MATCH ANALYSIS OF HIV/AIDS MODEL WITH REFERENCE TO KENYA

BY

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Abstract

Epidemiology models are used to combine complex data from various sources in order to study equally complex outcomes. The first simple HIV/AIDS epidemic model was by Anderson in 1986. Since then a lot has been done in terms of mathematical models to help understand the trends in the spread of the HIV/AIDS pandemic. Researchers over the years have concentrated on certain aspects of the disease at a time depending on the area of interest of the researcher sometimes with conflicting results partly because only some aspects of the disease were considered. No single model incorporating social behaviour, treatment, vaccination, stages of infection age structures and vertical transmission has been developed. In this study a comprehensive deterministic HIV/AIDS transmission model incorporating all the above has been formulated using differential equations. Different parameter values have been simulated numerically and the spread of the disease monitored against time to determine their effects on the HIV/AIDS spread using the Wolfram Research Mathematica Software. This study harmonizes the existing models into a single model to study various aspects of the HIV/AIDS Pandemic.

Chapter 1

Introduction

1.1 Introduction to Mathematical Models

Mathematical models have become important tools in analyzing the spread and control of infectious diseases. Computer simulations and mathematical models are useful experimental tools for building and testing theories, assessing quantitative conjectures, determining sensitivities to changes in parameter values and answering specific questions. They provide a broad conceptual framework within which we can formulate sensible questions and hopefully find meaningful answers.

HIV disease is characterized by a gradual deterioration of the immune function of a human being. HIV causes AIDS by triggering events that weaken a person's immune function. It has no cure and no vaccine as yet. The magnitude and severity of the problem of HIV/AIDS became increasingly evident in the early 1990's with a prevalence rate of about 12 percent in Africa [37]. In some communities i.e in Uganda a whole generation was wiped out [33]. Therefore the need to develop effective

strategies to prevent and control HIV infection became more urgent. The trend changed in the late 1990's with most Afican countries recording lower incidence rates and prevalence rates [34].

Mathematical modeling of HIV/AIDS usually takes two broad approaches. The first approach involves modeling HIV/AIDS transmission from Infectives to susceptibles over some period of time. The second approach models HIV/AIDS at the cellular or molecular level within an infected individual. The basis for most National HIV projections is usually a simple mathematical model often based on a single-stage (SS) model to fit the observed prevalence patterns yet HIV dynamics are quite complex. The infectiousness of HIV infected individuals is known to vary with stage of infection, being highly infectious in the first few weeks after becoming infected, then having low infectivity for many years, and finally becoming gradually more infectious as the immune system of those infected with HIV breaks down. The infection rates for the first and third stages have been documented to be several times higher than those for the second stage. Models that account for such stages are called staged progression (SP) models.

The first simple HIV Mathematical epidemic model goes back to Anderson 1986 [1]. By then behaviour change was recognized as the major way of combating the spread of HIV/AIDS epidemic given that there was no treatment or vaccine to the virus. This was later followed in the 1990's by Raw et al, [24] who modeled the effect of combination of anti-retroviral treatments with different levels of unsafe sex on HIV incidence among homosexual men in Australia when effective Anti-retroviral first became widely available. Recent models have involved aspects such as

stage of infection, vertical transmission, age structure, social and sexual mixing groups, treatment, vaccination etc.

In Kenya, HIV/AIDS epidemic peaked in the 1997/98 with an overall prevalence rate of 10 percent among adults but has halved in a decade - a dramatic and sustained decline that has rarely been seen in Africa. The most recent modeling of sentinel surveillance data from the websites of The Kenya National Aids Control Council [20] indicates that prevalence stood at 5.1 percent among adults at the end of 2006 compared with 10 percent in 1997/98. This turn-around can be attributed to greater awareness and resulting behavior change as well as a lower incidence of new infections and higher death rates. There is a strong evidence to suggest that there has been a reduction in risky behavior such as through increased condom use, delay in sexual debut and fewer partners [20]. These are indicators of a change in social behaviour due to counseling leading to a reduction in the rate of infection. The number of those receiving Antiretroviral treatment has also increased from 50,000 in 2005 to 600000 by 2008 [17]. Anti- retroviral therapy has the effect of increasing the incubation period of HIV before developing AIDS meaning that the infectives live for more years before they die which may lead to increases in new infections if there is no change in social behaviour. Research has shown that ARV's increases the lifespan of the infectives from 8 years to 15 years [13]. Treatment also has the effect of reducing the viral load among the infectives which could reduce the probability of getting an infection from a new sexual partner. Prevention of mother to child transmission (PMCT) have reduced the risk of transmission of HIV infected mothers to there babies by seventy percent [9].

Mathematical modeling of HIV/AIDS incorporating vaccination is not difficult. This is because vaccination reduces the number of susceptibles in the population thus reducing the number of new infections. The struggle for a safe and effective vaccine has been on going for over twenty years but still remains a difficult target, however recent studies done in Thailand hinted that some vaccine for some strain of the virus has been found with an efficacy level of 30 percent [4].

A lot of work has been done involving HIV/AIDS Mathematical modeling with researchers over the years concentrating on certain aspects of the disease one at a time depending on the areas of interest i.e counseling, treatment, circumcision, vaccination e.t.c. This study has developed a comprehensive deterministic HIV/AIDS transmission model incorporating counseling, treatment, vaccination, stages of infection, Age structures and vertical transmission.

1.2 Statement of the problem

A lot of work has been done involving HIV/AIDS mathematical modeling worldwide. Researchers over the years have concentrated on certain aspects of the disease one at a time depending on the area of interest i.e social behaviour, treatment, vaccination, stages of infection, circumcision, age structures, vertical transmission, population size e.t.c sometimes with parallel results partly because only some aspects of the disease are considered. Some models incorporating counseling, anti-retroviral therapy and vertical transmission assuming stages of infection have shown that treatment that is not accompanied by a positive change in social be-

haviour increases the number of both child and adult infectives in the population while other models incorporating stages of infection have shown that treatment is very significant in controlling the spread of HIV/AIDS scourge.

The struggle for a safe and effective vaccine has also been on going for many years but still remains elusive, however recent studies done in Thailand hinted that some vaccine for some strain of the HIV/AIDS virus has been found with an efficacy level of 30 percent. This study matches and harmonizes the existing models so that a single model can now be used to study various aspects of the HIV/AIDS Pandemic.

1.3 Objectives of the study

The aim of this study is to provide a comprehensive model incorporating counseling, treatment, vaccination, stages of infection, age structures, vertical transmission, and the population size.

The specific objective are as follows:

1. Determine wether a trade off exists between vaccination and treatment.

2. Investigate the effectiveness of a vaccine with an efficacy level of 30 percent in combating the the HIV/AIDS epidemic when treatment is being applied

1.4 Research methodology

In this study, we have modeled HIV/AIDS epidemic incorporating counseling, treatment, vaccination, stages of infection, age structures and vertical transmission using differential equations. A comprehensive model harmonizing the existing models presented in the literature review has been developed. The model is analyzed to determine the possible existence of equilibria and stability. Secondary data obtained from [20] and [17], have been simulated numerically incorporating different values for the parameters using the Wolfram Research Mathematica Software.

1.5 Significance of the study

This study harmonizes the existing models so that a single model can now be used to study all or most aspects of the HIV/AIDS Pandemic. The study also shows the relationships of the variables and parameters incorporated especially vaccination and treatment and hence predict the effectiveness of a vaccine with an efficacy level of 30 percent.

1.6 Outline of the thesis

Chapter one gives background information on the genesis of mathematical models.

Chapter two presents literature review of the researches that have been done in developing epidemic models.

Chapter three gives basic concepts in Mathematical epidemiology and the comprehensive model formulated.

In chapter four, we analyze the stability of the disease free equilibrium and calculate the basic reproduction number, R_0.

Chapter five and six presents numerical simulations of the model.

Chapter seven gives summery, recommendation and conclusion.

Chapter 2

Literature Review

Some of the first epidemiology models go back to Bernoulli 1760 [5]. He formulated and solved a mathematical model for smallpox in order to evaluate the effectiveness of variolation of heathy people with the smallpox virus. Although a model for small pox was formulated in 1760 by Bernoulli, deterministic epidemiology modeling seems to have started in the 20th century. In 1906 Hamer [14] formulated and analyzed a discrete time model in his attempt to understand the recurrence of measles epidemics. His model may have been the first to assume that the incidence (number of new cases per unit time) depends on the product of the densities of the susceptibles and infectives. Starting in 1926, Kermack and Mackendric [21] published papers on epidemic models and obtained the epidemic threshold result that the density of susceptibles must exceed a critical value in order for an epidemic outbreak to occur. The first simple HIV epidemic model was by Anderson 1986 [1]. Behaviour change was recognized as the major way of combating the spread of HIV/AIDS epidemic given that there was no treatment or vaccine to the virus. He showed that counseling is an effective method in the control of HIV/AIDS.

After the discovery of Anti-retroviral treatment, modeling of HIV/AIDS was directed towards incorporating behaviour change and effects of treatment. Incorporating treatment and social behaviour posed a challenge to HIV/AIDS mathematical modeling because treatment acts both in the positive and negative direction. It reduces the infectiousness of an infected individual reducing the probability of transmission from an infective to a susceptible. On the contrary anti-retroviral therapies increases the lifespan of the HIV infectives and as such they can infect more people if there is no change in their social behaviour.

These directions included the models by Hernandez and Hsieh 1994 [35], who concluded that only significant reductions in the transmission probability can contain the spread of the epidemic. Such reductions could be through adoption of safer sexual practices or through reductions in viral load due to treatment. A model by Yen and Cooke 2000 [38], on behaviour change and treatment of core groups and its effects on the spread of HIV/AIDS showed that behaviour change and treatment can eradicate the disease however if the treatment and behaviour change levels do not reach critical values, detrimental effects could be realized resulting from slower progresion to AIDS without sufficiently lower transmission rates resulting in increased spread of HIV infection. Raw et al. 2001 [24], modeled the effect of combination of anti-retroviral treatments with different levels of unsafe sex on HIV incidence in Australia when effective anti-retroviral treatments first became widely available in Australia. The results suggested that decreases in HIV incidences through large decreases in infectiousness as a result of combination anti-retroviral treatment could be counterbalanced by much more modest increases in the levels of unsafe

sex.

Anti-retroviral treatment also reduces the risk of transmission of HIV from HIV infected mothers to there unborn babies (Vertical transmission or mother to child transmission) by over seventy percent [9]. Models incorporating vertical transmission have been developed by many scholars. Among them were the models by Mugisha and Luboobi 2003 [25], who modeled the effect of vertical transmission in the dynamics of HIV/AIDS in an age structured population.

Age structured population models for the dynamics of HIV/AIDS are of importance in understanding the actual impact the spread has on a particular age group of interest. Such models do give a clear clue as to which age group should be concentrated on in terms of treatment, education and the kind of strategies for containing the spread. The most vulnerable age group for HIV/AIDS are those between 15 - 49 years [20]. These age structured models have something in common: delay in sexual maturity. This makes the formulation of the models have delay differential equations. Delay differential equations tend to have analytical complications due to the nature of the formulations involved and as such obtaining important population estimations becomes difficult.

Blynthe and Anderson 1988 [7], formulated age - structured models to study the effect of sexual activity levels in a continuous age structured population. Anderson et al 1991 [1] developed age structured models to study the role of sexual contact and proportionate mixing in a population of HIV/AIDS. Mugisha and Luboobi 2003 [25], derived a deterministic age-structured model in a two age-groups population with a constant

HIV prevalence rate and a variable prevalence rate (Force of infection to behave as a mass action).

HIV/AIDS models have been formulated to incorporate vaccination though a vaccine does not exist as yet, however recent studies done in Thailand hinted that some vaccine for some strain of the HIV virus has been discovered with an efficacy level of 30 percent. [4].

Wells 1998 [36], in his paper, "even imperfect vaccines could be valuable", suggested that vaccines against the AIDS causing virus could save money, extend lives and prevent deaths even if these vaccines are only moderately effective in preventing or treating the infection. Mills et al. 2001 [6], studied the effects of the live attenuated HIV vaccines in protecting against wild-type strains and concluded that the vaccine may not be completely safe because the attenuated strain could cause AIDS in some vaccinated individuals so there has to be a trade-off between efficacy and safety. Odeny 2003 [28], incorporated the "all or nothing" type of HIV vaccine in Kenya that covers a fraction of the vaccinated completely. Truphosa 2005, [31] modeled HIV AIDS in Kenya incorporating the "leaky type" of vaccine that protects everyone vaccinated partially. Kgosimore and Lungu 2006 [23], modeled the spread of HIV/AIDS in the application of treatment and vaccination with different levels of vaccine efficacy.

