Mathematical Modeling the Dynamics of HIV/AIDS with Treatment and Vertical Transmission

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Approval sheet 1

This is to certify that the thesis titled “MATHEMATICAL MODELINGTHE DAYNAMICS OF HIV/AIDS WITH TREATMENT AND VERTICAL TRANSMISSION” submitted in partial fulfillment of the requirement for the degree of Master of Science in Applied Mathematics to the Department of Mathematics Addis Ababa University, and is record of original research carried out by AMARE ATINAFU, ID.GSK/1149/09 under my supervision and no part of the thesis has been submitted for another degree. The assistance and the help received during the course of this investigation have been duly acknowledged. Therefore, I recommended that it may be accepted as fulfilling the thesis requirement.

_____	_____	_____
Name of Advisor	Signature	Date

Approval sheet 2

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Name of Chairman	Signature	Date
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Name of Advisor	Signature	Date
_____	_____	_____
Name of Examiner 1	Signature	Date

Declaration letter

I declare that this thesis is my original work and that all source materials that I used for this thesis have been properly acknowledged. This thesis has been submitted in partial fulfillment of the requirements for M.Sc. degree in Computational Mathematics at Addis Ababa University. I earnestly declare that this thesis is submitted to any other institution anywhere for the award of any academic degree, diploma, or certificate I modified the parameter.

Name _____

Signature _____

Place: Addis Ababa,

Ethiopia

Date of Submission _____

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First of all, I would like to thanks to the almighty God for Her blessings, guidance and protection throughout my studies and being successful in both direction. I would like to sincerely thank my advisor, **Dr. Manalebish Debalkie** her knowledge and vision in the past years of my study, as well as her continuous guidance and encouragement throughout the creation of this thesis. This thesis could not be finished without her support and encouragement.

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Abstract

A non linear Mathematical model is proposed the dynamics of HIV/AIDS with treatment and vertical transmission in order to decrease mother to child transmission. The equilibrium points of the model are found and the stability analyses of the model around those equilibrium points are conducted.

The stability analysis on the model shows that the disease free equilibrium point E_0 *is locally asymptotically stable If* $R_0 < 1$. *The positive endemic equilibrium point* E^* *is locally asymptotically stable if* $R_0 > 1$. *This shows that the basic reproduction number of the present model is greater than the one which is obtained from the model modeled without vertical transmission. Vertical transmission contributes positively to the spread of the disease. Numerical simulation of the model is carried out to assess the dynamics of HIV/AIDS infected immigrants and vertical transmission (MTCT) in the spread of HIV/AIDS disease.*

The result showed that HIV infective immigrants and vertical transmission (MTCT) significantly affects the spread of the disease. The disease reduces the spread of HIV and also prevents mother to child transmission (PMTCT).It is well accepted that both vertical transmission and immigration contribute positively to the spread of the disease and these two parameters cannot be avoided in practice.

List of Abbreviations

HIV: Human Immunodeficiency Virus.

DFE: Disease Free Equilibrium.

AIDS: Acquired Immune Deficiency Syndrome.

DTC: Drug Treatment Center ART Anti Retro viral Therapy.

MTCT: Mother to child transmission.

PMTCT Prevention of Mother To Child Transmission.

FHAPCO: Federal HIV/AIDS Prevention and Control Program.

UNAIDS: United Nations Programme on HIV/AIDS.

UNICEF: United Nation International Children Emergency Fund.

WHO: World Health Organization

PEPFAR: President Emergency plan for AIDS relief

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CHAPTER ONE

1. Introduction

1.1 Background of the study

Viral infectious diseases can be transferred in different ways; a few of them may be categorized as either horizontal or vertical. In relation to HIV/AIDS, horizontal transfusion may be deduced. HIV can be transferred from close personal touch between a susceptible person and an infected one. Usually, HIV can spread through blood products. This is known as horizontal transmission. During pregnancy, child birth, or breastfeeding, an HIV-positive mother can pass the virus to her unborn child. The direct transmission of a viral infectious disease from an infected mother to a newborn can lead to this.

There are a lot of factors that can cause vertical transmission of HIV/AIDS, some of which are maternal viral load and a kind of delivery. Chagas, dengue fever, hepatitis B, and HIV/AIDS are some of the diseases that can be passed vertically. However, Our concern is HIV/AIDS through vertical transmission. Out of this 20% of the children living with HIV go to AIDS in the primary year of their prolonged life, and among those most of them died by the age of 4 years (4). The remaining up to 80% of infectious children move to the symptoms of HIV/AIDS during the entry age of school (7-9 years) or even a period of adolescence(1). It is the world's most infected with HIV/AIDS.

In Africa, HIV/AIDS transmission is primarily through heterosexual (occurred between different sexes) and vertical transmission (mother-to-child). 40 percent of the whole HIV/AIDS symptoms can be attributed to infected mother-to-child transmission. (6). in 1997, at least 300 infants in the U.S. (United States) were infected with HIV through mother-to-child transmission. Most of the time, those kids were exposed. Over 2.5 million children under the age of 15 have died from this lethal illness in Africa. HIV/AIDS during a period of labor or breastfeeding is a threat to future development in the world. In Africa, where the quality level of literacy is very poor, the poverty level is very high, and the quality of health service is generally very low, mother to child transmission of HIV/AIDS has been felt most (1).

There are a lot of studies (research) (2) concerning the treatment of pregnant women who are infected with HIV/AIDS results in a significant decrease in the number of children born infected.

Another work (3) Furthermore, research into the treatment of HIV-infected babies has demonstrated that a great treatment for these children may prolong their survival and dynamically improve the quality of their lives.

Infected children may reach adulthood and become sexually active because anti-retro viral drugs are effective in the lowest viral loads. The mathematical model on the dynamics of the HIV pandemic with vertical transmission was further investigated (4).

As a result, we study and analyze the dynamics of HIV/AIDS with treatment and vertical transmission. As well as, we formulate anon-linear system of differential equations that models the dynamics of transmission in different population. The Modeling of HIV/AIDS dynamics that avoid the impact of mother to child transmission, particularly the time of the current high usage of anti-retroviral drugs treatment, may fail to capture the actual impact of HIV/AIDS in a population.

1.1.1 Thesis Outline

1. Chapter 1: presents the introduction, motivation, methodology and outline of the thesis.

2. Chapter 2: presents the transmission means of HIV/AIDS, different research review literature a related to MTCT (mother to child transmission) and the definition of system of differential equation and equilibrium point.

3. Chapter 3: presents the model formulation, mathematical analysis and basic properties of the model, positivity and boundedness solution of the model, determining the disease-free equilibrium point and reproduction number(R_0), existence of equilibrium point, the local stability of disease-free equilibrium point and endemic equilibrium point and its existence.

4. Chapter 4: Presents the numerical simulation and verification of the model and Implementation of the best method on the result of non-linear differential equation as well asthe conclusion and recommendation the areas of further research.

1.2 Statement of the problem

The most dangerous form of the viral disease is HIV/AIDS, which is a major health challenge. It's common in emerging nations like Ethiopia. Although there are some encouraging signs that the epidemic is stabilizing, the current HIV/AIDS surveillance prediction shows that the epidemic is stabilizing. In developing countries, this remains a fundamental health issue (6).

We should talk about how COVID-19 has diverted the attention of HIV/AIDS and how it affects the transmission of HIV/AIDS in 2012 E.C. It's possible that the COVID-19 epidemic was aimed at assessing the impact of HIV treatment on individuals. There is also a number of evidence that the risk of death from COVID-19 among people with HIV/AIDS could be twice that of the general population. Recent evidence shows that HIV service has been decreased by 75% and the problem is more extensive in Ethiopia. The majority of logistics for HIV service and fund are denoted by the goodwill of none governmental organization.

In order to choose the best strategy for control and final eradication of HIV/AIDS, it is necessary to identify the main reasons or parameters that may affect HIV/AIDS transmission. However, there are a number of advances in the availability, accessibility and utilization of HIV/AIDS prevention methods, care, support and treatment services, and improvements in the management of the epidemic. However, they still face a condition unlikely to give us respite in the near future is within 22 years (6).

The study aims to give a partial solution using the following basic questions.

- What is the goal of preventing horizontal transmission and AIDS-related mortality?
- What are the main ways of the spread of HIV/AIDS?
- How can we show the mathematical model analysis of HIV/AIDS?
- How the model is coded using a Mat-Lab soft ware using ode45?
- How can the model be stable?

➢ Methodology of the study

The non linear mathematical model was studied in this thesis using qualitative data collected from the internet and secondary data.

1.3 The Significance of the study

The goal of the HIV infection control program is to decrease morbidity and mortality due to this disease and prepare planning to control transmission of HIV in the society. Knowing how HIV/AIDS works is crucial for securing control over the epidemic.

The output (result) of this thesis will help healthy care workers to give awareness (motivation) and inform infected persons about the possible transmission ways of this disease as they might encounter. Further, clinicians can reduce mortality between HIV positives by first diagnosis and important intervention (11).

Mainly, the outcome of this thesis will allow physicians and policy makers to improve the motivation of the community about the major factors which increase the probability of HIV. The output of this thesis is predicted to provide information for public health workers and stakeholders who are doing in the area of giving care, support and treatment for HIV/AIDS carriers.

Chapter Two

2. Review of literature

Acquired immunodeficiency syndrome (AIDS) is a disease of the human immune system caused by the primary infectious agent called HIV (the human Immune deficiency virus). The World Health Organization (WHO) states that the Human Immunodeficiency Virus (HIV) infects immune system cells, including macrophages, dendrite cells, and helper T cells (more specifically, CD4+ T cells) (12).

HIV is transmitted primarily via unprotected sexual intercourse, contaminated blood transfusions, hypodermic needles, and from mother to child during pregnancy, delivery, or breastfeeding. There is no cure or vaccine for HIV/AIDS. However, antiretroviral treatment improves health, prolongs life, and substantially reduces the risk of HIV transmission (14).

At the end of 2016, there were approximately 36.7 million people living with HIV, with 1.8 million people becoming newly infected globally. Out of these numbers, 17 million people were accessing antiretroviral therapy (ART), with coverage being 46% at the end of 2015 and 48% at the end of 2016. The African Region is the most affected region according to the WHO report, with 25.6 million people living with HIV in 2016 (13).

Vertical transmission is the most common way in which children become infected with Human Immune Deficiency Virus (HIV) globally. This happens in Uterus, during a period of labor and through breast feeding. The first approach to the prevention of mother-to-child transmission (PMTCT) has been to identify HIV-infected pregnant woman by voluntary counseling and testing (VCT) for HIV and then provide anti-retroviral (ARV) drug prophylaxis to them during delivery and to their new born infants (1). The highest risk of transmission happens at the time of labor where $10\% - 20\%$ infection occurs. Without interventions, HIV infection in infants born to HIV mothers ranges from (5 -20) (7).

