HIV PREVALENCE ESTIMATES AND THEIR USE IN REGRESSION MODELS: CAUTIONARY EVIDENCE FROM ZIMBABWE AND STUDIES OF THE RELATIONSHIP BETWEEN ARMED CONFLICT AND HIV.

Fidelis M. Hove

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Supervisor: Prof. Nicoli Nattrass

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ABSTRACT

This dissertation makes two central arguments. The first is that regressions on country-level HIV prevalence are compromised by the fact that the HIV data used are estimates and not empirical data points. The HIV prevalence rates published by UNAIDS are estimates derived from epidemiological modelling (using EPP and Spectrum) in which data from antenatal clinics (sometimes supplemented by population survey data) are translated into adult HIV prevalence estimates. The estimates thus depend on the quantity and quality of the underlying data, the modelling architecture and the assumptions made during the modelling process. A case study of Zimbabwe is provided to illustrate this process. In the case of Zimbabwe, it has been widely acknowledged that the decline in HIV prevalence observed over the past 10 years was due to a combination of a decline in new infections, increase in AIDS deaths and migration. However, this dissertation argues that the assumptions used by the National HIV and AIDS Estimates Working Group appear to underestimate out-migration on the progression of the epidemic. Supporting this analysis is a subsequent chapter which provides background information on Zimbabwe's socio economic decline (early 1990s to the present) which affected the provinces differently but overall would have incentivised many young Zimbabweans to leave the country. As a point of departure, this part of the thesis makes two important points. First it offers a cautionary statement against the drawing of conclusions on the reasons for observed trends in HIV prevalence estimates without having an appreciation of the assumptions used in the modelling process. Second, it provides a platform for analysis and further development of different regression models that try to come up with an overarching theory on the nature of the link between HIV prevalence and military conflict.

The second part of the thesis begins by looking at past studies on the link between HIV prevalence and military conflict. The literature review reveals that there is evidence both for a positive and a negative relationship between the two variables and that econometric research is similarly ambivalent. A key problem is that data and methodological challenges make this very difficult to pin down. A major limitation of standard regression approaches is that they impose a linear model on HIV estimates which – as shown in the earlier discussion of HIV modelling – are curvilinear as they already are the product of a curve fitting exercise using EPP and Spectrum. Further, the models fail to acknowledge the dynamic nature of the epidemic, notably that today's prevalence rate is a function of past prevalence rates (thereby making it more difficult to pin point any particular change in HIV prevalence as a consequence of conflict). It is argued that dynamic panel regression models are probably more useful than others (and several estimation models are presented), but that even so, analysis of the relationship between HIV and military conflict should pay particular attention to the country cases driving what appears to be a broadly negative relationship between HIV and military conflict in Africa. The final chapter thus briefly examines the key contextual factors shaping this relationship in Angola, Ethiopia, Rwanda, Liberia, the Sudan, the DRC and Somalia.

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ABBREVIATIONS

AIM	AIDS Impact Model
AIPPA	Access to Information and Protection of Privacy Act
ANC	Antenatal Clinic
ART	Antiretroviral Treatment
CSO	Central Statistical Office
COW	Correlates of War
CRS	Congressional Research Service
DFID	Department for International Development
DRC	Democratic Republic of Congo
EPP	Estimation and Projection Package
ESAP	Economic Structural Adjustment Program
FE	Fixed Effects
GMM	Generalized Method of Moments
GPS	Global Positioning Systems
HIVWRA	HIV positive women of reproductive age;
HNP	Health and National Population
IDP	Internally Displaced Persons
ICG	International Crisis Group
LSDV	Least Squares Dummy Variable
MDC	Movement for Democratic Chane
MOHCW	Ministry of Health and Child Welfare of Zimbabwe
MPLA	Popular Movement for the Liberation of Angola
MTP1	First Medium Term Plan
MTP2	Second Medium Term Plan
NACP	National AIDS Control Programme
NAC	National AIDS Council
NATF	National AIDS Trust Fund
NANGO	National Association of NGOs
NBTS	National Blood Transfusion Services
PCSE	Panel Corrected Standard Errors
PHR	Physicians for Human Rights
PDRC	Puntland Development Research Centre

PEPFAR	The U.S. President's Emergency Plan for AIDS Relief
PF ZAPU	Patriotic Front Zimbabwe African people’s Union
PMTCT	Prevention of Mother to Child Transmission
POSA	Public Order and Security Act
PSI	Population Services International
PSI	Population Services International
PTR	Perinatal Transmission Rate
RE	Random Effects
STP	Short Term Plan
SAMP	South African Migration Project
TFR	Total Fertility Rate
UCDP	Uppsala Conflict Data Program
UNAIDS	Joint United Nations Programme on HIV/AIDS
UNAIDS	Joint United Nations Programme on HIV/AIDS
UNDP	United Nations Development Program
UNGASS	United Nations General Assembly
UNHCR	United Nations High Commission for Refugees
UNSTATS	United Nations Statistics Division
UNPF	United Nations Population Fund
UNITA	National Union for the Total Independence of Angola
UNICEF	United Nations Children's Fund
USAID	U.S. Agency for International Development
WHO	World Health Organization
USIP	United States Institute of Peace
WHO	World Health Organisation
YAS	Young Adult Survey
ZADHR	Zimbabwe Association of Doctors for Human Rights
ZANU PF	Zimbabwe African National Union Patriotic Front
ZCTU	Zimbabwe Congress of Trade Unions
ZDHS	Zimbabwe Demographic and Health Survey
ZIMPREST	Zimbabwe Programme for Economic Transformation
ZNFPC	Zimbabwe National Family Planning Council

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1 INTRODUCTION

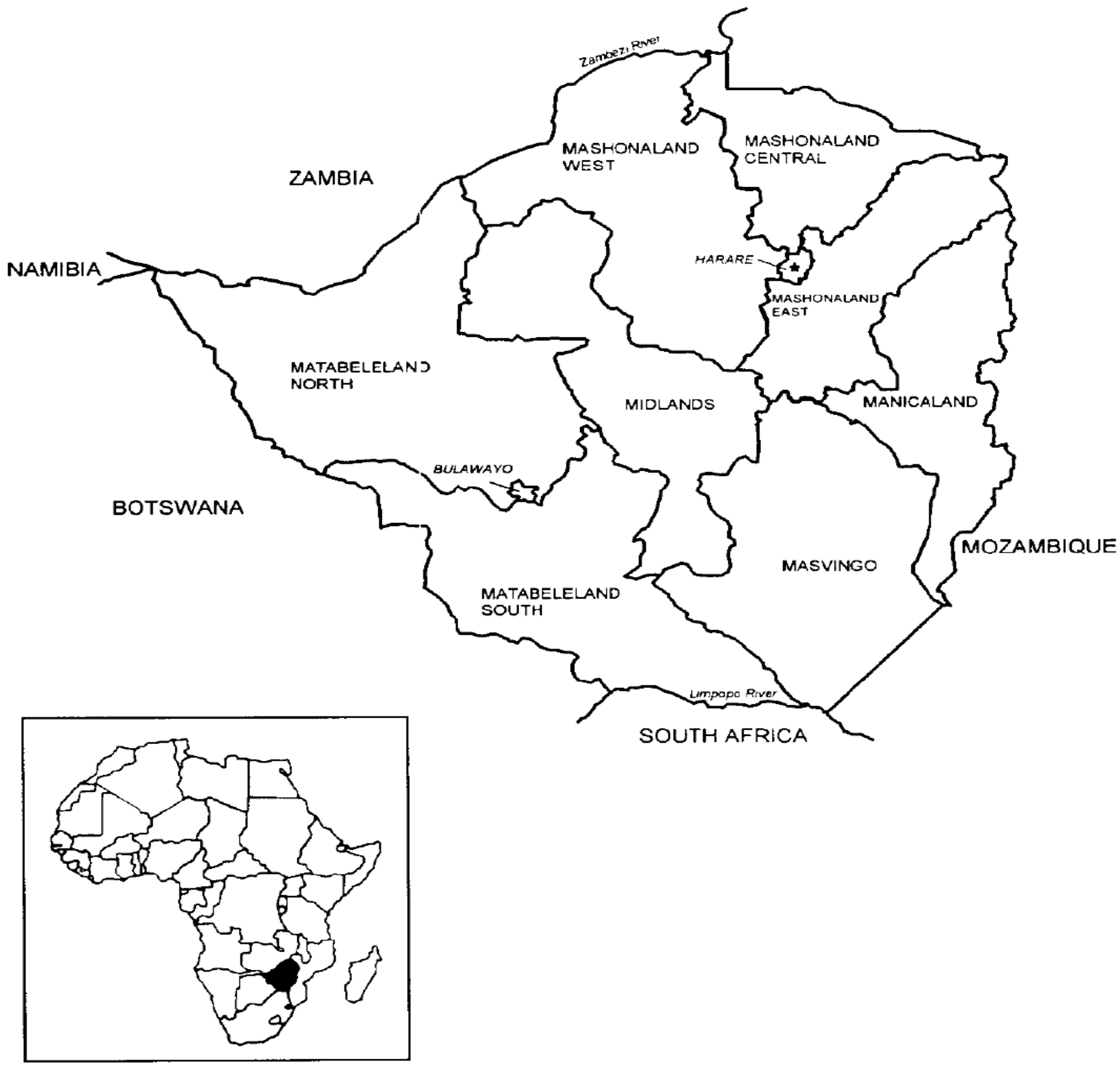
UNAIDS estimates that about 33.3 million people are living with the Human Immunodeficiency Virus (HIV), 68% of whom are in Sub-Saharan Africa (UNAIDS 2010: 18). Globally, the HIV epidemic (measured by HIV prevalence) is thought to have peaked around 1999 with sharp decreases in the number of new HIV infections (especially in Africa) pulling down HIV prevalence rates even as access to antiretroviral treatment (ART) expanded (UNAIDS, 2010:21-5).

Not only is Africa disproportionately affected by HIV, but it also suffers disproportionately from political instability and armed conflict. Indeed, there is an emerging body of empirical literature exploring whether armed conflict could have exacerbated the African AIDS epidemic. But in analysing the cross-country data on this topic, various problems emerge – not least of which pertain to the fact that HIV prevalence rates are not hard data, but are themselves a product of demographic modelling. Part one of the dissertation shows how HIV prevalence rates are derived using UNAIDS modelling packages (EPP and Spectrum), using Zimbabwe as a case study. This first half of the thesis forms a foundation for the second. Part two considers the relationship between armed conflict and HIV prevalence, drawing on lessons from part one – and arguing that data limitations suggest that researchers should be more cautious of cross-country regressions on HIV prevalence and that a mix of qualitative (including contextual and historical studies) and quantitative research is advisable in exploring the relationship between armed conflict and HIV prevalence.

1.1 Structure of the thesis

The thesis begins (Chapter 2) with a detailed explanation of how EPP and Spectrum, the two UNAIDS modelling packages, work to provide national level HIV prevalence and incidence estimates. Zimbabwe is used as a case study. The methodology is to ‘reverse engineer’ the UNAIDS estimates and to show how the implicit assumptions about migration, deaths and behaviour-change drive the results. I then go on to argue that the assumptions about out-migration may actually under-estimate how many people left the country (presumably because of the economic decline and socio-political instability beginning in the late 1990s). A model correcting for conservative, yet plausible migration rates suggests that migration played a much larger role (although behaviour change was a more significant factor) in the course of the epidemic.

Figure 1.1 A map of Zimbabwe



Source: ZDHS (1994)

A key element of this chapter is the estimation of HIV prevalence rates on a province by province basis. Zimbabwe is made up of 10 provinces and is bordered by Zambia and Malawi to the north, Botswana to the west, Mozambique to the east and South Africa to the south (see

Figure 1.1). The different sub epidemics in each province are expected to have followed different paths as each province's geography, demographic composition and economic characteristics are expected to matter. This is more pronounced for border provinces and those that host the major cities of Harare and Bulawayo where vibrant commercial sex industries and other activities linked to transport networks were crucial to the spread of the epidemic.

Chapter 3 gives a relatively detailed account of Zimbabwe's socio-economic history. It narrates how Zimbabwe's first 10 years after gaining independence from colonial rule were full of promise and how the country's socio-economic environment evolved into what we now know as the Zimbabwe "crisis". The main point of this chapter is to give a solid background to the discussions that follow in subsequent chapters.

Chapter 4 discusses the trajectory (both with regard to HIV prevalence and incidence) of the HIV epidemic in Zimbabwe in light of what we know about the country's economic and social history, and the available qualitative evidence for behaviour change, migration and AIDS related mortality. I begin with an outline of the various intervention programs by both government and non-profit organisations (including donor institutions) and how these may have affected the epidemic.

Aside the obvious issues of how resources were allocated (when they were available), I also look at the timing of different policy initiatives. In closing, I speculate on the possible explanations for the different trends in HIV prevalence and incidence observed in the different regions of the country. By reviewing literature on the topic as well as population-based surveys, I pay particular attention to changes in sexual behaviour including condom use and sexual concurrency. An area where HIV prevalence estimates have been used for cross country analysis is in the debate on the link between HIV and conflict. I use the lessons drawn on the estimation of HIV data from the Zimbabwe case (Chapters 2 to 4) to gain further insights into the HIV-conflict debate (part two of the thesis). The detailed description of Zimbabwe's epidemic illustrates how each country's socio-economic environment plays a significant role in determining the trajectory of epidemics such as HIV/AIDS. This complicates the search for an overarching relationship between HIV prevalence and military conflict.

Before looking at how econometricians have used cross country regression analysis to develop models that try to establish a link between HIV prevalence and conflict, I carry out a a review of past studies on the topic. Chapter 5 reviews the literature which posits two alternative scenarios: that armed conflict increases HIV (through rapes, the breakdown of support networks, the associated increase in survival sex, movement of militaries and refugees into new areas etc.); and that armed conflict reduces HIV rates by disrupting the economic and social networks that spread HIV.

Building on the previous chapter, Chapter 6 looks more closely at earlier attempts to model this relationship econometrically. I provide a much more detailed exposition of what these previous studies did. This includes a discussion of their regression output, choice of variables and overall modelling approach. The aim is to unpack the issues concerned with applying national HIV prevalence estimates (which aren't actual data points) to cross country regression models.

In Chapter 7, I provide an alternative set of regressions – both OLS (in which data for the period 1990 and 2005 is used in summary form) and various forms of panel regression models all using HIV prevalence trend data from UNAIDS. I explain in detail how these approaches differ from those discussed in Chapter 6. Finally, I discuss the pros and cons of using cross sectional and panel data. This includes the idea that the fact that HIV prevalence rates are estimates with wide error margins could result in many regressors being statistically insignificant.

Given that the results in Chapter 7 could also be driven by specific countries in the sample, Chapter 8 considers these in more detail. I present graphs of HIV prevalence as estimated by UNAIDS over time. The graphs also contain the data points for HIV prevalence sites to show the relationship between hard data and the estimated trend line. I use the EPP/Spectrum models to reverse engineer these estimates as done in Chapter 2. In addition to HIV prevalence estimates, I also produce incidence estimates as this is where the immediate impact of conflict is likely to be felt. The point of this exercise is not only to allow me to point to specific events or period of armed conflict that may have affected the trajectory of the epidemics in these countries but to show how complex estimates of HIV and incidence can be

– and how careful you have to be in concluding anything from cross-country regressions of the kind discussed in Chapters 6 and 7.

Chapter 9 concludes. It argues that great caution should be adopted towards using UNAIDS estimates of HIV prevalence and that it is important that scholars understand the basis (and limitations) of the demographic modelling that went into the production of HIV prevalence data over time. I also argue that rather than just look at the output, scholars should explore which countries are driving the effects they see – and to then look at the local level dynamics which may be affecting each country differently. In other words, the contribution the thesis makes is to argue that AIDS researchers need to understand the modelled nature of the HIV data they use, and that mixed methods (quantitative and qualitative) are useful in unpacking the relationship between HIV prevalence trends and various social determinants – be they armed conflict, or as in the case of Zimbabwe, mass migration following social and political disruption.

2 USING EPP AND SPECTRUM TO MODEL HIV PREVALENCE IN ZIMBABWE

Abstract

By the reverse-engineering of the UNGASS and MOHCW technical group's estimates on HIV prevalence, I argue that any such estimates produced using EPP and Spectrum packages should be used with caution. Using Zimbabwe as a case study, I show that national estimates of HIV prevalence and incidence obtained using EPP and Spectrum present an average picture of different sub epidemics in the different regions of a country. In Zimbabwe, the HIV epidemic was more menacing in economically vibrant provinces like Harare and Manicaland but not in less vibrant ones like Matabeleland North. EPP and Spectrum estimates also have a margin for error as they are based on ANC site data which is often inconsistently collected and so only few sites have data points for consecutive years. The accuracy of HIV prevalence and incidence estimates also relies on the model assumptions. The assumptions on migration that allow for the Spectrum model to produce prevalence estimates comparable to the UNGASS estimates however underestimate the level of out-migration from Zimbabwe since 2000. By changing this assumption to a conservative estimate of about 2 million out-migrations, I find that in addition to the decrease in new infections and an increase in AIDS deaths, out-migration was a significant (although not the main) contributor to the decline in prevalence in Zimbabwe observed between 1997 and 2010.

2.1 Introduction

The estimation of national HIV prevalence and incidence data for all countries affected by the HIV/AIDS epidemic has traditionally been led by UNAIDS. UNAIDS and WHO jointly publish annual epidemiological fact sheets on each country affected by the epidemic. These reports detail a number of general health indicators such as infant mortality rates, life expectancy but more importantly for this chapter, they also show estimates for the general trend in HIV prevalence and incidence (UNIADS/WHO, 2010). The modelling of HIV prevalence and incidence estimates is carried out by using data from antenatal clinic (ANC) and using it in the EPP and Spectrum models. Further, the process is based on methods and parameters that are informed by the UNAIDS Reference Group on HIV/AIDS Estimates, Modelling and Projections (UNAIDS, 2010). This is a group made up of leading researchers in HIV and AIDS, epidemiology, demography and related areas (UNSTATS, 2011). Drawing from research studies in different countries, the Reference Group assesses the most recent published and unpublished work and also reviews advances in the understanding of HIV epidemics and suggests methods to improve the quality and accuracy of the estimates (UNSTATS, 2011). In other words, although UNAIDS/WHO estimates are shared with national AIDS programmes for review and comments, they are not necessarily the official estimates used by national governments (UNAIDS, 2010). In fact, as is the case for Zimbabwe, some governments produce their own official HIV and AIDS estimates.

Using EPP and Spectrum, Zimbabwe's official HIV and AIDS estimates are produced by the National HIV and AIDS Estimates Working Group (MOHCW, 2009). The MOHCW AIDS and TB Unit leads this national working group but other partners include representatives from the National AIDS Council (NAC), University of Zimbabwe (UZ), Biomedical Research and Training Institute (BRTI), Imperial College London, CDC Zimbabwe, UNAIDS, UNFPA, the United Nations Children's Fund (UNICEF), WHO and Central Statistics Office (MOHCW, 2009:13). Zimbabwe's official HIV estimates are therefore also informed by the UNAIDS Reference Group on HIV/AIDS Estimates, Modelling and Projections. In its 2009 report, the MOHCW states that their report actually provides the figures that are published in the UNAIDS Global AIDS Report (MOHCW, 2009:8). This is part of a drive led by the UNAIDS Joint Programmes on HIV and AIDS aimed at helping countries develop the capacity to produce their own reports (MOHCW, 2009). This thesis refers to "working group" estimates and it should be clear that each time this term is used in reference to Zimbabwe, it applies to both UNAIDS/WHO estimates and the official MOHCW.

According to the 2010 United Nations General Assembly (UNGASS) report on HIV and AIDS in Zimbabwe, adult HIV prevalence (the percentage of adults living with HIV) has fallen from about a peak of 26.5% in 1997 to 14.3 in 2010 (UNGASS, 2010:10). These estimates are based on the UNAIDS supported work by the Zimbabwe National HIV and AIDS Estimates Working group (UNGASS, 2010:4). As is the case with UNAIDS/WHO estimates for other countries, these figures were obtained by obtaining data from antenatal clinic (ANC) sites and using these data in the EPP and Spectrum models (UNGASS, 2010). Using Zimbabwe as a case study, this thesis begins with a detailed explanation of how EPP and Spectrum, the two UNAIDS modelling packages, work to produce the national level HIV prevalence and incidence estimates.

The methodology is to 'reverse engineer'[1] the UNAIDS estimates and to show how assumptions about migration, deaths and behaviour-change drive the results. I describe the modelling strategy and associated projections of key demographic trends, notably HIV prevalence, incidence and AIDS-related mortality. I make a contribution to the on-going discussion of HIV prevalence in Zimbabwe by investigating what was probably assumed on the part of the producers of official HIV prevalence statistics about the underlying trends in AIDS-related deaths and HIV incidence when they used EPP and Spectrum.

As HIV prevalence can fall either because the number of new HIV infections slows down or because the number of people living with HIV declines (through death or emigration) relative to the rest of the population, it is important to understand the dynamics behind any fall in HIV prevalence as they suggest very different epidemic paths and appropriate policy responses. It is also important to note that with death, (and possibly migration, if those migrating are have higher HIV prevalence than those staying or are migrating to access HIV related health care), the more risky may be taken out of the population leading to lower population incidence.

In the case of Zimbabwe, it is important to know whether the key driver was behaviour change (to stem the inflow of new HIV infections), deaths or migration. I argue that the assumptions about out-migration probably under-estimate how many people left the country (presumably because of the economic decline and socio-political instability under President Mugabe). A model assuming slightly higher migration rates suggests that migration played a

[1] This means that an attempt is made at taking apart the various components / assumptions that make up these estimates so as to reproduce the estimates and see how sensitive they are to changes in the underlying assumptions.

larger role in the course of the epidemic although the decline in new infections and AIDS mortality both were significant contributors.

Using Zimbabwe as a case study for how HIV prevalence and incidence estimates are strongly affected by underlying assumptions highlights the caution with which these numbers should be used. I argue that the collection of ANC data is often not reported consistently over time and the modelling packages end up producing national HIV prevalence and incidence estimates that try to create levels and trends from very few data points.

2.2 Epidemiological Projection Package (EPP)

In order to analyse the impact of HIV on the population, one needs to first estimate a national HIV prevalence rate and trend over time (EPP is used in this chapter as well as by UNAIDS and the MOHCW working groups for this). The second step is to apply these estimates of prevalence to a demographic model and Spectrum will be used for this second task. EPP was developed by UNAIDS firstly to enable the capture of the main dynamics of an HIV epidemic in any country even without detailed knowledge of the transmission patterns in the country (Alkema, 2007:5). Secondly, EPP was designed to be simple enough to be used by national planning officials and other stakeholders. Even though the latest version of Spectrum has EPP embedded in it, to allow for a discussion of the EPP modelling process, modelling will be carried out in EPP first before moving to Spectrum. An alternative would have been to carry out all the modelling in Spectrum.

EPP is a software package that implements a differential equation model to reproduce key dynamics of HIV epidemics and considers only the population older than 15. Being a simple susceptible-infected model, the population at time t, (N(t)) is divided into three groups. These are a not-at-risk group X(t), an “at-risk” group Z(t) and an infected group Y(t) (Alkema, 2007:7). These are related as follows;

$$N(t) = X(t) + Z(t) + Y(t)$$

Individuals enter the population when they turn 15 years of age. Those entering the population are assigned to either the not-at-risk group X(t) or the at-risk group Z(t).

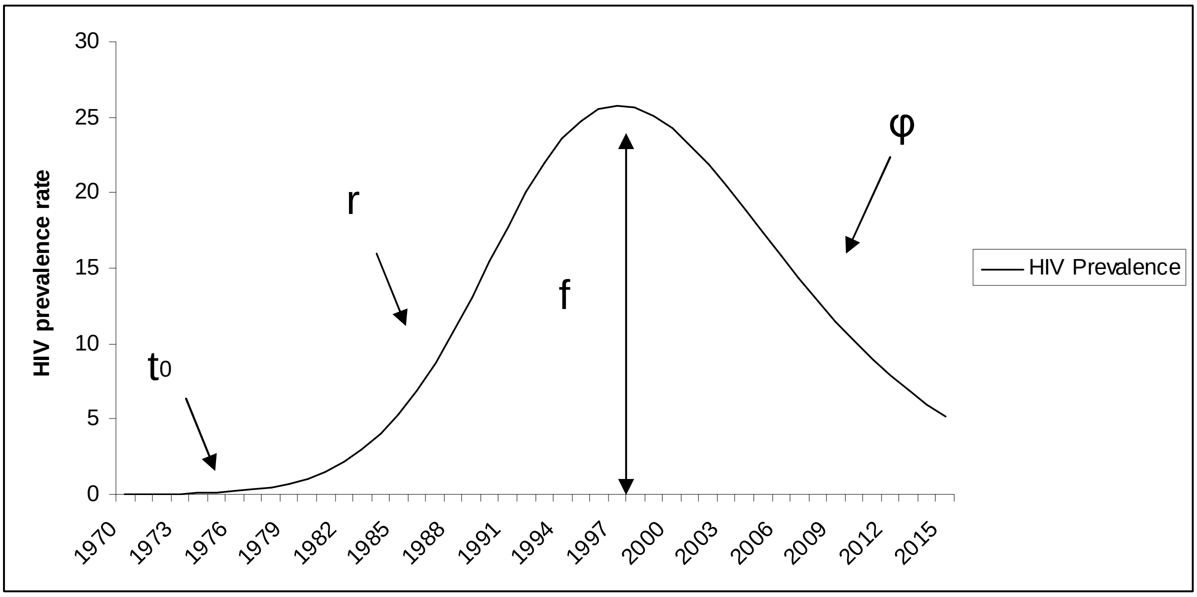
The population size 15 years earlier, the birth rate as well as the survival rate from birth to age 15 determine the total number of new entrants E(t) at time t. The birth rate is applied to both the uninfected and infected groups taking into account the HIV-related fertility reduction experienced by the infected group and the transmission of HIV from mother to child (Alkema, 2007:5). HIV prevalence rises as long as the number of new HIV infections is greater than the number of AIDS deaths. When the number of new infections or rate of incidence is about equal to the mortality rate the epidemic peaks. The epidemic stabilizes when the number of new HIV infections equals the number of AIDS deaths.

Four key parameters dominate the modelling architecture: the rate of infection (r), the start year of the epidemic (t_0), the behavioural response (φ), the fraction of the adult population at risk of infection at the start of the epidemic (f_0) and the rate of replenishment of the population at risk when it is depleted by AIDS deaths. EPP assumes a constant non-AIDS mortality rate (μ) and fertility rate b and does not represent migration or age structure (Alkema, 2007:6).

EPP generates 50,000 to 200,000 epidemic curves by randomly selecting values of the four key parameters from plausible distributions. However, it also allows one to make adjustments to these parameters in cases where the information is available. Using Figure 2.1, I attempt to explain these parameters. The behavioural response (φ) influences the level at which the epidemic levels off after the peak while changing the start date of the epidemic does not change its shape (the shape of the epidemic) but only its timing. The parameter f_0 (fraction of the population in the at-risk category when the epidemic starts) [2] influences when and at what prevalence the epidemic peaks. The epidemic will peak at a higher level if the proportion of the population initially at risk is higher.

[2] The graph shows f (the peak) and this is determined by $f_{0,}$ the fraction of the population in the at-risk category when the epidemic starts.

Figure 2.1 Modelling Parameters



Source: UNAIDS Reference Group on Estimates, Modelling and Projections, 2009

Explaining the behavioural response parameter requires slightly more effort. The behavioural response (φ) determines the level at which the epidemic levels off after the peak. Negative values of φ, correspond to a situation where new members of the population change their behaviour when they see others dying of AIDS, so that fewer of them enter the at-risk group. Positive values of φ correspond to the opposite situation in which a larger fraction of new members enter the at-risk group. The assumption here is that there are some in the general population who gradually enter the at risk population. These are people who for various reasons including culture or geography were not previously at risk. The faster the rate of infection r, the quicker prevalence increases at the start of the epidemic.

The not-at-risk group X(t), the at-risk group Z(t) and the infected group Y(t) are all dynamic in size. They are changed through recruitment, behaviour and infection and described by the following differential equations:

$$\frac{dX(t)}{dt} = \{1 - f(\frac{X(t)}{N(t)}, f_0, \Phi)\} \times E(t) - \mu \times X(t) \qquad \text{equation (1)}$$

$$\frac{dZ(t)}{dt} = f\{\frac{X(t)}{N(t)}, f_0, \Phi\} \times E(t) - \{\mu + r\frac{Y(t)}{N(t)} + \lambda(t)\} \times Z(t) \qquad \text{equation (2)}$$

$$\frac{dY(t)}{dt} = \{r\frac{Y(t)}{N(t)} + \lambda(t)\} \times Z(t) - \int_0^t \{r\frac{Y(\tau)}{N(\tau)} + \lambda(\tau)\} \times Z(\tau)g(t-\tau)d\tau \qquad \text{equation (3)}$$

Where μ represents the non-HIV death rate and g(τ) specifies the HIV death rate τ years after infection. Survival after infection is assumed to have a Weibull (2.4, 10.5) distribution (Alkema, 2007:7). Such an assumption is appropriate here because under these parameters, ($\alpha = 2.4$ and $\beta = 10.5$) the median of the distribution, i.e. the median survival time is 9 years. EPP assumes that the time at which the fraction $\lambda_{0,}$ of the at-risk group Z moves to infected group Y marks the start year of the epidemic. The function below represents the proportion of new 15 year olds who enter Z(t) at time t. The rest enter the not-at-risk group X(t) (Alkema, 2007:7). As mentioned earlier, the total number of new members at time t, E(t), depends on the population size 15 years ago, the birth rate as well as the survival rate from birth to age 15. In addition, the birth rate is applied to both the uninfected and infected groups taking into account the HIV-related fertility reduction experienced by the infected group and the transmission of HIV from mother to child. The fraction of new 15 year old members entering the at-risk group Z(t) at time t is given by the function f below. The rest of the new 15-year-olds enter the not-at-risk group X(t) (Alkema, 2007:7).

$$f\left(\frac{X(t)}{N(t)}, f_{0,} \Phi\right) \qquad \text{equation (4)}$$

2.2.2 Modelling Process

In order to project HIV prevalence using EPP one has to use the EPP program to input the modelling assumptions. Here the Zimbabwean AIDS epidemic is modelled as a “generalised epidemic”. An epidemic is labelled “generalised” if HIV prevalence amongst pregnant women is over 1%. ‘Concentrated epidemics’ are epidemics which are concentrated in high risk groups (e.g. prostitutes or injecting drug users). In such epidemics, it is assumed that HIV prevalence is 5% or higher in the group, and less than 1% amongst pregnant women (Wilson, 2006:4).

A generalised epidemic can be modelled in one of two ways. The epidemic can be modelled as the outcome of different processes of infection either defined by sub-population (e.g. rural/urban, injecting drug users etc.) or geographically (e.g. by province). It can also be modelled more broadly as a one country-wide process. EPP was used to model the epidemic using both. First, HIV prevalence was modelled for each of the ten provinces in Zimbabwe. EPP allows for the setting of different parameters for different sub-epidemics (by province) which allows the level and spread of HIV infection to differ within the general population. For this to be possible, demographic data for each sub-epidemic needs to be available.

The basic assumptions are set by the UN population division[3]. These include a proportion male (0 to 1) of 0.5, birth rate in 15+ (b) of 0.06408, survival to age 15 (l_{15}) of 0.86214, population growth rate (ages 15+) of 0.01120. These show little to no variation across the provincial populations. The population distributions were as follows; Harare - 16.4%, Bulawayo - 5.8%, Mashonaland East - 9.7%, Mashonaland Central - 8.6%, Mashonaland West – 10.5%, Matabeleland North – 6.0%, Matabeleland South - 5.6%, Manicaland - 13.5%, Masvingo - 11.3%, and Midlands - 12.6%. These are figures given in Zimbabwe's last national census which was held in 2002 (CSO, 2002).

After setting the demographic data, EPP then prompts you for HIV prevalence data. As alluded to earlier, the HIV data used is the ANC site data collected on women aged 15–49 years attending ante-natal clinics. This data is in Addendum A and is summarised in Figures 2.2 to 2.6. The first diagram in Figure 2.2 shows reported ANC HIV prevalence data for Harare province. Owing to the fact that HIV prevalence in rural and urban areas often follows different trends, it is important to highlight that all Harare ANC sites (13 sites) are categorised as urban. The dark line shows averages of all the reported HIV prevalence site data in the province. As the graph shows, some sites show slight deviations from the mean. These data points will be referred to as outliers. Noticeably, HIV prevalence data reported from Seke North Clinic seems to differ from the average especially in 1992 (the site reported 34 versus an average of 25.9). However, because the data from this site may contain useful information, it will not be thrown away in this study. As a general rule, only sites where irregularities have been reported will be candidates for exclusion.

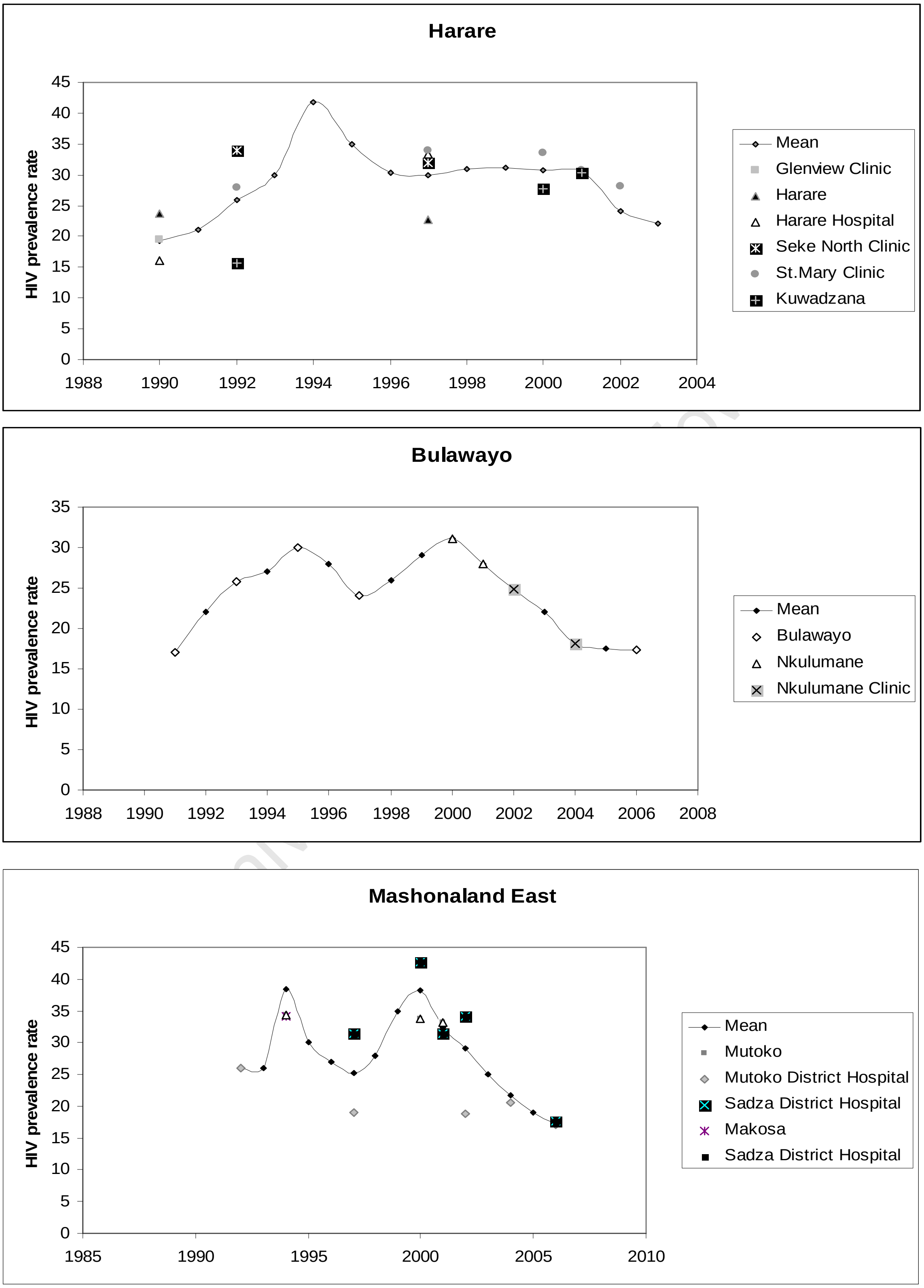
[3] See http://www.un.org/esa/population/unpop.htm or the United Nations Department of Economic and Social Affairs / Population Division PRED Bank 4.0 Country Profiles available at http://www.un.org/esa/population/publications/countryprofile/zimbabwe.pdf

Bulawayo province only reports 3 ANC sites with more than one data point. Not one of the sites reported HIV prevalence data consistently from 1990 to 2006. This poses a credibility problem. The fact that ANC data may have data points so few as to make it difficult to map the progression of the epidemic in the province, the modelling process often involves the calibration of the data with population survey data. This not only helps produce a trend line for cases such as Bulawayo where few data points exist but it also helps in some cases where ANC data may be disregarded because of irregularities either in the HIV testing or the handling of the testing results.

Of the 17 ANC sites reported from Mashonaland East, the most noticeable outlier is Katsukunye. Prevalence reported from this site seems particularly high in 1994 (54%) when compared to the average site prevalence for the province in that year (38.5 %). Data from this site seems suspect as there is only one data point reported by UNAIDS on this site and this makes it difficult for one to infer a trend (Figure 2.2 only shows sites for which there 2 or more data points). It therefore should not be objectionable to omit this site from the modelling. In fact as a general rule, all sites where only one test data point is available were omitted from the analysis. Mahomva et al (2006:i43-44) suggest that the quality of the blood samples from the Sadza ANC was compromised, most likely during transportation. As a result, instead of using the Genscreen test kit as was done for the other sites, two different ELISA test kits with a third kit as a tie breaker were employed to test the samples obtained from Sadza.

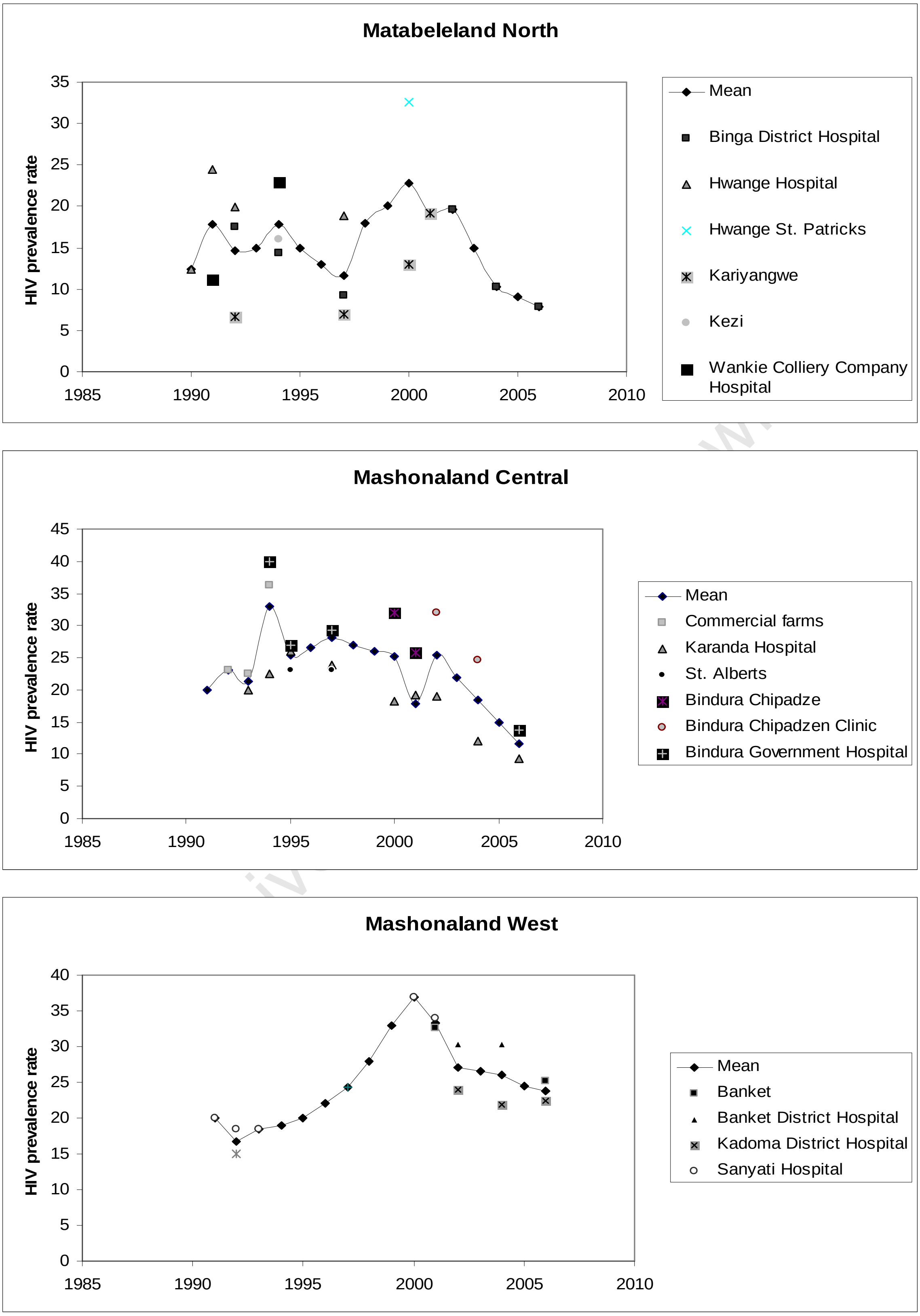
HIV prevalence in pregnant women in Mashonaland Central was particularly low at St. Albert’s mission Hospital in 2001 where prevalence averaged 8.3% whereas the average was 17.8%. As this is the only data point reported for St. Albert’s Mission Hospital in more than 15 years of ANC testing, it will be will be excluded from the modelling. Average HIV prevalence reported at Bindura Government Hospital drove mean prevalence up. Data on a total of 18 ANC sites was reported from Mashonaland Central (although only 5 had 2 or more data points). In Mashonaland West, data on only 6 ANC sites was reported (only 4 had 2 or more data points). As a result, the plot of site prevalence in Mashonaland West with the mean site prevalence does not show any outliers.

Figure 2.2 Mean ANC prevalence data by Province



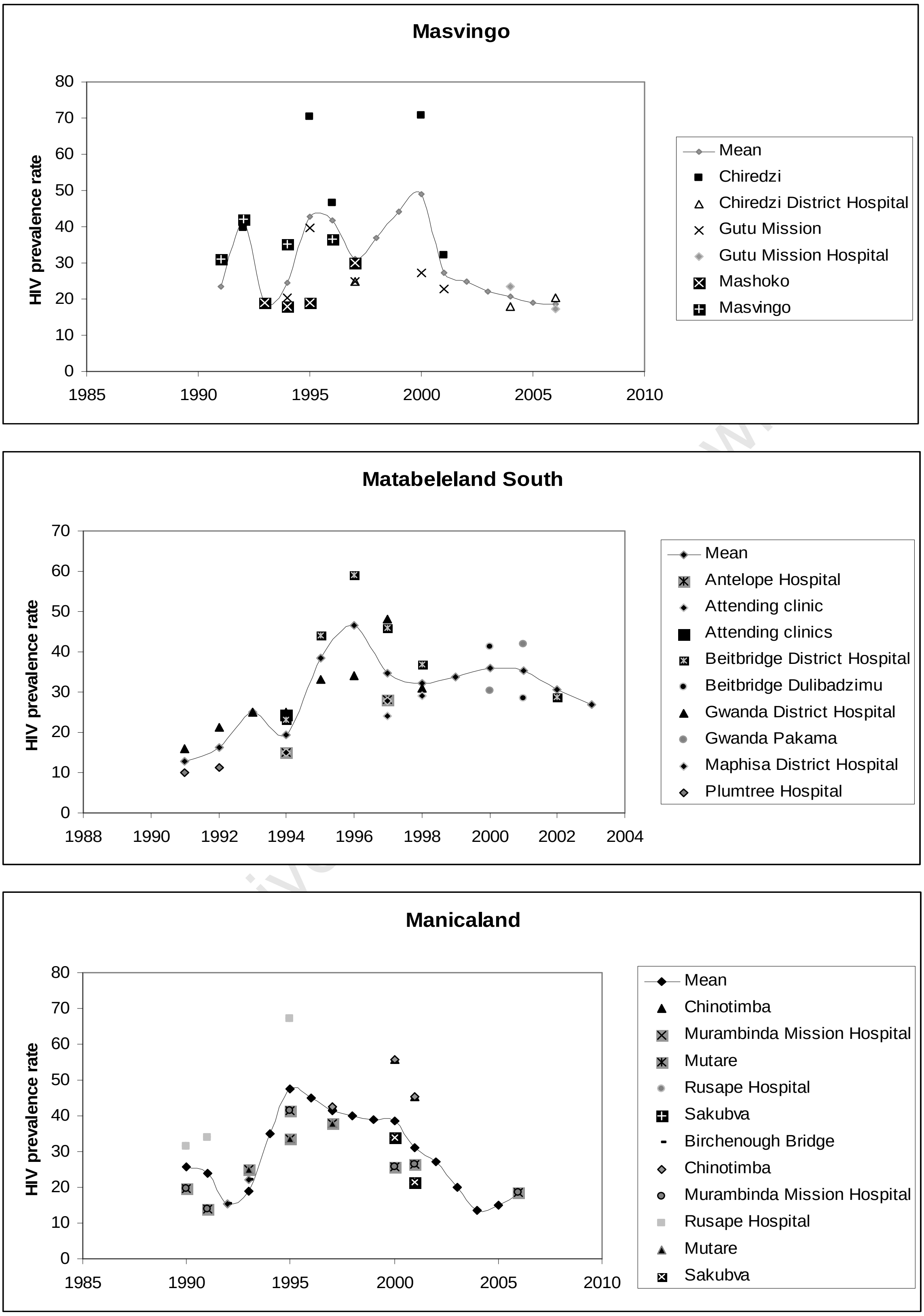
Source: UNAIDS Epidemiological Fact Sheet 2008

Figure 2.3 Mean ANC prevalence data by Province



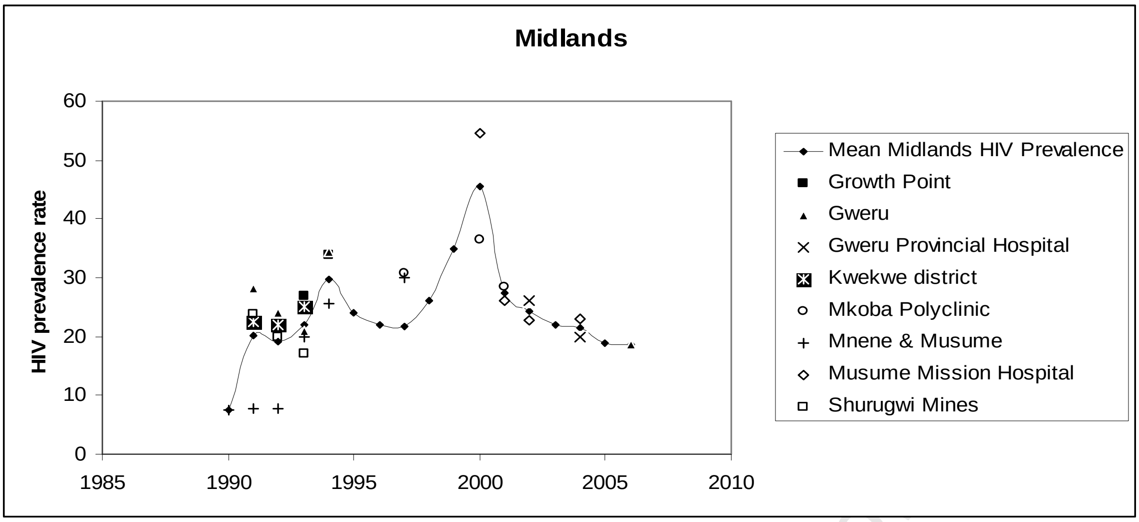
Source: UNAIDS Epidemiological Fact Sheet 2008

Figure 2.4 Mean ANC prevalence data by Province



Source: UNAIDS Epidemiological Fact Sheet 2008

Figure 2.5 Mean ANC prevalence data by Province



Source: UNAIDS Epidemiological Fact Sheet 2008

Beitbridge District hospital in Matabeleland South has 18 sites, with only 7 having more than 2 data points. The most interesting ANC site in Masvingo (20 sites, with 6 having more than 2 data points) is the Chiredzi ANC site. The data points for Chiredzi were not reported consistently over time. As a result, the National HIV and AIDS Estimates Working group excluded all data from this site prior to 2001 when they produced their estimates (MOHCW, 2009:38). Manicaland province has data on 32 ANC sites but only 11 had 2 or more data points. The visible outliers include Rusape which reported a 1994 site prevalence rate of 67% against a provincial average of 47.33% in the same year. Another extreme case is that of Chinotimba which reported a 2000 prevalence rate of 55.7% versus a provincial average of 38.43%. 13 ANC sites reported HIV prevalence data in the Midlands province (with 7 having more than 2 data points). The most noticeable outlier in the province is Musume Mission Hospital. As a matter of fact, the MOHCW working group excluded data for the Musume site in 2000 because the prevalence rate that year was not consistent with other years (MOHCW, 2009:38).

A number of sites have only a couple of years of reported data as alluded to earlier. In order to reverse engineer the MOHCW and UNAIDS estimates, an attempt will be made to use assumptions as close to those used by this working group as possible. The MOHCW decided to adjust down by 30%, prevalence rates calculated using results from the Genscreen HIV test (1989 through 2001) for the rural ANC sites Birchenough Bridge, Gutu, Hauna Growth Point, Murambinda, Mutoko and Sadza (MOHCW, 2009:38). Their argument here was that data from local epidemiological studies indicate that HIV prevalence rates are approximately 30% higher in these areas compared with the truly rural areas (MOHCW, 2009:38). These six sites are in rural regions but they typically are growth centres or are linked commercial centres via road networks. Most of the women attending ANCs at these sites live in or near a commercial centre and HIV prevalence at these ANC sites was 30% higher than in the ANC sites that the working group classified as truly rural (MOHCW, 2009:38).

In the modelling exercise presented here, only sites with a minimum of three data points will be included. This is in line with the modelling recommendations published by the UNAIDS Reference Group on Estimates, Modelling and Projections which advises that analyses of trends should be restricted to those sites with consistent reporting over time (UNAIDS, 2004:4). They suggest that three data points showing a consistent trend in prevalence are needed to conclude there is a trend. Another condition is that the time spanning the data points should be 3-6 years. It should also be noted that often the period of most interest for evaluation of a decline in prevalence will be some years after prevalence has peaked. This is because the decline in prevalence immediately following the peak prevalence is usually part of the natural epidemic due to mortality of those most at risk of infection who were infected early on in the epidemic (UNAIDS, 2004:4).

2.2.3 How representative is ANC HIV prevalence data?

When assessing the efficacy of EPP prevalence estimates it is important to consider that HIV prevalence among pregnant women at ANC clinics may not be the same as among all men and women in the general population. In other words, an argument can be made that it is not ideal to use ANC prevalence data to model HIV progression in the whole population. However, the UNAIDS Reference Group on Estimates reported that although on average, in sub-Saharan African populations, ANC estimates are approximately 10% lower for women in the general population and 10% too high for men, ANC estimates approximate the HIV prevalence among men and women combined reasonably well (UNAIDS, 2002).

Gregson et al (2002: 643-652) argue that ANC estimates may more closely approximate HIV prevalence among women in the general population. This means they substantially overestimate the prevalence in men and women combined in some locations especially in areas where risk factors are relatively lower e.g. locations where age at first sex is relatively high. However, spatial patterns of ANC clinic utilization may further complicate the pattern at the local level. The MOHCW working group on HIV estimates attempted to assess the representativeness of ANC data by investigating the relationship between HIV prevalence in ANC attendees and HIV prevalence in men and women in the general population in Zimbabwe as proxied by a survey study. They used data from the Zimbabwe Young Adult Survey (YAS), a population-based HIV prevalence and behavioural survey among 15 to 29 year olds in which pregnancy data were also collected (MOHCW, 2009:38). The MOHCW working group focused on ANC data from Manicaland and assessed studies of bias in ANC and population-based estimates conducted in Manicaland from 1998-2000.

What the working group did was to take the HIV prevalence rates in 2001 at the ANC sites in Harare (30.6%) and Bulawayo (27.9%) to obtain an average which was assumed to be an overall estimate for urban areas in 2001 (29.3%)(MOHCW, 2003)[4]. By extrapolating HIV prevalence among women aged 15 to 29 years in the 2001 YAS to that among women aged 15 to 49 years, using the ratio of HIV prevalence in these two age ranges for the more urban areas in the Manicaland project, the assessment showed that HIV prevalence in ANC attendees (29.3%) is a reasonable estimate of that among all women (approximately 27%) in urban areas (MOHCW, 2003; YAS, 2004; MOHCW, 2006).

Taking the same approach of extrapolation of YAS data for men yields an HIV prevalence of approximately 20% among men aged 15 to 49 years with the female-to-male HIV prevalence ratio at approximately 1.35:1[5](MOHCW, 2009:38). The data from large-scale commercial farms and subsistence farming areas in the Manicaland project showed that ANC estimates in both rural and urban areas understate the HIV prevalence among all women aged 15 to 49 years by 15% and overstate the HIV prevalence among all men aged 15 to 49 years by 15%

[4] It might have been methodologically preferable to take the different route of using some kind of weighted average instead of a simple average.

[5] (27%/20%)

(MOHCW, 2009:38; Gregson et al, 2002:643-652). This presents a case for calibration of the ANC data to make it more representative and this is an option available when modelling with EPP.

In the modelling exercise presented here, the ANC data is calibrated using the Zimbabwe Demographic and Health Survey of 2005-06 (ZDHS). The MOHCW working group also calibrated the ANC data using the ZDHS as will be discussed in further detail shortly. The 2005-06 ZDHS is according to the Zimbabwe Central Statistical office, a nationally representative survey of 8,907 women aged 15-49 and 7,175 men aged 15-54. A total of 9,778 households were sampled and the response rate was 95% with 9,285 being successfully interviewed. The Zimbabwe Master Sample (ZMS04) is the sampling frame used for the 2005-06 ZDHS. Developed by the Central Statistical Office (CSO), the ZMS04 had 1,200 enumeration areas (EAs which were allocated proportional to the total population and stratified by land use type (ZDHS, 2006:8). The sample was selected in two stages with the first stage being made up of 400 EAs that were selected with equal probability from the ZMS04 and stratified by urban and rural clusters for each of the 10 provinces. A complete household listing and mapping exercise was conducted in January 2005 within each of these 400 EAs (ZDHS, 2006:8).

Stage two involved the listing of households for each cluster. Each and every private household was listed and individuals residing in institutional households such as boarding schools, police camps or hospitals were excluded. After comprehensive training on the use of Global Positioning Systems (GPS), CSO provincial supervisors and their officers took the coordinates of the 2005-06 ZDHS sample clusters. All men aged 15-54 and all women aged 15-49 who were either visitors present in the household on the night before the survey or permanent residents of the households in the 2005-06 ZDHS sample were eligible to be interviewed (ZDHS, 2006:8). The ZDHS included an HIV testing toolkit and so eligible men and women were tested and results of their HIV status reported in the ZDHS report (ZDHS, 2006:10).

Upon obtaining their consent, anaemia and HIV testing was carried out on eligible women and men in each household. Children (age < 6) were only tested for anaemia but this was on condition that the parents or guardians gave their consent. The testing was anonymous and all women and men who were eligible for the individual interview were asked if they would consent to give a blood sample for HIV testing. For the 2005-06 ZDHS, the Dried Blood Spot (DBS) method of specimen collection was used. A one-use, disposable lancet was used to perform a finger prick, and the interviewer collected 5 drops of blood on specialized filter paper cards, also known as the DBS card. Placed on the DBS card were Barcode labels which kept track of the corresponding questionnaire for the respondent, and the blood transmittal sheet for the cluster. Details on how the cards were stored and finally taken for testing are provided by the MOHCW (ZDHS, 2006:10).

Table 2.1 2005-2006 ZDHS HIV prevalence and Mean ANC data by Province

Province	Women HIV+	Men HIV+	Total HIV+	ANC Avg	difference (1)	difference (2)	difference (3)
Manicaland	22.3	16.6	19.7	18.4	-4.0	1.8	-1.4
Mashonaland Central	22.9	13.8	18.5	11.6	-11.3	-2.2	-6.9
Mashonaland East	21.3	14.4	18	17.4	-4.0	3.0	-0.6
Mashonaland West	22.5	15.4	19.1	23.8	1.3	8.4	4.7
Matabeleland North	22.8	14.4	19	7.6	-15.2	-6.8	-11.4
Matabeleland South	24.6	15.6	20.8	25.8	1.2	10.2	5.0
Midlands	20.1	11.5	16.1	18.6	-1.5	7.1	2.5
Masvingo	17.3	12.1	15.1	18.8	1.5	6.7	3.7
Harare	21.1	17.3	19.3	15.6	-5.6	-1.8	-3.8
Bulawayo	19.6	12.8	16.8	17.3	-2.3	4.5	0.5
Average	21.5	14.4	18.2	17.5	-4.0	3.1	-0.8

Source: UNAIDS Epidemiological Fact Sheet 2008 and 2005-2006 ZDHS

Table 2.1 shows a comparison of the ANC data with data from the ZDHS. It is important to note that the actual ANC data had no HIV prevalence data reported for 2005. This means that the average ANC data above had to be obtained by averaging reported ANC data for 2004 and 2006. The columns labelled difference 1 to 3 show the difference in estimates between the ANC data and the DHS reported data. Difference 1 shows the difference between prevalence reported in the ANC data and that in women aged 15-49 reported in the ZDHS. This difference appears to be much larger than the difference between prevalence reported in the ANC data and that in men aged 15-54 reported in the ZDHS (difference 2). Prevalence reported in the ANC data does seem to be very close to prevalence in the general population overall (difference 3). This observation is somewhat comforting but it does not mean that the ANC data can be used without calibration[6].

Entering the ANC data into EPP is a very simple process and once this has been done for each site in every province, the program requires input of antiretroviral (ART) program data. The ART data and basic assumptions used for the modelling were obtained from the UNAIDS Epidemiological Fact Sheet (UNAIDS, 2008:12). Zimbabwe has implemented a treatment program that has expanded the number of adults receiving ART from an estimated 11000 in 2004 to 79813 by the end of 2007; about 19% of need (UNAIDS, 2008:2-6). Assuming this expansion rate was maintained (9.5% annually), the number of people on ART in 2010 is expected to be around 230 000. The base data used in the EPP has been reproduced below in Table 2.2.

Table 2.2 Estimated number of people receiving ART

Year	2004	2005	2006	2007	2008	2009	2010
Estimate	1,100	25867	55,761	79,813	133,854	210,000	230,000

Source: UNAIDS Epidemiological Fact Sheet 2008 (most recent), MOHCW report 2009.

Once data on ART interventions has been input, EPP allows for the projections to be calibrated using nationally representative surveys. For each of the provincial sub populations, EPP allows for up to 3 results from surveys to be entered. The 2005-2006 ZDHS was used in this study and once this was done, EPP then carries out the projections by fitting a maximum

[6] By comparing HIV seroprevalence estimates obtained from antenatal ANC sentinel surveillance surveys in Ethiopia, Kenya, Malawi, Tanzania and Uganda with those from population-based demographic and health surveys and AIDS indicator surveys, Montana et al (2008) find that ANC surveillance surveys tend to overestimate HIV prevalence compared to prevalence among women in the general population in DHS/AIS surveys. However, the ANC and DHS/AIS estimates are similar when restricted to women and men, or to women only, residing in catchment areas of ANC sites.

likelihood curve through both the ANC surveillance data and the ZDHS data through the process described in described in Section 2.2.

Once EPP produces its best fit, the program allows the user to try and improve it. This can be done by adjusting the four key modelling parameters namely; the rate of infection (r), the start year of the epidemic (t_0), the behavioural response (φ), the fraction of the adult population at risk of infection at the start of the epidemic (f_0) and the rate of replenishment of the population at risk when it is depleted by AIDS deaths. The EPP manual recommends that when modelling, these four parameters should not be adjusted until after the first curve with a fixed the behavioural response (φ) and phi and the start year of the epidemic (t_0) has been fitted. In this study no such adjustments were made.

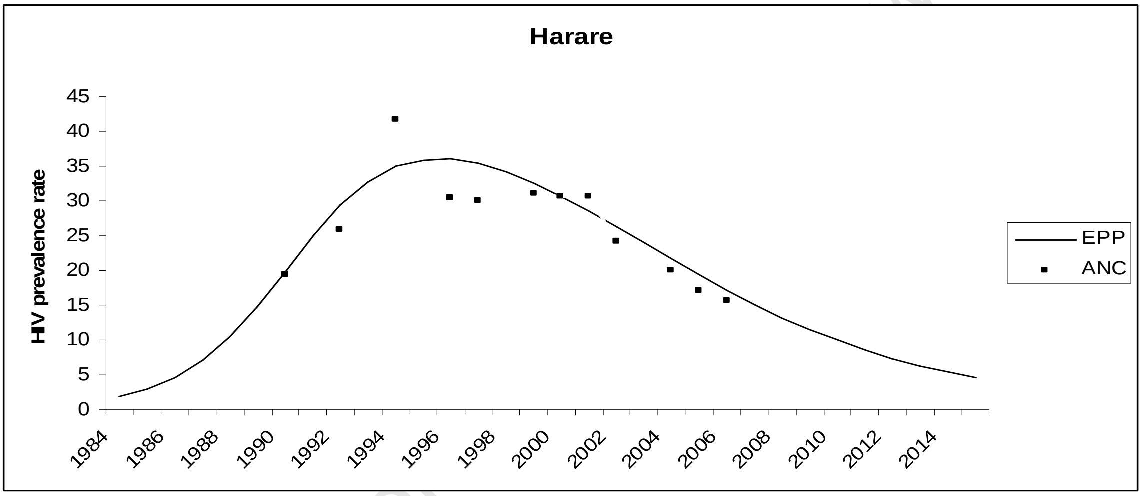
Once the user is satisfied with the final estimates produced by EPP, they can also do uncertainty analysis. This has been standard practice for UNAIDS estimates since 2003 when they included information on uncertainty in the estimates and projections by calculating and presenting plausibility bounds. The uncertainty/plausibility bounds were derived by combining the results of a bootstrap method with expert opinion regarding the range of possible epidemic curves (Alkema et al, 2008:2). This was first detailed by Grassly et al (2004) and further developed by Morgan et al (2006). However these plausibility bonds are not formal statistical confidence intervals and so Alkema et al (2007) proposed using Bayesian melding for uncertainty assessment in the model in EPP. Bayesian melding provides a way of including expert opinion while still giving formal statistical confidence intervals which reflect the uncertainty about the past and future Alkema, et al (2008:3). The confidence intervals reflect that the modelling results are based on imperfect information about the model's inputs and outputs Alkema, et al (2008:3).

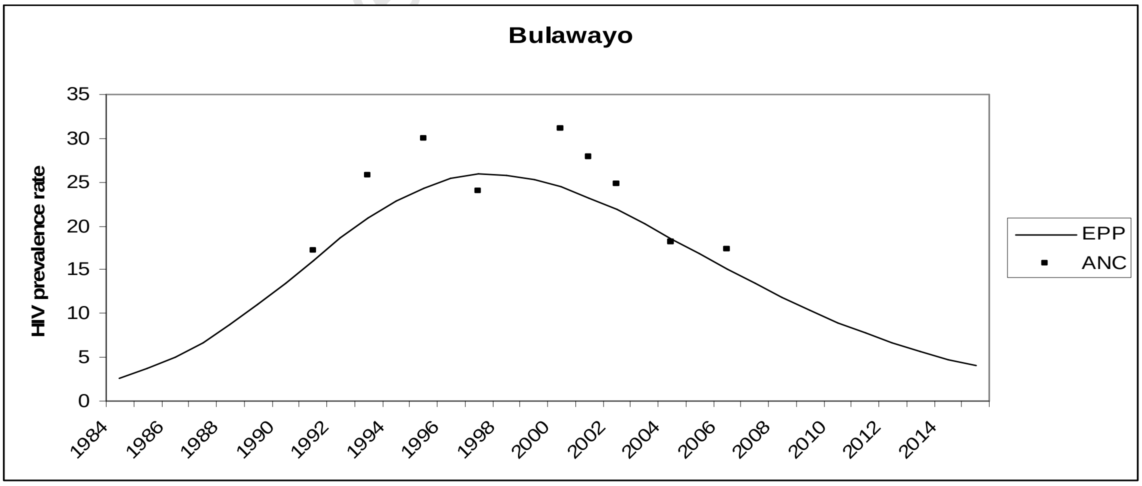
Finally, EPP allows the user to accommodate any known changes in the population that may have occured. It is recommended that these not be changed unless there is convincing evidence and data showing that there were changes in the populations. This cautionary point will be more useful in upcoming sections where the UN Population division's assumptions on migration in and out of Zimbabwe will be tested.

2.2.4 EPP Results

As already alluded to, a generalised epidemic can be modelled in one of two ways. The epidemic can be modelled as the outcome of different processes of infection either defined by sub-population (e.g. rural/urban, injecting drug users etc.) or geographically/by province. It can also be modelled more broadly as a one country-wide process. This study did both. First, HIV prevalence was modelled for each of the ten provinces in Zimbabwe using the ANC and ZDHS data described above. Figures 2.6 to 2.9 shows these results.

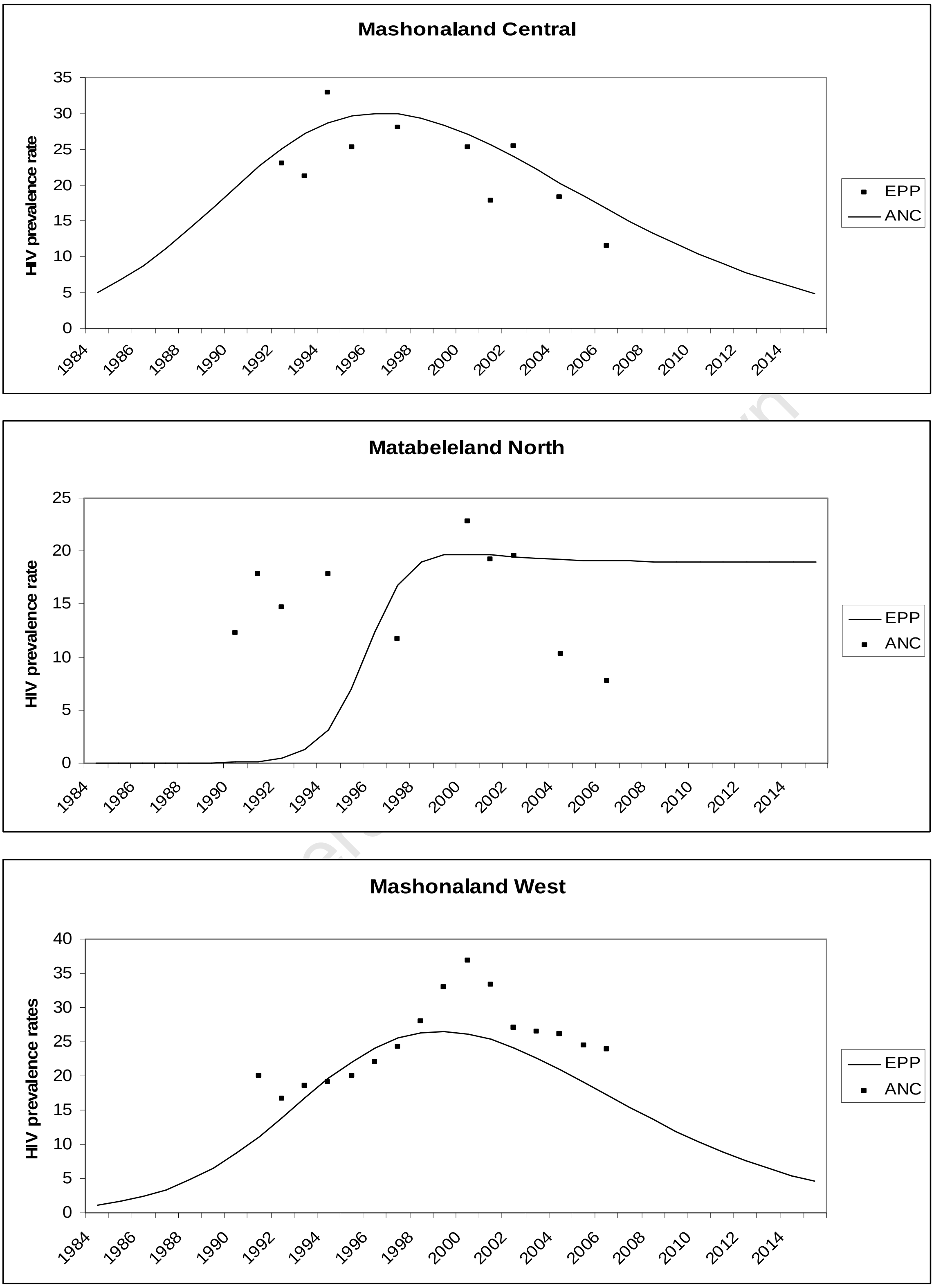
Figure 2.6 EPP HIV prevalence (age 15-49) projections by Province





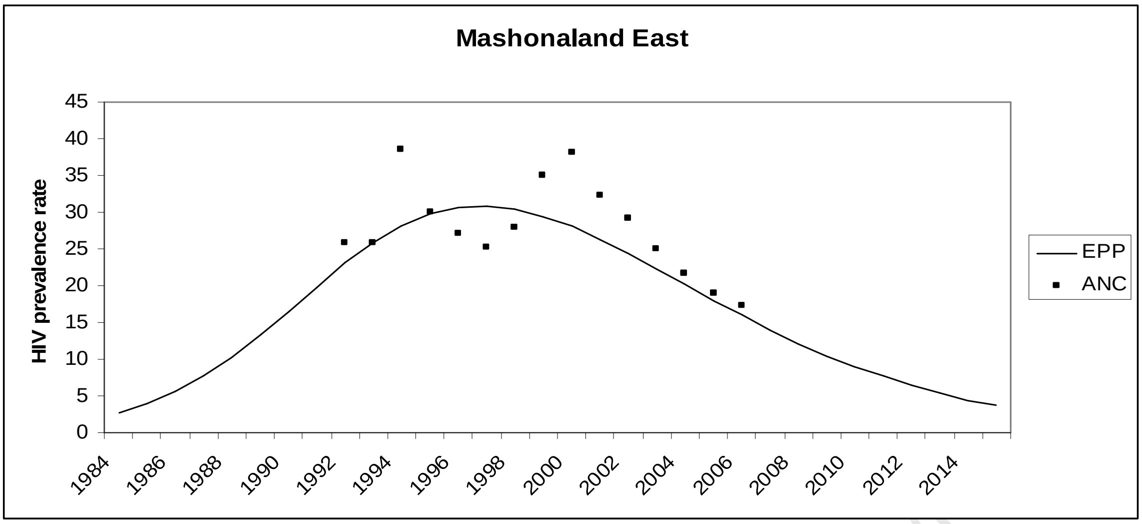
Source: EPP 2009 projections

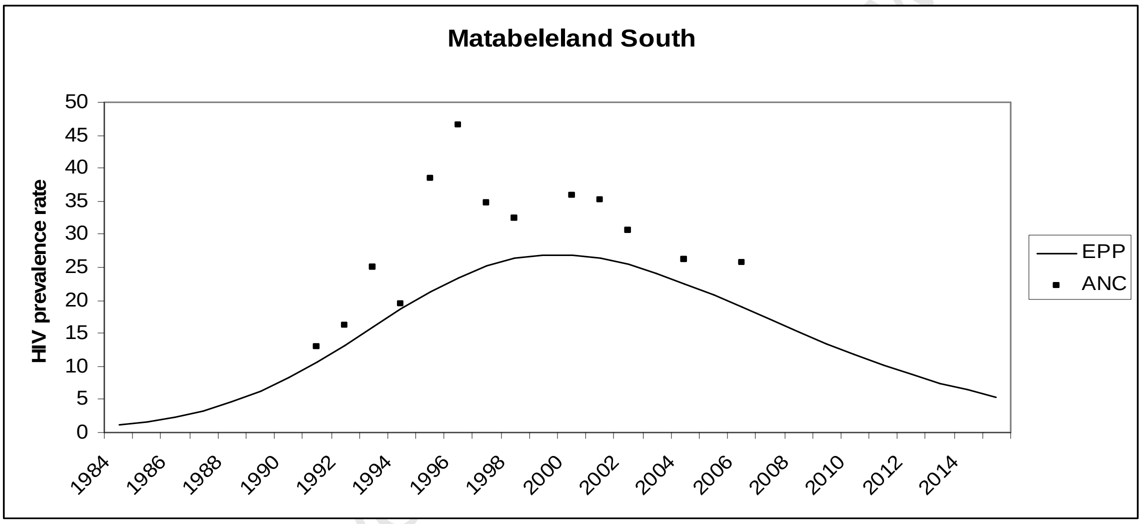
Figure 2.7 EPP HIV prevalence (age 15-49) projections by Province

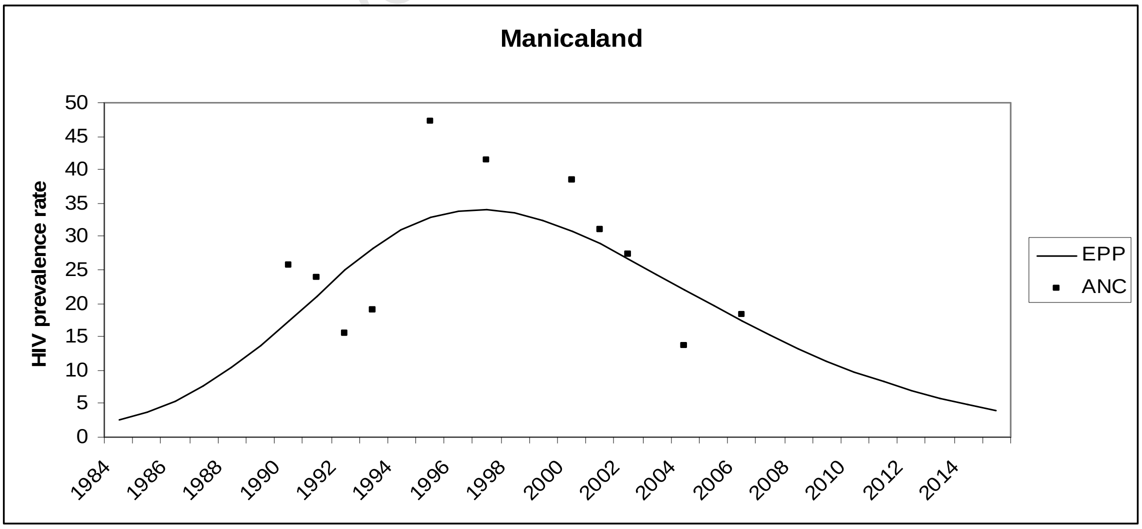


Source: EPP 2009 projections

Figure 2.8 EPP HIV prevalence (age 15-49) projections by Province

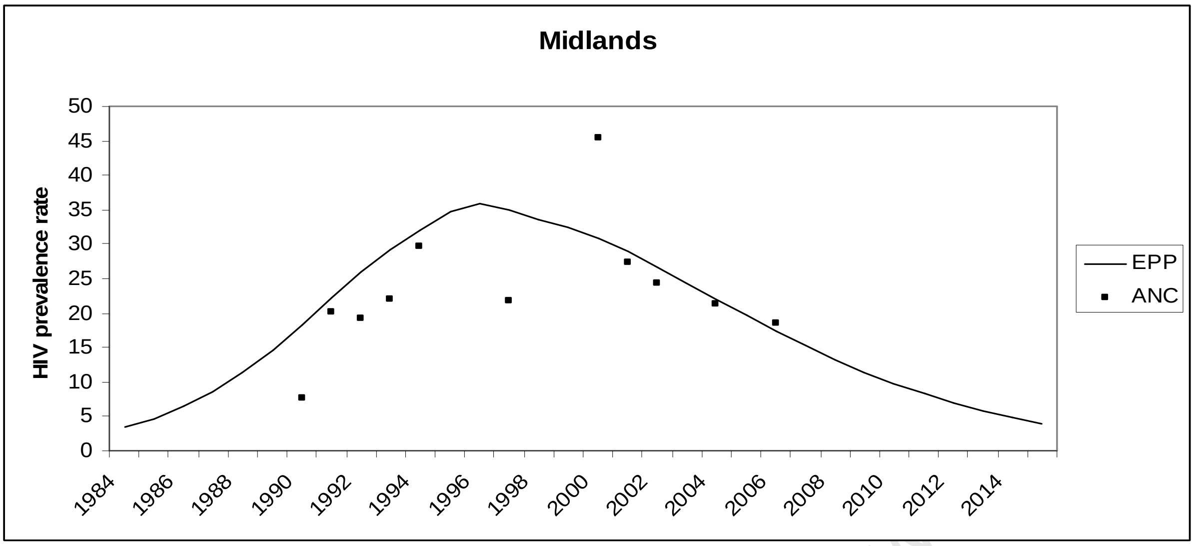


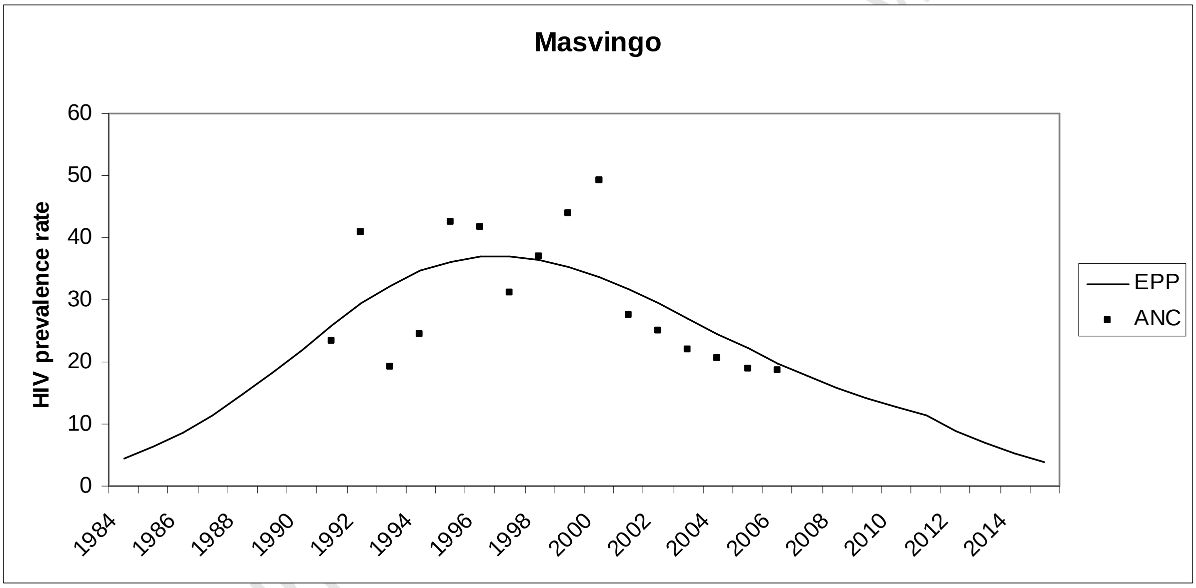




Source: EPP 2009 projections

Figure 2.9 EPP HIV prevalence (age 15-49) projections by Province





Source: EPP 2009 projections

One important point to note is that because there are differences between provinces with respect to consistency of reporting ANC data over time, province versus province comparisons have some limitations. Before analysing these results, it is also important to note that EPP estimates are more preliminary than they are final. In addition, because they are only estimates, there are error margins associated with the estimates. Even though EPP can produce these estimates, the current software does not allow the user to extract the actual estimates of the confidence intervals. They are shown in a chart in EPP format but they the figures are hard to unpick. However, the final HIV prevalence estimates and confidence intervals can be obtained by running the EPP results through the Spectrum modelling package. Spectrum does produce confidence intervals for the error margins (as presented in later sections) although this is only for national level estimates[7]. Notwithstanding, Figures 2.6 to 2.9 do give us a feel for the trends in HIV prevalence geographically. EPP prevalence estimates in Harare suggest that HIV prevalence peaked around 1996 (35.9%) having rapidly increased from around 1985 (2.9%). HIV prevalence seemed to peak at almost the same time in all non-border provinces. In the urban province of Bulawayo for instance, prevalence peaked in 1997 (25.9%) and the highest prevalence rates are estimated to have been in Masvingo (37% in 1997).