Chapter 3

Model Formulation

3.1 Basic Concepts

This chapter introduces the basic mathematical concepts that are fundamental to the understanding of the entire thesis. Our main source for this section was the book by Kgosimore and Lungu [23], although many other sources were used.

3.1.1 Epidemiology Models

Epidemic models are used to describe rapid outbreaks that occur in less than one year, while endemic models are used for studying diseases over longer periods during which there is a renewal of susceptibles by births or recovery from temporary immunity. The two classic (SIR) models provides an intuitive basis for understanding more complex epidemiology modeling results.

3.1.2 Formulating Epidemiology Models

If $S(t)$ is the number of susceptibles at time t, $I(t)$ is the number of infectives at time t, $R(t)$ is the number of the recovered at time t and N(t) is the total population size, $s(t) = \frac{S(t)}{N(t)}$ and $i(t) = \frac{I(t)}{N(t)}$, are the susceptible and infectious fractions respectively. If β, is the average number of adequate contacts (i.e contacts sufficient for transmission) of a person per unit time, then $\frac{\beta I(t)}{N(t)} = \beta i(t)$ is the average number of contacts with infectives per unit time due to the $S(t) = N(t)s(t)$ susceptibles. This form of horizontal incidence is called the standard incidence.

The Classic Epidemic Model

The classic epidemic model is the SIR model given by the initial value problem

$$\frac{dS}{dt} = \frac{-\beta IS}{N} \tag{3.1.1}$$

$$\frac{dI}{dt} = \frac{\beta IS}{N} - \gamma I \tag{3.1.2}$$

$$\frac{dR}{dt} = \gamma I \tag{3.1.3}$$

Where: $S(0) > 0,\ I(0) > 0,\ R(0) > 0$

S(t), I(t), and R(t) are the numbers in these classes so that $S(t) + I(t) + R(t) = N$. This model uses the standard incidence and has recovery at rate γI, corresponding to an exponential waiting time $e^{-\gamma t}$. Since the time period is short, this model has no vital dynamics births and deaths. Since R does not appear in the first two equations, the model can be reduced

to two equations. Dividing the equations above by constant population size N yields.

$$\frac{ds}{dt} = -\beta is \tag{3.1.4}$$

$$\frac{di}{dt} = \beta i(s - \frac{\gamma}{\beta}) \tag{3.1.5}$$

The Classic Endemic Model

The endemic model is the SIR model with vital dynamics (births and deaths) given by:

$$\frac{dS}{dt} = \mu N - \mu S - \frac{\beta IS}{N} \tag{3.1.6}$$

$$\frac{dI}{dt} = \frac{\beta IS}{N} - \gamma I - \mu I \tag{3.1.7}$$

$$\frac{dR}{dt} = \gamma I - \mu R \tag{3.1.8}$$

where: $S(0) > 0,\ I(0) > 0,\ R(0) > 0$

with S(t)+I(t)+R(t)= N. This SIR model is almost the same as the SIR epidemic model above except that it has an inflow of newborns into the susceptible class at rate μN and deaths at the rates μS, μI and μR. The deaths balance the births so that the population size N remains constant.

3.2 The Proposed Model

The population is divided into two age groups. Group I comprises of the sexually Immature children aged (0 - a) years and group II comprises of sexually mature and active adults aged (a) years and beyond. It is

group II that is responsible for the spread of the epidemic through sexual activity and for the spread in children through infected mothers (Vertical Transmission).

3.2.1 Assumptions

1. The transmission of HIV from an infective to a susceptible is through heterosexual mode and vertical transmission.
2. There is random mixing of individuals within the population.
3. AIDS cases which have full blown symptoms are easily noticeable and are not sexually interacted with and as such, they don't transmit the virus and do not give birth to new borns.
4. Individuals in group I comprise of sexually Immature children aged (0 - a) years and therefore do not transmit the disease.
5. The removed class are sexually interacted with but are not infectious and are immuned.
6. Treatment is done in the adult group only.

3.2.2 Parameters and Notations Used in the Model

Notations for the variables used in the model

- $W(t)$ - denotes the number of susceptible children at time t.
- $H(t)$ - The number of infected children at time t.

- $U(t)$ - The number of AIDS Cases at time t in group I.
- $S(t)$ - The number of susceptible adults at time t.
- $V(t)$ - The number of vaccinated adults at time t.
- $R(t)$ - The number of removed adults covered by the vaccine at time t.
- $X(t)$ - The number of infective adults at time t.
- $Z(t)$ - The number of infected adults who receive treatment at time t.
- $A(t)$ - The number of full blown AIDS Cases in group II at time t.
- $P(t)$ - The total population size at time t.

The Parameters used in the model and how they are obtained

- m - The rate at which the HIV infected children progress to AIDS in group I obtained from the average incubation period of HIV in children
- d - The disease related death rate obtained from the number of years it takes one who has shown full blown AIDS symptoms to die of the disease
- μ - Natural death rate referring to the number of deaths in a year per 1000 people, according to the Kenya demographics profile 2010 [30]

- α - The proportion of susceptible adults assumed to be vaccinated taken arbitrary.
- δ - vaccine efficacy, assuming the "All or Nothing type of vaccine". Obtained by getting the product of the vaccine efficacy (assuming the leaky vaccine) and the proportion of the susceptibles vaccinated. It represents the proportion of the vaccinated completely covered by the vaccine.
- ϵ - Proportion of the infectives receiving treatment.
- η - The rate at which the infectives who do not receive treatment progress to HIV/AIDS calculated from the average incubation period of HIV in adult infectives
- λ - Rate at which those treated progress to AIDS. (both the normal and vaccinated infectives) calculated from the average incubation period of treated adult infectives
- b - The per capita birth rate calculated as a ratio of the average live births per year to the general population size.
- g - Natural child mortality rate calculated as a ratio of the average number of children below 15 years who die per year to the total population below 15 years.
- v - Proportion of babies born with HIV from HIV infected mothers obtained from [27].
- b_1 -Per capita birth rate of the adults calculated as a ratio of the average live births per year among the adults to the general population size of the adults excluding AIDS cases

- a - Years at which an individual becomes sexually mature.
- β_1 - is the per partnership transmission probability of a normal infective who is not treated. Obtained from the prevalence of the disease in a given area and their sexual behaviour patterns [3].
- β_2 - is the per partnership transmission probability of an infective who is treated and counseled [3].
- c_1 - is the average number of new sexual partners acquired per unit time by those infected but not yet counseled and treated.
- c_2 - is the average number of new sexual partners acquired per unit time by those treated and counseled.
- θ - The proportion of the vaccinated protected by the vaccine assuming the "leaky type of vaccine" [4].

3.2.3 Age - Structures

Group I consists of children of age (0 - a) years which has three compartments namely $W(t)$ - the number of susceptible children at time t, $H(t)$ - the number of infected children at time t and $U(t)$ - the number of AIDS cases in children at time t. The differential equations for group I takes

the following form:-

$$\begin{aligned}
\frac{dW(t)}{dt} &= b_1N(t) - gW(t) - b_1v(X(t) + Z(t)) \\
&\quad -e^{(-ga)}b_1N(t-a) \qquad (3.2.1)\\
\frac{dH(t)}{dt} &= b_1v(X(t) + Z(t)) - (g+m)H(t) \qquad (3.2.2)\\
\frac{dU(t)}{dt} &= mH(t) - (d+g)U(t) \qquad (3.2.3)
\end{aligned}$$

Group II consists of adults aged (a) years and beyond. It has seven compartments comprising of $S(t)$ - the number of susceptible adults, $V(t)$ - the number of vaccinated adults, $R(t)$ - the number of removed adults, $X(t)$ - the number of infective adults, $Z(t)$ - the number of infected adults who receive treatment, $A(t)$ - the number of full blown AIDS cases in group II. The differential equations takes the following form:-

$$\begin{aligned}
\frac{dS(t)}{dt} &= e^{(-ga)}b_1N(t-a) - \left(\frac{\beta_1c_1S(t)(X(t))}{N(t)} + \frac{\beta_2c_2S(t)(Z(t))}{N(t)}\right) \\
&\quad -(\mu+\alpha)S(t) \qquad (3.2.4)\\
\frac{dV(t)}{dt} &= (\alpha-\delta)S(t) - \left((1-\theta)\left(\frac{\beta_1c_1V(t)(X(t))}{N(t)} + \frac{\beta_2c_2V(t)(Z(t))}{N(t)}\right)\right) \\
&\quad -\mu V(t) \qquad (3.2.5)\\
\frac{dR(t)}{dt} &= \delta S(t) - \mu R(t) \qquad (3.2.6)\\
\frac{dX(t)}{dt} &= \left(\frac{\beta_1c_1S(t)(X(t))}{N(t)} + \frac{\beta_2c_2S(t)(Z(t))}{N(t)}\right) \\
&\quad + \left((1-\theta)\left(\frac{\beta_1c_1V(t)(X(t))}{N(t)} + \frac{\beta_2c_2V(t)(Z(t))}{N(t)}\right)\right) \qquad (3.2.7)\\
&\quad -(\mu+\epsilon+\eta)X(t) \\
\frac{dZ(t)}{dt} &= \epsilon X(t) - (\mu+\lambda)Z(t) \qquad (3.2.8)\\
\frac{dA(t)}{dt} &= \eta X(t) + \lambda Z(t) - (\mu+d)A(t) \qquad (3.2.9)
\end{aligned}$$

With:

$P(t) = N(t) + W(t) + H(t) + U(t) + A(t), \quad N(t) = S(t) + V(t) + R(t) + X(t) + Z(t)$

and $b_1 = b\frac{P(t)}{N(t)}$

The population growth model is given by the following differential equation.

$$\frac{dP(t)}{dt} = (b_1 - \mu)N(t) - g(W(t) + U(t) + H(t)) - \mu(A(t)) - d(U(t) + A(t))$$

With $W(0) = f_1 P(0),\ H(0) = 0,\ U(0) = 0$

P(0) being the the total population at the start of the epidemic and f_1, is the fraction of children (a) years and below at the start of the epidemic where $0 < f_1 < 1$.

$S(0) = (1 - \phi_0) f_2 P(0),\ X(0) = \phi_0 f_2 P(0),\ A(0) = 0,$

with $0 < f_2 < 1$, being the fraction of the population that were adults at the start of the epidermic. ϕ_0, is the fraction of the adult population that were HIV infective at the start of the epidemic with $f_1 + f_2 = 1$.

In chapter four we analyze the stability of the disease free equilibrium points of equations 3.2.4, 3.2.5, 3.2.6, 3.2.7, 3.2.8 and calculate the basic reproduction number R_0.

Chapter 4

Analysis of the Disease Free Equilibrium (D.F.E)

4.1 Introduction

We analyze the stability of the D.F.E in group II (adults) since it is this group that is sexually active and responsible for the spread. We also assume that the AIDS cases $A(t)$ in the population can easily be identified from the full blown symptoms and are not associated with sexually and as such are not involved in the spread of the diseases though their projection will be investigated numerically to identify the impact they have on the population.

4.2 Proportional Variables

We use the proportions of the populations to enable us study the steady states. This is based on the assumption that it is more likely for the

population proportions to attain the steady states than an individual population class which perhaps may only happen when the carrying capacity is reached.

We set the proportions as follows: $s(t) = \frac{S[t]}{N(t)}$, $x(t) = \frac{X(t)}{N(t)}$, $z(t) = \frac{Z(t)}{N(t)}$, $r(t) = \frac{R(t)}{N(t)}$, $\nu(t) = \frac{V(t)}{N(t)}$.

Note: $s(t) + \nu(t) + r(t) + x(t) + z(t) = 1$

We set $e^{-ga} = \rho$

Given $\frac{S(t)}{N(t)} = s(t) \Longrightarrow S(t) = N(t)s(t)$,

Hence $\frac{dS(t)}{dt} = N(t)\frac{ds(t)}{dt} + s(t)\frac{dN(t)}{dt}$

$= \rho b_1 N(t-a) - (\beta_1 c_1 s(t) X(t] + \beta_2 c_2 s(t) Z(t)) - (\mu + \alpha) S(t)$

$\frac{dN(t)}{dt} = \rho b_1 N(t-a) - \mu N(t) - \eta X(t) - \lambda Z[t]$

.

