Gender, poverty and intimate partner violence in Southern Africa

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Abstract

In many parts of sub-Saharan Africa, and especially in southern Africa, HIV prevalence remains high despite a raft of interventions to curb its spread and persistence. A number of theories and explanations exist as to why this is the case, and among them are theories about how gender inequality, poverty and intimate partner violence link with HIV. This dissertation critiques the hypothesis that the disadvantages experienced by women in terms of income, political representation etc. renders them more vulnerable to HIV infection. Using literature reviews and quantitative research methods applied to Demographic and Household Survey data from Lesotho, Swaziland and Zimbabwe, we argue that this relationship varies from country to country and contest the proposition that either structural factors or individual factors mainly affect HIV.

It has become a conventional wisdom to hold that women in Southern Africa, by virtue of their disadvantaged social and economic position, are more vulnerable to HIV infection. But there is also a large body of work suggesting that individual sexual behaviour and bio-medical factors such as male circumcision shape the HIV terrain in sub-Saharan Africa and southern Africa. Chapters 2 and 3 review the literature on what drives the HIV epidemic. These chapters contest the posited monotonic relationship between higher HIV in countries with greater gender inequality. We argue that this relationship varies from country to country. We also contest the proposition that either structural factors or individual factors mainly affect HIV. Instead, we argue that this probably depends more on the underlying structural issues in different countries and their socio-economic backgrounds.

Chapter 4 explores the interplay between structural factors (notably socio-economic status (SES)), behavioural factors and HIV-status in southern Africa. The chapter presents multiple logistic regressions with HIV-status as the dependent variable, and various correlates of HIV

as the independent variables using Demographic and Health Survey data (DHS) from Lesotho, Swaziland and Zimbabwe. The purpose of this chapter is to answer two main questions. The first is whether structural factors affect individual HIV risk more than behavioural factors in the sub-region. The second pertains to whether these effects vary by gender. The main findings of the chapter, in accordance to our hypothesis, are that the effect of structural factors on individual level HIV risk is determined by country-specific factors. In Lesotho, a country with significant structural health problems, structural factors of household wealth, education, place of residence and employment status are the strongest predictors of HIV risk. In Zimbabwe, a relatively wealthier country[1], these variables had less of an influence, and instead, behavioural variables such as the number of lifetime partners, and age of marriage predicted higher HIV odds. Secondly, among women, there was no universal relationship between specific correlates of HIV across the three countries. Measures such as poverty predicted increased HIV risk for women in all the three countries, but to varied extents. For example, while for all three countries those that were just 'poor' had higher HIV rates than the 'poorest', in Lesotho, this increased risk was nearly twice as large as that found in Swaziland and Zimbabwe.

Chapter 5 looks at the relationship between gender inequality and HIV prevalence in sub-Saharan Africa. The chapter models HIV prevalence using cross-country data and explores the relationship between HIV prevalence and a wide array of gender inequality measures such as the proportion of women in parliament and proportion of women in secondary school relative to men. The chapter concludes that while it is clear that HIV prevalence is a function of some structural underlying factors unique to sub-Saharan Africa and Southern Africa, it is not clear that gender inequality is one of them.

[1] On page 21, the graph shows Zimbabwe to be poorer. This information about Zimbabwe is based on 2005 data, which is more relevant to the conclusion because it relates to the time around which the HIV data was collected. The graph on page 21 that shows Zimbabwe to be poorer is based on 2009 data.

Chapters 6 and 7 focus on intimate partner violence (IPV) and its links with HIV. The starting point of chapter 6 is that some of the research that argues that IPV leads to greater HIV risk also acknowledges that men who perpetrate IPV are likely to be more sexually risky than those that do not. Thus, if the relationship between IPV and HIV risk is positive and strong, this could be the result of having a sexually risky partner rather than the IPV itself. To elucidate this issue, as well as explore whether among southern African women IPV leads to HIV risk, we run multiple models to investigate whether men who perpetrate IPV are sexually risky and whether IPV predicts higher HIV odds among women. The main findings in this section are that men who perpetrate IPV in the sample do not engage in any riskier behaviour than those that do not. However, women who experience IPV show evidence of riskier sex (extramarital sex). Among women, IPV was associated with a 16 percent higher HIV risk. Although some of this additional risk was a result of the victims' own sexual behaviour, the magnitude of this effect was not strong and it only accounted for about a fifth of the overall incremental risk.

The dissertation concludes that what affects HIV varies from place to place and that any assumed unidirectional relationship between gender, inequality and abuse and HIV is probably unwarranted. Aligning HIV prevention objectives with gender equality objectives is not necessarily the best way to prevent HIV among women and there is need for more detailed studies that investigate the specific pathways through which IPV is associated with HIV.

Chapter 1. Introduction

1.1 Background

In sub-Saharan Africa, mortality from HIV among women far exceeds that from any other disease, infectious or otherwise. Data from 2009 shows that in that year alone, there were an estimated 1.3 million AIDS-related deaths in sub-Saharan Africa, with more than half these deaths occurring among women (UNAIDS, 2010:20). The effects of HIV on women are not restricted to mortality, they also include stigma, discrimination and violence (UNAIDS, 2009:10, UNAIDS, 2010:14) and these encounters all lead to undesirable life experiences, and sometimes death.

Research on the determinants of health shows that health is not only determined by biological processes, but is also influenced by social factors such as education, income, social connectedness and social capital, among others (e.g. Marmot & Wilkinson, 1999; Noone, 2009; Raphael, 2004; Wilkinson & Marmot, 2003). In many parts of the world, there is unequal access to these social resources and amenities, with women generally having less or restricted access than men. This unequal access, along gender lines, is a form of gender inequality. To the extent that health outcomes are affected by social factors that are in turn influenced by gender inequities, gender inequality is a determinant of health among women.

Given that African women bear the brunt of the HIV epidemic and are socially and economically disadvantaged relative to men, it is unsurprising that a substantial body of literature flags gender inequality as a root driver of HIV (e.g. Anderson, Cockroft & Shea, 2008; Campbell, 2000; Dowset, 2003; Maman, Campbell, Sweat et al, 2000; Outwater, Abrahams & Campbell 2005; UNAIDS, 2007; 2010; Wojcicki & Malala, 2001). The proposed causal pathways are varied and sometimes unclear, but the overarching reasoning

within this literature is that gender inequality restricts access to various social resources, amenities and privileges and that this hamstrings women in their efforts to protect themselves from, or seek treatment for, HIV. This literature cited above suggests a blanket, positive relationship between gender inequality and HIV: in countries where gender inequality is high, the most disadvantaged women are on average, at an individual level, exposed to higher HIV risk than men are, and subsequently have higher HIV prevalence and mortality.

Although national level HIV incidence data is generally not available, the HIV prevalence rate among young women (15-24) is considered a proxy for HIV incidence because sexual activity in this age group is expected to be recent and mortality from HIV is low (Mahy et al., 2010:1; UNAIDS 2010b:7). In this age group, HIV prevalence in Southern Africa is 3-6 times higher for young women than for young men (UNAIDS, 2010). Some literature makes a connection between gender and this disproportionate nature of the southern African epidemic (e.g. Gupta, 2000, 2002, 2006; Jewkes, Levin & Kekana, 2003; Kim, Proynk & Watts, 2008). This genre of literature points to gender inequality as one of the main determinants of the high HIV prevalence rates in Southern African countries – that higher HIV prevalence rates among women translate to a higher national HIV prevalence in these countries.

While biology or genetic make-up determines the 'sex' of an individual (which is generally either male or female and does not change), the distinct and acceptable roles and expectations of men and women determine 'gender'. Giving birth, for example, is a biological function, but the imperative or expectation to bear children, the number of children to bear and the division of parenting roles are all determined by gender (and this in turn determined by society). Gender is a social construct that embodies cultural values and entrenches social hierarchies that evolve through time, shaping and reshaping gender differences. It relates to the "*socially determined ideas and practices of what it is to be male or female*" (Reeves &

Baden, 2000:2) or "*the roles and expectations attributed to men and women in a given society, roles which change over time, place and life stage*" (Phillips, 2005:1).

It then follows from reason that, if gender roles (and necessarily gender inequality) vary from place to place, so should the relationship between gender inequality and any particular disease. Depending on the specific gender norms of a particular society, whether or not gender inequality positively or negatively affects disease incidence/prevalence should depend on the nature and extent of inequality as well as the specific characteristics of the disease in question (such as method and likelihood of transmission). If for example in one society women's sexuality is more tightly controlled than men's, it is not obvious why women in this society should have worse HIV outcomes than those from a different, more gender-equal society. It could well be that in the more equal society where women are free to have many sexual partners in different places, female HIV rates are higher than in the more repressive society. However, if men in these societies engage in risky sex with commercial sex-workers, then it is possible that a sex-worker/client epidemic could start which then gets transmitted to married women. Such context-specific dynamics are inadequately explored in the literature and yet they could prove to be vital in the determination of the HIV risk factors in southern Africa and the design of HIV interventions.

This dissertation argues that one is unlikely to find a consistent relationship within and across countries between HIV-status and gender-related factors because this relationship is likely to be mediated by country-level factors and society-specific factors: in some countries it could be positive, in others, negative or indeterminate. We further argue that the current literature on gender and HIV is inadequate as it does not provide a nuanced understanding of the association between gender inequality and HIV nor does it satisfactorily grapple with the possible relationship (which could be negligible) between gender inequality and the southern African epidemic.

The dissertation reviews the literature on gender and HIV in southern Africa and investigates various hypotheses as to how gender inequality and its associated themes of partner violence and discrimination fit into the broader relationship between HIV and its various potential determinants such as poverty and individual sexual behaviour. We endeavour to plug some of the gaps in the literature by drawing from a wealth of relevant knowledge and applying robust statistical tools to tease out important and yet unexplored underlying dynamics between HIV and gender inequality in southern Africa.

1.2 Structure of the dissertation

This dissertation comprises 8 chapters. Chapters 2 and 3 review the current research on the potential determinants of HIV in sub-Saharan Africa. We discuss these under two broad categories: structural determinants of HIV and behavioural determinants of HIV – and point to various short-comings in the literature.

Chapter 4 explores the relationship between socioeconomic status (SES) and HIV-status in southern Africa. The chapter uses nationally representative Demographic and Health Survey (DHS) data from Lesotho, Swaziland and Zimbabwe to explore whether socioeconomic status is linked to higher odds of being HIV positive, and whether this relationship differs between men and women. We also discuss the topical issue of whether structural factors (such as poverty, and income) matter at all in shaping the African HIV epidemic or whether HIV is driven largely by sexual behaviour among individuals.

Chapter 5 looks at the relationship between gender inequality and HIV prevalence in sub-Saharan Africa. The chapter models HIV prevalence using cross-country data and explores the relationship between HIV prevalence and a wide array of gender inequality measures. In this chapter, we firstly review all the econometric literature that uses cross-country multivariate analysis to model HIV prevalence. We then go on to model HIV prevalence and

examine whether Southern African countries have higher prevalence rates possibly because they have more gender inequality.

In chapters 6 and 7, we investigate the significance of intimate partner violence (IPV) as a determinant of HIV status among women in Zimbabwe. We probe two different, but complimentary issues surrounding IPV. The first concerns its character and reach. Here, we look at the prevalence of IPV in southern Africa as well as the motives and attitudes towards IPV among men and women. This is done through extensive searches and analyses of the relevant literature from Southern Africa. Secondly, we explore whether there is a statistically significant relationship between IPV and HIV-status in southern Africa through multivariate statistical analyses of nationally representative data from the Zimbabwe DHS survey. (There are notable data constraints that make it impossible to thoroughly probe the relationship between IPV and HIV in Lesotho and Swaziland. In the analysis, not only do we look at whether IPV affects HIV risk, but also probe the sexual behaviour of men who are likely to perpetrate IPV. Chapter 8 concludes the discussion.

Chapter 2. Women, poverty and HIV in sub-Saharan Africa

2.1 Introduction

Southern African countries bear a disproportionate share of the global HIV burden. Data from nationally representative surveys shows that in 2009, the nine countries with the highest national adult (15-49) HIV prevalence rates in the world were all in Southern Africa (UNAIDS, 2009: 27). Figure 2.1 below shows the regional distribution of HIV prevalence in Africa from the 2010 UNAIDS epidemiological update (2009 estimates).

The lowest adult (15-49) HIV prevalence rates in Africa are in North Africa. As Figure 2.1 shows, HIV prevalence in this region less than 0.1 per cent (UNAIDS, 2010:197). In sub-Saharan Africa, adult HIV prevalence rates are much higher than in North Africa. West Africa, with the lowest adult HIV prevalence rates south of the Sahara, has prevalence rates between 1 and 5 per cent. This is followed East Africa, with prevalence rates hovering between 3 and 6 per cent. The highest HIV prevalence rates in Africa are in southern Africa, where adult HIV prevalence ranges from 12 to 26 per cent.

Figure 2.1: Regional distribution of HIV in Africa[2] [3]

Source: UNAIDS epidemiological update, 2010 (2009 data). Annexure 1, pg. 181

In Southern Africa, women, especially young women between the ages of 15 and 24, bear a much larger share of the HIV burden. In Swaziland for example, approximately 31 per cent

[2] ? = missing data

[3] Figure does not intend to show data for every single country in Africa. The main objective is to pictorially show that there are notable regional differences in HIV prevalence across African regions.

of all the women who took HIV tests in 2007 tested positive, as compared to only 20 per cent of adult men (Mishra & Assche, 2009). In Lesotho, it is estimated that approximately 57 per cent of new HIV infections occur amongst women (UNAIDS, 2009). In the sub-region as a whole, up to 67 per cent of new infections in 2008 occurred to women (UNAIDS, 2008). Table 2.1 below shows the top 10 countries in the world ranked by adult national HIV prevalence as well as the corresponding burden borne by young women compared to similarly aged men.

Table 2:1: Adult (15-49) HIV prevalence and women's, young women's and young men's share of the HIV burden (2009 data)

Country	**Adult HIV Prevalence (15-49)**	**Women's proportion of people living with HIV**	**Young women's (15-24) proportion of people living with HIV**	**Young men's (15-24) proportion of people living with HIV**
Swaziland	25.9%	58%	15.6%	6.5%
Botswana	24.8%	57%	11.8%	5.2%
Lesotho	23.6%	62%	14.2%	5.4%
South Africa	17.8%	62%	13.6%	4.5%
Zimbabwe	14.3%	62%	6.9%	3.3%
Zambia	13.5%	57%	8.9%	4.2%
Namibia	13.1%	59%	5.8%	2.3%
Malawi	12.7%	59%	6.8%	3.1%
Mozambique	11.5%	63%	8.6%	3.1%
Uganda	6.5%	61%	4.8%	2.3%

Source: UNAIDS (2010) (2009 data)

Swaziland has the biggest HIV epidemic in the world with nearly 26 per cent of the adult population between the ages of 15 and 49 estimated to be HIV positive. In the sub-region as a

whole, women make up between 57 (Zambia and Botswana) and 63 per cent (Mozambique) of the HIV-positive population. The HIV burden on young women is higher than that borne by young men for all the countries in the region and in some cases, there is up to a threefold difference in the respective proportions infected by HIV. Uganda, the only country outside southern Africa on the list, serves an illustration of the severity of the southern African epidemic – its adult national prevalence is nearly half that of Mozambique, the southern African country ranked directly above it.

Table 2.1 and figure 2.1 underscore two facts about the HIV epidemic – the epidemic is disproportionately large in southern Africa compared to the rest of the continent and women in southern Africa bear a much larger share of this burden. To some researchers, this clustering of the highest HIV prevalence rates in one sub-region is evidence that 'structural drivers' must be shaping the HIV epidemic in Southern Africa (see e.g. Gupta, 2008:2). There is a sizeable body of literature that argues that in these countries, structural factors in the form of social, economic, political and cultural aspects of the environment, reduce the ability of individuals (especially women) in the countries to protect themselves from HIV (e.g. Blackenship, Bray & Merson, 2000; Gupta, Parkhurst, Ogden et al., 2008; Harrison, Newell, Imrie et al., 2010; Hatcher, de Wet, Bonell et al, 2010; Parker, Easton & Klein, 2000; Piot, Bartos, Larson et al., 2008; Sumartojo, 2000; Stillwaggon, 2006; Sawers & Stillwaggon, 2010).

Proponents of this point of view reason that in order to fight HIV effectively, intervention programs should focus on addressing these structural aspects of the environment, which include poverty, gender inequality, stigma and governance inadequacies. Their general argument is that changing the course of the HIV epidemic is only achievable through changing the conditions in which people live, as it is such conditions that primarily determine whether individuals are exposed, or become vulnerable to infection in the first place. These

authors advocate for 'structural' approaches to HIV prevention. Such approaches, rather than focusing on individual behaviour change, aim to, as examples, improve access to health and alleviate poverty (Stillwaggon, 2006; Sawers & Stillwaggon, 2010), reduce wealth inequality (Kim, Proynk and Watts, 2008) and eliminate gender inequality (Gupta, 2008; Coovadia & Hadingham, 2005).

Some authors suggest that poverty alleviation, for example, would improve diet and subsequently health, and this is likely to have a protective effect against HIV infection and other opportunistic infections (Sawers and Stillwaggon, 2010; Stillwaggon, 2006). It would also, potentially, reduce the need for women to engage in a wider range of coping activities (such as transactional sex) which are linked to higher HIV vulnerability (Gilbert & Walker, 2002; Gilbert, 1996; WHO, 2000). There is some basis for such arguments. For example, research has also shown that an early sexual debut among women from households that have experienced an income shock either through loss of job or death is common (Dinkelman, Lam & Leibbrandt, 2005). The hope is thus that interventions that minimise or alleviate the impact of such shocks may lower sexually risky behaviour and mitigate HIV risk among families.

Gender equality supposedly offers a wide range of benefits militating against HIV risk for women. Some argue that it would enable women to insist on condom use, refuse unsafe sex and desist from being in multi-partner sexual relationships (e.g. Gupta, 2000, Jewkes, Levin and Kekana, 2003). To others, gender equality would improve their income generating potential, leading to less dependency on men for financial resources (Gillespie, Kadiyala & Greener, 2007; Piot, Greener & Russell, 2007). Studies have shown that financial dependency on men can predict worse HIV and general health outcomes for women (Piot, Greener & Russell, 2007; Gillespie et al., 2007). According to some of the literature above, gender

equity would potentially reduce HIV prevalence among women and reduce the share of the HIV burden they bear in Southern Africa.

Implicit in the structural approach towards reducing HIV infections is the assumption that simple behaviour change interventions will not work, and that this is a key reason for the disappointing performance of conventional HIV prevention programs. However, Piot and colleagues argue that HIV/AIDS research over the past 25 years has generated enough knowledge about the transmission and prevention dynamics of HIV but essential programmes and services have not had sufficient coverage, follow up or funding (Piot et al, 2008). The authors accept that there are 'structural drivers' but focus on the failure to influence individuals (for example educating young people forthrightly about sex), arguing that the "*the tragic reality is that we have not provided a clear focus to ensure that all young people have the information they need before and while they are engaging in sex, especially in light of the high infection rates in young people, especially girls, and the early ages of sexual initiation — in many countries, 14–15 years is the median age of first sexual intercourse*" (Piot et al., 2008:5).

Other recommendations pertaining to the direction of HIV prevention methods noted in the above research include dealing "rationally" with drug abuse, eliminating mother to child transmission, creating strong, consistent and focused political leadership, reaching all those in need of help and treating HIV in hyper-endemic conditions as a full-scale emergency (Piot et al., 2008:8). Piot and colleagues reason that political leadership in countries such as Thailand, Uganda and Brazil in the 1990s was enlightened and these countries experienced a notable change in HIV infections patterns compared to other countries without such focused leadership (see Nattrass, 2008b also). Piot et al suggest then, that the main problem with fighting HIV rests with the implementation and monitoring of prevention programmes, and

that enough knowledge has already been generated through research to be able to effect better HIV prevention programs.

It is crucial to clarify at this point that even where the literature acknowledges that structural drivers may be important, there is no agreement as to which structural drivers actually matter or matter the most. What this literature has in common is the underlying argument that the conditions in which people live in are so influential that they shape and drive HIV outcomes for both men and women but more negatively for women. At the extreme end of this argument is the claim that structural factors are so powerful that they render individual level dynamics such as sexual behaviour choice impossible and thus unimportant in determining HIV infection levels.

If structural factors indeed dictate – or at least profoundly shape HIV risk, the implication of this 'structural-drivers-matter' argument is that a discernable pattern or HIV gradient between countries that have commensurate structural problems (relevant to HIV prevention), should be observable. If individual poverty is a key determinant of national level HIV prevalence, then countries with higher proportions of poor people can be expected to have higher HIV prevalence. If education is crucial, then some relationship between measures of education and HIV prevalence should be expected among countries. If institutional factors, notably with regard to governance, are regarded as key structural factors shaping HIV risk, then the strength of social and governance institutions among countries, likewise, should have a bearing on HIV prevalence levels

Data from sub-Saharan Africa however suggests that such simple gradients are not evident As Figure 2.2 shows, Southern Africa actually has some of the wealthiest African countries

(using per capita gross national income (GNI)) and yet these countries[4] have the highest HIV prevalence rates.