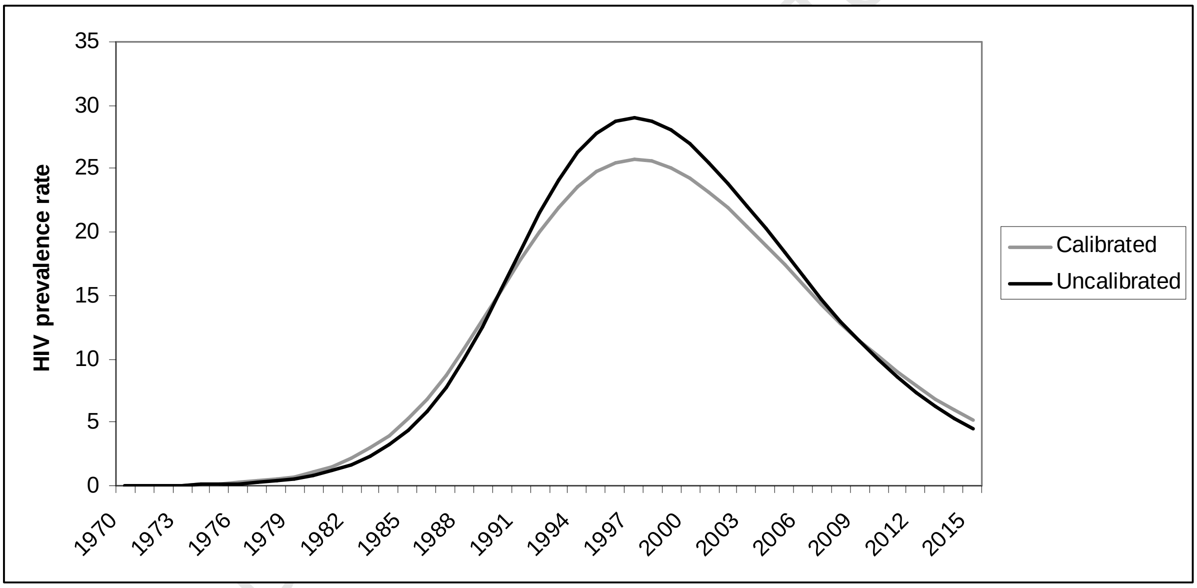
In the more rural but still non border province of Mashonaland East, prevalence also peaked in 1997 albeit at a much higher rate (30.9%). The story is no different for Mashonaland Central where prevalence peaked in 1997 (30.0%). Matabeleland North is a particularly interesting case because the peak (19.7% in 2000) in prevalence happened at a much lower level than in other provinces. The province is fairly economically isolated with but one commercial hub (Hwange, a mining town). Such areas also have poor communication infrastructure which probably hampers the developing of sexual networks (Iliffe, 2006:10). This point is explored further in Chapters 5 to 8.

EPP prevalence estimates for the Midlands provide a break from the general thesis that non border provinces peaked at much higher levels than the border ones. HIV prevalence in the province is estimated to have peaked around 1996 (35.9%). This means that the epidemiology of AIDS in the Midlands was closer to that of the Manicaland border province than that of Harare or Bulawayo. This is probably because the Midlands province is at the centre of much activity in Zimbabwe as it hosts two of Zimbabwe's six largest cities. A more critical analysis of these observations is presented in the next chapter. Figure 2.10 shows estimates for the trend in HIV prevalence in Zimbabwe modelled as the outcome of different processes of infection defined by province.

[7] To get Spectrum error margins and confidence intervals for the provinces, one would have to run each provincial sub-epidemic in Spectrum an exercise which is not particularly useful for the argument presented in this thesis. It would also require assumptions about provincial distribution of ART, PMTCT, etc, which were not readily available. Most reports provide aggregated data. The same point can be made by using the Spectrum national estimates as is done in the next sections.

As already mentioned, the ANC data was calibrated (actually forcing the curves to pass through specific data points). The MOHCW working group also calibrated the ANC data using the ZDHS. The MOHCW estimated are shown in Figure 2.12 and a comparison between these estimates and those from the modelling done in this thesis is also presented later. However, Figure 2.10 (based on the modelling done here) shows the difference between the calibrated EPP estimates of national HIV prevalence in Zimbabwe and the Estimates that would have been obtained without calibration. The model estimates for prevalence without calibration are much lower than the peak points for the calibrated data. This is because the ZDHS had the effect of making the rise slightly more gradual and the decline faster than would have been the case without calibration.

Figure 2.10 EPP HIV prevalence



Source: EPP 2009

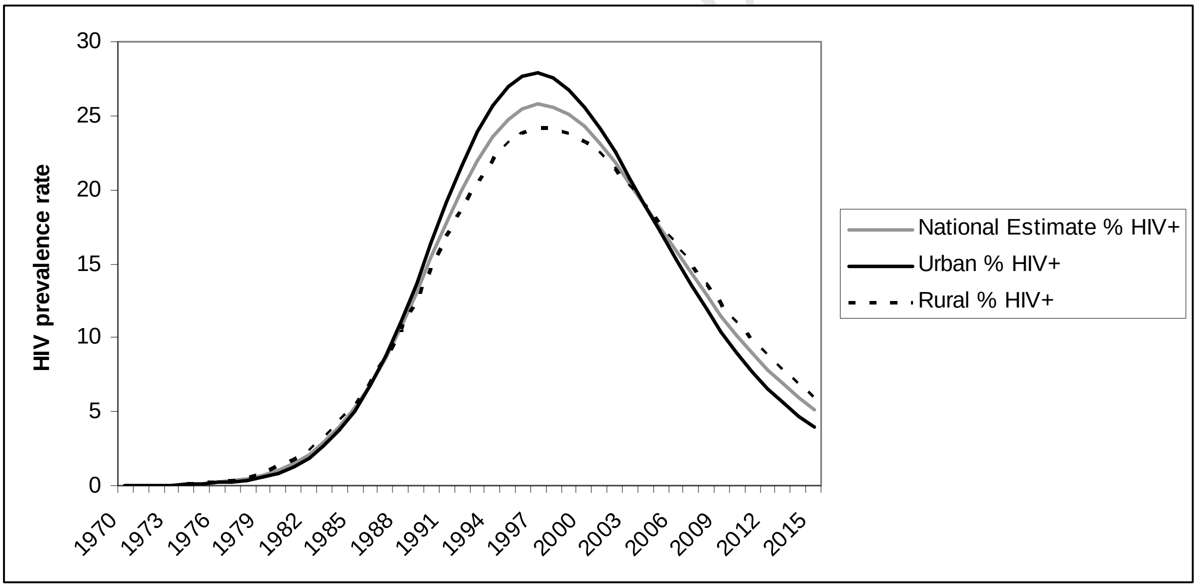
It is important to reiterate that the ANC site data is not free from bias given that it samples (only) pregnant women. Mentioned earlier was the fact that when assessing the accuracy of EPP prevalence estimates it is important to consider that HIV prevalence among pregnant women at ANC clinics may not be the same as among all men and women in the sexually active aged general population[8]. This makes calibration the only responsible option available

[8] Another point is that whereas ANC data is on all women attending the sites, the DHS used to calibrate the data is based on tests done on women 15-49 years old.

for a researcher. The justification for using calibrated data has already been addressed in the methodology and will not be revisited here.

Figure 2.11 shows trend lines for HIV prevalence estimates produced in EPP using a different modelling strategy. This time is assumed the epidemic is the outcome of different processes of infection defined by sub-population (rural/urban). These estimates suggest that adult HIV prevalence declined in both rural and urban areas since the late 1990s. Adult HIV prevalence has generally been higher in urban than in rural areas, peaking in urban areas at around 28% in 1996 and 24% in rural areas. This trend was however reversed around 2005 when prevalence in rural areas became higher. Currently at 8.9% in urban areas and 11.0% in rural areas, HIV prevalence looks to proceed on a downward trend with urban prevalence decreasing at a faster rate as shown in Figure 2.11.

Figure 2.11 Rural versus Urban prevalence



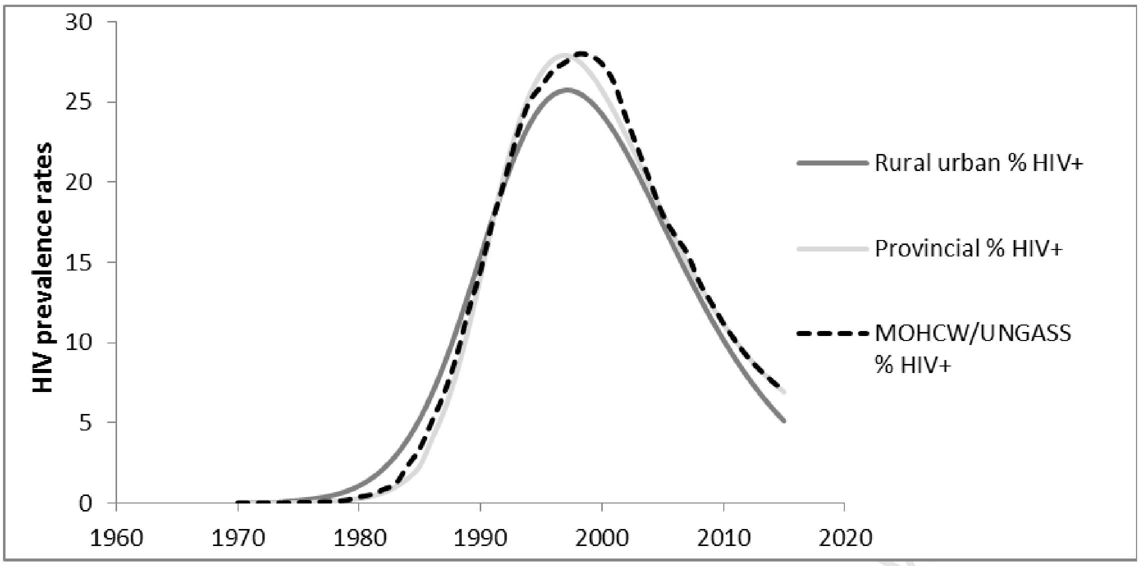
Source: EPP 2009 projections.

It is likely that the educated or those with means, living in urban areas are more likely to emigrate to South Africa, Botswana, United Kingdom or some other country in search for jobs and opportunities (especially given the circumstances in Zimbabwe at the time – see chapter 3). Some of the migration out of urban areas is within the country's borders as some who fall sick in urban areas are taken back to their rural villages to be nursed there.

Recalling the modelling assumptions used in estimating the above prevalence figures is useful at this point. This is because (as was done by the MOHCW), prevalence at Birchenough Bridge, Gutu, Hauna Growth Point, Murambinda, Mutoko and Sadza ANC sites was scaled down by 30% (MOHCW, 2009:16-38). The reasoning here was that data from local epidemiological studies indicate that HIV prevalence rates are approximately 30% higher in these areas compared with the truly rural areas. This scaling down of ANC prevalence sites would have depressed rural HIV prevalence estimates to some extent but it would be a stretch to conclude that these six sites are solely responsible for the rural-urban disparity.

Since EPP allows one to model the national epidemic either by building up from an urban rural template, or from a provincial template, results from the two approaches can be compared. The best fit curves produced by EPP for the urban, rural as well as the provincial analysis are shown in Figure 2.12. All the modelling and data points presented thus far suggest that Zimbabwe is experiencing a generalized epidemic that has been declining since the late 1990s. The national prevalence estimates obtained from the rural and urban modelling and those obtained from the provincial analysis are very similar. The provincial projections will be used as inputs to Spectrum because they give national estimates that resemble the UNGASS/MOHCW estimates produced by Zimbabwe's National Working Group on HIV and AIDS Estimates. Although the same can be said of the estimates from the from the rural urban projection, these estimates are not as close a match as the ones from the province by province projection. For instance, UNGASS reported a prevalence (age 15-49) rate of 15.6% for 2007 whereas the rural urban projection suggests a lower rate of 14.3%.

Figure 2.12 Rural versus Urban prevalence analysis



Source: EPP 2009 projections.

The national estimates produced from the province by province analysis give a prevalence rate of 15.1 % (2007) which is approximately equal to the UNGASS/MOHCW estimate. However, the modelling exercise is not yet complete. The EPP result needs to be run through a further estimation process using Spectrum in order to get estimates that are more comparable to the UNGASS/MOHCW estimates produced by the National Working Group on HIV and AIDS Estimates.

2.2.5 Limitations of the Epidemiological Projection Package (EPP)

The fundamental limitations inherent in EPP centre on issues of the quality and non-representative nature of the data used in projections. It has already been highlighted that the core of EPP input data, ANC site data, is not quite representative of the whole population. In addition, there is only limited data available for use in the calibration process. This is particularly the case in Zimbabwe, where to date, only the 2005-06 ZDHS has been used to calibrate ANC data. Ghys et al (2004:5) argue that the sophistication of model is both a strength and a weakness. As the model is based on an epidemiologically derived set of equations that model the transmission of HIV, it does a good job of fitting the full range of HIV epidemic types observed in the world. It is very advantageous that the model's parameters have a relatively simple interpretation such as the rate of growth of the epidemic or the proportion at risk of infection. But this simplicity can lead users to over interpret the implications of specific parameter fits (Ghys et al 2004:5).

An example of this is that in some instances, there may be many sets of r, f_0, t_0, and φ values that provide comparable fits and curves for any one data set. This is so particularly with widely varying values of r and f_0. But users may try to interpret these values as having a precise real world meaning. For example, the parameter for the initial fraction of the population at risk f_0, may be interpreted as a measure of risk in a given population (Ghys et al 2004:5). However, uncertainty about this estimate may make this interpretation meaningless. Thus, users must be cautioned against taking the model too literally (Ghys et al 2004:5).

Another concern that has been raised about earlier versions of the EPP program is that it assumes r, f_0, t_0, and φ are constant over time (Brown et al 2008). . Brown et al (2008) explain that of the four parameters mentioned, the two likely to change over time are the force of infection, r, and the behavioural change parameter, φ, which determines changes in the number of people entering the at-risk population over time. Changes in r could account for behaviour change as seen in increases in condom use, decreases in prevalence of other sexually transmitted infections acting as cofactors for HIV transmission, reductions in frequency of risky sex or other such changes (Brown et al 2008:6).

Changes in the size of the at-risk population over time could be covered by variations in φ. Brown et al (2008) found that in general, allowing for more flexibility in the model made for better fits in some cases, but not in others. This clearly still remains an area for further development. The new feature introduced in the 2009 version of the model is that it allows φ to shift to take into account a shift in behaviour change in generalized epidemics such as the one experienced in Zimbabwe. The parameter changes also to account for the use of antiretroviral in prevention programs (PMTCT) and in treatment programs (ART). The next section describes how the incidence and prevalence curves generated in EPP are used as inputs in order to generate estimates of prevalence and the impact of prevention and treatment programs using the Spectrum package.

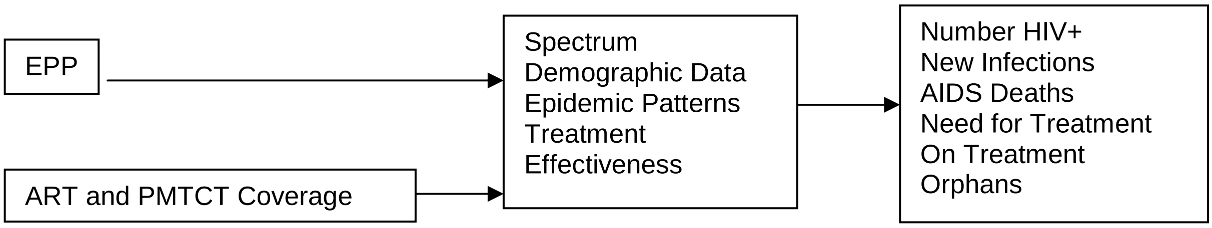
2.3 Spectrum

Spectrum is a modular software package that is used to examine the consequences of current trends and future program interventions in reproductive health. In addition to determining the consequences of HIV/AIDS, such as the number of AIDS deaths, the number of orphans as a result of AIDS, number of people living with HIV/AIDS by age and sex, Spectrum is also used to determine other demographic indicators of interest, such as life expectancy and mortality (Stover, 2004).

The Spectrum Policy Modelling System is an integrated package that contains a number of modules including Demography (DemProj), a program to make population projections based on the current population, and fertility, mortality, and migration rates for a country or region. Because it projects the population by age and sex, the core of the Spectrum package is DemProj, a demographic projection model. HIV/AIDS projections are then added to the demographic projections (see Figure 2.13). These are created by the AIDS Impact Module using estimates of HIV prevalence created by EPP as described earlier. Other assumptions required include the distribution of the time from infection until AIDS death, and the effect of HIV on fertility, the ratio of female: male prevalence and the distribution of infection by age.

The other module is the AIDS (AIDS Impact Model – AIM). It is used to project the consequences of the AIDS epidemic including: the number of people infected with HIV, AIDS deaths, the number of people needing treatment, and the number of orphans. The third module is the Family Planning (FamPlan), a program to project family planning requirements in order to achieve national goals for meeting couple‘s fertility intentions. A module called the Socioeconomic Impacts of High Fertility and Population Growth (RAPID) is used to project the social and economic consequences of high fertility and rapid population growth for sectors such as labour force, education, health, urbanization and agriculture. The costs and benefits of different programs intended to reduce the transmission of HIV from mothers to their new born children are estimated using the Prevention of Mother-to-Child Transmission of HIV (PMTCT). The last but equally important module is the Child survival (LiST), a program to estimate the effects of scaling up child survival intervention on the rate and number of deaths to children under the age of five.

Figure 2.13 How Spectrum works



Source: UNAIDS, 2009 Spectrum.

2.3.2 DemProj

DemProj is the demographic projection module in Spectrum. Population data has to be input before DemProj carries out the projections. This includes population by age and sex in the base year, life expectancy at birth in the absence of AIDS, the age pattern of mortality, and the number and distribution by age and sex of international migrants. Fertility data is also required including the total fertility rate over time and the age distribution of fertility. DemProj contains EasyProj which is designed to be used by non-demographers and none specialists. It creates a demographic projection using population data[9] from the United Nations Population Division. The user can set the start and end dates for the projections as well as select from a list, the distribution of fertility and mortality by age (Stover, 2004).

The module also allows the user to select an appropriate demographic life table. The modelling for Zimbabwe presented here was done based on the Coale-Demeny North Model life table. This is the Spectrum default setting. A life table is a table of information that attempts to give the the most complete description of mortality in any population. The data input needed for its construction are the age-specific death rates calculated from information on deaths by age and sex (from vital registration) and population by age and sex (from census) Murray et al, (2000:2). Although a useful tool, these life tables are not simple to construct owing to the fact that in a lot of developing countries, the required data either do not exist due to lack of functioning vital registration systems, or are unusable because of incompleteness of coverage or errors in reporting. Demographers have devised ways of

[9] See the United Nations Population Division. World population prospects: the 2002 revision. United Nations: New York, 2003.

deriving reasonably reliable life tables from incomplete data by using a variety of adjustment techniques Murray et al, (2000:2).

For cases where the data is unusable or cases where no vital registration data exist, indirect techniques for obtaining mortality rates are used (Murray et al (2000:2); Preston et al (2001:47-49)). Such methods are predicated on the observed similarities in the age-patterns of mortality for different populations, and range from the simple adoption of the mortality pattern of a neighbouring population with similar socio-biological characteristics, to the use of more complicated demographic models. The first set of model life tables, the UN model life tables, was published by the UN in 1955. The UN model life tables were constructed using 158 life tables for each sex, using statistical techniques to relate mortality at one age to mortality at another age for a range of mortality levels (Murray et al, 2000:3).

As demographic techniques improved, The Coale and Demeny regional model life tables were developed. These life tables were published initially in 1966. The Coale and Demeny life tables were derived from a set of 192 life tables, by sex, from actual populations. As described in detail by Murray et al (2000:3), the sets included life tables from several time periods (39 from before 1900 and 69 from after the Second World War) and mostly from Western countries. Europe, North America, Australia and New Zealand contributed a total of 176 tables. Three were from Israel; 6 from Japan, 3 from Taiwan; and 4 from the white population of South Africa. All of the 192 selected life tables were derived from registration data, and were subjected to very stringent standards of accuracy Murray et al (2000:3).

Analysis of the underlying relationships identified four typical age patterns of mortality, determined primarily by the geographical location of the population and also on the basis of their patterns of deviations from previously estimated regression equations. Those patterns were called: North, South, East, and West and each one had a characteristic pattern of child mortality (Murray et al (2000:3); Preston et al (2001:139-170)). Instead of the using the North table, we could have opted for the West model life table. Compilation of The West model is based on the residual tables not used in the other regional sets (i.e. countries of Western Europe and most of the non-European populations) and so is characterized by a pattern intermediate between North and the East patterns. Consensus among demographers is that because this model is derived from the largest number and broadest variety of cases, the Coale-Demeny West Model life table represents the most general mortality pattern (Indepth, 2002:10-250). It is therefore recommended for use when modelling African HIV epidemics (Stover, 1999). However, we follow the National HIV and AIDS Estimates Working group who used the Coale – Demeny North life table (MOHCW, 2009: 14).

Another assumption in the Spectrum modelling process is that on the level of net migration. In Zimbabwe, out-migration of highly skilled and educated professionals has occurred at a staggering rate since the late 1990s (Chetsanga, 2004; Tevera, 2003). However even unskilled Zimbabwean nationals left their mother country in search for better opportunities elsewhere. The Central Statistics Office accepts a low figure of 350,000 as an estimate for the number of out-migrants since 2002. This is despite the fact that another government arm the Reserve Bank of Zimbabwe (RBZ), has been using 3 million as a planning figure for the purpose of remittance calculations (IRIN, 2010). These numbers are just a tip of the iceberg as there are many more illegal immigrants in Botswana and the United Kingdom (Netsianda, 2007). Although the usual destinations include Botswana, USA, UK, Australia, New Zealand, South Africa was and continues to be the most popular. A great number of Zimbabweans, an estimated 1.7 million, are illegally settled in the South Africa alone (Netsianda, 2007). Others have estimated that between a total 500,000 and 3 million Zimbabweans are settled legally or illegally in South Africa (Crush, 2005:13).

Some have rejected these numbers, declaring them ill-informed exaggerations (Makina, 2007:5). A pilot study that interviewed migrants in South Africa at destination areas like Mussina has put the estimate of the number of Zimbabweans in the country at between 800,000 to just over one million (Makina, 2007:2). The World Bank's Health, Nutrition and Population data (HNP Stats) has estimated that Zimbabwe's net migration rate (per 1,000 people) has averaged -0.4 over the past ten years and will climb up to -0.5 between 2015 and 2020 (HNP Stats, 2010). These estimates are based on the HNP's population projections on Zimbabwe. This appears to be a very conservative estimate compared to the other figures being cited earlier (Makina, 2007:2; Netsianda, 2007; Crush, 2005:13; Chetsanga, 2004).

Chapter 3 gives context to the rise in out-migrations witnessed over the past 10 years. However, the modelling process is concerned more with the decision of which estimates to use than it is about exploring the reasons for the out-migration. The Spectrum (version 4) package states that the default migration figures it uses are based on, "intercensal net residuals and official statistics, population distribution by age and sex or simplified versions of Rogers-Castro migration age patterns, and incorporate statistical adjustment errors". There is no indication of the exact official statistics used for each country's default settings. This would not present much of a concern were it not for the fact that the level of out-migration that Zimbabwe has experienced over the past 11 years is a subject of hot debate.

Table 2.3 Default Spectrum migration statistics for Zimbabwe

Year	2004	2005	2006	2007	2008	2009	2010
Spectrum Default (all ages)							
Males	-87,472	-92,075	-96,679	-101,283	-90,101	-63,135	-36,168
Females	-64,351	-67,737	-71,124	-74,511	-65,716	-44,738	-23,761
Total	-151,822	-159,813	-167,803	-175,794	-155,817	-107,873	-59,928
Cumulative Total	-625,268	-785,081	-952,884	-1,128,678	-1,284,495	-1,392,367	-1,452,296

Source: 2009 Spectrum

The cumulative total row shows the cumulative total number of out-migrations from Zimbabwe since 2001. In other words, the default Spectrum assumptions are that out-migration from Zimbabwe has been to the order of 1.4 million since 2001. This according` to the figures already discussed is still quite conservative. Later on in this chapter an assessment will be made as to how much the modelling results change when the default assumptions are changed to an arbitrary and higher estimate of 2 million out-migrations (since 2001).

Before carrying out this analysis, it is important to refer to a recent study on the decline in HIV prevalence observed in Zimbabwe. Using a wide range of data sources Gregson et al (2010) assessed the contributions of rising mortality, falling HIV incidence and sexual behaviour change to the decline in HIV prevalence. Their study made a number of conclusions of which the most important for the modelling presented in this chapter is that international migration out of Zimbabwe is unlikely to have contributed greatly to the decline in HIV prevalence Gregson et al (2010:1318-1320). Data sources used include the 1988, 1994, 1999 and 2005 ZDHS surveys a series of bi-annual knowledge, attitudes, practices and beliefs surveys commissioned by Population Services International (PSI) between 1997 and 2007, the 2001-02 Zimbabwe Young Adult Survey, the 1997 National Youth and others (Gregson et al, 2010: 1315).

Because there is no data that shows the extent to which HIV positive persons migrated from the country, Gregson et al estimate the potential impact of this out-migration on HIV prevalence by fitting a published mathematical model (see Hallett et al (2006)) to the data listed above (ibid, 2010:1315). They projected trends of HIV prevalence under a number of different scenarios namely. Their modelling suggests that even with very large numbers of migrants, the effect of out-migrations was likely to have been small unless extreme assumptions are made (Gregson et al, 2010: 1318-1320). Even when assuming for instance that as the epidemic peaks, 10% of the national population migrates every year, with persons with AIDS 20 times more likely to migrate than uninfected individuals, and with 10% of asymptomatic HIV-positive individuals aware of their status and three times as likely to migrate as uninfected individuals, their modelling suggests that by 2010, prevalence would have been about 5% lower than under their default migration assumptions (Gregson et al, 2010:1319). Reference will be made to this result in Section 2.5 where focus is on using the result from the modelling in this chapter to assess the relative impact of out-migration on the estimated trend in HIV prevalence.

Gregson et al (2010) also tried to assess whether individuals migrating from Zimbabwe had higher prevalence rates than comparable groups in Zimbabwe. For this they used United Kingdom Health Protection Agency data on trends in Zimbabwe-born women tested at delivery at clinics in London (except south-west London) and the south-east and north-west regions of the UK between 2000 and 2006 (ibid, 2010:1315). They found that while on average the numbers of Zimbabwe-born women tested at ANCs in the UK increased between 2000 and 2006, HIV prevalence in these women remained stable at 9–11% (ibid, 2010:1319). This they argue suggests that HIV prevalence in Zimbabweans migrating to western countries could be lower than amongst non-migrants.

However they also acknowledge that migrants to southern and central African countries may have a different socio-economic profile due to the shorter distances and greater possibilities for unauthorised border crossings (ibid, 2010:1319-1320). This is an important point to make as the majority of out-migrations have been to countries in the region such as South Africa and Botswana. This means that the result presented by Gregson et al (2010) does not tell us anything about whether or not the individuals migrating out of Zimbabwe were likely to be from the population most at risk of infection (and so have higher HIV prevalence rates).

2.3.3 The AIM HIV/AIDS projection module

The AIM module is the module that creates projections of the consequences of the AIDS epidemic including: the number of people infected with HIV, AIDS deaths, the number of people needing treatment, and the number of orphans (Stover, 2004). The AIM module adds the HIV/AIDS projections to the demographic projections. The module uses the EPP output, projected adult HIV prevalence together with assumptions on the ratio of female to male prevalence, distribution of infection by age, distribution of the time from infection until AIDS death and the effect of HIV on fertility (Stover, 2004). The AIM manual by John Stover (Stover, 2003) explains in detail how the module carries out HIV/AIDS calculations and presents HIV/AIDS projections by age and sex.

The core of the AIM module can be summarised and simplified for non-demographers (Stove, 2004). In Stover’s description, the number of adults of age (a) and sex (s) infected with HIV in any year is given by the number of adults multiplied by the HIV prevalence (age 15-49):

$$\text{HIV}_{a,s,t} = \text{adult population}_{a,s,t} * \text{prevalence}_{a,s,t} \qquad \text{equation (5)}$$

The number of new infections each year is calculated as the number required to achieve the specified prevalence. In other words, new infections are calculated as the total number of infections expected in year t minus the number of infections surviving from year t-1. The number of surviving infections is given by the number of infections in the year t-1 minus deaths from AIDS or other causes occurring at time t-1.

$$\text{New HIV Infection}_{a,s,t} = \text{HIV}_{a,s,t} - (\text{HIV}_{a-1,s,t-1} - \text{AIDS deaths}_{a-1,s,t-1} - \text{non-AIDS deaths to HIV+}_{a-1,s,t-1})$$

The number of new infections in previous years and the rate of progression from infection to death determine the AIDS deaths:

$$\text{AIDS deaths}_{a,t-1} = \sum_{i=0}^{20} (\text{New HIV Infections}_{a\ i,t\ i} * \text{Proportion that die from i years after infection})$$

A key assumption made is that HIV infected people are subject to the same hazard of mortality from causes other than AIDS as are people who are not infected. During gestation, or after birth through breastfeeding, children may become infected with HIV.

$$\text{New Child HIV Infections}_t = \text{HIVWRA}_t * \text{TFR}_t * (1 - \text{TFRreduction}) * \text{PTR}_t$$

The terms in the equation above can be described as follows - HIVWRA is the number of HIV positive women of reproductive age; TFR reduction is the reduction in fertility caused by HIV infection; TFR is total fertility rate; and PTR is the perinatal transmission rate. The model assumes that the progression (process) from infection to AIDS and death is the same for both children and adults. However, the progression rates are not the same. The progression period describes the amount of time that elapses from the time a person becomes infected with HIV until he or she dies from AIDS (Stover, 2004).

The AIM module uses results from the UNAIDS Reference Group and studies such as the Collaborative Group on AIDS Incubation and Survival including the CASCADE EU Concerted Action to obtain a cumulative distribution of the progression from HIV infection to AIDS death (UNAIDS, 2002:16). The Collaborative Group on AIDS Incubation and Survival including the CASCADE EU Concerted Action carried out reviews of the time from infection to death from AIDS in the absence of antiretroviral therapy (ART) for developing and developed countries (Collaborative Group on AIDS Incubation and Survival, 2000). The results provide the basis for the distribution that is used by AIM which is described as the cumulative fraction of people infected with HIV who will die from AIDS. This is done by number of years since infection.

AIM has two default progression patterns that are based on the assumption that better health care leads to a longer, survival period in industrialised countries. The two default patterns are fast, which is for developing countries and slow, which is for industrialized ones (Stover, 2004). AIM assumes that the median time from infection to death is 9[10] years in developing countries and 11 for industrialised countries. As described by (Stover, 2004:3), survival times are assumed to follow a Weibull distribution in agreement with the data available. In addition, the progression to AIDS death can be slowed down by interventions such as ART. The Spectrum package delays progression to death provided the infected successfully manage to remain on treatment. In other words, the survival of patients on ART depends on the quality of treatment and the patient's status when starting ARTs. Spectrum assumes that the average survival on ART is in the range 3-7 years where the default assumption is that 80% of those on ART continue with it and survive into the following year (Stover, 2004:3).

The assumption used in this exercise is that the median time from infection to AIDS death without treatment is 11 years (MOHCW, 2009:53). Further, eligibility for ART is assumed to occur at a median of three years before AIDS death[11]. Receiving first- and/or second-line ART generally improves chances of survival and the ART program in Zimbabwe is fairly new having started in 2004. The UNAIDS Reference Group on Estimates, Models and Projections assumes that for most new programs the annual survival rates are 85% for the first year on ART and 95% for subsequent years, but the first year survival is likely to be lower in new programs with low ART coverage since most patients get started on ART very late, with low CD4[12] counts (Stover et al, 2008:21-24). These are key assumptions used in the modelling presented in this chapter.

Already mentioned is the fact that Spectrum assumes that although the progression from infection to AIDS and death is the same for children and adults, the progression rates are not the same. For instance, perinatally infected children generally progress to AIDS at a pace faster than that for adults. AIM assumes accordingly that some children will experience lower survival rates than others (Stover, 2004:3). This is based on UNAIDS reviews which suggested that the survival is best described by a rapid progression from infection to death for some children and much slower progression for others (UNAIDS, 2002; Stover, 2004:3).

[10] 8.6 years for males and 9.4 years for females.
[11] See Stover et al (2008)
[12] CD4 < 200

The epidemiology of HIV/AIDS is also influenced by gender. As Zimbabwe is modelled as a generalised epidemic, assumptions made by Spectrum on concentrated epidemics will be ignored in this discussion. Stover (2004) states that in generalised epidemics where most HIV transmission is through heterosexual contact there are usually many more male infections than female infections early in the epidemic but, over time, the number of female infections eventually exceeds the male infections.

Spectrum uses a pattern based on data from population based surveys throughout Africa (Mwaluko et al, 2003; Kwesigabo et al, 2000; Ministry of Health, Republic of Rwanda, 1997; Kilian et al, 1999; Kamali et al 2000; Buve´, 2003). These present ratios of female to male prevalence at different stages in the epidemic as shown by Stover (2004:4). As explained by Stover (2004:3), Spectrum by default, starts with the ratio of female to male prevalence at 0.23 in the first year of the epidemic and rises to 1.3 by the 15th year of the epidemic. In other words, Spectrum is set to assume by default, that the overall female to male ratio in mature epidemics is 1.3. Gregson et al (2002; 16:643–52) show that the female to male ratio in mixed urban and rural areas in Zimbabwe was 1.37 and this provides grounds for the changing of the default settings to 1.37 for Zimbabwe.

To model the epidemic in Zimbabwe, the default settings on the age distribution of HIV infection were maintained. Where data is available, Spectrum allows the user to replace the default settings. Regarding mother to child transmission of the virus, AIM assumes by default that the percentage of babies born to HIV infected mothers who are themselves infected (the perinatal transmission rate) is 32%. This is the assumption used in this chapter to model the epidemic in Zimbabwe. De Cock et al (2000; 283:1175-1182) and Zaba et al (1998) found that this percentage is around 13-32% in industrialised countries and 25-45% in developing countries.

Spectrum also takes into account the impact of HIV on fertility. Typically, HIV reduces fertility. Zaba et al (1998; 12: S41–50), Carpenter et al (1997; 2:113–26), Glynn et al (2000; 25:345–52) and Gray et al (1998; 351:98–103) show that HIV infection has a negative impact on fertility. The default pattern assumed by AIM is that HIV reduces fertility by 30% for women age >20 (Stover, 2004:4). In addition, because HIV infection signals that a woman is already sexually active; for such women (infected) aged 15–19 AIM assumes that fertility is 50% lower than that of uninfected women in the same age group.

The Spectrum package also calculates the number of orphans, including orphans as a result of AIDS in countries in sub-Saharan Africa and orphans due to all causes, and maternal, paternal, and dual orphans by age and sex. AIDS and non-AIDS orphans are estimated from the number of adult deaths each year, the fertility history of those who die and the rates of child survival. The methodology has been confirmed by comparing the results with the findings of national surveys (Grassly et al, 2003; Stover 2004:5).

The biggest advantage of the Spectrum package is undoubtedly its simple platform. It works in multiple languages, and has a full set of manuals and training materials. Spectrum produces key indicators of great importance to program planners and policy makers, including the number of new infections, the number of people living with HIV/AIDS, AIDS deaths, and orphans. The ease with which one can easily change the modelling assumptions makes Spectrum a powerful tool especially where the default assumptions set by the UNAIDS Reference Group are deemed outdated. These default assumptions e.g. the female to male ratio can be selected for countries with generalised or concentrated epidemics or country specific patterns can be used if data are available (Stover 2004:5). Spectrum is therefore a very useful tool for comparing the different outcomes of different assumptions as is done later in the chapter. Annex has a summary of the EPP and Spectrum assumptions used in the modelling process.

2.3.4 Limitations

One of the key disadvantages of Spectrum is that some of the model patterns used in the program are based on a small number of studies (Stover, 2004). An example is that the progression pattern for children is based on a few studies, none of which follow cohorts for very long and certainly not for 15 years (Stover, 2004:2-7). In addition, the database used to create the model patterns is not complete.

Another weakness is that Spectrum estimates incidence from the prevalence trends provided by EPP. The pattern of prevalence by age and sex is usually assumed to be constant over time and so the age specific patterns of incidence may not be reliable (Stover, 2004:2-7).

In general, the fact that data is limited makes the reliability of Spectrum estimates a debatable matter. A lot of the default distributions are based on limited data. An example of this is the default age distribution of HIV infection among injecting drug users. These distributions vary from country to country more specific to Zimbabwe is the problem of figuring out the extent of the emigration that has happened in the past ten years. It is expected that as the quality of the data improves, the efficacy of the Spectrum model will substantially improve.

2.3.5 Spectrum Results: HIV Prevalence (age 15-49), Incidence, AIDS deaths and Mortality

As alluded to in previous sections including Table 2.4, Spectrum requires EPP estimates of prevalence, Data on ART and PMTCT as input. These are run through the package's DemProj, AIDS Impact Model, FamPlan, Child survival (LiST) and RAPID modules. The resultant output are projections and estimates of HIV prevalence, AIDS deaths, need for treatment, number on treatment, new infections and various information on the epidemic's impact on orphans.

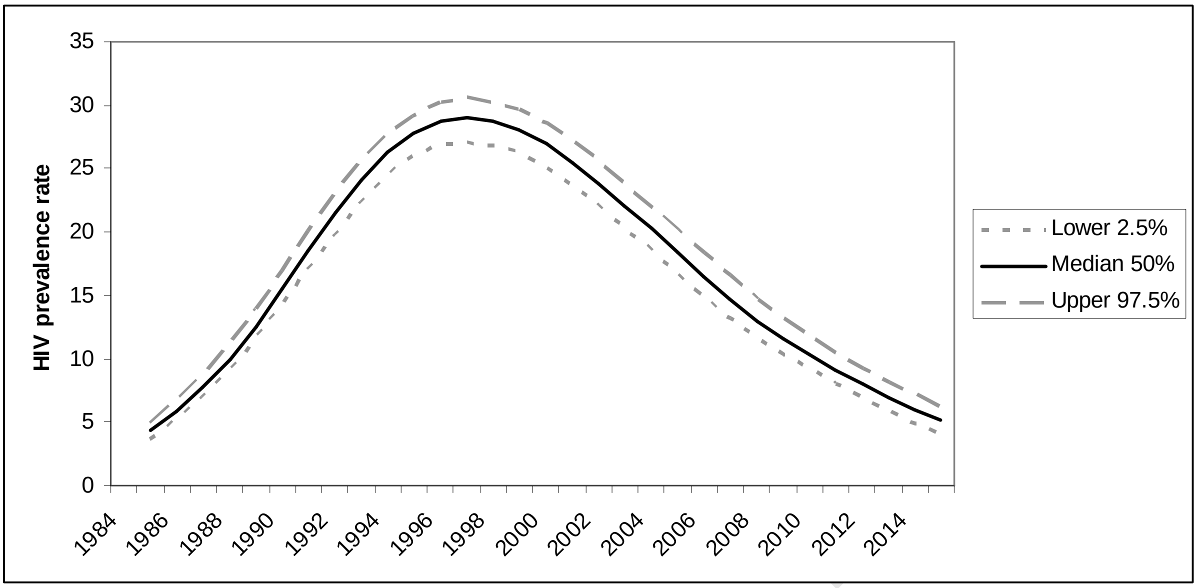
Table 2.4 Spectrum model results for Zimbabwe

	2007 Est	2007 Bounds	2009 Est	2009 Bounds
Total (adults and children)	1,332,440	1,250,110 - 1,382,400	1,156,361	1,024,220 - 1,288,02
Adults (15-49)	1,096,474	965,333 - 1,227,615	947,036	815,895 - 1,078,177
Women (15-49)	777,934	680,793 - 875,075	675,317	578,176 - 772,458
Children (15-49)	124,034	117,335 - 133,214	109,555	93,676 - 125,434
Adult Prevalence (15-49)	15.12	14.7 - 16.2	12.52	11.8 - 13.27
New HIV infections-Adult	39,321	37,755 - 41,288	38,744	37,178 - 40,310
New HIV Infections-Children	13,754	13,410 - 14,098	11,567	11,223 - 11,911
Annual AIDS deaths-Adult	120,061	117,059 - 123,063	117,028	114,026 - 120,029
Need for ART-Adult (15+)	349,139	340,410 - 357,867	354,813	346,085 - 363,541
AIDS orphans	990,450	965,689 - 1,015,211	983,528	958,767 - 1, 008,289

Source: Spectrum modelling

Table 2.4 shows that the number of new infections decreased from about 39,321 in 2007 to around 38,744 in 2009. AIDS deaths also decreased over the same years from 120,061 to 117,028 while the number of people in need of ART increased from 349,139 to 354,813. One of the more positive results from the modelling exercise is that the number of AIDS orphans decreased from 990,450 to 983,528. More results from the modelling are presented in Appendix A. The rest of the chapter focuses specifically on trends in HIV prevalence and incidence and the possible contribution of emigration to these trends.

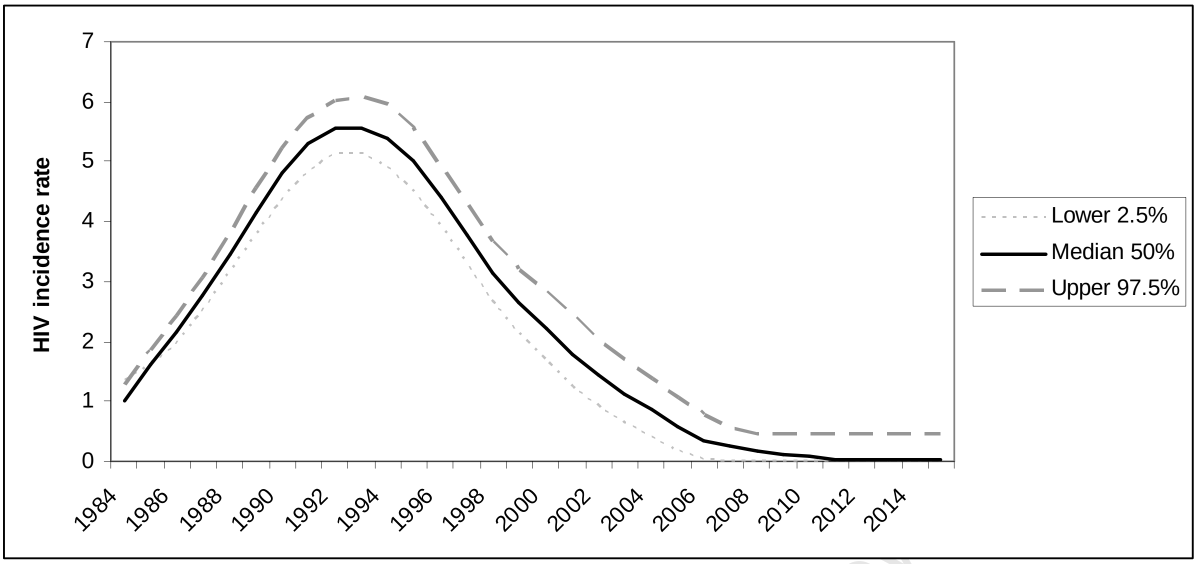
Figure 2.14 Spectrum HIV prevalence Estimates for Zimbabwe



Source: UNAIDS, 2009 Spectrum

Spectrum HIV prevalence estimates are shown in Figure 2.16. Reports by the Ministry of Health and Child Welfare (MOHCW) published in 2007, 2008 and 2009 based on the National HIV and AIDS Estimates Working group describe a trend in HIV prevalence in adults (age 15-49) that is similar to the one shown in Figure 2.16 (MOHCW, 2009:12-15). The MOHCW reported adult (age 15-49) prevalence for 2009 at 13.7%, 2008 at 14.1% and 2007 at 14.7% (MOHCW, 2009:7-8). Prevalence as shown in Figure 2.16 was estimated at 12.5% for 2009, 13.8% for 2008 and 15.1% for 2007. This suggests that the Spectrum model presented here successfully "reverse engineered" the MOHCW estimates.

Figure 2.15 Spectrum HIV incidence Estimates for Zimbabwe



Source: UNAIDS, 2009 Spectrum

Figure 2.16 shows that HIV prevalence increased quite rapidly from the mid-1980s all the way up to the late 1990s. Figure 2.17 shows the trends in HIV incidence which peaked around 1994.

In statistics and demography, a cohort is a group of subjects who have shared a particular experience during a particular time span (Dodge, 2003). Using the balancing equation of population growth, changes in the HIV positive population/cohort aged 15- 49 (HIV prevalence) will be disaggregated into change due to deaths, change due to new infections, change due to migration and finally change due to individuals aging into and out of the cohort.

2.4 Using the balancing equation of population growth to decompose the change in HIV prevalence in Zimbabwe

Preston et al (2001:2-4) restate the balancing equation of population change as;

$$N(T) = N(0) + B[0,T] - D[0,T] + I[0,T] - O[0,T] \quad \text{equation (6)}$$

Where

N (T) = number of persons alive in the population/cohort at time T,
N (0) = number of persons alive in the population/cohort at time 0,
B [0, T] = number of births in the population/cohort between time 0 and time T,
D [0, T] = number of deaths in the population/cohort between time 0 and time T,
I [0, T] = number of in-migrations between time 0 and time T,
O [0, T] = number of individuals aging into and out of the cohort between time 0 and time T,

The rationale is that there are at most two ways of leaving a population, death and out-migration and two ways of entering (birth and in-migration) (Preston et al, 2001:2-4). If we define our population or cohort as the number of people aged between 15 and 49 who are infected with HIV, then the balancing equation of population change can be used to investigate how this population changes with time. The balancing equation can then be restated as an "adapted balancing equation of population change";

$$N^{HIV+}(T) = N^{HIV+}(0) + E^{HIV+}[0,T] - D^{HIV+}[0,T] - D^{P}[0,T] + M^{HIV+}[0,T] + P^{HIV+}[0,T]$$

equation (7)

Where

$N^{HIV+}(T)$ = number of persons alive in the population[13] at time T,
$N^{HIV+}(0)$ = number of persons alive in the population/cohort at time 0,
$E^{HIV+}[0,T]$ = number of new infections (entrants) into the population between time 0 and time T,
$D^{HIV+}[0,T]$ = number of deaths in the population/cohort due to HIV between time 0 and time T,
$D^{P}[0,T]$ = number of deaths in the population due to non HIV causes between time 0 and time T,
$M^{HIV+}[0,T]$ = number of net migrations (in-migrations - out-migrations) between time 0 and time T,

[13] Unless otherwise stated, population here refers to those aged 15-49 who are HIV positive.

$P^{HIV+}[0,T]$ = number of individuals aging into and out of the cohort between time 0 and time T,

Because of the fact that as people age, they will leave the population (aged 15-49), the factor P^{HIV+} [0,T] tries to capture the proportion of HIV positive people (aged 15-49) who turn 50 or older. It also captures the proportion of the general population (aged < 15) who are infected and enter the population aged 15-49. The main difference between equation 6 and equation 7 is that in equation 7, individuals do not enter the population through birth but by being newly infected. Deaths in equation 7 have also been broken up into deaths due to HIV and non HIV related deaths. As was done by Stover (2004), a key assumption made is that HIV infected people are subject to the same hazard of mortality from causes other than AIDS as are people who are not infected. In addition, equation sums up in-migration and out-migration into a net migration component, $M^{HIV+}[0,T]$. Equation 7 can be rewritten as;

$$N^{HIV+}(T) = N^{HIV+}(0) + \Delta N^{HIV+}$$

$$N^{HIV+}(T) = N^{HIV+}(0) + \sum_0^T E_t^{HIV+} - \sum_0^T D_t^{HIV+} - \sum_0^T D_t^{P} + \sum_0^T M_t^{HIV+} + \sum_0^T P_t^{HIV+} \qquad \text{equation (8)}$$

At any point in time, *t*, the total adult population (aged 15-49), can be expressed as the sum of those infected and those not infected.

$$N(t) = N^{HIV+}(t) + N^{HIV-}(t)$$

Resultantly, the change in HIV prevalence (aged 15-49)[14] that is due to new infections can be calculated as

$$\text{Change due to new HIV infections} = \frac{\sum_0^T E_t^{HIV+}}{\Delta N[N(t)/T(t)] - \Delta N[N(0)/T(0)]}$$

$$\text{Change due to HIV Deaths} = \frac{\sum_0^T D_t^{HIV+}}{\Delta N[N(t)/T(t)] - \Delta N[N(0)/T(0)]}$$

[14] $\Delta N[N(t)/T(t)] - \Delta N[N(0)/T(0)]$ is the change in HIV prevalence between times *t* and *0*.

$$\text{Change due to Migration } = \frac{\sum_{0}^{T} \mathrm{M}_t^{\mathrm{HIV+}}}{\Delta N[N(t)/T(t)] - \Delta N[N(0)/T(0)]}$$

$$\text{Change due to Non HIV Deaths } = \frac{\sum_{0}^{T} \mathrm{D}_t^{\mathrm{P}}}{\Delta N[N(t)/T(t)] - \Delta N[N(0)/T(0)]}$$

$$\text{Change due to aging into and out of Cohort } = \frac{\sum_{0}^{T} \mathrm{P}_t^{\mathrm{HIV+}}}{\Delta N[N(t)/T(t)] - \Delta N[N(0)/T(0)]}$$

Using this approach, it is possible to characterise the change in HIV prevalence into different components. This will be done for two distinct time periods. The first is the time period from 1985 to 1997, when HIV prevalence was rising. The second is the period from 1997 to 2010 when HIV prevalence declined

2.4.1 The rise in HIV prevalence (age 15-49), 1985-1997

Table 2.5 shows that the rapid rise in prevalence was primarily (33.7%) because of an increase in new infections. Prevalence would have been higher were it not for HIV deaths which reduced prevalence by about 4.1%. Non HIV deaths reduced prevalence by about 0.7% and net migration accounted for around a 0.7% reduction in HIV prevalence between 1985 and 1997. The reduction in prevalence that was due to individuals aging into and out of the cohort was 2.6%. This shows that there were probably more infected people aging out of the cohort than there were infected people turning 15 years old (thus entering the cohort).

Table 2.5 Decomposition of changes in prevalence for Zimbabwe

Change due to New HIV infections, E^{HIV+}[1985,1997]	33.7%
Change due to HIV deaths, D^{HIV+}[1985,1997]	-4.0%
Change due to Non HIV deaths, D^{P}[1985,1997]	-0.7%
Change due to Migration, M^{HIV+}[1985,1997]	-0.7%
Change due to aging into and out of Cohort, P^{HIV+}[1985,1997]	-2.6%
Total Change in HIV Prevalence (age 15-49)	25.6%

Source: Own calculations on Spectrum output, Table 1 of the Appendix.

This is compelling evidence in support of arguments that the early stages of the HIV epidemic were characterised by an increase in incidence (new infections). As I describe in greater detail in the next chapter, the response to the epidemic was slow as educational campaigns did not gain much momentum until the mid-90s (Vos, 1994:193-203; Machaba-Hove, 2008). This was because of denial as well as a general under-appreciation of the severity of the crisis on the part of government. The economic and general prosperity of a Zimbabwe just liberated from its colonial power probably also facilitated growth of the flourishing commercial sex industry as well as sexual networks. Leading the pack were the Harare, Manicaland, Matabeleland South and Masvingo provinces. Changing sexual norms also contributed to the increase in new infections as traditional sex education became less common place. All of these factors would have led to a rapid increase in HIV prevalence and will be more critically discussed in the next chapter. As the huge emigrations out of the country only gained steam post 2000, an analysis of the impact of migration on the rise of HIV prevalence in Zimbabwe, for the period 1985-97 will not reveal much at this point.

2.4.2 Decline in Prevalence (age 15-49), 1997 to 2010

Existing research, which is discussed in more detail in later chapters suggests that the fall in HIV prevalence, beginning around 1997 was due to a couple of reasons. The first is that there was substantial behaviour change as people’s attitudes towards HIV and risky behaviour changed. Some reported changes such as delayed onset of sexual relations, increased monogamy and increased use of condoms (Gregson, 1997). Table 2.6 shows that new infections were a key driver of HIV prevalence. Given the implicit assumptions made in modelling the epidemic, the decline in prevalence (age 15-49) was due to a decrease in new infections although the more dominant reason was an increase in HIV deaths. Most deaths in the cohort estimated to be HIV related (29.4% of the change in HIV prevalence) and not non HIV related deaths. Further, emigration only contributed marginally (0.85%) to the decline in HIV prevalence. There were also more infected people aging out of the cohort than were turning 15 (and were infected already).

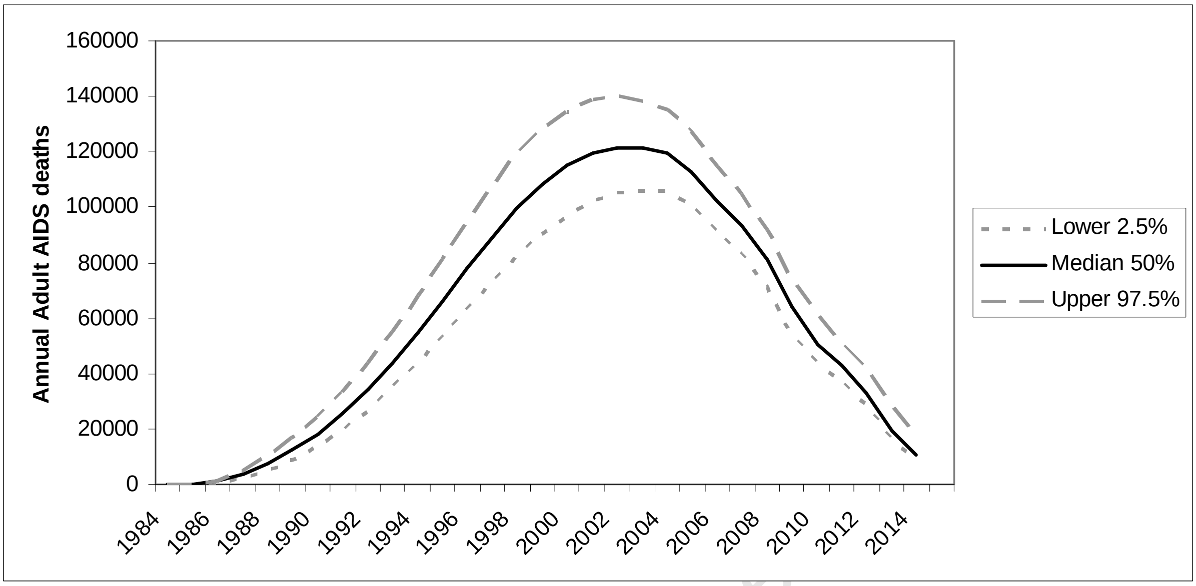
Table 2.6 Decomposition of changes in prevalence (age 15-49) with ART for Zimbabwe

Change due to New HIV infections, E^{HIV+}[1997, 2010]	24.6%
Change due to HIV deaths, D^{HIV+}[1997, 2010]	-29.4%
Change due to Non HIV deaths, D^{P}[1997, 2010]	-0.3%
Change due to Migration, M^{HIV+} [1997, 2010]	-0.9%
Change due to aging into and out of Cohort, P^{HIV+} [1997, 2010]	-10.3%
Total Change in HIV Prevalence (age 15-49)	-16.2%

Source: Own calculations, Spectrum output, Table 2 of the Appendix.

Zimbabwe implemented a treatment program that expanded the number of adults receiving ART from 8 000 in 2004 to 98 000 by the end of 2007, reaching about 19% in need of treatment (UNAIDS, 2008:2-6). Based on the Spectrum modelling done in this chapter, the provision of ART has averted an estimated 72,153 deaths from 2004 to 2009 (summary in Appendix, Tables 5-7). With new infections in the same period totalling about 371,099, more effort needs to be put in expanding the ART rollout in order to meet the rising need.
Figure 2.17 shows that the number of AIDS deaths took a considerable dip in 2004 and has continued falling all the way up to 2010. The future projection of AIDS deaths show that the reduction in the numbers of those succumbing to AIDS will carry on decreasing.

Figure 2.16 Annual Adult AIDS deaths for Zimbabwe

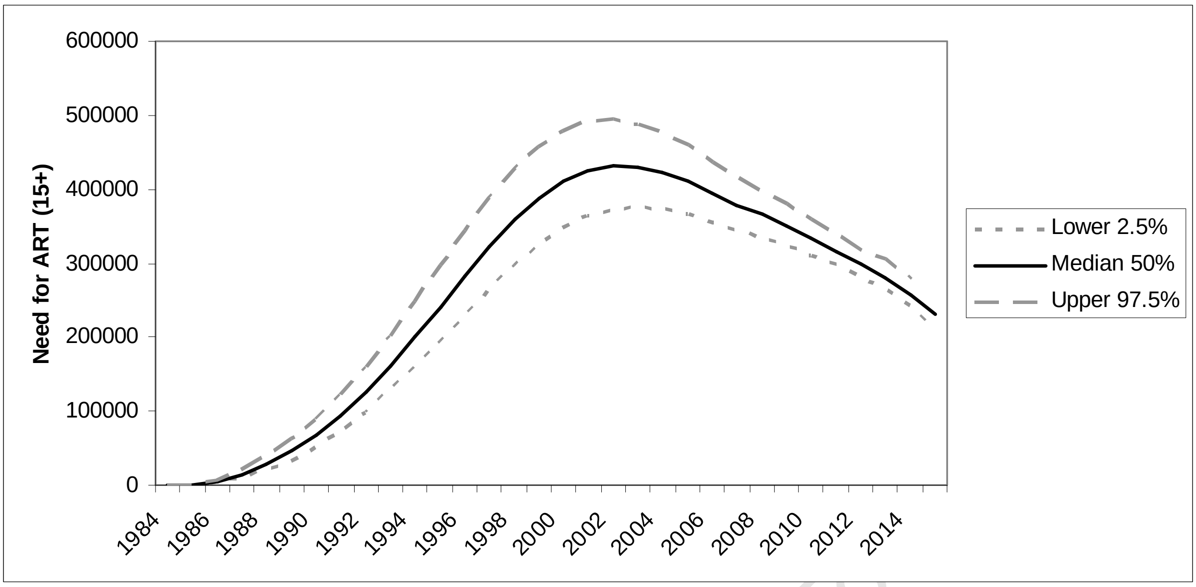


Source: Spectrum output

The number of people in need of treatment has been declining from the early 2000s as incidence began to fall. The need for ART is currently decreasing by about 8000 each year (see appendix). Figure 2.18 shows this trend clearly. This implies that the need for ART is expected to decrease by almost 35% from 350,935 in 2009 to about 230,526 by 2015 if the current rate of increase of coverage is maintained. This is important to consider given the cost and sustainability implications for the program and the evolving political and economic environment in Zimbabwe.[15]

[15] A Government of National Unity between R.G Mugabe and M Tsvangirai was formed late 2008.

Figure 2.17 :Need for ART for Zimbabwe



Source: Spectrum output

Had government not initiated the ART rollout, prevalence would have fallen by about 17.4 percentage points from 1997 to 2010 as shown in Table 2.7. This would have been driven primarily by HIV deaths. The trade-off between saving lives by ensuring more infected people are on treatment versus the targeting of lower prevalence rates is evident. A desirable outcome would be to ensure that those who are infected are educated and counselled so that they adopt safe sex practices and help reduce the number of new infections (thus reducing the prevalence rate).

Table 2.7 Decomposition of changes in prevalence (age 15-49) with No ART for Zimbabwe

Change due to New HIV infections, E^{HIV+}[1997, 2010]	24.7%
Change due to HIV deaths, D^{HIV+}[1997, 2010]	-33.4%
Change due to Non HIV deaths, D^{μ}[1997, 2010]	-0.1%
Change due to Migration, M^{HIV+} [1997, 2010]	-0.9%
Change due to aging into and out of Cohort, P^{HIV+} [1997, 2010]	-7.8%
Total Change in HIV Prevalence (age 15-49)	-17.4%

Source: Own calculations, Spectrum output, Table 3 of the Appendix.

Assuming that since 2001, the approximate number of people emigrating out of the country has been to the order of 2 million people (arbitrary assumption); the decrease in HIV prevalence between 1997 and 2010 can be broken down as shown in Table 2.8. Making this assumption and using Spectrum to estimate the relevant statistics reveals some interesting details. First, it is clear that assuming emigrations over the past ten years have been about 2 million (not 1.4 million) implies that the overall share of the overall change in prevalence (aged 15-49) attributable to new infections is reduced from 24.6% to 22.6%.   A bigger assumption on emigration implies a relatively smaller effect of the decline in new infections and a bigger contribution of out-migration to the decline in overall prevalence rates than under the default[16]. Even then the effect of migration is dwarfed by that of the reduction in new infections and increase in AIDS deaths.

Table 2.8 Decomposition of changes in prevalence (age 15-49) with ART for Zimbabwe

	Adjusted	Default
Change due to New HIV infections, E^{HIV+}[1997, 2010]	22.6%	24.6%
Change due to HIV deaths, D^{HIV+}[1997, 2010]	-36.4%	-29.4%
Change due to Non HIV deaths, D^{P}[1997, 2010]	-2.4%	-0.3%
Change due to Migration, M^{HIV+} [1997, 2010]	-6.1%	-0.9%
Change due to aging into and out of Cohort, P^{HIV+} [1997, 2010]	6.2%	-10.3%
Total Change in HIV Prevalence (age 15-49)	-16.2%	-16.2%

Source: Own calculations, Spectrum output, Table 4 of the Appendix.

It also reveals that whereas the default Spectrum assumptions on migration imply that the people aging out or into the cohort decreased prevalence by 10.3%, using the still very modest estimates of 2 million out-migrations implies that this effect increased prevalence by 6.2%[17]. This reflects the possibility that a substantial number of infected children turned 15 (i.e. aged into the cohort) between 1997 and 2010. It could also reflect the possibility that more

[16] It is noteworthy that the modelling presented here ignores the possibility that during the period under discussion, HIV prevalence rates among immigrants may have been higher than that of emigrants. The current model assumes that the emigrants had higher HIV prevalence than immigrants, which is a likely scenario but one with little evidence to support it.

[17] It is also important to note that the overall HIV estimates or curve of HIV prevalence does not change even when we change the migration assumptions. Figure 2.8 shows that the overall decline in prevalence between 1997 and 2010 was 16.20 percentage points in each case.

uninfected adults aged out of the cohort in that same period thereby pushing HIV in the cohort up by about 6.2%.

It is clear from this analysis that using a larger estimate of total out-migrations results in a different conclusion on the relative effect of the different factors contributing to the decline in prevalence. Assuming a bigger estimate of out-migrations means that the relative contribution of migration to the decline in prevalence also becomes larger. This is possibly a controversial conclusion which indicates how important it is for any HIV prevalence estimates to be accompanied by an explanation of the modelling assumptions used. This will prevent those referring to these estimates from drawing inaccurate conclusions. For instance, simply saying that migration played a very minor role in the decline in HIV prevalence in Zimbabwe without indicating the migration assumptions used to model the HIV prevalence (which in the case of the National HIV and AIDS Estimates Working Group probably under-estimate true out-migration) is probably misleading. The analysis carried out in this section, coupled with the evidence from Gregson et al (2010) make this cautionary statement very important for any discussion of Zimbabwe's recent decline in HIV prevalence.

2.5 Conclusion

The work in this chapter shows that it is possible to reverse engineer the HIV prevalence estimates obtained by the UNGASS and MOHCW working group using reasonable modelling assumptions. These assumptions imply that the rise in HIV prevalence was primarily due to an increase in new infections. They also imply that the fall in HIV prevalence was due to a combination of factors including a drop in new infections, an increase in AIDS deaths as well as in increase in the out-migration of infected individuals.

I have argued that the assumptions on migration that allow for the Spectrum model to produce prevalence estimates comparable to the UNGASS estimates however underestimate the level of out-migration from Zimbabwe since 2000. The default migration estimates assume that between 2000 and 2010, the cumulative total of migrations out of Zimbabwe was 1.4 million. By changing this assumption to a still conservative estimate of about 2 million out-migrations, an interesting change is observed. Assuming more out-migration means that in addition to the decrease in new infections and an increase in AIDS deaths, out-migration also becomes a significant contributor to the decline in prevalence in Zimbabwe. However, the impact of out-migration on the epidemic in Zimbabwe should not be over-emphasized.

The Zimbabwe case study illustrates that EPP and Spectrum model estimates are sensitive to the assumptions applied and should be interpreted with caution. Even with reasonable assumptions, there are differences between the epidemics in each sub-region or province. For instance, in most provinces, HIV prevalence rates peaked around 1996/7 (Harare in 1996 at 35.9%, Bulawayo in 1997 at 25.9%, Mashonaland East in 1997 at 30.9%, Mashonaland Central in 1997 at 30.0%, Midlands in 1996 at 35.9% and Manicaland in 1997 at 34%). On the other hand, HIV prevalence in the more economically isolated, sparsely populated and poorly connected (communication and transport infrastructure) Matabeleland North province peaked much later in 2000 and at a much lower level (19.7%). These differences mean that overall national estimates have a substantial margin for error. Another issue with EPP and Spectrum estimates is that they are based on ANC data. These data are sometimes inconsistently collected over time and only a few ANC sites have data points for consecutive years. This means that the models have to fill in the gaps essentially increasing the error margin of the estimates.

The next chapter provides an account of Zimbabwe's socio-economic history and the virtual collapse of the country's economy, public infrastructure and health care system. It also outlines how the political and economic situation resulted in the migration of millions of young adult Zimbabweans to many countries primarily in SADC in search of work. This analysis provides a backdrop for an analysis of the reasons for the trends in HIV prevalence and incidence estimated in this chapter. Most importantly, it highlights how national trends in HIV prevalence are a product of a wide array of factors which invariably make it even more challenging to characterize any national epidemic. In cases like Zimbabwe where there is social disruption the task is even more challenging.

