > e.$

Hence, there is an integer $k_1' \geq k_0'$ such that $P\left(T_k' \leq T'\right) \geq e \forall k' \geq k_1'$

Let x be defined as set of the variables that is, (S,E,I,R) and V: $R_+^4 \to R$ such that

$$V(x) = \left(S - p - plog\frac{S}{p}\right) + (E - 1 - logE) + (I - 1 - logI) + (R - 1 - logR)$$

where p is a positive constant.

By Ito's formula, the diffusion matrix is defined as

$$A(x) = (A_{i,j}(x))_{1\leq i,j\leq 1}, A_{i,j}(x) = \Sigma_{r=1}^{d} \sigma_r^i(x)\sigma_r^j(x)$$

The differential operator L is given by

$$L = \Sigma_{r=1}^{l} \text{ط}_i(x)\frac{\partial}{\partial xi} + \frac{1}{2}\Sigma_{j=1}^{l} A_{i,j}(x)\frac{\partial^2}{\partial xi\, \partial xj}$$

If L acts on V, V $\varepsilon C^2(E_l\ X\ R_+;\ R)$ then

$$LV(x) = \Sigma_{r=1}^{l} \text{ь}_i(x) \frac{\partial v}{\partial xi} + \frac{1}{2} \Sigma_{j=1}^{l} A_{i,j}(x) \frac{\partial^2 v}{\partial xi\, \partial xj}$$

where $V_x = (\frac{\partial v}{\partial x}, \ldots . \frac{\partial v}{\partial xi} and\ V_{xx} = \left(\frac{\partial^2 v}{\partial xi\, \partial xj}\right)_{lxl}$

By Ito's formula we have,

$$dV(X(t)) = LV(X(t))dt + \Sigma_{r=1}^{d} V_x(X(t))\sigma_i(X(t))dB_r(t)$$

So from system 3,

$$LV_1 = \Lambda - \mu S - \mu E - eI - \mu I - \mu R + p(aI + \mu - \frac{\Lambda}{S} - \frac{cR}{S} - aS - \frac{\text{ь}E}{R} + \delta + c$$

$$+ p\Sigma_{j=1}^{N}(E\sigma_1 + \sigma_2)^2 + \Sigma_{j=1}^{N}(S\sigma_1 - \sigma_3)^2 + \sigma_4^2$$

Let $\alpha\beta \leq \mu$ then there is a constant C_1 so that

$$LV_1(x) \leq C_1 + \Sigma_{j=1}^{N}(E\sigma_1 + \sigma_2)^2 + \Sigma_{j=1}^{N}(S\sigma_1 - \sigma_3)^2$$

Let us define V_2 as function $V_2: R_+^4 \to R_+$ by

$V_2(x) = (S + E + I + R)^2$, X=(S,E,I,R) then

$$LV_2(x) = 2(S + E + I + R)(\Lambda - \mu S - \mu I + eI - \mu I - \mu R) + \Sigma_{j=1}^{N}(S\sigma_1 + E\sigma_2 + I\sigma_3$$

$$+ R\sigma_4)^2$$

$$\leq (S + E + I + R)^2 + \Lambda^2 + (\sigma_1^2 + \sigma_2^2 + \sigma_3^2 + \sigma_4^2)(S + E + I + R)^2$$

$$\leq C_2 + C_3(S + E + I + R)^2$$

where C_2, C_3 are positive constants. Let $V(x,t) = V_1(x) + V_2(x)$ and C a positive constant such that $LV(x) \leq C + CV(x)$

Let $V(x,t) = e^{-ct}(1 + V(x))$ then

$$LV(x) = -ce^{-ct}(1 + V(x)) + e^{-ct}LV(x) \leq 0$$

Let $X_*'(t) = (S_*'(t),\ E_*'(t),\ I_*'(t),\ R_*'(t)),\ t \geq 0$, by Ito's formula we have for any

$k' \geq k_1'$,

$$E\tilde{V}(x_*(q),(q)) = V(x_*(0)) + E\int_0^q LV(x_*(0),v)dx \leq \tilde{V}(x_*(0)$$

where $q = (t, T_k')$.

Now, let set $\Omega_k' = (T_k' \leq T)$ and by the assertion before that , $P(T_k' \leq T) \geq e$ for all $k' \geq k_1'$,then, $P(\Omega_k') \geq e$ for every ω' in Ω_k', $V(x(T_k', \omega)) \geq d_m$ in such a way $d_m = min(V(y))_y$ has at least a member as $\frac{1}{k}$, or k as k $\to \infty$.

This follows then that,

$ed_m \leq E[V(x(T_k', \omega)I\Omega_k)] \leq e^{CT}V(x(0)$and letting $k \to \infty$

which is $\neq \infty > e^{CT}V(x(0)) \geq \infty$ which contradicts the earlier claim. Therefore, $T_\infty = \infty$, almost everywhere shows that the stochastic model in system 3 is unique and has a positive solution.

3.10.6 Condition for the disease extinction

For the disease to go into extinction depends on the reproduction number R_0 if it is less than unity it will lose stability and the disease will go into extinction. The time to go into extinction is given as

$$t_e = \frac{\ln 0.5}{R_0} \tag{53}$$

By substituting equation 23 into equation 53 we have

$$t_e = \frac{\ln 0.5(\mu(\mu+\text{ь}+\delta)(\text{ь}+e+\mu))}{a\Lambda\delta} \tag{54}.$$

CHAPTER FOUR

RESULTS AND DISCUSSION

In this section, results of the numerical simulations of the dynamical behaviour of system 3 are presented by using Maple 18 and with the parameter values from existing literature. The initial conditions are put as S_0=21950, E_0=50, I_0=0, R_0=0 and the parameters varied we have:

Figure 4.1 shows the variation of the susceptible population without the vital dynamics such as migration into the population, the figure clearly shows the effect of migration. The susceptible population grows high as there is migration into the population. This is evident from the sensitivity analysis that the migration rate is highly sensitive

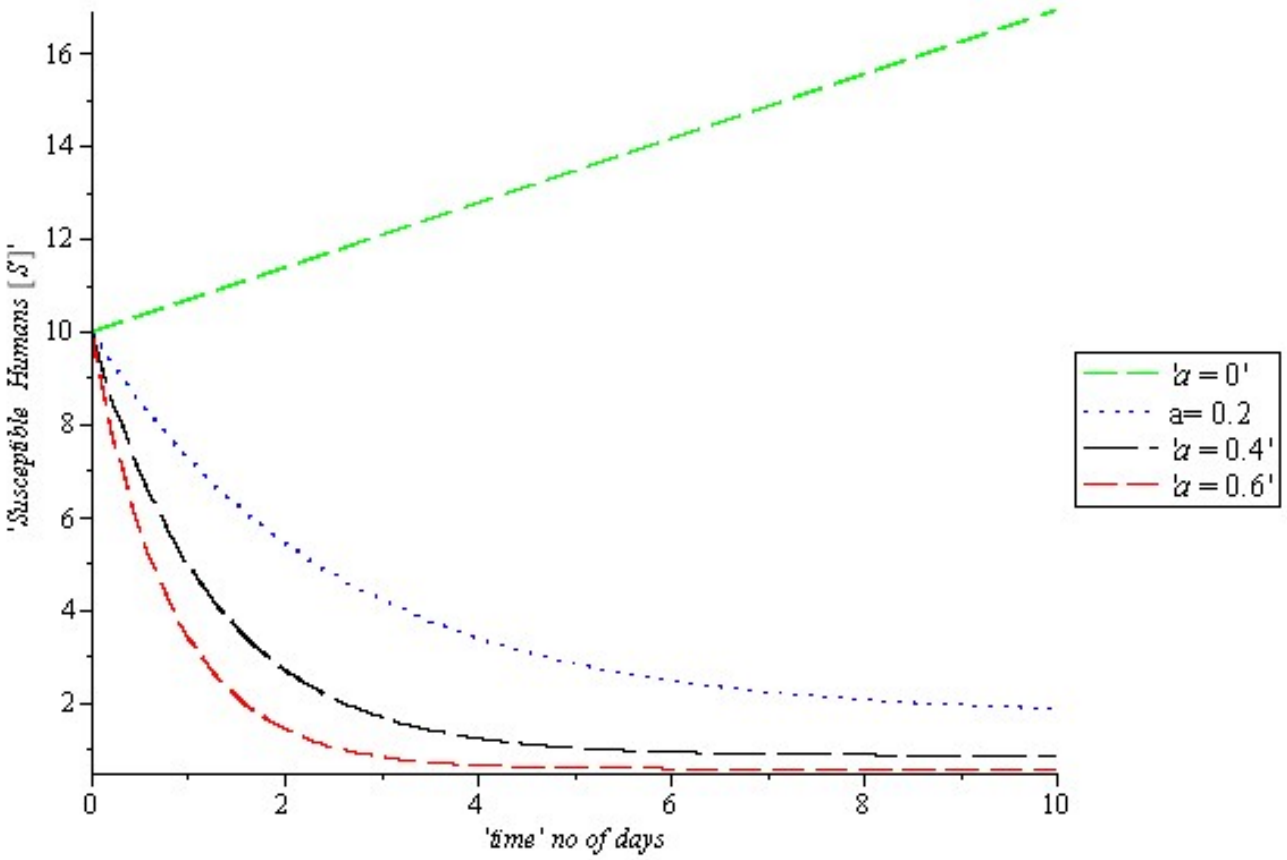


Figure 4.1 The variation of the susceptible population without the vital dynamics.

In Figure 4.1a,it is seen that the number of susceptible reduced when the transmission rate of the disease increased that is more susceptible individuals progress to the exposed and infected classes. This is true from the sensitivity analysis which revealed that the transmission parameter a, is one of the most sensitive parameters and that accounts for the rapid decline of the susceptible population.

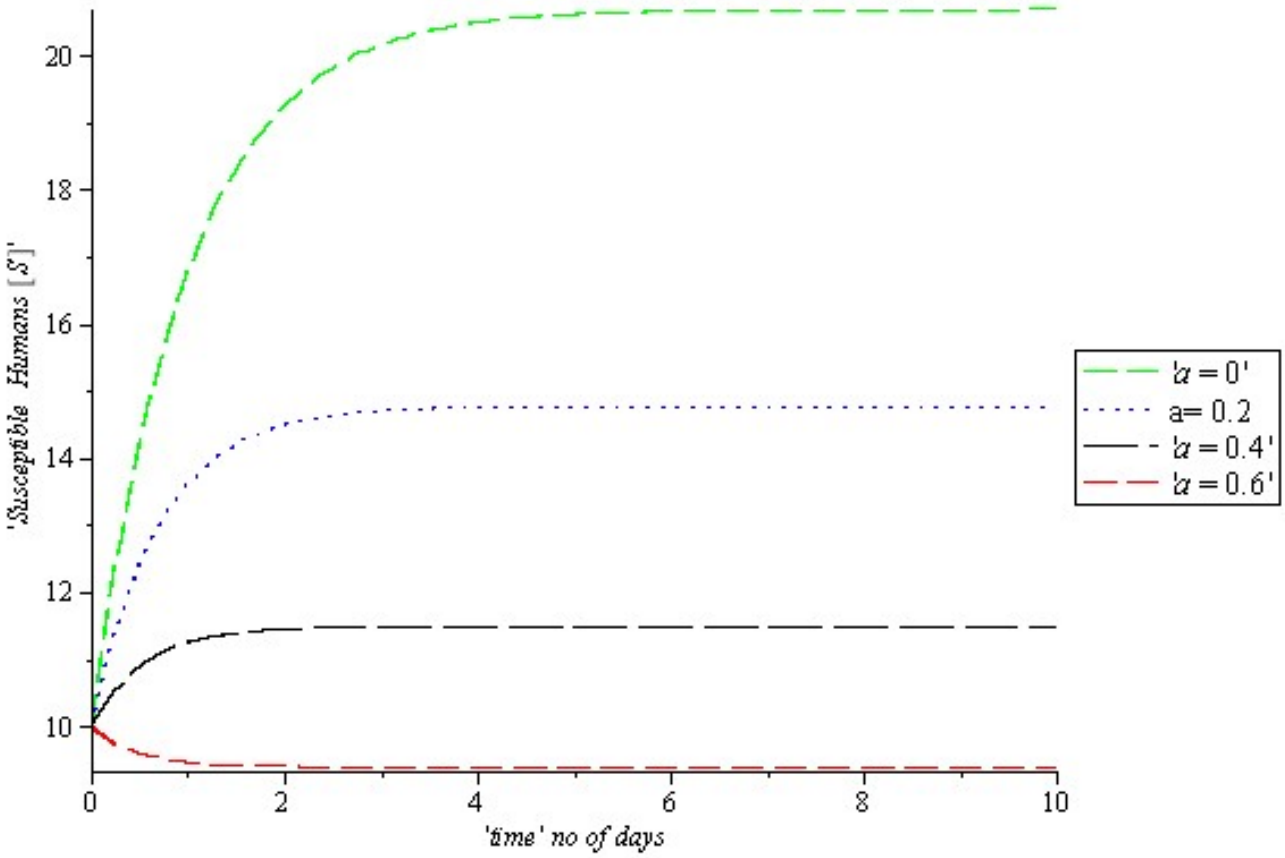


Figure 4.1a The variation of susceptible at various levels of transmission rates.

The simulation of the stochastic model for the susceptible is also used in Figure 4.1b to compare with the deterministic model of Figure 4. 1a The stochastic model of the susceptible shows an agreement in the shape as well as the rate of change of the transmission rate.

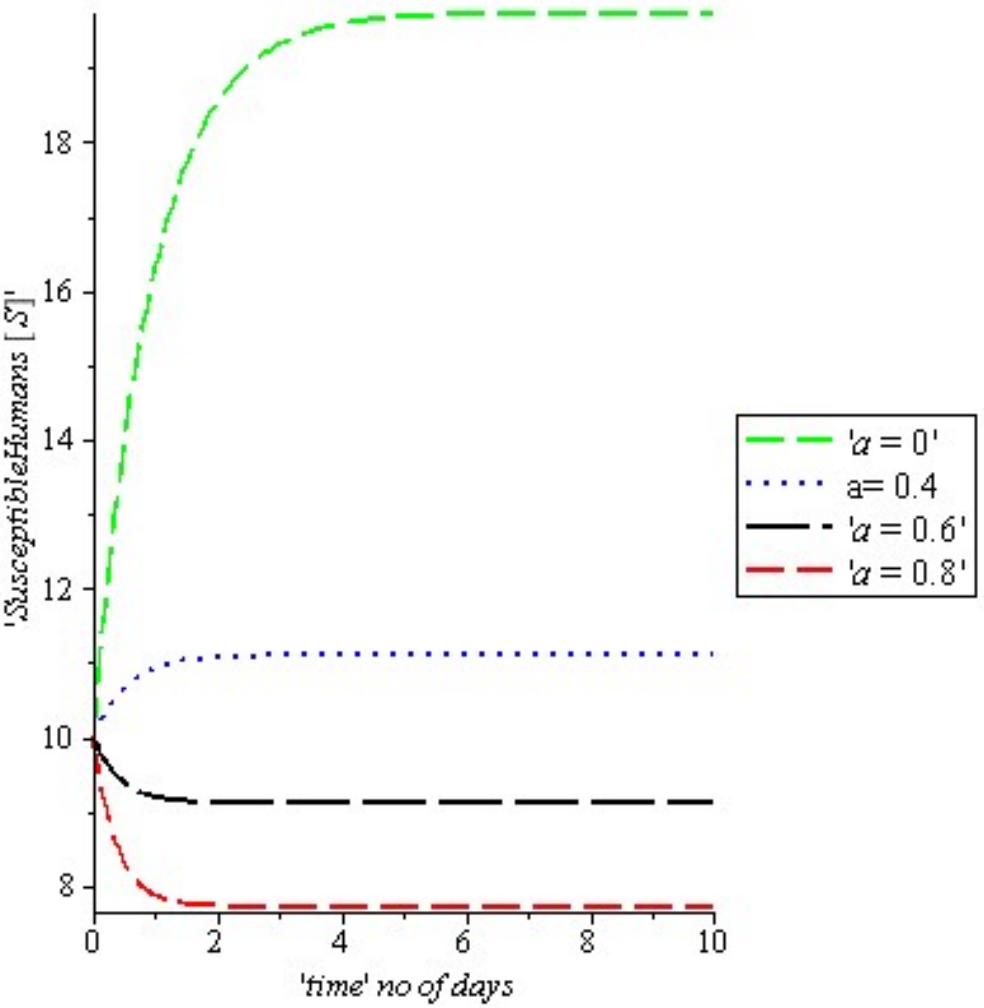


Figure 4.1b The variation of susceptible at various levels of a using stochastic model

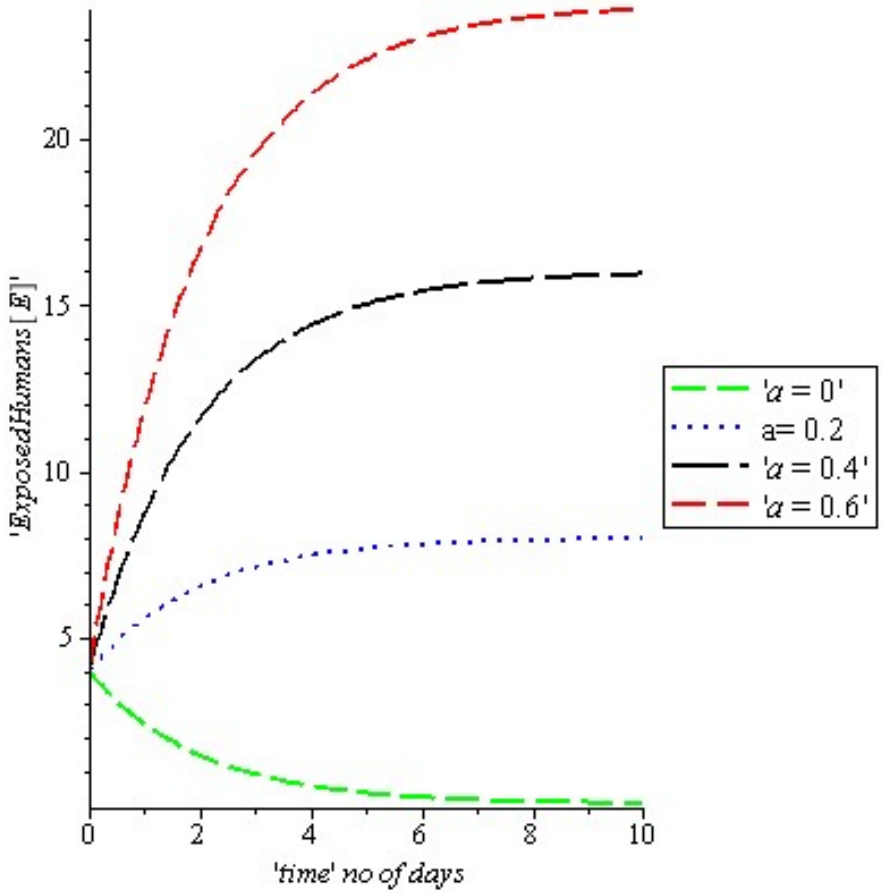


Figure 4.2aThe variation of exposed humans at various levels of a without the vital dynamics

In Figure 4.2a, when the transmission rate is increased, the number of the exposed increased because the susceptible individuals come to the exposed class to populate it.

