

The AIDS Epidemic in Tanzania:

A System Dynamics Approach for Policy Development

Alexander Alex Focus



Submitted in Partial Fulfilment of the Requirements for the Degree of
Master of Philosophy in System Dynamics

System Dynamics Group, Social Science Faculty, Department of
Geography, University of Bergen

Supervised by: David Wheat, Ph.D.
Associate Professor of System Dynamics

August, 2008

*To my daughter Lauryn and my wife Emma,
my source of strength and inspiration.*

The AIDS Epidemic in Tanzania: A System Dynamics Approach for Policy Development

Alexander Alex Focus

Abstract

Key Words: AIDS policy, epidemic, HIV, model, simulation, system dynamics, Tanzania.

Despite all the efforts made by the government and other agencies, the AIDS epidemic in Tanzania still remains a major challenge. Over the last two decades the numbers of people living with HIV/AIDS and the deaths caused by the epidemic has been increasing. The predominant mode of transmission is heterosexual contact. In Tanzania, HIV appears to be mostly diffused through heterosexual intercourse. Most infections occur in people between the ages 15 and 59. HIV/AIDS is also a disease of children and this is due to the high fertility of HIV-positive mothers. These make the epidemiological profile very different from HIV in the United States and Europe, where most of the HIV/AIDS policy modeling efforts have been concentrating. Using the method of system dynamics, a model of the spread of HIV in the Tanzania population has been developed. This model provides an increased understanding of HIV transmission dynamics and a way of judging the effectiveness of various intervention strategies. This model provides a policy tool that can be used in the ongoing debate about better management of the epidemic. The purpose of this model is to support the government of Tanzania and policy makers in their effort to slow down the spread of AIDS epidemic. The model replicates the historical data reasonably well and suggests that Highly Active Antiretroviral Therapy (HAART) alone, cannot significantly impact the number of HIV/AIDS infected individuals in the long run. The combination of two policies the HIV education and awareness program and Nevirapine treatment shows the maximum effectiveness.

Acknowledgements

Many people have been instrumental to make this work a reality. It is not possible to mention all names, in this regard, for those whose names will not be specified in this list of acknowledgement, please accept my sincere apologies and thank you all.

I'm greatly indebted and do not have sufficient way of expressing my deepest gratitude to my supervisor, Professor David Wheat, for his tireless dedication and highly qualified support, constructive comments, friendliness, inspiration and understanding. His continuous support, advice, encouragement gave me confidence to think critically. Without your inspiration and close guidance, it would have been difficult to accomplish this work. Professors Erling Moxnes, Paal Davidsen, and visiting professor Steve Alessi, I thank you very much for being a fuel in this process, for your moral and academic support throughout the entire programme. "mange takk skal dere ha "

My friends PhD students Mihaela Tabacaru, Mateo Pedercini, Andrea Bassi and John Pastor Ansah, all volunteered to help in one way or another throughout the entire programme, I do not have words to thank you. You have been a green light to my life throughout this long journey.

I greatly appreciate the friendliness, support and encouragement of my colleagues at the System Dynamics Group, Bergen, whom we shared all laughs and tears. My special thanks to Qian Hu, and Juan for great care and companion. My sincere appreciation also goes to the Ifakara Research and Development Centre, Tanzania (IRDC) for granting me permission to use their facilities including the office during data collection period.

To dearest friends Jonas, Lilian, Vonna, Patrick, Emma, Rodney, Vania and Viola, I thank you very much for your friendliness, continuous encouragement and moral support throughout my study period in Norway.

Finally, the greatest appreciation is reserved for my daughter Lauryn and my wife Emma, you have been a source of inspiration, support and encouragement throughout my study period. Without your love and support, this study would never have begun and would never have succeeded.

"If I have the gift of prophecy and can fathom all mysteries and all knowledge, and if I have a faith that can move mountains, but have not love, I am nothing". 1 Cor 13:2

Abbreviations/Acronyms

ABC	Abstain, Be faithful, Condom
AIDS	Acquired Immune Deficiency Syndrome
ANC	Antenatal Clinics
ART	Antiretroviral Therapy
ARVs	Antiretroviral drugs
CDC	U.S. Centers for Disease Control and Prevention
ELISA	Enzyme Linked Immunosorbent Assay
EPP	Estimation and Projection Package
GPA	Global Programme on AIDS
HAART	Highly Active Antiretroviral Therapy
HIV	Human Immuno-deficiency Virus
HIVNET 012	International HIV Networks for Prevention trials 012
IRDC	Ifakara Research and Development Centre
MOH	Ministry of Health
MTCT	Mother-to-Child Transmission
NACP	National AIDS Control Programme
PMTCT	Prevention of Mother-to-Child Transmission
PHPT	Perinatal HIV Transmission controlled Trial in Thailand
PLWHA	People Living with HIV and AIDS
TACAIDS	Tanzania Commission of AIDS
TDHS	Tanzania Demographic and Health Survey
UNAIDS	Joint United Nations Programme on AIDS
VCT	Voluntary Counselling and Testing
WHO	World Health Organisation

Table of Contents

1. INTRODUCTION	10
Brief history of HIV and AIDS	10
Biological and Clinical Aspects of HIV Infection.....	11
HIV and AIDS Global Situation	14
The HIV and AIDS situation in Tanzania	17
Government response on HIV and AIDS	19
A brief outline of this thesis	20
2. LITERATURE REVIEW.....	22
3. RESEARCH METHOD	29
Why is system dynamics applicable to the problem?.....	30
Building blocks of system dynamics.....	30
4. MODEL BUILDING	37
Key variables.....	37
Time Horizon	37
Reference Mode	38
Dynamic Hypothesis	40
Model Building	42
Model Building Overview.....	42
Model 1: An SI Model	44
Model 2: SIR model	51
Model 3: Introduce AIDS Deaths.....	54
Model 4. Births and Non AIDS deaths.....	57
Model 5: HIV-positive children	60
Model 6: HIV education and awareness program.	63
5. VALIDATION AND MODEL TESTING.....	68
Tests to Build Confidence	68
Verification	68
Face Validity	69
Historical Behavior	69
Extreme condition tests.....	70
Sensitivity analysis tests.....	75
Summary	84
6. POLICY ANALYSIS	85
Policy test 1: Reduce HIV infectivity and average HIV contact frequency.	85
Policy test 2: Reduce the mortality rate.....	88
Policy test 3. Reduce transmission of HIV from mother to child.....	92
Policy test 4. Reduce infectivity AND average HIV contact frequency AND transmission of mother to child.	93
Policy test 5. Reduce infectivity AND average HIV contact frequency AND mortality rate AND transmission of HIV from mother to child.	94

7. CONCLUSION: 96
 Contribution and Major Findings96
 Limitations and future research.....96

REFERENCES: 98

APPENDIX..... 107

1. INTRODUCTION

Tanzania, like many other countries in sub-Saharan Africa, is experiencing a serious epidemic, threatening its very survival. Today, despite many years of national response, the impact remains devastating, given the pace of spread of HIV/AIDS. Our thesis is aimed at using the method of system dynamics to create a model that will be used to assist in understanding the dynamics of the HIV transmission and help in developing policies to mitigate its impact on the Tanzanian population.

The first three sections of this chapter present epidemiological information on the human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome (AIDS), including a brief history followed by biological and clinical aspects of HIV and AIDS. Then there is a summary of the government response to the problem.

Brief history of HIV and AIDS

More than twenty five years ago the Centers for Diseases Control and Prevention (CDC) reported the unsettling news of five deaths in Los Angeles from *Pneumocystis Carinii* pneumonia (PCP) (CDC, 1981), the event that marked the beginning of a world-wide battle against HIV/AIDS epidemic. A similar virus was later identified by Luc Montagnier and colleagues at the Pasteur Institute in Paris (Barre-Sinoussi *et al.*, 1983). Since then numerous, co-existing epidemics have emerged throughout the globe – some of them highly volatile – all of them compromising the health and well-being of the communities they impact. HIV weakens the immune system by destroying T-Helper cells that play a major role in this system. A person carrying HIV will eventually develop the Acquired Immunodeficiency Syndrome (AIDS). When a person develops AIDS, the immune system gradually loses resistance against a variety of infectious diseases such as tuberculosis (TB). Because of weakened immune response, an AIDS infected person will eventually die of a disease the system no longer can handle.

The disease is characterised by a long and variable incubation period (the time from infection until the person develops AIDS) and the infected person may be infectious to a variable degree during this same incubation period. There are indications that the transmissibility of HIV infection varies greatly during the multi-year course of infection in an individual (Goedert *et al.*, 1987; Longini *et al.*, 1990; and Dangerfield *et al.*, 2001). Transmission may be more likely during the early flu-like illness, which occurs just after HIV

infection and before the body develops an antibody response. Since the HIV virus level in the blood decreases during the asymptomatic period, the transmissibility appears to be lower in this period (Hethcote, 1992 p.1; & Dangerfield *et al.*, 2001). Then the transmissibility seems to increase again as the Cluster of Differentiation 4+ or CD4+ or T-helper cell count gets low, the HIV virus level in the blood increases, and symptoms appear. The median incubation period is estimated to be about 7-10 years in previously healthy adults, though it is shorter for children, the elderly, and those with prior health problems (Cooley & Hamill, 1996; and Dangerfield *et al.*, 2001).

- a) We assume three main modes of transmission of HIV (e.g., see May & Anderson 1988; Hethcote, 1992; CDC, 2004; and UNAIDS/WHO, 2005). Modes of transmission vary between and within region. Sexual contact between people is the common mode of transmission. In Europe and the United States, such contacts have been typically homosexual but heterosexual contact has been a more common transmission mode in sub-Saharan Africa, Latin America and Asia.
- b) Blood transfusion. In the United States and Western Europe this has been an important transmission mechanism due to needle sharing among Intravenous Drug Users (IVDUs). In sub-Saharan Africa, non-sterile needles used for injection of medications have also resulted in numerous HIV infections.
- c) Infected mother to child transmission. Prenatal transmission from an HIV-infected mother to her child occurs before, during birth. Approximately 30 percent of the children of HIV infected mothers are also infected (Hethcote, 1992). In Africa, studies suggest that one in three newborns infected with HIV die before the age of one, over half die before reaching their second birthday, and most are dead before they are five years old (Newell *et al.*, 2004; and UNAIDS/WHO, 2005).

The first and the last mode of transmission is the main focus of this study.

Biological and Clinical Aspects of HIV Infection

The retrovirus called human immunodeficiency virus (HIV) was established in 1983 as the causative agent of AIDS. Isolates of HIV are molecularly and biologically heterogeneous, with some isolates being more virulent. Moreover, the HIV virus can change rapidly, even within an individual. The HIV infects a subpopulation of thymus-derived T-

lymphocytes called CD4⁺ lymphocytes or T4 cells, which are helper/inducer cells. These T-cells perform recognition and induction functions as part of the immune response to foreign stimuli. The HIV integrates into the CD4⁺ host cell DNA, where it can remain dormant for a long time. The CD4⁺ T-lymphocytes are eventually killed by the HIV while the HIV reproduces itself, so that the number of CD4⁺ cells gradually decreases from the normal number of about 900/ml (Hethcote and James, 1992) . This leads to severe immunodeficiency in persons infected with HIV. Thus the natural history of HIV infection is a gradual depletion of CD4⁺ cells, progressive unresponsiveness of the immune system, and increased susceptibility to opportunistic infections such as *Pneumocystis Carinii* pneumonia and malignancies such as Kaposi's sarcoma and tuberculosis.

Transmission of HIV infection can be through the transfer of either cell-free HIV virus or HIV-infected lymphocytes. The probability of transmission appears to depend on the stage of the HIV infection. In the first few weeks following infection, more HIV has been isolated from blood plasma than in the asymptomatic stage, so that people in the pre-antibody stage may be more infectious than people in the symptomatic stage. In the late stages of HIV infection and early stages of AIDS, the cell-free HIV virus is found more frequently in blood plasma, so that these people may also be more infectious.

The enzyme-linked immunoassay (ELISA) test for HIV antibodies is inexpensive and useful for screening large numbers of samples ; this test has almost no false negatives, but it is very sensitive (i.e., it has false positives at the rate of about 2 per 1000) . Samples which are positive by the ELISA test are then tested by the Western blot assay. This immunoblot method is very specific (i.e., false positives occur in no more than 0 .001% of the samples). Another test used is an indirect immunofluorescent antibody test involving microscopic examination of infected cell spots on glass slides. The combination of these and other tests such as PCR (polymerase chain reaction) are quite accurate in identifying HIV-positive individuals (Hethcote and James, 1992; and CDC, 2004).

There are many different clinical manifestations of HIV infection and AIDS. Acute HIV infection occurs just after infection in some patients and is characterized by an acute febrile illness with fever, sweats, lethargy, muscle ache, headache and sore throat. These symptoms may last 2 to 3 weeks. About 2 months after infection, the immune system has generated antibodies which are recognized by the ELISA test. After an asymptomatic period of about 5 years, an HIV-infected person may develop some symptoms such as oral candidiasis (thrush), hairy leukoplakia, herpes zoster, weight loss, diarrhoea, persistent generalized lymphadenopathy, neurologic diseases (dementia, myelopathy, polyneuropathy)

and tuberculosis. The HIV-infected person may die of these diseases, but most get one or more of the opportunistic infections or neoplasms which characterize AIDS. These include cytomegalovirus infection, *Pneumocystis Carinii* pneumonia, toxoplasmosis of the brain and Kaposi's sarcoma.

Previously, having AIDS was defined as having HIV infection and getting one of these additional diseases. Now it is additionally defined as a CD4+ count below 200, even without an opportunistic infection. Many other illnesses and corresponding symptoms may develop in addition to those listed here.

CD4+ count below 200 cells/ml

- *Pneumocystis carinii* pneumonia, "PCP pneumonia," now called *Pneumocystis jiroveci* pneumonia
- Candida esophagitis -- painful yeast infection of the esophagus
- Bacillary angiomatosis -- Skin lesions caused by a bacteria called *Bartonella*, which is usually acquired from cat scratches

The current surveillance definition replaced criteria published in 1987 that were based on clinical conditions and evidence of HIV infection but not on CD4+ T-helper cell determinations (CDC, 2003; and UNAIDS/WHO, 2005).

In many developing countries, where diagnostic facilities may be minimal, epidemiologists employ a case definition based on the presence of various clinical symptoms associated with immune deficiency and the exclusion of other known causes of immunosuppression, such as cancer or malnutrition (CDC, 2003). AIDS cases data used in this thesis were obtained from National Aids Control Programme-Tanzania (NACP, 2005) Surveillance report, where different method have been employed which includes antenatal clinics tests, blood donations tests and voluntarily control test for the period between 1983 and 2004 and the various clinical symptoms.

HIV and AIDS Global Situation

“In June of 1981 we saw a young gay man with the most devastating immune deficiency we had ever seen. We said, ‘We don’t know what this is, but we hope we don’t ever see another case like it again’.” (CDC, 1982).

Over 25 years on, HIV/AIDS epidemic continues to spread without relief, with more than 70 million persons infected worldwide since its beginning. The HIV/AIDS epidemic has become a health, socio-economic and development disaster, with far reaching implication for individuals, communities and countries. “No other disease has so dramatically highlighted the current disparities and inequalities in the health care access, economic opportunity and protection of basic human rights” (UNAIDS/WHO, 2007).

United Nations AIDS programme and World Health Organisation (UNAIDS/WHO) estimates that 33.2 million people were living with HIV/AIDS in the year 2007. Table 1.1 shows the estimated number of people living with HIV/AIDS, newly infected and AIDS death for the year 2007.

Number of people living with HIV in 2007		
	Total	33.2 million [30.6–36.1 million]
	Adults	30.8 million [28.2–33.6 million]
	Women	15.4 million [13.9–16.6 million]
	Children under 15 years	2.5 million [2.2–2.6 million]
People newly infected with HIV in 2007		
	Total	2.5 million [1.8–4.1 million]
	Adults	2.1 million [1.4–3.6 million]
	Children under 15 years	420 000 [350 000–540 000]
AIDS deaths in 2007		
	Total	2.1 million [1.9–2.4 million]
	Adults	1.7 million [1.6–2.1 million]
	Children under 15 years	330 000 [310 000–380 000]

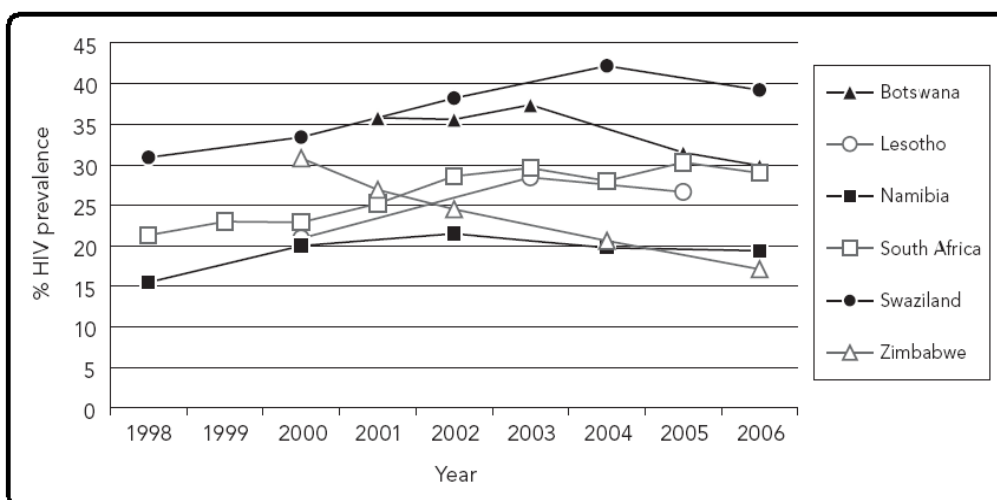
Source: UNAIDS/WHO report December 2007.

Table 1.1. Global summary of the HIV/AIDS epidemic December 2007.

Table 1.1 shows that the newly infected people in the year 2007 were estimated to be 2.5 million. In the same year, the HIV/AIDS epidemic claimed more than 2.1 million lives. Additionally, almost 6,800 adults and children are becoming infected each day, and close to half of these are young people under 24 years of age with over 95% occurring in poor and middle-income countries. Globally, the major mode of transmission remains; unprotected

sexual intercourse between men and heterosexual, needle sharing among Intravenous Drug Users (IVDUs), infected mother to child transmission (MTCT) and contaminated blood in health-care settings are other modes of HIV transmission although the relative importance of the modes of transmission varies between and within countries (UNAIDS/WHO, 2007). Data on Table 1.1 indicates that the HIV epidemic is far from over and requires more effort to stop the trend.

According to the UNAIDS/WHO 2007 report, sub-Saharan Africa remains the most affected region in the global AIDS epidemic. More than two thirds (68%) of all people HIV-positive live in this region where more than three quarters (76%) of all AIDS deaths in 2007 occurred. It is estimated that 1.7 million people were newly infected with HIV in 2007, bringing to 22.5 million the total number of people living with the virus. Unlike other regions, the majority of people living with HIV in sub-Saharan Africa (61%) are women (UNAIDS/WHO, 2007). The scale and trends of the epidemics in the region vary considerably, with southern Africa most seriously affected. This sub-region accounts for 35% of all people living with HIV and almost one third (32%) of all new HIV infections and AIDS deaths globally in 2007. According to the UNAIDS, life expectancy in sub-Saharan Africa, has fallen to below 50 years and nearly 10% of child mortality is HIV associated. “Current available data from sub-Saharan Africa show mostly stability, and declines at the country level have been observed in Zimbabwe, Kenya and Zambia” (UNAIDS/WHO, 2007). However, there are striking geographical differentials in HIV prevalence and trends within countries, and better understanding of local epidemiological and cultural contexts is vital for effective preventive interventions. National adult HIV prevalence exceeded 15% in eight countries in 2005 (Botswana, Lesotho, Mozambique, Namibia, South Africa, Swaziland, Zambia and Zimbabwe). Figure 1.1 shows the median HIV prevalence among women (15-49) attending antenatal clinics in Southern African countries.



Source: UNAIDS (2007) - Various antenatal clinic surveys.

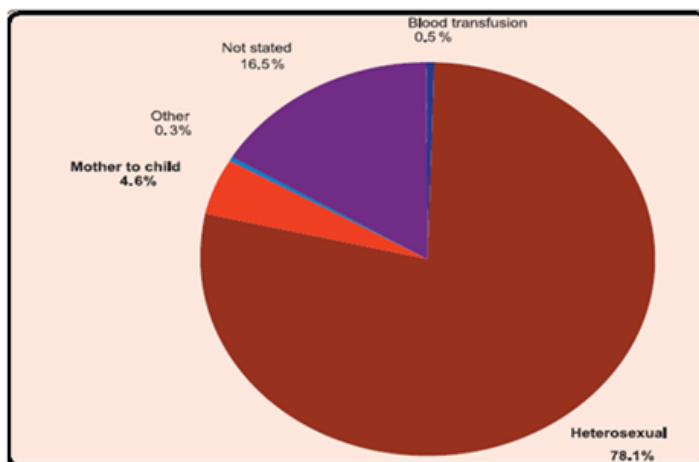
Figure 1.1. Median HIV prevalence among women (15-49) attending antenatal clinics in consistent sites in Southern African Countries, 1998-2006

Figure 1.1 shows the Median HIV prevalence among women (15–49 years) attending antenatal clinics in consistent sites in southern African countries, 1998–2006. Swaziland has the overall highest HIV prevalence at around 40% while Zimbabwe has the overall lowest HIV prevalence at around 20%.

In Asia, the epidemics are not driven by sexual behaviour in the general population, but mainly by injecting needles and sexual behaviour among sexual workers. The 92,000 adults and children estimated to be newly infected with HIV in East Asia in 2007 represent an increase of almost 20% over the 77,000 people who acquired HIV in 2001. Oceania also saw an increase in estimated new infections—from 3800 in 2001 to 14,000 in 2007. In the Caribbean, Latin America, the Middle East and North Africa, North America and Western Europe, the numbers of new HIV infections in 2007 remained approximately stable. However, “while the HIV epidemics in Asia appear stable, overall there are declines in Cambodia and Thailand (and possibly Burma), apparently as a result of successful application of the 100% condom use approach” (UNAIDS/WHO, 2007). Also, the epidemics in Latin America and the Caribbean are well established with nearly 1.6 million people already infected and almost 100,000 newly infected (UNAIDS/WHO, 2007).

The HIV and AIDS situation in Tanzania

The HIV/AIDS situation in Tanzania is the focus of this thesis. Like its neighbours in Sub-Saharan Africa, the country has been severely affected by HIV/AIDS epidemic. According to National AIDS Control Programme (NACP) in Tanzania, the first 3 cases of AIDS in the country were diagnosed in 1983 in Kagera region (NACP, 2005; TACAIDS, 2005). Since then, epidemiological data show an escalating epidemic, such that by 1986 all regions of the Tanzania had reported HIV/AIDS cases. With approximately one in ten adults aged between 15 and 59 living with HIV by the end of year 2000 (WHO, 2001), Tanzania is among the twenty-five countries with the highest HIV prevalence in the World. As is the case in other parts of Sub-Saharan Africa, almost 80% of HIV transmission is through heterosexual contact, and young people and women are particularly vulnerable (NACP, 2004; TACAIDS, 2004; & UNAIDS/WHO, 2004).



Source: NACP report, 2005

Figure 1.2. Possible source of infection for reported AIDS cases in Tanzania

Figure 1.2 shows that there are two main mode of transmission in Tanzania namely; heterosexual contacts and mother to child (MTCT). In the year 2004, heterosexual contacts accounted for 78.1% of all newly infections while mother to child accounted for 4.6%. However, mode of transmission for 16.5% of the newly infected could not be identified. The infection through blood transfusion is very small, only 0.5%.

In 2000, according to the Ministry of Health in Tanzania (MOH), adult morbidity and mortality study in three districts revealed that AIDS was the leading cause of death among adults aged 15-59 years. That means that the impact of HIV/AIDS is beginning to be felt in

all over the country. It is estimated that life expectancy will drop from 57 to 47 years by the year 2010 due to HIV/AIDS (Flessa, 2003). In the health division, the impact is being felt through the proportion of hospital beds occupied by patients with AIDS related conditions which are about 50% in urban areas, justifying the extension of the medical care for HIV/AIDS patient to their communities. In all areas, the loss of experienced and skilled manpower is increasingly being felt although exactly data for this group is difficult to find (NACP 2004; TACAIDS, 2004; and UNAIDS/WHO, 2004). Figure 1.3 shows the estimated top ten causes of death in Tanzania in the year 2002.

Top ten causes of death, all ages United Republic of Tanzania, 2002			
Causes	Deaths		Years of Life Lost
	(000)	(%)	(%)
All causes	583	100	100
HIV/AIDS	166	29	29
Lower respiratory infections	67	12	13
Malaria	56	10	12
Diarrhoeal diseases	31	6	6
Perinatal conditions	24	4	5
Tuberculosis	18	3	3
Cerebrovascular disease	16	3	1
Ischaemic heart disease	14	3	1
Syphilis	11	2	2
Road traffic accidents	10	2	2

Source: [Death and DALY estimates by cause, 2002](http://www.who.int/entity/healthinfo/statistics/bodgbddeathdalyestimates.xls)
<http://www.who.int/entity/healthinfo/statistics/bodgbddeathdalyestimates.xls>

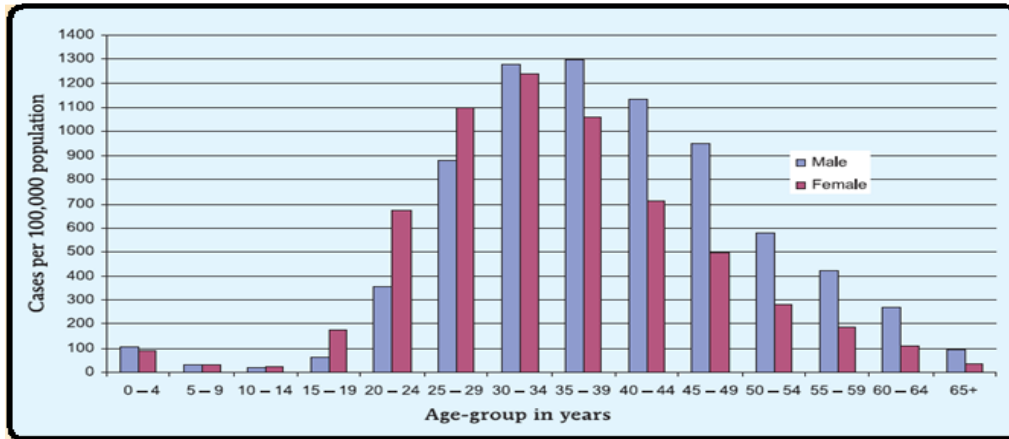
Source: Death and DALY estimates by cause, 2002, Tanzania

Figure 1.3. Top ten causes of death, all ages Tanzania, 2002

Figure 1.3 shows that HIV/AIDS was the leading cause of death in the year 2002 contributing to almost 29% of all deaths.

Using estimations and projections package (EPP) and spectrum model developed by WHO it is estimated that, in year 2004, between 1.8 and 2 million people were living with HIV in Tanzania. This resulted into a cumulative total of 192,532 reported AIDS cases from 21 regions since 1983. In December 2007, Tanzania was estimated to have around 2-2.2 million people with HIV and 220,000 people with AIDS (MOH, 2007). AIDS is widespread in both urban and rural communities. HIV prevalence studies conducted by (Soderberg et al. 1994; and Killewo, 1990) analysed the situation in the two most seriously affected rural regions in Tanzania. They found a HIV prevalence of 24.2% in Bukoba and 13.6% for male as well as 15.0% for female in Mbeya. These figures are not representative for the entire population, but they give us the picture about the spread of the epidemic in the rural

communities. (MUTAN 1994) almost confirms these figures for Arusha (9.5%) and Moshi (15.1%). These studies show that the epidemic is quickly spreading all over the country and needs a quick attention. The NACP report shows that AIDS cases are mostly concentrated on a certain groups see the following figure.



Sources: NACP report, 2005.

Figure 1.4. Case rates for cumulative AIDS cases by age and sex, Tanzania, 1987-2004.

Figure 1.4 shows that most of the AIDS cases are concentrated on the age group between 15 and 59. This is the age group that contain the labour force and the biological reproductive that means the future economy and population growth of the country could be in danger. The following section briefly highlights how the government of Tanzania has responded on HIV/AIDS epidemic.

Government response on HIV and AIDS

In response to the HIV/AIDS epidemic, the Government of Tanzania, with technical support from the World Health Organization’s Global Programme on AIDS (WHO-GPA), formed the Task Force on AIDS in 1985. In 1988, National Aids Control Programme (NACP) was established under the Ministry of Health to coordinate the implementation of laid plan. Initially, HIV/AIDS was perceived purely as a health problem and the campaign to deal with it involved the health ministry only through the National AIDS Control Programme. The national response consisted of developing strategies to prevent, control and mitigate the impact of the HIV/AIDS epidemic, through health education, multi-division response and community participation.

Other studies on the HIV dynamics

A number of good studies in the academic literature have dealt either with models for the spread of the AIDS epidemic, or policy models that analyzed both the spread of the epidemic and the control of AIDS epidemic. In addition to that, a great number of mathematical models have been used for predicting future AIDS cases. These and other modeling policy studies will be discussed in the literature review chapter 2.

Because of the severity of the problem, intervention includes cooperative efforts by development partners and civil society, including the private sector, share responsibility to complement government efforts. Prompted by the work of Dangerfield *et al.*, (2001) we use a system dynamics approach to model the AIDS epidemic in Tanzania. The system dynamics modeling approach has a number of significant characteristics that differentiate it from other techniques. A key characteristic, suggested by its name, is a concern with understanding and changing behaviours that occur over time. System dynamics models make explicit the causal factors responsible for changes occurring in systems from one point in time to the next and thereby enable the behaviour of those systems to be analyzed over time.

The general goal of our thesis is to create a model of the HIV/AIDS epidemic in Tanzania, obtain insight into the dynamics of the epidemic transmission, and suggest policies for reducing rate of infections. Our model is an SD adaptation of standard diffusion models used in the study of epidemics. An SD model proposed in this thesis uses data from the NACP surveillance report (2005) and the WHO report in the case of Tanzania.

A brief outline of this thesis

Our thesis is divided into seven chapters and two appendixes. *Chapter one* has provided the epidemiological information, clinical aspects of the disease, and the historical background of the HIV/AIDS epidemic, globally and locally, include the Tanzanian policy respond.

The goal of *Chapter two* is to describe and analyze the literature on the main approaches to HIV and AIDS policy modeling. The *third chapter* introduces the system dynamics method and its contribution to understanding complex systems and policy design. *Chapter 4* presents the model and the following *chapter 5* presents the validation process and its results. In *Chapter 6*, different policies are tested and their results and impacts on the

future development of the epidemic are discussed. The paper concludes with a summary and directions are given for future research.

2. LITERATURE REVIEW

AIDS epidemic and HIV dynamics modeling can be traced back to 1980's soon after the AIDS epidemic was recognized. In AIDS epidemic modeling, both stochastic and deterministic differential or difference equations have been used to describe the person-to-person transmission and the epidemic in high risk populations as well as in the general population. Some statistical and epidemiological approaches such as back-calculation have been proposed to estimate the AIDS epidemic and AIDS management. In modeling HIV dynamics within a host, bio-mathematicians and theoretical biologists have made great advances in the development of mathematical models to study the characteristics of HIV replication, HIV evolution and control policy. System dynamics models and computer simulations have become useful in analyzing the spread and control of infectious diseases such as AIDS. They together, build and test theories that are involved with complex biological systems related disease, getting quantitative speculations, determining parameter sensitivities due to change and estimating parameters from data.

“The first and most direct approach to predicting AIDS cases in the future is extrapolation” (Morgan and Curran, 1986; Karon *et al.*, 1988, 1989). This method is to fit an assumed form of the AIDS incidence curve to the AIDS incidence data in recent years and then to extend this curve for several years as a prediction of AIDS cases in the future. This method assumes that the current trends will continue for at least a few years into the future. Often separate curves and extrapolations are done for various risk groups. Advantages of extrapolation are its simplicity and ease of use. The extrapolation method has been a good predictor of AIDS incidence for a few years into the future, but it is not good for longer forecasts since it does not consider changes in the HIV epidemic due to factors such as behavioural changes or saturation in the high risk groups. Another disadvantage is that it does not give any information on HIV incidence or any understanding of the HIV transmission mechanisms i.e. it ignores the underlying structure that is producing the behaviour of the disease. Extrapolation has been used on United Kingdom (UK) data by Healy and Tillett (1988) and on European data by Downs *et al.*, (1987). Extrapolation has not worked as well in recent years because there have been clear changes in trends with a decreasing rate of growth and the incidence has reached a plateau in some risk groups.

The second method to modeling AIDS incidence is usually called back calculation (Brookmeyer and Gail, 1986 and 1988; Gail and Brookmeyer, 1988; Brookmeyer and Damiano, 1989). Using this approach, the total number of cases of AIDS at time t is the

summation up to time t of the product of the HIV incidence at time r and the probability of developing AIDS within $t - r$ years after infection. Thus if the HIV incidence and the distribution of the AIDS incubation period were known up to time t , then the cumulative AIDS cases would be calculated in a straight-forward way using the complexity summation above. This HIV incidence up to time t and its extrapolation for a few years are then used to forecast the AIDS incidence for a few years. The distribution of the incubation period for AIDS can be estimated parametrically or non-parametrically. The back calculation procedure is often applied to separate risk groups. Back calculation has been used for forecasting AIDS incidence for a few years and does have the advantage that it also yields estimates of HIV incidence; however, there are several disadvantages. It does not yield any information on the HIV transmission dynamics or estimates of parameter values. Estimated distributions for the AIDS incubation period are uncertain and the back calculation procedure is very sensitive to the distribution used (Brookmeyer and Damiano, 1988; and Hyman and Stanley, 1988). The instability of the back calculation process implies that the confidence intervals of the estimates of HIV incidence and future AIDS incidence are very wide.

The third approach to modeling AIDS is to use HIV transmission dynamics models which include the progression to AIDS. Using this method, the population is divided into different groups. Hethcote *et al.*, (1982); and Hethcote and Yorke, (1984), used this method in studying the sexual activity levels in the population. The population considered here consists of homosexual men who change male sex partners frequently, i.e., at least once every few years. This group is subdivided into men who have many different male sex partners (very-active) and those who have only a few different partners (active). The two activity levels used here do not introduce lots of parameters which cannot be estimated and are consistent with the existence of a small fraction of homosexual men who are very active sexually. The mixing structure, sexual activity level and progression to AIDS in a population may depend on the age of individuals, but there were not enough data available to justify the incorporation of age structure into the model.

Ahlgren *et al.*, (1990), developed a dynamic transmission model and found parameter values which optimised the fit to the sero-conversion data and AIDS incidence in homosexual men in San Francisco for 1978 to 1986. Their modeling experiments suggested that the high infectivity of the short-lived, antigen bearing first stage of HIV infection may have caused the rapid rise in the early epidemic in San Francisco. But this method yields no more information on HIV transmission dynamics on other stages of the epidemic.