Annex A EPP AND SPECTRUM RESULTS

Table A.1 Changes in the HIV + population (1985-1997) for Zimbabwe

	adult HIV+	adult HIV-	adult HIV+ & HIV-
$N^{HIV+(}$1997)	1877786	4845401	6723187
$N^{HIV+(}$1985)	96780	4092830	4189610
E[1985,1997]	2342632	-2342632	0
D^{HIV+}[1985,1997]	-281212	0	-281212
D^{P}[1985,1997]	-48798	-287949	336747
M^{HIV+}[1985,1997]	-50679	-125765	-176444
P[1985,1997]	-180938	3508916	2765555

Source: Spectrum modelling

Table A.2 Changes in the HIV + population (1997-2010) for Zimbabwe

	adult HIV+	adult HIV-	adult HIV+ & HIV-
N^{HIV+}(2010)	1074893	8353993	9428886
N^{HIV}+(1997)	1855599	4867587	6723187
E^{HIV+}[1997,2010]	1185680	-1185680	0
D^{HIV+}[1997,2010]	-1416743	0	-1416743
D^{P}[1997,2010]	-14560	-70839	-85399
M^{HIV+}[1997,2010]	-41116	-141884	-183000
P[1997,2010]	-493967	4884809	1386557

Source: Spectrum modelling

Table A.3 Changes in the HIV + population (1997-2010) for Zimbabwe

	adult HIV+	adult HIV-	adult HIV+ & HIV-
N^{HIV+}(2010)	1074893	8696862	9771755
N^{HIV+}(1997)	1855599	4673238	6528838
E^{HIV+}[1997,2010]	1106838	-1106838	0
D^{HIV+}[1997,2010]	-1495763	0	-1495763
D^{P}[1997,2010]	-2888	-3492	-6379.499465
M^{HIV+}[1997,2010]	-41116	-141884	-183000
P[1997,2010]	-347777	5275837	1923775

Source: Spectrum modelling

Table A.4 Changes in the HIV + population (1997-2010) for Zimbabwe

	adult HIV+	adult HIV-	adult HIV+ & HIV-
$N^{HIV+(}$2010)	1074893	8353993	9428886
N^{HIV+}(1997)	1855599	4867587	6723187
E^{HIV+}[1997,2010]	1208716	-1208716	0
D^{HIV+}[1997,2010]	-1815911	0	-1815911
D^{P}[1997,2010]	-177621	-728241	-905862
$M^{HIV+[}$1997,2010]	-238207	-992840	-1231047
P[1997,2010]	242316	6416203	1214973

Source: Spectrum modelling

Table A.5 Spectrum initial output summary (2007, 2009 estimates) for Zimbabwe

	2007 Est	2007 Bounds	2009 Est	2009 Bounds
Total (adults and children)	1,332,440	1,250,110 - 1,382,400	1,156,361	1,024,220 - 1,288,02
Adults (15-49)	1,096,474	965,333 - 1,227,615	947,036	815,895 - 1,078,177
Women (15-49)	777,934	680,793 - 875,075	675,317	578,176 - 772,458
Children (15-49)	124,034	117,335 - 133,214	109,555	93,676 - 125,434
Adult Prevalence (15-49)	15.12	14.7 - 16.2	12.52	11.8 - 13.27
New HIV infections-Adult	39,321	37,755 - 41,288	38,744	37,178 - 40,310
New HIV Infections-Children	13,754	13,410 - 14,098	11,567	11,223 - 11,911
Annual AIDS deaths-Adult	120,061	117,059 - 123,063	117,028	114,026 - 120,029
Need for ART-Adult (15+)	349,139	340,410 - 357,867	354,813	346,085 - 363,541
AIDS orphans	990,450	965,689 - 1,015,211	983,528	958,767 - 1, 008,289

Source: Spectrum modelling

Table A.6 Spectrum projections for Zimbabwe (1986-1995)

Year	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995
HIV population										
Total	174,497	261,621	381,846	538,992	729,305	942,672	1,158,185	1,358,474	1,532,920	1,679,149
Male	75,010	111,139	161,093	224,739	301,996	389,497	477,054	557,695	629,983	690,417
Females	99,487	150,482	220,754	314,252	427,309	553,175	681,131	800,778	902,938	988,731
Prevalence (15-49)	4.01	5.75	8.04	10.88	14.13	17.52	20.69	23.35	25.39	26.82
Annual HIV + Births										
Total	4,565	6,614	9,330	12,824	16,855	21,098	24,972	28,179	30,491	32,090
Percent	1.22	1.75	2.44	3.31	4.29	5.32	6.25	7.01	7.56	7.92
Cumulative AIDS deaths										
Total	9,245	14,778	23,127	35,519	53,427	78,515	112,504	157,150	214,145	285,037
Males	4,783	7,576	11,758	17,923	26,768	39,060	55,569	77,045	104,192	137,626
Females	4,462	7,201	11,368	17,596	26,659	39,454	56,935	80,104	109,953	147,412

Source: Spectrum modelling

Table A.7 Spectrum projections for Zimbabwe (1996-2005)

Year	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005
HIV population										
Total	1,797,089	1,877,786	1,919,497	1,926,161	1,902,790	1,854,541	1,786,521	1,703,643	1,611,866	1,515,247
Male	739,504	773,789	791,878	795,297	786,569	767,702	740,557	707,064	669,625	629,898
Females	1,057,585	1,103,997	1,127,619	1,130,864	1,116,221	1,086,838	1,045,963	996,580	942,241	885,349
Prevalence (15-49)	27.67	27.93	27.61	26.84	25.74	24.41	22.91	21.33	19.7	18.09
Annual HIV + Births										
Total	32,920	32,990	32,313	31,208	29,780	27,994	26,006	23,991	21,790	19,572
Percent	8.12	8.17	8.01	7.75	7.41	7.01	6.56	6.08	5.5	4.93
Cumulative AIDS deaths										
Total	370,967	472,465	589,310	720,612	864,809	1,019,701	1,182,636	1,350,757	1,518,066	1,681,655
Males	177,752	224,690	278,223	337,859	402,829	472,106	544,490	618,709	692,160	763,619
Females	193,215	247,774	311,087	382,753	461,980	547,595	638,146	732,049	825,907	918,036

Source: Spectrum modelling

Table A.8 Spectrum projections for Zimbabwe (2006-2015)

Year	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015
HIV population										
Total	1,419,414	1,332,440	1,241,395	1,156,361	1,074,893	997,753	926,969	862,822	804,985	752,393
Male	590,408	554,505	516,620	481,044	446,770	414,168	384,216	357,138	332,820	310,767
Females	829,006	777,934	724,775	675,317	628,123	583,585	542,753	505,684	472,165	441,626
Prevalence (15-49)	16.52	15.12	13.76	12.52	11.4	10.37	9.46	8.65	7.92	7.27
Annual HIV + Births										
Total	16,433	13,754	12,643	11,567	10,628	9,767	9,033	8,391	7,799	7,252
Percent	4.14	3.46	3.17	2.89	2.64	2.43	2.24	2.08	1.93	1.79
Cumulative AIDS deaths										
Total	1,828,499	1,962,864	2,100,063	2,230,391	2,358,743	2,482,689	2,599,831	2,708,586	2,808,362	2,899,356
Males	827,542	885,747	945,008	1,001,119	1,056,214	1,109,324	1,159,497	1,206,121	1,248,984	1,288,187
Females	1,000,957	1,077,118	1,155,055	1,229,272	1,302,528	1,373,365	1,440,334	1,502,465	1,559,378	1,611,169

Source: Spectrum modelling

Table A.9 Spectrum projections for Zimbabwe (1985-1993)

Year	1985	1986	1987	1988	1989	1990	1991	1992	1993
Maternal Orphans									
AIDS	1,927	3,228	5,360	8,861	14,515	23,376	36,780	56,231	83,274
Non AIDS	172,414	172,206	171,888	171,165	169,715	167,438	164,287	160,217	155,886
Total	174,341	175,433	177,248	180,026	184,229	190,815	201,067	216,448	239,160
Paternal Orphans									
AIDS	2,100	3,274	5,084	7,867	12,116	18,464	27,705	40,781	58,633
Non AIDS	266,957	265,232	263,310	260,008	254,847	248,091	240,192	231,879	224,144
Total	269,057	268,506	268,394	267,875	266,962	266,555	267,898	272,660	282,778
Double Orphans									
AIDS	563	888	1,389	2,580	5,150	8,304	12,917	18,397	25,999
Non AIDS	37,657	36,104	34,381	32,292	30,447	28,473	26,441	24,437	22,615
Total	38,220	36,991	35,769	34,872	35,597	36,777	39,359	42,834	48,614
Total Orphans	405,178	406,948	409,872	413,029	415,595	420,592	429,606	446,274	473,323
All AIDS orphans	3,644	5,896	9,495	14,829	22,527	35,115	53,898	81,962	120,592

Source: Spectrum modelling

Table A.10 Spectrum projections for Zimbabwe (1994-2002)

Year	1994	1995	1996	1997	1998	1999	2000	2001	2002
Maternal Orphans									
AIDS	119,226	164,875	220,422	285,072	357,047	433,667	511,657	587,961	659,647
Non AIDS	151,919	148,218	144,781	141,628	138,782	136,225	133,860	131,695	129,822
Total	271,145	313,092	365,203	426,700	495,830	569,892	645,517	719,655	789,468
Paternal Orphans									
AIDS	82,109	111,694	147,471	189,033	235,440	285,293	336,856	388,514	438,575
Non AIDS	217,886	212,808	208,738	205,777	203,888	202,880	202,442	202,433	202,840
Total	299,996	324,502	356,208	394,809	439,329	488,173	539,298	590,947	641,415
Double Orphans									
AIDS	36,719	51,539	71,598	97,923	131,244	171,599	218,186	269,639	323,694
Non AIDS	21,085	19,780	18,655	17,714	16,954	16,353	15,862	15,439	15,082
Total	57,804	71,319	90,253	115,638	148,198	187,951	234,048	285,078	338,776
Total Orphans	513,337	566,275	631,158	705,872	786,960	870,114	950,767	1,025,524	1,092,107
All AIDS orphans	171,011	233,523	307,270	389,997	478,196	567,658	654,044	733,931	804,854

Source: Spectrum modelling

Table A.11 Spectrum projections for Zimbabwe (2003-2011)

Year	2003	2004	2005	2006	2007	2008	2009	2010	2011
Maternal Orphans									
AIDS	724,172	778,350	820,638	847,968	847,968	859,591	855,134	841,478	822,929
Non AIDS	128,048	126,191	124,292	122,407	122,407	120,590	118,783	116,926	115,069
Total	852,220	904,541	944,930	970,375	970,375	980,181	973,917	958,404	937,999
Paternal Orphans									
AIDS	485,490	528,784	566,870	603,583	603,583	630,466	636,126	635,956	631,136
Non AIDS	203,439	204,106	204,724	205,635	205,635	206,131	205,761	205,069	204,118
Total	688,929	732,890	771,594	809,218	809,218	836,598	841,887	841,025	835,253
Double Orphans									
AIDS	377,445	428,323	473,288	513,199	513,199	539,500	540,775	532,614	517,566
Non AIDS	14,761	14,462	14,178	13,941	13,941	13,708	13,436	13,155	12,869
Total	392,205	442,785	487,466	527,139	527,139	553,208	554,211	545,769	530,434
Total Orphans	1,148,944	1,194,646	1,229,058	1,252,453	1,252,453	1,263,571	1,261,593	1,253,660	1,242,818
All AIDS orphans	865,485	914,580	951,956	977,556	977,556	990,450	990,020	983,528	974,104

Source: Spectrum modelling

Summary of assumptions and methodology

Below is a summary of the modelling process carried out in Chapter 2.

Epidemic Projection Package (EPP)

1. Zimbabwe is modelled as a generalized epidemic (i.e. the national HIV prevalence is consistently over 1% in pregnant women making the use of ANC data appropriate).
2. HIV prevalence from the ZDHS 2005-06 (with 2005 specified as the survey year). This is used to calibrate the ANC data
3. Using the Spectrum DemProj, the population statistics from the Zimbabwe 2002 Census and 2008 Inter-censal Demographic Survey data were projected up to 2010.
4. Prevalence rates calculated using results from the Genscreen HIV test (1989 through 2001) for the rural ANC sites Birchenough Bridge, Gutu, Hauna Growth Point, Murambinda, Mutoko and Sadza were adjusted down by 30%.
5. All data points for Chiredzi prior to 2001 are excluded.
6. Data from Musume site in 2000 are also excluded.
7. Base data on the number of people receiving ART was taken from the UNAIDS Epidemiological Fact Sheet.

Spectrum 4.41, the most recent version of Spectrum released on the 6th of July 2011 is the software package that is used to produce estimates of HIV and AIDS prevalence for adults and children, the number of new HIV infections, new AIDS cases, AIDS mortality and orphans. This latest version of Spectrum has EPP built in to allow the use to do all the modelling in one modelling package. However, the in-built EPP is optional and users can still do initial modelling in EPP (2009) then take the prevalence or incidence estimates to Spectrum 4,4. In this chapter the second option is chosen as it allows for a more systematic discussion of the modelling process.

Spectrum

1. For 1970, the base year, United Nations Population Division estimates of the population size and distribution in Zimbabwe were used.
2. The Coale-Demeny North life Table was used
3. Spectrum default assumptions on the Total Fertility Rate reduction were used.

4. The Spectrum default Total Fertility and Age-Specific Fertility Rates (TFR and ASFR) are similar to data from the 1988, 1994 and 1999 Zimbabwe Demographic and Health Surveys (ZDHS) and the 2002 Zimbabwe Census and so these were not changed
5. Life Expectancy at Birth assumptions were left at the default Spectrum assumption.
6. Adult HIV prevalence was imported from the EPP 2009 projections.
7. Following Zimbabwe's National HIV and AIDS Estimates Working Group, the net survival of people living with HIV is 11 years instead of the previous 9 years.
8. As per the default Spectrum assumption, in the absence of antiretroviral treatment, people living with HIV will become eligible for treatment an average of 3 years before they are expected to die from an AIDS related cause, instead of previously 2 years.
9. Following Zimbabwe's National HIV and AIDS Estimates Working Group, the AIM default setting regarding the HIV age distribution was used with the sex distribution adjusted using data from the YAS 2001-2002 and the ZDHS 2005-2006.
10. UNAIDS Zimbabwe Epidemiological Fact sheet Mother to Child Transmission data was used in the projection. The Spectrum default assumptions for the probability of transmission were used.
11. UNAIDS Zimbabwe Epidemiological Fact sheet data on ART rollout and Child Treatment services were used.
12. Following Zimbabwe's National HIV and AIDS Estimates Working Group, the proportion of women never married was taken from the ZDHS while the Spectrum default for the proportion of married women in monogamous marriages was applied. These assumptions are for the estimation of data relating to orphan-hood.

3 AN OVER-VIEW OF ZIMBABWE'S SOCIO-ECONOMIC DECLINE

Abstract

In this chapter, I provide some background on Zimbabwe's socio economic decline (early 1990s to the present). Much of the conventional wisdom and received opinion is that the political turmoil and economic collapse negatively impacted health care delivery, HIV prevention programs and led many of Zimbabwe's working aged population to leave the country in search for opportunities elsewhere.. The political climate in Zimbabwe also evolved such that the government became increasingly mistrustful of civil society, a situation that had an impact on the government- civil society partnerships and efforts at fighting the epidemic. This chapter focuses less on analysing whether many of the conventional arguments are true and more on describing the trajectory of the economy so as provide a political economy background to the work in subsequent chapters.

3.1 Introduction

Zimbabwe has one of the highest HIV prevalence rates in the world. It is ranked 5th (in ascending order) after South Africa, Lesotho, Botswana and Swaziland (UNAIDS, 2009:6).The epidemic has imposed a terrible burden on Zimbabweans due to increased mortality levels, reduced quality of life and large numbers of orphans. With help from the UN and other development partners, efforts have been launched to prevent the transmission of HIV from mothers to their babies (MTCTP) and to provide ART to those who need it. However, the unstable socio-political environment in Zimbabwe has made this difficult.

In this chapter, I introduce the political, economic and social context of the rise and the decline in HIV prevalence since the mid-1990s. I conduct a review of available literature on Zimbabwe in an attempt to conduct a preliminary account of the various political, economic and social forces that may have shaped the HIV epidemic. I use this chapter to create a foundation for further exploration of factors influencing trends in HIV prevalence in Zimbabwe (done in the next chapter).

In particular, I look at the extent to which the economic prosperity and relative political stability that characterized early post-colonial Zimbabwe may have set the platform for a rapid rise in HIV prevalence. Conversely, the political turmoil, beginning in the early 1990s negatively impacted health care delivery, HIV prevention programs, communication and transport infrastructure and migration thereby setting the stage for a fast decline in HIV prevalence.

The key developments in Zimbabwe's socio-political climate that may have had a significant impact on the progression of the epidemic are shown in Figure 3.1 and explained further in the rest of the chapter. The most notable of these were the economic structural adjustment program (ESAP) implemented in 1991.the Land Acquisition Act of 1992, the beginning of land expropriations in 1997, 1998 food riots, and the violence-plagued elections of 2000, 2005 and 2008. It is however important to note that Figure 3.1 does not purpote to show a relationship between GDP growth rates and prevalence[18]. Rather it tries to show the evolution of Zimbabwe's politics during different stages of the epidemic.

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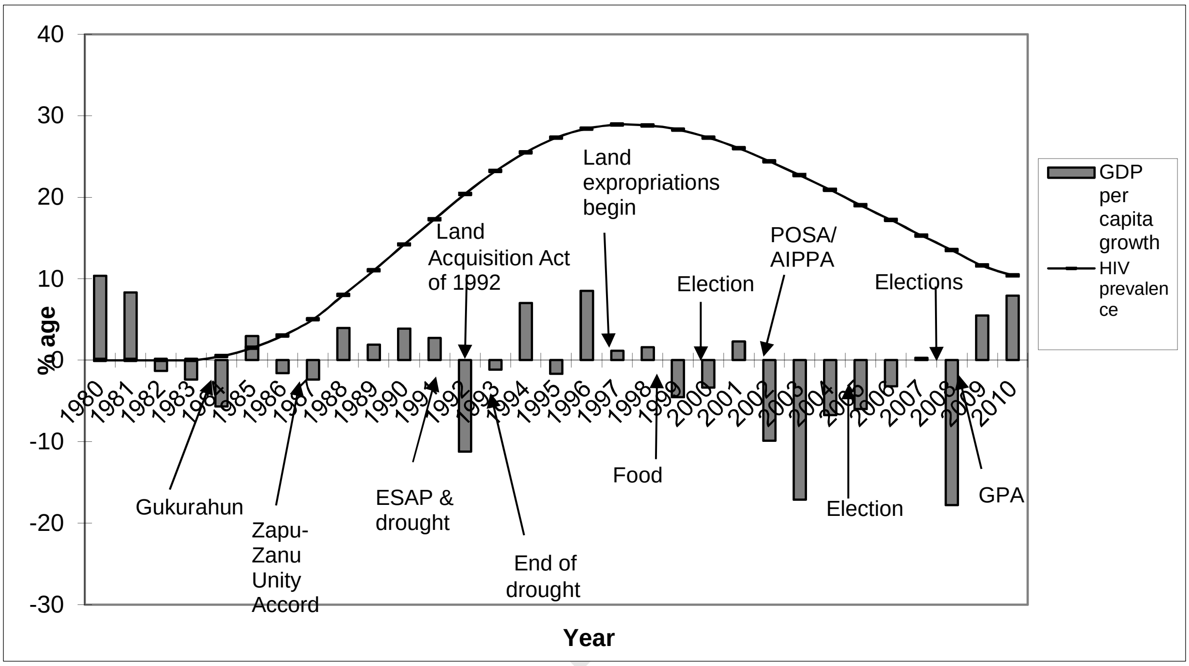
Figure 3.1 HIV prevalence versus GDP per capita growth (%)



Source: World Bank (2010), EPP and spectrum modelling (from Chapter 2)

Because the political economy of Zimbabwe has been anything but simple and straight forward, I discuss Zimbabwe's post colonial history in three parts. First is the period from 1980 to 1990, which was characterised by the consolidation of power by Mugabe and the crafting of a number of substantial growth and development intiatives. The second period, from 1990 to 1997 was characterised by the growing influence of civil society, non governmental organisations and a decline of Zimbabwe African National Union Patriotic Front's (ZANU PF) hold on power. It was also a period when corruption, structural adjustment programmes and gross mismanagement of the economy led to inflation, unempoyment and general economic collapse (Muzondidya, 2009; Kanyeze, 2003; Dhliwayo, 2001). Finally, I describe the deterioration of the socio economic situation into the much

[18]In any case, it is unlikely that HIV prevalence in one year is significantly determined by political economy in that year. However, the diagram does make the temptation to interpret the diagram as showing some kind of relationship between the two. Such an interpretation is not appropriate.

popularised "Zimbabwean crisis" (from 1998 to 2008)(Raftolpolous, 2009; Ndlovu-Gatsheni, 2008).

3.2 Early post-colonial Zimbabwe (1980-1990)

Zanu PF came into power in 1980 after a protracted guerilla war against white minority rule. The first democratic elections gave Zanu PF, led by Robert Mugabe, a majority in the house of parliament (57 seats) and PF Zapu led by Joshua Nkomo, 20 seats (Sibanda, 2005:222). Negotiations leading to the severing of British colonial influence over Zimbabwe had resulted in an agreement to reserve 20 white seats in the new parliament (Lodge et al, 2002:437-439). As early as 1982, Mugabe dismissed all PF Zapu members of cabinet accusing the party of attemping to overthrow the government (Institute for War and Peace Reporting, 2008:2; Alexander, 1998:151-182;CCJP, 1999:4-14). It was alleged that former freedom fighters from Zapu were perpetrating acts of violence in the Matabeleland and Midlands provinces (CCJP, 1999:4-14; New Zimbabwe, 2010). By 1984, Mugabe had dispatched a North Korea trained special forces army unit to quell the alleged dissent in an operatioin popularly known as Gukurahundi (CCJP, 1999:4-14; Mail & Guardian, 2010). Human rights activists now estimate that this army unit was responsible for the deaths of over 20 000 people, while thousands more were displaced from their homes in Matabeleland and Midlands, the provinces where Zapu had overwhelming support (New Zimbabwe, 2010; Institute for War and Peace Reporting, 2008:2).

The key characteristics of the Zanu PF post-independence state were lack of tolerance for political diversity and dissent, heavy reliance on force for mobilization and a narrow, monolithic interpretation of citizenship, nationalism and national unity (Raftopolous et al 2009:179; Raftopolous, 2004:4). In 1987, the government abolished the reservation of 20 white seats in parliament and 10 in the senate and a year later, Mugabe and Nkomo signed a unity agreement that led to the merging of PF Zapu and Zanu PF into Zanu PF (Ndlovu-Gatsheni, 2008:4). The agreement aimed at establishing a Marxist Leninist one party state even though President Mugabe himself publicly admitted that the idea faced internal opposition (Moyo, 1991:99).

The return to peace in Zimbabwe stabilized the economy and between 1987 and 1990, the economy experienced substantial growth (see Figure 3.1). It is also during this period that the HIV epidemic gained much momentum. With the first case having been recorded in 1985, HIV prevalence rose rapidly, reaching 14.2% in 1990 (UNAIDS/WHO, 2008). A discussion of the different interventions that government attempted to control the spread of the epidemic during this early phase is discussed at length in the next chapters.

In addition to trying to consolidate power, between 1980 and 1990, Robert Mugabe had the difficult task of ensuring sustainable post war reconstruction and growth. He also attempted to redress racial imbalances and reform inherited state institutions (Muzondidya, 2009:167). Government attempted to reduce the inequalities inherited from the colonial era by broadening black participation in the economy and making it more inclusive by integrating blacks through black economic empowerment, the employment of more blacks in the public service and the development of a black middle class (Muzondidya, 2009:167; Raftopoulous, 1996).

Mugabe introduced a gradual land resettlement programme aimed at solving both rural poverty as well as racial inequality in land ownership (Mumbengegwi, 2002:240). Government also tried to empower rural peasant farmers through subsidisation of inputs and opening up of lines of credit. These pro-rural agricultural policies improved rural agricultural output and in the early years after independence, communal farmers became the largest producers of maize and cotton (Moyo, 2000:24).

In the early 1980s, government, with the help of Scandinavian countries (Denmark, Norway and Sweden) expanded the provision of health and educational facilities to areas previously neglected by the colonial regime (Muzondidya, 2009:168). It focused on building roads, clinics, boreholes in rural communities and by 1990; substantial progress had been made in terms of infrastructure development (Muzondidya, 2009:168; Mlambo, 1997:55-82). The government’s efforts were noticeable internationally and it won praise from WHO and UNICEF who awarded it a prize for improving access to water and sanitation to rural households (84% of the national population had access to clean water by 1988) (Musemwa, 2008:6).

Health services were also dramatically improved after independence: real per capita recurrent expenditure on health almost doubled from 1979/80 to 1990/91 and this led to improvement in other social indicators such as the under-five mortality rate (Chisvo, 1993:3-20). Real per capita expenditure during the first five years after independence increased a whopping 51% as shown by the figures in Table 3.1.

Table 3.1 Real Recurrent Health Expenditure (at 1990 prices)

Year	Total Real Health Exp.(Z$ millions)	Per capita Real Health Exp. (Z$)	Health as % of GDP	Health Exp. As % of Gov.
1980/81	256.24	35.62	2.0	5.3
1981/82	306.60	41.44	2.2	5.6
1982/83	300.10	39.44	2.1	4.8
1983/84	289.61	36.74	2.2	4.8
1984/85	297.97	53.92	2.2	4.9
1985/86	331.00	39.48	2.3	5.3
1986/87	355.64	41.14	2.5	5.1
1987/88	384.10	43.08	2.8	5.5
1988/89	412.18	44.82	2.8	5.4
1989/90	478.88	50.5	2.6	5.9
1990/91	564.49	57.72	2.8	6.2
1991/92	511.97	50.72	3.0	5.1
1992/93	458.18	44.00	2.5	5.3
1993/94	412.88	38.45	2.4	5.1
1994/95	424.33	38.31	2.2	4.5
1995/96	409.71	35.86	2.2	4.2

Source: MOHCW, 1998; Central Statistical Office, 1985-1996.

One of Mugabe’s other positive achievements was the broadening of access to education. Enrolment in primary schools rose from 82,000 in 1979 to 2,216,878 in 1985 (Mlambo, 1997:59). Secondary school enrolment rose from 66,000 to 482,000 and, during the same period, the number of primary and secondary schools grew by 80% from 3,358 in 1980 to 6,042 in 1990 (Mlambo, 1997:59). As shown in Table 3.2, the gains in education services were on the back of a marked increase in education expenditure. Between 1980 and 1985, education expenditure as a percentage of GDP almost doubled (from 1.8 to 3.1).

The improvements in education did not lead to substantial increases in employment during the first decade of independence. Academics have cited the fact that the economy was growing at a rate slower than the rate of population growth (as shown in Table 3.1) and so the absorption of young educated Zimbabweans into the workforce was limited (Moyo, 2000: 26; Stoneman, 1988:47).

Table 3.2 Education Expenditure

Year	Education exp. as % of GDP	Education exp. as % of Gov. Exp
1980	1.8	10.3
1985	3.1	15.8
1990	1.9	16.9
1995	0.7	16.3
1996	0.5	15.8

Source: Report of the Comptroller & Auditor General (various years)

From 1982 to 1990, GDP growth rates averaged 1.3% (1.4% between 1980 and 1987) per annum while the population grew at an average of 3.3% (2.3% between 1980 and 1987) per annum (Stoneman, 1988:47). In the ten years after independence, only 10000 jobs were created yet there were thousands more leavers (Muzondidya, 2009:169; Stoneman, 1988:47). Table 3.3 shows that between 1980 and 1987, the rate of growth of formal employment was only half (0.7%) of the rate of growth of GDP.

Table 3.3 Employment, GDP and population growth

	1980	1981	1982	1983	1984	1985	1986	1987	Avg.(%)
Population[1]	7480	7730	7517	7729	7949	8175	8406	8639	
Pop. Growth rate (%)		3.3	2.84	2.84	2.84	2.84	2.76	2.76	2.3
Labour force (incl. com. Farmers)			2484	2623	2721	2823	2930	2039	
Labour force growth rate (%)				5.3	3.7	3.8	3.8	3.7	3.7
Formal employment	1010	1038	1046	1033	1036	1060	1065	1093	
Formal employment growth rate (%)	2.5	2.8	0.8	-1.2	0.3	2.3	0.5	2.6	0.7
GDP growth rate (%) (1980) constant prices	11	13	-1.2	-3.5	2.3	7.6	2.3	0.3	1.4

Source: CSO, Quarterly Digest of Statistics, June 1988: CSO, Population Projections, Medium Variant. Dec 1985: CSO, 1992 Census/ 10% Sample

Notwithstanding the challenge of job creation, government tried to improve the plight of those in employment. Wages and working conditions were improved (Muzondidya, 2009:168). This was done by the introduction of a minimum wage and the strengthening of workers bargaining strength through the introduction of collective bargaining (Dansereau, 2003:30). Government made it mandatory for companies to improve the working conditions for employees and their families (Kanyeze, 2003). Although the wage laws increased people’s incomes, Table 3.4 shows that in most sectors, real minimum wages did not always increase. In fact, minimum wages were sometimes decreased depending on the general health of each sector and the overall economy as shown by the GDP growth rates in Table 3.3.

Table 3.4 Minimum Real Wages: Indices (July 1980=100)

Date of order	Agriculture and Domestic	Mining	Industry and Commerce
Jan-1980	100.0	100.0	100.0
Jan-1981	96.8	117.5	117.5
Jan-1982	141.2	127.1	127.1
Sep-1983	107.3	92.0	96.1
Jul-1984	116.7	92.3	96.2
Jul-1985	125.9	103.6	103.6
Jul-1986	121.7	97.0	97.0
Jul-1988	116.3	90.7	90.7

Source: Knight (1996)

3.2.2 Increasing discontent

The biggest challenge facing government was that the economic boom of the immediate post-independence period did not last long (Mlambo, 1997:40-1; Burdette et al, 1987:79). The economy had grown quite rapidly (averaging 12% per year) during the first two years of independence but owing to droughts, weakening terms of trade and high oil prices, the growth was not sustained (Muzondidya, 2009:169). In addition, government was under pressure from the IMF and World Bank that were opposed to government's socialist policies (Dhliwayo, 2001:3-6; Muzondidya, 2009:169).

As government faced mounting challenges of slow economic growth, rising unemployment, it also faced increasing pressure from the public regarding the racially unequal distribution of resources. Elite groups in both rural (including richer peasants and farmers) and urban sociaties benefited the most from post independent government poliy (Mandaza 1986:23). Poverty remained a problem in both urban and rural areas (see Table 3.6).

Table 3.5 Size and structure of the Labour force

	1982		1992		1995	
	thousands	%	thousands	%	thousands	%
Total Labour-force	2483	100.0	3502	100	4045	100
Employment	1177	47.4	1236	35.3	1250	30.9
Communal Farmers	1038	41.8	1502	42.9	1600	39.6
Unemployment	268	10.8	764	21.8	1195	29.5

Sources: Government of Zimbabwe (1988:1996)

Table 3.5 shows that employment in communal farming areas increased between 1982 and 1992 while overall unemployment doubled (from 10.8% in 1982 to 21.8% in 1992) in the same period. The increase in employment in communal farming areas is likely because those losing their jobs in the urban areas migrated to communal farm lands. This would probably would have put them into poverty.

Table 3.6 Poverty in Zimbabwe in 1990

Geographic region	History	Total population (1990)	Percentage households below poverty line (1990)
Rural areas		7,720,000	31 %
Communal Farm Areas	Created by pre-independence policies which moved small scale farmers to communally owned land areas as commercial farms expanded	5,550,000	33 %
Resettlement Areas	Created by limited post independence redistribution of land bought by the government from commercial farmers and used to resettle landless households	370,000	41 %
Large-Scale Commercial Farms	Result of pre and post independence commercial farm activity (tobacco, sugar, cattle, soya, maize) which concentrated wage labour in camps around these farms	1,800,000	16 %
Urban areas		3,080,000	10 %
Country		10,800,000	25 %

Source: World Bank, 1995a. Vol II. Table 2.2 p. 27 and Table 4.1 p.83[19]

Although the data shown in Table 3.6 cannot be held as evidence of this claim, it does show

[19] based on the 1990 Census and 1990-91 ICES

that by 1990, communal farm areas and resettlement populations had much larger populations than urban areas yet they had the highest proportions of households below the poverty line.

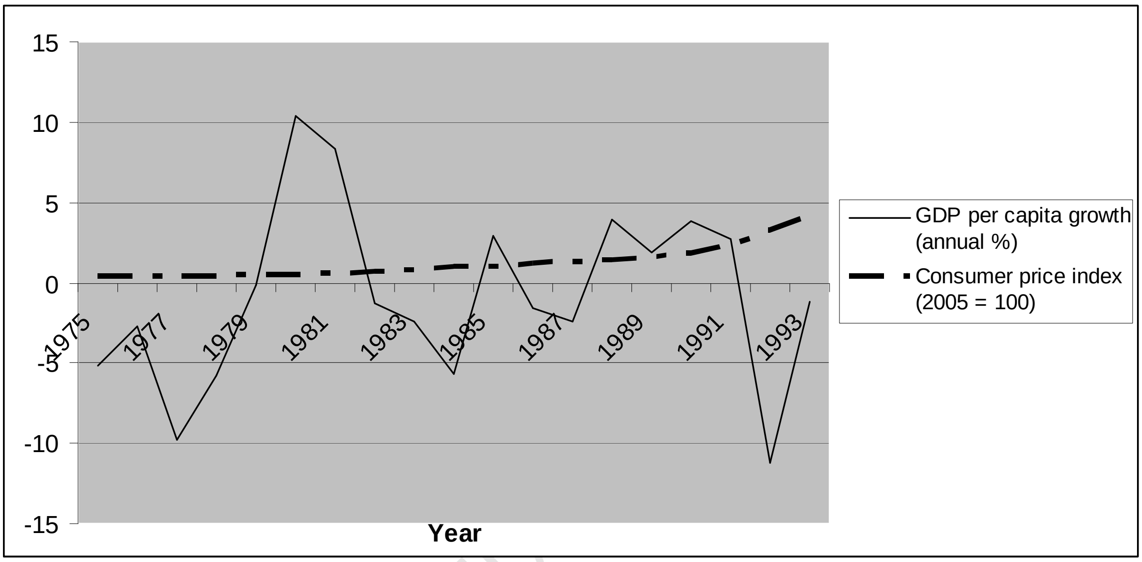
Poverty was mainly centred in provinces or regions such as Matabeleland North that were/are predominantly rural. While Harare and Bulawayo were the most urbanised provinces, Midlands, Manicaland and Masvingo were semi urbanised. Most of the other 6 provinces including Matabeleland North, Matabeleland South, Mashonaland Central, Mashonaland East and West are predominantly rural. These provinces have more people living in communal lands and to a less extent, resettlement areas (see Annex B and Table B.1 for more details).

However, in the urban areas many were not satisfied with government's policies and the black middle class faced difficulty in securing loans from white and foreign owned banks and so could not start their own enterprises (Maphosa, 1998:176-8). The main reasons suggested for the lack of access of the indigenous people to credit were lack of collateral security and institutional racism although the low levels of financial literacy also contributed (Maphosa, 1998: 181). While the latter reason is harder to prove from an empirical perspective, the lack of collateral security stemed directly from past colonial injustuces.

In 1989, a report on black economic empowerement revealed the following racial distribution of jobs in the private sector; senior management: 62.5% white, 37.5% black; middle management: 35.5% white, 64.5% black; junior management; 22% white, 78% black (Raftopolous, 1996:6).The rapid Africanization of the economy in the early years of independence took place predominantly in the public sector where government had direct control (Muzondidya, 2009:169). The 1980s saw little structural change in the economy which remained controlled by British and South African based multinational corporations (Stoneman, 1988:54-5; Economist Intelligence Unit, 1981:87). In 1985, 90% of the mining and 48% of the manufacturing industries were foreign owned (Burdette et al, 1987:78).

The urban black middle class grew increasingly impatient with the lack of opportunities owing to both a low growth in private sector employment and difficulties in accessing credit. The discontent was however not confined to the urban middle class. The slow pace of the land reform program also meant that by 1990, much of the rural black population remained below the poverty line (Muzondidya, 2009:172; Moyo, 2000:13) (see Table 3.6). After ten years of independence, government had acquired 3.5m hectares of land under the willing buyer willing seller approach and had only resettled 52,000 out of the 162,000 families it initially targeted (Moyo, 2000: 13). Of this acquired land, only 19% was prime arable land with good rainfall (Moyo, 2000: 13). Table 3.6 shows that in 1990, not only were too few people (370,000) resettled but about two fifths of these households lived in below the poverty line.

Figure 3.2 GDP per capita and CPI in Zimbabwe (1975-1993)



Source: World Bank Databank (2010)

In summary, (and as shown in Figure 3.2), the first two years of independence in Zimbabwe were characterized by a huge initial economic boom (as shown by Figures 3.1 and 3.2), which was then followed by mixed economic fortunes in the late 1980s. In addition, issues regarding the economic empowerment of previously disadvantaged groups notably, access to white collar private sector employment and capacity to start viable businesses were not seriously addressed in the first years of independence, as there was little political pressure (the economy performed well) on the government at a time (Muzondidya, 2009:174).

In 1982, Kaplan wrote, “whites, acknowledging their loss of political primacy, have focused on maintaining their economic status but have made few attempts to accommodate themselves to a changing social order” (Kaplan, 1991:16). Whether or not this statement was based on fact is of little consequence. What is important is that government did believe that after the political emancipation of the black majority in 1980, the next struggle was for the economic emancipation to be realized. Resultantly the next decade (1990-2000) was to be dominated by government's struggle to address racial economic imbalances in the face of massive resistance (perceived or otherwise) from the white community as well as consolidation of power in the wake of an increase in political and social activism (Raftopolous et al 2009:170-192; Muzondidya, 2009:174). This struggle manifested in a series of disastrous economic policies that led to the collapse of health and education infrastructure at a time when the AIDS epidemic was picking up steam across the country.

3.3 Economic Structural Adjustment Program (ESAP)

As a condition for financial aid from the Breton Woods institutions, in 1990 the government adopted the Economic Structural Adjustment Program (ESAP) (Dhliwayo, 2001:3; Raftopolous, 2009:189). In exchange for funding, the government had to privatise all government parastatals. ESAP was in full swing by the end of 1991 and the program aimed to abolish all subsidies and promote free markets (Dhliwayo, 2001:30). To sell the policy to the public, the government promised to reduce unemployment, bureaucratic red tape and facilitate increases in productivity (Saunders, 1996:3-5).

Instead, what happened were social service cutbacks. Local companies were put under intense pressure from imports (clothing and textiles, beverages and motor vehicle assembly) (Saunders, 1996:2). Government could not subsidise producers so prices started rising (Muzondidya, 2009:189). The downsizing of parastatals and industry caused massive unemployment, which rose from 32.2% in 1990 to 44% in 1993 (Mlambo, 1997:91). Government statistics released in 1994 showed that 20,710 workers had lost their jobs since the start of ESAP and the Zimbabwe Congress of Trade Unions (ZCTU) put this number at over 30,000 (Mlambo, 1997:91). The economy grew a paltry 1% in real terms during the ESAP period (1991-1995) compared to 4% during the pre ESAP period (1985-90) (Mlambo, 1997:90-91) (see Figure 2). Table 3.7 shows how the percentage growth in paid employment went into negative territory between 1991 and 1992.

Table 3.7 Paid employment (in 000's) by gender

Year	Men	(%)	Women	(%)	Total	% Growth
1986	889.8	82.3	191.2	17.7	1081.0	-
1987	889.6	82.0	195.5	18.0	1085.4	0.4
1988	929.5	82.2	201.6	17.8	1131.4	4.2
1989	957.0	82.0	210.1	18.0	1167.1	3.2
1990	977.6	82.0	214.5	18.0	1192.2	2.2
1991	1015.8	77.5	228.2	22.5	1244.0	4.3
1992	1012.5	77.7	223.8	22.3	1236.2	-0.6
1993	997.6	80.4	242.7	19.6	1240.3	0.3
1994	1018.0	80.0	252.2	20.0	1270.2	2.4

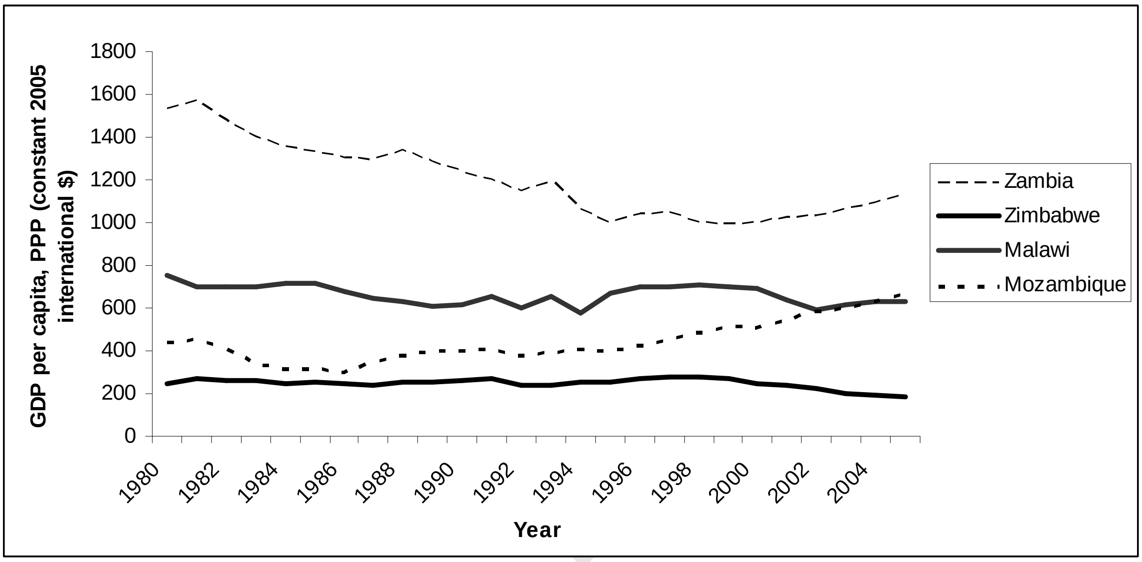
Source: ILO Yearbook of Labour Statistics, 1996

The ESAP had a negative impact on almost every sector of the economy. The manufacturing production index which stood at 131 in 1989, 138 in 1990 and 143 in 1991 (1980=100) fell to 130 in 1992 (Gibbon, 1995:9). Domestic consumer spending also fell between 25-30% in the first six months of 1992. Zimbabwe's trade balance was also negatively impacted by ESAP and while exports fell from USD 1785 million in 1991 to 1543 million in 1992 and imports rose from USD 1700 million to 1782 million (Gibbon, 1995:9). This was mainly because of an increase in commercial grain imports. Industrial input imports actually collapsed during this period as the country's currency weakened.

Some of the reasons put forward for the collapse in export capacity include high interest rates, credit restrictions, the phased removal of export incentives, and the tight monetary policies implemented by the central bank. The same issues also affected non exporting companies and the decline in the economy affected government revenues which for example, were 12.6 % below those forecast for the first half of 1992-93(Gibbon, 1995:9). The fall in revenue resulted in increases in the budget deficit. Compared to government's target of 5%, deficits stood at around 11% in 1990-1991, 10% in 1991-1992 and 11 % cent in 1992-93 (Gibbon, 1995:9).

Figure 3.3 however shows that countries like Zambia which adopted structural adjustment programs in 1991 also had negative economic performance exacerbated by several droughts and falling copper production. Between 1991 and 1995, real per capita GDP in Zambia fell by more than 20% only to pick up (with real GDP expanding by 6.5%) in 1996 due in part the near doubling of the maize that year (IMF and World Bank, 1999:2). However, the economies (measured by GDP per capita) of Mozambique and Malawi did not experience similar recessions in the 1991-1994 period[20].

Figure 3.3 GDP per capita, PPP Regional comparison



Source: World Bank (2010)

In Zimbabwe, when ESAP was in place (1991-5), indicators of the quality of government services also declined. The economic decline of the 1990s reversed the gains made in the provision of social services during the 1980s (Muzondidya, 2009:174). As shown in Table 3.9, GDP per capita fell a whopping 11% between 1991 and 1992. Two of the most important sectors that deteriorated rapidly were health and education. The number of nurses in government service per 1,000 population fell by 10% between 1991 and 1992 and government salaries (measured in U.S dollars) in the health sector fell by 26% (from 4,415 to 3,259) as shown (Central Statistical Office, 1996).

[20] Malawi for instance implemented its structural adjustment programs in 1981 whereas Mozambique implemented these in 1987 (Munthali, 2004: IMF and World Bank, 1999:1-3)

Table 3.8 Annual Earnings (USD) Per Employee

Year	Health	Education
1990	4,321	4,934
1991	3,641	4,415
1992	2,742	3,259
1993	2,330	2,725
1994	2,183	2,386
1995	2,546	2,516
1996	2,408	2,249

Source: Central Statistical Office, 1985-1996, 1985-1996 National Accounts

As shown in Table 3.1, real per capita public expenditure on health care declined by 38% (from 57.72 to 35.86) between 1990 and 1996. This decrease implied diminished spending on common drugs, extension and preventative health services, specialist facilities and treatment, and other components of quality health care delivery (Dhliwayo, 2001:4-9). Access to health services was further restricted by government's stricter enforcement of a user fees system, which erected barriers to health care for poorer social groups (Saunders, 1996:2).

It has also been reported that the situation in public hospitals became so dire that as early as 1992, doctors and nurses began referring to “ESAP deaths” (Saunders, 1996:2). These are deaths that were caused by the inability of patients to pay for the minimal length of time in hospital, or for prescription medicine. The next chapter will use the results from the modelling done in Chapter 2 to explore the extent to which actual AIDS deaths increased during this period and how they would have affected the progression of the epidemic. In short, a decrease in the population infected beginning in 1991 would have resulted in new infections slowing down and might explain the tapering off of HIV prevalence (around 1995, 1996 and 1997) that was observed in Figure 1.1.

The fact that Zimbabwe had one of its worst droughts in the 1991-1992 meant that the introduction of ESAP could not have been timed more disastrously. There were interactive as well as additive effects between ESAP and the drought (Marquette, 1997:1145). The drought resulted in widespread crop failure, declines in agricultural production and rising food prices. Resultantly, government could not immediately lift many of the food subsidies on staple foods such as maize meal, bread and sugar. However, a successful 1992-1993 growing season, allowed government to remove these subsidies leading to the real price of staples such as bread and sugar rising by 40% and 50% respectively (Minot, 1994:6)

In the field of education, government also cut spending between 1990 and 1996 as shown in Table 3.2. In the primary education sector in particular, real per capita spending and average spending per pupil fell to the lowest levels since independence (Dhliwayo, 2001:4-9). ESAP not only compromised the quality of education but its imposition of user fees barred access to education from hundreds of thousands of students from poorer backgrounds (Saunders, 1996:2-3).

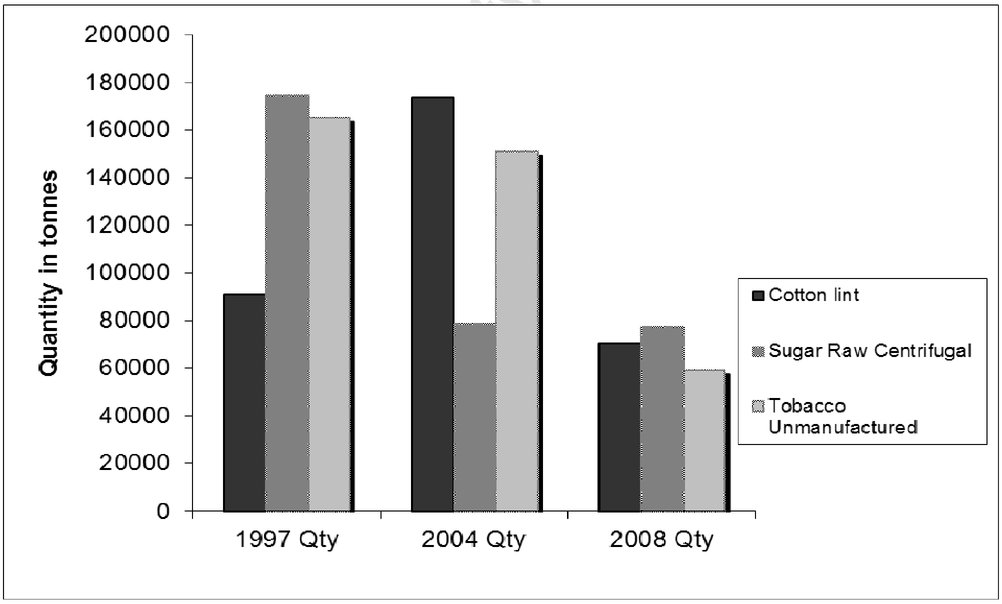
By the mid-1990s, the effects of ESAP had pushed pressure groups to the limit. The discontent had already been voiced as early as 1992, with a relatively poorly attended march against ESAP that was organised by the Zimbabwe Congress of Trade Unions (ZCTU) (Saunders, 2001:135-40). The protests were brutally squashed by the police and the relevance of the ZCTU and its ability to mobilize workers came into question (Raftopolous, 2001:11). The trade unions however combined with student groups, human rights organisations churches and government workers in the June 1996 public-sector strike which was largely successful (Muzondidya, 2009:194; Raftopolous, 2001:10-11). The biggest general strike (both private and public sector) came in December 1997 and it brought the whole economy to a stop (Muzondidya, 2009:194). Facing the real threat of losing power, the government adopted survivalist policies that further decayed the economy.

3.4 Politics of patronage

To deal with growing opposition from inside and outside the party, the government turned to the war veterans i.e. (those who had fought in the war of liberation in ZANU or ZAPU) and members of the security services (police, military, intelligence). Ndlovu-Gatsheni (2006) explains that the ruling party and the military had always maintained close ties. War veterans and the ruling party relied on violence and appeals to a liberation war discourse to establish their power and privilege in the army, the police and among other workers (Kriger, 2003:103). However, the veterans did not always agree with government policy and many in government feared the power of a united and organized veterans group (Chung, 2005:302).

To regain control and appease the electorate, government abandoned the inclusive nationalism of the early period and began to adopt a radical exclusive nationalist stance where it was primarily those aligned to the ruling party who benefited (Alexander, 2006:180-90). Those who had fought in the liberation struggle felt betrayed by government because they were both jobless and did not have good farming land (Muzondidya, 2009:190-192). To appease this constituency, Mugabe promised to allocate them some of the 1,471 large scale white owned commercial farms the government had identified for expropriation in 1997 under the Land Acquisition Act of 1992 (EISA, 2009)[21]. The beginning of land expropriations signalled the end to respect of property rights in Zimbabwe and made it one of the riskiest investment destinations. In the long term, the chaotic land reform program turned the country from being an exporter of many food crops including maize to being a net importer. Tobacco exports, which had been a main foreign currency earner also declined sharply from 164, 980 tonnes in 1997 to 59,103 tonnes to in 2008 (FAO, 2010). This is shown in Figure 3.4.

Figure 3.4 Top Agricultural Exports



Source: FAO (2010)

[21] EISA, The land issue in Zimbabwe. See http://www.eisa.org.za/WEP/zimland2.htm. [Accessed 24 January, 2009].

The shrinking of the agricultural sector output (since the late 1990s) by more than 50% resulted food shortages and increases in food prices which fed into the subsequent hyperinflation which eventually crippled the economy (New Agriculturalist, 2008).

Table 3.9 Zimbabwe Economic data

Year	Real GDP Per Capita Growth	Consumer Price Inflation	Lending Rate	Real interest Rate
1990	3.7	15.5	11.7	-5.6
1991	3.8	46.5	15.5	-8.5
1992	-11.2	46.3	19.7	-21.8
1993	-1.4	18.6	36.6	8.1
1994	2.3	21.1	34.8	12.6
1995	-3.1	25.8	34.7	12.2
1996	6.2	16.4	34.2	12.6
1997	2.4	20.1	32.5	13.7
1998	0.4	46.7	42	10.7

Sources: International Monetary Fund, World Economic Outlook database[22]

The politics of patronage was cemented with government's decision to pay unbudgeted gratuities of Z$50,000 (US$4,500 at 1997 prices) and monthly pensions of Z$2,000 (USD $ 180 at 1997 prices) to disgruntled war veterans (Muzondidya, 2009:198)[23]. Table 3.9 shows the relationship between real GDP growth, inflation, lending and the real interest rate in Zimbabwe between 1990 and 2007. The doubling of inflation in the period, 1997 to 1998 was partly because of a bloated budget as well as general fiscal mismanagement (Hanke, 2008:908-914).

Government had instituted (between 1996 and 2000) the Zimbabwe Programme for Economic Transformation (ZIMPREST) that tried to focus more on empowerment, indigenisation of the economy and land reform (Dhliwayo, 2001:2). In essence, it tried to undo some of the harm caused by the ESAP and earlier transformation policies. However, government spread itself too thin by also involving itself in the civil war in the Democratic Republic of the Congo

[22] Accessed on April 2008, http://www.imf.org/external.

[23] Each war veteran would get Z$50 000 cash and a tax-free monthly pension of Z$2 000. (Initially there were estimated to be around 32 000 claimants but this figure was eventually initiated to over 70 000). The program would cost a total of Z$5, 3 billion and put the annual budget deficit at 12.5% of GDP billion instead of the targeted 9.8%.

further worsening the budget deficit (Raftopolous, 2009:219). Some have questioned the motives behind the move especially given the reports of rampant looting of Congolese mineral wealth by Zimbabwean generals and Ministry of Defence officials (Kisangani, 2003: UN Security Council Report, 2002; The Insider, 2002). The Congo war was another opportunity that Mugabe used to ensure continued support from the security establishment.

The economy continued to decline during the ZIMPREST (see Tables 3.9 and 3.10). The expropriation of land, beginning in 1997 was done in such a corrupt manner that the country's once prosperous agricultural sector was almost completely destroyed (USDS, 2009). In its early years, land expropriation was more corrupt than it was violent as senior government official underutilized the land they had been allocated. Within a year, the country started feeling the effects of declining agricultural production and unprecedented food riots erupted (Raftopolus, 2009:202-204; Shaw, 2003). The riots, organised by the ZCTU, were in response to rising maize meal prices and a general shortage of the staple food (Shaw, 2003).

3.5 Zimbabwe's socio-political climate (from 1998-2009)

After the 1998 riots, the ZCTU became more vocal and by the end of 1999, its Secretary-General, Morgan Tsvangirai together with leaders from the National Constitutional Assembly (NCA) and other civic groups formed the Movement for Democratic Change (MDC). White commercial farmers were an important source of support in the 2000 general election for the MDC as they helped mobilize the rural population (their farm workers) (Selby, 2006:277; Raftoplous, 2009:210). To garner support for the 2000 parliamentary elections, the government encouraged war veterans to invade and occupy more white owned farms (Kriger, 2003:2-4). Violence erupted and Zanu PF won a widely contested and narrow general election in 2000 in which Mugabe received 56 per cent to Morgan Tsvangirai's 42 per cent of the presidential vote (Raftopolous, 2009:215 Moore, 2004:405-26).

The strength of the opposition caught the government off guard and they began crafting a repressive order to restrict the activities of the opposition and civic forces in the public sphere, including controlling the private press (Raftopolous, 2009:214). In 2002, laws including the Public Order and Security Act (POSA) [Chapter 11:17] and the Access to

Information and Protection of Privacy Act (AIPPA) [Chapter 11:17] (McGregor, 2002:9-37), were passed. In July of 2004, government also tried to pass the Non-governmental Organizations Bill which aimed to criminalize NGOs whose objectives include "issues of governance", or "the promotion and protection of human rights" (CRS, 2005:6). The laws were the basis of yet another violent and fraudulent victory at the polls for ZANU PF in the 2005 general elections in which ZANU PF secured a two thirds majority in parliament [24](Scarcecchia, 2006:221-37). This allowed ZANU PF the ability to amend the constitution without opposition support.

Despite its victory, government subsequently carried out a massive onslaught against the urban poor in May 2005 under Operation Murambatsvina ('drive out filth') which displaced an estimated 650,000-700,000 from their homes in cities across Zimbabwe (United Nations, 2005:2-5). The government demolished people's homes in high density areas and townships in Zimbabwe's main cities and moved them into transit camps, after which they assigned people to rural areas on the basis of their identity numbers (Pearce, 2011). Brian Raftopolus and others argue that the operation was meant to punish those who had supported the MDC in the 2005 elections and effectively force them to return to their rural homes (Raftopolus, 2009:221; Potts, 2006:273-91). The Catholic Archbishop of Bulawayo, Pius Ncube was quoted as saying

"*They want total political control - they want to peasantify people like [former Cambodian leader] Pol Pot - force them into the country so they can control them……………………… In the countryside they have no newspaper or radio except Zanu-PF propaganda, and they are controlled by the chiefs, who support the government …*,"

The operation had the real effect of further impoverishing those who already lived in rural areas and were dependent on remittances from their urban relatives (Raftopolus, 2009:220).

The rapid decline of the Zimbabwean economy from the late 1990s to 2008 has been attributed to two main causes (Bond, 2007:149-81; Davies, 2004:19-42). The first is that the long-term legacies of colonial resource inequalities and narrow forms of capital accumulation failed to build a broader productive base (Raftopolous, 2009:219; Davies, 2004:19-42). This resulted in the welfarist policies that characterised the 1980s and the neoliberal policies and

[24] This allowed ZANU PF the ability to amend the constitution without opposition support.

structural adjustment programme of the 1990s (Davies, 2004:19-42). The second cause stems from a combination of the political “threat” around the land reform as well as the ruling party’s patronage culture (Bond, 2007:149-81). The deterioration of Zimbabwe’s political and economic environment coincided with decline in HIV prevalence which began in 1998 after peaking in 1997 at 28.9% (see Figure 1.1). A more rigorous discuss of this link is presented in the next two chapters.

Table 3.10 Zimbabwe Economic data

Year	Real GDP Per Capita Growth	Consumer Price Inflation	Lending Rate	Real interest Rate
1998	0.4	46.7	42	10.7
1999	-3.3	56.9	55.3	-2.6
2000	-7.0	55.2	68.2	12.6
2001	-2.4	112.1	38	-35.3
2002	-4.1	198.9	36.4	-96.7
2003	-11.3	598.7	97.2	-267.7
2004	-3.3	132.7	278.9	-71
2005	-4.0	585.8	235.6	-2.1
2006	-5.4	1,281.1	496.4	-250.2
2007	-6.1	108,844.1	N/A	N/A
2008[2]	N/A	230 million	N/A	N/A

Sources: International Monetary Fund, World Economic Outlook database[25]

The IMF real GDP per capita data shown in Table 3.10 shows that Zimbabwe’s economy has been shrinking since 1999. As of 2006, GDP per capita was 47% lower than its 1980 level (see Table 3.10) (Robinson, 2007:4). Formal sector incomes also fell and at the end of December 2006, the average minimum wage for agricultural and domestic workers of Z$2,800 was only 3 per cent of the Food Datum Line (Raftopolous, 2009:219). Formal sector employment also declined from 1.4 million in 1998 to 998,000 in 2004 (Kanyeze, 2007:31-5). The Fast Track land reform program led to gross underutilisation of land, vandalism of agricultural infrastructure and huge declines in maize production (Matondi, 2008:13; Raftopolus, 2009;217). Mugabe maintained support in the ruling party and government by

[25] Accessed on April 2008, http://www.imf.org/external.

giving the best farms to officials in the ZANU PF, military and civil-service hierarchy (Raftopolous, 2009:218).

The current Reserve Bank Governor, Gideon Gono took office in 2003 when inflation stood at 599 per cent (see Table 3.10) and through printing money extended the ruling party's politics of patronage resulting in inflation reaching over 7,000 per cent in 2007 (Robinson, 2007:6). He took over economic policy-making not just in the recognised areas of responsibility of a central bank (monetary and exchange rate policy) but across the spectrum of fiscal and sectoral policies including food and farming input procurement (Robinson, 2007:6; Hanke, 2008; 1-2). At this point patronage became institutionalized and Gono used reserve bank funds at his discretion (Hanke, 2008; 2-4). This is exemplified by how, towards the end of 2007, he bought a fleet of new vehicles, generators, sets of televisions, and full sets of satellite dishes for the sitting judges to "improve their conditions of service" (The Zimbabwe Times, 2008).

The government under ZANU PF developed a track record of suppressing health information as a way to manage the political crisis (PHR, 2009:21). As already mentioned, this was facilitated by the Access to Information and Protection of Privacy Act (AIPPA) (McGregor, 2002:9-37). A classic example of this is government's public denial of the cholera epidemic that began in August of 2008. The erratic water supplies, shortages of water purification chemicals, broken water and sewer pipes and uncollected garbage in most parts of the country exacerbated the epidemic and by the end of the year, it had affected an estimated 60,000 people and killed 600 countrywide (Newsweek, 2008).

It was at this time that the then Minister of Information and Publicity, Sikhanyiso Ndlovu, reportedly ordered government-controlled media to downplay the cholera epidemic, which he said had afforded

"*... the country's enemies a chance to exert more pressure on President Robert Mugabe to leave office*" (Radio Voice of the People, 2008).

The minister was also reported to have ordered the media to turn a blind eye to the number of people who had died or [had become] infected with cholera, and instead focus on what the Government and NGOs were doing to contain the epidemic (PHR, 2009:21). Government's control of information facilitated by AIPPA makes it difficult to carry out a detailed analysis of the decline in the health-care system from 1997 to 2009. It also shows the ZANU PF government's paranoid distrust of western governments as well as its unwillingness to work with donors and NGOs.

Between 1998 and 2008, the health delivery system nearly came to a virtual standstill. Even if nurses and doctors wanted to go to work, the transport costs were prohibitive (MSF, 2009:3-4). The shortage of staffing at hospitals was also worsened by the massive exodus of Zimbabwe's health professionals. As the chairperson of the Zimbabwe Association of Doctors for Human Rights (ZADHR) remarked in 2008,
"*Zimbabwe now has just one doctor for 12 000 people. There are only 800 doctors registered in the country.......doctors are leaving on a weekly basis and nobody can blame them*" (Meldrum, 2008:1059-1060).

Only 25% of the 425 doctor posts in the state medical system in 2008 were filled, according to official figures (Meldrum, 2008:1059-1060). The dollarization of the economy (i.e. adoption of multiple currencies, primarily the U.S dollar as the official currency), in 2009 further worsened the plight of Zimbabweans as it made access to health care impossible for most (PHR, 2009:21-22).

Hospitals were frequently closed not only because of a shortage of staff but also on account of lack of running water and inadequate sanitation (PHR, 2009:21). NGOs however tried to step in albeit under intense monitoring by government (PHR, 2009:21-22). NGOs and donors were officially banned by the government in June of 2008 at a great cost to ordinary people who depended on them for food and medicines (NANGO, 2008). They were accused of campaigning against the government and using food and medicines to buy votes for the MDC (Zimbabwe Independent, 2008). Government however quickly moved to relax the ban on NGOs who worked with HIV/AIDS, children, the disabled and the elderly (IRIN, 2009).

The onslaught on the country's health system and support structures such as NGOs would have led to an increase in AIDS deaths. The next chapters will attempt to see if the rapid decline in HIV prevalence that characterized this turbulent period can be linked to increases in AIDS deaths. The aim would be see what proportion of the decrease in HIV prevalence can be attributed to an increase in AIDS mortality versus that which is attributable to decreases in new infections as a result of a reduction in risky behavioural practices.

The 2008 elections took place in the context of SADC brokered mediation framework led by Thabo Mbeki (Raftopolus, 2009; 227). Mbeki's "quiet diplomacy" was focused on creating conditions for a broadly acceptable election that would lead to a normalization of the Zimbabwean situation (Mbeki, 2007). The elections took place with little violence but failed to deliver an outright (with 50% plus one votes) winner with Mugabe polling 43.2 per cent versus Tsvangirai's 47.9 per cent (CBS News, 2008; Raftopolus, 2009; 229). This meant a runoff or second round of elections had to be carried out to decide the winner.

The period between the election and the runoff election was one of the most violent in Zimbabwean history since Gukurahundi and was again directed by the security apparatus under the Joint Operations Command (Alexander et al, 2008:80; Solidarity Peace Trust, 2008). Mugabe won the runoff after Tsvangirai withdrew from the elections and Mbeki began another mediation process which culminated a Global Political Agreement between ZANU and the MDC in September of 2008 [26](Raftopolus, 2009; 229). The agreement set the parameters for the formation of a Government of National Unity which has been in power since January 2009 (Raftopolus, 2009; 229).

The new government faces the difficult task of uniting the country as well reversing the decline of the Zimbabwean economy over the past 12 years. This includes attracting back all the skilled labour that left the economy as a result of the economic and social crises of the past 12 years. Although the out-migrations may actually have contributed (albeit to a less extent than the decrease in new infections and the increase in AIDS deaths) to the decline in HIV prevalence over the past decade as shown in Chapter 2, reversing the brain drain will help Zimbabwe rebound of its economic slump.

[26] The MDC had split into two factions namely MDC-T and MDC-M led by Tsvangirai and Arthur Mutambara respectively.

3.6 Conclusion

The development of social and economic policy in Zimbabwe can be best understood as having happened in three phases. The first was characterized by an early economic boom (1980-1982), which created a foundation for the expansion of most social services including health care and education. Government instituted redistributive policies although these began losing momentum between 1982 and 1986 as two recessions limited government's ability to spend. The later part of the 1980s was characterized by further economic prosperity although the conflict between Zanu PF and PF Zapu threatened to dampen economic growth. It was on back of this relative prosperity that the HIV epidemic emerged, with HIV prevalence continuing to rise until the late 1990s.

The second phase began with the disastrous Economic Structural Adjustment Program which resulted in cuts in government spending on health care and education. The removal of government subsidies in various sectors put local companies under pressure from imports. The clothing and textiles, beverages and motor vehicle assembly industries were hardest hit. This also caused unemployment rates to begin rising and the downsizing of parastatals further increased the jobless rate. The period was also characterized by Zimbabwe's worst drought in decades and many, particularly those in rural areas faced starvation. Political opposition to the liberation government also gained steam and by the late 1990s, countless strikes by labour movements as well as civil society groups had shaken government's hold on power.

The third phase of the development of social and economic policy in Zimbabwe was characterized by Zanu PF's survivalist policies, patronage and corruption, all leading to what has been termed the "Zimbabwe Crisis". During this phase, a series of disastrous policies including the expropriation of land, repressive laws such as the Public Order and Security Act and the Access to Information and Protection of Privacy Act, war veterans' gratuity payments and Operation Murambatsvina all lead to a virtual collapse of the country's health, education and economic system. The economic hardship coupled with the spats of violence that flared up during election periods led to massive exodus of professionals and non-professionals alike to neighbouring countries as well as the United States of America and the United Kingdom.

Despite the empirics being weak on the matter, Zimbabwe's socio-economic decline of probably occurred in a disparate manner. Policies such as Murambatsvina that resulted in massive relocation of the urban poor to communal and resettlement areas had an effect of increasing the population of provinces such as Matabeleland North, Matabeleland South, Mashonaland Central, Mashonaland East and West were average earnings have historically been lower than in Harare and Bulawayo.

Chapter 4 looks at why new infections increased so rapidly between 1984 and 1997. Arguments that there was widespread denial (within government and communities in general will be explored. The timeliness and appropriateness of government's will also be analysed. The decline in HIV prevalence post 1997 will be interrogated with aim of linking Zimbabwe's socio-economic decline and political instability to the reduction in new infections, increase in AIDS deaths and out-migrations. The decline in health, communication and transport infrastructure is central to this thesis. This discussion further illustrates the importance of understand the sub-epidemics in each country when interpreting HIV prevalence and incidence estimates.

4 EXPLAINING THE TRAJECTORY OF ZIMBABWE'S HIV EPIDEMIC

Abstract

In this chapter I argue that in addition to the economic crises discussed in Chapter 3, the Zimbabwe government's response to the HIV epidemic was delayed by a combination of denial and a desire to control any interventions (such as educational, media campaigns and condom distribution). The eventual multi-sectoral effort to fight the epidemic was late in coming and Zimbabwe fared poorly compared to Uganda. I also argue that available evidence accords with the demographic modelling presented in Chapter 2; i.e. that the decline in HIV prevalence was primarily due to a reduction in new infections. Secondary factors contributing to the decline include migration and AIDS mortality. The socio economic decline had an impact on all of these three factors and each of the provinces had unique experiences particularly regarding changes in condom use, sexual onset and sexual concurrency. Structural factors also probably played a role in the progression of the epidemic and provinces such as Matabeleland North which are sparsely populated, have poor communication and transport infrastructure and economically disadvantaged (making it hard to maintain sexual networks) had lower peaking epidemics. All of these lessons have implications for how national HIV prevalence estimates should be interpreted given that they are a combination of different regional sub-epidemics(as described in Chapter 2).

4.1 Introduction

As discussed in Chapter 3, Zimbabwe's socio-economic environment has been anything but stable over the past 15 years. The instability has not only undermined the effectiveness of HIV/AIDS intervention programs but it probably also had a significant impact on behaviour change, AIDS mortality and migration. These are the three factors which the EPP/Spectrum modelling exercise (Chapter 2) suggests were the dominant causes of the dramatic decline in HIV prevalence from almost 28.9% in the late 1990s to 14.3% in 2010.

In this chapter, I present a summary of government's HIV intervention programs including the Emergency Short Term Plan (STP), the First Medium Term Plan (MTP1) and others. I proceed to assess whether they were effective often highlighting differences between the Zimbabwean and Ugandan approaches to national HIV/AIDS policy. Having set a basic foundation on who did what in the HIV invention effort, I then look at available evidence about the factors that are most likely to have led to the dramatic fall in HIV prevalence in Zimbabwe.

I begin with a discussion of the impact of the AIDS intervention programs on behaviour change. Drawing on different empirical sources including the Demographic and Health Survey (DHS), this chapter investigates trends in sexual concurrency, condom use as well as early sexual debut. This analysis is done on both a national as well as provincial level. Looking at the provincial differences in behaviour change is a way to unmask some of the reasons why the epidemic followed different paths in different provinces/regions.

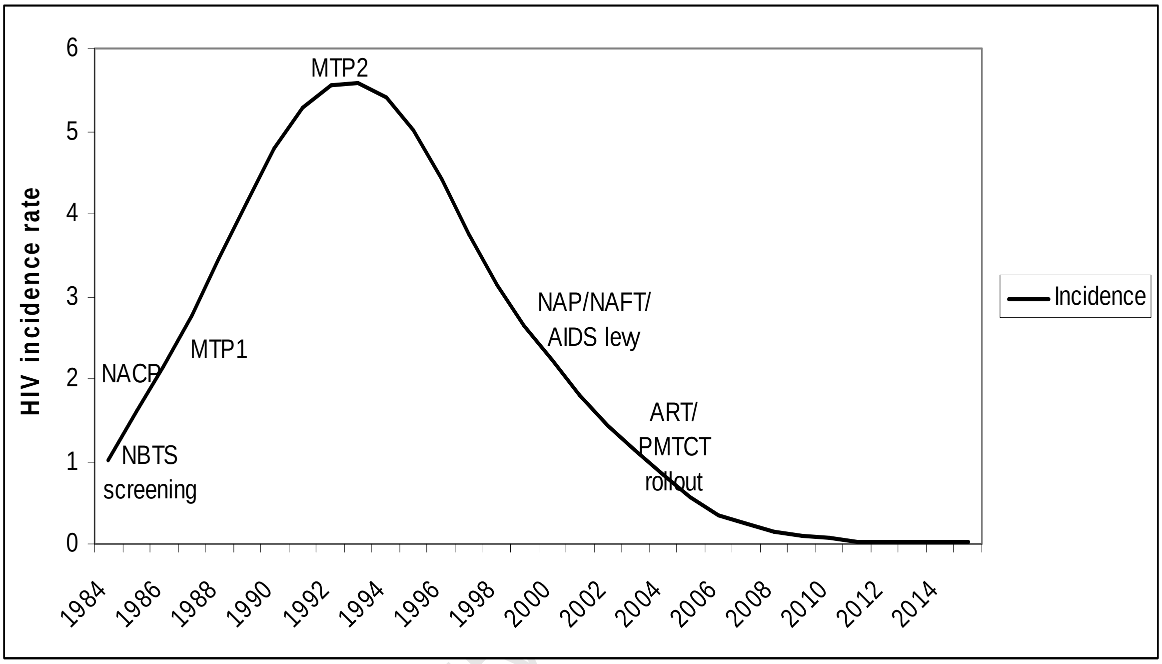
Finally, drawing from the incidence and prevalence modelling presented in Chapter 2 as well as the description of Zimbabwe's socio-economic decline in Chapter 3, lessons are drawn about how AIDS mortality and migration may have affected the trajectory of the HIV epidemic.

4.2 Government's Intervention

4.2.1 Government's early control of the response to the epidemic

One of the first official responses to the HIV epidemic was in 1985 when the Ministry of Health, through the National Blood Transfusion Services (NBTS) began focusing on intensifying the screening of donated blood and blood products (NAC, 2006:17). The move aimed at reducing the risk of infection via blood donations. Figure 4.1 shows various government policy intitiatives and situates them relative to the trend in the rate of new HIV infections.

Figure 4.1 HIV incidence and Intervention programs



Source: Spectrum modelling (Chapter 2)

In the epidemic’s early years, there seemed to be little evidence of a major drive to encourage sexual behavioural change (to reduce new infections). From the mid 1980s until around 1990 (a period when new infections increased), there was much denial by government about the existance of HIV/AIDS (Machaba-Hove, 2006:160; Rodriguez, 2003). Discussion of HIV and AIDS was minimal and President Mugabe rarely addressed the subject in public speeches (Avert, 2010a:3). In her article in Time magazine titled “Death Stalks a Continent,” Johanna McGeary bemoans the lack of leadership on the AIDS issue under the presidency of Robert Mugabe and how it aided the progression of the disease (McGeary, 2001).

Authors such as Alex de Waal have argued that some African governments managed the HIV/AIDS epidemic in a way that minimised political threats rather than confronting the epidemic head on (de Waal, 2006:3). De Waal suggests that this encouraged leader to implement policies aimed at managing the disease rather than resolving it (de Waal, 2006:3). This certainly appears to be the case in Zimbabwe in the early stages of the epidemic as there was no multi-sector plan involving non-governmental organisations and other stakeholders which probably would have been more effective at getting the message across to the people. In this regard, Zimbabwe's response was very different to that of Uganda's where the government reacted by taking a leading role in educating the population about safe sex and the risks of multiple sexual partnerships etc. (de Waal, 2006:118-120).

Uganda's widely praised first AIDS control programme was set up in 1987 and it was based on the ABC approach (abstain, be faithful, use condoms) (Avert, 2011:2). Although its main focus was on encouraging behaviour change, it also had many other components including the introduction of HIV surveillance and the monitoring of the safety of the blood supply (Avert, 2011:2). Fogelberg, (2005) argues that strong political will, as well as an inclusive approach to fighting the epidemic were the cornerstones of the intervention's success.

Museveni created a sense of nationalism, united the country and rallied popular support for the fight against the epidemic (Fogelberg, 2005:4). Under Museveni's leadership, government created a multi-sector approach involving, involving various sections of the media, schools, churches, local governance structures. It also created bodies such as the Uganda AIDS Commission (UAC) was formed in 1992 to coordinate and monitor the national AIDS strategy across all sectors (Hogle 2002:4). Multiple organisations were also created within different ministries and government also reached out to NGOs and community-based organizations (CBOs) Fogelberg (2005:5). CBOs are a special kind of NGO whose membership is comprised of individuals with similar characteristics e.g. HIV positive persons who come together to offer a local response to the epidemic Fogelberg (2005:5).

Murphy et al (2006:1) argue that policies to advance women's status were crucial to the ABCs' success. The passing of gender empowerment laws such as the constitutional amendment reserving a minimum of a third of all parliamentary seats to woman further bolstered women's efforts to fight the disease (Hogle, 2002:5-6). There were policy shifts not only at a national or constitutional level but local level greater financial and political support was given to social, economic, and political women's organizations (Tripp, 1998). A key aim of the ABC approach was to also empower women economically and this was a recognition that most economically insecure women had very limited control over their sexual relationships (Murpy et al, 2006:2). In addition to the creation of credit facilities for women aimed at giving them capital to improve their income and increase the financial independence, fees for primary education were scrapped, allowing for more young girls to finish school (Monico 2003:2).