The equations 3.2.4, 3.2.5, 3.2.6, 3.2.7, 3.2.8, with $e^{-ga} = \rho$ become

$$\frac{ds(t)}{dt} = \frac{\rho b_1 N(t-a)}{N(t)}(1-s(t)) - (\beta_1 c_1 s(t)x(t) + \beta_2 c_2 s(t)z(t)) \quad (4.2.1)$$
$$-(\mu+\alpha)s(t) + \mu s(t) + \eta s(t)x(t) + \lambda s(t)z(t)$$

$$\frac{d\nu(t)}{dt} = (\alpha-\delta)s(t) - ((1-\theta)(\beta_1 c_1 \nu(t)x(t) + \beta_2 c_2 \nu(t)z(t))) \quad (4.2.2)$$
$$-\mu\nu(t) - \frac{\rho b_1 N(t-a)}{N(t)}\nu(t) + \mu\nu(t) + \eta x(t)\nu[t] + \lambda z(t)\nu(t)$$

$$\frac{dr(t)}{dt} = \delta s(t) - \mu r(t) - \frac{\rho b_1 N(t-a)}{N(t)}r(t) + \mu r(t) \quad (4.2.3)$$
$$+\eta x(t)r(t) + \lambda r(t)z(t)$$

$$\frac{dx(t)}{dt} = (\beta_1 c_1 x(t)s(t)) + \beta_2 c_2 s(t)z(t))$$
$$+((1-\theta)(\beta_1 c_1 \nu(t)x(t) + \beta_2 c_2 \nu(t)z(t))) \quad (4.2.4)$$
$$-(\mu+\epsilon+\eta)x(t) - \frac{\rho b_1 N(t-a)}{N(t)}x(t) + \mu x(t) + \eta(x(t))^2 + \lambda x(t)z(t)$$

$$\frac{dz(t)}{dt} = \epsilon x(t) - (\mu+\lambda)z(t) - \frac{\rho b_1 N(t-a)}{N(t)}z(t) \quad (4.2.5)$$
$$+\mu z(t) + \eta x(t)z(t) + \lambda(z(t))^2$$

In a Disease free system, we have

$$N(t) = N_0 e^{(rt)} \Longrightarrow N(t) = N(t-a)e^{(ra)}$$

with $r = b - \mu$

We set $w_1 N(t) = N(t-a)$,

where w_1, denotes the weight given to the population size at time (a).

Again we let $s^*(t), \nu^*(t), r^*(t), x^*(t), z^*(t)$, be the equilibrium points. The

equilibrium equations 4.2.1, 4.2.2, 4.2.3, 4.2.4, 4.2.5, become

$$\begin{aligned}
0 &= \rho b_1 w_1(1-s^*(t)) - (\beta_1 c_1 s^*(t)x^*(t) + \beta_2 c_2 s^*(t)z^*(t)) && (4.2.6)\\
&\quad -(\mu+\alpha)s^*(t) + \mu s^*(t) + \eta s^*(t)x^*(t) + \lambda s^*(t)z^*(t)\\
0 &= (\alpha-\delta)s^*(t) - ((1-\theta)(\beta_1 c_1 \nu^*(t)x^*[t] + \beta_2 c_2 \nu^*(t)z^*(t))) && (4.2.7)\\
&\quad -\mu\nu^*(t) - \rho b_1 w_1 \nu^*(t) + \mu\nu^*(t) + \eta x^*(t)\nu^*(t) + \lambda z^*(t)\nu^*(t)\\
0 &= \delta s^*(t) - \mu r^*(t) - \rho b_1 w_1 r^*(t) + \mu r^*(t) + \eta x^*(t) r^*(t) && (4.2.8)\\
&\quad +\lambda r^*(t)z^*(t)\\
0 &= (\beta_1 c_1 x^*(t)s^*(t)) + \beta_2 c_2 s^*(t)z^*(t))\\
&\quad +((1-\theta)(\beta_1 c_1 \nu^*(t)x^*(t) + \beta_2 c_2 \nu^*(t)z^*(t))) && (4.2.9)\\
&\quad -(\mu+\epsilon+\eta)x^*(t) - \rho b_1 w_1 x^*(t) + \mu x^*(t) + \eta(x^*(t))^2 + \lambda x^*(t)z^*(t)\\
0 &= \epsilon x^*(t) - (\mu+\lambda)z^*(t) - \rho b_1 w_1 z^*(t) && (4.2.10)\\
&\quad +\mu z^*(t) + \eta x^*(t)z^*(t) + \lambda(z^*(t))^2
\end{aligned}$$

4.3 Testing the Stability of the Steady state solutions of the D.F.E

At the disease free equilibrium (D.F.E), we have
$[s^*(t), \nu^*(t), r^*(t), x^*(t), z^*(t)] = [1, 0, 0, 0, 0]$,
We find the eigenvalues of the jacobian of the equations 4.2.6, 4.2.7, 4.2.8, 4.2.9, 4.2.10 by obtaining their partial derivatives with respect to each variable. The corresponding matrix is therefore given by J.

$$\text{Where } J = \begin{pmatrix} -\rho b_1 w_1 - \alpha & 0 & 0 & \eta - \beta_1 c_1 & \lambda - \beta_2 c_2 \\ (\alpha - \delta) & -\rho b_1 w_1 & 0 & 0 & 0 \\ \delta & 0 & -\rho b_1 w_1 & 0 & 0 \\ 0 & 0 & 0 & \beta_1 c_1 - \rho b_1 w_1 - (\epsilon + \eta) & \beta_2 c_2 \\ 0 & 0 & 0 & \epsilon & -\lambda - \rho b_1 w_1 \end{pmatrix}$$

Using the Wolfram Research Mathematica Software the eigenvalues are given as:

$$\begin{aligned} \lambda_1 &= -\rho b_1 w_1 \\ \lambda_2 &= -\rho b_1 w_1 \\ \lambda_3 &= -\rho b_1 w_1 - \alpha \\ \lambda_4 &= \frac{1}{2}(-2\rho b_1 w_1 + c_1\beta_1 - \epsilon - \eta - \lambda) - \frac{1}{2}\Omega \\ \lambda_5 &= \frac{1}{2}(-2\rho b_1 w_1 + c_1\beta_1 - \epsilon - \eta - \lambda) + \frac{1}{2}\Omega \end{aligned}$$

where $\Omega = \sqrt{c_1^2\beta_1^2 - 2c_1\beta_1\epsilon + \epsilon^2 - 2c_1\beta_1\eta + 2\epsilon\eta + \eta^2 + 2c_1\beta_1\lambda - 2\epsilon\lambda - 2\eta\lambda + \lambda^2 + 4\epsilon\beta_2 c_2}$

The disease free equilibrium (D.F.E) is locally asymptotically stable when any of the following conditions are satisfied:

1. $c_1\beta_1 + \frac{1}{2}\Omega < (\epsilon + \eta + \lambda + 2\rho b_1 w_1)$.
2. $c_1\beta_1 < (\epsilon + \eta + \lambda + 2\rho b_1 w_1)$ *and* Ω *is not a real number.*

This means that if an infective is introduced in a host population of susceptibles, the infective will not invade the host population, i.e the disease will die out.

4.4 Threshold Quantities

The Threshold for many epidemiology models is the basic reproduction number R_0 defined as the average number of secondary infections produced when one infected individual is introduced into a host population where everyone is susceptible [24].

For many deterministic epidemiology models, an infection can get started in a fully susceptible population if and only if $R_0 > 1$. Thus the basic Reproduction number R_0, is often considered as the threshold quantity that determines when an infection can invade and persist in a new host population. It is also called the Basic Reproduction Ratio or Basic Reproduction Rate. The contact number is defined as the average number of adequate contacts of atypical infective during the infectious period. Adequate contacts is one that is sufficient for transmission if the individual contacted by the susceptible is an infective.

The reproduction number could be obtained by inspection if we have only one infective class. If the number of infective classes are two or more, then the technique due to Diekmann [12] 1990, called the next generation matrix is more appropriate. The technique has been studied by a number of researchers among them Kgosimore and Lungu [23].

4.4.1 The Next Generation Matrix

Define X_s, be the set of all disease free states, that is

$X_s = \{\dot{x}_i \geq 0,\ i = 1, 2, ..m\}$

In order to compute R_0, it is important to distinguish new infections from all other changes in the population.

- Let $F_i(x)$, be the rate of appearance of new infections in compartments i.
- V_i^+, be the rate of transfer of individuals into compartment i by all other means.
- V_i^-, be the rate of transfer of individuals out of compartments i.

It is assumed that each function $F_i(x)$, V_i^+, V_i^-, is continuously differentiable at least twice with respect to each variable.

The transmission Model consists of the non-negative initial conditions together with the following systems of equations.

$$\dot{x}_i = f_i(x) \tag{4.4.1}$$

Where $\dot{x}_i = f_i(x) = F_i(x) - V_i(x), i = 1, 2..n$, and $V_i = V_i^- - V_i^+$.

If x_0, is a (D.F.E), of 4.4.1 then the derivatives $DF(x_0)$, $DV(x_0)$, where F and V are the $m * m$, matrices defined by.

$$F = \frac{\partial F x_0}{\partial x_i} \text{ and } V = \frac{\partial V x_0}{\partial x_i}.$$

With $1 \leq i \leq m$. F is non-negative and V is non-singular m - matrix. m is

the number of the infective compartments. Following Diekmann (1990), [21], we call FV^{-1}, the next generation matrix for the model and R_0, is set to be the spectral radius of FV^{-1}. i.e $R_0 = \rho(FV^{-1})$.

4.4.2 Use of Proportional Populations

Applying this technique to our model we have two infected compartments $m = 2$, in group II given below.

$$\begin{aligned}
\frac{dx(t)}{dt} &= (\beta_1 c_1 x(t)s(t) + \beta_2 c_2 s(t)z(t)) + ((1-\theta)(\beta_1 c_1 \nu(t)x(t) + \beta_2 c_2 \nu(t)z(t))) \\
&\quad -(\epsilon+\eta)x(t) - \rho b_1 w_1 x(t) + \eta(x(t))^2 + \lambda x(t)z(t) \qquad (4.4.2) \\
\frac{dz(t)}{dt} &= \epsilon x(t) - (\mu+\lambda)z(t) - \rho b_1 w_1 z(t) + \\
&\quad \mu z(t) + \eta x(t)z(t) + \lambda(z(t))^2 \qquad (4.4.3)
\end{aligned}$$

We obtain matrix F given by:

$$F = \begin{pmatrix} (\beta_1 c_1 x(t)s(t) + \beta_2 c_2 s(t)z(t)) + ((1-\theta)(\beta_1 c_1 \nu(t)x(t) + \beta_2 c_2 \nu(t)z(t))) \\ 0 \end{pmatrix}$$

and matrix V given by:

$$V = V_i^- - V_i^+ = \begin{pmatrix} (\epsilon+\eta)x(t) + \rho b_1 w_1 x(t) - \eta(x(t))^2 - \lambda x(t)z(t) \\ (\lambda)z(t) + \rho b_1 w_1 z(t) - \epsilon x(t) - \eta x(t)z(t) - \lambda(z(t))^2 \end{pmatrix}$$

The (D.F.E), point of the system has coordinates

$[s^*(t), \nu^*(t), r^*(t), x^*(t), z^*(t)] = [1, 0, 0, 0, 0]$

The derivatives of matrices F and V at $[1, 0, 0, 0]$, are given by:

$$F_1 = \begin{pmatrix} \beta_1 c_1 & \beta_2 c_2 \\ 0 & 0 \end{pmatrix} \tag{4.4.4}$$

$$V_1 = \begin{pmatrix} (\epsilon + \eta + \rho b_1 w_1) & 0 \\ -\epsilon & (\lambda + \rho b_1 w_1) \end{pmatrix} \tag{4.4.5}$$

Using the Wolfram research mathematica software

$$V_1^{-1} = \begin{pmatrix} (\frac{1}{\rho b_1 w_1 + \epsilon + \eta}) & 0 \\ \frac{\epsilon}{(\rho b_1 w_1 + \epsilon\eta)(\rho b_1 w_1 + \lambda)} & \frac{1}{(\rho b_1 w_1 + \lambda)} \end{pmatrix} \tag{4.4.6}$$

$$F_1 * V_1^{-1} = \begin{pmatrix} (\frac{c_1\beta_1}{\rho b_1 w_1 + \epsilon + \eta} + \frac{\epsilon + \beta_1 c_1}{(\rho b_1 w_1 + \epsilon + \eta)(\rho b_1 w_1 + \lambda)}) & (\frac{\beta_1 c_1)}{(\rho b_1 w_1 + \lambda)} \\ 0 & 0 \end{pmatrix} \tag{4.4.7}$$

The eigenvalues of $F_1 * V_1^{-1}$ are given as

$\lambda_1 = 0$

$$\lambda_2 = \left(\frac{\beta_1 c_1}{(\rho b_1 w_1 + \epsilon + \eta)} + \frac{\epsilon \beta_2 c_2}{(\rho b_1 w_1 + \epsilon + \eta)(\rho b_1 w_1 + \lambda)}\right)$$

The spectral radius of $(F_1 * V_1^{-1}) = R_0 = \left(\frac{\beta_1 c_1}{(\rho b_1 w_1 + \epsilon + \eta)} + \frac{\epsilon \beta_2 c_2}{(\rho b_1 w_1 + \epsilon + \eta)(\rho b_1 w_1 + \lambda)}\right)$

According to [2], [1], [7], [23], [8] and [12] if $(F_1 * V_1^{-1}) = R_0 > 1$, the infection will invade and persist in a host population of susceptibles. However, if $R_0 < 1$, the infection will die out.