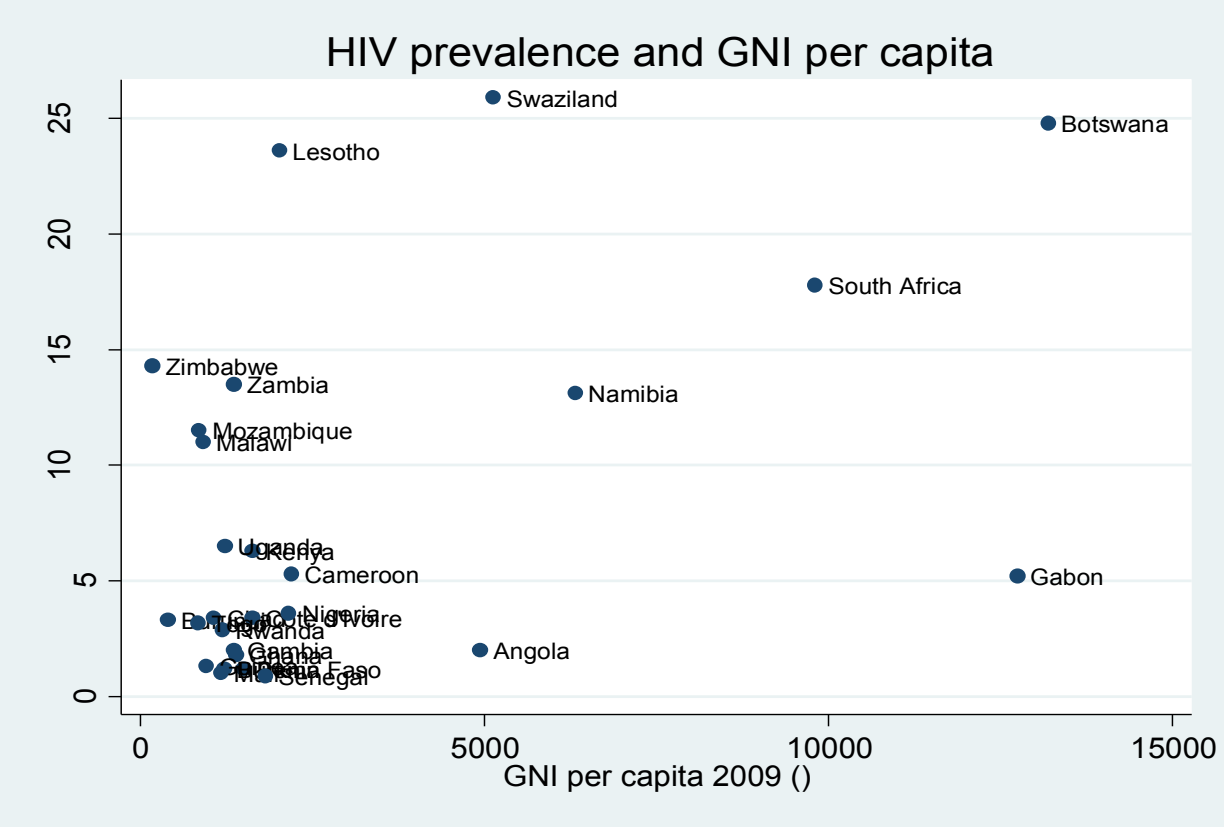

Figure 2.2: Adult (15-49) HIV prevalence and GNI per capita ($US) in sub-Saharan Africa. Source: hdr.undp.org/en/statistics and UNAIDS (2010)

As figure 2.2 shows, there is no clear relationship between HIV prevalence and income, at least not at a cross-country level among sub-Saharan African countries. The graph suggests a positive relationship between gross national income (GNI) and HIV in Southern Africa, but no clear relationship among other sub-Saharan African countries. Among those countries in southern Africa, there appears to be two different patterns of this relationship, with

[4] Angola, Benin Botswana, Burkina Faso, Burundi, Cameroon, Cape Verde, Chad, Côte d'Ivoire, Gabon, Ghana, Guinea, Kenya, Lesotho, Malawi, Mali, Mozambique, Namibia, Nigeria, Rwanda, Senegal, South Africa, Swaziland, Togo, Uganda, Zambia and Zimbabwe

Zimbabwe, Lesotho and Swaziland seemingly exhibiting a different HIV/GNI relationship to Namibia, South Africa and Botswana.

Across sub-Saharan African countries, no discernable relationship between education (measured by the literacy rate) and HIV prevalence is apparent either.

Figure 2.3 below shows the relationship between the literacy rate and adult HIV prevalence in sub-Saharan Africa[5].

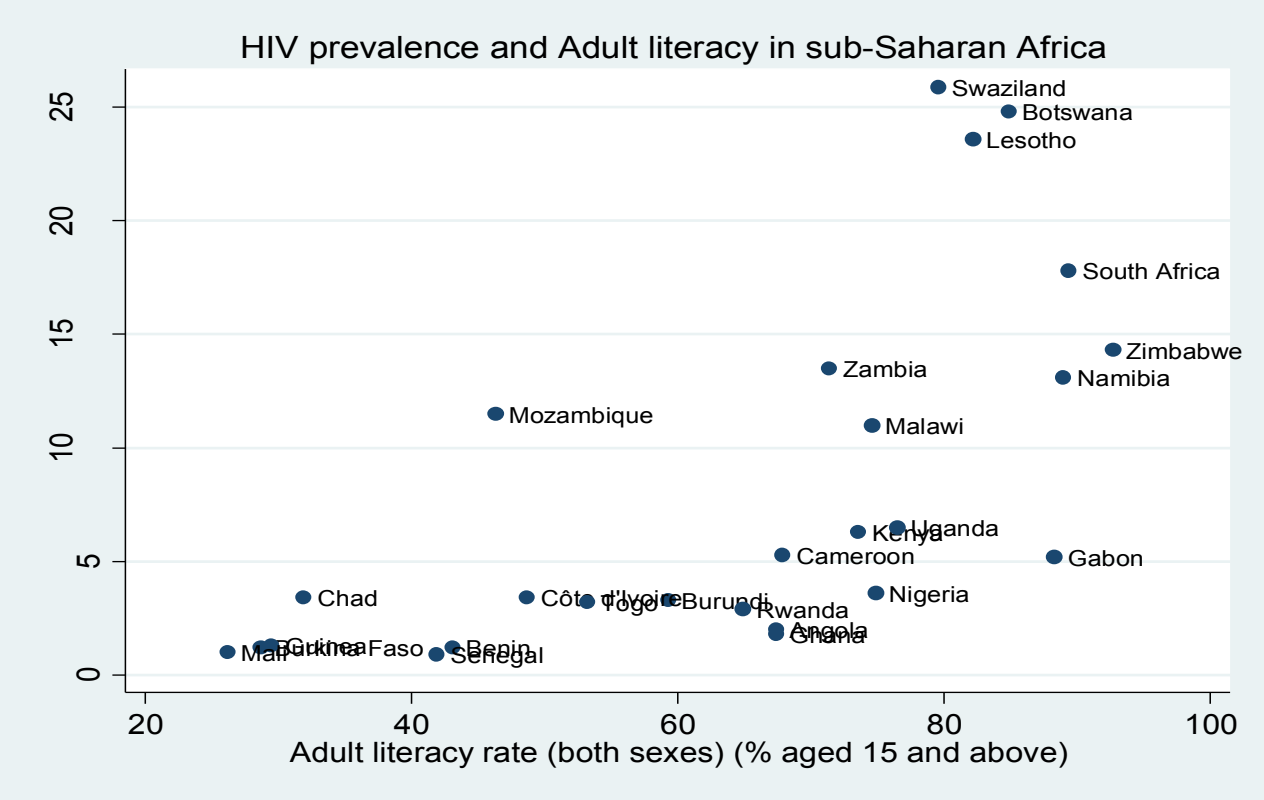

Figure 2.3: Adult (15-49) HIV prevalence and adult literacy rate (2008)

Source: UNAIDS (2010) and hdr.undp.org/en/statistics

[5] Angola, Benin Botswana, Burkina Faso, Burundi, Cameroon, Chad, Côte d'Ivoire, Gabon, Ghana, Guinea, Kenya, Lesotho, Malawi, Mali, Mozambique, Namibia, Nigeria, Rwanda, Senegal, South Africa, Swaziland, Togo, Uganda, Zambia and Zimbabwe

With the exception of Mozambique, which has an adult literacy rate of less than 50 per cent, southern African countries typically have adult literacy rates in excess of 70 per cent. They coincidentally report, however, much higher HIV prevalence rates of above 10 per cent of the adult population, on average – although this relationship is not systematic. Depending on the sample selected, one could fit a negative relationship between adult HIV prevalence and the adult literacy rate (e.g. if the sample is restricted to South Africa, Botswana, Lesotho, Swaziland, Namibia and Zimbabwe). But one could easily also find positive relationship if this sample changes to Mozambique, Zambia, Malawi, Namibia and Zimbabwe.

The latest gender inequality data from the human development report (HDR) does not show a close relationship between national HIV prevalence and gender inequality either. The HDR compiles a gender inequality index that tries to capture the level of inequality by looking at three dimensions of women's overall well-being – reproductive health, empowerment and labour market participation. Reproductive health is measured by two variables, adolescent fertility rate and maternal mortality. Empowerment is measured by educational attainment (percentage of women with secondary education and above) and parliamentary representation, while labour market participation is measured by the labour force participation rate of women. Using these five variables, a single index, ranked from zero to one, with zero representing no gender differences and one representing absolute gender inequality is computed. Figure 2.4 below shows the relationship between HIV prevalence and the gender inequality index for 22[6] sub-Saharan African countries for which data exists. Countries with lower gender inequality scores, such as Botswana, South Africa and Namibia are among those with the highest HIV prevalence rates.

[6] Benin, Botswana, Burundi, Cameroon, Côte d'Ivoire, Gabon, Gambia, Ghana, Kenya, Lesotho, Malawi, Mali, Mozambique, Namibia, Rwanda, Senegal, South Africa, Swaziland, Togo, Uganda, Zambia and Zimbabwe.

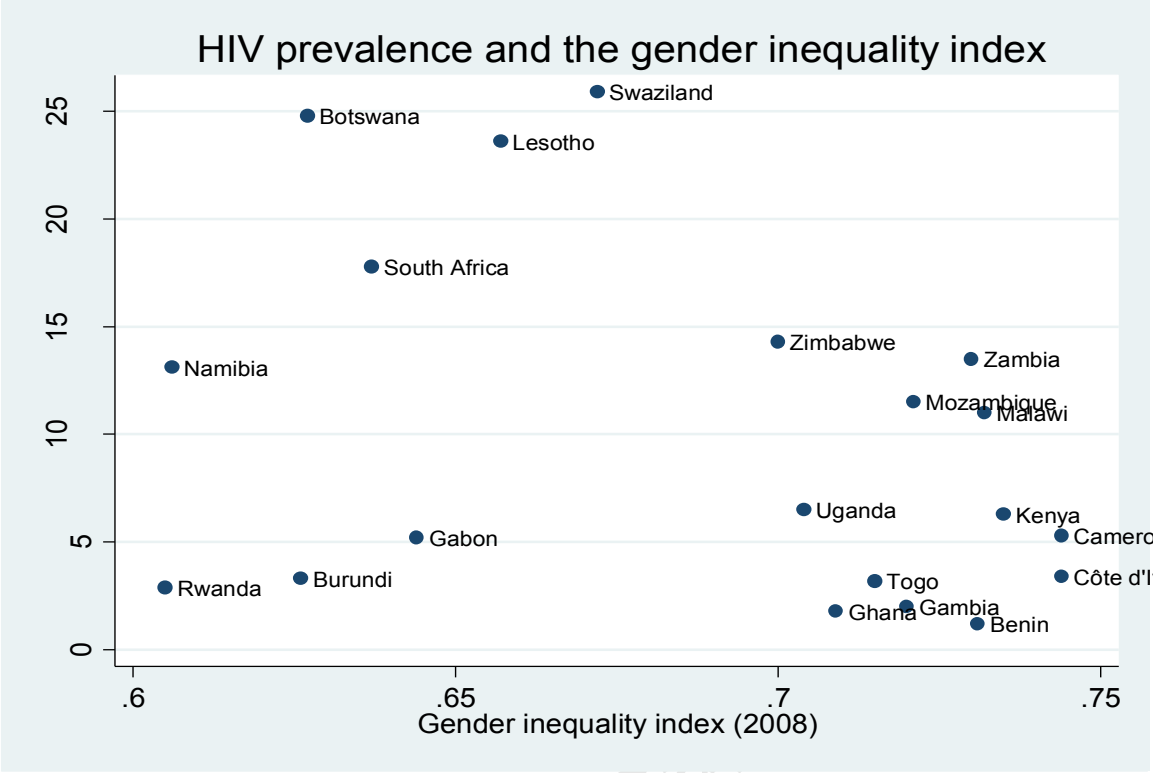

Figure 2.4: Adult (15-49) HIV prevalence (2009) and gender inequality index (2008) in sub-Saharan Africa

Compared to the rest of sub-Saharan Africa, there seems to be a positive relationship between gender inequality and HIV prevalence in Southern Africa. Countries with higher gender inequality scores appear to have low prevalence rates while the countries from southern Africa have lower gender inequality but higher HIV prevalence. However, the relationship between the gender inequality index and HIV prevalence is weak and far from convincing.

These graphical representations of the relationships between national HIV prevalence and education, gender inequality and income are very simplified. For one, they only show univariate correlations between the specific measure and HIV prevalence, thus ignoring interactions between HIV prevalence and other factors. They also ignore unique cultural factors that may influence HIV. Simple as they may be however, they raise doubts about any

simple acclaimed relationship between HIV and structural factors. Are structural factors that important after all? If they are, how strong, and how does this relationship differ within and across countries? Given the aggregate sketches provided in Figures 2.2 to 2.4 it is unsurprising that there is a counter argument that structural factors do not matter (or at least are of secondary importance) in driving the HIV epidemic in southern Africa.

While alluding to the fact that structural issues such as gender inequality, poverty and access to education are important millennium developmental goals that need addressing, some literature argues that structural drivers do not, in fact, hold sway over HIV (e.g. Auvert et al, 2001; Boerma et al., 2003; Chan 2005; Mah & Halperin 2010; Potts, Halperin, Kirby et al 2008; Shelton, 2007). This large body of research argues that African HIV epidemics are instead driven by biomedical and behavioural determinants that include circumcision (Halperin & Epstein, 2007; Klausner, Wamai, Bowa et al., 2008; Weiss, Halperin, Bailey et al., 2007), sexual concurrency (Shelton, 2006; Halperin & Epstein, 2007; Stoneburner & Low-Beer, 2004) and condom use (Hearst & Chen, 2004; Shelton et al., 2004).

In similar fashion with the research cited earlier in support of the 'structural drivers matter' argument, this second genre of literature does not agree about which individual factors are the most important in shaping HIV risk in sub-Saharan Africa. What they all have in common though is the contention that it is individual-level factors that affect African HIV epidemics more than any contextual factors and, intervention should thus be focused on individual behaviour such as getting more men circumcised, reducing the number of concurrent partnerships and promoting more consistent use of condoms.

Some of the literature shows, for example, that where male circumcision is high and sexual networks restricted, HIV prevalence tends to be lower, regardless of poverty, wealth or inequality. As an example to this, a widely cited commentary notes that:

"*Niger, a Muslim country where sexual behaviour is relatively constrained and MC*[7] *is universal, has an adult HIV prevalence of 0.7 per cent, despite being the lowest ranking country in the Human Development Index. Botswana, the second wealthiest country in Sub-Saharan Africa, has high levels of multiple concurrent partnerships among both sexes and lack of MC, with an HIV prevalence of 25 per cent"* (Potts, Halperin, Kirby, Swidler et.al, 2008:1).

Such claims are corroborated by research that adds that even within those countries with the highest HIV prevalence rates, the highest infection rates are not necessarily among the poorest, nor are they among those in the most gender-unequal settings (Fox, 2010:2; Parkhurst, 2010). Using DHS data from 13 sub-Saharan African countries, Fox (2010) shows a positive wealth gradient of HIV, with individuals from wealthier households more likely to be HIV-positive, though the relationship was not systematic.

Parkhurst (2010) investigates the relationship between HIV prevalence and poverty and wealth using data from 12 DHS and AIDS Indicator Surveys (AIS) from sub-Saharan Africa[8]. He does not find a consistent trend between the household wealth quintile measure and rates of HIV infection. While HIV prevalence systematically increased by wealth quintile among women from Cote d' Ivoire and both men and women from Rwanda, in all the other countries the relationship was not straight forward (ibid: 5). Among women in Lesotho, for example, those in the second and fifth wealth quintiles had the highest HIV prevalence rates while among men in the same country, HIV prevalence peaked in the third and fourth quintiles (loc. cit).

[7] Male circumcision
[8] Cameroon, Côte d'Ivoire, Ghana, Kenya, Lesotho, Malawi, Rwanda, Swaziland, the United Republic of Tanzania, Uganda, Zambia and Zimbabwe.

With Southern Africa being the epicentre of HIV infection, key questions about the factors that shape and drive HIV risk in the sub-region remain unsatisfactorily answered. This chapter poses two questions:

- What plausible empirical evidence is there to support the argument that structural factors, notably socioeconomic status (SES) shape the HIV epidemic in Southern Africa, and if so, which ones are crucial?
- Is there enough evidence to support the hypothesis that gender-related disadvantages could be the reason behind the high HIV prevalence rates amongst women in southern Africa?

In this chapter, we review the literature that strongly argues that structural drivers matter in explaining the HIV epidemic in southern Africa and in the next chapter, contrast it to the literature that argues that individual and biomedical factors actually play a more central role in driving the African HIV epidemic. In this review, we present some of the conceptual underpinnings that support the argument that structural drivers should matter as well as the supporting evidence from studies in southern Africa. In the concluding discussion, we weigh the evidence in respect of the presented arguments.

2.2 What are structural factors and how do they relate to HIV risk?

That health is greatly affected by the conditions in which people live has received plentiful scholarly attention over the past decades (e.g. Bell, et al., 2008; Braveman, Ergeter & Mockenhaupt, 2011; Marmot, 2000, 2005, Marmot, Friel, Solar, Orielle & Irwin, 2007; Marmot & Wilkinson, 2003; WHO, 2008; Williams, 2003). The general argument this literature makes is that there are differences in health status between individuals, and these health differences follow differences in social factors, most notably relative and absolute poverty.

Wilkinson and Marmot (2003) start their discussion by noting that even in the wealthiest countries, those people that are less well-off live shorter and are more susceptible to bouts of disease than the rest of the population (Wilkinson & Marmot, 2003:7). Drawing on articles and reports (many from the World Health organisation), the authors summarise what they observe to be the most common determinants of health – defined largely by life expectancy and frequency of illness (loc. cit). They observe that poor social and economic circumstances are a negative driver of health, "*with people down the social ladder usually run(ning) at least twice the risk of serious illness and premature death as those near or at the top*" (Wilkinson & Marmot, 2003:7). They also observe these differences within the same relative class levels, e.g. among those in the middle class, those closer to the top have better health outcomes than those at the lower end within this same class.

In this book, the authors discuss stress, early life support (or lack thereof), social exclusion, work, unemployment, social support, addiction, food and transport as the most influential factors on individuals' health (Wilkinson & Marmot, 2003). Through variable pathways (which we will explore later) some absolute and others relative, and with varying degrees of impact on health, the authors argue that all these factors ultimately determine how frequently individuals fall sick and how early they die.

Stressful situations, arising from worry and anxiety pertaining to economic insecurity, low self-esteem and social isolation all can affect health, in particular mental health (ibid:12). The authors argue that even in industrialised countries, the likelihood of experiencing these stressful situations is higher among those in lower social hierarchies. Early life support, even before birth, affects health and extreme examples are children born with foetal alcohol syndrome and other conditions arising from inadequate pre-natal care. Unemployment disposes individuals to all kinds of health disadvantages, ranging from more direct ones that include lack of sufficient finances to access health services for treatment and prevention to

more indirect health risks such as alcoholism, drug abuse and risky income generating activities such as crime (Wilkinson & Marmot, 2003). In short, this book argues that all those social factors listed above are the main determinants of health and that public policy should strive to address these if public expenditure programs towards improving health are to work. Notably missing from this piece though, are precise definitions for 'social hierarchy' or 'the social ladder', often referred to.

There is other complimentary literature, (e.g. Raphael, 2004) that contends that the economic and social determinants of morbidity include "*conditions of childhood, income, availability of food, housing, employment and working conditions, and health and social services*" (Raphael, 2004:1). Other, broader definitions have also been put forward and Blackenship and colleagues, for example, use an outcomes approach that defines structural factors simply as those issues that affect '*individuals' and communities' societal health outcomes*' (Blackenship, Bray & Merson 2000:1).