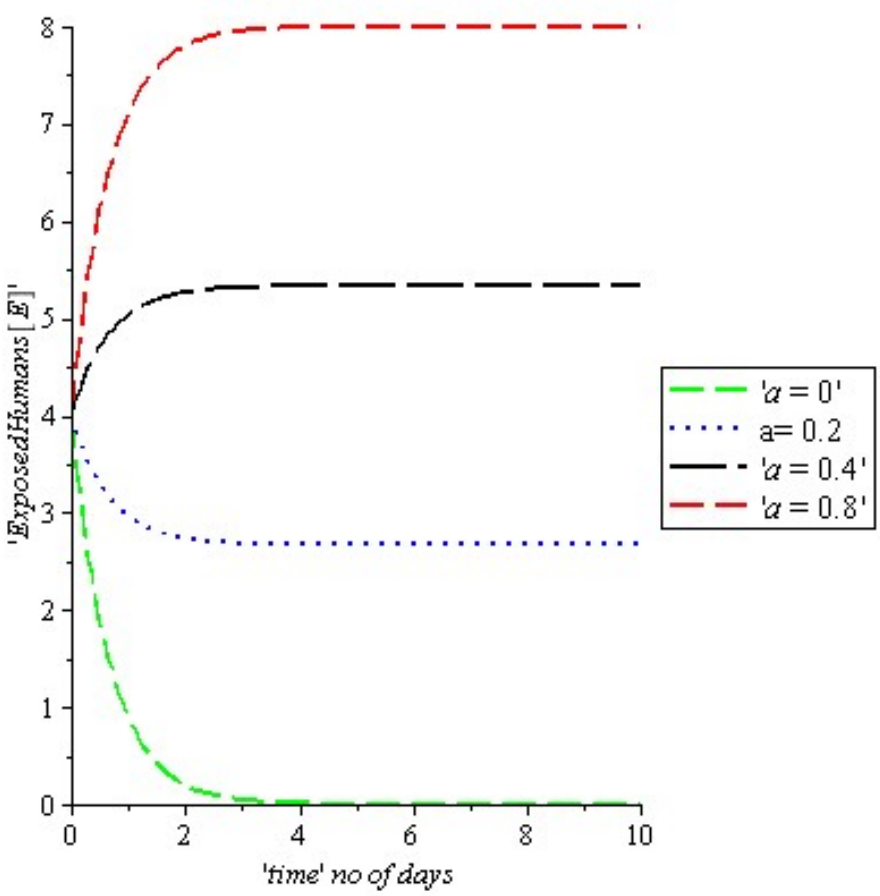


Figure 4.2bThe variation of exposed humans at various levels of a

In Figure 4.2b, the stochastic graph shows an agreement to what happened in the Figure 4.2a of the deterministic one

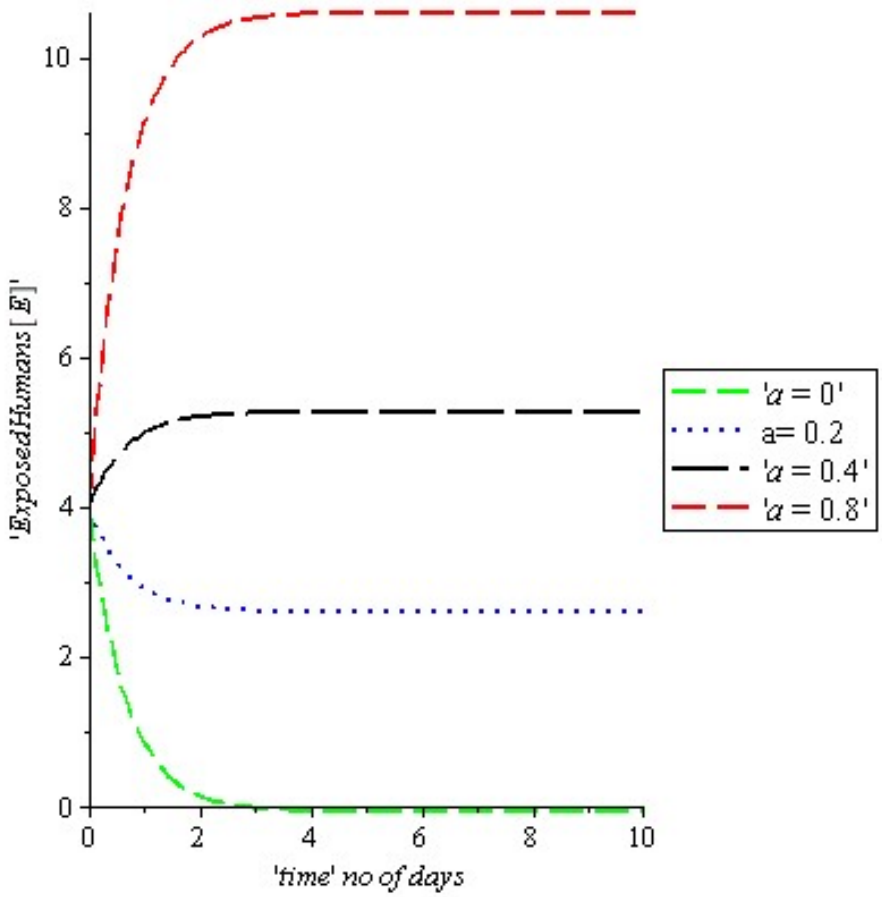


Figure 4.2cThe variation of the exposed using stochastic model

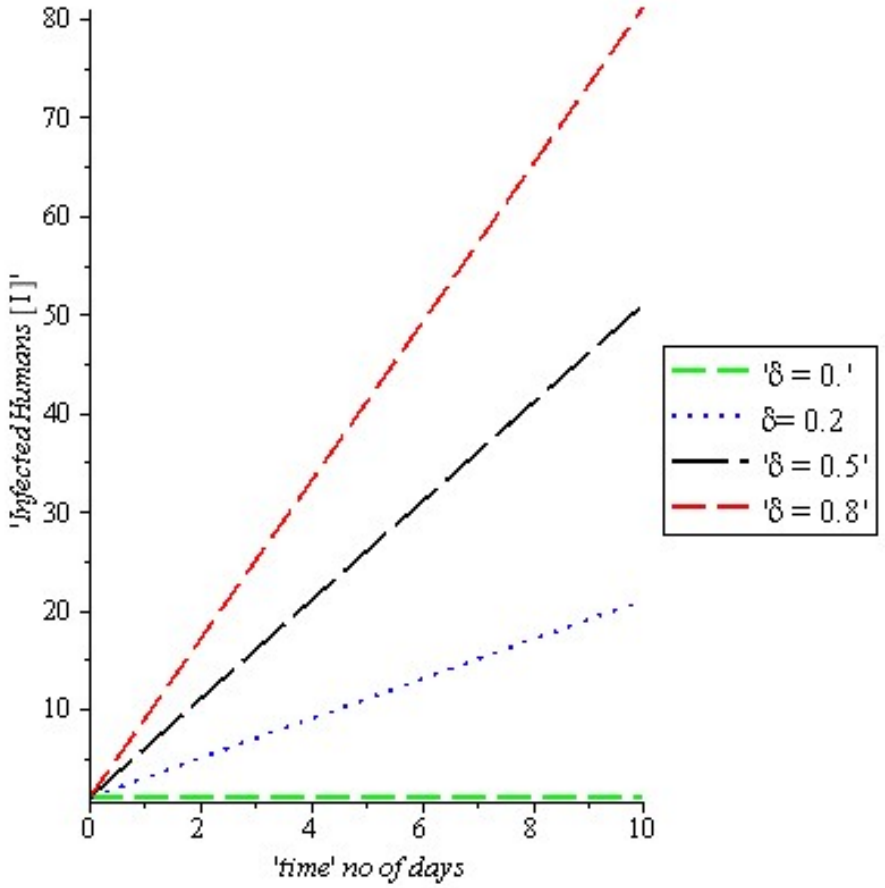


Figure 4.3aThe variation of infected humans without the vital dynamics

In Figure 4.3b, when the rate of progression of the exposed class into the infected class was varied from 0.0 to 0.8, it is clear that the number of infected humans varied considerably and increased when $\delta = 0.8$ that means the control measures to combat the disease should be increased in order to reduce the infected humans.

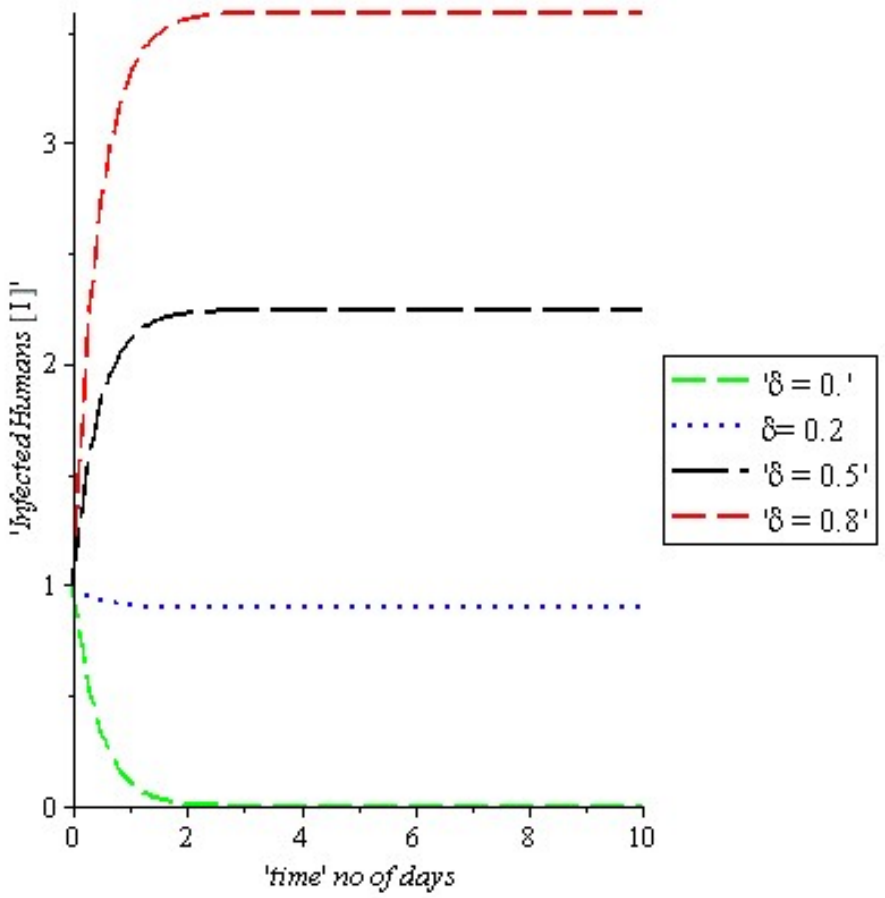


Figure 4.3b. The variation of infected humans while keeping the treatment rate constant

In Figure 4.3b, the stochastic variation of infected individuals, the stochastic model is in agreement with the deterministic one but with a slight change in shape which is due to the random variation which is expected

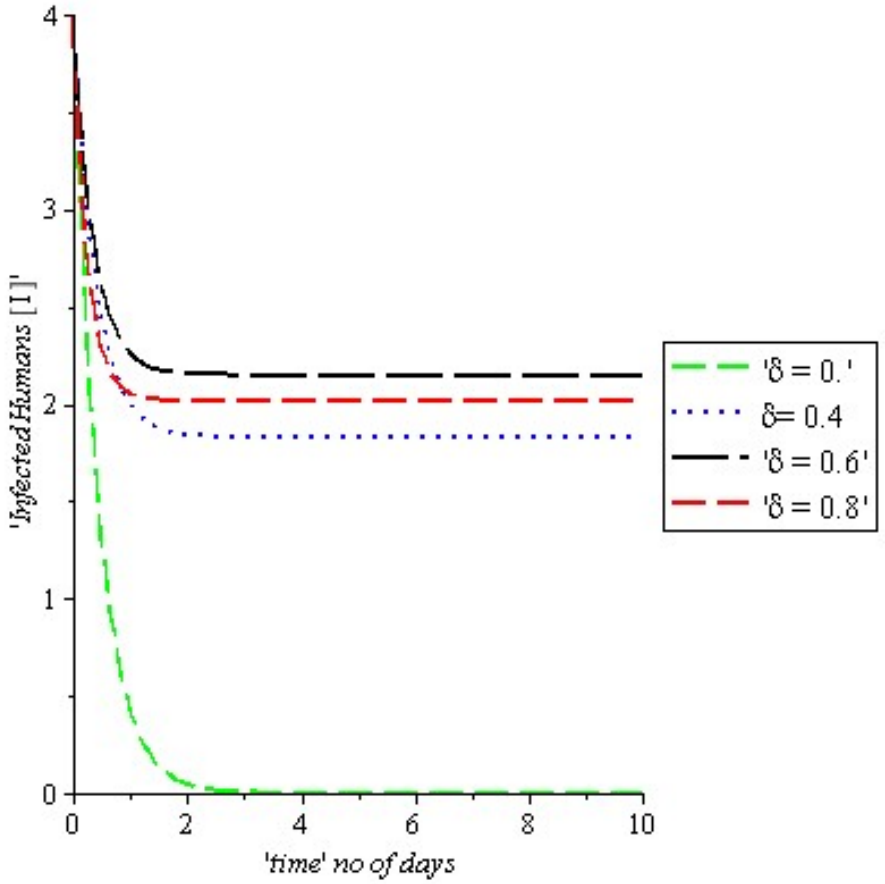


Figure 4.3c The variation of the infected humans using stochastic model

Figure 4.4 shows the variation of recovered population as the control measures are intensified. That is, when the rate of recovery varies as the control measures increases, the number of recovered individuals increases.

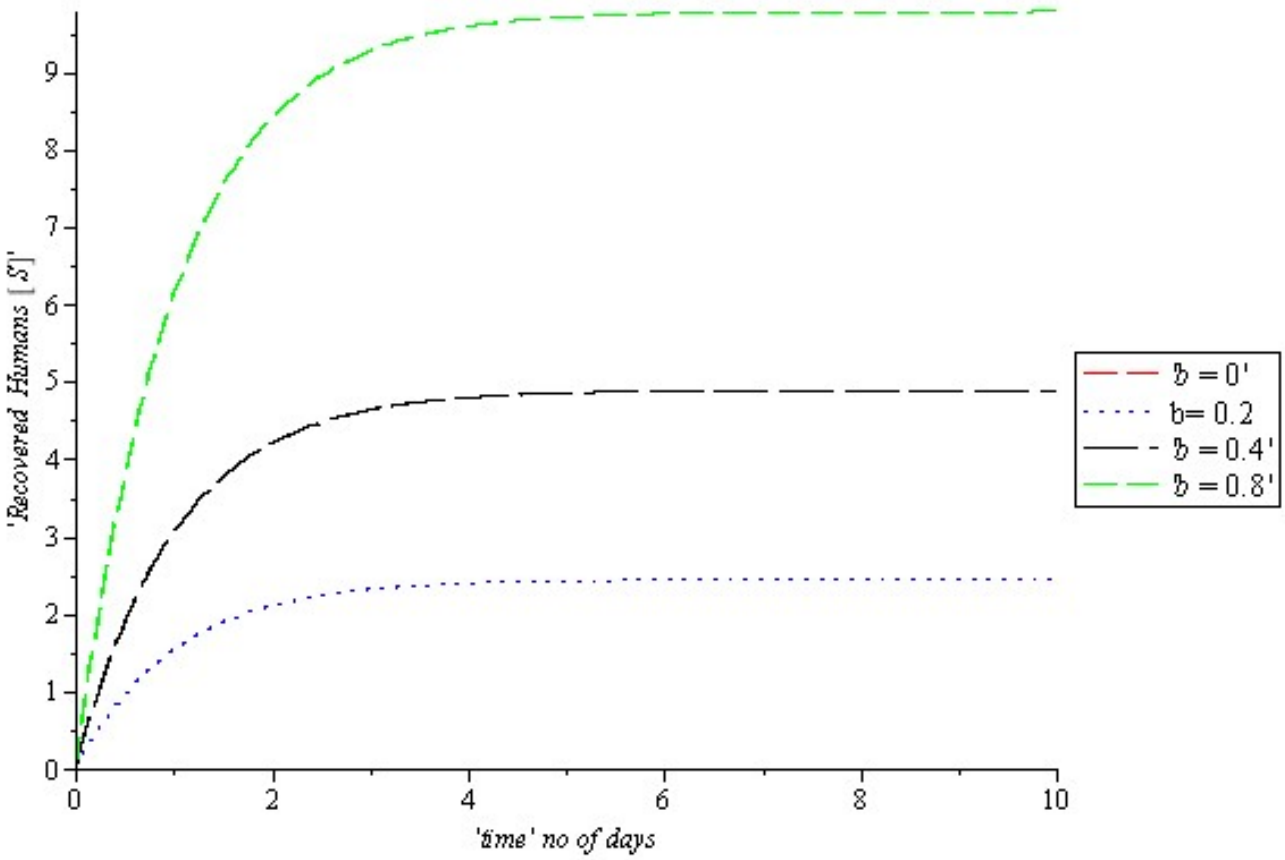


Figure 4.4 The variation of recovered humans at various levels of b

Figure 4.4a shows the stochastic variation of the recovered individuals as the control measures were put in place. It showed that as the control measures are put in place the recovered individuals increases which is in agreement with the deterministic counterpart

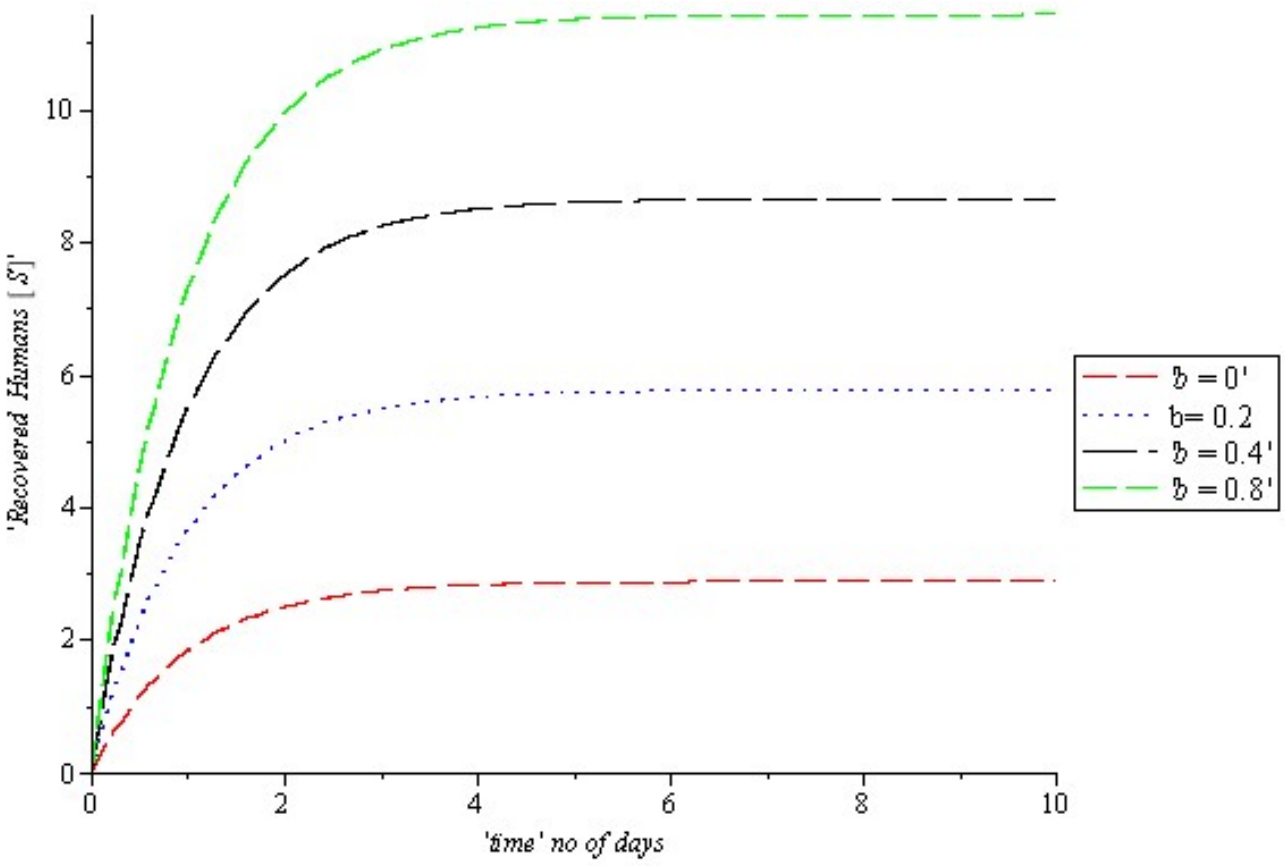
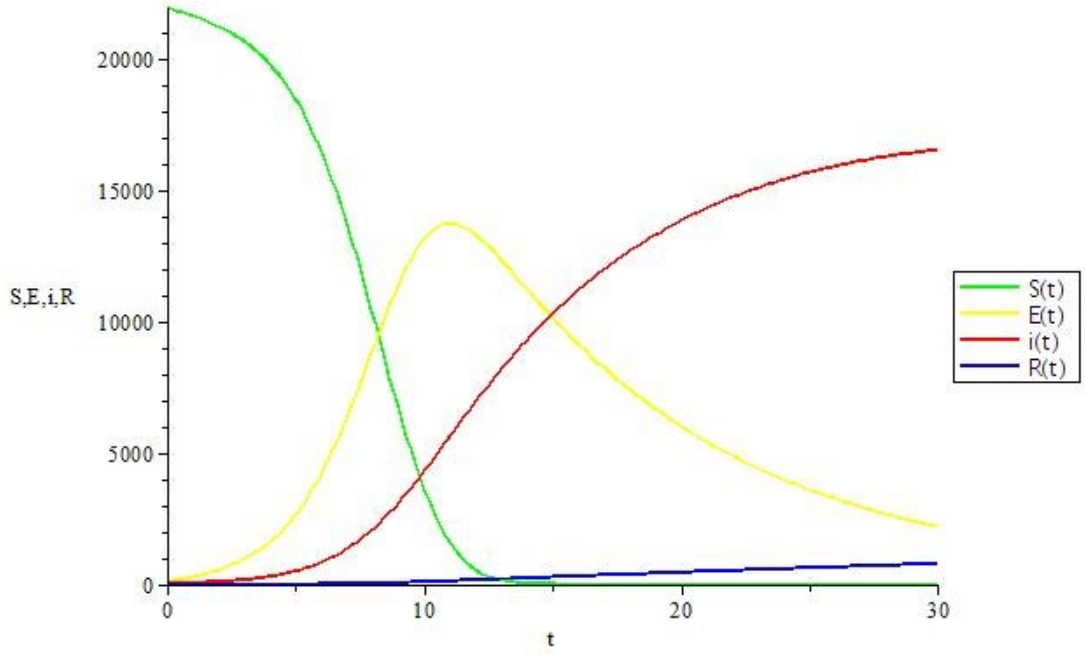


Figure 4.4a The stochastic variation of the recovered individuals.