4.4.3 Use of Individual Populations

From the work of Kgosimore and Lungu [23], we have two infected compartments $m = 2$, in group II given below.

$$\begin{aligned}
\frac{dX(t)}{dt} &= \left(\frac{\beta_1 c_1 S(t)(X(t))}{N(t)} + \frac{\beta_2 c_2 S(t)(Z(t))}{N(t)}\right) \\
&\quad + \left((1-\theta)\left(\frac{\beta_1 c_1 V(t)(X(t))}{N(t)} + \frac{\beta_2 c_2 V(t)(Z(t))}{N(t)}\right)\right) \\
&\quad -(\mu + \epsilon + \eta)X(t) \qquad (4.4.8) \\
\frac{dZ(t)}{dt} &= \epsilon X(t) - (\mu + \lambda)Z(t) \qquad (4.4.9)
\end{aligned}$$

We obtain matrix F given by:

$$F = \begin{pmatrix} \left(\frac{\beta_1 c_1 S(t)(X(t))}{N(t)} + \frac{\beta_2 c_2 S(t)(Z(t))}{N(t)}\right) + \left((1-\theta)\left(\frac{\beta_1 c_1 V(t)(X(t))}{N(t)} + \frac{\beta_2 c_2 V(t)(Z(t))}{N(t)}\right)\right) \\ 0 \end{pmatrix}$$

and

$$V = V_i^- - V_i^+ = \begin{pmatrix} (\epsilon+\eta)X(t) + \mu X(t) \\ (\lambda+\mu)Z(t) - \epsilon X(t) \end{pmatrix}$$

The (D.F.E), point of the system has coordinates

$[S^*(t), V^*(t), R^*(t), X^*(t), Z^*(t)] = [S^*(t), 0, 0, 0, 0]$

The derivatives of matrices F and V at $(S^*(t), 0, 0, 0)$, are given by:

$$F_1 = \begin{pmatrix} \beta_1 c_1 \frac{S^*(t)}{N^*(t)} & \beta_2 c_2 \frac{S^*(t)}{N^*(t)} \\ 0 & 0 \end{pmatrix} \tag{4.4.10}$$

In a disease free system, $S^*(t) = N^*(t)$, thus

$$F_1 = \begin{pmatrix} \beta_1 c_1 & \beta_2 c_2 \\ 0 & 0 \end{pmatrix} \tag{4.4.11}$$

$$V_1 = \begin{pmatrix} (\epsilon+\eta+\mu) & 0 \\ -\epsilon & (\lambda+\mu) \end{pmatrix} \tag{4.4.12}$$

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$$V_1^{-1} = \begin{pmatrix} (\frac{1}{\mu+\epsilon+\eta}) & 0 \\ \frac{\epsilon}{(\mu+\epsilon+\eta)(\mu+\lambda)} & \frac{1}{(\mu+\lambda)} \end{pmatrix} \tag{4.4.13}$$

$$F_1 * V_1^{-1} = \begin{pmatrix} (\frac{c_1\beta_1}{\mu+\epsilon+\eta} + \frac{\epsilon\beta_1 c_1}{(\mu+\epsilon+\eta)(\mu+\lambda)}) & (\frac{\beta_1 c_1}{\mu+\lambda}) \\ 0 & 0 \end{pmatrix} \tag{4.4.14}$$

The eigenvalues of $F_1 * V_1^{-1}$ are given as

$\lambda_1 = 0$

$\lambda_2 = (\frac{\beta_1 c_1}{(\mu+\epsilon+\eta)} + \frac{\epsilon\beta_2 c_2}{(\rho b_1 w_1+\epsilon+\eta)(\mu+\lambda)})$

The spectral radius of $(F_1 * V_1^{-1}) = R_0 = (\frac{\beta_1 c_1}{(\mu+\epsilon+\eta)} + \frac{\epsilon\beta_2 c_2}{(\mu+\epsilon+\eta)(\mu+\lambda)})$

Hence if $R_0 = (\frac{\beta_1 c_1}{(\mu+\epsilon+\eta)} + \frac{\epsilon\beta_2 c_2}{(\mu+\epsilon+\eta)(\mu+\lambda)}) > 1$
then the infection will invade and persist in a host population of susceptibles. The disease remains endemic in the population. However, if $R_0 < 1$, then the infection will die out.

Chapter 5

Numerical simulations of the Model

5.1 Introduction

In order to illustrate some of the analytical results in this thesis, numerous simulations of the full model were carried out using a set of parameter values given in section 5.2. During the early stages of the disease, the force of infection was estimated to be $\beta_1 c_1 = 0.6667$ deduced from the disease doubling time of 1.5 years [27], which gives the value of $R_0 > 1$ according to our analytical results from section 4.4.1 and 4.4.2. Numerical simulations confirms the same and shows that the disease remains endemic in the population (section 5.3.1). Treatment and counseling that reduces infectiousness when $R_0 < 1$ eliminates the disease as indicated in section 5.4. These simulations also shows that a trade-off exists between vaccination and treatment (chapter 6).

5.2 Initial parameter values from literature review

We present numerical simulations of the model using the following default parameter values unless specified otherwise:

- $b = 0.03664$ - The birth rate referring to the number of live births in a year per 1000 people. The value used is obtained from the Kenya demographics profile 2010 [30]. The value of 0.04 used in section 5.2 is adjusted to ensure that the ratio of the adults and those under 15 years is at steady state.
- $\mu = 0.00973$ - Natural death rate referring to the number of deaths in a year per 1000 people. Obtained from the Kenya demographics profile 2010 [30].
- $g = 0.015$ - Natural under 15 child mortality rate estimated to be 1.5 times higher than the natural death rate. Obtained from[3]. The estimation is done because the statistics for the under 15 mortality rate is not available in Kenya.
- $v = 0.3$ - Proportion of babies born with HIV from HIV infected mothers obtained from [27].
- $m = 0.2$ - The rate at which the HIV infected children progress to AIDS in group I. Calculated from the average incubation period of HIV in children. Obtained from [3].
- $d = 0.4$ - The disease related death rate. Calculated from the number of years it takes one who has shown full blown AIDS symptoms

to die of the disease. Obtained from [27].

- $\beta_1 = 0.019$ - The probability of getting an infection from a new sexual partner. Obtained from [3].

- $c_1 = 9.9$ - The rate at which an individual acquires new sexual partners per year. Calculated from the rate of new infections of 55000 per year obtained from the Kenya National Aids Control Council Report of 2007 and the transmission probability of 0.019 above [3].

- $\beta_1 c_1 = 0.1881$ - The force of infection of the infectives who are not treated and counseled. The value used is obtained from [3] and the Kenya National Aids Control Council Report of 2007. A force of infection of 0.6667 used in section 5.4.1 is calculated from the disease doubling time of 1.5 years experienced during the early stages of the disease.

- $\delta = 0.12$ - Vaccine efficacy, assuming the "All or Nothing type of vaccine". Obtained by getting the product of the vaccine efficacy assuming the leaky vaccine and the proportion of the susceptible vaccinated [4].

- $\theta = 0.3$ - The proportion of the vaccinated protected by the vaccine assuming the "leaky type of vaccine". Obtained from [4]

- $\eta = 0.125$ - The rate at which the normal infectives who do not receive treatment progress to AIDS. Calculated from the average incubation period of HIV in adult infectives. Obtained from [3].

- $\beta_2 c_2 = 0.03762$ - The force of infection for those treated and counseled. It is estimated that treatment reduces infectiousness by 50

percent From AIDS science [9]. The combined effects of treatment and counseling (Condom use) is assumed to reduce infectiousness further.

- $\lambda = 0.08$ - Rate at which those treated progress to AIDS. Calculated from the average incubation period of HIV in treated adult infectives. Obtained from AIDS science [11].
- $\epsilon = 0.44$ - The proportion of the infectives who receive treatment obtained from [16].
- $\alpha = 0.4$ - The proportion of the susceptibles assumed to be vaccinated since no practical vaccine exist as yet.
- $a = 15$ - Age at which one becomes sexually mature. Obtained from [3].
- $k = e^{-(b-\mu)*a}$ - The weight given to the population size (a) years ago, calculated from the values of μ and (a) above.
- $\rho = e^{(-g*a)}$ - Is the proportion of uninfected children who survive the developmental stage of (0 - a) years, calculated from the values of (a) and (g) above.

The initial conditions are obtained from the Kenya demographics profile 2010 [30], the Kenya National Aids Control Council Report 2007 [20] and the Kenyan population census report of 1999 - 2009 [10].

5.3 Population model

To develop a population model when the disease is not incorporated, we set all the disease parameters to zero. We begin by presenting the graph of the population proportions with time to investigate wether our parameter values gives us steady state solutions of the population proportions. We need the steady state solutions as a background to study the effects of HIV/AIDS on the stable states. Using the birth rate of 0.03664, the graph of the population proportions appears as shown in figure 5.3.1

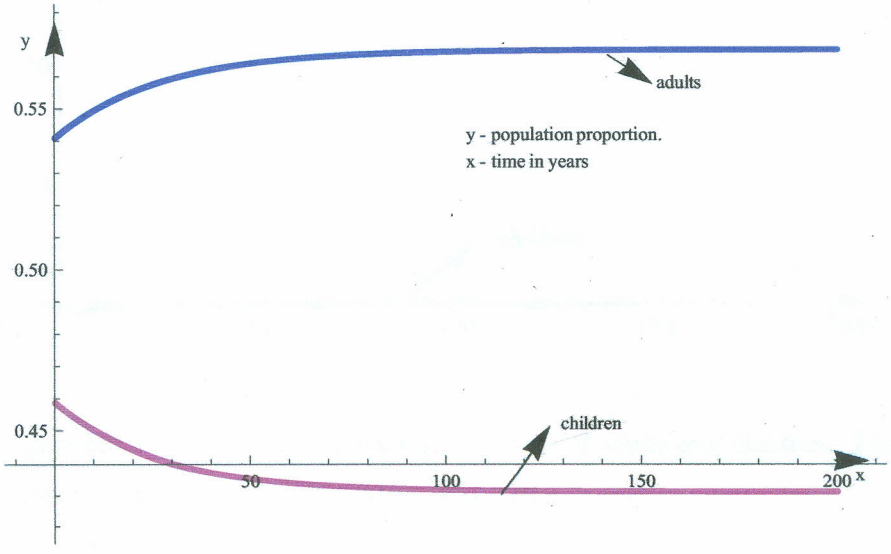

Figure 5.3.1: *Graph of Population proportions of adults and children.*

We observe that the stable solution either requires different initial conditions or different birth and death rates. To obtain a constant population proportion (stable solution) between adults and those under 15 years we adjust the birth rate to 0.04. The new graph is shown in figure 5.3.2.

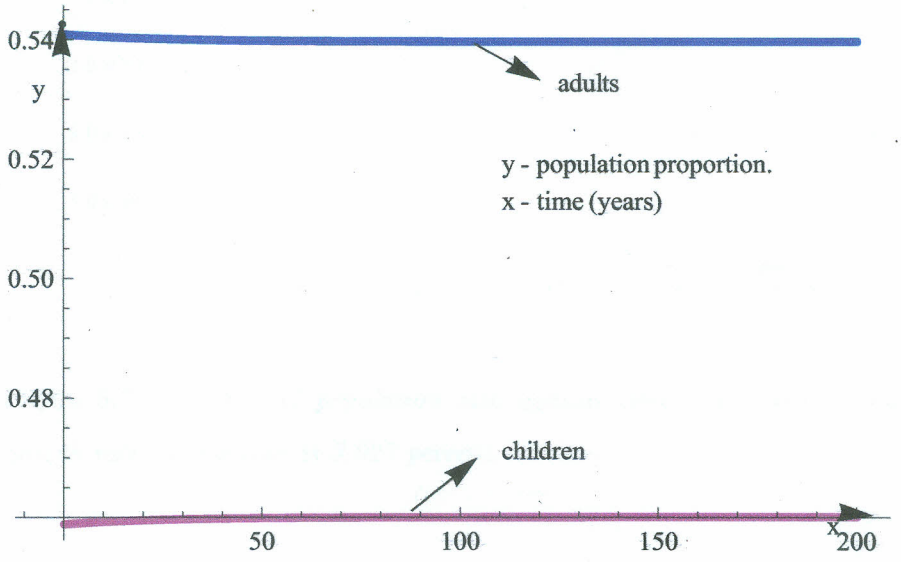

Figure 5.3.2: *Graph of Population proportions of adults and children. The parameter for the birth rate (b) used is 0.04.*

Using the birth rate of 0.04, the population model is shown in figure 5.3.3.