In a 2008 report, the WHO states that in all countries, and irrespective of income level, health and illness follow a social gradient with those in a lower socioeconomic status with poorer health (WHO, 2008:1). The report states that there are three main kinds of health differences across and within populations: the poor health that is experienced by the poor, the social gradient in health experienced within countries and the substantial health inequities between countries (ibid: 1). These inequities, the report argues, "*are caused by the unequal distribution of power, income, goods, and services, globally and nationally, the consequent unfairness in the immediate, visible circumstances of people's lives – their access to health care, schools, and education, their conditions of work and leisure, their homes, communities, towns, or cities – and their chances of leading a flourishing life*" (loc. cit).

This WHO report recommends three principles of action to alleviate health inequities. The first is to improve the circumstances in which people are born, grow, live, work and age – ultimately improving the conditions of daily life. The second is to address the inequitable distribution of the structural drivers of these conditions of daily life – distribution of power and resources, globally, nationally and locally. Lastly, the report recommends that policy makers develop a workforce that is well trained in the determinants of health and that public awareness about these social determinants is raised. These three main recommendations for alleviating health inequities (and ultimately improving health among individuals) clearly show that the focus is on changing the broader set of conditions under which people live.

In more recent research, Braveman et al (2011) make the argument that economic and social opportunities and resources, living and working conditions in homes and communities, access to medical care and personal behaviour all interact with age, gender and genetic make-up to affect health (ibid: 9). The authors argue that efforts that are only aimed at informing or urging individuals to adopt healthier lifestyles without considering where and how people live have been shown to be ineffective, as health is ultimately influenced by living conditions (loc. cit). In conclusion, they advocate for "*broadening the focus*" to improve the physical and social environments at home, school, work and neighbourhoods in order to improve health among individuals ultimately (ibid: S15).

In the HIV literature, there have been several attempts to define structural factors and although the definitions are not uniform, they generally consistently refer to those factors that impede the capability of individuals to reduce HIV risk (Gupta, 2008; Blackenship, Bray & Merson 2000; O'Leary & Martins, 2000). One study defines structural factors as "*barriers to or facilitators of individual's HIV prevention behaviours*" (O'Leary & Martins, 2000:1) and adds that these factors, which include physical, organisational, community, economic and others, primarily affect *exposure* to HIV (O'Leary & Martins, 2000). Important to note here

therefore is that these structural factors themselves are not directly linked to HIV risk, they influence behaviour (such as sexual) which then affects the risk of HIV infection.

The links between structural factors and HIV are complex, and several frameworks have been proposed to explain this relationship. Barnett and Whiteside (2002) propose a model in which different structural factors operate along a continuum of risk (Barnett and Whiteside, 2002). Along this continuum, there are distal factors and proximal factors that both affect HIV risk. The distal factors affect HIV risk through more diverse pathways, characterised by multiple cause and effects relationships than the closer, or more direct proximal factors (Barnett and Whiteside, 2002:2).

Denison and Sweat (1995) propose an alternative model that works in top down hierarchical levels, with super structural factors atop, affecting whole nations (e.g. level of economic development), followed by structural factors affecting a segment of the population (e.g. laws and policies) and environmental factors at the bottom, affecting individuals within the population (e.g. living conditions and opportunities) (Denison and Sweat, 1995).

Gupta (2008), alternatively, talks about structural approaches to HIV prevention and these are "*structural actions implemented as single policies or programmes that aim to change the conditions in which people live, multiple structural actions of this type implemented simultaneously, or community processes that catalyse social and political change*" (Gupta, 2008:3). The author portrays the main feature of such HIV prevention interventions as those that are not directly targeted at individual behaviour, but in the end, result in reduced HIV risk for individuals through shaping the environment individuals live in.

In figure 2.5 below, we present a framework proposed by Poundstone, Strathdee & Celentano (2004) which effectively combines elements of the frameworks discussed above. In this framework, the concentric rings represent the different levels at which the structural factors

operate and the distance between the factor and HIV risk/infection indicates how distal or proximal the factor is (Poundstone, Strathdee & Celentano, 2004).

In this framework, the outermost ring represents the most distal factors that affect the environment in which people live. The structural factors in this model in fact do more than just affect the environment – they actually make up the environment. They cover the broader context and encompass all the social, economic and biologic aspects that affect individuals. War, militarization and demographic change affect where people physically live, for example, whether they live in an urban area or a rural area, in their home country or abroad or even more specific locations such as refugee camps or other habitat.

These factors, through pathways that are complex, affect HIV transmission dynamics directly and indirectly. Take war for example. Through civil dislocation, mobility of armed forces and a higher incidence of prostitution and rape, the incidence of war may increase HIV risk within a population (Fourie, 2001; Fleshman, 2001). Military conflicts often include involuntary movement of refugees and displaced persons as well as separation of families, collapse of health services and dramatically increased instances of rape and prostitution (Fleshman, 2001:16) and these may increase HIV prevalence. The rate of spread of HIV may also be heightened by high rates of sexual interaction between military personnel and civilians through the use of rape as a weapon of war or through prostitution (Fourie, 2001:7).

Military personnel may also have much higher rates of HIV, possibly with prevalence rates of up to 80 per cent (Bisseker, 1998:34; International Crisis Group 2001; UNFPA 2003; U.S. National Intelligence Council 2000), but more recent literature contests the claim that southern African militaries have higher prevalence rates than the general population (Whiteside et al., 2006:1).

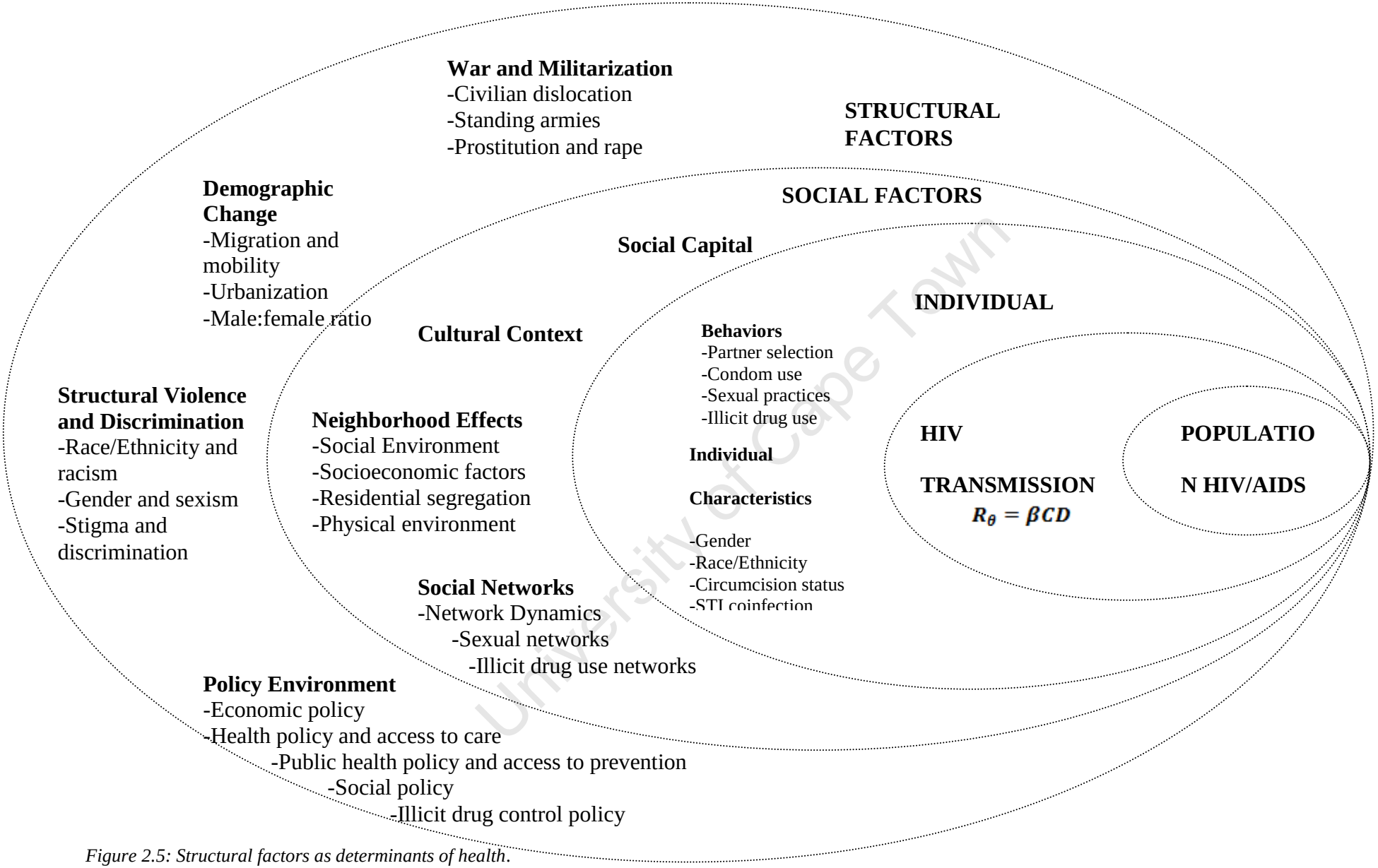

Figure 2.5: Structural factors as determinants of health.
Source: Poundstone, Strathdee & Celentano, 2004:3

Whiteside and colleagues (2006) sum up the reasons why military personnel may have higher HIV prevalence rates and contend that they may have higher rates because they:

"*may be posted away from their regular partners; are not as subject to behavioural constraints of family and community and are influenced by peer pressure; have a value set that encourages risky behaviour; are paid and so may be 'wealthy' in poor surroundings and have the cash to purchase sex; and may sustain injuries in combat and thus be exposed to unscreened blood through direct contamination or through transfusion*" (Whiteside et al., 2006:4). Although it remains difficult to ascertain the real level of HIV prevalence in militaries as there are a few published sources and where the data are available the militaries do not readily disclose the statistics (Whiteside et al 2006:1; Rupiya & Simapuka 2006:9; Barzegan, 2004), it is quite plausible that war may lead to larger HIV epidemics

However, Whiteside et al point out that the social disruption that war and militarization causes may also reduce HIV prevalence by restricting movement within the population (perhaps due to the presence of curfews or other mobility restrictions imposed directly by armed forces or indirectly due to security concerns (2006:3). It could also limit urbanisation and contain epidemics within localised regions and in southern Africa. Angola is an example of how militarization and war can possibly shield a nation from an explosive HIV epidemic. Prolonged civil war through all of the 1980s and most of the 1990s changed the HIV landscape and Angola has an HIV epidemic much lower than that of its neighbours who enjoyed peace over the same (Strand, Fernandes, Bergstrom et al., 2007). Iliffe (2006) also makes the same point about the Democratic Republic of Congo, arguing that war and civil unrest and the decimation of transport networks slowed down the spread of HIV from Kinshasa to the surrounding rural areas (ibid: 10-18).

Structural violence and discrimination includes racism, ethnic tension, xenophobia, sexism and stigma (Poundstone et al., 2004:3). The effects of stigma on HIV are widely documented and it prevents people from getting tested, seeking treatment and persisting with treatment from fear of victimisation (Deacon, Uys & Mohlahlane, 2010; Campbell, Skovdal, Madanhire et al., 2010; Dlamini, Wantland, Makoae et al., 2009; Kalichman & Simbayi, 2003; Simbayi, Kalichman ,Strebel et al., 2007; Campbell, Foulis, Maimane & Sibiya; 2007). All these may increase the persistence of an HIV epidemic.

Using data from a black township in Cape Town (224 men and 276 women), Kalichman and Simbayi (2003) look for the association between HIV testing and stigmas[9] related to HIV. The authors find that those not tested for HIV held more stigmatising attitudes than those that had been tested (Kalichman & Simbayi, 2003:4). Their analysis also showed that those who had never tested for HIV (coincidentally the group that held more HIV/AIDS stigmatising attitudes) were less likely to have ever used a condom and more likely to have used injected drugs.

Campbell and colleagues (2007) argue that stigma can serve as a form of "*social and psychological policing*" meant to punish those in breach of the underlying power relations of gender, generation and ethnicity (Campbell, et al., 2007:1). If it is a general belief in a society that HIV is the result of promiscuity, for example, then in such a society, those living with HIV may find it difficult to get moral (or any other support) from the community. Using focus group discussions among church youth (Christian Youth Alliance) from KwaZulu Natal (South Africa), the study examines the effects of HIV stigma on HIV related behaviour and finds stigma to be a deterrent of HIV preventative behaviours. Probing the reasons for a

[9] HIV related stigmas were measured using questions such as: Is AIDS spread by kissing, Can a person get AIDS by sharing kitchens and bathrooms with someone with AIDS, Can men give AIDS to women. For the full list of questions, refer to Kalichman & Simbayi, 2003:3

low turn-out at an HIV prevention meeting, the respondents suggested that there was stigma even from the church leadership as one church alliance worker said "*When we ask to talk about HIV in the churches they say we are encouraging the youth to sin. We recently called youth to attend a meeting on life skills [and had] a very poor turnout. Later we were told by youth that the minister said if they attended the workshop they would be demoted in the church*" (Campbell et al., 2007:3).

The policy environment encompasses health and economic and social policies that can affect HIV prevalence both directly and indirectly. Economic policy affects income and education levels for example, and the effects these have on HIV prevalence will be discussed later. Economic policy also affects health policy in as much as health policy is dependent on what resources are available to carry out the different health programs. Health policy in itself affects HIV and an example is the timing and scale of antiretroviral (ARV) treatment rollouts. In South Africa for example, Nattrass (2008c) used the Actuarial Society of South Africa (ASSA) model to predict that about 343 000 lives and 171,000 new infections could have been saved in South Africa, if the national government had rolled out highly active antiretroviral treatment (HAART) at the same time and pace as the Western Cape. In a study evaluating the relationship between country reputations for good and bad leadership on AIDS, Nattrass (2008b) finds that those countries with established reputations on AIDS leadership performed better, on average, on HAART coverage and prevention of mother to child transmission programs than those with bad reputations.

These distal factors do no only influence HIV directly; they also affect it through their effect on the social environment. They, according to the framework, affect aspects such as sexual networks, neighbourhood effects, the cultural context and social capital. Where the policy environment effectively monitors and controls illicit drug use, for example, illicit drug use

networks are likely to be smaller, and in epidemics that are mainly driven by injecting drug use this will likely reduce HIV infection.

In racially and ethnically harmonious societies where sexism, stigma and discrimination are low, social capital could be higher as communities can effectively mobilise against HIV infection and are able to support individuals through church, community centres, health centres and other such organisations (Barnett & Whiteside, 2006). Conversely, mobilising against HIV is more difficult in a society where social tension and mistrust is high.

It is not possible to exhaust all the possible interactions between the structural factors and the social factors and outline how each of them relates to HIV risk, but the underlying feature of the structural approach is that structural factors interact with each other and across the different layers of risk to shape population and individual HIV dynamics. The social factors then influence individual behaviours and characteristics, such as condom use, circumcision status, sexual practices, partner selection, income and education. This last group of factors primarily differs from the earlier groups in than none of the factors in the earlier groups actually directly affects HIV transmission –they merely affect exposure to HIV. Individual factors however directly affect HIV transmission as circumcision, sexual practices, partner selection and presence of sexually transmitted infections (STIs), for example, determines the likelihood of HIV transmission.

In the context of the theoretical framework, the key issues surrounding this debate hover around how powerful the various factors are. Very little research, if any, doubts that the proposed links between structural factors and eventual HIV- status are there. The issue centres on the relative strengths, or the importance, of the factors; i.e. is the influence of the structural factors on behavioural factors so strong that individual behaviour cannot not be targeted on its own to reduce HIV risk? This is the argument that the ‘structural-drivers-

matter' proponents make – that the impact of poverty, gender inequality or bad governance is so strong on individuals that it renders any attempts to modify behaviour useless. The counter argument is that gender inequality, poverty and endemic disease are problems that in their own right merit adequate attention, but for the purposes of fighting HIV, programs should target individual behaviour. In the following section, we present and critique the evidence in support of structural factors shaping HIV risk.

2.3 Poverty and HIV in sub-Saharan Africa

There is substantial literature claiming that poverty and lower SES for women are key drivers of the epidemic in Southern Africa and the rest of sub-Saharan Africa (Rodrigo & Rajapakse, 2010; Kim et al., 2008; Gabrysch, Edwards & Glynn, 2008; Wojcicki, 2005; Zierler, 1997; Gupta 2002; 2008). This literature argues that poorer individuals are more likely to be HIV-positive or to engage in riskier sexual practices. The main hypothesized pathways between poverty and higher risk sexual practices is that poor people, especially women, lack bargaining power to insist on safe sex in sexual relationships and they are more likely to be engaged in multiple partnerships as multiple partnerships have economic and social benefits (Gupta, 2008, Dinkelman et al., 2007; Wojcicki, 2005; Jewkes et al., 2007; Kim et al., 2008).

In a study of young adults in Cape Town, and using sexual debut, multiple sex partners in past year and condom use last sex as outcome variables, Dinkelman and colleagues (2007) find that household income is negatively associated with early sexual debut for both boys and girls. The same study also showed that among young girls, unexpected economic shocks due to the death of the main breadwinner or loss of job are associated with multiple sexual partnerships (Dinkelman, Lam & Leibbrandt, 2007). Among girls, per capita household income was negatively correlated with the probability of sexual debut. Women from those households that experienced a 10 per cent increase in income in 2002 were 0.6 per cent less

likely to sexual debut by 2005 (and this was in comparison to those that did not have increase in household income). Young men from poorer communities were less likely to report condom use, with a 5 per cent lower likelihood of condom use during last sex for every 10 per cent reduction in community wealth level (Dinkelman et al., 2007:1).

In rural KwaZulu-Natal, South Africa, Dunkle and colleagues (2007) examine the prevalence and predictors of transactional sex with casual partners and main girlfriends among 1 288 men aged 15-26 from the Eastern Cape (Dunkle, Jewkes, Nduna, Jama, Levin, Sikweyiya & Koss, 2007). They find that poorer women are more likely to engage in transactional sex, and were less likely to use condoms in their last sexual encounter (Dunkle et al., 2007). This study adds to those studies that suggest that poverty, especially among women prompts them to enter into transactional relationships for survival and in these relationships, they are more vulnerable to HIV due to limited negotiating power.

In Zimbabwe, Lopman and colleagues (2007), using a follow up cohort study (respondents were recruited in Manicaland between 1998 and 2001, and follow up was done between 2001 and 2003), investigate the association between the wealth index and HIV incidence, HIV mortality, sexual risk behaviour and sexual mixing patterns in Zimbabwe. The authors observe a decrease in HIV incidence across all asset wealth groups (for both men and women) during the study period, but that the largest decrease in HIV incidence was observed in the highest wealth tercile, again for both men and women.[10] The research also finds that wealthier women reported fewer partners and were less likely to engage in transactional sex (Lopman et al., 2007). The study attributes the decline in HIV incidence among the wealthier individuals possibly to a greater education level and better access to communication about

[10] 25% reduction for men and 21 % reduction for women.

HIV prevention and argues that the relatively wealthy are empowered and are able to change their sexual behaviour more easily than the poor are.

Weiser and colleagues (2007) find food insufficiency in the past twelve months to be associated with inconsistent condom use among both men and women in Botswana and Swaziland (Weiser et al., 2007). Food insufficiency defined as not having enough to eat over the previous 12 months, after controlling for income and education, was strongly related to risky sexual behaviour among women. In both countries, women who reported food insufficiency in the past year were twice as likely to have engaged in transactional sex and more likely to report a lack of control in sexual relationships. Among men, the authors find a weak association between food insufficiency and inconsistent condom use and no relationship between food insufficiency and other risky behaviours.

Often, the actual mechanisms between food security and HIV risk are not detailed, but a qualitative study in Uganda conducted face-to-face interviews with 25 women and 16 men who had reported food insecurity within the past year (Miller, Bangsberg, Tuller et al., 2010). The focus study interviews suggested that there were distinct pathways linking food insufficiency to HIV risk especially among women. Firstly, food insufficiency directly prompted women to engage in transactional sex. The study suggests for women without food, transactional sex becomes a viable means for one to provide oneself or their family with food. In the study, one of the respondents, a 35-year-old widow confesses:

"Yes, truly it happens to a lot of women. I've thought about it myself. Most of the time, when I'm desperate for something to eat, something to drink…And then the devil tempts you, and then you see a man, and he's there and you ask yourself, 'If I slept with him, couldn't I get 10,000 shillings [approximately $5.00 US] to buy maybe a sack of charcoal?'" (Miller et al., 2010:3)

Secondly, food insecurity compromised women's ability to insist on safer sex practices, especially condom use, even in primary relationships (Miller et al., 2010). Reports of men directly withholding food as punishment or control of condom use are also made and in a telling report a 44-year-old widow, with four children reports that:

"*If I'd insisted on condoms he would have been angry and would have beaten me, maybe. And the times I would really refuse to have sex with him, he would refuse to bring food and refuse to take care of the children. I tried it like two times, and both occasions he didn't bring home any food, he would go and spend the day elsewhere*." (Miller et al., 2010:4)

The same study also found, in addition to the above mechanisms, that widows were at an increased risk of food insecurity and of being approached for transactional sex and that food insecurity also acted as a barrier for women intending to leave abusive partners (Miller et al., 2010). This study is one of the few that outline the specific mechanisms through which poverty may be linked to HIV– it creates a need for primary resources, such as food, and transactional sex becomes a viable resource procurement means. Secondly, it limits negotiation power of women in relationships, as men use food (or any other resource) as a bargaining chip.