Infection through breast feeding is decided based on the health status of Mother, the duration of breast feeding and the condition of the breast. Before or during pregnancy, the risk of transmission is twice high in newly infected mothers than previously infected mothers.

USAID is an implementing agency of the president's emergency plan for AIDS relief, which supports the implementation of HIV services for women, infants and children. The goal of USAID is to end preventable child and maternal deaths by providing comprehensive maternal and infant health. HIV prevention care and treatment services to mothers living with HIV and their infants exposed to HIV are provided to mothers living with HIV and their infants. Since 2015, USAID support has enabled 3.3 million babies to be born HIV free to mothers living with HIV, thanks to USAID support. (16).

To expand coverage and deliver simple and effective PMTCT interventions in all settings, even with limited capacity, prevention from vertical transmission strategy (PMTCTS) is crucial. The WHO/UNICEF/UNAIDS 4-prolonged prevention of mother to child transmission has been adopted by Ethiopia. (6).

The anti-retroviral medication taken by either the mother or the infant reduces the risk of transmission in those who don't nurse. If women are not untreated or are treated, two years of breast feeding results in an HIV/AIDS risk in their baby decreasing 17% treatment risk to 1% - 2% per year. Due to increased risk of death without breast feeding in many areas in developing countries, The World Health Organization recommends either the mother or the baby being treated with anti-retroviral medication while breast feeding is continued or the prevention of the safe formula. At that time Infection with HIV would occur during pregnancy (8).

The researcher has brought with him numerous mathematical models of HIV/AIDS dynamics with treatment and mother-to-child transmission based on previous workers on epidemics. The researcher will analyze the propagation rate of individuals affected by HIV from one compartment to another compartment and the impact of each parameter in the model.

2.1 Objectives of the study

- General objective

■The basic aim of this thesis is to construct and present a mathematical model that reflects the dynamic of HIV/AIDS with treatment and vertical transmission.

- The Specific objectives are:

■ To find the equilibrium points and their stability.

■ To compute the reproduction number, disease-free equilibrium point, and endemic equilibrium point.

■ Carrying out model simulation in mat lab software and verifying the outcome.

■ To suggest ways to slow down the transmission rate.

2.2 Ordinary differential equations

An ordinary differential equation of order n is an equation which contains derivatives of unknown functions $y(t)$ which is denoted as

$$f\left(y, \frac{dy}{dt}, \frac{d^2y}{dt^2}, \frac{d^3y}{dt^3}, \dots, \frac{d^ny}{dt^n}, t\right) = 0$$

It is an equation which studies the change in the unknown variable y with respect to time.

2.2.1 System of ordinary differential equation

A differential equation system consisting of two or more equations simultaneously is known as a system of ordinary differential equations $\frac{dx_i}{dt} = F(x(t),\ t)$

Where

$x(t) = (x_1(t), x_2(t), x_3(t), \dots, x_n(t))^T$

$F = (f_1,\ f_2,\ f_3,\ \dots,\ f_n)^T$ and $f_i = f_i(x_1(t),\ x_2(t),\ x_3(t),\ \dots,\ x_n(t),\ t)$.

$$f(y) = \begin{bmatrix} f_1(y) \\ f_2(y) \\ . \\ . \\ . \\ f_n(y) \end{bmatrix}$$

The function f_i are called $components\ off$. we define

$$Df(p) == \begin{bmatrix} \frac{\partial f_1(p)}{\partial y_1} & \frac{\partial f_1(p)}{\partial y_2} & \dots & \frac{\partial f_1(p)}{\partial y_n} \\ \frac{\partial f_2(p)}{\partial y_1} & \frac{\partial f_2(p)}{\partial y_2} & \dots & \frac{\partial f_2(p)}{\partial y_n} \\ . & . & & . \\ . & . & & . \\ . & . & & . \\ \frac{\partial f_n(p)}{\partial y_1} & \frac{\partial f_n(p)}{\partial y_2} & \dots & \frac{\partial f_n(p)}{\partial y_n} \end{bmatrix}$$ is the linearization of f called the jacobian matrix.

2.3 Next generation matrix

The next-generation operator approach is utilized to establish the reproduction number (R_0). It is applied in a population consisting of compartments that are mutually exclusive. As described by Driessche and Watmough in their paper (9), it follows.

$Let\ x_i, i = 1, \dots, m,$ ($where,$ m (infected compartment) < n (total number of compartments)) be the number of proportion of individuals in each compartment and X_s={ x $\geq 0/x_i \geq 0,\ i = 1, 2, \dots, m$} be the disease free states (non-infected state variables) of the model, where $x = (x_1, \dots, x_n)$ and

- $V_i(x)$ be the difference between rate of transfer of individuals out of and into $the\ i^{th}$compartments.
- $F_i(x)$ are determined by the rates of appearance of new infection compartment i.
- $V_i^+(x)$ are determined by the rate of transfer of individuals in to $the\ i^{th}$compartments.
- $V_i^-(x)$ are determined by rate of transfer of individuals out of the i^{th} compartments.
- $f_i(x)\ be$ the proportional rate of infected individuals in the i^{th} compartments.

Then, we have $\frac{dx_i}{dt} = f_i(x) = F_i(x) - V_i(x)$.

$Where, V_i = V_i^-(x) - V_i^+(x)$. The functions F_i(x), V_i^+(x) and V_i^-(x)) are made the following assumptions

• $Assumption1\ (C1). If\ x \geq 0,$ Implies $F_i(x)$, $V_i^+(x)$, $V_i^-(x) \geq 0.\ for\ i = 1, 2, \ldots, m.$ which means that, the rate of transfer is non-negative.

• $Asumption2\ (C2).$ If x = 0, then $V_i^-(x) = 0.$ In particular, if x $\in$ X_S (disease Free State variables), Then $V_i^-(x)\ exist.\ For\ i = 1, 2, \ldots, m.$This means that, for an empty compartment, there cannot be transfer of individuals out of the compartments.

• $Assumption3\ (C3). F_i(x) = 0\ i > m.$This means that there is no appearance of infections in uninfected compartments.

• $Assumption4\ (C4). If x \in X_S,$ ThenF_i(x) = 0 and $V_i^+(X)$ = 0.$For\ i = 1, 2, \ldots, m.$ This means that, if a population is not infected, then it will remain free of the disease.

• $Assumption5\ (C5).$ If F(x) is becomes to zero, then all $eigen\ values\ the\ Jacobian\ matrix$ at the disease-free equilibrium has negative real parts. This means that, the disease-free equilibrium is locally-asymptotically stable if there is no incidence of infection into the compartment.

$If X^*$ is a disease-free equilibrium of C (1) of $f(x)$ and $f_i(x)\ satisfies(C1)\ through\ (C5)$, then the $Jacobian\ matrices\ F(x) and\ V\ (x)$ at the equilibrium point X^* are given by:F= $\left[\frac{\partial F_i}{\partial x_j}(x^*)\right]$ and V =$\left[\frac{\partial Vi}{\partial x_j}(x^*)\right]$ with $1 \leq i,\ j \leq m.$Thus, the reproduction number R_o is the spectral radius of the next generation matrix $FV^{-1},\ given\ R_o = \rho(FV^{-1})$. Where ρ is the spectral radius (dominant Eigen value).

CHAPTER THREE

3. Introduction of the model

The study of HIV transmission and dynamics of this disease has been of great interest to both applied mathematics and biology. The World Health Organization (WHO) has classified the symptoms of a human with HIV/AIDS carrier as major and minor symptoms. Humans with major and minor symptoms known as pre-AIDS. Pre-AIDS is a condition triggered by HIV that hasn't been included in severe AIDS. The immune system reduces until the carrier population suffers serious disease (5). HIV AIDS still has no cure, so that there are still individual deaths due to AIDS. Treatment for human infected by HIV/AIDS is a treatment which aims to develop the immune system (6).

3.1 The mathematical model formulation

First we consider HIV/AIDS model in which the total population divided in to two compartments namely; susceptible individuals S and $I_H$infectious individuals in SI model.

$$\frac{ds}{dt} = \Lambda - \lambda_H S - \mu S$$

$\frac{dI_H}{dt} = \lambda_H S - (\mu + d_H)I_H$. d_H is per capital induced death rate. β_H is Probability of HIV infection per contact with a person who is HIV infections and c is per- capital contact rate for HIV.

$\lambda_H = c\beta_H \frac{I_H}{N}$ is the force infection. The above model is also expressed using diagram as follows:

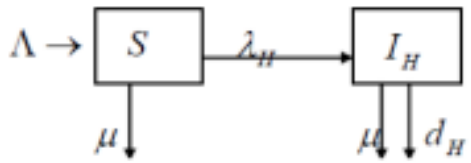

Figure 3.1: Initial (sub) Flow diagram of the SI compartments of HIV model (10).

3.2 Flow diagram of the present model

Using the above assumptions we develop the following flow diagram. Here we develop SI model in to SITA model. Where T is the treatment population and A is population who infected by AIDS.

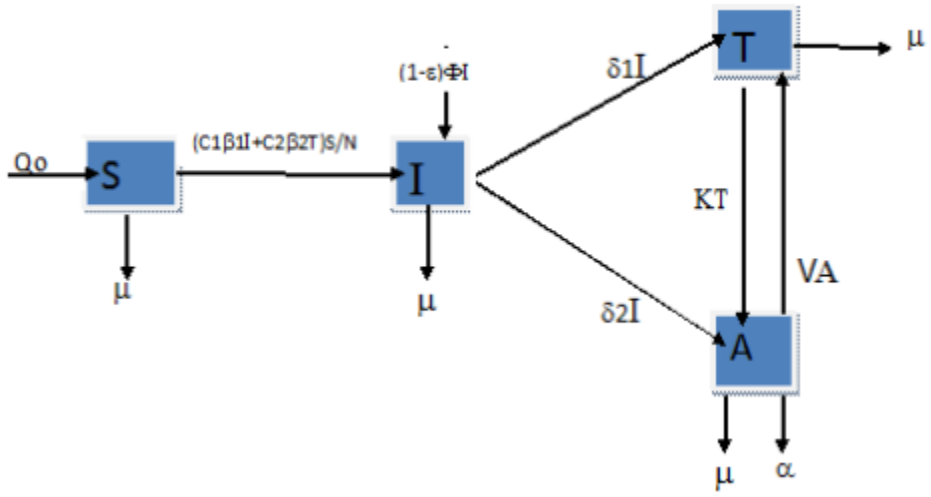

Figure 3.2 Flow diagram of the present model (11).