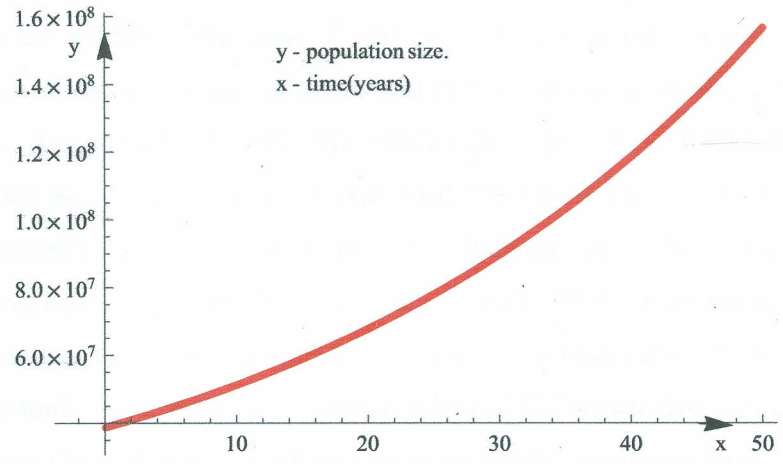

Figure 5.3.3: *Graph of population size against time. The population growth rate in this case is 3.027 percent.*

At the current growth rate of 2.691 percent, [30] the Kenyan population is expected to double in about 26 years assuming that the birth and death rates remains constant. Running our simulations for 50 years with the adjusted birth rate of 0.04 (growth rate of 3.027 percent), we obtain a population size of about 160 million people as shown in figure 5.3.3. According to the HIV/AIDS Policy fact sheet of October 2009 [17] and the Kenya demographics profile 2010 [13] deaths due to HIV/AIDS is estimated to be about 150,000 per year (2003 estimate). Given that the growth rate of 2.691 percent is inclusive of HIV/AIDS in the population; the underlying growth rate of 3.027 percent is a reasonable estimate for the population growth rate exclusive of HIV/AIDS which gives a doubling time of about 23 years. We thus have a consistent population model from which we can start our analysis.

5.4 Disease Incorporated

In this section we factor in the disease alone without any intervention into the model and investigate its effects on the population size and structure.

5.4.1 Early stages of the disease.

During the early stages of HIV/AIDS pandemic, the force of infection was estimated to be 0.6667 which was deduced from the disease doubling time of 1.5 years obtained from [27]. Using this force of infection of 0.6667 and the 2009 Kenyan population size obtained from the Kenyan population

census report of 1999 - 2009, the simulations for the population size in 50 years would appear as in figure 5.4.1.

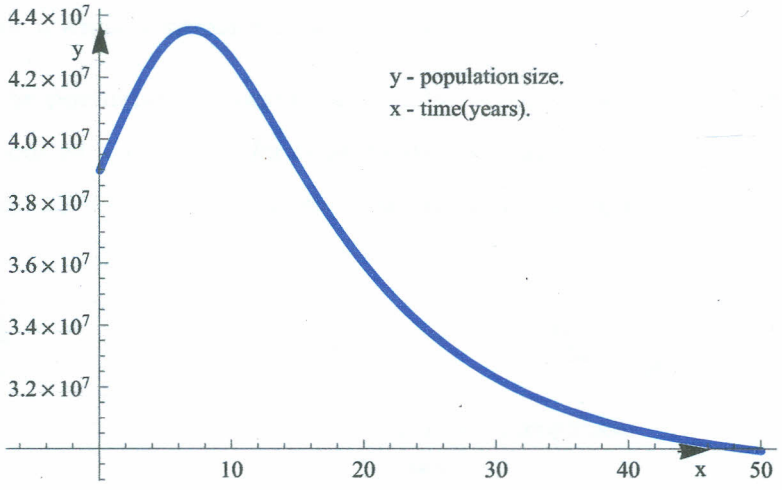

Figure 5.4.1: *Graph of Population size against time with the force of infection* $\beta_1 c_1 = 0.6667$. *In this case* $R_o = 4.94842$

We observe that the population begins to decline after about 12 years. This reflects the scenarios witnessed in the 1990's in Uganda, South Africa and other African countries where the scourge of HIV/AIDS wiped out almost a whole generation in some communities.

The population proportion for adults would reduce from 56 percent to about 24 percent and for those below 15 years would increase from 44 percent to 76 percent in 50 years time as shown in figure 5.4.2.

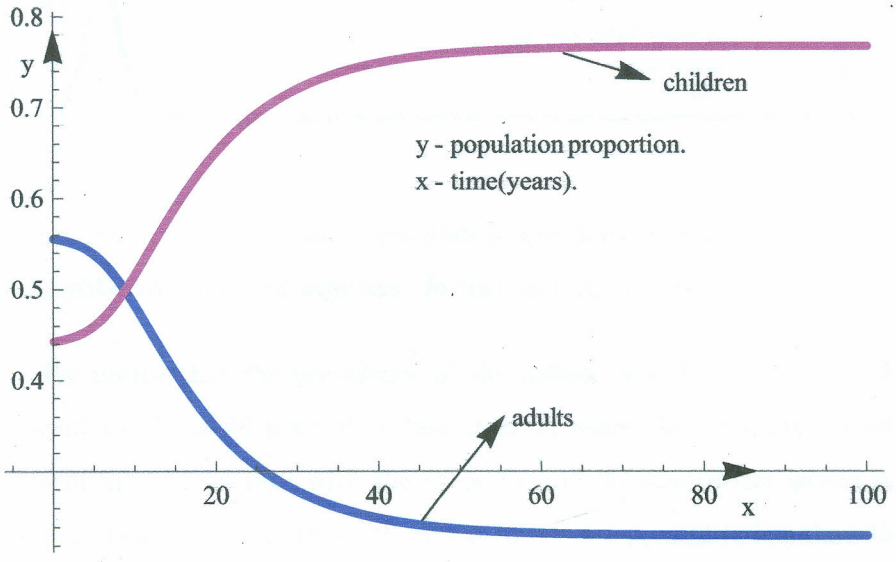

Figure 5.4.2: *Graph of the Population proportions against time.* $R_o = 4.94842$

The graph of the population proportions for the susceptible adults, susceptible under 15, and the infectives would also appear as shown in figure 5.4.3.

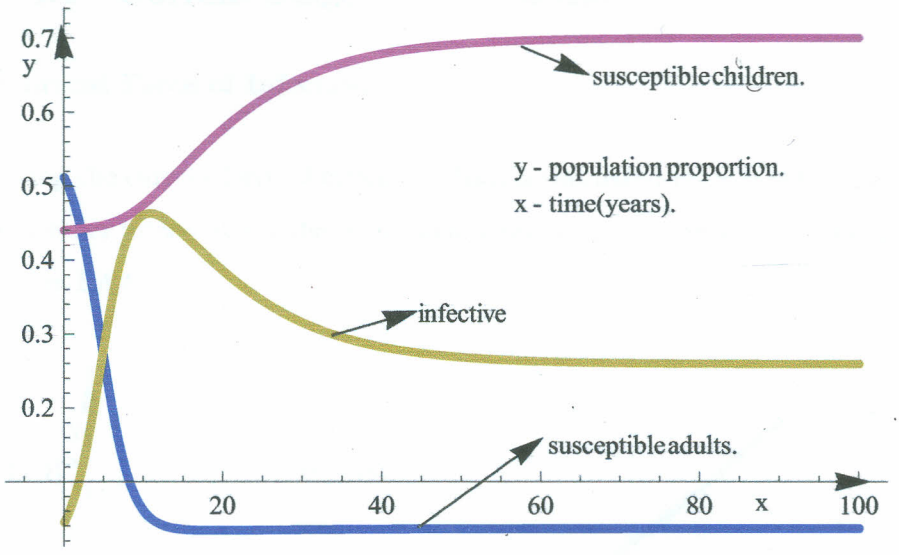

Figure 5.4.3: *Graph of the Population proportions of susceptible adults, susceptible children and infective. In this case* $R_o = 4.94842$.

We notice that the prevalence of the disease would increase from 5 percent to about 46 percent in less than 15 years then stabilize at 28 percent in 50 years time with the proportion of the susceptible adults in the population reducing from 52 percent to below 5 percent in less than 15 years. The steady states of the population proportions are attained in 50 years time. The model suggests that in 10 years time, the proportion of the HIV/AIDS victims in the population will be equal to the proportion of the susceptible under 15 years with the susceptible adults almost wiped out in the population. This reflects the scenarios that transpired in the early stages of the disease in some African countries however, everything changed thereafter.

5.4.2 Current Stage of the Disease.

Current Force of Infection.

Using the current force of infection which is estimated to be 0.1881 without other interventions the population growth curve appears as shown in figure 5.4.4.

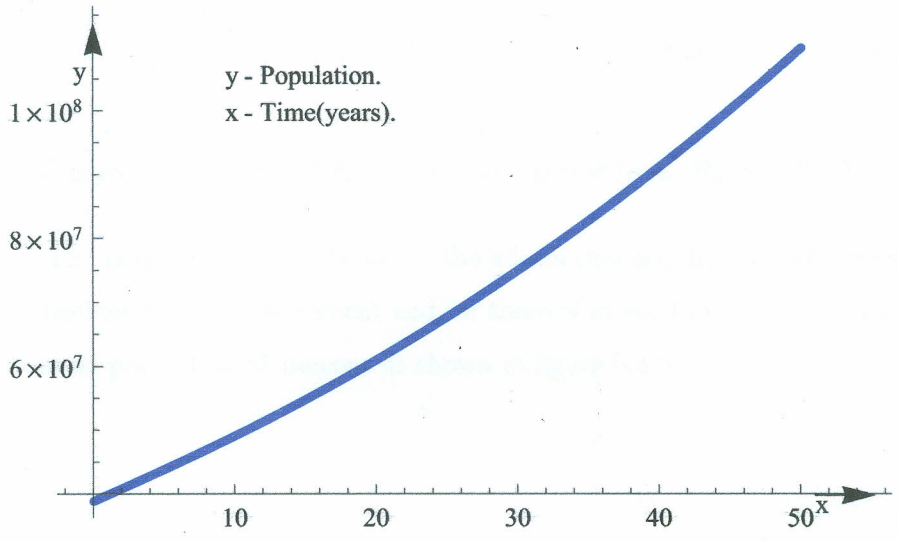

Figure 5.4.4: *Graph of Population size against time with the force of infection* $\beta_1 c_1 = 0.1881$. $R_o = 1.39613$.

If there is no intervention at the current estimated force of infection of 0.1881, the increase in the population size would be about 48 million people less than it could have been in the absence of the disease in 50 years. (figure 5.3.3. and 5.4.4.)

According to this model, the prevalence (The fraction of the popula-

tion infected) of the disease would increase from 5 percent and stabilize at 12 percent as shown in figure 5.4.5.

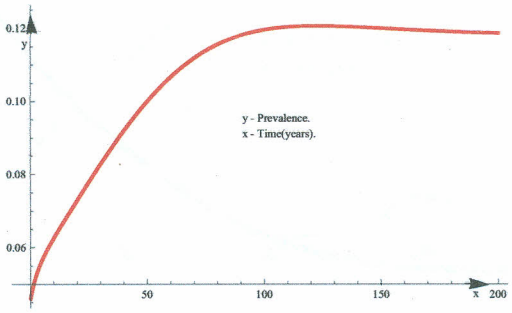

Figure 5.4.5: *Graph of the Prevalence against time.* $R_o = 1.39613$.

The population proportions for the adults changes dramatically from 56 percent to about 43 percent and for those who are 15 years and below from 44 percent to 57 percent as shown in figure 5.4.6.

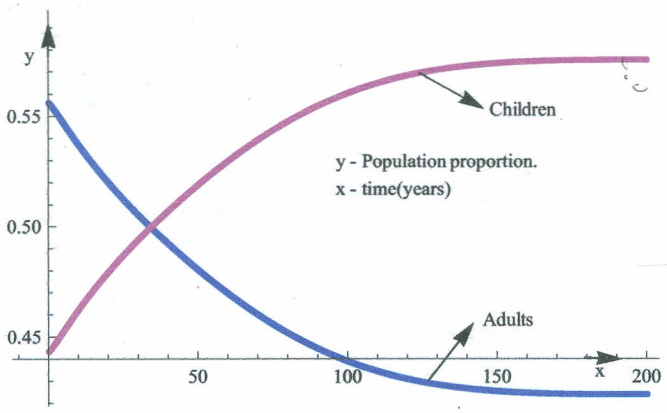

Figure 5.4.6: *Graph of the Population proportions against time.* $R_o = 1.39613$.