School curricula were also changed to emphasize the dangers of transactional sex and other risky behaviour (Monico 2003:2). However, Murphy et al, (2006:2) argue that the reasons the ABC messages were remarkably successful is not just about the content of the messages themselves but more about the extensive social mobilization at every level and strong political leadership from its president. Although the effect of AIDS mortality should not be understated, the ABC campaign managed to change sexual behaviour significantly. Epstein argues that casual partner reduction was the major behavioural change during the HIV decline in Uganda during the 1990s (Epstein, 2009:3). Increasing and consistent use of condoms especially in causal relationships also contributed to the decline in prevalence (Epstein et al, 2005:3-4).

It is important to note that this brief reference to Uganda's experience is not exhaustive and that its aim is to provide a comparative lense through which Zimbabwe's experience may be viewed. Key differences between the two cases include the fact that Yoweri Museveni could have had more political space to be assertive since the HIV epidemic was exploding at the time he came into power via military means. This gave him "policy space" although the actual implementation of interventions would necessarily have to involve international development partners and NGOs on whose funding his government depended.

In the case of Zimbabwe, the epidemic exploded at a time when the economy was still doing relatively well (see previous chapters) with the political leadership not having to depend on development partners for help in crafting policy. A narration of the Zimbabwean response to the rise if the epidemic reveals a number of insights.

In Zimbabwe, the first major step in the fight against AIDS was the establishment of the National AIDS Control Programme (NACP) in 1987. The programme was domiciled in the Ministry of Health and it formulated a one year Emergency Short Term Plan (STP) that aimed at creating public awareness of HIV and AIDS. The STP was designed to be comprehensive and included the training of health personnel in the different aspects of HIV and AIDS interventions to include promotion of appropriate behaviour change (such as condom use) among targeted population groups (NAC, 2006:17).

There is little evidence to suggest that the STP and NACP's efforts were successful at stemming the increase in new infections (certainly the modelling done in Chapter 2 and presented in Figure 2.17 shows that new infections increased from 1985 to 1993). However it is also possible for the programs to have had some impact even though the country continued to experience rising HIV incidence. Regardless, it is likely that the effectiveness of the STP may have been limited by the government's inability to be open about the disease and to incorporate other stakeholders in the development of a solution. In stark contrast, Uganda has been praised for having introduced a timely policy framework that had strong government leadership, broad-based partnerships and effective public education campaigns all contributing to a decline in the number of people living with HIV and AIDS in the 1990s (Avert, 2011).

Zimbabwe's government attempted to maintain control and, as De Waal argues, the responses appeared to have been motivated more by the need to coordinate institutions (to maintain some control over any intervention) than to produce tangible outcomes (De Waal, 2006:3). The weak response by government could not have been more disastrously timed as it came at a time when the masses were increasingly agitated first by the failure of empowerment programs (in the 1980s as explained in the previous chapter) and later by the disastrous structural adjustment programs (ESAP).

Although it wasn't as comprehensive and timely as Uganda's ABC (abstain, be faithful, use condom) policy, Zimbabwe's STP sowed the seeds for the monitoring of the epidemic through epidemiological surveillance (NAC, 2006:17). The first antenatal clinic surveys were carried out (owing to pressure from WHO) in 1989 with the launching of the First Medium Term Plan (MTP1) (Mahomva et al, 2006:2; NAC, 2006). With the launching of the MTP1, government relinquished some control over the AIDS responses. The main aim of MTP1 was, " *to limit transmission of HIV and other sexually transmitted diseases, mitigate the medical and psychological effects of the epidemic and establish a multi-sectoral approach to programme implementation"* (NACP, 1998:65).

The MTP1 (1989-1993) was probably crippled by a lack of transparency and government's attempt to manage the epidemic rather than consult widely to nip it in the bud. The media was equally complacent, possibly owing to pressure from government although there is not enough evidence to conclude that government unduly influenced them (as discussed in the previous chapter, government only began its ruthless crackdown on the media in the 2000-2009 period). A study by Pitts et al (1993:223-230) investigated newspaper coverage in Zimbabwe on HIV/AIDS and related topics and revealed that in the late 1980s to early 1990s, the media gave little attention to the epidemic.

There are however, some indications that Government sought actively to suppress knowledge about the extent of the epidemic during the late 1980s by instructing doctors in public service not to mention AIDS as a cause of death on death certificates (Leshomo et al, 2007:139). A 1990 report in Parade Magazine accused government of suppressing AIDS data and misinforming the public (Houston, 1990, 5). It may have attempted to maintain control of the epidemic by playing down AIDS statistics as has been argued by Alan Whiteside (Whiteside, 2008:81). Given such attitudes on the part of government and the media towards the disease in its early stages, it is little wonder that nothing substantial was done to prevent the rise in HIV incidence and prevalence in the 1980s into the late 1990s.

The MTP1 and the STP did not prevent the rise in the rate of new infections and the UNGASS and MOHCW working group estimate that incidence (adults, 15-49) peaked nationally at 4.6% in 1993. This result was also shown in the EPP and Spectrum modelling done in the Chapter 2 (see Figure 2.17 for trends in incidence). Gregson et al (2008) estimated incidence in the cities of Bulawayo and Harare and found that incidence peaked between 1988 and 1990 at levels between 3% and 6% in Harare. The peak was slightly later in

Bulawayo (between 1992 and 1994) although the levels were comparable, with incidence rates between 3% and 6% (Gregson et al, 2008:5). Despite the formulation of various policies and projects, the fight against AIDS was viewed as being compromised by poor organisation and limited commitment from government (Avert 2010:2).

The Second Medium Term Plan (MTP2), which remained official policy from 1994 to 1998, succeeded the MTP1 (NAC, 2006:17). It was with the formation of the MTCP2 that government began consulting widely to formulate a more effective broad-based solution to the epidemic. The MTP2 aimed at creating strategies to allow for the mobilization of non-health sectors to integrate HIV and AIDS issues for example, requiring the military to implement education programs to rank and file. The policy helped give impetus to efforts focused on reducing the transmission of HIV and other STIs in the military (Machaba-Hove, 2006:160). It appears to have been a long overdue admission by government that it could not fight the epidemic on its own and that it had to incorporate non-governmental stakeholders in the formation of policy. As shown by Figure 4.1, the launching of the MTP1 coincided with the beginning of the decline in the rate of new infections but whether it was crucial in assisting the decline or whether this was just a fortunate coincidence remains unclear

4.2.2 National AIDS policy (1999)

A significant step in the fight against AIDS was the belated formulation of the National AIDS Policy (1999) (Rodriguez, 2003:1; Avert, 2010a:2; NAC, 2006:17). This was after a widely consultative process that involved stakeholders such as donors and non-governmental organisations. With the creation of the NAC, government introduced the National AIDS Trust Fund (NATF) or “AIDS levy” which was collected through a 3% tax on all taxable income nationally (NAC, 2006:17). The NAC was given the responsibility of allocating resources for HIV/AIDS (including the AIDS levy) and tasked with the coordination of AIDS programs funded through government and donor agencies such as the Global Fund (NAC, 2008; Avert, 2010a).

The decentralisation of the AIDS response came with the creation of structures with a focus on district service delivery. The NAC has the responsibility of disbursing funds to local structures for implementation of HIV and AIDS interventions (UNGASS, 2008:12). This is done through decentralised local coordinating structures called AIDS Action Committees and they are represented in all the administrative levels including provincial, district, ward and village levels (UNGASS, 2008:11-13)

However, increasingly sceptical of Zimbabwe's management of the AIDS epidemic, international donors either withdrew their funding from government institutions such as the NAC, or channelled funding direct to non-governmental organisations (Kasambala et al, 2006:59-60). The Global Fund continued to support the NAC, but in a fairly limited manner while countries like Zambia, which have lower HIV prevalence rates than Zimbabwe (Moyo et al, 2007a:18; IRIN 2009) got more support. Even so, Zimbabwe remained heavily dependent on foreign assistance (discussed more below). By 2006, the total amount of funding coming into Zimbabwe stood at about $100 million while about $10 million was raised internally (primarily through the AIDS levy) (Nyahonda, 2006: 3-4).

The modelling exercise presented in Chapter 2, suggests that Zimbabwe's implementation of an ART program from 2004 (UNAIDS, 2008:2-6:25) probably managed to reduce AIDS mortality. The ART rollout came after government announced a State of Emergency around AIDS which allowed it to satisfy World Trade Organization regulations governing the import or manufacture of generic versions of patented pharmaceuticals (Khor, 2007:9-10). To allow local production of ART, government's Medicine Control Authority protected local manufacturers from cheaper imports (Makiwa, 2004). Varichem, the first African Pharmaceutical company to manufacture ARVs was a beneficiary of this policy and in October 2010, it became the fourth company in sub-Saharan Africa to achieve WHO prequalification for its ARV products[27] (WHO, 2011). Prequalification means that Varichem can make generic copies of products that are patented in most developed and developing countries.

The distribution of ART has been marred by a lot of controversy as NGOs, the MDC and government all tried to gain political leverage from the crisis. I address these issues later. In summary, because of Zimbabwe's political and economic decline, by 2007, government had

[27] The prequalified drugs are Lamivudine/Zidovudine - 150mg/300mg.
See http://apps.who.int/prequal/query/ProductRegistry.aspx

fallen short of its target having been able to give ART to only 100,000 (out of a 250,000 target) (GOZ, 2008:5). The signing of a global political agreement between ZANU PF and the MDC in 2008 however led to an increase in donor funding and government targeted to increase public sector provision from 150,000 in 2008 to 343,000 in 2009 (number in need in 2009 is estimated at 343,000) (UNAIDS, 2008:25).

The ART rollout program ran parallel to the provision of PMTCT. A multi-sectoral national PMTCT partnership forum, which meets regularly to improve coordination of the program, was established in 2004 (UNGASS, 2008:19). Zimbabwe has gradually improved provision of PMTCT and at the end of 2006, 1,422 health institutions were offering PMTCT services of which 547 offered "comprehensive PMTCT services" (UNGASS, 2008:19). This means that at these sites, the women had access to on site HIV testing and counselling as well as ARV prophylaxis. The remaining 875 sites offered a minimum/supportive package of PMTCT services that included on-site supportive counselling as well as ARV prophylaxis but with testing done at one of the other 547 sites (UNGASS, 2008:19). UNAIDS publications from which Chapter 2's modelling assumptions drew, report that PMTCT coverage has improved steadily from 8% in 2004, 13% in 2005 17% in 2006 to 29% in 2007 (UNAIDS, 2008:15). However, Nevirapine (NVP) uptake has remained low due to poor male participation in ANC and critical staff shortages in the public health sector particularly at the primary health care level (UNGASS, 2008:19). In general, the same funding or political problems that have characterised the ART rollout have also had a similar impact on the PMTCT programmes.

4.2.3 The politicisation of Zimbabwe's AIDS response

As discussed in the previous chapter, in the period after 1998, the government was desperate for political survival and it identified NGOs and donors as the enemy. After the 1998 riots, the Zimbabwe Congress of Trade Unions (ZCTU) civic leaders and churches became more militant and started organising protests and mass action culminating in the formation of the MDC in 1999 (Raftopolous, 2009:209). Unlike in Uganda, government not only tried to manage the epidemic and claim credit for the success of the interventions but it actively sought to neutralise NGOs and donors. This strategy came at a huge cost in the form of a rapidly declining health delivery system and increases in AIDS mortality. The battle against the NGOs was carried out on two fronts with the state using repressive legislation as detailed in Chapter 3 and restricting the flow of funding to these institutions.

The Zimbabwe government used repressive parliamentary legislation as well as political intimidation to wrestle control of the intervention programs from NGOs (despite it having little capacity to manage on its own). It also used the same tools to control the flow of information. As we discussed in earlier sections, this stifling of the AIDS response became obvious in 2002 when government passed the Public Order and Security Act (POSA) and the Access to Information and Protection of Privacy Act (AIPPA) (McGregor, 2002:9-37). These restricted the movement of all NGOs, opposition parties and also allowed the government to manage the flow of information.

These laws also allowed government to manipulate the media and falsely seek credit for the decline in prevalence with the President claiming that prevalence had declined despite Zimbabwe not receiving external funding (President Mugabe 2004). At the time, government only contributed 10% of total the total funding for AIDS interventions (Nyahoda, 2006:3-4). There are many other instances where government either misinformed the public or accused NGOs of impropriety (Gore, 2003; Nherera, 2003; Gundani, 2004; Rödlach, 2006). In 2004, government also attempted to pass the Non-governmental Organizations Bill which aimed to criminalize NGOs whose objectives include "issues of governance", or "the promotion and protection of human rights" (CRS, 2005:6). This was later done in July of 2008 a few weeks before the presidential run-off election (IRIN, 2010).

Even after the formation of the inclusive government, government continued to threaten NGOs. On July 27 2009 Mugabe remarked;

"*We have now a phenomenon of NGOs, or shall I call them phenomena, for they really are a type of government in the background of a formal government. I don't know whether this creature is for the better or for the worse, but in our country we have seen a situation where they have exceeded their terms of reference and perhaps we might have to reconsider the advisability of having NGOs*." (IRIN, 2010).

NGOs have been the corner stone of the AIDS response in Zimbabwe as they provide millions of Zimbabweans with food, medication, education and human rights support

(Ngirande, 2010). The government's onslaught on NGOs has had the effect of reducing their efficiency thus putting the lives of the millions infection with HIV or suffering from full blown AIDS at risk.

The second battle front was that of funding. Because the AIDS response was funded by both government and non-governmental donors, government's control of the AIDS response was strongly challenged. As a signatory to the Abuja declaration of 1998, government committed to put at least 15% of the government budget to health care (UNGASS, 2008; 5-6). This combined with the takings from the AIDS levy (National AIDS Trust Fund (NATF)) allowed government to contribute US $ 14,7 million in 2005, US $ 63,4 million in 2006 and US $ 86 ,256 in 2007 (UNGASS, 2008;5). International bilateral and multilateral donors contributed about US $ 74, 7 million in 2005, and US $ 66 million in 2006 and over US $ 110 million (UNGASS, 2008; 5-6).

The government's persecution of the opposition and NGOs led to more external funding being funnelled through NGOs further weakening government's control of the AIDS response. Government responded by levelling charges of corruption against a number of NGOs in an effort to discredit them (Kasambala et al, 2006:62-63). For instance, the Minister of Health alleged that the Zimbabwe National Network for People Living with HIV and AIDS abused $96 million allocated to it though the NAC and had used part of the money to buy property (The Herald, 2004). All these efforts made international donors more sceptical and Zimbabwe received the lowest amount of funding (per capita) in comparison with other countries with high prevalence rates (Loewenson, 2008:20).

The distribution of ART was also marred by controversy as government was accused of giving exclusive access to the lifesaving drugs only to those in government circles (Chipunza 2005). Similar accusations surfaced in 2008 where some government officials were accused of selling the drugs on the black market (Sollom, 2009:33; Chimunhu 2010). The scandals surrounding the ART rollout are not confined to misappropriation of drugs but extend to misuse of AIDS funding as illustrated by the Reserve Bank Governor's unauthorised withdrawal of US$7.3 million from the National AIDS Council's official account (IRIN 2009, IRIN 2009a). These funds had been donated by The Global Fund for the purchase of ARV drugs and the central bank later returned them (IRIN 2009a).

The Reserve Bank Governor also admitted to having taken hundreds of millions of Euros from private bank accounts, including 300,000 Euros from a bank account belonging to Hivos, a Dutch development organisation in order to "keep the country afloat" (Radio Nederland Wereldomroep, 2009). Reporter Peter Vermaas, alleges that at the time he wrote the article, Hivos was yet to be reimbursed an amount totalling 90,000 Euros (Radio Nederland Wereldomroep, 2009). All of these scandals illustrate how government's approach to the AIDS response may have actually exacerbated AIDS mortality as it stifled attempts by NGOs to distribute ARV drugs, food and other forms of support.

4.3 Behaviour change

The incidence and prevalence modelling done in chapter 2 suggest that in Zimbabwe, there were two periods with different characteristics with respect to behaviour change. The rise in prevalence (between 1984 and 1997) was due to an increase in new infections shown by Figure 2.15. The subsequent fall (post 1997) in HIV prevalence was due various factors such as an increase in AIDS mortality, migration and to a larger extent, a reduction in new infections. In this section, I review the available evidence from other sources to explore whether it supports this story.

4.3.1 The impact of the AIDS intervention programs

There are grounds for assuming that government action did not stem the AIDS epidemic in its early stages and that the anti AIDS campaigns were largely ineffective (Avert, 2010a; Vos, 1994; Siziya et al, 1999). A cross sectional study of commercial farm workers in Manicaland, Mashonaland Central, Mashonaland West and Mashonaland East provinces of Zimbabwe comprising 406 men and 411 women revealed that even in the late 1990s, behaviour change was not yet noticeable (Siziya et al 1999:316–20). The authors concluded that despite the high sero-prevalence rates in Zimbabwe, many of those sampled did not perceive themselves to be at risk. This kind of denialism would have made them persist in high-risk behaviour.

Vos proposes that it was likely that the early prevention campaigns were based on incorrect assumptions about sexual behaviour – notably under-estimating risk factors such as sexual concurrency and that this would have reduced the efficacy of the education campaigns[28] (Vos, 1994:196-201). Vos' observations were based on a survey with questions on sexual practices including sexual concurrency, condom use, anal sex and other practices (Vos, 1994:193). It comprised 111 structured interviews with hospital patients, secondary school students and teachers, and 11 focus group discussions with traditional healers, midwives, village community workers, secondary school students and teachers, and commercial sex workers in a rural district of Matabeleland in Zimbabwe (Vos, 1994:193). The aim of the study was to investigate attitudes towards sex and sexual behaviour in order to define more appropriate health education messages (Vos, 1994:193-203). As already mentioned, research and empirical evidence on the prevalence and spread of HIV in the early stages of the epidemic in Zimbabwe is quite thin and the above study is arguably one of the most comprehensive studies on sexual behaviour in Zimbabwe in the early stages of the epidemic.

The study suggested that there had been a break down in traditional norms and that this is one of the reasons for supposing that there had been an increase in risky behaviour (between the late 1980s to early 1990s). Traditional sex education (where teenagers were groomed to become sexually responsible adults) no longer took place and communication between sexual partners was, in his assessment, limited (Vos, 1994:196). He argued that large-scale migrant labour had increased the demand for transactional sex and the women engaging in transactional sex largely did not negotiate condom use. Some men reported a "biological" need for multiple sexual partners, a belief supposedly rooted in traditional customs where a man showed his wealth by having multiple partners. Vos also found that only penetrative sex was acceptable and other types of sexual behaviour (such as thigh sex – which had been promoted by adults in the past as acceptable sexual experimentation for young unmarried adults) were now seen as deviant. Vos (1994:193) argued that more effort should have been put into restoring traditional communication about sexual matters across generations and to urge partners to discuss sex (Vos, 1994:193).

[28] Initial campaigns primarily emphasized condom use and abstinence (NAC, 2006)

With the switch from the MTP1 to MTP2 (1993-1994), AIDS policy interventions were made more multi-sectoral as government partnered many different non-governmental organisations (much like Uganda had done in the late 1980s). As the educational campaigns and other efforts picked up pace and the epidemic progressed, there were some indications of behaviour change and improved attitudes towards HIV/AIDS were noticeable from the mid-1990s. A survey study carried out between March and June 1994 in the Manicaland Province of Zimbabwe showed that there was evidence of delayed onset of sexual relations, increased monogamy and increased use of condoms (Gregson et al, 1997:327-329).

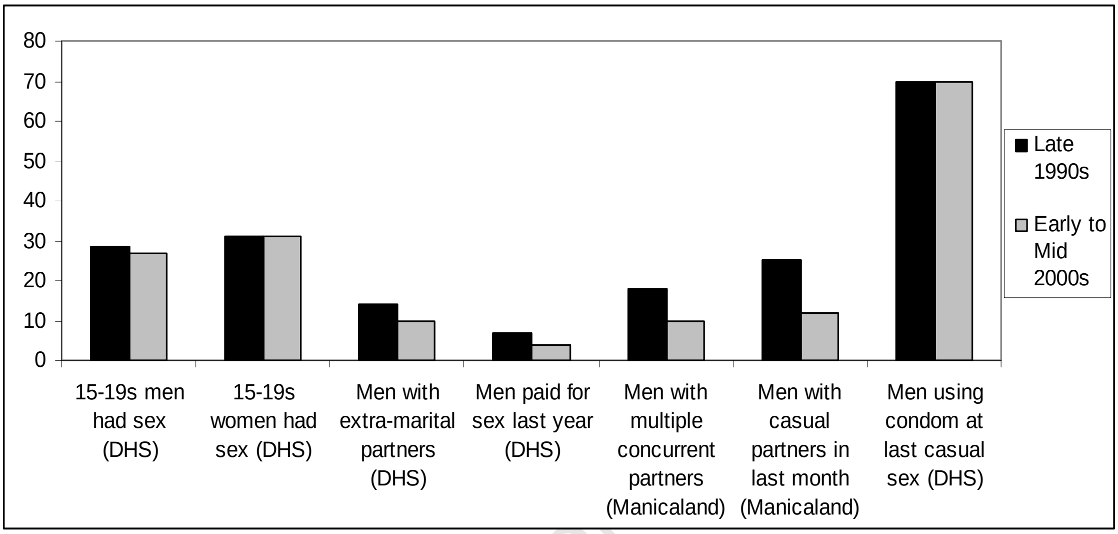
The study took the form of a household census of 1294 women of childbearing age (13-49 years) in the Mutasa and Chimanimani areas (Gregson et al, 1997:322). The questions asked sought to obtain socio-demographic information as well as detailed data on breastfeeding and births Gregson et al, 1997:322). There were also questions on the women's' sexual activity and union histories, attitudes towards, as well as their use of methods of contraception and knowledge on HIV (Gregson et al, 1997:322). The study came with a caveat. The authors stress that using self-reported data to gauge behaviour change is not an optimal methodology because it probably exaggerates the change in behaviour (Gregson et al, 1997:321)

Gregson et al argued that behaviour change was probably facilitated by greater knowledge, experience and personal risk perception but hampered by low female autonomy, marital status and economic status, and by male labour migration and alcohol consumption (1997:328). Personal risk perception was quite high (42%) and correlated with non-marriage, media exposure and contact with medical services (ibid 1997:326). There was a positive relationship between knowledge on HIV and AIDS and education, religion, travel and media exposure (ibid 1997:326-8). Gregson et al (1997:328) argue that the association between improved knowledge and contact with the media show that national information campaigns had started to have a significant impact on behavioural trends. Since the results of this study coincide with the shift in government policy from MTP1 to the multi-sectoral MTP2 (1994 to 1998), this may very well be the case.

Figure 4.2 is taken from Halperin et al (2011) and shows results from the 1999 ZDHS, 2005/6 ZDHS and surveys carried out in rural Manicaland between 1998 and 2000 as well as

2001 and 2003. The 2005 ZDHS was described in greater detail in Chapter 2. The 1999 ZDHS is a nationally representative survey that was implemented by the Central Statistical Office (CSO) from August to November 1999. These epidemiological findings are consistent with the decrease in new infections (as modelled in Chapter 2) was probably to a great extent due to behaviour change, primarily a reduction in sexual concurrency.

Figure 4.2 Epidemiological findings on sexual behaviour



Source: Multiple sources in Halperin et al (2011)

Figure 4.2 shows that between the late 1990s and mid-2000s, the percentage of men with extra marital partners decreased and so did that of men paying for sex. However, in the same time period, the percentage of men using a condom during casual sex remained constant. The Manicaland surveys echo this trend. They show a decrease in the percentage of men with multiple concurrent partners between the late 1990s and the mid-2000s. In order to allow for a discussion of how the epidemics were different in each of Zimbabwe's 10 provinces, I will use evidence from the Zimbabwe Demographic and Health Survey (ZDHS) of 1994, 1999 and 2005.

4.3.2 Sexual concurrency

While it is generally accepted that the first ten years of Zimbabwe's independence were characterised by relative success and development, these were followed by at least 15 years of general socio economic decline. According to an argument made by Iliffe (2006) this could have protected Zimbabwe to some extent from an even greater rise in HIV prevalence. He suggests that poor communication infrastructure and other such characteristics of under development probably slowed the spread of HIV infection for example, in the sparsely populated and often forested regions of the DR Congo (Iliffe, 2006:10).

The Iliffe argument can be applied in the Zimbabwean context to suggest that the economic prosperity, infrastructure growth and modernisation of the 1980s was primarily responsible for the spread of HIV rather than the subsequent economic crisis. Before analysing the implications of the Iliffe argument regarding Zimbabwe, one must first accept the premise that sexual networks increase the risk of HIV infection and that economic development and modernisation (which improves infrastructure and incomes) foster the creation and maintenance of such networks.

There is a substantial body of research that argues that the creation of sexual and other social networks might increase the risk of infection (Epstein, 2008**;** Morris, 2008; Halperin, 2004; Rothenberg, 2001; Morris, 1997 & 2000). Mah and Halperin (2008) and others have proposed that concurrent sexual relationships are a key driver of the epidemic in sub-Saharan Africa. Using network modelling, (Morris and Kretzchmar 1997a b; Kretzschmar 2000; Epstein 2008, Potts et al. 2008) demonstrated that sexual concurrency drives HIV prevalence to a far greater degree than the number of sexual partners. However, there have recently been dissenting voices who charge that the evidence on which Mah and Halperin base their arguments is weak and that more research is required to bolster their argument (Lurie et al 2010). In particular, they point out that there is limited evidence that levels of concurrency are significantly higher in Africa than elsewhere and there is no empirical proof that the observed levels of concurrency explain the African epidemics (Lurie et al, 2010:18).

The debate is also centred on differing views on the definition of sexual concurrency (Lurie et al, 2010:18). Mah and Halperin affirm that HIV spreads more rapidly in sexual networks with more partnerships that overlap in time compared to networks in which serial monogamy predominates (Mah and Halperin, 2008). They suggest that the definition of concurrency be the overlap of one or more sexual partnerships for a period of one month or longer (Mah and Halperin, 2008:12). This according to Lurie is too loose a definition and employing a more strict definition will improve our understanding of the impact of concurrent sexual relationships on the AIDS epidemic (Lurie et al 2010). For instance, using the Mah and Halperin definition, participants in concurrent sexual relationships can have as few as two partners in their lifetime or as many as hundreds. All these are pooled and given the same definition despite the fact that some of these would have been one time encounters (Lurie et al, 2010:18).

Most recently, Tanser F et al (2011) carried out an analysis of field data which adds an interesting dimension to the debate. They used data from the Africa Centre demographic surveillance site in KwaZulu-Natal, South Africa and applied a multi variable statistical model to contrast the increased risk of infection in an HIV-negative woman living in a community with high numbers of reported lifetime sexual partners in men (a widely accepted and robust index of risky sexual behaviour) against that of a woman living in a community with a high prevalence of male concurrent sexual partnerships. They followed up 7284 women who were HIV negative at baseline, were tested at least twice during the study period (2004–09), and were resident in the surveillance area at least half of the time[29]. Tanser F et al (2011) found no evidence to suggest that concurrent partnerships are an important driver of HIV incidence in this typical high-prevalence rural African population. Their conclusion was that there should be a push to reduce multiple partnerships, irrespective of whether those partnerships are concurrent.

Despite the doubt that has been cast over the size of the impact of sexual concurrency on the epidemic, there is broader consensus that it played some role in the spread of the disease. More interestingly, there are authors who argue that high incomes are linked to the development of such networks (Serwadda et al, 1992). In Uganda, employment in the formal sector has been associated with higher HIV infection as those who gain employment also attain economic freedom, which facilitates their maintenance of concurrent sexual partnerships (Serwadda et al, 1992). The history of the epidemic (early stages) has been no different in other parts of sub-Saharan Africa. Studies by Killewo et al (1990) and Neequaye et al (1991) in Tanzania and Ghana respectively identified sexual intercourse with prostitutes,

[29] They note that only 273 did not meet the reside in the surveillance area at least 50% of the time.

history of sexually transmitted diseases, multiple sexual partners and traditional practices as risk factors in adults. For commercial sex workers to stay in business, there must be buyers willing and able to pay for their services and so economic vibrancy has often been accompanied by the growth of the commercial sex industry, particularly in south and south east Asian countries (Phongpaichit et al 1998:196; Clarke, 2006; Clarke et al, 2004: 153).

Iliffe's argument that poor communication infrastructure and other such characteristics of under-development actually slow down the spread of the epidemic has two possible but speculative implications for the epidemiology of HIV in Zimbabwe and other developing countries. The first is that as Zimbabwe became more prosperous (1980-1990), modernization led to the improvement of communication infrastructure, which would have helped the creation and maintenance of sexual networks (and would have given sex workers access to a wider, more mobile, and population of clients).

Second, it is consistent with the fact that as prevalence rose rapidly, the epidemic was relatively less menacing in rural Zimbabwe (where development was slower) than in urban Zimbabwe. This is true for the period 1985 to around 1990 as is shown by the ANC sentinel surveillance data presented in Chapter 2. An interesting example is how the epidemic in sparsely populated and economically disadvantaged Matabeleland North was more subdued than that in capital provinces of Harare and Bulawayo.

The economic decline (post 1990), precipitated by droughts and the structural adjustment programs not only slowed down development, but it initiated a reversal of the gains made in the first years after independence. I argue, rather speculatively that this might have caused a break down in infrastructure which would have been felt less by those in already underdeveloped rural regions. Therefore post 1990, prevalence would have continued to rise in rural areas (and predominantly rural provinces) albeit at a slower pace than in urban areas. This also conforms to trends that have been witnessed in other sub-Saharan countries were prevalence has generally been higher in urban areas (Dyson, 2003:427). It is worth noting that just as HIV prevalence rose at a faster rate in urban areas, the decline in prevalence was also slower in rural areas. Figure 2.11 shows that while in 1984 HIV prevalence was around 3.8 % and 4.2% in urban and rural areas respectively, by 1987 the trend had reversed with rural areas having prevalence rates at 8.6% versus 8.7% in urban areas. Urban areas continued to have higher prevalence rates until after prevalence had long peaked in 1997 and the modelling suggests that around 2006 HIV prevalence in urban areas stood at 15.5% versus 16.2 % in rural areas.

Iliffe's point is that lack of infrastructure slows down the epidemic by hampering the maintenance of sexual networks(Iliffe, 2006:10). However, maybe investigating the incentives for engaging in such relationships might reveal more. Several authors have pointed to the presence of transactional sex across the income distribution in Africa, ranging from sex for food amongst poor women to sex for luxuries amongst the better off (Swidler et al, 2007; Leclerc-Madlala, 2003; Caldwell et al. 1989; Ankomah, 1999). For instance Shelton et al (2005) intimated that the poor especially women are vulnerable to sexual exploitation because HIV prevalence is partly a function of survival. Further Lugalla et al (1999) argue that gender inequality and poverty deprives women of their ability to fulfil their socially designated responsibilities, and therefore debases them, often forcing them into commercial sex work. Scholarly research on the relationship between transactional sex and income has shown that HIV prevalence is not only highest in the relatively wealthy Southern African countries but HIV prevalence is highest among the wealthiest groups within each country (Shelton et al. 2005; NSO and ORC Macro 2005 for Malawi).

When the economy grows and living standards rise, transactional sex affords enterprising women access to both basic foodstuffs and luxuries (Leclerc-Madlala, 2003; Ankomah, 1992). Leclerc-Madlala, (2003), carried out a small study in Durban, South Africa aimed at exploring the incentives driving sexual exchange for material gain. The study concluded that acquiring the "commodities of modernity" is the new incentive for transactional sex. It may well be the case that as incomes rise, transactional sex for luxuries rises too as men have more disposable income, and women find new ways to obtain a share of it. This could be a reason for the rise in prevalence in Zimbabwe between 1985 and 1997.

However, work based on rural Malawi revealed that transactional sex is not only about women's poverty or about the need for social mobility but also about men who are relatively well off and so feel compelled to have multiple sex partners (Swidler et al, 2007). Swidler et al also argue that transactional sex can be thought of as a way of establishing and maintaining a binding social relationship, which can be activated or deactivated when the need arises. The most important conclusion they make is that transactional sex is about ties of dependence (investing in people and trusting they will reciprocate) that have been long established. This suggests that as long as pervasive economic and social insecurity and under developed labour markets persist, patron-client ties will continue to be a feature of African societies rendering them vulnerable to HIV epidemics (Swidler et al, 2007:158).

The Swidler et al argument highlights he incentives poorer people have to get into networks. This could explain why prevalence continued to rise rapidly in both rural and urban areas even as the economy faltered. More importantly, the above arguments suggest that whether a particular country has higher urban or rural epidemics is a matter of their particular history, cultural dynamics and development. In order to understand some of these differences, it is useful to look at some empirics on the change in sexual behaviour in Zimbabwe (and differences between provinces), particularly pertaining to sexual concurrency.

Figure 4.2, taken from Halperin et al, (2011) shows that between the late 1990s and early 2000s there was nearly a 30% reduction in the proportion of men reporting extra-marital partners. Evidence presented in a comprehensive review of various surveys[30] showed that the proportion of males and females aged 15-29 reporting non regular sexual partners in Zimbabwe (in the past 12 months) decreased between the late 1990s and mid-2000s (Mahomva et al, 2006:1-2).

Table 4.1 shows the percent of people who had more than 2 sexual partners in the 1994, 1999 and 2005 ZDHS. Regrettably, the ZDHS do not contain data on sexual concurrency but one may have a rough idea of the level of sexual concurrency based on the number of multiple partners an individual has. Despite that only multiple sexual partnerships running at the same time may be interpreted as concurrency, a higher number of sexual partners may indicate a greater level of risky sexual behaviour and an increased chance that some of the sexual partnerships run concurrently. The 1994 and 1999 surveys asked respondents the number of sexual partners they had in the previous 12 months. However, the 2005 survey asked

[30] These were the 1999 ZDHS, 2001 and 2003 Population Services International (PSI) youth survey, the 2000–2004 ANC and the 2001/2002 YAS (Young Adult Survey).

respondents the number of sexual partners they had in the previous month. Despite not being a perfect statistic, the 2005 ZDHS provides a more reliable measure than the 1994 and 1999 ZDHS data.

Table 4.1 Reported number of sexual partners

DHS surveys			Men			Women
Province	1994	1999	2005	1994	1999	2005
Manicaland	15.7	5.8	16.8	2	0.4	0.7
Mashonaland Central	5.3	10.4	16.8	1.3	1.4	1.8
Mashonaland East	9.2	4.2	4.2	1.1	0.4	0.8
Mashonaland West	5.6	11.9	15.8	3.4	1.1	1.4
Matabeleland North	19.4	12.5	13.2	1.6	0.7	0.4
Matabeleland South	9.7	9.9	2.4	0.5	2.8	3.3
Midlands	13.2	10.3	13.4	3	1.4	0.9
Masvingo	5.9	8.5	18.9	1.8	0.5	0.9
Harare	10.6	6.1	15.3	1	1.1	2.2
Bulawayo	6.2	10.5	11.5	1.2	0.9	1.2

Sources: ZDHS (1994, 1999, 2005)

Between 1994 and 1999, the ZDHS data shows that the number of people who had more than 2 sexual partners in the previous year decreased. Matabeleland North, Midlands and Harare provinces had the highest rates of men with more than 2 sexual partners over the previous year. Given that for Harare and Midlands, HIV incidence and consequently prevalence peaked at relatively high levels (as shown in figures 2.2 to 2.6 prevalence in both provinces peaked at 35.9%), the numbers shown in Table 4.1 make sense. However, the puzzle comes when one considers that Matabeleland North had the lowest peaking epidemic yet it had the highest percentage (in both 1994 and 1999) of men who had more than 2 sexual partners in the previous year.

This flies in the face of the idea that sparsely populated regions with poor infrastructure such as Matabeleland North would have had slower epidemics. Despite them not having road and communication infrastructure (which makes the maintenance of sexual networks easier) as efficient as that of other provinces such as Harare, it may be that there are cultural beliefs and practices common place in Matabeleland North which encouraged multiple sexual partnerships. Sambisa et al (2010) used cross logistics regression analysis on the Zimbabwe

Demographic and Health Survey data to show that Shona youth were less likely to have engaged in risky sexual behaviour than their Ndebele counterparts[31]. Another explanation could be that self-reported data on sensitive issues such as sexuality are generally unreliable. It is also likely that the provincial level is too broad for thinking about the linkages between infrastructure, population density and sexual networks. Large parts of Matabeleland North are unpopulated and so the picture might be different if one looked at village level dynamics.

Compared to DHS data for women, there were proportionally more men than women who had more than 2 sexual partners in the previous year. Mashonaland West and Masvingo had the highest rates of women with more than 2 sexual partners over the previous year in 1994. However in 1999, Matabeleland South, Mashonaland Central and the Midlands provinces had higher percentages of women with more than 2 sexual partners.

The percentage of women with more than 2 sexual partners in the 1999 ZDHS follows a trend comparable to that for men in the same survey. These numbers are in tune with the HIV prevalence rate estimates modelled in Chapter 2 (see Figures 2.2 to 2.6). These graphs show the Midlands and Mashonaland Central provinces as having had higher peaks (35.9% and 27.0% respectively) in HIV prevalence than other sub-epidemics such as Matabeleland North (peaked at 19.6%) where the percentage of men and women reporting to have had more than 2 sexual partners over the previous year was higher.

Table 4.1 also shows the percentage of men and women who reported having had more than two non-regular sexual partners in Zimbabwe (in the past month) as reported in the 2005/6 ZDHS. The phrasing of the question presented in the survey clearly makes the 2005 data a more stringent measure of sexual networks than those presented from1994 and 1999. However, all the numbers presented in this sub-section are far from ideal because in order to interpret the number of sexual partners (which is what is shown in Table 4.1) as sexual concurrency, one needs data on overlapping dates.

Consistent with the 1999 figures, the percentage of men who reported having had more than two non-regular sexual partners in Zimbabwe in the previous month was lowest in

[31] Matabeleland is predominantly Ndebele.

Mashonaland East where as shown in Figure 2.7, prevalence peaked at a relatively high rate of 31% in 1997/8. The 2005 DHS data shown in Table 4.1 further cements the notion that each province experienced its own sub-epidemic which was a function of many province/region specific factors including opportunities for development of sexual networks

4.3.3 Condom use

A UNAIDS review in 2005 claimed that an increase in condom use could have contributed to a decline in HIV incidence during the 1990s and hence to the fall in prevalence (age 15-49) from 2000 to 2007 (UNAIDS, 2005:39). Incidentaly, the review down plays migration and mortality as possible causes of the decline in prevalence. In the review, UNAIDS argue that mathematical model simulations of the HIV epidemic in Zimbabwe show that the pace of the declines in prevalence and incidence could not have occurred without substantial changes in risky behaviour including a decrease in non-regular sexual partners and significant increase in condom use among non-cohabiting partners (UNAIDS, 2005:39). The EPP and Spectrum modelling done in Chapter 2 which reverse engineers the UNAIDS/MOHCW modelled results however shows that although the decrease in new infections was a significant factor in the overall decline in HIV prevalence in Zimbabwe, migration and mortality were influential factors as well.

In Zimbabwe, distribution of condoms by the public sector is done through the Zimbabwe National Family Planning Council (ZNFPC). One of the biggest proponents of the notion that a change in sexual behaviour is the major reason why prevalence has fallen rapidly since 2000 is Population Services International (PSI). PSI reports that Zimbabwe is one of the few success stories in female condom programming and PSI/Zimbabwe’s social marketing program has been praised by donors and the news media for its different but supposedly effective approach (IRIN, 2010). In 2001 PSI distributed (sold) 455,566 female condoms, which quadrupled to 1,806,760 by 2007 (UNPF, 2008:12). In 2006, more than half of all PSI/Zimbabwe’s female condoms were distributed through a network of 823 hair salons and 1,045 hair dressers mainly in low income areas (DFID , 2006:1). By 2006, it is reported that the hair salon program had increased knowledge of and demand for the female condom massively, contributing to the 240% increase in use between 2001 and 2006 (DFID, 2006:1).

The distribution of male condoms has also grown phenomenally. In 1996 PSI started a condom social marketing program with the launch of subsidized “protector plus male condoms”, which were sold through existing private sector channels, including non-traditional retail outlets such as liquor stores and kiosks (PSI, 2010). Male condom distribution increased from 7,593 930 in 2001 to 48,134,700 in 2007 (UNPF, 2008:12). The ZNFPC complimented the PSI’s efforts and in 2006, they distributed (free) 980,560 female condoms and 55,567,000 male condoms (UNPF, 2008:12). However, 2006 UNGASS interviews indicated that young people preferred socially marketed branded condoms, sold by PSI which they perceived to be safer than the free condoms given out by ZNFPC and affordable (UNPF, 2008:12-13).

Data from a range of national surveys (including the 1994 & 1999 Demographic Health Survey, the Zimbabwe Young Adult Survey (2002) and recent PSI population-based surveys (2005)), reveal high and increasing levels of condom use with non-cohabiting sexual partners (Mahomva et al 2006:1-6; PSI, 2007). The same surveys also happen to show that condom use for family planning purposes was very low and one might conclude that non-cohabiting sexual partners were the biggest users of condoms (PSI, 2007).

Halperin et al, (2011) as well as Hallet et al (2009) and Muchini (2010) argue that condom distribution and promotion efforts that began in the early 1990s are likely to have contributed through helping build high levels of condom use for commercial and casual sex. PSI attributes the increase in condom use to changed attitudes to condoms, changing social norms and improvements in the marketing of product (PSI, 2007). Condom use (self-reported) increased steadily during the 1990s. For instance, Figure 4.2 shows that in most provinces, prevalence of condom use (in the last 4 weeks) among sexually men surveyed in the 1994 ZDHS was above 60%.

It is also interesting to investigate how condom use may have varied from province to province. This is an important exercise as the EPP modelling in Chapter 2 revealed how epidemics in the different regions followed different trajectories. This analysis is best done on a per survey basis (looking across provinces) rather than comparing one survey to the next as the questionnaires used often had questions phrased in ways that made the numbers hard to compare (as highlighted in this section). For some questions, the sample sizes of the respondents were very low (shown by the dashes in Table 4.2. Some of the results from the 1994, 1999 and 2005 ZDHS questions on condom use are shown in Table 4.2.

Table 4.2 Reported condom use

DHS surveys	Men			Women		
Province	1994 (% of sexually active)	1999 (% of married)	2005 (% of youth)	1994 (% of sexually active)	1999 (% of married)	2005 (% of youth)
Manicaland	63.5	4.7	79.3	29.4	1.4	-
Mashonaland Central	71.6	4.0	79.3	34.1	1.5	-
Mashonaland East	80.7	7.9	68.8	36.5	1.3	-
Mashonaland West	74.1	4.8	78.7	35.5	0.5	-
Matabeleland North	49.9	4.7	38.7	19.7	1.6	17.4
Matabeleland South	57.9	4.2	64.3	34.3	2.3	31.0
Midlands	75.9	10.5	53.4	35.8	1.9	25.6
Masvingo	54.5	1.8	46.8	23.6	1.5	-
Harare	73.1	6.3	91.0	38.4	2.3	49.5
Bulawayo	63.6	5.3	75.4	34.5	4.0	56.1

Sources: ZDHS (1994, 1999, 2005)[32]

In 1994, the ZDHS asked all sexually active individuals if they had used a condom in the previous 4 weeks. Condom use among men was highest in Mashonaland East (80.7% for men and 36.5% for women) and the Midlands (75.9% for men and 35.8% for women). However, condom use was especially low in Masvingo (54.5% for men and 23.6% for women) and the Matabeleland North (49.9% for men and 19.7% for women).

On the one hand, it is understandable why Matabeleland North, which had lower HIV prevalence rates (as shown in Table 2.7) than all other provinces would also have lower rates of condom use. It might be that there was low perceived risk of infection via unprotected sex. But if this is the case, then it is unclear why Masvingo, which had much higher HIV prevalence rates (peaking at 37% in 1997) had very low rates of condom use. What is plausible is that the low rates of condom use in the province may have contributed to the high prevalence rates estimated in Chapter 2. As a caveat, it is important to note that self-reported data on condom use is problematic. One of the concerns is that even if one uses a condom, it is impossible to know whether or not it was used properly. Another point to make is that

[32] CSO (1994), CSO (1999), CSO (2006) also report similar figures

condom use may be a poor indicator where couples do not use condoms but both partners are monogamous. In such cases, not using a condom is a poor indicator for the risk of infection.

Table 4.2 shows slightly different numbers from the 1999 ZDHS. It shows the percentage of married individuals who were “currently” (i.e. in the last sexual encounter) using condoms. The table shows that condom use among married people was highest in the Midlands (10.5% for men and 1.9% for women) and Mashonaland East (7.9% for men and 1.3% for women). Condom use among married people was lowest in Masvingo (1.8% for men and 1.5% for women). This is particularly concerning given that Masvingo’s prevalence rates peaked at a level higher than that for all other provinces (see Chapter 2). It may be that the low rates of condom use contributed to the high prevalence rates although this would only be one of many factors.

The 2005 figures show the percentage of youth (aged 15-26) that used a condom in their last sexual act. Most notable are the numbers for males in Masvingo (46.8%) and Matabeleland (38.7%). These are extremely low although this is in keeping with the trends shown in the 1994 and 1999 surveys. The numbers for women are also much lower than those for men so much so that some provinces such as Manicaland had so few respondents (less than 20) than working out percentages would not make sense.

Overall, Table 4.2 not only shows that there were differences in reported condom use across provinces over the years, but it also highlights how low rates of condom use were for women compared to men. This held true for married couples as well as for unmarried youths. In all the surveys and across the different questions on condom use posed, it is clear that reported condom use was more prevalent among men than women. Further, provinces like Matabeleland had lower reported condom use despite having relatively lower peaking epidemics (see Figures 2.6 to 2.9). Turning to reported data on other markers of sexual behaviour in the ZDHS, the only surveys where one can attempt a province by province analysis might help give some explanations for these observations.

4.3.4 Sexual debut

Results of a review of the 1999 ZDHS, 2001 and 2003 Population Services International (PSI) youth survey, the 2000–2004 ANC and the 2001/2002 YAS (Young Adult Survey) concluded that there had been a reduction (between the late 1990s to mid-2000s) in the number of people having sex before age 15 among males and females (aged 15-19 years) (Mahomva et al, 2006:1-2).

Table 4.3 shows the median age at sexual debut for the different ZDHSs. The table also presents median ages at sexual debut for women who were aged 20-49 and men aged 25-49. According to these surveys, there was an increase in the median age at sexual debut for Manicaland (19.5 in 1994 to 21.5 in 1999), Midlands (19,6 in 1994 to 19.8 in 1999) and Matabeleland North (18,0 in 1994 to 18.9 in 1999). These encouraging statistics would have aided a decline in new infections which probably led to the decrease in HIV prevalence observed in the provinces. Unlike for men, the median age at sexual debut increased most provinces. Only Mashonaland Central saw a decrease in these numbers (18.5 in 1994 to 18.3 in 1999).

Table 4.3 Reported sexual debut

DHS surveys			Men			Women
Province	1994	1999	2005	1994	1999	2005
Manicaland	19.5	21.5	22.3	18.6	18.2	37.0
Mashonaland Central	20.8	18.1	33.5	18.5	18.3	57.0
Mashonaland East	20.1	18.3	19.2	18.2	18.9	36.9
Mashonaland West	19.2	18.7	34.5	17.5	18.6	50.2
Matabeleland North	18.0	18.9	40.0	17.4	18.1	51.6
Matabeleland South	18.9	18.3	22.3	17.3	18.0	47.1
Midlands	19.6	19.8	29.4	18.7	19.2	38.8
Masvingo	20.3	19.5	20.8	18.2	18.9	39.0
Harare	19.4	18.9	25.2	19.4	19.8	21.4
Bulawayo	20.0	19.7	36.4	18.6	18.8	19.3

Sources: ZDHS (1994, 1999, 2005)

Table 4.3 also shows some statistics from the 2005 DHS. The table shows the percentage of women aged 18-24 had their sexual debut before age 18. By a significant margin, prevalence of early sexual debut for males was highest males in Matabeleland North. For women, Mashonaland Central had higher rates of early sexual debut although Matabeleland North was a close second. The fact that these numbers were more than 30% for most provinces is a cause for concern. The fact that Matabeleland North, in addition to having lower condom use, it had relatively high prevalence of early sexual debut is of particular interest[33].

4.4 AIDS mortality

4.4.1 Decline in Health Service delivery and the "ESAP" deaths

The state of the health delivery system no doubt contributed to AIDS related mortality, which in turn was a significant cause of the decline in adult prevalence (from 1997 to date). This rise in AIDS deaths continued unabated until the early 2000s. MOHCW (2009:7-8)[34] reported that AIDS deaths most likely peaked around 1999 which was just after the economy had begun slipping into recession. There is a temptation to immediately make a causal link between the rapid increase in AIDS deaths and the decline in health system but this thesis does not carry out an exhaustive, scientific interrogation of this. It merely describes the context around the evolution of the epidemic and points out interesting issues that would warrant further investigation.

All the gains in health delivery that the first ten years of independence brought had been reversed by the end of 1992 (Chisvo, 1993:3-20). As discussed in Chapter 3, the initial decline of the health sector was precipitated by the structural adjustment programmes prescribed by the IMF and World Bank (Saunders, 1996:2). Per capita public expenditure on health care declined and so did spending on drugs (Dhliwayo, 2001:4-9).

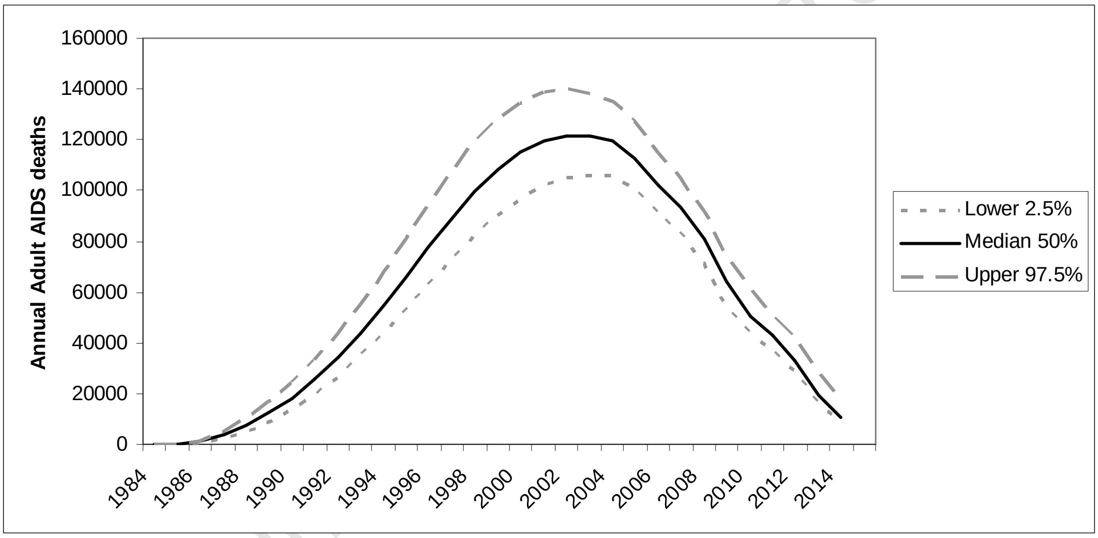
Figure 4.3 shows the trend in adult AIDS deaths as estimated by the Spectrum modelling done in Chapter 2. The trend suggests that the decline of the health system (from1991 onwards) coincided with the increase in mortality and hence may have exacerbated it[35]. AIDS

[33] It is important to note work by Lewis et al (2007) which highlights that the timing of an individual's sexual behaviour in their lifetime is very important. Sexual concurrency, low condom use or early sexual debut present different of risk where the epidemic is in its infancy versus when it is at its peak.
[34] The modelling presented in Chapter 2 shows the same.
[35] Although this is beyond the scope of this thesis, further investigation of a possible causal link will be an interesting exercise.

deaths appear to have risen quite rapidly during the ESAP years (1991-6) which would corroborate the notion of "ESAP deaths" expressed by doctors and nurses in the early 1990s (Saunders, 1996:2).

An area for further investigation would be an analysis of what extent these "ESAP deaths" existed and whether a causal link exists between the decline in health systems and AIDS mortality. This would have to be a separate exercise as the link if it exists is probably a complex one. Given the discussions (in previous chapters) on the lags between infection, sero-conversion and death, particularly in the pre-ARV period days, an causal link will be difficult to establish.

Figure 4.3 Estimates of AIDS Deaths



Source: MOHCW 2009 report (MOHCW, 2009:23)

Table 4.4 shows that the decline in health services continued even after ESAP had been replaced by Zimbabwe Programme for Economic Transformation (ZIMPREST 1996 - 2000). Total hospital admissions declined by 4.7% (477,530 to 454,927) from 1997 to 1998. Between 1998 and 1997, the fall in admissions was even higher at 12.8% (454,927 to 396,511). As the AIDS epidemic would have been placing greater pressure on the health sector over this period, the decline in admissions is an indication of collapsing capacity to treat sick people rather than any drop in the demand for health services.

Table 4.4 Decline in Hospital Admissions

Year	Total Hospital Admissions
1997	477,530
1998	454,927
1999	396,511

Source: QDS, CSO, June/Dec 1999

A study by Gregson et al (2007) showed that despite towns having higher prevalence rates than agricultural estates and villages between 1998 and 2005, villages had much higher rates of AIDS mortality. As Zimbabwe's socio-economic situation deteriorated, the health delivery system further deteriorated. In October 2008, the Zimbabwe Association of Doctors for Human Rights (ZADHR) expressed outrage at the halting of admissions at one of the country's largest referral hospitals (Gwatidzo, 2008). The organization chairperson, Dr Douglas Gwatidzo said the disheartening move had been necessitated by a chronic shortage of drugs, food and staff that had left the country for greener pastures (Gwatidzo, 2008).

Poor nutrition and lack of adequate medical care has been known to result in higher mortality in high risk groups (Bendich, 1998:125-47). This coupled with UN estimates that in 2008, 45% of the population was malnourished could be interpreted to mean that more AIDS deaths (of those who would have lived longer given better nutrition etc) are to be expected beyond 2010 as more HIV cases will progress into AIDS (Chelala, 2008). This is a very likely scenario given that as reported in the latest UNAIDS Zimbabwe epidemiological fact sheet, between 2004 to 2007, the number of people receiving ART increased from just 8000 to 98000 (about 19% of need) (UNAIDS, 2008:2-6).

One often ignored link between increases in AIDS mortality and the change in behaviour observed in Zimbabwe is that high AIDS mortality may have been a dominant factor for stimulating behaviour change. Muchini et al, (2010) argue, using evidence from men and women in focus groups and interviews who reported (repeatedly and consistently) that personal exposure to AIDS mortality and the resulting fear of contracting the virus were the primary motivation for changes in sexual behaviour. This was true for reductions in casual sex and other multiple sexual partnerships (Muchini et al, 2010). Further, some have suggested that the government's early adoption of a home-based care policy may inadvertently have catalysed the process of behaviour change (Hansen et al, 1998). The argument is that the reality of AIDS mortality that confronts care givers when a relative dies at home is more likely to result in a tangible fear of death among family and friends than when patients are primarily cared for in clinical facilities (Low-Beer et al, 2004:165-185).

4.5 Political instability and dislocation

Iliffe (2006:10) has argued that poor communication infrastructure and other such characteristics of under development probably slowed the spread of HIV in conflict regions such as the Democratic Republic of Congo. As has already been described in Chapter 3, the period from 1998 to 2009 was characterised by violence and political intimidation as the Zimbabwean government tried to hold on to power. This could have acted to assist the rapid decline in HIV prevalence over the same period. Dislocation of masses of people to the rural areas through policies such as the 2005 Operation Murambatsvina may have led to a decline in new infections as sexual networks and ties of dependence broke up.

This is particularly so as the pattern of violence around Murambitsvina was such that the urban poor were forcibly relocated to impoverished communal and resettlement areas. In these rural areas, the violent farm evictions and pre-election intimidation by government militia would have also led to dislocation of workers in commercial farming communities. The differences between the patterns of violence in urban provinces such Harare and Bulawayo and rural ones such as Mashonaland West, Matabeleland North and South may have led to the HIV epidemic peaking at different levels in each. However, data constraints (it is very difficult to get hard data on accounts of violence) make this discussion more speculative than analytical. Although available data does not allow us to make conclusive statements regarding the effect of the internal migration on HIV prevalence in each region, Chapter 2 showed how migration of Zimbabwe's prime working age population (15-49) to neighbouring countries and the UK. And USA probably contributed to the decline in HIV prevalence beginning in the late 1990s.

4.6 Conclusion

In the final analysis, STP and MTP1 were ill timed, ineffectual and severely handicapped by the lack of inclusivity of their approach to HIV interventions. They came after a period of denial and insecurity of the part of government which probably contributed to new infections rising quite rapidly from the mid-1990s to early 1990s. Uganda's ABC and "zero grazing" policies were in contrast put in place early enough in the epidemic's history and included other stake holders such as civil society organizations and international donors. The more inclusive MTP2 signalled a change in Zimbabwe's AIDS response, coincided with a decrease in new infections (which started around 1994). Further decentralisation of the response which came with the launching of the 1999 NAP (much like Uganda had done in the late 1980s) helped drive the success of the HIV education messages although the efforts were hampered by government's crackdown on non-governmental organisations).

As the educational, media campaigns and other efforts picked up pace and the epidemic progressed, there were some indications of behaviour change and improved attitudes towards HIV/AIDS were noticeable from the mid-1990s (based on evidence from the DHS and other surveys). These show decreases in sexual concurrency, improvements in condom use (self-reported) and increases in the number of individuals using condoms during casual sex. One puzzling issue with regard to behaviour change is the case of Matabeleland North, a province that had the lowest peaking epidemic yet it had the highest rates of sexual concurrency.

Given how some have argued that sparsely populated regions with poor infrastructure such as Matabeleland North would have had slower epidemics (as it is difficult to maintain sexual networks), one would have expected lower prevalence of multiple sexual partnerships. However, this interesting case highlights the fact that no two provinces are the same and other, more nuanced factors such as regional cultural differences probably influence the sub-epidemics. Although there is no discussion of possible cultural differences between the provinces, a pattern whereby Matabeleland North and to a less extent Masvingo have lower reported condom use and earlier sexual debut confirms these differences

Another cause of the recent decline in HIV prevalence in Zimbabwe is AIDS mortality. AIDS deaths appear to have risen quite rapidly during the early 1990s as the ESAP kicked in and was probably worsened by the virtual collapse of Zimbabwe's health system. Personal experiences with AIDS mortality may also have had the effect of scaring individuals into changing their behaviour. Zimbabwe's economic collapse and political instability marked by such polices as Operation Murambatsvina might have also played a role in the decline in HIV prevalence through dismantling social networks. As noted earlier, these polices also had different implications for different provinces. However, the country as a whole suffered from large out migration of people primarily into neighbouring countries. Arguments that migration had little effect on the epidemic that are based on HIV prevalence data from Zimbabwean's in places like the United Kingdom miss the point that only a fractions of Zimbabweans leaving the country (who could afford) settled over-seas. The overwhelming majority settled in Botswana and South Africa.

Instability and economic decline in Zimbabwe resulted in gaps in information sources (e.g. last national census was completed in 2002) that make a more detailed analysis of the epidemic particularly at regional level nearly impossible to carry out. The analyses in Chapters 2 through to 4 show that in disrupted countries, even where there is no full scale armed conflict or war, it is still very difficult to characterise national HIV prevalence trends. This is because not only are they based on often very sparse data points from sub- regions with their own sub-epidemics but also because of the myriad of nuanced factors that are at play in unstable countries.

The central argument presented in the second part of the dissertation is that the analysis of the link between HIV prevalence and potential social determinants should be carried out with full cognisance of the limits of the available data. I illustrate the point through an analysis of the relationship between armed conflict and HIV prevalence. This chapter reviews the literature on the relationship between armed conflict and HIV prevalence and chapter six continues the review but with specific reference to cross country quantitative studies. Chapter 7 takes a look at the cross-country analysis of the relationship between HIV prevalence and armed conflict in Sub-Saharan Africa further, while chapter 8 pays particular attention to the seven countries which drive the aggregate results.

5 A REVIEW OF THE LITERATURE ON ARMED CONFLICT AND HIV PREVALENCE IN AFRICA

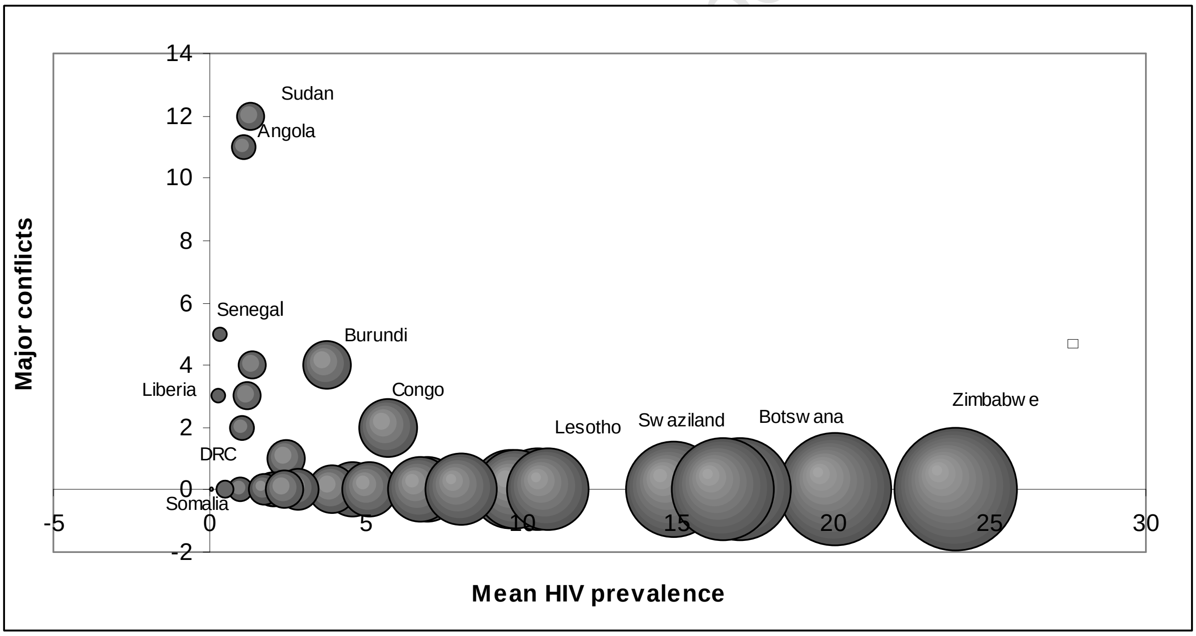
Abstract

In this chapter, I review the literature that suggests that conflict can exacerbate the HIV epidemic through sex with infected soldiers, war-related rape, poverty-related unsafe sex, transactional sex, etc. I also review the literature arguing the contrary. This literature argues that militaries do not always have higher prevalence rates than the surrounding populations and that war can disrupt sexual networks. What I draw from the literature is that the issue is complex and probably varies from case to case. Further, data collection during conflict is very difficult, as these conflict zones are either unreachable (for safety reasons) or violence afflicted people may not be willing to be surveyed or to provide an HIV test. These constraints mean that gross generalizations should be avoided.

5.1 Introduction

Africa has the distinction of being ravaged by a plethora of problems ranging from civil wars, widespread poverty and the HIV epidemic. In sub-Saharan Africa, it appears that counties afflicted by armed conflict and political instability have not been as hard hit by the HIV epidemic as the more stable ones. Putting aside the complexities of how countries are classified as “fragile” or in “armed conflict,” the evidence suggests that the link between armed conflict or state fragility and national level HIV prevalence rates is an extremely vexing one.

Figure 5.1 HIV and armed conflict in sub-Saharan Africa



Source: UNAIDS (2008) and UCDP (2010)[36]

Figure 5.1 shows the relationship between armed conflict and HIV prevalence in 30 sub-Saharan countries. The HIV prevalence rates shown are actually an average of the national estimates produced by UNAIDS for each country over 15 years (1990 - 2005). The conflict

[36] See online: http://www.pcr.uu.se/research/UCDP/data_and_publications/definitions... 2010/05/17 UCDP – Definitions

data shown is the total of major conflicts that each country had in the same time period. The data is the UCDP/PRIO major conflict variable, which is a dummy variable that assigns a value of 1 to countries that have had more than 1000 battle related [37]deaths during the year.

Granted each country's epidemic is a function of many other factors such as population density, religion, culture, the average standards of living and state of health care systems, Figure 5.1 suggests that it is countries that had more armed conflicts which had lower HIV prevalence rate estimates. These include predominantly Muslim countries such as Somalia as well as other countries like Angola. De Waal et al, (2010) show that by using existing indices of state fragility, those countries with the highest HIV prevalence are not ranked among the most fragile, and those considered most fragile do not report the highest rates of HIV. But rather than admitting the possibility that fragility might actually protect a country from the epidemic as has been argued by Iliffe (2006) , Melo, (2000), Strand, (2007) and others, De Waal et al, (2010:17, 24-18) propose that their result is a due to how fragility is measured. They argue that commonly used indicators of state fragility do not capture the impact of HIV and AIDS on local governance, human resources, service delivery and community survival (De Waal, 2010:15). These measures focus primarily on measuring national level institutions and so fail to capture the significance of local governance structures and local level service delivery which are the corner stone of any efforts to prevent the spread of HIV and the extension of prevention, care and treatment.

Many of the big global stakeholders in the HIV-armed conflict/fragility debate have in fact, tended to support the notion that these factors actually worsen the spread of the disease. Conventional opinion has been that the heavy militarization of fragile states and conflict zones on the continent has helped increase HIV prevalence (UNAIDS, 2003; Ba et al, 2008; Eric et al, 2003). Infection rates in African military personnel have been cited as being significantly higher than those among civilian populations, and therefore a cause of the spread of HIV in conflict zones (Abebe et al, 2003: 25-30: ICG, 2004). Postulated reasons for this are that military personnel tend to spend a lot of time away from home and have higher incomes than their surrounding communities making them potential clients for commercial sex workers (Matchaba-Hove, 2006: p.104). Armed conflicts also create environments where

[37] The definition of this variable and "battle related deaths" is discussed in Chapter 7.

women are prone to rape and abuse which further aids the spread of the disease (Carballo et al, 2001:2-6).

Indeed this view has received a lot of support especially from political circles with the distinguished Graça Maçhel, former Minister of Education in Mozambique and now wife of Nelson Mandela remarking, "*The chaotic and brutal circumstances of war aggravate all the factors that fuel the HIV/AIDS crisis. War breaks up families and communities, creating millions of refugees and placing women and children in great peril of sexual attack or systematic rape used to terrorise opposing forces*" (Maçhel, 2009). As stated in a 2001 United States Institute of Peace report, ''*No-one denies the role of conflict in the spread of the virus*'' (USIP 2001:8). Such views have been echoed by researchers such as such as Jewkes, (2007: 2140) and were first raised by the likes of Copley (1999:4). More recently, econometric models have also suggested that conflict might worsen HIV prevalence rates (UNAIDS estimates) at a country level (see Iqbal and Zorn, 2010, and Davenport and Loyle, 2009) – this is discussed in more detail in chapter 6.

However, a rival body of opinion suggests that the relationship between military conflict and HIV may be more complex than this. John Iliffe (2006: 10-18) suggests that the war and disruption of transport and communication networks in the Democratic Republic of the Congo (DRC) slowed down the epidemic by limiting the linkage of sexual networks across space. Aside from the DRC, the case of Angola is another where the country was "protected" from AIDS by nearly 40 years of war (Strand, 2007: 467-471). Other examples where HIV prevalence was very low during periods of military conflict include Liberia (Spiegel, 2008: 5), Ethiopia (Iliffe, 2006), and Mozambique (Melo, 2000: 203–207).

The rest of the chapter explores the broad evidence for and against these rival propositions about the relationship between HIV and armed conflict.

5.2 How military conflict may exacerbate the spread of HIV

The first school of thought argues that military conflict and the resulting social dislocation worsens the spread of HIV through the militarization of society, migration of infected people, breakdown of social norms and support structures, as well as the collapse of health and education infrastructure. This section will discuss each in turn.

5.2.1 Militarization

One of the key reasons why military conflict is assumed to exacerbate the spread of HIV is the belief that HIV prevalence rates in militaries are relatively high (Hankins, 2002; Elbe, 2002). Infection rates in Sub-Saharan Africa amongst the military have often been cited as being especially high, with claims that a number of militaries are experiencing rates above 50% (e.g. Eric et al. 2003: 578; ICG, 2004; UNAIDS 2003). The International Crisis Group was confident enough of this position to declare, "*It has been an accepted assumption that the rates of HIV are higher among the military and other uniformed forces than among the general population*" (ICG, 2004).

It has been argued that prevalence rates in the security forces are especially high because military culture and training encourage machismo, courage and a willingness to take risks: values that may encourage participation in riskier sexual behaviour (Tripodi, 2004: 196-200; Matchaba-Hove, 2006: 104). Troops are often away from home for long periods and may resort to commercial sex especially in regions where their disposable income is higher than those around them (Foreman, 2002; Abebe et al. 2003;Nwokoji et al 2004: 5; Spiegel, 2004: 223-30; Matchaba-Hove, 2006: 104; Plus News, 2009).

Ba et al. (2008) argued that HIV is a huge security threat given that estimates of the rate of HIV within African militaries are as high as 90%. After conducting a random effects pooled analysis on 21 African militaries, they found that HIV prevalence within the military is elevated compared to the comparable age cohorts in the general population (Ba et al. 2008: 90-98). In 1997, UNAIDS estimated that sexually transmitted disease rates among armed forces are generally 2 to 5 times higher than in civilian populations; the difference perhaps being even greater in times of conflict (Kavvoura et al. 2006: 79). The fact that young recruits may be socially inexperienced and that military culture tends to favour risk-taking behaviour is a reason brought forward to explain the high prevalence rates in the Zimbabwean military (Matchaba-Hove, 2006: 172). There is also the possibility of occupational infection through caring for the wounded and the possibility of receiving contaminated blood during emergency transfusions although this may be a relatively small risk (Matchaba-Hove, 2006: 104).

Although recent estimates are hard to come by, the literature indicates that in the early 2000s, about half of Zimbabwe's soldiers were HIV-positive (SAFAIDS, 2003; UNDP, 2003: 31; Carballo et al, 2003: 14). Carballo et al, (2003) present three fronts through which the military can affect the progression of the epidemic in the civilian population. First, young girls and women are at increased risk of sexual abuse by armed personnel during both conflict and peace times (Carballo et al, 2003: 11-15). This can be in the form of poor and vulnerable women and girls being drawn to military barracks and resorting to survival sex in search of money and food (Matchaba-Hove, 2006: 174). In countries like Zimbabwe, where unemployment rates are very high, military personnel often have higher incomes than the people in the surrounding communities (Matchaba-Hove, 2006: 174). In addition, many military barracks are single sex accommodation facilities meaning that when away from home for long periods; military personnel may visit local brothels (Matchaba-Hove, 2006: 174; Abebe et al. 2003: 25-27). The presence of large numbers of armed men in uniforms often means a sex industry springs up, increasing HIV risk for sex workers and uniformed services personnel (UNAIDS, 2004: 175). There are also (still on the first front) cases of sex in exchange for allowing women, especially cross-border traders with no documents, to cross checkpoints and borders (SAMP, 2005: 28; Dodson, 2000: 40-6).

A second front is the spread of the disease from military personnel to their partners upon their return from duty (PEPFAR, 2010; Medecins du Monde report, 2010; Calderon, 1997: 27). The third front, which actually feeds into the first two, is that of military personnel infecting each other. For instance, young female recruits in the military are at great risk of sexual abuse from their male superiors UNDP (2003: 29-33). Thus far, our discussion does not prove that heavy militarization results in a marked increase in HIV incidence and prevalence. However, it does bring to light the danger that military activity poses in the fight against AIDS/HIV. In other words, the level of militarization of a society could be a factor driving country-level HIV prevalence.

5.2.2 Migration and social disruption

Military conflict can also spread HIV through the resulting forced migration of refugees and the attendant social disruption. More specifically, HIV is likely to spread if HIV prevalence amongst the refugees is higher than in the destination areas and spread HIV there (Salama, 2001: 4-12; Smith, 2002: 38; Hankins, 2002), or if refugees flee from low-prevalence to high-prevalence regions and become infected (Becker et al, 2008: 2). Furthermore, displacement places people in chaotic circumstances where condoms are likely to be scarce and where social institutions and support structures which protect women from sexual abuse are disrupted (UNAIDS, 2004: 175).

Armed conflict can also increase the likelihood of exposure to HIV infection through the breakdown of traditional sexual norms (UNAIDS, 2004: 174). As Hankins et al. (2002) argue, the chaotic conditions associated with conflict can lead to the disintegration of traditional values and norms regarding sexual behaviour thereby leading to an overall increase in risk of HIV exposure (Hankins et al, 2002: 2245-52). Women and girls are particularly vulnerable in conflict situations. For instance, armed conflict can create conditions of such severe deprivation that women and girls, in particular, are coerced into exchanging sex for money, food or protection (UNAIDS, 2004: 175). These transactions can happen between unarmed men and women in refugee camps and other such civilian scenarios. Resultantly, this point can be seen as separate from the one mentioned earlier as to how women are particularly vulnerable to armed personnel in and around military barracks and establishments.

Rape has been used as weapon of war and subjugation across the world[38], most recently in Bosnia-Herzegovina, DRC, Liberia, and Rwanda (UNAIDS, 2004: 175; UN Dispatch, 2010; OHCHR, 2010). Assuming that prevalence rates in the armed forces are indeed higher than in the general population, these rapes would increase the risk of infection in the civilian population. A report on a study carried out in Rwanda revealed that 17% of women who had been raped tested HIV-positive, compared with 11% of women who had not been raped (de Waal, 2005: 8; UNAIDS/UNHCR, 2003). The Stop Rape Now Campaign, a United Nations initiative, reports that in some conflict areas, such as the DRC, it is not just the women who

[38] Here rape is viewed as a strategic/political tool and this is a separate point from that already raised of armed personal raping women for other reasons

have been abused and that young men and boys have also been raped (Stop Rape Now, 2010). This highlights the importance of broadening the discussion on the impact of sexual abuse on HIV incidence to include studies of how prevalent abuse of males is.

