A MATHEMATICAL MODEL OF HIV/AIDS EPIDEMIC IN THE PRESENCE OF IRRESPONSIBLE INFECTIVES

By:

Mohammed Ibrahim Daabo
UDS/MM/0002/08

UNIVERSITY FOR DEVELOPMENT STUDIES

April 2010

A MATHEMATICAL MODEL OF HIV/AIDS EPIDEMIC IN THE PRESENCE OF IRRESPONSIBLE INFECTIVES

A THESIS SUBMITTED TO THE DEPARTMEMNT OF MATHEMATICS, UNIVERSITY FOR DEVELOPMENT STUDIES, GHANA

IN PARTIAL FULFILMENT OF THE REQUIREMENT FOR THE DEGREE OF MASTER OF SCIENCE

By:

Mohammed Ibrahim Daabo

UDS/MM/0002/08

April 2010

Dedication

I dedicate this work to my children, Abdul Basit, Faiza and my newly born son Habab

Declaration

I hereby declare that this submission is my own work and to the best of my knowledge, it contains no material previously published or written by another person nor material which to a substantial extent has been accepted for the award of any other degree or diploma at UDS or any other educational institution, except where due acknowledgement is made in the thesis. Any contribution made to the research by colleagues, with whom I have worked at UDS or elsewhere, during my candidature, is fully acknowledged.

I also declare that the intellectual content of this thesis is the product of my own work, except to the extent that assistance from others in the project's design and conception or in style, presentation and linguistic expression is acknowledged.

MOHAMMED I. DAABO		12/05/2010
Name of Student	Signature	Date

Certified by:

Professor Oluwole Daniel Makinde		12/05/2010
Name of Supervisor	Signature	Date

Certified by:

Name of Head of department	Signature	Date

Abstract

In this thesis, a non-linear mathematical model is proposed and analyzed to study the effect of irresponsible infectives in the spread of HIV/AIDS in a variable size population. In considering the modeling dynamics, the population is divided into four subclasses, of susceptibles (HIV negatives who can contract the disease), irresponsible infectives (people who are infected with the virus but do not know or live irresponsible life styles) , responsible infectives (HIV positives who know they are infected and are careful) and full-blown AIDS patients. Susceptibles are assumed to be infected through sexual contact with infectives and all infectives develop AIDS at a constant rate. The stability theory of differential equations and computer simulations are used to analyze the model. The model analysis shows that the disease-free equilibrium is always locally asymptotically stable and in such a case the basic reproductive number $R_0 < 1$ and the endemic equilibrium does not exist. The disease is thus eliminated from the system. If $R_0 > 1$, the endemic equilibrium exists and the disease remains in the system. It is shown that the endemicity of the disease is reduced when irresponsible infectives become responsible infectives who are more likely not to take part in sexual interactions.

A numerical simulation of the model is also used to investigate the influence of certain other parameters on the transmission dynamics of HIV/AIDS.

List of Tables

List of Figures

Acknowledgments

All thanks and praise are due to ALLAH (Almighty GOD) who gave us life, health, and strength and sustained us during the period of the study.

My foremost gratitude goes to my supervisor, Professor Oluwole Daniel Makinde of Cape Penisula University of Technology, South Africa, who painstakingly guided and directed the study at the time he could barely make any time for anything beside his work. I say may the almighty God bless you. In a similar vein, I am grateful to the Vice Chancellor Professor K. S. Nokoe for the support and the opportunity given to me to study in UDS. He is a mentor and a father. May the good Lord continue to bless him. I must take this opportunity to acknowledge my great debt to the Dean, Faculty of Mathematical Sciences, Dr. O. E. Oyetunji, the vice Dean Dr. Alhassan Seidu for their keen interest in research.

I am also thankful to the postgraduate coordinator, Mr. Kazeem A. Gbolagade for all the courtesy, understanding and support extended to me during this project.

I am equally grateful to my friend Mr. Baba Seidu, a senior research assistant in the department of Mathematics for the invaluable discussions we have had over the course of this work.

I wish to express my gratitude and appreciation to all persons especially my friend Abukari Alhassan and lecturers in the Faculty of Mathematical Sciences without whose support, the production of this work would have been a fiasco.

Finally, I am indebted to my wife, Mumuni Nashiratu for the sacrifices she has made in ensuring my success and for always been there for me.

Contents

CHAPTER 1

Introduction

1.1 Introduction

Human Immunodeficiency Virus (HIV) is the agent that causes Acquired Immunodeficiency Syndrome (AIDS). HIV is transmitted through sexual contact with an infected individual, through exchange of infected blood or blood products, or to the newborn from an infected mother. HIV infected persons may harbor the virus for many years with no clinical signs of the disease. Eventually, HIV destroys the body's immune system, mainly by impairing a class of white blood cells whose regulatory activities are essential for immune protection. As a result, people who have AIDS are prone to lung infections, brain abscesses, and a variety of other infections caused by microorganisms that usually do not produce disease in healthy people. Those who have AIDS also are prone to cancers such as Kaposi's sarcoma, a skin cancer rarely seen in non-HIV-infected populations (Chin and Lwanga, 1991). HIV/AIDS is one of the most destructive diseases humankind has ever faced, with profound social, economic and public health consequences. Since the beginning of the pandemic over 25 years ago, more than 25 million people have died of AIDS-related illnesses and an estimated 33 million people are now living with HIV (International Aids Society [IAS], 2009). Sub-Saharan Africa remains the most severely affected of the pandemic with an estimated 22.5 million people with HIV, or 68% of the global total, are in Sub-Saharan Africa (World Health Organization Media Centre, 2007). The AIDS epidemic is estimated to be one of the leading causes of death globally and the major cause of death in Africa.

In Ghana, the first HIV/AIDS case was reported in 1986 and as of the end of 2003, an estimated 350,000 people were living with the disease in the country with about 30,000 death cases recorded

(The Henry J. Kaiser Family Foundation, 2005). Currently, heterosexual sex remains the predominant mode of transmission of HIV in Ghana, accounting for 75-80 per cent of all infections, with an estimate 240, 802 HIV positive cases as against 236, 151 recorded in 2003. The annual infection rate has over the years reduced from 3.6% in 2003 to 1.7% in 2008 (Donkor, 2009). The AIDS epidemic is estimated to be one of the leading causes of death in sub-Saharan Africa and the fourth leading cause of death globally. The pandemic has cut life expectancy significantly in many countries in sub-Saharan Africa. For example life expectancy in Botswana decreased from 65 years in 1985-1990 to 40 years in 2000-2005 (Sharomi, 2006). In addition to being a serious public health problem, HIV has far reaching consequences to all social and economic sectors and society. It exacerbates poverty, reduces educational opportunities, devastates the work force, creates large numbers of orphans and exerts tremendous pressure on the limited health and social services (Sharomi, 2006). For example, HIV/AIDS has cut annual growth rates in Africa by 2-4% per year, (Dixon et al., 2002). Mathematical models have been used extensively in research into the epidemiology of HIV/AIDS, to help improve our understanding of the main contributing factors in a given epidemic of the disease. According to Glenn Ledder, (2005) "A mathematical model is a set of formulas or equations based on a quantitative description of a real life or physical problem and created in the hope that the behavior it predicts will resemble the real behavior on which it is based". Mathematical models of the transmission dynamics of HIV play an important role in our better understanding of epidemiological patterns for disease control as they provide short and long term predication of HIV/AIDS incidence. May and Anderson, (1986,1987,1988) initial work on modeling saw various refinements being made into modeling frameworks and over the years specific issues have been looked at by researchers. Makinde (2009) studied the transmission dynamics of infectious diseases with waning immunity using the non-purtubative approach. Makinde (2007) again looked at the Adomian decomposition approach to a SIR epidemic model with constant vaccination strategy. Alexander et al. (2006) also studied the effect of the booster vaccination on disease epidemiology. Flessa (1999) developed a model on decision support for malaria control programmes. Mogadas and Gumel (2003) proposed a mathematical model to study childhood diseases with non-permanent immunity. Misra and Mishra (2009) considered the effect of booster vaccination on the transmission dynamics of diseases that spread by droplet infection.

Agraj et al (2006) studied the spread of AIDS epidemic with vertical transmission by considering a non-linear mathematical model. Arazoza and Lounes (2002) considered a non-linear model on sexually transmitted diseases with contact tracing. Busenburg et al (1995) considered a simple model on HIV transmission in India. Coutsoudis et al (2002) studied on the free formula milk for infants of HIV infected women. Dunn et al. (1998) looked at the variations in maternal infectivity on mother-to-child transmission of HIV. Kribs-Zeleta (1999) considered a structured model for heterosexual disease transmission. Newell (1998) studied the mechanism and timing of mother-to-child transmission of HIV infection. Perelson and Nelson (1999) also studied a mathematical analysis of HIV dynamics. Rajesh and Nafees (2006) derived a primary sequence and secondary structures of responsive elements from HIV infective mothers and infants on vertical transmission. Rouzious et al (1995) considered an estimated timing of mother-to-child HIV transmission by using markov model. Van de Perre (1999) studied the transmission of the human immune-deficiency virus through breast-feeding. Busenberg and Cooke (1993) discussed a variety of diseases that transmit both horizontally and vertically, and gave a comprehensive survey of the formulation and the mathematical analysis of compartmental models that also incorporate vertical transmission. Hyman et al. (1999) studied the impact of variations in infectiousness by taking into account different levels of virus between individuals during the Chronic phase of infection. Greenhalgh et al. (2001) studied the impact of condom use on sexual transmission of HIV/AIDS amongst a homogeneously mixing male homosexual population. Li et al. (2001) proposed a model for an infectious disease that spreads in the host population through both horizontal and vertical transmission. Hsieh and Chen (2004) developed a mathematical model for a model community which has the structure of two classes of commercial sex workers and two classes of sexually active male customers with different levels of sexual activity. Agarwala (2002) developed a density dependent HIV transmission model for a Canadian population by taking into account the vertical transmission and by using simple mass action type interaction. Naresh and Tripathi (2005) studied the spread of HIV infection in a population in the presence of tuberculosis. Research is still going on in the area of modeling. The importance of this is to partner with health experts and policy makers to see how best the spread of the disease can be reduced through medical intervention and behavioral change. This research seeks to develop a mathematical model to study the impact of irresponsible infectives

on the spread of HIV/AIDS infection and then offer possible intervention strategies. The research will also develop a theoretical framework that would predict the possible intervention strategies to prevent the spread of HIV/AIDS infection resulting from infective immigrants. It will provide a numerical solution for non-linear systems of differential equations resulting from the modeling of the impact of irresponsible infectives and infective immigrants on the spread of HIV/AIDS. The aim is to offer both short and long term strategies to control the spread of HIV/AIDS.

1.2 Mathematical Modeling

Mathematical modeling has to do with using Mathematics to explore topics outside Mathematics. Thus, mathematical modeling is an activity of translating a real life problem into mathematical form for subsequent analysis. Mathematical models arise in every field of study that is of human interest, including Science, Engineering, Economics and sometimes the non-sciences such as History. By nature, a mathematical model could be as simple as a single equation relating two (2) variables or as complicated as a set of n equations with n unknowns. Mathematical modeling usually begins with a conceptual thinking of a real life problem which leads to an idealized characterization of it. Thus, the mathematical model is a mathematical description of the conceptual model and most often not the real situation (Ledder, 2005)

1.2.1 Mathematical Models in Science

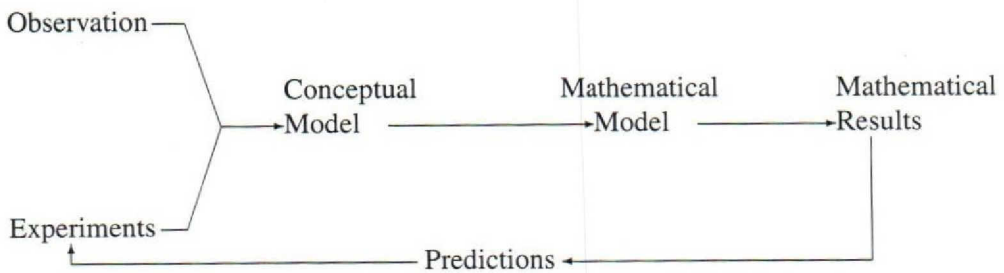

Figure 1.1: Schematic mathematical model for science (Ledder, 2005)

Figure 1.1 shows a schematic mathematical model that illustrates a typical scientific model. The process begins with a careful observation and scientific experimentation which leads to a conceptual thinking about a physical problem. The conceptual model is then transformed into the appropriate mathematical equation/model, which when further solved produces the required

mathematical results. These results can be validated with existing results and experiments before they can be used in making predictions about the physical problem (Ledder, 2005).

1.2.2 Components of Mathematical Models

Variables, constants, parameters and input functions are generally considered as components of a mathematical model (Ledder, 2005).

Independent variables: An independent variable is a mathematical quantity that takes on a range of values. Such quantities are measures of time.

Dependent variables: A dependent variable is a quantity that changes during a given problem, depending on the values of the independent variable.

Constant: This is a quantity in modeling that has a single fixed value and does not change during a given problem.

Domain of the model: This includes the set of all possible values of the dependent variable.

Parameters: A model parameter is a quantity whose value is fixed throughout the domain of the model but can be varied to give a family of related problems.

1.2.3 Model Development Steps

Different problems may require specific steps in their model formulation and development. Glenn Ledder (2005) considered the following outline as a guide for model formulation.

1. Determine the purpose of the mathematical model.
2. Outline a conceptual model to describe an idealized representation of the situation being modeled.
3. Define symbols to represent the various mathematical quantities in the conceptual model.
4. Derive mathematical equations to represent the model.
5. Make simplifications of the original equations to conform with solvable equations.
6. Analyze the model by using analytical, graphical, and/or numerical techniques.
7. Critique the model by agreeing or disagreeing with its predictions on experimental data, common sense or everyday experience.

1.3 Statement of the Problem

HIV/AIDS was first widely recognized in the early 1980s and has since not gotten any cure. Researchers, Governments, Health professionals and individuals have gone on various campaign messages to sensitize and educate the public on the dangers and transmission dynamics of the disease; but HIV/AIDS still remains the world number one health problem today. Mathematicians have also joined to educate and offer control strategies of HIV/AIDS transmission dynamics using the knowledge of mathematical modeling and epidemiology. Several mathematical modeling problems have been looked at in the literature. However, there are many more areas in which research is continuing especially with respect to the non linear behavior of infectives.

The transmission dynamics of HIV/AIDS infection in the presence of irresponsible infectives in a population is the research problem that this thesis seeks to address using mathematical modeling.

1.4 Aim of the Study

This study aims to investigate theoretically the impact of irresponsible infectives on the transmission dynamics of HIV/AIDS within a given population.

1.4.1 Specific Objectives of the Study

1. To develop a mathematical model to study the impact of irresponsible infectives on the risk of HIV/AIDS infection and then offer possible intervention strategies.
2. To develop a theoretical framework that would predict the possible intervention strategies to prevent the spread of HIV/AIDS infection resulting from infective immigrants.
3. To provide a numerical solution for non-linear systems of differential equation modeling the impact of irresponsible infectives and infective immigrants on the spread of HIV/AIDS.

1.5 Significance of the Study

Modeling the impact of irresponsible infectives on transmission dynamics of HIV/AIDS is extremely important for the following reasons. It will help;

- Describe patterns of infection and HIV/AIDS occurrence in a population.
- Identify outbreaks or unusual rates of HIV/AIDS occurrence.
- Assist in the understanding and control of HIV/AIDS transmission dynamics.

- Identify and characterize factors that contribute to the spread of HIV infection.
- Develop and evaluate mathematical models on HIV/AIDS transmission dynamics.
- Develop and evaluate primary, secondary and tertiary prevention and control measures of HIV/AIDS for individuals.

1.6 Theoretical Framework

The operational definitions of concepts relating to the transmission dynamics of infectious diseases will be presented in this section.

1.6.1 Threshold Theorem (The Threshold theorem of Epidemiology):

This has to do with a situation where a small group of people having an infectious disease is inserted into a large population that is capable of catching the disease. The question is: What happens as time evolves? Will the disease die out rapidly, or will an epidemic occur? How many people will ultimately catch the disease? To answer these questions, a system of differential equations, which govern the spread of infectious diseases within a population is derived, and the behavior of its solution is analyzed. This approach will then lead to the Threshold Theorem of epidemiology, which states that an epidemic will occur only if the number of people who are susceptible to the disease exceeds a certain threshold value.

1.6.2 Some Definitions

Stability Condition: These are the conditions that has to do with long-term behavior of the system which is disturbed, with regard to whether or not it will differ from the undisturbed behavior by an acceptably small amount.