This model suggests that even with the current reduced rates of infection the adult population would reduce to much lower levels compared with the children's population in the next one hundred years which would lead to more dependants than the work force.

The graph of the population proportions for the susceptible adults, susceptible under 15, and the infectives is shown in figure 5.4.7.

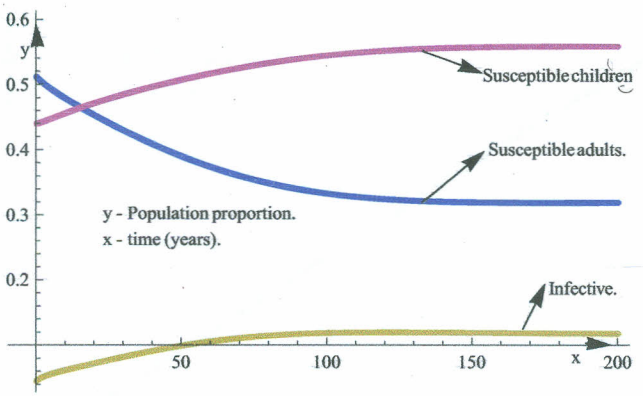

Figure 5.4.7: *Graph of the Population proportions of susceptible adults, children and infective.* $R_o = 1.39613$.

5.5 Treatment Incorporated

Treatment of adult infectives with no behavioral change and no change in infectiousness

We investigate the effects of treatment which does not reduce infectiousness or which is counterbalanced by reckless sexual practices with no counseling on those treated. The population growth curve appears as shown in figure 5.5.1.

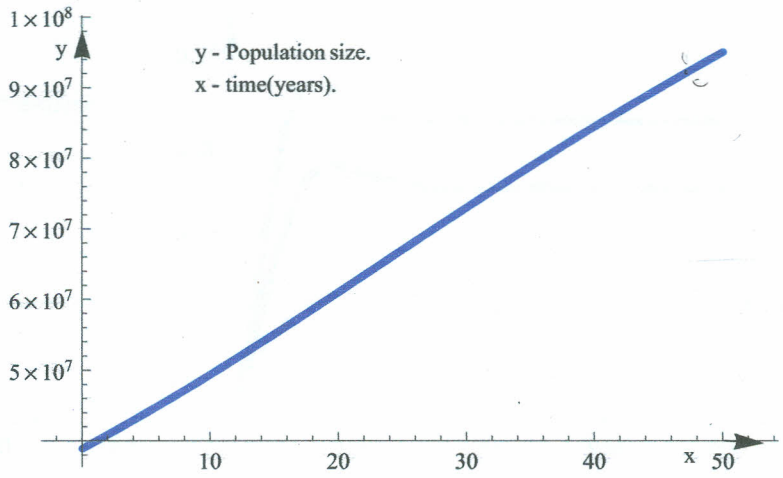

Figure 5.5.1: *Graph of Population against time in years with* $\beta_1 c_1 = 0.1881, R_0 = 1.93215$..

According to this model, treatment that does not reduce infectiousness dramatically changes the population size as shown above. The increase in the population size would be about 62 million people less than it could have been in the absence of the disease and less than 14 million people in the presence of the disease without treatment in 50 years, (Figure 5.3.3, 5.4.4 and 5.5.1).

The parameter ϵ is the proportion of infectives who receive treatment. Different parameter values for treatment are simulated and the results are shown in figure 5.5.2.

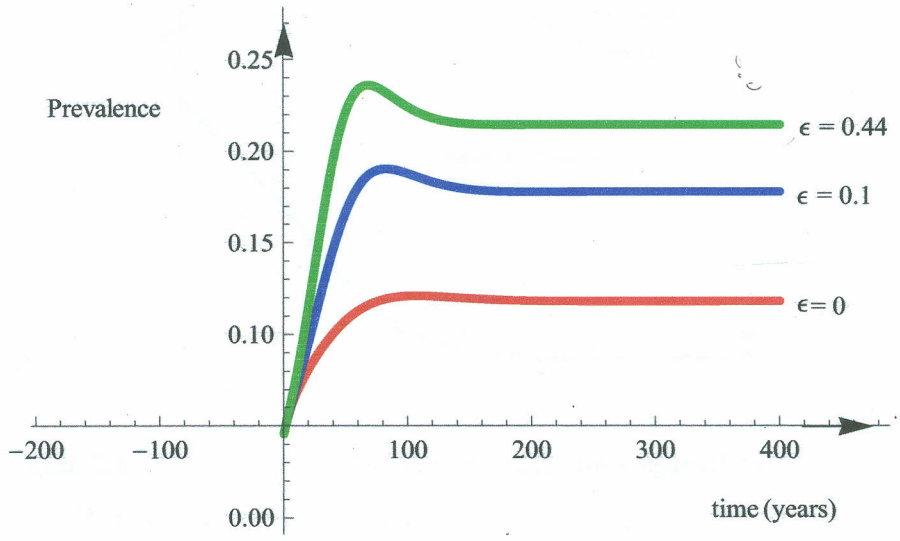

Figure 5.5.2: *Graph of the disease prevalence against time. When* $\epsilon = 0, R_0 = 1.39613$. *When* $\epsilon = 0.1, R_0 = 1.69441$.*When* $\epsilon = 0.44, R_0 = 1.93215$.

The increase in the disease prevalence level with treatment may be due to the fact that treatment lengthens the lives of the infectives and as such those who would have died of AIDS do become healthier and continue to spread the disease if they are not counseled or the counseling has no effect in changing there social behaviour.

The population proportions for adults and for those under 15 years is shown in figure 5.5.3.

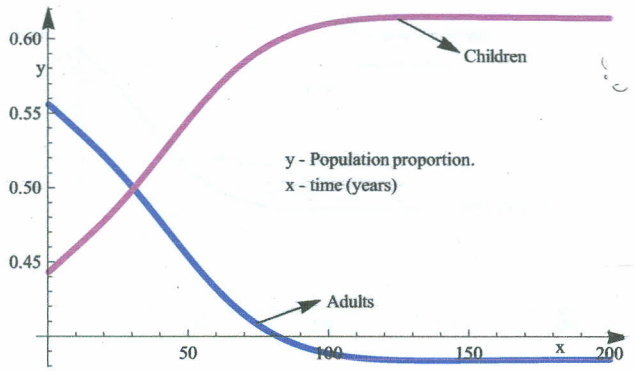

Figure 5.5.3: *Graph of the Population proportions against time. In this case, R_o is calculated to be* 1.93215.

The steady states of the population proportions changes drastically from 56 percent to 38 percent for adults and from 43 percent to 62 percent for those below 15 years after about 100 years.

The population proportions of those below 15 years, adults and HIV/AIDS victims are shown in figure 5.5.4.

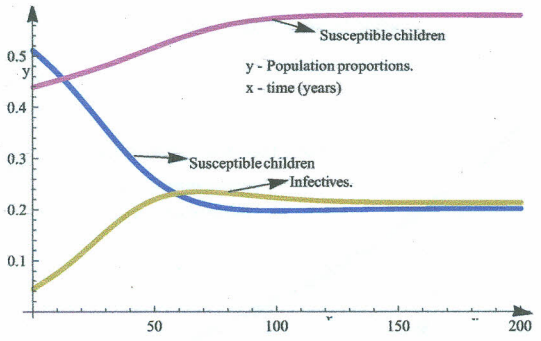

Figure 5.5.4: *Graph of the population proportions against time,* $R_0 = 1.93215$.

This model suggests that the proportion of HIV/AIDS victims in the population would be higher than the susceptible adults (figure 5.5.4).

Treatment of adult infectives with behavioral change and change in infectiousness

This reflects the current state of HIV/AIDS in Kenya where 44 percent of HIV/AIDS infectives receive treatment and counseling. According to medical research done in the U.S.A [13] treatment alone reduces infectiousness by 50 percent. In Kenya, no data exist to estimate by which percentage treatment coupled with counseling (zero grazing,condom use and circumcision) reduces infectiousness. We begin our simulations by varying the effectiveness of counseling and treatment from 50 percent ($\beta_2 c_2 = 0.0905$) to 80 percent($\beta_2 c_2 = 0.03762$). The simulations for the population size is shown in figure 5.5.5.

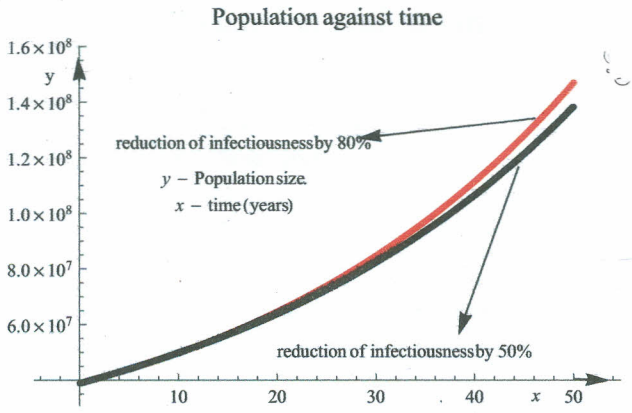

Figure 5.5.5: *Red line represents treatment and counseling where* $\beta_2 c_2 = 0.03762$. *Black line represents treatment alone where* $\beta_2 c_2 = 0.09405$.

The application of treatment alone that reduces infectiousness by 50 percent would increase the population size by about 20 million people less than it could have been in the absence of the disease and more than 28 million people in the presence of the disease without treatment in 50 years, however, when the treatment is coupled with counseling that reduces infectiousness by 80 percent, then the increase in the population size would be about 10 million people less than it could have been in the absence of the disease and more than 38 million people in the presence of the disease without treatment in 50 years. (figure 5.3.3, 5.4.4 and 5.5.5).

We observe here that the population size in 50 years time, assuming treatment and counseling that reduces infectiousness by 80 percent is equivalent to the expected population size of Kenya in the next 50 years (150 million people) at the current growth rate of 2.691 percent. This is clearly shown in figure 5.5.6.

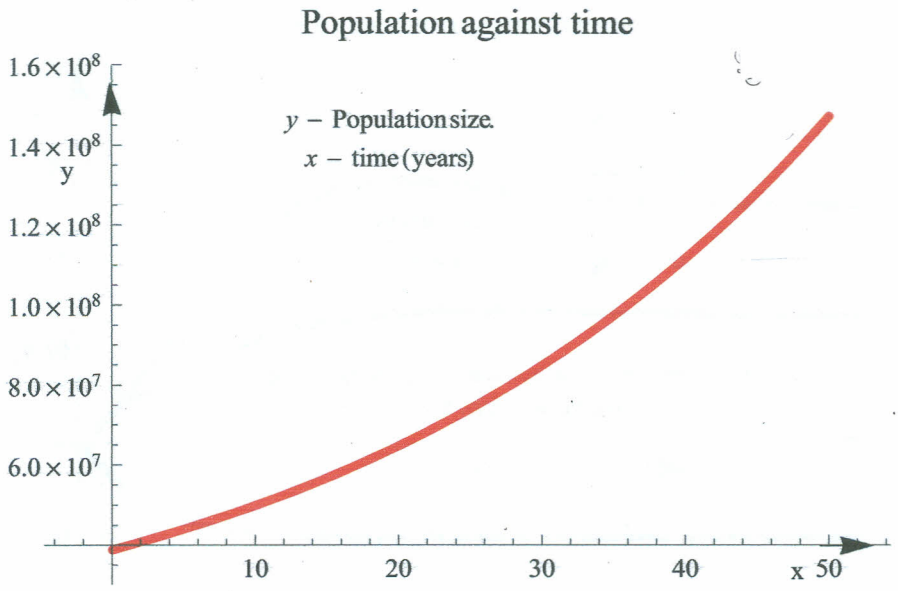

Figure 5.5.6: *Population growth rate is 2.691 percent,* $\beta_2 c_2 = 0.03762$ $R_0 = 0.648258$

In this stage we simulate the prevalence of the disease with time in the application of treatment alone that reduces infectiousness by 50 percent, treatment coupled with counseling that further reduces infectiousness by 80 percent and treatment that does not reduce infectiousness as shown in figure 5.5.7.