At the end of 1980's and early 1990's years there has been a tremendous number of modeling papers which use dynamic models of HIV transmission and progression to AIDS. Two authors who have contributed to HIV/AIDS modeling are Anderson and May (e.g., Anderson, 1988; Anderson and May, 1988 ; May, 1988); some of their modeling results are incorporated into their encyclopaedic book (Anderson and May, 1991), which is built from their numerous epidemiological papers during the past ten years. Their models have covered not only homosexual and heterosexual populations in the United States and United Kingdom, but also heterosexual populations in Africa. They have estimated reproduction numbers (contact numbers), doubling times and demographic consequences. But because of lack of good data during that time these models did not reflect the HIV transmission dynamics in sub-Saharan Africa, and in addition to that new trend of AIDS development make this model of little use.

Using the method of system dynamics, Dangerfield and Roberts, (1990), developed a model of the spread of AIDS in the UK homosexual population. The purpose of the model is to provide policy support tool. This model is not primarily intended to possess a forecasting role. Rather it examines the progress of the in an at-risk cohort of one million male homosexuals, allowing the investigator to easily compare the relative epidemiological consequences of crucial virological and behavioral aspects of the infection. The advantage of this model is that it fits the historical data well. But the disadvantage is that this model cannot be applied to the heterosexual transmission dynamics in sub-Saharan Africa because of the differences in infectivity and contact frequency.

Dangerfield *et al.*, (2001) developed another system dynamics model of HIV/AIDS epidemiology designed to simulate the effects of triple combination antiretroviral therapy in the treatment of HIV/AIDS. They used the epidemic data on homosexual men in the UK (1981-1998) to fit the baseline model. They draw a conclusion that the new combination therapies, which supplanted antiretroviral mono and dual therapy in 1996, were proving to be the most effective prophylaxis yet for halting viral replication *in vivo*, but they also indicated that many uncertainties still surround their use. They generated a range of model scenarios as a means of considering these uncertainties. This model did not pay attention on the issue of heterosexual contacts therefore is not suitable in our case study.

David and Ralph, (2002), developed a system dynamics model for the purpose of fostering a greater understanding about the psychosocial dynamics of HIV/AIDS prevention and care in the community over a twenty year time horizon, from the epidemic's inception

(circa 1981 – 2002). In particular, the psychosocial dynamics of perceived stigma, complacency, and disempowerment were studied in relation to the epidemiology of HIV/AIDS in Michigan. The study was informed by the results of an extensive qualitative research project that explored the current and emerging needs of persons living with HIV/AIDS (PLWHA) and by the insight and knowledge of a group of ten core key informants from Michigan's HIV community. They concluded that the process of modeling for understanding successfully generated an explicit picture of the dynamic complexity of the psychosocial context of HIV/AIDS prevention and care, opened a common space for the candid exchange of ideas about what can and ought to be done about it, and increased the potential of the community to work together in the future in a manner of enlightened collective action. Again this model was purely focused on the homosexual contacts.

Booz Allen, (2005), in partnership with leading academics from Brown, Emory, and Wayne State Universities, developed an integrated System Dynamics model that combined disease epidemiology and economics together with a variety of policy and program options. The model was based on peer-reviewed scientific literature and its predictions were validated against prevalence and economic data from India between 1987 and 2003. This model was designed to cultivate an understanding of both the epidemiological and economic dynamics of HIV/AIDS in India. Additionally, the strategic simulation acted as a catalyst, bringing together the most appropriate national and international government, non-government and business stakeholders for India's social, political and economic future. The HIV/AIDS model has two key modules: the disease progression module and the economic module, but the approach consists of a collection of integrated analytic solutions. The strategic simulation held in India was not intended to be a predictor of the future of the AIDS epidemic in India, but an analysis of potential scenarios that could occur based on actions taken in the present.

Only few policy models have been developed for low-income countries, although these countries (refer to Table 1.1) are most severely affected by this pandemic. Until the end of year 1990, there were only two models to examine the likely trends in HIV infection and AIDS prevalence in Tanzania. The models generally are based on data from the middle and late 1980s (World Bank, 1992). One model, prepared by Rodolfo Bulatao (1990), simulates two alternative scenarios—one involving a low degree of mutual monogamy in the society (an assumption that 15 percent of all married couples are mutually faithful) and the one involving a high degree of mutual monogamy (an assumption that 45 percent of couples are faithful). The two scenarios share estimates of incidence of specific risk behaviour, such as rate of

partner change, frequency of blood transfusions and proportion of the population engaged in prostitution.

Another model, developed by James Chin and F. Sonnenberg (1990), uses current data (1990 data) on HIV infection rates to estimate the number of past AIDS cases and the near term future growth in the HIV infection and AIDS. The two models projected the future impact of HIV/AIDS for 10 years that means until year 2000. We believe this time is not long enough for the epidemic which such a long incubating time as HIV/AIDS but could save for short term purpose.

In 1993, Heidenberger and Flessa developed the first system dynamics model for AIDS epidemic in Tanzania mainland (Heidenberger and Flessa, 1993). The purpose of this model was to provide policy support to the Lutheran Church, one of the main providers of health care in Tanzania mainland. This model assumed two policies; condom and vaccine or a suppressor, the later was not available yet. This model had to be revised by Flessa in 1996 in order to supply an answer to the contemporary planning needs of the church health care in Tanzania (Flessa, 1996). Data used in this model were obtained by the hospitals run by Lutheran church. One of the issues discussed in the revised model is the question of whether the spread of AIDS could be efficiently restrained by the use of condoms whereby this discussion in the church is mainly a moral topic. Today, the use of condoms still is a critical discussion in the churches of Tanzania. Moral issue made it difficult to formulate and apply the condom policy. These models did not consider the use of advanced therapies as there were not available to the general public. Flessa argued further that *“The fact that the simulation of 1989, based on 1988 data, gave an acceptable forecast for the years 1989 to 1994 encourages us to believe that the estimates by our enriched model of 1994 will be valid until the year 2000 and will present a proper foundation of decision making for the churches of Tanzania”*. In 2003, Flessa developed another SD model which allows assessing the impact of different interventions on a pattern population in Eastern Africa (Uganda, Kenya and Tanzania). The model predicts the spread of AIDS only in a highly-epidemic region of Eastern Africa and in Tanzania, the model covered only three areas (Mbeya region, Umbwe-Kilimanjaro region and Songea-Ruvuma region). However, the three regions are not representative for the entire population, and, consequently, should not be the basis of planning and decision making and the reliability of these data is very low, as laboratory procedures are commonly of low quality Flessa (2003).

In 2004, UNAIDS/WHO released a mathematical model for AIDS epidemic in Tanzania (NACP, 2005). The model consists of two computer software; the estimations and projections package (EPP) and the SPECTRUM. The Estimations and Projections Package (EPP 2005) for HIV/AIDS estimates and projects HIV prevalence, number of people living with HIV and new HIV infections and AIDS cases using antenatal clinic (ANC) surveillance data. The prevalence projection produced by EPP can be transferred to SPECTRUM, a demographic projection model, to calculate the number of AIDS deaths. This model does indicate neither the impact of different policies nor the complex biological and behavioural traits of the epidemic. The choice of parameters in this model (EPP) does not allow it to deal with issues such as behavioural change in response to interventions (for example increased condom use, the use of HAART and nevirapine for HIV-positive children, and likely changes in the size of the at-risk population as a result of vaccine introduction).

Summary:

In this chapter we have reviewed literature showing different methods of modeling AIDS epidemic and HIV transmission dynamics. Different methods have been used such as analytical, numerical and computer simulation models including those based on system dynamics. For example some mathematical models involve different approaches such as direct approach and back calculation. There are several advantages associated with these models which includes; simplicity and easy to use, ability to forecast future HIV incidence and ability to fit the historical data. These models have some disadvantages too such as inability to forecast longer term HIV incidence, most of these models are designed for the situation in the Europe and the US, and they ignores the underlying structure that produces the behaviour and do not yield any information about the HIV transmission. Computer simulation models have become popular in recent years and they have several advantages; they exhibit the causal relationship that exists between the structure and its behaviour. Population can be disaggregated into several categories that represent different modes of HIV/AIDS transmission, and dynamics of HIV transmission is clearly described. Unlike other models, computer simulation models are able to forecast for longer term. But most of the existing computer simulation models are related to the situation in the Europe and the US.

The system dynamics model in this thesis focused on the epidemic situation in Tanzania one of the developing countries. The main mode of transmission in this country is heterosexual and mother-to-child transmission unlike many models in the literature where the main modes of transmission were homosexuals and intravenous drug users. The major

emphasis in this thesis is to obtain data from the NACP Tanzania and from multiple sources and then to see if all of these parameters are consistent in a simulation model with estimated HIV and AIDS incidences. The available data on the NACP report has only been used to project the future cases until year 2010. The current available model (EPP) has some disadvantages which include failure to indicate the impact of different policies in future, failure to establish the dynamics transmission of the epidemic and has limited choice of parameters. This model aimed only to forecast the future trend of the epidemic until year 2010. Thus, our goal is to provide HIV/AIDS forecasts until year 2050 and to provide a dynamic simulation model with parameter values which reconstructs what has occurred up to the year 2004 and be able to test the impact of different policies. When interpreting modeling results, one must be aware of the approximations involved in the formulation of the model.

In the next chapter we present our research method applied in this thesis and explicitly show how we believe this method is relevant to the problem.

3. RESEARCH METHOD

In this thesis, we focus on the HIV/AIDS epidemic situation in Tanzania. We develop a system dynamics (SD) model to replicate the historical data and design policies. This study applies a ‘system dynamics approach’, a research methodology explicitly intended to promote in-depth learning about dynamically complex problems (Ford, 1999; Sterman, 1994 & 2000). The system dynamics modeling technique has been extensively applied to modeling the HIV/AIDS epidemic before (see also the work of Heidenberger and Flessa, 1993; Brandeau *et al.*, 1990; Hethcote and Van, 1992; Hethcote and Van, 1992; Heidenberger and Roth, 1998; Bernstein, *et al.*, 1998; Flessa, 1995 and 2003; and Dangerfield, *et al.*, 2001) and for more literature refer to the literature review chapter 2.

The model covers the historical period (circa 1980 to 2004) and project to 2050. A system dynamics HIV/AIDS model like all models is not designed to predict the future with certainty but as a policy tool which is used as a component in the ongoing debate about the better control of the epidemic. The model is also used as a device for obtaining an increased understanding of HIV transmission dynamics and as a way of judging the effectiveness of various intervention strategies. As Sterman argued, system dynamics models reduce the probability that policy-makers will be surprised by unanticipated delays or other “unanticipated” events and permit more adequate preparation (Sterman, 2000). In addition, our simulation model enables policy makers to test different assumptions, explore potential scenarios, and examine impact of their decisions in an effective way.

Like most policy models, this type of model requires large amount of data, and several assumptions especially when applied to a developing country with limited data like Tanzania. Using his experience from working in Tanzania, Flessa argued that the “reliability of data is very low, as laboratory procedures are commonly of low quality” (Flessa, 2003). Therefore, lack of data may be the obstacle, but reasonable assumptions are considered to fill the gap. “No matter how many resources one has, one can envision a complex enough model to render resources insufficient to the task” (Donella Meadows *et al.*, 1982 p.197). The rest of this chapter goes by presenting the system dynamics method and its contribution to the understanding of complex systems and policy design in these systems.

Why is system dynamics applicable to the problem?

The HIV/AIDS epidemic is complex. This may explain the disappointing results of policies to prevent the new infections. From an SD point of view, there are three drivers of dynamic complexity in the HIV/AIDS model systems: (1) presence of feedback loops; (2) time delays between the cause and effect of an action, and (3) existence of non-linear relationships among the system's elements. In this context, system dynamics “provides a strategic tool that can be used to predict the spread of epidemic and effectiveness of general prevention and treatment programs targeted to either individuals or the entire population” (Sterman, 2000).

System dynamics simulation is now used routinely throughout the natural and social sciences, hailed as a “third branch of science”, standing alongside theory and experiment as a unique and vital method to advance human knowledge (Pool, 1992). Jay Forrester emphasizes in one of his papers “If the model is a good representation of an actual situation, then it becomes the theory of how that part of the real world operates” (J. Forrester, 1968).

System dynamics models in general “involve set of simultaneous nonlinear differential equations and these relationships are notoriously difficult to handle mathematically” (Donella Meadows, 1980). We will use the main building blocks of system dynamics to emphasise its usefulness.

Building blocks of system dynamics

Stock and flow

Stock and flows are the main building blocks of system dynamics models. A simple, everyday illustration of stocks and flows is human population as stock and birth as flow. Sterman, (2000, p.192) described stocks, as “accumulations, that represent the state of the system and generate the information upon which the decisions and actions are based”. In other words stock accumulates the difference between the inflow to a process and its outflow. Figure 3.1 shows that the stock of Human Population increases with the inflow of human births and decreases with the outflow of human deaths the same applied to the stock of Bathtub which increases with the inflow of water and decreases with the outflow of water.

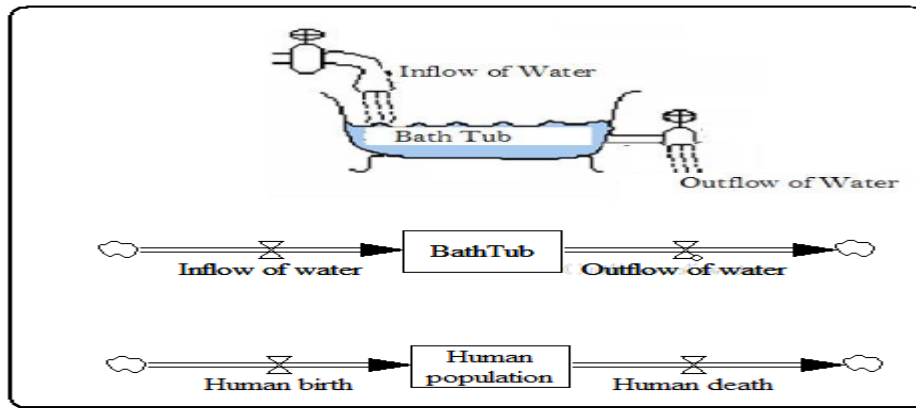


Figure 3.1: The stock and flow diagram using bathtub analogy to represent model of human population growth

Figure 3.1 shows example of two types of variables:

- Stock – the bathtub and human population
- Flow - the inflow of water and human births/Outflow of water and human death

The double line represents material flow – in this case, the flow of human from a cloud into the stock. The cloud represents the infinite source of human births. A cloud may also be viewed as a stock that is outside the system boundary, so we don't bother to keep track of it at least for the moment. The cloud on the left represents a source; the cloud on the right is an infinite sink. We may interpret figure 3.1 to mean that this model of population does not represent where people come from when they are born or where they go when they die. In this case, stocks are also critical in generating the dynamics of systems because they provide systems with inertia and memory, being the source of delays and creating disequilibrium dynamics by decoupling rates of flow (Mass 1980, *cited in Sterman 2000*). Delays will be described later in this chapter. In addition to that, the next chapter 4.0 demonstrate how the two building blocks (stock and flow) could be applied to represent the case in our system model.

Feedback loop

“The feedback loop is circle of interactions, a closed loop of action and information” (Richardson, 1999). The patterns of behaviour of any two variables in such a closed loop are linked, each influencing, and in turn responding to, the behaviour of each other. This circular relationship, which indicates that an influence is both a cause and an effect, is known as

“feedback” and lies at the heart of system dynamics approach (Sterman, 1994, 2000, & 2001; Senge, 1990; and Forrester, 1961).

All endogenous dynamics arise from the interaction of just two types of feedback loops, positive (contagion or self-reinforcing) and negative (balancing or self-correcting) loop. A causal loop that characteristically tends to reinforce or amplify a change in any one of its element is called a positive loop. Arrows in causal loop diagrams are labelled + or – depending on whether the causal influence is positive or negative. We use the + to represent a cause-and-effect relationship in which the two variables change in the same direction. In a positive loop, an increase in an element X feeds around the loop and tends to cause X to increase still further; likewise a decrease in X tends to cause X to decrease still further.

In contrast, a causal loop that characteristically tends to diminish or counteract a change in any one of its elements is called a negative loop. In a negative loop, an increase in X feeds around the loop and tends to cause X to slow or reverse its increase. The motivation for the positive and negative labels comes from the way loop polarities can be obtained from the polarities of the individual causal links that combine to form the loop. The “arithmetic” of causal links parallels the arithmetic of multiplying signed numbers. To establish the parallelism, define a causal influence from X to Y to be positive if a change in X tends to produce an increase in Y, a decrease in X tends to produce a decrease in Y. Similarly, define a causal influence from X to Y to be negative if a change in X tends to produce a change a decrease in Y, a decrease in X tends to produce an increase in Y. It is then easy to argue that the polarity of causal loop is the product of the polarities of its links. Although it is easy to infer the behaviour of each of these loops in isolation, if a system includes many interacting feedback loops, as is often the case, it becomes impossible to predict how the system will behave by merely examining the diagram of loops. In fact, dynamics observed in systems often arise from shifts in loop dominance as the system evolves over time (Ford, 1999; Richardson, 1995). Numerical simulation is necessary to confirm the net effects of the various loops in the model.

The easiest way to explain the concept of feedback is with an example. Figure 3.2 shows a human population stock that is fed by the flow of human births and drained by the flow of human deaths.

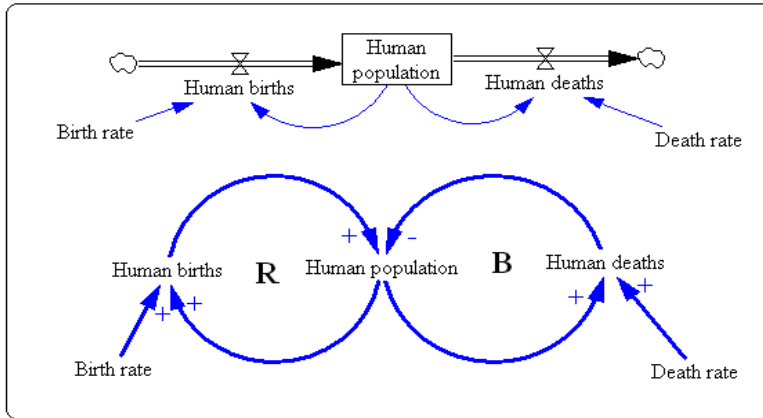


Figure 3.2. Flow diagram and corresponding causal loop diagram

The causal loop diagram shows two feedback loops. The loop on the left is the positive (or self-reinforcing) and we label positive feedback loops with **R** in the middle of the loop. Positive feedback in Figure 3.2 shows a closed chain of cause and effect in which a higher human population leads to more human births, and more human births lead to still higher human population in the future. Arrows from human births to the human population and from human population to the human births are all labelled with a positive polarity. The loop on the right shows the causal links between human deaths and human population. This means the human deaths will reduce the size of the human population. The arrow from human deaths to the human population is labelled with a – to stand for negative polarity. This means the human deaths will reduce the size of the population. The arrow from the human population to the human deaths is a positive arrow. It stands for the fact that a larger human population will tend to have a greater number of deaths (given a fixed value of the death rate). The closed chain of cause and effect is labelled with a **B** to stand for negative feedback (or self-correcting).

Delay

“A delay is a process whose output, or result, falls behind its input in some way” (Sterman, 1989 & 2000 p.411; and Scott, 1993). Commonly, it is assumed that an action immediately follows its trigger. However, in reality, causes and effects are often not close in time and space (Sterman 2000; and Sengupta *et al.*, 1999). These delays make systems more “dynamically complex as they slow the learning process by reducing the ability to accumulate experience, test hypotheses, and apply findings to intervene to improve a

particular situation” (Sterman, 2000). Further, if consequences of actions are not immediately apparent, agents will continue to take actions to make the system converge to a desired state without giving it the necessary time to absorb the effects of these actions and respond adequately. The result may be an oscillating behavior in which systems either overshoot or lag behind their equilibrium. This behavior becomes even more dramatic in situations where some delays are “unobservable”: a context in which effective decision-making based on intuition or experience becomes an elusive goal. As pointed out by Sengupta *et al.*, (1999), “delays constitute one of the most important characteristics of dynamic tasks, and the ability to handle them is essential for effective performance in such environments”. See the example below.

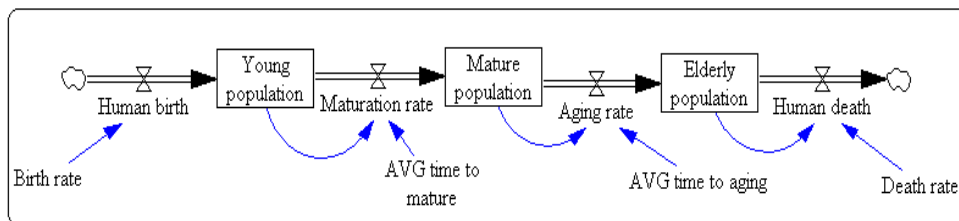


Figure 3.3: Simple population model with time delay between stocks

An example in Figure 3.3 shows a combination of transfer rate and average time intervals used in a simple human population model. There are three stocks in this diagram, young population, mature population and elderly population. The time intervals are the average time to mature and average time to aging. These inputs control the flow of people from one stock to the next within the system. This means that there is a delay from one stock to another and the input to the delay is the people who need to mature and to age. Another good example of delay is people contracting a disease say HIV may not immediately show its symptoms. The input to the delay is the people becoming infected. The result, or output, is the rate at which people are transforming into another stage (stock) of the disease say AIDS. In between these two delays is the stock of HIV people that need to be filled in the recruitment process. The resulting delay between becoming infected and showing symptoms can be related to the first order material delay or any higher order depending on the type of the system.

Nonlinear relationship

“Non-linearities” are considered important in explaining system behavior because they are source of dynamic complexity in the system. This source of dynamic complexity means that the response (effect) of the system to an action (cause) is not always linearly proportional. The presence of such relationships in a system increases dynamic complexity because the response of the system to a disturbance will be different, as it will depend on its current state. The same action may trigger completely unpredictable consequences, as the response of the system is contingent upon the current balance of power among its feedback loops. Non-linear relationships may enable an action to become the trigger of a shift in dominance from one loop to another, which exacerbates the frequency of changes of power among the system’s feedback loops, hence increasing its dynamic complexity.

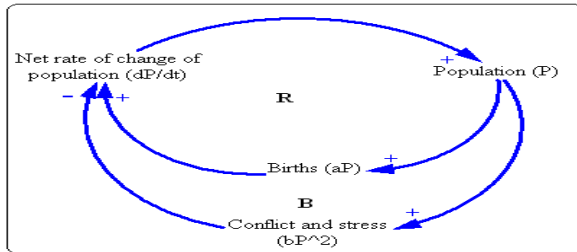
“Non-linear relationships can cause feedback loops to vary in strength, depending on the state of the rest of the system” (Medows D.H, and Robinson, 1985; Ford, 1999; and Sterman 1989 & 2000). Linked non-linear feedback loops thus form patterns of shifting loop dominance - under some conditions one part of the system is very active, and under other conditions another set of relationships takes control and shifts the entire system behavior. A model composed of several feedback loops linked non-linearly can produce a wide variety of complex behavior patterns, and can represent an evolving or adapting system structure. “Every system initially exhibiting exponential growth will eventually approach the carrying capacity of its environment, whether that is food supply for a population of moose, the number of people susceptible to infection by a virus, or the potential market for a new product” (Sterman, 2000 p.285). As the system approaches its limits to growth, it goes through a nonlinear transition from a regime where positive feedback dominates to a regime where negative feedback dominates. The result is often a smooth transition from exponential growth to equilibrium, which is S-shaped growth.

The logistic population equation of P.F. Verhulst (1838), cited also in Richardson (1999, p.32) provides an instructive population example of the non-linearity concept embedded in differential equations applied to societal phenomena.

$$dP/dt = aP - bP^2.$$

The term aP in this equation represents the tendency of population to grow at a rate proportional to the size of the population, the standard assumption of exponential growth.

Verhulst intended the term involving the square of population to represent conflict and stress arising from contacts between people, which he assumed would be roughly proportional to P times P . The result is the familiar nonlinear logistic equation, which exhibits sigmoid growth.



Source: Richardson, (1999 p.33)

Figure 3.4: Feedback loop structure of the Verhulst population equation

Figure 3.4 shows a representation of the equation above as a pair of feedback loops. The positive loop **R** corresponds to the tendency of the population to grow at a rate proportional to itself. The negative loop **B** corresponds to the growth-limiting effects Verhulst envisioned in conflict and stress. For low levels of population the growth effects predominate, and the population would appear to exhibit essentially unrestricted exponential growth. The term bP^2 representing conflict and stress would grow more rapidly than aP , however.

The initial purpose for our model is to explain the dynamics of problematic behaviour associated with AIDS epidemic and the ultimate purpose is to identify policy variables or structural elements by which we may modify that behaviour. System dynamics enable us to explicitly identify the presence of feedback loops, delays coupled with the presence of interconnected feedback loops, and non-linear relationships. In the next chapter we start the model building process.

4. MODEL BUILDING

In the previous chapters, we have presented the motivation for this thesis, the related literature helping us to build our case, and the method we employed in our case. In this chapter, a connection is established between structures and their dynamic behaviour and this is important in terms of generating insights about the situation. First, we present the main variables and time horizon of the model and then followed by epidemiological information in form of reference mode and the assumption it is based on. We continue with a basic model overview and main feedback loops meant to help the reader grasp the basic dynamics involved in the HIV transmission. We will continue with the model building process until we are able to replicate the reference behaviour. Finally we describe and discuss the details of the last model.

Key variables

The two main key variables in this study are the number of people living with HIV and AIDS, termed as “stock of HIV population” and “stock of AIDS population”. Despite the massive effort and careful work of the National AIDS Control Programme in Tanzania, the data for HIV and AIDS are highly uncertain. In addition, the definitions of AIDS have changed over the years as understanding of the disease has improved. Previously, having AIDS was defined as having HIV infection and getting one of the additional diseases. Now it is additionally defined as a CD4+ count below 200, even without an opportunistic infection (WHO, 2003). That means initially there were some infected people who were placed in a wrong group of either HIV or AIDS. This assumption is reasonable if we consider the real situation in Tanzania. Therefore, the available data on the NACP report cannot be considered as the exactly trend of HIV and AIDS cases in the country rather as an indication of the general behaviour of the growing problem. The reference mode for the AIDS population will be displayed using available data on the NACP Surveillance Reports in Tanzania 2005, and WHO report in 2005 and the hypothesized reference mode for HIV population will be based on the estimated percentage of the population assumed to be living with HIV overtime.

Time Horizon

The time horizon for the reference model is set at 70 years (from 1980 to 2050). “The time horizon should extend far back in history to show how the problem emerged and describe its symptoms” (Sterman, 2000 p.90), and “it should also extend far enough into

future to capture the delayed and indirect effects of potential policies” (Ford, 1999 p.172). The choice of time horizon influences our perception of the problem. In our study case, the first three cases of AIDS in the country were diagnosed in 1983 and the choice of year 2050 is reasonably far enough to capture delayed and indirect effects of potential policies.

Reference Mode

Reference mode in this work describes the HIV/AIDS problem through a set of graphs showing how it develops over time. So far drawing the reference mode has been identified as the best way to be sure about the nature of the dynamic problem because the problem is characterised dynamically, showing how the problem arose and how it might evolve in the future (Ford, 1999; Sterman, 2000). As Sterman (2000) highlight in his book, we will “refer back to the reference mode throughout the modeling process”.

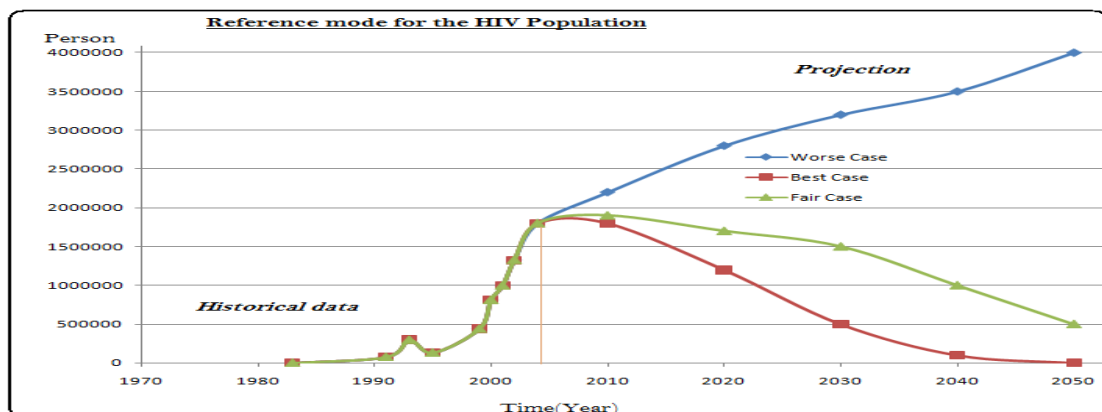


Figure 4.1a. Hypothesized Reference Mode for the HIV infected population

The hypothesized reference mode for the HIV infected population in Figure 4.1a is based on the estimated percentage of people believed to be living with HIV in the country overtime (NACP, 2005). Figure 4.1a shows that in the year 1983 the estimated number of people living with the HIV virus were 725 or 0.003% of the total population, in the year 1993 it increases to 449,561 or 2% of the total population, and in the year 2004 the estimate increased to about 1.8 million or 5% of total population (NACP, 2005; WHO, 2005). Figure 4.1a also shows three possible trajectories of the future scenarios; the worse case predicts that HIV infected population will grow to reach 4 million by the year 2050, the fair case predicts that HIV infected population will fall to below 500,000 by the year 2050, and the best case predicts that HIV infected population will fall to zero before the year 2050.

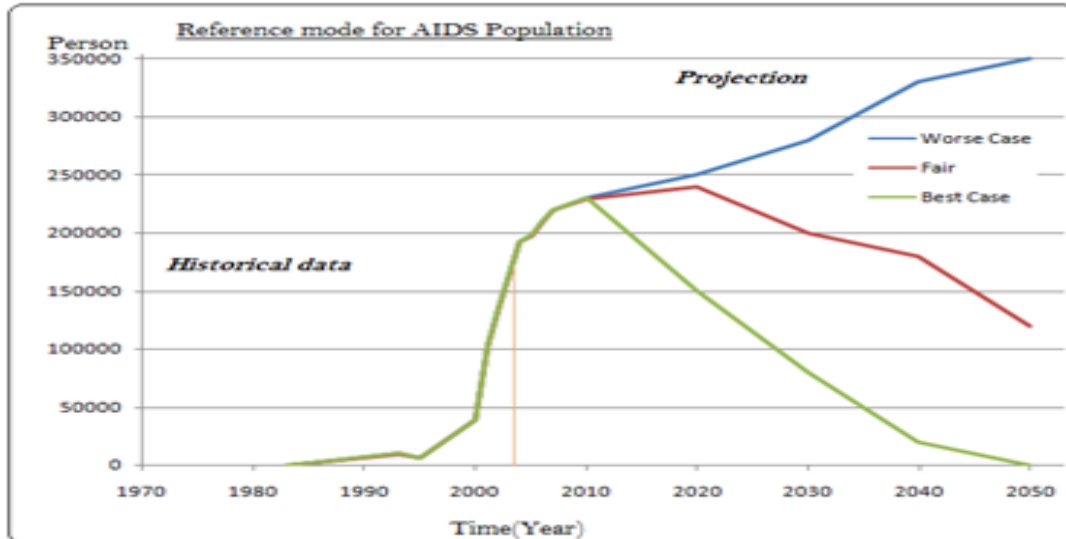


Figure 4.1b. Reference Mode for AIDS population

Figure 4.1b is generated using available data from the Surveillance Report 2005 in Tanzania and TACAIDS report 2005, where the first 3 cases of AIDS in the country were diagnosed in 1983. Figure 4.1b shows that in the period between 1983 and 1986 few cases were reported in the country. This is because the epidemic was not well known (NACP, 2005). Between year 1991 and 1993 many cases were reported to the NACP. Overall, there has been a large increase in the number of reported cases from 1983 – 2004 for AIDS. In year 2004 a total of 192,532 AIDS cases were reported to NACP Tanzania. Figure 4.1b also shows three possible trajectories of the future scenarios; the worse case predicts that AIDS population will grow to reach 350,000 by the year 2050, the fair case predicts that AIDS population will fall to below 150,000 by the year 2050, and the best case predicts that AIDS population will fall to zero by the year 2050.

In the two figures above 4.1a and 4.1b, there was an increase in the number of reported cases for both HIV and AIDS between 1990 and 1993. This peak just reflects aggressive data collection during this period and does not represent a peak increase in the infection rate (NACP, 2005).

Dynamic Hypothesis

In this section, we present a hypothesis regarding the structure responsible for generating the behavior graphed in the reference mode. (Figure 4.1a and 4.1b). We begin developing the dynamic hypothesis simply by thinking about the behavior in Figure 4.1a. The reference mode for the HIV infected population in Figure 4.1a shows the exponential-like growth and we assume that the infection rate moves people from the susceptible population into the HIV population, that is, people who are infected with the disease but do not yet exhibit any symptoms. The reference mode for the AIDS population in figure 4.1b shows the also the exponential-like growth, we assume that after the incubation period, people begin to exhibit symptoms (typically while remaining infectious) and move into the AIDS population category. We assume that the rate at which people exhibit symptoms is a first order delay process with a constant average incubation period. The simplified causal loop diagram for the reference mode in Figure 4.1a and 4.1b is shown below

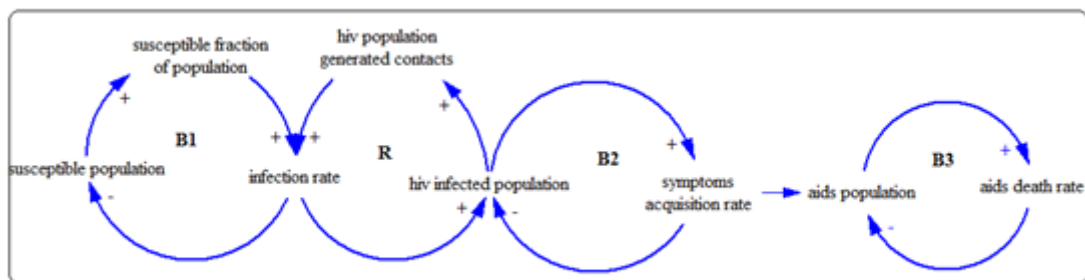


Figure 4.2. Simplified Causal Loop Diagram.

This simplified causal loop in Figure 4.2 above highlights the main feedback loops relevant in the system. There are four loops on Figure 4.2; one contagion (R) and three depletion loops (B1, B2 and B3). Normally, the relative strength of the two loops (Contagion-R and Depletion-B1) will not be constant during the course of the epidemic: particularly important is the switch in dominance between the two loops which causes the system, under normal conditions, to produce the typical S-shaped behaviour in the total number of HIV infected population. At the beginning of the epidemic the reinforcing loop will dominate, driving an exponential growth in the number of infected people. In the mean time, the balancing loop will become stronger and stronger while subjects are moved from the susceptible to the infected population, and it will finally become dominant. The cumulative

number of HIV infected population follows an S-shaped curve while the rate at which new cases occurs rises exponentially, peaks, then falls as the epidemic ends.