Lewis and colleagues, using a population based survey conclude that poor women in towns and in rural areas were more likely to be HIV-positive than wealthier women were but that the reasons for the higher prevalence varied by context. In rural areas, poor women were more likely to have started having sex earlier (the study does not explain the reasons for the varied sexual behaviour but shows that sexual behaviour varied by place of residence) while in towns they were more likely to be engaged in sex work than wealthier women (Lewis et al., 2006). Whether place of residence indicates the general access to social amenities, or lack

thereof, or is a proxy of the choices that individuals make in the face of poverty (or wealth), it appears to be a determinant of HIV risk.

This study does not make a distinction between the underlying motivations for an early sexual debut in rural areas and sex work among poor women in towns. An earlier sexual debut in the rural areas could indicate a form of transactional sex – the kind surrounded by and exchange of gifts such food for sex with those that are well off while in towns poor women are able to ask directly for cash in exchange for sex.

However, there is a significant body of research questioning the theoretical and empirical basis for explaining women's disproportionate vulnerability in terms of transactional and survival sex (Leclerc-Madlala, 2008, Luke, 2005, 2003). Luke (2003) performs a meta-review of studies from sub-Saharan Africa that are linked to age-disparate relationships and the motivations for adolescent girls to be in such relationships. The author concludes that although the motivations for adolescent girls are varied, they can be grouped into three main categories: economic assistance, securing longer-term opportunities and increasing social status among friends (Luke, 2003:7). Older men on the other hand, had a different set of motives. Luke identifies "*regular access to sex, enhancement of prestige, domestic help and maintenance of health*" as some of the main motivations for age-disparate relationships among men (Luke, 2003:8).

The author also finds that young girls appeared to have a great degree of control with regards the number and types of partners, and often successfully employed tactics to delay the onset of sex (such as false promises) (ibid: 9). They also had a high degree of power in terminating these relationships, or to refuse sex if they did not continue receiving gifts. Within the relationships however, across the many studies, older men are typically seen as having more

negotiating power regarding when to have sex and issues surrounding condom use and other safe sex practices.

What this meta-review therefore brings out, is that the power dynamic between men and women in these kinds of transactional relationships is one that is not simplistically one sided. In areas of key interest to the young girls, such as economic assistance and an improvement in social status, girls had a lot of bargaining power and thus would choose who they want to be with, how many people they want to be with and also even determine the extent and nature of gifts. Likewise, in areas of key interest to the older men, such as frequency of sex (and presumably the kind of sex e.g. unprotected); older men virtually had all the negotiating power. All else equal, such an interaction would be thus be beneficial to both partners – but the critical problem comes with the asymmetrical HIV risk levels that exist prior to the sexual relationships. Older men may well have higher HIV prevalence rates than the young women. This, together with the fact that women are biologically more vulnerable to infection from a single act of unprotected sex than men, renders young women in these relations especially vulnerable to HIV.

In a follow up study in Kisumu, Kenya, Luke (2005) used a sample of 1 614 partnerships to probe the prevalence of the "sugar daddy" phenomenon and to investigate the sexual interactions between these men and young women (Luke, 2005). One of the key findings of this research was that there was no evidence, contrary to widely held perceptions, that the higher HIV risk among adolescent girls in age-disparate relationships was due to their inability to negotiate safe sex with older men (Luke, 2005:12). The author finds that relationships with adolescent girls did not necessarily entail lower condom use per se – this was only true in relationships in which there was more than a 10-year age difference in the partners. The author reports that 'sugar daddies' did not seem to make decisions

independently of their partners, and concludes that, "*sexual behavior is an outcome dependent on the characteristics of both partners and the differences between them*" (ibid:13).

In a review of studies from southern Africa, Leclerc-Madlala (2008) identifies young women's agency in transactional sexual relationships and argues that women are able to make choices and bargain, based on their aspirations in life, and so are not necessarily powerless in their interactions with older men. This research argues that many young women in transactional and age-disparate relationships do not see themselves as victims and although young women often do not have sufficient negotiating power in sexual relationships they can freely choose the number and type of partners they have. Leclerk-Madlala also refers to what she calls "third-party" patronage, where among rural women, obtaining resources from older men through casual partnerships is a legitimate way for young women to improve their social image and become more attractive to young men with the hope that it might lead to marriage (Leclerc-Madlala, 2008:S19).

In Swaziland, Jones shows that among young women, transactional relationships show transfers of necessities, such as school fees or books, but more frequently, girls use them to obtain goods such as mobile phones, jewellery and fashion clothing (Jones, 2006).

In corroborating research, Wamoyi and colleagues find that transactional sex was widely accepted by both parents and young people in Tanzania – although the perception of gains was not the same among young men and young women (Wamoyi, Fenwick, Urassa et al., 2010). Young men saw transactional relationships as a way through which women could easily satisfy their sexual needs and men prove their masculinity while young women felt that men were "stupid" for having to pay for sex (Wamoyi et al., 2010:1). The notion that parents and both young men and women widely accept transactional sex and can identify with the

benefits arising from it (as well as the costs) suggests that that motives of engaging in transactional sex for women are not simplistically explained in terms of absolute poverty. They may be a result of a culturally entrenched system that, in this case, acts as a means of wealth transfer between families and offers a platform for individual and social experimentation for both young men and women.

In a widely cited piece from Malawi, Swidler & Watkins (2007) carry out a comprehensive theme search of journals collected by field workers recording conversations in rural Malawi pertaining to HIV. Drawing on these recorded conversations, mostly by women, the authors argue that transactional relationships occur within a cultural context, and within the African culture are not simplistically about poor women trying to survive. Instead, they are also about three things. Firstly, they are about relatively wealthier men, whose circumstances drive them to seek and maintain multiple partners, and, about women who are not extremely poor but who aspire to climb the social hierarchy, get economic independence or just improve their life with non-essential but admirable commodities in life such as soap and lotions (Swidler & Watkins, 2007: 11). Secondly, they are also about creating and maintaining ties of interdependence and although these ties may be unequal at the time of exchange, they may be reactivated in the future (loc. cit). Lastly, and most importantly in as much as it relates to HIV risk the authors argue, the relationships that evolve from the exchange of sex for money are just a subset of "*patron–client interactions that are pervasive through sub-Saharan Africa and proved to be resilient through time*" (loc. cit).

In rural Uganda, findings from research on discordant couples further suggest that women's sexual behaviour cannot simply be explained in terms hyper vulnerability. From 141 discordant couples used in the study, 57 per cent of those living in trading centres (who on average were wealthier than couples elsewhere) had the woman as the sero-positive partner

(Serwadda, Gray, Wawer, et al., 1995). This compared to 52 per cent of discordant couples living in trading villages and 20 per cent from those in agricultural communities (Serwadda, Gray, Wawer, et al., 1995). While this study is not recent and does not give direct evidence for women's agency, it definitely suggests a more complicated sexual behaviour pattern than the rather simplistic 'poor women are necessarily vulnerable' hypothesis.

The issue of agency versus vulnerability is complicated. Recently, this story was published in a popular South African women's online magazine:

> "*I'm a 16 year old school girl. Some time ago my neighbour, 25 and separated, started to talk to me about sex, what she did, she was then still married, and how she loved doing these things. She asked if I had had sex, I said no. Then her husband left and I would spend a lot of time with her.*
>
> *One day she invited me to shower with her saying she was going to show me what I was missing. She touched me in certain places and I felt wonderful, weird but nice. We watched adult movies and she explained that with a man it was even better.*
>
> *She then started to speak a lot about her boss and later told me that he was looking for a young girl.*
>
> *After some time she asked if I would be interested. At first I said no, but with her persistence and the fact that he would give me lots of money (I needed some things that my friends had and my parents could not give) I eventually agreed.*
>
> *We went to his house and she watched TV whilst I had sex with him - a man in his 50s. At first it was painful, but then I started to enjoy it. We now go to his house whenever his wife is away. Now all the girls in my school envy me as I have the best of everything and now I love the old man. Is it wrong? I love the benefits. What do you*

think of this?" Source: http://www.women24.com/LoveAndSex/SexAndSizzle/Sex-in-exchange-for-money-20120308 [Accessed on Thursday 08 March 2012]

We have presented this story here as it appeared in the article, devoid of any underlying background characteristics such as setting, race, nationality and any identifiers. This story perhaps sums up the complexity of the interplay between gender, agency, poverty, parenting roles and possibly HIV risk. Is this a story of abuse, where older men and women use their privileged social position (in this case, financial), to exploit unassuming young girls? Is it one of agency, where a young woman is exposed to life choices, and picks a choice that elevates her to a higher social standing among her peers (and is so comfortable with the arrangement she actually falls in love)? Is it about the influence of a lack of parental roles in society, where young women do not have enough guidance to make fully well informed decisions? Or, does it typify a case of 'hyper-vulnerability' of young women, where a nested web of push and pull factors spell out potentially harmful health outcomes for young women?[11]

There is also a growing body of empirical research disputing any putative positive aggregate relationship between poverty and HIV (e.g. Fox, 2010; Hattori & Nii-Amoo Dodoo, 2007; Hargreaves et al., 2007; Mishra et al., 2007; Msisha, Kapiga, Earls et al., 2008; Parkhurst, 2010). Mishra and colleagues find that in eight sub-Saharan African countries, adults in the wealthiest 25 per cent of the population had higher HIV prevalence than the poorer ones, but the positive association between HIV and wealth status was not statistically significant in multivariate models (Mishra et al., 2007). In this study, the authors use household wealth levels as the measure of poverty, the underlying assumption being that household wealth is

[11] Interestingly, at the end of the article, readers could anonymously vote from a list what the girl should do, and the list contained 1. She should tell her parents. 2. She should call helpline. 3. She should go to the police. 4. All the above and, lastly, 5. She's 16 – she knows what she is doing. As of 16 April 2012, 2 232 votes had been cast and 8% thought she should tell her parents, 6% were in favour of calling Childline (an organisation to assist children combat abuse), 5% reporting to the police while 50% voted "all the above" and 31% voted "she is 16, she knows what she is doing".

indicative of individuals' capability to afford and access health services. This study makes use of nationally representative data, thus adding weight to the conclusion that in general there is no clear association between wealth and HIV.

In Limpopo province, South Africa, Hargreaves et al., (2007) analyse a cohort study and find that sero-conversion among women was not associated with wealth (Hargreaves et al., 2007). The study finds education had a much larger protective effect on HIV incidence than poverty. A large nationally representative survey in Tanzania Msisha et al (2008) found a positive relationship between the standard of living and HIV-infection. In adjusted models, individuals in the highest quintile of standard of living had increased likelihoods of being HIV-positive, with odds ratios for men (2.38) and that for women (3.74). The relationship between occupation status and HIV differed between and women, with women in professional jobs more likely to be HIV positive whereas unemployed men were more likely than their employed counterparts to be HIV-positive.

In a study in Kenya conducted among Nairobi's poorest residents, slum dwellers, the authors looked at the prevalence of sexual exclusivity among married, cohabiting and co-residence couples (Hattori & Nii-Amoo Dodoo, 2007). The rationale for the research setting was to look at sexual behaviour in a context where poverty, hypothetically, would invoke risky sexual behaviour, specifically multiple partnerships. From the study, the authors conclude that marriage is associated with higher rates of sexual exclusivity for both men and women, and fail to find any evidence that poverty necessarily made men or women have many sexual partners.

How poverty relates to HIV risk in sub-Saharan Africa is clearly not well understood. Is it about household wealth, and if so, what is the behavioural link to HIV risk? Is it about food, or is it about the lack of access to information on how individuals may protect themselves

from HIV if they are poor? As the plethora of studies above show, researchers tend to focus on many of niche dimensions of what can generally be construed to be poverty (e.g. lack of food or low household wealth score), and then argue how these might be related to HIV risk in that particular setting. Weighing up the literature is thus problematic as the context and measurement of 'poverty' are far from consistent between studies. It would have been more helpful if poverty was measured consistently and if pathways between food insufficiency and HIV, household wealth and HIV, or monthly income and HIV were spelled out explicitly and then tested empirically. The literature on education and HIV risk is better in this respect.

2.4 Education and HIV in sub-Saharan Africa

Education is another potential structural determinant of HIV risk and the literature on the relationship between education and HIV risk in southern Africa is complex and sometimes contradictory. The literature suggests that there are two distinct effects of education on HIV risk – educational attainment and merely attending school (see e.g. Jukes et al, 2009:2). Educational attainment can affect HIV risk through three main pathways. First, it may modify sexual behaviour through improved knowledge and more accurate perceptions of the link between biological processes and HIV transmission among individuals (Jukes et al, 2009: 2). A higher educational attainment would thus enable individuals to understand the link between sexual behaviour such as unprotected sex and its end result (such as HIV infection) and may, as a result, encourage individuals to behave in ways that would protect them from HIV (Jukes et al, 2009: 3).

The studies discussed below provide some evidence for this hypothesis. In a study in Manicaland, Zimbabwe, women with higher levels of education reported higher levels of condom use and had lower levels of HIV infection than those less educated over a two-year follow up period (Gregson, Terceira, Mushati et al. 2004). In another study across 32

countries, Vandemoortele and Delamonica (2000), established that literate women were three times more likely than illiterate women to know that a healthy looking person can have HIV, and four times more likely to know the main ways to avoid HIV infection (Vandemoortele & Delamonica, 2000). The authors did not directly link these findings to lower HIV prevalence rates among women, but inferred that this would lead to lower infection HIV rates among them.

Educational attainment may also alter HIV risk through changing the socio-economic status of individuals by virtue of affording them better income-generating opportunities. Educated women may thus have lower HIV risk as they have better access to information about HIV prevention and have the means to purchase condoms and the economic independence to refuse to have unprotected sex with men they might otherwise be dependent upon.

The third pathway through which educational attainment may affect HIV risk is through establishing or improving social capital. A study in Manicaland, Zimbabwe investigated the relationship between HIV and belonging to well-functioning social groups related to churches, political parties and other organisations. The study finds that educated women were more likely to belong to such groups and that among women in these groups, educated women got a larger HIV protection effect than uneducated women (Gregson et al., 2004). Educated women were 1.5 times less likely to experience sero-conversion over a two-year follow up period, as compared to uneducated women who were also members of such groups.

The other effect education has on HIV is through schooling i.e. just being in school, regardless of what is learnt in school. There is a very limited discussion on this so far, but there are studies suggesting that school policies that lead to exclusion should one fall pregnant, for example, are a reason why schoolgirls may associate negative outcomes with unprotected sex and thus desist from it (Were, 2007; Jukes 2009:4).

Educational attainment has however also been attributed to exacerbating HIV risk among women. For example, several African studies have found that better educated women are more likely to be HIV-positive (e.g. Kelly et al. 1999; Gillespie Kadiyala & Greener, 2007; Gregson et al., 2006; Hargreaves & Glynn, 2002). It is argued that although more educated women may delay the onset of sexual debut, they delay marriage by an even greater extent and thus will have more lifetime partners than lesser educated women (Blanc & Way, 1998). And, although more educated women are more likely to use modern contraceptives than less educated women overall, they are also more likely to use hormonal forms of contraception which do not protect against HIV infection (Jukes and Desai, 2005:4) – and in fact may increase the risk of becoming HIV positive (Heffron et al, 2011).

Whether or not educational attainment ultimately lowers or increases HIV risk depends on which effect is greater, the risk-mitigating factors or the risk-enhancing factors. There is no plausible evidence so far suggesting which effect should be greater, but an increasingly large number of studies hypothesise that the age of the epidemic determines which effect is greater (e.g. Gillespie, 2008; Msisha et al., 2008; Jewkes et al., 2009). The latter research argues that at earlier stages of an epidemic, where limited information on HIV prevention is available, education is bound to be associated with higher HIV risk, as individuals are likely to be mobile and exchanging sex partners more (Jewkes et al., 2009:5). In later stages of the epidemic though, those who have attained a higher level of education should respond better to calls for behaviour change and can assimilate prevention messages faster (Gregson et al., 2006).

A systematic review of the literature on education and HIV reinforces this last point, concluding that prior to 1996, most studies found no, or a positive relationship, between education and HIV, but after 1996 the majority of the studies now find a protective impact

(Hargreaves et al., 2007:8). In this meta-review of the relationship between education and HIV, the authors reviewed over 4 000 abstracts and 1200 full articles. After eliminating those that did not meet the search criteria, eligible studies were grouped according to two time-periods, those before 1996 and those after 1996. The authors found that among the eligible studies before 1996, 15 studies in sub-Saharan Africa reported increased HIV infection among those with more education, 16 found no association and 1 found decreased risk among the more educated. Among the studies post 1996 (to 2008), 5 found increased risk of HIV among the more educated, 28 found no association and seven found decreased HIV risk among the more educated. Overall, out of the 72 studies under review, the majority (44) of the studies show no relationship between HIV and education.

The above findings are also corroborated by a study in Uganda that explores the association between schooling levels and HIV-1 levels in a rural population cohort (De Walque,Nyikayingi-Miiro, Busingye et al., 2005). The authors explore the association between schooling, measured by the highest grade of schooling attained, and HIV-status, using a sample of young adults from rural villages in Uganda recruited in 1989/1990. They run logistic regressions comparing the association between schooling on HIV-status controlling for age, gender and marital status among young adults. Next, they perform the same analysis but this time using young adults between the ages of 18-29 in 1999/2000. The authors find that controlling for the above covariates, there was a significant relationship between a higher educational attainment and lower HIV prevalence among women age 18-29 (De Walque et al., 2005).

Education and poverty do not operate in isolation. As the framework outlined earlier in figure 2.5 shows, wealth, education or poverty also affect each other and jointly affect HIV risk. There is a wider body of literature that looks at the relationship between economic status,

which is largely determined by education and income/wealth, and HIV. Other literature goes beyond economic status to look at socioeconomic status (e.g. Hargreaves et al., 2007; Collinson et al., 2006; Meier et al., 2006; Braveman et al., 2005 Murrain & Barker, 1997; Kaplan, 1996; Feinstein 1993) which is the influence of "*a composite measure that typically incorporates economic status, measured by income; social status, measured by education; and work status, measured by occupation*" (Dutton & Levine, 1989:30) on HIV. We discuss the link between SES and HIV below.

2.4.1 SES and HIV in sub-Saharan Africa

At the outset it is useful to distinguish between economic status and SES. Economic status is purely a function of how much an individual earns, or is reasonably able to spend. It is therefore primarily a function of one's job, occupation or other method that an individual has at their disposal to make raise income. Such methods could even be illegal or socially condemned (e.g. dealing drugs, sex-work, crime). There are thus likely to be serious problems with regard to the quality/accuracy of data on these activities.

Economic status may affect HIV in that it enables individuals to afford healthcare services. Holding external factors constant, economic status allows for the accumulation of household goods such as radio and television, allows for travel, higher education and other financial benefits. The studies reviewed thus far focus on this relationship between economic status and HIV, and a number show that a low economic status can lead to higher risk sexual practices for women.

However, economic status is not an accurate representation of individual's means to afford or not afford healthcare services. In the presence of gender inequality for example, while a woman may earn an income, the husband or family may confiscate or withhold part or all of her income. The husband and family may also dictate how her income is to be used and in

such cases, economic status in itself ceases to be a reflective indicator of the woman's ability to afford health services. Some studies therefore incorporate this social dynamic into economic status and seek to measure socioeconomic status instead of just economic status (e.g. Hargreaves et al., 2002; 2008 Braveman et al., 2005; Feinsten, 1993; Soobader, LeClere, Hadden & Maury, 2001). Certain occupations for example, may provide health insurance and while this does not change the economic status of that individual, it definitely changes the ease of access to health facilities. Occupations that need a higher level of education may also foster a greater capacity of home care through the knowledge gained from books and other materials (Rutstein & Johnson, 2004: 15). While a teacher and a drug dealer could have the same economic status, the teacher might be seen as having a higher socio-economic status as, in addition to probably being more likely to be afforded credit within the community, the community may be more willing to assist in terms of financial or other problems the teacher may have. However, as is clear from this discussion, the idea of SES is rather nebulous, difficult to measure, and measures are likely to vary from study to study.

A number of studies investigate the relationship between SES and HIV status (e.g. Barnighausen, Hosegood, Timaeus et al., 2007; Tladi, 2006; Msisha et al., 2008; Hargreaves et al., 2002; Wojcicki, 2005; Amuri, Mitchell, Cockroft et al., 2011). In 2005, Wojcicki (2005) performed a meta-review of the studies that investigate relationship between HIV and SES. The authors reviewed 36 studies from sub-Saharan Africa and of these 30 were cross-sectional, one a case control study and five were cohort or nested case-control studies. Fifteen of the studies found no association between SES and HIV infection, 12 found an association between higher SES and higher HIV risk; eight found that low SES contributed to higher HIV and in one, the evidence was mixed (Wojcicki, 2005).