➢ Model equations and compartmentalization of the model

Depending on the consideration and the flow diagram of the present model, the dynamics of HIV/AIDS transmission is governed by a system of non-linear ordinary differential equations as follows.

$$\frac{dS}{dt} = Q_0 - \frac{(C_1\beta_1 I + C_2\beta_2 T)}{N}\mathrm{S} - \mu S \qquad (1)$$

$$\frac{dI}{dt} = \frac{(C_1\beta_1 I + C_2\beta_2 T)S}{N} + (1-\epsilon)\phi I - (\delta_1 + \delta_2 + \mu)I \qquad (2)$$

$$\frac{dT}{dt} = \delta_1 I + \upsilon A - (K + \mu)T \qquad (3)$$

$$\frac{dA}{dt} = \delta_2 I + KT - (\upsilon + \alpha + \mu)A \qquad (4)$$

Let $\Lambda = \frac{C_1\beta_1 I}{N} + \frac{c_2\beta_2 T}{N}$, From the system of the equation (1) to (4), the initial condition are assumed to be

$S(0) = S_0, T(0) = T_0$ and $A(0) = A_0$. The above system of equations is known as model equations

Four compartments are used to group the total population. People's movements within these compartments have been depicted. This was used to derive the notation and parameters. A better understanding of the model is provided by the flow diagram and direction. A set of nonlinear ordinary differential equations that are constructed describe the model. The model is analyzed mathematically, and the observation is included.

➢ Variable descriptions of variables and parameters

The people were recruited into the susceptible class at a fixed rate of Q_0. This is because of natural births. This grouped in to $I(t)$ and $T(t)$classes.Thus people from $S(t)$ moves to $I(t)$ with rate of $\frac{(C_1\beta_1 I + C_2\beta_2 T)S}{N}$ and due to mother to child transmission at a rate of$(1-\epsilon)\phi I$.

Parameters	Descriptions
β_1	The probab*i*lity per one contact with that the disease transmits to susceptible human
β_2	The probab*i*lity per one contact with that the disease transfer to treated human
δ_1	The rate of treated person from infected compartment to treatment human
δ_2	The rate of AIDS people from infected compartment to AIDS classes
c_1	Per capital contact rate of susceptible classes
c_2	Per capital contact rate of infected classes
ϵ	The natural death rate of newly born infected children
μ	The natural reason death rate of susceptible **,** infected **,** treated and AIDS class
φ	The sexual contact rate between susceptible and infected person that leads to infected children.
K	The rate of treated person from the treatment compartment to AIDS class
V	The rate of Anti-retroviral treatment of AIDS people
α	The natural death rate of AIDS classes

Table 3.1: description of modeling parameters

From this study $\beta_1 > \beta_2$, the chance of transferring the disease to a susceptible population infected human is higher than the chance of transferring the disease to treatment humans. Additionally, we assume that $\delta_2 > k$ since infected person goes to AIDS more than the treated people. In this thesis, the mathematical model of the HIV epidemic divides the population into four distinct groups. Those are prone to infection, treatment, and HIV/AIDS.

None (free) infected population is considered **susceptible.**

Infected refers to a person who is HIV-positive.

Treatment refers to carriers who are performing the treatment in to ART.

AIDS refers to a person who has acquired immune deficiency syndrome.

Model Assumptions

■*The population is constant and closed*. This means that it can increase or reduce due to birth and death.

■ The direct transfer of HIV/AIDS by sexual intercourse happens in individuals susceptible to infection, treatment, and AIDS.

■The population mortality occurred due to natural deaths in each class and due to AIDS.

■ There are different populations under study that vary with time.

■ The total human population is categorized in to four classes (SITA).

3.3 Important properties and mathematical analysis of the model

We interpret the mathematical analysis of the model as mentioned by system (1) to (4) here. The total number of individuals N(t) is represented by:

$$N(t) = s(t) + I(t) + T(t) + A(t)$$

$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dT}{dt} + \frac{dA}{dt}$ Where,

$$\frac{dN}{dt} = Q_0 - \Lambda S - \mu S + \Lambda S + (1-\epsilon)\phi I - (\delta_1 + \delta_2)I - \mu I + \delta_1 I + \upsilon A - (k+\mu)T + \delta_2 \mathrm{I} + kT$$

$$-(\upsilon + \alpha + \mu)A \qquad (5)$$

$$\frac{dN}{dt} = Q_0 + (1-\epsilon)\phi I - \alpha A - \mu N$$

Several important properties of the model (5) that is crucial for our doing for the next part of such existence of equilibrium points and the stability analysis of equilibrium points are included in this subsection. The region in which the solution of (5) is biologically interpreted and well posed is explained by the boundedness of the solution. The non-negative of the solution is expressed in the positivity solution.

3.3.1 Positivity and boundedness of the solution

In order to prove that the general equation of the model (1) up to (4) is useful and mathematically unique, it is required to show that the population sizes of total compartments are positive. It's a theorem that shows this fact.

Theorem1:

If $S(0) > 0, I(0) \geq 0, T(0) \geq 0\ and A(0) \geq 0$,then the solution $\{S(t), I(t), T(t), A(t)\}$ of the system of equation (1) to (4) is always positive for $\forall t > 0$.

Proof

We should consider and verify each differential equation to prove that their solution is positive from the equations (1) to (4) the differential equation (1) of the dynamical system can be written as follows.

$\frac{dS}{dt} + (\Lambda + \mu)S = Q_0$. Where,

$\Lambda(t) = \left[\frac{C_1\beta_1 I + C_2\beta_2 T}{N(t)}\right]$ is the force of infection rate that the first order linear ordinary differential equation, which can be solved to obtain a particular solution.

$$\int_0^t \frac{dS}{S} + \int_0^t (\Lambda + \mu)dt = \int_0^t \frac{Q_0}{S} dt \qquad (6)$$

$$ln\frac{S(t)}{S(0)} = \int_0^t -(\Lambda + \mu)dt + \int_0^t \frac{Q_0}{S} dt$$

$$S(t) = S(0)e^{\int_0^t -(\Lambda+\mu)dt} + S(0)e^{\int_0^t \frac{tQ_0}{S} dt} > 0$$

It implies that S(t)is positive since $S(0) > 0,\ Q_0 > 0\ and$an exponential function is always positive. Now as $t \to \infty$, the analytical solution now becomes $S(t) \geq 0$. this results in always a positive solution or population size for the susceptible compartment S (t).

From the second differential equation of the system gives us a solution.$\frac{dI}{dt} = \frac{(C_1\beta_1 I + C_2\beta_2 T)}{N} + (1 - \epsilon)\varphi I - (\delta_1 + \delta_2 + \mu)I$. Since $I \geq 0$, with out loss of generality this can be written as an inequality

$$\frac{dI}{dt} \geq -(\delta_1 + \delta_2 + \mu)I \qquad (7)$$

Up to the integrating inequality (8) using the method of separation of variables, the method of separation of variables is used. The outcome of an analytical approach is:

$$\int \frac{dI}{I} = \int -(\delta_1 + \delta_2 + \mu)\, dt \qquad (8)$$

$I(t) = I(0)e^{-(\delta_1+\delta_2+\mu)t}$. Where, I(0) is the integral constant and it represents the initial population of an infectious compartment. It is, therefore, a positive quantity

The analytic solution leads to I(t)≥ 0. Now. Hence, the solution or population size of the infected compartment I(t) is always positive. From the third differential equation of the system (3) we have the following

$\frac{dT}{dt} = \delta_1 I + \upsilon A - (K + \mu)T$ Since $I(t) \geq 0 \Rightarrow \delta_1 I > 0, with\ out\ loss\ of generality$it can be expressed as in the above equation as an inequality:

$$\int \frac{dT}{T} \geq \int -(K + \mu)dt \qquad (9)$$

The analytic solution for an inequality (9) represents the initial population of the treatment class and vertical transmission (MTCT) and infected compartments. Therefore, it's also a positive number.

$$T(t) \geq T(0)e^{-(K+\mu)t} \qquad (10)$$

Now as the limit $t \to \infty$, The analytical solution of the inequality leads to T(t)≥ 0 The population size of the compartment T(t) is always positive. From the last differential equation of the system.

$$\frac{dA}{dt} = \delta_2 I + KT - (\upsilon + \alpha + \mu)A$$

Since$I(t) \geq 0\ and T(t) \geq 0$. without loss of generality by using separation of variables this can be expressed as follows:

$$\frac{dA}{dt} \geq -(\upsilon + \alpha + \mu)A \qquad (11)$$

$\int \frac{dA}{A} \geq \int -(\upsilon + \alpha + \mu)dt$ (12)

From an integration of (12) an analytic solution is obtained as$A(t) \geq A(0)e^{-(\upsilon+\alpha+\mu)t}$. Here the initial population of the AIDS compartment is represented by A(0) It is a positive value. Now as t goes to ∞, the analytic solution goes to$A(t) \geq 0$. Since the solution or the population size of AIDS compartment $A(t)$is always positive. Therefore, it is obvious to conclude that the solution S(t), T(t), I(t) and A(t) are positive for t ≥ 0.

Remark: $e^k \geq 0$, for all real number k .

Boundedness: The whole human population size $N(t)$of the system is the sum of the population of the four compartments. i.e. $(Biological\ fiseable\ region)\Omega = \{S(t), I(t), T(t), A(t) \leq \mathscr{R}_+^4\}$

Proof

To start let us consider$N(t) = S(t) + I(t) + A(t) + T(t)$.

Upon differentiating (derivation) with respect to time it can be gained as follows:

$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dT}{dt} + \frac{dA}{dt}$.

$\frac{dN}{dt} = Q_0 + (1-\epsilon)\phi I - \alpha A - \mu N$…..From equation (5).

$\frac{dN}{dt} \leq Q_0 + (1-\epsilon)\phi I - \alpha A - \mu N$

$\frac{dN}{dt} \leq Q_0 + [(1-\epsilon)\phi - \mu)]N - \alpha A$

$\frac{dN}{dt} \leq Q_0 + [(1-\epsilon)\phi - \mu]N$

$\frac{dN}{dt} \leq Q_0 - \mu_0 N$

Since, $-\mu_0 = (1-\epsilon)\phi - \mu$.

Then using integration by integrating factor

$N(t) \leq \frac{Q_0}{\mu_0} - e^{-\mu_0 t}(\frac{Q_0}{\mu_0} - N_0)$.Then as $t \to \infty, the\ total\ population\ N(t) \leq \frac{Q_0}{\mu_0}$, then the biological feasible region $\Omega \leq \mathscr{R}_+^4\ expressed\ as\ follows$:

$\Omega = \{S(t), I(t), T(t), A(t) \leq \mathcal{R}_+^4\}$

3.4 Equilibrium point and its existence

The basic reproduction number is obtained by employing the next-generation matrix approach.to find the reproduction number we should proceed the following steps.