CLD representing the HIV infected population (Figure 4.1a)

In Figure 4.2 above the only reinforcing loop is the “contagion” loop **R**, which highlights the idea that if the number of the HIV infected population increases, assuming that the infectivity and the number of risky contacts each person is able to generate remains constant, the total number of risky contacts generated by HIV infected population will increase, that *ceteris paribus*, would increase the infection rate, that would increase again the number of the HIV infected population, this is the process represented by loop **R**. *This loop could be associated with the behavior we have seen in the reference mode Figure 4.1a where the number of HIV infected population seems to be increasing in the exponential-like growth from year 1983 through 2004.* However, as more people get infected, the number of subjects in the susceptible population stock will decrease, therefore, susceptible population increases the susceptible fraction of population that increases the infection rate while in turn the infection rate reduces the number of the susceptible population and this is the effect described by the susceptible population “depletion” loop **B1**. *However, the effect of loop B1 cannot be observed clearly because in the Figure 4.1a, loop R is stronger and the number of HIV infected population keep increasing in the exponential-like growth.*

CLD representing the AIDS population (Figure 4.1b)

When the HIV infected population increases, with a certain delay (incubation period) the number of people acquiring AIDS symptoms will increase and in turn this will lower the HIV infected population. This process reveals another important *negative loop B2* in the model. As the population of people living with AIDS increases so does the number of people dying from AIDS which depends also on the average duration of AIDS and in turn, this lower the AIDS population. This process reveals the last *negative loop B3* in this simplified representation of the system. *This loop could be associated with the behavior we have seen in the Figure 4.1b where the number of AIDS population seems to be increasing in the exponential-like growth from year 1983 through 2004.* That means the number of moving into AIDS population is higher than people dying from AIDS.

The net effect of these loops could be responsible for the behavior in the Figure 4.1a and 4.1b and this is our dynamic hypothesis. The description of the stock-and-flow version begins in the next section.

Model Building

In this part of chapter four, we present a system dynamics model that we hypothesize is responsible for the historical behaviour pattern. To create the model of the HIV/AIDS epidemic, we have based the analysis upon a classic example of the common epidemic model, (SIR model of epidemiology) which is used in varying guises to study the epidemiological theories (W. O. Kermack and A. G. McKendrick, 1927, cited also in Sterman, 2000).

The SIR model of epidemiology is one of the simplest models of infectious diseases. The SIR model is a nonlinear model which considers three classes of people in a population. This model is called SIR for Susceptible - Infected – Recovered (e.g., chicken pox). As a variant on this title SIR can also stand for Susceptible - Infected - Removed for example, HIV/AIDS, where individuals die from contracting the disease. In order to keep the initial process of building the model simple, we will start by concentrating on the two variables the SI model (Susceptible and Infected) and we keep the population constant across time. And, later on, we will be able to directly extend the model to one of a changing population (with births and deaths) without much difficulty. The following Table 4.1 summarizes our model building process and the main variables involved.

Model Building Overview

Models	Type of model	Main Variables Involved
Model 1	SI Model	Susceptible Population (S), HIV infected population (S), Infection rate , Contact frequency, Infectivity, susceptible fraction of population
Model 2	SIR Model	All variables in “Model 1” + AIDS Population, symptoms acquisition rate , Incubation period
Model 3	Modified SIR Model	All variables in “Model 2” + AIDS death rate , Average duration of AIDS

Model 4	Modified SIR Model	All variables in “Model 3” + birth rate , inflow to susceptible population, Susceptible normal death rate , HIV normal death rate
Model 5	Modified SIR Model	All variables in “Model 4” + HIV-positive children
Model 6	Modified SIR Model	All variables in “Model 5” + effect of HIV education and awareness program

Table 4.1: Model building overview and main variables involved

To illustrate, Table 4.1 shows list of variables and different models designed to study the HIV/AIDS epidemic in Tanzania. The variables we have decided to include in the model are those that we consider to be vital for the behavior we have seen in the reference mode section at the beginning of this chapter (Figure 4.1a and Figure 4.1b). Sterman (2000) emphasizes that a broad model boundary that is able to capture all important feedbacks loops is more important than a model with a lot of details in the specification of individual components.

From Table 4.1 above the susceptible population, HIV infected population, AIDS population, infection rate, inflow to susceptible population, AIDS symptoms rate, AIDS deaths rate and HIV-positive children birth rate are variables considered to be endogenous variables in the “Model 5”. The first two models (“Model 1” and “Model 2”) consider the fixed population with no births and deaths taking place. The third model (“Model 3”) assumes that people living with AIDS will finally die after average time with AIDS. The “Model 4” includes births and non AIDS deaths, and we assume further that fractional birth rate is exogenous to the model because our model does not deal with the dynamics of the whole population growth, but the HIV transmission dynamics. The average duration of AIDS, total population, incubation period, infectivity HIV contact frequency and HIV education and awareness are the variables considered to be exogenous to the Model 6. The normal death rates are considered to be exogenous to the model as well. In fact their estimation is based on the fractional death rate of the particular stock. The model does not separate the population between rural and urban. This chapter will explain the endogenous

variables and the structure of their relationships. We will begin our model building process by describing the dynamics of the two stocks in an SI model based on Sterman's formulation (Sterman, 2000).

Model 1: An SI Model

In this section we consider an SI model of the spread of a disease. This model illustrates a common method of interaction of two populations (Susceptible and Infected). We consider the spread of a disease in a population using the original formulation and later the modified formulation. To develop Model 1, we initially make the following assumptions, which can be relaxed later:

- 1) The size of the population remains fixed at any time; births, deaths and migration are ignored or, births plus in-migration exactly equal deaths plus out-migration.
- 2) We consider just two classes of individuals: those who have the disease and are infectious, and those who do not have the disease but are susceptible to it. Each individual is in one of these two classes. (In this model, no one is immune, and once an individual has the disease, the individual remains infectious.)
- 3) We assume that the disease spreads through interactions between pairs of individuals. If one is susceptible and one is infected, the susceptible might become infected.
- 4) The population is assumed to be homogenous: all members of the community are assumed to interact at the same amount (there are no groups that remain isolated from the community or whose behavior is different from others).

The "Model 1" is based on the assumption that the infected population is increased by the infection rate while the susceptible population is decreased by it. The dynamics of the system is represented by the infection rate.

$$I = \text{INTEGRAL} (IR, I_0)$$

$$S = \text{INTEGRAL} (-IR, N - I_0)$$

Where N is the total population in the community and I_0 is the initial number of infectious population (a small number or even a single individual). To formulate the infection rate, consider the process by which susceptible population become infected.

People in the community interact at a certain rate (the Contact frequency rate, c , measured in people contacted per person per time period, or $1/\text{time period}$). Thus the susceptible population generate S_c encounters per time period. Some of these encounters are with infectious people. If infected population interact at the same rate as susceptible population (they are not quarantined or confined), then the probability that any randomly selected encounter is an encounter with an infected individual is I/N . The infectivity, i , of the disease is the probability that a person becomes infected after contact with an infectious person. Not every encounter with an infected person results in infection. The infectivity, i , of the disease is the probability that a person becomes infected after contact with an infectious person. The infection rate is therefore the total number of encounters S_c multiplied by the probability that any of those encounters is with an infected person I/N multiplied by the probability that an encounter with an infected person results in infection:

$$IR = (ciS)(I/N)$$

Note that dynamics can be determined by noting that without births, deaths, or migration, the total population is fixed:

$$S + I = N$$

Though the system contains two stocks, it is actually a first-order system because one of the stocks is completely determined by the other. Substituting $N-I$ for S in $IR = (ciS)(I/N)$ yields $IR = (c)(i)(1-I/N)$. Sterman's formulation structure (Sterman, 2000) is shown below.

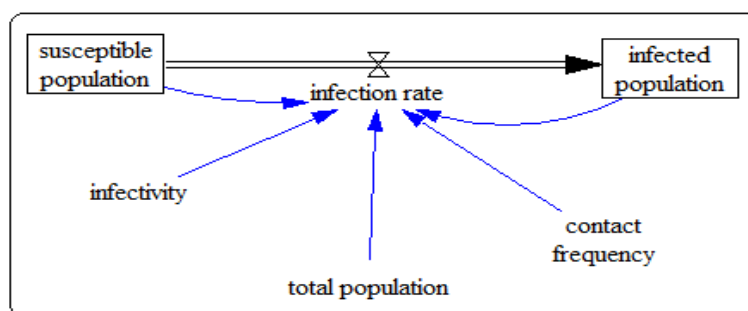


Figure 4.3 Sterman's formulation structure in chapter 9.

There are two stocks and one flow in the Figure 4.3, stocks of susceptible and infected population and a flow of infection rate. People move from the stock of susceptible population to the stock of infected population through a flow of infection rate; it takes both a susceptible and an infected person to generate a new infection. Sterman’s formulation structure shows clearly the two “policy parameters” (contact frequency and infectivity) in the system that might be reformulated as endogenous policy equations. However the structure above does not encourage a visual image of the process that generates infections. In the following section we will attempt to improve the Sterman version.

The figure below shows our attempt to improve the Sterman version, where the structure describes the process that generates infection more clearly.

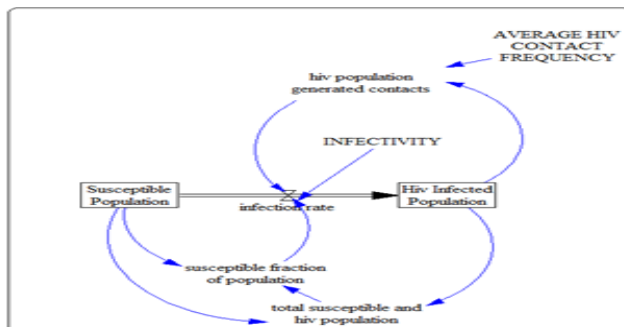


Figure 4.4. Modified structure of Model 1.

There are two stocks and one flow in the Figure 4.4 similar to Sterman’s formulation, stocks of susceptible and HIV infected population and a flow of infection rate. The infection rate in Figure 4.4 above is therefore the HIV population generated contacts multiplied by the probability of infection if contacts (infectivity) multiplied by the susceptible fraction of population. Susceptible fraction of population equals susceptible population divided by total susceptible and HIV infected population and HIV population generated contacts equals to HIV infected population multiplied by the average HIV contact frequency.

The following Figure 4.5 shows the causal loop diagram of the Model 1.

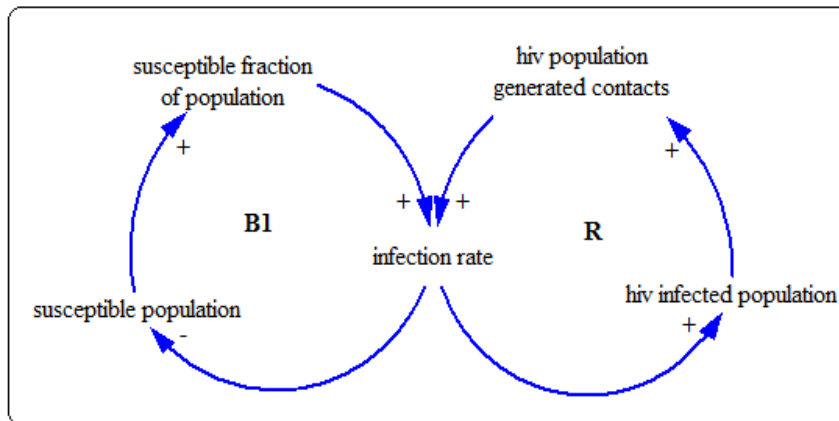


Figure 4.5. Basic Causal Loop Diagram of Model 1.

In Figure 4.5 the causal loop diagram shows two feedback loops, the positive Contagion loop **R** and the negative Depletion loop **B1**. Note that loop B1 and R in Figure 4.5 correspond to loop B1 and R in Figure 4.2 which describes our main hypothesis in this study. Having seen the structure of the Model 1 and its corresponding causal loop diagram, we will now move on to its corresponding behaviour.

Behavioural pattern of the Model 1.

The main mode of transmission in Tanzania is through heterosexual contacts, therefore susceptible and HIV infected population in our model are based on the age group between 15 and 59. This is the age group we consider to be sexually active in the population. To see the behavior of Model 1 and be able to compare with the reference mode behavior, we will now fit the model with real data from our study case, Tanzania.

Susceptible and Infected population:

In 1983, Tanzania had a total population of circa 19,730,000 people according to the National Bureau of Statistics, Tanzania (NBS, 2005). Our main focus is on the age group between 15 and 59 which is estimated to be 47% of the total population. Susceptible population in our model is therefore equals to 47% of 19,730,000 = 9,273,100. In the same year, the number of people living with HIV is thought to be 725 and 3 people were living with AIDS (NACP, 2005; TACAIDS, 2005). That means total infected population equals 725 + 3 = 728.

Infectivity and average contact frequency:

The probability of HIV transmission per sexual act of vaginal intercourse, or infectivity (i), has been estimated from prospective studies of HIV-discordant partners or male contacts with prostitutes (Brookmeyer *et al.*, 1994). Published estimates of transmission probabilities per act vary from $i = 0.0001$ to 0.0014 in the US and European studies of discordant couples (Royce *et al.*, 1998; Nicolos *et al.*, 1994; Padian *et al.*, 1991 & De Vincenzi, 1994) and to $i = 0.002$ in Thai couples (Duerr *et al.*, 1994). However, higher transmission probabilities ($i = 0.056 - 0.100$ per act) have been reported among men who had contacts with female prostitutes in Thailand (Mastro *et al.*, 1994) and Kenya (Cameron *et al.*, 1989). The study on the HIV transmission conducted in Rakai Uganda estimated the average probability of $i = 0.0011$ (Gray *et al.*, 2001). Many studies conclude that the higher transmission probabilities associated with commercial sex might be attributable to the presence of other sexually transmitted diseases (STDs), which are thought to increase infectivity of or susceptibility to HIV (Royce *et al.*, 1998; Cameron *et al.*, 1989) or to possible errors in reported risky contact frequency. Other factors that increase transmission include lower CD4 counts or AIDS in the HIV-positive partner (Royce *et al.*, 1998; Duerr *et al.*, 1994; Cameron *et al.*, 1989). Male-to-female HIV transmission is usually more efficient than female-to-male transmission in US and European populations, but the small numbers of HIV-positive female partners limit conclusive sex-specific estimates of transmission probabilities per sex act (Royce *et al.*, 1998; Nicolos *et al.*, 1994; Padian *et al.*, 1991; De Vincenzi, 1994; Mayer *et al.*, 1995; & O'Brien *et al.*, 1994)

There are not enough studies on per-contact probability of transmission from representative heterosexual couples in sub-Saharan Africa, and there is little information on the efficiency of transmission associated with HIV viral subtypes (Royce *et al.*, 1998). An estimate for female-to-male transmission is also not available but is presumably somewhat lower (hivinsite, 2005). We would therefore focus on the infectivity from male to female. Three different studies conducted in three different East African cities reported that the transmission probability per sex act from male to female varies from 0.0011 to 0.24 (Gray *et al.*, 2001; Grosslurth *et al.*, 1995; and Quinn *et al.*, 2000), using this report the estimated weighted average of the HIV transmission probability is around 0.03 per sex act. We will adopt the infectivity value of 0.03 in our model. All three reports conclude that not all sex acts result into infectivity but it also depends on other factors such as higher viral load and

genital ulceration. The estimated infectivity value above assumes the presence of other sexual transmitted diseases (STDs) to serve as facilitating factors. STDs were observed in most of the HIV infected people in all three cities (Quinn *et al.*, 2000). Untreated STDs, particularly herpes and syphilis, cause open genital sores that dramatically increase the probability of blood transmission during sex, and therefore increase the probability of HIV transmission, see (Kapiga and Aitken, 2003).

When considering HIV sexual contact frequency data in Tanzania, issues of underreporting of sexual behaviour become more relevant. What is important is that the levels of sexual behaviour reported here are almost similar to other studies of sexual behaviour in the regions (in particular, the Four Cities Study (Ferry *et al.*, 2001). Using data from Demographic Health Survey between 1999 and 2003, people reporting having had sex without condom or with a person who they do not know their health status varies from year to year, in Tanzania we estimate an average of 19.25 unsafe contacts (risky contacts) per year. That means yearly average weighted unsafe sexual (risky) contacts between 1999 and 2003 equals $(21+20+19+17 = 77)$, therefore $77(\text{contacts}) / 4(\text{years}) = 19.25$ unsafe sexual contacts per year). The data for sexual behaviour among sex workers may be underreported because prostitution is illegal in Tanzania (DHS, 2004). We assume that only risky sexual contact with HIV infected person results into HIV infection and that susceptible population have similar unsafe sexual contact as infected. This assumption is reasonable if we assume that many people do not know their HIV status. Although it is difficult to come to a conclusion on this contact frequency without further studies in the people's sexual behaviour, a few things are worth noting. The first is that there exists people having unsafe sex in the general population and some of these contacts could be with infected people. Further, many people do not know their HIV status therefore they do not realise the danger they are posing on others.

In the Model 1, once an infected individual arrives in the community, every susceptible person eventually becomes infected, and the total HIV infected population following the classic S-shaped pattern of the logistic curve.

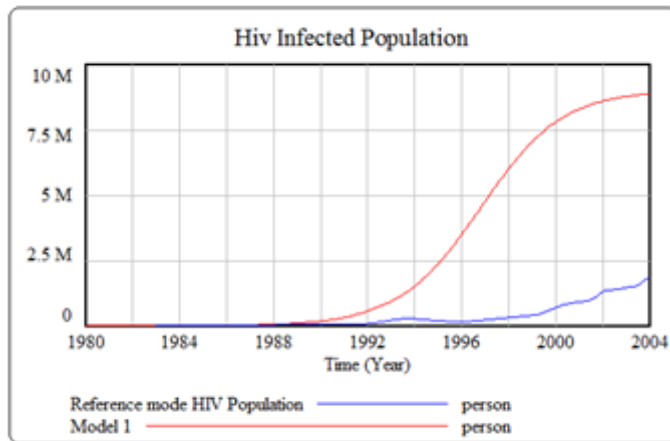


Figure 4.6. Behaviour of Model 1 and the reference mode for HIV/AIDS population

Figure 4.6 above display the graph for the Model 1 behaviour and the reference mode for HIV/AIDS population. In order to compare the two behaviours we have assumed that the infected population in our case is the combination of people living with HIV and AIDS. The behaviours in both graphs reveal that the reinforcing loop dominates; driving an exponential growth in the number of HIV/AIDS infected population. The peak in the simulated HIV/AIDS infected population reach almost 8.2 million by the year 2004 while the peak in the reference mode behaviour for HIV/AIDS population reached around 2 million. The difference between two behaviours is clearly seen on Figure 4.6.

Model 1 captures the most fundamental feature of infectious diseases: the disease spreads through contact between infected and susceptible individuals. It is the interaction of these two groups that creates the positive and negative loops and the non linear flow equation is responsible for the shift in the loop dominance as the susceptible population is depleted. The nonlinearities arise because the two populations are multiplied together; it takes both a susceptible and an infectious person to generate a new case.

While the Model 1 captures the basic process of infection, it contains many simplifying and restrictive assumptions. The model does not represent births, deaths, or migration and assume no incubation period. The population is assumed to be homogeneous. The disease does not alter people’s lifestyle: infected population are assumed to interact at the same average rate as susceptible population. Therefore, in an attempt to replicate the reference mode, some of these assumptions can be relaxed by considering Model 2 with some additional features.

Model 2: SIR model

We now consider the SIR model of epidemiology. The model contains the basic three stocks: The Susceptible population, S, the HIV Infected population, I, and Recovered/Removed population in our case AIDS population. The susceptible population, as in the Model 1, is reduced by the infection rate. The HIV infected population accumulates the infection rate less the symptoms acquisition rate and the AIDS population accumulates the symptoms acquisition rate. The dynamics of the system is represented by the infection rate. Sterman's formulation of SIR model (Sterman, 2000) is shown below.

$$S = \text{INTEGRAL} (- IR, N - I_0 - R_0)$$

$$I = \text{INTEGRAL} (IR - RR, I_0)$$

$$R = \text{INTEGRAL} (RR, R_0)$$

The initial susceptible population is the total population within the age group 15 - 59 less the initial number of infected and any initially AIDS individuals.

In our case the symptoms acquisition rate can be modeled in several ways. In the Model 2, the incubation period, is assumed to be constant and the symptoms acquisition process is assumed to follow a first-order delay, negative feedback process.

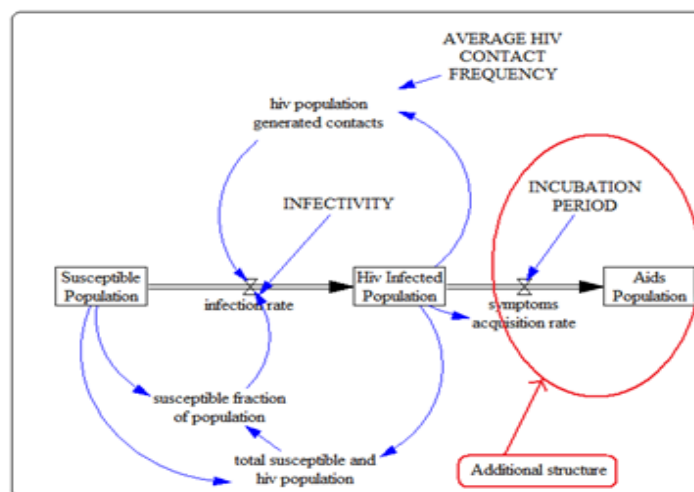


Figure 4.7. Structure of Model 2.

The structure of “Model 2” in Figure 4.7 contains three stocks and two flows: The Susceptible population, the HIV Infected population and the AIDS population. The two flows

are the infection rate and symptoms acquisition rate. Everything else remains the same as in the Model 1 except that we have included the flow of symptoms acquisition rate and the stock of the AIDS population.

$$\text{Symptoms acquisition rate} = \text{HIV Infected population} / \text{Incubation period}$$

Adding structure influence model and that can change the behaviour of the system due to the delay process between the two stocks (HIV infected population and AIDS population) we will describe this in the behaviour section.

Causal Loop Diagram of the Model 2.

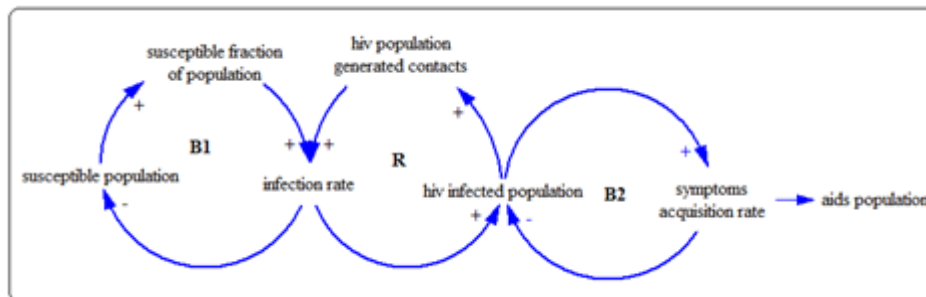


Figure 4.8. Causal Loop Diagram of the Model 2.

The causal loop diagram highlights the extra loop formed by the symptoms acquisition process. The structure of the Model 2 shows that those contracting the disease become infectious for a certain period of time (incubation period) before showing symptoms and then they move to another stage of the disease AIDS. Note that loop B1, R and B2 in Figure 4.8 correspond to loop B1, R, and B2 in the Figure 4.2 which are the main loops in the dynamic hypothesis.

Behavioural pattern of the Model 2.

The behaviour of Model 2 is displayed in the figure below where all assumptions of the Model 1 are retained. The initial value of the AIDS population is considered to be 3 (NACP, 2005), and the incubation period is assumed to be 8 years (Gray *et al.*, 2001; Grosslurth *et al.*, 1995; and Quinn *et al.*, 2000).

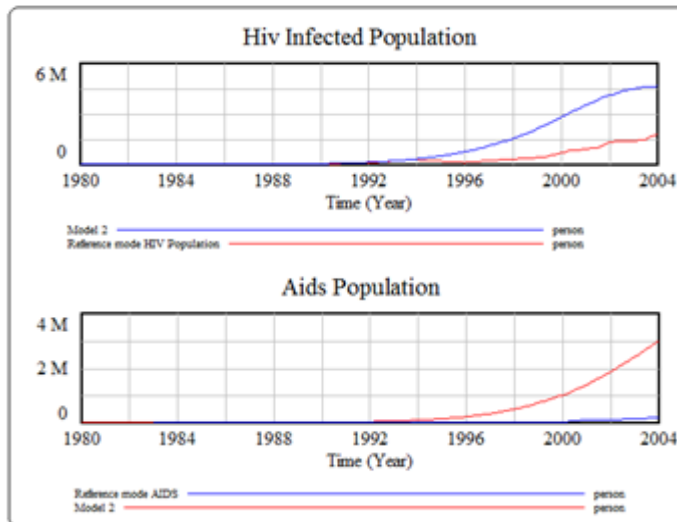


Figure 4.9. Behaviour of the “Model 2” and the reference mode for the HIV population and reference mode for AIDS population.

The simulation behaviour of an epidemic in the Model 2 and the reference mode for HIV population and AIDS population are shown on Figure 4.9. The graph representing the simulation for the HIV infected population in Model 2 grows faster and reaches the peak of almost 4.6 million by the year 2004. At that time the reference mode for HIV population reaches the peak of 1.8 million. The simulated behaviour of AIDS population in Model 2 grows to reach a peak of above 3 million while the reference mode for AIDS population grows to reach a peak of almost 200,000.

Model 1 and Model 2 differ in both structure and behaviour. The former assumes that when infectious come in the community the whole community will be infected because there is nobody who is immune. The later assumes that people do move from HIV population to AIDS population after incubation period. In addition, the dynamics in the Model 2 can be determined by noting that without births, or deaths, the total population is fixed. The simulated behaviour of Model 2 is still far from the reference mode behaviour, although it is closer than Model 1 behaviour. Nevertheless, it is clear that the Model 2 structure is not adequate. We will improve the model by considering the changing population, first by introducing the deaths in the Model 3.

Model 3: Introduce AIDS Deaths

Model 2 involves number of restrictive assumptions. Therefore, we modify the Model 2 by introducing AIDS deaths people on the AIDS population.

Structure of Model 3: Introducing deaths

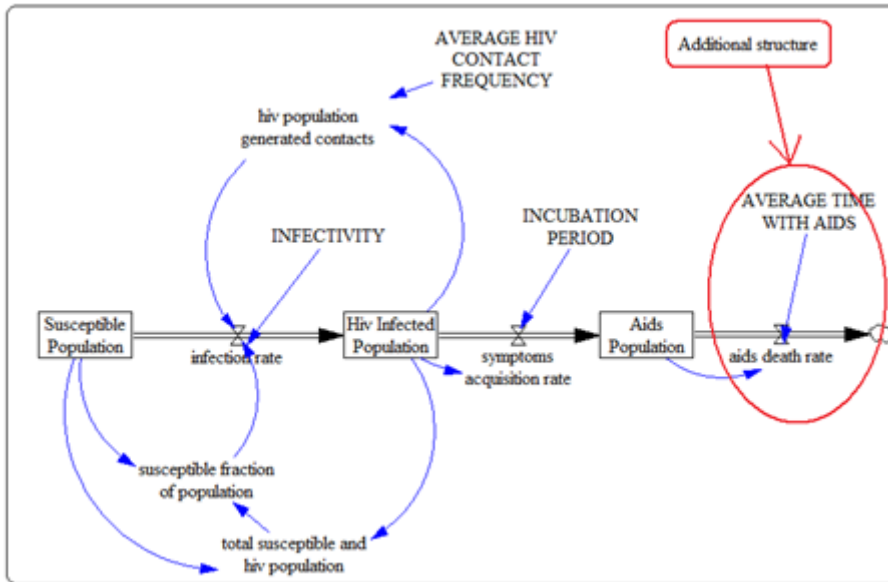


Figure 4.10. Structure of the Model 3.

The structure of the Model 3 is displayed in Figure 4.10 above. The new structure allows people in the AIDS stock to die. There are three stocks and three flows in the Figure 4.10, stock of the susceptible population, stock of the HIV infected population and stock of the AIDS population and the flow of infection rate, symptoms acquisition rate and aids death rate. Births are omitted in Model 3 so the total population change only through deaths and people with AIDS remain infected until death. People move from the stock of the susceptible population to the stock of the HIV infected population through a flow of infection rate. They move further from the stock of the HIV infected population to the stock of the AIDS population after an incubation period and finally this people will die.

$$\text{AIDS death rate} = \text{AIDS population} / \text{Average time with AIDS.}$$

The following diagram shows the causal loop diagram of the Model 3.

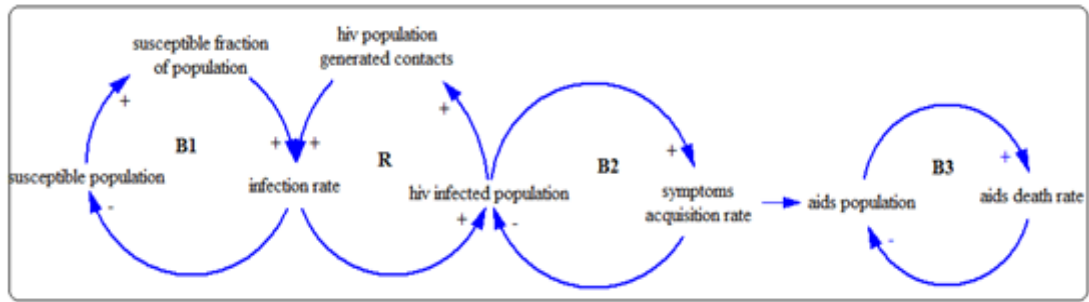


Figure 4.11. Causal Loop Diagram of the Model 3.

The summarized causal loop diagram Figure 4.11 highlights the extra loop formed by the AIDS death rate. The causal loop diagram in Figure 4.11 shows that people acquiring AIDS symptoms will stay in the stock of AIDS population for a certain period of time (average duration of AIDS) and they finally die.

Loop B1, R and B2 and B3 in Figure 4.11 correspond to (Depletion loop- B1), (Contagion loop-R), (AIDS Symptoms loop B2), and (AIDS death loop B3) in Figure 4.2. Thus, we have now demonstrated that our dynamic hypothesis is based on the stock-and – flow structure in Figure 4.10.

Behavioural pattern of the Model 3.

To see the behavior of the Model 3 and be able to compare with the reference mode behavior, we will now fit the model with the same information we have used in the Model 1 and Model 2. In addition to that we have now introduced the flow of AIDS death rate. The assumption for the average time with AIDS parameter is that without any treatment it takes only 1 year or less for a person suffering from AIDS disease to die (UNAIDS/WHO, 2007; Gray *et al.*, 2001), and this has also been observed by NACP report in Tanzania. The simulation results for Model 3 are displayed in Figure 4.12, along with the reference modes for both stocks.

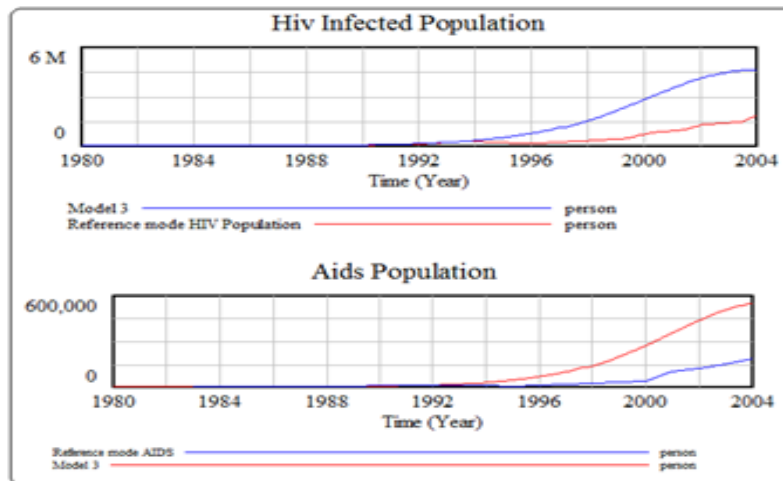


Figure 4.12. The behaviour of Model 3 and the reference mode for the HIV population and AIDS population.

The HIV simulation results for Model 3 Figure 4.12 and Model 2 Figure 4.9 are the same; nothing in that part of the structure changed when we went from Model 2 to Model 3, the results are the same.

The AIDS population grows to approximately 551,000 people by the year 2004. The reference mode for AIDS population grows to around 200,000 by the year 2004.

The behavior we have seen in Figure 4.12 does not yet replicate the reference mode behavior; therefore we need we consider how the structure might inadequate and then make improvements. If we try to run the model for longer enough say until year 2050 we may realize that the whole susceptible population will diminish and that is due to the fact there are no inflow to the stock of susceptible population. Therefore, in this system when everybody is infected and no people entering into the susceptible population that means after an incubation period, everybody will acquire AIDS symptoms and die, which obviously unrealistic. This requires the model to be integrated with a basic population model, taking into account the evolution of the susceptible base and of the population as a whole. We also point out that people do not die only through AIDS, there are several causes of deaths, and therefore we will consider all this in the next model.

Model 4. Births and Non AIDS deaths

To simulate the births and non AIDS deaths, let us adopt the simple approach to simulating population from chapter 6 in Ford, (1999) and simulating coflows from chapter 12 in Sterman, (2000). Figure 4.13 shows a population model.

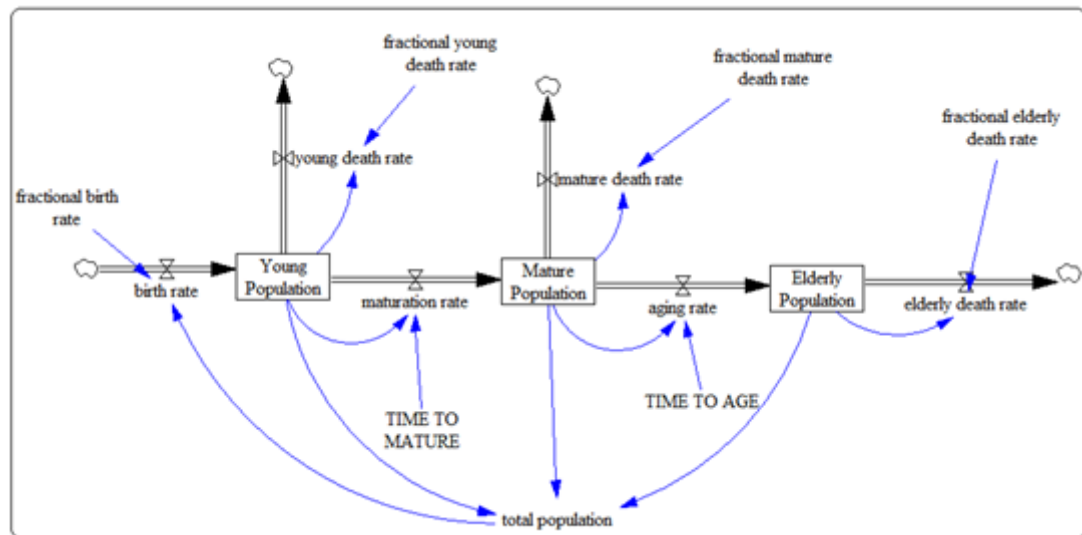


Figure 4.13. The population model

Young Population age 0 - 14:

A simple population model in Figure 4.13 begins with the stock of the young population. The stock of young population is increased by birth rate and decreased by young death rate and maturation rate. For simplicity, assume all the two outflow rates are first-order:

Young Population = INTEGRAL(birth rate – young death rate - maturation rate, Young Population(to)).

Initial value for the young population equals to the total population in 1983 multiplied by the fraction of young population. Total population is thought to be 19,730,000 (NBS, 2005).

$$19,730,000 * 0.49 = 9,667,700$$

We consider the average fractional birth rate to be 42 births per 1000 people this assumption is based on the report by the U.S. Bureau of Census, (1995), and this corresponds to the estimate by the population statistics information for Africa (2001) and the National Bureau of Statistics, Tanzania (NBS, 2005).