Using longitudinal data, a study in Kwa-Zulu Natal investigates the link between three measures of SES (educational attainment, household wealth categories and per capita household expenditure) and HIV incidence (Barnighausen et al., 2007). Among the 3 325 individuals who tested HIV-negative at the start of observation, members from households that fell into the middle 40 per cent of relative wealth had a 72 per cent higher probability of HIV acquisition than members from the poorest 40 per cent households (controlling for sex, age, rural vs. urban residence, migration status and partnership status (Barnighausen et al., 2007). In another analyses based on the same sample, one additional year of education reduced the probability of seroconverting by seven per cent over the 1.3-year follow up period. In this study, per capita household expenditure did not influence HIV incidence. The authors conclude that while reducing poverty could have an impact on reducing new HIV infections, much bigger reductions in HIV incidence can be attained from investing in education.

In Tanzania, Amuri and colleagues find a positive association between HIV prevalence and stigma levels and poverty (Amuri, Mitchell, Cockroft et al., 2011). HIV was higher among women, those with higher education and those who reside in urban areas while HIV stigma was higher among the poorer, the less educated and those in the rural areas.

Tladi (2006) investigates the relationship between poverty and HIV using the 1998 South African DHS. The author finds a positive relationship between being poor and being HIV-positive, explained by poverty-related[12] characteristics such as lower levels of education and limited knowledge about the transmission pathways of HIV. The study also finds that poor women financially dependent on their husbands were at a higher risk for HIV as they were less likely to use condoms than those not financially dependent. However, while these

[12] These measures do not actually measure poverty themselves, but are usually associated with poverty

poverty-related measures predicted higher HIV risk, the study also notes that high SES was also linked to higher HIV infection, exhibiting a "U" shaped relationship between SES and HIV.

In rural Manicaland, Zimbabwe, Hallet, Lewis, Lopman, et al (2007) find that poor young women from rural areas had a lower age of sexual debut, and this was associated with higher rates of HIV infection. The study uses cross-sectional data with behavioural data linked to HIV test results for 4 138 men and 4 948 women. First, women that were unskilled and unemployed, not associated with any church and uneducated were more likely to have sex earlier than other women. Secondly, an earlier age of sexual debut was associated with higher HIV prevalence. In univariate models adjusted for years of sexual activity, sexual debut between the ages of 12 and 16 was associated with higher HIV prevalence than sexual debut at 21. (It is not clear in the study why these cut off points were made.) However, in the multivariate model, factoring in sexual behaviour, this age differential was not statistically significant. Among older women, women with a young age of sexual debut had higher HIV prevalence, and the authors explain this in terms of a higher number of lifetime partners for those that debut early.

Among such women who report early sexual debut, Pettifor and colleagues (2004) find higher HIV infection in Zimbabwe, but this time using data from Harare, the capital (Pettifor et al., 2004). Early sexual debut, defined as sex before the age of 15, was associated with higher HIV prevalence in a cross-sectional survey including 4 393 women. The study found 30 per cent higher HIV prevalence among those that debuted before the age of 15, controlling for duration of sexual activity and current age (Pettifor et al., 2004). Women with an early sexual debut reported higher risk behaviours such as multiple lifetime partners and not completing school.

Stillwaggon and Sawers argue that the gravity of the HIV epidemic in sub-Saharan Africa is due to poverty, but that this is because of malnourishment, endemic parasitic disease (such as malaria, schistosomiasis, and worms) and the high prevalence of cofactor infections due to poor health infrastructure (Stillwaggon, 2002, 2006, Stillwaggon, Sawers & Hertz, 2008; Sawers and Stillwaggon, 2010). In her 2002 article, which forms the core of an argument that has not changed substantially but has been refined in later publications, Stillwaggon argues that conditions of general poverty in Africa, characterised by insecure food security, poor health-care systems and a high prevalence of parasitic disease effectively made Africa a "*fertile terrain*" for HIV to flourish (Stillwaggon, 2002:1). In this piece, the author makes the claim that sub-Saharan Africa provided the set of conditions that would make any infectious disease spread quickly i.e. it is not that there is anything special about HIV– it is that there is something unique about Africa, and it is poverty and endemic disease (loc. cit.).

Firstly, Stillwaggon argues, sub-Saharan Africa in contrast to the rest of the world, experienced declining caloric intake (especially protein and vitamin micronutrients) between 1970 and 2002 (Stillwaggon, 2002:2). She hypothesises that this decline in nutrition, especially micronutrients responsible for protecting the body against infection, successively weakened the immune system of Africans, making HIV transmission among individuals in the region more likely. Secondly, parasitic diseases such as malaria, schistosomiasis, trypanosomiasis and intestinal parasites are highly prevalent in Africa, (with about 90 per cent of Malaria infections in tropical Africa, for example) (Stillwaggon, 2002:7). These parasitic infections have two main effects on the immune system. First, she argues, they further deplete nutrition in the individuals infected (ibid: 10) and secondly, in some cases, possibly make the HIV pathogen more virulent (loc. cit). Lastly, many STIs in sub-Saharan

Africa go untreated, especially those that are cofactors of HIV infection (e.g. chancroid and gonorrhoea), again supposedly increasing HIV transmission in the sub region (ibid: 9).

To summarise, Stillwaggon argues that the socio-economic and biological terrain in sub-Saharan Africa facilitated an explosive HIV epidemic. Most people are poor and subject to malnourishment and frequent bouts of infectious diseases. The weak immune systems, depleted by poor nourishment and continually ravaged by a series of parasitic infections and disease as well as untreated STIs naturally makes individuals in sub-Saharan Africa highly prone not only to HIV infection, but to other infectious diseases as well.

While contending that there may be some benefits arising from reducing co-infections, Nattrass (2009) disputes the argument that co-infections in themselves are one of the main reasons the HIV epidemic in Africa is so large (ibid: 2). Firstly, she notes that there have been no large-scale field studies that test for the association between HIV and parasitic worms (loc. cit). Secondly, she points to "*thin and contradictory*" evidence on the relationship between poor nutrition and HIV susceptibility, citing (Moore et al., 2003) who shows that in a study in Rwanda, there was no association between seroconversion and SES or blood vitamin levels (loc. cit). Lastly, Nattrass (2009) points out that the existing trials of micronutrient supplements have been unsuccessful in reducing HIV transmission, with some of them actually being stopped prematurely because they showed signs of harm (ibid: 2).

In subsequent work, Stillwaggon has expanded the argument to point out that the affluent countries in southern Africa, such as Botswana and South Africa, have high inequality levels and that the relationship between HIV and poverty still holds despite there being no obvious relationship at national level between average income and HIV prevalence (Sawers & Stillwaggon, 2010:7). In this respect, measures of national inequality (such as the Gini coefficient) are a proxy variable for the persistence of poverty

Using cross-country data, Stillwaggon (2002) and Sawers & Stillwaggon (2010) show a negative correlation between calorie intake and HIV prevalence in developing countries and a positive relationship between infectious disease and HIV (Stillwaggon, 2001; Sawers & Stillwaggon, 2010). Chapter 5 will analyse these results in detail, but apart from these studies, the evidence that nutrition is driving HIV infection in southern Africa and the rest of the sub-continent is otherwise not convincing. In two randomised control trials for example, one in Tanzania and another in South Africa, no evidence of lowered mother to child transmission was observed among HIV-positive mothers supplemented with vitamins[13] (Coutsoudis, Pillay, Spooner, et al., 1999; Fawzi, Msamanga, Hunter, et al., 2000).

2.5 Gender as a determinant of HIV

Research is divided when it comes to the relationship between HIV and gender inequality. On the one hand is the literature that suggests gender is not an important determinant of HIV in sub-Saharan Africa (e.g. Epstein 2007; Green et al.,2006; Kirby 2008; Oster 2007; Persson & Sjöstedt 2008; Potts et al. 2008; Stillwaggon and Sawers, 2010:8[14]; Stillwaggon, 2008:4). In this body of literature, some studies (notably Stillwaggon and Sawers, 2010 & Stillwagon, 2008) categorically state that gender inequality is not at all important in explaining HIV prevalence in sub-Saharan Africa (Stillwaggon & Sawers, 2010:8; Stillwaggon, 2008:4). The other literature cited here, though without the assertion that gender does not matter, infers (mostly through discussing their own 'main determinants' of HIV that are not gender) that gender is not the main determinant of HIV in sub-Saharan Africa (e.g. Epstein, 2007; Green et al., 2006; Halperin & Epstein, 2008:7; Kirby, 2008; Oster, 2007; Potts et al., 2008).

There is also a literature that strongly argues that the oppression of women and their relatively limited income, political representation and power renders them more vulnerable

[13] Generally, vitamins are responsible for boosting the immune system.
[14] Citing a lack of direct evidence from any of the studies that argue gender is important

to HIV (Brummer, 2002; Gillespie, Kadiyala and Greener, 2007; Hunter, 2007; Over, 1998; Tsafack, 2006,2008; Kim et al, 2008; Jewkes & Dunkle, 2006 Maman, 2000; Sibanda, 2000; Grieg & Koopman, 2003; Jewkes, Levin, & Penn-Kekana, 2003; Mill & Anarfi, 2002;Orubuloye, Caldwell, & Caldwell, 1993; Seidel, 1993). This literature generally argues that there is a positive relationship between gender inequality and HIV, with countries that oppress women more likely to have worse HIV epidemics because women are in a weak social and economic position and are unable to adequately protect themselves from HIV. We discuss these studies below.

The hypothesised pathways through which gender inequality leads to HIV are varied, but some literature makes the strong claim that cultural practices and norms carry entrenched discrimination, which vilify women and exposes them to higher levels of infection than men (Peitersen, 2009; Jursut & Kalipeni, 2010, Rassjo & Kiwanuka, 2010). One of these norms is the prevalent expectation that within a sexual relationships men should determine or take charge of the important decisions about sex – the how and the when to have sex. Research from Malawi, South Africa, Kenya and Uganda shows that men usually have control over condom use (Bingenheimer, 2010; Bühler & Kohler, 2003; Camlin & Chimbwete, 2003; Kishindo, 1995; Maharaj & Cleland, 2004), and that many women are brought up to view their husbands as the dominant sexual partner (Lawson,1999).

It is also claimed that social institutions such as marriage sometimes uphold a form of institutionalised control of men over women, as it is common in sub-Saharan Africa for men to "*make it extremely difficult for wives to refuse sex, insist upon condom use, or confront their husbands about extramarital partnerships*" (Bingenheimer, 2010:2). Cultural systems sometimes may thus dictate that women have no control over their sex lives or those of their husbands' outside marriage (Caldwell, Caldwell & Quinn, 1989; Smith & Cohen, 2000). As

an example, while extramarital affairs are tolerated by many cultures in sub-Saharan Africa, in others, there are rules requiring women to have no or limited sexual experience before marriage and expected to be monogamous thereafter, while no such restrictions are imposed on men (Buve, Bishikwabo-Nsarhaza & Mutangadura 2002:4).

In Uganda, Rassjo and Kiwanuka (2010) conducted focus group discussions on views about social and cultural influences on sexuality. Sexual health group discussions among adolescents and adolescents voiced some of the pressures that include forced marriages and pressure to marry and transactional sex (sometimes even with relatives) (Rassjo & Kiwanuka, 2010). As one boy put it: "*sir, the problem here, our culture will not allow a girl who is above 18 years to stay in the family when she is not married. It is a curse and it is a taboo. Any person who is out of school, if you have developed breasts and you started menstruation you are supposed to get married. So to avoid being cursed and being a taboo, that is why young girls tend to marry so early. And the moment they get married they have their first pregnancy and they have to produce (Boy in Wakiso*)". (Rassjo and Kiwanuka, 2010:6).

There is also literature that claims that women's social burden – through culturally imposed family responsibilities – leads to worse HIV outcomes among women (e.g. Rankin & Lingren, 2004; Desai & Potter, 2001). The issue here is that women's roles[15], dictated by social norms or culture, may lead women to be poor (especially in the annulment of marriage) as they do not have the time nor the opportunity to enter the mainstream economic market by either going to school, participating in skills training or obtaining formal work (Rankin & Lingren, 2004). They are then left in a cycle of poverty that could lead to transactional sex in

[15] In a study in Malawi, Rankin and Lindgren identify three main sources of burden for women, namely: the family, poverty and women's roles (Rankin and Lindgren, 2004). Women care for children, men and frequently for their in-laws, and lately this burden has increased as they now also take care of most of those with HIV/AIDS (Rankin and Lindgren, 2004). Other research has also identifies the "triple burden" on women: household chores, community work and household responsibilities (Desai & Potter 2001)

the mechanisms explained earlier and this leads to worse HIV odds among them than among men.

Other cultural practices such as polygyny are also implicated in worsening the HIV epidemic among women in sub-Saharan Africa (Lawson, 1999; Runganga et al., 1992; Bove & Valeggia, 2008). Polygyny is very prevalent in much of East and Southern Africa (Dalton and Leung, 2011:1) and is linked to a faster rate of spread of STIs that include HIV[16] (Bove & Valeggia, 2008). There are a number of possible ways through which polygyny may result in higher HIV prevalence among women. Bove and colleagues suggest that in polygamous relationships, HIV risk is higher as there are documented low levels of condom use in these relationships, poor communication among spouses and the relationships often involve multiple partners[17] (Bove & Valeggia, 2008:1). Reniers and colleagues find that men in polygamous relationships report a higher level of extra-marital sex and that there is an "adverse selection" of women living with HIV into polygamous relationships (Reniers & Tfaily, 2008:1)

Research has frequently disputed that polygamy leads to increased HIV prevalence. From earlier research that argues that polygyny lowers the rate of HIV infection because it limits the incidence of extra-marital sex (e.g. Caldwell, et al., 1993; Lawson 1999) to more recent research that gives evidence disputing that polygyny increases HIV infection (e.g. Mitsuanga, 2005; Reniers & Watkins 2010; Reniers, Georgesa, Watkins et al., 2010), there is substantial literature that argues polygyny might actually have a protective effect against HIV.

[16] There is other literature that associates polygyny and a worse African epidemic, but this literature discusses this as concurrency problem and not as a cultural problem. We will highlight this literature in the section on individual behaviour and HIV risk

[17] The authors make the assumption the multiple partnerships are inherently more risky

Using 19 DHS and AIDS Indicator survey (AIS) data, Reniers and colleagues examine the national and sub-national relationship between polygamy and HIV across sub-Saharan Africa (Reniers & Watkins 2010). The authors find that at both the national level and sub-national level, HIV prevalence is low in areas that polygamy is more common, and, within those regions with higher levels of polygamy, HIV prevalence is lower (Reniers & Watkins, 2010). The authors explain their findings in terms of closed sexual networks that may result from polygamy, disproportionate recruitment of HIV-positive women into polygamous marriages and lower sex frequency in those relationships (Reniers & Watkins, 2010:1).

First, they argue that in many of these relationships, sexual relationships are largely restricted within the marriages. This, they argue, has a larger protective effect at an individual and community level than the additional HIV risk that may result from the multiple partnership nature of the marital unions (ibid:6). Secondly, they observe that in polygamous relationships, there was a disproportionate recruitment of HIV-positive women, and this could have the effect of protecting HIV-negative people (and the community as a whole) as it lowers the likelihood that HIV-negative individuals meet with those that are HIV-positive. Lastly, they also observed that polygamous relationships reported lower sex frequency and this may lower the likelihood of HIV transmission within some of the marriages. Polygamy, therefore, they conclude, is a form of "*benign*" concurrency (Reniers & Watkins, 2010:6)

It is hardly prudent to base generalised conclusions on a single study, but this study suggests a sexual network story – that in cases where many women can be married to the same man, this is not itself necessarily risky. If everyone involved in the marital union restricts sexual activity to within that marital union, then they are locked in a low HIV-risk network. If none of the members is HIV-positive, then such a network will have a protective effect for those individuals. More so, even if any of the members are positive, a protective effect at the

population level can still be experienced for as long as the network is closed. The observed tendency of many HIV-positive women to disproportionately cluster in polygamous marriages could result in lowered community HIV risk as HIV infected people are also locked in their own sexual network. However, this study does not give any insight as to the magnitudes of these protection levels.

The practice of 'dry sex' where women use plants or cloth to dry up vaginal fluid during sex is common in southern Africa[18] and some authors suggest it increases HIV risk among women (Hilber, Hull, Preston-Whyte et al., 2010; McClelland et al. 2006; Baleta 1998; Van De Wijgert, Morrison, Salata, & Padian, 2006; Runganga et al., 1992). These studies hypothesise that dry sex leads to higher transmission probabilities as the risk of genital tearing is higher and susceptibility to bacterial vaginosis (which facilitates faster HIV transmission) is higher among women who practice dry sex (McClelland, Lavreys, et al., 2006; Van De Wijgert, Morrison, Salata, & Padian, 2006). However, this literature is still in its infancy and lacks direct evidence of the association between dry sex and HIV infection.

Widow inheritance at a time where one of the main causes of death is sub-Saharan Africa is HIV, may expose women to sexual partners already infected with HIV (Agot, Stoep, Tracy et al., 2010; Floyd, Crampin, Glynn et al., 2008). Using a prospective cohort study to investigate if widow inheritance increases HIV risk, Agot and colleagues find increased odds of HIV infection, after adjusting for age and education, among inherited wives than among those not inherited (Agot, Stoep, Tracy et al., 2010). The authors investigate, using 1 987 widows with an average age of 35, the relationship between the type of widow inheritance and HIV-status, controlling for a wide range of bio-markers such as number and type of

[18] There is ethnographic research from sub-Saharan Africa that documents men's preference for "tight sex" (Brown, Ayowa, and Brown 1993; Morar and Karim 1998; Wojcicki and Malala 2001). Vaginal practices of drying up the vagina are also "supported by cultural beliefs about sexual health and gender ….and strongly influenced by local notions of cleanliness and sexual readines" Scorgie, Smit, Martin-Hilber et al, 2010:4

sexual partners, frequency of sex, condom use, a history of transactional sex, age, occupation, place of residence, among others. HIV-status was the outcome variable in logistic regressions, and type of widow inheritance a categorical variable with not inherited, inherited by family members for companionship, inherited by family members for ritual practices, inherited by non-family members for companionship and inherited by non-family members for ritual practices as the categories for widow inheritance.

Controlling for age and educational attainment, there were consistent differences in the likelihood for being HIV-positive among the inherited and the non-inherited, although there were further differences in HIV risk depending on the motive for inheritance and the person who inherited. Widows that were inherited were more likely to be HIV-positive than those that were not inherited, but this was not controlling for HIV status at the time of husband's death. In an attempt to factor in the HIV status at the time of husband's death, the authors restricted a regression to a subsample of widows who had been widowed for 10 or more years – which is the average life expectancy for those living with HIV from the onset of HIV infection in Kenya. From this subsample of 235 women who were most likely HIV-negative at the time of their husband's death, those inherited by a non-relative for sexual ritual purposes were at a higher risk of being HIV positive than those not inherited.

The study showed that among the Luo, in Kenya widow inheritance exposed women to a higher likelihood of being HIV-positive, especially for those women who were inherited by non-relatives for ritual purposes. The actual reason for this however, was unclear, as the authors note the existence of "*jokowiny*" or professional inheritors (ibid: 2) who are men who serially and sometimes concurrently inherit many wives, in response to the lower numbers of in-laws willing to inherit. Thus, whether the heightened risk levels among inherited wives

was a concurrency story or a function of other, yet unclear, links between inheritance and HIV is unknown.

In Malawi, Floyd and colleagues find that widows are often obliged to be inherited by the dead family's relatives as this is one of the few traditional ways for them to achieve financial security (Floyd, Crampin, Glynn et al., 2008) but the authors do not hypothesize if this would increase vulnerability to HIV or not.

There are very limited studies on the prevalence of widow inheritance in southern Africa (and on how much it affects HIV prevalence) but the few studies available elsewhere suggest that in some societies, such as the Luo in Kenya, it is very high (Prince, 2011:1, Agot et al., 2010:1) but in others low or undocumented (Agot et al., 2010:2). It is thus hard to know what impact this cultural practice has on individual level HIV risk but the possibility of a relationship between HIV and widow inheritance has been discussed as a possible source of additional HIV risk among southern African women.

The tradition of paying bride price in cash or kind may negatively affect women's ability to negotiate abstinence, faithfulness or condom use by their partners (Bishai, Falb, Pariyo, et al., 2009:3; Bishai, 2006; Wendo, 2004; Lawson, 1999). Bride price payments range from token gifts of esteem to outright purchase that can result in a refund if the husband is unsatisfied with the marriage (Bishai et al., 2009:1). In the case where these payments are substantially large, relatives or family may dictate when a woman gets married, to whom they get married and also influence decisions about condom use, abstinence and negotiations for fidelity (Anderson 2007:1).