1. First, identify the existence of equilibrium points, which means compute the disease-free and endemic equilibrium points.
2. Analyze the local stability of disease-free equilibrium points as well as the local stability of disease-endemic equilibrium points and
3. Calculate the equation for the basic reproduction number R_0. The right hand side of the system of equation model is set to zero. to obtain the equilibrium points.

   i.e., $\frac{dS}{dt} = 0,\ \frac{dI}{dt} = 0,\ \frac{dT}{dt} = 0\ and\ \frac{dA}{dt} = 0$

This is a condition for the requirement of disease free equilibrium point.

Therefore, the system of the equation (1-4) set as follows:

$$Q_0 - \Lambda S - \mu S = 0$$

$$\Lambda S + [(1-\epsilon)\varphi - (\delta_1 + \delta_2 + \mu)]I = 0$$

$$\delta_1 I + \upsilon A - (k + \mu)T = 0$$

$$\delta_2 I + kT - (\upsilon + \alpha + \mu)A = 0$$

3.4.1 The disease free equilibrium point

To achieve a disease-free condition, the right-hand side's equilibrium point setting system (1) must be zero. Setting the disease-free equilibrium point of the model (1) to (4) yields the disease-free equilibrium point.

$\frac{dS}{dt} = 0,\ \frac{dI}{dt} = 0,\ \frac{dT}{dt} = 0\ and\ \frac{dA}{dt} = 0$. In addition at the disease free equilibrium point there are neither infected people nor AIDS patients. i.e. $I = T = A = 0$. Evaluating this into (1)

$$\frac{dS}{dt} = Q_0 - \frac{(C_1\beta_1 I\ +\ C_2\beta_2 T)}{N} - \mu S \Rightarrow [Q_0 - \mu S] = 0 \Rightarrow Q_0 = \mu S \Rightarrow S = \frac{Q_0}{\mu}$$

Therefore, the disease free equilibrium point of this model is given by $(\frac{Q_0}{\mu}, 0, 0, 0)$

3.4.2 Reproduction number R_0

The threshold parameter that governs the spread of a disease is called the reproduction number (R_0), and we employ the next-generation matrix approach to obtain the reproduction number.

In this model, the infected class is divided into I and T, and the differential equation is used as follows.

$$\frac{dI}{dt} = \frac{(C_1\beta_1 I\ + C_2\beta_2 T)S}{N} + (1-\epsilon)\phi I - (\delta_1 + \delta_2 + \mu)I$$

$$\frac{dT}{dt} = \delta_1 I + \upsilon A - (K + \mu)T$$

There are infected compartments here. The rates of change of the population of those compartments are given by (2) and (3) of the system. We can express F_i and V_i as follows:

$$F_i = \begin{bmatrix} \frac{(C_1\beta_1 I + C_2\beta_2 T)S}{N} \\ 0 \end{bmatrix} \text{And } V_i = \begin{bmatrix} (\delta_1 + \delta_2 + \mu)I - ((1-\epsilon)\varphi)I \\ (K+\mu)T - \delta_1 I - \upsilon A \end{bmatrix}$$

Then F and V were achieved by linearization. Then computing the partial derivative (difference) with respect to I and T, then evaluating them at the disease-free equilibrium pointE_0, which gives the Jacobian matrix.

$$F = \begin{bmatrix} C_1\beta_1 & C_2\beta_2 \\ 0 & 0 \end{bmatrix} \text{And } V = \begin{bmatrix} \delta_1 + \delta_2 + \mu - (1-\epsilon)\varphi & 0 \\ -\delta_1 & k+\mu \end{bmatrix}$$

$$V^{-1} = \left[\frac{\partial V_i}{\partial x_j}(E_0)\right]^{-1} = \begin{bmatrix} \frac{1}{\delta_1 + \delta_2 + \mu - (1-\epsilon)\varphi} & 0 \\ \frac{\delta_1}{\delta_1 + \delta_2 + \mu - (1-\epsilon)\varphi(k+\mu)} & \frac{1}{k+\mu} \end{bmatrix}$$

$$FV^{-1} = \left[\frac{\partial F_i}{\partial x_j}(E_0)\right]\left[\frac{\partial V_i}{\partial x_j}(E_0)\right]^{-1} = \begin{bmatrix} \frac{C_1\beta_1(k+\mu) + C_2\beta_2\delta_1}{(\delta_1+\delta_2+\mu-(1-\epsilon)\varphi)(k+\mu)} & \frac{C_2\beta_2}{(k+\mu)} \\ 0 & 0 \end{bmatrix}$$

$R_0 = \rho(FV^{-1})$, where ρ is the spectral radius (dominant matrix).

$R_0 = \max\{\lambda_1, \lambda_2\} = \lambda_2$

$$R_0 = \frac{C_1\beta_1(k+\mu) + C_2\beta_2\delta_1}{(\delta_1+\delta_2+\mu-(1-\epsilon)\varphi)(k+\mu)}$$

From here the subtraction of the positive term (1-ϵ)φ in the denominators comes from the vertical transmission that increases the value of the reproduction number (R_0). Moreover, the reproduction number R_0^* without vertical transmission modelled is given by

$$R_0^* = \left\{\frac{C_1\beta_1(k+\mu) + C_2\beta_2\delta_1}{(\delta_1+\delta_2+\mu)(k+\mu)}\right\}$$

From this we conclude that $R_0 > R_0^*$. This shows that the basic reproduction number of the present model is greater than the one that is obtained from the model without vertical transmission. This demonstrates that the spread of HIV/AIDS accelerates due to the horizontal transmission from the infected mother to the uninfected children.

3.5 The local stability of disease free equilibrium point

Theorem: The disease free equilibrium of the system is locally asymptotically stable if $R_0 < 1 and\ unstable\ if R_0 > 1$.

Proof

The local stability of E_0 needs to be obtained. The Variation matrix is calculated based on the equilibrium point E_0.

$J(E_0)$

$$= \begin{bmatrix} \frac{-(C_1\beta_1 I + C_2\beta_2 T)}{N} - \mu & -C_1\beta_1 & -C_2\beta_2 & 0 \\ \frac{(C_1\beta_1 I + C_2\beta_2 T)}{N} & C_1\beta_1 + (1-\varepsilon)\varphi - (\delta_1 + \delta_2 + \mu) & C_2\beta_2 & 0 \\ 0 & \delta_1 & -(k+\mu) & \nu \\ 0 & \delta_2 & k & -(\nu + \alpha + \mu) \end{bmatrix}$$

Since the $jacobian$ of at disease free equilibrium point $S = \frac{Q_0}{\mu}, \mathrm{I} = 0, \mathrm{T} = 0,\ \mathrm{A} = 0$

$$J(E_0) = \begin{bmatrix} -\mu & -C_1\beta_1 & -C_2\beta_2 & 0 \\ 0 & C_1\beta_1 + (1-\varepsilon)\varphi - (\delta_1 + \delta_2 + \mu) & C_2\beta_2 & 0 \\ 0 & \delta_1 & -(k+\mu) & \nu \\ 0 & \delta_2 & k & -(\nu + \alpha + \mu) \end{bmatrix}$$

Using the characteristics equation $(J(E_0) - \lambda I) = 0, \lambda_1 = -\mu,$ Then $(J(E_0) - \lambda I) = 0$

$f(\lambda) = \left(c_1\beta_1 + (1-\epsilon)\varphi - (\delta_1 + \delta_2 + \mu) - \lambda\right)(-(\mathrm{k} + \mu) - \lambda) + \delta_1 \mathrm{c}_2\beta_2 = 0$

$[\lambda^2 + \left[(\mathrm{k} + \mu) + (\delta_1 + \delta_2 + \mu) - \left(\mathrm{c}_1\beta_1 + (1-\epsilon)\right)\varphi\right]\lambda + (\mathrm{k} + \mu)((\delta_1 + \delta_2 + \mu) - (\mathrm{C}_1\beta_1 + (1-\epsilon)\varphi) + \delta_1 \mathrm{C}_2\beta_2 = 0$

$if\, c_1\beta_1 + (1-\epsilon)\varphi - (\delta_1 + \delta_2 + \mu) < 0$

$c_1\beta_1 + (1-\epsilon)\varphi < (\delta_1 + \delta_2 + \mu)$

$$\frac{c_1\beta_1 + (1-\epsilon)\varphi}{(\delta_1 + \delta_2 + \mu)} < 1$$

The model systems (1) to (4) are locally asymptotically stable according to the Routh Hurwitz criteria. It can be seen that all coefficients of the characteristics equation are positive (none are negative) and all roots of the polynomial f(λ) are negative or have negative real part provided that they are negative.

$R_0 = \frac{\mathrm{c}_1\beta_1(\mathrm{k}+\mu) + \mathrm{c}_2\beta_2\delta_1}{(\delta_1 + \delta_2 + \mu - (1-\epsilon)\varphi)(\mathrm{k}+\mu)}$ There fore $R_0 < 1$.

Proofs of the above theorem that is the disease free equilibrium of the system is locally asymptotically stable.

3.6 The endemic equilibrium point

The existence of the endemic equilibrium point of the modified model should now be studied. There is an equilibrium point E* where the disease persists in the population or if the carrier population is present in the system. For the existence and the uniqueness of endemic equilibrium point: $E^* = (S^*,\ I^*,\ T^*,\ A^*)$ its co-ordinate should be satisfying the following conditions:

$E^* = (S^*, \quad I^*, \quad T^*, \quad A^*) \neq 0.$

Where, $S^* > 0, I^* > 0, T^* > 0$ and $A^* > 0$. the endemic equilibrium point is obtained by solving the system of equations as:

$$Q_0 - \frac{(c_1\beta_1 I + c_2\beta_2 T)S}{N} - \mu S = 0 \qquad (13)$$

$$\frac{(c_1\beta_1 I + c_2\beta_2 T)S}{N} + (1-\epsilon)\varphi I - (\delta_1 + \delta_2 + \mu)I = 0 \qquad (14)$$

$$\delta_1 I - (k+\mu)T = 0 \qquad (15)$$

$$\delta_2 I + kT - (v+\alpha+\mu)A = 0 \qquad (16)$$

By adding the system of the equation (13) and (14) we get

$$Q_0 - \mu S + (1-\epsilon)\varphi I - (\delta_1 + \delta_2 + \mu)I = 0$$

$$S^* = \frac{Q_0 - (\delta_1 + \delta_2 + \mu - (1-\epsilon)\varphi)I^*}{\mu} \qquad (17)$$