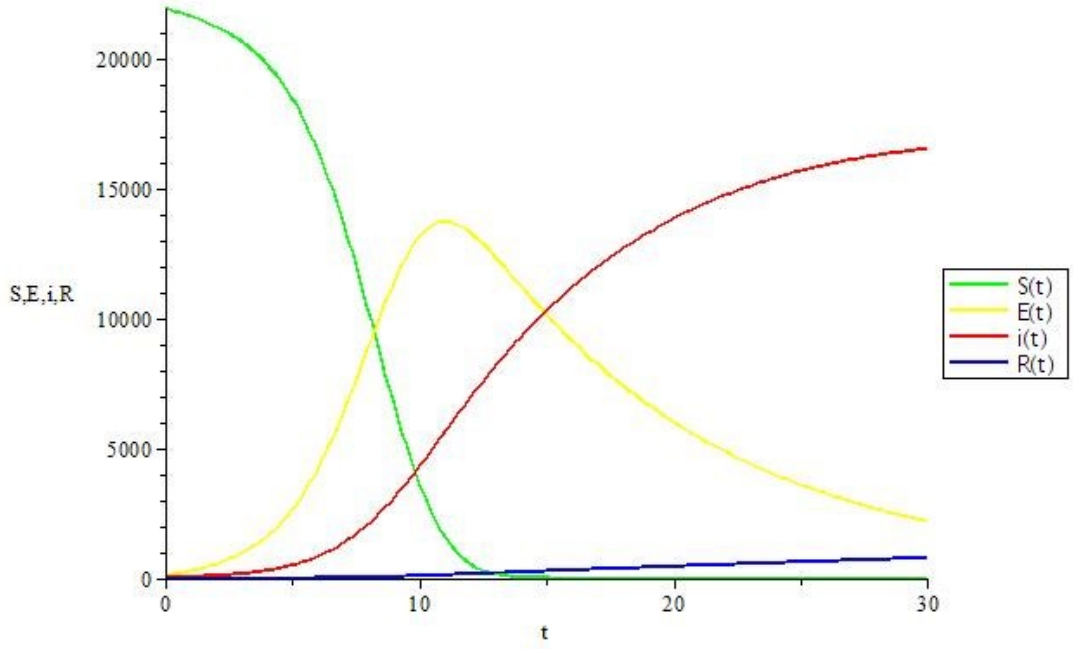
To know how the total population will look like if the reproduction number $R_0 > 1$, in the Figure 4.5 below, the infected population increased as the susceptible individuals decreased and the exposed decreased. It implies that, efforts must be intensified to control the most sensitive parameters so that the reproduction number can be brought below 1 so that the disease can die out.



t (time in months)

Figure 4.5 The variation of the total population when $R_0 > 1$

In Figure 4.5a, the stochastic variation of the total population when the basic reproduction number is greater than one showed that the recovered is at the minimum which increased the number of infected.



t (time in months)

Figure 4.5a The stochastic variation of the total population when $R_0 > 1$

We have established that the model of system 2 exhibited two equilibria : disease free and the endemic points. In the Figure 4.6 below, the disease free is stable when the system is kept below 1 and unstable as it is more than 1. Also, the endemic is unstable a bit around 1 and stable when it is more than 1.

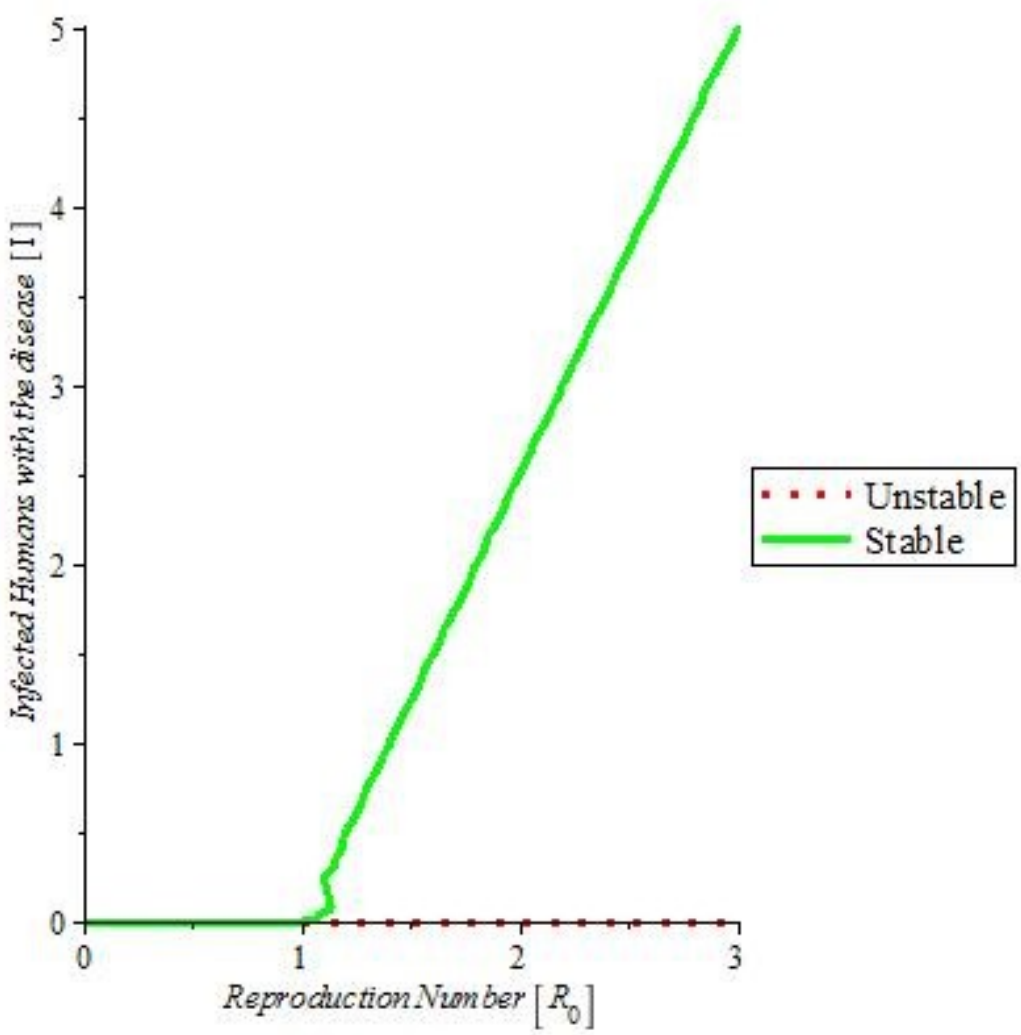


Figure 4.6 Bifurcation diagram of the model

In Figure 4.7, when the control measures were intensified, those that were exposed to the disease reduced.

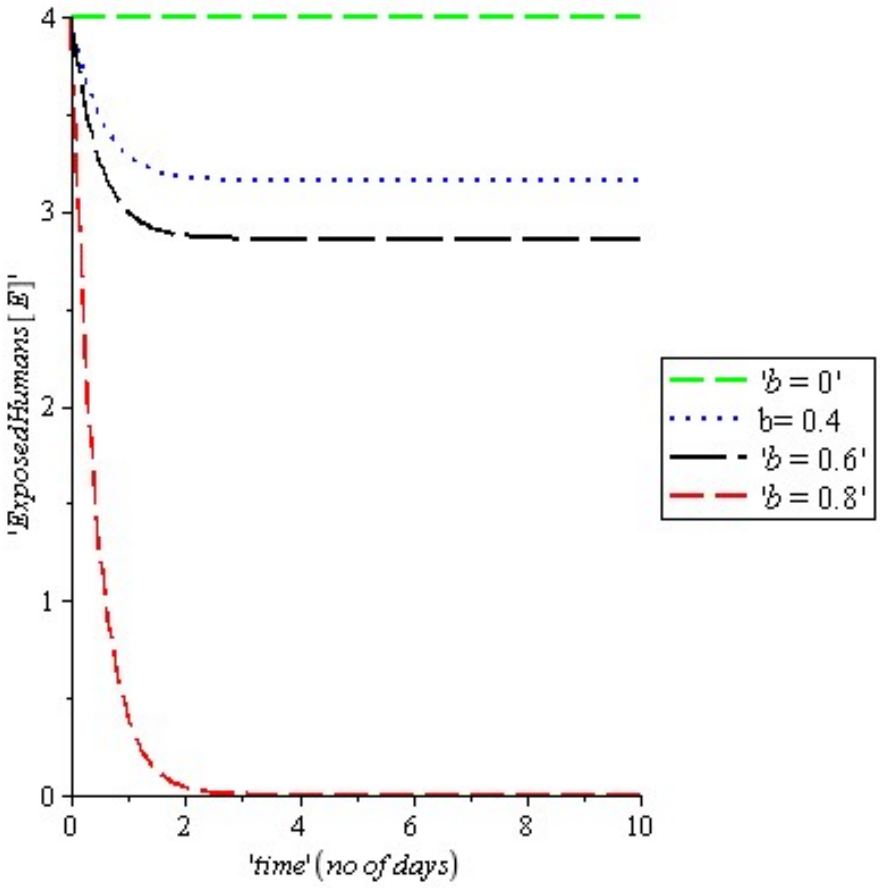


Figure 4.7 The effect of treatment on the exposed individuals.

Figure 4.8 shows effect of treatment on the infected individuals, as the rate of treatment "b" changes, the number of infected individuals reduces.

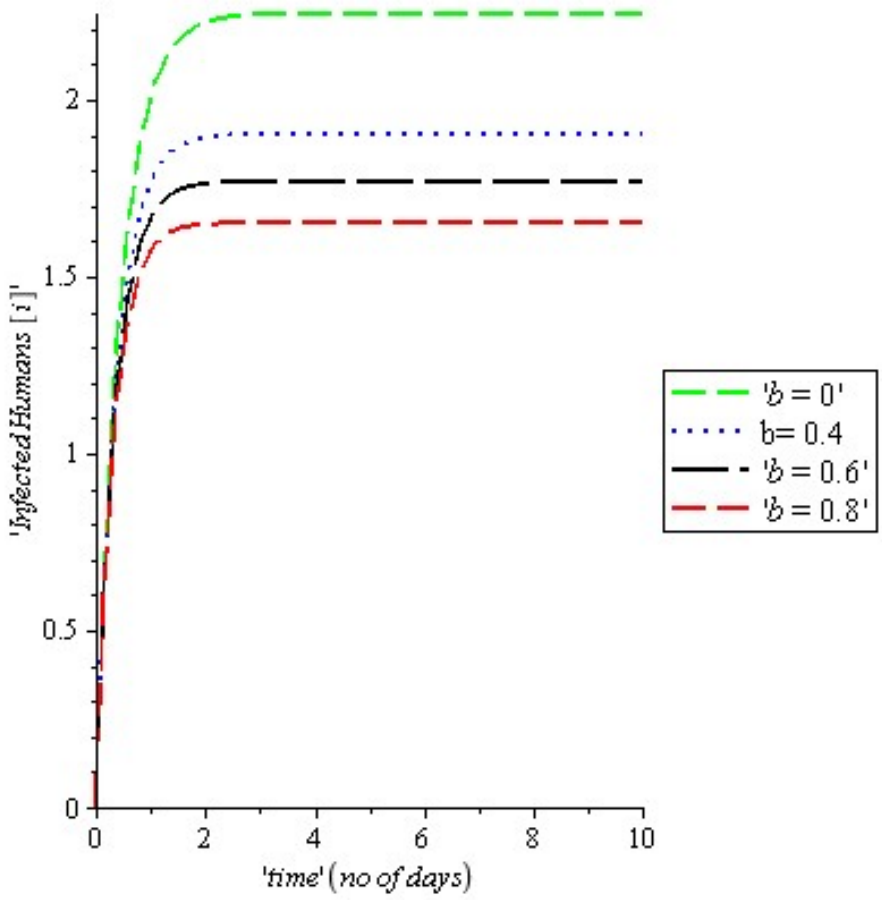


Figure 4.8 The effect of treatment on the infected humans.

Figures 4.9 and 4.10 are the overview of the total population in the long run of deterministic and stochastic models respectively. When the disease is minimized or negligible, the susceptible individuals with right information and good control measures put in place, will increase and the population will tend to a constant value while the exposed and the infected individuals decrease.

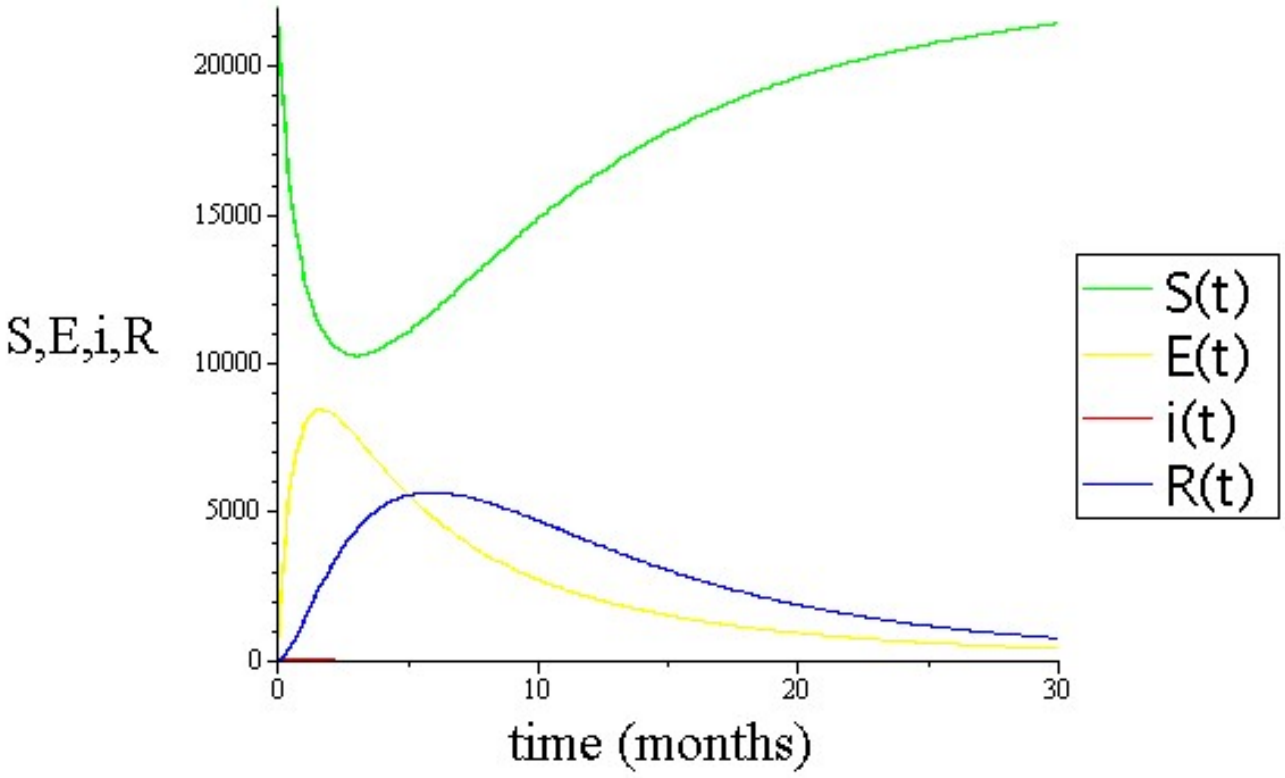


Figure 4.9 Variation of the total population

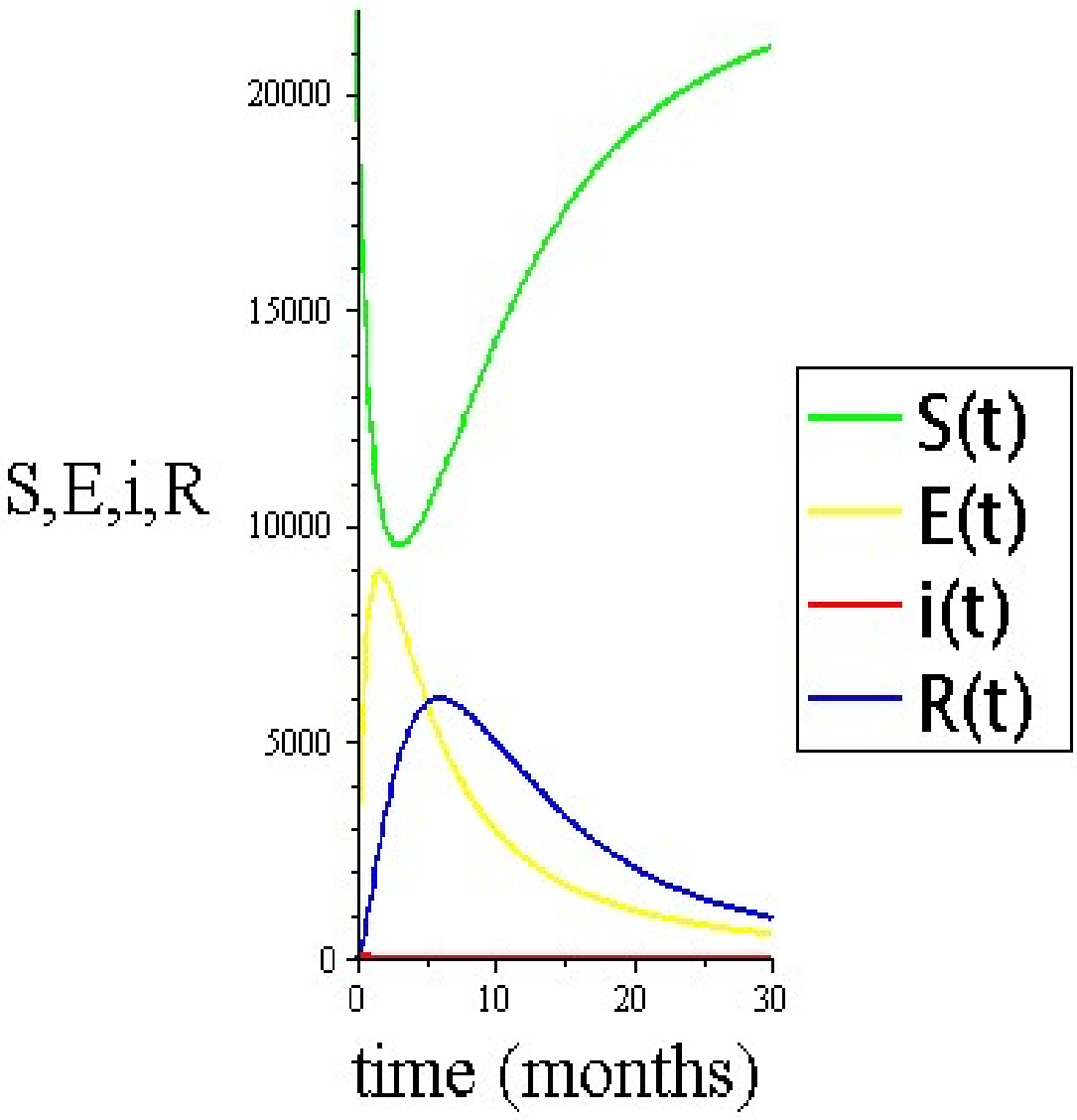


Figure 4.10 Stochastic variation of the total population

Figure 4.11 shows the effect of optimal control strategy 1, which is the treatment of infected individuals. It shows that with the introduction of the control; the effect of the disease is reduced