Autonomous System: Suppose $x^{'} = (x_1, x_2, , x_n)^{'}$ is a system of differential equations describing the changes in the variables characterizing an epidemiological problem. The system is said to be autonomous if $(x_1, x_2, , x_n)$ does not depend on t explicitly, where t is the time variable.

Equilibrium Point: An equilibrium point x_0 of an autonomous system is a point which simultaneously satisfies $x^{'} = 0$.

A critical point (x_0, y_0) of a system of differential equations $x^{'} = Ax$ is **stable** if given a number $\varepsilon > 0$, there exists a number $\delta > 0$ such that $x = \phi(t), y = \psi(t)$ of the system at $t = 0$, satisfies

$[(\phi(0)-x_0)^2+(\psi(0)-y_0)^2]^{1/2}<\delta$ and $[(\phi(0)-x_0)^2+(\psi(0)-y_0)^2]^{1/2}<\varepsilon$ for all $t>0$. This definition implies that all solutions that are sufficiently close to (x_0,y_0) stay close to (x_0,y_0)

A critical point (x_0,y_0) is **asymptotically stable** if it is stable and if there exists δ_0, such that $0<\delta_0<\delta$ and such that if a solution $x=\phi(t)$, $y=\psi(t)$ satisfy $[(\phi(0)-x_0)^2+(\psi(0)-y_0)^2]^{1/2}<\delta_0]$, then $\lim\psi(t)=y_0$ as $t\to\infty$. This definition means that the solution curves that start close to (x_0,y_0) must not only stay close to (x_0,y_0) but must approach (x_0,y_0) as $t\to\infty$

Basic Reproduction Number: The average number of secondary infections produced when one infected individual is introduced into the whole population where everyone is susceptible; or threshold quantity that determines when an infection can invade and persist in a new host population.

Endemic Disease: The habitual presence of a disease or infectious agent in a defined geographical area or population.

Epidemic: Rates of disease clearly in excess of normal or expected frequency in a defined geographic area.

Epidemiology: The study of the distribution and determinants of health-related conditions and events in a population, and the application of this study to the control of health problems.

Horizontal Transmission: Transmission that typically occurs through direct or indirect physical contact with infectious hosts or through disease vectors such as mosquitoes, ticks, or other biting insects.

Incubation Period: A time beginning with invasion by an infectious agent and continuing until the organism multiplies to a sufficient number to produce a host reaction and clinical symptoms.

Induction Period: The period of time from causal action of a factor (exposure) to initiation of a disease.

Infection: The entry and establishment of an infectious agent in a host (synchronization).

Latency: The time between exposure to a disease-producing agent and manifestation of the disease.

Pandemic: Epidemics that involves population in widespread geographic areas of the world.

Prevalence: Measure of the number of cases of a given disease in a specified Population at a designated time; usually a rate measured at a point in time.

Susceptibility: State or quality of lacking resistance to an agent and therefore being likely to develop effects if exposed.

1.7 Thesis Outline

This thesis is organized into four chapters. Chapter one consists of an introduction and background study of the epidemiology of HIV/ AIDS transmission. Basic mathematical definitions needed to qualitatively analyze the models in the thesis are also included in chapter one. In chapter two, HIV/AIDS transmission model in the presence of irresponsible infectives is modeled and analyzed. The effect of infective immigrants is considered in the modeling process in chapter three. Chapter four discusses the findings, conclusions and contributions for future research work.

CHAPTER 2

Modeling HIV/AIDS transmission in the presence of irresponsible infectives

2.1 Introduction

In this chapter, a non-linear mathematical model has been proposed and analyzed to study the effect of the presence of irresponsible infectives in the spread of HIV/AIDS in a variable size population N. Irresponsible infectives are HIV positives who may not know that they have the disease. It also includes people who have the disease and are seriously involved in drug abuse and alcoholism. Ignorance, drugs abuse and alcoholism impairs judgment and good decision making, leaving people more prone to engage in HIV risk behavior such as unsafe sex, injection and non adherence to HIV treatment. There can be linkages between ignorance, alcoholism, drug abuse and HIV infections and therefore, the socio-behavioral intervention of society can help change infectives behavior and minimize the risk of infections.

2.2 Model formulation and Assumptions

2.2.1 Assumptions

The following assumptions are made in order to construct the mathematical models for the problem

1. The population N under study is heterogeneous and varying with time
2. The population N under study is subdivided into four classes

3. The HIV can only be transmitted through sexual intercourse or through infection from an infected needle and blood.
4. The full-blown AIDS group is sexually inactive
5. The rate at which irresponsible infectives infect people with the disease is higher than that of responsible infectives

2.2.2 Description of model parameters

N(t) = Total population size at time t

S(t) = The size of the Susceptible population at time t

$I_1(t)$ = The size of the Irresponsible infective population at time t

$I_2(t)$ = The size of the Responsible infective population at time t

A(t) =The size of the Full blown AIDS population at time t

c = The number of sexual partners an infective individual has

β_1 = The contact rate of irresponsible infectives

β_2 = The contact rate of responsible infectives

μ =The natural death rate (Natural mortality rate of an individual in the population)

θ = The conversion rate of irresponsible infectives to responsible infectives

δ = The conversion rate of infectives to full-blown AIDS

α =The AIDS-induced mortality rate

Q_0 = The rate of recruitment of Susceptibles into the population.

For clarity sake, we represent $N(t)$, $I_1(t)$, I_2 and $A(t)$ by N, I_1, I_2 and A respectively.

2.3 Model Formulation

In this model we want to study the effect of irresponsible infectives on the spread of HIV/AIDS in a variable-size population at time t in which there is recruitment of susceptibles into the population at a rate Q_0. The population N is subdivided into four classes of susceptibles $S(t)$, irresponsible infectives $I_1(t)$, responsible infectives $I_2(t)$ and AIDS patients $A(t)$ with a natural mortality rate of μ. The AIDS patients class is assumed to have an AIDS induced mortality rate α. It is also assumed that all infectives progress to develop AIDS at the rate δ with time. The irresponsible infectives can become responsible as time evolves at the rate θ. Susceptibles become HIV infected

through sexual contact with infectives and are directly recruited into the irresponsible population at the rate proportional to the susceptible population and to the ratio between the number of infected populations and the total population. Thus, $\frac{dI_1}{dt} \propto \frac{SI_1}{N}$ and $\frac{dI_1}{dt} = \beta_1 \frac{SI_1}{N}$. Also, $\frac{dI_2}{dt} \propto \frac{SI_2}{N}$ and $\frac{dI_2}{dt} = \beta_2 \frac{SI_2}{N}$. β_1 and β_2 are the effective contact rate of an infective individual. We also consider the parameter c, that is the number of sexual partners an infective individual has to play an important role in the spread of the disease. The above considerations and discussions can be summarized and illustrated on the flowchart in figure 2.1 below.

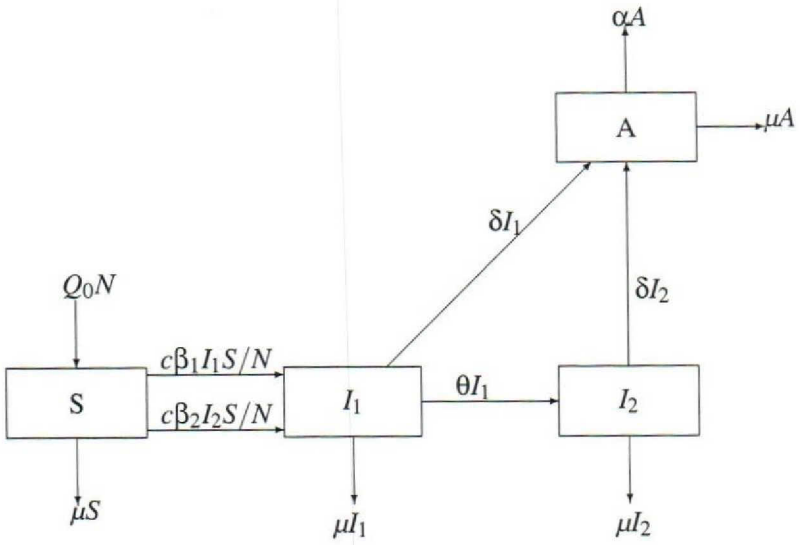

Figure 2.1: Proposed Flow chart for irresponsible infectives model

From figure 2.1, the susceptible population changes with time according to the differential equation

$$\frac{dS}{dt} = Q_0 N - \frac{c(\beta_1 I_1 + \beta_2 I_2)S}{N} - \mu S.$$

Similarly, the population of infective individuals change with time according to the differential equations

$$\frac{dI_1}{dt} = \frac{c(\beta_1 I_1 + \beta_2 I_2)S}{N} - (\delta + \theta + \mu)I_1 \text{ and } \frac{dI_2}{dt} = \theta I_1 - (\delta + \mu)I_2.$$

Finally, the AIDS population changes with time according to the first order differential equation

$$\frac{dA}{dt} = \delta(I_1 + I_2) - (\alpha + \mu)A.$$

Thus the proposed model is governed by the system of first order differential equations

$$\frac{dS}{dt} = Q_0 N - \frac{c(\beta_1 I_1 + \beta_2 I_2)S}{N} - \mu S \tag{2.1}$$

$$\frac{dI_1}{dt} = \frac{c(\beta_1 I_1 + \beta_2 I_2)S}{N} - (\delta + \theta + \mu)I_1 \tag{2.2}$$

$$\frac{dI_2}{dt} = \theta I_1 - (\delta + \mu)I_2 \tag{2.3}$$

$$\frac{dA}{dt} = \delta(I_1 + I_2) - (\alpha + \mu)A \tag{2.4}$$

With initial conditions given by $S(0) = S_0\, I_1(0) = I_{10}\, I_2(0) = I_{20}\, A(0) = A_0$, $\beta_1 > \beta_2$. Note that if the total population size is given by $N = S + I_1 + I_2 + A$ then we have

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI_1}{dt} + \frac{dI_2}{dt} + \frac{dA}{dt} = (Q_0 - \mu)N - \alpha A$$

$$\therefore \frac{dN}{dt} = (Q_0 - \mu)N - \alpha A$$

The questions that we might want to address using this model include;

- What will be the effect of a small proportion of irresponsible HIV/AIDS infectives present in a given population? Will the disease die out? or will there be an epidemic as time evolves?
- What is the effect of the model parameters α, $\gamma\,\delta$, β_1, β_2, θ and c on the transmission dynamics of HIV/AIDS?

2.4 Solution of the Model

Since the model equations are nonlinear in nature, it is convenient to solve the system qualitatively and numerically (Simulation).

2.5 Qualitative Analysis

2.5.1 Normalization

The dimensions of the model variables S, I_1, I_2 and A are people. The population size N also has people as its dimension. Therefore, in order to solve the system of equations, we need to normalize the model by defining new variables s, i_1, i_2 and a.

It must be noted that i_1 and i_2 are the fractions of the population that are infected. a is the fraction of AIDS population and s is the fraction of the susceptible population. Since the fraction infected can, in principle, range between 0 and 1, then $s + i_1 + i_2 + a = 1$.

Thus, to normalize the model, we set $s = \frac{S}{N}, i_1 = \frac{I_1}{N}\ i_2 = \frac{I_2}{N}$ and $a = \frac{A}{N}$

Then we obtain

$\frac{dS}{dt} = s\frac{dN}{dt} + N\frac{ds}{dt}$, $\frac{dI_1}{dt} = i_1\frac{dN}{dt} + N\frac{di_1}{dt}$, $\frac{dI_2}{dt} = i_2\frac{dN}{dt} + N\frac{di_2}{dt}$, and $\frac{dA}{dt} = a\frac{dN}{dt} + N\frac{da}{dt}$

Subtituting these identities into the model (2.1)-(2.4) we have:

$$N\frac{ds}{dt} = \frac{dS}{dt} - s\frac{dN}{dt} = Q_0N - \frac{c(\beta_1I_1 + \beta_2I_2)S}{N} - \mu S - s[(Q_0 - \mu)N - \alpha A]$$

$$\Rightarrow N\frac{ds}{dt} = Q_0N - c(\beta_1i_1 + \beta_2i_2)sN - \mu sN - s[(Q_0 - \mu)N - \alpha aN]$$

$$\Rightarrow \frac{ds}{dt} = Q_0 - c(\beta_1i_1 + \beta_2i_2)s - \mu s - s[(Q_0 - \mu) - \alpha a]$$

$$\therefore \frac{ds}{dt} = (1 - s)Q_0 - c(\beta_1i_1 + \beta_2i_2)s + \alpha as$$

Also

$$N\frac{di_1}{dt} = \frac{dI_1}{dt} - i_1\frac{dN}{dt} = \frac{c(\beta_1I_1 + \beta_2I_2)S}{N} - (\delta + \theta + \mu)I_1 - i_1[(Q_0 - \mu)N - \alpha A]$$

$$\Rightarrow N\frac{di_1}{dt} = c(\beta_1i_1 + \beta_2i_2)sN - (\delta + \theta + \mu)i_1N - i_1[(Q_0 - \mu)N - \alpha aN]$$

$$\Rightarrow \frac{di_1}{dt} = c(\beta_1i_1 + \beta_2i_2)s - (\delta + \theta + \mu)i_1 - i_1[(Q_0 - \mu) - \alpha a]$$

$$\therefore \frac{di_1}{dt} = c(\beta_1i_1 + \beta_2i_2)s - (Q_0 + \delta + \theta)i_1 + \alpha ai_1$$

Also

$$N\frac{di_2}{dt} = \frac{dI_2}{dt} - i_2\frac{dN}{dt} = \theta I_1 - (\delta + \mu)I_2 - i_2[(Q_0 - \mu)N - \alpha A]$$

$$\Rightarrow N\frac{di_2}{dt} = \theta i_1N - (\delta + \mu)i_2N - i_2[(Q_0 - \mu)N - \alpha aN]$$

$$\Rightarrow \frac{di_2}{dt} = \theta i_1 - (\delta + \mu)i_2 - i_2[(Q_0 - \mu) - \alpha a]$$

$$\therefore \frac{di_2}{dt} = \theta i_1 - (Q_o + \delta)i_2 + \alpha ai_2$$

Finally

$$N\frac{da}{dt} = \frac{dA}{dt} - a\frac{dN}{dt} = \delta(I_1 + I_2) - (\alpha + \mu)A - a[(Q_0 - \mu)N - \alpha A]$$

$$\Rightarrow N\frac{da}{dt} = \delta(i_1 + i_2)N - (\alpha + \mu)aN - a[(Q_0 - \mu)N - \alpha aN]$$

$$\therefore \frac{da}{dt} = \delta(i_1 + i_2) - (Q_0 + \alpha)a + \alpha a^2$$

Thus, a normalized form of the model (2.1)-(2.4) is given by

$$\frac{ds}{dt} = Q_0 - c(\beta_1i_1 + \beta_2i_2)s - Q_0s + \alpha as \tag{2.5}$$

$$\frac{di_1}{dt} = c(\beta_1i_1 + \beta_2i_2)s - (Q_0 + \delta + \theta)i_1 + \alpha ai_1 \tag{2.6}$$

$$\frac{di_2}{dt} = \theta i_1 - (Q_o + \delta)i_2 + \alpha ai_2 \tag{2.7}$$

$$\frac{da}{dt} = \delta(i_1 + i_2) - (Q_0 + \alpha)a + \alpha a^2 \tag{2.8}$$

With initial conditions $s(0) = s_0$, $i_1(0) = i_{10}$; $i_2(0) = i_{20}$ and $a(0) = a_0$ where $s + i_1 + i_1 + a \leq 1$

2.5.2 Linearization

The Jacobian that appropriately linearizes the governing equations of the model is given by.

$$J(s, i_1, i_2, a) = \begin{pmatrix} -Q_0 - c(\beta_1 i_1 + \beta_2 i_2) + \alpha a & -c\beta_1 s & -c\beta_2 s & \alpha s \\ c(\beta_1 i_1 + \beta_2 i_2) & c\beta_1 s - Q_0 - \delta - \theta + \alpha a & c\beta_2 s & 0 \\ 0 & \theta & -Q_0 - \delta + \alpha a & \alpha i_2 \\ 0 & \delta & \delta & -Q_0 - \alpha + 2\alpha a \end{pmatrix} \tag{2.9}$$

We study the system in the closed set $\Gamma = \{(s, i_1, i_2, a) \in R_+^4 | s + i_1 + i_2 + a \leq 1\}$.

The system will have either of two long-term behaviour; disease-free equilibrium and the endemic equilibrium.