In South Africa, research using focus group discussions failed to find any evidence of sexual behaviour change among men, induced by paying lobola (i.e. bride price) (Heeren, Jemmott,

Tyler, Tshabe & Ngwane, 2011). The authors held 11 focus group discussions with 11 to 14 participants in each discussion comprising of both men and women. The key objective of the study was to investigate whether paying lobola was associated with sexual behaviour change among men (Hereen et al., 2011). The results showed that it was generally agreed among the respondents, both men and women, that husbands would typically have other sexual partners, regardless of whether they paid lobola or not. Lobola as a cultural custom was not identified as a reason for infidelity. Rather, it was mutual respect that determined whether a husband had other partners or not – and this was not necessarily linked to paying lobola.

Closely tied to cultural norms and practices are the social constructions of masculinity and femininity. Not all masculinities are the same (Connell, 1987, 2000), but in many societies, a dominant or hegemonic masculinity is present – with implications for both men and women. We will not engage in a comprehensive discussion of how masculinities are formed – this has been widely discussed and debated elsewhere (e.g. Connell, 1987, 2000, 2005; Donaldson, 1993; Barret 1996). However, there is sizeable body of literature that argues that in Africa, masculine and associated feminine identities may be responsible for heightened HIV risk levels, particularly among women (e.g. Sorrell & Raffaelli, 2005; Jewkes & Morrell, 2010; Wood & Jewkes, 2001; Hunter, 2006).

In South Africa, qualitative research has shown "*denial of weakness, virility, the appearance of being strong, and emotional and physical control and respect*" (Sorrell & Raffaelli, 2005: 3) to be viewed widely as characteristics that define manhood. Jewkes & Morrell (2010) also support this finding and contend that the dominant ideal of an African man "*emphasizes toughness, strength and expression of prodigious success*" (Jewkes & Morell, 2010:1). Various authors discuss the existence of the "*isoka*" masculinity among the Zulu – a dominant masculinity among Zulu-speaking people, that prescribes that a 'real' man was one

with many sexual partnerships (Leclerc-Madlala 2001, 2012; Hunter, 2006; Iliffe, 2007). Iliffe (2007), for example, portrays the isoka as "*the handsome, popular, and irresponsible hero who displayed his masculinity, in one of the few ways available in a township, by having penetrative sex with girlfriends whom he could not afford to marry*" (Iliffe, 2007:46).

Responses from qualitative research among gold miners also brought up the association between manhood and toughness, with a common metaphor among the miners likening men to sheep, and "*…no matter how hard you hit a sheep or slaughter it you will not hear it cry. The animal that can cry is a goat. So, that is a comparison that whatever pain you can inflict on a man you will not see him cry*" as according to one miner (Campbell, 1997: 6). Using studies from South Africa, and contending that the same holds for Southern Africa, Wood and Jewkes (2001) argue that in South Africa, sexual prowess among young men is often seen as a sign of virility, and sexual inexperience a sign of purity among women (Wood & Jewkes, 2001).

Constructions of masculinities and femininities can affect HIV risk through two general ways. The one is where the construction itself perpetuates higher risk sexual behaviour – e.g. by encouraging multiple sexual partners or by desisting from condom use. Varga (2001) notes that among young Zulu men for example, while a significant minority actually state a preference for abstinence until marriage, they often end up engaging in penetrative sex because they feel obliged to for fear of social rejection (Varga, 2001). Campbell (1997) highlights the challenge of improving condom use among gold miners, where virility is part of what makes one a man, but men feel using condoms compromises virility (Campbell, 1997:2). In such cases, the social construct itself directly promotes a behaviour that exposes the individual to higher HIV risk.

The second, and perhaps the more complex and trickier to elucidate, is the higher HIV risk that arises from the combination of 'harmless' constructions of masculinity and femininity – which although nothing may be intrinsically problematic with these constructions per se (obedience or commanding respect do not in themselves change HIV risk levels), the combination spells higher HIV risk. Jewkes and Morrell (2010) succinctly describe this when they argue that

"*The women partners of hegemonically masculine men are at risk of HIV because they lack control of the circumstances of sex during particularly risky encounters. They often present their acquiescence to their partners' behaviour as a trade-off made to secure social or material rewards, for this ideal of femininity is upheld, not by violence per se, by a cultural system of sanctions and rewards. Thus, men and women who adopt these gender identities are following ideals with deep roots in social and cultural processes, and thus, they are models of behaviour that may be hard for individuals to critique and in which to exercise choice. Women who are materially and emotionally vulnerable are least able to risk experiencing sanctions or foregoing these rewards and thus are most vulnerable to their men folk.*" (Jewkes & Morell, 2010:1)

What the authors highlight here is the complex interplay of HIV risk, social constructions of masculinity and femininity and the underlying society-specific conditions of wealth, poverty or vulnerability. On the one hand, women desire those perceived to be 'masculine', because these are the attractive men in society as society prescribes. They fulfil many or most of their gender roles and likewise are respected and revered. However, the notions of masculinity these men follow may include a prescription of risky sexual behaviour, which, even if the women are opposed to it, are not readily able to speak out, as speaking out (as an example) may not be a socially desirable feminine characteristic in some places. In cases they may

want to speak out, underlying conditions of vulnerability may negate the willingness to speak out as it could come at a cost of being ostracised.

Gender inequality against women also occurs outside the cultural context. At a national level, for example, the representation of women in parliament in sub-Saharan Africa is poor. According to the 2009 HDR data, some countries, e.g. Rwanda did not have a single woman representative in parliament (hdr.undp.org/en/statistics). The argument here is that women are not adequately represented in political decision-making (and although participation in parliament is a single measure, it reflects the general state of women's representation) and therefore national laws, such as those pertaining to rape, abuse and inheritance inadequately protect women (Wood and Jewkes, 2001, HDR, 1995).

To summarise, there are two general pathways linking gendered cultural norms and practices to enhanced HIV risk for women. The first is the poverty story – that cultural practices result in a gendered bias in the allocation of resources and women are 'forced' into survival or transactional sex, which increases their risk of contracting HIV. This poverty also necessitates a dependence on men, which severely limits their bargaining power with men in issues concerning fidelity and use of condoms for fear of abandonment and destitution. More so, in cases where women know that they are at risk, perhaps due to infidelity by the spouse, they cannot readily refuse sex or leave. In some cases, they may be forced to stay with a promiscuous husband because they cannot afford to reimburse the husband or his family the bride price and may thus be stuck in an HIV risky marriage.

Secondly, many cultural norms in themselves impose the direct risk to women. Dry sex, for example, increases the chances of abrasion during sex, and thus may increase the likelihood of HIV transmission. Polygyny places married women in a sexual network that may be risky (but the literature also suggests that such arrangements could be protective). Constructions of

femininity may encourage being pure, sexually inexperienced and passive about matters concerning sex with their partners or husbands and thus leaving women in a weak position to negotiate safer sex.

2.6 Discussion

There is a wide range of evidence from a number of southern African countries and from other countries elsewhere that suggests that poverty, and low SES especially among women, is a key driver of HIV infection. In the studies we reviewed in this chapter, some of which are meta reviews and thus cover a wide scope of the literature suggest that poverty increases the pressure among women to resort to transactional sex and that it also reduces bargaining power in relationships. Poverty has also been linked to HIV through increased malnutrition and a higher prevalence of presence of STIs among the poor. In summary, the literature that suggests that poverty and low SES is responsible for high HIV rates in sub-Saharan Africa argues that at a national level:

- Poor countries have underdeveloped health infrastructure and therefore the people in those countries have higher parasitic disease incidence and successive bouts of infection continuously compromise their resistance to all infections and this weakens their immune system. The weak immune system makes disease incidence for all infections, including HIV, higher.
- Untreated STIs are common due to the poor health infrastructure, and these untreated STIs increase the chances of HIV transmission because some of them are cofactors to HIV

At an individual level, poverty:

- Leads to insufficient resources for survival, such as food, and poor women resort to transactional sex. In these transactional relationships, bargaining for safer sex is limited, as the consequences of not having enough food are immediate
- Poor women also face restricted negotiating power even in their primary relationships due to the combination of poverty and gender norms and this feeds into the cycle of poverty and vulnerability to HIV
- Poverty also increases the likelihood that men offer transactional sex
- Can induce stigma, perhaps due to inadequate or limited access to media information about the true transmission pathways of HIV and how one may protect oneself from HIV.

Apart from poverty and SES, other structural factors that are crucial include discrimination and inequality. Discrimination may lead to poverty, and through the summed ways above, lead to higher HIV risk. Alternatively, or concurrently, unequal societal norms and customs may directly increase HIV risk among individuals.

In the upcoming chapter, we review the literature that contests the hypothesis that poverty and gender are the key drivers of the HIV epidemic in sub-Saharan Africa.

Chapter 3. Concurrency, male circumcision and individual sexual behaviour as key determinants of HIV in sub-Saharan Africa

3.1 Introduction

In chapter 2, we gave a background of the HIV epidemic in sub-Saharan Africa and review the literature that argues structural factors including gender inequality and lower SES has a stronger influence on the HIV epidemics in the region and in Southern Africa. As we pointed out, there are those that believe instead that it is concurrency, behavioural and biomedical factors that affect HIV more. In this chapter, we review these studies.

3.2 Concurrency and HIV in sub-Saharan Africa

Many authors argue that the high HIV rates in sub-Saharan Africa are a direct result of concurrent sexual partnerships (Kretzschmar & Morris, 1996, 1997, 2000; Halperin & Epstein 2004, 2007; Potts et al. 2008; Shelton, 2007, 2009; Southern Africa Development Community 2006; UNAIDS/WHO 2006; Stoneburner & Low-Beer, 2004; Green, Halperin, Nantuluya & Hogle, 2006). In a discussion that has received widespread support (and critique), this literature flags concurrent sexual partnerships, especially long-term concurrent sexual partnerships, as the main determinant of high HIV prevalence rates in sub-Saharan Africa. The basic argument here is that concurrent sexual relationships, where one man has more than one sexual partner at a time, result in a faster rate of transmission of HIV than serial sexual relationships, where one person can have many partners, but one at a time.

Using a mathematical model that simulated the rate of spread of HIV in a population with a high proportion of concurrent partnerships, Kretzschmar and Morris attributed the high HIV prevalence rates in many sub-Saharan African countries to concurrency (Kretzschmar & Morris, 1996). In this widely cited article, the authors hypothesized that HIV in sub-Saharan

owed its rate of spread primarily to sexual network dynamics (Kretzschmar & Morris, 1996:1). In the mathematical simulation, with a set of assumptions discussed below, the authors reach a conclusion that under concurrent partnerships, the rate of spread of HIV in a population would be exponential, as compared to linear growth path for a population that is characterised by serial monogamy.

The authors had to rely on a number of assumptions, and they made assumptions about the rate of formation of new partnerships, rate of dissolution of concurrent partnerships, the probability of HIV transmission per day and the number of sexual contacts a day (Kretzschmar & Morris, 1996:17). They assumed that:

- There is a one per cent probability that a new partnership starts every day
- The frequency of sexual contact between all partners is one a day (i.e. that every individual in the concurrent relationship has sex with every member in the union once every day)
- There is a five per cent probability that partner dissolution occurs every day
- There is an HIV transmission probability of five per cent per day (Kretzschmar & Morris, 1996:17)

The model allowed for two types of partnerships. Serially monogamous relationships in which individuals would not have any sexual contact outside of the relationship for the duration of that relationship and concurrent partnerships where the average length of the partnerships was about 2 years (Kretzschmar & Morris, 1996:17).

Kretzchmar & Morris, (1996) concluded that the spread of an epidemic not only depended on the number of sexual partnerships but also on whether these occurred simultaneously or in sequence over time (ibid:27).

The 1996 model was followed by another in 1997, (Morris &Kretzschmar, 1997) which was aimed at examining "*how concurrent partnerships amplify the rate of HIV spread"* and potentially being a *"useful and practical tool for evaluation and intervention at the beginning of an epidemic*" (Morris &Kretzschmar, 1997:1). Much of the underlying methodology between this model and the one preceding it was the same (ibid: 3) but the key emphasis of this model was on rate of HIV spread in the early years of the epidemic.

In this research, the authors simulated the spread of the HIV using 10 simulations, the first with serial monogamy only and then with each successive simulation performed with a higher concurrency measure. The authors reach to the same conclusions as before, and find that concurrent partnerships exponentially increase the number of individuals infected by HIV in the initial stages of an epidemic. In one simulation, where one-half of the partnerships in a population are concurrent, the size of the epidemic after 5 years was 10 times larger than that simulated under serial monogamy.

These models showed one main thing – that under concurrent partnering, the rate of growth of HIV infection in a population would be exponential while under serial monogamy it would be linear. The models did very little to explain specifically why this was the case and alluded that more epidemiological research was needed to elucidate the exact population level dynamics at play.

Various studies in support of the concurrency argument were subsequently published (e.g. Halperin & Epstein, 2004, 2007; Mah & Halperin, 2010a; 2010b; Epstein, 2007, 2008). Early

support for the concurrency argument relied heavily on the mathematical models of Morris and Kretzschmar whilst providing additional reasons why concurrency was likely to be driving HIV patterns in Africa (e.g. Potts et al. 2008; Shelton, 2007; Halperin & Epstein, 2004, 2007). Epstein, (2007) for example explains how concurrency could plausibly explain how in Africa, HIV managed to spread generously among the general population and was not confined to high-risk population groups, as was the case outside Africa, for example in Europe and the USA (Epstein, 2007:54).

Halperin and Epstein (2007) note that national HIV prevalence is lower in countries with concentrated epidemics (among men who have sex with men and injecting drug users) whereas in the generalised African epidemics, HIV prevalence is driven by heterosexual sex and that high levels of concurrent partnerships exacerbate its spread. The authors point to the WHO (1995) study that shows that 18%, 22% and 55% of men in Tanzania, Zambia and Lesotho, respectively, reported more frequent concurrent partnerships lasting more than a year, compared to adult men in Sri Lanka and Thailand, whose respective figures were 2% and 3%(Halperin and Epstein, 2007:2). Among women, the corresponding figures for Tanzania, Zambia, Lesotho, Thailand and Sri Lanka were 9% 11% 39% 0.2% and 1% respectively (WHO, 1995). The authors argue that such differences account for the severity of the HIV epidemic in Southern and East African regions relative to the low population prevalence in Thailand and Sri Lanka. While noting that polygamy (a form of concurrent partnerships) is common in North and West Africa, and yet these regions have lower HIV rates, the same authors hypothesize that this is due to the near universal circumcision among men and the tight control of women's sexuality in these predominantly Muslim populations (Halperin and Epstein, 2007:3).

Potts, Halperin, Kirby et al, (2008) use a number of studies to present an argument that treatment of STIs, use of vaccines and microbides do not work in preventing HIV infection. Instead, male circumcision and reducing multiple partners should be the target for HIV intervention programs. They present secondary evidence from Gray & Wawer (2008), a study which shows that of the six randomised control trials (RCTs) on the efficacy of treatment of STIs that had been conducted by 2008, only one trial, (Grosskurth, Mosha, Todd et al., 1995), showed a reduction in HIV transmission (40%). The other five, (Wawer, Sewankwambo, Serwadda et al., 1999; Kamali, Quigley, Nakiyingi et al., 2003; Gregson, Adamson, Papaya et al., 2007; Kaul Kimanji, Negelkerke et al., 2004 and Gray, Wabwire-Mangen, Kigozi et al., 2004), showed no change in HIV transmission after controlling for the treatment of STIs[19].

Potts and colleagues (2008) then go on to cite Cohen (2008) and Halperin et al, (2004) who argue that the use of microbides and vaccines has been inefficient and sometimes could be even harmful and that abstinence does not work, respectively (Potts et al., 2008:2). Using the above studies as evidence of what does not work, they turn to the successful RCTs for male circumcision and successful studies on reducing multiple partners (e.g. the Zero Grazing campaign in Uganda) to argue that multiple sexual partners and low levels of circumcision are the main drivers of the African HIV epidemic. .

Shelton (2007) talks about the 10 myths and the one truth about HIV epidemics in Africa, the truth being that the "*key driver*" of generalised HIV epidemics is concurrent partnerships (Shelton, 2007:2). He argues that potential drivers such as inequality and discrimination, poverty, treatment of STIs, among others are dwarfed by the impact of concurrent

[19] In their commentary, Gray and Wawer (2008) add that the one trial that showed the 40 percent decline in HIV transmission was conducted in a low HIV prevalence area, and thus the results can hardly be generalised to represent the effect STI treatment can have on HIV transmission in general.

partnerships on generalised HIV epidemics. The author argues that reducing multiple partnerships is the key, but this topic has been neglected "*because of the culture wars between advocates of condoms and advocates of abstinence, because it smacks of moralising, because mass behavioural change is alien to most medical professionals, and because of the competing priorities of HIV programmes*" (Shelton, 2007:3).

The bulk of the early literature in support of concurrency drew heavily on the Kretzschmar and Morris models, and used as evidence studies from different settings that could offer support for the theory – even though many of these did not test for the impact of concurrency on HIV transmission themselves.

This 'early support' of concurrency as a key determinant of HIV prevalence in sub-Saharan Africa triggered criticism, mainly citing a lack of direct evidence (Lurie & Rosenthal, 2009; 2010; Stillwaggon & Sawers, 2010; Stillwaggon, 2010) and the use of inappropriate assumptions (Stillwaggon & Sawers, 2010). Stillwaggon and Sawers (2010), for example, argue that most of the "*direct*" evidence in support of the concurrency argument stems from the Kretzschmar and Morris model from 1996, but that this model: "*makes several crucial but completely unrealistic assumptions: that women and men are equally likely to have concurrent partners, that all partners have sexual contact with every partner every day, that partnerships on average last only a few months, and that each sexual contact has a 5 per cent chance of transmitting HIV"* (Stillwaggon & Sawers, 2010: 9).

In a later piece (Sawers and Stillwaggon, 2010b) the authors present their "systematic" critique of the evidence against concurrency. They list the main assumptions in the 1996 Kretzschmar and Morris model and use a raft of ethnographic and epidemiological studies to present their evidence that:

1. The frequency of sexual contact used in the mathematical modelling was highly unrealistic (Lurie & Rosenthal, 2010; Carael, 1999; Harrison, Cleland & Frohlich, 2008; Shisana et al., 2005; Brewis & Meyer, 2005; Stewart, Morrison & White, 2002)

2. The transmission rate of 0.05 per cent assumed in the model is about 50 times higher than generally accepted 0.001 transmission probability per sex act (Deuchert & Brody, 2007)

3. The levels of concurrency assumed was also higher than those from epidemiological studies (Mishra & Bignami-Van Assche, 2009; Kapiga & Lugalla, 2002)

In other research contesting the concurrency hypothesis, another widely cited article failed to find evidence of the importance of concurrency in determining HIV prevalence in select sub-Saharan African countries (Senegal (Dakar), Benin (Cotonou), Cameroon (Yaounde), Kenya (Kisimu), Zambia (Ndola)) (Lagarde E, Auvert B, Caraël M, et al., 2001). This multi-centre study conducted under the auspices of WHO employed a questionnaire that documented the number (including the dates of start and end of partnership and the number of sexual acts) of concurrent partnerships over the 12 months preceding the survey. Using this data, an index[20] of concurrency was computed.

First, the study reported that there were no marked differences in the mean concurrency index for HIV negative and HIV positive people (ibid: 3). Secondly, the study explored the concurrency index as a risk factor for HIV infection and adjusting for age, marital status, chlamydia and gonorrhoea prevalence, there was no statistical relationship between HIV and

[20] This index was meant fulfil the following criteria: "*(i)summarizes the individual propensity to keep or dissolve on-going partnership(s) before engaging in another one; (ii) does not depend on the number of partnerships; (iii) does not depend on the length of partnerships; (iv) covers the 12-month period preceding the interview*." (Lagarde et al., 2001:2)

the concurrency index. The authors subsequently conclude that concurrency does not seem to be driving HIV prevalence in the selected countries.

This study has been widely cited by those that dispute that concurrency is significant as a driver of HIV (e.g. Stillwaggon, 2010; Lurie & Rosenthal, 2010; Tanser, Barnighausen, Hund et al., 2011). The underlying rationale for their support is that if concurrent partnerships are a major determinant of HIV prevalence, then cities that report the highest levels of concurrency should also report the highest HIV prevalence rates. This is not necessarily the case according to the results of this study. Yaoundé, with an HIV prevalence of about 5.9 per cent recorded the most concurrency. Ndola, which had the highest HIV prevalence in the sample, recorded concurrency levels lower than three of the centres.

In another study, using nationally representative DHS and AIDS Indicator Survey (AIS) data from 22 countries (21 from Africa), Mishra and colleagues could not find any correlation between HIV prevalence and concurrency at the community level or at the national level and neither among women nor among men (Mishra & Bignami-Van Assche, 2009). In this study, concurrency was measured as having two or more sexual partners over the past twelve months preceding the survey *and* if the sexual relationship with the current partner was longer than twelve months. It is thus not a clear indicator of concurrency per se, as people who report having had more than one sexual partner may have done so on a serial basis. At an individual level, no association between this measure of concurrency and HIV-status was observed after controlling for individual biomarkers such as age and education (Mishra & Bignami-Van Assche, 2009).