Young death rate = Young population * fractional young death rate

Fractional young death rate equals to 30 per 1000 people (NBS, 2005). Infant mortality rate is reported to be high in developing countries.

Maturation rate = young population/time to mature

We assume that it takes 14 years to mature.

Mature population 15 – 59 (Susceptible population)

Mature Population = INTEGRAL(maturation rate – mature death rate - aging rate, Mature Population(to)).

Mature population (1983) = 19,730,000*0.47 (fraction of mature population) = 9,273,100

Mature death rate = Mature population * fractional mature death rate

Fractional mature death rate equals 10 per 1000 people (NBS, 2005)

Aging rate = mature population/time to age

We assume that it takes 44 years to age

Elderly Population age 60 and above:

Elderly Population = INTEGRAL(aging rate – elderly death rate, Elderly Population(to)).

Elderly population (1983) = 19,730,000*0.04(fractional elderly population) = 789,200

Elderly death rate = Elderly population * fractional elderly death rate

Fractional elderly death rate equals 15 per 1000 people.

Now, we need to integrate the population model with the HIV/AIDS Model 3. As we have indicated earlier on, our model considers heterosexual contacts which occur within the age group between 15 and 59 (mature population in the population model). Therefore, we assume that the stock of mature population in the population model is similar to the stock of susceptible population in the HIV/AIDS model that means the flow of maturation rate is also similar to the inflow to the susceptible population. In addition to that we assume that the

outflow of mature death rate in the stock of mature population is similar to the outflow of susceptible normal death rate in the HIV/AIDS population.

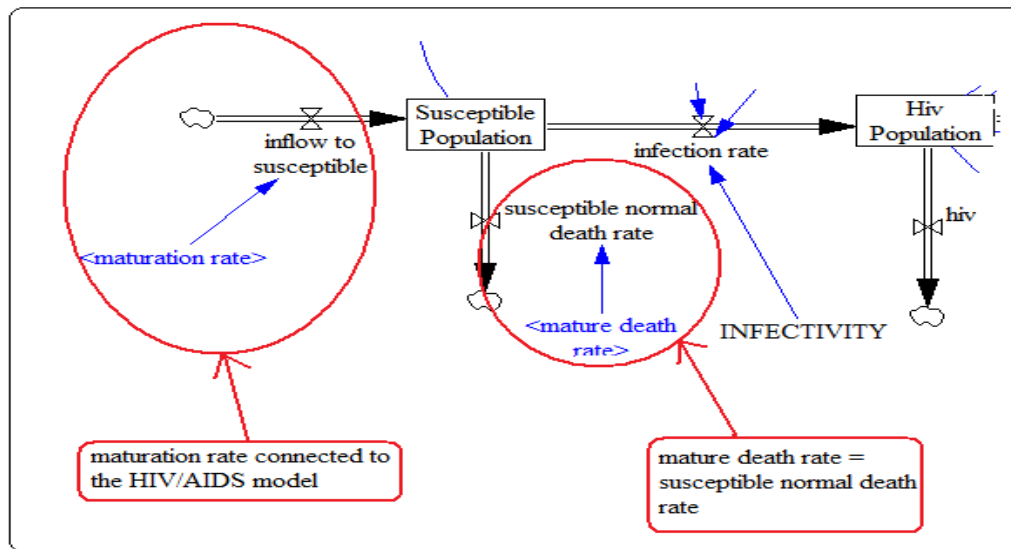


Figure 4.14. The structure to show how population is attached to the Model 4

Figure 4.14 shows how the population model is attached to the rest of the model with the use of coflows. The coflow is a stock and flow structure exactly mirroring the main stock and flow as new people enter into the population stock and others leave the population when they die.

Inflow to susceptible = maturation rate

The new model structure Model 4 is able to keep track of births and deaths occurring in the population.

Behavioural pattern of the Model 4

All Model 4 assumptions are the same as in the previous Model 3, except that we have included the population model, and the normal deaths for population stock.

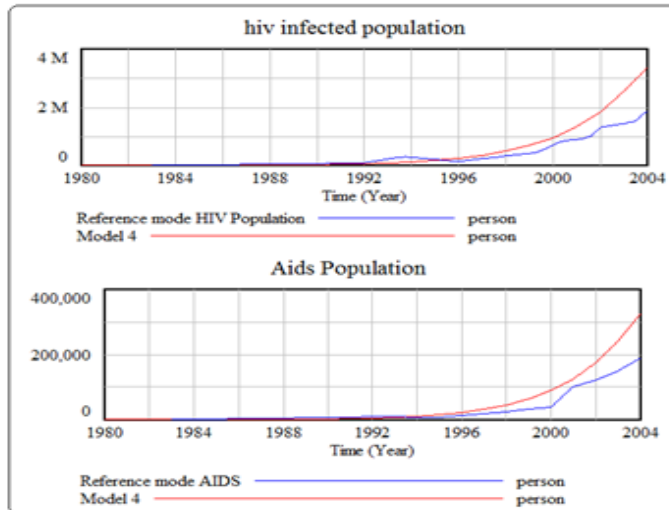


Figure 4.15. Behaviour of the Model 4

Figure 4.15 shows that HIV population grows to around 3 million in Model 4 by the year 2004. The reference mode for HIV population grows to around 1.8 million by the year 2004. The AIDS population grows to approximately 300,000 people by the year 2004. The reference mode for AIDS population grows to around 200,000 by the year 2004.

The Model 4 behavior in Figure 4.15, while an improvement over the behavior of Model 3 does not yet replicate the reference mode behavior; thus causing us to consider what additional structure might be appropriate. We remember that some of the births are from HIV-positive mothers therefore we need to track these people. The next model will consider all these factors.

Model 5: HIV-positive children

We may realise that some of the births are from HIV positive mothers; therefore we should be able to separate between children who are HIV-positive and HIV-negative children. The children of HIV-positive mothers can be HIV-positive or not depending on several factors, as hygienic conditions during the delivery of the child or as the duration of the breast-feeding period, among others: in most of cases this is strongly depending on the quality of the medical assistance, before and after the delivery (WHO, 2007). The overall probability of transmission of the virus from mother to child is represented by the prenatal transmission fraction. This is the fraction of new born children that is HIV-positive and this

fraction is dimensionless. The fraction of children of HIV-positive mothers that is not HIV-positive will flow into the stock of young population.

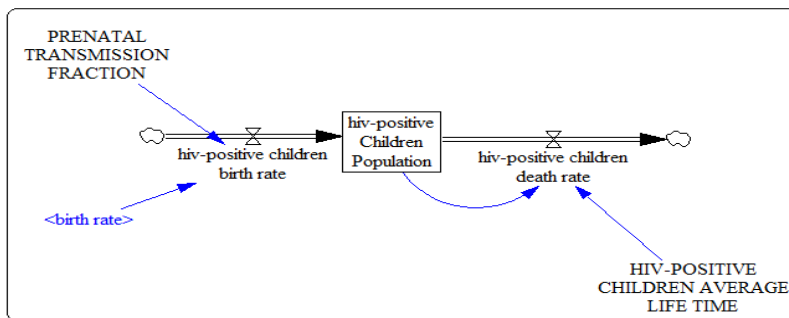


Figure 4.16. The stock of the HIV-positive children population

Figure 4.16 shows the structure of the HIV-positive children population. The stock of HIV-positive children is increased by the flow of HIV-positive children birth rate and is reduced by the flow of HIV-positive children death rate. The flow of the HIV-positive children birth rate is shown to depend on the birth rate and prenatal transmission fraction

Those who are infected, on the other side, will flow into the stock of the HIV-positive children population. Here they will stay for the maximum of 2 years, before eventually dying from one or several causes associated to HIV. In Africa, 30 – 50% of all untreated HIV-positive children die prematurely before their first birthday (Dray-Spira et al. 2000). Note that a particular observation must be made on the utilization of the same fractional birth rates for infected and uninfected people. This is a reasonable assumption if we assume that most of the HIV positive mothers have no idea about their HIV status. Therefore;

$$\text{HIV-positive children population} = \text{INTEGRAL} (\text{HIV-positive children birth rate} - \text{HIV-positive children death rate}, \text{HIV-positive children (to)})$$

Initial HIV-positive children = 0. This estimate is based on the assumption that at the beginning of the epidemic those infected were adults.

$$\text{HIV-positive children birth rate} = \text{birth rate} * \text{prenatal transmission fraction}$$

$$\text{Prenatal transmission fraction} = \text{HIV-positive children born in (2004)}/\text{total births (2004)}$$

TACAIDS, (2005) estimates that 3.19% of children born in 2004 were HIV-positive and that in the same year there were a total of 1,357,680 new births. That means 3.19% of 1,357,680 equals 43,310 HIV-positive children born during 2004. Therefore;

Prenatal transmission fraction = $43,310/1,357,680 = 0.0319$ round off. 0.32 and the unit is dimensionless.

HIV-positive children death rate = HIV-positive children/HIV-positive children average life time.

HIV-positive children average life time is estimated at 2 years (NACP, 2005; TACAIDS, 2005; WHO, 2005). We assume that HIV-positive children will die through HIV related complications.

Therefore, we need to change the structure of the population model, see the following Figure 4.17 below.

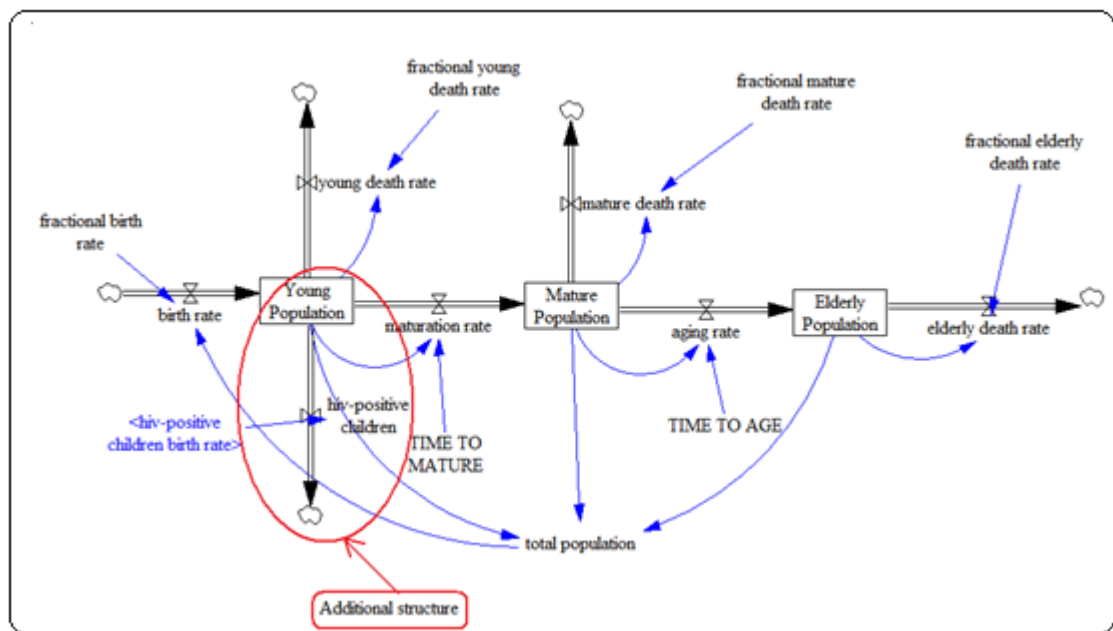


Figure 4.17. Additional structure to the stock of the Young population.

Young population = (birth rate – young death rate – maturation rate – HIV-positive children, Young population (to))

Behavioural pattern of the Model 5.

All Model 5 assumptions are the same as in the previous model Model 4, except that we have included the HIV-positive children population. Figure 4.18 below shows the behaviour of the Model 5.

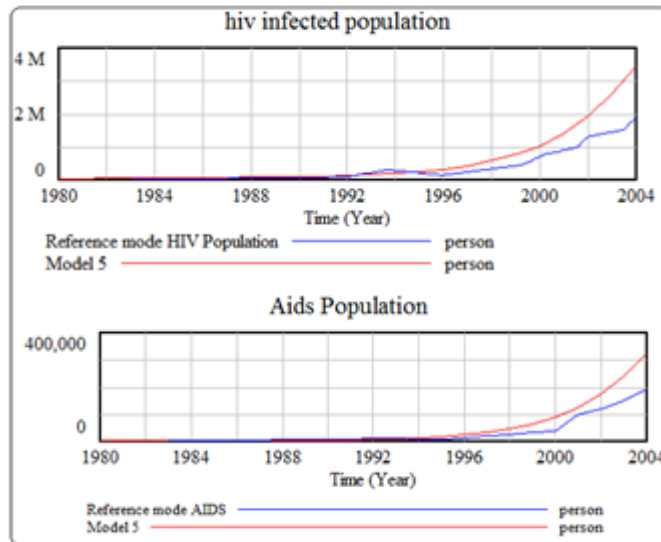


Figure 4.18. Behaviour of the Model 5

The HIV/AIDS simulation results for Model 5 Figure 4.18 and Model 4 Figure 4.15 are almost the same; there is little improvement apart from the additional structure changed when we went from Model 4 to Model 5. This is due to the fact that fraction of HIV-positive children is still little.

Model 5, while it matches the behavior pattern, nevertheless overestimates the historical trend; thus causing us to consider what additional structure might be appropriate. We hypothesize that the HIV education and awareness program has probably had some beneficial effect. We assume further that some of the “error” in Model 5 is due to failure to reflect the beneficial effect of the HIV education and awareness program. The next Model 6 will consider all these factors.

Model 6: HIV education and awareness program.

Studies have shown that despite a high level of knowledge of HIV transmission and ways to prevent it, only 16 per cent of men reported using condoms during casual sex (Stromblad and Zaar, 2005). This finding appeared to be partly related to various myths surrounding the use of condoms (UNAIDS and UNHCR, 2003). Using findings from 1996

Tanzania Demographic and Health Survey (TDHS) and 1999 Tanzania Reproductive and Child Health Survey (TRCHS) indicates that knowledge about ways to avoid HIV/AIDS increased among the general population. TACAIDS, (2001) indicates a low reduction in HIV infectivity in the general population of about 5 – 10% per cent due to the increase in HIV education and awareness program. Studies conducted by Stromblad revealed that some people found condoms uncomfortable and others suggest that available condoms are of particularly bad quality and commonly tend to burst during sexual contact (Stromblad and Zaar, 2005). On the other hand while the government and other agencies focused on educating people about the use of condoms, Tanzanian’s churches do not share the same idea instead they preach against the use of condoms. Therefore, to close the gap between the simulation in the Model 5 and the reference mode we have estimated the effect of HIV education and awareness program on the infectivity of about 10% at the time of the survey. We will consider making a nonlinear curve in the table function show the effect. Therefore, we assume a goal-seeking behaviour that approaches 10%. This is reasonable behaviour if we consider the situation during the survey.

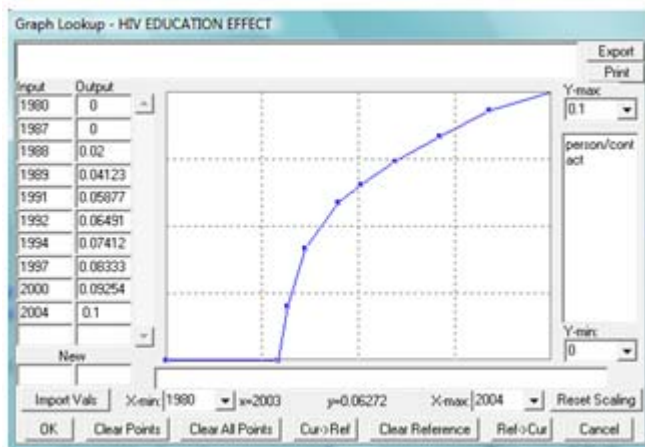


Figure 4.19. HIV education and awareness effect table function

Figure 4.19 shows that before the introduction of the HIV education and awareness program there were no effect on the infectivity. The input is time (year) and the output is the effect measured in percent. Before year 1988 there were no effect on the estimated infectivity. From the year 1988 the effect of HIV education and awareness program could be seen and it started increasing in a goal-seeking behaviour, at this time people wanted to know much about the disease. The effect is high at the beginning but from around year 2000 the effect seems to decrease. We believe that HIV education and awareness program reduces the HIV infectivity because some people may opt to use condom or abstain from having sex.

HIV education effect = 1980-1987 = 0% and from the year 1988 the effect started increasing to reach the maximum of about 10% in 2004.

Therefore, Infectivity = $0.03 \cdot (1 - \text{HIV education effect})$

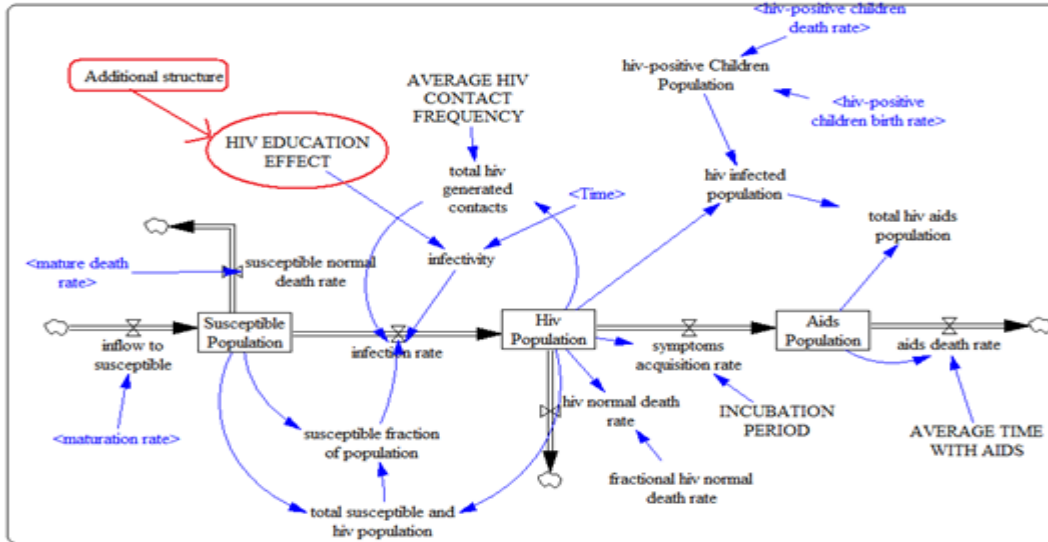


Figure 4.20. HIV education effect attached to the Model 5.

Figure 4.20 shows how the HIV education effect structure is attached to the Model 5.

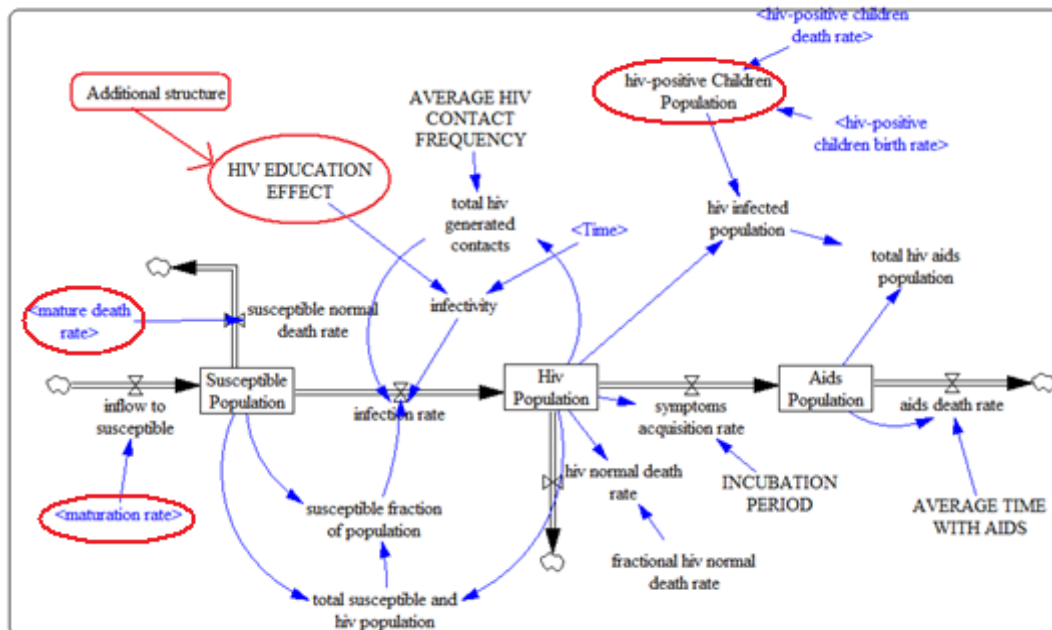


Figure 4.21. The structure of the Model 6.

HIV infected population = HIV population + HIV-positive children population

Total HIV/AIDS population = HIV infected population + AIDS population

Behavioural pattern of the Model 6.

All Model 6 assumptions are the same as in the previous Model 5, except that we have included the effect of HIV education and awareness. Figure 4.22 and Figure 4.23 below shows the behaviour of the Model 6.

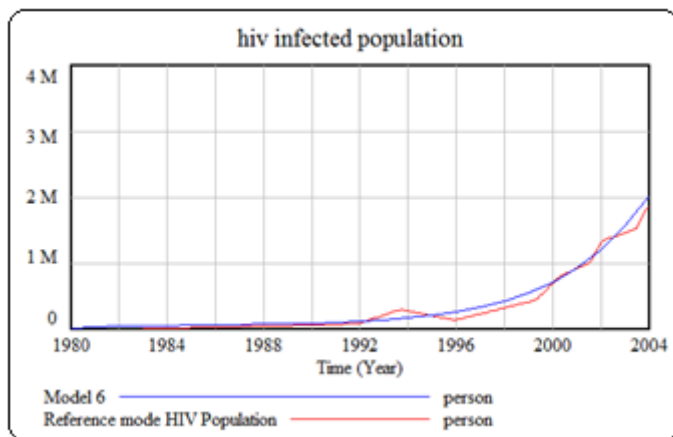


Figure 4.22: Behaviour of Model 6 and the reference mode for HIV population.

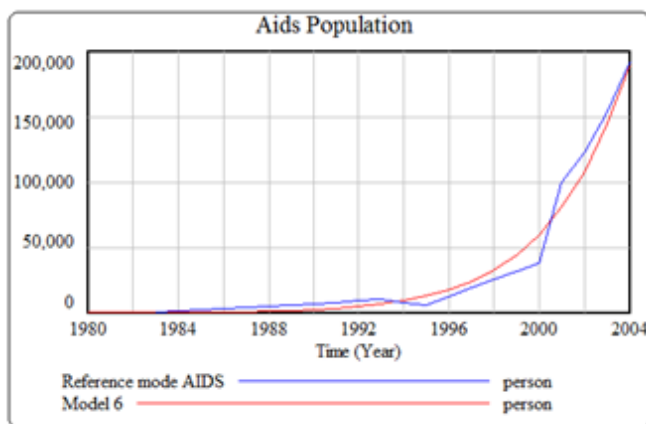


Figure 4.23: Behaviour of Model 6 and the reference mode for AIDS population.

Figure 4.22 and Figure 4.23 shows the simulation results of the Model 6 which includes the effect of HIV education on the average HIV contact frequency.

The simulated HIV population peak to reach around 2 million at the year 2004 which is approximate 1.8 million for the reference mode for HIV population. The simulated AIDS population peak to reach almost 190,000 by the year 2004 which is approximate the reference mode for AIDS cases which 200,000.

At this point, we have reached a major milestone in the modeling process; namely, a reasonably accurate replication of the reference mode. We want to acknowledge that the table function structure we have introduced in the Model 6 represent a highly uncertain parameter. However, the HIV education effect of maximum 10% we have estimated appear to be partly related to the various studies conducted in the country (TRCHS, 1999 and Stromblad and Zaar, 2005). Such a low effect could be on one hand the result of the standoff between the government, churches and other agencies regarding the issue of whether to use condom or not and on the other hand the unavailability and poor quality of the available condoms. The two Figures 4.22 and Figure 4.23 shows the reference mode established at the start of the simulation. We set out with the “dynamic hypothesis” that the HIV population and susceptible population could interact to produce the exponential growth-like behaviour shown in the reference mode and that we observe in the Figure 4.22. At the moment the reinforcing loop **R** proves to be much stronger than the balancing loop draining more and more people from the susceptible population into the HIV population.

For the matter of simplicity, we have ignored some of the issues such as tracking the population separately between rural and urban, track male and female separately and the influence of the general level of education attained on the average HIV contact frequency rate and infectivity.

Summary

This chapter has presented the building of the model, from the hypothetical reference mode, to the structure that we have built using Vensim software (a complete list of equations is listed in the appendix of this paper). We will now address the validation issue.

5. VALIDATION AND MODEL TESTING

Validation and model tests in this chapter help us to build confidence that a model is useful. It also help us understand the underlying structure; find out the robustness and sensitivity of the results according to the assumptions that we made with regarding to the model boundary and interactions among variables. Following Forrester (1973) and (Forrester and Senge, 1980), we “validate” the model by trying to build confidence in the soundness and usefulness of our model. We will now validate our model by testing its soundness and usefulness.

Tests to Build Confidence

Matching historical behavior is only one of several tests that can be used to build confidence in our model. Many researchers have described a wide assortment of useful tests (Forrester and Senge 1980; Richardson and Pugh; 1981; Kitching 1983). We will perform several tests in our model which include among others: verification, face validity, historical behavior, extreme behavior, integration error, sensitivity analysis, behavior tests and policy tests.

Verification

Models may be “verified” when they are run in an independent manner (naturally by different group on a different computer) to learn if the results match the published results. The purpose of this test (verification) is to learn if the computer models “run as intended” (House and McLeod, 1977 p.66). Greenberger *et al.*, (1976 p.70) describe verification as a “test of whether the model has been synthesized exactly as intended. Verification of a model indicates that is has been faithful to its conception, irrespective of whether or not it and its conception are valid”. However, Kitching (1983 p.42) warns that verification may sound tautological but is “nevertheless a necessary check that the mechanisms of the model are in fact doing what the modeler thinks they are doing.”

For an independent part to be able to test our model we have provided adequate documentation for all the variables used and we are providing a CD containing the complete model, plus all of the validation tests. Therefore, our model can be fully tested by an independent part at any point of time.

Face Validity

Ford described the “face validity” test as a common sense test (Ford, 1999). We have performed a face validity tests to see if the parameters and our model structure make sense. All the stocks used in the model such as the stock of population, the stock of susceptible population, the stock of people living with HIV, the stock of people living with AIDS and the stocks of the HIV-positive children represents the three main groups in the epidemic. All the flows pointed in the right direction, parameters that must be positive such as birth rate has positive values throughout the simulation. However, Ford (199) believe that in large organization, many models grow to become so complex in such a way that we cannot “zoom inside the model” to perform this kind of simple test, but our model is a simple and small model therefore to perform face validity is possible.

Historical Behavior

In system dynamics model testing, one of the most common and important tests is to set the inputs to the mode at their historical values and observe if the simulation results match the history (Ford, 1999). Indeed, this test is usually what people normally think of when discussing about model validation. Kitching (1983 p.43) describes several variations in the historical behavior test for ecological systems. Model parameters in our model are mostly based on data from the National AIDS Control Programme in Tanzania, and World Health Organization. We simulated our model to learn if the model simulation matches data from these sources.

The historical behavior test is especially informative if the model is designed with a large number of endogenous variables and a limited number of exogenous inputs. Table 4.1 in chapter 4 shows that most of the variables in the model are endogenously generated. Our model falls into this category because it is constructed from a system dynamics perspective, which encourages us to simulate the feedback loops in the system. In his book Ford (1999) insists that an internally generated behavior that matches history is one of the more important tests of any model. We have built our model in a step by step process until the model generated pattern that matches historical behavior in the Model 6. Below is the behavior of the two reference mode and simulated behavior of the Model 6. Tests in Model 6 include the “HIV education policy” structure, and we would admit that this is more subjective in its formulation than the other parts of the model.

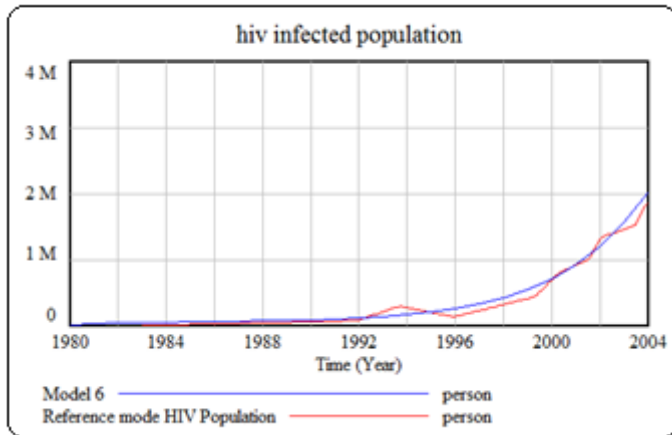


Figure 4.22: Behaviour of Model 6 and the reference mode for HIV population.

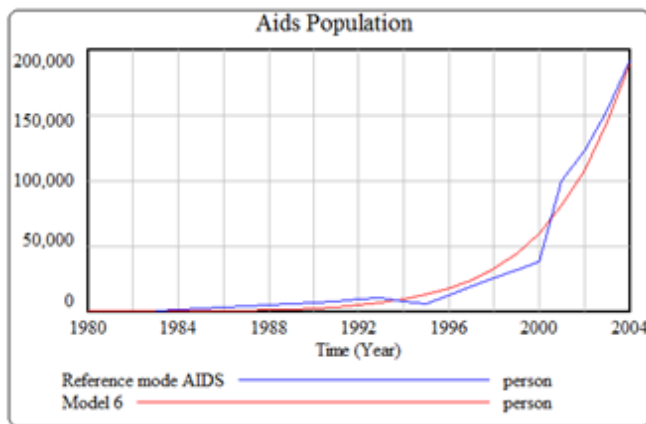


Figure 4.23: Behaviour of Model 6 and the reference mode for AIDS population.

The two Figures 4.22 and Figure 4.23 shows the reference mode established at the start of the simulation and that we managed to generate it reasonably well.

Extreme condition tests

One of the revealing tests is to make change in model parameters and see if the model’s response is plausible. Extreme behavior testing may be facilitated by the software in the Vensim software. If the model is structurally flawed, the flaws will probably be revealed by a simulation with clearly spurious behavior. We will concentrate on the following indicators: the susceptible population, the HIV infected population, the contact frequency and the prenatal transmission fraction. Such tests, termed “reality checks” by two authors Peterson and Eberlein (1994), quickly uncover flaws, a great advantage in a large model. We will compare the values of the extreme testing with the values of the model in baseline run.

Zero susceptible population.

If there are no people who are susceptible to the HIV at the beginning of simulation, but there exist an inflow to the susceptible population, we expect that at the beginning, the graph of the HIV infected population to start growing at the below the Model 6. The two graphs will have the same behaviour S-shaped form and that at a certain point the two graphs will be similar. At this point the initial value does not affect the behaviour anymore. And we expect that the stock of the AIDS population will also follow the same behaviour. We will do the test by multiplying the initial value of susceptible population by zero and everything else remain the same. If there are no initial susceptible population it means that there are no prenatal infections as well, therefore prenatal transmission fraction equal to zero.

Susceptible population = initial susceptible population * 0 from year 1980

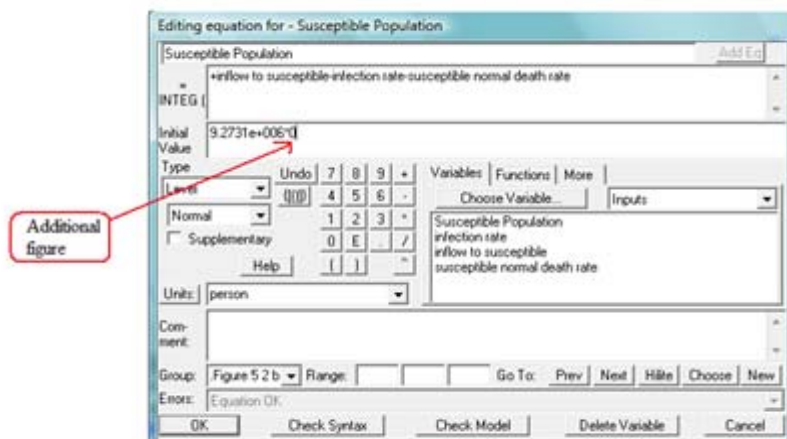


Figure 5.1. Additional figure in the susceptible population initial value.

The graph below displays the behavior generated

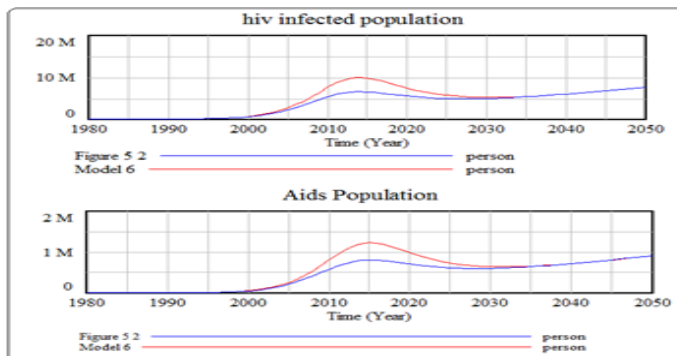


Figure 5.2. Extreme condition tests zero initial susceptible population.

The model behaves as expected: the stock of HIV infected population in Figure 5.2 grows below the graph in Model 6 but at around year 2035 the two graphs grow at the same level. And the stock of AIDS population in the Figure 5.2 grows below the graph in Model 6 but the two graphs grow at the same level at around year 2035.

Zero HIV infected population from 1980

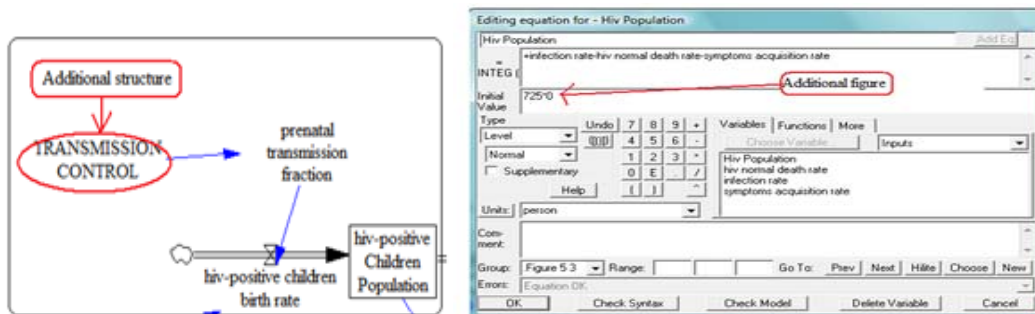
If there are no people who are infected, we expect the stock of the HIV infected population to remain at zero throughout. We expect that the stock of the AIDS population to start diminishing immediately because there were few people already living with AIDS but there will be no more people developing AIDS symptoms. We will perform the test by setting the value of HIV population at zero from the beginning of the simulation year 1980.

$$\text{HIV population} = 0 \text{ from 1980}$$

We have assumed a prenatal transmission fraction of 0.032, but if there are no susceptible population initially, there will be no children born with HIV

$$\text{Prenatal transmission fraction} = 0.032 * \text{transmission control}$$

$$\text{Transmission control} = 0 \text{ from year 1980}$$



Transmission control structure and zero initial susceptible added to the Model 6.