2.5.2.1 Disease-free Equilibrium

The disease-free equilibrium is attained when there are no infectives and no full-blown AIDS patients. That is, disease-free equilibrium is attained when $i_1 = i_2 = a = 0$.Thus, the disease-free equilibrium will be given by $E_0 = (1, 0, 0, 0)$

When the disease is completely eliminated from the system, the solutions approaches the disease free equilibrium of the form of E_0.

The Jacobian evaluated at E_0 is given by

$$J(E_0) = \begin{pmatrix} -Q_0 & -c\beta_1 & -c\beta_2 & \alpha \\ 0 & c\beta_1 - Q_0 - \delta - \theta & c\beta_2 & 0 \\ 0 & \theta & -Q_0 - \delta & 0 \\ 0 & \delta & \delta & -Q_0 - \alpha \end{pmatrix} \tag{2.10}$$

2.5.3 Local stability analysis of disease-free equilibrium

To study the local stability of the disease-free equilibrium, we evaluate the solution of the characteristic equation of the Jacobian of the normalised system at the disease-free equilibrium as follows.

$$f(\lambda)=\begin{vmatrix} -Q_0-\lambda & -c\beta_1 & -c\beta_2 & \alpha \\ 0 & c\beta_1-Q_0-\delta-\theta-\lambda & c\beta_2 & 0 \\ 0 & \theta & -Q_0-\delta-\lambda & 0 \\ 0 & \delta & \delta & -Q_0-\alpha-\lambda \end{vmatrix}=0$$

$$\Rightarrow f(\lambda)=-(Q_0+\lambda)\begin{vmatrix} c\beta_1-Q_0-\delta-\theta-\lambda & c\beta_2 & 0 \\ \theta & -Q_0-\delta-\lambda & 0 \\ \delta & \delta & -Q_0-\alpha-\lambda \end{vmatrix}=0$$

$$\Rightarrow f(\lambda)=(Q_0+\lambda)(Q_0+\alpha+\lambda)\begin{vmatrix} c\beta_1-Q_0-\delta-\theta-\lambda & c\beta_2 \\ \theta & -Q_0-\delta-\lambda \end{vmatrix}=0$$

$$\Rightarrow f(\lambda)=(Q_0+\lambda)(Q_0+\alpha+\lambda)[(-c\beta_1+Q_0+\delta+\theta+\lambda)(Q_0+\delta+\lambda)-c\beta_2\theta]$$

$$\Rightarrow f(\lambda)=(Q_0+\lambda)(Q_0+\alpha+\lambda)(\lambda^2+\nu\lambda+\rho)$$

Where

$\nu=-c\beta_1+2Q_0+2\delta+\theta$, and $\rho=(-c\beta_1+Q_0+\delta+\theta)(Q_0+\delta)-c\beta_2\theta$

$R_0=\frac{c\beta_1(Q_0+\delta)+c\beta_2\theta}{(Q_0+\delta+\theta)(Q_0+\delta)}$

The disease-free equilibrium is locally asymptotically stable if $\nu>0$ and $\rho>0$. However, $\rho>0$ is sufficient for E_0 to be locally asymptotically stable.

Thus E_0 is locally asymptotically stable if

$(-c\beta_1+Q_0+\delta+\theta)(Q_0+\delta)-c\beta_2\theta>0$ or $c\beta_1(Q_0+\delta)+c\beta_2\theta<(Q_0+\delta+\theta)(Q_0+\delta)$.

This condition corresponds with $R_0<1$

2.5.4 Endemic Equilibrium

Recall that $\frac{dN}{dt}=(Q_0-\mu)N-\alpha A$. Then the system 2.1-2.4 can be rewritten as

$$\frac{dN}{dt}=(Q_0-\mu)N-\alpha A \tag{2.11}$$

$$\frac{dI_1}{dt}=\frac{c(\beta_1I_1+\beta_2I_2)(N-I_1-I_2-A)}{N}-(\delta+\theta+\mu)I_1 \tag{2.12}$$

$$\frac{dI_2}{dt}=\theta I_1-(\delta+\mu)I_2 \tag{2.13}$$

$$\frac{dA}{dt}=\delta(I_1+I_2)-(\alpha+\mu)A \tag{2.14}$$

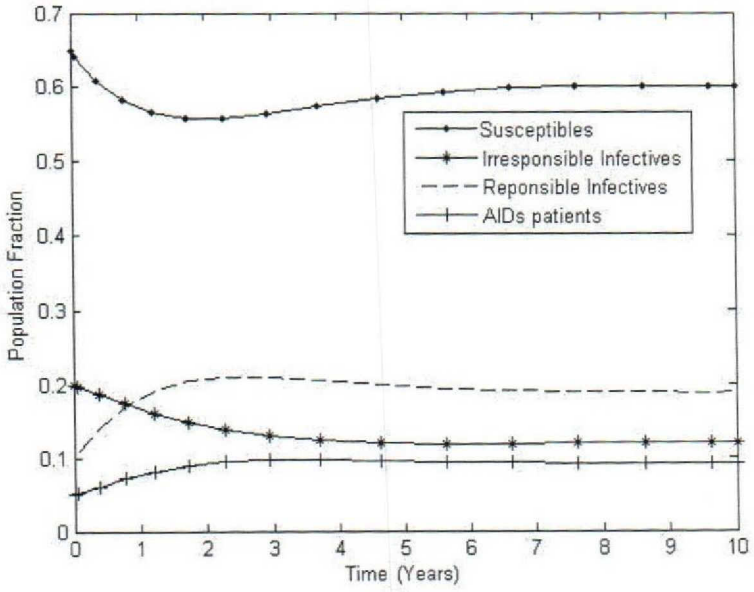

Figure 2.3: Variation of population in different classes for c = 25, $\theta = 0.95$

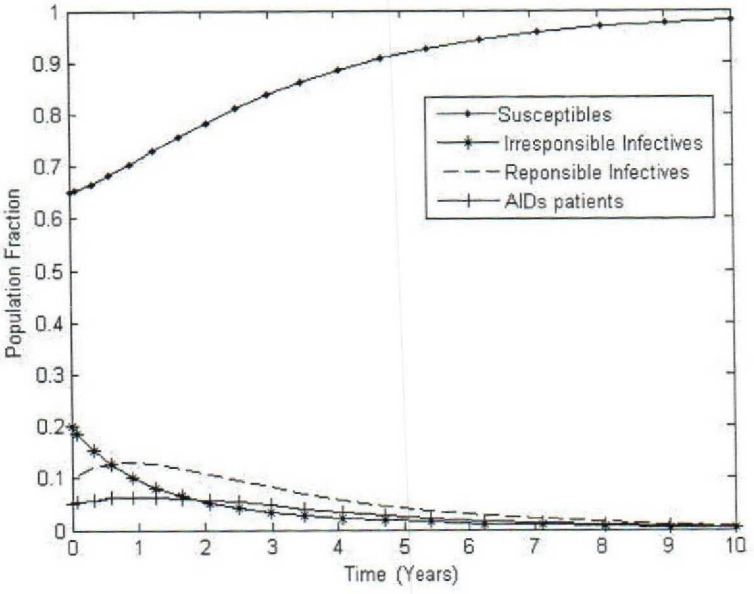

Figure 2.4: Variation of population in different classes for c = 10, $\theta = 0.75$

Let the endemic equilibrium be $E^* = (N^*, I_1^*, I_2^*, A)$

At equilibrium $\frac{dN^*}{dt} = \frac{dI_1^*}{dt} = \frac{dI_2^*}{dt} = \frac{dA^*}{dt} = 0$. Thus system (2.11)-(2.14) becomes

$$(Q_0 - \mu)N^* - \alpha A^* = 0 \tag{2.15}$$

$$\frac{c(\beta_1 I_1^* + \beta_2 I_2^*)(N^* - I_1^* - I_2^* - A^*)}{N^*} - (\delta + \theta + \mu)I_1^* = 0 \tag{2.16}$$

$$\theta I_1^* - (\delta + \mu)I_2^* = 0 \tag{2.17}$$

$$\delta(I_1^* + I_2^*) - (\alpha + \mu)A^* = 0 \tag{2.18}$$

From equation 2.17 we have $I_1^* = \frac{(\delta+\mu)I_2^*}{\theta}$

Substituting I_1 into 2.18 we have $\delta(\frac{(\delta+\mu)I_2^*}{\theta} + I_2^*) - (\alpha+\mu)A^* = 0$

$\Rightarrow \delta(\frac{(\delta+\mu)}{\theta} + 1)I_2^* - (\alpha+\mu)A^* = 0$

$\Rightarrow \delta(\frac{\delta+\mu+\theta}{\theta})I_2^* - (\alpha+\mu)A^* = 0$

$\Rightarrow (\alpha+\mu)A^* = \delta(\frac{\delta+\mu+\theta}{\theta})I_2^*$

$\therefore A^* = \frac{\delta(\delta+\mu+\theta)}{\theta(\alpha+\mu)}I_2^*$

Finally, substituting A^* into 2.15 we have $(Q_0 - \mu)N^* - \frac{\alpha\delta(\delta+\mu+\theta)}{\theta(\alpha+\mu)}I_2^* = 0$

$\therefore N^* = \frac{\alpha\delta(\delta+\mu+\theta)}{\theta(\alpha+\mu)(Q_0-\mu)}I_2^*$

Thus, the endemic equilibrium of the system 2.11-2.14 is given by

$N^* = \frac{\alpha\delta(\delta+\mu+\theta)}{\theta(\alpha+\mu)(Q_0-\mu)}I_2^*$

I_2^*

$I_1^* = \frac{(\delta+\mu)}{\theta}I_2^*$

$A^* = \frac{\delta(\delta+\mu+\theta)}{\theta(\alpha+\mu)}I_2^*$

2.5.5 Analytical Results

It is found that, the system's reproduction number, R_0, is given by

$R_0 = \frac{c\beta_1(Q_0+\delta)+c\beta_2\theta}{(Q_0+\delta+\theta)(Q_0+\delta)}$.

The system is stable if $R_0 < 1$ and the spread of the disease can even die out if $\beta_1 = \beta_2 = c = 0$. That is, when infectives present good characters and engage in safe sex, then the basic reproductive number is significantly reduced and can even approach zero. If however, $R_0 > 1$ then the system becomes unstable and the infection of the disease persists in the population. In this case HIV/AIDS infectives actively take part in the spread of the disease by sexual interaction in which $\beta_1 = \beta_2 =$

$c \neq 0$. Thus, the endemicity of the disease can be reduced when infectives present positive attitude towards preventive measures and do not spread the disease.

2.6 Numerical Simulation of the model

To observe the dynamics of the system, the model (2.5)-(2.8) is numerically integrated using the fourth order Runge-Kutta method using the following parameters

$\beta_2 = 0.015$, $\alpha = .5$, $\mu = 0.02$, $Q_0 = 0.40$, $\delta = 0.25$, $\beta_1 = .08$, $\theta = .955$ and $c = 10$

With initial conditions $s(0) = 0.65$, $i_1(0) = 0.20$, $i_2(0) = 0.10$ and $a(0) = 0.05$

Here, it must be noted that s, i_1, i_2 and a are fractions of which their sum should not exceed unity. Therefore the choice of the values of the initial conditions and the parameter values are done randomly just for the purpose of illustration of the model. It is however possible for the model to be implemented on observed data. The results of the computer simulations are shown in tables A.1 to table A.4 in appendix A and graphically displayed in figure 2.2 to figure 2.15

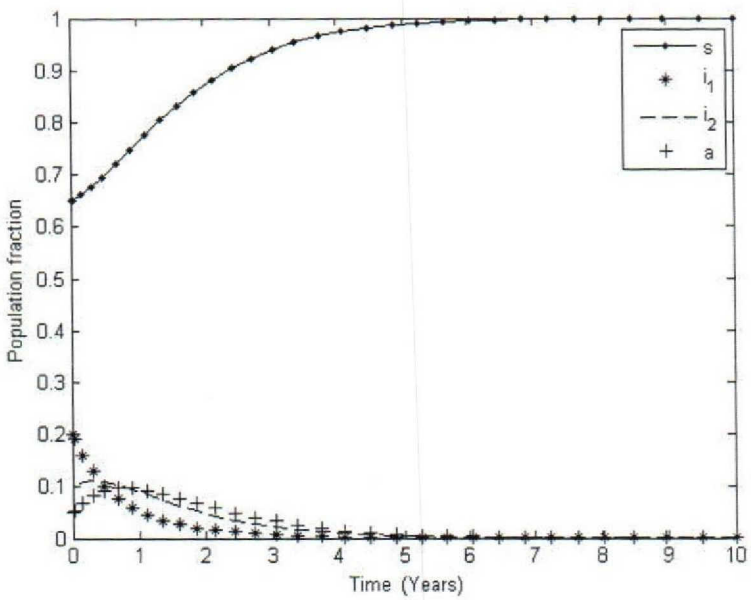

Figure 2.2: Variation of population in different classes for c = 10, $\theta = 0.95$

It is observed from figures 2.2 to 2.8 that increasing θ, the conversion rate of irresponsible infectives to responsible infectives, reduces both the irresponsible infectives population and AIDS patients population and increases the responsible infectives population. That is, increasing the rate

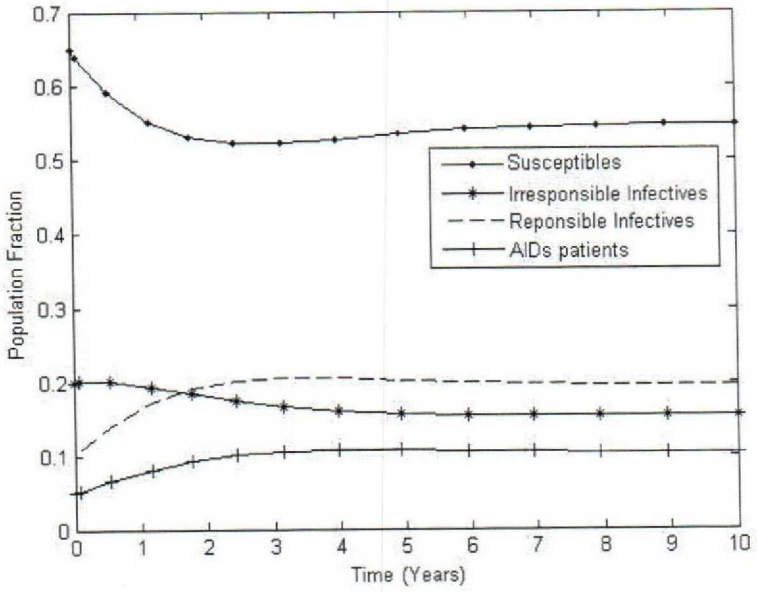

Figure 2.5: Variation of population in different classes for c = 25, $\theta = 0.75$

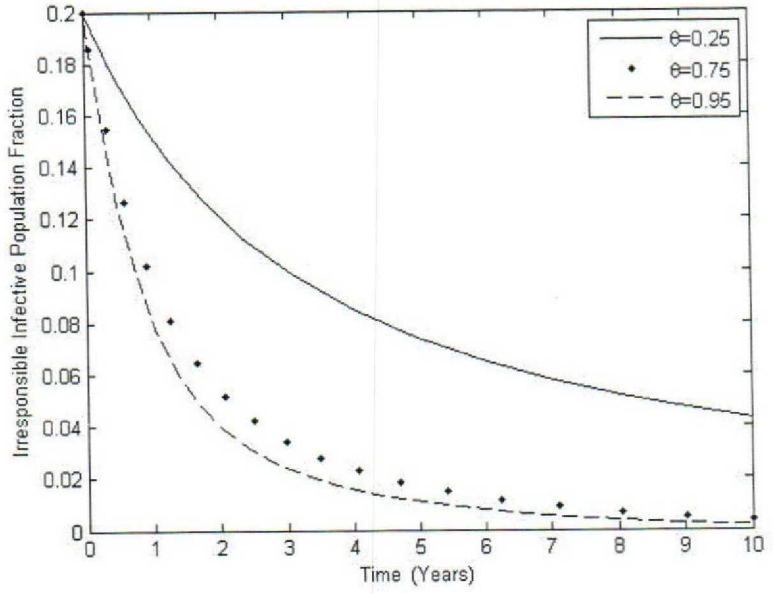

Figure 2.6: Variation of Irresponsible Infective population for different values of θ