The same study also went a step further to test the relationship between concurrency and HIV, but this time at a community level. The study used the DHS and AIS population clusters, with each cluster considered a community. Concurrency prevalence rates per

community were computed as the percentage of those respondents in the cluster that, according to the authors' concurrency definition above, were in concurrent relationships. A comparison was then made between the respective concurrency prevalence rates and the corresponding population cluster HIV prevalence rate. No association of a link between community level concurrency and community level prevalence was found.

However, more recent literature in support of the concurrency argument argues that those who are sceptical about the role of concurrency do not actually present much relevant evidence (e.g. Mah & Shelton, 2011; Morris, 2009; Mah & Halperin 2010; Epstein & Morris, 2011). This literature puts forth a clearer argument that the link between concurrency and HIV prevalence lies in the subtle, yet critical interaction of the "*relationship between behaviour (at the individual level) and disease transmission dynamics (at the population level*)." (Morris, 2009:1). The current version of the concurrency argument emphasises that it is not one's own concurrency that renders one more exposed to HIV – it is the concurrency of those with whom one is having a relationship with that matters. Thus, showing that there is "no evidence" of a relationship between individual concurrency and individual HIV-status essentially misses the point (Mah & Shelton, 2011; Morris, 2009; Mah & Halperin 2010; Epstein & Morris, 2011). More so, while the Kretszchmar and Morris models may have relied on many assumptions that are not realistic, this just formed the basis of an argument that has since evolved (Epstein & Morris, 2011:5; Goodreau, 2011).

A recent study conducted in KwaZulu Natal, (Tanser, Barnighausen, Hund et al., 2011), however stands up to the refined recent concurrency argument. Using a rather complicated technique, the study first mapped each male respondent in the survey onto their geographical location (using their household's physical location). A questionnaire (administered in 2004) then collected sexual behaviour information for these men (such as multiple partners and any

concurrent sexual relationships). This information was then overlaid on the geographical map to have a "point" concurrency estimate measure (i.e. for every homestead on the map in which a man in the survey lived, there would be a concurrency measure[21]). This mapping covered all the homesteads in a rural community covering an area of up to 45.4km^2. Next, HIV incidence among women was tracked over a 5 year period (testing for HIV done in 2004 and then in 2009) and this was compared to the point concurrency estimates in the community.

Over the 5 years, HIV incidence among women was 3.60 cases per 100 person years. Among sexually active men, the average point prevalence of men reporting concurrent partners in 2004 was 23.2 per cent and the median number of reported lifetime partners was 5 (Tanser et al., 2011:3). Adjusting for individual-level socioeconomic, demographic and environmental factors associated with HIV acquisition, a high prevalence of concurrency in the same local community was associated with haphazard HIV infection. The study found no additional risk among women arising from living in a specific locale where concurrency was high (and 61 per cent of the women in the survey had sexual partners within the immediate village) with high rates of concurrency.

Evidence from discordant couples has been put forth in support of the concurrency theory (Mah & Shelton, 2011:3). Using nationally representative DHS data from Burkina Faso, Cameroon, Ghana, Kenya and Tanzania, De Walque (2007) finds that for all the countries, at least two-thirds of the infected couples (sample size ranged from 1 086 couples in Kenya to 2 214 couples in Tanzania) are discordant (de Walque, 2007:5). In a cohort study in Tanzania, Hugonnet and colleagues (2002) observed a 67 per cent seroconversion rate among

[21] This concurrency measure was calculated using a weighted two-dimensional standard Gaussian kernel of search radius 3km (i.e. different weights were applied to each point's correspondent sexual behaviour, with the largest weighting given to those homesteads at the centre (hub) of the village).

concordant HIV negative couples and suggest that this could be because of "*extramarital exposure*" Hugonnet, Frank, Todd et al., 2002:6). In another, much larger study, approximately 51 900 couples of unknown HIV-1 status were tested and 6 543 HIV-1 discordant couples were found (12.6 per cent) (Lingappa, Kahle, Mugo et al., 2009). Mah and Shelton (2011) argue that high levels of discordance reported widely in sub-Saharan Africa and the high rates of seroconversion among HIV negative concordant couples is evidence of the significant role concurrency plays in the region (Mah & Shelton, 2011:3)

Debate on the concurrency debacle still rages on, but in the balance of the evidence, only one study addresses the more recent concurrency argument that it is strictly speaking not the individual's concurrency that matters in determining their HIV risk, but that of their sexual partners. However, even in this study, proxy measures are used.

3.3 Condom use, male circumcision and HIV

Going back to our wider discussion on the relationship between individual behaviour and HIV, Talbott (2007) makes the claim that sexual behaviour is responsible for the high prevalence rates in Africa, but that the issue is not so much about concurrency as it is about the number of commercial sex workers (Talbott, 2007). In this research, the author argues that the HIV rates in Southern Africa are not disproportional, if one compares the number of commercial sex workers as a percentage of the female adult population (Talbott, 2007). The research makes the point that sexual mixing between low-risk populations and high-risk populations, especially commercial sex workers, necessarily exacerbates HIV in Southern Africa. Using cross-country multivariate regression analysis the study models HIV prevalence in African countries as a function of the prevalence of sex workers, infection rates among sex workers in each country among other explanatory variables.[22] The author

[22] percentage of population that is Muslim, GDP, Gini coefficient and the illiteracy rate

concludes that HIV in sub-Saharan Africa is largely due to the numbers of sex workers and the HIV prevalence among them.

This study however has a couple of shortcomings. Firstly, and crucially, the data on the prevalence of commercial sex workers is poor. Due to difficulties in obtaining accurate data on the number of commercial sex workers in different countries, the paper uses estimates, but sometimes from highly localised areas, and these are then generalised to city-level and country-level estimates. While the research uses the best data available, the data are not that great. Secondly, as the paper points out as well, for every heterosexual contact with a commercial sex worker there is a man, and without any analysis of the behaviour patterns of the men who frequently pay for sex, there is only so much that can be drawn from the prevalence of sex workers in a country alone.

In a comparison of the epidemics in Cambodia and South Africa, Buvé concludes that the lack of condom use by sex workers in South Africa, as opposed to those in Cambodia is what could have made the South African epidemic bigger than the Cambodian epidemic (Buvé, 2001). This study strengthens the argument that in order to make accurate correlations between the prevalence of prostitutes and the size of the epidemic, the rates of condom use in the sexual encounters may actually play a more central role than the mere prevalence of commercial sex workers.

Research suggests that condom use may reduce HIV infection by up to 69 per cent (Szabo & Short, 2000). In a study among discordant couples, consistent use of condoms was associated with a 69 per cent smaller HIV transmission probability (Szabo and Short, 2000). In many countries, condoms are prescribed as the single most effective way to prevent HIV transmission among sexually active individuals (e.g. www.avert.org/condom.htm). The actual protective effect could be higher and it is likely that this low level of protection observed in

the study may have had to do with the over reporting of actual condom use among the respondents. Although condoms are highly effective at reducing HIV transmission, their impact on generalised epidemics has not been as strong as it can potentially be if everyone used them. Shelton, (2007) argues that although condom use, especially by sex workers may have an impact on generalised epidemics, they have a limited impact in the same epidemics because many people do not like them for varying reasons including imperfect protection (Shelton, 2007:2).

Using focus group discussions from South Africa, Zimbabwe and Lesotho, among other research objectives, Kacanek, Dennis, Sahin-Hodogluigil et al (2012) probe the reasons why condom use is low among a sample of HIV negative men and women. The main reasons for not using condoms were disruption of romance, mistrust of condoms and sabotage, with the responses such as the following bearing testimony to those reasons: "*The condom requires a lot of work, especially for me. When I am tired and in the mood to have sex . . . the condom [is] something that I don't agree on*" (man, Harare). "*There is no one to trust if you find yourself using condoms on your wife at home and with one from the world [extramarital partner] you do not. That's impossible* (man, Harare). "*He does . . . things at times tearing the condom so that you have sex with him thinking that you are protected when you are not*" (woman, Harare) (Kacanek, Dennis Sahin-Hodogluigil et al., 2012:8).

Male circumcision is another individual biomedical factor widely linked to HIV infection (Auvert, Taljaard, Largarde, et al., 2005; Bailey, Moses, Parker, et al., 2007; Maughan-Brown, Venkataramani, Nattrass et al., 2011; Halperin & Epstein, 2007; Weiss et al., 2008; Klausner et al., 2008). The inner side of the foreskin is rich in Langerhans cells and these cells are an important entry point for HIV (Szabo, 2000; Soto-Ramirez, 1996). Without the

foreskin therefore, the surface area under exposure is smaller and this can protect against HIV.

Evidence of the protective effect of circumcision from the studies that have tested it is powerful. In three randomised controlled trials, one in South Africa, one in Kenya and another in Uganda, the average protective effect was greater than 50 per cent (Auvert, Taljaard, Largarde, et al., 2005; Bailey, Moses, Parker, et al., 2007). Auvert et al (2005) use a sample of 3 274 men, grouped into a control and intervention group with follow up visits at three, 12 and 21 months. While controlling for condom use, health-seeking behaviour, age and five sexual behaviour variables[23] the authors find an incidence rate of 0.85 per 100 person-years in the intervention group and 2.1 per 100-person years in the control group – a protective effect of 61 per cent. The study concludes that the extent of protection from male circumcision is as high as what a high efficacy HIV vaccine would provide. In Kisumu, Kenya, Bailey et al., (2007) found a protective effect of about 53 per cent among 1 391 circumcised men.

In the multicentre study comparing the determinants of HIV among four cities cited earlier, the authors find that lack of circumcision was significantly more pronounced in the high HIV prevalence areas than in the low prevalence areas (Buvé, 2001). This study, however, does not isolate the protective impact of circumcision alone, it merely shows that male circumcision, among other factors, was more common in the low prevalence cities than in the high prevalence cities.

A more recent study conducted in Cape Town also finds a traditional circumcision to have a protective effect against HIV among men (Maughan-Brown et al., 2011). Using data from

[23] These were: having a spousal partner, number of non-spousal sexual contacts, having at least one sexual contact without a condom, having at least one sexual relationship with at least one sexual partner and the number of sexual contacts.

473 young adults in Cape Town, this study finds those with partial circumcision with a 7 per cent greater risk of being HIV-positive compared to fully circumcised men (ibid:1). This study uses the Cape Area Panel Study (CAPS) and in wave 5 of the study, respondents were asked whether they were circumcised. For those that were, an additional follow up question probing how much of their foreskin was removed was asked (ibid: 2). Fully circumcised men were those that reported that they were circumcised and that "*the entire foreskin*" was removed while partially circumcised men were those that responded yes to "*only some of the foreskin*" being removed (loc. cit). Despite no statistical differences in the sexual behaviour of those partially circumcised and those fully circumcised, those that were fully circumcised had a lower risk of being HIV-positive – attesting to the strength of male circumcision as an HIV prevention method at the individual level.

Research also argues that male circumcision has both a population level effect and an individual-level effect (Halperin & Epstein, 2007; Weiss et al., 2008; Klausner et al., 2008). Due to the lower prevalence rates among circumcised men (individual effect), it is logical that circumcision results in a secondary, population level protective benefit arising from the fact that circumcised men have lower prevalence and this reduces the chances of HIV transmission among the women they have sex with as well.

In a literature review on the studies that investigate the efficacy of male circumcision, Klausner concludes that the actual protective effect of male circumcision is about 75 per cent, owing to the large numbers of men in the RCTs that are not captured in the follow up and those who are circumcised during the follow up period (Klausner, 2008). On top of being an effective HIV prevention method, various studies have also supported male circumcision by arguing that it is cost effective (Gray, 2007; Kahn 2006; Auvert 2008; Fieno, 2008; Bollinger; 2009).

However, although there seems to be little doubt that male circumcision does reduce individual level female to male HIV transmission, it has been suggested that this may not necessarily translate to lower HIV prevalence rates at the population level in the long-term (Garenne, 2009). This research uses DHS data from 13 African countries to compare HIV prevalence between circumcised and uncircumcised men. In eight of the countries, Garenne (2009) fails to find a statistical difference between HIV prevalence among circumcised men and uncircumcised men, while in three countries (Cameroon, Lesotho & Malawi) circumcised men had higher HIV prevalence. Only in two countries (Kenya and Uganda) was there evidence of a protective effect at population level from male circumcision. Warren (2010) also finds circumcised men in Lesotho to be at higher HIV risk than uncircumcised men after controlling for age, location and education (ibid:5). The author explains these findings in terms of higher sexual risk-taking among circumcised men, which then negates any benefits that may accrue from circumcision.

3.4 Early age of marriage and age at first sex as determinants of HIV

An early age of marriage and an early age of first sex have been found to be underlying determinants of HIV infection in southern Africa (Clark, 2004; Pettifor, Audrey, van der Straten et al., 2004) and elsewhere in sub-Saharan Africa (Bongaarts, 2007; Robert, Ronald, Sewankambo et al., 2003). Clark (2004) investigates the relationship between an early age of marriage (defined as marriage before the age of 20) and HIV among sexually active adolescent girls in Zambia (762) and Kenya (1137). The author finds that married adolescent girls in urban centres (Kisumu and Ndola) in the two countries had higher HIV prevalence although they, on average, reported fewer sexual partners than single, sexually active girls. The author however finds that in both countries, marriage increased frequency of sex, decreased condom use and eliminated the option of secondary abstinence among girls (Clark,

2004). More-so, adolescent girls were married to older men, who on average had a three-fold higher HIV prevalence than young girls' boyfriends.

In a contrasting set of findings however, Bongaarts (2007) finds that a positive correlation between age at median age at first marriage and HIV prevalence exists, as well as a positive correlation between the time between age of sexual debut and age of marriage. The study uses ecological studies from 33 sub-Saharan African countries and explains this finding in terms of higher HIV exposure per year for unmarried women compared to married women.

3.5 Discussion

Proponents of a behavioural and biomedical approach to HIV prevention argue that:

- The evidence that shows poverty is a lead driver of HIV is highly contestable and inadequate. There are many counter examples and studies where the poor or poorest are not necessarily the most vulnerable to HIV
- The argument that gender inequality drives women to transactional sex (and any other HIV risk enhancing behaviour) is simplistic and ignores women's own agency and evidence from discordant couples that show that a large proportion of men, even migrating men, in fact contract HIV from their wives
- Where adequate data is available, behavioural and biomedical approaches to fighting HIV have shown to work, in particular male circumcision, reduction in multiple partners and condom use

In chapter 2 we discussed gender, poverty and other structural factors as determinants of HIV and in this chapter discuss the impact of concurrency and a number of behavioural variables such as male circumcision and age at first sex on HIV. It is still unclear whether behavioural and biomedical issues matter in shaping the HIV epidemic, or structural issues such as

poverty and inequality are what is driving the HIV epidemic in Southern Africa. It would appear that this is an empirical question and in the next chapter, we perform our own quantitative analysis of this relationship between structural and individual level behaviour with HIV-status using nationally representative data.

Chapter 4. An empirical assessment of the impact of structural factors and behavioural factors on HIV-status among men and women in Lesotho, Swaziland and Zimbabwe

4.1 Introduction

In this chapter, we continue the discussion on the main determinants of HIV under hyper-epidemic conditions, but using a quantitative approach. We assess the empirical evidence from southern Africa on the potential determinants of HIV-status at an individual level. Using nationally representative cross-sectional DHS data, we probe the strength of relationship between structural factors such as poverty, education and place of residence and HIV-status and that between behavioural and biomedical factors such as the number of lifetime partners, age at first sex and age of marriage and HIV-status. By carefully tracking the evolution of coefficients as more variables are included, and paying special attention to women, we explore the extent to which nationally representative data can shed light on the relationship between HIV status and socio-economic and demographic factors.

The chapter focuses on the relationship between SES and HIV infection on individuals from Southern Africa, with the spotlight falling strongly on women as they bear the brunt of the African HIV epidemic. We run pooled logistic regressions, first for both men and women, then later for female respondents only, on the measures of SES on HIV-status, while controlling for an array of covariates.

4.2 Sample

We limit the data analysis to DHS Lesotho (2004), DHS Swaziland (2006) and DHS Zimbabwe (2005/2006) because these are the countries in Southern Africa that have DHS

surveys that include HIV testing and test results that can be anonymously linked to the background socio-economic characteristics of the individuals.

All three studies employ a two-stage sampling design and approximately 10 800 households were selected for the Zimbabwe survey, 9 000 households for the Lesotho survey and 4 800 households for the Swaziland survey (MOHSW [Zimbabwe], 2006; MOHSW [Lesotho], 2006; CSO [Swaziland] 2006-07). In all analyses, we restrict age to between 15-49 for consistency and comparability with most other studies.

According to the DHS protocol, blood was collected from respondents using a finger prick lancet and collected onto filter paper. HIV testing was done using two HIV enzyme immunosorbent assays based on different antigens and specimens with an initial indeterminate result were resolved by Western Blot testing. For quality control, all HIV-positive specimens and a sample of HIV-negative specimens (usually 5 per cent) were subject to a re-test at a different laboratory using the same testing procedure (Macro International 2007). More information on the type of questionnaire, sample design, and other technical information is discussed elsewhere (CSO, Zimbabwe and Macro International 2007; Ministry Of Health and Social Welfare (MOHSW), Lesotho.

4.3 Operationalising variables

Understanding the relationship between SES and HIV poses some difficulties for research, the first being a conceptual or definitional problem. SES does not have a conventional definition so across different quantitative studies different measurements are in use (Wojciki, 2005:6). These differences make it difficult to assess the consistency, and sometimes validity, of different conclusions, as researchers base conclusions on differently constructed measures. Table 4.1 below shows some of the measurements used for SES in the literature on SES and HIV that we reviewed in chapter 2. The measures of SES vary from simple measurements

such as having enough food over the past week or not, to SES measures that vary depending on occupation, age and life stage.

Table 4:1: SES measurements in recent literature

Study	**Measurement of SES**
Amuri et al., 2011: Socio-economic status and HIV/AIDS in Tanzania	Food insufficiency in the past week ("had enough food in the past week" versus " did not have enough food in the past week)
Tladi 2006: Poverty and HIV/AIDS in South Africa: an empirical contribution	Monthly income. 0-R600 "very poor"; R601-R1000 "poor"; R1000+ "not poor"
Msisha et al., 2008: Socioeconomic status and HIV sero-prevalence in Tanzania: a counterintuitive relationship	Educational attainment in years of education, occupational status, rural/urban residence and household living standard measure
Mishra et al., 2008: HIV infection does not disproportionately affect the poorer in sub-Saharan Africa	DHS provided wealth index. Uses a composite of household assets and then computes a cumulative wealth measure using principal component analysis (PCA)
Barnighausen et al., 2008: The socioeconomic determinants of HIV incidence: evidence from longitudinal, population-based study in rural South Africa	Educational attainment, household wealth and per capita household expenditure
Hargreaves et al., 2002: Socioeconomic status and risk of HIV infection in an urban population in Kenya	Study employs a 3 stage process to calculate SES First stage uses household amenities of running water and electricity. Those individuals with no water and no electricity in their households score 0, either water or electricity, 1 and those with both water and electricity score 2. Second stage uses educational attainment. Individuals with incomplete primary score 0, completed primary education, 1 and secondary education and higher score 2 Third stage uses employment status. Not employed 0, low-income employment, 1 and high income employment 2. Final SES ranks are then computed from the above scores. For women and male students, a cumulative score of 0 or 1 is

	considered "low SES", cumulative score of 2= "medium SES" and a cumulative score of 3 or 4 = "high SES". For other men, a cumulative score of 0-2 ="low SES", 3-4 = "medium SES" and 5-6 considered "high SES"[24]

The above studies typically show the variation in the methods in use to measure SES. This variation in measures for SES has generated some discussion into which measures of SES are the most appropriate (e.g. Braveman, Cubbin, Egerter et al., 2005; Vyas & Kumaranayake, 2006; Howe, Hargreaves, Ploubidis et al., 2010). Braveman and colleagues (2005) highlight that SES is "*complex and multifactorial*" and that a lot of the available data available to researchers who wish to investigate the links between SES and health outcomes often has many limitations (Braveman et al., 2005:1). In this widely cited article, the authors recommend an "*outcome-and-social group-specific*" approach in measuring SES that involves: "*(1) considering plausible explanatory pathways and mechanisms, (2) measuring as much relevant socioeconomic information as possible, (3) specifying the particular socioeconomic factors measured (rather than SES overall), and (4) systematically considering how potentially important unmeasured socioeconomic factors may affect conclusions.*" (Braveman et al., 2005:1)

In recent research, this approach has been widely applied, with researchers picking SES variables that are expansive and capture individual- as well as community-level (aggregate) aspects of SES (e.g. Johnson et al., 2009; Msisha et al., 2008; Krieger, Williams & Moss, 1997; Armstrong, 2000).

[24] A male student with just running water and electricity would be considered to have medium SES while a man who was not a student and had the same characteristics would be considered to be of low SES.