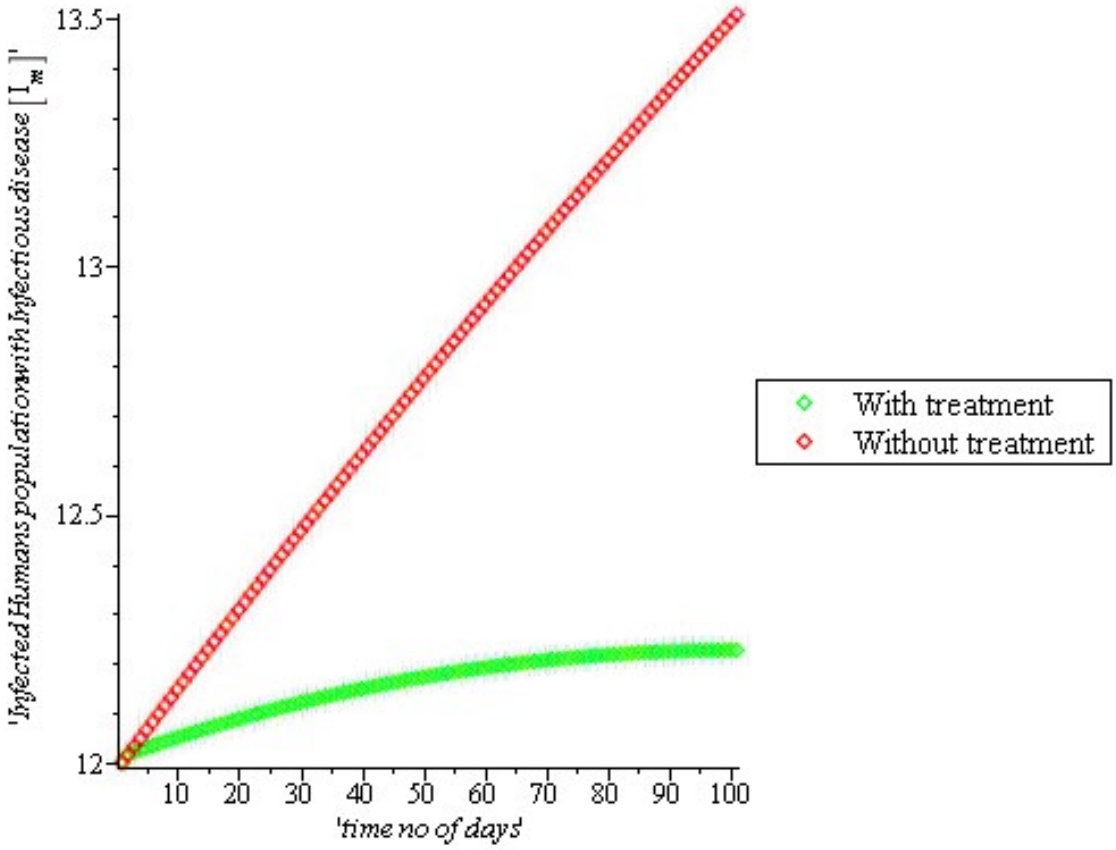


Figure 4.11 Optimal control of the model using treatment as a control measure.

The optimal control strategy 2

In Figure 4.12,the effect of the spread of the infectious disease is reduced as the introduction of the optimal control strategy using vaccination as a control measure is implemented.

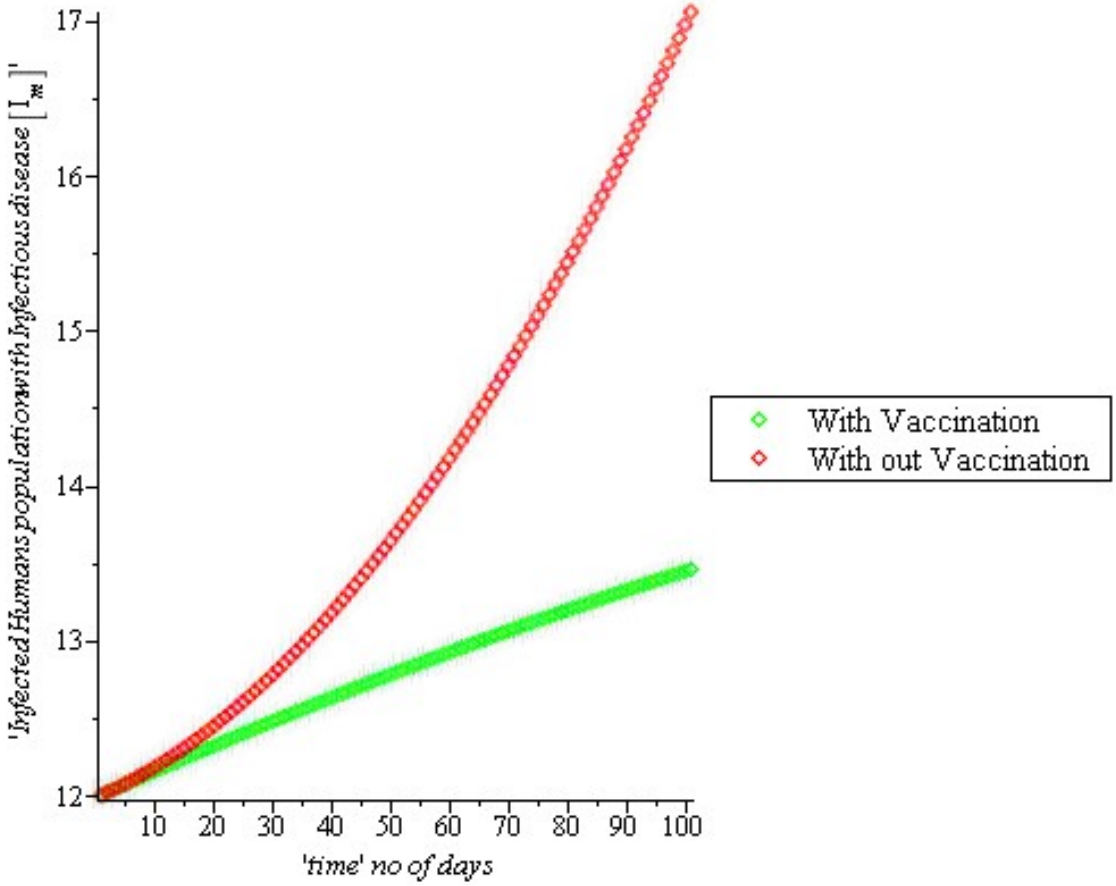


Figure 4.12 Optimal control of the model using vaccination as a control measure

The optimal control system using the combination of the two control measures in Figure 4.13 shows the optimal control strategy using the combination of the two control strategies. This proves more effective than using any one of them singly

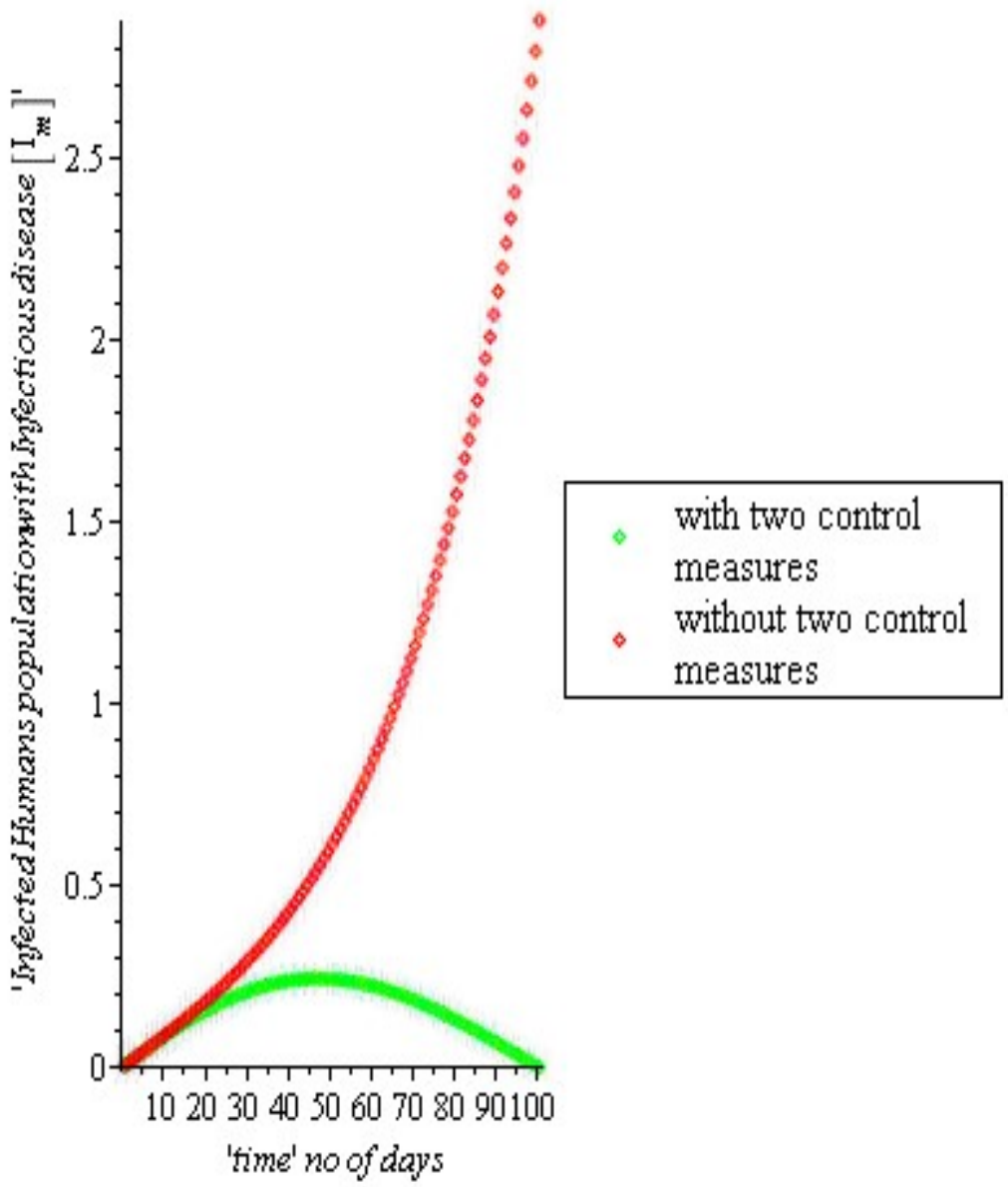


Figure 4.13 Optimal control strategy using the combination of the two strategies.

The model is applied to a live data of 2014 Ebola virus outbreak in Liberia, Sierra-Leonne and Guinea The parameters estimated from the live data were in agreement with the ones in the existing literature The parameters are given below with their respective values gotten from the least squares estimation method used.

Parameters	Values
a	0.000025
ط	0.48941
c	1.963907
δ	0.0498
μ	0.002165
Λ	1034
e	0.05019
σ_1	0.038
σ_2	0.24388
σ_3	0.269304
σ_4	0.630522

Table 2 The parameter values

The variation of total population using the deterministic model with the Ebola data of 2014 in Guinea, Sierra Leonne and Liberia is given in the Figures 4.14 and 4.15 below using deterministic and stochastic models respectively.

Figure 4.14 shows that after about 30 days of the outbreak, the susceptible population started to decrease and after about 61 days, the recovered individuals started increasing, this may be due to the intervention and awareness. This in turn reduced the exposed and infected individuals.

Using the stochastic model however,Figure 4.15 shows after about 25 days the susceptible population started decreasing and in about 31 days recovered individuals started to increase. The difference may be due to the randomness in the model.

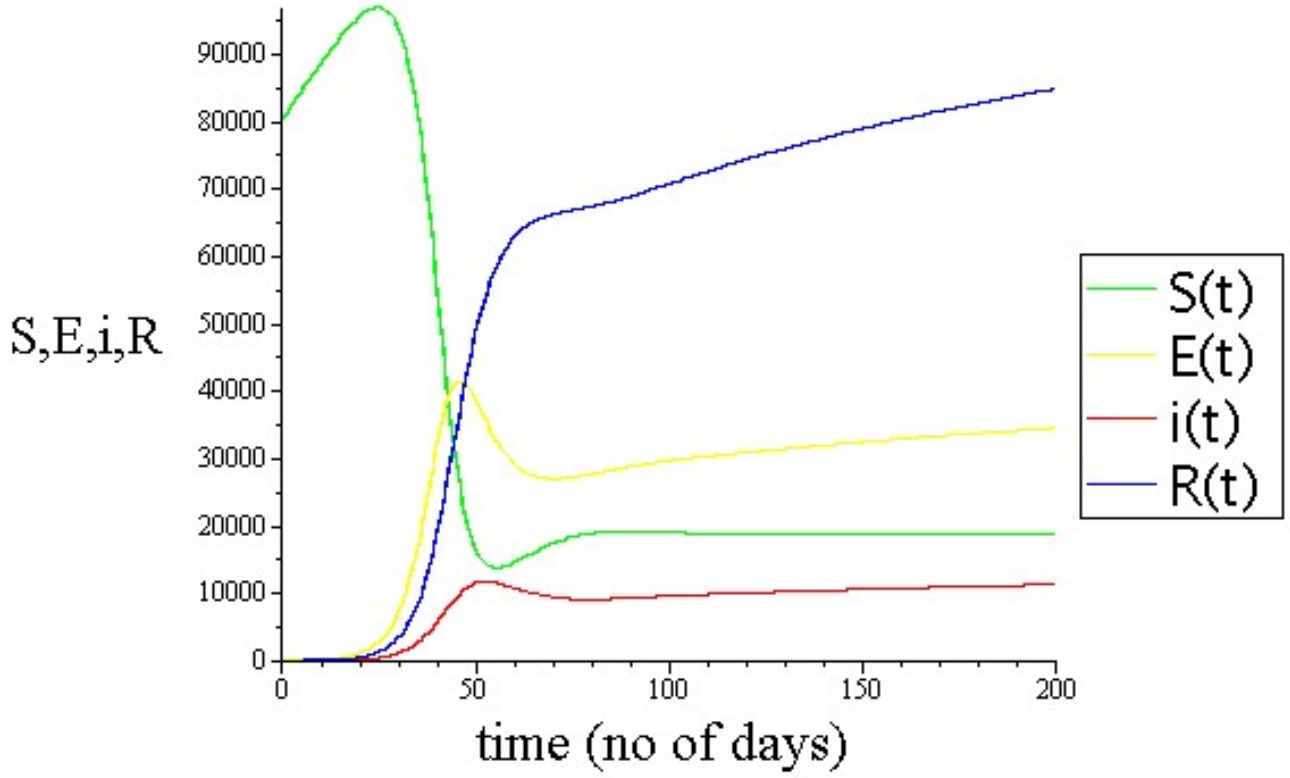


Figure 4.14 Variation of total population against time using Ebola data of 2014

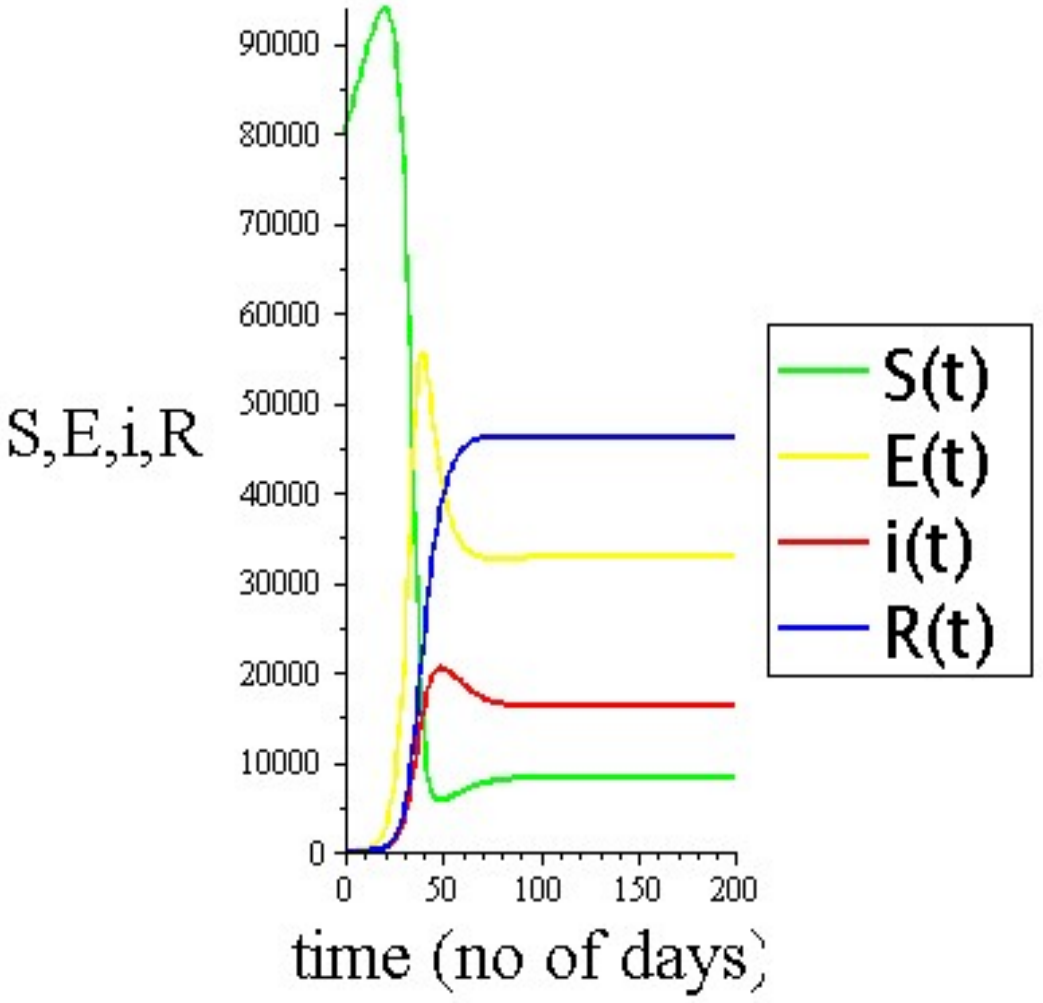


Figure 4.15 Stochastic variation of total population against time using Ebola data of 2014

The simulated R_0 was constructed using different values of the parameter simulated by varying the values of Λ, a and δ using uniform distribution. Each of the parameters was simulated for n = 1000 times. The varying values of the parameters are Λ U(500,1500), a U(0,0.00005), and δ U(0,0.0996).

The mean value for the three simulated R_0 is given by R_0 = 1.9873 The 95 % confidence interval for the R_0 is 1.9399 $\leq R_0 \leq$ 2.0346 The skewness of R_0 is 0.6933 and the measure of kurtosis is 1.5 The R_0 calculated with the parameters from the data is R_0 = 2.027 which is within the interval of the one gotten from the numerical simulation

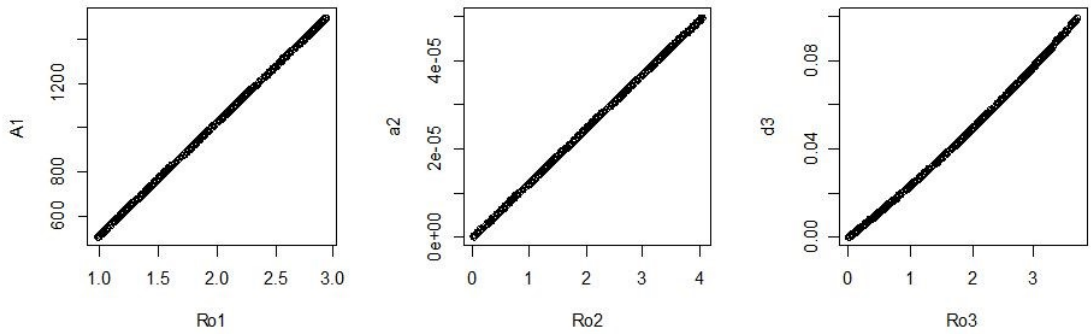


Figure 4.16 Graphs of Λ(A1) against R_0,a (a2) against R_0 and δ(d3) against R_0

In Figure 4.17, the histograms were presented which showed that the data is skewed positively.