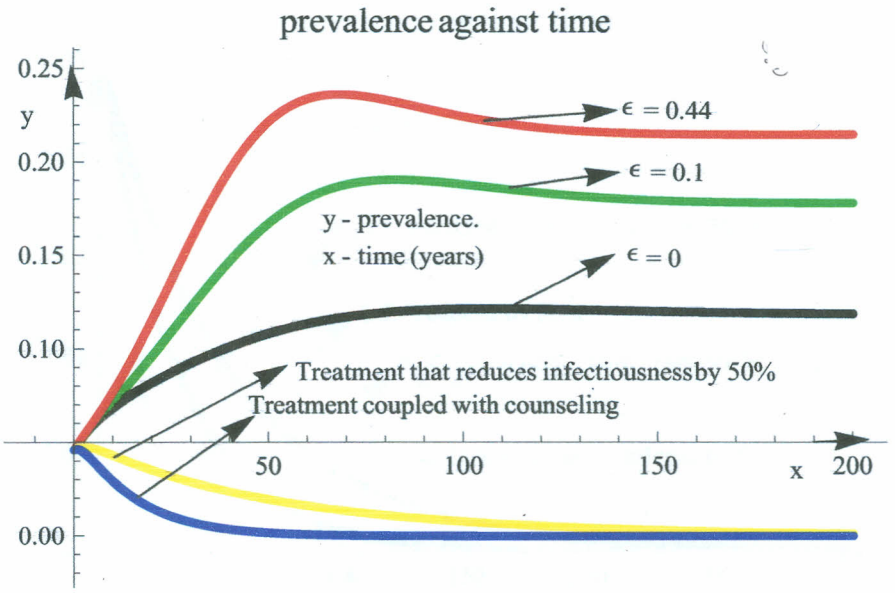

Figure 5.5.7:

According to this model as observed in figure 5.5.2, increasing the proportion of the infectives who receive treatment when that treatment does not reduce infectiousness is disastrous, however when the treatment reduces infectiousness, then it would be helpful (yellow line). Treatment coupled with counseling that further reduces infectiousness is even much better (blue line).

We again simulate the prevalence of the disease with time for $\beta_2 c_2 = 0.0905$ (reducing infectiousness by 50 percent), $\beta_2 c_2 = 0.07524$ (reducing infectiousness by 60 percent), $\beta_2 c_2 = 0.03762$ (reducing infectiousness by 80 percent) and $\beta_2 c_2 = 0.01881$ (reducing infectiousness by 90 percent) as shown in figure 5.5.8.

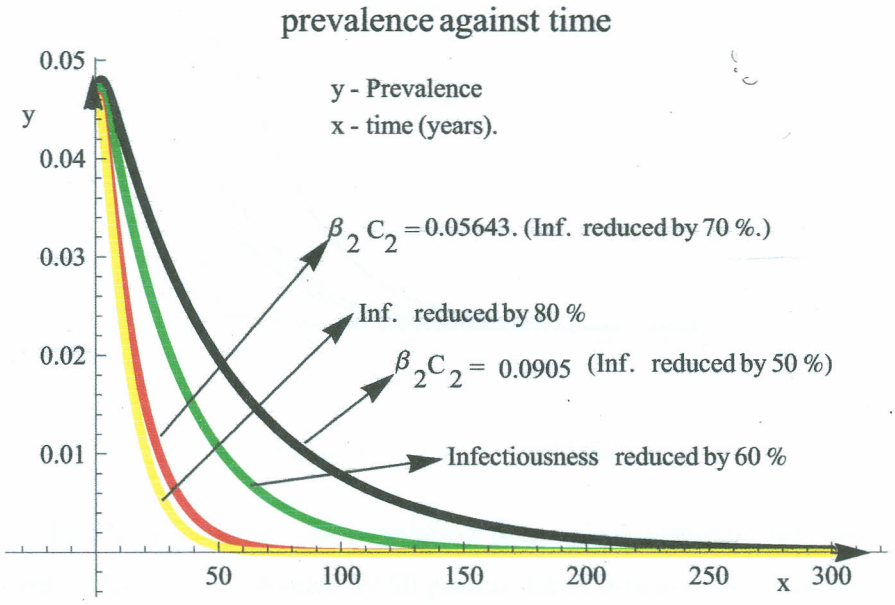

Figure 5.5.8:

We observe that at the estimated current force of infection of $\beta_1 c_1 = 0.1881$, coupled with treatment alone that reduces infectiousness by 50 percent, the disease will die out in about 200 years time (figure 5.5.8), however if effective counseling that further reduces infectiousness by 80 percent is incorporated, the disease would die out in less than 70 years (figure 5.5.8). We thus conclude that counseling and treatment that reduces infectiousness is very effective in controlling the spread of the disease.

Using the force of infection of $\beta_1 c_1 = 0.1881$, coupled with treatment and counseling that reduces infectiousness by 80 percent ($\beta_2 c_2 = 0.03762$), the disease will always die out even with different initial conditions as shown in figure 5.5.9.

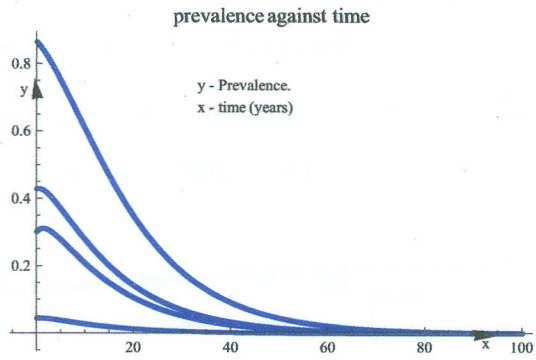

Figure 5.5.9: $R_0 = 0.648258$.

The simulations for the population proportions assuming that treatment reduces infectiousness by 50 percent for adults and for those under 15 years is shown in figure 5.5.10.

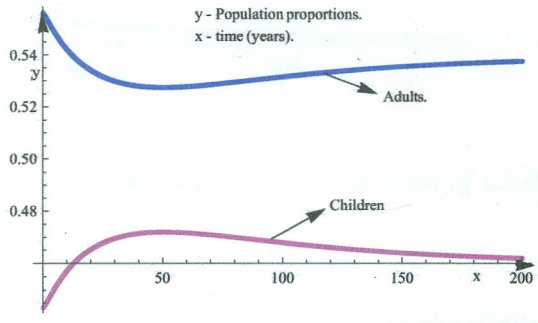

Figure 5.5.10: *Graph of the Population proportions of adults and children against time.*

It appears that the proportion of the adults in the population is declining while the population proportion of those under 15 years is increasing. It is also important to note that after 200 years, the proportion of adults

added together with the proportion of children equals to one implying that the disease must have been eliminated.

The population proportions of those below 15 years, adults and HIV/AIDS victims appears as shown in figure 5.5.11.

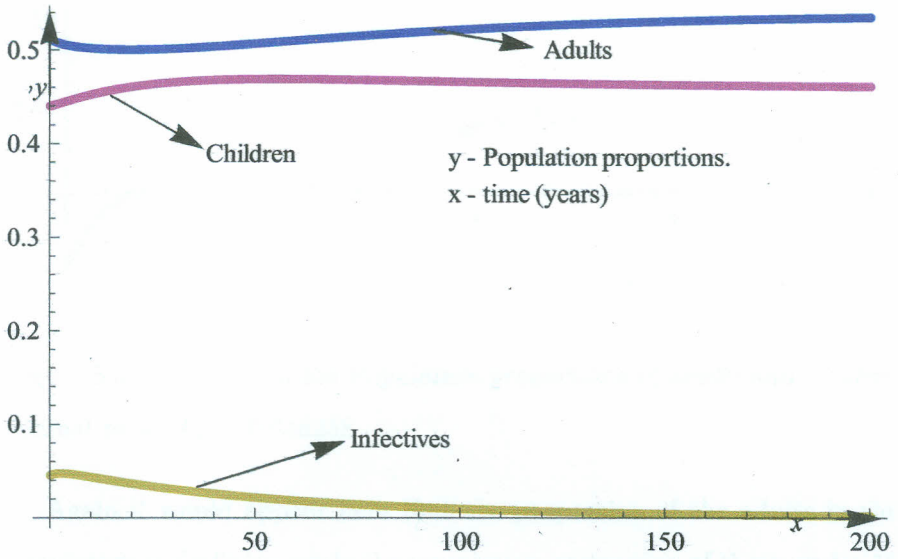

Figure 5.5.11: *Graph of the population proportions of adults, children and infectives against time.*

Here we confirm that in the next 200 years the disease would be no more.

The simulations for the population proportions assuming that treatment coupled with counseling reduces infectiousness by 80 percent for adults and for those under 15 years is shown in figure 5.5.12.

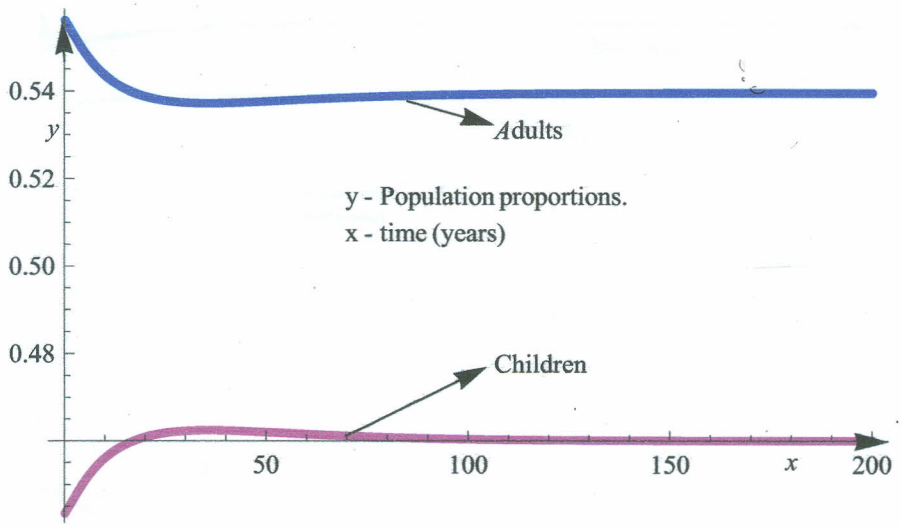

Figure 5.5.12: *Graph of the Population proportions of adults and children against time.* $R_0 = 0.648258$

Again it would appear here that the proportion of the adults in the population is declining while the population proportion of those under 15 years is increasing then stabilize in the next 30 years when the disease has been eliminated, however the graph of the the population proportions of those below 15 years, adults and HIV/AIDS victims reveals that the proportion of the adults in the population is actually increasing but appears to be reducing because the infectives are being eliminated as shown in figure 5.5.13.

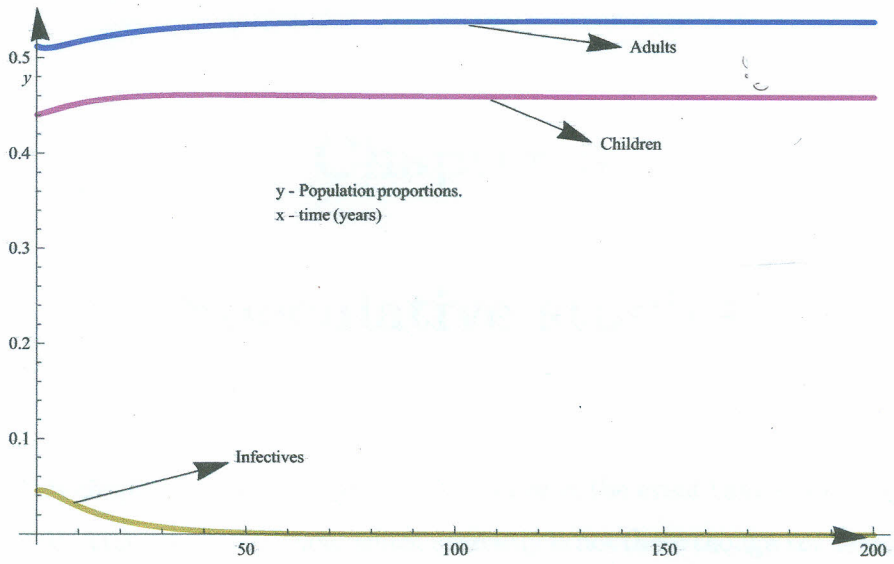

Figure 5.5.13: *Graph of the population proportions of adults, children and infectives against time.* $R_0 = 0.648258$

Chapter 6

Speculative studies

This chapter is based on speculative studies in the event that an effective HIV/AIDS vaccine is found which currently is not there though the search for an effective HIV/AIDS vaccine has been on-going for over 20 years with the latest findings in Thailand [4] indicating that some vaccine for some strain of the HIV/AIDS virus has been obtained with an efficacy level of 30 percent. It is not known however that the vaccine acts as the "Leaky type" or as the "All or Nothing type" or both.

6.1 Vaccination incorporated

In this section we assume that 40 percent of the susceptible are vaccinated and the vaccine acts both as the "leaky" type and the "All or Nothing" type of Vaccine. We also assume that one million susceptibles are vaccinated with 300,000 of them completely covered by the vaccine. We further assume that the vaccine is 30 percent effective [4]. We begin by investigating the effects of treatment alone without vaccination then simulate for vaccination alone without treatment in a single graph to determine

which of them would be more effective using the parameters in chapter 6 assuming that treatment alone reduces infectiousness by 50 percent, i.e $\beta_2 c_2 = 0.09405$. The simulations are shown in figure 6.1.1.