From the equation (15) we have the following

$$T^* = \frac{\delta_1 I^*}{k+\mu} \qquad (18)$$

Here v= 0 since v is the rate of Aids population that transfers to treated

From equation (16) we have

$$A^* = \frac{\delta_2 I^* + kT^*}{v + \alpha + \mu} \tag{19}$$

Evaluate equation (17) and (18) into (14) and assume that $I = I^* = 1$we get

$$1 = \frac{(c_1\beta_1(k+\mu) + c_2\beta_2\delta_1)Q_0 - \mu N(k+\mu)(\delta_1 + \delta_2 + \mu - (1-\epsilon)\varphi)}{(c_1\beta_1(k+\mu) + c_2\beta_2\delta_1)(\delta_1 + \delta_2 + \mu - (1-\epsilon)\varphi)} \tag{20}$$

$$I^* = \frac{R_0Q_0 - \mu N}{R_0(\delta_1 + \delta_2 + \mu - (1-\epsilon)\varphi)} \quad \text{Since, } I^* = 1 \tag{21}$$

Evaluate (21) into (17) we have

$$S^* = \frac{N}{R_0} \tag{22}$$

Evaluating (21) in to (18)

$$T^* = \frac{\delta_1(R_0Q_0 - \mu N)}{R_0(k+\mu)(\delta_1 + \delta_2 + \mu - (1-\epsilon)\varphi)} \tag{23}$$

From the equation (19) $A^* becomes$:

$$A^* = \frac{1}{R_0}\left(\frac{(R_0Q_0 - \mu N)[(k+\mu)\delta_2 + k\delta_1]}{(k+\mu)(v+\alpha+\mu)[\delta_1 + \delta_2 + \mu - (1-\epsilon)\varphi]}\right) \tag{24}$$

$Therefore,$

$$S^* = \left\{\frac{N}{R_0} = \frac{N(\delta_1 + \delta_2 + \mu - (1-\epsilon)\varphi(k+\mu)}{c_1\beta_1(k+\mu) + c_2\beta_2\delta_1}\right\} \tag{25}$$

$$I^* = \left\{\frac{R_0Q_0 - \mu N}{R_0(\delta_1 + \delta_2 + \mu - (1-\epsilon)\varphi)}\right\} \tag{26}$$

$$T^* = \frac{\delta_1(R_0Q_0 - \mu N)}{R_0(k+\mu)(\delta_1 + \delta_2 + \mu - (1-\epsilon)\varphi)} \tag{27}$$

$$A^* = \frac{1}{R_0}\left(\frac{(R_0Q_0 - \mu N)[(k+\mu)\delta_2 + k\delta_1]}{(k+\mu)(v+\alpha+\mu)[\delta_1 + \delta_2 + \mu - (1-\epsilon)\varphi]}\right) \tag{28}$$

$LetX = (k+\mu)(v+\alpha+\mu) and Y = (\delta_1+\delta_2+\mu-(1-\epsilon)\varphi)$

$$A^* = \frac{1}{R_0}\left(\frac{(R_0Q_0-\mu N)[(k+\mu)\delta_2+k\delta_1]}{xy}\right) \quad (29)$$

3.6.1 Local stability of endemic equilibrium point

From (22) if $R_0 > 1, we\ see\ that\ I^* will\ be\ positive$. we also note that $E^* = (S^*, I^*, T^*, A^*)$is unique endemic equilibrium point which exists and positive whenever

$R_0 > 1, S^* > 0, I^* > 0, T^* > 0, A^* > 0$. We investigate the local stability of the endemic equilibrium point E^*. the disease presents (exists) in the population.

Theorem: The positive endemic equilibrium point E^* of the system of equation (1) to (4) is locally asymptotically stable if $R_0 > 1$.

Proof

Now the subsystem equations from (1) to (4) are the linearization of those subsystems at the endemic equilibrium points as follows:

$$J(E^*) = \begin{bmatrix} -q-\mu & \frac{-(c_1\beta_1 S^*)}{N} & \frac{-(c_2\beta_2 S^*)}{N} & 0 \\ q & \frac{c_1\beta_1 S^*}{N}+(1-\varepsilon)\varphi-(\delta_1+\delta_2+\mu) & \frac{(c_2\beta_2 S^*)}{N} & 0 \\ 0 & \delta_1 & -(k+\mu) & v \\ 0 & \delta_2 & k & -(v+\alpha+\mu) \end{bmatrix}$$

$Here\ q = \frac{c_1\beta_1 I^* + c_2\beta_2 T^*}{N}$.

$I^* > 0$, Since the disease persists (exist) in the population.

$\frac{R_0Q_0-\mu N}{R_0(\delta_1+\delta_2+\mu-(1-\epsilon)\varphi)} > 0,\ R_0Q_0 > \mu N, R_0 > \frac{\mu N}{Q_0}, R_0 > 1\ if and\ only$ if $\mu N = Q_0$.

Similarly, all endemic equilibrium points are expressed in terms of $R_0 and\ all\ are\ positive$. Therefore the system (1) to (4) is stable.

$parameter$	$value$	$source$	$Parameter$	$Value$	$source$
μ	0.4	$Estimate$	$\beta - 1$	0.3	$Estimate$
φ	0.04	$Estimate$	$\beta - 2$	0.04	$Estimate$
$\delta - 1$	0.4	$Estimate$	$C - 1$	1	$Estimate$
$\delta - 2$	0.03	$Estimate$	ϵ	0.2	$Estimate$
V	0.2	$Estimate$	K	0.09	$Estimate$
α	0.9	$Estimate$	$C - 2$	2	$Estimate$

Table 3.2: The parameter values by estimation.

Since, from the above parameter values we calculated the reproduction number as follows $R_0 = \frac{c_1\beta_1(k+\mu)+c_2\beta_2\delta_1}{(\delta_1+\delta_2+\mu-(1-\epsilon)\varphi)(k+\mu)} = \frac{1(0.3)(0.09+0.4)+2(0.04)(0.4)}{(0.4+0.03+0.4-(1-0.2)0.04)(0.09+0.4)} = \frac{0.147+0.032}{(0.83-0.032)(0.49)}$

$$= 0.45777709$$

CHAPTER FOUR

4. Numerical simulation

Data was obtained using ode45 in the Mat lab software for model simulation.

The starting value for the proportion of the values of used parameters in the simulation will be obtained based on these problems.

$$S = 300, I = 450, T = 650, A = 200, at\ time\ t_0 = 0\ and t_f = 15$$

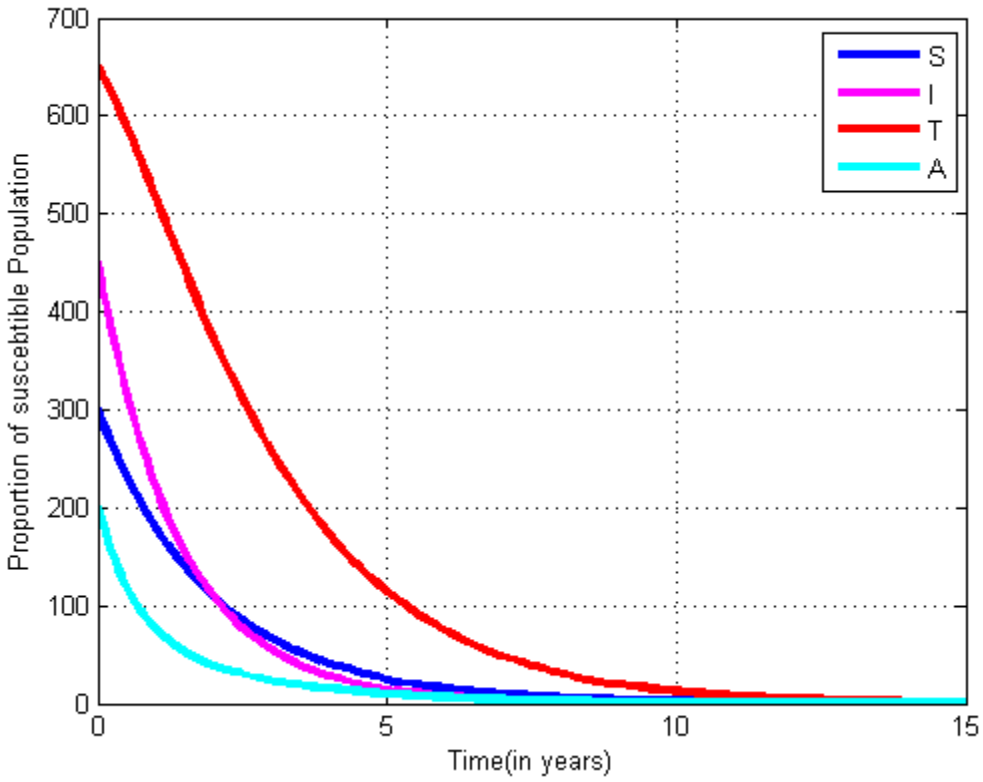

Figure 4.1: Variation of the different class $R_0 < 1$

From the above figure as time increase, the rate of treatment class is increasing and the susceptible population decrease due to inflow of infective immigrants and vertical transmission. Also the infected and AIDS class decrease at equilibrium point. Implies that the susceptible class is inversely related to the treated class.

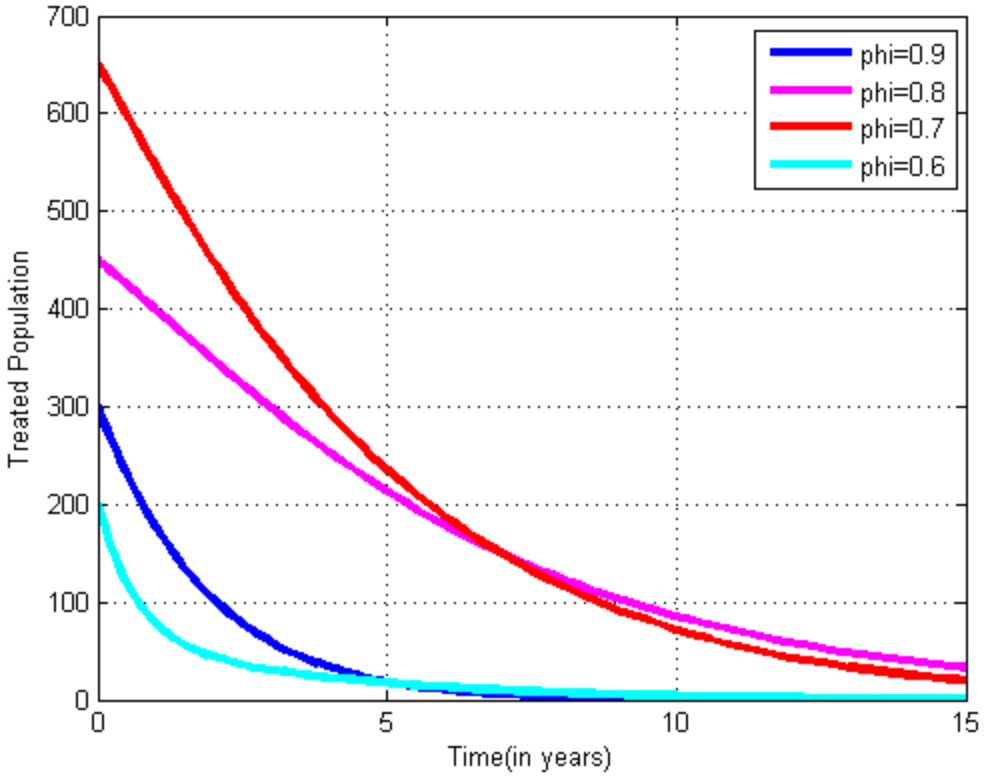

Figure 4.2: Depending on the given parameter$R_0 < 1$

The relation between phi (φ) with respect to the susceptible class, infected and treated class is given by: here as phi (φ) increase the susceptible class decrease. But the infected and the treated class are increase. Also the AIDS class decrease at equilibrium (i.e., as the value of φ increase T$> I > S > A$). But after certain period of time as time increase the susceptible and the treated class decrease whereas the infected class increases.