Other potential links between military conflict and HIV include the use of illicit drugs (to cope with the stress of war) which diminish responsibility and in the case of injected drugs, increase the risk of HIV directly through shared needles (Strathdee et al. 2002; Smith, 2002: 4-7; Hankins et al. 2002: 2250). However, given the low average income levels in most Sub-Saharan countries, it is unlikely that intravenous drug use is a major vector for HIV transmission in the armed forces.

The collapse of health systems that results from conflict almost inevitably worsens the progression of the HIV/AIDS epidemic. This is because when conflict triggers health system malfunction and collapse, national blood supply safety is threatened, and HIV prevention and care programs can disintegrate (UNAIDS 2004). AIDS programs are also poorly managed during conflicts and often under-staffed as skilled personnel leave the conflict zones (Ellman et al. 2005; Barnett, 2005). Although this might lead to lower prevalence rates, (as AIDS deaths increase because of shortages of antiretroviral drugs and other medications) they also may lead to higher prevalence rates, as preventative programs (including condom distribution) are incapacitated and HIV incidence rises. Resistant forms of the virus may also arise as fighting undermines the supply of antiretroviral drugs to conflict regions. This means that those who would have started taking these drugs often find it hard to get them resulting in them skipping doses and this would greatly increase the chance of them developing resistance. That the prevention and care of HIV patients is greatly diminished as a direct result of conflict is further evidenced by the fact that sexually transmitted infections (STIs) often rise in post-conflict situations (Betsi et al 2004: 363).

5.3 How military conflict may slow the spread of HIV

In contrast to the evidence and hypotheses presented above, a rival literature maintains that the relationship between HIV and military conflict is complex, often contradictory, and possibly even protective.

5.3.1 Is HIV in African militaries really higher than in civilian populations?

Spiegel et al. (2007) and Whiteside et al. (2006) observe that because data on the military are difficult to obtain and verify, the analysis of prevalence in militaries is especially challenging. They point out that in the case of South Africa, HIV prevalence in the military may actually be lower than that in the civilian population (Whiteside et al. 2006: 202). Population based surveys such as South Africa's 2004 Reproductive Health Research Unit (RHRU) survey of 15-24 year olds shows that in the general population, HIV prevalence was 7.3% for males (aged 15-19) compared to 24.5% for women (aged 15-19) (Pettifor, 2004: 31). They thus conclude that because the majority of recruits are males between the ages of 17-22, most South Africa army recruits probably have lower prevalence rates than the average national (male plus female) adult prevalence rates (Whiteside et al. 2006: 202)

However, the argument by Whiteside et al. (2006) is not convincing in that it does nothing to clarify whether men in the SA military have higher prevalence than their similarly aged male counterparts in the general population. Indeed some reports have argued that infection rates in the South African military are slightly higher than among the general population (Plus News, 2006). As much as 40% of the South African military was reported to have been HIV positive in 2001 (compared to 16.9% among 15-49 year olds in the general population) (Lovgren, 2001: 1).

Whiteside et al. (2006: 203) also suggest that HIV prevalence in African militaries is a function of the number of years of service. This makes it hard to make generalizations about HIV prevalence in armies with different age structures and typical length of service. They argue that the longer serving officers have often been more prone to infection owing to exposure to higher prevalence areas during multiple foreign missions (Whiteside et al, 2006: 203). Evidence from a 1989/90 study of Nigerian troops returning home from foreign missions in Liberia and Sierra Leone found that infection rates in these peace keepers were more than double of those in non-peace keeping troops (Whiteside et al, 2006: 206). The study also showed that a service member's risk of infection doubled each year spent on deployment in war zones (Fleshman, 2004: 10). This implies that even if young recruits came in with lower prevalence rates, after more years of service in the military, they are likely to have infection rates comparable to or even higher than that of the general population (Whiteside et al, 2006).

Secondly, there are reports of some militaries, notably Uganda, screening recruits for HIV to exclude infected applicants (Whiteside, 2006: 203). In countries like South Africa and Namibia, such measures are illegal and there have been court cases debating the constitutionality of such screening (South African AIDS Law Project, 2003). The screening of recruits would mean that HIV prevalence in the army on entry would be minimal (Whiteside, 2006: 203). A case in point is the HIV screening system that was in place in Ethiopia between 1998 and 2000. Only those testing negative, (93% of all candidates tested) were admitted to the army (Abebe et al, 2003: 1835-40). As a result, HIV prevalence in the Ethiopian military during this mobilization would probably have been as low as 2.8% (Whiteside et al, 2005: 203; Berhe, 2005: 107–14). In other words, the issue of HIV prevalence in the military is complex and probably varies from case to case. Simplistic claims about HIV prevalence always being higher in the military should be avoided.

5.3.2 Military Conflict may help prevent the spread of HIV

Iliffe (2006) suggests that war can have a protective effect on an HIV epidemic by disrupting transport and communication networks. He argues that this was the case in the DRC where war prevented the linking of sexual networks between Kinshasa (the epicentre of the epidemic) and the surrounding forested areas (Iliffe, 2006: 10-18). This argument for a ‘protective’ effect of war has been bolstered by evidence from empirical studies in other countries like Angola, Liberia and Mozambique where HIV prevalence was subdued during the conflicts in those regions, but started rising in the post conflict reconstruction period (Spiegel, 2008: 5; Melo, 2000: 203-207: Strand, 2007: 467-471; Plus News, 2010; WHO, 2000).

Mock (2004) has observed that in most cases, conflict countries seem to have lower levels of HIV infection than those with relative peace (Mock, 2004: 6). One of the most significant contributions to the debate on whether conflict lowers HIV prevalence in affected areas was by Spiegel et al (2007). They compared HIV prevalence in populations directly affected by conflict with that in those areas not directly affected. They also compared HIV prevalence in refugee camps and in the closest surrounding host communities in sub-Saharan Africa. Their search of the international literature obtained 295 articles on conflict and HIV prevalence in Africa which identified seven countries with a history of widespread conflict that had original data on HIV prevalence in the five years from 2001 to 2007 (Spiegel et al., 2007: 2188). These were the DRC, Sudan, Rwanda, Uganda, Sierra Leone, Somalia, and Burundi (see Tables 5.1 – 5.3).

Table 5.1 Prevalence of HIV infection in eastern DRC (2004) and in nearest neighbouring country sentinel sites

	Prevalence (95% Confidence Interval)	Neighboring country and nearest site	Prevalence
DRC			
Bukavu (urban)	3.1 % (1.9-5.1)	Burundi , Kayanza (semi-urban)	10.2%
		Muramvya (rural)	14.7%
Bunia (urban)	3.2 % (2.0-5.1)	Uganda, Arua (rural)	5.2%
Goma (urban)	5.4 % (3.8-7.6)	Rwanda, Gisenyi (rural)	7.1%
Kindu (urban)$^{+}$	3.7 % (2.4-5.8)		
Kisangani (urban)$^{+}$	6.3 % (4.4-8.8)		
Lodja (rural)$^{+}$	6.6 % (4.8-9.1)		
Neisu (rural)	6.7 % (4.7-9.2)	No site near border in Sudan	
Karawa (rural)	4.5 % (2.9-6.6)	Central African Republic, Bangassou (rural)	9.0%

Source: Spiegel et al (2007)

Table 5.1 shows that HIV prevalence in both rural and urban sites in the DRC was generally lower than that in comparable sentinel sites in neighbouring Uganda and Burundi (Spiegel et al, 2007: 2189). The DRC rural sites of Lodja, Neisu and Karawa had lower prevalence rates than the Arua (Uganda), Gisenti (Rwanda) and Muramvya (Burundi). This speaks to the argument by Iliffe (2006) and others which suggests that conflict actually protects against an explosive HIV epidemic. Even the semi-urban Kayanza site in Burundi had higher prevalence rates than the DRC's Bunia, Bukavu and Goma urban sites.

Table 5.2 shows that Burundian refugees at the Mtabila and Muyovosi camps had higher prevalence rates than that in the comparable Kigoma region of Tanzania. These are exceptions to the rule as all the other refugee camps in Tanzania show that prevalence was lower than in the host Kagera population. Resultantly, Spiegel et al, (2007: 2192) conclude that there was insufficient evidence that HIV prevalence was greater in refugee populations (from Burundi and DRC) than in the host population (Tanzania) Most of the refugee sites (in the column to the left) show much lower prevalence rates than the host sites to the right.

Table 5.2 Prevalence of HIV infection in Tanzania (refugee host country)

	Prevalence 95 % CI	Year	Host population (Refugee camps in Tanzania)	Prevalence 95 % CI	
Burundian refugees Mtabila and Muyovosi Camps	1.70%	2001	Kigoma	2.00%	2003
	4.50%	2003	region*[1]		
Nduta and Mtendeli	1.30%	2001			
	1.60%	2002			
	1.70%	2003			
Lukole Camp	4.80%	2001	Kagera	3.70%	2003
	3.10%	2002	region*		
	1.60%	2003			
DRC refugees Lugufu and Nyaragusu Camps	1.00%	2001	Kagera region*	3.70%	2003
	2.50%	2002			
	1.80%	2003			

Source: Spiegel et al (2007)

Table 5.3 supports the contention that there is insufficient evidence that HIV transmission is higher in refugee populations (refugees from Eritrea, Somalia etc. hosted in Rwanda, Sudan Kenya and Zambia) than in comparable sites in the host country (Spiegel et al, 2007: 2192). Most of the conflict sites (in the column to the left) show much lower prevalence rates than the host sites to the right. Another observation they made is that there was no evidence that the extensive rape incidences reported in a number of sites in Rwanda, Sierra Leone and the DRC fuelled the epidemic (Spiegel et al, 2007: 2193).

Data collection during conflict is very difficult, therefore Tables 5.1 – 5.3 should be treated with caution because violence afflicted people may not be willing to be surveyed or to provide an HIV test (Spiegel et al, 2007:2192- 2193). In other words, uncertainties with data quality mean gross generalizations should be avoided.

Table 5.3 Prevalence of HIV infection in Rwanda, Sudan Kenya and Zambia (refugee host countries).

	Prevalence 95 % CI	Year	Host population	Prevalence 95 % CI	
DRC refugees in Rwanda Gilembe camp	1.50% (0.4-3.8)	2002	Byumba site	6.70% (4.7-9.4)	2002
DRC refugees in Zambia Mwange camp Kala camp	1.20% 3.40%	2005 2005	Nchelenge site	18.90%	2002
Eritrean refugees in Sudan Several camps in eastern Sudan	4.10%	2002	El Gadarif site	4.00%	1998
Sudaneese refugees in Uganda Palorinya settlement Kyangwali settlement	1.00% (0.3-1.8) 2.70% (1.3-4.0)	2004 2004	Surrounding population Moyo site Surrounding population Hoima site	5.90% (1.7-10.1) 4.30% 2.80% (1.0-6.6) 4.60%	2004 2002 2004 2002
Sudaneese refugees in Kenya Kakuma camp	5.00% (3.5-7.0)	2002	Lodwar site	18.00%	2002
Somali refugees in Kenya Dadaab camps	0.60% (0.01-1.1) 1.40% (0.5-2.2)	2003 2005	Garissa site	26.00% 11.00%	2002 2004

Source: Spiegel et al (2007)

The argument that conflict helps spread the epidemic may hold true in certain countries but as we have seen, the counter argument put forward by Whiteside et al. (2006), Spiegel et al, (2007), Iliffe (2006) and others has some empirical backing. Evidence from the above study by Spiegel et al (2007) leans towards the Iliffe view that military conflict may have helped slow the HIV epidemic in Africa. However, the approach used did not provide an analysis of the extent to which each country's level of governance and the strength of state institutions may have influenced these community-level results. As de Waal et al. (2010) point out, the sheer diversity of HIV epidemics particularly in states considered to be fragile point to the importance of tracing context-specific links between HIV and weak governance. In as much as it is important to "know your epidemic", knowing your social, economic and political context is equally important (Buse et al, 2008; De Waal et al. 2010:34).

5.4 Conclusion

In this chapter I reviewed two opposing arguments regarding the nature of the relationship between HIV and armed conflict. One suggests that armed conflict increases the spread of HIV (through rapes, the breakdown of support networks, the associated increase in survival sex, movement of militaries and refugees into new areas etc.). On the other hand, some argue that armed conflict reduces HIV rates by disrupting the economic and social networks that spread HIV. An emerging consensus appears to be that it is important to understand the political social and demographic characteristics of particular epidemics – i.e. the 'know your local epidemic' argument. Nevertheless, there are a couple of bold attempts to pin down a general relationship between armed conflict and HIV prevalence. I review these in chapter 6 and then attempt to push this line of enquiry further in chapter 7.

6 A FURTHER REVIEW OF THE LITERATURE: THE USE OF HIV PREVALENCE ESTIMATES IN REGRESSION ANALYSIS

Abstract

The use of regression models to investigate the extent to which conflict affects the HIV epidemic is a fairly new development. In this chapter, I review the work by Iqbal and Zorn (2010), Davenport and Loyle (2009 and Paxton (2009). The three econometric studies use different model specifications and find dissimilar results. Iqbal and Zorn as well as Davenport and Loyle find a positive relationship between interstate and intrastate armed conflict and HIV prevalence (i.e. their work supports the view that conflict can exacerbate the HIV epidemic). I argue that there are methodological difficulties with such studies – especially with regard to the assumption that a linear relationship exists between the dependent and independent variables – when, as shown in Chapter 2, HIV prevalence data are generated by demographic models which assume that HIV prevalence follows a curvilinear pattern over time. Other conceptual concerns are also highlighted in the analysis. The study by Paxton (2009) finds a positive (statistically insignificant) relationship between HIV prevalence and interstate conflict. Further, Paxton also finds a statistically significant negative relationship between intrastate (domestic) conflict and HIV prevalence. I also discuss conceptual concerns regarding Paxton's work and how his work differs from that by Iqbal and Zorn.

6.1 Introduction

The previous chapter showed that there has been much interest in the relationship between HIV and conflict. But in the main, the literature has comprised national reports and country specific recommendations and has not specifically been intended to evaluate the conflict-HIV relationship across time and space (Davenport and Loyle, 2009). Much of the work done has been informative and suggestive rather than quantitatively rigorous. This absence of rigorous econometric literature on the link between HIV and armed conflict is not surprising given the possible reverse causality between the two as well as the spatial and temporal aspects of the disease (Iqbal and Zorn, 2010).

In the wake of the fascinating and predominantly qualitative research, three econometric studies have emerged that have tried to model the armed conflict-HIV nexus. The first of these was by Iqbal and Zorn (2010). Davenport and Loyle (2009) reviewed the work by Iqbal and Zorn and also presented a different modelling approach. The work by Iqbal and Zorn actually came before that by Davenport and Loyle although Iqbal and Zorn were only published a year after Davenport and Loyle (hence the somewhat confusing publication dates). Davenport and Loyle (2009) however seem to ignore another econometric paper by Paxton (2009) published by Harvard University in January 2009. Before attempting my own econometric modelling (in the next chapter), I take time here to discuss the modelling strategies adopted by these scholars.

Iqbal and Zorn (2010) use Ordinary Least Squares (OLS) regression models to look at the link between HIV prevalence (as estimated by UNAIDS) and armed conflict in 43. African countries for the years 1997 to 2005. When they pool all the data together, they find that the occurrence of international military conflict (i.e. wars between countries) has a large and statistically significant positive relationship with HIV prevalence. The occurrence of domestic conflict (i.e. intrastate / armed conflict within countries) was not, however, statistically significantly associated with HIV prevalence.

Davenport & Loyle (2009) build on Iqbal and Zorn (2010) by presenting their own econometric model. However, they measure conflict using variables for the duration, magnitude and geographic scope of each conflict. They also included other contextual variables like the health expenditure of the each of the 197 countries in their sample. Their study found that the direct effects of conflict, measured in magnitude, duration and geographical scope, had a positive, but statistically insignificant relationship with HIV prevalence. Davenport & Loyle (2009) find this result to be consistent for their global dataset as well as one for a sub-Saharan Africa one. These papers thus provide a counter-weight against arguments that armed conflict has a protective effect in reducing the spread of HIV.

Although Davenport and Loyle (2010) do acknowledge it, their work and that of Iqbal and Zorn does not adequately address the issue of how the dependant variable, HIV prevalence is measured, estimated and how best to use it in econometric analysis. As discussed in Chapter 2, UNAIDS HIV estimates are obtained from ANC data using the EPP and Spectrum modelling packages. The estimates are thus already the product of modelling and should be acknowledged as such, rather than implicitly treated as hard data points. Addressing the implication of using UNAIDS HIV estimates in regression analysis is a gap in the literature that needs to be filled. There is a great temptation to fixate on figuring out how other independent variables or factors were or indeed on finding out how best to modify or adjust the variables used. But far too little attention is paid to understanding what each model implies or assumes on the part of the dependent variable (HIV prevalence estimates).

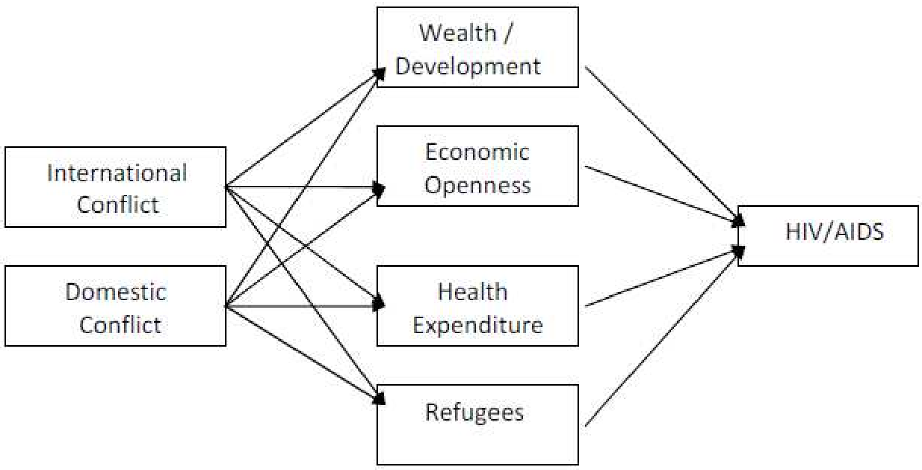
Paxton's work which precedes that of Davenport and Loyle uses very parsimonious panel data Fixed Effects and Random Effects regression models. The merits and demerits of Fixed Effects and Random Effects models are discussed in detail later on in the chapter. In the models he presents, Paxton (2009) finds a significant negative association between HIV prevalence and intrastate / domestic conflict. He also finds the relationship between HIV prevalence and interstate conflict to be positive but statistically insignificant. The lack of consistency in the results from these three econometric attempts has led to some including the AIDS, Security and Conflict Initiative (ASCI) dismissing population-level econometric analysis as a useful methodology for investigating the link between armed conflict and HIV prevalence (see de Waal et al, 2010).

6.2 Taking a first stab: Iqbal and Zorn (2010)

6.2.1 Overall argument and conceptual framework

As shown by Figure 6.1 Iqbal and Zorn argue the link between conflict and HIV is best understood by distinguishing between international armed conflict and domestic armed conflict.

Figure 6.1 The Iqbal and Zorn framework



Source: Iqbal and Zorn (2010)

The authors posit that international and domestic armed conflict influence the progression of the epidemic in two ways. Through the movement of displaced people and that of troops from one country to another, conflict increases the chances of infected and uninfected people interacting. The displacement of non-combatant/civilian populations is a main feature of armed conflict. Civil wars are also particularly notorious for resulting in large outflows of refugees (Davenport et al, 2003; Moore and Shellman, 2004). Chapters 2 to 4 show using Zimbabwe as a case study that reasons for migration can be varied as people can flee their home countries not only on account of the violence that conflict brings, but also in cases where the socio economic situation of a country deteriorates. Displaced populations will invariably be prone to disease and infection. Lurie et al. (2003) note a correlation between the presence of migrant populations and increasing HIV infection while Decosas and Adrien

(1997), argue a similar correlation between refugee flows and HIV infection. This raises a few concerns as will be discussed later in this chapter.[39]

The second point Iqbal and Zorn make is that during conflict, not only do troops come into contact with other populations (refugees or otherwise) but this contact is often in a context that makes HIV/AIDS transmission more likely. They present arguments similar to those discussed in the previous chapter (5) about how soldiers are of a sexually active age and being away from home for extended periods of time makes them more likely to engage in casual sexual relations. They also bring up arguments about how the military valorises violent and risky behaviour (which might lead to rapes etc.) (Iqbal and Zorn, 2009:9).

Other factors are controlled for in their analysis. They argue that the level of economic and infrastructural development and economic openness are linked to domestic resources for HIV prevention programs. They control for factors such as religion, GDP per capita, population density and the level of education. They assume that population density is likely to raise the prevalence of HIV infection. They explain that more densely populated areas are likely to experience higher spread of HIV since the levels of interaction among people in such areas often exceeds those in less dense areas (Iqbal and Zorn, 2010: 153). This might be a reason why HIV prevalence rates are higher in urban than in rural areas (Buve´, Bishikwabo-Nsarhaza, and Mutangadura, 2002).

Iqbal and Zorn also argue that communities in which sexual behaviour is generally governed by strict religious standards that prohibit casual sexual relationships are likely to have lower rates of sexually transmitted infections (e.g. Allain et al. 2004). They therefore expect a negative relationship between Islam and HIV prevalence since Muslim countries generally have lower HIV prevalence rates (Iqbal and Zorn, 2010: 153).

In including education, Iqbal and Zorn argue that educated individuals respond better to HIV prevention programs and are likely to make informed decisions regarding safer sexual practices (Peterman, Lindsey, and Selik, 2005). Education also improves economic opportunities and therefore reduces the likelihood of women engaging in prostitution and

[39] While the theory here is very intuitive, Chapter 5, and in particular, Spiegel et al (2007) warns that emerging evidence is to the contrary.

perhaps other forms of transactional sex. Regime type is also controlled for because good governance and democracy has been linked to more positive health outcomes (e.g., Przeworski, Cheibub, and Limongi, 2000). Stable countries generally prioritize health and other social expenditure as opposed to less stable countries which are likely to prioritise defence and security. For instance, Ghobarah et al, (2004a) show a link between levels of democratic governance and health spending. Consequently, Iqbal and Zorn expect that democratic states where security is not a priority, will have lower prevalence rates given their capacity to avail resources to combat the factors that increase the spread of the epidemic (Iqbal and Zorn, 2010:154).

Economic openness also allows a country access to external markets and its ability to mobilize foreign resources. These then have an impact on the extent the epidemic can be kept in check. However, openness determines whether the country will have significant number of people crossing its borders and this may increase or reduce prevalence rates depending on whether they come from lower or higher prevalence countries.

6.2.2 Research design

Using HIV and armed conflict data on 43 African countries for the years 1997 to 2005, Iqbal and Zorn apply Ordinary Least Squares (OLS) regression models to operationalize their framework. They do not provide a list of countries in their study so it is impossible to replicate their results. In prepping their data for analysis, Iqbal and Zorn find that spatial correlations was more pronounced in the north and the south of Africa where they found strong relationships between infection rates from one nation to the next (Iqbal and Zorn, 2010: 154). Spatial correlation was however, lower in central and western Africa, where cross-country rates were found to be more variable (Iqbal and Zorn, 2010: 154). Resultantly, they estimate a pooled OLS model, a spatial lag model as well as a spatial error model.

The spatial error model is one that assumes that it is the error terms (or disturbances) across spatial units (countries) which are correlated. This means that the OLS assumptions are violated and the estimates are inefficient. The spatial lag model assumes that the dependent variable HIV prevalence (estimates) for one country are affected by the independent variables in both that country and other countries, especially its neighbours.. This again violates the OLS assumptions rendering the estimates biased and inefficient.

They also lag their conflict variables to accord with their hypothesis that conflict has a causal effect on HIV prevalence, but not immediately. Iqbal and Zorn use HIV prevalence estimates obtained from the *Report on the Global HIV/AIDS Epidemic* (2002) as released by the World Health Organization (WHO) and UNAIDS. The key objective of Chapters 2 to 4 was to illustrate how these estimates are obtained for each country and to highlight how national estimates are the modelled combined effects of the sub-epidemics in each country. The treatment of these HIV prevalence estimates as if they are actual data points as is done in the interpretation of the results from both by Iqbal and Zorn (2010) and Davenport and Loyle (2009) is also particularly worrisome.

The Davenport and Loyle measure of armed conflict is taken from the International Peace Research Institute in Oslo (Gleditsch et al. 2002) and includes all armed conflict resulting in at least 25-battle deaths per year. Iqbal and Zorn (2010) also use this dataset to distinguish between domestic and international conflicts[40]. Drawing from various sources in the cross national social science literature, they also include variables proxying for wealth or development (for this they use GDP per capita), education, regime type (lagged), economic openness, heath expenditures, refugees, population density, and culture (Muslim religion). How each of these variable is calculated and the source of the data are shown in Table 6.1

For instance, Population density is measured as the natural logarithm of population per square kilometre while the Muslim Population variable measures the percentage of each country's population that is Muslim (Iqbal and Zorn, 2010).   Economic openness, was measured calculating the natural logarithm of the sum of total imports plus exports, as a percentage of total GDP while Education is measured as the percentage of adults (age 15+) in the population who are literate (Iqbal and Zorn, 2010). To measure the level of influx of refugees, they use the natural logarithm of stocks of refugees present in the country for that specific year (Iqbal and Zorn, 2010) in question. It is worth noting how the conflict variables are measured as they are the primary independent variables of interest. The conflict variables are

[40] See online: http://www.pcr.uu.se/research/UCDP/data_and_publications/definitions... 2010/05/17 UCDP – Definitions

4-point ordinal scales that show both the presence and the intensity of internal or external violent conflict. More than 1000 battle related deaths in every year of a conflict indicates a major conflict (i.e. value 3 on the scale).

Table 6.1 Variable sources and how the variables are used

Variable	Operationalization (Source)	Mean	Standard Deviation	Expected Influence
Year	Recoded to 1997 = 1	5.18	2.84	Negative
Population Density*	Population per km^2 (World Bank)	74.8	102.2	Positive
Muslim Population	Muslim percent of the population (CIA World Factbook 2008)	36.4	33.8	Negative
Education	Adult literacy rate (WDI, UNESCO 2005)	58.6	18.1	Negative
Democracy$_{t-1}$	Lagged POLITY score (Marshall and Jaggers 2004)	0.70	5.32	Negative
Wealth/Development*	Per capita GDP, in US $s (World Development Indicators)	2380	3096	Negative
Economic Openness*	$\frac{\text{Imports+Exports}}{\text{GDP}} \times 100$ (World Development Indicators)	68.9	31.4	Negative
Health Expenditures	Public and private health expenditures, % of GDP (WDI)	4.81	1.75	Negative
Refugees*	Refugees present, in thousands (UNHCR *Statistical Yearbook*)	57.8	105.7	Positive
International Conflict$_{t-1}$	Lagged Armed Conflict Score (Strand et. al. 2003)	0.03	0.16	Positive
Domestic Conflict$_{t-1}$	Lagged Armed Conflict Score (Strand et. al. 2003)	0.19	0.46	Positive

Source: Iqbal and Zorn (2010). The asterisks represent variable which are logged

6.2.3 Results

In interpreting their results, Iqbal and Zorn state that Table 6.2 can be interpreted by viewing the coefficients as elasticities or percentage changes in the dependent variable. The dependant variable itself is a percentage change but they opt to interpret their results as changes in predicted HIV/AIDS rates in a typical median country where the prevalence rate is 5% (Iqbal and Zorn, 2010:158). In this median or typical country, a one-unit increase in international conflict increases expected HIV/AIDS rates by 5.7% in the spatial lag model and 5.9% in the spatial error model.

In their full models, (pooled OLS, spatial lag and spatial error) Iqbal and Zorn find that while the effect of domestic conflict is consistently small and statistically significant, that of international conflict is large, positive and statistically significant in most cases. This can be interpreted to mean that while domestic conflict has little impact on national level HIV prevalence, interstate conflict seems to be associated with higher HIV prevalence rate estimates.

Table 6.2 Iqbal and Zorn (2010) model results

Dependant variable: Logged UNAIDS HIV prevalence estimates (1997-2005)

	Reduced-Form Models			Full Models		
Variable	OLS	Spatial Lag	Spatial Error	OLS	Spatial Lag	Spatial Error
(Constant)	4.27 (0.84)	2.33 (0.75)	2.18 (0.74)	0.54 (2.44)	−2.29 (1.46)	−0.67 (0.37)
Year	−0.02 (0.02)	−0.02 (0.03)	−0.02 (0.03)	−0.05* (0.02)	−0.05* (0.02)	−0.03 (0.03)
Population Density	−0.19 (0.11)	−0.26 (0.06)	−0.28 (0.07)	−0.24 (0.09)	−0.27 (0.05)	−0.24 (0.07)
Muslim Population	−0.04** (0.006)	−0.02** (0.006)	−0.03** (0.004)	−0.03** (0.005)	−0.02** (0.004)	−0.03** (0.003)
Education	−0.016 (0.010)	−0.009* (0.005)	−0.001 (0.006)	−0.018* (0.011)	−0.016** (0.006)	−0.017** (0.006)
Democracy$_{t-1}$	−0.025 (0.037)	−0.013 (0.018)	−0.018 (0.018)	−0.019 (0.027)	−0.006 (0.014)	−0.034* (0.019)
Wealth/ Development$_{t-1}$	—	—	—	−0.24 (0.22)	−0.17 (0.11)	−0.18* (0.08)
Economic Openness$_{t-1}$	—	—	—	0.76* (0.37)	0.99** (0.22)	0.72** (0.14)
Health Expenditures	—	—	—	0.28 (0.07)	0.27 (0.04)	0.20 (0.05)
Refugees$_{t-1}$	—	—	—	0.14* (0.08)	0.11** (0.03)	0.12** (0.03)
International Conflict$_{t-1}$	0.46 (0.29)	0.70* (0.33)	0.82* (0.39)	0.50* (0.28)	0.76** (0.27)	0.78** (0.29)
Domestic Conflict$_{t-1}$	0.18 (0.17)	0.08 (0.16)	0.24 (0.15)	0.33* (0.14)	0.34* (0.15)	0.33* (0.16)
$\hat{\rho}$	—	0.11 (0.04)	—	—	0.11 (0.03)	—
$\hat{\lambda}$	—	—	0.04 (0.02)	—	—	0.23 (0.08)

Source: Iqbal and Zorn (2010)[41]

As Table 6.1 shows, their model also finds consistent results across specifications in respect to the Muslim and education variables (Iqbal and Zorn, 2010:158). They find that Muslim countries and those with relatively high adult levels of education/ literacy are associated with lower HIV prevalence rates. Iqbal and Zorn find that for a typical country (with median HIV prevalence rate at 5%), each 10% increase in the Muslim population corresponds to a predicted decrease in the HIV/AIDS rate of roughly 1%. They also find that each 10% increase in adult literacy decreases the predicted prevalence rate by roughly three-quarters of 1%. The complete models (with all variables) show that wealth/GDP per capita is negatively

[41] Note: NT = 181. Numbers is parentheses are robust (White 1980) standard errors. One asterisk indicates p < 0.05, two indicate p < 0.01 (0ne-tailed, except for Economic Openness, where tests are two tailed.

related to HIV prevalence. They find that a one percentage increase in GDP lowers expected HIV/AIDS rates by nearly 1% (ibid, 2010:158). Health expenditures are however, positively related to HIV prevalence rate estimates. These expenditures are most likely responding to rather than driving the high levels of HIV prevalence. Finally, their model shows a positive effect of refugee influxes that is consistent across all three models (OLS, spatial lag, and spatial error models). This is in line with their theoretical framework (see Figure 6.1).

6.2.4 Limitations

One of the biggest limitations of the work by Iqbal and Zorn is their choice of UNAIDS/WHO HIV prevalence rate estimates as their dependent variable. Chapter 2 illustrated how these are obtained and warns about their uncritical use in any analysis that tests aggregate level relationships.

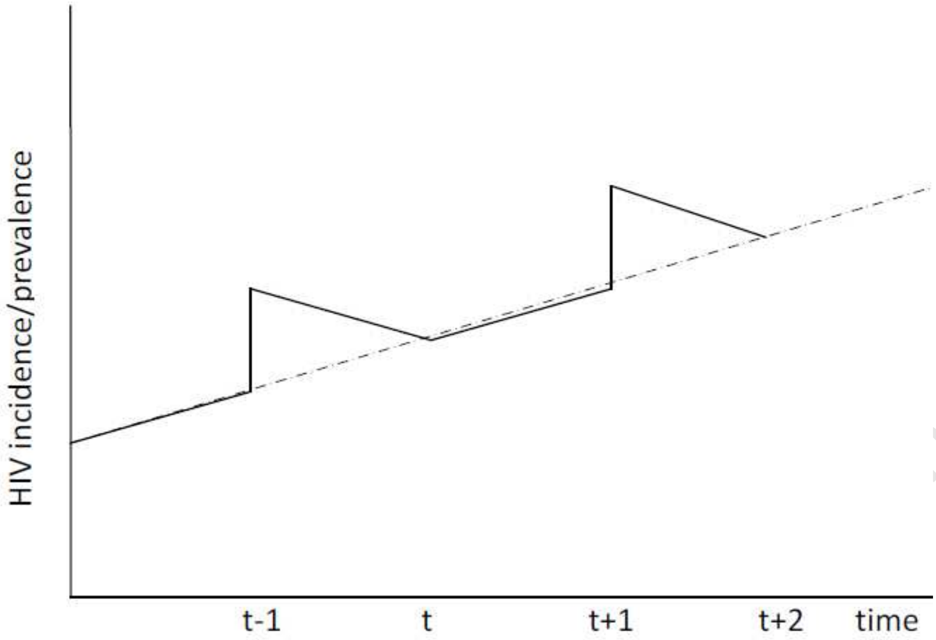
6.2.4.1 HIV prevalence estimates

Firstly, let us consider the use of UNAIDS/WHO HIV prevalence rate estimates that rely on antenatal surveys in host countries. Each country has different capacity and quality constraints which compromise the comparability of the estimates across countries. In conflict countries, some regions have been unstable for so long that it is conceivable that they have lost the capacity to collect such data in any systematic manner. Furthermore data collection in conflict areas is very difficult. For instance, violence afflicted people may not be willing to be surveyed or to provide an HIV test (Spiegel et al, 2007:2192- 2193). This would reduce the sample sizes for the ANC estimates. The margins of error around those HIV estimates are thus likely to be wide. Even when they are accurately collected, a country typically has different sub-epidemics (as shown for Zimbabwe in Chapter 2). A second potential source of error is introduced when these sub-epidemics are brought together by EPP/Spectrum algorithms and assumptions to produce national estimates.

Davenport and Loyle (2009: 11) argue that UNAIDS estimates of HIV prevalence are not comparable over time – supposedly because of yearly updates to the UNAIDS estimation models. This, however, would only apply if one takes HIV estimates from different UNAIDS reports, rather than using the most recently generated projections of HIV prevalence over time (which by that very construction are consistent over time). Their comment thus suggests a lack of appreciation of how UNAIDS HIV prevalence estimates are derived. Today's estimates for each country over time are derived using EPP/Spectrum modelling which produces one curve over time. This was one of the main points illustrated in the modelling done in Chapter 2. These curves and estimates are what are then presented in the UNAIDS/WHO Report on the Global HIV/AIDS Epidemic (2008). This means one can choose to take estimates of HIV prevalence for any country for the years 1997 to 2005, all from one curve, based on the same model assumptions – and hence that the HIV prevalence rates are consistently estimated over time and are thus 'comparable'. The disadvantage, however, is that the HIV estimates are very much the product of demographic modelling, and thus reflect curvilinear assumptions about their path over time. As discussed in more detail below, their modelled smooth path over time thus introduces noise into subsequent attempts to isolate the impact of particular events, such as armed conflict, on HIV prevalence.

Iqbal and Zorn use a linear model which, by its very design, assumes that armed conflict in year t will affect HIV prevalence or incidence in the following year (t+1) and that if year t+1 is not a conflict year, that HIV prevalence goes back down in year t+2. However, the modelled nature of HIV prevalence rates means that such clear up and down trends will never be evident. This is because even if hypothetically in a particular country, HIV prevalence rates had declined in the year after a conflict year and that this was reflected in a dip in ANC prevalence rates, the country's *estimated* HIV prevalence rates would have eliminated the dip through the smoothing curvilinear modelling methodology of EPP and Spectrum. Hence, any researcher using a linear model to regress variables on UNAIDS-generated HIV prevalence rates over time, is less likely to find a relationship between the independent variables and HIV prevalence than would be the case if the dependent variable had comprised *real* data points. Researchers who forget, or do not have an adequate appreciation of the nature of estimated HIV prevalence rates – *and their path over time* – are expecting too much of their linear models.

Figure 6.2 Conflict shocks to the model (assuming a positive relationship between HIV prevalence and conflict)



Source: Own conceptualisation

Figure 6.2 shows how the OLS regression framework assumes that HIV prevalence is affected by armed conflict. For the purposes the exercise this diagram assumes that the relationship between HIV prevalence and conflict is positive. The dashed line shows the assumed non conflict related momentum of the HIV epidemic. Shocks are assumed to affect HIV prevalence the following year (since all conflict variables are lagged) but going back down in the next. The curvilinear modelled nature of HIV prevalence estimates act to confound such attempts to model the relationship between HIV prevalence and conflict using linear OLS models because the EPP/Spectrum modelling approach smoothens out such disturbances if and when they take place. An alternative approach is to aggregate HIV data over time (as done by Davenport and Loyle) or to experiment with different dynamic specifications of the model (see Chapter 7). But while such approaches are better, they nevertheless suffer from the same problem of using a dependent variable that is itself already the product of demographic modelling.

6.2.4.2 The independent variables: additional criticisms

The Iqbal and Zorn (2010) model suggests that there are differences between domestic and interstate types of armed conflict without looking at how the duration or intensity of each episode of armed conflict affects their model. This, makes their model incomplete (Davenport and Loyle, 2009:11), Another concern is that their theoretical approach takes a snap shot of the countries over the same stretch of time without accounting for the conflict histories of each country. The time period chosen, 1997-2005, will not capture the impact of conflict in earlier years. Furthermore, the model implicitly assumes that the HIV epidemics are at the same stage, whereas in practice, countries with older epidemics (those closer to the DRC) are more likely to have constant or falling HIV rates, whereas the new ones (in Southern Africa) are more likely to be experiencing rising epidemics.

The conceptual framework presented by Iqbal and Zorn makes an important conceptual contribution in that it includes the impact of refugee populations on host countries. However, their measure is crude in that they simply use the total number of refugees in the host country as their proxy – when it would have been much better (and would have aligned better with their own framework) if they had distinguished between refugees from higher prevalence (higher than the host) countries and those from lower prevalence countries (Davenport and Loyle, 2009). This is important because HIV prevalence would only rise in the host country if refugees come from predominantly higher prevalence nations. Another weakness of the Iqbal and Zorn study is that they do not provide a list of countries in their sample or indicate how the countries were selected. This makes it impossible to replicate their results.

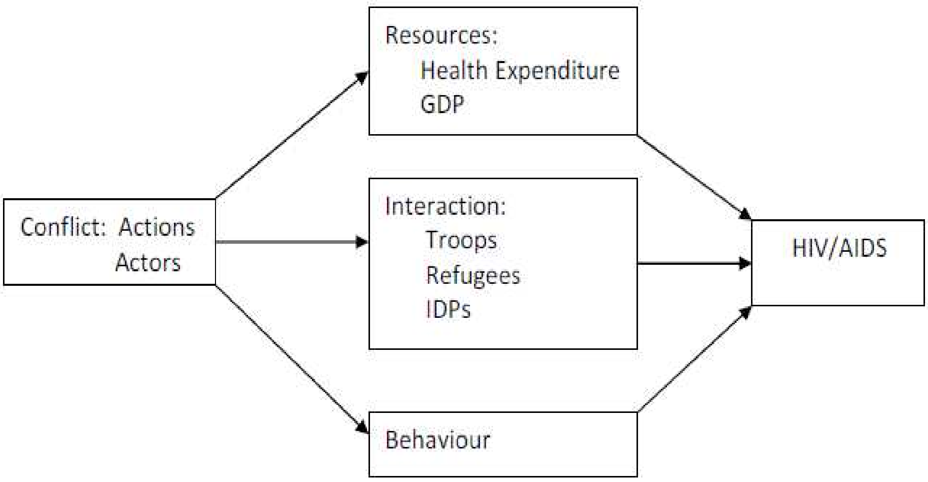
6.3 The follow up study

As already mentioned, another econometric contribution to the literature on HIV and armed conflict was by Davenport and Loyle (2009). The scope of the study was global although they also tested their results using a smaller sample of Sub-Saharan African countries. Their work deviates from the Iqbal and Zorn study in a number of theoretical and methodological aspects.

6.3.1 Conceptual framework

Davenport and Loyle explore three mechanisms that influence HIV prevalence. These three are behaviour, resources and interaction. As shown in Figure 6.3 these factors are assumed to be influenced by conflict and they in turn are assumed to influence HIV prevalence rates. Their incorporation of a behavioural dimension is a major difference between their work and that of Iqbal and Zorn (2010). Individual perceptions of the future are assumed to determine changes in each person's risk calculus. Their theoretical argument is that when there is very little sense of a future at all, individuals might disregard their long-term personal safety by not practicing safe behaviour to prevent HIV infection (Davenport and Loyle, 2010:13). They also argue that armed conflict results in a breakdown of social networks and family structures that act as additional support against risky behaviour (Davenport and Loyle, 2010:13). However, the discussion in the previous chapter highlights how it is exactly this breakdown of social networks that might also slow down the spread of the epidemic.

Figure 6.3 The Davenport and Loyle framework



Source: Davenport and Loyle (2010)

In their model, Davenport and Loyle combine level of development, economic openness and health expenditures into one mechanism which they term "resources" (see next section for their research design). They present the case that armed conflict results in the channelling of resources away from public health/social services and towards security concerns (Davenport and Loyle, 2010:13). This prioritisation of security over the prevention and treatment of HIV is assumed to hamper efforts to combat the HIV epidemic. Other complementary services and public responsibilities such as education and general health services also suffer. But while this may well result in higher levels of HIV infections (HIV incidence) Davenport and Loyle assume too quickly that this will be reflected in higher HIV prevalence rates. As the analysis in Chapter 3 showed, countries experiencing severe disruption in services (as was the case in Zimbabwe) the collapse of health systems might lead to more AIDS deaths which in turn would lead to a reduction in HIV prevalence rates.

Their "interaction" mechanism tries to capture the way different populations interact with each other. The idea here is to operationalize the interactions theorized (but not implemented in the modelling) by Iqbal and Zorn who, as pointed out above, only included raw numbers of total refugees in their model. Davenport and Loyle also include internally displaced persons (IDPs), peace keeping efforts (troops) and cross border refugees (Davenport and Loyle, 2010).

Davenport and Loyle argue that interaction can occur in a variety of ways. First there is interaction between soldiers from either side of the conflict and the civilian population (Davenport and Loyle, 2010:15). Troops (foreign or domestic) can bring HIV into or take it out of previously segregated communities. Many of the arguments discussed in more detail in Chapter 5 are touched on by Davenport and Loyle – such as the notion that soldiers are generally more risk taking and spend so much time away from home that they seek sexual gratification in highly risky ways. Chapter 5 revealed that the evidence to support such arguments is very thin.

Davenport and Loyle note that HIV infections might occur even where armed personal are not the vectors because conflict also leads to the displacement of people and entrants (high HIV) into previously isolated low HIV prevalence communities might spread the disease (Davenport and Loyle, 2010:15). These may be internally displaced persons or refugees. However, evidence from Spiegel et al. (2007) and others, presented in Chapter 5, does not support this hypothesis.

The key difference between the theoretical approach of Davenport and Loyle vs. Iqbal and Zorn is that Iqbal and Zorn look at the relationship between armed conflict and HIV prevalence from the perspective of interstate versus domestic types of conflict, whereas

Davenport and Loyle do so through the lens of actions and actors (as shown in Figure 6.3). They point out that the actions carried out in armed conflicts vary in terms of the duration of the fighting, geographical scope of the conflict and the magnitude of violence (Davenport and Loyle, 2010:15). This, in turn, is assumed to affect HIV prevalence through its impact on behaviour, resources and interactions.

Following Iqbal and Zorn, Davenport and Loyle argue that the higher the magnitude of violence, the greater the chance of HIV prevalence increasing and that the longer the duration of the conflict, the greater the increase in HIV prevalence. They also hypothesize that HIV prevalence in a country will increase as the geographical scope of the conflict increases. All of these are arguments in favour of the notion that militarization, armed conflict and social disruption act to worsen the spread of the epidemic (although as shown in chapter 5, there are strong counter-arguments and counter-evidence to these claims). .

With regard to behaviour, Davenport and Loyle hypothesise that different actors have different impacts on resources, behaviour and interactions. They distinguish between three types of actors namely, soldiers, interveners and refugees. First, Davenport and Loyle (2010:16) argue that as the number of soldiers in a conflict increase, so will HIV prevalence rates. As the number of domestic or/and foreign soldiers increases, the rates of interactions of these soldiers with the population also increase. Contrary to the Iqbal and Zorn framework, Davenport and Loyle suggest that it is not instructive to classify a conflict as international versus domestic. Rather, they focus on the presence of international forces (intervening forces, peacekeepers), the idea being that HIV prevalence in the conflict country is likely to increase if there are soldiers and interveners from countries with higher prevalence rates (Davenport and Loyle, 2010:16).

Davenport and Loyle also argue that even in non-conflict countries, HIV prevalence might rise if the country hosts refugees from countries with higher prevalence rates. Unlike Iqbal and Zorn who look at overall numbers of refugees in a country, Davenport and Loyle argue that there should be a distinction between those refugees from high versus refugees from low prevalence countries. They state that, “International refugees from origin countries with a higher rate of HIV prevalence will increase the HIV prevalence rate in the host country”. (This, however, is contrary to the work published by Spiegel et al. (2007) and others – see Chapter 5).

6.3.2 Research design

Davenport and Loyle run a cross sectional OLS regression on 2007 HIV prevalence rate estimates and a series of conflict and other control variables. Like Iqbal and Zorn, Davenport and Loyle also do not provide a list of the counties in their sample. It is therefore impossible to replicate their results. Presumably they included all countries recording cases of HIV in their global sample and all sub-Saharan countries in their sub-Saharan sample. They use the UNAIDS/WHO 2007 estimates of HIV prevalence. All of their explanatory variables are averages (or aggregates) of the period 1997 to 2007. This is shown in Table 6.2. Their logic here is that AIDS has an 8 to 10 year incubation period and so the one year lag applied with most social science statistical models may not be appropriate (Davenport and Loyle, 2009: 19). While this is correct, they fail to consider the fact (discussed in more detail above) that HIV prevalence rates are already the product of demographic modelling and that even if the impact of conflict takes more time than a year, the modelled nature of the dependent variable will obscure the relationship to an important extent.

The explanatory variables they use include conflict data from the Centre for the Study of Civil War at the International Peace Research Institute and the Uppsala Conflict Data Program.[42] These conflict variables (scope, duration, magnitude, interstate, intrastate) collectively measure behavioural breakdown (Davenport and Loyle, 2009: 21). Davenport and Loyle (2009) ingeniously use a scope (conflict) variable which is created by dividing the total area of the armed conflict by the total land area of the country. They also incorporate other variables including one to measure conflict duration (number of years of civil conflict between 1997 and 2007), and the number of battle related deaths to measure the magnitude of the conflict.

[42] See online: http://www.pcr.uu.se/research/UCDP/data_and_publications/definitions... 2010/05/17 UCDP – Definitions

Table 6.3 How data was aggregated

Variable	Aggregation
High HIV Prevalence Refugees	Average over 10 Years
Medium and Low HIV Prevalence Refugees	Average over 10 Years
GDP	Average over 10 Years
Population Density	Average over 10 Years
Trade as Percentage of GDP	Average over 10 Years
Health Expenditures as Percentage of GDP	Average over 10 Years
Regime Type	N/A
Civil War Years	Number of Years over 10 Years
Battle Deaths	Sum
Peacekeeping Operations	Number of Years over 10 Years
Scope of the Conflict	Average over 10 Years
State Repression	N/A
Interstate War	Number of Years over 10 Years

Source: Davenport and Loyle (2009)

Davenport and Loyle measure "interaction" using international intervention as well as refugee variables. Refugee data was obtained from the dyadic UNHCR global refugee data[43]. They split this data so as to divide it into three, separating refugee origin countries into high, medium and low HIV rate countries[44] (Davenport and Loyle, 2009: 21). For each of these, they counted the number total number of refugees from the relevant category into the country in question between 1997 and 2007. Davenport and Loyle also include variables that measure population density, trade, health expenditure, region, presence of peace keeping forces[45] and the level of state repression.

6.3.3 Results

As shown by Table 6.3, their study found that the direct effects of conflict, measured in magnitude, duration and geographical scope, had no statistically significant correlation with national HIV prevalence estimates. Davenport & Loyle (2009) find this result to be consistent for the global dataset and for one considering only countries in sub-Saharan Africa. The relationship was positive (but statistically insignificant) for magnitude and duration of conflict, but negative (and insignificant) for geographical scope. No explanation of these

[43] See http://www.unhcr.org/statistics.html
[44] High if HIV prev > 2.2 %; Medium if 0.2 < HIV prev < 2.2 %; Low if HIV prev < 0.2 %
[45] See online: http://www.un.org/Depts/dpko/dpko/pastops.shtml. For this variable they used a tally the number of years the country had a peace keeping mission in the ten years sampled.

trends is provided. In any event, the results do not support the hypothesis that conflict is associated with higher HIV prevalence rates.

Table 6.4 Davenport and Loyle global sample regression results

Variable	Coefficient	Standard Error	T-score	P-value
High HIV refugees	2.59e-07	(7.79e-08)	3.320	0.010
Medium and Low HIV Refugees	-2.36e-08	(1.72e-08)	-1.370	0.173
GDP	-4.71e-04	(1.00e-04)	-3.260	0.001
Population Density	-8.95e-06	(5.42e-06)	-1.650	0.102
Trade	0.006	(0.003)	2.410	0.018
Health Expenditures	0.198	(0.073)	2.700	0.008
Regime Type	0.015	(0.312)	0.050	0.963
Region	2.268	(0.275)	8.260	0.000
Intrastate Conflict	0.056	(0.059)	0.930	0.354
Battle Deaths	3.02e-06	(0.000)	0.170	0.868
Scope	-53.72	(169.1)	0.320	0.751
Peacekeeping Operation	-0.088	(0.038)	2.310	0.022
State Repression	0.243	(0.342)	0.710	0.479
Interstate Conflict	0.065	(0.096)	0.680	0.499
Constant	2.620	(0.566)	4.630	0.000
R^2	0.5699			

Source: Davenport and Loyle (2009)

Another fundamental finding of the Davenport and Loyle study is that both at a global and Sub-Saharan Africa level, domestic and international conflict have a positive but not statistically significant impact on HIV prevalence rates. Additionally, the conflict duration, scope and magnitude variables were also found to have a positive, but statistically insignificant, relationship with conflict. Davenport and Loyle conclude that regardless of what aspect of conflict one highlights, the presence of armed conflict itself does not appear to have a significant influence on the HIV prevalence rate of a given country. But they do find that refugees from high HIV prevalence countries have a positive and statistically significant effect on a country’s HIV prevalence rate estimates[46]. Davenport and Loyle find that as the

[46] On the other hand, when refugees from medium or low HIV prevalence countries are tested, the effect of interaction on the host country is not significant.

number of high HIV prevalence refugees increases from its minimum to its maximum value the expected value of HIV prevalence for the host country increases from 1.08 to 3.43 (Davenport and Loyle, 2009:24). In other words, countries with the highest levels of refugees from high risk countries have a HIV prevalence rate 281%[47] higher than countries without refugees (ibid, 2009:24).

Of the other variables they find statistically significant, Davenport and Loyle find that the expected HIV prevalence rate for a country with the minimum level of GDP to be 1.38. The expected HIV prevalence rate for a country with the maximum level of GDP to be 0.179 with the difference of -1.2 resulting in an 87% reduction in the HIV prevalence rate of the host country as GDP increases from its minimum value to its maximum (Davenport and Loyle, 2009:24). Although they disagreed with the methodology employed by Iqbal and Zorn, Davenport and Loyle acknowledge that their results are consistent with Iqbal and Zorn’s findings (ibid, 2009:25).

With respect to their theory on the effect of peacekeeping operations on the host country’s HIV prevalence rates, they find a rather surprising result. Davenport and Loyle find that the presence of peace keeping troops results in a 58.7% reduction in the HIV prevalence rate of the host country. This attribute to the resources that peace keeping missions often bring to th host country. These often include public health projects which would assist in lowering prevalence.

They go further to explain that some of the countries in their study, for example, Georgia, Morocco, Lebanon and Israel, all hosted a UN peacekeeping mission for the period sampled yet all four countries have an HIV prevalence rate of 0.1 % (ibid, 2009:26). This highlights the sensitivity of their results to the countries in their sample as some of these countries have HIV prevalence rates that are so low that it is more likely that other factors, many of which are not controlled for in the regression, would explain the low prevalence rates.

[47] This is not a typo, this is a result from Davenport et al (2009)

6.3.4 Limitations

In building on Iqbal and Zorn, Davenport and Loyle (2010) focus primarily on improving the independent variables (conceptual framework). They also avoid using the pooled OLS approach used by Iqbal and Zorn by using prevalence rates from one year. However, their independent variables are aggregated (over the 10 years) and this brings forth a few concerns.

6.3.4.1 Aggregation of the independent variables

It is likely that the dynamics of the independent variables i.e. how they interact with HIV prevalence over time will get drowned out in the aggregation employed by Davenport and Loyle. This would make it difficult to find statistically significant country-level relationships between indicators of conflict, or disruption and HIV trends. In essence, by choosing a cross sectional OLS model of this kind, Davenport and Loyle ignore the dynamics at play within each country. Their model implicitly looks at variations between rather than within countries because for each country, conflict history, GDP and other variables are all averaged/summed over 10 years. However, in their defence, the reason for taking this route was to avoid the flaw in Iqbal and Zorn. Regardless, the aggregation of the variables presents a different suite of issues.

Their model can be summarised as follows;
$Y_i = \alpha + X_i\beta + v_i$

Where Y_i is the logarithm of the 2007 HIV prevalence estimates for the i^{th} country. And X_i represents all the independent variables (aggregated by averaging or summing over the 10 years) in their analysis. The model works by comparing each county's characteristics as defined by the aggregates of the independent variables (X_i) to those for other countries in the sample. This means in a global sample that includes most countries in the world, the results they find are not surprising. One would expect to find a positive relationship between HIV and conflict as many of the world's conflict countries are in sub-Saharan Africa which also has most of the world's HIV cases. The unfortunate coincidence that Africa seems to have proportionally more conflict countries as well as more and bigger HIV epidemics is one Davenport and Loyle take time to discuss in their introduction (Davenport and Loyle (2009).

However, they do not interpret their results in light of this extremely important point. . In other words, their model is misleading if not interpreted appropriately. It merely proclaims that when one compares each country's HIV prevalence rate estimates for the year 2007, one finds that conflict countries defined as those that had more than 1 conflict in the 10 years under their study (many of whom are sub-Saharan) are associated with higher HIV prevalence rates (predominantly sub-Saharan). This does little in terms of helping us understand the effect that conflict has on the HIV epidemic.

6.3.4.2 Other issues

Davenport and Loyle's methodology also fails to take into account that HIV epidemics are likely to be at different stages for the countries in the sample. Countries with older epidemics might have lower prevalence rates than those whose epidemics are relatively new as the latter will still have increases in new infections until the epidemic peaks. As data becomes more available and the literature advances, models such as those by Iqbal and Zorn as well as Davenport and Loyle may well be able to be expanded to include more independent variables allowing for more factors to be controlled for. However, it is important to learn from these two studies so as to develop econometric models that are appropriate for the kind of limitations we currently face regarding the measuring and estimation of HIV data. Other more subtle issues like the fact that many countries launched their ART and PMTCT from 2005 onwards are also easy to miss and may confound Davenport and Loyle's result as these interventions would have had had an impact on AIDS mortality and HIV prevalence rate estimates.

6.4 Fixed Effect and Random Effect panel models: Paxton (2009)

Around the same time as Davenport and Loyle study, another attempt at modelling the relationship between HIV and conflict was carried out by Nathan Paxton at Harvard University. Paxton (2009) uses a different econometric methodology to that employed by both Iqbal and Zorn as well Davenport and Loyle. Using panel data, Paxton presents fixed effects and random effects models. Although his models are parsimonious and are not hard to follow, his work assumes too much on the part of the reader and does not explain the merits or demerits of using either fixed effects or random effects models. Before discussing his findings, I will use the next sub-section of this chapter to give a quick round up of what fixed effects and random effects models are and how they differ from the cross sectional and pooled regression models by Iqbal and Zorn as well Davenport and Loyle.

6.4.1 Fixed Effects (within country effects)

When using fixed effects models, each country is assumed to have its own, immeasurable, individual characteristics that may or may not influence the independent/predictor variables (for example a particular history of ethnic conflict). Fixed effects models assume that something within each country may influence or bias the predictor (conflict variables) or outcome/dependent variables (HIV prevalence estimates) and we need to control for this. Resultantly, fixed effects models assume that there is a correlation between each country's error term and the predictor variables. This is the corner stone of fixed effects estimation. Fixed effects remove the effect of those time-invariant[48] characteristics from the predictor variables so we can assess the independent variables/predictors' net effect.

A country fixed effects model is appropriate if each country has a significantly different level of HIV (prevalence estimates) when all regressors/independent variables i.e. conflict and other controls are set to zero. It is reasonable that the HIV data (prevalence estimates) vary dramatically at country level (see next chapter). Because each country is different, the country's error term and the constant (which captures individual country characteristics) should not be correlated with that of the other countries. If we opt to use a fixed effects model, we would have to use a within effect model to avoid the incidental parameter problem[49] (an issue when n>T). In short, the model exploits within-country variation over time. Time invariant characteristics cannot cause such a change, because they are constant for each country (Kohler et al, 2009:245).

[48] "The key insight is that if the unobserved variable does not change over time, then any changes in the dependent variable must be due to influences other than these fixed characteristics." (Stock and Watson, 2007:289-290).

[49] Least Squares Dummy Variable regression models are problematic when there are many groups or n>T. See Baltagi, (2008) for more on the incidental parameter problem.

The fixed effects functional form is given as follows[50]

$Y_{it} = (\alpha + \mu_i) + X_{it}\beta + v_{it}$

Where v_{it} are normally distributed (Park, 2009:16). α is the intercept whereas μ_i is the time invariant country effect. Each β represents the change in Y (prevalence estimates) for each 1-unit increase in X within each country.

6.4.2 Random Effects (within and between country effects)

A random effects model by contrast, estimates variance components for countries and the country error terms, assuming the same intercept and slopes. μ_i is a part of the errors and is assumed uncorrelated with any regressor/independent variable. Thus, the difference among countries lies in their variance of the error term, not in their intercepts. If there is reason to believe that some omitted variables may be constant over time but vary between countries, and others may be fixed between countries but vary over time, then one can control for both types by using random effects[51]. Given that HIV prevalence estimates vary over time in each case as the epidemic progresses, a random effects model may be more appropriate. One other important advantage of random effects is that you can include time invariant variables (i.e. religion or cultural differences). These are absorbed by the intercept in the fixed effects model.

The functional form of the random effects model is given as follows

$Y_{it} = \alpha + X_{it}\beta + (v_{it} + \mu_{it})$ where v_{it} are normally distributed (Park, 2009:16).

The β represents the change in Y (incidence or prevalence) for a 1-unit increase in X either within each country or between countries. The Haussmann specification test compares the fixed effect and random effect models under the null hypothesis that the individual effects are

[50] "In the case of time-series cross-sectional data the interpretation of the beta coefficients would be "…for a given country, as X varies across time by one unit, Y increases or decreases by β units". (see Bartels, 2008).
[51] Stata's random-effects estimator is a weighted average of fixed and between effects (See Park (2009:4-10).

not correlated with the other regressors (Haussmann, 1978). If H_o is rejected, then the individual effects are correlated with the other regressors in the model and a random effects model produces biased estimators violating one of the Gauss-Markov assumptions. This would mean a fixed effects model is preferred. However, if cannot reject H_o, then both the fixed effects and random effects estimators are consistent, but only the fixed effects estimator is efficient.

Table 6.5 Summary of models discussed thus far

Model	Advantages	Disadvantages
Iqbal and Zorn (2010)	1. Allowed for the first econometric analysis of the HIV-conflict nexus. The model allowed for an analysis of both time dependent and time invariant factors.	1. The model does not accommodate the fact that the epidemic has its own momentum (driven by other factors other than conflict). 2. The pooled linear OLS regression model asks too much of HIV estimates drawn from curves.
Davenport and Loyle (2009)	1. The aggregation of data in a cross country OLS analysis avoided the complications of interpreting how each of the independent variables affect the HIV prevalence estimates over time.	1. Aggregation of data drowns out much of the dynamics between conflict, other independent variables and the HIV prevalence estimates. 2. Their model implicitly looks at variations between rather than within countries.
Fixed Effects	1. Panel approach (time and cross country dimension). 2. Assumes every country has a significantly different level of HIV prevalence estimates (Y variable) when all independent variables (Xs) are set to zero. 3. Removes effects of time invariant factors such as religion.	1. As a non dynamic model it assumes that HIV prevalence estimates have no momentum of their own (not determined by conflict. 2. Linear regression models ask too much of HIV estimates drawn from curves.
Random Effects	1. Panel approach (time and cross country dimension). 2.Can include time invariant characteristics (these are absorbed by the intercept in a FE model). 3. Controls for both effects that are constant over time but vary between countries (religion etc) and those that may be fixed between countries but vary over time (e.g. level of health infrastructure).	1. As a non dynamic model it assumes that HIV prevalence estimates have no momentum of their own (not determined by conflict. 2. Linear regression models ask too much of HIV estimates drawn from curves.

Source: Previous sub-sections in this chapter

The advantages and disadvantages of the models discussed thus far are listed in Table 6.4.

In general there are a few reasons why one would prefer to use a random effects specification over a fixed effect one. The first is if there is reason to believe that the unobserved effects are not correlated with the independent variables. This can either be because theory suggests that it is not or because the model has good controls or many time invariant variables. Random effects models are also preferred when one wants to focus primarily on between rather than within country variation or on time constant variables. If the Haussmann test shows that random effects and fixed effects yield similar results, then one might also want to go with the random effects specification

6.4.3 Paxton's overall argument and conceptual framework

By presenting both bivariate and multivariate regression models, Paxton argues that the key predictors of HIV prevalence rates (population level) can be grouped into three categories. The first of these are what he calls "economic transitions". The variables included as measures of economic transition are changes in GDP or GDP growth and the level of price inflation. Regarding the use of GDP growth rates as a dependent variable, Paxton gives the example of China as one where the growth in GDP had brought about many changes. These include the migration of people from rural or sparsely populated regions to more populous urban centres (Paxton, 2009:4). This presents greater opportunities for interactions with infected individuals via intravenous drug use, commercial sex or men having sex with men. Paxton also uses the inflation (hyperinflation) rates as a measure of the status or general wellbeing of a country. These two economic factors are measured using data from the United Nations Statistics Division (2008)[52] and the World Bank.

The second category of predictors is what Paxton calls "political transitions". These are measured using the Polity IV project scores of the authority (autocracy) characteristics of different countries over time. The measures are split into three separate categories (three separate variables). First are major democratic transitions. This is when the polity score (which ranges from -10 to 10) changes six or more points in a positive direction as well as a shift from an autocratic score (-10 to 0) to a partially democratic (1 to 6) or fully democratic (7 to 10) score. The minor democratic transition variable measures periods defined in much

[52]See www.undata.org

the same way as the major democratic transition variable except that the total size of the shift is 3–5 points. The third variable, measures major slides into autocracy by considering only periods where the polity score drops 6 or more points.

The third category of regressors measures the conflict status of a state. Paxton uses the Major Episodes of Political Violence (MEPV) dataset (Marshall 2006a). This is a dataset that has been used extensively to measure the occurrence of war and civil violence, Further, the MEPV categorises conflict into three types: conflicts between or among two or more states, conflicts within the state primarily over political identity and ethnic conflicts within the state primarily over social identity attributes (Paxton, 2009:8). The dependant variable, HIV prevalence rates are taken from UNAIDS (2008) and cover the period from 1990–2007. All in all, the data (all variables) spans 168 countries. Paxton presents two sets of results, first from the bivariate analysis and lastly those from the multivariate analysis.

6.4.4 Results

Paxton's bivariate regressions show the estimated effect on economic growth of varying levels or of hyperinflation upon HIV prevalence to be statistically insignificant and small. These results are shown in Figure 6.6. A similar result is observed for the relationship between hyperinflation and estimated HIV prevalence rates. Paxton concludes that rapid economic transitions do not have a measurable effect on the spread of HIV/AIDS. Regarding political transitions, Paxton finds that rapid political transitions have statistically significant and positive effect on HIV prevalence levels.

Table 6.6 Bivariate regression results

	β	N	Groups
Economic			
GDP growth positive (F)	0.005 (0.012)	1673	132
GDP growth over 6 (F)	0.013 (0.016)	482	101
GDP growth over 8 (F)	0.011 [p=0.12] (0.007)	242	69
Inflation over 100% /year (F)	-0.000 (0.000)	102	32
Political (Polity IV score)			
Major democratic transition (R)	0.069 *** (0.015)	65	30
Minor democratic transition (F)	0.786 ** (0.247)	11	9
Major autocratic transition (R)	0.055 [p=0.13] (0.036)	20	17
Conflict			
Interstate War (R)	0.047 (0.160)	1585	125
Civil War (R)	-0.279 ** (0.092)	1585	125
Ethnic War (R)	-0.201 * (0.089)	1585	125

Source: Paxton (2009). The table entries are FE or RE linear panel regression results which are chosen based on the Hausman test on the FE and RE coefficient vectors of each model, Standard errors are shown in parenthesis. ***p< 0.001 **P<0.01 *p<0.05

With regards to the conflict variables, Paxton finds that interstate war does not have a measurable effect upon HIV prevalence. However, civil and ethnic wars have strong negative and statistically significant effects on population level estimated HIV prevalence. Fully acknowledging that the models do not explain what it is about ethnic or domestic conflict that slows down the HIV epidemic, Paxton speculates on some of such reasons. One idea is what he calls "numerator effects" which is where the numbers of HIV+ people, relative to their proportion in the population, decreases faster than those of HIV- people. Reasons for such a decrease could be that these HIV+ people are for some reason clustered in areas where more conflict activity occurs. Such persons may be secluded geographically or their movements may be restricted by the fighting and this cuts off their access to medical care. Paxton notes that in all such cases, HIV+ persons would bear the same level of risk as other members of the society for death from the conflict, and so being HIV positive further adds to these risks.

Table 6.7 Multivariate regression results

Variable	Model 1	Model 2	Model 3
Annual GDP Growth	0.003	0.023 **	-0.028 **
	(0.009)	(0.009)	(0.010)
Polity IV Score	0.194 ***	0.189 ***	
	(0.021)	(0.021)	
Major Democratic shift			0.061 ***
			(0.015)
International War	0.033	0.108	
	(0.166)	(0.164)	
Civil War	-0.147 [p=0.11]	-0.203 *	-0.372
	(0.093)	(0.092)	No obs.
Ethnic War	-0.169	-0.244 **	
	(0.095)	(0.094)	
Controls			
Pop. Growth / year		-0.484 ***	0.328
		(0.067)	(0.450)
N	1548	1548	51
k	0.067	0.100	0.598
Multi. R^2	-0.279	1585	125

Source: Paxton (2009)

The multivariate fixed effects regression models confirm the results observed in the bivariate analysis. They show that major democratic transitions and intrastate wars have a statistically significant and large effect upon HIV prevalence. Model 3 shows that a 1 % shift towards democratisation is associated with a 0.06% decrease in HIV prevalence (Paxton, 2009:19). Further, while the transition to democracy drives HIV prevalence upward, intrastate war and ethnic conflict are associated with lower population level estimated HIV prevalence rates.

6.4.5 Limitations

Because the models are presented by Paxton are all relatively parsimonious compared to those by Iqbal and Zorn as well as by Davenport and Loyle, they omit many relevant controls notably, time variant variables. This omission might be confounding the results observed by

Paxton and would perhaps account for the fact he finds a negative relationship between conflict and HIV prevalence. Another weakness is that much like Davenport and Loyle, Paxton uses HIV data covering periods after 2004-5 when many countries launched their ART and PMTCT interventions. This too might be confounding the results.

However, the Paxton fixed effects multivariate models have the advantage that they allow for one to focus only on within country variation (as explained in previous sections). This might be the reason for the difference between the results shown in Tables 6.5 and 6.6 and those from the Iqbal and Zorn as well as Davenport and Loyle. The focus on within country variation removes the confounding effect of having numerous time invariant controls which might be poorly measured. As I mention in the introduction to this chapter, the fact that Paxton's results go against conventional wisdom (backed by the Iqbal and Zorn as well as Davenport and Loyle) has been used as evidence that there is little to be gained from population level regression analysis. For example De Waal et al (2010) encourage a move away from aggregate population level analysis as it fails "*to capture the dynamic and complex nature of the social and epidemiological changes taking place and evolving over time*" (de Waal, 2010:50).

In the chapters that follow, I argue that there is still much to be gained from such aggregate level analysis because it provides context to any micro analysis of the link between HIV and conflict. Granted, part of the reason for the differences in the results produced by econometric analysis is the unimpressive quality of the data used. However, the choice of econometric model is just as important. The next section explores the plausibility of dynamic panel regression models as an alternative to the models presented thus far.

6.5 Discussion and conclusion

A key argument in Chapters 1 to 5 is that we should use HIV prevalence rates cautiously and in the full knowledge of how they are estimated. Although the pioneering work by Iqbal and Zorn, Paxton as well as Davenport and Loyle has advanced literature on the link between HIV and conflict, a few questions still have to be answered. The first question is regarding whether HIV prevalence rate estimates are the right data to use to investigate the link between HIV and conflict. Could we learn more by using estimated HIV incidence data? If we were to use estimated incidence data in regression analysis, would we still have to make assumptions as strong as those used by Iqbal and Zorn as well as Davenport and Loyle when they use prevalence estimates?

Incidence is theoretically more appropriate than HIV prevalence for measuring the impact of conflict on the epidemic because it is a direct measure of new infections, whereas HIV prevalence is affected also by AIDS mortality. However, HIV incidence measures are derived from HIV prevalence estimates (using demographic modelling packages such as EPP and Spectrum) so the same problems discussed earlier for OLS regression on HIV prevalence estimates will apply also to HIV incidence estimates.

In any event, the HIV/AIDS incidence estimates presented in the UNAIDS/WHO Report on the Global HIV/AIDS Epidemic (2009) do not have numbers for many of the conflict countries. For instance, UNAIDS do not provide incidence estimates for Ethiopia. There are many countries for which UNAIDS reports confidence intervals for the incidence figures but not actual estimated points. These include Sudan, Nigeria, Somalia, DRC, Burundi, Chad, Liberia, Madagascar, Mauritius and Equatorial Guinea. This leaves HIV prevalence estimates as the only practical choice (unless one estimates an epidemic curve for each country and uses Spectrum to estimate HIV incidence). The second question that may be posed is if we are to use estimated HIV data (incidence or prevalence), how best can we apply regression techniques to better understand the HIV-conflict nexus?

The studies presented in this chapter show how various techniques have been used to model the relationship between HIV and armed conflict ranging from cross sectional OLS, pooled OLS, fixed effects and random effects models. These have produced varied results and each has strengths and weaknesses. In the next chapter, I attempt to use all the models described in this chapter, to ascertain whether population level regression methods are hopelessly inconsistent and should be forgotten about or whether there technical and theoretical reasons for the different results presented by the few econometric studies discussed thus far. To this end I also use the same data set to run dynamic panel models. These models account for the fact that trends in HIV prevalence have their own momentum which is not always captured by the independent variables in regressions.

7 RELOOKING AT THE USE OF HIV PREVALENCE AND INCIDENCE ESTIMATES IN MODELLING THE RELATIONSHIP BETWEEN ARMED CONFLICT AND HIV

Abstract

Building on past econometric work, this chapter provides cross-country OLS and dynamic panel data regression analysis of HIV prevalence and conflict in 34 sub-Saharan countries with generalised epidemics. The panel data regressions corroborate the results presented by Paxton (2009). The results therefore cast doubt on the argument that conflict worsens the epidemic, particularly at a country (population) level. I find a negative, statistically significant association between domestic, armed conflict, militarization, the magnitude of conflict, and HIV prevalence (estimated). However, as did Paxton (2009) I find the relationship between estimated HIV prevalence and interstate conflict to be positive but statistically insignificant. I argue that the sample of countries is likely to be crucial in driving the estimated relationship between HIV prevalence and armed conflict. The descriptive statistics highlight that it is Sudan, Angola, Rwanda, Ethiopia, Liberia, Somalia, and the Democratic Republic of the Congo that drive the result found in this chapter. I also caution that the OLS, fixed effects and random effects models presented in this chapter suffer from the same problems highlighted in chapter 6.

Despite dynamic models being a step in the right direction because they account for the fact that trends in HIV prevalence have their own momentum, they require adequate attention to issues of inconsistency that may arise when relevant variables are omitted. Most importantly, all these models are linear regression models and so ignore the fact that the HIV prevalence rates used are estimates based on curvilinear projections. This makes the model specifications far from ideal. Finally, every model is dependent on the quality of its inputs and the lack of actual data on HIV prevalence for most countries is a cause for caution. However, any micro level analysis of contextual factors must be informed by a separate study of the underlying socio-economic, demographic and socio cultural factors central to the spread of the epidemic. Appreciating the limitations of econometric modelling when interpreting the results makes them more useful as a tool for creating a foundation on which more contextual, micro analysis can be carried out.