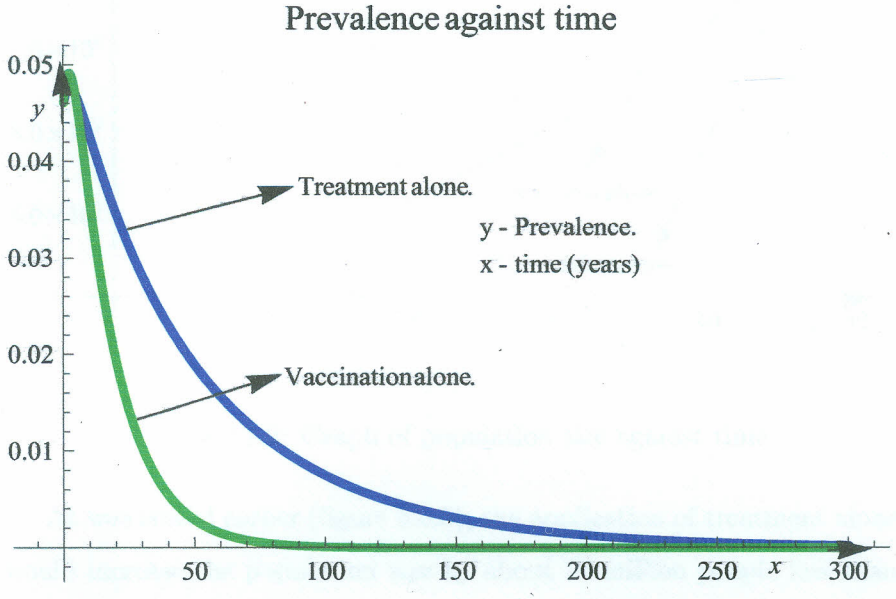

Figure 6.1.1:

This model suggests that vaccination alone (Green line) without treatment is more effective in controlling HIV spread using the vaccination and treatment parameters above.

The graphs for the population size in separate application of treatment and vaccination is shown in figure 6.1.2.

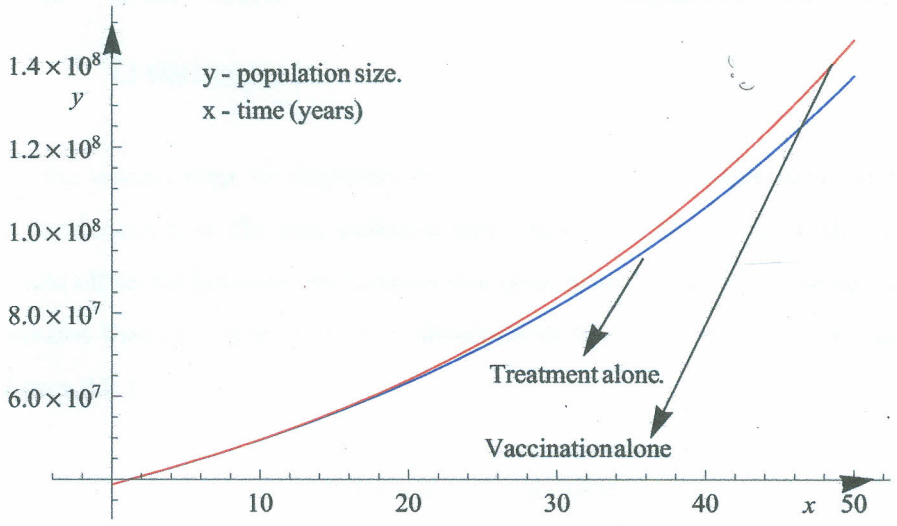

Figure 6.1.2: Graph of population size against time

As was stated earlier (figure 5.5.5), the application of treatment alone would increase the population size by about 20 million people less than it could have been in the absence of the disease and more than 28 million people in the presence of the disease without any other intervention in 50 years, whereas vaccination when applied alone, would increase the population size by about 15 million people less than it could have been in the absence of the disease and more than 33 million people in the presence of the disease without any other intervention in 50 years, (figure 5.3.3, 5.4.4 and 6.1.2).

6.2 The trade - off between vaccination and treatment

In the second step we simulate vaccination alone, treatment alone and a combination of the two within a single graph to determine wether a trade off exists between vaccination and treatment. In this case we again assume that treatment reduces infectiousness by 50 percent as shown in figure 6.2.1.

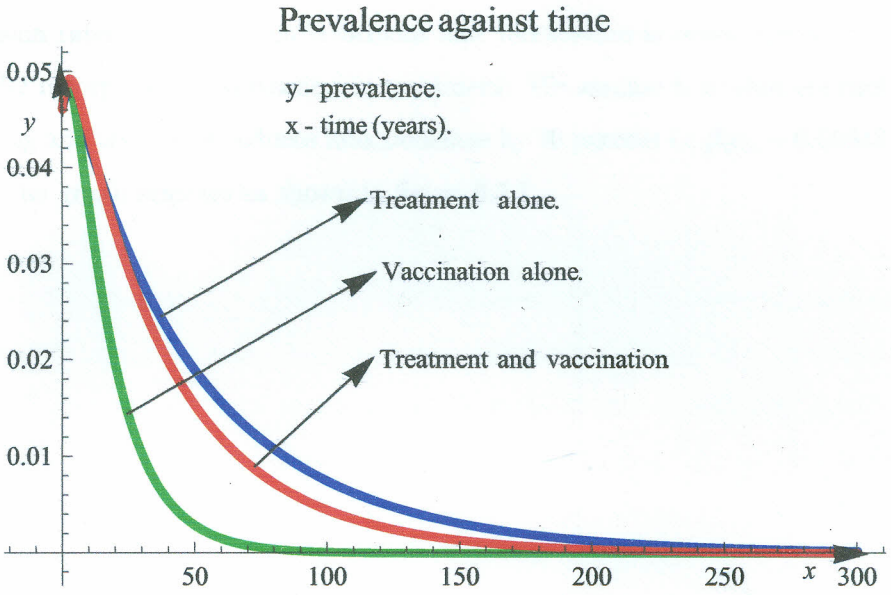

Figure 6.2.1:

We note that a trade - off seems to exist between vaccination and treatment using our parameter values for treatment and vaccination. We observe that vaccination alone (Green line) is still more effective than a combination of treatment and vaccination (Red line) implying that treatment would be counter productive when applying vaccination.

6.3 Threshold Parameters

We set to numerically investigate the thresholds of the disease transmission rates beyond which treatment and vaccination is counterproductive by incorporating counseling in treatment. We assume here that counseling and treatment reduces infectiousness by 70 percent i.e $\beta_2 c_2 = 0.05643$. The graph appears as shown in figure 6.3.1.

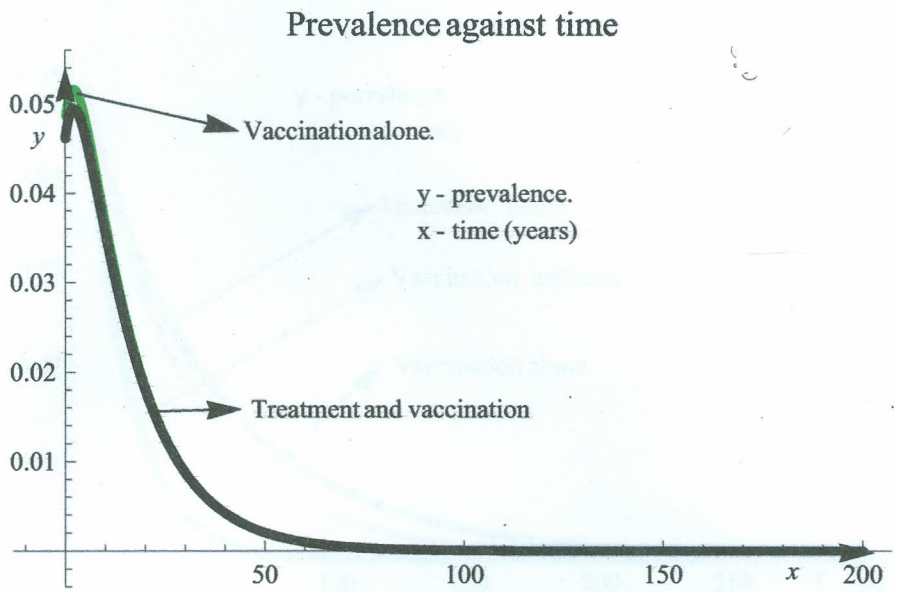

Figure 6.3.1:

We observe that treatment and counseling that reduces infectiousness by 70 percent coupled together with vaccination (Black line) is equivalent to applying vaccination alone (Green line) thus Counseling coupled with treatment that reduces infectiousness by less than 70 percent will be counterproductive if applied together with vaccination whereas if infectiousness is reduced by more than 70 percent as a result of treatment and counseling then a combination of treatment,counseling and vaccination would not be counterproductive using our parameters above.

Assuming that treatment coupled with counseling reduces infectiousness by 80 percent, we set to simulate the effects of the same in terms of prevalence. The simulations for the prevalence appears as shown in figure 6.3.2.

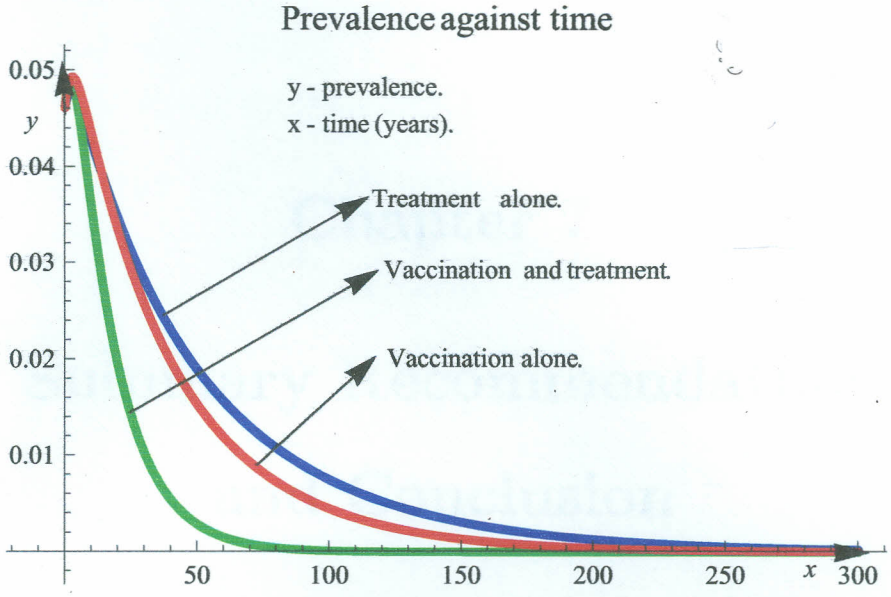

Figure 6.3.2:

It is important to note here that a combination of treatment, counseling and vaccination (Green line) would be more effective than each one of them applied separately.

Chapter 7

Summary Recommendation and Conclusion

7.1 Summary

We formulated a comprehensive HIV/AIDS transmission model incorporating counseling, treatment, vaccination, stages of infection, age structures, vertical transmission, and the population size with reference to the Kenyan situation and according to this model, we observed the following:

- If there is no intervention even at the current reduced rates of HIV/AIDS infection, our model predicts that we might get a lower proportion of adults in the population than those who are 15 years and below which would mean that we have more dependants than the workforce in the country.
- Treatment that does not reduce infectiousness is worse than when the treatment is not applied at all, however when coupled with

effective counseling, then it is very effective in combating the spread of the disease and finally eliminating it.

- Our speculative studies for vaccination showed that careful considerations should be made when a combination of vaccination and treatment is to be applied because a combination of the two could be counterproductive or helpful depending on how it is implemented.

7.2 Recommendation

HIV/AIDS still remains a very serious problem in Kenya though a lot of effort has been put to combat the spread of the disease through condom use, male circumcision e.t.c., but more still needs to be done in terms of awareness campaigns. It is also important to ensure that any treatment applied reduces infectiousness. This is perhaps a possible area of research in clinical health to identify possible ways of improving the effectiveness of HIV/AIDS drugs.

7.3 Conclusion

It should be stressed that mathematical models have become important tools in analyzing the spread and control of infectious diseases and non mathematicians should be able to appreciate the usefulness of these models, however deterministic Mathematical modeling is more useful in predicting trends of diseases usually over longer periods of time but might not be very accurate in the short run because of changing circumstances.

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