The graph below displays the behavior of the tests and we will use Model 6.

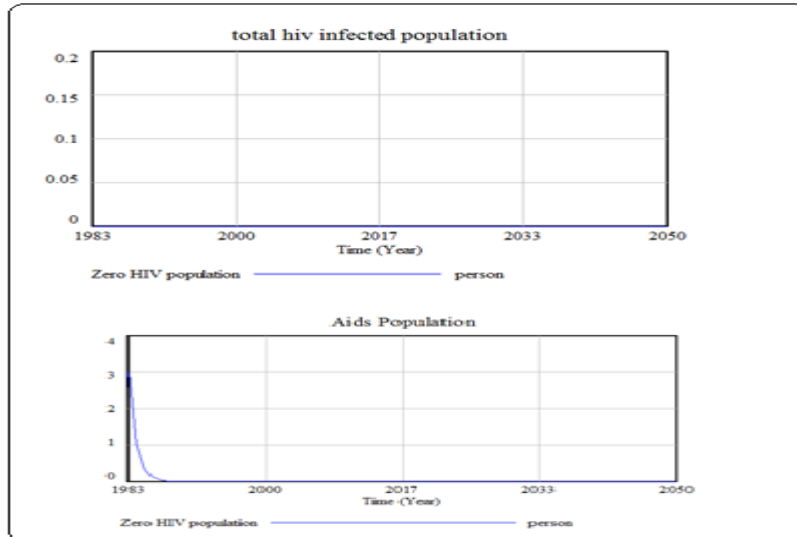


Figure 5.3. Extreme condition tests HIV population = zero

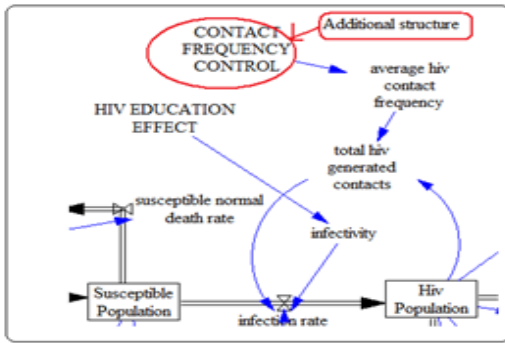
The model behaves as expected in the real world: the stock of HIV population remains at zero and the stock of AIDS population go to zero because no more people developing AIDS symptoms. Without susceptible population there will be no goal seeking or exponential-like behaviour because it takes both susceptible and infected to create a new infection.

Zero average HIV contacts frequency and zero prenatal transmission fraction.

If average HIV contact frequency and prenatal transmission fraction equal to zero from year 1980, that means the behaviour of HIV infected population will start diminishing to zero from its initial value. At the beginning of the simulation the stock of AIDS population will start to grow because there were few people living with HIV but will soon start decreasing again because no more people contacting HIV.

$$\text{average HIV contact frequency} = 19.25 * \text{contact frequency control}$$

$$\text{Contact frequency control} = 0 \text{ from } 1980$$



Contact frequency control added to the Model 6.

Prenatal transmission fraction = 0.032 * transmission control

Transmission control = 0 from year 1980

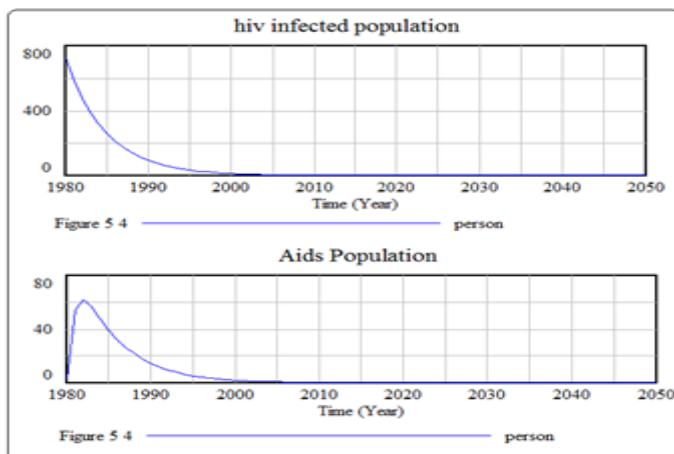


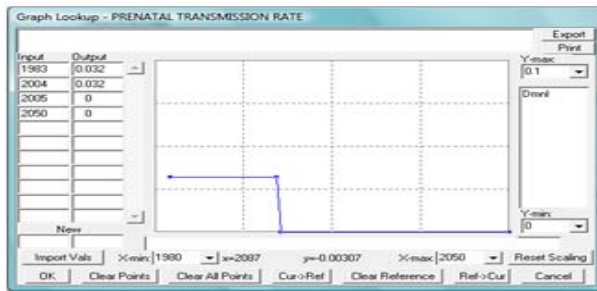
Figure 5.4. HIV contacts frequency and prenatal transmission fraction equals zero.

The model behaves as expected: the stock of the HIV infected population diminishes to zero and the stock of AIDS population increases and then drops towards zero because the whole population has been infected and no new infection moving into HIV population.

Prenatal transmission fraction equal to zero from the year 2005

If prenatal transmission fraction drops to zero from year 2005 that means there will be no more children contracting the HIV infection. Stock of the HIV-positive children must go to zero after year 2005. We will perform the test using Model 6 with an additional structure.

Change in the prenatal transmission is shown using the following function



Change in the prenatal transmission fraction added to the Model 6.

Figure 5.5 below shows the results of the test

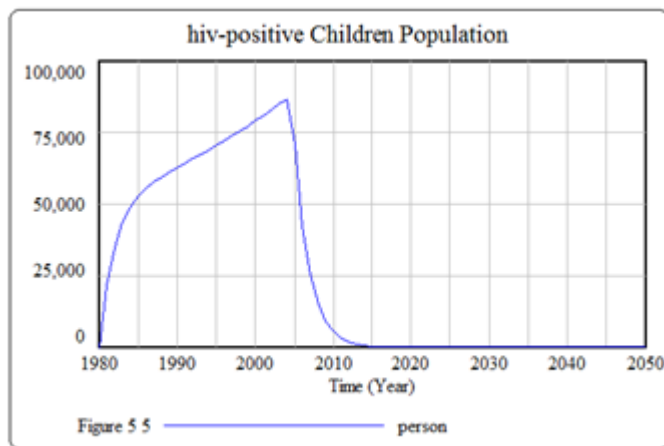


Figure 5.5. Zero prenatal transmission fraction from year 2005 to 2050

The model behaves as expected: the stock of the HIV-positive children started falling immediately after year 2005.

Sensitivity analysis tests

The purpose of this test is check if the model behavior is sensitive to parameter value permutations. We will identify parameters that influence the strength of feedback loops. Then we will vary parameter values and evaluate the effects on the feedback loops during simulations. Our goal is to learn if the basic pattern of results is sensitive to changes in the uncertain parameters. We will check to see if we get the reference mode after each test. A model is called robust when it generates the same general behavior despite the great uncertainty in parameter values (Sterman, 2000; Ford, 1999).

Integration error

The result of our model should not be sensitive to the choice of the time step or integration method. We used the “DT error” test suggested by Sterman, (2000) and we found no sensitivity to the time step, not to the integration method. The time step we used throughout the modelling process was .125.

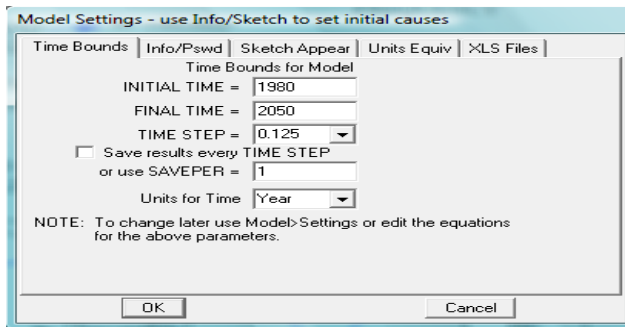


Figure 5.6. Time step

Policy sensitivity test

We performed a policy sensitivity test and policy parameters to see if the policies we suggest are sensitive to extreme values. (See also policy tests in the chapter 6, Policy analysis).

Influences on the Feedback Loops – Policy parameters

We will now identify parameters that influence the strength of feedback loops and then vary value parameters and evaluate the effects on the feedback loops during simulation

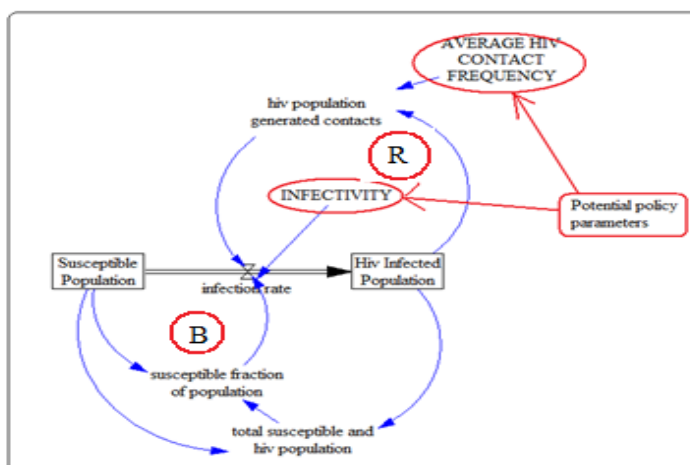


Figure 5.7. Potential Policy Parameters.

Figure 5.7 shows our policy parameters. In principle, they are manageable, and it is hoped that by changing them, the behavior of the model will be different; i.e., it is possible to improve the model's behavior.

Loop B

In our model, two parameters affecting loop B are infectivity and average HIV contact frequency. Average HIV contact frequency affects loop B (indirectly) and infectivity affects loop B (directly). It is possible to “vary” the populations that influence the feedback loops. For human populations, however, that does not typically translate into real-world options. Note, that “eradication” of non-human carriers of disease is often considered a policy option.

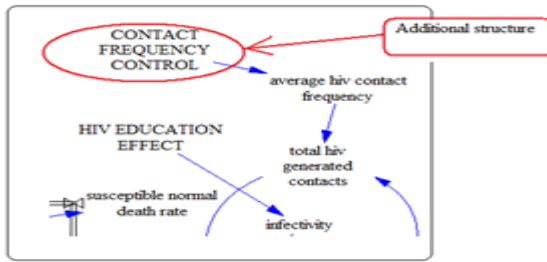
Loop R

In our model, the same parameters affect loop R. Average HIV contact frequency affects loop R (directly) and infectivity affects loop R (directly).

Both parameters affect both loops. And it is not possible in the real world to vary the parameter effects on one loop without also varying the effects on the other loop. Therefore, it is not necessary in our case to examine the separate sensitivity of each loop to changes in parameter values. (In some cases, it may be important to examine loops separately in the policy search for ways to strengthen “good” loops and weaken “bad” loops.)

Sensitivity analysis test- average HIV contact frequency

Heterosexual contact has been identified as the main mode of transmission in Tanzania. We have assumed the average contact frequency to be 19.25 unsafe sexual contacts per year (DHS, 2004). We assume that many people do not know their HIV status so the average unsafe sexual contacts estimated will involve contacts with HIV infected people. To test the sensitivity of the model to changes in this parameter we initially simply run a simulation with three different values. As Sterman (2000) suggested we should be able to test the sensitivity over a wide range. We will now conduct the sensitivity of the level of HIV infected persons to variations in the number of HIV contact frequency. During this test we assume that prenatal transmission fraction equal to zero and we will use the Model 6 with an additional structure. We will perform three tests with 10, 15 and 20 average HIV contact frequency per year.



Additional structure to the Model 6 (contact frequency control)

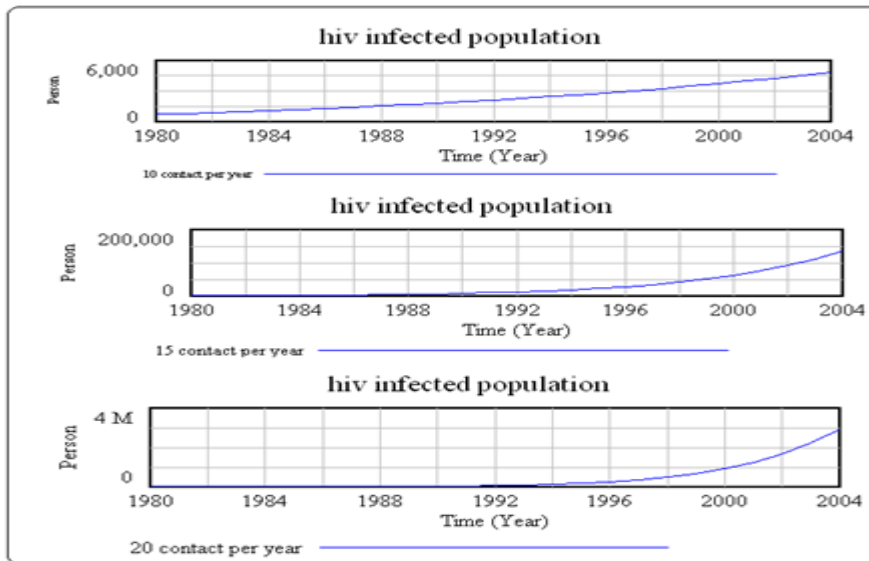


Figure 5.8. Sensitivity to the level of HIV infected population to variations in the average HIV contact frequency.

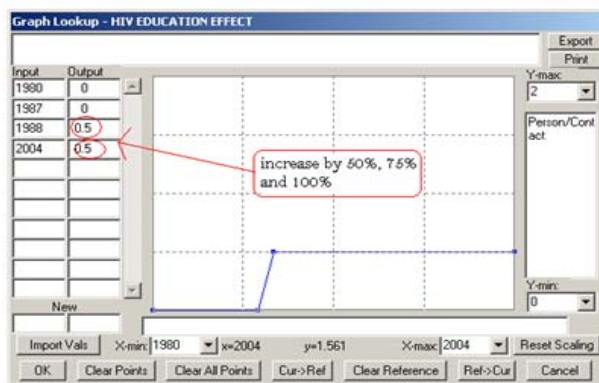
Figure 5.8 shows that with average of 10 HIV contact frequency per year, the HIV infected population grows to reach a peak of around 5,000 by the year 2004. If we increased the average HIV contact frequency to 15 per year, the HIV infected population grows to reach a peak of around 150,000 by the year 2004. Finally, if we increase the average HIV contact frequency to 20 contacts per year, the HIV infected population grows to reach a peak of around 3 million by the year 2004.

The test indicates that controlling the unsafe sexual contact have the potential for dramatically reducing the spread of the HIV.

Sensitivity analysis test- Infectivity.

In our model, infectivity is determined by a biological and social factor and we set it to be 0.03 although as we have indicated in the model building process, the infectivity may fall as people become aware of the risk of HIV and ways in which it can be transmitted; that is, some people may reduce or abstain from risky sexual contacts or use condoms. We have estimated a reduction in HIV infectivity of about 10 per cent in the year 2004 due to the ongoing HIV education and awareness program.

We will now examine the sensitivity of the HIV infected population to the variations in the infectivity. We will assume a maximum reduction in the infectivity by 50%, 75% and 100% from the year 1988 to 2004 due to HIV education program and we will keep the prenatal transmission fraction at zero value throughout. We will conduct the test using Model 6.



Structure showing the value to be changed in the year 2004

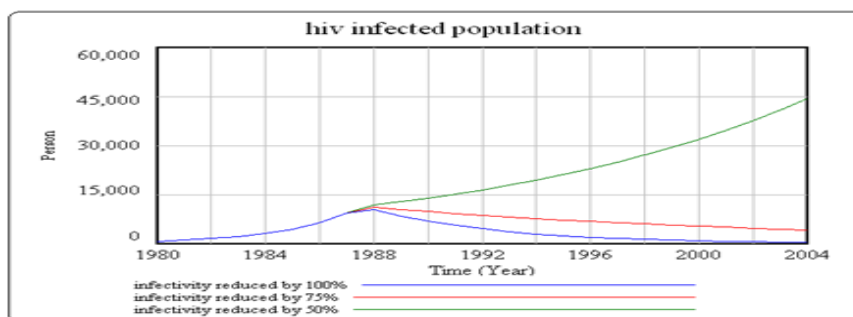


Figure 5.9. Sensitivity of the level of the HIV population to variations in the infectivity.

Figure 5.9 shows that if infectivity is reduced by 50% from the year 1988 to 2004, the HIV infected population grows to reach a peak of around 45,000 by the year 2004. If the infectivity is reduced by 75%, the HIV infected population falls to less than 15,000 by the

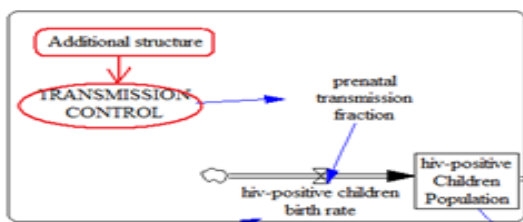
year 2004. Finally, if HIV education program has 100% effect on infectivity from the year 1988 there will be no more HIV infections and HIV infected population falls to zero before year 2004.

Figure 5.9 shows that reducing the chances of transmitting the HIV during sexual contact (infectivity) have the potential dramatically reducing the spread of the disease.

The model is very sensitive to the two variables above infectivity and average contact frequency.

Sensitivity analysis test - Prenatal transmission fraction

We will now examine the sensitivity of the HIV-positive children population to the variations in the prenatal transmission fraction. We will reduce the prenatal transmission fraction by 25%, 50%, 75% and 100% from the beginning of the simulation. We will conduct the test using Model 6 with an additional structure.



Transmission control structure added to the Model 6.

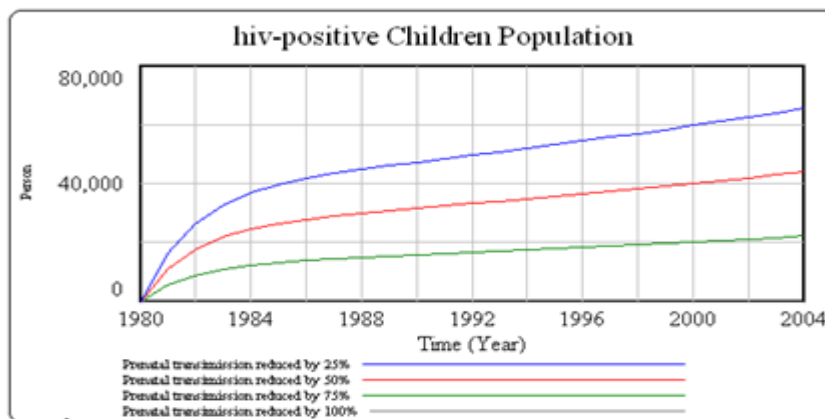


Figure 5.10. Sensitivity of the level of the HIV-positive children population to variations in the prenatal transmission fraction.

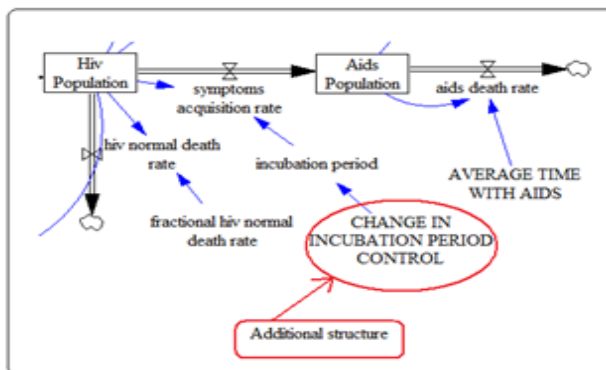
Figure 5.10 shows that if prenatal transmission fraction is reduced by 25% from the current value, the HIV-positive children population grows to reach a peak of around 75,000

by the year 2004. If the prenatal transmission fraction is reduced by 50%, the HIV-positive children population grows to reach a peak of around 45,000 by the year 2004. If the prenatal transmission fraction is reduced by 75%, the HIV-positive children population grows to reach a peak of around 20,000 by the year 2004. Finally, if the prenatal transmission fraction is reduced by 100%, the HIV-positive children population remains at zero level throughout.

Figure 5.10 shows that reducing the chances of transmitting the HIV from the mother to child have the potential dramatically reducing the spread of the disease to the children population. The model is not sensitive to the prenatal transmission fraction parameter.

Incubation period.

We will now examine the sensitivity of the HIV-positive children population to the variations in the incubation period. The current value of the incubation period equals 8 years. We will reduce and increase the incubation period by 25%. We will conduct the test using Model 6. We assume prenatal transmission fraction equals zero.



Change in incubation period control added to the Model 6.

Reduced by 25% * 8 incubation period equals 6 years

Increased by 25% * 8 incubation period equals 10 years

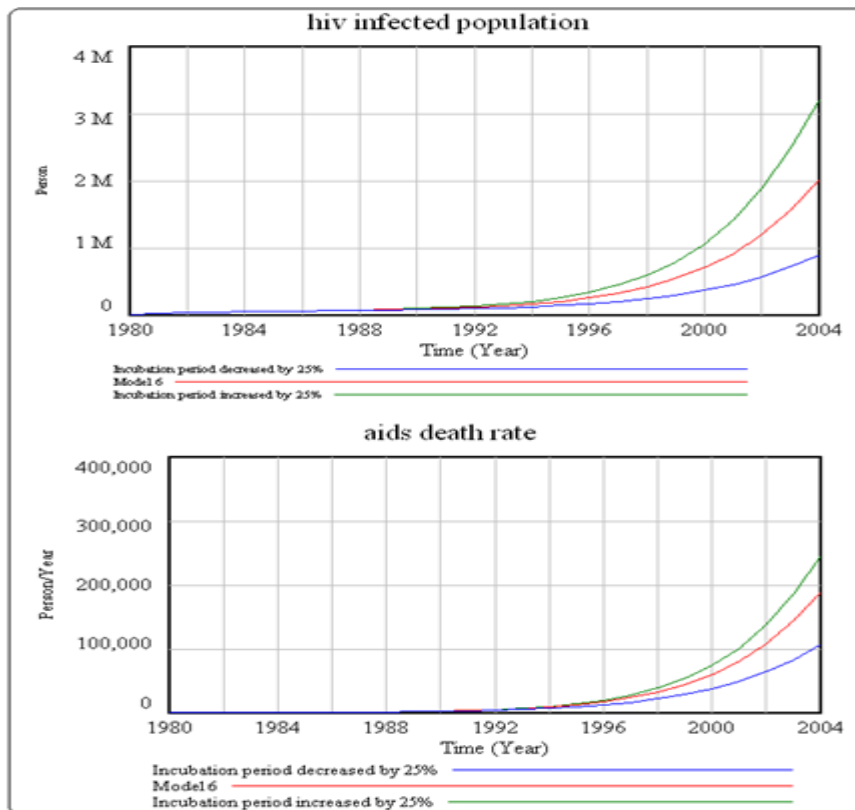


Figure 5.11. Sensitivity of the level of the HIV infected population and AIDS death rate to variations in the incubation period.

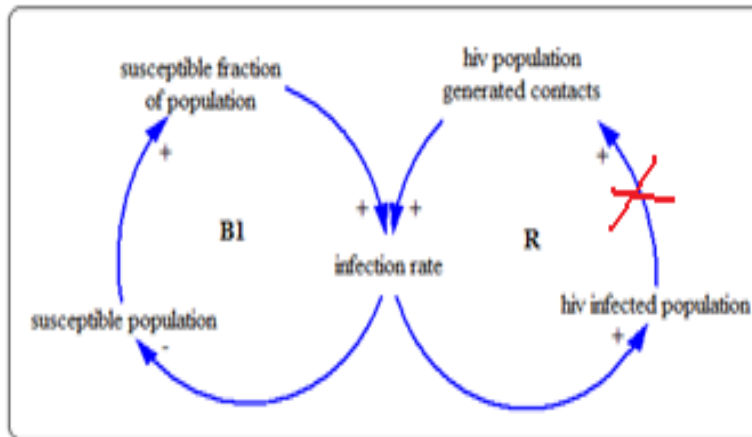
Figure 5.11 shows that if the incubation period is decreased by 25% from the current value, the HIV infected population grows to reach a peak of around 1 million by the year 2004. If the incubation period is increased by 25%, the HIV infected population grows to reach a peak of around 3 million by the year 2004.

Increasing the incubation period has also an immediate impact in reducing the mortality rate for the people living with AIDS. We assume no side effect from people living with HIV, i.e., increasing the infection rate.

Structure – Behaviour Tests

Cut the Reinforcing Loop

We start by describing the characteristics behavior during the early part of the simulation and in the last stages. We will use the loop to explain why the characteristic behavior changes. The model produces the exponential-like growth behavior and we believe that the following causal loop diagram is responsible for the behavior.



We will neutralize the R loop without affecting the B1 loop. We will do that by holding constant the average HIV contact frequency at 19.25 and eliminate feedback from HIV infected population. Therefore; HIV population generated contacts = (HIV infected population/HIV infected population) * average contact frequency.

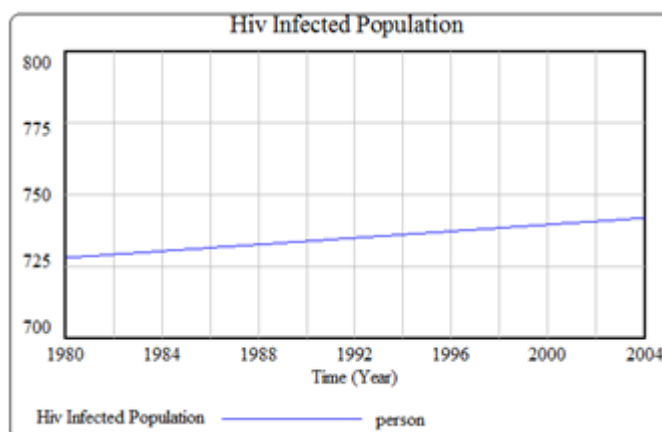


Figure 5.12. Cut the reinforcing loop

Figure 5.12 shows that by cutting the R loop, the exponential growth-like behaviour is replaced by goal seeking behaviour and there is no evidence of exponential-like growth. The

R loop was responsible for the exponential-like behaviour in previous simulations. We performed another tests by cutting loop B1 and also cutting both loops and in all these tests the behaviour of the model changed, but the results are not presented here.

Summary

Based on the purpose of testing, we implement several tests in this chapter. After all kinds of tests, we can say, in general, the model is useful for the purpose of showing the dynamics of the HIV transmission in Tanzania.

We will present, in the next chapter, some of the policy analysis we performed.

6. POLICY ANALYSIS

The focus of the study is policy development and the final step in our model building process is the most interesting and the most important to use the model to develop policies and test how changes in policies might improve the simulated behaviour of the system. “Policy design includes the creation of entirely new strategies, structures, and decision rules” (Sterman, 2000).

Today we do not have any vaccine or cure for HIV/AIDS and with this in mind, it becomes important for policy makers to find other ways to stop or at least to slow down the future spread of the HIV epidemic. A principle deficiency in policy makers’ mental models is the tendency to think of cause and effect as local and immediate. But in dynamically complex systems, cause and effects are distant in time and space. “Most of the unintended effects of decisions leading to policy resistance involve feedbacks with long delays, far removed from the point of decision or problem symptom” (Sterman, 2000 p.91). We believe that implementing policies to reduce future increase in the stock of HIV population will eventually reduce the growth trend on the stock of AIDS population. One of the obvious goals will be to prevent more HIV cases. Another goal will be to prolong life for those who are infected. We will start our policy tests policy by applying one test at a time and combined policies.

Policy test 1: Reduce HIV infectivity and average HIV contact frequency.

HIV education and awareness program.

The first type of intervention is designed to decrease the probability of being infected during sexual contacts and reduce unsafe sexual behaviour. In this case, we consider a scaled-up version of the Ugandan experience. Through HIV educational campaigns, Uganda appears to have decreased most aspects of unsafe sexual behaviour, and estimates suggest that the HIV prevalence there has gone down substantially (details of the intervention can be found in (Hogle, 2002). The Ugandan experience involves the ABC program (Abstinence, Be faithful and or use Condom), promotion of voluntary counselling and HIV testing (centres and services) and the provision/storage and distribution of HIV test kits. We also consider the current HIV education and awareness in Tanzania which we assume to have decreased infectivity by 5%. The current HIV education and awareness should be extended to reach more people in the country side for example there has been an outcry in Tanzania over a

woman who was badly injured by her husband after she took an HIV test which is being encouraged nationwide <http://news.bbc.co.uk/2/hi/africa/7117184.stm>. The above example shows that there are still some people who do not understand the risk of HIV therefore they will be able to prevent themselves from being infected. We will perform this test by assuming that the HIV education and awareness program are extended to the rural areas and that the government and other agencies will provide quality condoms either for free or at affordable price to the general population. In addition to that, we assume that the churches in Tanzania will reach a point where they will allow their members to use condoms to prevent HIV infection. Currently, churches do not accept the issue of educating people about the use of condom. We assume the HIV education and awareness program will reduce the HIV infectivity by 50% and 75% from the year 2005. We will perform this test using Model 6 with the additional structure and in order to see the impact clearly we assume that children do not use condom therefore we assume zero prenatal transmission fraction.

In this case we assume that Tanzania will adopt the Cambodia and Thailand experience, where the infectivity has decreased to more than 75%, apparently as a result of successful application of the 100% condom use approach” (UNAIDS/WHO, 2007).

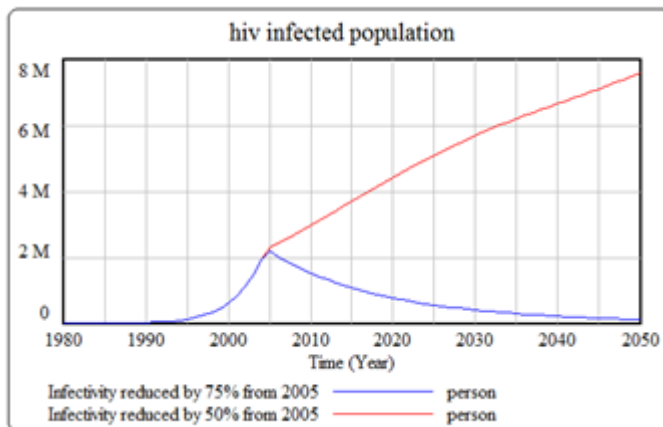
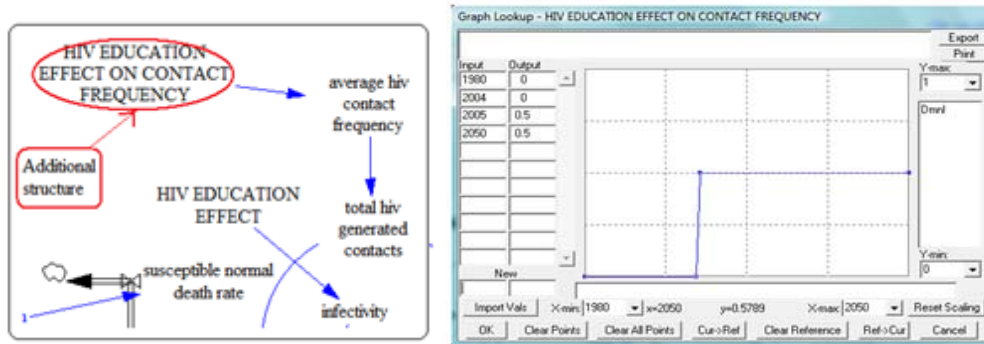


Figure 6.1a. HIV education and awareness effect on Infectivity

Figure 6.1a shows that increase in the HIV education and awareness program among the population aged between 15 and 59 have dramatic impact on reducing the population of people living with HIV. A decrease of about 75% from year 2005 in infectivity has an impact of reducing the HIV infected population to almost zero by the year 2050.

Following the Ugandan experience, we assume further that proper HIV education and awareness program will also reduce the average HIV contact frequency by 50% from year

2005. That means some people will reduce practising extramarital sexual contacts which is very common in some areas, or some people will prefer to have only one sex partner. We will add the following structure to the Model 6.



Additional structure in the Model 6.

We will now present the behaviour of the Model

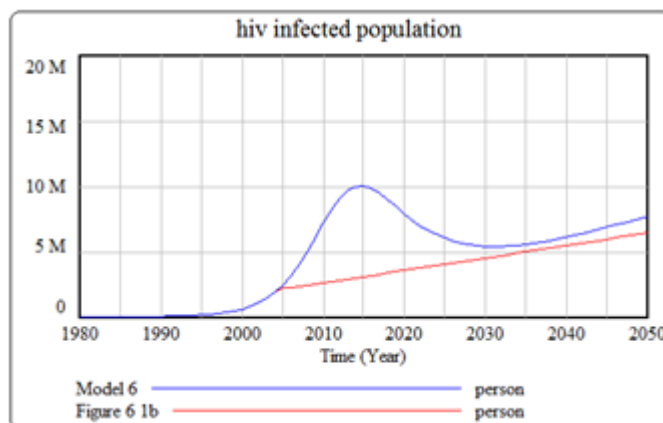


Figure 6.1b. HIV education and awareness Model 6 (baseline), and 50% reduced HIV contact frequency.

Figure 6.1b shows that reduced HIV contact frequency due to the HIV education and awareness program has an effect on the HIV infected population. The graph showing the effect grows at the lower level than in the Model 6 throughout from year 2005. We assume that proper HIV education and awareness program will reduce both infectivity and the average HIV contact frequency by 50%.

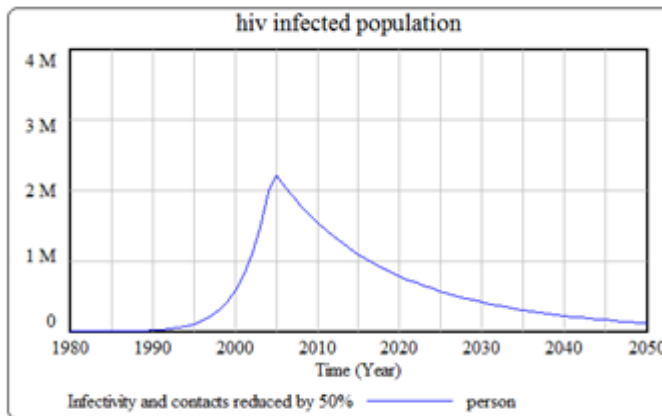


Figure 6.1c. Combined policy: HIV education and awareness program effect on the Infectivity and average HIV contact frequency.

Figure 6.1c shows that increase in the HIV education and awareness programs will have an impact on the infectivity and on the average HIV contact frequency. A decrease of about 50% from year 2005 has an immediate effect in reducing the stock of HIV infected population both in the long and short run.

Policy test 2: Reduce the mortality rate.

Highly Active Anti-Retroviral Therapy (HAART).

Triple combination antiretroviral therapy (also known as Highly Active Anti-Retroviral Therapy (HAART)) is currently the most appropriate treatment for advanced HIV disease and AIDS in the countries of Europe and the US (WHO, 2000). It works by attacking HIV at different points in its life cycle so as to prevent it reproducing. Three classes of antiretroviral drugs are involved and the patient is required to take these at regular points throughout the day. The introduction of this therapy, together with the viral load test, represents an encouraging milestone in the treatment of HIV/AIDS. Before the advent of the viral load test in clinics, both the health of people with HIV and the effect of treatments were assessed by monitoring changes in CD4 cell count, as well as physical signs and symptoms.