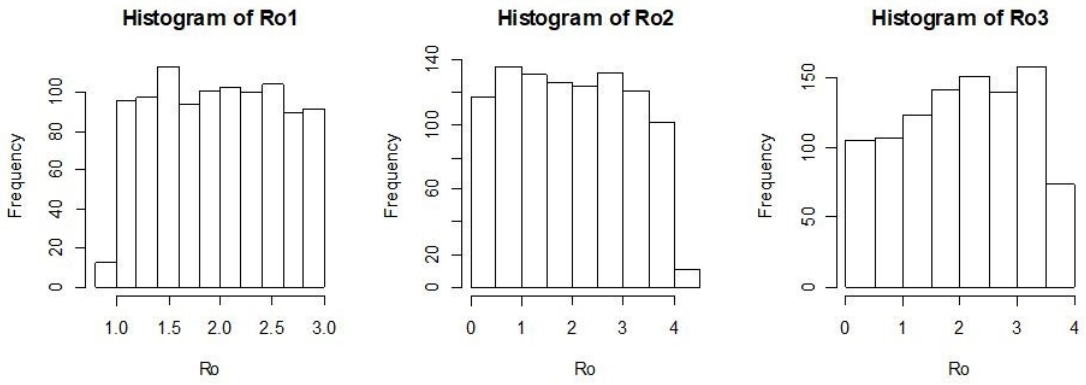


Figure 4.17 Histogram of the migration, transmission and progression rates against reproduction number

CHAPTER FIVE

SUMMARY,CONCLUSIONANDRECOMMENDATION

5.1 SUMMARY

In this study, a new non linear differential S-E-I-R model has been proposed and analysed to study and examine the transmission dynamics of infectious diseases in a dynamic population with migration into the susceptible population.The equilibrium points of the model were found to be E=0 and

$$S = \frac{(\mu+\text{ь}+\delta)(\text{ь}+e+\mu)}{a\delta}$$

by equating the non-linear differential equations of system 3 together. The disease free and endemic equilibria were obtained and their stabilities investigated. A numerical study of the model has been conducted to see the effect of certain parameters on the spread of infectious diseases. Model parameters have been estimated by applying Least squares estimation by linearising the non-linear differential equations on the live data of Ebola 2014. It is observed that if there is an increase in the transmission rate of the infection, there would be a sharp decrease of the susceptible population which would in turn increase the exposed population and the infected population. However, if efforts and control measures are intensified, this would bring about the increase in the recovered individuals and the infected population would decrease. It was found out that if R_0 is reduced to be less than unity (1), the disease would die out and the treated infected humans will increase which would lead to the eradication of the disease.

5.2 CONCLUSION

In this study, a non linear differential model had been proposed and analysed to study and examine the spread and transmission of infectious diseases in a dynamic population with migration in to the susceptible class. It is assumed that there is no constant population, due to the unpredictability nature of infectious diseases on the start of the epidemic, and for the fact that migration plays important role in the transmission of infectious diseases, migration rate has been introduced in to the model. By doing the analysis of the model,the equilibrium points were found to be

$$E = 0$$

or

$$S = \frac{(\mu+\text{ь}+\delta)(\text{ь}+e+\mu)}{a\delta}$$

The reproduction number R_0 is found to be

$$R_0 = \frac{a\Lambda\delta}{\mu(\mu+\text{ь}+\delta)(\text{ь}+e+\mu)}$$

by applying next generation matrix method. It is observed that when $R_0 < 1$, the disease dies out and when $R_0 > 1$, the disease persists in the system which is endemic. The model has two non-negative equilibria namely the disease free P^0 and the endemic P^*. The stability analysis of the model revealed that, the disease free equilibrium P^0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. Analysing the endemic equilibrium P^* by obtaining the derivative of the Lyapunov function showed that the endemic equilibrium is globally stable when $R_0 > 1$. The bifurcation of the model was examined and it showed that the model of system 3 has forward bifurcation. The sensitivity analysis showed that the migration rate (Λ) and transmission rate (a) are the most sensitive of all the parameters. In the study also, the optimal control system to reduce the burden of

the epidemic on the susceptible population was tested and it was found out that using a combination of treatment and vaccination proved to be more effective than using only one control of either of the two. The stochasticity of the model showed that the intensity of the white noise incorporated in the model made the results as close as possible to the deterministic one which is expected.It also showed that the effect of white noise affected the stability of the model. The deterministic and stochastic models were tested using both the numerical simulation and 2014 Ebola outbreak data outbreak in West Africa. The models performed closely as possible in the simulation as well as in the data. The reproduction number of the 2014 Ebola outbreak in some parts of West Africa was found to be 2.027 which revealed persistence of the disease over model without migration of 1.88. The value of the reproduction number of model with migration is within the interval of the one found in the simulation which is $1.9399 \leq R_0 \leq 2.0346$ at 95 percent confidence interval. The skewness of R_0 is 0.6933 and the measure of kurtosis is 1.5.

The value of the reproduction number with migration is in agreement with what was obtained in Althaus (2014) which falls within the interval of 1.5 and 2.5

5.3 RECOMMENDATION

Due to the dynamical nature of infectious diseases and the porousity of the borders of the developing countries, this research found out that an increase in the transmission rate of the infection would bring about a sharp decrease in the susceptible population and in turn increase the exposed population that are at risk and infected population , thus, the transmission rate of the infection should be controlled by effective treatment, public enlightenment, washing of hands regularly, quarantine, vaccination etc to keep the infected

population under control. Also,to keep the migration / inflows of individuals into the susceptible class in check, immunization of new born against the disease at the hospitals should be done, immigrants into the community/ country should be controlled by contact tracing, effective treatment / check at the boarders, to prevent the spread of the disease.

5.4 CONTRIBUTION TO KNOWLEDGE

A review of compartmental model S-E-I-R that is useful in infectious modelling wasanalysed. Since the classical assumption of stable population has been violated, a robust model that incorporated migration in and out of the population was proposed and introduced. The model was extended to stochastic model by using stochastic differential equation to allow for randomenvironmental fluctuation

5.5 SUGGESTION FOR FURTHER STUDIES

Due to the deadly nature of infectious diseases and the sporadic nature of its spread, it is important for more researches in the study of the spread and control to explore new ways/ strategies of its control.Based on the model of this study, we proposed that further study should be on

- The maximum likelihood estimation of the parameters using Weibull distribution and other probability distributions in a stochastic epidemic model.
- Expansion of the model to incorporate the vaccination or immunization of the immigrants and new born.
- Persistence and spatial study of the spread of infectious disease using stochastic differential equation.
- Effective study of stochastic stability on the reproduction number of S-E-I-R model.

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APPENDIX

Maple code for the variation of susceptible without the dynamics

```
restart;
equ1 := diff(S(t), t)-Lambda+a*S(t)*i(t)+mu*S(t)-c*R(t);
```

$$\frac{d}{dt} S(t) - \text{Lambda} + a\, S(t)\, i(t) + \text{mu}\, S(t) - c\, R(t)$$

```
equ2 := evalf(subs(i(t) = 2, R(t) = 3, a = 0, b = 0.5e-2, e = 1.229, mu = 0, c = .23,
delta = .5, Lambda = 0, equ1));
```

$$\frac{d}{dt} S(t) - 0.69$$

```
equ3IVP := evalf(rhs(dsolve({equ2, S(0) = 10}, S(t))));
```

$$0.6900000000\, t + 10.$$

```
D1 := evalf(equ3IVP);
```

$$0.6900000000\, t + 10.$$

```
plot(D1, t = 0 .. 10, labels = ["time", "S"], title = "Figure 1: The variation of
susceptible population at value a, a=0 ");
equ1 := diff(S(t), t)-Lambda+a*S(t)*i(t)+mu*S(t)-c*R(t);
```

$$\frac{d}{dt} S(t) - \text{Lambda} + a\, S(t)\, i(t) + \text{mu}\, S(t) - c\, R(t)$$

equ2 := evalf(subs(i(t) = 2, R(t) = 3, a = .2, b = 0.5e-2, e = 1.229, mu = 0, c = .23, delta = .5, Lambda = 0, equ1));

$$\frac{d}{dt}S(t) - 0.69 + 0.4\,S(t)$$

equ3IVP := evalf(rhs(dsolve({equ2, S(0) = 10}, S(t))));

$$1.725000000 + 8.275000000\,\exp(-0.4000000000\,t)$$

D2 := evalf(equ3IVP);

$$1.725000000 + 8.275000000\,\exp(-0.4000000000\,t)$$

plot(D2, t = 0 .. 10, labels = ["time", "S"], title = "Figure 1: The variation of susceptible population at value a , a ");

equ1 := diff(S(t), t)-Lambda+a*S(t)*i(t)+mu*S(t)-c*R(t);

$$\frac{d}{dt}S(t) - \mathrm{Lambda} + a\,S(t)\,i(t) + \mu\,S(t) - c\,R(t)$$

equ2 := evalf(subs(i(t) = 2, R(t) = 3, a = .4, b = 0.5e-2, e = 1.229, mu = 0, c = .23, delta = .5, Lambda = 0, equ1));

$$\frac{d}{dt}S(t) - 0.69 + 0.8\,S(t)$$

equ3IVP := evalf(rhs(dsolve({equ2, S(0) = 10}, S(t))));

0.8625000000 + 9.137500000 exp(-0.8000000000 t)

D3 := evalf(equ3IVP);

0.8625000000 + 9.137500000 exp(-0.8000000000 t)

plot(D3, t = 0 .. 10, labels = ["time", "S"], title = "Figure 1: The variation of susceptible population at value a, a ");

equ1 := diff(S(t), t)-Lambda+a*S(t)*i(t)+mu*S(t)-c*R(t);

$$\frac{d}{dt} S(t) - \text{Lambda} + a\,S(t)\,i(t) + \text{mu}\,S(t) - c\,R(t)$$

equ2 := evalf(subs(i(t) = 2, R(t) = 3, a = .6, b = 0.5e-2, e = 1.229, mu = 0, c = .23, delta = .5, Lambda = 0, equ1));

$$\frac{d}{dt} S(t) - 0.69 + 1.2\,S(t)$$

equ3IVP := evalf(rhs(dsolve({equ2, S(0) = 10}, S(t))));

0.5750000000 + 9.425000000 exp(-1.200000000 t)

D4 := evalf(equ3IVP);

0.5750000000 + 9.425000000 exp(-1.200000000 t)

plot(D4, t = 0 .. 10, labels = ["time", "S"], title = "Figure 1: The variation of

susceptible population at value a , a ")

plot([D1, D2, D3, D4], t = 0 .. 10, labels = [''time'*no*of*days', ''Susceptible*Humans*[S]''], labeldirections = ["horizontal", "vertical"], linestyle = [dash, dot, longdash], color = [green, blue, black, red], legend = [''a = 0'', "a= 0.2", ''a = .4'', ''a = .6'']);

Maple code for the variation of susceptible population at various levels of a

```
restart;
equ1 := diff(S(t), t)-Lambda+a*S(t)*i(t)+mu*S(t)-c*R(t);
        |--- S(t)| - Lambda + a S(t) i(t) + mu S(t) - c R(t)
equ2 := evalf(subs(i(t) = 2, R(t) = 3, a = 0, b = 0.5e-2, e = 1.229, mu = 1, c = .23,
delta = .5, Lambda = 20, equ1));
equ3IVP := evalf(rhs(dsolve({equ2, S(0) = 10}, S(t))));
D1 := evalf(equ3IVP);
     plot(D1, t = 0 .. 10, labels = ["time", "S"], title = "Figure 1: The variation of
susceptible population at value a, a=0 ");
equ1 := diff(S(t), t)-Lambda+a*S(t)*i(t)+mu*S(t)-c*R(t);
        |--- S(t)| - Lambda + a S(t) i(t) + mu S(t) - c R(t)
equ2 := evalf(subs(i(t) = 2, R(t) = 3, a = .2, b = 0.5e-2, e = 1.229, mu = 1, c = .23,
delta = .5, Lambda = 20, equ1)
equ3IVP := evalf(rhs(dsolve({equ2, S(0) = 10}, S(t))));
D2 := evalf(equ3IVP);
plot(D2, t = 0 .. 10, labels = ["time", "S"], title = "Figure 1: The variation of
susceptible population at value a , a ");
```

```
equ1 := diff(S(t), t)-Lambda+a*S(t)*i(t)+mu*S(t)-c*R(t);
 |--- S(t)| - Lambda + a S(t) i(t) + mu S(t) - c R(t)
equ2 := evalf(subs(i(t) = 2, R(t) = 3, a = .4, b = 0.5e-2, e = 1.229, mu = 1, c = .23,
delta = .5, Lambda = 20, equ1));     /
equ3IVP := evalf(rhs(dsolve({equ2, S(0) = 10}, S(t))));
D3 := evalf(equ3IVP);
plot(D3, t = 0 .. 10, labels = ["time", "S"], title = "Figure 1: The variation of
susceptible population at value a, a ");
equ1 := diff(S(t), t)-Lambda+a*S(t)*i(t)+mu*S(t)-c*R(t);
equ2 := evalf(subs(i(t) = 2, R(t) = 3, a = .6, b = 0.5e-2, e = 1.229, mu = 1, c = .23,
delta = .5, Lambda = 20, equ1));
equ3IVP := evalf(rhs(dsolve({equ2, S(0) = 10}, S(t))));
D4 := evalf(equ3IVP);
plot(D4, t = 0 .. 10, labels = ["time", "S"], title = "Figure 1: The variation of
susceptible population at value a , a ");
plot([D1, D2, D3, D4], t = 0 .. 10, labels = ["time", "Susceptible*Humans*[S]"],
labeldirections = ["horizontal", "vertical"], caption = "Figure 4.1: The variation of
susceptible population at various levels of a ", linestyle = [dash, dot, longdash],
color = [green, blue, black, red], legend = ["a = 0", "a= 0.2", "a = .4", "a = .6"]);
```

Maple code for the variation of susceptible population at various levels of a

```
restart;
equ1 diff(S(t),
t)-Lambda+a*S(t)*i(t)+mu*S(t)-c*R(t)+`&sigma;__1`*a*S(t)*i(t)*dB1+`&sigma
```

;__2`*mu*S(t)*dB2; + σ__1 a S(t) i(t) dB1 + σ__2 mu S(t) dB2

equ2 := evalf(subs(i(t) = 2, R(t) = 3, a = 0, b = 0., e = 1.229, mu = 1, c = .23, delta = .5, `&sigma;__1` = 0.38e-1, `&sigma;__2` = .24388, dB1 = .5, dB2 = .2, Lambda = 20, equ1));

equ3IVP := evalf(rhs(dsolve({equ2, S(0) = 10}, S(t))));

D1 := evalf(equ3IVP);

plot(D1, t = 0 .. 500, labels = ["time", "S"], title = "Figure 1: The variation of susceptible population at value a, a=0 ");

equ1 := diff(S(t), t)-Lambda+a*S(t)*i(t)+mu*S(t)-c*R(t)+`&sigma;__1`*a*S(t)*i(t)*dB1+`&sigma;__2`*mu*S(t)*dB2;

+ σ__1 a S(t) i(t) dB1 + σ__2 mu S(t) dB2

equ2 := evalf(subs(i(t) = 2, R(t) = 3, a = .4, b = 0., e = 1.229, mu = 1, c = .23, delta = .5, ` &sigma;__1` = 0.38e-1, `&sigma;__2` = .24388, dB1 = .5, dB2 = .2, Lambda = 20, equ1));

equ3IVP := evalf(rhs(dsolve({equ2, S(0) = 10}, S(t))));

D2 := evalf(equ3IVP);

plot(D2, t = 0 .. 10, labels = ["time", "S"], title = "Figure 1: The variation of susceptible population at value a , a ");

equ1 := diff(S(t), t)-Lambda+a*S(t)*i(t)+mu*S(t)-c*R(t)+`&sigma;__1`*a*S(t)*i(t)*dB1+`&sigma;__2`*mu*S(t)*dB2

```
 |--- S(t)| - Lambda + a S(t) i(t) + mu S(t) - c R(t)
 + &sigma;__1 a S(t) i(t) dB1 + &sigma;__2 mu S(t) dB2
equ2 := evalf(subs(i(t) = 2, R(t) = 3, a = .6, b = 0., e = 1.229, mu = 1, c = .23, delta
= .5, `&sigma;__1` = 0.38e-1, `&sigma;__2` = .24388, dB1 = .5, dB2 = .2, Lambda
= 20, equ1));
 |--- S(t)| - 20.69 + 2.271576 S(t)
equ3IVP := evalf(rhs(dsolve({equ2, S(0) = 10}, S(t))));
D3 := evalf(equ3IVP);
plot(D3, t = 0 .. 10, labels = ["time", "S"], title = "Figure 1: The variation of
susceptible population at value a, a ");
equ1 diff(S(t),
t)-Lambda+a*S(t)*i(t)+mu*S(t)-c*R(t)+`&sigma;__1`*a*S(t)*i(t)*dB1+`&sigma
;__2`*mu*S(t)*dB2
 |--- S(t)| - Lambda + a S(t) i(t) + mu S(t) - c R(t)
      + &sigma;__1 a S(t) i(t) dB1 + &sigma;__2 mu S(t) dB2
equ2 := evalf(subs(i(t) = 2, R(t) = 3, a = .8, b = 0., e = 1.229, mu = 1, c = .23, delta
= .5, `&sigma;__1` = 0.38e-1, `&sigma;__2` = .24388, dB1 = .5, dB2 = .2, Lambda
= 20, equ1));
equ3IVP := evalf(rhs(dsolve({equ2, S(0) = 10}, S(t))));
D4 := evalf(equ3IVP);
plot(D4, t = 0 .. 10, labels = ["time", "S"], title = "Figure 1: The variation of
susceptible population at value a , a ");
plot([D1, D2, D3, D4], t = 0 .. 10, labels = ["time", "Susceptible*Humans*[S]"],
```

labeldirections = ["horizontal", "vertical"], caption = "Figure 1a: The variation of susceptible population at various levels of a using stochastic model ", linestyle = [dash, dot, longdash], color = [green, blue, black, red], legend = ["a = 0", "a= 0.4", "a = .6", "a = .8"]);