7.1 Introduction

Chapter 6 explained that aside from the models presented by Iqbal and Zorn, Davenport and Loyle as well as those by Paxton, there are other model specifications that are worth exploring. These include fixed effects and random effects. Using a different dataset, this chapter presents fixed effects, random effects as well as dynamic panel models. The point of this section of the thesis is not to improve Iqbal and Zorn, Paxton or Davenport and Loyle's conceptual frameworks. Rather, the models presented in this chapter aim to highlight the difficulties associated with the use of HIV estimates as dependent variables in regression models. The analysis highlights the imperfections of many of the independent variables typically used in such regressions and their implications for the interpretation of the model results.

In addition to showing the different ways in which the conflict /HIV relationship can be modelled, the analysis also attempts to reconcile the literature in Chapter 4 (stressing the relative importance of militarization and the role the military plays in the HIV-conflict relationship) with the modelling options presented in Chapter 6. Despite arguments that suggest that there is nothing to be gained from country level regression models, I suggest that there remains something to be gained from such modelling especially when the limitations and assumptions behind the models are appreciated. After presenting the rationale for using cross country or population level analysis, I proceed first with a description of the data in section 7.2 and another of the methodology in sections 7.3 and 7.4. This is followed by a presentation of the results in section 7.5 and section 7.6. discusses the results and concludes the chapter.

7.1.1 The relevance of cross country regressions

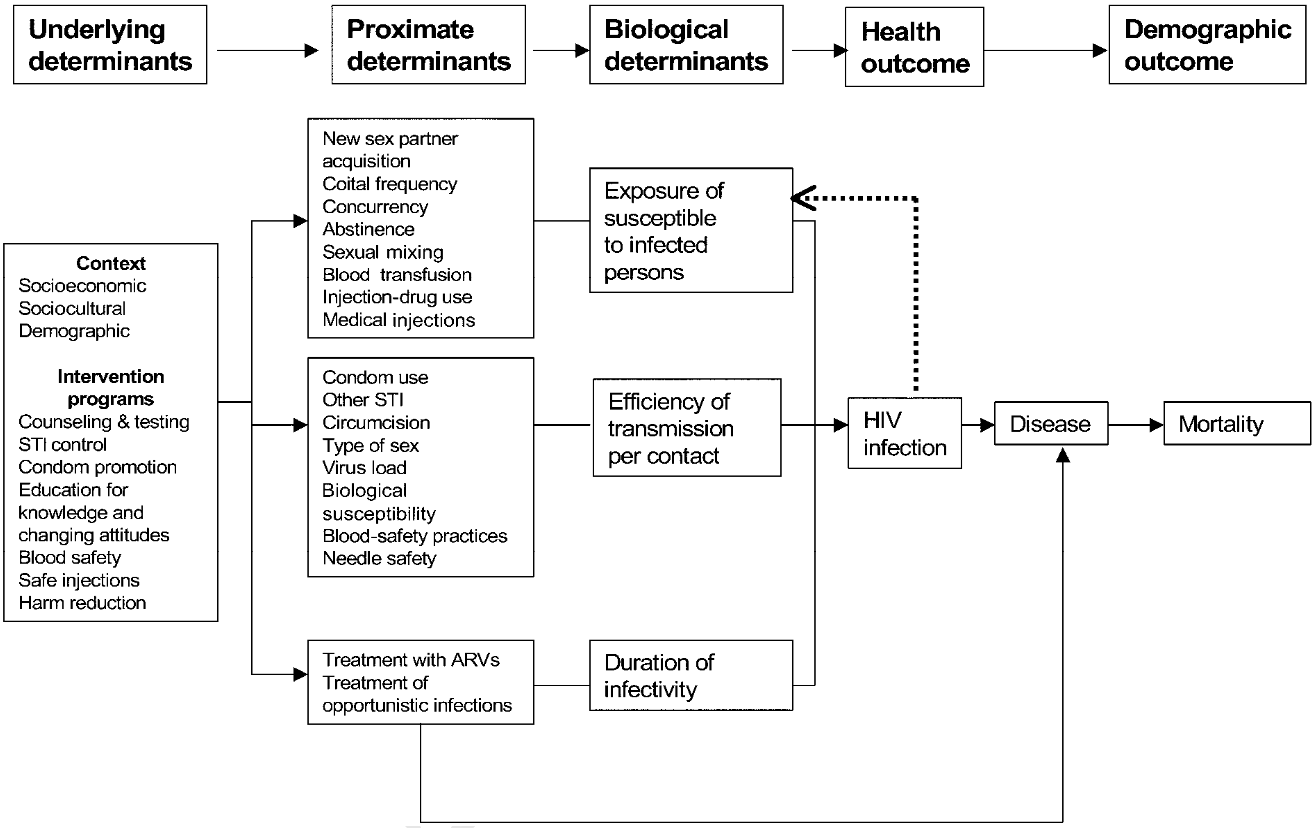
As highlighted by Lewis et al, (2007: 61) in infectious disease epidemiological studies, the focus is on whether individuals are exposed to infection and whether they actually get infected upon exposure. However as Lewis et al continue, it is often the case that while attempting to identify various inter relationships and pathways such as social, demographic, economic, cultural, behavioural and biological factors, studies include large numbers of variables without a coherent conceptual framework. Given the complexity of issues that surface particularly when HIV is the infection of interest, I will draw from established frameworks in epidemiology to map out a conceptual framework that carries forward the lessons from the studies by Iqbal and Zorn, Paxton as well as Davenport and Loyle. Despite the challenges associated with population level regressions, they form an integral component of epidemiological study.

In fact, the role of socioeconomic, demographic determinants in addition to those that are more contextual has been well acknowledged (see Aral et al, 1999; Boerma and Weir, 2005; Lewis et al, 2007 and others). A part of the interest has stemmed from the fact that while many advances have been made in understanding the spread of HIV from studies of individual risk factors in different populations, there is less known about why different populations have had different epidemics (Lewis, et al 2007). While this is probably sufficient to justify population level (cross country) regression analysis of the HIV epidemic, there is yet another reason for understanding population level determinants of the spread of HIV. This is that the population level factors are the starting point for any comprehensive epidemiological study as they structure the other more micro or individual level behavioural factors in potentially important ways.

The formulation of the proximate frameworks for studying the transmission of HIV draws on precisely this insight. These have been presented by Lewis et al (2007) as well as Boerma and Weir (2005). They draw on proximate frameworks in fertility studies (see Bongaarts et al, 1983; Bongaarts, 1978), child survival (see Mosley and Chen, 1984; Van Norren et al, 1989; Becker and Black, 1996) and other literature in demographic and epidemiology. In explaining their proximate determinants framework, Boerma and Weir map a mechanism through which social and environmental factors can be linked with more biological factors. Sociocultural, intervention program characteristics and economic determinants are population or country-level factors which influence the proximate determinants and these in turn have both behavioural and biological components (Boerma and Weir, 2007:63).

In the framework, HIV infection is determined by biological determinants. They identify three biological determinants namely; exposure of susceptible persons to those who are already infected, the probability or efficiency of transmission per contact, and finally the duration of infectivity. These are shown in Figure 7.1. These three determinants are influenced by the proximate determinants.

Figure 7.1 Proximate determinants framework



Source: Boerma and Weir (2005)

The proximate determinants include factors such as condom use, injection drug use and sexual concurrency. Proximate factors are influenced by what Boerma and Weir call "underlying determinants." These underlying determinants include demographics, sociocultural characteristics and HIV prevention campaigns. They note that it is important to understand the distinction between the underlying and proximate determinants in statistical analyses and that mixing underlying and proximate determinant models in the same model is likely to produce results that are difficult to interpret. Further the system shown in Figure (7.1) does not as Boerma and Weir (2005:64) stress "try to show either the complex interactions that may occur between the underlying, proximate, and biological determinants or the feedback mechanisms that link outcomes on the right with the determinants on the left". They advise that researchers should try and take full advantage of the multilevel structure. In essence, methods that determine patterns of underlying or contextual variables can serve as "markers" of the proximate determinants that are relevant (Boerma and Weir, 2005: 65). Following on the discussions in Chapter 5, lessons from Iqbal and Zorn, Paxton as well as Davenport and Loyle, in looking at the link between HIV prevalence and conflict, it is important to identify specific underlying factors. These include socioeconomic factors (income/ GDP per capita, economic openness, health expenditures, conflict, democracy, and militarization), demographic factors (population density, age of the epidemic, migration and movement of refugees) and socio cultural factors (religion etc.). The rest of this chapter will focus on finding out which underlying factors might be viewed as central to gaining an understanding of the link between HIV and armed conflict.

7.2 The data

My analysis uses 34 Sub-Saharan countries which had HIV prevalence rates (for adults aged 15-49) higher than 1% (i.e. had generalised HIV epidemics) in at least one year between 1990 and 2005. HIV prevalence estimates for the 34 countries for the 16-year period (1990 to 2005) were drawn from the 2008 UNAIDS/WHO report on the global AIDS epidemic (UNAIDS 2008). In addition to the variables included in the analyses by Iqbal and Zorn, Davenport and Loyle as well as Paxton, I include a few new variables which I describe in this section.

As there is very little data on HIV prevalence in militaries, I turn to data on the level of militarization of a country to see if the level of militarization is associated with the trend in HIV prevalence in the general population over time. The national force ratio and national military expenditure are the two measures of militarization commonly used in social science research in this field (see Omitoogun, 2001 and Onyeiwu, 2004). The national force ratio is the number of people in the military divided by the total population. For comparison between countries, military expenditure/GNP data are also used. The force ratio is preferable for our purposes because military expenditure on advanced fighter jets and other equipment is often for reasons other than internal threats and may not reflect the internal security situation of a country or be a reliable indicator of the number of people involved in the military. It has also been argued that in less developed countries, expenditure-based measures tend to be inflated while the size of the economy is underestimated (Weede, 1995: 229) hence the ratio itself is probably unreliable.

Time series data (1990-2005) on force ratios for each of the 34 countries was obtained from the Stockholm International Peace Research Institute (SIPRI)[53]. To account for the possibility that economically developed countries might have greater capacity to maintain a bigger military even when they are not engaged in armed conflicts, GDP per capita data is included in the analysis. To proxy the size of the economy, time series data on per capita GDP (in constant $US) for each of the 34 countries were obtained from the World Bank's Databank[54]. The force ratio, HIV prevalence and GDP per capita variables were logged to create normal distributions and allow easy interpretation of the coefficients in the models.

A good amount of the research that has been done on conflict has worked with and built on the Correlates of War (COW) database (see Miguel et al, 2004; Sambanis, 2002). However, this database has been reported to be inconsistent. For instance, it is not clear whether the COW database uses a minimum of 1000 cumulative deaths or a minimum of 1000 deaths per year to classify a country as a conflict country (Sarkees et al, 2003: 50-9). Furthermore, the COW criterion risks excluding armed conflicts that may be major for smaller countries, including many African countries (Miguel et al, 2004: 730). While Paxton (2009) uses the MEPV database, it is not ideal here as it uses a threshold of 500 directly related persons and so includes civilian deaths. In the analysis presented here, I opt for the UCDP/PRIO conflict data as it has a more transparent definition of conflict-related deaths and it takes into account countries with less than 1000 reported deaths (Miguel et al, 2004: 730). It is also unique in that a time series can be constructed so that it matches the consistent HIV prevalence data (HIV prevalence estimates are available for each country for each year in the period 1990-2005).

The UCDP/PRIO conflict data comprises many variables but I select four in particular. The UCDP/PRIO minor conflict variable is a simple binary variable, which assigns a value of 1 to countries that have had at least 25 but not more than 1000 battle related deaths during the year. A battle is defined as armed conflict carried out with the purpose of realizing

[53] See http://www.sipri.org/
[54] See http://databank.worldbank.org/ddp/home.do

domination of a government or a specific territory.[55] The second variable I use is the UCDP/PRIO major conflict variable, which is also a dummy variable that assigns a value of 1 to countries that have had more than 1000 battle related deaths during the year. The minor and major conflict dummy variables are crude measures of the intensity of armed conflict and I use them together with the militarization, GDP per capita and HIV prevalence estimates to create a balanced panel data set comprising 34 countries and spanning 16 years (1990-2005).

I include dummy variables to indicate whether the conflict was interstate or domestic. Following Iqbal and Zorn (2010) as well as Davenport and Loyle (2009), I also incorporate a variable measuring the geographical scope of each conflict. This is calculated by dividing the radius of the region affected by the conflict by the total land area of the country. I also include a population density control variable obtained from the World Bank Databank[56]. My analysis also includes a variable measuring the distance between each country's capital city and the capital city of the DRC. The idea here is that since it is generally accepted that the epidemic started in the DRC, proximity to the DRC might be an important factor determining the level of the epidemic in any given country. Intuitively, one would expect that that older epidemics would have more matured epidemics that are on the decline (therefore have lower prevalence rates).

Finally, I also include a variable which measures the percentage of the male population that is circumcised. The evidence that male circumcision reduces the chance of HIV infection is a new but substantially researched area. Aside from observational studies demonstrating a clear link between reduced HIV prevalence and circumcision (Siegfried et al. 2005), there have been clinical trials that showed that circumcision reduces the risk of HIV infection by about 60% (see Gray et al. 2007; Bailey et al. 2007; Auvert et al. 2005). The circumcision variable I use in this analysis is taken from the Circumcision Independent Reference and Commentary Service.[57]

[55] See online: http://www.pcr.uu.se/research/UCDP/data_and_publications/definitions... 2010/05/17 UCDP – Definitions
[56] See http://databank.worldbank.org/ddp/home.do?Step=12&id=4&CNO=2 (World Development Indicators)
[57] See www.circs.org

Table 7.1 shows the summary statistics for each of the variables in the sample. As is expected, there is great variation in HIV prevalence (as measured by the UNAIDS estimates) in the dataset as a whole (log of HIV prevalence has a standard deviation of 1.55). In addition, there is more variation in HIV prevalence between countries than within each country over time. This is unsurprising given the broad spectrum of countries in the sample (see Table 7.2 for a list of countries in the sample). There are not that many countries whose epidemic drastically went up, peaked, and came down in the period 1995 to 2005. Examples of countries that had such experiences include Zimbabwe as shown in Chapter 2. Some countries had much higher prevalence rates/level of the epidemic than others as shown in Table 7.2. Countries like Cote d'Ivoire had an average prevalence rate of around 0.3% in the 16-year period versus Zimbabwe with an average prevalence rate at 23.9%.

The level of militarization also exhibits more variation between countries than within each country. It is likely to be a function of each country's specific policies, socio-political priorities and economic prosperity. Note also that the level of militarization is not necessarily a function of the number of conflicts a country has had. As shown in Table 7.2, Somalia was the most militarized country (if one takes an arithmetic mean of the force ratio over the 16 years) and it did have a relatively high number of conflict years. However, some heavily militarized countries such as Djibouti, Gabon, Namibia, Botswana and Zimbabwe had no conflicts between 1990 and 2005. Angola and Burundi are the only other heavily militarized countries to have had more than ten conflict years (major and minor) from 1990 to 2005.

Table 7.1 Summary Statistics for the main variables

Variable		Mean	Std. Dev.	Min	Max	Observations
Log of HIV prevalence	Overall	1.004	1.554	-4.605	3.364	N= 544
	between		1.444	-3.290	3.156	N= 34
	Within		0.623	-1.670	2.964	N= 16
Log of GDP per capita	Overall	1.866	1.475	-6.663	9.048	N= 544
	between		0.078	1.670	1.932	N= 34
	Within		1.473	-6.467	9.098	N= 16
Minor conflict	Overall	0.208	0.406	0	1	N= 544
	between		0.271	0	0.938	N= 34
	Within		0.306	-0.730	1.145	N= 16
Major conflict	Overall	0.112	0.316	0	1	N= 544
	between		0.196	0	0.813	N= 34
	Within		0.250	-0.700	1.050	N= 16
Domestic conflict	Overall	0.176	0.392	0	2	N= 544
	between		0.297	0	1	N= 34
	Within		0.259	-0.761	2.051	N= 16
Interstate conflict	Overall	0.026	0.158	0	1	N= 544
	between		0.069	0	0.313	N= 34
	Within		0.143	-0.287	0.963	N= 16
Conflict scope	Overall	0.048	0.134	0	1.878	N= 544
	between		0.083	0	0.329	N= 34
	Within		0.107	-0.280	1.809	N= 16
Log of force ratio	Overall	0.911	0.896	-1.096	3.541	N= 544
	between		0.832	-0.394	2.679	N= 34
	Within		0.363	-1.070	2.660	N= 16
Log population density	Overall	3.363	1.281	0.543	6.417	N= 544
	between		1.293	0.731	6.341	N= 34
	Within		0.120	3.101	3.671	N= 16

Source: Stata summary statistics

Table 7.2 Summary Statistics for the main conflict variables

Country (mean force ratios)	Country (minor conflicts)	Country (major conflicts)	Country (mean HIV prevalence)
Somalia (17.38)	**Ethiopia (14)**	**Sudan (12)**	Zimbabwe (23.91)
Djibouti (15.21)	Uganda (13)	**Angola (11)**	Botswana (20.06)
Mauritius (14.0)	Chad (11)	Senegal (5)	Swaziland (16.97)
Cameroon (14.0)	Burundi (10)	**Rwanda (5)**	Lesotho (16.44)
Angola (9.42)	Senegal (9)	Burundi (4)	Zambia (14.86)
Gabon (7.0)	**Somalia (8)**	**DRC (4)**	Malawi (10.84)
Namibia (5.89)	Sierra Leone (8)	Uganda (3)	South Africa (10.52)
Liberia (5.62)	**Liberia (7)**	**Somalia (3)**	Uganda (10.2)
Burundi (5.47)	**Rwanda (6)**	**Liberia (3)**	Namibia (9.66)
Botswana (5.39)	Niger (5)	Sierra Leone (2)	Kenya (8.07)
Cote d'Ivoire (5.17)	Congo, Rep. (4)	Congo, Rep. (2)	Mozambique (6.95)
Chad (4.87)	CAR (4)	Chad (1)	Tanzania (6.74)
Zimbabwe (4.6)	Cote d'Ivoire (3)	**Ethiopia (0)**	Congo, Rep. (5.74)
Congo, Rep. (4.29)	**Sudan (3)**	Eq. Guinea (0)	CAR (5.12)
Sudan (3.12)	**Angola (2)**	Niger (0)	Cameroon (4.6)
Swaziland (2.98)	Nigeria (2)	CAR (0)	Gabon (3.93)
Ethiopia (2.97)	Cameroon (1)	Cote d'Ivoire (0)	Burundi (3.79)
South Africa (2.81)	**DRC (1)**	Nigeria (0)	Eq. Guinea (2.82)
Uganda (2.49)	Lesotho (1)	Cameroon (0)	Chad (2.47)
Zambia (2.03)	Djibouti (0)	Lesotho (0)	Nigeria (2.42)
Sierra Leone (1.99)	Eq. Guinea (0)	Djibouti (0)	**Ethiopia (2.09)**
Senegal (1.87)	Mauritius (0)	Mauritius (0)	Djibouti (2.04)
Rwanda (1.81)	Gabon (0)	Gabon (0)	**Rwanda (1.73)**
Madagascar (1.71)	Namibia (0)	Namibia (0)	**DRC (1.36)**
DRC (1.44)	Botswana (0)	Botswana (0)	**Sudan (1.34)**
Mozambique (1.41)	Zimbabwe (0)	Zimbabwe (0)	**Liberia (1.21)**
CAR (1.20)	Swaziland (0)	Swaziland (0)	**Angola (1.12)**
Tanzania (1.17)	South Africa (0)	South Africa (0)	Sierra Leone (1.03)
Lesotho (1.11)	Zambia (0)	Zambia (0)	Cote d'Ivoire (1)
Eq. Guinea (1.0)	Madagascar (0)	Madagascar (0)	Mauritius (0.51)
Nigeria (0.96)	Mozambique (0)	Mozambique (0)	Niger (0.48)
Kenya (0.91)	Tanzania (0)	Tanzania (0)	Senegal (0.32)
Niger (0.85)	Kenya (0)	Kenya (0)	**Somalia (0.3)**
Malawi (0.71)	Malawi (0)	Malawi (0)	Madagascar (0.06)

Source: Own calculations, the numbers in the minor and major conflict columns represent the sum total of conflict years in each country between 1990 and 2005.

Tables 7.1 and 7.2 shows that the minor, major, domestic, and interstate conflict variables exhibit more variation within than across countries. Only a few countries such as Ethiopia, Sudan, Angola, Somalia, Uganda, Burundi, and Chad had sustained conflicts in the 16-year period (UCDP, 2010). These are the countries with the highest number of conflict years, as shown in Table 7.2.

To understand the broad relationship among the variables, I calculate the pair-wise correlation between each of the variables and present them in Table 7.3. Following Iqbal and Zorn (2010), I lag all conflict variables by 1 year. GDP per capita is positively correlated with HIV prevalence estimates, a partial correlation result also found by Nattrass (2009: 2) when she looked at the cross country relationship between HIV and poverty (using 2005 data on HIV prevalence and GDP per capita)[58].

Table 7.3 Summary statistics for the main variables

	Log of HIV	Log of GDP per cap	Minor conflict $_{t-1}$	Major conflict $_{t-1}$	Domestic conflict $_{t-1}$	Interstate conflict $_{t-1}$
Log of GDP per cap	0.190	1				
P value	0.000					
Minor conflict $_{t-1}$	-0.133	-0.174	1			
P value	0.001	0.000				
Major conflict $_{t-1}$	-0.202	0.006	-0.124	1		
P value	0.000	0.893	0.004			
Domestic conflict $_{t-1}$	-0.286	-0.101	0.453	0.287	1	
P value	0.000	0.018	0.000	0.000		
Interstate conflict $_{t-1}$	0.034	0.035	-0.060	-0.273	-0.045	1
P value	0.425	0.415	0.163	0.000	0.290	
Conflict scope $_{t-1}$	-0.039	-0.057	0.364	0.325	0.041	0.167
P value	0.359	0.187	0.000	0.000	0.338	0.000
Log of force ratio $_{t-1}$	-0.072	0.335	0.113	0.248	0.124	-0.003
P value	0.092	0.000	0.008	0.000	0.004	0.949
Log of population density $_{t-1}$	0.082	0.006	0.157	0.0831	-0.113	-0.070
P value	0.645	0.9717	0.3758	0.64	0.525	0.698

Source: Own calculations in Stata 10[59].

[58] This result does not hold later in our panel regression models, just as in Nattrass (2009)

[59] Following Iqbal and Zorn (2010), the conflict variables have a one year lag. See Chapter 6 for arguments to support this modelling decision.

Table 7.4 Within country correlation coefficients (correlation with HIV prevalence)

Country	Force ratio $_{t-1}$ coefficient	P value	Minor conflict $_{t-1}$ Coefficient	P value	Major conflict $_{t-1}$ coefficient	P value
Somalia	-0.32	0.23	0.21	0.44	-0.85	0.00
Djibouti	0.79	0.00	0.00	0.00	0.00	0.00
Angola	-0.85	0.00	0.23	0.39	-0.47	0.07
Gabon	-0.68	0.04	0.00	0.00	0.00	0.00
Namibia	0.33	0.21	0.00	0.00	0.00	0.00
Liberia	0.39	0.14	-0.05	0.85	-0.49	0.05
Burundi	-0.24	0.38	0.11	0.68	0.05	0.87
Botswana	0.83	0.00	0.00	0.00	0.00	0.00
Cote d'Ivoire	-0.93	0.00	0.11	0.68	0.00	0.00
Chad	-0.93	0.00	0.06	0.82	-0.57	0.02
Zimbabwe	0.61	0.01	0.00	0.00	0.00	0.00
Congo	0.35	0.19	0.18	0.51	0.12	0.66
Cameroon	0.03	0.91	0.13	0.64	0.00	0.00
Mauritius	0.79	0.00	0.00	0.00	0.00	0.00
Sudan	0.65	0.01	0.05	0.85	-0.05	0.85
Swaziland	-0.94	0.00	0.00	0.00	0.00	0.00
Ethiopia	-0.25	0.35	0.04	0.90	0.00	0.00
South Africa	-0.20	0.45	0.00	0.00	0.00	0.00
Uganda	0.80	0.00	0.33	0.21	-0.33	0.21
Zambia	0.13	0.63	0.00	0.00	0.00	0.00
Sierra Leone	0.38	0.15	-0.30	0.26	0.15	0.57
Senegal	-0.78	0.00	-0.19	0.48	0.01	0.98
Rwanda	-0.51	0.05	0.34	0.19	0.09	0.73
Madagascar	-0.61	0.01	0.00	0.00	0.00	0.00
DRC	-0.05	0.85	-0.12	0.65	-0.28	0.30
Mozambique	-0.89	0.00	0.00	0.00	0.00	0.00
CAR	-0.36	0.17	0.33	0.22	0.00	0.00
Tanzania	-0.29	0.28	0.00	0.00	0.00	0.00
Lesotho	-0.85	0.00	0.16	0.55	0.00	0.00
Nigeria	0.47	0.07	0.20	0.46	0.00	0.00
Kenya	0.56	0.02	0.00	0.00	0.00	0.00
Niger	0.68	0.00	-0.51	0.04	0.00	0.00
Equatorial Guinea	-0.75	0.00	0.00	0.00	0.00	0.00
Malawi	-0.43	0.10	0.00	0.00	0.00	0.00

Source: Own calculations in Stata 10

Although it is good practice to carry out an analysis of the descriptive statistics of the variables before using them in any regression analysis, when the variable of interest are HIV prevalence estimates, there is need to be cautious. Although researchers must avoid reading too much from bivariate correlation coefficients, they are still useful indicators of the direction and size of the relationships between the variables. The signs on these coefficients might give an indication of what to expect the nature of the relation between the regressors and the dependant variable to be once we run regressions although they speak little to the strength of the relationship. For instance, it is most likely that our regressions will find a negative relationship between the force ratio and HIV prevalence estimates. This thesis argues that researchers must avoid doing too much with HIV prevalence estimates or avoid interpreting coefficients from these estimates as these are not drawn from real measured data points. However one needs to a get a sense of the data by running correlation coefficients

In addition, the minor and major conflict dummy variables also have negative and statistically significant partial correlations with the logarithm of HIV prevalence. This is not surprising given that most of the conflict countries had low average HIV prevalence rates (as estimated by UNGASS) between 1990 and 2005. Indeed, seven countries (marked in bold) with high conflict measures and low HIV prevalence, drive the negative relationship. These are Angola, Rwanda, Somalia, Liberia, Sudan, Ethiopia and the DRC. For this reason, I explore these countries in particular in Chapter 8.

Niger, Sierra Leone, Senegal, Ethiopia, and Cote d'Ivoire are some of the countries that had minor conflicts. The fact that these countries also had low average HIV prevalence rates drives the negative correlation coefficient between the minor conflict dummy variable and the logarithm of HIV prevalence variable. Of these countries, Ethiopia probably had the biggest influence on the correlation result as it had minor conflicts, mostly in the Oromiya region throughout most of the 1990 to 2005 period.

The benefit of panel data is that I can run partial correlations for each country over the 16 years sampled. Table 7.4 actually shows that it is primarily countries which had conflict (minor or major) between 1990 and 2005 that show a negative and statistically significant linear relationship between militarization (force ratio) and estimated HIV prevalence. These include Sudan, Somali, Chad etc. as shown in Chapter 8. Countries such as Mauritius, Namibia, Botswana, Zimbabwe, Kenya, and Uganda are among those that are not classified as having had conflict between 1990 and 2005 according to the UCDP/PRIO criterion. For these countries, estimated HIV prevalence has a positive linear relationship with the level of militarization or force ratio.

Although partial correlations cannot be used to draw definitive conclusions, the correlation matrices give us broad indications of the link between the different variables. I turn to regression models in order to explore the link between conflict, militarization and estimated HIV prevalence more comprehensively.

7.3 Empirical Methodology

Using a short panel data set (i.e. the number of countries is larger than the number of years for the data series, n>T), I investigate the link between conflict, militarization and estimated HIV prevalence. As explained in Chapter 6, panel data allows one to control for variables you cannot observe or measure like cultural factors (when comparing countries or states within a country –i.e. Ethiopia versus South Africa). In addition, panel data helps to control for unobservable variables that change over time within but not across countries (i.e. national policies and HIV interventions, federal regulations).

To start with, I present Specification 1 (regressions 1.1 to 1.5), which is a pooled OLS regression model that is close to that presented by Iqbal and Zorn (2010). Unfortunately, as noted in chapter 6, the actual model presented by Iqbal and Zorn cannot be replicated because they do not provide details of which countries they include in their analysis. However, I include in the regressions in this chapter, many of the independent variables that are in the Iqbal and Zorn model. Furthermore, unlike Iqbal and Zorn, I include regional dummy variables (in Specification 1) distinguishing between east, west, central and southern Africa. It also includes variables proxying for the level of militarization of a country, another proxying for the geographical scope of the conflict (following Davenport and Loyle), as well as the major and minor conflict variables described in the previous section.

Specification 2 (regressions 2.1 to 2.5), follows Davenport and Loyle. Again, it is not possible to replicate their results as they did not give a list of the countries in their sample. One main difference between Specification 2 and Davenport and Loyle is that while they aggregate their independent variables for the 10 years between 1997 and 2007, Specification 2 does the same for the years 1990 to 2005. As discussed in Chapter 2, a number of countries had already begun implementing ART and PMTCT programs by 2007. The different scales, efficacy and timing of the programs are country specific and so would confound our results. As opposed to Davenport and Loyle, the dependent variable used in Specification 2 is 2005 HIV prevalence rate estimates (rather than the 2007 values). Instead of having the variable indicating the number of battle related deaths, Specification 2 includes the major conflict and minor conflict variables discussed in the previous section. These are derived directly from the number of battle related deaths. Specification 2 includes the trade, health expenditures, refugee and peacekeeping variables included by Davenport and Loyle.

Specification 3 is a fixed effects model specification that allows for a comparison between my panel fixed effects model, Paxton's panel fixed effects model and Specifications 1 and 2 that follow the Iqbal and Zorn as well as Davenport and Loyle specifications. As indicated in Chapter 6, fixed effects models remove effects of time invariant factors such as religion and cultural differences and so acknowledge the fact that each country has a significantly different level of HIV prevalence estimates when all the independent variables are set to zero. Hausman tests (described in Chapter 5) are also presented to assess whether the random effects is more appropriate than the fixed effects model.

Specification 4 is the dynamic panel model specification. Before this model is presented, a description of how it differ from the pooled OLS, cross country OLS, fixed effects and random effects models is given (see section 7.5). The dynamic model (unlike Specifications 1, 2, and 3) has the lag of HIV prevalence rate (estimates) as an independent variable. In other words, Specifications 4 assumes that if conflict at a time t, results in an increase/decrease in HIV prevalence at time t+1, then the prevalence rate estimates at time t+2 will depend on prevalence rates at time t+1 regardless of whether there is another conflict at time t+1. Specification 4 is the system GMM estimator, also known as the Arellano and Bover/Blundell estimator. As discussed in more detail in Chapter 6, the system GMM assume that there is no autocorrelation in the idiosyncratic errors and requires the initial condition that the panel level effects are uncorrelated with the first difference of the first observation of the dependent variables.

7.4 Results

7.4.1 Pooled OLS

Model specification 1 shows, that HIV prevalence estimates have a statistically insignificant relationship with population density. Regression 1.1 controls for population density, religion, education and the level of democracy in a country. Regression 1.2 adds to these, controls for economic openness, health expenditure, proportion of refugees in the country as well as the interstate and domestic conflict variables. Contrary to the theoretical frameworks and literature discussed in Chapters 5 and 6, regressions 1.1 and 1.2 seem to show the relationship between estimated HIV prevalence rates and population density to be negative. However, regressions 1.3, 1.4 and 1.5 which not only include more conflict variables but most crucially have dummy variables capturing regional differences in the epidemic show the relationship between HIV prevalence estimates and population density to be positive. This not only makes intuitive sense but because the result is statistically insignificant, it does not really corroborate the literature discussed in prior chapters which suggests that more densely populated areas are likely to have more sexual interactions (some resulting in infection). Comparing these five regressions, it appears that it is the inclusion of the regional dummies, which results in the observed positive link between population density and HIV prevalence estimates.

However, the surprise comes in that regression 1.2 which is the model close to that presented by Iqbal and Zorn as their full OLS model, shows a *negative* but still statistically insignificant relationship between estimated HIV prevalence rate and population density. One plausible reason for the difference in the results is that the data sets used in the two regressions cover two different periods. Iqbal and Zorn use a shorter time series (1997-2005) than that used in this thesis (1990-2005). Another reason could be that the two regressions include different countries in the analysis as the sample used here is smaller than that used by Iqbal and Zorn. However, all these are speculative points as without knowing which countries were included in the Iqabal and Zorn study, it is impossible to replicate their results and make a comparison. Regardless, not including regional dummies or some other method of accounting for spatial dependence is likely to be inappropriate and regressions 1.3 to 1.5 are probably better specifications than 1.1 and 1.2.

Table 7.5 Pooled OLS regressions (Specification)

	Model									
Variable	**1.1**		**1.2**		**1.3**		**1.4**		**1.5**	
Year	0.077	***	0.050	***	0.053	***	0.052	***	0.055	***
	(0.013)		(0.005)		(0.008)		(0.008)		(0.007)	
Log of Population density	-0.058		-0.021		0.054		0.058		0.040	
	(0.048)		(0.039)		(0.050)		(0.047)		(0.045)	
Muslim religion	-0.952	***	-1.191	***	-0.889	***	-0.750	***	-1.111	***
	(0.150)		(0.124)		(0.122)		(0.107)		(0.127)	
Education	0.012	***	2.2E-3		-1.6E-3		-5.3E-3	**	-0.012	***
	(3.9E-3)		(3.3E-3)		(2.6E-3)		(2.7E-3)		(3.0E-3)	
Democracy $_{t-1}$	7.7E-3	***	0.011	***	6.9E-3	***	6.8E-3	***	4.2E-3	***
	(2.1E-3)		(1.7E-3)		(1.5E-3)		(1.5E-03)		(1.3E-3)	
Log GDP per capita $_{t-1}$			0.142	***	-0.005		-0.011		-0.038	
			(0.029)		(0.032)		(0.026)		(0.027)	
Economic openness $_{t-1}$			-1.2E-3	**	-1.1E-7		1.0E-3	***	6.1E-4	**
			(4.1E-4)		(3.2E-4)		(3.1E-4)		(3.0E-4)	
Health expenditures			0.633	***	-0.029		-0.016		-0.254	**
			(0.148)		(0.170)		(0.137)		(0.135)	
Log of Refugees $_{t-1}$			0.043		0.021	*	-0.006		-0.001	
			(0.019)		(0.018)		(0.017)		(0.108)	
West Africa					-2.006	***	-2.070	***	-1.810	***
					(0.175)		(0.161)		(0.159)	
East Africa					-0.827	***	-0.764	***	-0.575	***
					(0.130)		(0.124)		(0.128)	
Central Africa					-1.294	***	-1.814	***	-1.615	***
					(0.117)		(0.149)		(0.146)	
Log of Distance from the DRC							-3.4E-4		-2.4E-4	***
							(6.8E-5)		(7.1E-5)	
Log force ratio $_{t-1}$							-0.165		-0.126	***
							(0.043)		(0.041)	
Interstate Conflict $_{t-1}$			-0.664	***	-0.455		-0.486	**	0.100	
			(0.220)		(0.289)		(0.231)		(0.168)	
Domestic Conflict $_{t-1}$			-0.399	***	-0.413	***	-0.285	**	-0.067	
			(0.142)		(0.147)		(0.122)		(0.106)	
Major Conflict $_{t-1}$									-0.586	***
									(0.107)	
Minor Conflict $_{t-1}$									-0.144	
									(0.102)	
Log of Conflict Scope $_{t-1}$							-0.126	**		
							(0.043)			
Log of Peace keepers $_{t-1}$									-0.092	***
									(0.015)	
R^2	0.2276		0.5345		0.6925		0.7519		0.7826	
Standard errors	robust		robust		robust		robust		Robust	
P- value	0.0000		0.0000		0.0000		0.0000		0.0000	
No of observations	510		431		431		431		431	

Source: Own regression analysis. *All figures smaller than 0.01 are written in scientific form to avoid loss of information. The P-values for model specification are based on the F-test.

Model specification 1 also shows, as did Iqbal Zorn, that the HIV prevalence estimates have a statistically significant and negative association with the Muslim religion dummy. As discussed in chapters 5 and 6, this is hardly surprising as Muslim countries have on average lower HIV prevalence rates than non-Muslim countries. The negative result is relatively consistent throughout all Specification 1 regressions (1.1 to 1.5) although the size of the coefficient fluctuates. While regression 1.1 shows that Muslim countries are associated with prevalence rates 61.4 % lower than non-Muslim countries, regression 1.5 show this effect to be slightly larger (at 67.1 %) when accounting for all the other conflict variables . These estimates are derived by taking the anti-log of the coefficient on Muslim religion variable.

Regressions 1.1 and 1.2 show a positive and small link between education (as measured by adult literacy rates) and HIV prevalence estimates. This is at best weak evidence against the theory presented by Iqbal and Zorn which suggests that the relationship should be negative. This is because education enables the population most at risk to obtain relevant information that allow them to make informed decisions about safer sexual behaviour and contraception (Iqbal and Zorn, 2010:153). Iqbal and Zorn base their argument on literature suggesting that education greatly enhances the effectiveness of prevention programs (see Peterman, Lindsey and Selik, 2005). Various studies have also shown that there is a positive association between education and condom use (see Ukwuani, Tsui, and Suchindran (2003) as well as Global Campaign for Education (2004)). Iqbal and Zorn also cite the example of work by Appleton (2000) where the argument is that education better educated women are likely to delay pregnancy and eventually have fewer children than those with lower education levels (and lower incomes).

Regressions 1.3 to 1.5 show a negative link between HIV prevalence estimates and education although the relationship is only statistically significant in regressions 1.4 and 1.5. While regression 1.4 shows that a 1 % increase in the adult literacy rate is associated with a 0.5 % decrease in HIV prevalence, regression 1.5 shows that the same increase in adult literacy is associated with a 1.2 % decrease in HIV prevalence rate. As already noted, these two are the regressions that control for the regional differences in the levels of the epidemic that are not captured by the other explanatory variables in the regressions. This is further evidence that the inclusion of regional factors makes a material difference to the results. Not accounting for these factors as is done in models 1.1 and 1.2 probably leads to misspecification error or confounding. It is therefore likely that the link between HIV prevalence and education is very contextual and more nuanced than the above regression result reveal.

Model Specification 1 (regressions 1.1 to 1.5) shows that the regional dummy variables each have a negative and statistically significant association with the HIV prevalence estimates. This underscores the fact that HIV prevalence in West, East and Central Africa is generally lower than that in the base case, Southern Africa[60]. The fact that the models control for religion based cultural differences means that these (cultural differences) probably aren't the reason for the differences in HIV prevalence between Southern African countries and those in the rest of sub-Saharan Africa.

There probably are some other social drivers such as traditional beliefs that are being captured by the West, East and Central Africa dummy variables. These are likely to shape divergent views on death, personal risk as well as issues regarding stigma (see Iliffe, 2006:112-124). There are also likely to be differences across the regions regarding traditional views on sexual behaviour. Here it is crucial not to focus on a comparison of traditional measures of sexual behaviour such as the frequency of sex or the number of sexual partners in a lifetime. One reason for this is that survey based evidence on frequency of sex or the number of sexual partners in a lifetime shows little significant variation across country (Wellings et al. 2006). Despite the emergence of literature on the topic (see Poku 2005:73–75; Epstein 2007;Iliffe 2006; Hunter 2002, 2006; Parker et al. 2007; Mah and Halperin 2008; Mah 2008), it is hard to ascertain if there are differences in the patterns of sexual networks and concurrent sexual partnerships across the regions. The problem here is that it is very difficult to obtain cross country data that tells us something about long-term concurrent sexual relationships. Even country data based on national surveys of more limited data on sexual behaviour is suspect (see Chapter 4.3).

Another possible explanation for the regional differences in the level of HIV prevalence found in different regions could be the variation in HIV strains and sub-strains across these

[60] The descriptive analysis of the countries sampled in this chapter showed how South Africa, Zimbabwe, Zambia, Malawi and other Southern African countries topped the list of high prevalence countries.

regions. HIV comes in two types, HIV-1 and HIV-2. Worldwide, HIV-1 is the most common type although both types are transmitted by sexual contact, through blood, and from mother to child, and their resultant AIDS cases are clinically indistinguishable (Avert, 2010b). Concentrated in West Africa, HIV-2 is less easily transmitted, and the period between initial infection and illness is longer than in the case of HIV-1 (Avert, 2010b). This might be one of the many possible of factors that explain the coefficient on the West Africa dummy variable. However, explaining why Southern Africa has much higher prevalence rates than East and Central Africa is even harder. While all three regions have the HIV-1 strain, Subtypes A and D are most common in East and Central Africa. Subtype C is predominant in Southern and some parts of East Africa and about half of the world's infections are HIV-1, subtype C (Avert, 2010b).

The differences in the types of HIV in each region might help explain why Southern Africa (with subtype C) generally has worse epidemics especially given evidence that suggests that other subtypes (e.g. D) are more virulent and so mortality is higher among those infected. For instance, a study presented in 2006 found that Ugandans infected with subtype D (which is more effective at binding to immune cells) developed AIDS sooner than those infected with subtype A, and also died sooner (if not on ART) (Laeyendecker et al, 2006). Other research corroborated the Ugandan study and found that Kenyan women infected with subtype D had more than twice the risk of death over six years compared with those infected with subtype A. Baeten et al 2007. Two studies also found that subtype C (predominant in Southern Africa) was more transmissible than D (Blackard et al, 2001: Renjifo et al, 2004).

Iqbal and Zorn theorize that it is likely that HIV prevalence will have a negative relationship with measures of democratic governance. They argue that not only are democratic governments better able to respond to the needs of their people as argued by Lake and Baum (2001), Olson (1993) and Bueno de Mesquita et al. (2003), there is a link between democratic governance and increased health spending (Russett, 2004). This theory is supported by their regression models which show a negative relationship between HIV prevalence estimates and democratic governance (as measured by the POLITY SCALE). However, democratic governance often promotes an environment conducive to economic growth and investment factors which may lead to improvements in transport and communication infrastructure all of which may actually lead to increases in new HIV infections. This is because the development of such infrastructure makes sexual networks easier to maintain (see Chapter 5). When controlling for many other factors including the different conflict variables, Specification 1 (all regressions 1.1 to 1.5) shows a positive and statistically significant relationship between the democracy (POLITY IV) score and estimated HIV prevalence rates. The lack of reliable data on the development of transport and communication infrastructure prevents us from distinguishing how much of the effect of democracy on the epidemic can be attributed to development of such infrastructure.

Specification 1 (regressions 1.3 to 1.5) shows a negative albeit statistically insignificant relationship between HIV prevalence estimates and GDP per capita. Iqbal and Zorn also find the same. The "poverty causes AIDS" debate has long been a heated one. Stillwaggon (2000, 2002, 2006) argues that malnourished people, especially those infected with worms, weakened by Tuberculosis (TB) and malaria, and burdened by untreated sexually transmitted infections (STIs) are especially biologically vulnerable to infection. The logic is that the genital lesions caused by STIs and malnutrition weaken mucosal and skin defences which allows for more entry points for the virus (Stillwaggon, 2002:10). In addition, infection with parasites and other diseases over activates the immune system and so creates more target cells for HIV infection (Stillwaggon, 2002:11-12). It has also been argued by Bentwich et al. (2000) as well as Borkow and Bentwich (2006) that individuals who are infected with parasites and other endemic diseases have an elevated immune response which may increase their susceptibility to infection by the virus.[61]

Such arguments present a case for there to be greater emphasis on the treatment of STIs and other parasites but do not lead one to conclude that there are poverty-related biological pathways in driving the epidemic Nattrass (2009:2). There has been debate on whether malnutrition increases the susceptibility to HIV infection. The studies done have concluded differently. Although there has not been any consistent evidence linking vitamin deficiency and HIV infection (Dreyfuss and Fawzi 2002), some studies have shown evidence that improvements in nutrition might slow down the progression of disease (Fawzi et al. 2004). On the other hand, some randomized controlled trials where subjects were given

[61] Due to unavailability of data, none of the regressions in this thesis include measures of malnutrition or prevalence of STIs.

micronutrient supplements did not show a protective effect. Instead, they may have been more harmful than anticipated (Mills et al. 2005).

It is interesting to note that the evidence against a “poverty causes AIDS” argument has been mounting. For example, a study of malnourished children in Zambia showed that HIV negative malnourished children had normal CD4 counts (Hughes et al. 2009:349-50). On the other hand, the HIV positive malnourished children had below-normal CD4 counts and that these counts did not improve with better nutrition (Hughes et al. 2009:349-50). In her review of the literature, Nattrass (2009:2) concludes that although it is possible that people living in poor conditions with inadequate public health services are at greater risk of HIV infection, the evidence is limited and contradictory. Her analysis shows that there is no obvious relationship between a country’s per capita income and its HIV prevalence. In some instances poverty is driving risky behaviour, but in others, it is the richer people with the resources and income who get infected (Gillespie et al, 2007; Mishra et al, 2007; Potts et al, 2008). Clearly and as Nattrass, (2009) argues, the link between HIV and a country’s per capita income varies in different contexts (Nattrass, 2009:3). Indeed the debate is much more nuanced than this chapter attempts to explore[62].

Iqbal and Zorn argue that international trade has a wealth creating potential, is a source of government revenue and so allows for conditions that help fight the HIV epidemic (Iqbal and Zorn, 2010:151). They therefore expected to find a negative relationship between HIV prevalence estimates and economic openness (trade). However their regression models show a positive relationship between HIV prevalence estimates and economic openness. Iqbal and Zorn acknowledge the alternative argument that economic openness may actually lead to an upsurge in HIV prevalence as it allows for greater interaction of HIV infected persons crossing borders into lower prevalence regions (countries).[63] The regressions this chapter follow Iqbal and Zorn where economic openness is measured as a percentage. The percentage

[62] Only regression 1.2 shows the link between HIV prevalence rate estimates and GDP per capita as being statistically significant. Regressions 1.3 to 1.5 show that the relationship between HIV prevalence estimates and GDP per capita is not statistically significant when we control for regional difference in the epidemic (regional dummies).

[63] Iqbal and Zorn draw attention to the example of small-scale studies by Bwayo et al. (1994), Gysels, Pool, and Bwanika (2001) that point to the importance of long-haul truck drivers in the spread of HIV in southern and eastern Africa.

is obtained by dividing the sum of exports and imports by the level of GDP and multiplying this ratio by 100.

Specification 1 shows an interesting picture. Although regressions 1.2 and 1.3 show a small and negative relationship between HIV prevalence estimates and economic openness, regressions 1.4 and 1.5 which control for the level of militarization, the magnitude of the conflict (measured by using the major/minor conflict variables or the conflict scope variable) and presence of peace keepers (regression 1.5) show this to be a positive, small and statistically significant relationship. Regression 1.4 shows that holding all other factors constant, a 1 % increase in economic openness is associated with a 0.001% increase in HIV prevalence in the next year. Regression 1.5 shows that holding all other factors constant, a 1 % increase in economic openness is associated with an even smaller and almost negligible (0.0004 %) increase in HIV prevalence in the next year.

Assuming that the regressions are not mis-specified, this result could be interpreted to mean that in regressions 1.2 and 1.3, the economic openness variable might be capturing the inflow of resources linked to increases in the force ratio (number of armed personnel per capita). The trade in munitions is also linked to the magnitude of the conflict as bigger conflicts would need more resources to be mobilised. These factors may be confounding the regressions that do not control for increases in the force ratio. Overall, the fact that the coefficients are so small means that economic openness is probably not a key factor in the conflict –HIV nexus[64].

Unlike in Iqbal and Zorn, regressions 1.3, 1.4 and 1.5 show a negative relationship between health expenditure (total public and private health expenditures as a percentage of GDP) and estimates of HIV prevalence. However, only regression 1.5 shows this to be statistically significant. The regression shows that holding all other factors constant, a 1% increase in health expenditures is associated with a 0.3 % decrease in HIV prevalence in the next year. The results shown in these regressions (1.3, 1.4 and 1.5) are in line with what Iqbal and Zorn

[64] That said, it would be interesting to explore a model that distinguishes between different types of trade. There could be a difference between the effects of formal versus informal trade where the nature of informal trade is such that the returns on smuggling, and indirectly on access to foreign exchange, are so high that it incentives cross border sex work.

theorized their models would show (Iqbal and Zorn, 2010:156). Iqbal and Zorn expected to find the relationship between HIV prevalence estimates and health expenditures to be negative suggesting that higher health expenditures go a long way in the fight against the epidemic. However, as Iqbal and Zorn correctly note, the health expenditure variable is not an ideal measure as it does not distinguish between curative and preventative health expenditures. Therefore one cannot not make the conclusion that increases in overall health expenditures lead to positive outcomes (reduction in HIV prevalence) in the attempts to fight the epidemic.

Unlike in Iqbal and Zorn, regressions 1.2 to 1.5 show an inconsistent albeit statistically insignificant relationship between HIV prevalence estimates and total number of refugees in a country. The relationship is positive in regressions 1.2 and 1.3 and negative in regressions 1.4 and 1.5. The difference between these regressions is that models 1.4 and 1.5 contain not only the distance from the DRC variable but the variables controlling for the magnitude of the conflict, presence of peace keepers as well as the force ratio. Regardless, the inconsistency in the sign of the coefficient on the refugee variable is not entirely surprising. The refugee variable is not ideal as it fails to capture a key determinant of the spread of HIV. Refugees will only spread the epidemic if they come from home areas with particularly high prevalence rates. Therefore using two “refugee” variables where one notes the number of refugees from countries with HIV prevalence higher than the host while the other notes the number of refugees from countries with HIV prevalence lower than the host is likely to be more useful. This is the approach suggested and implemented by Davenport and Loyle (2009).

Specification 1 (regressions 1.4 and 1.5) shows a negative association between HIV prevalence estimates and the maturity of the epidemic. For instance, regression 1.5 shows that holding all other factors constant, a 1 % increase in the distance from the DRC’s capital city is associated with a 0.002 % decrease in estimated HIV prevalence rates. Since maturity is measured by the distance of each country from the DRC, Specification 1 shows that older (those closer to the DRC) epidemics generally have higher prevalence rates than younger ones. This is somewhat counter intuitive as one would have expected that older epidemics would have more matured epidemics that are on the decline (therefore have lower prevalence rates). However, countries closer to the DRC, more specifically those in the Southern African region have higher average prevalence rates than others such as Ethiopia, Chad and Nigeria.

Since the models capture regional differences in HIV prevalence by including West, East and Central Africa dummies, the distance from DRC variable probably explains the variation in HIV prevalence (estimates) attributable to the country's proximity to the DRC after controlling for other characteristics that a country shares with its neighbours (such as sharing particular trains and sub-strains of the virus). Therefore it is probably not because countries close to the DRC share similar strains of virus as the DRC that we see the result in regressions 1.4 and 1.5. Rather it probably shows the importance of transport infrastructure with regards to the spread of a virus like HIV. Although it is unquestionable that the virus spread to most corners of the continent, the traditional transport links (and close proximity) between the DRC and its Southern African neighbours allowed the virus to spread faster to those countries.

Regressions 1.4 and 1.5 show a negative association between HIV prevalence estimates and the level of militarization as measured by the force ratio. However the result is only statistically significant in regression 1.5. The regression shows that holding all other factors constant, a 1% increase in the force ratio is associated with a 0.13 % decrease in HIV prevalence rates in the next year. This result is consistent with the descriptive statistics presented in Section 7.2. While this result is interesting and may be seen as further evidence suggesting that HIV prevalence in the military is a statistically significant determinant of national HIV prevalence rates, there is major cause for caution. Such a conclusion cannot be reached until mechanisms or polices allowing for the testing of those in the military are established. This however, is highly unlikely as many of those to be tested are in unstable (or at war) regions. Even for those in peaceful environments, there may be ethical considerations that hamper the testing of individuals in the military.

Regressions 1.2 to 1.4 show that unlike in Iqbal and Zorn or Davenport and Loyle, domestic conflict has a statistically significant negative relationship with estimated HIV prevalence rates. For instance, regression 1.4 shows that holding all other factors constant, the presence of domestic conflict is associated with 24.8 % decrease in HIV prevalence rates in the next year. This is evidence in support of the argument that domestic conflict might actually slow down the epidemic (more is discussed in Section 7.5). The same is observed for the relationship between interstate conflict and HIV prevalence estimates in all regressions except 1.5. Regression 1.5 shows that when one controls for the presence of peacekeepers, the relationship between HIV prevalence and interstate conflict becomes positive, albeit statistically insignificant. Although it may be that much of the variation in estimated HIV prevalence rates explained by the interstate conflict variable in regressions 1.2 to 1.4 was actually due to the presence of peacekeepers, this is a speculative point. It is however interesting to note that the result (with respect to interstate conflict) corroborates that presented by Paxton (2009).

Further, regression 1.5 shows that major conflicts have a negative and statistically significant relationship with HIV prevalence estimates. Holding all other factors constant, a major conflict is associated with a 44.3 % decrease in HIV prevalence in the next year. Although the relationship is not statistically significant, minor conflicts also have a negative relationship with conflict. More on the possible reasons why the coefficients on the conflict variables in the regressions presented in this chapter are different from Iqbal and Zorn (2010) as well as Davenport and Loyle (2009) is covered in Section 7.5.

One key difference between regression 1.4 and 1.5 is that whereas regression 1.5 uses the minor/major conflict variables to proxy the magnitude of the conflict, regression 1.4 uses the geographical scope variable. This scope variable (calculated by dividing the radius of the region affected by the conflict by the total land area of the country) also has a negative and statistically significant relationship with the HIV prevalence estimates. Holding all other factors constant, a 1 % increase in the geographical scope of a conflict is associated with a 11.8 % decrease in HIV prevalence in the next year. This might be interpreted to show that whether the intensity of conflict is measured either by a count of battle deaths (major/minor conflict variables) or by the size of the geographical region affected, the magnitude of a conflict is negatively associated with HIV prevalence estimates.

Regression 1.5 also includes a variable showing each year a conflict involved peacekeepers from other countries. Holding all other factors constant, a 1 % increase in the number of peace keepers in a conflict country is associated with a 8.8 % decrease in HIV prevalence in the next year. The result shows a negative and statistically significant relationship between the presence of peace keepers and HIV prevalence estimates. However, it must be noted that this variable is less than ideal as it does not distinguish between peacekeepers from high and those from low prevalence countries. This might be confounding the results observed in regression 1.5. For instance if one interprets the result shown in regression 1.5 as meaning that the inflow of peace keepers is associated with increases in the level of the epidemic, then one is implicitly assuming that the peace keepers have infection rates high enough to influence the trajectory of the epidemic in the host nation.

7.4.1.2 Discussion

It is well and good to have a well thought out theoretical model or framework but the results will be biased or determined not only by the choice of model specification but also by the choice of sample as well as the choice and quality of variables used.

Table 7.6 Comparison of the Pooled OLS regressions

	Pooled OLS	
Similarities	**Iqbal and Zorn** Linear model Same variables used No control for spatial dependence	**Regression 1.2** Linear model Same variables used No control for spatial dependence
Differences	Sample : 43 African countries Sample : 1997 - 2005	Sample : 34 Sub-Saharan countries with generalised epidemics Sample : 1990 - 2005
Results	Domestic conflict (positive) Interstate conflict (positive) Muslim religion (negative) Education (negative) Economic Openness (positive) Refugees (positive)	Domestic conflict (negative) Interstate conflict (negative, but positive in reg 1.5) Muslim religion (negative) Education (positive, not stat sig) Economic Openness (negative, not stat sig) Refugees (positive, not stat sig)

Source: Own regressions. Only key variables from Iqbal and Zorn's and model 1 are shown.

The regression presented by Iqbal and Zorn as their Full OLS model is different from regression 1.2 primarily because the two models are based on different samples with respect to the countries in the study as well as the time periods studied. However when one looks solely at regressions 1.1 to 1.5, it is evident that it is a hard ask to draw definitive conclusions from these regressions. The nature of the individual relationships of the independent variables with HIV prevalence rate estimates changes depending on the context i.e. on which control variables have been used. Therefore, researchers ought to present a series of models showing these differences and be cautious in drawing firm conclusions.

Indeed a number of the control variables used in regressions 1.1 to 1.5 are inadequate on the grounds that they do not contain explicit information from which we can draw direct (let alone linear) relationships with estimated HIV prevalence data. Important questions to answer would be for instance whether we can infer something about the potential effectiveness of HIV education campaigns from adult literacy rates? Do measures of international trade such as economic openness provide an adequate indication of inflow of resources that might help fight the epidemic or do they, in some instances represent investments in security and defence (arms etc.). Does an increase in aggregate health expenditures indicate an increase in resources put to HIV preventative care, curative care for opportunistic infections or infrastructure projects?

These questions are difficult to answer as is and using these controls when modelling the relationship between armed conflict and estimated HIV prevalence data (not actual data points) is probably asking too much of both the dependent and independent variables. In the event that some gain can be found in pursuing regression approaches to investigating the link between estimated HIV prevalence rates and conflict, it is important to assess what type of regression model is better suited to providing reasonable results. This is part of the motivation for presenting the different types of regressions that follow.

7.4.2 Cross sectional OLS

Chapter 6 detailed how Davenport and Loyle (2009) attempted to improve on both the theoretical framework and the model specification presented by Iqbal and Zorn (2010). Specification 2 (based on Davenport and Loyle's cross sectional OLS regression and shown in Table 7.7) shows results similar to those shown in Specification 1. However, the interpretation of the coefficients is not the same. The key difference between Specifications 1 and 2 is that Specification 2 uses aggregated data to look at variation in different factors (the controls) between countries over a fixed period and how they affect HIV prevalence estimates.

One of the other differences in model specification between Iqbal and Zorn and Davenport and Loyle is that the latter don't use a simple count of refugees in a country as a variable. Instead they make a distinction between refugees from medium and lower (than host country) HIV prevalence countries and those from higher HIV prevalence countries. Specification 2 (regressions 2.3 to 2.5) controls for population density, religion, education and the level of democracy in a country, trade, health expenditure, as well as conflict by using either the major, minor, domestic or interstate conflict variables. As was the case with Davenport and Loyle in Specification 2, I take the average of high HIV prevalence refugees variable (logged), medium and low HIV prevalence refugee (logged), log of GDP, log of population density, trade as a percentage of GDP, health expenditures as a percentage of GDP, log of the force ratio and log of conflict scope as dependant variables. Unlike Davenport and Loyle, these averages are taken over the period between 1990 and 2005. For the conflict variables, I use the total number of conflict years in the period 1990 to 2005 which were classified as domestic, intrastate, major and minor conflict. The distance from DRC, East Africa, West Africa, Central Africa and the Muslim religion variables are all time invariant and so the 2005 values were used. Since the dependant variable is logged, the coefficients in the regression can be interpreted as elasticities.

The Specification 2 models show that having refugees from a higher prevalence region is associated with higher prevalence rates in the host country[65]. However, unlike in Davenport and Loyle, the relationship is not statistically significant. In addition, the size of the coefficient is very small indicating that if an effect exists at all, it is all but minute.

Specification 2 also shows a negative relationship between HIV prevalence estimates and GDP per capita. Davenport and Loyle as well as Iqbal and Zorn also find the same[66].

[65] Regressions 1.1 and 1.2 which do not account for conflict show this relationship as a negative one

[66] Regression 1.1 which does not account for region specific factors (West, East and Central Africa dummy variables) shows this as a negative relationship.

However, Specification 2 shows that unlike in Davenport and Loyle, the link between GDP per capita and estimated HIV prevalence is not statistically significant. As already discussed, the relationship between measures of a country's wealth such as GDP per capita and HIV varies in different contexts and it is unlikely that regression analysis involving estimated (and not actual) HIV prevalence will help understand the nuanced nature of the link.
Regression 2.1 shows, that HIV prevalence estimates have a statistically insignificant and negative relationship with population density. This corroborates the result found by Davenport and Loyle (2010) as well as in regressions 1.1 and 1.2. However, as was the case in Specification 1, once we control for regional differences using the West, East and Central Africa dummies the relationship becomes positive although still statistically significant. This is a more intuitive result as prior chapters indicated that the epidemic is likely to spread faster in more closely populated areas.

Regression 1.5 shows that holding all other factors constant, West African countries are associated with HIV prevalence rates 77.8% lower than those in Southern Africa. Further, holding all other factors constant, East African countries are associated with HIV prevalence rates 66.7 % lower than those in Southern Africa. The coefficient on the Central Africa dummy variable is more statistically significant than that for the West and East Africa dummy variables. Regression 1.5 shows that holding all other factors constant, Central African countries are associated with HIV prevalence rates 70.9 % lower than those in Southern Africa

The difference between the result found by Davenport and Loyle and Specification 2 is that Davenport and Loyle use a regional dummy which lumps all sub-Saharan countries together. This, as noted in the discussion of the results from regressions 1.2 to 1.5, ignores the difference between different countries in the sub-Saharan countries. Regressions 2.2 to 2.5 show that West, East and Central African countries have certain characteristics (not controlled for in the regressions) which lead them to have lower prevalence rates than those in Southern Africa. As already highlighted, these factors could include regional difference in sexual practice (not attributable to religion) as well as the regional difference in strains and sub strains of the epidemic.

Table 7.7 Cross sectional OLS (Specification 2)

	Model				
Variable	**2.1**	**2.2**	**2.3**	**2.4**	**2.5**
Log of High HIV refugees	5.9E-6	1.5E-6	7.3E-6	7.6E-6	2.2E-6
	(4.3E-6)	(6.3E-6)	(9.7E-6)	(6.7E-6)	(4.7E-6)
Log of Medium and Low HIV refugees	3.3E-6	3.7E-6	5.3E-6	3.0E-6	3.0E-6
	(1.3E-6)	(2.1E-6)	(3.0E-6)	(2.2E-6)	(2.0E-06)
Log GDP per capita	0.011	-0.134	-0.061	-0.162	-0.079
	(0.123)	(0.175)	(0.230)	(0.258)	(0.172)
Log of Population density	-0.100	0.083	0.032	0.020	-0.121
	(0.175)	(0.224)	(0.284)	(0.302)	(0.291)
Trade		0.001	0.005	-6.2E-4	-0.002
		(0.005)	(0.005)	(0.005)	(0.003)
Health expenditures		1.350 *	1.317	0.319	0.348
		(0.742)	(0.977)	(1.014)	(0.691)
Regime type (democracy)		0.014	0.056	0.014	0.012
		(0.018)	(0.032)	(0.015)	(0.016)
West Africa		-2.320 **	-1.711 ***	-1.329 ***	-1.507 *
		(0.891)	(0.653)	(0.736)	(0.734)
East Africa		-1.999 **	-1.953 **	-0.541 **	-1.099 *
		(0.797)	(1.153)	(1.122)	(0.602)
Central Africa		-0.626	-0.146	-1.204	-1.235 **
		(0.843)	(0.770)	(0.817)	(0.465)
Domestic Conflict			0.049	-0.021	-0.031
			(0.104)	(0.108)	(0.043)
Major Conflict			-0.177	-0.126	
			(0.144)	(0.143)	
Minor Conflict			-0.040	-0.039	
			(0.091)	(0.094)	
Log of Conflict Scope			1.161		0.135
			(0.759)		(0.426)
Log of Peace keeping operation			0.164	0.168	-0.025
			(0.128)	(0.136)	(0.071)
Interstate Conflict			0.009	-0.126	0.085
			(0.551)	(0.597)	(0.424)
Muslim religion	-1.566 ***	-0.816 ***		-1.040	-1.021 **
	(0.384)	(0.875)		(0.727)	(0.447)
Log force ratio				-0.157	
				(0.407)	
Log of Distance from the DRC	-1.3E-4 ***				-1.3E-4 ***
	(6.5E-6)			0.407	(2.7E-5)
R^2	0.5904	0.5334	0.6221	0.6238	0.8394
Standard errors	Robust	Robust	robust	robust	Robust
P-value	0.0000	0.0000	0.0000	0.0000	0.0000
No of observations	33	32	32	32	32

Source: Own regression analysis

Specification 2 shows that as was found by Davenport and Loyle, there is a positive relationship between the level of trade and estimated HIV prevalence. The effect is very small and unlike in Davenport and Loyle, Specification 2 shows that it is not statistically significant. The result provides at best, weak support for their theory that HIV may be spread via trade as infected people travel to less infected regions. This is an argument also acknowledged by Iqbal and Zorn as trade allows for greater interaction of HIV infected persons crossing borders into lower prevalence countries. However, regression 2.4 and 2.5 show a different picture. They show a negative relationship between HIV prevalence estimates and the level of Trade. This instead supports the arguments presented by Iqbal and Zorn that international trade has a wealth creating potential that helps government fight the HIV epidemic (Iqbal and Zorn, 2010:151).

As is the case with Davenport and Loyle, regressions 2.2 to 2.5 show a positive relationship between health expenditure and estimates of HIV prevalence. However, the result is not statistically significant. Another similarity between Specification 2 and Davenport and Loyle is that they find a positive statistically insignificant relationship between the regime type indicator and estimated HIV prevalence.

Specification 2 (regressions 2.1 and 2.5) shows a negative association between HIV prevalence estimates and the maturity of the epidemic. The regressions show, as did Specification 1, that older (those closer to the DRC) epidemics generally have higher prevalence rates than younger ones. For instance, regression 1.5 shows that holding all other factors constant, a 1 % increase in the distance from the DRC's capital city is associated with a 0.001 % decrease in estimated HIV prevalence rates. It would be interesting to see whether the panel regressions to follow show a similar result which, as already discussed is somewhat counter intuitive.

Regression 2.4 shows a negative association between HIV prevalence estimates and the level of militarization. Unlike in Specification 1, this result is not statistically significant. In other words, the result is less powerful than that from Specification 1. Both regressions however do not help explain how increases in militarization lead to the spread of the epidemic. They do little to clarify the debate touched on in Chapter 5 as to whether armed personnel have higher prevalence rates than comparable civilian populations and whether their purported riskier sexual behaviour has contributed to this. Indeed because the regressions do not account for these factors, one could speculate that there are a myriad of factors (e.g. risky sexual practices) associated with militarization (and not controlled for here) which are being captured by the militarization variable.

Like Specification 1, Specification 2 (regressions 2.3 to 2.5) shows that domestic conflict has a statistically insignificant negative relationship with the estimated HIV prevalence rates[67]. Had the result been statistically significant, it would have provided evidence in support of the argument that domestic conflict might actually slow down the epidemic. The same is not observed for the relationship between interstate conflict and HIV prevalence estimates. Regressions 2.3 to 2.5 show this to be positive[68]. It must be stressed however, that in both cases the relationships are not statistically significant. Regressions 2.3 and 2.4 show that major conflicts have a negative and statistically insignificant relationship with HIV prevalence estimates. The same is observed for the relationship between minor conflicts and HIV prevalence estimates. As was the case with Davenport and Loyle, the scope variable has a negative and statistically insignificant relationship with estimated HIV prevalence. Section 7.5 discusses these results in more detail.

Specification 2 (regressions 2.3 to 2.4) show a positive, albeit statistically insignificant link between hosting peace keeping forces and increases in HIV prevalence. Whereas Davenport and Loyle find this relationship to be negative (as does regression 2.5), they posit that their result differs from their hypothesis because the missions are often in the form of non-combat UN programs and public health projects (Davenport and Loyle, 2009:28). Further they explain that these are used for HIV education and prevention and so this potentially counters the negative effects of peacekeeper/civilian interaction (Davenport and Loyle, 2009:28). This explanation is far from convincing. It is a far stretch to assume that a variable that simply counts the presence of peace keeping troops also is a good proxy for the number and reach of other auxiliary humanitarian assistance.

[67] Paxton finds a similar result, although it is statistically significant
[68] Similar to the result shown by Paxton

However the difference between the results presented here (and hypothesized by Davenport and Loyle) and those presented by Davenport and Loyle is hard to pin down. On the one hand it could be as a result of the difference in the time period sampled while it could also be because of the difference in the countries sampled.[69] In any case, the variable used to proxy the presence of peace keepers is a poor one as the presence of peace keepers will only increase prevalence if these troops are infected (or at least from higher prevalence countries) by the virus when they enter the host country.

7.4.2.2 Discussion

Table 7.8 Comparison of the Cross sectional OLS regressions

	Cross sectional OLS	
Similarities	**Davenport and Loyle** Linear model Similar but not identical variables used	**Regression 2.3** Linear model Similar but not identical variables used
Differences	Sample : Global and Sub-Saharan countries Sample : 1997 - 2007	Sample : 34 Sub-Saharan countries with generalised epidemics Sample : 1990 - 2005
Results	High HIV refugees (positive) GDP (negative) Trade (positive) Health expenditures (positive) Peace keepers (positive) Region (negative)	High HIV refugees (positive, not stat sig) Log GDP per capita (negative, not stat sig) Trade (positive, not stat sig) Health expenditures (positive, not stat sig) Peace keeping operation (positive, not stat sig) West Africa (negative) East Africa (negative) Central Africa (negative)

Source: Own regressions. Only variables that are statistically significant in Davenport and Loyle's model are shown in Table 7.8

Two key lessons can be drawn from the summary of the Pooled OLS regressions. The first is that the choice of sample (countries in the study as well as the time period under consideration) may bias the results of any regression analysis. Table 7.8 indicates the differences between the similar but not identical regressions; regression 2.3 in Table 7.7 and that presented by Davenport and Loyle (2010). These were exhausted in the discussion in

[69] While the regressions presented here are for the period 1990 to 2005, Davenport and Loyle only consider the period 1997 to 2007. Further they use a global sample while this study only considers 34 sub-Saharan countries.

Section 7.4.2. The second lesson was that the quality of both the independent and dependent variable is very important.

7.4.3 Fixed Effect models

Fixed effects models have the advantage of allowing one to focus only on within country variation in HIV prevalence estimates. This means that the results in Table 7.9 should be interpreted as relationships between HIV prevalence estimates and the independent variables over time assuming that there are no cross country differences in time invariant characteristics. Further, each coefficient in the regression represents the change in the dependant variable (HIV prevalence estimates) for each 1-unit increase in the independent variable within each country. However, because the dependant variable is logged, the regression coefficients may be interpreted as elasticities. In terms of the direction of association between HIV prevalence estimates and the conflict variables (major, minor, domestic interstate, geographical scope) the result of all Specification 3 regressions (3.1 to 3.5) are similar to that of Specifications 1 (especially 1.5) and 2.

The major and minor conflict variables both have negative associations with estimated HIV prevalence rates. However, only the major conflict variables are statistically significant. Regressions 3.3 and 3.4 show that when we control for the independent variables in Table 7. and also focus only at within country variation in HIV prevalence, major conflict years are associated with future (next year) prevalence rates 18.0 % lower than non-major conflict years. Specification 3 also shows that the domestic conflict variable has a statistically insignificant negative relationship with the estimated HIV prevalence rates[70]. Regressions 3.3 to 3.5 show the relationship between interstate conflict and HIV prevalence to be positive and statistically insignificant. As was the case with Davenport and Loyle, the scope variable has a negative and statistically insignificant relationship with estimated HIV prevalence. Section 7.5 discusses these results in more detail.

With regards to the other control variables, the fixed effects models show a positive, large and statistically significant relationship between HIV prevalence estimates and population

[70] This result is similar to that presented by Paxton (2009) although these regressions are not directly comparable since Paxton uses a different sample and the Ethnic War control variable not used here. The regressions presented here also control for the presence of refugees which are missing in Paxton

density. This relationship was not statistically significant in Specifications 1 and 2. This shows that there are some country specific factors not captured by Specifications 1 and 2 that conflate the significance of the relationship between estimated HIV prevalence and population density[71].

Table 7.9 also shows that under a fixed effects model such as regression 3.2 where we don't control for any of the measures of armed conflict, a 1% increase in GDP per capita is associated with a 0.3 % increase in estimated HIV prevalence rates. When controlling for conflict using the major, minor, domestic, interstate and force ratio variables as is done in regression in regression 3.4, a 1 % increase in GDP per capita is also associated with about 0.3 % decrease (within country) in estimated HIV prevalence. The fixed effects models also show a positive and statistically significant relationship between estimated HIV prevalence and economic openness. However this effect is small and on average, the regressions show that holding all other factors constant, a 1 % in economic openness is associated with a 0.001 % increase (within country) in estimated HIV prevalence .

This result corroborates the theory posited by Davenport and Loyle as well as Iqbal and Zorn that HIV may be spread via trade as infected people travel to less infected regions. As is the case with Specification 2 as well as Davenport and Loyle the fixed effects models show a positive statistically insignificant relationship between health expenditure and estimates of HIV prevalence.

A similarity between Specification 3, Iqbal and Zorn and the fixed effects models presented here is that the relationship between the democracy variable and the estimated HIV prevalence is both positive and statistically insignificant. Regression 3.4 shows a negative and statistically insignificant association between HIV prevalence estimates and the level of militarization. This a result also found in Specification 2.

[71] These factors would not conflate the FE model results since FE control for all such country specific factors.