Prior to the introduction of the HAART treatment, no treatment regime had succeeded in satisfactorily stopping the progress of HIV disease (WHO, 2005). For many HIV-infected people, HAART can reduce the viral load in their blood to a level undetectable except by the most sensitive tests recently developed. Therefore, many HIV patients were switched from their original therapy over to HAART treatment almost as soon as HAART treatment was shown to be effective (WHO, 2000).

“The influence of the HAART treatment on the HIV epidemic has both positive and negative aspects” (Dangerfield *et.al.*, 2001). On the one hand, the therapy is effective in delaying or halting disease progression. Logically, this will have two consequences. First, the number of diagnosed AIDS cases will exhibit a sharp decrease consequent upon the widespread adoption of the HAART treatment. On the hand these infectious people will increase and if they do not change the behaviour they will increase the infection rate even further.

The HAART treatment was introduced in Tanzania in the year 2000 and by year 2004 only around 100,000 people are reported to be under HAART treatment (TACAIDS, 2005). Dangerfield *et al.*, (2001) suggested that the stock of infected population be divided into 5 stocks in order to test the impact of HAART on different HIV stages but to obtain data for every HIV stage in Tanzania is almost impossible may be in the future (interview, 2007). We will therefore introduce the impact of HAART to the same structure we have built in our model. We assume that the Tanzanian government and other agencies will be able to provide HAART treatment to more HIV infected people and this is only possible if people have access to HIV testing and counselling centres which are currently not available in some rural areas or not well equipped. We assume the government and other agencies to follow the Ugandan experience by proving the HIV test kits to all Heath centres. We assume further that once a person is classified as having AIDS, that does not change, regardless of any (or vast) clinical improvement in their condition.

To test the HAART policy the model’s parameters have been set as in the base case (Model 6). First, we assume that from the beginning of 2005 that the incubation period will increase by 50% and 75%. This percentage is based on the assumption that 50% and 75% of people living with HIV have access to HAART treatment. It takes almost 3 months for HIV patient to recover after start receiving HAART treatment (Interview). We will perform this test by adding structure on the Model 6 as shown below.

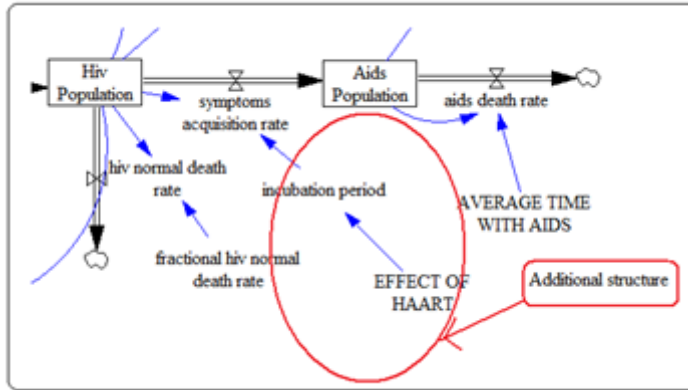


Figure 6.2. HAART structure

The development of the total HIV population is shown in the figure below.

50% increase * 8 years = 12 years

75% increase * 8 years = 14 years

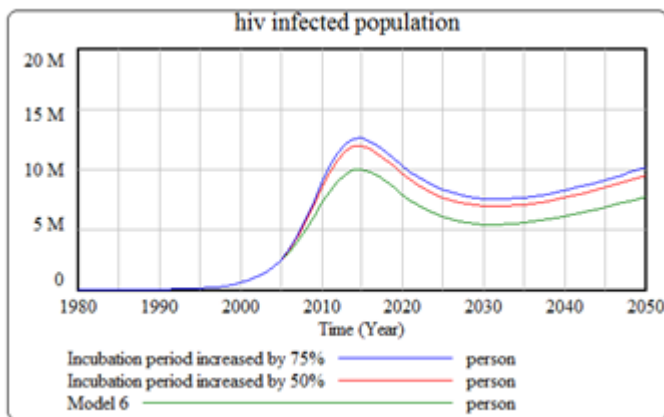


Figure 6.3. HAART treatment increase incubation period by 50% and 75%

From Figure 6.3 we can observe that right after the introduction of the HAART treatment in year 2005, the total number of people living with HIV increased because HAART has improved their life. We should remember that these are people who do have AIDS symptoms. Increase in HIV means a corresponding decrease in AIDS population see Figure 6.4 below.

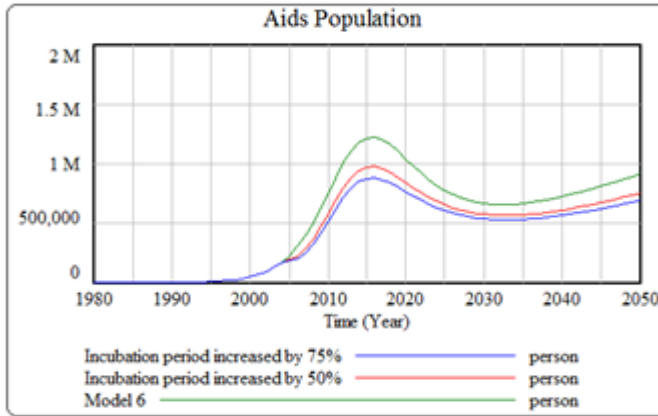


Figure 6.4. HAART treatment increased incubation period by 50% and 75% effect on the AIDS Population.

Figure 6.4 shows that as the incubation period increase by 50% and 75% the AIDS death rate falls, as we would expect. This causes the total HIV/AIDS population to increase faster than in the base case, owing to the fact that the death rate drains a lower number of subjects from these stocks. See figure below

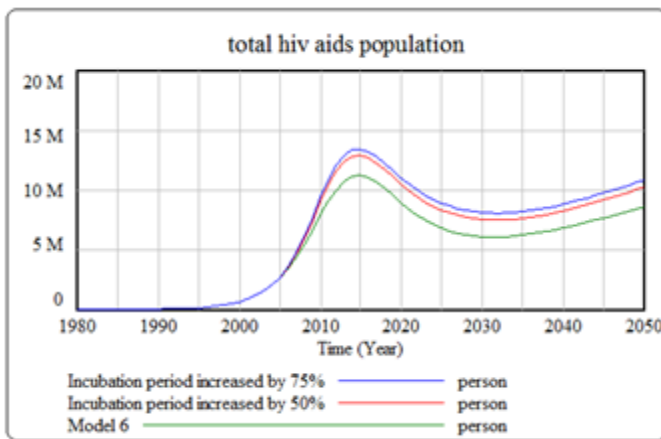


Figure 6.5. HAART treatment increased incubation period by 50% and 75% effect on HIV/AIDS population

This increase in the total HIV/AIDS population is accompanied by a parallel increase in the infection rate, due to the fact that a higher number of infectious people will now generate a higher number of contacts and will eventually infect a higher number of persons.

In this test, HAART treatment allows the infected individuals to live longer and for some to resume risky sexual behaviours and infect more people thus maintaining a high number of infectious individuals in the population.

We have tried the HAART treatment policy test with different values of incubation period and the result would not change qualitatively. However the sensitivity of the model to

the choice of this parameter (duration of infectivity) is only numerical, and there are absolutely no behaviour mode or policy sensitivity.

We conclude that HAART treatment should be extended to help more infected people because it allows the infected individuals to live longer.

Policy test 3. Reduce transmission of HIV from mother to child.

Nevirapine PCMTC regimens

The third policy that will be tested concerns the use of the Nevirapine, a drug particularly efficient in reducing the risk of virus transmission from mother to child. The children of HIV positive mothers can be HIV positive or not depending on several factors, as hygienic conditions during the delivery of the kid or as the duration of the breast-feeding period, among others. In most of cases this is strongly depending on the quality of the medical assistance, before and after the delivery. The overall probability of transmission of the virus from mother to child is represented by the prenatal transmission fraction. The reference value for this parameter has been estimated to be 0.032. In order for an infected pregnant mother to be treated with Nevirapine regimes, the mother should be tested for HIV at the early stage of the pregnancy. In Tanzania, especially in the rural areas, most of mothers do not know about their HIV status, therefore they are not undergoing the treatment. In some cases some mothers knows about their HIV status but they do not have access to Nevirapine regimes and even if they have access they cannot follow it properly. We assume that the Tanzanian government and other agencies will be able to test more pregnant mothers for HIV and provide the required treatment on time.

To test the effect of the introduction of a Nevirapine treatment on pregnant women, we assume a reduction of the prenatal transmission fraction by 50% (from 0.32 to 0.016) from the year 2005. In reality, if well conducted, this treatment can reduce much more than 50% of the transmission fraction on a single woman, but here a reduction of 50% has been considered assuming that only a part of the pregnant women can afford this treatment and that not all of them will follow it perfectly. Here we assume that the government will follow the Ugandan experience.

For this new simulation only the prenatal transmission fraction parameter has been modified, while the rest of the parameters setting corresponds to the one used to represent the Model 6 with the additional structure.



Modified prenatal transmission fraction added to the Model 6

Figure 6.6 shows the immediate results of the introduction of such a treatment.

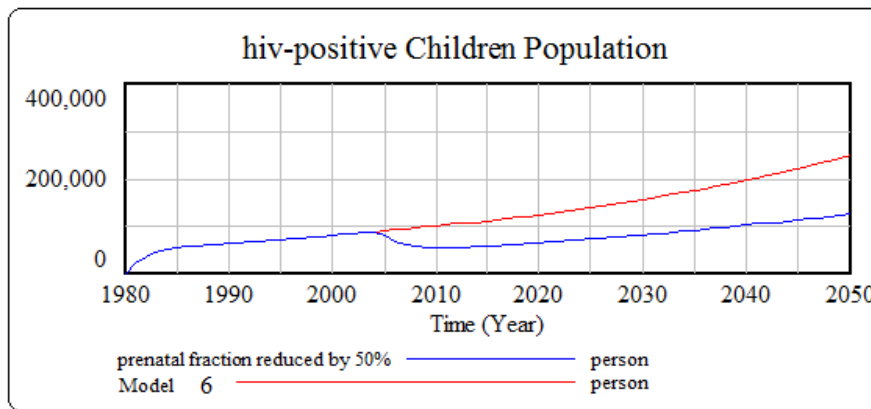


Figure 6.6. Reduced prenatal transmission fraction by 50%.

Figure 6.6 shows the behaviour of the HIV-positive children. As it clearly appears, the implementation of this kind of Nevirapine, strongly reduced the number of children born with HIV. The magnitude of the effect is particularly relevant and the simulated number of HIV-positive children in the year 2050 is in this case about half of what is shown without this treatment.

Our conclusion is that the use of nevirapine will have a major impact on reducing the number of children contracting the disease through prenatal transmission and that means a corresponding decrease in the deaths of the HIV-positive children.

Policy test 4. Reduce infectivity AND average HIV contact frequency AND transmission of mother to child.

Combined policies HIV education and awareness program and NEVIRAPINE

We assume a combination of two policies; HIV education and awareness program, and nevirapine treatment for the pregnant infected mothers. We assume the implementation

of policies from year 2005. We assume a decrease of about 50% in the infectivity and average HIV contact frequency and that Nevirapine treatment will reduce prenatal transmission fraction by 50%.

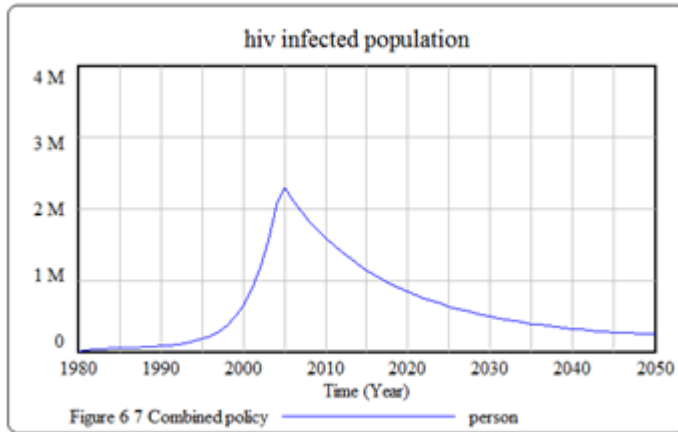


Figure 6.7. Combined policies HIV education and awareness program AND Nevirapine treatment.

Figure 6.7 shows that combination of two policies has dramatically impact in reducing the HIV infected population both in the short and long term.

Policy test 5. Reduce infectivity AND average HIV contact frequency AND mortality rate AND transmission of HIV from mother to child.

HIV education and awareness, HAART, and NEVIRAPINE.

We assume a combination of three policies; HIV education and awareness program, HAART treatment on the HIV infected adults, HAART and Nevirapine treatment for the infected pregnant mothers. We assume the implementation of policies from year 2005. We assume that HIV education and awareness program will reduce the infectivity and average HIV contact frequency by 50%, HAART treatment will increase the incubation period by 50% and Nevirapine will reduce prenatal transmission rate by 50%.

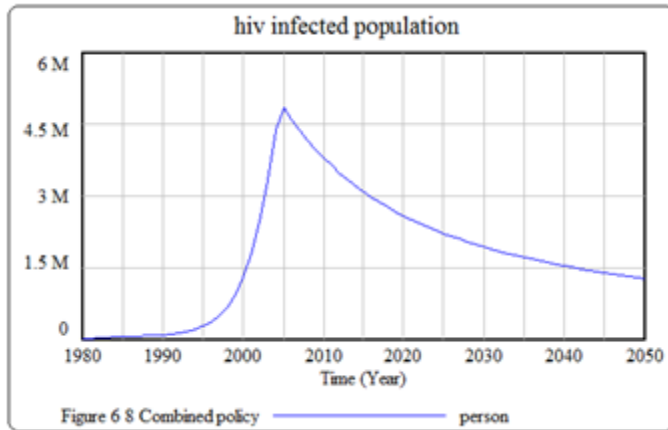


Figure 6.8. Combined policies

Figure 6.8 shows that combination of three policies has good efficiency of reducing the impact of HIV epidemic by prolonging life to those living with HIV and reducing the future infection.

Policy conclusion

With the current model setting it appears that HAART alone, even when the rate of treatment is increased, cannot tackle the HIV epidemics on its own. In fact, HAART allows the infected individuals to live longer and for some to resume risky sexual behaviours and infect more people thus maintaining a high number of infectious individuals in the population. The combination of the HIV education and awareness program and Nevirapine shows the best results in reducing the new infections for both adults and children. HIV education and awareness program have also been promoted as a remedy against HIV/AIDS epidemic (Green, 2003). Indeed HIV education and awareness program shows in our model a high efficiency in the short term but we assume only a transitory positive effect of the campaign on unsafe sexual behaviours.

7. CONCLUSION:

Contribution and Major Findings

This thesis has illuminated the complexity of HIV and modes of transmission from both adults and HIV-positive mothers' to child perspectives. It has investigated the biological and clinical aspects of HIV as well as the government's efforts to combat the epidemic. This thesis used an SD adaptation of standard diffusion model. It has generated a step by step system dynamics model that helps in understanding the dynamics of the HIV transmission. In this thesis, Model 6 is our last model in the building process and that replicate the reference mode. Based on the purpose of testing, we implemented several tests in this thesis. In general the model is useful for the purpose of showing the dynamics of the HIV transmissions. The study provides insight into ways in which together the Tanzanian government and other operating agencies would combat the epidemic. The methodology used in the study provides a strategic tool that can be used to predict spread of epidemic and effectiveness of general prevention and treatment policies.

To our knowledge our model is the first system dynamics model in Tanzania which incorporates the dynamics of HIV transmission, be able to replicate the historical data, and test different policy scenarios.

Our model allows the testing of several combinations of various policies. The strategy consisting of mixing several combinations seems to be the best and appropriate way to tackle HIV/AIDS epidemic in Tanzania. Combination of two policies HIV education and awareness program and Nevirapine treatment has good efficiency of reducing the impact of HIV epidemic by reducing the future infection for both adults and children. The study underscores the importance of adopting the Ugandan experience through HIV education and awareness program and the government and churches should adopt similar policies on HIV prevention.

Limitations and future research

The "Model 6" in this study is built in a simplified way with a simple structure and many simplified assumptions. For the future work, some assumptions like combined population for rural and urban can be relaxed to make the model more convincing. Population should be disaggregated between female and male in order to keep track of both groups; in

this model we did not get enough details about the population distribution age cohort that could allow us to build such a model but we consider expanding the model in the future.

We will also consider to change the infectivity parameter in future as mentioned by Kaplan (1990) that assuming a constant HIV infectivity per partner is reasonable, while assuming a constant infectivity per general population (sex act) is not. The first contacts largely influence the overall infectivity risk. The major recommendation related to model structure springing out of the present work is that future model should disaggregate the stock of infected population into 5 stages (Dangerfield, 2001) to allow the utilization of different infectivity and contact frequency in different stages of infectiousness. We could not implement this idea in our model because of lack of data.

Recent studies suggest that there is a close relationship between level of education and availability of health services and risky contact frequency (WHO, 2005). A study in South Africa shows that the infection rate among educated people is slowing down (WHO, 2007) and that people with better access to basic health service are more aware of the risk of HIV and ways in which it can be transmitted. We would recommend that the future model should consider the impact of level of education, access to health service on the contact frequency.

Finally, we would gladly work with others who are interested in extending this model and improving it.

References:

- Anderson, R. M. & May, R. M. (1988). Epidemiological parameters of HIV transmission. *Nature* 333, vaccination. *Science* 215, 1053-1060.
- Auvert, B. (1998). The Auvert approach : a stochastic model for the heterosexual spread of the human immunodeficiency in The AIDS epidemic and its demographic consequences, Proceedings of the United Nations/World Health Organization workshop on modeling the demographic impact of AIDS in Pattern II countries, New York, 13-15 ST/ESA/SER.A/119
- Bagni, R. Berchi, R. & Cariello, P. (2002). A comparison of simulation models applied to epidemics. *Journal of Artificial Societies and Social Simulations* 5(3).
- Barre-Sinoussi, F. *et al.*, (1983). Isolation of a T-Lymphotropic retrovirus from a patient at risk for Acquired Immune Deficiency Syndrome (AIDS) *Science* 220, 868 – 871.
- Bernstein, R. S. Sokal, D. C. Seitz, S.T. Auvert, B. Stover, J. & Naamara, W. (1998). Simulating the control of a heterosexual HIV epidemic in a severely affected East African city, *Interfaces* 28; 101-126
- Brandeau, M. L. (1998). AIDS policy modeling. A social role for operations research, *Ricerca Operativa* 27: (81–82):5–33.
- Brandeau, M. L. Lee, H. L. & Owens, D. K. (1990). Policy analysis of human immunodeficiency virus screening and intervention: A review of modeling approaches. *AIDS and Public Policy Journal* 5(2):119–131.
- Brookmeyer, R. & Gail, M. H. (1994). AIDS epidemiology: a quantitative approach. New York: Oxford University Press 36-50.
- Booz -Allen, (2005). The AIDS Epidemic: Integrating System Dynamics and Gaming for Strategic Simulation, McLean, USA
- Bulatao, R. A. (1989). The Bulatao approach: projecting the demographic impact of the HIV epidemic using standard parameters in The AIDS epidemic and its demographic consequences, Proceedings of the United Nations/World Health Organization workshop on modeling the demographic impact of AIDS in Pattern II countries, New York, 13-15 ST/ESA/SER.A/119
- Blower, S. M. & Gerberding, J.L. (1998). “Understanding, predicting, and controlling the emergence of drug-resistant tuberculosis: a theoretical framework”. *Journal of Molecular Medicine*, V 76, pp 624-636.
- Cameron, D. W. Simonsen, J. N. D'Costa, L. J. *et al.*, (1989). Female to male transmission of human immunodeficiency virus type 1: risk factors for seroconversion in men. 2: 403-07.
- Center for Disease Control and Prevention. (1998). Guidelines for the use of antiretroviral agents in HIV-infected adults and adolescents. *Morbidity and Mortality Weekly Report* 47(RR-5):42-82.
- _____. (September 1982) "Current Trends Update on Acquired Immune Deficiency Syndrome (AIDS) --United States".

- _____. (1985). Revision of the case definition of the acquired immune deficiency syndrome for national reporting – U.S. MMWR 34, 373-375
- _____. (1987). Revision of the CDC surveillance case definition for AIDS. MMWR 36, 3S-15S
- _____. (1993). Revised Classification System for HIV Infection and Expanded Surveillance Case Definition for AIDS Among Adolescents and Adults". MMWR
- _____. (2003). HIV/AIDS Surveillance Report, (Vol. 15).
- _____. (2003). "What causes AIDS?", December Updated Report
- Chin, J. & Sonnenberg, F. (1991). "The epidemiology and projected mortality of AIDS in the United Republic of Tanzania". Background paper prepared for Tanzania AIDS assessment and planning study, Washington DC: World Bank.
- Cooley, P. Myers, L. & Hamill, D. (1996). A meta-analysis of estimates of the AIDS incubation distribution, *European Journal of Epidemiology* **12** (3), 229-235
- Clark, R. (1988). System dynamics and modeling. INFORMS, Boston
- Clemens J, *et al.* (1996). Evaluating new vaccines for developing countries: Efficacy or effectiveness? *The Journal of the American Medical Association* **275**(5): 390–397
- Dangerfield, B. (1992). The system dynamics modeling process and DYSMAP2. *European Journal of Operational Research* **59**(1): 203–209
- Dangerfield, B. Fang, Y. & Roberts, C. (2001). Model based scenarios for the epidemiology of HIV/AIDS: the consequences of highly active antiretroviral therapy. *System Dynamics Review*; **17**(2):119-150
- _____ (2002). Model-based scenarios for the epidemiology of HIV/AIDS: the consequences of highly active antiretroviral therapy. *System Dynamics Review* (in press)
- David, W. L. & Ralph, L., (2002). Understanding the Psychosocial Dynamics of HIV/AIDS Prevention and Care in the Community: Base Case Model Finding and Implications. NY. US
- De Vincenzi I, for the European Study Group in Heterosexual Transmission of HIV. A longitudinal study of human immunodeficiency virus transmission by heterosexual couples. *N Engl J Med* **331**: 341-46.
- Dray-Spira, R. *et al.*, (2000). "Prevention of infectious complications of paediatric HIV infection in Africa." *Aids* **14**(9): 1091-9
- Duerr, A. Xia, Z. Nagachinta, T. Tovanbutra, S. Tansuhaj, A. & Nelson, K. (1994). Probability of male-to-female HIV transmission among married couples in Chiang Mai, Thailand. 10th International Conference on AIDS, Yokahama, Japan

- Ferry, B. M. Carael, A. Buve, B. Auvert, M. Laourou, L. Kanhonou, M. De Loenzien, E. Akam, J. Chege, & F. Kaona, (2001) "Comparison of Key Parameters of Sexual Behavior in Four African Urban Populations with Different Levels of HIV Infection," *AIDS*, XV, S41–50.
- Forsythe, S. S. (1998). The affordability of antiretroviral therapy in developing countries: what policymakers need to know. *AIDS* **12**: (Supplement 2): 11-18.
- Ford, A. (1999). *Modeling the Environment* (paperback ed.). Washington, D.C.: Island Press.
- Forrester, J. W. (1968). *Principles of Systems*. Cambridge, MA,: The MIT Press
- _____. (1969). *Urban Dynamics* Pegasus Communication, Waltham, MA
- _____. (1971). *Principles of Systems*. Cambridge, Mass. Wright-Allen Press, Inc.,
- _____. (1975). *Collected Papers of Jay W. Forrester*. Cambridge, Massachusetts: Wright-Allen Press Inc.
- _____. (1976). Business structure, economic cycles, and national policy. *Futures* **8**: 195-214
- _____. (1979). An alternative approach to economy policy: macrobehavior from micro-structure. In *Economic Issues of the eighties*, Kamrany NM, Day RH (eds). Johns Hopkins University Press: Baltimore, MD; 80-108.
- _____. (1992). "From the Ranch to System Dynamics: An Autobiography," In: Arthur G. Bedeian, ed., Management Laureates: A Collection of Autobiographical Essays. Volume 1 of 3. Greenwich, CT: JAI Press.
- Forrester, J. W. Mass, N. J. & Ryan, C. (1980). The system dynamics national model: understanding socio-economic behavior and policy alternatives. *Technology Forecasting and Social Change* **9**: 51-68
- Forrester, N. B. (1982). A dynamic synthesis of basic macroeconomic theory: implications for stabilization policy analysis. PhD dissertation, Massachusetts Institute of Technology, Cambridge, MA.
- Flessa, S. (1996). HIV/AIDS care in Tanzania: *Planning and policies based on sound data rather than speculation and moral principles offers the only hope for a desperate situation*, Volume 23 number 3.
- Flessa, S. (2003). Decision support for AIDS control programmes in Eastern Africa, Nurnmberg, Germany
- Goedert, J. J. Eyster, M. E. Biggar, R. J. & Blattner, W. A. (1987). Heterosexual transmission of human immunodeficiency virus: association with severe depletion of T-Helper lymphocytes in men with haemophilia. *AIDS Res Human Retrov* **3** #4, 355-361.
- Gray, et al. & and the Rakai Project Team, (2001) "Probability of HIV-1 Transmission per Coital Act in Monogamous, Heterosexual, HIV-1 Discordant Couples in Rakai, Uganda," *The Lancet*, CCCLVII 1149–1153.

- Green, E. C. (2003). Rethinking AIDS prevention. Praeger publishers, Westport, CT
- Grosskurth, et al., (1988). "Impact of Improved Treatment of Sexually Transmitted Diseases of HIV Infection in Rural Tanzania: Randomized Controlled Trial," *The Lancet*, CCCXLVI (1995), 530–536.
- Healy, M. J. R & Tillet, H. E. (1988). Short-term extrapolation of the AIDS epidemic. *J.R Stat Soc A* 151, 50-61
- Heidenberger, K. & Flessa, S. (1993). A system dynamics model for AIDS policy support in Tanzania. *European Journal of Operational Research* **70**(2):167–176
- Heidenberger, K. & Roth, M. (1998). Taxonomies in the strategic management of health technologies: the case of multiperiod compartmental HIV/AIDS policy models. *International Journal of Technology Management* **15**(3/4/5):336–358
- Hethcote, H. W. & Van Ark, J. W. (1992). *Modeling HIV transmission and AIDS in the United States*. Springer, Berlin Heidelberg New York
- Hogle, Janice. (2002). "What Happened in Uganda?" manuscript, USAID.
- Homer, J. B. & Hirsch, G. B. (2006). System Dynamics modeling for public health: background and opportunities. *American Journal of Public Health*. **96**(3): 452-458
- IBRD/World Bank. (2000). AIDS Assessment and planning Study, *Annual report*, Washington, D.C.
- Joint United Nation programme on HIV and AIDS (UNAIDS). (1998). Spread of HIV over time in sub-Saharan Africa, 1982–1997.
- Jourdain, G. N. *et al.*, (2004). "Intrapartum exposure to nevirapine and subsequent maternal responses to nevirapine-based antiretroviral therapy. " *N Engl J Med* **351** (3): 229-40.
- Kapiga, Saidi, and Iain Aitken, *Role of Sexually Transmitted Diseases in HIV-1 Transmission* (New York: Kluwer Academic Publisher, 2003).
- Kaplan, E.H.(1990). Modelling HIV infectivity: must sex acts be counted? *Journal of Acquired Immune Deficiency Syndromes* **3**:55-61
- Killewo, J. (1990) Prevalence of HIV-1 infection in the Kagera region of Tanzania: a population based study. *AIDS* **4**: 1081–1085
- Leshabari, S.C. (2008). Infant feeding in the context of HIV infection. (Mothers' experiences and programme implications for maternal and child health services in Tanzania). Dissertation for degree of philosophie doctor (PhD). University of Bergen, Norway
- Leroy, V. L. *et al.*, (2002). Uptake of infant feeding interventions to reduce postnatal transmission of HIV-1 in Abidjan, Cote d'voire. *HIV and Infant Feeding: A Compilation of Programmatic Evidence, 2004* (pp. 32- 33). USAID, UNICEF & QAP-URC. B. B.P. Koniz-Booher, A. De Wagt, P. Illiff & J. Willumsen
Accessed at: www.hiv.gov.gy/edocs/compilation_hivinfantfeeding.pdf

- Low, G. (1980). The multiplier-accelerator model of business cycles interpreted from a system dynamics perspective. In *Elements of the System Dynamics Method*, Randers J (ed.) MIT Press: Cambridge, MA 76-94.
- Longini, I. M. Clark, W. S. Haber, M. *et al.*, (1990). The stages of HIV infection: Waiting times and infection transmission probabilities. In: *Mathematical and Statistical Approaches to AIDS epidemiology*, C Castillo-Chavez, ED., *Lecture Notes in Biomathematics 83*, Springer-Verlag, Germany, p. 111-137.
- Mass, N. J. (1975). *Economic Cycles: An analysis of Underlying Causes*. Wright-Allen Press: Cambridge, MA.
- _____. (1980). Stock and flow variables and the dynamics of supply and demand . In *Elements of the System Dynamics Method*, Rander J (ed.). MIT Press: Cambridge MA; 95-112.
- Mastro, T. D. & Kitayaporn, D. (1998). HIV type 1 transmission probabilities: estimates from epidemiologic studies. *AIDS Res Hum Retrovirol* **14** (Suppl 3): S223-27.
- Mastro, T. D. Satten, G. A. Nopkesorn, T. Sangkharomya, S. & Longini, I. M. (1994). Probability of female-to-male transmission of HIV-1 in Thailand **434**: 204-07.
- Mayer, K. H. & Anderson, D. J. (1995). Heterosexual HIV transmission. *Infect Agents Dis* **4**: 273-84.
- Meadow, D. H. (1980). *The Unavoidable A Priori*, MA,: The MIT Press
- Meadow, D. H. Richardson, J. & Bruckmann, G. (1982). *Grouping in the Dark*. Chichester, England: John Wiley and Sons.
- Meadows, D.H. & Robinson, J. (1985). *The Electronic Oracle: Computer Models and Social Decisions*. Chichester, England: John Wiley and Sons.
- Merson, M. H. & Dayton, J. M. (2002). Overview of HIV prevention programs in developing countries in *Quantitative evaluation of HIV prevention programs*, eds E.H. Kaplan and R. Brookmeyer , Yale University Press.
- Ministry of Health Tanzania, (2002). National Policy on HIV/AIDS TACAIDS, December report
- Ministry of Health Tanzania, (2004). National Policy on HIV/AIDS TACAIDS, December report
- Ministry of Health Tanzania, (2005). NACP Surveillance Report. *Report Number 19*, Tanzania
- Moodley, D. Moodley, J. *et al.*, (2003). “A multicenter randomised controlled trial of nevirapine versus a combination of Zidovudine and lamivudine to reduce intrapartum and early postpartum mother-to-child transmission of human immunodeficiency virus type 1.” *J Infect Dis* **187**(5): 725-35.
- Moxnes, E. & Saysel, A. K. (2004). Misperceptions of Basic Climate Change Dynamics: Information Policies, WPSD 1/04, System Dynamics Group, University of Bergen.

- Moxnes, E. (2004). Misperceptions of Basic Dynamics: The Case of Renewable Resource Management. *System Dynamics Review* **20** (2): 139-162
- MUTAN, (1994). The AIDS-situation in Kilimanjaro. Vortrag, 15.03.1994, Masoka Management Training Institute, Moshi, Tanzania
- Nancy, R. David, F.A. Ralph, M.D.I. & William, A. S. (1983). Introduction to Computer Simulation. Reading, MA: Addison-Wesley, p.16.
- Newell, M. L. *et al.*, (2004). 'Mortality of infected and uninfected infants born to HIV-infected mothers in Africa: a pooled analysis', *The Lancet* 364(9441)
- Nicolosi, A. Leite, M. L. C. Musicco, M. Arici, C. Gavazzeni, G. & Lazzarin, A. (1994) For the Italian Study Group on HIV Heterosexual Transmission. The efficiency of male-to-female and female-to-male sexual transmission of the human immunodeficiency virus: a study of 730 stable couples. *Epidemiology* **5**: 570-75.
- O'Brien, T. R. Busch, M. P. Donegan, E. *et al.*, (1994). Heterosexual transmission of human immunodeficiency virus type 1 from transfusion recipients to their sex partners. *J Acquir Immune Defic Syndr Hum Retrovirol* **7**: 705-10.
- Padian, N. Shiboski, S. C. & Jewell, N. P. Female-to-male transmission of human immunodeficiency virus. *JAMA* **266**: 1664-67.
- Palloni, A. & Lamas, L. (1989). The Palloni approach: a duration-dependant model of the spread of HIV/AIDS in Africa in The AIDS epidemic and its demographic consequences, Proceedings of the United Nations/World Health Organization workshop on modeling the demographic impact of AIDS in Pattern II countries, New York, 13-15 ST/ESA/SER.A/119
- Pool, R. (1992). The Third branch of science debuts. *Science* 256 (3rd April): 44-47
- Population statistics information for Africa. (2001).
- Accessed at. <http://www.populstat.info/Africa/tanzanip.htm>
- Peterson, D. & Eberlein, R. (1994). Reality check: A bridge between systems thinking and system dynamics, *System Dynamics Review* **10**(2-3), 159-174
- Radzicki, M. (1993). A System Dynamics approach to macroeconomics. Guest lecture at the Department of Information Science, University of Bergen.
- Royce RA, Sena A, Cates W, Cohen MS. Sexual transmission of HIV. *N Engl J Med* **336**: 1072-78.
- Qu, W. & Gerald, Barney. O. (2000). T21 China: An Initial Application and Analysis. *Millennium Institute*, Arlington, VA, USA
- Quinn, *et al.*, (2000). "Viral Load and Heterosexual Transmission of Human Immunodeficiency Virus Type 1," *New England Journal of Medicine*, CCCXLII, 921-929. Ragni, Margaret, Lawrence Kingsley, Pamela Nimorwicz, Phalguni

- Rauner, M. & Brandeau, M. L. (2001). AIDS Policy Modeling for the 21st Century: An Overview of Key Issues. *Health Care Management Science*. **4**: 165-180
- ReVelle, C. F. Feldmann, F. & Lynn, W. (1969). An optimization model of tuberculosis epidemiology, *Management Sciences*, **16**, 190-210
- Richardson, G. P. (1995). “ Loop polarity, loop dominance and the concept of dominant polarity”. *System Dynamics Review*, V 11(1), pp. 67-88.
- _____. (1999). *Feedback Thought in Social Science and Systems Theory*. Pegasus Communications, Inc. Waltham, MA. US
- Richardson, G. P & Pugh, A. L. (1981). “*Introduction to system dynamics with DYNAMO*”. The MIT press, Cambridge, MA
- Richmond, B. (1987). “An academic user’s guide to STELLA. Software documentation”. High Performance Systems, Hanover, USA.
- Roberts, C. & B. Dangerfield. (1990). Modelling the epidemiological consequences of HIV infection and AIDS: A contribution from operational research, *Journal of the Operational Research Society* **41**(4), 273-289.
- Rowley, J. T. Anderson, R. M. & Ng, W. (1990). Reducing the spread of HIV infection in sub-Saharan Africa: some demographic and economic implications. *AIDS* **4**: 47–56
- _____. (1994) Modelling the impact and cost-effectiveness of HIV prevention efforts. *AIDS* **8**: 539–548
- Sengupta, K. Abdelhamid, T. K. & Bosley, M. (1999). “Coping with staffing delays in software project management: An experimental Investigation”. *IEEE Transactions on systems, Man, and Cybernetics, Part A*, v 29(1), pp 77-91.
- Soderberg, S. *et al.*, (1994). Prevalence of HIV-1 infection in rural, semi-urban and urban villages in South–Western Tanzania: estimates from a blood donor study. Unpublished
- Somi, R. G. (2005). Estimating and projecting HIV prevalence and AIDS deaths in Tanzania using antenatal surveillance data. Accessed at: <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1471785>
- Stanley, E. A. Seitz, S.T. Johnson, P. D. Way, P. O. & Curry, T. F. (1989). The United States Inter-Agency Working Group approach: The iwg model for the hereosexual spread of HIV and the demographic impact of the AIDS epidemic in The AIDS epidemic and its demographic consequences, Proceedings of the United Nations/World Health Organization workshop on modeling the demographic impact of AIDS in Pattern II countries, New York, 13-15 ST/ESA/SER.A/119
- Scott, S. (1993). Dealing with delay claims : a survey. *Int J Project Mngt* **11**: 143–153.
- Sterman, J. D. (1985). A behavioral model of the economic long wave. *Journal of Economic Behavior and Organization* **6**: 17-53.