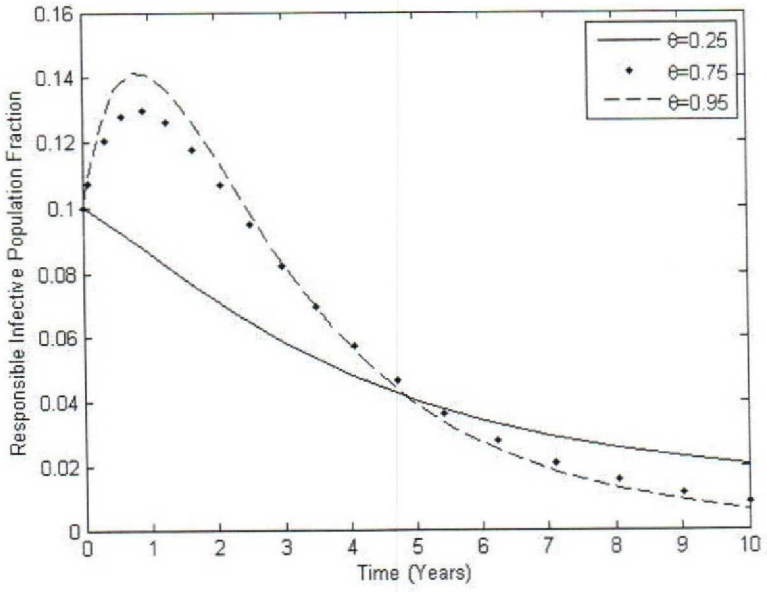

Figure 2.7: Variation of Responsible Infective population for different values of θ

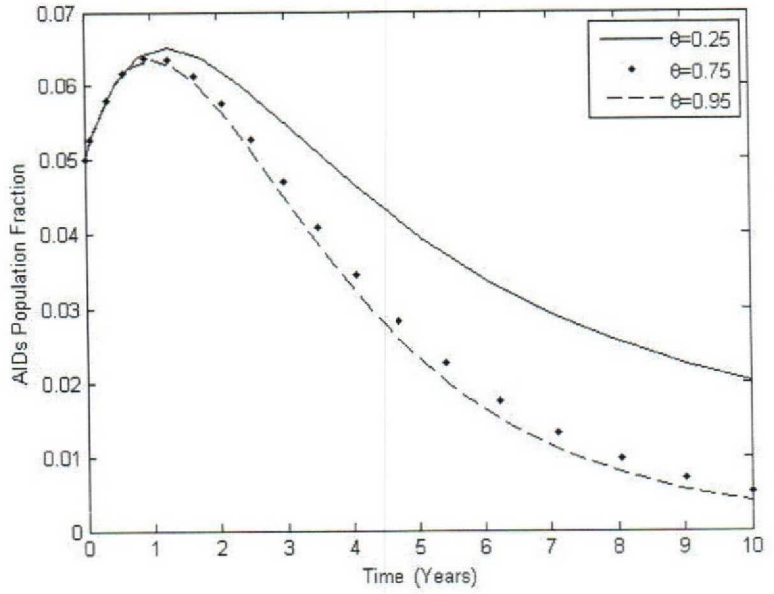

Figure 2.8: Variation of AIDS population for different values of θ

at which irresponsible infectives convert to responsible infectives results in more of the infectives becoming responsible and thus not spreading the disease. This goes to reduce the number of people contracting the disease. Thus, to help check the spread of the disease, policies that can lead to an increase in θ such as mass education, mass screening and the development of strategies that could lead to sexual inactivity of infectives, should be considered.

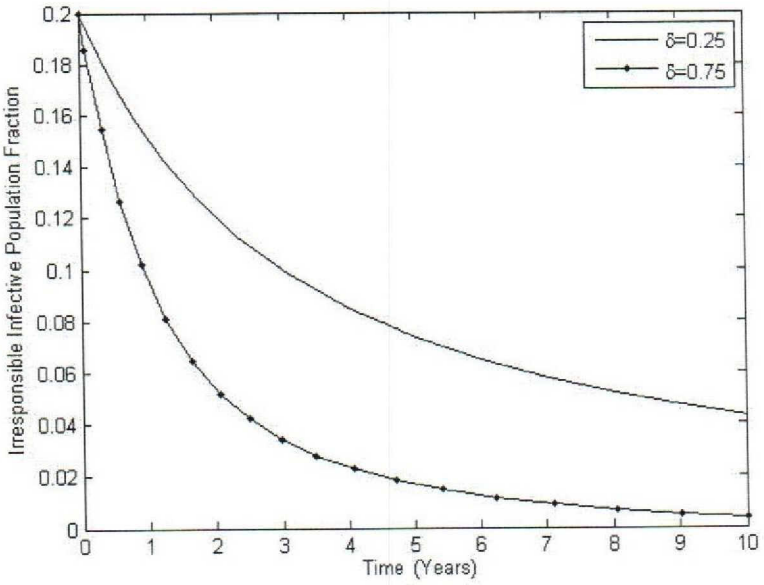

Figure 2.9: Variation of Irresponsible Infective population for different values of δ

Also, we observed from figures 2.9, 2.10 and 2.11 that increasing δ, the rate at which infectives convert to full-blown AIDS patients, reduces both the irresponsible and responsible infectives populations but increases the full-blown AIDS population. Thus, if infectives could be made to quickly develop AIDS, the spread of the disease could be checked and brought under control.

Again, it can be seen from figure 2.12 to figure 2.15 that increasing c, the number of sexual partners, results in a decrease in the susceptible population and an accompanying increase in the infective and AIDS populations. That is, if the number of sexual partners increases, there will be the tendency for more people to get infected with the virus. This will lead to a reduction in the susceptible population with a concomitant increase in the infective and AIDS populations.

Simulating R_0 under the prevailing values of the other parameters by varying c showed, as in table

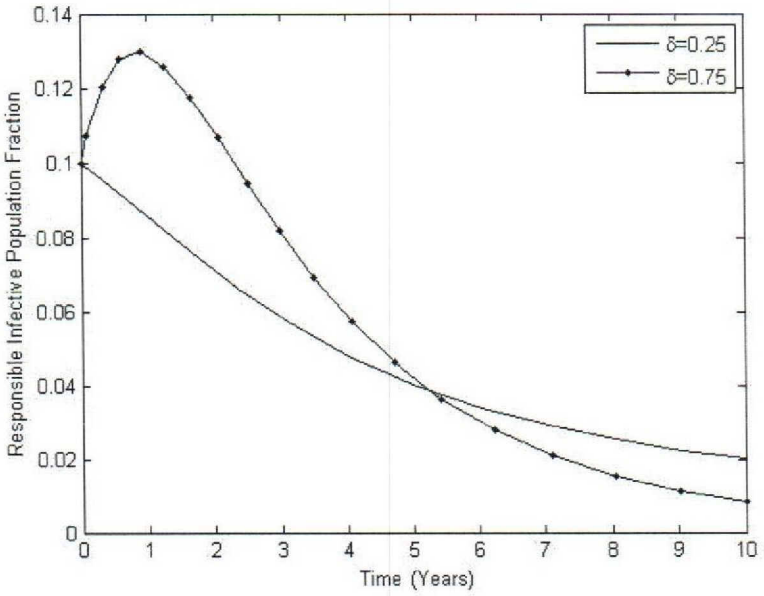

Figure 2.10: Variation of Responsible Infective population for different values of δ

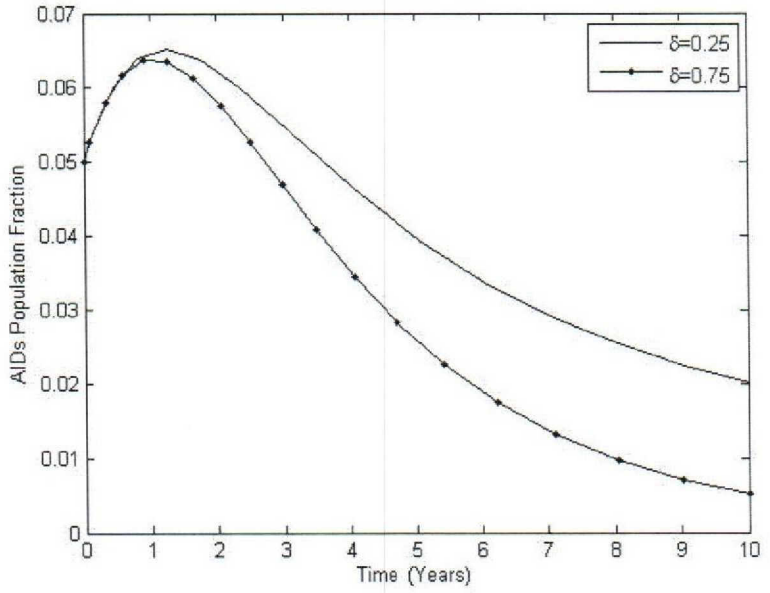

Figure 2.11: Variation of AIDS population for different values of δ

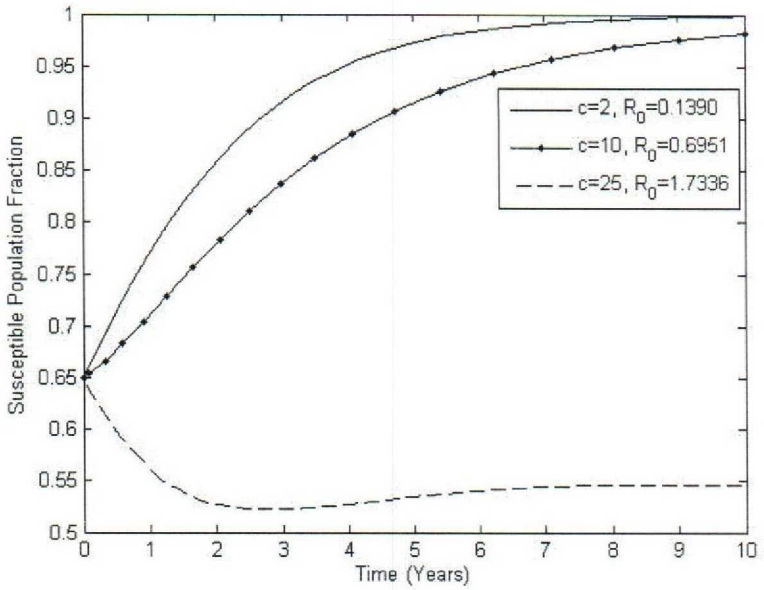

Figure 2.12: Variation of Susceptible population for different values of c

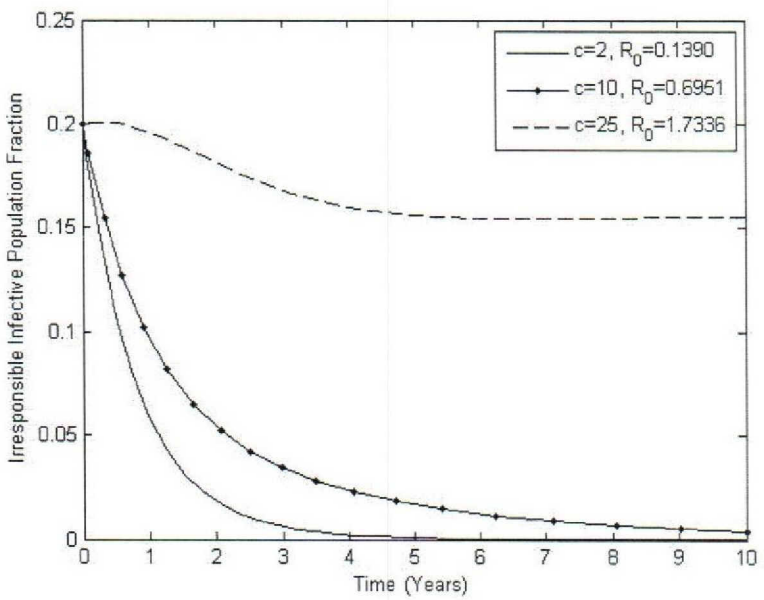

Figure 2.13: Variation of Irresponsible Infective population for different values of c

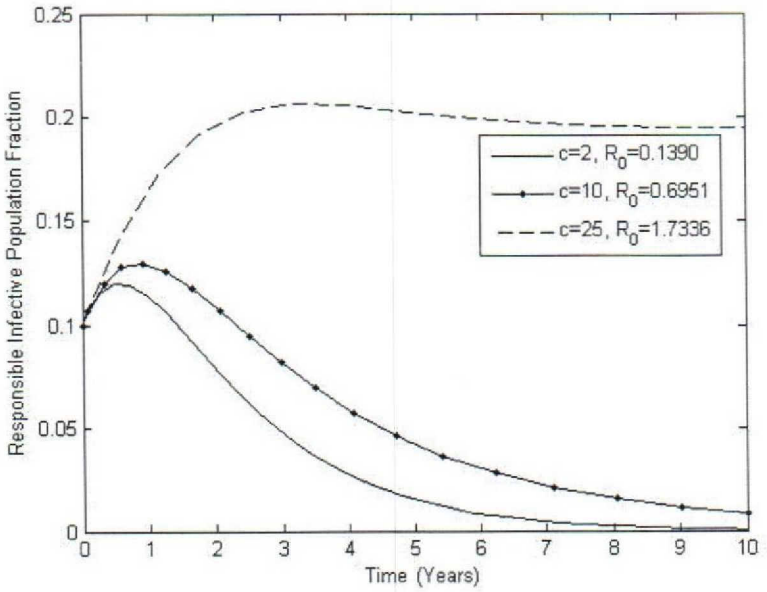

Figure 2.14: Variation of Responsible Infective population for different values of c

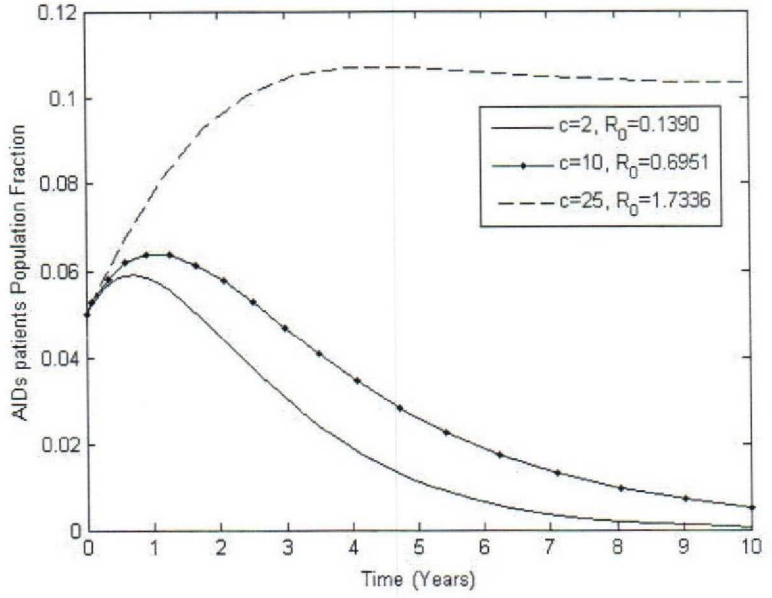

Figure 2.15: Variation of AIDS population for different values of c

A.4, that the number of sexual partners should not exceed 14 if we need to keep the system stable. Thus, measures should be kept in place to ensure that the number of sexual partners be kept to the bearest practicable minimal.

CHAPTER 3

Effects of infective immigrant on the transmission dynamics of HIV/AIDS

3.1 Introduction

In the world today, immigration poses a significant risk for disease dissemination including HIV/AIDS. The effect may be substantial in developing countries which normally do not check the complete health status of immigrants. These immigrants place their sex partners in their home countries and their destination countries at risk of the HIV/AIDS epidemic. This claim however has not been established and evaluated adequately to see the effect and exact mechanism that immigration contributes in the spread of HIV/AIDS. Coffee *et al* (2007) developed a model to study the impact of migration on the spread of HIV/AIDS in South Africa using observed data. This chapter looks at a nonlinear mathematical model that has been proposed and analyzed to study the effect of infective immigrants in the spread of HIV/AIDS in a population.

3.2 Model formulation and Assumption

3.2.1 Assumptions

The following assumptions are made in order to construct the mathematical model for the problem

1. The population under study is heterogeneous and varying with time
2. The population under study is subdivided into four groups

3. The HIV can only be transmitted through sexual intercourse or through infection from infected needle and blood.
4. The full-blown AIDS class is sexually inactive
5. The rate at which irresponsible infectives infect people with the disease is higher than that of responsible infectives

3.2.2 Description of model parameters

γ = Rate of recruitment of infective immigrants into the population. All other parameters are as defined in chapter two

3.3 Model Formulation

In modeling the effect of infective immigrants on the population, we consider a modification of the model in chapter two by incorporating the recruitment of infective immigrants into the irresponsible infective population. The assumption is that infectives coming into the population might not know their HIV status and have the tendency of behaving irresponsibly. It is also assumed that the recruitment of susceptible individuals is at a constant rate Q_0. The modified flowchart from figure 2.1 in chapter two is shown in figure 3.1 below.