Table 7.9 Fixed Effects model (Specification 3)

	Model				
Variable	**3.1**	**3.2**	**3.3**	**3.4**	**3.5**
Log of High HIV refugees $_{t-1}$	1.4E-7	3.7E-7	3.4E-7	3.2E-7	3.8E-7
	(2.4E-7)	(2.4E-7)	(2.4E-7)	(2.4E-7)	(2.4E-7)
Log of Medium and Low HIV refugees $_{t-1}$	-1.2E-7	-1.3E-7	-1.7E-7	-1.8E-7	-1.2E-7
	(2.1E-7)	(2.0E-7)	(2.1E-7)	(2.1E-7)	(2.1E-7)
Log of GDP per capita $_{t-1}$	0.052	-0.317 *	-0.295 *	-0.344 **	-0.300 **
	(0.136)	(0.167)	(0.168)	(0.171)	(0.169)
Log of Population density	2.553 ***	2.541 ***	2.508 ***	2.481 ***	2.515 ***
	(0.184)	(0.181)	(0.183)	(0.184)	(0.184)
Economic openness $_{t-1}$		1.0E-3 **	9.6E-4 **	9.7E-4 **	1.0E-3 **
		(4.6E-4)	(4.6E-4)	(4.6E-4)	(4.6E-4)
Health expenditures		-0.121	-0.153	-0.166	-0.146
		(0.554)	(0.556)	(0.555)	(0.557)
Democracy t-1		2.5E-3 ***	2.3E-3 **	2.4E-3 **	2.4E-3 **
		(9.2E-4)	(9.4E-4)	(9.4E-4)	(9.4E-4)
Domestic Conflict t-1			-7.8E-3	-8.3E-3	-5.3E-3
			(0.091)	(0.091)	(0.078)
Major Conflict t-1			-0.198 *	-0.193 **	
			(0.103)	(0.103)	
Minor Conflict t-1			-0.014	-0.014	
			(0.093)	(0.093)	
Log of Conflict Scope t-1			0.012	0.011	-0.003
			(0.029)	(0.029)	(0.025)
Log Peace keeping operation t-1			0.084	0.080	0.083
			(0.110)	(0.110)	(0.110)
Interstate Conflict t-1			0.154	0.149	0.106
			(0.142)	(0.142)	(0.138)
Muslim religion	-1.566 ***				-1.021 **
	0.384				0.447
Log force ratio $_{t-1}$				-0.090	
				(0.059)	
R2					
Between	0.0012	0.0135	0.016	0.0171	0.0167
Within	0.3137	0.3399	0.3493	0.3528	0.3417
Pesaran's p value	0	0	0	0	0
Standard errors	robust	robust	robust	robust	robust
F test	51.76	32.07	17.76	16.71	20.38
No of observations	490	475	475	475	475

Source: Own regression analysis

Regressions 3.3 to 3.5 show a positive, and statistically insignificant link between hosting peace keeping forces and increases in HIV prevalence. This is in tune with most other regressions presented thus far (except for e.g. regression 2.5). Despite the advantage of allowing one to assume away all time invariant factors that may influence or confound the relationships between HIV prevalence estimates and time variant independent variables, fixed effects models like the ones presented in Table 7.7 ignore the fact that the HIV epidemic is dynamic. They ignore the fact that this year's estimated prevalence rates are a function of previous years' rates.

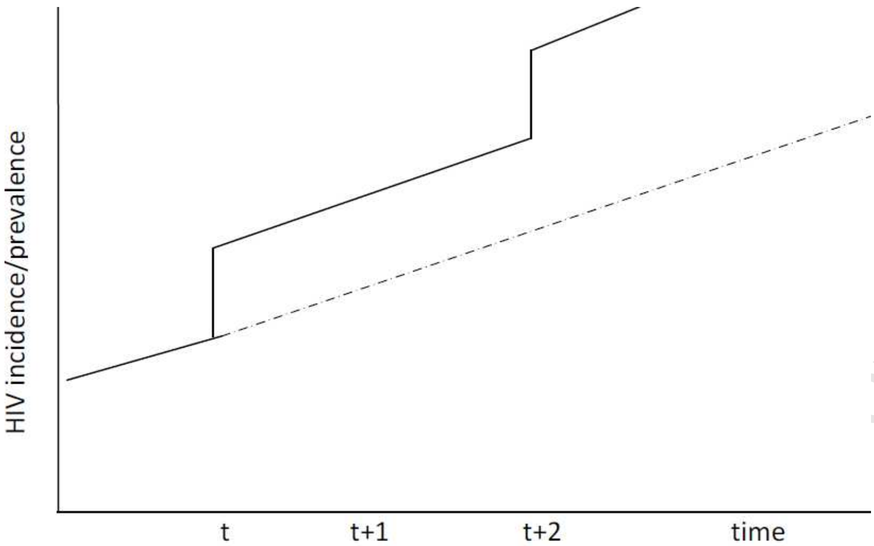
7.5 An alternative Approach: Dynamic Panel Models

While the fixed effects and random effects models (the Paxton models) have a lot to offer compared to cross sectional or pooled OLS models, they too do not take into account that the trend in the dependent variable (HIV prevalence estimates) have their own dynamic over time. This limitation was discussed in greater detail in section 6.2.4. One way of attempting to take some of this into account is to employ dynamic panel models.

A number of aspects of politics are inherently dynamic and there have been attempts to model the dynamics of political phenomena by including lags of the dependent variables as regressors (Wawro, 2002:2). These include dynamic models of party identification (Green and Palmquist, 1990; Green and Yoon, 2002) and campaign finance (Krasno et al, 1994). However as noted by Wawro (2003:3), these studies estimate models on a period by period basis and do not take advantage of the panel structure of the data and the efficiency it provides (as they estimated a separate, cross sectional model for each time period). Dynamic panel models however, include both lagged dependent variables and unobserved individual-specific effects. In addition, these models allow for dynamics to be modelled while accounting for individual level heterogeneity.

Dynamic panel analysis is especially attractive in that it can accommodate the dynamic nature of HIV prevalence (though the problem of having an estimated dependent variable still remains). In a dynamic panel model, if conflict at time t results in an increase in HIV prevalence estimates at time t+1, then regardless of whether there is another conflict at time t+1, HIV prevalence estimates at time t+2 will be a function of HIV prevalence estimates the year before (t+1). This is shown in Figure 7.2. In other words, HIV prevalence is inherently dynamic in these models. Secondly, because epidemics in different countries are likely to be influenced by country-specific characteristics, a dynamic model would serve us well as it includes as part of its specification, unobserved individual level heterogeneity (Wawro, 2002:3).

Figure 7.2 Shocks to dynamic models



Source: Own diagram

Dynamic panel models explicitly include variables to account for past levels of HIV prevalence and time invariant country specific effects. This affords us an opportunity to understand better what factors drive HIV prevalence over time whilst distinguishing between what Wawro (2002:3) call "true" dynamics and factors that vary across, but not within, countries over time. The basic dynamic panel model can be estimated as follows;

$$Y_{it} = \alpha Y_{it-1} + X_{it}\beta + \mu_i + v_{it}$$

Where i, denotes the cross sectional units, t the time periods, X_{it} and a vector of exogenous explanatory variables, μ_i is an unobservable country level effect which is constant across time within individuals and v_{it} is a random disturbance term.

While models such as that by Iqbal and Zorn, Davenport and Loyle's OLS regression can be adapted into dynamic models by adding lags of the dependent variable as regressors, OLS models produce biased and inconsistent estimates when used in dynamic panel data analysis.

Other standard within groups or Least Squares Dummy Variable (LSDV) transformations remove the individual effects but produce biased and inconsistent estimates because there is correlation between the transformed lagged dependent variable and the transformed disturbance (Baltagi, 1995:125-126; Kiviet, 1995). OLS estimates will be biased upwards whilst the within group estimates are biased downwards (Bond, 2002:7). The Generalised Method of Moments (GMM) estimator is an attempt to find middle ground (Bond, 2002:7).

The Arellano and Bond (1991) estimator is a GMM estimator that includes p lags of the dependent variable as covariates and contains unobserved panel-level effects (fixed or random). Arellano and Bond (1991) derived a consistent generalized method-of-moments (GMM) estimator that deals with the correlation between the lagged dependent variables and the unobserved panel-level effects. This was later improved by Arellano and Bover (1995).

Another option is the Arellano and Bover/Blundell and Bond (1995, 1998) estimator, which built on the Arellano and Bond (1991) by using additional moment conditions so as to improve the performance of the Arellano and Bond (1991) model. These dynamic models can be modelled strictly as GMM estimators or with an option for robust standard errors, corrected for panel level autocorrelation and heteroscedasticity. The system GMM assumes that there is no autocorrelation in the idiosyncratic errors and requires the initial condition that the panel-level effects be uncorrelated with the first difference of the first observation of the dependent variable. Unless the Blundell-Bond (1998) estimator is adjusted for spatial dependence, it might produce inconsistent results.

Table 7.10 Summary of the panel regression models

Model	Transformation	Regressors	Consistency
LSDV/FE	Within	$Y_{i,t-1}$, X_{it}	No
PCSE (Prais-Winsten)	Within, Between	$Y_{i,t-1}$, X_{it}	No
First Difference GMM (Arellano-Bond (1991))	Δ	$\Delta Y_{i,t-1}$, ΔX_{it}	Yes
System GMM (Arellano-Bover/Blundell-Bond (1995,1998)	Δ	$\Delta Y_{i,t-1}$, ΔX_{it}, $Y_{i,t-1}$, X_{it}	Yes

Source: Adapted from Eigner (2009)

Table 7.10 shows some of the models that may be used in dynamic panel analysis. First, the OLS models presented by Iqbal and Zorn (2010) and Davenport and Loyle (2009) as well as new fixed effects models can be adjusted by adding lags of the dependent variable as regressors but this will produce inconsistent estimates as shown in Table 7.10. The more appropriate models to use for dynamic analysis would be the First Difference GMM and System GMM (Bover/Blundell and Bond estimator) as these will be consistent.

7.5.2 System Generalized Method of Moments (GMM) models

Specification 4 shows the results obtained from the dynamic regression models (system GMM). Table 7.11 shows these results. A number of tests were run in order to justify the use of the system dynamic models. The most crucial would be the Arellano-Bond serial correlation tests. As shown in Table 7.11, the Arellano-Bond (AR1) statistics were significant while the Arellano-Bond (AR2) statistics were insignificant at a 5% level of significance. Thus means that there was no serial correlation in the level residuals and so the system GMM and its moment conditions are appropriate (Cheng et al, 2000:392). The reader may also refer to Blundell and Bond (1998) for a detailed explanation of the system GMM. Other standard regression tests were also carried out[72].

Table 7.11 highlights a very strong and possibly overwhelming self-reinforcing effect of estimated HIV prevalence rates' past values on their current value. The coefficient on the lagged HIV prevalence rate variable is stable and very high (at about 0.98). This means that for all the other variables, we expect to see smaller coefficient than was the case for Specifications 1, 2 and 3. This is because current HIV prevalence rates are primarily a function of previous HIV prevalence rates and are on the short term determined less by shocks such as conflict or a refugee influx.

[72] In Stata xtdpdsys is used for the Arellano-Bover/Blundell-Bond (1995, 1998) estimator and xtabond estimates the Arellano and Bond (1991). Using the Wald test, we no found evidence of heteroscedasticity and also no found evidence of serial correlation (AR1) using the Wooldridge test. There also was no evidence of multi-colinearity.

Regardless, the system GMM model shows that unlike other models presented thus far, the relationship between estimated HIV prevalence and the High HIV refugees variable is statistically significant. Although the effect is very small, it is fairly consistent across all regressions 4.1 to 4.5. For instance, regressions 4.1, 4.3 and 4.5 show that holding all other factors constant, a 1 % increase in the high HIV prevalence rate variable is associated with a 0.0000001 % increase in estimated HIV prevalence rates. However the association between estimated HIV prevalence and the Medium to Low HIV refugees variable is not statistically significant. As was the case with Specifications 2, 3 and Davenport and Loyle, the small size of the coefficients suggests that the link between HIV prevalence estimates and the presence of refugees should not be overstated.

The system GMM models also show that even when one accounts for the dynamic nature of HIV prevalence estimates, there is a negative relationship between HIV prevalence estimates and GDP per capita. However, like the results from Specification 2 and the random effects models, the relationship is not statistically significant. The system GMM models also show that like the other models already presented, the relationship between population density and HIV prevalence estimates is not only positive but also statistically insignificant. Clearly the link between population level HIV prevalence and population density depends on the context. Unlike infections such as the cold that spread with minimal contact between persons, HIV is spread commonly through exchange of body fluids. Whereas it may be easier to link increases in population density to increases in infections of highly infectious diseases such as the common cold, the link is much more complex when looking at the spreading of HIV.

Specification 4 also shows that West, East and Central African countries are associated with prevalence rates lower than those in Southern African countries. However, unlike in the other models presented thus far, the coefficients on the West, East and Central Africa are much smaller and not statistically significant. This is probably because the inclusion of the lag of HIV prevalence estimates as a control (introduction of dynamics) explains much of the variation in estimated HIV prevalence. More interestingly, the regressions show that once the conflict variables are added, only the Central Africa regional dummy remains statistically significant. The explanation here could be that because most of the conflict countries in the sample are in West and East Africa (excluding Angola and DRC)[73], the conflict variables actually explain some of the influence these regional dummies had on the estimated HIV prevalence variable.

Specification 4 shows that the relationship between the measure of economic openness and estimated HIV prevalence varies. The fact that the relationship is negative for all models 4.3 to 4.5 is contradictory to the results observed for the fixed effects and random effects models. These are the models that control for conflict using the different variables already discussed.

The interpretation here could be that when one controls for different factors including conflict, increases in economic openness are associated with decreases in the level of (estimated) HIV prevalence rates. Regression 4.3 shows that holding all other factors constant, 1 % increase in economic openness is associated with a 0.00004 % increase in estimated HIV prevalence rates in the next year. In other words, Specification 4 shows that there is hardly any merits to what Iqbal and Zorn argue, which is that international trade helps a country mobilise resources that help fight the HIV epidemic (Iqbal and Zorn, 2010:151).[74]

The health expenditures variable (a poor measure for reasons already discussed) does not have a statistically significant relationship with estimated HIV prevalence. The system GMM models show similar results to all other models with regard to the relationship between the democratic status of a country and estimated HIV prevalence estimates. Specification 4 shows a positive and statistically significant relationship between the level of democracy in a country and estimated HIV prevalence rates. However the effect is very small. For instance, regression 4.4 shows that holding all other factors constant, a 1 % increase in democracy (the variable is on a 1 to 100 scale) is associated with a 0.00081 % increase in estimated HIV prevalence rates in the next year.

Specification 4 shows that the relationship between the distance from the DRC variable and estimated prevalence rates is negative, small and statistically significant. The same is observed for the other models presented in this thesis. In other words, even when one accounts for the dynamic nature of HIV prevalence estimates, older epidemics generally have

[73] In the sample, DRC is classified as Central African whilst Angola is Southern African.
[74]

higher prevalence rates than younger ones. As to be expected, the dynamic analysis shows a negative and statistically significant relationship between the prevalence of male circumcision and estimated HIV prevalence. Regression 4.3 shows that holding all other factors constant, a 1 % increase in prevalence of male circumcision is associated with a 0.154 % decrease in estimated HIV prevalence rates.

The relationship between the level of militarization/force ratio and estimated HIV prevalence is generally consistent for all specifications presented thus far. The system GMM models show that there is a negative association between estimated HIV prevalence rates and the force ratios even if one assumes that HIV prevalence rates are dynamic in nature. However, since the relationship is not statistically significant, the relationship should not be overstated.

Unlike in Davenport and Loyle (but in tune with the models presented in this chapter), Specification 4 shows that domestic conflict has a negative relationship with the estimated HIV prevalence rates. Despite being statistically significant, the relationship is relatively weak (small coefficients). For instance, regression 4.3 predicts that holding all other factors constant, major conflict years are associated with future (next year) prevalence rates 2.3 % lower than non-major conflict years.

Table 7.11 System GMM model (Specification 4)

	Model				
Variable	**4.1**	**4.2**	**4.3**	**4.4**	**4.5**
Log of HIV prevalence $_{t-1}$	0.860 ***	0.890 ***	0.890 ***	0.890 ***	0.890 ***
	(0.026)	(0.027)	(0.027)	(0.029)	(0.029)
Log of High HIV refugees $_{t-1}$	1.3E-8	1.4E-7 **	1.3E-7 **	1.2E-7 **	1.3E-7 **
	(8.8E-8)	(1.4E-7)	(1.2E-7)	(1.0E-7)	(1.1E-7)
Log Medium and Low HIV refugees $_{t-1}$	5.0E-7	3.6E-7	2.9E-7	2.0E-7	2.1E-7
	(1.4E-7)	(1.7E-7)	(1.5E-7)	(9.5E-8)	(1.1E-7)
Log GDP per capita $_{t-1}$	-0.061	-0.084	-0.069	-0.058	-0.056
	(5.1E-2)	(7.0E-2)	(0.040)	(0.087)	(0.090)
Log Population density	0.182	0.250 **	0.302 *	-0.329 **	-0.331 *
	(0.136)	(0.085)	(0.143)	(0.122)	(0.126)
Economic openness $_{t-1}$		1.3E-4	4.2E-5 *	-5.1E-5	-4.5E-5
		(2.3E-4)	(2.6E-4)	(2.4E-4)	(2.2E-4)
Health expenditures		-0.197	-0.133	-0.252	-0.257
		(0.249)	(0.364)	(0.240)	(0.241)
Democracy $_{t-1}$		5.1E-4 *	5.6E-4 **	8.1E-4 **	8.1E-4 **
		(2.6E-4)	(9.3E-4)	(3.3E-4)	(1.0E-3)
West Africa		-0.334 *	-0.904 *	-0.407	-0.413
		(0.535)	(0.971)	(1.029)	(1.018)
East Africa		-0.365* *	-0.116 *	-0.110	-0.148
		(0.258)	(0.410)	(0.466)	(0.455)
Central Africa		-0.884 *	-2.052 *	-1.619 *	-1.638 *
		(0.474)	(1.160)	(0.996)	(0.978)
Domestic Conflict $_{t-1}$				-0.004	-0.014
				(0.017)	(0.026)
Major Conflict $_{t-1}$				-0.023 *	
				(0.014)	
Minor Conflict $_{t-1}$				-0.019	
				(2.8E-2)	
Log of Conflict Scope $_{t-1}$					-3.5E-3
					(3.3E-3)
Log of Peace keeping operation $_{t-1}$				0.039	0.040
				(7.3E-2)	(0.072)
Interstate Conflict $_{t-1}$				0.032	0.028 *
				(0.022)	(0.014)
Muslim religion				-1.185 **	-1.159 **
				(0.564)	(0.547)
Male circumcision			-0.154 **		
			(0.008)		
Log force ratio $_{t-1}$				-0.047	-0.047
				(0.029)	(0.030)
Log Distance from the DRC				-2.5E-5 *	-2.4E-5 *
				(1.4E-5)	(1.4E-5)
No of Instruments	124	127	127	133	132
Arello-Bond test (AR1)	0.0418	0.0483	0.0469	0.0462	0.0478
Arello-Bond test (AR2)	0.0742	0.0891	0.0889	0.0923	0.0871
Standard errors	robust	robust	robust	robust	robust
Wald test	1399.54	2355.16	2159.14	4310.87	2976.21
No of observations	490	475	475	475	475

Source: Own regression analysis

The same cannot be said of the relationship between interstate conflict and HIV prevalence estimates. This relationship is, as is the case with the fixed effects and random effects models a positive one. Further, regression 4.4 shows that major conflicts have a negative and statistically significant relationship with HIV prevalence estimates. The same is observed for the relationship between minor conflicts and HIV prevalence estimates although it is not statistically significant. As was the case with Davenport and Loyle, regression 4.5 shows that the scope variable has a negative and statistically insignificant relationship with estimated HIV prevalence. As is observed for all other models presented in this chapter and in contrast to the findings of Davenport and Loyle, regressions 4.4 and 4.5 show a positive, albeit statistically insignificant link between hosting peace keeping forces and increases in HIV prevalence.

7.6 Discussion and Conclusion

Chapters 2 to 6 highlight that HIV prevalence estimates are not ideal candidates for use in regression analyses where they are the dependent variable. Problems with HIV data collection and estimation are detailed in Chapters 2 and 3 using Zimbabwe as a case study while Chapters 3 to 4 discuss how these estimates are shaped by the EPP/Spectrum modelling architecture and assumptions made about behaviour, migration, etc. The models proposed in the previous chapter and tested in this chapter produce many different results but nevertheless suggest that some findings are more consistent across the models. This in turn suggests that analysis of the relationship between armed conflict and HIV prevalence might be a useful starting point or base for more contextual micro analysis. However, careful attention must be given to both the modelling decisions made and the interpretation of the results.

Three questions are at the core of any regression analysis. The first concerns the choice of model type or specification. Firstly, OLS models used by Iqbal and Zorn (2010) and Davenport and Loyle (2009) as well as those presented in this chapter have the fundamental flaw of ignoring the fact that HIV prevalence will have its own momentum (determined by other factors not included in the model). This is a weakness of the fixed effects and effects models that however allows for a focus on either within country variation fixed effects and primarily between country variation random effects in the relationship between HIV and conflict.

Dynamic model specifications allow for the lag of the dependent variable (HIV prevalence estimates) to be included as an independent variable. This allows for the models to account for the dynamic nature of the epidemic. However, even after accounting for dynamics, the models presented are fundamentally linear models. Given that the HIV estimates are obtained from curvilinear approximations (as shown in Chapter 2), they might be inadequately capturing the link between HIV and conflict. The models assume conflict and HIV have a linear relationship yet the HIV epidemics typically follow a curved path.

The second question at the heart of any regression analysis involving estimated HIV prevalence data concerns the choice of sample. Despite the fact that none of the specifications are perfect, the overarching theme they highlight is that the association between HIV prevalence and factors such as the level of militarization of a country and conflict (especially domestic conflict) may be negative. Previous studies that carried out an econometric analysis of the link between HIV and conflict did not reveal which countries were in their sample. This makes the replication of their results impossible.

The sample used in this chapter is restricted only to sub-Saharan Africa (which is the region hardest hit by HIV in the world) countries with generalised epidemics and this avoids the results being confounded by other countries that do not have HIV prevalence rates high enough to allow for an analysis of the link between conflict and HIV. The major and minor conflict variables were consistently found to have a negative and statistically significant association with estimated HIV prevalence. This is in tune with our dataset's summary statistics that suggest this result is driven by countries such as Angola, Liberia, Somalia, and the Democratic Republic of the Congo. This shows that the regression results in this chapter are probably influenced (and hence are different from Iqbal and Zorn (2010) and Davenport and Loyle (2009)) by the sample of countries included.

The third important question to answer when carrying out regression analysis involving estimated HIV prevalence rates is whether the control variables used are appropriate. The dynamic panel models find a very small but statistically significant and positive relationship between estimated HIV prevalence rates and the inflow of refugees from high prevalence countries. The models also confirm that countries with relatively high proportions of Muslims or a higher proportion of circumcised males are associated with lower prevalence rates even when controlling for factors such as conflict, economic openness etc. They also suggest that much of the variation in estimated HIV prevalence levels between Southern, East and West African countries is explained away by the conflict variables.

The aim of the next chapter is to try and explore how the individual cases of countries such as Somalia, DRC and others drive the result observed here. The controls contained in the regressions presented here are probably not capturing other important factors. Factors such as the level of transport and communication infrastructure were assumed to be implicitly captured by variables such as GDP per capita or economic openness. However, this assumption may be inaccurate. Although data constraints prevent us from being able put variables measuring the level of transport infrastructure or changes in sexual behaviour in cross country regressions, looking at the specific circumstances facing the high conflict countries, together with the little available data on these factors (and many others) is a useful exercise to supplement and expand the results from cross country regression analysis.

Annex B Random Effects model

As discussed in Chapter 6, the use of random effects models is often advisable only in instances where the model specification is likely to have some material controls that it does not account for. This could be because the data is not available (e.g. country specific factors like the level of communication infrastructure in conflict regions) or because its quality is questionable (e.g. cultural differences in sexual practices).

The number of controls matters less for the fixed effects where we assume away time invariant factors but more important for the random effects where we would need to have a less parsimonious model (have independent variables that control for most factors affecting HIV prevalence estimates). Random effects models unlike their fixed effects counterparts allow us to focus on time invariant variables and so are appealing where between country variability is important. Random effects might be insightful as our descriptive analysis in Section 7.2 showed that the data has both within and within country variation[75].

The Hausman specification test results shown in Table 7.9 make the decision on which specification is appropriate easier to make. As discussed in Chapter 6, the Hausman test compares the fixed effect and random effect models under the null hypothesis (H_o) that the individual effects are not correlated with the other regressors (Haussmann, 1978). If H_o is rejected, then the individual effects are correlated with the other regressors in the model and a fixed effects model is preferred. The results of the Hausman tests shown in Table 7.9 show that the null hypothesis is rejected. This means the fixed effects models are preferable and it is probably advisable for one to look at the fixed effects model results more instructively than one would the random effects.

[75] There is an even spread between HIV prevalence estimates, GDP, population density, domestic conflict and the level of militarization which show greater variation within countries and the interstate, minor and major conflict and conflict scope variables that show more between country variation.

Table 7.12 Random effects model

	Model				
Variable	**B.1**	**B.2**	**B.3**	**B.4**	**B.5**
Log of High HIV refugees $_{t-1}$	-1.3E-7	1.4E-7	1.4E-7	3.0E-8	8.5E-8
	(2.7E-7)	(2.6E-7)	(1.1E-7)	(2.7E-7)	(2.7E-7)
Log of Medium and Low HIV refugees $_{t-1}$	-3.4E-7	-2.2E-7	-2.4E-7	3.1E-6	-2.6E-7
	(2.3E-7)	(2.2E-7)	(1.6E-7)	(2.2E-7)	(2.3E-7)
Log GDP per capita $_{t-1}$	0.301 ***	-0.091	-0.087	-0.074	-0.057
	(0.094)	(0.119)	(0.111)	(0.108)	(0.104)
Log of Population density	1.012 ***	1.474 ***	1.509 ***	1.144 ***	1.082 ***
	(0.127)	(0.147)	(0.174)	(0.141)	(0.136)
Economic openness $_{t-1}$		8.9E-4 *	8.6E-4 *	8.6E-4 *	8.1E-4 *
		(4.7E-4)	(2.4E-4)	(4.7E-4)	(4.8E-4)
Health expenditures		-0.158	-0.201	-0.273	-0.261
		(0.468)	(0.413)	(0.448)	(0.438)
Democracy $_{t-1}$		3.0E-3 ***	2.7E-3 ***	2.9E-3 ***	3.0E-3 ***
		(9.7E-4)	(9.3E-4)	(1.0E-3)	(1.0E-3)
West Africa		-4.235 ***	-4.292 ***	-4.156 ***	-3.873 ***
		(0.820)	(0.830)	(0.791)	(0.750)
East Africa		-3.989 ***	-4.031 ***	-3.325 ***	-3.142 ***
		(0.667)	(0.724)	(0.628)	(0.597)
Central Africa		-1.001	-1.056	-1.453 **	-1.447 **
		(0.736)	(0.714)	(0.647)	(0.614)
Domestic Conflict $_{t-1}$			0.007	-0.001	-0.027
			(0.074)	(0.099)	(0.086)
Major Conflict $_{t-1}$			-0.209 *	-0.215 **	
			(0.093)	(0.113)	
Minor Conflict $_{t-1}$			-0.026	-0.034	
			(0.078)	(0.102)	
Log of Conflict Scope $_{t-1}$			-0.003	-0.009	-0.027
			(0.020)	(0.032)	(0.028)
Log of Peace keeping operation $_{t-1}$			0.023	0.016	-0.002
			(0.054)	(0.067)	(0.064)
Interstate Conflict			0.226	0.243	0.195
			(0.079)	(0.156)	(0.152)
Muslim religion	-0.975 **			0.448	0.297
	(0.470)			(0.510)	(0.482)
Log of force ratio $_{t-1}$				-0.109 *	
				(0.063)	
Log of Distance from the DRC	-1.5E-4 ***			-1.0E-4 ***	-1.0E-4 ***
	(3.3E-5)			(3.2E-5)	(3.0E-5)
R^2					
Between	0.2016	0.1595	0.1531	0.3681	0.3885
Within	0.2698	0.3241	0.3316	0.3067	0.2933
Pesaran's p value	0	0	0	0	0
Standard errors	robust	robust	robust	robust	Robust
Wald test	98.94	138.76	149.48	148.54	137.39
Hausman (P- value)	0	0	0	0	0
No of observations	490	475	475	475	475

Source: Own regression analysis

Notwithstanding, it may be of interest to discuss some of the differences in the results produced by the fixed effects and the random effects models. Indeed the only differences lie in the inclusion of time invariant variables in the random effects model. The results of the random effects regressions are therefore presented in Table 7.12. As was the case for Specifications 1 and 2, the regional dummy variables all have a negative relationship with estimated HIV prevalence. The possible explanations for this result are probably similar to those put forward to explain the results observed for Specification 1. As was the case with the other regressions (Specifications 1 and 2), the distance from the DRC variable has a negative relationship with estimated HIV prevalence.

The random effects specification shows that major and minor conflict variables both have negative associations with estimated HIV prevalence rates. As is the case with the fixed effects models, only the major conflict variables are statistically significant. Similarly, Table 7.12 shows that the domestic conflict variable has a statistically insignificant negative relationship with the estimated HIV prevalence rates. However, as is the case with the fixed effects models, regressions B.3 to B.5 show this to be positive and statistically insignificant.

The RE models also show a positive and statistically significant relationship between HIV prevalence estimates population density. This result is similar to that observed for the FE models. With respect to the measure of economic openness, the random effects models (as do the fixed effects models) show a positive and statistically significant relationship between estimated HIV prevalence and economic openness. As is the case with Specification 3, the FE models as well as Davenport and Loyle models, the random effects models show a positive statistically insignificant relationship between health expenditure and estimates of HIV prevalence.

The same is true of the relationship between the democracy variable and the estimated HIV prevalence which is both positive and statistically insignificant. Regression B.4 shows a negative association between HIV prevalence estimates and the level of militarization. Unlike in the fixed effects model, the random effects show that this relationship is statistically significant.

Regressions B.3 and B.4 show a positive, and statistically insignificant link between hosting peace keeping forces and increases in HIV prevalence. The result is in synch with that found for similar fixed effects models. However, regression 4.5 shows a negative relationship between the presence of peace keeping forces and estimated HIV prevalence. This is probably because regression B.5 does not control for changes in the force ratio controlled for by regression B.4. Resultantly, the peace keeping operation variable might be capturing overall increases in troop activity and not just that by foreign peace keeping interveners.

Just as is the case with fixed effects models, random effects models like the ones presented in Table 7.12 ignore the fact that the HIV epidemic is dynamic. This and the fact that they assume the relationship between the independent variables and HIV prevalence estimates is linear makes these regression models far from ideal. Many of the inconsistencies or puzzles we observe in Table 7.12 may be as a result of these issues.

8 UNDERSTANDING THE HIV-ARMED CONFLICT NEXUS: A LOOK AT SEVEN COUNTRY CASES

Abstract

This chapter advances the findings from the regression models presented in the previous chapter & by looking at the seven countries (Angola, Rwanda, Somalia, Liberia, DRC, Sudan and Ethiopia) which drive the cross-country correlation between armed conflict and HIV prevalence in Africa. The brief case histories suggest that the link between HIV prevalence and conflict is far more nuanced than suggested by econometric research on the topic. Following a framework first proposed by McInnes (2009), the case studies suggest that each conflict is unique in its geographical scope and effect on sexual behaviour and dislocation and that each country has its own specific pre conflict conditions. These 'background factors' or conditions such as poor transport and communication infrastructure as well as population density probably shape the effect the conflict may have on the epidemic as they determine the level of human mobility and mixing during the conflict. But these are particularly difficult to measure especially during conditions of armed conflict.

Introduction

Chapter 7 showed that modelling the link between HIV prevalence and armed conflict at a global level is confounded by the fact that most conflict countries are in Africa where HIV prevalence is high. Restricting the sample to 34 sub-Saharan countries with generalised epidemics is more meaningful and reveals that there is a statistically significant negative relationship between HIV prevalence estimates and armed conflict. Further, these results are driven primarily by a number of conflict countries including Sudan, Angola, Liberia, Somalia, and the DRC (see Table 7.2). All these had generalised epidemics and at least one minor or major conflict in the years between 1990 and 2005. As these countries are primarily responsible for the negative correlation between HIV prevalence and conflict, it makes sense to explore their particular case histories in order to understand why there is an overall negative relationship between HIV and conflict in Africa.

The seven country cases are presented in a way that allows for gaps between the conceptual framework and the results presented in Chapter 7. Regression analysis can never account for the many possible factors that influence the progression of the epidemic in different countries. At most, it can (within the necessarily limited realm of linear regression methods) provide broad insights into the HIV-conflict nexus. In this chapter I explore some of the country-level factors that may explain why the regression results in Chapter 7 reveal a negative association between HIV prevalence estimates and armed conflict.

In so doing I estimate HIV prevalence and incidence for each country (using EPP and Spectrum) and then locate the trend over time in the context of the specific history of conflict in each country. Graphs showing the link between HIV prevalence and incidence estimates and average rural and urban ANC site data are presented in Addendum C. However, because of the disparity between the ANC data and the more smoothed EPP and Spectrum estimates, the graphs are difficult to read and so only graphs of the estimates are presented in the main text (to allow a discussion of these estimates). I construct a narrative which draws on McInnes (2009) and suggests that the epidemics in each country were influenced both by background (structural) factors as well as changes that occurred during the conflict. Whereas some of these background factors such as population density, the level of the epidemic when conflict starts are relatively easy to capture in regressions, others such as the level of development of transport, communication and health infrastructure are difficult to capture especially in a panel analysis requiring time series data. Some, such as the scale, duration and nature of conflict are somewhere in between as data on the number of battle related deaths and duration of conflict are accessible although not ideal measures.

Capturing the changes that occur in conflicts is even harder and just as is it is difficult to find data on the level of development of transport, communication and health infrastructure before conflict starts, it is even harder to find this data for the years when the counties are unstable. Furthermore, there are no meaningful ways to measure the changes in sexual behaviour or amount of sexual mixing between individuals be they armed personnel, displaced individuals or refugee host populations.

All of these shortcomings or data inadequacies mean that the regressions in Chapter 7 only tell part of the story. Given the issues that come with using HIV prevalence estimates for cross country regression analysis, the fact that the control variables themselves are difficult to measure further bolsters the need for caution when interpreting regression analyses such as those presented in Chapter 7. Furthermore, the fact that seven countries drive the negative correlation between conflict and HIV prevalence (see Table 7.2) points to the role that case specific and inherently immeasurable factors play in shaping the overall result. In these country sketches presented below, I draw on sources such as the CIA Country Fact Book,[76] and other secondary literature. The intention is not to provide a comprehensive history, but rather to provide a succinct sketch of the different dynamics pertaining to conflict in these countries and to place these within the context of what we know about behaviour, infrastructure etc. which have a bearing on the path of the HIV epidemic. In sketching the relevant background factors, I follow the framework provided by McInnes (2009).

8.1.1 McInnes Framework

The central argument made by McInnes is that despite the presence of risk factors (such as those discussed in Chapter 5), it is not always the case that conflict acts a vector for HIV (McInnes, 2009:1). McInnes reaches this conclusion by developing a framework which

[76] See CIA FACTBOOK https://www.cia.gov/library/publications/the-world-factbook/

distinguishes between susceptibility and vulnerability which explains the circumstances where HIV might and might not spread given these risk factors. In order to understand the framework, one must first understand McInnes' description of what these "risk factors" are as well as what the terms "susceptibility" and "vulnerability" mean in the context of the framework.

8.1.1.1 The risk factors

The first risk factor mentioned by McInnes is regarding HIV and the military (McInnes, 2009:3). Chapter 5 already covered the different arguments often brought up on this debate. These range from the contested argument that militaries have higher HIV prevalence rates than comparable civilian groups to the unproven notions that those in militaries engage in particularly riskier sexual practices (more than civilians).

The second risk factor mentioned by McInnes is the dislocation and migration that result from conflict. Much of the arguments on this risk factor were also covered in Chapters 5 to 7. In short, McInnes notes that refugee camps are particularly prone to the spread of disease, including HIV. This is because they lack the support and capacity to fight disease as basic needs such as sanitation are often in a poor state. He argues that women and children are also vulnerable in refugee areas and may be forced to engage in transactional sex (or are subject to sexual violence). A third risk factor, often connected to the second is that conflict may cause changes in sexual behaviour as argued in Chapter 5. McInnes draws on examples that also feature in the review of the literature in Chapter 5 including the use of rape as a weapon of war.

McInnes argues that the destruction of health systems that conflict often leads to is another risk factor. Conflict may affect everything from the delivery of drugs, destruction of infrastructure, interruption of prevention and treatment of HIV and often times health systems switch from primary care to secondary and tertiary care. Finally, as was mentioned in Chapter 6, and argued by Davenport and Loyle (2009) the presence of peace keepers may be another risk factor. This is particularly so when peacekeepers come from high prevalence countries and serve in missions in lower prevalence countries[77].

McInnes acknowledges that although there has been much discussion on the risk factors mentioned thus far, the complexity of the HIV-conflict nexus has resulted in there being little explanation as to why even in the presence of these risk factors HIV prevalence rates do not always increase (McInnes, 2009:11). The previous chapter (Chapter 7) of this thesis highlighted that not only has conflict not increased in some cases but regression analysis suggests that domestic conflict may be linked with a decrease in HIV prevalence although country-level regression analysis is far from an ideal way of exploring the relationship. Chapter 7 also concludes, as does McInnes that a more nuanced explanation of the relationship between conflict and the spread of HIV is required (McInnes, 2009:11).

The cornerstone of the McInnes framework is an appreciation of the fact that it is not conflict but specific types of changes which might occur because of conflict which lead to increased HIV prevalence (McInnes, 2009:11). He argues that pre-existing background and related contextual factors are important in shaping “vulnerability” and “susceptibility”.

8.1.1.2 Background factors (Susceptibility)

There are a number of background factors which may make a country “susceptible” to increased prevalence during a conflict but these do not in themselves automatically result in an increase in prevalence. These background factors merely determine how susceptible to increases in HIV prevalence a country is (McInnes, 2009:11). However, there are changes introduced by conflict that will supposedly make a country “vulnerable” to increased prevalence. For conflict to be a significant risk factor, a country has to be both susceptible and vulnerable (McInnes, 2009:11).

McInnes mentions five background factors that determine susceptibility. The first of these is the level of HIV prevalence when the conflict starts (McInnes, 2009:12). His suggestion is

[77] Despite our regressions not distinguishing between peace keepers from high prevalence countries from those from low prevalence countries, the dynamic regression models presented in Chapter 7 show a positive albeit statistically insignificant association between HIV prevalence and the number of peace keeping troops.

that if HIV prevalence levels are low, then conflict is unlikely to lead to an upsurge in HIV prevalence as there are too few infected people for this to happen. He also argues that the stage (where it is peaking an epidemic is at is important since the virus is more easily spread at certain times such as the period immediately after infection when the viral load is high. McInnes however admits that it is hard to say the extent to which this is true at community level.

Another background factor highlighted by McInnes is the state of transport and communication infrastructure (2009:12). These determine human mobility, which is an important factor in the spread of HIV (McInnes, 2009:12). If transport and communication infrastructure is poor, then remote communities will be isolated and may actually be protected from the HIV epidemic. On the other hand, good communication and transport infrastructure allows for easier movement of people (fleeing the conflict or the troops engaged in the fighting) during the conflict which might spread the disease.

Population density is seen as another important factor. According to McInnes, HIV is unlikely to spread slower if conflict is conducted in a remote, rural area with low population density (McInnes 2009:12). Conversely, if the conflict is in a high population density area, the chances for increased interaction between infected and uninfected individuals increase. This positive relationship between population density and HIV prevalence rate (estimates) was supported by the fixed effect and system GMM dynamic models presented in Chapter 7.

A very intuitive determinant of susceptibility (i.e. the fourth background factor) is the scale, duration and nature of conflict. McInnes (2009:12) argues that lower intensity conflicts (fewer individuals involved), that cover a smaller geographical scale may lack the effective mass to generate the spread of the disease. However, the regression results presented in the previous chapter show mixed results. The measures of conflict magnitude which are based on battle related deaths consistently show a negative and statistically significant association with estimated HIV prevalence rates. The fixed effect models and the dynamic models also show a negative link between the geographical scope of a conflict and estimated HIV prevalence although this is not statistically significant.

McInnes also argues that the nature of the conflict, in terms of the level of antipathy between communities also determines the progression of the HIV epidemic. The point here is that conflicts of identity within states are likely to generate higher levels of antipathy which could make acts of sexual violence more likely McInnes (2009:12). This is in contrast with wars of acquisition between states. Prolonged conflict may lead to further destruction of infrastructure, increase the isolation of certain communities and so insulate them from the epidemic.

Finally, McInnes argues that poverty is an important background factor which determines the extent to which conflict affects the HIV epidemic. The regression analysis in Chapter 7 showed that not only is this a weak factor, but the relationship between poverty and HIV prevalence is not best analysed using measures of the standard of living such as per capita GDP. The linkages are far more nuanced. McInnes also acknowledges that poverty is probably the weakest risk factor in his analysis although he suggests that poverty may force vulnerable women into transactional sex (McInnes, 2009).

8.1.1.3 Changes during conflict (Vulnerability)

As already mentioned, McInnes argues that in addition to these background factors, changes brought about by conflict determine the impact that conflict will have on HIV prevalence. One such change is disassortive mixing[78]. War results in mobile groups mixing in new ways. This can be through movements of refugees and troops from one area to another where new sexual networks may be created. As alluded to in Chapters 4 and 5, the nature of such sexual contact will vary from commercial, consensual, casual, transactional or more violent forms such as rape. McInnes (2009:13) suggests that what is crucial for the spread of HIV is not so much the movement of people but the degree to which they mix with new communities. This rational is obviously dependant on whether or not the population groups mixing actually have different prevalence rates.

[78] In networking, mixing can be classified broadly as assortative or disassortive. *Assortative mixing* is the tendency for nodes to connect to like nodes, while *disassortive mixing* captures the opposite case in which very different nodes are connected.

Another consideration is the change in sexual behaviour that might occur as a result of conflict. McInnes (2009:13) proposes that these may range from casual sexual encounters, infidelity, sexual predation and increases in the numbers of partners often as a consequence of stress, changing norms over acceptable sexual behaviour, peer group pressure, hyper masculinity etc. There may also be increased instances of rape and sexual violence perpetrated on women and children.

McInnes argues that during conflicts, there may be an increase in depression and trauma which might affect libido particularly in post conflict situations. McInnes also mentions that the depression and trauma might also lead to increased drug abuse. This may be in the form of alcoholism or addition to narcotics. McInnes also mentions increased injecting drug abuse as another factor that greatly increases the risk of infection. However this is unlikely to be a major factor in Sub-Saharan Africa and so this is unlikely to lead to significant upsurges in prevalence rates. Further, McInnes states that conflicts in Sub-Saharan Africa have been characterised by low incidence of injectable drug use (McInnes, 2009:14).

A more fundamental change that may occur during conflict is the destruction of health services which would compromise the effectiveness of HIV interventions. McInnes (2009:13) singles out changes in health support, including education as well as the availability of diagnostic kits, supply (and use) of condoms and the care and treatment of people living with HIV as being crucial. If these health and ancillary services are interrupted by conflict, the trajectory of the epidemic will be affected. A shift in resource allocation might also occur with resources being diverted within the health services from primary to secondary and tertiary care. Increases in AIDS mortality might reduce the number of infected people in the population while on the other hand, the unavailability of condoms combined with a reduction in resources put to AIDS education and awareness campaigns might all act to increase the number of new infections.

Table 8.1 provides a summary of the core ideas espoused by McInnes (2009). To test his framework, McInnes uses four country cases namely; Sierra Leone, Angola, Rwanda and the DRC. He argues that in these countries, prevalence did not rise during the conflicts despite the presence of traditional risk factors. Of these countries, Angola, Rwanda and the DRC were identified in Chapters 7 as having influenced the result found in the regression analysis suggesting that there may be a negative link between HIV prevalence and conflict. The discussion of the country cases presented here will make reference to the minor, major, domestic and interstate conflict variables used in the econometric analysis presented in Chapter 7.

Table 8.1 Some of the risk, susceptibility and vulnerability factors discussed by McInnes

Traditional risk factors
During conflict
HIV and the military
Human mobility, migration, dislocation
Sexual violence against women
Destruction of health services and reduction of a focus on primary health care
Post conflict
Reintegration of previously isolated communities
Presence of peace keepers
Background factors (Susceptibility)
Current level of HIV prevalence when conflict begins
Population density
Transport and communication
Scale and nature of conflict
Poverty
Changes during conflict (Vulnerability)
Disassortive mixing
Changes in sexual behaviour
Changes in health support

Source: McInnes (2009)

Other countries include Liberia, Sudan, Ethiopia and Somalia. I will proceed to use the McInnes framework to investigate possible explanations for the regression results observed in Chapter 7. Inevitably some of my analysis will coincide with that by McInnes particularly regarding Angola, Rwanda and the DRC although my approach also includes the modelling of HIV prevalence and incidence estimates using EPP and Spectrum for the years 1990 to 2009, as this is the period for which the most recent input data are available for all country cases. However, since the regression analysis took estimates up to 2005, the discussion will not discuss estimates beyond the year 2005. These estimates are then used to try and map changes in the epidemic to developments in each country's conflict environment. Another important difference between my approach and McInnes is that I allow for the possibility that conflict related disruption may not only have not rendered countries vulnerable and susceptible, but may actually have insulated or protected them to some extent from an HIV epidemic.

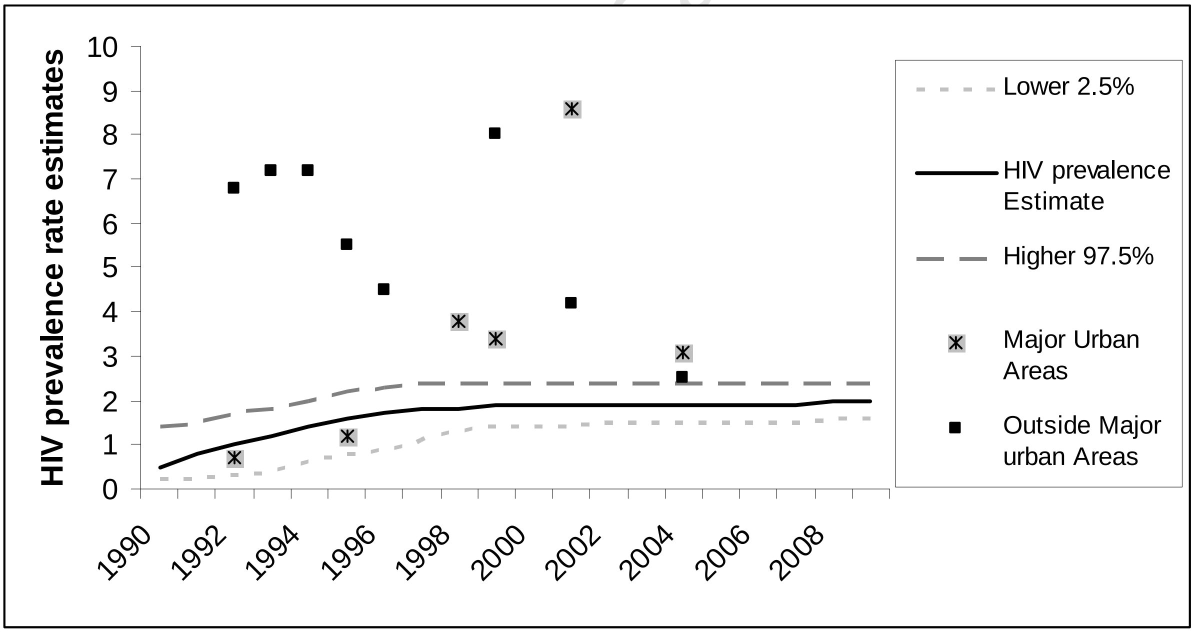
8.2 Angola

The first case of HIV in Angola was diagnosed in 1985 (UNAIDS, 2005). HIV prevalence was reported to be at 2.5% in 2005, making it the lowest rate of HIV prevalence in continental southern Africa (USAID, 2008:1; see also own estimates in Figure 8.1). As of December 2005, UNAIDS estimates put the number of people living with HIV at 320 000 (USAID, 2008:1). However, the National Institute against HIV/AIDS estimates that 400 950 people were living with AIDS in 2006 (USAID, 2008:1). The modelling done in this section focused more on producing HIV incidence and prevalence estimates and less on other estimates such as the number of people in need of ART treatment or the number of AIDS deaths.

In modelling the HIV prevalence and incidence estimates, a number of assumptions had to be made. As was described in Chapter 2, EPP is used to produce HIV prevalence estimates which are then used in Spectrum to produce the final HIV prevalence and incidence estimates with confidence intervals. In EPP, the epidemic in Angola was modelled as a generalised epidemic. The ANC data was also down scaled by a factor of 0.8. This is an assumption recommended for epidemics where there is no HIV data based on national population based surveys, HIV prevalence data from antenatal clinic attendees should be adjusted downwards by this factor (UNAIDS Reference Group on Estimates, Modelling and Projections, 2006; UNAIDS, 2007:10). This recommendation is based on a UNAIDS report that found that in countries with generalised epidemics, in both urban and rural areas, HIV prevalence among adults in population-based surveys is approximately 80% of the prevalence among antenatal clinic attendees (UNAIDS, 2007). The obvious result of this assumption is that ANC data are generally much higher that the HIV prevalence trends shown in Figure 8.1. In this chapter and following UNAIDS, this assumption is also applied to the DRC, Somalia and Sudan where no national population-based survey data on HIV is available (UNAIDS, 2007:10).

In Spectrum one of the important assumptions made is that the Coale-Demeny North life table was used as the model life table. Spectrum default assumptions on the total fertility rate reduction and life expectancy at birth. As per the UNAIDS recommendation, it was assumed that in the absence of antiretroviral treatment, people living with HIV will become eligible for treatment an average of 3 years before they are expected to die from an AIDS related cause (UNAIDS Reference Group on Estimates, Modelling and Projections, 2006). It was also assumed that the net survival of people living with HIV is 11 years (UNAIDS Reference Group on Estimates, Modelling and Projections, 2006). Lastly the 2008 UNAIDS Angola epidemiological fact sheet was the main source of the data on ART rollout, PMTCT and child treatment services were used.

Figure 8.1 HIV prevalence in Angola (1990-2009)



Source: Own EPP and Spectrum modelling.

Figure 8.1 shows that HIV prevalence estimates rose steadily throughout the 1990s. However, in the 2000s, HIV prevalence remained fairly constant. Note the wide error margins around this estimate. This indicates that the underlying ANC data is patchy (see Figure 8.1) and that the ‘true’ adult HIV prevalence rate could be more than double or half the estimated rate (the upper 97.5% estimate). Similarly wide error margins are common in Africa, and are reflected also in the other country cases discussed in this chapter.

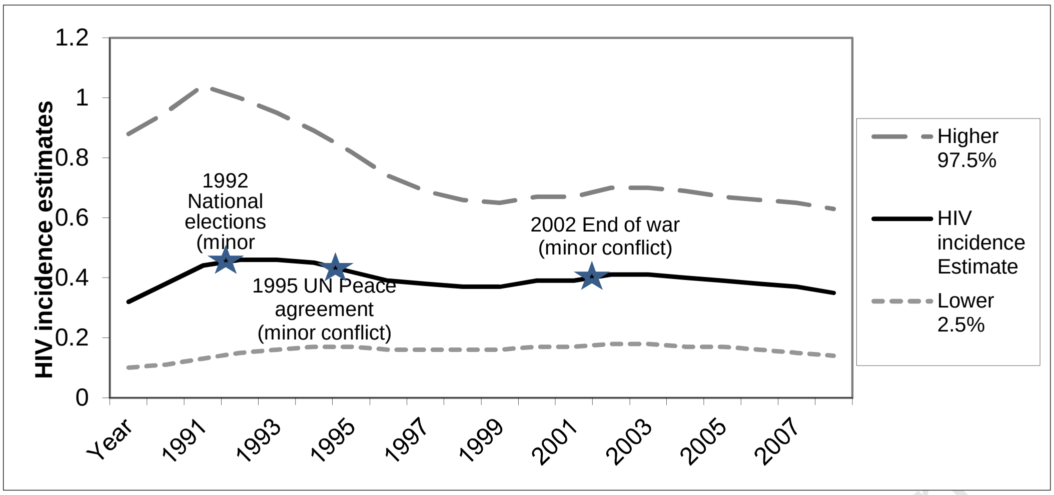
8.2.2 The civil war in Angola

Angola is still in the process of rebuilding its country after the end of a 27-year civil war in 2002. Fighting between the Popular Movement for the Liberation of Angola (MPLA), led by Jose Eduardo Dos Santos, and the National Union for the Total Independence of Angola (UNITA), led by Jonas Savimbi followed independence from Portugal in 1975. As early as 1961, the nationalist MPLA spear-headed the “First War of National Liberation”. Other nationalist groups then joined the anti-colonial struggle leading to the attainment of independence in 1975. However, the MPLA soon monopolized power and civil war erupted between the then “Marxist” MPLA government, backed by Cuba and the Soviet Union and the South Africa and U.S supported UNITA. (Agadjanian, 2001:2)

There was hope of peace in 1992 (see Figure 8.2) when Angola held national elections, but fighting picked up again by 1996. Up to 1.5 million lives may have been lost - and 4 million people displaced - in the quarter century of fighting. Savimbi's death in 2002 ended UNITA's insurgency and strengthened the MPLA's hold on power (CIA fact book, 2010). In the period from 1990 to 2005, Angola had a major conflict (more than 1000 battle related deaths) in each year except for 1995, 1998 and 2004 which had minor conflicts (therefore only minor conflicts are marked in Figure 8.2). These minor conflicts have primarily been internal[79] contestations over territory (UCDP, 2010). The country has enjoyed relative peace and since 2002.

[79] While UNITA controlled areas were mainly rural, MPLA held many of the major towns for a long while.

Figure 8.2 HIV incidence in Angola (1990-2005)



Source: Own EPP and Spectrum modelling.

The literature discussed in Chapter 5 on the HIV-conflict nexus focuses on potential links between risky sexual behaviour and armed conflict. The relevant outcome variable is thus ideally HIV incidence (new HIV infections) rather than HIV prevalence. Figure 8.2 shows the link between HIV incidence estimates and key events in the evolution of Angola's political situation. The HIV incidence estimates were generated from the EPP/Spectrum modelling discussed earlier. Because the data are so poor, and incidence estimates subject to big margins of error, Figure 8.2 should be interpreted with caution. It also means that it is very hard to discern the link between the minor or major conflict years and the trend in estimated incidence. However, Figure 8.2 seems to suggest around the time of the 1992 elections, estimated incidence was on the rise but only declined between 1993 and 1996 when there were major conflicts. Further, estimated incidence rates continued on a downward trend from 1996 up to around 1999 a time when each year in that period was a major conflict year.

8.2.3 Traditional risk factors

Despite the long and protracted war in Angola, there is little evidence that Angola was particularly at risk of an explosive epidemic (McInnes, 2009). In terms of the HIV and the military, there was little testing done during the conflict. However, in the post conflict period there was little evidence to suggest that HIV prevalence was higher among armed personnel than in comparable civilian populations. For instance, in 2003, the 3.9% prevalence rates found amongst urban based military personnel was identical to prevalence in the general population (UNAIDS, 2008). There is also little evidence to suggest that dislocation and migration were main features of the conflict. However, UNDP gave estimates, which it conceded to be uncertain, that in 2006, there were still about 62 000 internally displaced persons (IDPs) (UNDP, 2010)[80]. Reports of sexual violence against woman in the conflict in Angola are scarce (McInnes, 2009). Further, aside from Cuban and South African involvement in the conflict in the 1980s, Namibia's involvement in 1999, the conflict was predominantly a domestic factor and there is little evidence to link these interventions to the HIV epidemic in the country.

8.2.4 Background factors

The conflict in Angola predated the emergence of the HIV epidemic and this may have restricted its spread across the country. In terms of transport and communication infrastructure, Angola has always struggled with poor transport and communication infrastructure. This is evidenced by the fact that the percentage of paved roads was virtually zero for the 1980s and 1990s (World Bank Databank, 2008). [81]Although there is little evidence to support this, it is conceivable that the poor infrastructure is one of the reasons Angola was protected from an epidemic as explosive as that in Botswana or other Southern African countries.[82]

Population density has always been very low in Angola with estimates being put at 5 people per square kilometre in 1975, 11 in 2000 and 13 in 2005 (UN, 2006)[83]. The low population density coupled with the difficulty of cross country movement would have made it difficult for the epidemic to spread. However, it is worth mentioning that the regression analysis in Chapter 7 was inconclusive as to the nature of the relationship between HIV prevalence (estimates) and conflict.

[80] Data mined from http://hdrstats.undp.org/buildtables/rc_report.cfm
[81] Data from World Bank http://ddp-ext.worldbank.org/ext/DDPQQ
[82] The regressions presented in the previous chapter did not investigate this factor due to the lack of reliable time series data.
[83] http://esa.un.org/unpp/p2k0data.asp.

The conflict in Angola was protracted and widespread with the MPLA controlling most of the major towns while UNITA dominated in the rural regions for long periods (UCDP, 2008). However, even the regions not directly affected by war were impacted mainly through the overall decay of their economic and social infrastructure and general impoverishment (Agadjanian, 2001:9).

As already noted, the prolonged conflict may have led to further destruction of infrastructure, increased the isolation of certain communities and so insulated them to some extent from the epidemic. Regarding poverty as a background factor, the evidence is also limited. Although Angola was one of the poorest countries in the world during its conflict period, there are scarcely any reports or evidence of this having caused an increase in transactional sex or any other poverty-linked risky behaviour.

All of the background factors (except for poverty) suggest that Angola was unlikely to realise an upsurge of HIV prevalence during the conflict period. Indeed, it is likely that war protected Angola to some extent from such an increase.

8.2.5 Changes during conflict

During the 1975-2002 period, the civil war made cross country travel nearly impossible (USAID, 2008:1). Owing to the poor infrastructure it is plausible that the degree of mixing and creation of new sexual networks as refugees and troops moved from one area to another was limited. The conflict in Angola is seen as having limited human mobility rather than causing large refugee flows (Bing et al, 2003:578).

It is also very difficult to investigate whether there was any change in sexual behaviour as a result of the conflict. Unlike the cases of the DRC and Rwanda, literature on the sexual abuse that occurred during the Angolan conflict is limited. There is evidence from post conflict research which suggests that Angolan soldiers engaged in risky sexual practices such as unprotected sex with multiple partners (Bing et al, 2003:393). We cannot however assume that their sexual behaviour was different before or during the conflict.

With regard to the destruction of health services, there is general consensus that the war reduced the public health system in much of Angola to shambles[84] with most people relying primarily on non-governmental organizations for the supply of basic food and medical care (Medicins Sans Frontiers, 2000; Publico, 2000). The post conflict data does show that Angola has lower than average numbers of key health professionals and lower life expectancy than the regional average (McInnes, 2009:21).

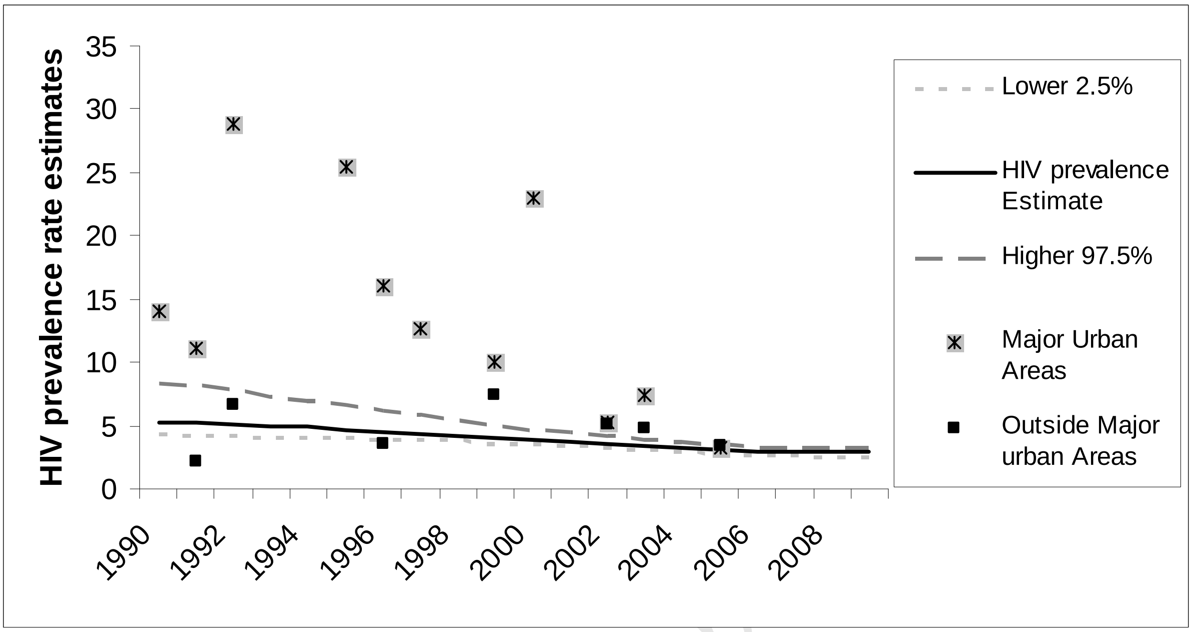
All in all, background factors such Angola's poor communication and transport infrastructure may have made the possibility of an upsurge in HIV prevalence during the conflict remote. In fact, they may have protected Angola from an explosive epidemic as shown by the econometric analysis in Chapter 7. Speculatively, Angola's low population density coupled with the collapse of health infrastructure further complicates any attempt to explain why Angola was seemingly protected from an upsurge in HIV prevalence during the conflict. The changes in sexual behaviour that occurred during the conflict are also very difficult to assess.

8.3 Ethiopia

HIV was first reported in Ethiopia in 1984. In 2007, the estimated adult HIV/AIDS prevalence in Ethiopia was 2.1 percent (USAID, 2010).[85] In 2008, this figure had reduced to 1.4 percent (PEPFAR, 2007)[86]. This estimate is based on testing done on a sample of 5,780 men and 5,300 women age 15 to 49 who gave informed consent. The survey found that HIV prevalence varies from region to region with the Southern Nations, Nationalities, and People's Region (SNNPR) having prevalence rates of 0.2 percent versus rates of about 6 percent found in the Gambela region (PEPFAR, 2007). Estimates (own estimates shown in Figure 8.3), produced in EPP and Spectrum show that HIV prevalence peaked around 1997. HIV is increasingly becoming a major national concern as projections (national level) estimate that in 2009, approximately 1.1 million Ethiopians were living with HIV and the HIV prevalence has begun picking up again (see Figure 8.3) reaching 2.3 percent in 2009 (USAID, 2010).

[84] This is evidenced by the high levels of child malnutrition and low levels of immunization (even by the very low sub-Saharan standards).
[85] http://www.usaid.gov/our_work/global_health/aids/Countries/africa/ethiopia.html
[86] http://www.pepfar.gov/documents/organization/116219.pdf

Figure 8.3 HIV prevalence in Ethiopia (1990-2005)



Source: Own EPP and Spectrum modelling

In modelling the HIV prevalence and incidence estimates in EPP, the epidemic in Ethiopia was modelled as a generalised epidemic. The ANC data was not down scaled by a factor of 0.8 as was the case for Angola but survey data was used to calibrate it. The calibration was done using the 2005 Demographic and Health Survey results (see Central Statistical Agency & ORC Macro (2006). Resultantly, the trend lines shown in Figure 8.3 seem to ignore outlier ANC sites such as Gondar which recorded prevalence rates at 25.3 percent in 1995 versus the national average of 2.4 percent in the same year estimated in this thesis. In Spectrum, the Coale-Demeny North life table was used as the model life table applicable to Ethiopia. Spectrum default assumptions on the total fertility rate reduction and life expectancy at birth were also used. Further, it was assumed that in the absence of antiretroviral treatment, people living with HIV will become eligible for treatment an average of 3 years before they are expected to die from an AIDS related cause (UNAIDS Reference Group on Estimates, Modelling and Projections, 2006). Information on ART rollout, PMTCT and child treatment services were obtained from the 2008 UNAIDS Ethiopia epidemiological fact sheet.

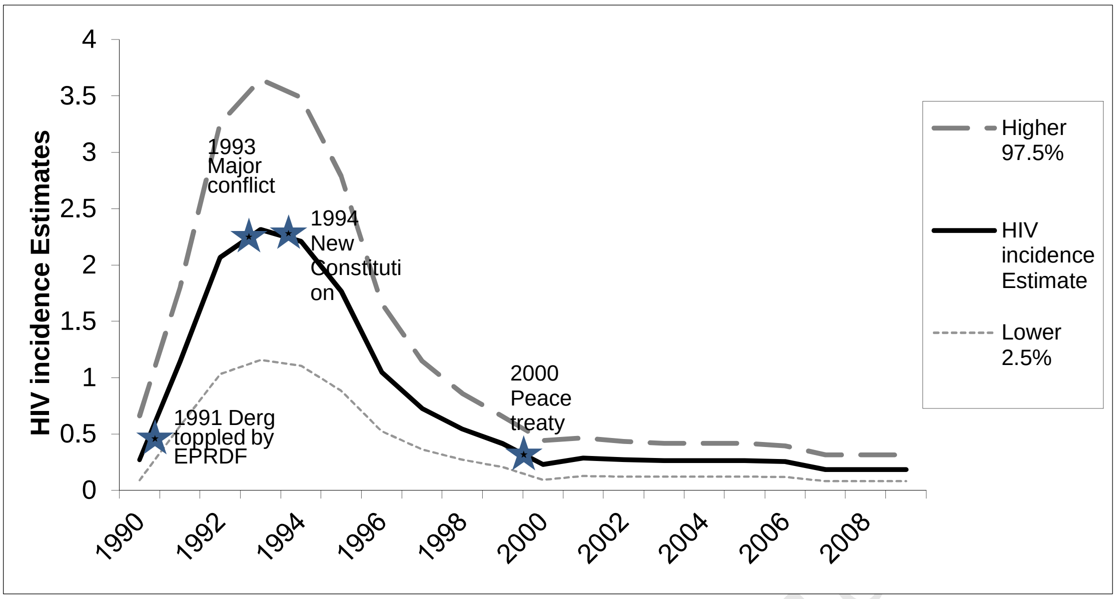
8.3.2 Conflict history

With the exception of a short lived Italian occupation from 1936-1941, Ethiopia is the only African country to not have been colonized (Embassy of Ethiopia, 2010)[87]. In 1974, the Derg, a military junta chaired by Major Mengistu Haile Mariam removed Emperor Haile Selassie who had ruled Ethiopia since 1930 (Embassy of Ethiopia, 2010). The military junta established a socialist state which, over 17 years survived numerous coups and uprisings. A combination of wide scale droughts, increased political resistance and refugee problems weakened the Derg and in 1991 they were toppled by a rebel group, the Ethiopian People's Revolutionary Democratic Front (EPRDF).

A constitution was adopted in 1994, and Ethiopia's first multiparty elections were held in 1995. A border war with Eritrea late in the 1990s ended with a peace treaty in December 2000[88]. In the period from 1990 to 2005, Ethiopia had a minor conflict (between 25 and 1000 battle related deaths) in each year except for 1993. These were primarily internal[89] contestations over territory or control of government (UCDP, 2010[90]). Figure 8.4 shows that HIV incidence rates are estimated to have peaked at 2.4% in 1993 and rapidly declined to levels below 0.5% in the early 2000s. The graph suggests that the major conflict in 1993 coincided with the turn in the trajectory of the epidemic. In the subsequent years, the rate of new infections fell except for a slight increase in incidence following the 2000 peace treaty. This would explain the negative relationship HIV and conflict that was shown in the regression analysis.

[87] http://ethiopianembassyabidjan.org/profile
[88] CIA, The World fact book https://www.cia.gov/library/publications/the-world-factbook/
[89] Captured by the domestic conflict variable used in the regressions in Chapter 7
[90] UCDP is the Uppsala Conflict Data Program.

Figure 8.4 HIV incidence in Ethiopia (1990-2005)



Source: Own EPP and Spectrum modelling

8.3.3 Traditional risk factors

Regarding the traditional risk factors, there is little data to allow a meaningful analysis. Chapter 5 revealed that the Ethiopian military is one of the few that went through a period (between 1998 to 2000) of pre-testing potential recruits for the purposes of excluding those who test positive (Abebe et al, 2003). Although this might lead to the expectation of generally lower prevalence rates in the military, it is impossible to clarify this in the absence of actual HIV tests on serving members. There is also very little evidence of sexual violence against women although this by no means suggests that such acts were not prevalent. Although Ethiopia had UN peace keeping missions in each year between 2000 and 2005 (where the dataset ends), there is no way of telling whether many of them came from higher prevalence countries or how much of an effect the peace keeping missions had on the trajectory of the epidemic.

8.3.4 Background factors

The conflict in Ethiopia predated the HIV epidemic which may have made it less likely for Ethiopia to witness increases in new infections. Further, the poor transport and communication infrastructure that characterizes Ethiopia would have made the mixing of different groups and the formation or maintenance of sexual networks difficult. For instance in the 1990s, the whole of Ethiopia was served by a total of 4,109 km of asphalt road, 9,287 of gravel and 5,610 rural road (FAO, 1995)[91]. This was an average of 31 km. of road (all types) per 1000 square kilometre (FAO, 1995). To paint a clearer picture, the situation meant that nearly 75% of Ethiopians lived more than a day's walk from all-weather roads (FAO, 1995). The war further ravaged this poor infrastructure.

A factor that may have made Ethiopia more susceptible to increases in HIV prevalence is its population density which has always been slightly above regional averages on account of refugees from Eritrea and Somalia. However, the regression results in Chapter showed that the link between estimated HIV prevalence and conflict is far from clear. According to PEPFAR (2007), there were an estimated 97,300 refugees in Ethiopia in early 2007. However, many of these were from Eritrea and Somalia, countries with lower HIV prevalence rates than Ethiopia making it unlikely that their settling in Ethiopia would lead to increases in HIV prevalence. Overall, the background factors in Ethiopia suggest it may have been protected from and upsurge in HIV prevalence although looking at the changes that occurred (vulnerability) during conflict twill help clarify this conclusion

8.3.5 Effect of conflict

The conflicts in Ethiopia have been less wide spread in recent years with most of the fighting occurring on the country's borders. The biggest effect of this has been the influx of refugees although as already stated, there is little information available to ascertain what kind of an impact this had on sexual behaviour. Ethiopia has a growing commercial sex industry particularly in urban centres. Plus News (2009)[92] reports that many of the women entering into commercial sex work are rural migrants that are either unemployed, lowly paid in their rural homes or are running away from unwanted marriages. Years of instability in these remote parts of the country would have hampered economic growth and prosperity restricting most development to the capital city Addis Ababa. The commercial sex industry in Addis

[91] http://www.africa.upenn.edu/eue_web/faoinfra.htm
[92] http://www.irinnews.org/report.aspx?ReportId=86718

Ababa is particularly vibrant, with sex workers earning up to 30 times a domestic workers salary (Plus News, 2009).

The prolonged conflicts in and around (on the borders) Ethiopia also severely damaged the country's health infrastructure. Ethiopia has long suffered from wide scale shortages of health workers and counsellors, in addition to poor access to sparse health services, inadequate sanitation, inefficient procurement systems as well as weak monitoring and evaluation systems (PEPFAR, 2007). Such inadequacies hamper the effectiveness of HIV prevention campaigns and treatment efforts thereby improving the chances for increases in HIV prevalence. They also often translate to higher AIDS mortality which would decrease HIV prevalence.

Overall, the decline in the health care system, growth of the commercial sex industry (in urban areas) and relatively high population density suggest a positive correlation between HIV prevalence and the occurrence of the conflict. However, the sparseness of the conflicts as well as the destruction of an already weak communications and transport infrastructure explains why although HIV prevalence rose initially in the early 1990s, it fell in the 2000s when Ethiopia had sustained minor conflicts[93].

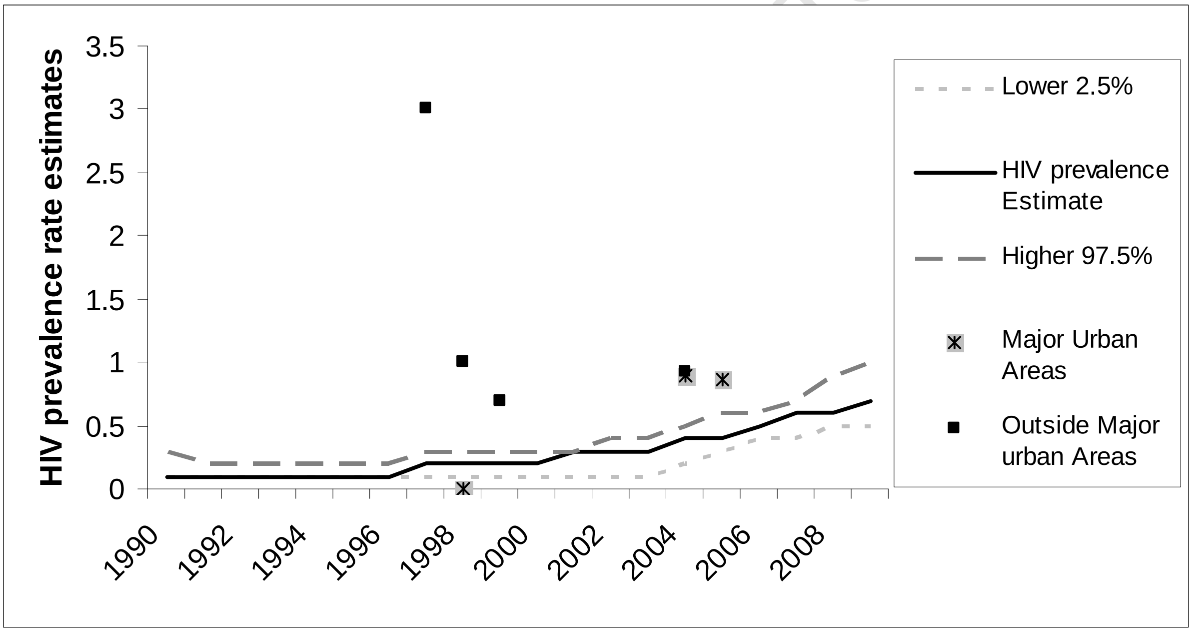
8.4 Somalia

In Somalia HIV was first noticeable in the 1980s. Studies carried out then in Mogadishu and two southern cities between 1985 and 1989 found no HIV infection in prostitutes, STD patients, or tuberculosis (Burans et al, 1990; Jama et al, 1987; Scott et al, 1991). However, the virus was noticed by 1995 studies in Mogadishu and two other southern cities (Watts et al, 1994). They found that 5 out of 245 prostitutes, 1 out of 79 military personnel and none of 43 tuberculosis or 80 STD patients was infected (Watts et al, 1994). Figure 8.5 shows the HIV prevalence estimates rates produced in this dissertation by using the EPP and Spectrum packages.

[93] Figure 8.4 also shows that the 1993 major conflict coincided with the beginning of a fast decline in HIV incidence.