- _____. (1989). Modeling managerial behavior: misperceptions of feedback in a dynamic decision making experiment. *Mngt Sci* **35**: 321–339.
- _____. (2000). *Business Dynamics: Systems Thinking and Modelling for a Complex World*. McGraw-Hill: Boston, MA.
- Stromblad, O & E. Zaar. (2005). HIV awareness and risk behaviour among men in rural Tanzania, Goteborg, Sweden
- Swager, S. J. Castillo-Chaves, C. & Hethcote, H.W. (1989). Statistical and Mathematical Approaches in HIV/AIDS Modeling. In: *Mathematical and Statistical Approaches to AIDS Epidemiology*, C Castillo-Chaves, Ed., Lecture Notes in Biomathematics 83, Springer-Verlag, Germany, 2-35.
- Su, M. K. Chen, K. H. & Wang, H. C. (1992). The spread of '88 Shanghai type-A hepatitis; a system dynamics model and analysis. *System Dynamics Review* **8** (3): 213-232
- Tanzania Commission for AIDS. (2005). National Bureau of Statistics and ORC Macro: HIV/AIDS Indicator Survey 2003–2004. 2005
- Tanzania Demographic and Health Survey. (1999). December report
- _____. (2001). December report
- Udo, H. M. J. Asgedom, A. H. & Viets, T. C. (2006). Modeling the impact of interventions on the dynamics in village poultry systems. *Agricultural Systems* **88**(2-3): 255-269
- UN/WHO, (1989). The AIDS epidemic and its demographic consequences, Proceedings of the United Nations/World Health Organization workshop on modeling the demographic impact of AIDS in Pattern II countries, New York, 13-15 ST/ESA/SER.A/119
- _____. (2000). AIDS epidemic update: December 2000. UNAIDS/WHO, Geneva 2000. UNAIDS/00
- _____. (2001). AIDS epidemic update: December 2001. UNAIDS/WHO, Geneva 2001. UNAIDS/01
- _____. (2004). AIDS epidemic update: December 2004. UNAIDS/WHO, Geneva 2004. UNAIDS/04. . Accessed at: <http://www.unaids.org>
- _____. (2005). AIDS epidemic update: December 2005. UNAIDS/WHO, Geneva 2005. UNAIDS/05.19E. Accessed at: http://www.unaids.org/epi2005/doc/report_pdf.html.
- _____. (2006). AIDS epidemic update: December 2006. UNAIDS, Geneva 2006. UNAIDS/06.29E. ISBN 92 9173542 6.
- _____. (2007). *Comparing adult antenatal-clinic based HIV prevalence with prevalence from national population based surveys in sub-Saharan Africa*. UNAIDS presentation. Accessed 17 November 2007 at http://data.unaids.org/pub/Presentation/2007/survey_anc_2007_en.pdf.

_____. (2007). *AIDS epidemic update: December 2007*. UNAIDS, Geneva 2007.
UNAIDS/07.27E / JC1322E (December 2007).

United Nations Secretariat/Department of Economic and Social Affairs/Population Division,
World Population Prospects: The 2002 Revision: Highlights. 2003: New York.
Accessed at: <http://www.un.org/esa/population/publications/wpp2002/wpp2002annextables.PDF>

United Republic of Tanzania. (2002). The National Bureau of Statistics. Population and
housing census. Accessed at: <http://http.www.tanzania.go.tz>

UNAIDS and UNHCR, (2003). *HIV/AIDS and STI prevention and care in Rwandan refugee
camps in the United Republic of Tanzania*. Geneva.

Accessed at <http://www.dec.org/pdf_docs/PNACS782.pdf>

U.S. Bureau of the Census, International Programs Center. (1994). HIV/AIDS Surveillance
Database. December 1994 Release.

Van der Ploeg, C. P. B *et al.*, (1998). STDSIM: a microsimulation model for decision support
in STD control. *Interfaces* **28**: 84–100

Wheat, I. D. (2007). *The Feedback Method: A System Dynamics Approach To Teaching
Macroeconomics*. Ph.D. dissertation, University of Bergen Open Research Archive
(<https://bora.uib.no/handle/1956/2239>), Bergen, Norway.

Worldbank. (1992). Tanzania: AIDS assessment and planning study. Worldbank, Washington,
DC

World Health Organisation. (2003). 3 by 5 Global initiatives providing Anti retroviral therapy
in Tanzania. World Health Organisation report on Tanzania

World Bank. (1992). *Tanzania AIDS Assessment and Planning Study*, Washington, D.C.

Zikmund, W. G. (2000). *Business Research Methods*, sixth edition, Oklahoma, Harcourt
College Publishers

Websites:

<http://news.bbc.co.uk/2/hi/africa/7117184.stm>

APPENDIX

Model Equations and Annotations

Model 1

{UTF-8}

average HIV contact frequency=

19.25

~ person/person/Year

~ |

hiv infected population= INTEG (

infection rate,

728)

~ person

~ |

hiv population generated contacts=

hiv infected population*average HIV contact frequency

~ person/Year

~ |

infection rate=

hiv population generated contacts*infectivity*susceptible fraction of population

~ person/Year

~ |

infectivity=

0.03

~ Dmnl

~ |

susceptible fraction of population=

susceptible population/total susceptible and hiv population

~ Dmnl

~ |

susceptible population= INTEG (

-infection rate,

9e+006)

~ person

~ |

total susceptible and hiv population=

hiv infected population+susceptible population

~ person

~ |

.Control

*****~

Simulation Control Parameters

|

FINAL TIME = 2004

~ Year

~ The final time for the simulation.

|

INITIAL TIME = 1980

~ Year
~ The initial time for the simulation.
|

SAVEPER = 0.125

~ Year [0,?]
~ The frequency with which output is stored.
|

TIME STEP = 0.125

~ Year [0,?]
~ The time step for the simulation.
|

\\--// Sketch information - do not modify anything except names

V300 Do not put anything below this section - it will be ignored

*View 1

\$192-192-192,0,Times New Roman|12||0-0-0|0-0-0|0-0-255|-1--1--1|-1--1--1|96,96,100

10,1,susceptible population,302,355,40,20,3,3,0,0,0,0,0

10,2,hiv infected population,561,355,40,20,3,3,0,0,0,0,0

1,3,5,2,4,0,0,22,0,0,0,-1--1--1,,1|(479,355)|

1,4,5,1,100,0,0,22,0,0,0,-1--1--1,,1|(383,355)|

11,5,268,431,355,6,8,34,3,0,0,1,0,0,0

10,6,infection rate,431,374,41,11,40,3,0,0,-1,0,0,0

12,7,48,384,-49,10,8,0,3,0,0,-1,0,0,0

10,8,infectivity,508,271,50,11,8,3,0,0,0,0,0,0

10,9,average HIV contact frequency,651,121,55,28,8,3,0,0,0,0,0,0

10,10,hiv population generated contacts,491,178,69,28,8,3,0,0,0,0,0,0

1,11,9,10,0,0,0,0,64,0,-1--1--1,,1|(584,144)|

1,12,2,10,1,0,0,0,0,64,0,-1--1--1,,1|(611,218)|
 10,13,total susceptible and hiv population,479,541,65,19,8,3,0,0,0,0,0,0
 1,14,1,13,1,0,0,0,0,64,0,-1--1--1,,1|(311,501)|
 10,15,susceptible fraction of population,405,478,61,19,8,3,0,0,0,0,0,0
 1,16,15,5,1,0,0,0,0,64,0,-1--1--1,,1|(447,426)|
 1,17,8,6,0,0,0,0,0,64,0,-1--1--1,,1|(473,316)|
 1,18,10,5,1,0,0,0,0,64,0,-1--1--1,,1|(406,265)|
 1,19,2,13,1,0,0,0,0,64,0,-1--1--1,,1|(598,448)|
 1,20,13,15,0,0,0,0,0,64,0,-1--1--1,,1|(447,513)|
 1,21,1,15,1,0,0,0,0,64,0,-1--1--1,,1|(319,446)|

Model 2

{UTF-8}

aids population= INTEG (

symptoms acquisition rate,

3)

~ person

~ |

average HIV contact frequency=

19.25

~ person/person/Year

~ |

hiv infected population= INTEG (

infection rate-symptoms acquisition rate,

725)

~ person

~ |

hiv population generated contacts=

hiv infected population*average HIV contact frequency

~ person/Year

~ |

incubation period=

8

~ Year

~ |

infection rate=

hiv population generated contacts*infectivity*susceptible fraction of population

~ person/Year

~ |

infectivity=

0.03

~ Dmnl

~ |

susceptible fraction of population=

susceptible population/total susceptible and hiv population

~ Dmnl

~ |

susceptible population= INTEG (

-infection rate,

9e+006)

~ person

~ |

symptoms acquisition rate=

hiv infected population/incubation period

~ person/Year

~ |

total susceptible and hiv population=

hiv infected population+susceptible population

~ person

~ |

.Control

*****~

Simulation Control Parameters

|

FINAL TIME = 2004

~ Year

~ The final time for the simulation.

|

INITIAL TIME = 1980

~ Year

~ The initial time for the simulation.

|

SAVEPER = 0.125

- ~ Year [0,?]
- ~ The frequency with which output is stored.
- |

TIME STEP = 0.125

- ~ Year [0,?]
- ~ The time step for the simulation.
- |

\\|--// Sketch information - do not modify anything except names

V300 Do not put anything below this section - it will be ignored

*View 1

\$192-192-192,0,Times New Roman|12||0-0-0|0-0-0|0-0-255|-1--1--1|-1--1--1|96,96,100

10,1,susceptible population,116,307,40,20,3,3,0,0,0,0,0

10,2,hiv infected population,375,307,40,20,3,3,0,0,0,0,0

1,3,5,2,4,0,0,22,0,0,0,-1--1--1,,1|(293,307)|

1,4,5,1,100,0,0,22,0,0,0,-1--1--1,,1|(197,307)|

11,5,300,245,307,6,8,34,3,0,0,1,0,0,0

10,6,infection rate,245,326,41,11,40,3,0,0,-1,0,0,0

12,7,48,384,-49,10,8,0,3,0,0,-1,0,0,0

10,8,infectivity,322,223,50,11,8,3,0,0,0,0,0

10,9,average HIV contact frequency,465,73,55,28,8,3,0,0,0,0,0

10,10,hiv population generated contacts,305,130,69,28,8,3,0,0,0,0,0

1,11,9,10,0,0,0,0,64,0,-1--1--1,,1|(398,96)|

1,12,2,10,1,0,0,0,64,0,-1--1--1,,1|(425,170)|

10,13,total susceptible and hiv population,293,493,65,19,8,3,0,0,0,0,0

1,14,1,13,1,0,0,0,64,0,-1--1--1,,1|(125,453)|

10,15,susceptible fraction of population,219,430,61,19,8,3,0,0,0,0,0

1,16,15,5,1,0,0,0,0,64,0,-1--1--1,,1|(261,378)|
 1,17,8,6,0,0,0,0,0,64,0,-1--1--1,,1|(287,268)|
 1,18,10,5,1,0,0,0,0,64,0,-1--1--1,,1|(220,217)|
 1,19,2,13,1,0,0,0,0,64,0,-1--1--1,,1|(412,400)|
 1,20,13,15,0,0,0,0,0,64,0,-1--1--1,,1|(261,465)|
 1,21,1,15,1,0,0,0,0,64,0,-1--1--1,,1|(133,398)|
 10,22,aids population,592,307,40,20,3,3,0,0,0,0,0,0
 1,23,25,22,4,0,0,22,0,0,0,-1--1--1,,1|(520,307)|
 1,24,25,2,100,0,0,22,0,0,0,-1--1--1,,1|(446,307)|
 11,25,220,483,307,6,8,34,3,0,0,1,0,0,0
 10,26,symptoms acquisition rate,483,334,48,19,40,3,0,0,-1,0,0,0
 10,27,incubation period,536,215,52,19,8,3,0,0,0,0,0,0
 1,28,27,25,0,0,0,0,0,64,0,-1--1--1,,1|(509,261)|
 1,29,2,26,1,0,0,0,0,64,0,-1--1--1,,1|(442,340)|

Model 3

{UTF-8}

aids death rate=

aids population/average duration of aids

~ person/Year

~ |

aids population= INTEG (

symptoms acquisition rate-aids death rate,

3)

~ person

~ |

average duration of aids=

1
~ Year
~ |

average HIV contact frequency=

19.25
~ person/person/Year
~ |

hiv infected population= INTEG (

infection rate-symptoms acquisition rate,
725)
~ person
~ |

hiv population generated contacts=

hiv infected population*average HIV contact frequency
~ person/Year
~ |

incubation period=

8
~ Year
~ |

infection rate=

hiv population generated contacts*infectivity*susceptible fraction of population

~ person/Year

~ |

infectivity=

0.03

~ Dmnl

~ |

susceptible fraction of population=

susceptible population/total susceptible and hiv population

~ Dmnl

~ |

susceptible population= INTEG (

-infection rate,

9e+006)

~ person

~ |

symptoms acquisition rate=

hiv infected population/incubation period

~ person/Year

~ |

total susceptible and hiv population=

hiv infected population+susceptible population

~ person

~ |

.Control

*****~

Simulation Control Parameters

|

FINAL TIME = 2004

~ Year

~ The final time for the simulation.

|

INITIAL TIME = 1980

~ Year

~ The initial time for the simulation.

|

SAVEPER = 0.125

~ Year [0,?]

~ The frequency with which output is stored.

|

TIME STEP = 0.125

~ Year [0,?]

~ The time step for the simulation.

|

\\/--// Sketch information - do not modify anything except names

V300 Do not put anything below this section - it will be ignored

*View 1

\$192-192-192,0,Times New Roman|12||0-0-0|0-0-0|0-0-255|-1--1--1|-1--1--1|96,96,100

10,1,susceptible population,116,307,40,20,3,3,0,0,0,0,0

10,2,hiv infected population,375,307,40,20,3,3,0,0,0,0,0

1,3,5,2,4,0,0,22,0,0,0,-1--1--1,,1|(293,307)|

1,4,5,1,100,0,0,22,0,0,0,-1--1--1,,1|(197,307)|

11,5,332,245,307,6,8,34,3,0,0,1,0,0,0

10,6,infection rate,245,326,41,11,40,3,0,0,-1,0,0,0

12,7,48,384,-49,10,8,0,3,0,0,-1,0,0,0

10,8,infectivity,322,223,50,11,8,3,0,0,0,0,0

10,9,average HIV contact frequency,465,73,55,28,8,3,0,0,0,0,0

10,10,hiv population generated contacts,305,130,69,28,8,3,0,0,0,0,0

1,11,9,10,0,0,0,0,64,0,-1--1--1,,1|(398,96)|

1,12,2,10,1,0,0,0,64,0,-1--1--1,,1|(425,170)|

10,13,total susceptible and hiv population,293,493,65,19,8,3,0,0,0,0,0

1,14,1,13,1,0,0,0,64,0,-1--1--1,,1|(125,453)|

10,15,susceptible fraction of population,219,430,61,19,8,3,0,0,0,0,0

1,16,15,5,1,0,0,0,64,0,-1--1--1,,1|(261,378)|

1,17,8,6,0,0,0,64,0,-1--1--1,,1|(287,268)|

1,18,10,5,1,0,0,0,64,0,-1--1--1,,1|(220,217)|

1,19,2,13,1,0,0,0,64,0,-1--1--1,,1|(412,400)|

1,20,13,15,0,0,0,64,0,-1--1--1,,1|(261,465)|

1,21,1,15,1,0,0,0,64,0,-1--1--1,,1|(133,398)|

10,22,aids population,592,307,40,20,3,3,0,0,0,0,0,0

1,23,25,22,4,0,0,22,0,0,0,-1--1--1,,1|(520,307)|

1,24,25,2,100,0,0,22,0,0,0,-1--1--1,,1|(446,307)|

11,25,236,483,307,6,8,34,3,0,0,1,0,0,0

10,26,symptoms acquisition rate,483,334,48,19,40,3,0,0,-1,0,0,0

10,27,incubation period,536,215,52,19,8,3,0,0,0,0,0,0

1,28,27,25,0,0,0,0,64,0,-1--1--1,,1|(509,261)|

1,29,2,26,1,0,0,0,64,0,-1--1--1,,1|(442,340)|

12,30,48,791,306,10,8,0,3,0,0,-1,0,0,0

1,31,33,30,4,0,0,22,0,0,0,-1--1--1,,1|(746,306)|

1,32,33,22,100,0,0,22,0,0,0,-1--1--1,,1|(666,306)|

11,33,48,706,306,6,8,34,3,0,0,1,0,0,0

10,34,aids death rate,706,325,46,11,40,3,0,0,-1,0,0,0

10,35,average duration of aids,730,202,77,19,8,3,0,0,0,0,0,0

1,36,35,34,0,0,0,0,64,0,-1--1--1,,1|(718,260)|

1,37,22,34,1,0,0,0,64,0,-1--1--1,,1|(646,343)|

Model 4

aging rate=

mature population/time to age

~ person/Year

~ |

aids death rate=

AIDS population/average time with aids

~ person/Year

~ |

AIDS population= INTEG (
+symptoms acquisition rate-aids death rate,

3)

~ person

~ |

average hiv contact frequency=

19.25

~ 1/Year

~ |

average time with aids=

1

~ Year

~ |

birth rate=

total population*fractional birth rate

~ person/Year

~ |

contact rate= INTEG (

-net change in contact rate,

initial average hiv contact frequency)

~ contact/(Year*person)

~ |

effect of hiv education=

effect of hiv education on contact rate table(hiv education)

~ Dmnl

~ |

effect of hiv education on contact rate table(

[(0,0)-(1.8,0.5)],(0,0),(1,0.05),(1.2,0.065),(1.4,0.08),(1.6,0.095),(1.8,0.105))

~ Dmnl

~ |

elderly death rate=

elderly population*fractional elderly death rate

~ person/Year

~ |

elderly population= INTEG (

aging rate-elderly death rate,

789200)

~ person

~ |

fractional birth rate=

42/1000

~ 1/Year

~ |

fractional elderly death rate=

15/1000

~ 1/Year

~ |

fractional hiv normal death rate=

80/1000

~ 1/Year

~ |

fractional mature death rate=

10/1000

~ 1/Year

~ |

fractional young death rate=

30/1000

~ 1/Year

~ |

hiv education=

HIV education table(Time)

~ Dmnl

~ |

HIV education table(

[(1983,0)-(2050,5)],(1980,0),(1988,0.1),(1993,0.2),(1998,0.3),(2002,0.4),(2004,0.5))

~ Dmnl

~ |

hiv infected population=

HIV population+"hiv-positive children population"

~ person

~ |

hiv normal death rate=

HIV population*fractional hiv normal death rate

~ person/Year

~ |

HIV population= INTEG (

+infection rate-hiv normal death rate-symptoms acquisition rate,

725)

~ person

~ |

"hiv-positive children"=

"hiv-positive children birth rate"

~ person/Year

~ |

"hiv-positive children average life time"=

2

~ Year

~ |

"hiv-positive children birth rate"=

birth rate*prenatal transmission rate

~ person/Year

~ |

"hiv-positive children death rate"=

"hiv-positive children population"/"hiv-positive children average life time"

~ person/Year

~ |

"hiv-positive children population"= INTEG (

"hiv-positive children birth rate"-"hiv-positive children death rate",

0)

~ person

~ |

incubation period=

8

~ Year

~ |

infection rate=

infectivity*susceptible fraction of population*total hiv generated contacts

~ person/Year

~ |

infectivity=

0.03

~ person/contact

~ |

inflow to susceptible=

maturation rate

~ person/Year

~ |

initial average hiv contact frequency=

19.25

~ contact/(person*Year)

~ |

maturation rate=

young population/time to mature

~ person/Year

~ |

mature death rate=

mature population*fractional mature death rate

~ person/Year

~ |

mature population= INTEG (

+maturation rate-aging rate-mature death rate,

9.2731e+006)

~ person

~ |

net change in contact rate=

contact rate*effect of hiv education

~ Dmnl

~ |

prenatal transmission rate=

0.032*0

~ Dmnl

~ |

susceptible fraction of population=

susceptible population/total susceptible and hiv population

~ Dmnl

~ |

susceptible normal death rate=

mature death rate

~ person/Year

~ |

susceptible population= INTEG (

+inflow to susceptible-infection rate-susceptible normal death rate,

9.2731e+006)

~ person

~ |

symptoms acquisition rate=

HIV population/incubation period

~ person/Year

~ |

time to age=

44

~ Year

~ |

time to mature=

14

~ Year

~ |

total hiv aids population=

AIDS population+hiv infected population

~ person

~ ~ :SUPPLEMENTARY

|

total hiv generated contacts=

HIV population*average hiv contact frequency

~ contact/Year

~ |

total population=

elderly population+mature population+young population

~ person

~ |

total susceptible and hiv population=

HIV population+susceptible population

~ person

~ |

young death rate=

young population*fractional young death rate

~ person/Year

~ |

young population= INTEG (

birth rate-maturation rate-young death rate,

9.6677e+006)

~ person

~ |

.Control

*****~

Simulation Control Parameters

|

FINAL TIME = 2004

~ Year

~ The final time for the simulation.

|

INITIAL TIME = 1980

~ Year

~ The initial time for the simulation.

|

SAVEPER = 1

~ Year [0,?]

~ The frequency with which output is stored.

|

TIME STEP = 0.125

~ Year [0,?]

~ The time step for the simulation.

|

\\/--// Sketch information - do not modify anything except names

V300 Do not put anything below this section - it will be ignored

*Population

\$192-192-192,0,Times New Roman|12||0-0-0|0-0-0|0-0-255|-1--1--1|-1--1--1|96,96,100

10,1,young population,256,244,40,20,3,3,0,0,0,0,0

10,2,mature population,481,248,40,20,3,3,0,0,0,0,0,0

10,3,elderly population,709,254,40,20,3,3,0,0,0,0,0,0

12,4,48,89,237,10,8,0,3,0,0,-1,0,0,0

1,5,7,1,4,0,0,22,0,0,0,-1--1--1,,1|(189,237)|

1,6,7,4,100,0,0,22,0,0,0,-1--1--1,,1|(125,237)|

11,7,48,157,237,6,8,34,3,0,0,1,0,0,0

10,8,birth rate,157,256,29,11,40,3,0,0,-1,0,0,0

1,9,11,2,4,0,0,22,0,0,0,-1--1--1,,1|(407,246)|

1,10,11,1,100,0,0,22,0,0,0,-1--1--1,,1|(329,246)|

11,11,380,368,246,6,8,34,3,0,0,1,0,0,0

10,12,maturation rate,368,265,47,11,40,3,0,0,-1,0,0,0

1,13,15,3,4,0,0,22,0,0,0,-1--1--1,,1|(635,251)|

1,14,15,2,100,0,0,22,0,0,0,-1--1--1,,1|(555,251)|

11,15,908,595,251,6,8,34,3,0,0,1,0,0,0

10,16,aging rate,595,270,31,11,40,3,0,0,-1,0,0,0

12,17,48,923,241,10,8,0,3,0,0,-1,0,0,0

1,18,20,17,4,0,0,22,0,0,0,-1--1--1,,1|(877,248)|

1,19,20,3,100,0,0,22,0,0,0,-1--1--1,,1|(789,248)|

11,20,48,835,248,6,8,34,3,0,0,1,0,0,0

10,21,elderly death rate,835,267,54,11,40,3,0,0,-1,0,0,0

12,22,48,254,67,10,8,0,3,0,0,-1,0,0,0

1,23,25,22,4,0,0,22,0,0,0,-1--1--1,,1|(256,106)|

1,24,25,1,100,0,0,22,0,0,0,-1--1--1,,1|(256,186)|

11,25,48,256,143,8,6,33,3,0,0,4,0,0,0

10,26,young death rate,316,143,52,11,40,3,0,0,-1,0,0,0

12,27,48,481,82,10,8,0,3,0,0,-1,0,0,0

1,28,30,27,4,0,0,22,0,0,0,-1--1--1,,1|(480,122)|

1,29,30,2,100,0,0,22,0,0,0,-1--1--1,,1|(480,197)|

11,30,48,480,161,8,6,33,3,0,0,4,0,0,0

10,31,mature death rate,543,161,55,11,40,3,0,0,-1,0,0,0

10,32,fractional birth rate,73,185,53,19,8,3,0,0,0,0,0,0

10,33,fractional young death rate,390,54,59,28,8,3,0,0,0,0,0,0

10,34,fractional mature death rate,672,66,63,28,8,3,0,0,0,0,0,0

10,35,fractional elderly death rate,866,117,65,28,8,3,0,0,0,0,0,0

1,36,32,8,0,0,0,0,0,64,0,-1--1--1,,1|(113,219)|

1,37,1,26,1,0,0,0,0,64,0,-1--1--1,,1|(305,201)|

1,38,33,26,0,0,0,0,0,64,0,-1--1--1,,1|(350,101)|

1,39,2,31,1,0,0,0,0,64,0,-1--1--1,,1|(539,207)|

1,40,2,16,1,0,0,0,0,64,0,-1--1--1,,1|(518,303)|

1,41,34,31,0,0,0,0,0,64,0,-1--1--1,,1|(601,117)|

1,42,3,21,1,0,0,0,0,64,0,-1--1--1,,1|(757,291)|

1,43,35,21,0,0,0,0,0,64,0,-1--1--1,,1|(850,193)|

10,44,total population,489,450,53,19,8,3,0,0,0,0,0,0

1,45,1,44,1,0,0,0,0,64,0,-1--1--1,,1|(360,415)|

1,46,2,44,0,0,0,0,0,64,0,-1--1--1,,1|(484,342)|

1,47,3,44,1,0,0,0,0,64,0,-1--1--1,,1|(647,372)|

1,48,44,8,1,0,0,0,0,64,0,-1--1--1,,1|(220,358)|

10,49,time to mature,374,374,34,19,8,3,0,0,0,0,0,0

10,50,time to age,612,350,51,11,8,3,0,0,0,0,0,0

1,51,49,12,0,0,0,0,0,64,0,-1--1--1,,1|(370,322)|

1,52,50,16,0,0,0,0,0,64,0,-1--1--1,,1|(605,316)|

10,53,"hiv-positive children birth rate",309,379,60,19,8,2,1,3,-1,0,0,0,0-255,0-0-0,|12||0-0-255

1,54,1,12,0,0,0,0,0,0,0,-1--1--1,,1|(301,252)|

\\--// Sketch information - do not modify anything except names

V300 Do not put anything below this section - it will be ignored

*HIV AIDS

\$192-192-192,0,Times New Roman|12||0-0-0|0-0-0|0-0-255|-1--1--1|-1--1--1|96,96,100

10,1,susceptible population,238,316,40,20,3,3,0,0,0,0,0,0

10,2,HIV population,496,313,40,20,3,3,0,0,0,0,0,0

10,3,AIDS population,729,314,40,20,3,3,0,0,0,0,0,0

12,4,48,78,311,10,8,0,3,0,0,-1,0,0,0

1,5,7,1,4,0,0,22,0,0,0,-1--1--1,,1|(173,311)|

1,6,7,4,100,0,0,22,0,0,0,-1--1--1,,1|(112,311)|

11,7,48,143,311,6,8,34,3,0,0,1,0,0,0

10,8,inflow to susceptible,143,338,35,19,40,3,0,0,-1,0,0,0

1,9,11,2,4,0,0,22,0,0,0,-1--1--1,,1|(415,317)|

1,10,11,1,100,0,0,22,0,0,0,-1--1--1,,1|(320,317)|

11,11,892,368,317,6,8,34,3,0,0,1,0,0,0

10,12,infection rate,368,336,41,11,40,3,0,0,-1,0,0,0

1,13,15,3,4,0,0,22,0,0,0,-1--1--1,,1|(655,311)|

1,14,15,2,100,0,0,22,0,0,0,-1--1--1,,1|(572,311)|

11,15,732,615,311,6,8,34,3,0,0,1,0,0,0

10,16,symptoms acquisition rate,615,338,48,19,40,3,0,0,-1,0,0,0

12,17,48,914,309,10,8,0,3,0,0,-1,0,0,0

1,18,20,17,4,0,0,22,0,0,0,-1--1--1,,1|(873,309)|

1,19,20,3,100,0,0,22,0,0,0,-1--1--1,,1|(799,309)|

11,20,48,836,309,6,8,34,3,0,0,1,0,0,0

10,21,aids death rate,836,328,46,11,40,3,0,0,-1,0,0,0

12,22,48,163,210,10,8,0,3,0,0,-1,0,0,0

1,23,25,22,4,0,0,22,0,0,0,-1--1--1,,1|(196,217)|

1,24,25,1,100,0,0,22,0,0,0,-1--1--1,,1|(228,259)|

11,25,48,228,217,8,6,33,3,0,0,4,0,0,0

10,26,susceptible normal death rate,295,217,59,19,40,3,0,0,-1,0,0,0

12,27,48,497,463,10,8,0,3,0,0,-1,0,0,0

1,28,30,27,4,0,0,22,0,0,0,-1--1--1,,1|(497,427)|

1,29,30,2,100,0,0,22,0,0,0,-1--1--1,,1|(497,360)|

11,30,48,497,394,8,6,33,3,0,0,4,0,0,0

10,31,hiv normal death rate,558,394,53,19,40,3,0,0,-1,0,0,0

10,32,maturation rate,129,422,56,11,8,2,0,3,-1,0,0,0,0-0-255,0-0-0,|12||0-0-255

10,33,mature death rate,96,256,46,19,8,2,0,3,-1,0,0,0,0-0-255,0-0-0,|12||0-0-255

1,34,32,8,0,0,0,0,0,64,0,-1--1--1,,1|(133,390)|

1,35,33,26,0,0,0,0,0,64,0,-1--1--1,,1|(182,238)|

1,36,2,31,0,0,0,0,0,64,0,-1--1--1,,1|(522,348)|

1,37,3,21,1,0,0,0,0,64,0,-1--1--1,,1|(776,346)|

10,38,fractional hiv normal death rate,609,471,69,28,8,3,0,0,0,0,0,0

10,39,average time with aids,860,424,60,19,8,3,0,0,0,0,0,0

10,40,incubation period,690,404,52,19,8,3,0,0,0,0,0,0

1,41,38,31,0,0,0,0,0,64,0,-1--1--1,,1|(584,433)|

1,42,40,16,0,0,0,0,0,64,0,-1--1--1,,1|(657,375)|

1,43,39,21,0,0,0,0,0,64,0,-1--1--1,,1|(848,378)|

1,44,2,16,0,0,0,0,0,64,0,-1--1--1,,1|(544,322)|

10,45,total hiv generated contacts,436,173,48,28,8,3,0,0,0,0,0,0

1,46,2,45,1,0,0,0,0,64,0,-1--1--1,,1|(518,232)|

10,47,susceptible fraction of population,347,453,61,19,8,3,0,0,0,0,0,0

1,48,47,11,1,0,0,0,0,64,0,-1--1--1,,1|(370,372)|

1,49,45,12,1,0,0,0,0,64,0,-1--1--1,,1|(354,220)|

10,50,infectivity,420,243,50,11,8,3,0,0,0,0,0,0

1,51,50,12,0,0,0,0,0,64,0,-1--1--1,,1|(397,283)|

10,52,hiv infected population,663,168,36,19,8,3,0,0,0,0,0,0

1,53,2,52,0,0,0,0,0,64,0,-1--1--1,,1|(574,244)|

10,54,total hiv aids population,806,209,64,19,8,3,0,0,0,0,0,0

1,55,52,54,0,0,0,0,0,64,0,-1--1--1,,1|(713,182)|

1,56,3,54,0,0,0,0,0,64,0,-1--1--1,,1|(763,266)|

1,57,1,58,1,0,0,0,0,64,0,-1--1--1,,1|(342,536)|

10,58,total susceptible and hiv population,361,535,65,19,8,3,0,0,0,0,0,0

1,59,1,47,1,0,0,0,0,64,0,-1--1--1,,1|(268,428)|

1,60,58,47,1,0,0,0,0,64,0,-1--1--1,,1|(360,496)|

1,61,2,58,0,1,0,0,0,0,0,-1--1--1,,1|(431,418)|

1,62,2,58,1,0,0,0,0,64,0,-1--1--1,,1|(495,473)|

10,63,average hiv contact frequency,321,95,55,28,8,3,0,0,0,0,0,0

1,64,63,45,0,0,0,0,0,64,0,-1--1--1,,1|(372,130)|

10,65,"hiv-positive children birth rate",621,97,60,19,8,2,1,3,-1,0,0,0,0-0-255,0-0-0,|12||0-0-255

10,66,"hiv-positive children death rate",621,97,68,19,8,2,1,3,-1,0,0,0,0-0-255,0-0-0,|12||0-0-255

10,67,"hiv-positive children population",663,206,68,19,8,2,1,3,-1,0,0,0,0-0-255,0-0-0,|12||0-0-255

1,68,67,52,0,1,0,0,0,0,0,-1--1--1,,1|(663,187)|

\\|--// Sketch information - do not modify anything except names

V300 Do not put anything below this section - it will be ignored

*HIV-positive children

\$192-192-192,0,Times New Roman|12||0-0-0|0-0-0|0-0-255|-1--1--1|-1--1--1|96,96,100

Model 5

aging rate=

mature population/time to age

~ person/Year

~ |

aids death rate=

AIDS population/average time with aids

~ person/Year

~ |

AIDS population= INTEG (

+symptoms acquisition rate-aids death rate,

3)

~ person

~ |

average hiv contact frequency=

19.25

~ 1/Year

~ |

average time with aids=

1

~ Year

~ |

birth rate=

total population*fractional birth rate

~ person/Year

~ |

contact rate= INTEG (

 -net change in contact rate,

 initial average hiv contact frequency)

 ~ contact/(Year*person)

 ~ |

effect of hiv education=

 effect of hiv education on contact rate table(hiv education)

 ~ Dmnl

 ~ |

effect of hiv education on contact rate table(

 [(0,0)-(1.8,0.5)],(0,0),(1,0.05),(1.2,0.065),(1.4,0.08),(1.6,0.095),(1.8,0.105))

 ~ Dmnl

 ~ |

elderly death rate=

 elderly population*fractional elderly death rate

 ~ person/Year

 ~ |

elderly population= INTEG (

 aging rate-elderly death rate,

 789200)

 ~ person

 ~ |

fractional birth rate=

 42/1000

~ 1/Year

~ |

fractional elderly death rate=

15/1000

~ 1/Year

~ |

fractional hiv normal death rate=

80/1000

~ 1/Year

~ |

fractional mature death rate=

10/1000

~ 1/Year

~ |

fractional young death rate=

30/1000

~ 1/Year

~ |

hiv education=

HIV education table(Time)

~ Dmnl

~ |

HIV education table(

[(1983,0)-(2050,5)],(1980,0),(1988,0.1),(1993,0.2),(1998,0.3),(2002,0.4),(2004,0.5))

~ Dmnl

~ |

hiv infected population=

HIV population+"hiv-positive children population"

~ person

~ |

hiv normal death rate=

HIV population*fractional hiv normal death rate

~ person/Year

~ |

HIV population= INTEG (

+infection rate-hiv normal death rate-symptoms acquisition rate,

725)

~ person

~ |

"hiv-positive children"=

"hiv-positive children birth rate"

~ person/Year

~ |

"hiv-positive children average life time"=

2

~ Year

~ |

"hiv-positive children birth rate"=

birth rate*prenatal transmission rate

~ person/Year

~ |

"hiv-positive children death rate"=

"hiv-positive children population"/"hiv-positive children average life time"

~ person/Year

~ |

"hiv-positive children population"= INTEG (

"hiv-positive children birth rate"-"hiv-positive children death rate",

0)

~ person

~ |

incubation period=

8

~ Year

~ |

infection rate=

infectivity*susceptible fraction of population*total hiv generated contacts

~ person/Year

~ |

infectivity=

0.03

~ person/contact

~ |

inflow to susceptible=

maturation rate

~ person/Year

~ |

initial average hiv contact frequency=

19.25

~ contact/(person*Year)

~ |

maturation rate=

young population/time to mature

~ person/Year

~ |

mature death rate=

mature population*fractional mature death rate

~ person/Year

~ |

mature population= INTEG (

+maturation rate-aging rate-mature death rate,

9.2731e+006)

~ person

~ |

net change in contact rate=

contact rate*effect of hiv education

~ Dmnl

~ |

prenatal transmission rate=

0.032

~ Dmnl

~ |

susceptible fraction of population=

susceptible population/total susceptible and hiv population

~ Dmnl

~ |

susceptible normal death rate=

mature death rate

~ person/Year

~ |

susceptible population= INTEG (

+inflow to susceptible-infection rate-susceptible normal death rate,

9.2731e+006)

~ person

~ |

symptoms acquisition rate=

HIV population/incubation period

~ person/Year

~ |

time to age=

44

~ Year

~ |

time to mature=

14

~ Year

~ |

total hiv aids population=

AIDS population+hiv infected population

~ person

~ ~ :SUPPLEMENTARY

|

total hiv generated contacts=

HIV population*average hiv contact frequency

~ contact/Year

~ |

total population=

elderly population+mature population+young population

~ person

~ |

total susceptible and hiv population=

HIV population+susceptible population

~ person

~ |

young death rate=

young population*fractional young death rate

~ person/Year

~ |

young population= INTEG (

birth rate-maturation rate-young death rate-"hiv-positive children",

9.6677e+006)

~ person

~ |

.Control

*****~

Simulation Control Parameters

|

FINAL TIME = 2004

~ Year

~ The final time for the simulation.

|

INITIAL TIME = 1980

~ Year

~ The initial time for the simulation.

|

SAVEPER = 1

~ Year [0,?]