Maple code for the stochastic variation of the infected humans

```
restart;

equ1 := diff(i(t), t)-delta*E(t)+b*i(t)+e*i(t)+mu*i(t)-`&sigma;__4`*delta*E(t)*dB4+`&sigma;__3`*b*i(t)*dB3+`&sigma;__2`*mu*i(t)*dB2;
      / d         \
      |--- i(t)| - delta E(t) + b i(t) + e i(t) + mu i(t)
      \ dt        /

          - &sigma;__4 delta E(t) dB4 + &sigma;__3 b i(t) dB3

          + &sigma;__2 mu i(t) dB2
equ2 := evalf(subs(E(t) = 10, a = 0, b = 0., e = 1.229, mu = 1, c = 0.1e-1, delta = 0., `&sigma;__1` = 0.38e-1, `&sigma;__2` = 5.24388, `&sigma;__3` = .269304, `&sigma;__4` = .630522, dB1 = 0.5e-1, dB2 = 0.2e-2, dB3 = 0.75e-3, dB4 = .25, Lambda = 20, equ1));
                      / d         \
```

```
                    |--- i(t)| + 2.23948776 i(t)
                    \ dt       /
equ3IVP := evalf(rhs(dsolve({equ2, i(0) = 4}, i(t))));
                              4. exp(-2.239487760 t)
D1 := evalf(equ3IVP);
                              4. exp(-2.239487760 t)
plot(D1, t = 0 .. 10, labels = ["time", "E"], title = "Figure 13: The variation of
infected individuals at value of b , b=0 ");

equ1                                    :=                                    diff(i(t),
t)-delta*E(t)+b*i(t)+e*i(t)+mu*i(t)-`&sigma;__4`*delta*E(t)*dB4+`&sigma;__3`
*b*i(t)*dB3+`&sigma;__2`*mu*i(t)*dB2;
    / d        \
    |--- i(t)| - delta E(t) + b i(t) + e i(t) + mu i(t)
    \ dt       /

        - &sigma;__4 delta E(t) dB4 + &sigma;__3 b i(t) dB3

        + &sigma;__2 mu i(t) dB2
equ2 := evalf(subs(E(t) = 10, S(t) = 50, a = 0, b = .2, e = 1.229, mu = 1, c = 0.1e-1,
delta = .4, `&sigma;__1` = 0.38e-1, `&sigma;__2` = 5.24388, `&sigma;__3` =
.269304, `&sigma;__4` = .630522, dB1 = 0.5e-1, dB2 = 0.2e-1, dB3 = 0.75e-3, dB4
= .25, Lambda = 20, equ1));
```

```
/ d          \
|--- i(t)| - 4.630522000 + 2.533917996 i(t)
\ dt         /
```

```
equ3IVP := evalf(rhs(dsolve({equ2, i(0) = 4}, i(t))));
```

```
1.827415886 + 2.172584114 exp(-2.533917996 t)
```

```
D2 := evalf(equ3IVP);
```

```
1.827415886 + 2.172584114 exp(-2.533917996 t)
```

```
plot(D2, t = 0 .. 10, labels = ["time", "E"], title = "Figure 2: The variation of Infected
population    for value , a ");
```

```
equ1                    :=                    diff(i(t),
t)-delta*E(t)+b*i(t)+e*i(t)+mu*i(t)-`&sigma;__4`*delta*E(t)*dB4+`&sigma;__3`
*b*i(t)*dB3+`&sigma;__2`*mu*i(t)*dB2;
```

```
/ d          \
|--- i(t)| - delta E(t) + b i(t) + e i(t) + mu i(t)
\ dt         /
```

```
- &sigma;__4 delta E(t) dB4 + &sigma;__3 b i(t) dB3
```

```
+ &sigma;__2 mu i(t) dB2
```

```
equ2 := evalf(subs(E(t) = 10, S(t) = 50, a = 0, b = .4, e = 1.229, mu = 1, c = 0.1e-1,
delta = .6, `&sigma;__1` = 0.38e-1, `&sigma;__2` = 5.24388, `&sigma;__3` =
```

.269304, `&sigma;__4` = 0.30522e-1, dB1 = 0.5e-1, dB2 = 0.2e-1, dB3 = .75, dB4 = .25, Lambda = 20, equ1));

$$\left|\frac{d}{dt} i(t)\right| - 6.045783000 + 2.814668800\, i(t)$$

equ3IVP := evalf(rhs(dsolve({equ2, i(0) = 4}, i(t))));

$$2.147955383 + 1.852044617 \exp(-2.814668800\, t)$$

D3 := evalf(equ3IVP);

$$2.147955383 + 1.852044617 \exp(-2.814668800\, t)$$

plot(D3, t = 0 .. 10, labels = ["time", "E"], title = "Figure 3: The variation of Infected population for value , a ");

equ1 := diff(i(t), t)-delta*E(t)+b*i(t)+e*i(t)+mu*i(t)-`&sigma;__4`*delta*E(t)*dB4+`&sigma;__3`*b*i(t)*dB3+`&sigma;__2`*mu*i(t)*dB2;

$$\left|\frac{d}{dt} i(t)\right| - \text{delta}\, E(t) + b\, i(t) + e\, i(t) + \text{mu}\, i(t)$$

$$- \&\text{sigma};\_\_4\ \text{delta}\ E(t)\ dB4 + \&\text{sigma};\_\_3\ b\ i(t)\ dB3$$

$$+ \&\text{sigma};\_\_2\ \text{mu}\ i(t)\ dB2$$

```
equ2 := evalf(subs(E(t) = 10, S(t) = 50, a = 0, b = .6, e = 1.229, mu = 1, c = 0.1e-1,
delta = .8, `&sigma;__1` = 0.38e-1, `&sigma;__2` = 5.24388, `&sigma;__3` =
.269304, `&sigma;__4` = 0.30522e-1, dB1 = 0.5e-1, dB2 = .2, dB3 = .75, dB4 =
.25, Lambda = 20, equ1));
                / d         \
                |--- i(t)| - 8.061044000 + 3.998962800 i(t)
                \ dt        /

equ3IVP := evalf(rhs(dsolve({equ2, i(0) = 4}, i(t))));
                2.015783693 + 1.984216307 exp(-3.998962800 t)

D4 := evalf(equ3IVP);
                2.015783693 + 1.984216307 exp(-3.998962800 t)
plot(D4, t = 0 .. 10, labels = ["time", "E"], title = "Figure 3: The variation of Infected
population   for value , a ");
plot([D1, D2, D3, D4], t = 0 .. 10, labels = ["time"*no*of*days,
"Infected*Humans*[I]"], labeldirections = ["horizontal", "vertical"], linestyle =
[dash, dot, longdash], color = [green, blue, black, red], legend = ["delta = 0.",
"&delta;= 0.4", "delta = .6", "delta = .8"]);
```

Maple code for the effect of treatment on the exposed individuals

```
restart;
equ1 := diff(E(t), t)-a*S(t)*i(t)+(mu+b+delta)*E(t);
              / d         \
```

```
|--- E(t)| - a S(t) i(t) + (mu + b + delta) E(t)
\ dt       /
```

```
equ2 := evalf(subs(i(t) = 2, S(t) = 10, a = .3, b = 0., e = 1.229, mu = 1, c = 0.1e-1,
delta = .5, Lambda = 20, equ1));
```

```
/ d         \
|--- E(t)| - 6.0 + 1.5 E(t)
\ dt       /
```

```
equ3IVP := evalf(rhs(dsolve({equ2, E(0) = 4}, E(t))));
```

4.

```
D1 := evalf(equ3IVP);
```

4.

```
plot(D1, t = 0 .. 10, labels = ["time", "E"], title = "Figure 1: The variation of
proportion of exposed human population    for value , a=0 ");
equ1 := diff(E(t), t)-a*S(t)*i(t)+(mu+b+delta)*E(t);
```

```
/ d         \
|--- E(t)| - a S(t) i(t) + (mu + b + delta) E(t)
\ dt       /
```

```
equ2 := evalf(subs(i(t) = 2, S(t) = 10, a = .3, b = .4, e = 1.229, mu = 1, c = 0.1e-1,
delta = .5, Lambda = 20, equ1));
```

```
/ d         \
|--- E(t)| - 6.0 + 1.9 E(t)
\ dt       /
```

```
equ3IVP := evalf(rhs(dsolve({equ2, E(0) = 4}, E(t))));

        3.157894737 + 0.8421052632 exp(-1.900000000 t)

D2 := evalf(equ3IVP);

        3.157894737 + 0.8421052632 exp(-1.900000000 t)

plot(D2, t = 0 .. 10, labels = ["time", "E"], title = "Figure 1: The variation of
proportion of exposed human population    for value , a ");

equ1 := diff(E(t), t)-a*S(t)*i(t)+(mu+b+delta)*E(t);

        / d         \
        |--- E(t)| - a S(t) i(t) + (mu + b + delta) E(t)
        \ dt        /

equ2 := evalf(subs(i(t) = 2, S(t) = 10, a = .3, b = .6, e = 1.229, mu = 1, c = 0.1e-1,
delta = .5, Lambda = 20, equ1));

                / d         \
                |--- E(t)| - 6.0 + 2.1 E(t)
                \ dt        /


equ3IVP := evalf(rhs(dsolve({equ2, E(0) = 4}, E(t))));

        2.857142857 + 1.142857143 exp(-2.100000000 t)


D3 := evalf(equ3IVP);

        2.857142857 + 1.142857143 exp(-2.100000000 t)

plot(D3, t = 0 .. 10, labels = ["time", "E"], title = "Figure 1: The variation of
proportion of exposed human population    for value , a ");
```

```
equ1 := diff(E(t), t)-a*S(t)*i(t)+(mu+b+delta)*E(t);
```

$$\frac{d}{dt} E(t) - a\,S(t)\,i(t) + (mu + b + delta)\,E(t)$$

```
equ2 := evalf(subs(i(t) = 2, S(t) = 10, a = 0., b = .8, e = 1.229, mu = 1, c = 0.1e-1,
delta = .5, Lambda = 20, equ1));
```

$$\frac{d}{dt} E(t) + 2.3\,E(t)$$

```
equ3IVP := evalf(rhs(dsolve({equ2, E(0) = 4}, E(t))));
```

$$4.\exp(-2.300000000\,t)$$

```
D4 := evalf(equ3IVP);
```

$$4.\exp(-2.300000000\,t)$$

```
plot(D4, t = 0 .. 10, labels = ["time", "E"], title = "Figure 1: The variation of
proportion of exposed human population  for value , a ");
plot([D1, D2, D3, D4], t = 0 .. 10, labels = [("time")(no*of*days),
"ExposedHumans*[E]"], labeldirections = ["horizontal", "vertical"], linestyle =
[dash, dot, longdash], color = [green, blue, black, red], legend = ["b = 0", "b= 0.4",
"b = .6", "b = .8"]);
```

Maple code for the effect of treatment on the infected humans

```
restart;
equ1 := diff(i(t), t)-delta*E(t)+(b+e+mu)*i(t);
```

```
                / d         \
                |--- i(t)| - delta E(t) + (b + e + mu) i(t)
                \ dt        /
equ2 := evalf(subs(S(t) = 20, E(t) = 10, a = .3, b = 0., e = 1.229, mu = 1, c = 0.1e-1,
delta = .5, Lambda = 20, equ1));
                        / d         \
                        |--- i(t)| - 5.0 + 2.229 i(t)
                        \ dt        /
equ3IVP := evalf(rhs(dsolve({equ2, i(0) = 0}, i(t))));
                2.243158367 - 2.243158367 exp(-2.229000000 t)
D1 := evalf(equ3IVP);
                2.243158367 - 2.243158367 exp(-2.229000000 t)
plot(D1, t = 0 .. 10, labels = ["time", "i"], title = "Figure 9: The Effect of treatment
on the infected   population   for value , b=0 ");
equ1 := diff(i(t), t)-delta*E(t)+(b+e+mu)*i(t);
                / d         \
                |--- i(t)| - delta E(t) + (b + e + mu) i(t)
                \ dt        /
equ2 := evalf(subs(S(t) = 20, E(t) = 10, a = .3, b = .4, e = 1.229, mu = 1, c = 0.1e-1,
delta = .5, Lambda = 20, equ1));
                        / d         \
                        |--- i(t)| - 5.0 + 2.629 i(t)
                        \ dt        /
```

```
equ3IVP := evalf(rhs(dsolve({equ2, i(0) = 0}, i(t))));

        1.901863827 - 1.901863827 exp(-2.629000000 t)

D2 := evalf(equ3IVP);

        1.901863827 - 1.901863827 exp(-2.629000000 t)

plot(D2, t = 0 .. 10, labels = ["time", "i"], title = "Figure 9: The effect of treatment on
the infected individuals for value , i");

equ1 := diff(i(t), t)-delta*E(t)+(b+e+mu)*i(t);

          / d         \
          |--- i(t)| - delta E(t) + (b + e + mu) i(t)
          \ dt        /

equ2 := evalf(subs(S(t) = 20, E(t) = 10, a = .3, b = .6, e = 1.229, mu = 1, c = 0.1e-1,
delta = .5, Lambda = 20, equ1));

                  / d         \
                  |--- i(t)| - 5.0 + 2.829 i(t)
                  \ dt        /

equ3IVP := evalf(rhs(dsolve({equ2, i(0) = 0}, i(t))));

        1.767408978 - 1.767408978 exp(-2.829000000 t)

D3 := evalf(equ3IVP);

        1.767408978 - 1.767408978 exp(-2.829000000 t)

plot(D3, t = 0 .. 10, labels = ["time", "i"], title = "Figure 9: the effect of treatment on
the infected individuals    for value , i");

equ1 := diff(i(t), t)-delta*E(t)+(b+e+mu)*i(t);

          / d         \
```

```
        |--- i(t)| - delta E(t) + (b + e + mu) i(t)
        \ dt       /
equ2 := evalf(subs(S(t) = 20, E(t) = 10, a = .3, b = .8, e = 1.229, mu = 1, c = 0.1e-1,
delta = .5, Lambda = 20, equ1));
                    / d        \
                    |--- i(t)| - 5.0 + 3.029 i(t)
                    \ dt       /

equ3IVP := evalf(rhs(dsolve({equ2, i(0) = 0}, i(t))));
          1.650709805 - 1.650709805 exp(-3.029000000 t)

D4 := evalf(equ3IVP);
          1.650709805 - 1.650709805 exp(-3.029000000 t)
plot(D4, t = 0 .. 10, labels = ["time", "i"], title = "Figure 9: The effect of treatment on
the infected individuals    for value , i");
plot([D1, D2, D3, D4], t = 0 .. 10, labels = [("time")(no*of*days),
"Infected*Humans*[i]"], labeldirections = ["horizontal", "vertical"], linestyle =
[dash, dot, longdash], color = [green, blue, black, red], legend = ["b = 0", "b= 0.4",
"b = .6", "b = .8"])
```

Maple code for the bifurcation diagram

```
restart;
with(plots);
print(`output redirected...`); # input placeholder
```

```
with(DEtools);
print(`output redirected...`); # input placeholder
f                                                                                    :=
(a*delta*c(delta*b+b*(b+e+mu))-a*delta*(c+mu)*(b+mu+delta)*(b+e+mu))*i^2
+mu*delta*(c+mu)*(b+mu+delta)*(b+e+mu)*(R[0]-1)*i;
print(`output redirected...`); # input placeholder
(a delta c(delta b + b (b + e + mu))- a delta (c + mu) (b + mu + delta) (b + e + mu))
i
 + mu delta (c + mu) (b + mu + delta) (b + e + mu) (R[0] - 1) i
print(??); # input placeholder
g := evalf(subs(a = .4, b = 0.5e-2, e = 1.229, mu = 1, c = 0.1e-1, delta = .5, f));
print(`output redirected...`); # input placeholder
EQ1 := g = 0;
print(`output redirected...`); # input placeholder
      print(??); # input placeholder
EQ2 := diff(g, i[m]) = 0;
print(`output redirected...`); # input placeholder
 print(??); # input placeholder
EQ2 := factor(EQ2);
print(`output redirected...`); # input placeholder
print(??); # input placeholder
BIFpt := solve({EQ1, EQ2}, {i, R[0]});
print(`output redirected...`); # input placeholder
```

{i = 0., R[0] = R[0]},

print(??); # input placeholder

BIFdiag := implicitplot(g = 0, R[0] = 0 .. 1, i = -0.1e-1 .. 0., linestyle = solid, color = green, thickness = 3);

print(`output redirected...`); # input placeholder

PLOT(CURVES(Array(1..25, 1..2, {(1, 1) = .0, (1, 2) = .0, (2,

print(`output redirected...`); # input placeholder

display(BIFdiag, BIFdiag1, BIFdiag3, labels = ["Reproduction*Number*[R[0]]", "Infected*Humans*with*the*disease*[I]"], labeldirections = ["horizontal", "vertical"], caption = "Figure 6: The bifurcation diagram for infectious disease with parameter values ");

Maple code for the stochastic variation of the population against time

$$dequ1 := \frac{d}{dt}S(t) - \Lambda + a\cdot S(t)\cdot i(t) + \mu\cdot S(t) - c\cdot R(t) + \sigma_1\cdot a\cdot S(t)\cdot i(t)\cdot dB1 + \sigma_2\cdot\mu\cdot S(t)\cdot dB2, \frac{d}{dt}E(t) - a\cdot S(t)\cdot i(t) + (\mu + b + \delta)\cdot E(t) - \sigma_1\cdot a\cdot S(t)\cdot i(t)\cdot dB1 + \sigma_2\cdot\mu\cdot S(t)\cdot dB2 + \sigma_3\cdot b\cdot E(t)\cdot dB3 + \sigma_4\cdot\delta\cdot E(t)\cdot dB4, \frac{d}{dt}i(t) - \delta\cdot E(t) + b\cdot i(t) + e\cdot i(t) + \mu\cdot i(t) - \sigma_4\cdot\delta\cdot E(t)\cdot dB4 + \sigma_3\cdot b\cdot i(t)\cdot dB3 + \sigma_2\cdot\mu\cdot i(t)\cdot dB2, \frac{d}{dt}R(t) - b\cdot i(t) - b\cdot E(t) + \mu\cdot R(t) + c\cdot R(t) - \sigma_3\cdot b\cdot E(t)\cdot dB3 - \sigma_3\cdot b\cdot i(t)\cdot dB3 - \sigma_2\cdot\mu\cdot R(t)\cdot dB2$$

$$\frac{d}{dt}S(t) - \Lambda + a\,S(t)\,i(t) + \mu\,S(t) - c\,R(t) + \sigma_1\,a\,S(t)\,i(t)\,dB1 + \sigma_2\,\mu\,S(t)\,dB2, \frac{d}{dt}E(t) - a\,S(t)\,i(t) + (\mu + b + \delta)\,E(t) - \sigma_1\,a\,S(t)\,i(t)\,dB1 + \sigma_2\,\mu\,S(t)\,dB2 + \sigma_3\,b\,E(t)\,dB3 + \sigma_4\,\delta\,E(t)\,dB4, \frac{d}{dt}i(t) - \delta\,E(t) + b\,i(t) + e\,i(t) + \mu\,i(t) - \sigma_4\,\delta\,E(t)\,dB4 + \sigma_3\,b\,i(t)\,dB3 + \sigma_2\,\mu\,i(t)\,dB2, \frac{d}{dt}R(t) - b\,i(t) - b\,E(t) + \mu\,R(t) + c\,R(t) - \sigma_3\,b\,E(t)\,dB3 - \sigma_3\,b\,i(t)\,dB3 - \sigma_2\,\mu\,R(t)\,dB2$$

$$deq := eval\left(dequ1, \left\{a = 0.0165, b = 0.27, e = 0.73, \mu = 0.0001, c = 0.23, \delta = 0.0005, \sigma_1 = 1.0, \sigma_2 = 1.0, \sigma_3 = 1.0, \sigma_4 = 1.630522, dB1 = 0.09, dB2 = 0.002, dB3 = 0.00075, dB4 = 0.0025, \Lambda = 20\right\}\right)$$

$$\frac{d}{dt}S(t) - 20 + 0.0179850\,S(t)\,i(t) + 0.00010020\,S(t) - 0.23\,R(t), \frac{d}{dt}E(t)$$
$$- 0.0179850\,S(t)\,i(t) + 0.2708045382\,E(t) + 2.0\,10^{-7}\,S(t), \frac{d}{dt}i(t)$$
$$- 0.0005020381525\,E(t) + 1.00030270\,i(t), \frac{d}{dt}R(t) - 0.27020250\,i(t)$$
$$- 0.27020250\,E(t) + 0.23009980\,R(t)$$

$ic1 := S(0) = 21950, i(0) = 50, R(0) = 0, E(0) = 100$

$S(0) = 21950, i(0) = 50, R(0) = 0, E(0) = 100$

$dsol1 := dsolve(\{deq, ic1\}, numeric, output = array([0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50]));$

```
> with(plots): sys := deq;
fcns := {S(t) , E(t) , i(t) , R(t) }:
p:= dsolve({sys,S(0) = 21950, i(0) = 50, R(0) = 0, E(0) =
100},fcns,type=numeric,method=classical):
```

$$sys := \frac{d}{dt}S(t) - 20 + 0.0179850\,S(t)\,i(t) + 0.00010020\,S(t) - 0.23\,R(t), \frac{d}{dt}E(t)$$
$$- 0.0179850\,S(t)\,i(t) + 0.2708045382\,E(t) + 2.0\,10^{-7}\,S(t), \frac{d}{dt}i(t)$$
$$- 0.0005020381525\,E(t) + 1.00030270\,i(t), \frac{d}{dt}R(t) - 0.27020250\,i(t)$$
$$- 0.27020250\,E(t) + 0.23009980\,R(t)$$

```
> odeplot(p,
[[t,S(t)],[t,E(t)],[t,i(t)],[t,R(t)]],0..30,titlefont =
["ROMAN", 15], labelfont = ["ROMAN", 20],labels = ["time
(months) ", "S,E,i,R "], labeldirections = ["horizontal",
"horizontal"],linestyle=[solid,solid,solid,solid],color=[gr
een,yellow,red,blue],legend = [ "S(t) ","E(t) ","i(t) ","R(t)
"],legendstyle = [font = ["HELVETICA", 20], location =
right]);;;

>
```