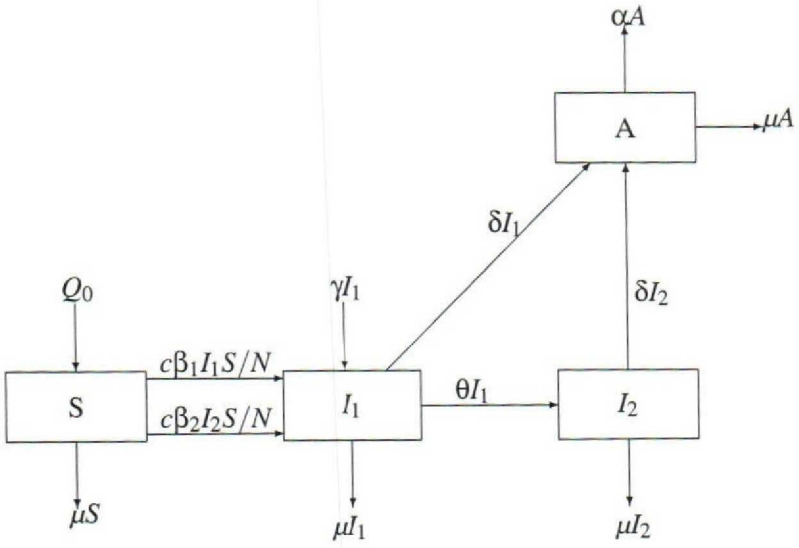

Figure 3.1: Proposed Flowchart for infective immigrants model

From figure 3.1, the proposed differential equations representing the model are given by;

$$\frac{dS}{dt} = Q_0 - \frac{c(\beta_1 I_1 + \beta_2 I_2)S}{N} - \mu S \tag{3.1}$$

$$\frac{dI_1}{dt} = \frac{c(\beta_1 I_1 + \beta_2 I_2)}{N} S + \gamma I_1 - (\theta + \delta + \mu) I_1 \tag{3.2}$$

$$\frac{dI_2}{dt} = \theta I_1 - (\delta + \mu) I_2 \tag{3.3}$$

$$\frac{dA}{dt} = \delta(I_1 + I_2) - (\alpha + \mu) A \tag{3.4}$$

With initial conditions given by $S(0) = S_0\ I_1(0) = I_{10}\ I_2(0) = I_{20}\ A(0) = A_0\ \beta_1 > \beta_2$.

If the total population size is given by $N = S + I_1 + I_2 + A$, then we have $\frac{dN}{dt} = (Q_0 - \mu)N - \alpha A + \gamma I_1$ and the model (3.3)-(3.4) can be re-writen as follows

$$\frac{dN}{dt} = Q_0 - \mu N - \alpha A + \gamma I_1 \tag{3.5}$$

$$\frac{dI_1}{dt} = \frac{c(\beta_1 I_1 + \beta_2 I_2)(N - I_1 - I_2 - A)}{N} + \gamma I_1 - (\theta + \delta + \mu) I_1 \tag{3.6}$$

$$\frac{dI_2}{dt} = \theta I_1 - (\delta + \mu) I_2 \tag{3.7}$$

$$\frac{dA}{dt} = \delta(I_1 + I_2) - (\alpha + \mu) A \tag{3.8}$$

With initial conditions given by $N(0) = N_0\ I_1(0) = I_{10}\ I_2(0) = I_{20}\ A(0) = A_0\ \beta_1 > \beta_2$.

3.4 Qualitative Analysis

3.4.1 Equilibrium Analysis

The system exhibits two equilibria; the disease-free equilibrium and the endemic equilibrium.

3.4.1.1 Disease-free Equilibrium

At the disease-free equilibrium, there are no infectives and full-blown aids patients. Hence $I_1 = I_2 = A = 0$ and $N = \frac{Q_0}{\mu}$. Hence the disease-free equilibrium is $E_0 = (\frac{Q_0}{\mu}, 0, 0, 0)$

3.4.1.2 Endemic equilibrium

Let the endemic equilibrium be $E^* = (N^*, I_1^*, I_2^*, A^*)$.

Note that at equilibrium, $\frac{dN}{dt} = \frac{dI_1}{dt} = \frac{dI_2}{dt} = \frac{dA}{dt} = 0$.

Hence system (3.5)-(3.8) becomes

$$Q_0 - \mu N^* - \alpha A^* + \gamma I_1^* = 0 \tag{3.9}$$

$$\frac{c(\beta_1 I_1^* + \beta_2 I_2^*)(N^* - I_1^* - I_2^* - A^*)}{N^*} + \gamma I_1^* - (\theta + \delta + \mu) I_1^* = 0 \tag{3.10}$$

$$\theta I_1^* - (\delta + \mu) I_2^* = 0 \tag{3.11}$$

$$\delta(I_1^* + I_2^*) - (\alpha + \mu) A^* = 0 \tag{3.12}$$

From equation (3.11) we have

$I_2^* = \frac{\theta}{\delta+\mu} I_1^*$.

Substituting I_2^* into equation (3.12) we have

$\delta(I_1^* + \frac{\theta}{\delta+\mu} I_1^*) - (\alpha+\mu)A^* = 0$

$\Rightarrow \frac{\delta(\theta+\delta+\mu)}{\delta+\mu} I_1^* - (\alpha+\mu)A^* = 0$

$\therefore A^* = \frac{\delta(\theta+\delta+\mu)}{(\delta+\mu)(\alpha+\mu)} I_1^*$

Putting I_2^* and A^* into equation (3.10) we have:

$\frac{c(\beta_1 I_1 + \beta_2 I_2)(N^* - I_1^* - I_2^* - A^*)}{N^*} + \gamma I_1^* - (\theta+\delta+\mu) I_1^* = 0$

$\frac{c(\beta_1 I_1 + \beta_2 I_2)(N^* - I_1^* - I_2^* - A^*)}{N^*} + \gamma I_1^* - (\theta+\delta+\mu) I_1^* = 0$

$\frac{c(\beta_1 + \beta_2 \frac{\theta}{\delta+\mu})(N^* - I_1^* - I_2^* - A^*)}{N^*} I_1^* + \gamma I_1^* - (\theta+\delta+\mu) I_1^* = 0$

$\Rightarrow \frac{c(\beta_1 + \beta_2 \frac{\theta}{\delta+\mu})(N^* - I_1^* - I_2^* - A^*)}{N^*} + \gamma - (\theta+\delta+\mu) = 0$

$\Rightarrow c(\beta_1 + \beta_2 \frac{\theta}{\delta+\mu})(N^* - I_1^* - I_2^* - A^*) + N^*(\gamma - \theta - \delta - \mu) = 0$

$\Rightarrow c(\frac{\beta_1(\delta+\mu) + \beta_2\theta}{\delta+\mu})(N^* - I_1^* - I_2^* - A^*) + N^*(\gamma - \theta - \delta - \mu) = 0$

$\Rightarrow c(\beta_1(\delta+\mu) + \beta_2\theta)(N^* - I_1^* - I_2^* - A^*) + N^*(\gamma - \theta - \delta - \mu)(\delta+\mu) = 0$

$$\Rightarrow c(\beta_1(\delta+\mu)+\beta_2\theta)(-I_1^*-I_2^*-A^*)+N^*[c\beta_1(\delta+\mu)+c\beta_2\theta+(\gamma-\theta-\delta-\mu)(\delta+\mu)]=0$$

$$\Rightarrow N^*[c\beta_1(\delta+\mu)+c\beta_2\theta+(\gamma-\theta-\delta-\mu)(\delta+\mu)]=c(\beta_1(\delta+\mu)+\beta_2\theta)(I_1^*+I_2^*+A^*)$$

$$\Rightarrow N^*[c\beta_1(\delta+\mu)+c\beta_2\theta+(\gamma-\theta-\delta-\mu)(\delta+\mu)]=c[\beta_1(\delta+\mu)+\beta_2\theta][I_1^*+\tfrac{\theta}{\delta+\mu}I_1^*+\tfrac{\delta(\theta+\delta+\mu)}{(\delta+\mu)(\alpha+\mu)}I_1^*]$$

$$\Rightarrow N^*[c\beta_1(\delta+\mu)+c\beta_2\theta+(\gamma-\theta-\delta-\mu)(\delta+\mu)]=c[\beta_1(\delta+\mu)+\beta_2\theta][1+\tfrac{\theta}{\delta+\mu}+\tfrac{\delta(\theta+\delta+\mu)}{(\delta+\mu)(\alpha+\mu)}]I_1^*$$

$$\Rightarrow N^*[c\beta_1(\delta+\mu)+c\beta_2\theta+(\gamma-\theta-\delta-\mu)(\delta+\mu)]=c[\beta_1(\delta+\mu)+\beta_2\theta][\tfrac{(\delta+\mu)(\alpha+\mu)+\theta(\alpha+\mu)+\delta(\theta+\delta+\mu)}{(\delta+\mu)(\alpha+\mu)}]I_1^*$$

$$\Rightarrow N^*[c\beta_1(\delta+\mu)+c\beta_2\theta+(\gamma-\theta-\delta-\mu)(\delta+\mu)]=c[\beta_1(\delta+\mu)+\beta_2\theta][\tfrac{(\theta+\delta+\mu)(\alpha+\mu)+\delta(\theta+\delta+\mu)}{(\delta+\mu)(\alpha+\mu)}]I_1^*$$

$$\Rightarrow N^*[c\beta_1(\delta+\mu)+c\beta_2\theta+(\gamma-\theta-\delta-\mu)(\delta+\mu)]=c[\beta_1(\delta+\mu)+\beta_2\theta][\tfrac{(\theta+\delta+\mu)(\alpha+\mu+\delta)}{(\delta+\mu)(\alpha+\mu)}]I_1^*$$

$$\Rightarrow N^*[c\beta_1(\delta+\mu)+c\beta_2\theta+(\gamma-\theta-\delta-\mu)(\delta+\mu)]=c[\beta_1(\delta+\mu)+\beta_2\theta][\tfrac{(\theta+\delta+\mu)(\alpha+\mu+\delta)}{(\delta+\mu)(\alpha+\mu)}]I_1^*$$

$$\therefore N^*=\tfrac{c[\beta_1(\delta+\mu)+\beta_2\theta](\theta+\delta+\mu)(\alpha+\mu+\delta)}{(\delta+\mu)(\alpha+\mu)\xi}I_1^*$$

Where $\xi=c\beta_1(\delta+\mu)+c\beta_2\theta+(\gamma-\theta-\delta-\mu)(\delta+\mu)$

From equation (3.9) we have

$$Q_0-\mu N^*-\alpha A^*+\gamma I_1^*=0$$

Substituting N^* and A^* we have:

$$Q_0-\tfrac{c\mu[\beta_1(\delta+\mu)+\beta_2\theta](\theta+\delta+\mu)(\alpha+\mu+\delta)}{(\delta+\mu)(\alpha+\mu)\xi}I_1^*-\tfrac{\alpha\delta(\theta+\delta+\mu)}{(\delta+\mu)(\alpha+\mu)}I_1^*+\gamma I_1^*=0$$

$$\Rightarrow Q_0-\{\tfrac{c\mu[\beta_1(\delta+\mu)+\beta_2\theta](\theta+\delta+\mu)(\alpha+\mu+\delta)}{(\delta+\mu)(\alpha+\mu)\xi}+\tfrac{\alpha\delta(\theta+\delta+\mu)}{(\delta+\mu)(\alpha+\mu)}-\gamma\}I_1^*=0$$

$$\Rightarrow \tfrac{c\mu[\beta_1(\delta+\mu)+\beta_2\theta](\theta+\delta+\mu)(\alpha+\mu+\delta)+\alpha\delta(\theta+\delta+\mu)\xi-(\delta+\mu)(\alpha+\mu)\xi\gamma}{(\delta+\mu)(\alpha+\mu)\xi}I_1^*=Q_0$$

$$\therefore I_1^*=\frac{Q_0(\delta+\mu)(\alpha+\mu)\xi}{c\mu[\beta_1(\delta+\mu)+\beta_2\theta](\theta+\delta+\mu)(\alpha+\mu+\delta)+\alpha\delta(\theta+\delta+\mu)\xi-(\delta+\mu)(\alpha+\mu)\xi\gamma}$$

Thus, the model (3.5)-(3.8) has two equilibria namely the disease-free equilibrium $E_0=(Q_0/\mu,0,0,0)$ and the endemic equilibrium $E^*=(N^*,I_1^*,I_2^*,A^*)$.

Where

$$N^*=\tfrac{c[\beta_1(\delta+\mu)+\beta_2\theta](\theta+\delta+\mu)(\alpha+\mu+\delta)}{(\delta+\mu)(\alpha+\mu)\xi}I_1^*$$

$$I_1^*=\frac{Q_0(\delta+\mu)(\alpha+\mu)\xi}{c\mu[\beta_1(\delta+\mu)+\beta_2\theta](\theta+\delta+\mu)(\alpha+\mu+\delta)+\alpha\delta(\theta+\delta+\mu)\xi-(\delta+\mu)(\alpha+\mu)\xi\gamma}$$

$$I_2^*=\tfrac{\theta}{\delta+\mu}I_1^*.$$

$$A^*=\tfrac{\delta(\theta+\delta+\mu)}{(\delta+\mu)(\alpha+\mu)}I_1^*$$

Where $\xi=c\beta_1(\delta+\mu)+c\beta_2\theta+(\gamma-\theta-\delta-\mu)(\delta+\mu)$

We note here that E^* is positive only when $\xi>0$ or $R_0>1$, and $\alpha\delta(\theta+\delta+\mu)-(\delta+\mu)(\alpha+\mu)\gamma>0$

Where $R_0 = \frac{(\gamma+c\beta_1)(\delta+\mu)+c\beta_2\theta}{(\theta+\delta+\mu)(\delta+\mu)}$

3.4.2 Local stability analysis

To determine the local stability of the equilibria, we evaluate the jacobian matrix, M, of the system (3.5)-(3.8)

$$M = \begin{bmatrix} -\mu & \gamma & 0 & -\alpha \\ m_{21} & m_{22} & m_{23} & m_{24} \\ 0 & \theta & -\delta-\mu & 0 \\ 0 & \delta & \delta & -\alpha-\mu \end{bmatrix} \tag{3.13}$$

Where

$$m_{21} = \frac{c(\beta_1 I_1+\beta_2 I_2)}{N} - \frac{c(\beta_1 I_1+\beta_2 I_2)(N-I_1-I_2-A)}{N^2} = \frac{c(\beta_1 I_1+\beta_2 I_2)(I_1+I_2+A)}{N^2} > 0$$

$$m_{22} = \frac{c\beta_1 (N-I_1-I_2-A)}{N} - \frac{c(\beta_1 I_1+\beta_2 I_2)}{N} + (\gamma-\theta-\delta-\mu)$$

$$m_{23} = \frac{c\beta_2 (N-I_1-I_2-A)}{N} - \frac{c(\beta_1 I_1+\beta_2 I_2)}{N}$$

$$m_{24} = -\frac{c(\beta_1 I_1+\beta_2 I_2)}{N}$$

Let $q = \frac{c(\beta_1+\beta_2 I_2)}{N}$, $r = \frac{c\beta_2 (N-I_1-I_2-A)}{N}$ and $p = \frac{c\beta_1 (N-I_1-I_2-A)}{N} + (\gamma-\theta-\delta-\mu)$

Then $m_{21} = \frac{q(I_1+I_2+A)}{N} > 0$, $m_{22} = -(p+q) < 0$, $m_{23} = r-q$, $m_{24} = -q < 0$

3.4.2.1 Local stability analysis of E_0

The Jacobian matrix corresponding to E_0 is given by

$$M_0 = \begin{bmatrix} -\mu & \gamma & 0 & -\alpha \\ 0 & c\beta_1-(\theta+\delta+\mu-\gamma) & c\beta_2 & 0 \\ 0 & \theta & -(\delta+\mu) & 0 \\ 0 & \delta & \delta & -(\alpha+\mu) \end{bmatrix} \tag{3.14}$$

The characteristic equation corresponding to M_0 is given by

$$f(\lambda) = \begin{vmatrix} -\mu-\lambda & \gamma & 0 & -\alpha \\ 0 & c\beta_1-(\theta+\delta+\mu-\gamma)-\lambda & c\beta_2 & 0 \\ 0 & \theta & -(\delta+\mu)-\lambda & 0 \\ 0 & \delta & \delta & -(\alpha+\mu)-\lambda \end{vmatrix} = 0$$

$$\Rightarrow f(\lambda) = -(\mu+\lambda)\begin{vmatrix} c\beta_1-(\theta+\delta+\mu-\gamma)-\lambda & c\beta_2 & 0 \\ \theta & -(\delta+\mu)-\lambda & 0 \\ \delta & \delta & -(\alpha+\mu)-\lambda \end{vmatrix} = 0$$

$$\Rightarrow f(\lambda) = (\mu+\lambda)(\alpha+\mu+\lambda)\begin{vmatrix} c\beta_1-(\theta+\delta+\mu-\gamma)-\lambda & c\beta_2 \\ \theta & -(\delta+\mu)-\lambda \end{vmatrix} = 0$$

$$\Rightarrow f(\lambda) = (\mu+\lambda)(\alpha+\mu+\lambda)[(\delta+\mu+\lambda)(\theta+\delta+\mu-\gamma-c\beta_1+\lambda)-c\beta_2\theta] = 0$$

$$\Rightarrow f(\lambda) = (\mu+\lambda)(\alpha+\mu+\lambda)(\lambda^2+\nu\lambda+\rho)$$

Where

$\nu = (\delta+\mu)+(\theta+\delta+\mu-\gamma-c\beta_1)$ and $\rho = (\delta+\mu)(\theta+\delta+\mu-\gamma-c\beta_1)-c\beta_2\theta$

We note that E_0 is locally asymptotically stable when $\nu > 0$ and $\rho > 0$.