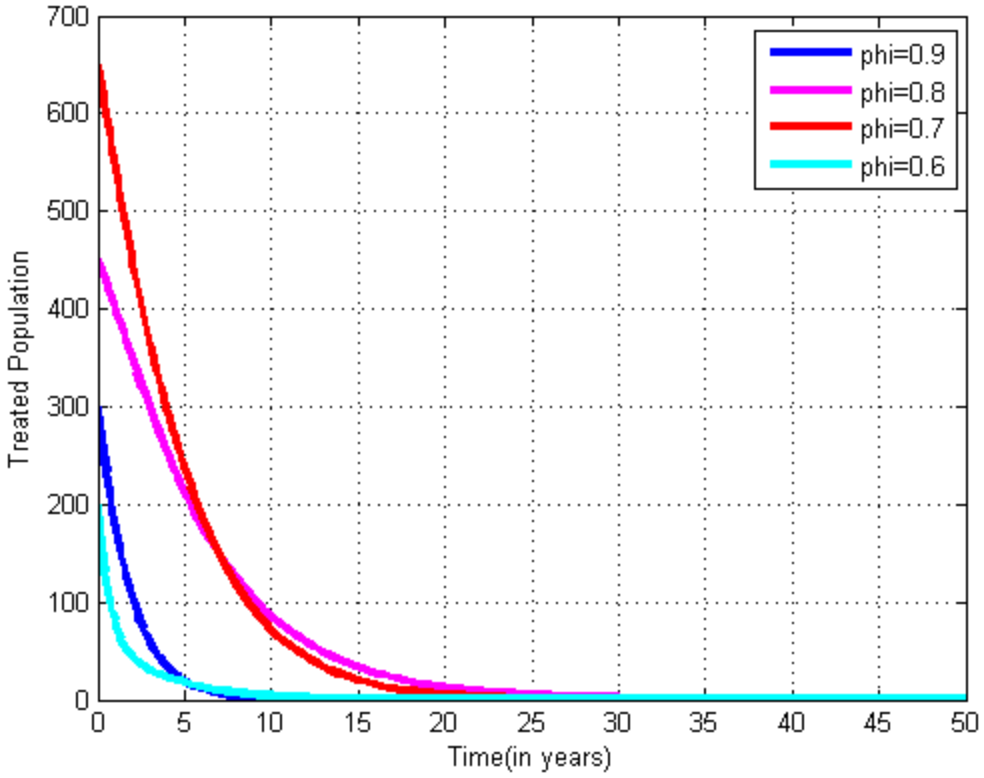

Figure 4. 3: the impact of vertical transmission in the given $R_0 < 1$

Here the relation between susceptible, infected and AIDS class with respect to time:

In a variety of time, the susceptible and the treated class decrease where as the infected class increase and the AIDS class decrease to zero at equilibrium position.

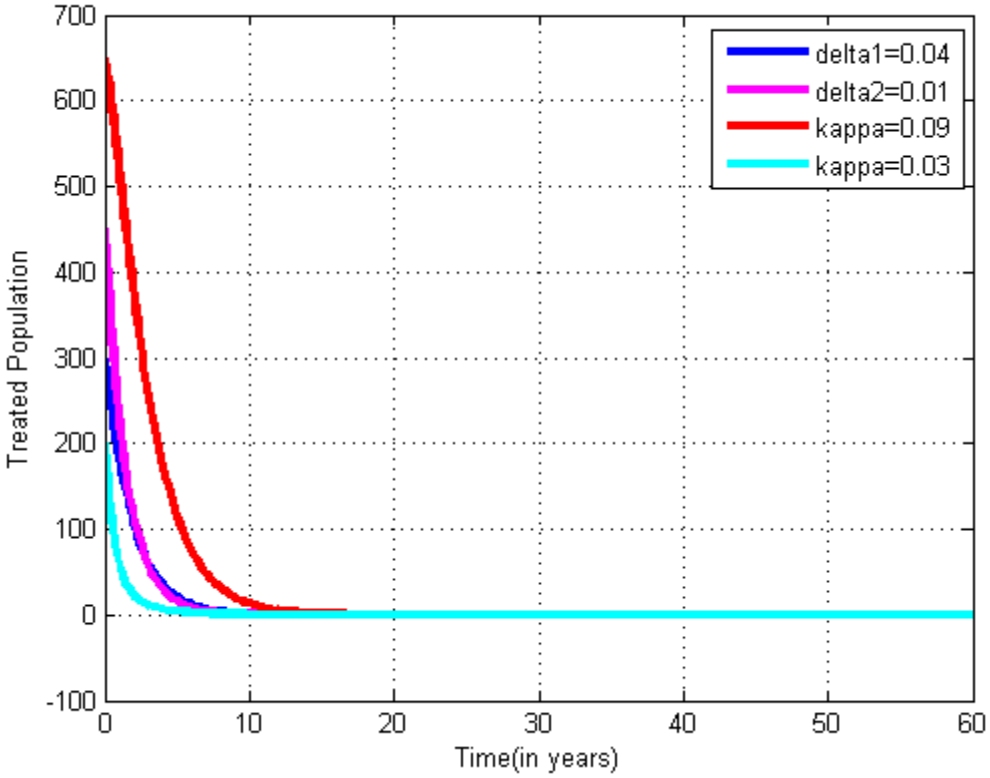

Figure 4.4: when $R_0 > 1$ in the impact of treatment and AIDS class.

According to the given values of parameters, the infected, treated and ADS class approaches to zero at equilibrium point as the susceptible class decreases.

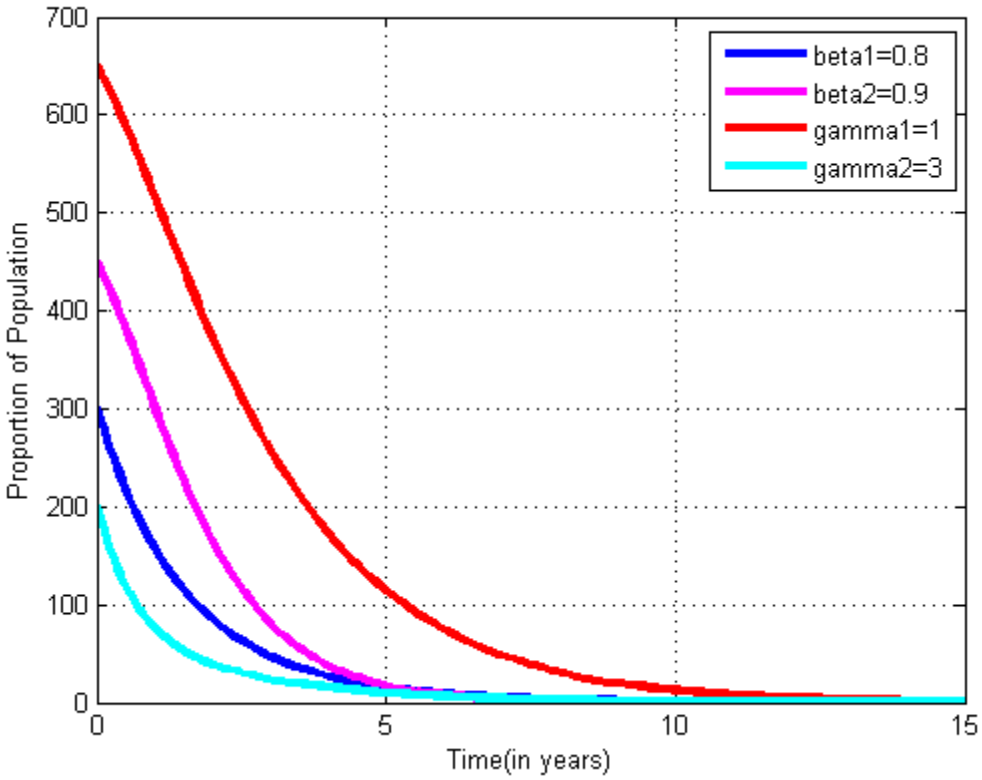

Figure 4.5: the relation between susceptible and infected class for $R_0 > 1$

Here the susceptible population decreases with time where as the rate of infection class increase due to inflow of infective immigrants and vertical transmission. Also the AIDS class decrease with time and it reaches at equilibrium position.

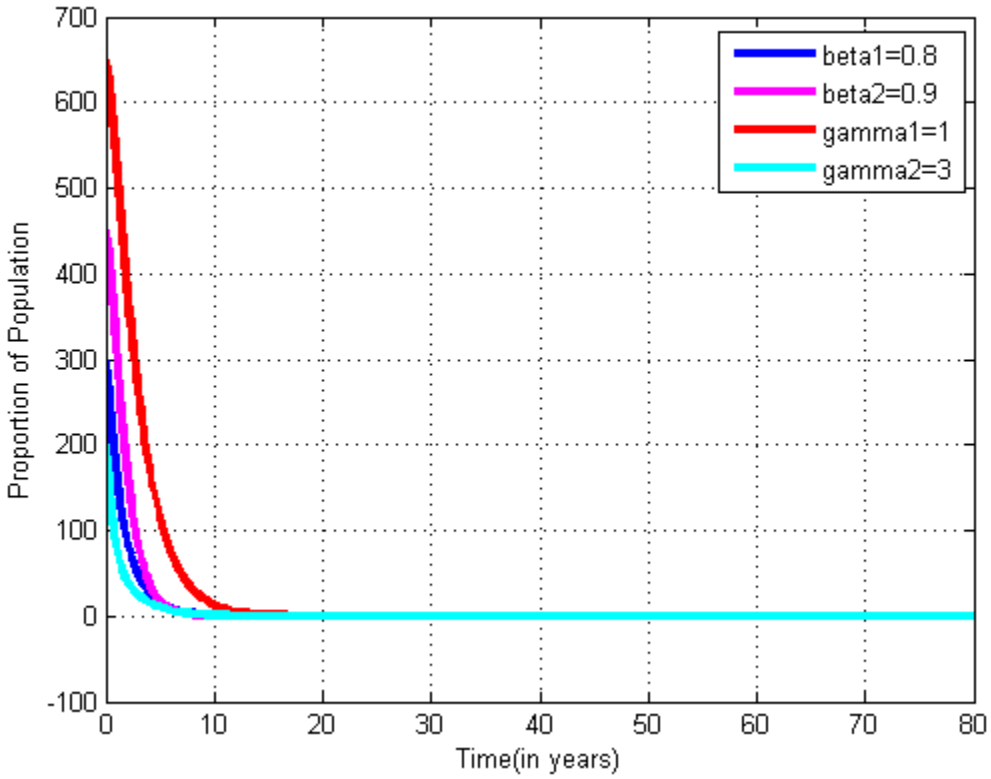

Figure 4.6: The variation of susceptible and infected for $R_0 > 1$

Here as the time increases all four different classes decreases and approaches to zero at equilibrium point.