Maple code for the optimal control using one control strategy

restart; unprotect(Pi);

a := 0.318e-3; b := 0.175e-1; e := .576; mu := 0.5e-1; c := 0.1e-1; delta := .8; Lambda := 20; m[1] := .1; m[2] := .2; m[3] := .4; m[4] := .1; u[1][0] := 0; u[2][0] := 0; N := 0.1e-1; Q := 900; P1[100] := 0; P2[100] := 0; P3[100] := 0; P4[100] := 0; S[0] := 460;

```
E[0] := 30; i[0] := 12; R[0] := 0; h := 0.1e-2;


    for n from 0 to 100 do S[n+1] :=
    S[n]+h*(Lambda-(1-u[1][n]-u[2][n])*a*i[n]*S[n]-mu*S[n]+c*R[n]); E[n+1] :=
    E[n]+h*((1-u[1][n]-u[2][n])*a*i[n]*S[n+1]-(u[1][n]+u[2][n]+mu+delta)*E[n]);
    i[n+1] := i[n]+h*(delta*E[n+1]-(u[1][n]+e+mu)*i[n]); R[n+1] :=
    R[n]+h*(u[1][n]*i[n+1]+u[1][n]*E[n+1]+u[2][n]*E[n+1]-(c+mu)*R[n]);
    P1[100-n-1] :=
    P1[100-n]+h*((P1[100-n]-P2[100-n])*(1-u[1][n]-u[2][n])*a*i[n+1]-mu*P1[100-n
    ]); P2[100-n-1] :=
    P2[100-n]+h*(P2[100-n]*(u[1][n]+u[2][n]+mu+delta)-P4[100-n]*(u[1][n]+u[2][n
    ])-P3[100-n]*delta-Q); P3[100-n-1] :=
    P3[100-n]+h*(P3[100-n]*(u[1][n]+e+mu)-P4[100-n]*u[1][n]-N); P4[100-n-1] :=
    P4[100-n]+h*(P4[100-n]*(c+mu)-P1[100-n-1]*c); A[1][n] :=
    -(1/2)*((P2[100-n-1]-P1[100-n-1])*a*S[n+1]*i[n+1]+(P2[100-n-1]-P4[100-n-1])*
    E[n+1]+(P3[100-n-1]-P4[100-n-1])*i[n+1])/m[3]; A[2][n] :=
    -(1/2)*((P2[100-n-1]-P1[100-n-1])*a*S[n+1]*i[n+1]+(P2[100-n-1]-P4[100-n-1])*
    E[n+1])/m[4]; u[1][n+1] := min(1, max(0, A[1][n])); u[2][n+1] := 0 end do;
    S[0] := 460; E[0] := 30; i[0] := 12; R[0] := 0;
    C1 := [seq(S[n], n = 0 .. 100)]; C2 := [seq(E[n], n = 0 .. 100)]; C3 := [seq(i[n], n = 0 ..
    100)]; C4 := [seq(R[n], n = 0 .. 100)];
    with(Statistics); A1 := PointPlot(C1, color = green, thickness = 1, symbol =
    default);
    %;
    PLOT(CURVES([[1., 460.], [2., 459.9952446], [3., 459.9922448],

      [4., 459.9892456], [5., 459.9862470], [6., 459.9832489],

      [7., 459.9802514], [8., 459.9772545], [9., 459.9742581],

      [10., 459.9712623], [11., 459.9682671], [12., 459.9652724],

      [13., 459.9622783], [14., 459.9592848], [15., 459.9562918],

      [16., 459.9532994], [17., 459.9503076], [18., 459.9473163],

      [19., 459.9443256], [20., 459.9413354], [21., 459.9383458],

      [22., 459.9353568], [23., 459.9323683], [24., 459.9293804],
```

[25., 459.9263930], [26., 459.9234062], [27., 459.9204199],

[28., 459.9174342], [29., 459.9144491], [30., 459.9114645],

[31., 459.9084805], [32., 459.9054970], [33., 459.9025141],

[34., 459.8995317], [35., 459.8965499], [36., 459.8935686],

[37., 459.8905879], [38., 459.8876077], [39., 459.8846281],

[40., 459.8816490], [41., 459.8786705], [42., 459.8756925],

[43., 459.8727151], [44., 459.8697382], [45., 459.8667619],

[46., 459.8637861], [47., 459.8608109], [48., 459.8578362],

[49., 459.8548621], [50., 459.8518885], [51., 459.8489154],

[52., 459.8459429], [53., 459.8429709], [54., 459.8399995],

[55., 459.8370286], [56., 459.8340583], [57., 459.8310885],

[58., 459.8281192], [59., 459.8251505], [60., 459.8221823],

[61., 459.8192146], [62., 459.8162475], [63., 459.8132809],

[64., 459.8103149], [65., 459.8073494], [66., 459.8043844],

[67., 459.8014200], [68., 459.7984561], [69., 459.7954927],

[70., 459.7925299], [71., 459.7895676], [72., 459.7866058],

[73., 459.7836446], [74., 459.7806839], [75., 459.7777237],

[76., 459.7747641], [77., 459.7718050], [78., 459.7688464],

[79., 459.7658883], [80., 459.7629308], [81., 459.7599738],

[82., 459.7570173], [83., 459.7540614], [84., 459.7511060],

```
   [85., 459.7481511], [86., 459.7451967], [87., 459.7422429],

   [88., 459.7392896], [89., 459.7363368], [90., 459.7333845],

   [91., 459.7304327], [92., 459.7274815], [93., 459.7245308],

   [94., 459.7215806], [95., 459.7186309], [96., 459.7156818],

   [97., 459.7127332], [98., 459.7097851], [99., 459.7068375],

   [100., 459.7038904], [101., 459.7009438]],

   COLOUR(RGB, 0., 1.00000000, 0.),

   LEGEND("__never_display_this_legend_entry")), STYLE(POINT),

   SYMBOL(DEFAULT), THICKNESS(1))
with(Statistics); A2 := PointPlot(C2, color = green, thickness = 1, symbol =
default);
print(`output redirected...`); # input placeholder
  PLOT(CURVES([[1., 30.], [2., 29.97625534], [3., 29.92079927],

    [4., 29.86544579], [5., 29.81019472], [6., 29.75504586],

    [7., 29.69999903], [8., 29.64505403], [9., 29.59021068],

    [10., 29.53546879], [11., 29.48082817], [12., 29.42628864],

    [13., 29.37185001], [14., 29.31751209], [15., 29.26327469],

    [16., 29.20913763], [17., 29.15510073], [18., 29.10116379],

    [19., 29.04732664], [20., 28.99358909], [21., 28.93995095],

    [22., 28.88641204], [23., 28.83297218], [24., 28.77963118],

    [25., 28.72638886], [26., 28.67324504], [27., 28.62019954],

    [28., 28.56725217], [29., 28.51440275], [30., 28.46165110],
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[31., 28.40899705], [32., 28.35644041], [33., 28.30398100],

[34., 28.25161864], [35., 28.19935315], [36., 28.14718435],

[37., 28.09511206], [38., 28.04313610], [39., 27.99125630],

[40., 27.93947248], [41., 27.88778446], [42., 27.83619206],

[43., 27.78469510], [44., 27.73329341], [45., 27.68198682],

[46., 27.63077514], [47., 27.57965821], [48., 27.52863584],

[49., 27.47770786], [50., 27.42687410], [51., 27.37613438],

[52., 27.32548853], [53., 27.27493638], [54., 27.22447775],

[55., 27.17411247], [56., 27.12384036], [57., 27.07366126],

[58., 27.02357499], [59., 26.97358138], [60., 26.92368025],

[61., 26.87387144], [62., 26.82415478], [63., 26.77453009],

[64., 26.72499721], [65., 26.67555597], [66., 26.62620619],

[67., 26.57694771], [68., 26.52778036], [69., 26.47870397],

[70., 26.42971837], [71., 26.38082339], [72., 26.33201887],

[73., 26.28330464], [74., 26.23468053], [75., 26.18614637],

[76., 26.13770200], [77., 26.08934725], [78., 26.04108196],

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[82., 25.84891204], [83., 25.80109155], [84., 25.75335953],

[85., 25.70571581], [86., 25.65816024], [87., 25.61069264],

[88., 25.56331286], [89., 25.51602073], [90., 25.46881609],

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    [94., 25.28086921], [95., 25.23409960], [96., 25.18741652],

    [97., 25.14081980], [98., 25.09430928], [99., 25.04788481],

    [100., 25.00154622], [101., 24.95529336]],

    COLOUR(RGB, 0., 1.00000000, 0.),

    LEGEND("__never_display_this_legend_entry")), STYLE(POINT),

    SYMBOL(DEFAULT), THICKNESS(1))
with(Statistics); A3 := PointPlot(C3, color = green, thickness = 1, symbol =
default);
print(`output redirected...`); # input placeholder
  PLOT(CURVES([[1., 12.], [2., 12.01646900], [3., 12.02086686],

    [4., 12.02521329], [5., 12.02950845], [6., 12.03375251],

    [7., 12.03794563], [8., 12.04208797], [9., 12.04617970],

    [10., 12.05022099], [11., 12.05421199], [12., 12.05815287],

    [13., 12.06204379], [14., 12.06588492], [15., 12.06967641],

    [16., 12.07341843], [17., 12.07711113], [18., 12.08075468],

    [19., 12.08434923], [20., 12.08789495], [21., 12.09139199],

    [22., 12.09484052], [23., 12.09824069], [24., 12.10159266],

    [25., 12.10489658], [26., 12.10815261], [27., 12.11136091],

    [28., 12.11452164], [29., 12.11763495], [30., 12.12070100],

    [31., 12.12371994], [32., 12.12669192], [33., 12.12961710],

    [34., 12.13249564], [35., 12.13532768], [36., 12.13811338],
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[37., 12.14085290], [38., 12.14354638], [39., 12.14619398],

[40., 12.14879585], [41., 12.15135214], [42., 12.15386300],

[43., 12.15632857], [44., 12.15874901], [45., 12.16112447],

[46., 12.16345510], [47., 12.16574105], [48., 12.16798246],

[49., 12.17017949], [50., 12.17233228], [51., 12.17444098],

[52., 12.17650573], [53., 12.17852668], [54., 12.18050398],

[55., 12.18243777], [56., 12.18432820], [57., 12.18617541],

[58., 12.18797955], [59., 12.18974076], [60., 12.19145919],

[61., 12.19313497], [62., 12.19476826], [63., 12.19635919],

[64., 12.19790791], [65., 12.19941456], [66., 12.20087928],

[67., 12.20230221], [68., 12.20368349], [69., 12.20502326],

[70., 12.20632167], [71., 12.20757885], [72., 12.20879494],

[73., 12.20997008], [74., 12.21110441], [75., 12.21219807],

[76., 12.21325120], [77., 12.21426393], [78., 12.21523640],

[79., 12.21616875], [80., 12.21706111], [81., 12.21791363],

[82., 12.21872643], [83., 12.21949965], [84., 12.22023343],

[85., 12.22092790], [86., 12.22158320], [87., 12.22219946],

[88., 12.22277681], [89., 12.22331539], [90., 12.22381533],

[91., 12.22427677], [92., 12.22469983], [93., 12.22508465],

[94., 12.22543136], [95., 12.22574009], [96., 12.22601097],

```
    [97., 12.22624413], [98., 12.22643970], [99., 12.22659782],

    [100., 12.22671861], [101., 12.22680220]],

    COLOUR(RGB, 0., 1.00000000, 0.),

    LEGEND("__never_display_this_legend_entry")), STYLE(POINT),

    SYMBOL(DEFAULT), THICKNESS(1))
with(Statistics); A4 := PointPlot(C4, color = green, thickness = 1, symbol =
default);
%;
PLOT(CURVES([[1., 0.], [2., 0.], [3., 0.04194166613],

   [4., 0.08382980871], [5., 0.1256644821], [6., 0.1674457406],

   [7., 0.2091736385], [8., 0.2508482301], [9., 0.2924695696],

   [10., 0.3340377112], [11., 0.3755527091], [12., 0.4170146174],

   [13., 0.4584234903], [14., 0.4997793819], [15., 0.5410823462],

   [16., 0.5823324373], [17., 0.6235297092], [18., 0.6646742159],

   [19., 0.7057660113], [20., 0.7468051494], [21., 0.7877916840],

   [22., 0.8287256691], [23., 0.8696071584], [24., 0.9104362058],

   [25., 0.9512128651], [26., 0.9919371900], [27., 1.032609234],

   [28., 1.073229051], [29., 1.113796695], [30., 1.154312219],

   [31., 1.194775677], [32., 1.235187123], [33., 1.275546610],

   [34., 1.315854191], [35., 1.356109921], [36., 1.396313852],

   [37., 1.436466038], [38., 1.476566533], [39., 1.516615389],

   [40., 1.556612660], [41., 1.596558400], [42., 1.636452662],
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[43., 1.676295499], [44., 1.716086964], [45., 1.755827110],

[46., 1.795515991], [47., 1.835153659], [48., 1.874740168],

[49., 1.914275571], [50., 1.953759921], [51., 1.993193271],

[52., 2.032575674], [53., 2.071907183], [54., 2.111187850],

[55., 2.150417729], [56., 2.189596872], [57., 2.228725333],

[58., 2.267803164], [59., 2.306830418], [60., 2.345807148],

[61., 2.384733406], [62., 2.423609245], [63., 2.462434718],

[64., 2.501209877], [65., 2.539934775], [66., 2.578609464],

[67., 2.617233997], [68., 2.655808427], [69., 2.694332806],

[70., 2.732807186], [71., 2.771231620], [72., 2.809606160],

[73., 2.847930858], [74., 2.886205767], [75., 2.924430939],

[76., 2.962606426], [77., 3.000732281], [78., 3.038808555],

[79., 3.076835301], [80., 3.114812571], [81., 3.152740417],

[82., 3.190618891], [83., 3.228448045], [84., 3.266227931],

[85., 3.303958601], [86., 3.341640107], [87., 3.379272501],

[88., 3.416855834], [89., 3.454390159], [90., 3.491875527],

[91., 3.529311990], [92., 3.566699600], [93., 3.604038408],

[94., 3.641328466], [95., 3.678569826], [96., 3.715762539],

[97., 3.752906657], [98., 3.790002232], [99., 3.827049314],

[100., 3.864047956], [101., 3.900998209]],

```
   COLOUR(RGB, 0., 1.00000000, 0.),

   LEGEND("__never_display_this_legend_entry")), STYLE(POINT),

   SYMBOL(DEFAULT), THICKNESS(1))
```

```
for n from 0 to 100 do S[n+1] := S[n]+h*(-a*S[n]*i[n]+c*R[n]-mu*S[n]+Lambda);
E[n+1] := E[n]+h*(a*i[n]*S[n+1]-(mu+delta)*E[n]); i[n+1] :=
i[n]+h*(delta*E[n+1]-(e+mu)*i[n]); R[n+1] := R[n]-h*(c+mu)*R[n] end do;
S[0] := 460; E[0] := 30; i[0] := 12; R[0] := 0;
B1 := [seq(S[n], n = 0 .. 100)]; B2 := [seq(E[n], n = 0 .. 100)]; B3 := [seq(i[n], n = 0 ..
100)]; B4 := [seq(R[n], n = 0 .. 100)];
with(Statistics); D1 := PointPlot(B1, color = red, thickness = 1, symbol = default);
%;
PLOT(CURVES([[1., 460.], [2., 459.9952446], [3., 459.9904871],

   [4., 459.9857274], [5., 459.9809656], [6., 459.9762017],

   [7., 459.9714356], [8., 459.9666674], [9., 459.9618971],

   [10., 459.9571246], [11., 459.9523500], [12., 459.9475733],

   [13., 459.9427945], [14., 459.9380136], [15., 459.9332306],

   [16., 459.9284455], [17., 459.9236583], [18., 459.9188690],

   [19., 459.9140776], [20., 459.9092842], [21., 459.9044887],

   [22., 459.8996911], [23., 459.8948915], [24., 459.8900898],

   [25., 459.8852860], [26., 459.8804802], [27., 459.8756723],

   [28., 459.8708624], [29., 459.8660505], [30., 459.8612365],

   [31., 459.8564205], [32., 459.8516025], [33., 459.8467824],
```

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[43., 459.7984715], [44., 459.7936295], [45., 459.7887855],

[46., 459.7839395], [47., 459.7790916], [48., 459.7742417],

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[55., 459.7402381], [56., 459.7353727], [57., 459.7305054],

[58., 459.7256362], [59., 459.7207650], [60., 459.7158919],

[61., 459.7110169], [62., 459.7061400], [63., 459.7012612],

[64., 459.6963805], [65., 459.6914980], [66., 459.6866136],

[67., 459.6817273], [68., 459.6768391], [69., 459.6719491],

[70., 459.6670572], [71., 459.6621634], [72., 459.6572678],

[73., 459.6523703], [74., 459.6474710], [75., 459.6425698],

[76., 459.6376668], [77., 459.6327619], [78., 459.6278552],

[79., 459.6229467], [80., 459.6180363], [81., 459.6131241],

[82., 459.6082101], [83., 459.6032943], [84., 459.5983767],

[85., 459.5934573], [86., 459.5885361], [87., 459.5836131],

[88., 459.5786883], [89., 459.5737617], [90., 459.5688333],

[91., 459.5639031], [92., 459.5589711], [93., 459.5540374],

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   [94., 459.5491019], [95., 459.5441646], [96., 459.5392256],

   [97., 459.5342848], [98., 459.5293422], [99., 459.5243979],

   [100., 459.5194518], [101., 459.5145040]],

   COLOUR(RGB, 1.00000000, 0., 0.),

   LEGEND("__never_display_this_legend_entry")), STYLE(POINT),

   SYMBOL(DEFAULT), THICKNESS(1))
with(Statistics); D2 := PointPlot(B2, color = red, thickness = 1, symbol = default);
print(`output redirected...`); # input placeholder
  PLOT(CURVES([[1., 30.], [2., 29.97625534], [3., 29.95253326],

    [4., 29.92883373], [5., 29.90515672], [6., 29.88150222],

    [7., 29.85787020], [8., 29.83426063], [9., 29.81067350],

    [10., 29.78710878], [11., 29.76356644], [12., 29.74004646],

    [13., 29.71654882], [14., 29.69307350], [15., 29.66962047],

    [16., 29.64618971], [17., 29.62278120], [18., 29.59939491],

    [19., 29.57603082], [20., 29.55268891], [21., 29.52936916],

    [22., 29.50607154], [23., 29.48279602], [24., 29.45954259],

    [25., 29.43631122], [26., 29.41310189], [27., 29.38991458],

    [28., 29.36674926], [29., 29.34360591], [30., 29.32048451],

    [31., 29.29738503], [32., 29.27430745], [33., 29.25125175],

    [34., 29.22821791], [35., 29.20520590], [36., 29.18221570],

    [37., 29.15924729], [38., 29.13630065], [39., 29.11337575],
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[55., 28.74951593], [56., 28.72695721], [57., 28.70441983],

[58., 28.68190377], [59., 28.65940900], [60., 28.63693550],

[61., 28.61448325], [62., 28.59205223], [63., 28.56964242],

[64., 28.54725379], [65., 28.52488632], [66., 28.50253999],

[67., 28.48021478], [68., 28.45791067], [69., 28.43562763],

[70., 28.41336564], [71., 28.39112469], [72., 28.36890474],

[73., 28.34670578], [74., 28.32452779], [75., 28.30237074],

[76., 28.28023461], [77., 28.25811938], [78., 28.23602503],

[79., 28.21395153], [80., 28.19189887], [81., 28.16986702],

[82., 28.14785596], [83., 28.12586567], [84., 28.10389613],

[85., 28.08194732], [86., 28.06001921], [87., 28.03811178],

[88., 28.01622502], [89., 27.99435890], [90., 27.97251340],

[91., 27.95068849], [92., 27.92888416], [93., 27.90710038],

[94., 27.88533714], [95., 27.86359441], [96., 27.84187217],

[97., 27.82017040], [98., 27.79848908], [99., 27.77682819],

[100., 27.75518770], [101., 27.73356760]],

COLOUR(RGB, 1.00000000, 0., 0.),

LEGEND("__never_display_this_legend_entry")), STYLE(POINT),

SYMBOL(DEFAULT), THICKNESS(1))
with(Statistics); D3 := PointPlot(B3, color = red, thickness = 1, symbol = default);
print(`output redirected...`); # input placeholder
PLOT(CURVES([[1., 12.], [2., 12.01646900], [3., 12.03290872],