However, the condition (3.15) is sufficient to make E_0 locally asymptotically stable.

$$(\delta+\mu)(\theta+\delta+\mu) > (\delta+\mu)(\gamma+c\beta_1)+c\beta_2\theta \tag{3.15}$$

It is clear that for $R_0 < 1$ which corresponds to the condition (3.15), the disease-free equilibrium is locally asymptotically stable so that infection fades out from the population and thus the endemic equilibrium does not exist. However, for $R_0 > 1$ E_0 is unstable and then the infection is maintained in the population.

3.4.3 Analytical Results

If we write equation 3.15 as $R_0 < \frac{(\gamma+c\beta_1)(\delta+\mu)+c\beta_2\theta}{(\theta+\delta+\mu)(\delta+\mu)}$, then it is clear that $\beta_1 = \beta_2 = \theta = c = 0$ does not show the disappearance of the disease from the system as in the case of chapter two. This is due to the presence of γ, the recruitment rate of infective immigrants in the expression. This implies that even if infectives show responsible character by engaging in safe sex and do not spread the disease, but the presence of infective immigrants will maintain the disease in the population. Therefore as part of efforts to keep the spread of HIV/AIDS on check, the screening of immigrants into the population should be encouraged by policy makers. Thus, a reduction in the value of γ can reduce the value of R_0 thereby making the system more stable. That is, the spread of the disease can be reduced. Hence the spread of the disease can die out of the population if $\beta_1 = \beta_2 = \theta = c = \gamma = 0$.

3.5 Numerical Simulation of the model

To observe the dynamics of the system, the model (3.5)-(3.8) is numerically integrated using the fourth order Runge-Kutta method using the parameters

$Q_0 = 2000$, $\mu = 0.02$, $\beta_1 = 0.08$, $\beta_2 = 0.015$, $\alpha = 1$, $\delta = 0.75$, $c = 10$, $\gamma = 0.97$ and $\theta = 0.95$

with initial values $N(0) = 15000$, $I_1(0) = 10000$, $I_2(0) = 2000$ and $A(0) = 500$

The endemic equilibrium values are computed as

$N^* = 33896$, $I_1^* = 1909$, $I_2^* = 2355$ and $A^* = 3135$

It must be noted that the above parameter values and the initial conditions were chosen at random for illustration purposes as in the case of most modeling problems. It is however possible for the model to be implemented on observed data. The computer simulations are performed for different initial values in the following four cases as shown in table 3.1.

Table 3.1: Different initial conditions used for simulation

Case	N(0)	$I_1(0)$	$I_2(0)$	A(0)
1.	15000	10000	2000	500
2.	19000	16500	2000	100
3.	15000	3000	2500	1000
4.	10000	8000	1800	300

The results of the computer simulations are graphically displayed in figures 3.2 to 3.16.

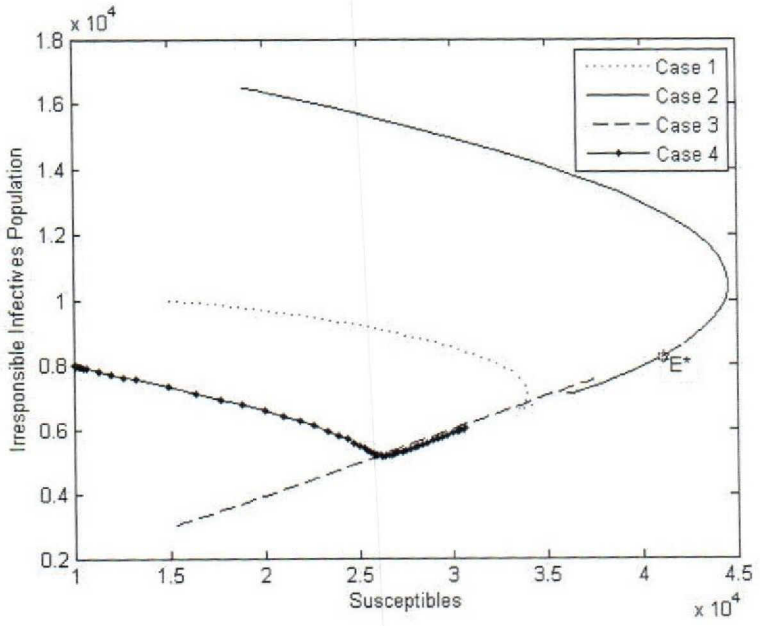

Figure 3.2: Variation of Irresponsible infectives population against Susceptible population

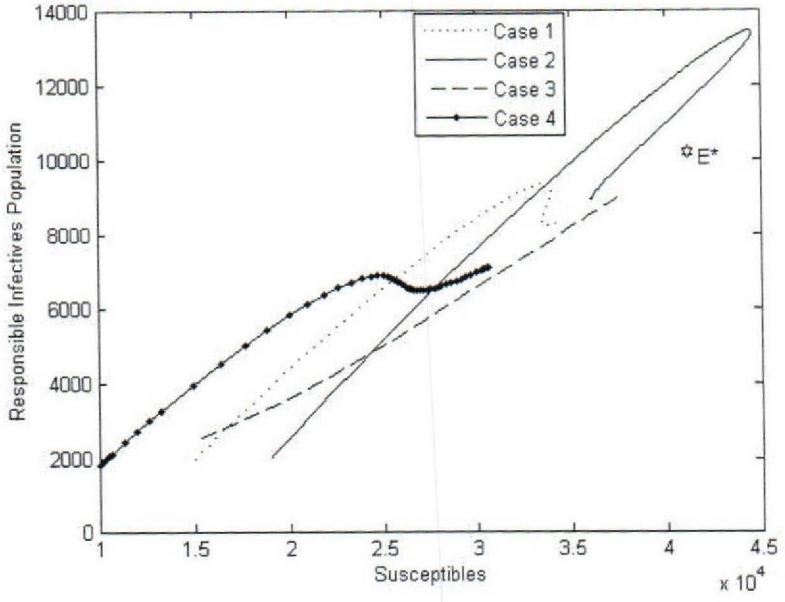

Figure 3.3: Variation of Responsible infectives population against Susceptible population

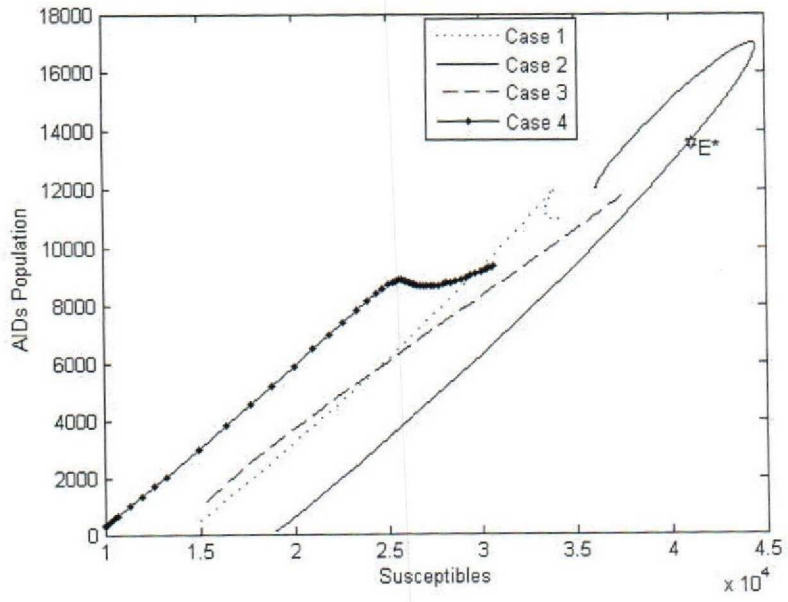

Figure 3.4: Variation of AIDS patients population against Susceptible population

In figures 3.2, 3.3 and 3.4 the irresponsible infectives population, responsible infectives population and AIDS patients population are plotted against the susceptible population respectively for various initial values. It is seen from these figures that irrespective of the initial conditions chosen, the solutions curves all always tend towards the equilibrium point E^*. This implies that the system (3.5)-(3.8) is globally stable about the endemic equilibrium point E^* .

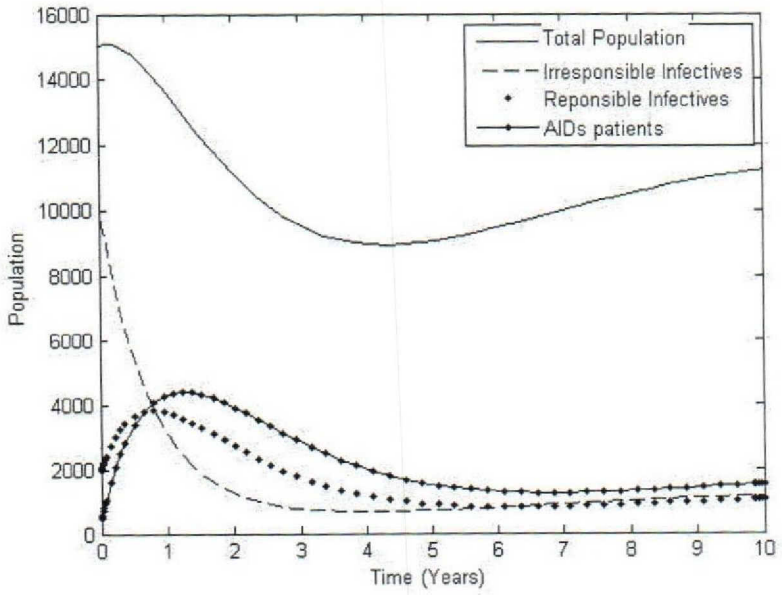

Figure 3.5: Variation of population classes without Infective immigrants ($\gamma = 0,\ Q_0 = 2000$)

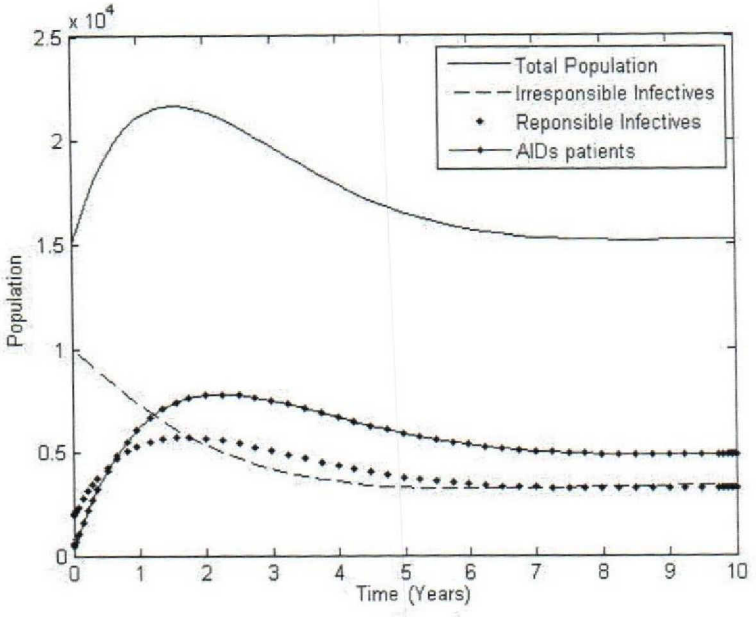

Figure 3.6: Variation of population classes with Infective immigrants ($\gamma = 0.95,\ Q_0 = 2000$)

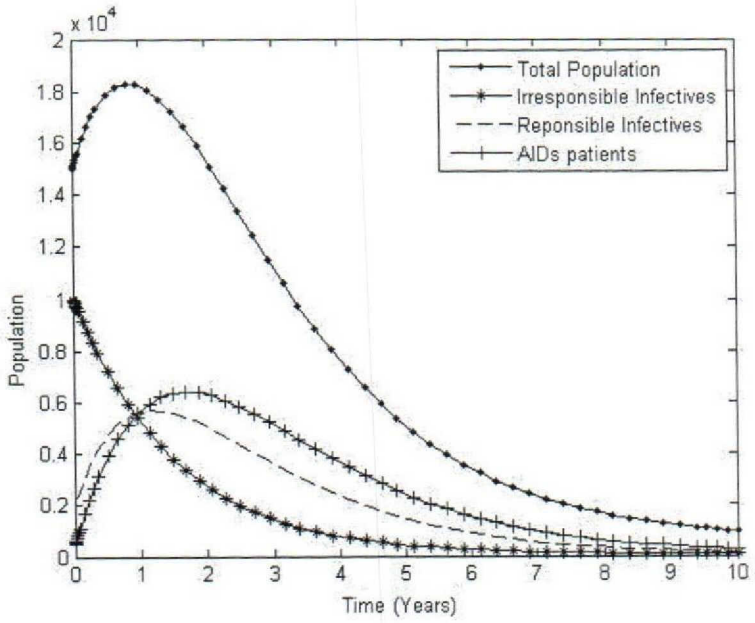

Figure 3.7: Variation of population classes with Infective immigrants ($\gamma = 0.97$, $Q_0 = 0$)

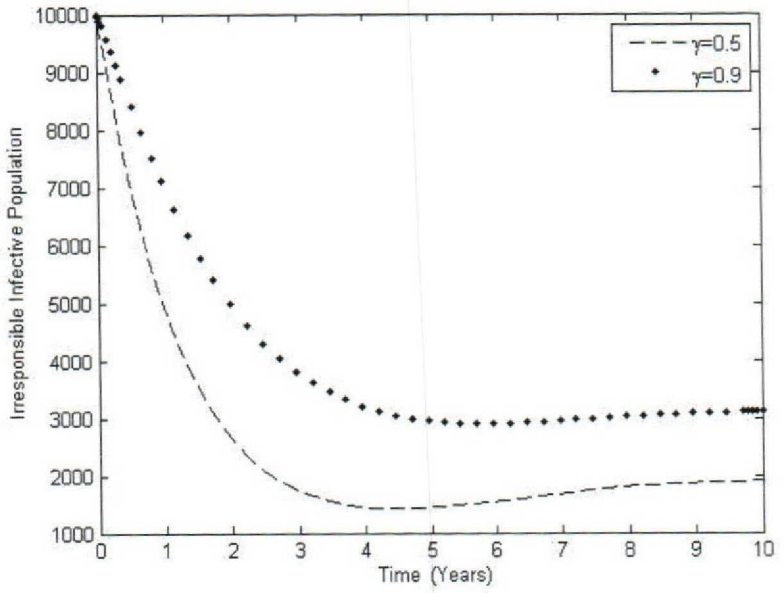

Figure 3.8: Variation of Irresponsible Infective population for different values of γ

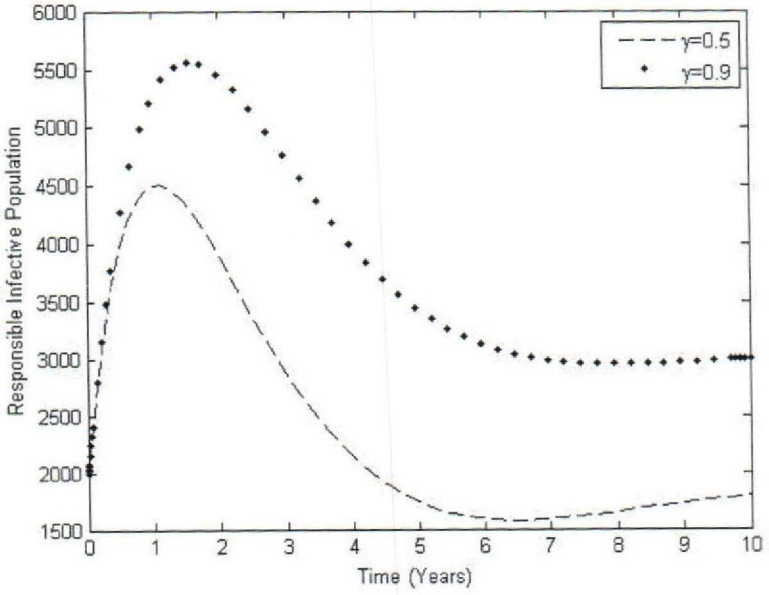

Figure 3.9: Variation of Responsible Infective population for different values of γ