4.1 Limitation of the thesis

The non linear mathematical model was studied in this thesis using qualitative data collected from the internet and secondary data.

One limitation is the limited access to internet in our area, which makes it difficult to get necessary information on time. Secondary data used in the study could contain inaccuracies or biases. Data recording on the different patient charts was poor. Baseline values of the variables are included in the study The study does not include CD4 cell count stability or improvement, weight loss or gain, treatment adherence, treatment switches or substitution, which are associated with mortality of AIDS patients.

4.2 Conclusion and result

We developed a non-linear deterministic model to study the dynamics of AIDS with treatment and vertical transmission in this thesis. The causes and symptoms of HIV/AIDS are described in the literature review as well as the mathematical model assumption, model formulation, the positivity of the solution, the disease free equilibrium point, and the basic reproduction number for the non-linear deterministic model. The model's local stability and the endemic equilibrium point are discussed as well.

In this thesis a non- linear mathematical model is proposed and analyzed in order to study the transmission of HIV/AIDS in a population of varying size with treatment and vertical transmission under the hypothesis that due to the sexual interaction of the susceptible with the infected one. The infected babies are born to increase the growth of infective population. It is assumed that people in AIDS classes are exposed and incapable of producing children.

By analyzing the model, we have found the threshold parameter R_0. It is noted that the disease dies out (the number of infected individual will decrease from generation to next asymptotically) when $R_0 < 1$ and the disease becomes endemic $when\ R_0 > 1$. so that the model has two non-negative equilibrium namely the disease free equilibrium $E_0 = (\frac{Q_0}{\mu}, 0, 0, 0)$and the endemic equilibrium$E^* = (S^*, I^*, T^*, A^*)$. It is found that the disease free equilibrium E_0 is asymptotically stable if $R_0 < 1$ and if $R_0 > 1$, the disease free equilibrium is unstable implies it will spread

without control measures and higher number are more likely to cause epidemics. In unstable equilibrium the infection is maintained in the population. The endemic equilibrium E^*that exists when $R_0 > 1$is always locally asymptotically stable.

If $R_0 = 1$, The disease becomes endemic, means the disease remains in the population at a constant rate, as one infected individual transmits the disease to one susceptible.

Here as the time increase all four different classes' decreases and approaches to zero at equilibrium point.

4.3 Recommendation

We recommend that the policy makers, healthy care workers, and an individual should be giving conclusion and education to the population to goes the clinic during birth and avoiding traditional birth, need to appreciate to the population to use ART treatment to decrease the transmission of HIV – AIDS. In addition to this Giving ART and medicine to the AIDS infectious because it decreases the number of AIDS stage.

To control the spread of the disease in the community we have to be sure that the numerical value of basic reproduction numbers are less than unity.

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APPENDIX

MATLAB CODE FOR NUMERICAL SIMULATIONS:

```
functiondy =sita1(t,y)
global alpha beta1 beta2 gamma1 gamma2 delta1 delta2 epsilon kappa mu nu rho
phi
alpha=0.9; beta1=0.3; beta2=0.04; gamma1=1; gamma2=2; delta1=0.4;
delta2=0.03; epsilon=0.2;
kappa=0.09; mu=0.4; nu=0.2; rho=0.04; phi=0.04;
dy=[0 0 0 0]';
N=y(1)+y(2)+y(3)+y(4);
dy(1)=rho-mu*y(1)-(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N;
dy(2)=(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N+(1-epsilon)*phi*y(2)-
(mu+delta1+delta2)*y(2);
dy(3)=delta1*y(2)+nu*y(4)-(mu+kappa)*y(3);
dy(4)=delta2*y(2)+kappa*y(3)-(mu+alpha+nu)*y(4);
R0=(gamma1*beta1*(kappa+mu)+gamma2*beta2*delta1)/(kappa+mu)*(delta1+delta2+mu
-(1-epsilon)*phi)
end
```