[4., 12.04931919], [5., 12.06570044], [6., 12.08205251],

[7., 12.09837544], [8., 12.11466927], [9., 12.13093403],

[10., 12.14716975], [11., 12.16337647], [12., 12.17955423],

[13., 12.19570307], [14., 12.21182302], [15., 12.22791412],

[16., 12.24397640], [17., 12.26000990], [18., 12.27601465],

[19., 12.29199069], [20., 12.30793805], [21., 12.32385678],

[22., 12.33974690], [23., 12.35560846], [24., 12.37144148],

[25., 12.38724601], [26., 12.40302208], [27., 12.41876972],

[28., 12.43448897], [29., 12.45017986], [30., 12.46584244],

[31., 12.48147673], [32., 12.49708277], [33., 12.51266060],

[34., 12.52821025], [35., 12.54373176], [36., 12.55922516],

[37., 12.57469048], [38., 12.59012776], [39., 12.60553704],

[40., 12.62091835], [41., 12.63627173], [42., 12.65159721],

[43., 12.66689482], [44., 12.68216461], [45., 12.69740660],

[46., 12.71262083], [47., 12.72780733], [48., 12.74296614],

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[55., 12.84830539], [56., 12.86324392], [57., 12.87815507],

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[61., 12.93752652], [62., 12.95230127], [63., 12.96704884],

[64., 12.98176927], [65., 12.99646259], [66., 13.01112884],

[67., 13.02576805], [68., 13.04038025], [69., 13.05496547],

[70., 13.06952375], [71., 13.08405513], [72., 13.09855964],

[73., 13.11303731], [74., 13.12748817], [75., 13.14191226],

[76., 13.15630961], [77., 13.17068026], [78., 13.18502423],

[79., 13.19934157], [80., 13.21363230], [81., 13.22789646],

[82., 13.24213408], [83., 13.25634520], [84., 13.27052984],

[85., 13.28468805], [86., 13.29881985], [87., 13.31292528],

[88., 13.32700437], [89., 13.34105715], [90., 13.35508366],

[91., 13.36908393], [92., 13.38305799], [93., 13.39700588],

[94., 13.41092762], [95., 13.42482325], [96., 13.43869281],

[97., 13.45253632], [98., 13.46635382], [99., 13.48014535],

[100., 13.49391093], [101., 13.50765060]],

COLOUR(RGB, 1.00000000, 0., 0.),

LEGEND("__never_display_this_legend_entry")), STYLE(POINT),

```
    SYMBOL(DEFAULT), THICKNESS(1))
with(Statistics); D4 := PointPlot(B4, color = red, thickness = 1, symbol = default);
%;
PLOT(CURVES([[1., 0.], [2., 0.], [3., 0.], [4., 0.], [5., 0.],

   [6., 0.], [7., 0.], [8., 0.], [9., 0.], [10., 0.], [11., 0.],

   [12., 0.], [13., 0.], [14., 0.], [15., 0.], [16., 0.],

   [17., 0.], [18., 0.], [19., 0.], [20., 0.], [21., 0.],

   [22., 0.], [23., 0.], [24., 0.], [25., 0.], [26., 0.],

   [27., 0.], [28., 0.], [29., 0.], [30., 0.], [31., 0.],

   [32., 0.], [33., 0.], [34., 0.], [35., 0.], [36., 0.],

   [37., 0.], [38., 0.], [39., 0.], [40., 0.], [41., 0.],

   [42., 0.], [43., 0.], [44., 0.], [45., 0.], [46., 0.],

   [47., 0.], [48., 0.], [49., 0.], [50., 0.], [51., 0.],

   [52., 0.], [53., 0.], [54., 0.], [55., 0.], [56., 0.],

   [57., 0.], [58., 0.], [59., 0.], [60., 0.], [61., 0.],

   [62., 0.], [63., 0.], [64., 0.], [65., 0.], [66., 0.],

   [67., 0.], [68., 0.], [69., 0.], [70., 0.], [71., 0.],

   [72., 0.], [73., 0.], [74., 0.], [75., 0.], [76., 0.],

   [77., 0.], [78., 0.], [79., 0.], [80., 0.], [81., 0.],

   [82., 0.], [83., 0.], [84., 0.], [85., 0.], [86., 0.],

   [87., 0.], [88., 0.], [89., 0.], [90., 0.], [91., 0.],
```

```
    [92., 0.], [93., 0.], [94., 0.], [95., 0.], [96., 0.],

    [97., 0.], [98., 0.], [99., 0.], [100., 0.], [101., 0.]],

    COLOUR(RGB, 1.00000000, 0., 0.),

    LEGEND("__never_display_this_legend_entry")), STYLE(POINT),

    SYMBOL(DEFAULT), THICKNESS(1))

print(??); # input placeholder




plots[display](A3, D3, labels = ["time*no*of*days",
"Infected*Humans*population*with*Infectious*disease*[I[m]]"], labeldirections
= ["horizontal", "vertical"]);
```

Maple code for the optimal control strategy 2

Maple code for the optimal control using two control strategies

```
restart; unprotect(Pi);
a := .165; b := .27; e := 0.1e-1; mu := 1; c := 0.1e-1; delta := .29; Lambda := 20; m[1] := .1;
m[2] := .2; m[3] := .4; m[4] := .1; u[1][0] := 0; u[2][0] := 0; N := 0.1e-1; Q := 900; P1[100]
:= 0; P2[100] := 0; P3[100] := 0; P4[100] := 0; S[0] := 21950; E[0] := 30; i[0] := 0; R[0] :=
0; h := 0.1e-2;

for n from 0 to 100 do S[n+1] :=
S[n]+h*(Lambda-(1-u[1][n]-u[2][n])*a*i[n]*S[n]-mu*S[n]+c*R[n]); E[n+1] :=
E[n]+h*((1-u[1][n]-u[2][n])*a*i[n]*S[n+1]-(u[1][n]+u[2][n]+mu+delta)*E[n]); i[n+1] :=
i[n]+h*(delta*E[n+1]-(u[1][n]+e+mu)*i[n]); R[n+1] :=
R[n]+h*(u[1][n]*i[n+1]+u[1][n]*E[n+1]+u[2][n]*E[n+1]-(c+mu)*R[n]); P1[100-n-1] :=
P1[100-n]+h*((P1[100-n]-P2[100-n])*(1-u[1][n]-u[2][n])*a*i[n+1]-mu*P1[100-n]);
P2[100-n-1] :=
P2[100-n]+h*(P2[100-n]*(u[1][n]+u[2][n]+mu+delta)-P4[100-n]*(u[1][n]+u[2][n])-P3[1
00-n]*delta-Q); P3[100-n-1] :=
P3[100-n]+h*(P3[100-n]*(u[1][n]+e+mu)-P4[100-n]*u[1][n]-N); P4[100-n-1] :=
P4[100-n]+h*(P4[100-n]*(c+mu)-P1[100-n-1]*c); A[1][n] :=
-(1/2)*((P2[100-n-1]-P1[100-n-1])*a*S[n+1]*i[n+1]+(P2[100-n-1]-P4[100-n-1])*E[n+1]
+(P3[100-n-1]-P4[100-n-1])*i[n+1])/m[3]; A[2][n] :=
-(1/2)*((P2[100-n-1]-P1[100-n-1])*a*S[n+1]*i[n+1]+(P2[100-n-1]-P4[100-n-1])*E[n+1]
)/m[4]; u[1][n+1] := min(1, max(0, A[1][n])); u[2][n+1] := min(1, max(0, A[2][n])) end
do;
S[0] := 21950; E[0] := 30; i[0] := 0; R[0] := 0;
C1 := [seq(S[n], n = 0 .. 100)]; C2 := [seq(E[n], n = 0 .. 100)]; C3 := [seq(i[n], n = 0 .. 100)];
C4 := [seq(R[n], n = 0 .. 100)];
with(Statistics); A1 := PointPlot(C1, color = green, thickness = 1, symbol = default);
%;
PLOT(CURVES([[1., 21950.], [2., 21928.070], [3., 21906.19337],

   SYMBOL(DEFAULT), THICKNESS(1))
with(Statistics); A2 := PointPlot(C2, color = green, thickness = 1, symbol = default);
print(`output redirected...`); # input placeholder
PLOT(CURVES([[1., 30.], [2., 29.96130], [3., 29.83132155],



   LEGEND("__never_display_this_legend_entry")), STYLE(POINT),

   SYMBOL(DEFAULT), THICKNESS(1))
with(Statistics); A3 := PointPlot(C3, color = green, thickness = 1, symbol = default);
print(`output redirected...`); # input placeholder
PLOT(CURVES([[1., 0.], [2., 0.0086887770], [3., 0.01732239581],
```

```
   COLOUR(RGB, 0., 1.00000000, 0.),

   LEGEND("__never_display_this_legend_entry")), STYLE(POINT),

   SYMBOL(DEFAULT), THICKNESS(1))

for n from 0 to 100 do S[n+1] := S[n]+h*(-a*S[n]*i[n]+c*R[n]-mu*S[n]+Lambda);
E[n+1] := E[n]+h*(a*i[n]*S[n+1]-(mu+delta)*E[n]); i[n+1] :=
i[n]+h*(delta*E[n+1]-(e+mu)*i[n]); R[n+1] := R[n]-h*(c+mu)*R[n] end do;
S[0] := 21950; E[0] := 30; i[0] := 0; R[0] := 0;
B1 := [seq(S[n], n = 0 .. 100)]; B2 := [seq(E[n], n = 0 .. 100)]; B3 := [seq(i[n], n = 0 .. 100)];
B4 := [seq(R[n], n = 0 .. 100)];
with(Statistics); D1 := PointPlot(B1, color = red, thickness = 1, symbol = default);
%;
PLOT(CURVES([[1., 21950.], [2., 21928.070], [3., 21906.13049],

deq1 := diff(S(t), t)-Lambda+a*S(t)*i(t)+mu*S(t)-c*R(t), diff(E(t),
t)-a*S(t)*i(t)+(mu+b+delta)*E(t), diff(i(t), t)-delta*E(t)+(b+e+mu)*i(t), diff(R(t),
t)-b*i(t)-b*E(t)+c*R(t)+mu*R(t);
print(`output redirected...`); # input placeholder
       / d         \
       |--- S(t)| - Lambda + a S(t) i(t) + mu S(t) - c R(t),
       \ dt        /

          / d         \
          |--- E(t)| - a S(t) i(t) + (mu + b + delta) E(t),
          \ dt        /

          / d         \
          |--- i(t)| - delta E(t) + (b + e + mu) i(t),
          \ dt        /

          / d         \
          |--- R(t)| - b i(t) - b E(t) + c R(t) + mu R(t)
          \ dt        /
deq := eval(deq1, {Lambda = 1034, a = 0.25e-4, b = 0.98941e-1, c = 0.5e-1, delta =
0.498e-1, e = 0.5019e-1, mu = 0.2165e-2});
print(`output redirected...`); # input placeholder
/ d         \
|--- S(t)| - 1034 + 0.000025 S(t) i(t) + 0.002165 S(t)
\ dt        /

                      / d         \
    - 0.05 R(t), |--- E(t)| - 0.000025 S(t) i(t) + 0.150906 E(t),
                      \ dt        /
```

```
  / d          \
  |--- i(t)| - 0.0498 E(t) + 0.151296 i(t),
  \ dt         /

  / d          \
  |--- R(t)| - 0.098941 i(t) - 0.098941 E(t) + 0.052165 R(t)
  \ dt         /
ic1 := S(0) = 80000, i(0) = 0, R(0) = 10, E(0) = 50;
print(`output redirected...`); # input placeholder
              S(0) = 80000, i(0) = 0, R(0) = 10, E(0) = 50
dsol1 := dsolve({deq, ic1}, numeric);
%;
proc(x_rkf45)   ...   end;
```

Maple code for the deterministic variation using data

```
dsol1 := dsolve({deq, ic1}, numeric, output = array([0, 1, 2, 3,

  4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20,

  21, 22, 23, 24, 25, 26, 27, 28, 29, 3031, 32, 33, 34, 35, 36,

  37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50]))

dsol1 := dsolve({deq,ic1}, numeric,
output=array([0,1,2,3,4,5,6,7,8,9,10,11,12,13,14,15,16,17,18,19,20,21,22,23,24,25,26,27,
28,29,30,31,32,33,34,35,36,37,38,39,40,41,42,43,44,45,46,47,48,49,50,51,52,53,54,55,56
,57,58,59,60,61,62,63,64,65,66,67,68,69,70,71,72,73,74,75,76,77,78,79,80,81,82,83,84,8
5,86,87,88,89,90,91,92,93,94,95,96,97,98,99,100]));

with(plots): sys := deq;
fcns := {S(t) , E(t) , i(t) , R(t) }:
p:= dsolve({sys,S(0) = 80000, i(0) =0, R(0) = 10, E(0) =
50},fcns,type=numeric,method=classical):
/ d          \
|--- S(t)| - 1034 + 0.000025 S(t) i(t) + 0.002165 S(t)
\ dt         /

                        / d          \
    - 0.05 R(t), |--- E(t)| - 0.000025 S(t) i(t) + 0.150906 E(t),
                        \ dt         /

  / d          \
  |--- i(t)| - 0.0498 E(t) + 0.151296 i(t),
```

```
  \ dt       /

  / d        \
  |--- R(t)| - 0.098941 i(t) - 0.098941 E(t) + 0.052165 R(t)
  \ dt       /
odeplot(p, [[t,S(t)],[t,E(t)],[t,i(t)],[t,R(t)]],0..200,titlefont = ["ROMAN", 15], labelfont =
["ROMAN", 20],labels = ["time (no of days) ", "S,E,i,R "], labeldirections = ["horizontal",
"horizontal"],linestyle=[solid,solid,solid,solid],color=[green,yellow,red,blue],legend = [
"S(t) ","E(t) ","i(t) ","R(t) "],legendstyle = [font = ["HELVETICA", 20], location =
right]);;;
```

Maple code for the stochastic variation using data

```
>
```

$$dequ1 := \frac{d}{dt}S(t) - \Lambda + a\cdot S(t)\cdot i(t) + \mu\cdot S(t) - c\cdot R(t) + \sigma_1\cdot a\cdot S(t)\cdot i(t)\cdot dB1 + \sigma_2\cdot\mu\cdot S(t)\cdot dB2, \frac{d}{dt}E(t) - a\cdot S(t)\cdot i(t) + (\mu + b + \delta)\cdot E(t) - \sigma_1\cdot a\cdot S(t)\cdot i(t)\cdot dB1 + \sigma_2\cdot\mu\cdot S(t)\cdot dB2 + \sigma_3\cdot b\cdot E(t)\cdot dB3 + \sigma_4\cdot\delta\cdot E(t)\cdot dB4, \frac{d}{dt}i(t) - \delta\cdot E(t) + b\cdot i(t) + e\cdot i(t) + \mu\cdot i(t) - \sigma_4\cdot\delta\cdot E(t)\cdot dB4 + \sigma_3\cdot b\cdot i(t)\cdot dB3 + \sigma_2\cdot\mu\cdot i(t)\cdot dB2, \frac{d}{dt}R(t) - b\cdot i(t) - b\cdot E(t) + \mu\cdot R(t) + c\cdot R(t) - \sigma_3\cdot b\cdot E(t)\cdot dB3 - \sigma_3\cdot b\cdot i(t)\cdot dB3 - \sigma_2\cdot\mu\cdot R(t)\cdot dB2$$

$$\frac{d}{dt}S(t) - \Lambda + a\,S(t)\,i(t) + \mu\,S(t) - c\,R(t) + \sigma_1\,a\,S(t)\,i(t)\,dB1 + \sigma_2\,\mu\,S(t)\,dB2, \frac{d}{dt}E(t) - a\,S(t)\,i(t) + (\mu + b + \delta)\,E(t) - \sigma_1\,a\,S(t)\,i(t)\,dB1 + \sigma_2\,\mu\,S(t)\,dB2 + \sigma_3\,b\,E(t)\,dB3 + \sigma_4\,\delta\,E(t)\,dB4, \frac{d}{dt}i(t) - \delta\,E(t) + b\,i(t) + e\,i(t) + \mu\,i(t) - \sigma_4\,\delta\,E(t)\,dB4 + \sigma_3\,b\,i(t)\,dB3 + \sigma_2\,\mu\,i(t)\,dB2, \frac{d}{dt}R(t) - b\,i(t) - b\,E(t) + \mu\,R(t) + c\,R(t) - \sigma_3\,b\,E(t)\,dB3 - \sigma_3\,b\,i(t)\,dB3 - \sigma_2\,\mu\,R(t)\,dB2$$

$$deq := eval\left(dequ1, \left\{a = 0.000025, b = 0.048941, e = 0.05019, \mu = 0.002165, c = 0.05, \delta = 0.0498, \sigma_1 = 0.038, \sigma_2 = 5.24388, \sigma_3 = 0.269304, \sigma_4 = 0.630522, dB1 = 0.09, dB2 = 0.002, dB3 = 0.00075, dB4 = 0.0025, \Lambda = 1034\right\}\right)$$

$$\frac{d}{dt}S(t) - 1034 + 0.00002508550\,S(t)\,i(t) + 0.002187706000\,S(t) - 0.05\,R(t), \frac{d}{dt}E(t)$$

$$- 0.00002508550\,S(t)\,i(t) + 0.1009943850\,E(t) + 0.00002270600040\,S(t), \frac{d}{dt}i(t)$$

$$- 0.04987849999\,E(t) + 0.1013285910\,i(t), \frac{d}{dt}R(t) - 0.04895088501\,i(t)$$

$$- 0.04895088501\,E(t) + 0.05214229400\,R(t)$$

$ic1 := S(0) = 80000, i(0) = 0, R(0) = 10, E(0) = 50$

$$S(0) = 80000, i(0) = 0, R(0) = 10, E(0) = 50$$

```
> dsol1 := dsolve({deq, ic1}, numeric, output = array([0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13,
  14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37,
  38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61,
  62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85,
  86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100]));
```

R CODE FOR THE HISTOGRAM

```
library(moments)

set.seed(1234)

#Simulate Ro using simulated values of the parameters

par(mfrow = c(2, 3))

#Simulate Ro varying the recruiment rate at which the susceptible class is being

populated

a1 = 0.000025

b1 = 0.48941

d1 = 0.0498

e1 = 0.05019

u1 = 0.002165

A1 = sort(runif(1000, 500,1500))

Ro1 = (a1*A1*d1)/(u1*(u1+b1+a1)*(b1+e1+u1))

Ro1
```

```
m1 = mean(Ro1)

#Simulate Ro varying the rate of infection

a2 = sort(runif(1000, 0, 2*a1))

b2 = 0.48941

d2 = 0.0498

e2 = 0.05019

u2 = 0.002165

A2 = 1034

Ro2 = (a2*A2*d2)/(u2*(u2+b2+a2)*(b2+e2+u2))

Ro2

m2 = mean(Ro2)



#Simulate Ro varying the rate at which the exposed class is being populated

a3 = 0.000025

b3 = 0.48941

d3 = 0.0498

d3 = sort(runif(1000,0,2*d1))

e3 = 0.05019

u3 = 0.002165

A3 = 1034

Ro3 = (a3*A3*d3)/(u3*(u3+b3+a3)*(b3+e3+u3))

Ro3
```

```
m3 = mean(Ro3)

sRo = c(m1, m2, m3)

mn = mean(sRo)

sdRo= sd(sRo)

skewness(sRo)

kurtosis(sRo)

LCI = mn - 1.96*sdRo

UCI = mn + 1.96*sdRo

mn

LCI

UCI


plot(Ro1, A1)

plot(Ro2, a2)

plot(Ro3,d3)

hist(Ro1, xlab = "Ro")

hist(Ro2,   xlab = "Ro")

hist(Ro3,   xlab = "Ro")

#Simulate Ro with the parameters gotten from data

a = 0.000025

b = 0.48941

d = 0.0498

e = 0.05019
```

u = 0.002165

A = 1034

Ro = (a*A*d)/(u*(u+b+a)*(b+e+u))

Ro