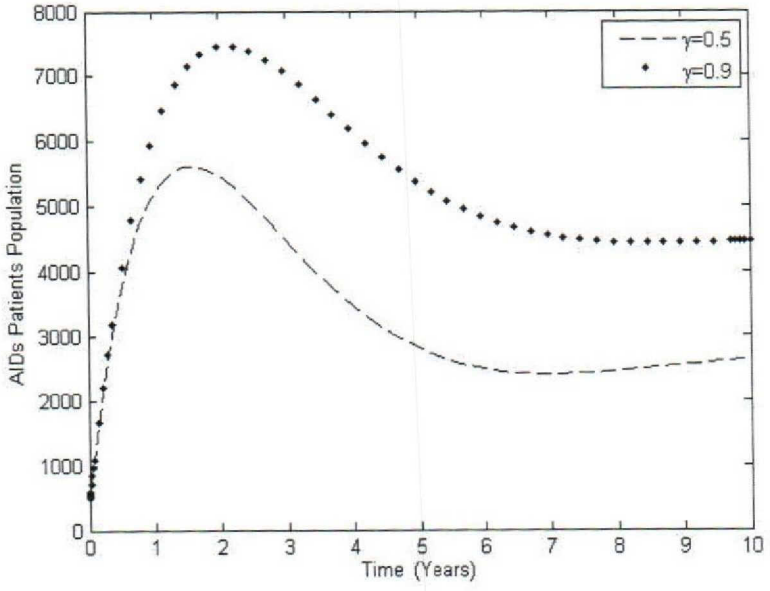

Figure 3.10: Variation of AIDS population for different values of γ

It is also seen from figure 3.5 to figure 3.10 that the presence of infective immigrants in the system increases the total population initially. But as time goes on more susceptible individuals get infected with the disease and then develop into AIDS. They eventually die thereby reducing the total population in the long-run. In the case of figure 3.7 there are no recruitment of susceptibles into the population. Therefore, the total population, the infected classes and the AIDS class all initially increase but later reduce to zero. Therefore, in order to minimize the spread of the disease and prevent the total population from being wiped away as in the case of figure 3.7, effective immigration policies such as screening should be put in place.

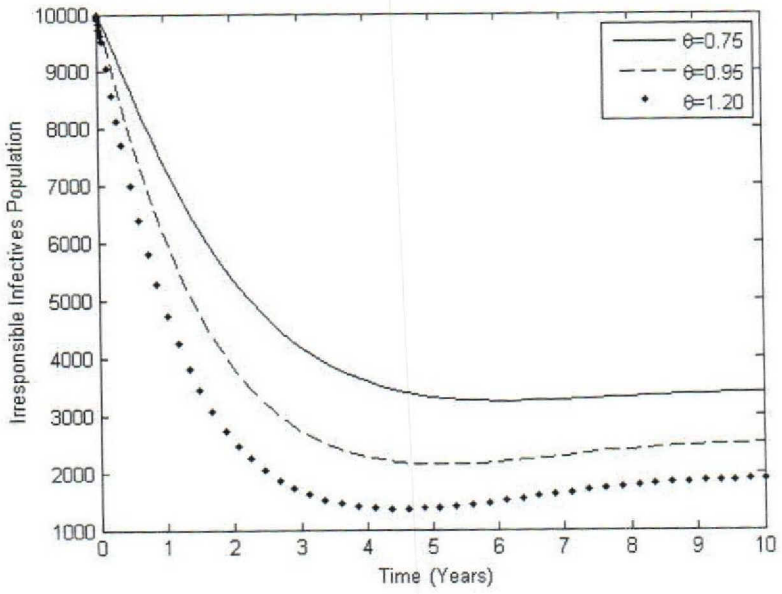

Figure 3.11: Variation of Irresponsible Infective population for different values of θ

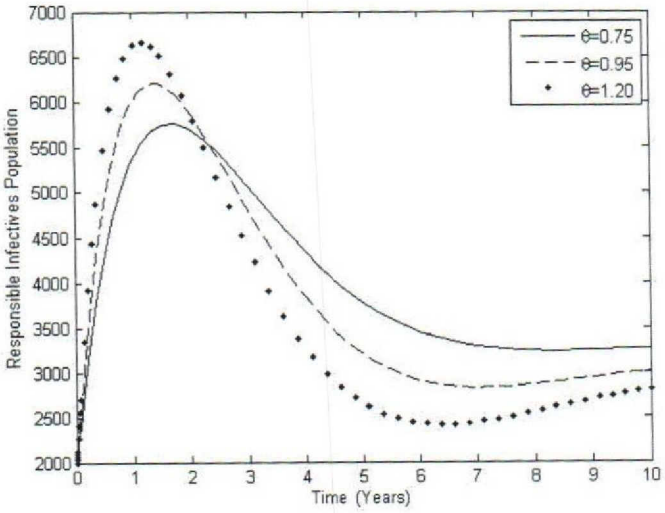

Figure 3.12: Variation of Responsible Infective population for different values of θ

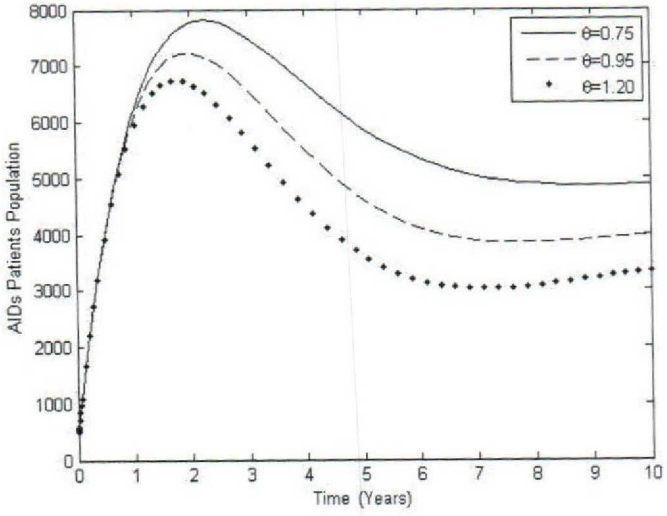

Figure 3.13: Variation of AIDS population for different values of θ

From figures 3.11 to figure 3.13 it can be observed that increasing θ, the conversion rate of irresponsible infectives to responsible infectives, reduces both the irresponsible infectives and the AIDS patients population. However, the responsible infectives population increases with increasing θ in the short-term but decreases with increasing θ in the long-term. That is, increasing θ will lead to more of the irresponsible infectives becoming responsible. This reduces the irresponsible infective population and consequently reduces the AIDS patients population. The responsible

infectives population will at first increase,but as less people get infected due to the reduced irresponsible infectives, the long term result will be a total reduction in the infectives (both irresponsible and responsible). Thus, efforts should be made towards increasing the rate of conversion of irresponsible infectives to responsible infectives. Such efforts could be mass education, mass screening and some other "radical" ways like administration of anaphrodisiacs to infectives.

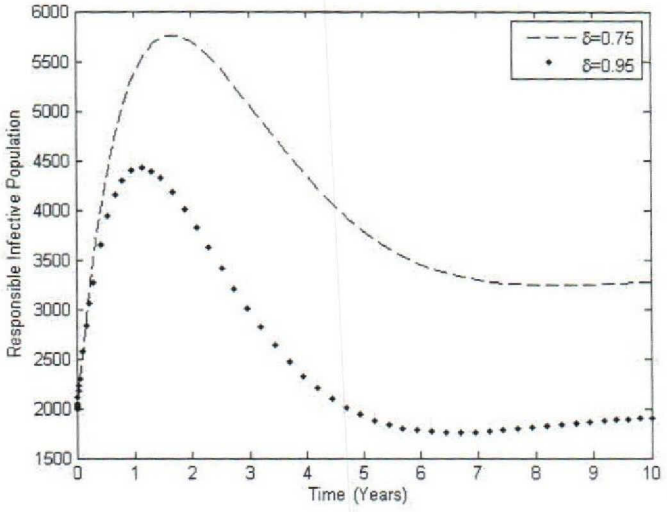

Figure 3.14: Variation of Irresponsible Infective population for different values of δ

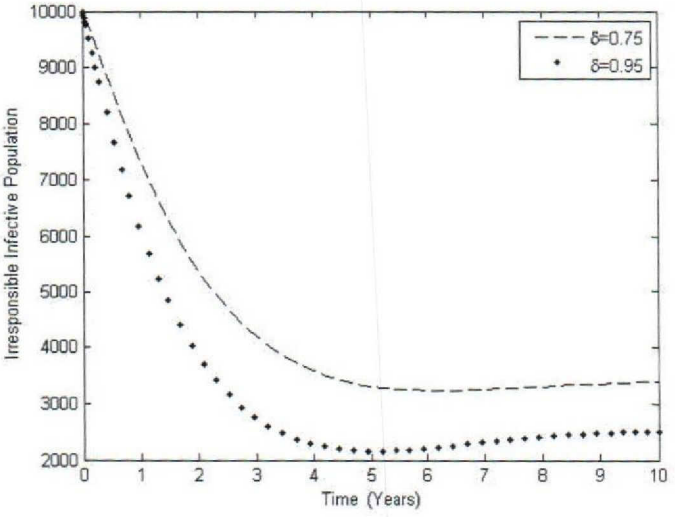

Figure 3.15: Variation of Responsible Infective population for different values of δ

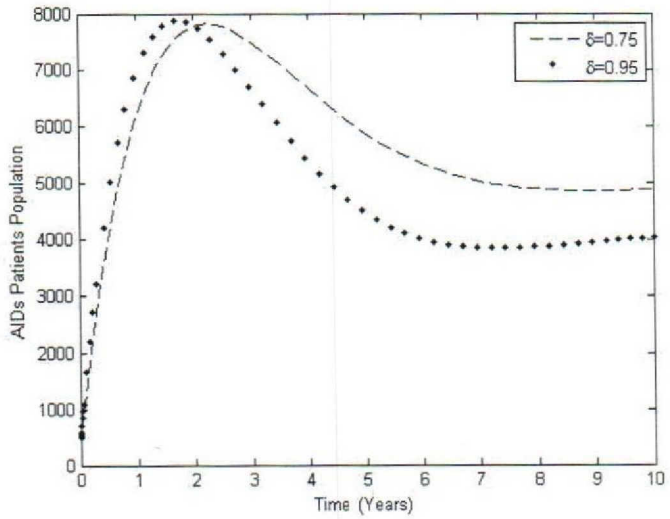

Figure 3.16: Variation of AIDS population for different values of δ

It is observed from figure 3.14 to figure 3.16 that increasing δ, the rate of conversion of infectives to full-blown AIDS patients results in a reduction of the infectives population and an increase in the AIDS patients population in the short term. However, in the long run, increasing δ results in a reduction of the AIDS population. That is if infectives become full-blown AIDS patients at a higher rate there will be a reduction in the infectives class and a corresponding increase in the AIDS class. However, as more infectives develop AIDS and pass out of the system(through AIDS-induced death) there will be a reduction in the AIDS class in the long-term.

The MATLAB codes that were used for the integration can be found in the appendices.

3.6 Summary of results

In summary,the analysis and simulations in chapters two and three show the following results:

1. Increase in contact rate of infectives, β_1 and β_2 increases R_0, thereby making the system unstable. Thus, efforts should be made to discourage people from having so many sexual partners in order to reduce the spread of HIV/AIDS.
2. An increase in the number of sexual partners, c, of infectives increases both responsible and irresponsible infectives populations which leads to an increase in the AIDS population.
3. Two equilibria, the disease-free equilibrium $E_0 = (1, 0, 0, 0)$ and the endemic equilibrium, $E^* = (N^*, I_1^*, I_2^*, A^*)$, have been established.
4. The system is locally asymptotically stable about E_0 and globally asymptotically stable about E^*.
5. A critical quantity, R_0, the basic reproductive number has been established. It is found that, if $R_0 < 1$ then the disease dies out of the population and if $R_0 > 1$ the disease persists in the population.
6. An increase in δ, the conversion rate of infectives to full-blown AIDS patients reduces both responsible and irresponsible infectives population but increases the AIDS population. Thus, HIV infection rate can be reduced if the rate at which infected classes move to full-blown class increases.
7. An increase in θ, the conversion rate of irresponsible infectives to responsible infectives reduces the irresponsible infectives population and subsequently reduces the AIDS population. Thus, if there are effective policies such as mass education, mass screening, use of condoms and abstinence put in place then the spread of HIV/AIDS could be reduced.
8. The research has established that the numerical results agrees well with the qualitative results.

CHAPTER 4

Conclusion, Contribution and future work

4.1 Conclusion

In this thesis, a non-linear mathematical model is proposed to study the spread of HIV/AIDS in the presence of irresponsible infectives in a variable size population with constant recruitment of susceptibles and infectives. By analyzing the model analytically, a threshold quantity, R_0 is established. It is found that this threshold value determines the endemicity or otherwise of the disease. The model has two (2) equilibria namely; the disease-free equilibrium and the endemic equilibrium. It is found that the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$, corresponding to the disappearance of the disease from the system. Also, $R_0 > 1$ shows that the system is unstable and the spread of the disease is maintained in the population.

The computer simulation of the model further shows that changes in the model parameters have a greater influence at better understanding of the spread and control of HIV/AIDS. For instance, an increase in the number of sexual partners reduces the entire population by way of spreading the disease. That is, increase in the number of sexual partners increases the number of infectives populations thereby reducing the susceptible population. Therefore, in order to reduce the spread of HIV/AIDS, the number of sexual partners should be reduced. It is also found that the disease becomes endemic due to immigration. Therefore, if the rate of immigration into the population is restricted and strict screening policies are put in place by policy makers such as Governments and immigration officials then the spread of the disease could be kept under control.

Further more, increase in the conversion rate of irresponsible infectives to responsible infectives reduces the irresponsible infectives population thereby reducing the spread of the disease.

Therefore, it is recommended that serious campaign messages be put in place to make people responsible by way of abstaining from unprotected sex, alcoholism, drugs and all other activities which influence people to make impaired judgment thereby becoming irresponsible.

Also, an increase in the conversion rate of infectives to AIDS can reduce the spread of the disease as the full-blown AIDS patients are sexually inactive. Therefore, if there could be a way of making all infectives move to full-blown AIDS at a very fast rate then the disease could even be eliminated from the system.

It is further found that the analytical method of analyzing the system under the prevailing conditions agrees perfectly with the numerical simulations.

Finally, the analysis shows that the most effective way to lower the spread of HIV/AIDS is to educate the population by making them aware of the various ways of contracting the disease. Therefore, Governments, health professionals, policymakers and researchers should continue to institute educative programmes that will reach communities at desired levels in order to increase the awareness of the population about the transmission dynamics of the disease so that the HIV/AIDS pandemic can be controlled. Thus, the socio-behavioral change remains the most effective treatment for HIV/AIDS.

4.2 Contribution of thesis

The research contributes in three main categories or ways. The first is in the design of the appropriate mathematical models for the transmission of HIV/AIDS in the presence of irresponsible infectives. The second contribution is the rigorous analysis of the nonlinear differential equations that have been modeled. The third has to do with the use of the analyzed results to interpret real life situations in the population. Thus, the main specific contributions made by this research are listed below.

1. The design of a mathematical model to study the transmission dynamics of HIV/AIDS in the presence of irresponsible infectives.
2. The design of a mathematical model to study the effect of infective immigrants on the transmission of HIV/AIDS in a population.
3. The research has also established the existence of the local stability of the model.

4. The research established a threshold parameter, R_0, the basic reproductive number which determines the endemicity or otherwise of the disease.
5. Establishing that certain model parameters such as contact rate (β_1, β_2), number of sexual partners (c) and the conversion rate of irresponsible infectives to responsible infectives(θ) influence the spread of the disease.
6. The research has established that, immigration increases the spread of the disease.

4.3 Future work

Although this research shows that there are prospects of the effective control of HIV/AIDS through education, strict screening policies, reduction in number of sexual partners and the increase in the conversion rate of irresponsible infectives to responsible infectives, more work need to be done in the following areas.