~ The frequency with which output is stored.

|

TIME STEP = 0.125

~ Year [0,?]

~ The time step for the simulation.

|

\\!---// Sketch information - do not modify anything except names

V300 Do not put anything below this section - it will be ignored

*Population

\$192-192-192,0,Times New Roman|12||0-0-0|0-0-0|0-0-255|-1--1--1|-1--1--1|96,96,100

10,1,young population,256,244,40,20,3,3,0,0,0,0,0

10,2,mature population,481,248,40,20,3,3,0,0,0,0,0

10,3,elderly population,709,254,40,20,3,3,0,0,0,0,0

12,4,48,89,237,10,8,0,3,0,0,-1,0,0,0

1,5,7,1,4,0,0,22,0,0,0,-1--1--1,,1|(189,237)|

1,6,7,4,100,0,0,22,0,0,0,-1--1--1,,1|(125,237)|

11,7,48,157,237,6,8,34,3,0,0,1,0,0,0

10,8,birth rate,157,256,29,11,40,3,0,0,-1,0,0,0

1,9,11,2,4,0,0,22,0,0,0,-1--1--1,,1|(407,246)|

1,10,11,1,100,0,0,22,0,0,0,-1--1--1,,1|(329,246)|

11,11,380,368,246,6,8,34,3,0,0,1,0,0,0

10,12,maturation rate,368,265,47,11,40,3,0,0,-1,0,0,0

1,13,15,3,4,0,0,22,0,0,0,-1--1--1,,1|(635,251)|

1,14,15,2,100,0,0,22,0,0,0,-1--1--1,,1|(555,251)|
 11,15,908,595,251,6,8,34,3,0,0,1,0,0,0
 10,16,aging rate,595,270,31,11,40,3,0,0,-1,0,0,0
 12,17,48,923,241,10,8,0,3,0,0,-1,0,0,0
 1,18,20,17,4,0,0,22,0,0,0,-1--1--1,,1|(877,248)|
 1,19,20,3,100,0,0,22,0,0,0,-1--1--1,,1|(789,248)|
 11,20,48,835,248,6,8,34,3,0,0,1,0,0,0
 10,21,elderly death rate,835,267,54,11,40,3,0,0,-1,0,0,0
 12,22,48,254,67,10,8,0,3,0,0,-1,0,0,0
 1,23,25,22,4,0,0,22,0,0,0,-1--1--1,,1|(256,106)|
 1,24,25,1,100,0,0,22,0,0,0,-1--1--1,,1|(256,186)|
 11,25,48,256,143,8,6,33,3,0,0,4,0,0,0
 10,26,young death rate,316,143,52,11,40,3,0,0,-1,0,0,0
 12,27,48,481,82,10,8,0,3,0,0,-1,0,0,0
 1,28,30,27,4,0,0,22,0,0,0,-1--1--1,,1|(480,122)|
 1,29,30,2,100,0,0,22,0,0,0,-1--1--1,,1|(480,197)|
 11,30,48,480,161,8,6,33,3,0,0,4,0,0,0
 10,31,mature death rate,543,161,55,11,40,3,0,0,-1,0,0,0
 10,32,fractional birth rate,73,185,53,19,8,3,0,0,0,0,0,0
 10,33,fractional young death rate,390,54,59,28,8,3,0,0,0,0,0,0
 10,34,fractional mature death rate,672,66,63,28,8,3,0,0,0,0,0,0
 10,35,fractional elderly death rate,866,117,65,28,8,3,0,0,0,0,0,0
 1,36,32,8,0,0,0,0,0,64,0,-1--1--1,,1|(113,219)|
 1,37,1,26,1,0,0,0,0,64,0,-1--1--1,,1|(305,201)|
 1,38,33,26,0,0,0,0,0,64,0,-1--1--1,,1|(350,101)|
 1,39,1,12,1,0,0,0,0,64,0,-1--1--1,,1|(292,297)|
 1,40,2,31,1,0,0,0,0,64,0,-1--1--1,,1|(539,207)|
 1,41,2,16,1,0,0,0,0,64,0,-1--1--1,,1|(518,303)|
 1,42,34,31,0,0,0,0,0,64,0,-1--1--1,,1|(601,117)|

1,43,3,21,1,0,0,0,0,64,0,-1--1--1,,1|(757,291)|

1,44,35,21,0,0,0,0,0,64,0,-1--1--1,,1|(850,193)|

10,45,total population,489,450,53,19,8,3,0,0,0,0,0,0

1,46,1,45,1,0,0,0,0,64,0,-1--1--1,,1|(360,415)|

1,47,2,45,0,0,0,0,0,64,0,-1--1--1,,1|(484,342)|

1,48,3,45,1,0,0,0,0,64,0,-1--1--1,,1|(647,372)|

1,49,45,8,1,0,0,0,0,64,0,-1--1--1,,1|(220,358)|

10,50,time to mature,374,374,34,19,8,3,0,0,0,0,0,0

10,51,time to age,612,350,51,11,8,3,0,0,0,0,0,0

1,52,50,12,0,0,0,0,0,64,0,-1--1--1,,1|(370,322)|

1,53,51,16,0,0,0,0,0,64,0,-1--1--1,,1|(605,316)|

12,54,48,259,426,10,8,0,3,0,0,-1,0,0,0

1,55,57,54,4,0,0,22,0,0,0,-1--1--1,,1|(259,382)|

1,56,57,1,100,0,0,22,0,0,0,-1--1--1,,1|(259,299)|

11,57,48,259,341,8,6,33,3,0,0,4,0,0,0

10,58,"hiv-positive children",309,341,37,19,40,3,0,0,-1,0,0,0

10,59,"hiv-positive children birth rate",144,356,60,19,8,2,0,3,-1,0,0,0,0-255,0-0-0,|12|0-0-255

1,60,59,58,0,0,0,0,0,64,0,-1--1--1,,1|(231,348)|

\\|--// Sketch information - do not modify anything except names

V300 Do not put anything below this section - it will be ignored

*HIV AIDS

\$192-192-192,0,Times New Roman|12||0-0-0|0-0-0|0-0-255|-1--1--1|-1--1--1|96,96,100

10,1,susceptible population,238,384,40,20,3,3,0,0,0,0,0,0

10,2,HIV population,496,381,40,20,3,3,0,0,0,0,0,0

10,3,AIDS population,729,382,40,20,3,3,0,0,0,0,0,0

12,4,48,78,379,10,8,0,3,0,0,-1,0,0,0

1,5,7,1,4,0,0,22,0,0,0,-1--1--1,,1|(173,379)|

1,6,7,4,100,0,0,22,0,0,0,-1--1--1,,1|(112,379)|

11,7,48,143,379,6,8,34,3,0,0,1,0,0,0

10,8,inflow to susceptible,143,406,35,19,40,3,0,0,-1,0,0,0
 1,9,11,2,4,0,0,22,0,0,0,-1--1--1,,1|(415,385)|
 1,10,11,1,100,0,0,22,0,0,0,-1--1--1,,1|(320,385)|
 11,11,892,368,385,6,8,34,3,0,0,1,0,0,0
 10,12,infection rate,368,404,41,11,40,3,0,0,-1,0,0,0
 1,13,15,3,4,0,0,22,0,0,0,-1--1--1,,1|(655,379)|
 1,14,15,2,100,0,0,22,0,0,0,-1--1--1,,1|(572,379)|
 11,15,732,615,379,6,8,34,3,0,0,1,0,0,0
 10,16,symptoms acquisition rate,615,406,48,19,40,3,0,0,-1,0,0,0
 12,17,48,914,377,10,8,0,3,0,0,-1,0,0,0
 1,18,20,17,4,0,0,22,0,0,0,-1--1--1,,1|(873,377)|
 1,19,20,3,100,0,0,22,0,0,0,-1--1--1,,1|(799,377)|
 11,20,48,836,377,6,8,34,3,0,0,1,0,0,0
 10,21,aids death rate,836,396,46,11,40,3,0,0,-1,0,0,0
 12,22,48,163,278,10,8,0,3,0,0,-1,0,0,0
 1,23,25,22,4,0,0,22,0,0,0,-1--1--1,,1|(196,285)|
 1,24,25,1,100,0,0,22,0,0,0,-1--1--1,,1|(228,327)|
 11,25,48,228,285,8,6,33,3,0,0,4,0,0,0
 10,26,susceptible normal death rate,295,285,59,19,40,3,0,0,-1,0,0,0
 12,27,48,497,531,10,8,0,3,0,0,-1,0,0,0
 1,28,30,27,4,0,0,22,0,0,0,-1--1--1,,1|(497,495)|
 1,29,30,2,100,0,0,22,0,0,0,-1--1--1,,1|(497,428)|
 11,30,48,497,462,8,6,33,3,0,0,4,0,0,0
 10,31,hiv normal death rate,558,462,53,19,40,3,0,0,-1,0,0,0
 10,32,maturation rate,129,490,56,11,8,2,0,3,-1,0,0,0,0-0-255,0-0-0,|12||0-0-255
 10,33,mature death rate,96,324,46,19,8,2,0,3,-1,0,0,0,0-0-255,0-0-0,|12||0-0-255
 1,34,32,8,0,0,0,0,64,0,-1--1--1,,1|(133,458)|
 1,35,33,26,0,0,0,0,64,0,-1--1--1,,1|(182,306)|
 1,36,2,31,0,0,0,0,64,0,-1--1--1,,1|(522,416)|

1,37,3,21,1,0,0,0,0,64,0,-1--1--1,,1|(776,414)|

10,38,fractional hiv normal death rate,609,539,69,28,8,3,0,0,0,0,0,0

10,39,average time with aids,860,492,60,19,8,3,0,0,0,0,0,0

10,40,incubation period,690,472,52,19,8,3,0,0,0,0,0,0

1,41,38,31,0,0,0,0,0,64,0,-1--1--1,,1|(584,501)|

1,42,40,16,0,0,0,0,0,64,0,-1--1--1,,1|(657,443)|

1,43,39,21,0,0,0,0,0,64,0,-1--1--1,,1|(848,446)|

1,44,2,16,0,0,0,0,0,64,0,-1--1--1,,1|(544,390)|

10,45,total hiv generated contacts,436,241,48,28,8,3,0,0,0,0,0,0

1,46,2,45,1,0,0,0,0,64,0,-1--1--1,,1|(518,300)|

10,47,susceptible fraction of population,347,521,61,19,8,3,0,0,0,0,0,0

1,48,47,11,1,0,0,0,0,64,0,-1--1--1,,1|(370,440)|

1,49,45,12,1,0,0,0,0,64,0,-1--1--1,,1|(354,288)|

10,50,infectivity,420,311,50,11,8,3,0,0,0,0,0,0

1,51,50,12,0,0,0,0,0,64,0,-1--1--1,,1|(397,351)|

10,52,hiv infected population,663,236,36,19,8,3,0,0,0,0,0,0

10,53,"hiv-positive children population",621,127,66,19,8,3,0,0,-1,0,0,0

10,54,"hiv-positive children birth rate",826,158,60,19,8,2,0,3,-1,0,0,0,0-255,0-0-0,|12||0-0-255

1,55,54,53,0,0,0,0,0,0,0,-1--1--1,,1|(733,143)|

10,56,"hiv-positive children death rate",778,58,68,19,8,2,0,3,-1,0,0,0,0-255,0-0-0,|12||0-0-255

1,57,56,53,0,0,0,0,0,0,0,-1--1--1,,1|(705,89)|

1,58,53,52,0,0,0,0,0,64,0,-1--1--1,,1|(639,174)|

1,59,2,52,0,0,0,0,0,64,0,-1--1--1,,1|(574,312)|

10,60,total hiv aids population,806,277,64,19,8,3,0,0,0,0,0,0

1,61,52,60,0,0,0,0,0,64,0,-1--1--1,,1|(713,250)|

1,62,3,60,0,0,0,0,0,64,0,-1--1--1,,1|(763,334)|

1,63,1,64,1,0,0,0,0,64,0,-1--1--1,,1|(342,604)|

10,64,total susceptible and hiv population,361,603,65,19,8,3,0,0,0,0,0,0

1,65,1,47,1,0,0,0,0,64,0,-1--1--1,,1|(268,496)|

1,66,64,47,1,0,0,0,0,64,0,-1--1--1,,1|(360,564)|

1,67,2,64,0,1,0,0,0,0,0,-1--1--1,,1|(431,486)|

1,68,2,64,1,0,0,0,0,64,0,-1--1--1,,1|(495,541)|

10,69,average hiv contact frequency,336,107,55,28,8,3,0,0,0,0,0

1,70,69,45,0,0,0,0,0,64,0,-1--1--1,,1|(381,168)|

\\--// Sketch information - do not modify anything except names

V300 Do not put anything below this section - it will be ignored

*HIV-positive children

\$192-192-192,0,Times New Roman|12||0-0-0|0-0-0|0-0-255|-1--1--1|-1--1--1|96,96,100

10,1,"hiv-positive children population",525,213,40,30,3,3,0,0,0,0,0

12,2,48,332,207,10,8,0,3,0,0,-1,0,0,0

1,3,5,1,4,0,0,22,0,0,0,-1--1--1,,1|(452,207)|

1,4,5,2,100,0,0,22,0,0,0,-1--1--1,,1|(374,207)|

11,5,48,413,207,6,8,34,3,0,0,1,0,0,0

10,6,"hiv-positive children birth rate",413,234,64,19,40,3,0,0,-1,0,0,0

12,7,48,756,210,10,8,0,3,0,0,-1,0,0,0

1,8,10,7,4,0,0,22,0,0,0,-1--1--1,,1|(703,210)|

1,9,10,1,100,0,0,22,0,0,0,-1--1--1,,1|(607,210)|

11,10,48,655,210,6,8,34,3,0,0,1,0,0,0

10,11,"hiv-positive children death rate",655,237,64,19,40,3,0,0,-1,0,0,0

10,12,birth rate,281,289,38,11,8,2,0,3,-1,0,0,0,0-0-255,0-0-0,|12||0-0-255

10,13,prenatal transmission rate,288,128,62,28,8,3,0,0,0,0,0,0

1,14,13,6,0,0,0,0,0,64,0,-1--1--1,,1|(350,180)|

1,15,12,6,0,0,0,0,0,64,0,-1--1--1,,1|(330,268)|

10,16,"hiv-positive children average life time",768,356,81,28,8,3,0,0,0,0,0,0

1,17,16,11,0,0,0,0,0,64,0,-1--1--1,,1|(712,297)|

1,18,1,11,1,0,0,0,0,64,0,-1--1--1,,1|(554,266)|

Model 6

aging rate=

mature population/time to age

~ person/Year

~ |

aids death rate=

AIDS population/average time with aids

~ person/Year

~ |

AIDS population= INTEG (

+symptoms acquisition rate-aids death rate,

3)

~ person

~ |

average hiv contact frequency=

19.25

~ contact/(person*Year)

~ |

average time with aids=

1

~ Year

~ |

birth rate=

total population*fractional birth rate

~ person/Year

~ |

elderly death rate=

elderly population*fractional elderly death rate

~ person/Year

~ |

elderly population= INTEG (

aging rate-elderly death rate,

789200)

~ person

~ |

fractional birth rate=

42/1000

~ 1/Year

~ |

fractional elderly death rate=

15/1000

~ 1/Year

~ |

fractional hiv normal death rate=

80/1000

~ 1/Year

~ |

fractional mature death rate=

10/1000

~ 1/Year

~ |

fractional young death rate=

30/1000

~ 1/Year

~ |

HIV education effect=

1

~ person/contact

~ |

hiv infected population=

HIV population+"hiv-positive children population"

~ person

~ |

hiv normal death rate=

HIV population*fractional hiv normal death rate

~ person/Year

~ |

HIV population= INTEG (

+infection rate-hiv normal death rate-symptoms acquisition rate,

725)

~ person

~ |

"hiv-positive children"=

"hiv-positive children birth rate"

~ person/Year

~ |

"hiv-positive children average life time"=

2

~ Year

~ |

"hiv-positive children birth rate"=

birth rate*prenatal transmission fraction

~ person/Year

~ |

"hiv-positive children death rate"=

"hiv-positive children population"/"hiv-positive children average life time"

~ person/Year

~ |

"hiv-positive children population"= INTEG (

"hiv-positive children birth rate"-"hiv-positive children death rate",

0)

~ person

~ |

incubation period=

8

~ Year

~ |

infection rate=

infectivity*susceptible fraction of population*total hiv generated contacts

~ person/Year

~ |

infectivity=

0.03*HIV education effect

~ person/contact

~ |

inflow to susceptible=

maturation rate

~ person/Year

~ |

maturation rate=

young population/time to mature

~ person/Year

~ |

mature death rate=

mature population*fractional mature death rate

~ person/Year

~ |

mature population= INTEG (
 +maturation rate-aging rate-mature death rate,
 9.2731e+006)
 ~ person
 ~ |

prenatal transmission fraction=
 0.032
 ~ Dmnl
 ~ |

susceptible fraction of population=
 susceptible population/total susceptible and hiv population
 ~ Dmnl
 ~ |

susceptible normal death rate=
 mature death rate
 ~ person/Year
 ~ |

susceptible population= INTEG (
 +inflow to susceptible-infection rate-susceptible normal death rate,
 9.2731e+006)
 ~ person
 ~ |

symptoms acquisition rate=
 HIV population/incubation period

~ person/Year

~ |

time to age=

44

~ Year

~ |

time to mature=

14

~ Year

~ |

total hiv aids population=

AIDS population+hiv infected population

~ person

~ ~ :SUPPLEMENTARY

|

total hiv generated contacts=

HIV population*average hiv contact frequency

~ contact/Year

~ |

total population=

elderly population+mature population+young population

~ person

~ |

total susceptible and hiv population=

HIV population+susceptible population

~ person

~ |

young death rate=

young population*fractional young death rate

~ person/Year

~ |

young population= INTEG (

birth rate-maturation rate-young death rate-"hiv-positive children",

9.6677e+006)

~ person

~ |

.Control

*****~

Simulation Control Parameters

|

FINAL TIME = 2004

~ Year

~ The final time for the simulation.

|

INITIAL TIME = 1980

~ Year

~ The initial time for the simulation.
|

SAVEPER = 1

~ Year [0,?]
~ The frequency with which output is stored.
|

TIME STEP = 0.125

~ Year [0,?]
~ The time step for the simulation.
|

\\|--// Sketch information - do not modify anything except names

V300 Do not put anything below this section - it will be ignored

*Population

\$192-192-192,0,Times New Roman|12||0-0-0|0-0-0|0-0-255|-1--1--1|-1--1--1|96,96,100

10,1,young population,256,244,40,20,3,3,0,0,0,0,0

10,2,mature population,481,248,40,20,3,3,0,0,0,0,0

10,3,elderly population,709,254,40,20,3,3,0,0,0,0,0

12,4,48,89,237,10,8,0,3,0,0,-1,0,0,0

1,5,7,1,4,0,0,22,0,0,0,-1--1--1,,1|(189,237)|

1,6,7,4,100,0,0,22,0,0,0,-1--1--1,,1|(125,237)|

11,7,48,157,237,6,8,34,3,0,0,1,0,0,0

10,8,birth rate,157,256,29,11,40,3,0,0,-1,0,0,0

1,9,11,2,4,0,0,22,0,0,0,-1--1--1,,1|(407,246)|

1,10,11,1,100,0,0,22,0,0,0,-1--1--1,,1|(329,246)|

11,11,380,368,246,6,8,34,3,0,0,1,0,0,0

10,12,maturation rate,368,265,47,11,40,3,0,0,-1,0,0,0

1,13,15,3,4,0,0,22,0,0,0,-1--1--1,,1|(635,251)|
 1,14,15,2,100,0,0,22,0,0,0,-1--1--1,,1|(555,251)|
 11,15,796,595,251,6,8,34,3,0,0,1,0,0,0
 10,16,aging rate,595,270,31,11,40,3,0,0,-1,0,0,0
 12,17,48,923,241,10,8,0,3,0,0,-1,0,0,0
 1,18,20,17,4,0,0,22,0,0,0,-1--1--1,,1|(877,248)|
 1,19,20,3,100,0,0,22,0,0,0,-1--1--1,,1|(789,248)|
 11,20,48,835,248,6,8,34,3,0,0,1,0,0,0
 10,21,elderly death rate,835,267,54,11,40,3,0,0,-1,0,0,0
 12,22,48,254,67,10,8,0,3,0,0,-1,0,0,0
 1,23,25,22,4,0,0,22,0,0,0,-1--1--1,,1|(256,106)|
 1,24,25,1,100,0,0,22,0,0,0,-1--1--1,,1|(256,186)|
 11,25,48,256,143,8,6,33,3,0,0,4,0,0,0
 10,26,young death rate,316,143,52,11,40,3,0,0,-1,0,0,0
 12,27,48,481,82,10,8,0,3,0,0,-1,0,0,0
 1,28,30,27,4,0,0,22,0,0,0,-1--1--1,,1|(480,122)|
 1,29,30,2,100,0,0,22,0,0,0,-1--1--1,,1|(480,197)|
 11,30,48,480,161,8,6,33,3,0,0,4,0,0,0
 10,31,mature death rate,543,161,55,11,40,3,0,0,-1,0,0,0
 10,32,fractional birth rate,73,185,53,19,8,3,0,0,0,0,0,0
 10,33,fractional young death rate,390,54,59,28,8,3,0,0,0,0,0,0
 10,34,fractional mature death rate,672,66,63,28,8,3,0,0,0,0,0,0
 10,35,fractional elderly death rate,866,117,65,28,8,3,0,0,0,0,0,0
 1,36,32,8,0,0,0,0,0,64,0,-1--1--1,,1|(113,219)|
 1,37,1,26,1,0,0,0,0,64,0,-1--1--1,,1|(305,201)|
 1,38,33,26,0,0,0,0,0,64,0,-1--1--1,,1|(350,101)|
 1,39,1,12,1,0,0,0,0,64,0,-1--1--1,,1|(292,297)|
 1,40,2,31,1,0,0,0,0,64,0,-1--1--1,,1|(539,207)|
 1,41,2,16,1,0,0,0,0,64,0,-1--1--1,,1|(518,303)|

1,42,34,31,0,0,0,0,64,0,-1--1--1,,1|(601,117)|

1,43,3,21,1,0,0,0,64,0,-1--1--1,,1|(757,291)|

1,44,35,21,0,0,0,0,64,0,-1--1--1,,1|(850,193)|

10,45,total population,489,450,53,19,8,3,0,0,0,0,0

1,46,1,45,1,0,0,0,64,0,-1--1--1,,1|(360,415)|

1,47,2,45,0,0,0,0,64,0,-1--1--1,,1|(484,342)|

1,48,3,45,1,0,0,0,64,0,-1--1--1,,1|(647,372)|

1,49,45,8,1,0,0,0,64,0,-1--1--1,,1|(220,358)|

10,50,time to mature,374,374,34,19,8,3,0,0,0,0,0

10,51,time to age,612,350,51,11,8,3,0,0,0,0,0

1,52,50,12,0,0,0,0,64,0,-1--1--1,,1|(370,322)|

1,53,51,16,0,0,0,0,64,0,-1--1--1,,1|(605,316)|

12,54,48,259,426,10,8,0,3,0,0,-1,0,0,0

1,55,57,54,4,0,0,22,0,0,0,-1--1--1,,1|(259,382)|

1,56,57,1,100,0,0,22,0,0,0,-1--1--1,,1|(259,299)|

11,57,48,259,341,8,6,33,3,0,0,4,0,0,0

10,58,"hiv-positive children",309,341,37,19,40,3,0,0,-1,0,0,0

10,59,"hiv-positive children birth rate",144,356,60,19,8,2,0,3,-1,0,0,0,0-255,0-0-0,|12|0-0-255

1,60,59,58,0,0,0,0,64,0,-1--1--1,,1|(231,348)|

\\|---| Sketch information - do not modify anything except names

V300 Do not put anything below this section - it will be ignored

*HIV AIDS

\$192-192-192,0,Times New Roman|12||0-0-0|0-0-0|0-0-255|-1--1--1|-1--1--1|96,96,100

10,1,susceptible population,238,384,40,20,3,3,0,0,0,0,0

10,2,HIV population,496,381,40,20,3,3,0,0,0,0,0

10,3,AIDS population,729,382,40,20,3,3,0,0,0,0,0

12,4,48,78,379,10,8,0,3,0,0,-1,0,0,0

1,5,7,1,4,0,0,22,0,0,0,-1--1--1,,1|(173,379)|

1,6,7,4,100,0,0,22,0,0,0,-1--1--1,,1|(112,379)|

11,7,48,143,379,6,8,34,3,0,0,1,0,0,0
 10,8,inflow to susceptible,143,406,35,19,40,3,0,0,-1,0,0,0
 1,9,11,2,4,0,0,22,0,0,0,-1--1--1,,1|(415,385)|
 1,10,11,1,100,0,0,22,0,0,0,-1--1--1,,1|(320,385)|
 11,11,780,368,385,6,8,34,3,0,0,1,0,0,0
 10,12,infection rate,368,404,41,11,40,3,0,0,-1,0,0,0
 1,13,15,3,4,0,0,22,0,0,0,-1--1--1,,1|(655,379)|
 1,14,15,2,100,0,0,22,0,0,0,-1--1--1,,1|(572,379)|
 11,15,620,615,379,6,8,34,3,0,0,1,0,0,0
 10,16,symptoms acquisition rate,615,406,48,19,40,3,0,0,-1,0,0,0
 12,17,48,914,377,10,8,0,3,0,0,-1,0,0,0
 1,18,20,17,4,0,0,22,0,0,0,-1--1--1,,1|(873,377)|
 1,19,20,3,100,0,0,22,0,0,0,-1--1--1,,1|(799,377)|
 11,20,48,836,377,6,8,34,3,0,0,1,0,0,0
 10,21,aids death rate,836,396,46,11,40,3,0,0,-1,0,0,0
 12,22,48,163,278,10,8,0,3,0,0,-1,0,0,0
 1,23,25,22,4,0,0,22,0,0,0,-1--1--1,,1|(227,278)|
 1,24,25,1,100,0,0,22,0,0,0,-1--1--1,,1|(227,346)|
 11,25,48,227,323,8,6,33,3,0,0,4,0,0,0
 10,26,susceptible normal death rate,294,323,59,19,40,3,0,0,-1,0,0,0
 12,27,48,497,531,10,8,0,3,0,0,-1,0,0,0
 1,28,30,27,4,0,0,22,0,0,0,-1--1--1,,1|(497,495)|
 1,29,30,2,100,0,0,22,0,0,0,-1--1--1,,1|(497,428)|
 11,30,48,497,462,8,6,33,3,0,0,4,0,0,0
 10,31,hiv normal death rate,558,462,53,19,40,3,0,0,-1,0,0,0
 10,32,maturation rate,129,490,56,11,8,2,0,3,-1,0,0,0,0-0-255,0-0-0,|12||0-0-255
 10,33,mature death rate,96,324,46,19,8,2,0,3,-1,0,0,0,0-0-255,0-0-0,|12||0-0-255
 1,34,32,8,0,0,0,0,64,0,-1--1--1,,1|(133,458)|
 1,35,33,26,0,0,0,0,64,0,-1--1--1,,1|(181,323)|

1,36,2,31,0,0,0,0,64,0,-1--1--1,,1|(522,416)|

1,37,3,21,1,0,0,0,64,0,-1--1--1,,1|(776,414)|

10,38,fractional hiv normal death rate,609,539,69,28,8,3,0,0,0,0,0

10,39,average time with aids,860,492,60,19,8,3,0,0,0,0,0

10,40,incubation period,690,472,52,19,8,3,0,0,0,0,0

1,41,38,31,0,0,0,0,64,0,-1--1--1,,1|(584,501)|

1,42,40,16,0,0,0,0,64,0,-1--1--1,,1|(657,443)|

1,43,39,21,0,0,0,0,64,0,-1--1--1,,1|(848,446)|

1,44,2,16,0,0,0,0,64,0,-1--1--1,,1|(544,390)|

10,45,average hiv contact frequency,428,151,55,28,8,3,0,0,0,0,0

10,46,total hiv generated contacts,436,241,48,28,8,3,0,0,0,0,0

1,47,2,46,1,0,0,0,64,0,-1--1--1,,1|(518,300)|

10,48,susceptible fraction of population,346,494,61,19,8,3,0,0,0,0,0

1,49,48,11,1,0,0,0,64,0,-1--1--1,,1|(366,429)|

1,50,46,12,1,0,0,0,64,0,-1--1--1,,1|(354,288)|

10,51,infectivity,422,311,50,11,8,3,0,0,0,0,0

1,52,51,12,0,0,0,0,64,0,-1--1--1,,1|(398,351)|

10,53,hiv infected population,663,236,36,19,8,3,0,0,0,0,0

10,54,"hiv-positive children population",621,127,66,19,8,3,0,0,-1,0,0,0

10,55,"hiv-positive children birth rate",776,163,60,19,8,2,0,3,-1,0,0,0,0-255,0-0-0,|12||0-0-255

1,56,55,54,0,0,0,0,64,0,-1--1--1,,1|(708,147)|

10,57,"hiv-positive children death rate",735,70,68,19,8,2,0,3,-1,0,0,0,0-255,0-0-0,|12||0-0-255

1,58,57,54,0,0,0,0,64,0,-1--1--1,,1|(684,95)|

1,59,54,53,0,0,0,0,64,0,-1--1--1,,1|(639,174)|

1,60,2,53,0,0,0,0,64,0,-1--1--1,,1|(574,312)|

10,61,total hiv aids population,806,277,64,19,8,3,0,0,0,0,0

1,62,53,61,0,0,0,0,64,0,-1--1--1,,1|(713,250)|

1,63,3,61,0,0,0,0,64,0,-1--1--1,,1|(763,334)|

1,64,1,65,1,0,0,0,0,64,0,-1--1--1,,1|(340,573)|

10,65,total susceptible and hiv population,355,572,65,19,8,3,0,0,0,0,0,0

1,66,1,48,1,0,0,0,0,64,0,-1--1--1,,1|(278,476)|

1,67,65,48,1,0,0,0,0,64,0,-1--1--1,,1|(356,535)|

1,68,2,65,0,1,0,0,0,0,0,-1--1--1,,1|(429,471)|

1,69,2,65,1,0,0,0,0,64,0,-1--1--1,,1|(476,522)|

1,70,45,46,0,0,0,0,0,64,0,-1--1--1,,1|(430,189)|

10,71,HIV education effect,283,204,65,19,8,3,0,0,0,0,0,0

1,72,71,51,0,0,0,0,0,64,0,-1--1--1,,1|(351,257)|

\\|--// Sketch information - do not modify anything except names

V300 Do not put anything below this section - it will be ignored

*HIV-positive children

\$192-192-192,0,Times New Roman|12||0-0-0|0-0-0|0-0-255|-1--1--1|-1--1--1|96,96,100

10,1,"hiv-positive children population",525,213,40,30,3,3,0,0,0,0,0,0

12,2,48,332,207,10,8,0,3,0,0,-1,0,0,0

1,3,5,1,4,0,0,22,0,0,0,-1--1--1,,1|(452,207)|

1,4,5,2,100,0,0,22,0,0,0,-1--1--1,,1|(374,207)|

11,5,48,413,207,6,8,34,3,0,0,1,0,0,0

10,6,"hiv-positive children birth rate",413,234,64,19,40,3,0,0,-1,0,0,0

12,7,48,756,210,10,8,0,3,0,0,-1,0,0,0

1,8,10,7,4,0,0,22,0,0,0,-1--1--1,,1|(703,210)|

1,9,10,1,100,0,0,22,0,0,0,-1--1--1,,1|(607,210)|

11,10,48,655,210,6,8,34,3,0,0,1,0,0,0

10,11,"hiv-positive children death rate",655,237,64,19,40,3,0,0,-1,0,0,0

10,12,birth rate,281,289,38,11,8,2,0,3,-1,0,0,0,0-0-255,0-0-0,|12||0-0-255

10,13,prenatal transmission fraction,288,128,62,28,8,3,0,0,0,0,0,0

1,14,13,6,0,0,0,0,0,64,0,-1--1--1,,1|(350,180)|

1,15,12,6,0,0,0,0,0,64,0,-1--1--1,,1|(330,268)|

10,16,"hiv-positive children average life time",768,356,81,28,8,3,0,0,0,0,0,0

1,17,16,11,0,0,0,0,64,0,-1--1--1,,1|(712,297)|

1,18,1,11,1,0,0,0,64,0,-1--1--1,,1|(554,266)|