Matlab Script

```
clear
closeall
formatlong
options=odeset('RelTol',1e-4,'AbsTol',[1e-4 1e-4 1e-4 1e-4]);
[t,y]=ode45('sita1',[0 15],[300 450 650 200],options);
plot(t,y(:,1),'-b',t,y(:,2),'-m',t,y(:,3),'-r',t,y(:,4),'-c','LineWidth',2)
xlabel('Time(in years)')
ylabel('Proportion of suscebtible Population')
legend('S','I','T','A')
gridon
functiondy =sita21(t,y)
global alpha beta1 beta2 gamma1 gamma2 delta1 delta2 epsilon kappa mu nu rho
phi
alpha=0.9; beta1=0.3; beta2=0.04; gamma1=1; gamma2=2; delta1=0.4;
delta2=0.03; epsilon=0.2;
kappa=0.09; mu=0.4; nu=0.2; rho=0.04; phi=0.9;
dy=[0 0 0 0]';
N=y(1)+y(2)+y(3)+y(4);
dy(1)=rho-mu*y(1)-(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N;
dy(2)=(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N+(1-epsilon)*phi*y(2)-
(mu+delta1+delta2)*y(2);
dy(3)=delta1*y(2)+nu*y(4)-(mu+kappa)*y(3);
dy(4)=delta2*y(2)+kappa*y(3)-(mu+alpha+nu)*y(4);
end
functiondy =sita22(t,y)
global alpha beta1 beta2 gamma1 gamma2 delta1 delta2 epsilon kappa mu nu rho
phi
alpha=0.9; beta1=0.3; beta2=0.04; gamma1=1; gamma2=2; delta1=0.4;
delta2=0.03; epsilon=0.2;
kappa=0.09; mu=0.4; nu=0.2; rho=0.04; phi=0.8;
dy=[0 0 0 0]';
N=y(1)+y(2)+y(3)+y(4);
dy(1)=rho-mu*y(1)-(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N;
dy(2)=(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N+(1-epsilon)*phi*y(2)-
(mu+delta1+delta2)*y(2);
```

```
dy(3)=delta1*y(2)+nu*y(4)-(mu+kappa)*y(3);
dy(4)=delta2*y(2)+kappa*y(3)-(mu+alpha+nu)*y(4);
end
functiondy =sita23(t,y)
global alpha beta1 beta2 gamma1 gamma2 delta1 delta2 epsilon kappa mu nu rho
phi
alpha=0.9; beta1=0.3; beta2=0.04; gamma1=1; gamma2=2; delta1=0.4;
delta2=0.03; epsilon=0.2;
kappa=0.09; mu=0.4; nu=0.2; rho=0.04; phi=0.7;
dy=[0 0 0 0]';
N=y(1)+y(2)+y(3)+y(4);
dy(1)=rho-mu*y(1)-(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N;
dy(2)=(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N+(1-epsilon)*phi*y(2)-
(mu+delta1+delta2)*y(2);
dy(3)=delta1*y(2)+nu*y(4)-(mu+kappa)*y(3);
dy(4)=delta2*y(2)+kappa*y(3)-(mu+alpha+nu)*y(4);
end
functiondy =sita24(t,y)
global alpha beta1 beta2 gamma1 gamma2 delta1 delta2 epsilon kappa mu nu rho
phi
alpha=0.9; beta1=0.3; beta2=0.04; gamma1=1; gamma2=2; delta1=0.4;
delta2=0.03; epsilon=0.2;
kappa=0.09; mu=0.4; nu=0.2; rho=0.04; phi=0.6;
dy=[0 0 0 0]';
N=y(1)+y(2)+y(3)+y(4);
dy(1)=rho-mu*y(1)-(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N;
dy(2)=(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N+(1-epsilon)*phi*y(2)-
(mu+delta1+delta2)*y(2);
dy(3)=delta1*y(2)+nu*y(4)-(mu+kappa)*y(3);
dy(4)=delta2*y(2)+kappa*y(3)-(mu+alpha+nu)*y(4);
end
clear
closeall
formatlong
options=odeset('RelTol',1e-4,'AbsTol',[1e-4 1e-4 1e-4 1e-4]);
[t1,y1]=ode45('sita21',[0 15],[300 450 650 200],options);
[t2,y2]=ode45('sita22',[0 15],[300 450 650 200],options);
[t3,y3]=ode45('sita23',[0 15],[300 450 650 200],options);
[t4,y4]=ode45('sita24',[0 15],[300 450 650 200],options);
plot(t1,y1(:,1),'-b',t2,y2(:,2),'-m',t3,y3(:,3),'-r',t4,y4(:,4),'-
c','LineWidth',2);
xlabel('Time(in years)');
ylabel('Treated Population');
legend('phi=0.9','phi=0.8','phi=0.7','phi=0.6');
gridon;
beta1=0.3; beta2=0.04; gamma1=1; gamma2=2; delta1=0.4; delta2=0.03;
epsilon=0.2;
kappa=0.09; mu=0.4; phi=0.75;
R0=(gamma1*beta1*(kappa+mu)+gamma2*beta2*delta1)/(kappa+mu)*(delta1+delta2+mu
-(1-epsilon)*phi)
clear
closeall
formatlong
options=odeset('RelTol',1e-4,'AbsTol',[1e-4 1e-4 1e-4 1e-4]);
[t1,y1]=ode45('sita21',[0 50],[300 450 650 200],options);
[t2,y2]=ode45('sita22',[0 50],[300 450 650 200],options);
[t3,y3]=ode45('sita23',[0 50],[300 450 650 200],options);
```

```
[t4,y4]=ode45('sita24',[0 50],[300 450 650 200],options);
plot(t1,y1(:,1),'-b',t2,y2(:,2),'-m',t3,y3(:,3),'-r',t4,y4(:,4),'-
c','LineWidth',2);
xlabel('Time(in years)');
ylabel('Treated Population');
legend('phi=0.9','phi=0.8','phi=0.7','phi=0.6');
gridon;
beta1=0.3; beta2=0.04; gamma1=1; gamma2=2; delta1=0.4; delta2=0.03;
epsilon=0.2;
kappa=0.09; mu=0.4; phi=0.75;
R0=(gamma1*beta1*(kappa+mu)+gamma2*beta2*delta1)/(kappa+mu)*(delta1+delta2+mu
-(1-epsilon)*phi)
functiondy =sita41(t,y)
global alpha beta1 beta2 gamma1 gamma2 delta1 delta2 epsilon kappa mu nu rho
phi
alpha=0.9; beta1=0.3; beta2=0.04; gamma1=1; gamma2=2; delta1=0.04;
delta2=0.03; epsilon=0.2;
kappa=0.09; mu=0.4; nu=0.2; rho=0.04; phi=0.04;
dy=[0 0 0 0]';
N=y(1)+y(2)+y(3)+y(4);
dy(1)=rho-mu*y(1)-(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N;
dy(2)=(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N+(1-epsilon)*phi*y(2)-
(mu+delta1+delta2)*y(2);
dy(3)=delta1*y(2)+nu*y(4)-(mu+kappa)*y(3);
dy(4)=delta2*y(2)+kappa*y(3)-(mu+alpha+nu)*y(4);
end
functiondy =sita42(t,y)
global alpha beta1 beta2 gamma1 gamma2 delta1 delta2 epsilon kappa mu nu rho
phi
alpha=0.9; beta1=0.3; beta2=0.04; gamma1=1; gamma2=2; delta1=0.4;
delta2=0.01; epsilon=0.2;
kappa=0.09; mu=0.4; nu=0.2; rho=0.04; phi=0.04;
dy=[0 0 0 0]';
N=y(1)+y(2)+y(3)+y(4);
dy(1)=rho-mu*y(1)-(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N;
dy(2)=(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N+(1-epsilon)*phi*y(2)-
(mu+delta1+delta2)*y(2);
dy(3)=delta1*y(2)+nu*y(4)-(mu+kappa)*y(3);
dy(4)=delta2*y(2)+kappa*y(3)-(mu+alpha+nu)*y(4);
end
functiondy =sita43(t,y)
global alpha beta1 beta2 gamma1 gamma2 delta1 delta2 epsilon kappa mu nu rho
phi
alpha=0.9; beta1=0.3; beta2=0.04; gamma1=1; gamma2=2; delta1=0.4;
delta2=0.03; epsilon=0.2;
kappa=0.09; mu=0.4; nu=0.2; rho=0.04; phi=0.04;
dy=[0 0 0 0]';
N=y(1)+y(2)+y(3)+y(4);
dy(1)=rho-mu*y(1)-(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N;
dy(2)=(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N+(1-epsilon)*phi*y(2)-
(mu+delta1+delta2)*y(2);
dy(3)=delta1*y(2)+nu*y(4)-(mu+kappa)*y(3);
dy(4)=delta2*y(2)+kappa*y(3)-(mu+alpha+nu)*y(4);
end
functiondy =sita44(t,y)
global alpha beta1 beta2 gamma1 gamma2 delta1 delta2 epsilon kappa mu nu rho
phi
```

```
alpha=0.9; beta1=0.3; beta2=0.04; gamma1=1; gamma2=2; delta1=0.4;
delta2=0.03; epsilon=0.2;
kappa=0.03; mu=0.4; nu=0.2; rho=0.04; phi=0.04;
dy=[0 0 0 0]';
N=y(1)+y(2)+y(3)+y(4);
dy(1)=rho-mu*y(1)-(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N;
dy(2)=(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N+(1-epsilon)*phi*y(2)-
(mu+delta1+delta2)*y(2);
dy(3)=delta1*y(2)+nu*y(4)-(mu+kappa)*y(3);
dy(4)=delta2*y(2)+kappa*y(3)-(mu+alpha+nu)*y(4);
end
clear
closeall
formatlong
options=odeset('RelTol',1e-4,'AbsTol',[1e-4 1e-4 1e-4 1e-4]);
[t1,y1]=ode45('sita41',[0 60],[300 450 650 200],options);
[t2,y2]=ode45('sita42',[0 60],[300 450 650 200],options);
[t3,y3]=ode45('sita43',[0 60],[300 450 650 200],options);
[t4,y4]=ode45('sita44',[0 60],[300 450 650 200],options);
plot(t1,y1(:,1),'-b',t2,y2(:,2),'-m',t3,y3(:,3),'-r',t4,y4(:,4),'-
c','LineWidth',2);
xlabel('Time(in years)');
ylabel('Treated Population');
legend('delta1=0.04','delta2=0.01','kappa=0.09','kappa=0.03');
gridon;
beta1=0.4; beta2=0.5; gamma1=1; gamma2=3; delta1=0.4; delta2=0.01;
epsilon=0.2;
kappa=0.06; mu=0.4; phi=0.001;
R0=(gamma1*beta1*(kappa+mu)+gamma2*beta2*delta1)/(kappa+mu)*(delta1+delta2+mu
-(1-epsilon)*phi)
functiondy =sita51(t,y)
global alpha beta1 beta2 gamma1 gamma2 delta1 delta2 epsilon kappa mu nu rho
phi
alpha=0.9; beta1=0.8; beta2=0.04; gamma1=1; gamma2=2; delta1=0.4;
delta2=0.03; epsilon=0.2;
kappa=0.09; mu=0.4; nu=0.2; rho=0.04; phi=0.04;
dy=[0 0 0 0]';
N=y(1)+y(2)+y(3)+y(4);
dy(1)=rho-mu*y(1)-(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N;
dy(2)=(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N+(1-epsilon)*phi*y(2)-
(mu+delta1+delta2)*y(2);
dy(3)=delta1*y(2)+nu*y(4)-(mu+kappa)*y(3);
dy(4)=delta2*y(2)+kappa*y(3)-(mu+alpha+nu)*y(4);
end
functiondy =sita52(t,y)
global alpha beta1 beta2 gamma1 gamma2 delta1 delta2 epsilon kappa mu nu rho
phi
alpha=0.9; beta1=0.3; beta2=0.9; gamma1=1; gamma2=2; delta1=0.4; delta2=0.03;
epsilon=0.2;
kappa=0.09; mu=0.4; nu=0.2; rho=0.04; phi=0.04;
dy=[0 0 0 0]';
N=y(1)+y(2)+y(3)+y(4);
dy(1)=rho-mu*y(1)-(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N;
dy(2)=(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N+(1-epsilon)*phi*y(2)-
(mu+delta1+delta2)*y(2);
dy(3)=delta1*y(2)+nu*y(4)-(mu+kappa)*y(3);
dy(4)=delta2*y(2)+kappa*y(3)-(mu+alpha+nu)*y(4);
```

```
end
functiondy =sita53(t,y)
global alpha beta1 beta2 gamma1 gamma2 delta1 delta2 epsilon kappa mu nu rho
phi
alpha=0.9; beta1=0.3; beta2=0.04; gamma1=1; gamma2=2; delta1=0.4;
delta2=0.03; epsilon=0.2;
kappa=0.09; mu=0.4; nu=0.2; rho=0.04; phi=0.04;
dy=[0 0 0 0]';
N=y(1)+y(2)+y(3)+y(4);
dy(1)=rho-mu*y(1)-(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N;
dy(2)=(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N+(1-epsilon)*phi*y(2)-
(mu+delta1+delta2)*y(2);
dy(3)=delta1*y(2)+nu*y(4)-(mu+kappa)*y(3);
dy(4)=delta2*y(2)+kappa*y(3)-(mu+alpha+nu)*y(4);
end
functiondy =sita54(t,y)
global alpha beta1 beta2 gamma1 gamma2 delta1 delta2 epsilon kappa mu nu rho
phi
alpha=0.9; beta1=0.3; beta2=0.04; gamma1=1; gamma2=3; delta1=0.4;
delta2=0.03; epsilon=0.2;
kappa=0.09; mu=0.4; nu=0.2; rho=0.04; phi=0.04;
dy=[0 0 0 0]';
N=y(1)+y(2)+y(3)+y(4);
dy(1)=rho-mu*y(1)-(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N;
dy(2)=(gamma1*beta1*y(2)+gamma2*beta2*y(3))*y(1)/N+(1-epsilon)*phi*y(2)-
(mu+delta1+delta2)*y(2);
dy(3)=delta1*y(2)+nu*y(4)-(mu+kappa)*y(3);
dy(4)=delta2*y(2)+kappa*y(3)-(mu+alpha+nu)*y(4);
end
clear
closeall
formatlong
options=odeset('RelTol',1e-4,'AbsTol',[1e-4 1e-4 1e-4 1e-4]);
[t1,y1]=ode45('sita51',[0 15],[300 450 650 200],options);
[t2,y2]=ode45('sita52',[0 15],[300 450 650 200],options);
[t3,y3]=ode45('sita53',[0 15],[300 450 650 200],options);
[t4,y4]=ode45('sita54',[0 15],[300 450 650 200],options);
plot(t1,y1(:,1),'-b',t2,y2(:,2),'-m',t3,y3(:,3),'-r',t4,y4(:,4),'-
c','LineWidth',2);
xlabel('Time(in years)');
ylabel(Proportion of Population');
legend('beta1=0.8','beta2=0.9','gamma1=1','gamma2=3');
gridon;
beta1=0.7; beta2=0.8; gamma1=1; gamma2=3; delta1=0.4; delta2=0.01;
epsilon=0.2;
kappa=0.06; mu=0.4; phi=0.001;
R0=(gamma1*beta1*(kappa+mu)+gamma2*beta2*delta1)/(kappa+mu)*(delta1+delta2+mu
-(1-epsilon)*phi)
clear
closeall
formatlong
options=odeset('RelTol',1e-4,'AbsTol',[1e-4 1e-4 1e-4 1e-4]);
[t1,y1]=ode45('sita51',[0 80],[300 450 650 200],options);
[t2,y2]=ode45('sita52',[0 80],[300 450 650 200],options);
[t3,y3]=ode45('sita53',[0 80],[300 450 650 200],options);
[t4,y4]=ode45('sita54',[0 80],[300 450 650 200],options);
```

```
plot(t1,y1(:,1),'-b',t2,y2(:,2),'-m',t3,y3(:,3),'-r',t4,y4(:,4),'-
c','LineWidth',2);
xlabel('Time(in years)');
ylabel('Proportion of Population');
legend('beta1=0.8','beta2=0.9','gamma1=1','gamma2=3');
gridon;
beta1=0.8; beta2=0.9; gamma1=1; gamma2=3; delta1=0.4; delta2=0.01;
epsilon=0.2;
kappa=0.06; mu=0.4; phi=0.001;
R0=(gamma1*beta1*(kappa+mu)+gamma2*beta2*delta1)/(kappa+mu)*(delta1+delta2+mu
-(1-epsilon)*phi)
```