1. A mathematical model study of HIV/AIDS transmission dynamics in the presence of irresponsible susceptibles.
2. A mathematical model study of HIV/AIDS transmission in the presence of Shari'ah (The Islamic system of governance)
3. A mathematical model study of the endemicity or otherwise of the HIV/AIDS pandemic in the presence of anti-retroviral admission.

Bibliography

Agarwala, B. D. (2002). On two ODE models for HIV/AIDS development in Canada and a logistic SEIR model. Far East J. Appl. Math., 6(1), 25-70.

Agraj Tripathi, Ram Naresh, and Sandip Omar. (2006). Modeling the spread of AIDS epidemic with vertical transmission. Applied Mathematics and Computation,178, 262-272.

Alexander, M. E., Mogadas, S. M., and Rohani, P. (2006). Modeling the effect of the booster vaccination on disease epidemiology. Journal of Mathematical Biology, 52(3), 290-306.

Anderson, R.M. (1988), The role of mathematical models in the study of HIV transmission and the epidemiology of AIDS. J. AIDS., 1, 241-256.

Anderson, R.M., Medley, G.F., May, R.M. and Johnson, A.M. (1986). A Preliminary study of the transmission dynamics of the human immunodeficiency virus (HIV) the causative agent of AIDS. IMA J. Math. Appl. med. Biol., 3, 229-263.

Arazoza, H.D. and Lounes, R. (2002). A nonlinear model for sexually transmitted diseases with contact tracing. IMA J. Math. Appl. Med. Biol.19, 221-234.

Busenberg, S., Cooke K., and Ying-Heng H. (1995). A model for HIV in Asia. Math Biosci., 128, 185-201.

Busenberg, S. and Cooke, K.L. (1993). Vertically transmitted diseases models and dynamics. Springer velag, New York.

Chin, J. and Lwanga, S.K. (1991). Estimation and projection of adult AIDS cases: A simple epidemiological model. Bulletin of the World Health Organization, 69(4), 399-406.

Coffee Megan, Lurie Mark, N. and Garnett Geoff, P. (2007). Modeling the impact of migration on the HIV epidemic in South Africa. AIDS, 21(3), 343-350.

Coutsoudis, A., Goga, A. E. and Rollins, N. et al. (2002). Free formula milk for infants of HIV infected women: Blessing or curse? Health policy plan, 17, 154-160.

Dixon S, McDonald S. and J. Roberts J. (2002). The impact of HIV and AIDS on Africa economic development. BMJ. 324, 232-234.

Donkor, S. (2009, December 2). Be part of the know your HIV status campaign: Daily Graphic, issue 18089 p. 29, Accra.

Dunn, D. T., Tess, B. H., Rodrigues, L. C. and Ades, A. E. (1998). Mother-to-child transmission of HIV: Implication of variation in maternal infectivity. AIDS, 12:2211-2216.

Flessa S. (1999). Decision supports for malaria-control programmes- a system dynamics model. Health care management Science, 2(3), 181-191.

Glenn Ledder (2005). Differential Equations: A modeling approach. McGraw-Hill Companies.

Greenhalgh, D., Doyle, M. and Lewis, F. (2001). A mathematical treatment of AIDS and condom use. IMA J. Math. Appl. Med. Biol., 18, 225-262.

Hsieh, Y.H and Chen, C.F. (2004). Modeling the social dynamics of a sex industry: its implications for spread of HIV/AIDS. Bull. Math. Biol., 66, 143-266.

Hyman, J. M., Lia, J. and Stanley, E.A. (1999). The differential infectivity and staged progression models for the transmission of HIV. Math. Biosci., 55, 77-109.

International AIDS Society (IAS) Report. (2009). Retrieved December 5, 2009 from *http//www.ias2009.rog/ad 08-07-09.pdf.*

Kribs-Zaleta, C. M. (1999), Structured models for heterosexual disease transmission. Math. Bioci., 160, 83-108.

Li, M.Y., Smith, H.L. and Wang, L. (2001). Global dynamics of an SEIR epidemic with vertical transmission. SIAM J. Appl. Math., 62 (1), 58-69.

Makinde, O. D. (2007). Adomian decomposition approach to a SIR epidemic model with constant vaccination strategy. Applied Mathematics and Computation, 184, 842-848.

Makinde, O. D. (2009). On non-purtubative approach to transmission dynamics of infectious diseases with waning immunity. International Journal of Nonlinear Science and Numerical Simulation, 10(4), 451-458.

May, R.M. and Anderson, R.M. (1987). Transmission dynamics of HIV infection. Nature, 3426, 137-142.

Misra, O. P. and Mishra, D. K. (2009). Modeling the effect of booster vaccination on the transmission dynamics of diseases that spread by droplet infection. Nonlinear analysis: Hybrid system, 3, 657-665.

Mogadas, S. M. and Gumel, A. B. (2003). Mathematical study of a model for childhood diseases with non-permanent immunity. Journal of computational and Applied Mathematics, 157(2), 347-363.

Naresh, R and Tripathi, A. (2005). Modeling and analysis of HIV-TB co-infection in a variable size population. Math. Model. Anal., 10, 275-286.

Newell, M. L. (1998). Mechanism and timing of mother-to-child transmission of HIV-1. AIDS, 12, 832-837.

Perelson A. S. and Nelson P. W. (1999). Mathematical analysis of HIV-1 dynamics in vivo. SIAMREV, 41, 3-44.

Rajesh Ramakrishna and Nafees Ahmad. (2006). Derivation of primary sequences and secondary structures of rev responsive elements from HIV-1 infected mothers and infants following vertical transmission. Technical report September, College of Medicine, The university of Arizona Health Sciences Center, Tucson, AZ85724, USA.

Rouzious C., Costagliola D., Burjard M., Blanche S., Mayaux M. J., Griscelli C., and Valleron A. J.(1995). Estimated timing of mother-to-child HIV- 1 transmission by use of markov model. AMJ Epidemiology, 142, 1330-1337.

Sharomi, O. Y. (2006). Mathematical analysis of models of HIV epidemiology. Masters thesis, University of Manitoba, Winnipeg.

The Henry J. Kaiser Family foundation Report (October, 2005). HIV/AIDS Policy Fact Sheet: "The HIV/AIDS epidemic in Ghana". Retrieved December 5, 2009 from *www.kff.org*

Van de perre, P. (1999). Transmission of human immunodeficiency virus type-1 through breast-feeding: how can it be prevented? J. Infect. Dis., 179(3), 405-407.

World Health Organization Media Centre. (2007). "Global HIV prevalence has leveled off". Retrieved December 6, 2009, from *http/www.who.int/mediacentre/news/release/2007/pr61/en/index.ht.*

APPENDIX A

Results of Numerical Simulation of Model for irresponsible Infectives

Table A.1: Variation of population fraction in different classes with irresponsible infectives

Time(Years)	s	i_1	i_2	a
0	0.650000	0.200000	0.100000	0.050000
1	0.715126	0.079942	0.141390	0.063542
2	0.788657	0.039496	0.114892	0.056954
3	0.849384	0.023421	0.082564	0.044631
4	0.894546	0.015507	0.057173	0.032774
5	0.926628	0.010809	0.039263	0.023300
6	0.948977	0.007684	0.027022	0.016318
7	0.964438	0.005493	0.018702	0.011366
8	0.975132	0.003929	0.013025	0.007914
9	0.982551	0.002806	0.009119	0.005523
10	0.98772	0.002002	0.006411	0.003867

Table A.2: Variation of population in different classes for various values of θ

Time	Irresponsible Infectives			Responsible Infectives			Full-blown AIDs		
	$\theta = 0.25$	$\theta = 0.75$	$\theta = 0.95$	$\theta = 0.25$	$\theta = 0.75$	$\theta = 0.95$	$\theta = 0.25$	$\theta = 0.75$	$\theta = 0.95$
0	0.2000	0.2000	0.2000	0.1000	0.1000	0.1000	0.0500	0.0500	0.0500
1	0.1516	0.0961	0.0803	0.0857	0.1293	0.1411	0.0648	0.0639	0.0635
2	0.1208	0.0537	0.0398	0.0711	0.1087	0.1148	0.0624	0.0582	0.0570
3	0.0999	0.0340	0.0236	0.0585	0.0816	0.0826	0.0549	0.0467	0.0447
4	0.0850	0.0235	0.0157	0.0483	0.0589	0.0572	0.0469	0.0353	0.0328
5	0.0737	0.0169	0.0109	0.0403	0.0421	0.0393	0.0398	0.0259	0.0234
6	0.0649	0.0125	0.0078	0.0342	0.0302	0.0271	0.0339	0.0188	0.0164
7	0.0578	0.0093	0.0056	0.0294	0.0217	0.0188	0.0293	0.0136	0.0114
8	0.0519	0.0069	0.0040	0.0256	0.0158	0.0131	0.0256	0.0099	0.0079
9	0.0470	0.0052	0.0028	0.0226	0.0115	0.0092	0.0226	0.0072	0.0056
10	0.0428	0.0039	0.0020	0.0202	0.0085	0.0064	0.0202	0.0053	0.0039

APPENDIX B

Results of Numerical Simulation of Model for Infective Immigrants

Table B.1: Variation of population in different classes without immigration ($\gamma = 0$, $Q_0 = 2000$)

Time	N	I_1	I_2	A
0	15000	10000	2000	500
1	13768	2170	3976	4073
2	11619	598	2565	3462
3	10533	248	1428	2288
4	10516	143	781	1388
5	11221	94	436	817
6	12352	63	252	480
7	13718	41	150	285
8	15206	27	91	171
9	16752	17	56	104
10	18319	11	35	64

Table B.2: Variation of population in different classes for various values of δ

Time	Susceptibles		Irresponsible Infectives		Responsible Infectives		AIDs patients	
	$\delta = 0.75$	$\delta = 0.95$	$\delta = 0.75$	$\delta = 0.95$	$\delta = 0.75$	$\delta = 0.95$	$\delta = 0.75$	$\delta = 0.95$
0	15000	15000	10000	10000	2000	2000	500	500
1	21072	19744	7334	6073	5369	4409	6187	6977
2	21375	18276	5356	3819	5699	3901	7779	7778
3	19719	15701	4193	2731	5068	2976	7499	6657
4	17904	13760	3580	2277	4340	2295	6670	5367
5	16550	12708	3308	2160	3791	1921	5885	4463
6	15736	12331	3233	2210	3457	1775	5335	3993
7	15342	12337	3256	2318	3296	1763	5024	3841
8	15210	12496	3309	2416	3245	1810	4889	3866
9	15206	12664	3357	2478	3248	1865	4858	3954
10	15244	12781	3387	2504	3269	1905	4871	4038

Table B.3: Variation of population classes with immigration ($\gamma = 0.95$, $Q_0 = 2000$)

Time	N	I_1	I_2	A
0	15000	10000	2000	500
1	26716	9037	7197	7173
2	31915	8173	8986	10548
3	33697	7523	9321	11810
4	33981	7073	9122	12003
5	33778	6791	8796	11770
6	33557	6641	8509	11445
7	33498	6593	8317	11177
8	33640	6618	8225	11016
9	33961	6694	8219	10965
10	34418	6803	8280	11009

Table B.4: Variation of Susceptible and Irresponsible Infective population for various values of θ

Time	Susceptible Population			Irresponsible Infectives		
	$\theta = 0.75$	$\theta = 0.95$	$\theta = 1.20$	$\theta = 0.75$	$\theta = 0.95$	$\theta = 1.20$
0	15000	15000	15000	10000	10000	10000
1	21072	20411	19695	7334	6081	4818
2	21375	19704	18113	5356	3825	2543
3	19719	17412	15475	4193	2730	1669
4	17904	15408	13552	3580	2265	1391
5	16550	14163	12588	3308	2136	1380
6	15736	13600	12347	3233	2179	1491
7	15342	13487	12520	3256	2289	1638
8	15210	13599	12849	3309	2397	1764
9	15206	13772	13168	3357	2470	1842
10	15244	13916	13398	3387	2504	1875

Table B.5: Variation of Responsible Infectives and AIDs patients population in different classes for various values of θ

Time	Responsible Infectives			AIDs population		
	$\theta = 0.75$	$\theta = 0.95$	$\theta = 1.20$	$\theta = 0.75$	$\theta = 0.95$	$\theta = 1.20$
0	2000	2000	2000	500	500	500
1	5369	6010	6607	6187	6048	5891
2	5699	5887	5878	7779	7229	6671
3	5068	4805	4378	7499	6550	5675
4	4340	3835	3267	6670	5482	4473
5	3791	3215	2659	5885	4616	3609
6	3457	2914	2433	5335	4095	3154
7	3296	2832	2443	5024	3871	3019
8	3245	2869	2564	4889	3842	3075
9	3248	2946	2704	4858	3903	3206
10	3269	3015	2812	4871	3984	3335

Table B.6: Variation of population in different classes for various values of γ

Time	Susceptibles		Irresponsible Infectives		Responsible Infectives		AIDs	
	$\gamma = 0.5$	$\gamma = 0.9$	$\gamma = 0.5$	$\gamma = 0.9$	$\gamma = 0.5$	$\gamma = 0.9$	$\gamma = 0.5$	$\gamma = 0.9$
0	15000	15000	10000	10000	2000	2000	500	500
1	16841	20519	4876	7007	4494	5259	5179	6061
2	14797	20404	2614	4934	3879	5442	5450	7454
3	12588	18552	1737	3772	2867	4726	4461	7035
4	11191	16704	1455	3191	2137	3971	3468	6143
5	10584	15404	1442	2956	1739	3427	2808	5350
6	10496	14674	1549	2914	1588	3115	2486	4820
7	10664	14362	1688	2962	1586	2979	2405	4541
8	10899	14294	1804	3030	1653	2948	2457	4438
9	11096	14332	1873	3085	1732	2967	2554	4431
10	11217	14392	1899	3116	1791	2997	2641	4461

APPENDIX C

MATLAB codes used in study


```
function dy = InfectiveImmigrants(t,y)
beta2=0.015;alpha=1;mu=0.02;Q0=0.00;delta=0.75;beta1=0.08;theta=.95;c=10;gama=0.97;
dy = zeros(4,1);
dy(1)=Q0-mu*y(1)-alpha*y(4)+gama*y(2);
dy(2)=c*(beta1*y(2)+beta2*y(3))*(y(1)-y(2)-y(3)-y(4))/y(1)+gama*y(2)-...
(theta+delta+mu)*y(2);
dy(3)=theta*y(2)-(delta+mu)*y(3);
dy(4)=delta*(y(2)+y(3))-(alpha+mu)*y(4);
% [T1,Y1]=ode45(@InfectiveImmigrants,[0 10],[15000,10000,2000 500]);
% plot(T1,Y1(:,1),'k.-',T1,Y1(:,2),'k*',T1,Y1(:,3),'k--',T1,Y1(:,4),'k+')
% [T1,Y1]=ode45(@InfectiveImmigrants,[0 10],[15000,10000,2000 500]);
% [T2,Y2]=ode45(@InfectiveImmigrants,[0 10],[19000 16500 2000 100]);
% [T3,Y3]=ode45(@InfectiveImmigrants,[0 10],[15000 3000 2500 1000]);
% [T4,Y4]=ode45(@InfectiveImmigrants,[0 10],[10000 8000 1800 300]);


function dy =IrresponsibleInfectives(t,y)
 beta2=0.015;alpha=.5;mu=0.02;Q0=0.40;delta=0.25;beta1=.08;theta=.75;c=25;
R0=(c*beta1*(delta+Q0)+c*beta2*theta)/((delta+Q0)*(delta+theta+Q0));
dy=zeros(4,1);
dy(1)=Q0-c*(beta1*y(2)+beta2*y(3))*y(1)-Q0*y(1)+alpha*y(4)*y(1);
dy(2)=c*(beta1*y(2)+beta2*y(3))*y(1)-(delta+theta+Q0)*y(2)+alpha*y(4)*y(2);
```

```
dy(3)=theta*y(2)-(delta+Q0)*y(3)+alpha*y(4)*y(3);
dy(4)=delta*(y(2)+y(3))-(alpha+Q0)*y(4)+alpha*(y(4))^2;

% [T1,Y1]=ode23(@IrresponsibleInfectives,[0 10],[0.65,0.2,0.1,0.05]);
% plot(T1,Y1(:,1),'k.-',T1,Y1(:,2),'k*',T1,Y1(:,3),'k--',T1,Y1(:,4),'k+')
% xlabel('Time(years)')
% ylabel('Population fraction')
% legend('s','i_1','i_2','a')
```