In EPP, the epidemic in Somalia was modelled as a generalised epidemic. Further, as was recommended by UNAIDS, the ANC data was also down scaled by a factor of 0.8. (UNAIDS, 2007:10). The Coale-Demeny North life table was used as the model life table in Spectrum. Further, Spectrum default assumptions on the total fertility rate reduction and life expectancy at birth and the net survival of people living with HIV were used. As per the UNAIDS recommendation, it was also assumed that in the absence of antiretroviral treatment, people living with HIV will become eligible for treatment an average of 3 years before they are expected to die from an AIDS related cause (UNAIDS Reference Group on Estimates, Modelling and Projections, 2006). Data on ART rollout, PMTCT and child treatment services were obtained from the 2008 UNAIDS Somalia epidemiological fact sheet.

Figure 8.5 HIV prevalence in Somalia (1990-2005)



Source: Own EPP and Spectrum modelling.

As shown in Figure 8.5, HIV prevalence remained low throughout the 1990s and a study carried out in Mogadishu found no HIV infection among 157 blood donors, 57 inpatient adults and 42 inpatient children (Nur et al, 2000)[94]. In 1998, another study in Mogadishu also reported no HIV infections (UNAIDS, 2002). In the north of the country, the situation was slightly different. Studies in the northern part of Somalia, also known as Somaliland showed

[94] The authors observe that 'during the civil war in Somalia, no evidence of an increase in HIV-1 infections was found (p. 137).

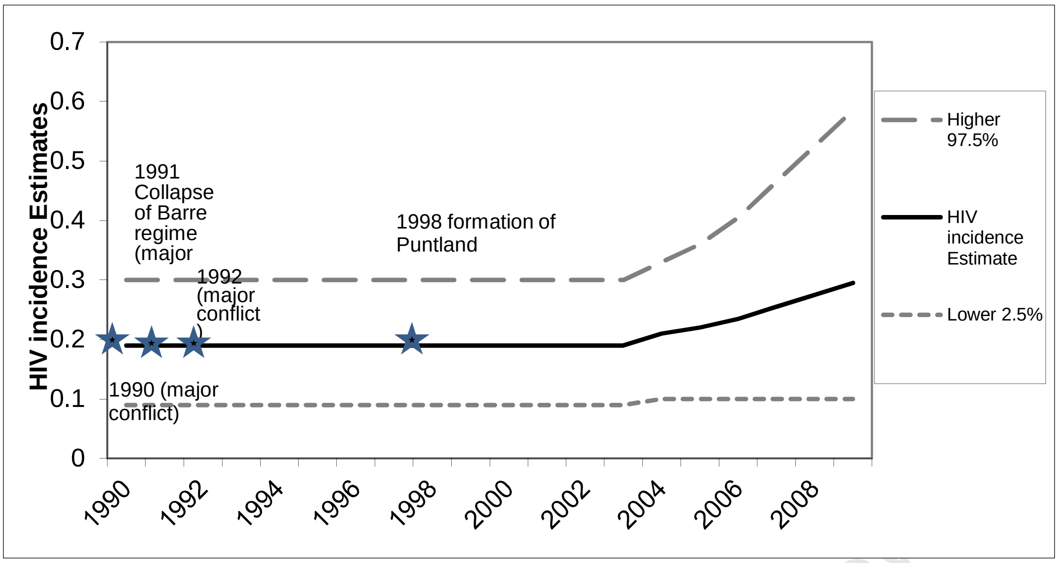
more substantial evidence of HIV in the general population. According to a study done on three northern cities in 1999, HIV prevalence was found to be 4.6% among 314 tuberculosis patients and 0.9% in antenatal clinics (Abokor, 2000)[95] . However, even in the north of Somalia, HIV prevalence rates are still much lower than in surrounding countries[96]. The EPP and Spectrum estimates of HIV prevalence shown in Figure 8.5 are national level estimates that weld the disparate Northern and Southern epidemics.

8.4.2 Conflict in Somalia

Just as many African countries have struggled in making a peaceful transition from colonial to democratic rule, Somalia's story is no different. Britain withdrew from British Somaliland (now part of the Republic of Somaliland) in 1960 allowing its former protectorate to join with Italian Somaliland and form the new nation of Somalia[97] . Following a 1969 coup led by Mohamed Siad Barre, socialist Somalia was relatively stable (for the next two decades) despite Barre adopting an authoritarian leadership style[98] . Barre's regime collapsed in 1991 (see Figure 8.6) and Somalia descended into factional fighting and turmoil. The opposition liberation movements included the United Somali Congress, USC (Hayiwe clan), the Somali Patriotic Movement (Ogadeni clan), and the Somali Salvation Democratic Movement (Majerten clan) (World Bank, 2005:10).

[95] http://www.iprt.org/HIV%20survey.htm
[96] Kenya's and Ethiopia's HIV epidemics reached double-digit rates of infection in at least one urban antenatal clinic by 1990 – 91 (UNAIDS, 2002d; UNAIDS, 2000e)
[97] CIA, The World fact book https://www.cia.gov/library/publications/the-world-factbook/
[98] CIA, The World fact book https://www.cia.gov/library/publications/the-world-factbook/

Figure 8.6 HIV incidence in Somalia (1990-2005)



Source: Own EPP and Spectrum modelling.

Northern clans declared an independent Republic of Somaliland which is comprised of the Awdal, Woqooyi Galbeed, Togdheer, Sanaag, and Sool regions in May of 1991. The Republic of Somaliland has maintained a stable existence and holds municipal, parliamentary, and presidential elections despite lacking recognition from any other government (World Bank, 2005:6). Somalia increasingly became fractured with a semi-autonomous state of Puntland, comprising the Bari, Nugaal, and northern Mudug regions being formed in 1998 (see Figure 8.6)[99]. Although it does not aim at independence, it has been self-governing since 1998 and has made some progress towards constructing a legitimate and representative government. Puntland not only claims parts of eastern Sool and Sanaag, it also disputes its border with Somaliland. Figure 8.6 shows that HIV incidence was fairly stagnant during the major conflict years and only began to rise in 2003.

In the period from 1990 to 2005, only 1990, 1991 and 1992 were classified as major (all domestic) conflict years according to the UCDP criterion (i.e. above 1000 battle related deaths were reported). This means that most intense fighting was carried out in the immediate

[99] CIA, The World fact book https://www.cia.gov/library/publications/the-world-factbook/

period leading to the collapse of the Barre regime. The subsequent territorial conflicts and ethnic fighting which resulted in the formation of Somaliland and Puntland have been classified as minor conflicts (Figure 8.6 therefore only shows the major conflict years).

By 2005, Somalia consisted three major parts namely, Somaliland with a population of 3 million, Puntland 1.5 million and South-central Somalia 5 million (World Bank, 2005:7; Ministry of National Planning and Coordination, 2004; PDRC, 2004). Further 8.6 shows that incidence rates were very low all throughout the fighting that followed the collapse of the Barre regime. Further, it shows that as relative peace began to develop in the mid-2000s, HIV incidence began rising. This suggests that conflict is not a plausible driver of HIV in Somalia and that it may even have had a protective effect.

8.4.3 Background factors

As already mentioned, one of the five background factors that McInnes argues contribute to a country's susceptibility is the level of HIV prevalence when the conflict starts (McInnes, 2009:12). However, in the case of Somalia, although the debut of major conflict was in the early 1990s, it already had a history of minor conflicts predating the first cases of HIV. The state of transport and communication infrastructure is another important background factor (Mock et al, 2004:11). Overall population density in Somalia is generally low. However, the population density has been on a steady rise from a 1993 low of 10 people per square metre (largely due to the displacement of people during the 1991 to 1993 major conflict) to a 2005 high of 13 people per square metre. Although rather speculative, these factors would have made a rise in overall prevalence rates highly unlikely to occur during the conflict period.

8.4.4 Impact of conflict

The major conflict years that followed the toppling of Barre provided the biggest opportunity for disassortive mixing. Barre's government was responsible for gross atrocities against civilians resulting in the deaths of an estimated 50000 to 60000 Somalis mainly from the Isaaq clan (World Bank, 2005:10; Africa Watch, 1990). In addition to forcing 400000 Somali's to flee to Ethiopia, government's assault on the southern city of Hargeysa and surrounding regions lead to a further 400000 Somali's being internally displaced (World Bank, 2005:10; Africa Watch, 1990). Important to note however, is that much of this migration was from the war ravaged south to northern parts of the country where HIV prevalence was/is generally higher.

Although the major conflict years devastated the whole of Somalia, the subsequent inter clan clashes were more prevalent in the turbulent south. The northwest and northeast of Somalia, Somaliland in particular, maintained the more robust authority of traditional clan elders and more political cohesion among the clans (World Bank, 2005:11).[100] This might explain why the epidemic thrived in the north as the social cohesion and opportunity for commerce would have facilitated the formation and maintenance of sexual networks.

Despite the state of health services and HIV awareness being other important factors, data on these is not available. Overall, Somalia was likely protected from an explosive epidemic by a combination of its poor transport and communication infrastructure, low population density and the difficulty of movement that was exacerbated by the conflict particularly in the South.

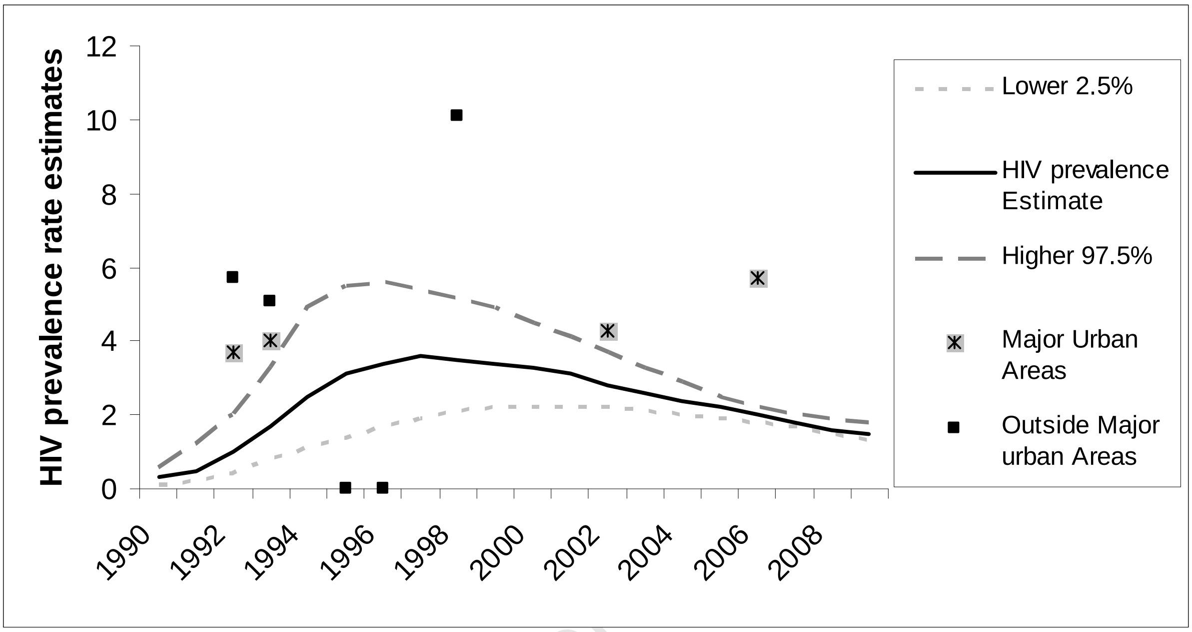
8.5 Liberia

Just like many other African countries, HIV became noticeable in Liberia as early as in the 1980s. From 1987 to 1989, studies showed that 1-4% of tuberculosis patients were infected while 0.6% of blood donors as well as 0.7% of visa applicants tested positive (US Census Bureau, 2003). However, other studies carried out in the period found no HIV in 941 healthy individuals, 35 STD patients and 30 prostitutes (US Census Bureau, 2003). UNGASS estimates of HIV prevalence in Liberia show that the epidemic has been very stable becoming generalised in 1994 and reaching about 1.9% in 2005 (UNGASS, 2008). These estimates are confirmed by my own EPP and Spectrum modelling results shown in Figure 8.7.

[100] There are clear differences between the epidemics in North versus that in the South with the war torn south having much lower prevalence rates that the North.

Liberia shares a border with Sierra Leone which has generally had lower prevalence rates than Liberia (Gisselquist, 2004:5). However, Liberia's other neighbour, Cote d'Ivoire has much higher prevalence rates[101].

Figure 8.7 HIV prevalence in Liberia (1990-2005)



Source: Own EPP and Spectrum modelling

When modelling the epidemic in EPP, the ANC site data was calibrated using the 2007 Demographic and Health Survey (Liberia Institute of Statistics and Geo-Information Services & Macro International, 2007). Further, the epidemic was modelled as a generalised one. Unlike for Zimbabwe and Angola, the Coale-Demeny West life table was used as the model life table. However, many of the default Spectrum assumptions such as those on fertility rates, mortality and life expectancy at birth were used. It was also assumed that in the absence of antiretroviral treatment, people living with HIV will become eligible for treatment an average of 3 years before they are expected to die from an AIDS related cause. The net survival of people living with HIV was assumed to be 11 years as is the case with all the modelling carried out in this thesis. Lastly the 2008 UNAIDS Liberia epidemiological fact sheet was the main source of the data on ART rollout, PMTCT and child treatment services.

[101] The median HIV prevalence in 28 sentinel antenatal clinics was 7.3% (WHO/AFRO, 2003).

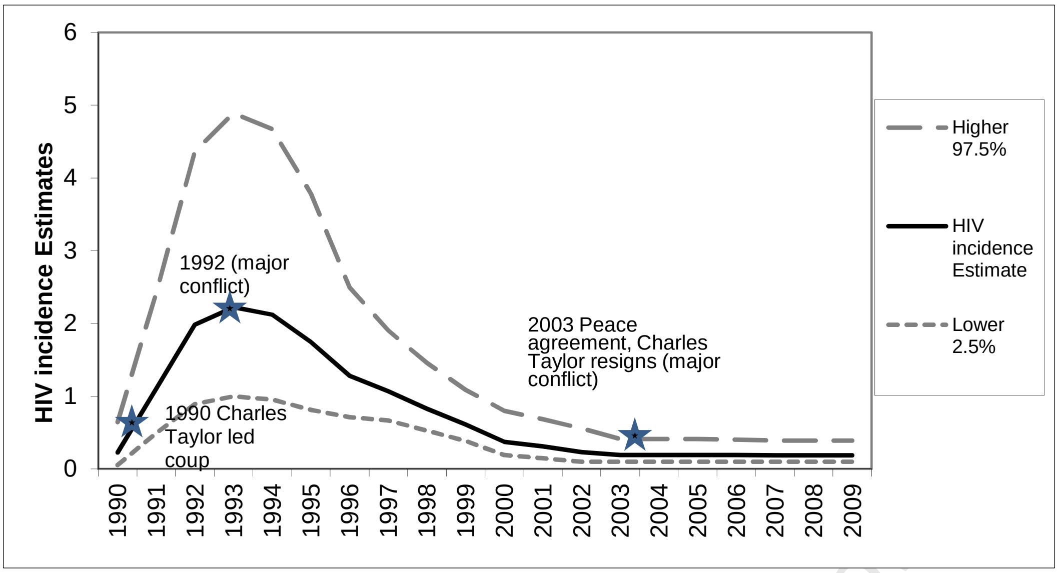
8.5.2 Liberia's conflict history

Liberia's civil war began in December 1989 when Charles Taylor's National Patriotic Front of Liberia entered the country from Côte d'Ivoire. Taylor seized power from Samuel Doe in 1990 (see Figure 8.8) (Gisselquist, 2004: 5). Samuel Doe's government, which came in through military coup in 1980 had governed with an iron fist and for years, competing armed factions controlled parts of the country (Gisselquist, 2004:5)[102]. As shown in Figure 8.8, HIV incidence rose rapidly after 1990 only to start falling after 1992 (a UCDP major conflict year shown in Figure 8.8. In 1997, there was a period of relative peace, which allowed for an election that brought Chares Taylor into power although major fighting resumed in 2000[103].

Another peace agreement in 2003 (see Figure 8.8) led to the resignation of Charles Taylor and he fled to Nigeria where he was later arrested and charged with war crimes for his involvement in Sierra Leone's civil war. President Taylor was accused of trading weapons for conflict diamonds from brutal Sierra Leone rebels, and prolonging the conflict. Taylor was indicted for war crimes at the United Nation's Tribunal in Sierra Leone on June 4, 2003 (Global Security, 2010). In the period from 1990 to 2005, Liberia had three major conflicts in 1990, 1992 and 2003 with the other conflict years between 1993 and 2002 being classified as minor conflict years (UCDP, 2010). Following two years of rule by a transitional government, democratic elections in late 2005 brought President Ellen Johnson Sirleaf to power.

[102] Also see CIA, The World fact book https://www.cia.gov/library/publications/the-world-factbook/
[103] See CIA, The World fact book https://www.cia.gov/library/publications/the-world-factbook/

Figure 8.8 HIV incidence in Liberia (1990-2005)



Source: Own EPP and Spectrum modelling

Figure 8.8 shows that despite estimated HIV incidence rising rapidly between 1990 and 1993, it declined at an equally fast rate between 1993 and 2000, a period where each year was a minor conflict year.

8.5.3 Background factors

In the case of Liberia, the HIV epidemic predated the conflict although the fact that prevalence was very low (UNGASS puts this number at 0.2% in 1990) and remained low after the conflict makes it difficult to assess how conflict affected it. The civil war in the early 1990s led to a decline in communication infrastructure as evidenced by the decline in the number of telephone lines in the country from 9380 in 1990 to 3300 in 1991 (World Bank databank, 2010). However, the road infrastructure was left intact and the number of useable roads actually increased all throughout the 1990s (World Bank databank, 2010). Notwithstanding, cross country travel was made impossible by the large presence of militia in both the urban and rural regions (Swiss et al, 1998).

As mentioned earlier, another background factor which might determine susceptibility to conflict-induced increases in HIV prevalence is population density. The population density in Liberia was relatively low (compared to other countries in the region) and ranged from 22.5 people per square kilometre 1990 to about 32 people per square kilometre at the end of the conflict (2003). This increase in population density may have set Liberia up for a rise in HIV (HIV prevalence rose from 0.2% in 1990 to 1.75% in 2003) although there is no hard evidence to support this. The increase in population density is likely to have been as a result of an influx of refugees from low prevalence countries such as Sierra Leone into Liberia.

8.5.4 Effect of conflict

Almost half of Liberia's 2.5 million people were forced to flee their homes at least once during the civil conflict (Agency for International Development, 1991:2). Resultantly, Liberia had the largest percentage of refugees and internally displaced people of any country in the world (Swiss et al, 1991:2). What is crucial for the spread of HIV is not so much the movement of people but the degree to which they mix with new communities (McInnes, 2009:13). Data constraints make it is difficult to ascertain the extent of the mixing between internally displaced groups across Liberia.

One of the main characteristics of the Liberian conflict was sexual violence. Women and young children were particularly vulnerable. When militia took control of a village and when women crossed check points, many were reportedly forced to cook for a soldier or fighter and this was often a precursor to them being abused (Swiss, et al, 1998:5). A study carried out in Monrovia comprising survey interviews of a random sample of 205 women and girls between the ages of 15 and 70 years (88% participation rate) revealed the extent of this problem. The result is powerful particularly because Monrovia was actually safer than most parts of Liberia as it was under a West African peacekeeping force (Swiss, et al, 1998:2). The study found that 49% of the women had experienced at least 1 act of physical or sexual violence by a soldier or fighter. 15% had been subjected to attempted rape or had been sexually coerced (15%). They also found that women who had been accused of belonging to a particular ethnic group of fighting faction were at increased risk of abuse (Swiss et al, 1998:5).

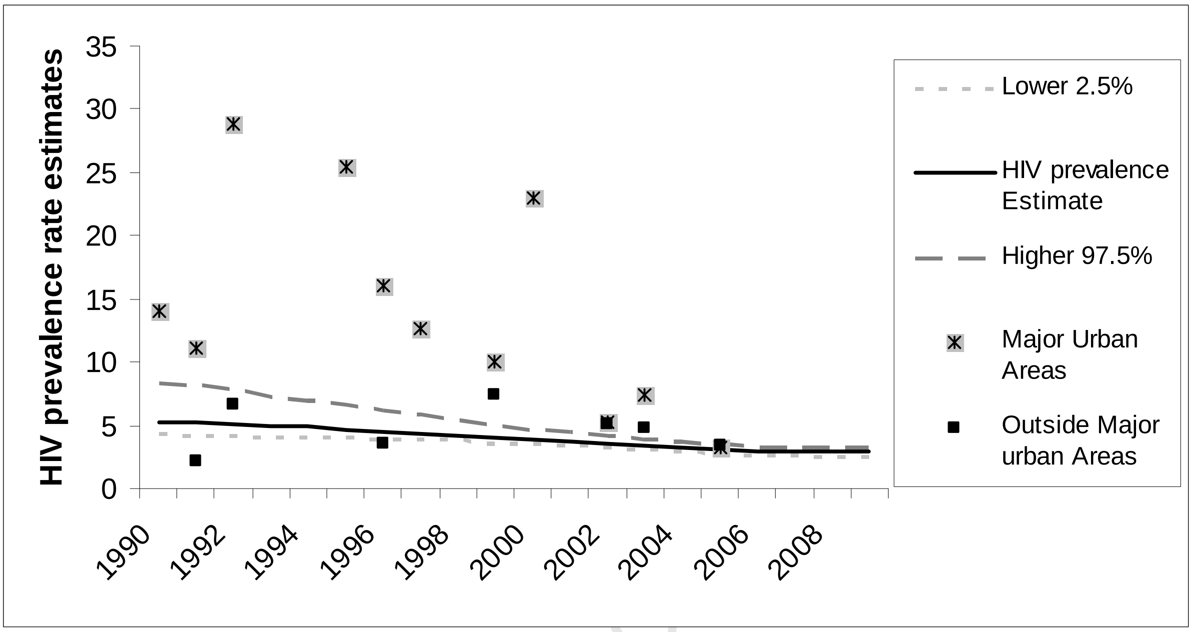
Just as is the case in most conflict ridden countries, Liberia's health care system was also ravaged by the fighting (Buseh, 2009) although the extent to which this affected the health care system was affected by war is hard to determine. Overall, despite being possibly vulnerable to an upsurge in HIV prevalence on account of the increase in population density and internally displaced persons, the degree of disassortive mixing that occurred is difficult to ascertain. In addition, the extent to which cases of rape and other forms of sexual violence may have affected national rates of HIV infection is unclear.

8.6 Rwanda

The first case of HIV/AIDS in Rwanda was probably discovered in 1983 (Davenport, 2009:29). Because of the data restrictions and poor record keeping, tracing the exact trajectory of the epidemic is difficult to accomplish (Davenport, 2009:29). However, 1991 data from 5 clinic sites reported prevalence rates of 27% in urban areas and 2.2% in rural areas making Rwanda one of the most infected HIV countries in the world (DHS, 2005). In the early 1990s, international organizations such as Population Services International (PSI) and Project San Francisco established surveillance and education programs that primarily serviced urban areas such as Kigali and Butare (Davenport, 2009:29). Unfortunately these organizations left Rwanda in 1992 as they closed their centres because of violence or lack of funding. They only returned after the 1994 genocide and the transitional government supported the setting up of a new HIV prevalence surveillance system. The first post genocide DHS survey (of 10 sites) revealed that prevalence in urban areas had remained constant at about 27% whereas it had gone up to 6.9% in rural areas (DHS, 2005).

Rwanda continued to improve its surveillance capacity and in 2002 a survey including 24 sites put HIV prevalence estimates at 7.0 – 8.5% in urban areas and 2.6 – 3.6% in rural areas (DHS, 2005). This probably reflected a dramatic change in the urban population (rural urban migration) (Davenport, 2009:29). Overall prevalence rate estimates shown in Figure 8.9 are national level estimates from EPP and Spectrum modelling. Although they show a steady decrease in prevalence from the 1990s to the 2000s, they are averages and fail to capture the rural versus urban differences in the epidemic.[104]

Figure 8.9 HIV prevalence in Rwanda (1990-2005)



Source: Own EPP and Spectrum modelling.

In modelling the HIV prevalence and incidence estimates shown in Figure 8.9 above, the epidemic in Rwanda was modelled as a generalised epidemic. Further The ANC data was calibrated using the 2005 DHS whose findings have already been highlighted in this section. Spectrum default assumptions on the total fertility rates, mortality, life expectancy at birth as well as the model life table (Coale-Demeny North life table) were used. It was assumed that in the absence of antiretroviral treatment, people living with HIV will become eligible for treatment an average of 3 years before they are expected to die from an AIDS related cause (UNAIDS Reference Group on Estimates, Modelling and Projections, 2006). Further it was also assumed that the net survival of people living with HIV is 11 years and the 2008 UNAIDS Rwanda epidemiological fact sheet was used as the main source of the data on ART rollout, child treatment services and PMTCT.

[104] This is one of the weaknesses with using national level estimates of HIV incidence and prevalence as they ignore region specific nuances

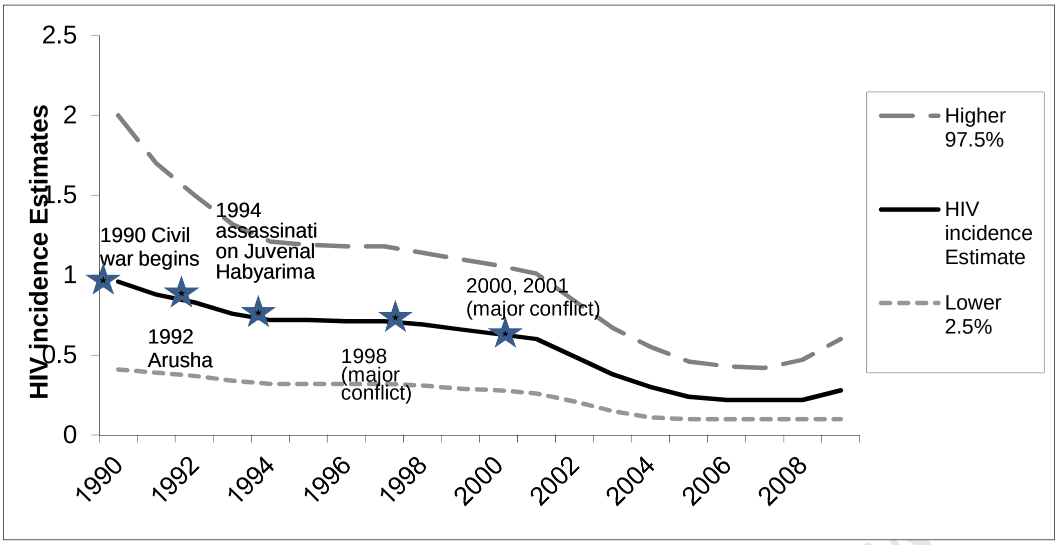
8.6.2 Conflict in Rwanda

Rwanda has been turbulent since attaining independence from Belgium in 1962. According to the UCDP conflict classifications, in the period from 1990 to 2005, Rwanda had minor conflicts (more than 25 but less than 1000 battle related deaths) in 1990, 1993, 1994, 1997, 1999 and 2000. In 1991, 1992, 2001 and 2002, Rwanda had major conflicts. The fighting in Rwanda has always had ethnic roots with the majority Hutu overthrowing the ruling Tutsi king in 1959[105]. In the post-colonial period, the Hutu ethnic group committed a series of government sponsored programs against minority Tutsis in 1964 and then again in 1972-73 (Davenport, 2009:29).

The Tutsis fled into Uganda and other neighbouring countries and their children later formed a rebel group, the Rwandan Patriotic Front (RPF) which began the civil war in October 1990 (see Figure 8.10). A two year civil war followed, ending with the signing of the 1992 Arusha ceasefire (see Figure 8.10) (Davenport, 2009:30). The ceasefire was not entirely successful and disagreements over the power sharing agreement led to the formation of extremist factions (within the old Rwandan government and Rwandan army) that were responsible for the 1994 genocide (Prunier, 1995).

[105] See CIA, The World fact book https://www.cia.gov/library/publications/the-world-factbook/

Figure 8.10 HIV incidence in Rwanda (1990-2005)



Source: Own EPP and Spectrum modelling.

The April 1994 assassination of the Rwandan President, Juvenal Habyarimana, sparked what was later known as the Rwandan genocide (Davenport, 2009:30). Tutsi rebels halted the genocide of 800,000 Tutsis and moderate Hutus in July 1994 but in the process, caused about 2 million Hutus to flee to neighbouring countries in fear of Tutsi retribution[106]. The first post war local elections were held in 1999 and the first post-genocide presidential and legislative elections in 2003. Overall, Figure 8.10 shows that HIV incidence rates fell rapidly during the years of civil war leading up to the 1994 genocide. However as peace keeping efforts returned the country to relative stability, HIV incidence stabilised only to dip again around 2001 (and 2002) which was a major conflict year according to the UCDP.

8.6.3 Background factors

The HIV epidemic sprung up around 1983 immediately prior to the conflict. The fact that HIV prevalence was low when the conflict started suggests that the conflict was unlikely to cause an upsurge in HIV prevalence as theorised by McInnes (2009). The epidemic seems to have been more menacing in urban areas although it was also widespread in the rural parts of

[106] See CIA, The World fact book https://www.cia.gov/library/publications/the-world-factbook/

the country. This later changed as the conflict led to the displacement of millions, with prevalence in urban areas falling as urban population densities increased. Rwanda has been argued to have one of the highest population densities in the world, although this declined slightly in the mid-1990s after the genocide (UNGASS, 2008:14; United Nations Population Division, 2008). This factor may have made it more likely for the HIV epidemic to spread although as noted earlier, the regression results do not find a strong link between population density and HIV prevalence.

It is particularly challenging to assess how the nature and scale of the conflict influenced the progression of Rwanda's HIV epidemic. Large areas of the country were ravaged by the war at different times and the degree of violence was varied (McInnes, 2009:28). As already highlighted, the UCDP conflict classification has a number of weaknesses as a measure of the scope and range of conflict. The more obvious one is that the UCDP classifies the1994 genocide as a minor conflict on account of it not having reported more than 25 battle related deaths. These aren't battle related deaths as there was only one aggressor (i.e. the genocide was not a battle). This is despite the fact over 800, 000 were killed in the ethnic cleansing. This is one shortcoming with the regression models presented in this chapter as the conflict variable used is not ideal.

The state of Rwandan transport infrastructure was poor throughout the conflict with a very low percentage of roads paved and small numbers of buses (McInnes, 2009:24). The war also destroyed Rwanda's communication infrastructure from 11'763 telephone lines in 1991 to 6'900 telephone lines in 1995 (World Bank databank, 2010). This would have reduced human mobility although the large flow of people fleeing the conflict signifies that mobility was still possible (McInnes, 2009:24).

Despite having poor transport and communication infrastructure, all the ingredients for a conflict-induced upsurge in HIV prevalence were present in Rwanda. According to McInnes, initial high prevalence rates and the high population density increased the chance of an upsurge in HIV prevalence (McInnes, 2009:24). The large number of rape and sexual assault cases throughout the conflict also increased susceptibility. Despite all these factors, HIV prevalence fell during the period of the conflict.

8.6.4 Impact of conflict

When discussing the potential of disassortive mixing during the conflict in Rwanda, McInnes (2009) makes a couple of points. Firstly, the genocide resulted in massive displacement of people. The instability that characterised the 1990s led to many people becoming refugees in neighbouring countries as well as internally. With this dislocation came the rape and abuse of women. These factors increased vulnerability. However it is important to investigate the nature of these movements further. McInnes (2009:25) notes that disassortive mixing may have been limited by the fact that refugee flows were along ethnic lines and communities may well have retained a degree of coherence as a consequence. The potential for further rises in HIV prevalence might also have been reduced by the fact that involvement of foreign armies was not high and the second part of the conflict (in the late 1990s to early 2000s) was fought almost entirely outside the country (McInnes, 2009:25).

Changes in sexual behaviour as a result of conflict are another factor that is particularly crucial given the nature of the conflict in Rwanda. Many have highlighted how rape was used as a weapon. Sexual violence against women and girls constituted a central part of the genocidal strategy (Amnesty International, 2004). Some have alleged that this was done so as deliberately infect Tutsi women with HIV. An example of this is a view by Paula Donovan (2002:17) that has repeatedly been cited by many scholars including McInnes (2009:15).

"Integral to the plan to annihilate the Tutsi population was the systematic sexual molestation, mutilation, and rape of women and girls...Most survivors describe the genocide as a bloodbath during which rape was inevitable for practically all females... Eyewitnesses recounted later that marauders carrying the virus described their intentions to their victims: they were going to rape and infect them as an ultimate punishment that would guarantee long-suffering and death."

However, Donovan concedes that it is impossible to calculate how many women who were raped were subsequently killed. It is also difficult to know how many rapists claiming to be HIV positive actually were primarily because testing was not extensive (Donovan, 2002:18).

All these factors make the task of assessing if such sexual violence had an impact on the trajectory of the epidemic very difficult, not least of all Donovan (2002:18) claiming that the rape of Tutsi women contributed to a subsequent increase in HIV prevalence. This is in spite of some data that shows that HIV prevalence amongst pregnant women in urban Rwanda declined in the years after the genocide as the overall national prevalence rate continued to fall (UNAIDS, 2007; McInnes, 2009:25).

In addition, some empirical work has shown that even in the most extreme cases, widespread rape in conflict counties in sub-Saharan Africa is not associated with direct population level changes in HIV prevalence (Anema et al, 2008). This however does not mean that in such situations women are not infected nor does it in anyway discount the plight of such victims. This as McInnes explains, means that that at a national population level, the use of rape as a weapon of war is not a sufficient change in sexual behaviour to significantly increase vulnerability to increases in HIV prevalence on a national scale (McInnes, 2009:25).

The evidence on behavioural change in Rwanda is also very hard to interpret. The high pre-conflict prevalence rates suggest that risky sexual behaviour was not uncommon. This may have been because of social restrictions on the use of condoms and low level of knowledge on how HIV is transmitted (McInnes, 2009:25). This is particularly concerning given the high HIV prevalence rates among commercial sex workers pre-conflict. For instance, two surveys in Butare in 1983 and 1984 revealed that among commercial sex workers, HIV prevalence was at 75% and 88% respectively (Kayirangwa et al, 2008:29)[107].

Although some have claimed that the AIDS response was not affected by the conflict, there is reason to be more cautious. McInnes (2009:26) argues that although the genocide had a devastating effect on these services[108], the impact appears to have been relatively short lived and major testing and preventative programmes were begun after the second wave of conflict. Others have argued that by the time the conflict ended, Rwanda was already experiencing a

[107] All of these factors explain why prevalence was extremely high in urban areas in the late 19980s and early 1990s

[108] Rwanda was estimated to have lost more than 80% of its health personnel through death or flight during the genocide.

rapid scale-up of HIV prevention, care, and treatment programmes (Kayirangwa et al, 2008:29).

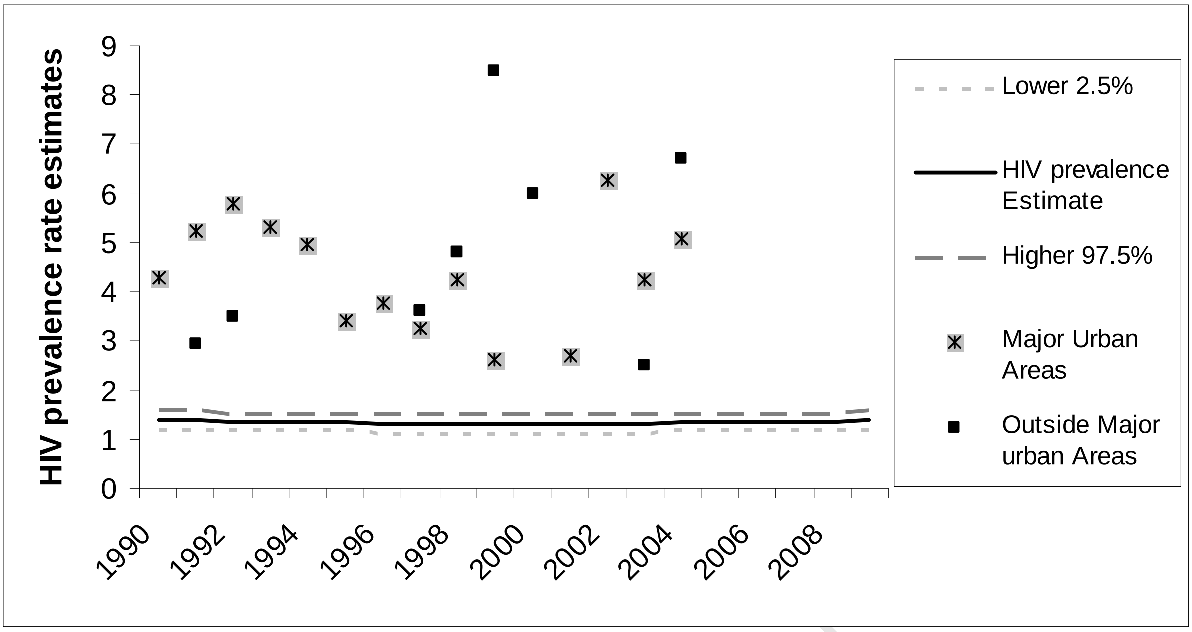
In the final analysis, the case of Rwanda is a puzzling one. Despite there being background factors such as high population density and the displacement of people which set it up for an increase in HIV prevalence, Rwanda's epidemic slowed down. The decline of an already poor communication and infrastructure network may have contributed to this slowdown in prevalence.

8.7 Democratic Republic of the Congo

One of the first African countries to recognize HIV/AIDS, the Democratic Republic of the Congo (DRC) started registering cases in 1983 (USAID, 2008:1). By 2003, the DRC had more than one million people living with HIV/AIDS (UNAIDS, 2004). However, it has had lower HIV prevalence rates than most other countries in the sub-Saharan Africa. The HIV/AIDS prevalence rate in the DRC was 4.2% in 2003, compared to 7.5% in sub-Saharan Africa and 1.1% globally (UNAIDS, 2004). The trends in HIV prevalence in the DRC are shown in Figure 8.11 which illustrates my own estimates of HIV prevalence drawn from EPP and Spectrum.

The epidemic in the DRC was modelled as a generalised epidemic and the ANC data was down scaled by a factor of 0.8. The Coale-Demeny North life table was used as the model life table and Spectrum default assumptions on the total fertility rate reduction and life expectancy at birth were employed. As per the UNAIDS recommendation, it was assumed that in the absence of antiretroviral treatment, people living with HIV will become eligible for treatment an average of 3 years before they are expected to die from an AIDS related cause (UNAIDS Reference Group on Estimates, Modelling and Projections, 2006). As is the case with all the modelling done in this thesis, it was also assumed that the net survival of people living with HIV is 11 years (UNAIDS Reference Group on Estimates, Modelling and Projections, 2006). The main source of the data on ART rollout, PMTCT and child treatment services was the 2008 UNAIDS DRC epidemiological fact sheet.

Figure 8.11 HIV prevalence in the DRC (1990-2005)



Source: Own EPP and Spectrum modelling

The primary mode of transmission of HIV in the DRC is heterosexual activity which in 2008, was estimated to have accounted for 87% of the cases (USAID, 2008:1). The conflict in the DRC has made research on the epidemiology of HIV in the country very difficult. Resultantly, HIV surveillance data are limited (UNAIDS, 2004). The little data available shows that the eastern region has generally had higher prevalence rates than elsewhere in the country (UNAIDS, 2004; Kaiser Family Foundation, 2005)[109].

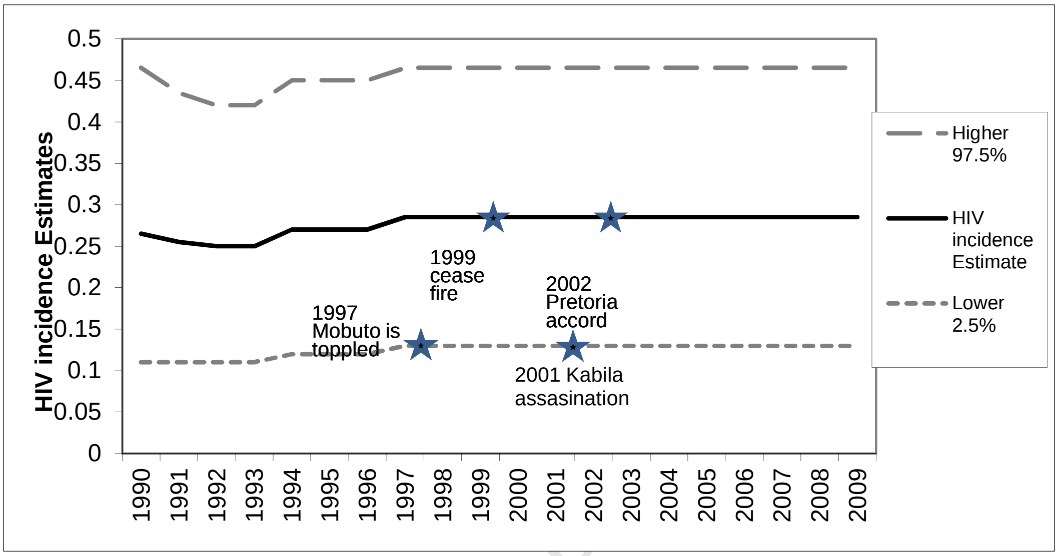
8.7.2 The conflict

First established as a Belgian colony in 1908, the Republic of the Congo gained its independence in 1960. However, its early years were marred by political and social instability. Colonel Joseph Mobutu seized power and declared himself president in a November 1965 coup ushering in a new era of political turmoil in the DRC. Mobutu entrenched his dictatorship of the DRC and managed to retain power for 32 years through several sham elections, as well as through brutal force (CIA factbook, 2011). The tensions in

[109] Available data indicate that prevalence rates range from approximately 1.8% in Mikalayi and 3% in Bunia and Bukavu to 6.3% and 7% in Kisangani and Lubumbashi, respectively. High HIV prevalence rates were also found in some rural areas, including Neisu (6.7%) and Lodja (6.5%).

the country simmered and in the 1990s, the Zairian army and local population began to attack the ethnic Tutsi Banyamulenges in the eastern part of the country (McInnes, 2009:27). Hutu refugees fleeing Rwanda further worsened the tensions and around October 1996, an alliance of Banyamulenges and anti-government groups mobilized against the Mobuto regime (McInnes, 2009:27).

Figure 8.12 HIV incidence in the DRC (1990-2005)



Source: Own EPP and Spectrum modelling

The rebellion, which resulted in the toppling of the Mobuto regime in May of 1997 (see Figure 8.12), was led by Laurent Kabila who had backing from Rwanda and Burundi[110] . With just over a year in power, the Mobuto government faced rebellion from within as factionalism rocked the former liberation heroes (McInnes, 2009:2007). This second insurrection was also backed by Rwanda and Uganda and troops from Angola, Chad, Namibia, Sudan, and Zimbabwe intervened to support Kabila's regime. This second phase of fighting continued until 2001 despite the signing of a cease-fire agreement by the DRC, Congolese armed rebel groups, Angola, Namibia, Rwanda, Uganda, and Zimbabwe in July 1999 (see Figure 8.12). During the second phase of the conflict, new militia groups consisting

[110] See CIA, The World fact book https://www.cia.gov/library/publications/the-world-factbook/

of disaffected youths from the eastern part of the country increasingly became involved in the violence (UCDP, 2008).

Joseph Kabila became president of the DRC in January of 2001 (see Figure 8.12) after his father Laurent was assassinated. In July 2003, after the withdrawal of Rwandan forces from eastern DRC and the signing of the Pretoria Accord in 2002, a transitional government was formed. As shown in Figure 8.12, estimated incidence rates have remained constant throughout the 1990s and 2000s. However these estimates are based on limited data. Regardless, it is important to find out the reason for this trend (assuming the estimates paint a reasonably accurate picture. Figure 8.12 also shows that it is difficult to see a pattern of how the conflict years may have impacted incidence rates as HIV incidence was consistently very low (averaging around 0.25%).

8.7.3 Background factors

The civil war in the DRC intensified in the 1990s, a time when the HIV epidemic was advanced and levelling off. The four years from 1997 to 2000 are classified as major conflict years (UCDP, 2010). In addition, the epidemic appears to have reached its peak in the 1990s, when life expectancy had dropped to 9% (USAID, 2008). This on its own means there are other factors predating the conflict year, which had limited the spread of the epidemic. Consequently, the debut of conflict was a less important factor in determining the trajectory of the epidemic (McInnes, 2009:27). One of these factors is possibly the poor road and communication infrastructure as highlighted by Iliffe (2006:5) and others. The DRC has notoriously poor transport infrastructure with less than 2% of the roads paved. The conflict possibly led to the decline in transport and communication infrastructure and so would therefore have limited the spread of the virus by hampering cross country movements. About 90% of the DRC's population live in the eastern parts of the country where surveys have shown that HIV prevalence is five times greater than the national average (USAID, 2008:1). The conflict, poor communication and transport infrastructure meant that communities in other regions were isolated from those in eastern side of the DRC which could explain their lower prevalence rates.

The population density in the DRC is relatively low and was at 19 per square kilometre in 1995 and 22 in 2000 (McInnes, 2009:29). This however, is not the full picture as more than 60% of these people live in rural areas where population density is even lower (UNGASS, 2005:3; WHO/UNAIDS, 2004:3). This means that the epidemic was unlikely to be high in these low population density and rural areas where conflict was more concentrated.
The ethnic dimension of the DRC conflict would have increased the possibility of an uptick in HIV prevalence, especially given the fact that many other countries took part in the war. This would have increased the level of antipathy, thereby possibly contributing to the worrying number of cases of rape and sexual violence. More on this will be discussed in the next section.

8.7.4 Effect of the conflict

As regards to disassortive mixing, it is conceivable that the large number of foreign parties involved in the conflict (foreign troops and internally displaced people) would have mixed to a substantial extent. The extent to which this mixing and creation of new commercial, consensual, casual, transactional or non-consensual sexual networks impacted HIV prevalence is difficult to ascertain. This is because there is little directly available data to inform an analysis of these factors (McInnes, 2009: 30).

Assessing whether there was an increase or decrease in risky sexual behaviour as a result of the conflict is a challenge. The DRC had a generalised epidemic and the conflict began when HIV prevalence had levelled off. This means that risky sexual practices were quite commonplace and there is little evidence to suggest that the conflict might not have changed this. Prevalence rates amongst female sex workers were 38% in Kinshasa as far back as 1989 (Vandepitte et al, 2002:4). The relatively low age of sexual debut, low condom use (overall condom use was 2.3% in 2001 and only 20% of young men used condoms with non-regular partners) with non-regular partners and high incidence of multiple sexual partnerships attest to this (UNGASS, 2005:14-15; WHO/UNAIDS, 2005:12,15).

Despite the evidence being largely anecdotal, the conflict probably did lead to more cases of sexual violence perpetrated primarily on women and children. There are estimates that put the average number of women raped everyday across the DRC during the conflict at 67 (USAID, 2008:1). However, the link between rape and an increase in generalized HIV prevalence is not robust (Anema et al, 2008). Other changes in sexual behaviour that may have occurred include a decrease in the use of commercial sex workers. This would have come about as a result of people losing their purchasing power and more generally, risky sexual behaviour may have been restricted by mobility constraints.

In the final analysis the DRC is likely to have been protected by an upsurge in HIV prevalence by a combination of factors. The low population density, difficulty of cross country movement, poor transport and communication infrastructure are the most likely factors that would have provided such protection. On the other hand, the reports of sexual abuse driven by the ethnic nature of the conflict suggest HIV prevalence should have risen. However, the conflict was not solely inter-ethnic making this a rather weak point (McInnes, 2009:29).

8.8 Sudan

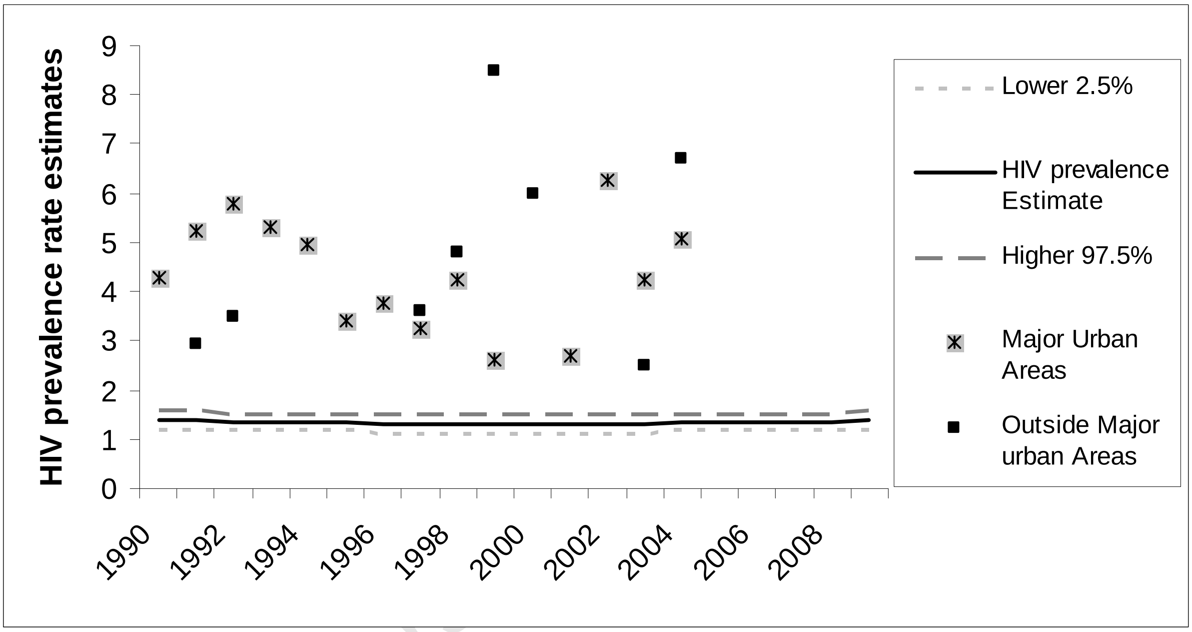
The first official case of HIV was reported in 1987 (UNDP, 2010)[111]. The Sudan is a very large and sparsely populated country and there are marked differences between HIV prevalence in the South and that in the North. The South has a generalized epidemic with a prevalence rate exceeding 1% whereas the North has a low to concentrated epidemic (UNDP, 2010). Despite this fact, overall prevalence rates for the country as a whole have often been reported. For instance, in 2008, UNAIDS estimated an overall prevalence of 1.4 % with 320,000 people living with HIV (UNAIDS, 2008). This is confirmed by Figure 8.13 which shows HIV prevalence estimates as modelled in EPP and Spectrum.

In modelling the HIV prevalence and incidence estimates, the epidemic in Sudan was modelled as a generalised epidemic. In addition, the ANC data was down scaled by a factor of 0.8. This recommended assumption is the reason why there is a big disparity between the trend lines shown in Figure 8.13 and the actual ANC data points. The Coale-Demeny North life table was used as the model life table and the Spectrum default assumptions on the total

[111] http://www.sd.undp.org/story_GFATM2_interview.htm

fertility rate reduction and life expectancy at birth were applied. It was also assumed that in the absence of antiretroviral treatment, people living with HIV will become eligible for treatment an average of 3 years before they are expected to die from an AIDS related cause (UNAIDS Reference Group on Estimates, Modelling and Projections, 2006). Lastly the 2008 UNAIDS Angola epidemiological fact sheet was the main source of the data on ART rollout, PMTCT and child treatment services were used.

Figure 8.13 HIV prevalence in Sudan (1990-2005)



Source: Own EPP and Spectrum modelling

Figure 8.13 shows that HIV prevalence rates at about 2% in 2009 while UNGASS estimates that the disparity between the North and Southern epidemics are expected to continue. While Northern prevalence rates were estimated to be at 0.67% in 2009, the epidemic will gradually become generalised with prevalence rates projected to reach 1.2% in 2015 (while national prevalence rates top 2.2%) (Sudan National Aids Control Program, 2010:7). There are also marked differences in prevalence rates between urban and rural areas as well as men and women. Towns have higher prevalence rates than rural areas and HIV prevalence may be higher in areas that have had greater population mobility (USAID, 2008)[112]. In terms of

[112] http://ghiqc.usaid.gov/tasc3/docs/650-09-313/annex_m.pdf

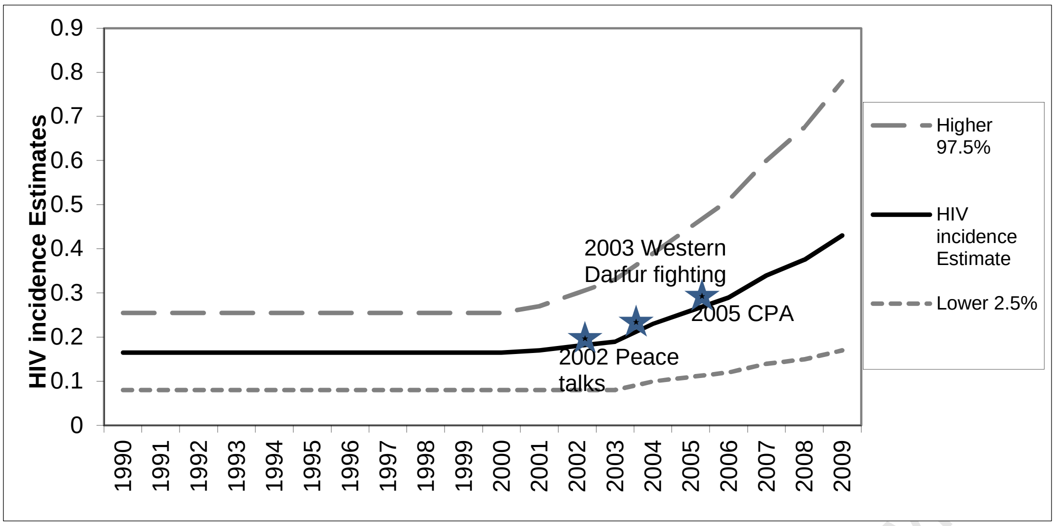
gender differences, women have markedly higher prevalence rates than their male counterparts (USAID, 2008).

8.8.2 Conflict in Sudan

Since independence in 1956, military regimes favouring Islamic-oriented governments have dominated national politics in Sudan. The country has been in a state of civil war for the greater part of the 20th century. The northern economic, political, and social domination of largely Christian non-Muslim, non-Arab southern Sudanese territories was the main cause of both the first war (ended in 1972) and the second. The second war (beginning in 1983) and drought resulted in more than four million people being displaced and, according to rebel estimates, more than two million deaths over a period of two decades. Peace talks gained momentum in 2002-04 with the signing of several accords (see Figure 8.14).

In 2003, a separate conflict broke (see Figure 8.14) out in the western region of Darfur displacing nearly two million people and causing an estimated 200,000 to 400,000 deaths (CIA factbook, 2010). Southern Sudan was granted autonomy for six years after the signing of the final North/South Comprehensive Peace Agreement (CPA) in January 2005 (see Figure 8.14). In the period from 1990 to 2005, Sudan has had a major conflict (more than 1000 battle related deaths) in each year except for 1993, 1994 and 2005 which had minor conflicts. These have primarily been internal contestations over territory (UCDP, 2010).

Figure 8.14 HIV incidence in Sudan (1990-2005)



Source: Own EPP and Spectrum modelling

Figure 8.14 shows that the periods of intense fighting were characterised by low HIV incidence rates. The first signs of an uptick in HIV incidence came after the first peace talks in the period from 2002 and 2004. The 2003 minor conflict did not have much of an impact on the epidemic and upward trend in incidence continued. In fact, the post peace agreement period (after the North/South CPA was signed) has been characterised by a steady increase in HIV incidence.

8.8.3 Background factors

Spiegel et al, (2007) argue that Sudan probably benefited from the protective effects of war against HIV transmission. This is because a number of people lived[113] in remote, hard to access areas and the destruction of the already poor transport infrastructure made it even harder for them to mix with other groups. The low population density in Sudan is another reason why the country was to some extent, protected from an upsurge in HIV prevalence.

[113] Some because they fled to such hard to access areas for security reasons

8.8.4 Effect of conflict

Like in the case of Rwanda, the Sudanese conflict was characterized by cases of rape and sexual abuse perpetrated primarily on women. A 2004 interview carried out by Amnesty International on Chad based refugees from Darfur revealed the extent of these abuses. One female refugee explained;

"I was sleeping when the attack on Disa started. I was taken away by the attackers, they were all in uniforms. They took dozens of other girls and made us walk for three hours. During the day we were beaten and they were telling us: "You, the black women, we will exterminate you, you have no god." At night we were raped several times. The Arabs guarded us with arms and we were not given food for three days." (Amnesty International, 2004)

Other evidence collected by Amnesty International explains the extent of the systematic and unlawful attacks on civilians by the government sponsored “Janjawid” where men are murdered, women are raped and villages are destroyed leaving many families displaced (Amnesty International, 2004). One factor that makes analysing the impact of the sexual abuse on the trajectory of the epidemic particularly hard to explore is that many of the women interviewed by organisations such as Amnesty International refuse to talk about their rapes. The stigma attached to being raped limits the ability of researchers to make estimates of the numbers of women raped and the extent of the sexual abuse. Women, especially those who are married, refuse to talk about rape for fear of being shunned and ostracized by their families and communities (Amnesty International, 2004). This coupled with the lack of infrastructure to test rape victims further complicates any attempt to link the rape and sexual abuse to increases in HIV prevalence in these communities or refugee camps let alone on a national level.

The prolonged period of civil strife in Sudan worsened an already weak transport, communications and health infrastructure. For this reason, the conflict may have had a protective impact on HIV prevalence rates (estimates). However, since the signing of the comprehensive peace agreement (CPA) which as is described in section 8.8.2 was in January 2005, Southern Sudan has seen an increase in population mobility, formation of sexual networks and heightening of exposure to HIV infection (ILO, 2008: Kaiser et al, 2006). The risk of a rise in HIV prevalence rates is also increased by the return of former combatants to civilian life and the arrival of foreign workers for business or aid/relief/charity work (USAID, 2008).

In the final analysis, Sudan (particularly the South) may have been protected from an upsurge in HIV prevalence by a combination of factors including the low population density and the sparseness of the settlements. The direct impact of the conflict on the epidemic is however, unclear.

8.9 Summary and conclusions

In Chapter 5, I discussed the two main theories regarding the link between HIV prevalence and conflict. One school of thought proposes that conflict exacerbates the HIV epidemic (through sex with infected soldiers, war-related rape, poverty-related unsafe sex, transactional sex, etc.). The other argues that conflict protects countries from a major HIV epidemic by limiting mobility, disrupting sexual networks and so forth. In Chapter 7, I found a negative but statistically weak association between different measures of armed conflict, militarization and HIV prevalence. I also find that of all the conflict countries in my analysis, seven had low prevalence generalized epidemics during conflict periods. These are Rwanda, DRC, Angola, Somalia, Ethiopia, Sudan and Liberia. It was these countries which drove the cross-country regression results, so for this reason each case was discussed in detail in this chapter.

The analysis in this chapter took McInne's framework as a guide. It suggests that background factors such as the level of HIV prevalence when the conflict starts, the state of transport and communication infrastructure, population density and the scale and duration of the conflict may have affected the way that conflict affected the trajectory of the epidemic. Each country case is different from the next although all seven case studies share a common characteristic in that where conditions limited mobility or prevented the mixing of different groups, HIV prevalence was unlikely to increase. This is true for sparsely populated Sudan as much as it is true for Ethiopia and Sudan.

Not only are background, structural factors important but the changes that occur during conflict combine with the background factors to determine the extent to which HIV prevalence rises or falls. In the seven cases I present, the most difficult issue to decipher is the extent to which conflict causes changes in risky sexual behaviour. Resultantly, many of my tentative conclusions are based on anecdotal evidence. This is a common challenge for any study on the social impacts of conflict as hard data is often poorly collected if ever.

This chapter adds to the emerging body of literature suggesting that it is not conflict that directly affects the progression of the HIV epidemic but rather context and possible background factors such as the level of HIV prevalence when the conflict starts, the state of transport and communication infrastructure, population density and the scale and duration of the conflict etc. This is important as some countries that would not be described as conflict countries (for e.g. Zimbabwe) had political and socio economic collapses that created conditions akin to those found in conflict countries in terms of the collapse of infrastructure and displacement of people and migration. Resultantly, it would be interesting to investigate the extent to which their (countries like Zimbabwe) socio economic decline, manifesting in the form of a collapse in the state of transport and communication infrastructure, decline in population density (as people emigrate), and decline in health care systems influenced the trajectory of the epidemic. The expectation is that as in the cases of the DRC, Angola, Somalia, Ethiopia, Sudan and Liberia, the spread of HIV would be limited and prevalence rates either drop or increase at a slower pace. However, the problems of collecting data during conflicts make such an investigation difficult to operationalize.

The chapter concludes, rather speculatively, that in all the countries studied (in this chapter), the negative association between conflict and HIV prevalence estimates found in Chapter 7 was likely a result of some of the factors which were not controlled for (or poorly measured) in the regressions. These include but are not limited to the state of health, transport and communication infrastructure, the level of sexual mixing, the impact of rape and sexual abuse and changes in sexual behaviour.

9 CONCLUSION

The aim of this thesis was to investigate the issues and concerns associated with the use of HIV estimates in analysing the progression of the HIV epidemic in conditions of social disruption or outright armed conflict. The main argument of this thesis is that despite them being useful, regressions on country-level HIV prevalence are profoundly compromised by the fact that the HIV data are estimates and not empirical data points. HIV prevalence estimates are a product of demographic modelling in which a scatter of data from antenatal clinic sites is welded together into a national aggregate using a simple modelling package (EPP and Spectrum). Although the antenatal clinic is supplemented by additional data from national HIV surveys to 'calibrate' the model, the estimates are subject to big error margins. Further, some countries, particularly those in conflict situations lack the capacity to carry out and produce national surveys statistics on HIV/AIDS.

Estimates of, say, the effect of civil war, on HIV prevalence are thus outcomes of a model regressed on the outcome of a demographic model which assumes that HIV prevalence is driven by behaviour change, mortality, and migration. Further, the construction of national HIV prevalence estimates assumes that a general epidemic can be constructed out of regional data points, despite the fact that local epidemics often follow unique region-specific paths. This has implications for how HIV estimates should be interpreted and what ought to be drawn from country-level analysis.

A decision should always be made regarding the approach to use when analysing different epidemics as for instance, when a civil war is concentrated in one particular region and we wanted to explore the impact of conflict on the HIV epidemic, then a regionally based methodology should be used. However, the data are often limited and scattered, making this impossible in anything other than a more qualitative set of speculations. In addition, if one looks instead at the national aggregates, it is possible that the dynamics between HIV and other factors will get drowned out in the aggregation, making it difficult to find significant country-level relationships between indicators of conflict, or disruption and HIV trends. It is therefore important to avoid the aggregation of data. Panel data methods mean the risk of aggregation is reduced and adding a dynamic element to the modelling allows for more consistent results. However these results are still constrained by the quality of data and although they help in pointing out the direction in which more micro level analysis should focus, researchers should not present them in ways that could be viewed by some as irresponsible.

9.1 Policy implications

This study by nature only has a few policy implications. It focuses more on how research has/ought to be conducted but some policy lessons do come up along the way. The chapters on Zimbabwe's epidemic (Chapters 2 to 4) reveal a number of points. Firstly, Chapter 2 shows that UNGASS technical working group estimates of HIV prevalence and incidence can be replicated (within a margin of error) based on fairly reasonable assumptions. However, any interpretation of these estimates must fully acknowledge the basic assumptions made in obtaining them and modellers must be appreciative of how sensitive model results are to the assumptions used. For instance, in as much as the decline in HIV prevalence in Zimbabwe was predominantly as a result of a behaviour change (decrease in sexual concurrency, delay of sexual debut), the role of migration has probably been downplayed (although it should not be overstated).

In addition to out-migration, Zimbabwe's socio economic decline had other implications for the epidemic in Zimbabwe. The country's political climate made government sensitive of civil society involvement in intervention programs and this hampered the emergence of a coherent multi-sectoral approach. However, when this was eventually achieved in the late 1990s, HIV prevalence began to fall, as the rate of new infections declined. The inventions including educational campaigns are likely to have been responsible for the decreases in sexual concurrency, increased condom use (self-reported evidence) and delay of sexual debut. The economic decline and implementation of structural adjustment programs rapidly destroyed public health infrastructure and primary health care facilities and this may have led to increased AIDS mortality. This accelerated the decline in HIV prevalence in Zimbabwe. Further, anecdotal evidence suggests that the increase in mortality may have created a “fear factor” where many of those seeing the effects of AIDS on close family and friends changed their sexual behaviour and practices.

The, recent stability in Zimbabwe, the result of the current Government of National Unity presents a challenge. The improvements in income, communication and transport infrastructure could mean that all the conditions that helped accelerate the decline in prevalence during the economic and political slump might be on the mend. This could signal the potential for an uptick in new HIV cases.

A recent Huffington Post article by Peter Navario warns of a possible upsurge in HIV prevalence in Zimbabwe. The report alleges that as Zimbabwe tries to recover from almost a decade of political turmoil, there has been in jump in the percentage of people seeking treatment for sexually transmitted diseases (Navario, 2010). This may be a sign that HIV prevalence will soon be on the rise again after having fallen dramatically from around 27% in 1997 to about 13.7% in 2009 (MOHCW, 2009:7-8). Navario argues that the country is at risk owing to the return of Zimbabweans returning home from other HIV afflicted neighbouring countries like Botswana and South Africa (Navario, 2010). Possible solutions to this include interventions that improve access to health services through the removal of user fees, as well as the recapitalization of preventative programs such as the public and private sector condom distribution programs.

9.1.1 National level policies

Chapters 5, 6, 7 and 8 explored the relationship between HIV and conflict. The key policy conclusion is that this relationship is not only highly contextual but cross country regression analyses that uses HIV prevalence estimates may not be the best methodology to use for establishing an overarching theory on the relationship.

Policy design should therefore not be based on overarching theories on the relationship between HIV and conflict. Rather, policy design should rely more on case by case exploration of the relationship and because of the different character of national epidemics, both quantitative and qualitative methods should be employed. Furthermore, due attention should be put on region specific factors that determine how each country's sub-epidemics evolve over time. Although cross country studies, particularly regressions may be useful at giving insights into differences (between) in the levels of the epidemic, there is a danger that the aggregation might drown out region specific nuances. Researchers should not use population level regressions to draw conclusions on the nature of more micro “proximate” or “biological” determinants.

Care must be taken to focus on understanding the assumptions behind any population level or cross country modelling. Regression results should be used to explore whether statistically significant relationships exist between potential socio-economic determinants (such as conflict) and HIV. Even so, care should also be taken to explore which countries drive the relationship and what contextual factors underpin their experience.

9.2 Avenues for future research

Chapter 7 presented several attempts at understanding the link between HIV conflict using econometric methods. As a result much of the results are preliminary and there is great room for advancement and further research to be carried out on the topic. The fundamental lesson from this study is that regression analyses that use HIV prevalence estimates as the dependant variable are to be interpreted with caution. However, there is potential to explore more studies that look at non-linear methods of modelling the link between HIV prevalence and conflict. OLS regression methods are largely inadequate at capturing the link between HIV prevalence and conflict. This is because non dynamic OLS models fail to capture the fact that HIV prevalence has its own momentum that is determined by other factors not included in the model. However, fixed effects, or simple OLS models with a lagged dependent variable as a regressor (with a dynamic component) produce biased and inconsistent results.

Dynamic model specifications are better suited to modelling the link between HIV prevalence estimates and conflict since they account for the dynamic nature of the epidemic, acknowledging that it has its own momentum driven by other factors not included in the model. However, they too are linear in nature and so are not ideal given that the HIV estimates are obtained from curvilinear approximations (as shown in Chapter 2). There is therefore room for the application of more appropriate, non-linear models.

The linear regression models in this study show that there is a need for researchers to pay careful attention to the countries they include in their sample. The regression in presented here show that the association between HIV prevalence and factors such as the level of militarization of a country and conflict may be negative. This result corresponds to our dataset's summary statistics that suggest this result is driven by countries such as Angola, Liberia, Somalia, and the Democratic Republic of the Congo. Past regression analyses of the link between HIV and conflict were larger than that presented in this study and also did not reveal which countries were included. The sample used in this chapter is restricted only to sub-Saharan Africa (which is the region hardest by HIV in the world) countries with generalised epidemics and this avoids the results being confounded by other countries that do not have HIV prevalence rates high enough to allow for an analysis of the link between conflict and HIV.

10 References

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B.1 Province by province Analysis

Table B2 shows that while Harare and Bulawayo were the most urbanised provinces, Midlands, Manicaland and Masvingo were semi urbanised. Most of the other 6 provinces including Matabeleland North, Matabeleland South, Mashonaland Central, Mashonaland East and West are predominantly rural. These provinces have more people living in communal lands and to a less extent, resettlement areas.

It is unclear to what extent the 2005 "Operation Murambatsvina" altered the population distributions in Table B2. Operation Murambitsvina [drive out filth], discussed in Chapter 3, was an operation by the government where it destroyed people's homes in high density areas and townships in Zimbabwe's main cities and moved them into transit camps, after which they assigned people to rural areas on the basis of their identity numbers.

Operation Murambatsvina further worsened the already serious food shortages. Critics of the government's policy argue that Murambatsvina was meant to reassert control over urban people who have voted overwhelmingly for the opposition in the 2001 and 2005 elections. As suggested by the Catholic Archbishop of Bulawayo, Pius Ncube.

"They *want total political control - they want to peasantify people like [former Cambodian leader] Pol Pot - force them into they country so they can control them.……………………… In the countryside they have no newspaper or radio except Zanu-PF propaganda, and they are controlled by the chiefs, who support the government …*,"

Table B.1 Provincial split by settlement type (1992 and 2002)

Province		Communal lands	Small-scale commercial farms	Large-scale commercial farms	Resett-lement areas	Urban council areas	Other urban areas	Total Urban	Total Population
Manicaland	92	991	33	202	126	160	16	177	1,537
	02	975	21	146	167	216	18	234	1,569
Mashonaland Central	92	503	12	229	33	21	55	76	857
	02	616	14	189	73	47	48	94	995
Mashonaland East	92	709	27	170	51	41	15	54	1,034
	02	752	31	143	83	84	8	92	1,127
Mashonaland West	92	398	23	335	70	172	100	271	1,113
	02	429	20	276	154	265	69	334	1,225
Matabeleland North	92	471	26	20	17	59	22	81	641
	02	524	13	30	35	67	14	82	705
Matabeleland South	92	466	8	49	16	28	20	48	592
	02	499	8	32	45	46	21	67	653
Midlands	92	865	13	70	56	255	40	295	1,308
	02	948	12	73	81	326	20	346	1,464
Masvingo	92	948	27	80	58	78	18	96	1,223
	02	919	29	78	161	95	19	115	1,320
Harare	92	--	1	21	--	1,464	--	1,464	1,486
	02	--	1	21	--	1,873	--	1,873	1,896
Bulawayo	92	--	--	--	--	622	--	622	622
	02	--	--	--	--	677	--	677	677
Total	92	5,352	170	1,177	427	2,901	284	3,185	10,413
	02	5,661	150	987	800	3,696	218	3,914	11,632
%	92	51.4	1.6	11.3	4.1	27.9	2.7	30.6	100.0
	02	48.7	1.3	8.5	6.9	31.8	1.9	33.6	100.0

Source: (CSO) Census, (2002) and Census, (1992)

Table B.2 Population density (2002) and average earnings by province (1993)

Province	Capital	Pop Density	Avg Earnings
Bulawayo	Bulawayo	1,413	1,815
Harare	Harare	2,183	5,051
Manicaland	Mutare	43	387
Mashonaland Central	Bindura	35	--
Mashonaland East	Marondera	35	106
Mashonaland West	Chinhoyi	21	95
Masvingo	Masvingo	23	190
Matabeleland North	Lupane	9	--
Matabeleland South	Gwanda	12	--
Midlands	Gweru	30	360

Source: (CSO) Census, (2002); (CSO) Quarterly Digest of Statistics (1993)

Overall, Harare and Bulawayo had the highest population densities. This is shown in Table B3. Further, the final column in the same table shows that Harare and Bulawayo also had higher average earnings than other provinces. This leads one suspect that the pattern in population density above is as a result of people moving from lower earning provinces to Harare and Bulawayo. Without making extraordinary leaps in reasoning, the fact that the earnings figures shown above are 1993 numbers, leads one to suspect they may have been a factor in determining the 2002 census based numbers on population density.

Table B.3 Average Earnings (1993)

	Industry	Amount
Average salary in 1993	Overall	1,020
Highest paid industry	Manufacturing	3,017
Lowest paid industry	Electricity and water	157
2nd Lowest paid industry	Private domestic workers	191
Rural	Agric, forestry, fishing	948

Source: (CSO) Quarterly Digest of Statistics (1993)

The average statistics shown above highlight that the highest paying industry in 1993 was the manufacturing industry. These industries were/are centred in Harare and Bulawayo, and to a less extent, Midlands province (Gweru). Harare and Bulawayo provinces had average salaries much larger than the average earnings in all other industries save for the manufacturing industry (Harare's average was higher than the manufacturing industry average earnings). Average rural/agricultural industry earnings (948) were much lower than the overall average earnings (1,020). Many of the provinces in Table B.2 which had very low average earnings such as Mashonaland Central, Matabeleland North (hence the blanks signalling very low sample numbers), Mashonaland West etc. are composed primarily of subsistence farmers.

Table B.4 Internal migration

Province	Census Population 2002	Population/ ever born in province	As % of population in 2002			
			Born and residing in province	Born elsewhere	Born in province, residing elsewhere	Net gain lifetime migration
Harare	1,820,675	128	48	52	-31	22
Bulawayo	664,973	123	49	51	-32	19
Manicaland	1,602,327	89	85	15	-27	-12
Mashonaland Central	996,694	102	76	24	-22	2
Mashonaland East	1,135,201	93	70	30	-38	-8
Mashonaland West	1,214,036	107	70	30	-24	6
Matabeleland North	704,540	99	80	20	-21	-1
Matabeleland South	639,721	92	81	19	-27	-8
Midlands	1,476,644	99	75	25	-25	-1
Masvingo	1,360,825	84	86	14	-33	-19

Source: (CSO) Census, (2002)

Table B5 shows evidence that Harare and Bulawayo are provinces that are comprised primarily by people who had been born elsewhere. This confirms that these provinces are the most popular destinations for those leaving other provinces such as Matabeleland North, Mashonaland East etc, in search for opportunities in the two provinces hosting the country’s capital cities.