Household wealth and education have been used most widely as measures of SES in research that explores the links between SES and HIV or HIV risk behaviours in Southern Africa (see e.g. Hargreaves et al., 2007; Lopman et al., 2007; Dinkelman eta al., 2007; Johnson & Way 2006). Employment status, where available, also provides important information on the individuals, and the rationale is that occupation is an even more direct proxy for expenditure than other wealth measures, such as household wealth, which may not readily reflect the spending power of the individuals in question (Hargreaves et al., 2002; Hope, 2000; Ayisi et al., 2002). It may also influence the ability to seek treatment (and this could affect HIV acquisition if STIs are not treated fully, for example). In chapter 2, we also presented literature that showed varied HIV rates between individuals in urban areas and rural areas (e.g. Lewis et al., 2006).

That said, there is also research highlighting some of the problems of using expansive measures of SES. Using data from the Malawi 2004/05 DHS (11 280 households) Howe et al., (2010) compares the DHS-provided wealth index to four subjective measures of socioeconomic position (SEP) namely perceived adequacy of food consumption, perceived consumption adequacy, perceived income sufficiency and an economic ladder question. These subjective SEP questions are based on the respondents' own perception of where they think they fall, socioeconomically for example, in the community. Subjective food adequacy questions involve asking respondents if they feel that they have adequate food and the ladder SEP question involves respondents being shown a pictorial ladder, with the bottom step representing the 'most poor' and the highest representing the 'wealthiest' and then asking the respondents to pick which step they think they fall on.

The authors then compare the DHS wealth index to these subjective measures of SEP in terms of how they are in agreement in terms of the ranking of households on a cumulative

score of wealth, how close they are in respect to the US$1-a-day poverty definition based on consumption and the socio-economic processes giving rise to the SEP scores (e.g. whether the respondent was educated or not, employed or not, and so forth).

The authors find that the household classification under each subjective score differed considerably to that of the wealth index, with three of the SEP measures (perceived food and income sufficiency indicators and the economic ladder question) accurately identifying more households living under less than a dollar than those predicted by the wealth index. The wealth index was also strongly influenced by community-level wealth levels (due to questions such as "do you have access to running water" (or to electricity)), while the subjective SEP measures were independent of community wealth level. Subjective SEP measures however carried inherent reporting biases and larger inconsistencies.

4.4 Measures

We measure educational attainment using years of education. This is a continuous variable that captures the number of years of education attained, ordered from zero for no education to the highest level of education attained. We do not assume that the relationship between education and HIV is linear, so we also include a second education measure, which is the square of the years of education variable. This variable will enable us to observe any evidence of a turning point in the relationship between education and HIV.

We measure employment status using a dummy variable distinguishing between those who were employed at the time of the survey and those who were not. Employment status affects or contributes to the SES of an individual, and as discussed earlier, may independently affect HIV risk. The last measure of SES we use is place of residence. In line with the discussion of the literature that recommends the use of an area-level measure of SES, we include a place of residence variable, classified as rural or urban and the reference category is urban residence.

We measure age as a continuous variable, representing the age of the individual at the time of survey. We do not make any prior assumption about the relationship between HIV and age so we include a secondary age variable, age squared. Age at first sexual intercourse is a continuous variable and so is age at first marriage and the number of lifetime sexual partners.

As a proxy for SEP, we use the DHS-provided household wealth index. This wealth index is constructed using Principal Component Analysis (PCA) and is developed using a composite measure of ownership of household items to construct a cumulative living standard measure that places individuals on a continuous scale of relative wealth. In the three countries, respondents were asked about the ownership of 12 household assets[25]. The individual wealth scores for each household are then computed using PCA and all the members from the same household allocated the same household score. Once each member is assigned a relative wealth score, quintiles are then constructed using the distribution of the household population.

A wealth index avoids the many problems income measures have such as seasonal volatility and reporting bias (Rutstein & Johnson, 2004:3). In all regressions, we use household wealth measured by population quintiles, quintile 1 for the poorest 20 per cent of the population up to quintile five, which represents the wealthiest 20 per cent of the population. In the regression models, the base category (omitted) will be the lowest wealth quintile[26].

Used this way, the wealth quintiles are a measure of both relative poverty and absolute poverty. Here is why. For each country, the total sample made up of all the people in wealth quintile one through to quintile five will contain the entire wealth spectrum in the country. In

[25] Type of flooring, water supply, sanitation facilities, electricity, radio, television, telephone, refrigerator, type of vehicle, persons per sleeping room, ownership of agricultural land and domestic servant.

[26] An alternative would be to use a continuous variable ranked from the lowest household wealth value to the highest, but this potentially suffers from one major problem. Using a continuous measure of household wealth imposes a linear relationship between the logit of HIV-status and household wealth, and this may not be the case.

this total sample, there will be those that are poor and those that are wealthy. Naturally, in some of the quintiles (most likely one and two), we would only find poor people and in others (most likely four and five), rich people. So while the difference between those people in quintile 1 and 2 may only show relative inequality (i.e. they are different wealth classes but both comprising poor people), that between quintile one and say quintile five would separate those that are poor from those that are not.

In the pooled sample however, the wealth quintiles (defined separately for each country) are only a measure of relative wealth. This is because for each country, the absolute wealth levels for each country are different, but regardless of this level of wealth, the sample is split into five wealth bands. So, while in a very poor country those in quintile one may be so poor they may often go without food, those in the same quintile in a better off country may not be in such desperate straits. In the pooled sample therefore, the wealth quintiles only serve as a measure the impact of relative wealth on HIV-status – whether being relatively poorer or richer than others makes one more or less vulnerable to HIV infection, regardless of the absolute wealth level.

Following from the above paragraph, for us to be able to analyse the results in the logistic regressions, we need a clearer understanding of the people in these quintiles – most importantly by country and by gender. Country-level analyses are important because despite there being a Southern African epidemic, different features of this sub-regional epidemic are more salient in some countries than another. The countries themselves happen to have structural differences, with Zimbabwe, for example being a wealthier country than the other two but struggling with governance issues whereas Lesotho has better governance but is a poorer country. The following section looks at and highlights these inter country differences

to shed light on the kinds of people in each of the wealth quintiles to give a clearer understanding of the regression results.

4.4.1 A glance at the country profiles and composition of wealth quintiles

In 2005, WHO compiled a number of country reports to represent the state of affairs in the health sector for different countries and outline some of the challenges these countries faced in fighting the HIV epidemic. Since the data from the countries we look at is from 2004 to 2006, these country reports give a relevant representation of the macroeconomic conditions in these three countries at about the time the DHS data were collected. Table 4.2 below presents some of the demographic, socioeconomic and HIV indicator data for Lesotho, Swaziland and Zimbabwe (latest available estimates as of 2005).

Table 4:2 Demographic, socioeconomic and HIV indicator data, Lesotho, Swaziland and Zimbabwe

Indicator[27]	**Lesotho**	**Swaziland**	**Zimbabwe**
Total population (millions)	2.2	1.1	12.9
Proportion of population urbanised (%)	18.2	23.8	35.8
Life expectancy at birth (years)	38	40-45	37
GDP per capita ($US)	402	1096	1385
Government budget spent on health (%)	6.9	7.5	12.2
Per capita expenditure on health ($US)	17	66	118
Human Development index	0.497	0.498	0.505
Adult HIV prevalence (15-49) (%)[28]	26-31	37-40	21-28
Reported condom use at last high risk sex (15-24 years) male (%)	53	NA	69
Reported condom use at last high risk sex (15-24 years) female (%)	53	NA	42

Source: WHO, (2005a:1); WHO, (2005b:1); WHO, (2005c:1)

Of the three countries, Lesotho has the lowest HDI ranking, and generally fares worst on all demographic and socioeconomic indicators. It has a largely rural population, with about 80

[27] The estimates for the different indicators are not all from the same year. They are all from 2002-2005. For the specific year to which the indicator refers, as well as the source of the estimate, please see the referenced sources below the table.

[28] Data given as a range in line with the original UN report from which the data comes from.

per cent of the population living in the rural areas. Its GDP per capita is less than half that of Swaziland and Zimbabwe, and the amount spent on healthcare is the lowest for all three countries – both as a percentage of GDP and per capita nominal expenditure. According to the 2004 Lesotho DHS, there were no marked differences in the proportion of young women and men who used condoms during the last high risk sexual encounter – defined as one in which there was a direct exchange of money or other gifts for sex. The most vulnerable group is young people, particularly young women between the ages of 20-29 (WHO, 2005a:2). Lesotho's first AIDS case was diagnosed in 1986 and since then, the government has faced huge challenges to come up with a solid action plan to tackle HIV due to inadequate finance and infrastructure (http://www.avert.org/aids-lesotho.htm). HIV prevalence steadily rose and reached a peak around 2005, and there has not been a major change since then (ibid). Most of the HIV prevention methods have been targeted towards sex workers, migrant labourers, factory workers, long distance truck drivers and taxi drivers (Population Services International, 'Southern Africa regional Social Marketing Program, 2005)

In 2009, a study performed a modes of transmission analysis in Lesotho (NAC, 2009) and found that there was a high frequency of multiple and concurrent partners. In that year, this study found that nearly half of all sexually active adult males and 26 per cent of all adult sexually active women reported having sex with more than two partners in the past 12 months. Subsequent analysis also exposed a number of cultural and socioeconomic factors that contribute towards a high HIV prevalence (MOHSW, 2009) including high levels poverty and inequality, traditional and cultural practices regarding female roles that tend to fuel discrimination towards women and difficult access to healthcare services (UNAIDS, 2012:31)

The country indicators for Lesotho thus paint the picture of a poor country, with very limited investment in healthcare. WHO (2005a) points out some of the key challenges in HIV

prevention in the country include seriously inadequate training of healthcare workers, lack of laboratory capacity to diagnose and monitor people on antiretroviral therapy and a constrained access to essential drugs and less than adequate communication strategies on HIV/AIDS (WHO, 2005a:2). Socially and culturally, the uptake of safer sexual behaviours appears to be slow.

For most macroeconomic indicators, Swaziland slots in between Lesotho and Zimbabwe, with a higher urbanised population, much higher per capita GDP, a higher proportion of government budget spending on health than Lesotho. In 2005, Swaziland had the highest life expectancy at birth estimates of the three countries, and per capita, spent nearly four times more on health than Lesotho. It however was (and still is) the most heavily HIV afflicted country in the world. HIV prevalence rose from a mere 3.9 per cent in 1992 to 42.6 per cent in 2004 (Mathunjwa & Gary, 2006:1). A strong adherence to cultural practices such as dominance of men over women and polygamy have been cited as some of the underlying reasons for the high HIV levels in the country (Manthunjwa & Gary, 2006; Ziyane & Ehlers, 2007). Ziyane and Ehlers (2007) for example, investigate Swazi men's contraceptive knowledge attitudes and practices and find that the men indicated that they were the sole decision makers about sexual health matters (ibid:1).

The WHO cites a lack of human resource capacity as a major challenge in increasing the scope of ARV therapy in the country (2005: 2). Due to the high rate of mortality from AIDS, the government was also failing to replace lost healthcare workers and recruitment and retention of staff was restricted by poor working conditions (loc. cit). HIV prevalence is worst among young women between 20-29 years old, with a national prevalence of 56 per cent among these women in 2004 (ibid:1).

In comparison to Lesotho then, Swaziland is a slightly wealthier country, able to spend more on healthcare and with a better health infrastructure but faces a serious human resource

challenges. It appears that a strong cultural dominance of men over women exists and that polygamy is common.

Generally, Zimbabwe has the best macroeconomic and demographic indicators of the three countries, but massive governance deficiencies. It has the highest HDI index and proportionally spends more on health than the other two countries. The proportion of urbanised population is the highest and so is the GDP per capita. As of 2006, the government spent nearly 7 times more on health per capita than Lesotho and about one and half times more per capita than Swaziland. The GDP per capita in the country remained the highest, despite nearly a decade long recession leading up to 2005 (when this report was made). Although health infrastructure was well developed in 2005 (in terms of availability of clinics, hospitals and transport networks to these places), WHO (2005c:2) reports major challenges in running these centres, with dwindling health workers due to emigration to other countries as well as a massive drop in donor funds due to the political and economic situation (ibid: 2). The same WHO situation report also cites an acute shortage of drugs, supplies and replacement laboratory equipment due to rising costs and unavailability of foreign exchange. HIV varies greatly by residence, with the most vulnerable people found in large-scale commercial farms, administrative centres, high-growth areas outside cities and towns and state lands and mines (loc. cit).

One of the first elucidatory research articles on sexual behaviour in Zimbabwe was conducted in 2005 (Mahomva, Greby, Dube et al., 2005). The aim of this paper was to bring together data from different reports and generate a picture of HIV prevalence and trends in Zimbabwe from 1997-2004. The study uses data from four antenatal clinic (ANC) surveys carried out between 2000 and 2004, four general population surveys from 1999-2003, two local studies from 1997 to 2003 and service statistics from 1990 to 2004 (ibid:1). Data from these sources highlighted a decline in HIV prevalence, and among some of the reasons for this decline were

a reduction in age at sexual debut (specifically sex before the age of 15) and a reduction in the proportion of men and women between 15-29 reporting non-regular sex partners over the past year (loc. cit). The study concluded that condom use with non-regular partners had remained high since 1999, despite HIV testing and counselling increasing in the same period. Other research also echoed the same behaviour-led decline in HIV prevalence in Zimbabwe. Using data from elsewhere in the country, Hallet, Arberle-Grasse, Bello et al (2005) corroborate the above findings, concluding that HIV prevalence declines in Zimbabwe could not have been achieved without the adoption of safer sex methods (ibid:1). Using survey data and mathematical back calculation techniques, Gregson, Gonese, Hallet et al., (2010) provide the "*first convincing evidence*" that HIV declines in Zimbabwe were accelerated by changes in sexual behaviour. This study shows that there has been declines on the numbers of people reporting casual partners from the late 1990s as well as an increase in condom use with regular partners between 1998 and 2007 (ibid: 1).

Notwithstanding the highly conservative culture that has been described as resistant to change and the governance issues that have resulted in the cutting of donor aid to fight HIV, there seems to be heightened awareness among Zimbabweans about HIV and this has been cited as one of the reasons why HIV has been on the fall.

Looking at the countries at the time of the DHS surveys, one was relatively wealthy, with somewhat better infrastructure, but plagued by governance and economic problems while the other two were poorer with Swaziland suffering from a human resource problem while Lesotho was suffering from a general capacity problem with constrained human and capital resources.

These country differences suggest that the population quintiles for the different countries probably contain people of differing socio-economic status. In the following section, we present a picture of who falls in these households. We outline some of the characteristics of

those households that lie along the wealth quintile cut-off points of the wealth index for each of the countries. Table 4.3 below shows the ownership of select household assets, availability of some social amenities as well as employment data (where available) for each of the countries. The households we present, for each country are the poorest household and a household at the 20, 40, 60, 80 and 100th percentile. These households are of course not necessarily representative of the quintile, but they provide a flavour of the socio-economic contours within the different countries and serve as qualitative markers of the nature and spread of the wealth distribution.

The first three households in the table are the poorest surveyed household in Lesotho, Swaziland and Zimbabwe. These three households are all rural, with the main family dwelling made from locally available mud, earth or dung. The households are relatively large, with at least seven household members and none of the households owns a radio, television, fridge, bicycle or car. There is no recognised toilet system, so respondents most likely use the bush as a toilet. There is no electricity in any of the houses. In Lesotho and Zimbabwe, the households fetch water from a protected well, while in Swaziland the main source of water is a nearby dam. There is no recorded occupation for any of the household heads in the three households.

There is a slight improvement in ownership of household assets as we move to the households at the cut-off income level for quintile 2. In the Swaziland and Zimbabwe DHS, the poorest household in this quintile owns a radio, while in Zimbabwe this household also owns a bicycle and has a traditional pit toilet. In Lesotho and Swaziland, there is a recorded occupation for the household head, skilled and self-employed (and both household heads are in their working age).

Table 4:3 Depiction of the individual households at the wealth quintile cut-off points in Lesotho, Swaziland and Zimbabwe

Wealth cut off (Percentiles**)**	**Building Materials**	**Household Assets**					**Amenities**				**Household characteristics**			**Country**
	Floor	*Radio*	*TV*	*Fridge*	*Bicycle*	*Car*	*Toilet*	*Elec*	*Water*	*How difficult is it to access health services?*	*Occupation of House hold head*	*Age and sex of household head*	*Household size*	
0	Mud/earth /dung	✗	✗	✗	✗	✗	No facility	✗	Protected well	Small problem	-	Male 37	8	**Lesotho**
	Other	✗	✗	✗	✗	✗	No facility	✗	Dam	Small problem	-	Male 55	7	**Swaziland**
	Mud/earth /dung	✗	✗	✗	✗	✗	No facility	✗	Protected well	Small problem	-	Female 41	8	**Zimbabwe**
20	Mud/earth /dung	✗	✗	✗	✗	✗	No facility	✗	Public tap	Small problem	Agriculture, self employed	Female 33	2	**Lesotho**
	Cement	✓	✗	✗	✗	✗	No facility	✗	Tanker truck	Big problem	Skilled	Female 26	5	**Swaziland**
	Mud/earth /dung	✓	✗	✗	✓	✗	Traditional pit toilet	✗	Open well	Small problem	-	Female 74	10	**Zimbabwe**
40	Cement	✗	✗	✗	✗	✗	Traditional pit toilet	✗	Open well	Big problem	No work	Female 59	7	**Lesotho**
	Cement	✗	✗	✗	✗	✗	Traditional pit toilet	✗	Piped in	Big problem	-	Female 61	9	**Swaziland**
	Cement	✗	✗	✗	✗	✗	No facility	✗	Open well	Big problem	Agriculture Self-employed	Female 63	4	**Zimbabwe**

60	Vinyl	✓	×	×	×	×	No facility	×	Public tap	Small problem	Professional, technical, managerial	Male 41	4	**Lesotho**
	Cement	✓	✓	×	×	×	Traditional pit toilet	✓	Protected well	Big problem	Services	Female 47	6	**Swaziland**
	Cement	✓	✓	×	✓	×	Traditional pit toilet	×	Open well	Big Problem	Sales	Female 40	6	**Zimbabwe**
80	Cement	✓	×	×	×	×	Ventilated pit latrine	×	Piped in	Small problem	Professional, technical, managerial	Male 29	3	**Lesotho**
	Cement	✓	✓	✓	×	×	Ventilated pit latrine	✓	Piped in	Small problem	Skilled	Male 38	6	**Swaziland**
	Cement	✓	✓	✓	×	×	Other	✓	Piped in	Small problem	-	Female 31	5	**Zimbabwe**
100	Tiles	✓	✓	✓	✓	✓	Flush toilet	✓	Piped in	Small problem	-	Male 60	12	**Lesotho**
	Carpet	✓	✓	✓	×	×	Flush toilet	✓	Piped in	Small problem	-	Male 45	5	**Swaziland**
	Tiles	✓	✓	✓	✓	✓	Flush toilet	✓	Piped in	Small Problem	Professional, technical, managerial	Male 46	4	**Zimbabwe**

The poorest households in quintile 3 for each country has no household assets, but all live in houses with cement floors (which clearly scores highly in the construction of the DHS wealth index). Unlike the households from the poorer quintiles, these households have a traditional pit toilet (Lesotho and Swaziland) and piped water (Swaziland).

There is a notable difference in household wealth and amenities between the cut-off households in quintile 3 and those in quintile 4. The poorest household in quintile 4 in Zimbabwe is a six member, female-headed household, living in a cement floor house that has a radio, television and a bicycle. The household head is of working age and works in sales and the household has a traditional pit toilet. In Swaziland, the corresponding household also has a radio and television, plus access to electricity and has a traditional pit toilet. It is also a female-headed household and the household head holds a position in sales.

The last quintile in all three countries typically comprises households that have many of the household assets, as well as access to electricity and piped water. The cut-off households for this quintile (five) have ventilated pit latrines and report that accessing health services is a "*small problem*". They are smaller than the cut-off households in the other quintiles, with three, six and five household members for Lesotho, Swaziland and Zimbabwe respectively. In these three households, all household heads are of working age.

Table 4.3 reveals some interesting features on the households in the sample. Before discussing some of these, it is crucial to reiterate that these households are merely to give the reader an idea of whom we are talking about. The first notable feature of these cut off points is that most of the households at the poorer end of the scale

(quintiles 1 and 2) have much larger families than those closer to the top end of the scale. The poorest households in all 3 countries have at least 7 household members while those at the higher end (in the case of Swaziland and Zimbabwe) have 5 and 4 household members, respectively. It is also quite notable that the poor households, and this applies for all the countries, are predominantly headed by a female, and in a significant number of cases, an old woman past their working age. These women are therefore most likely to be grandmother's left to take care of grandchildren (whose parents may have died of HIV). In contrast, for all three countries, those households in quintiles 4 and 5 are predominantly headed by a man, who is of working age.

To get a more accurate visualisation of how the quintiles compare across the countries, figure 4.1 below shows the kernel density estimates of the PCA generated household wealth scores. The kernel density estimates show the density function of a given variable and so are, simplistically, smoothed histograms depicting the number of respondents (density) at given wealth levels. On figure 4.1, we show the respective cut-off points for each of the quintiles for all three countries.

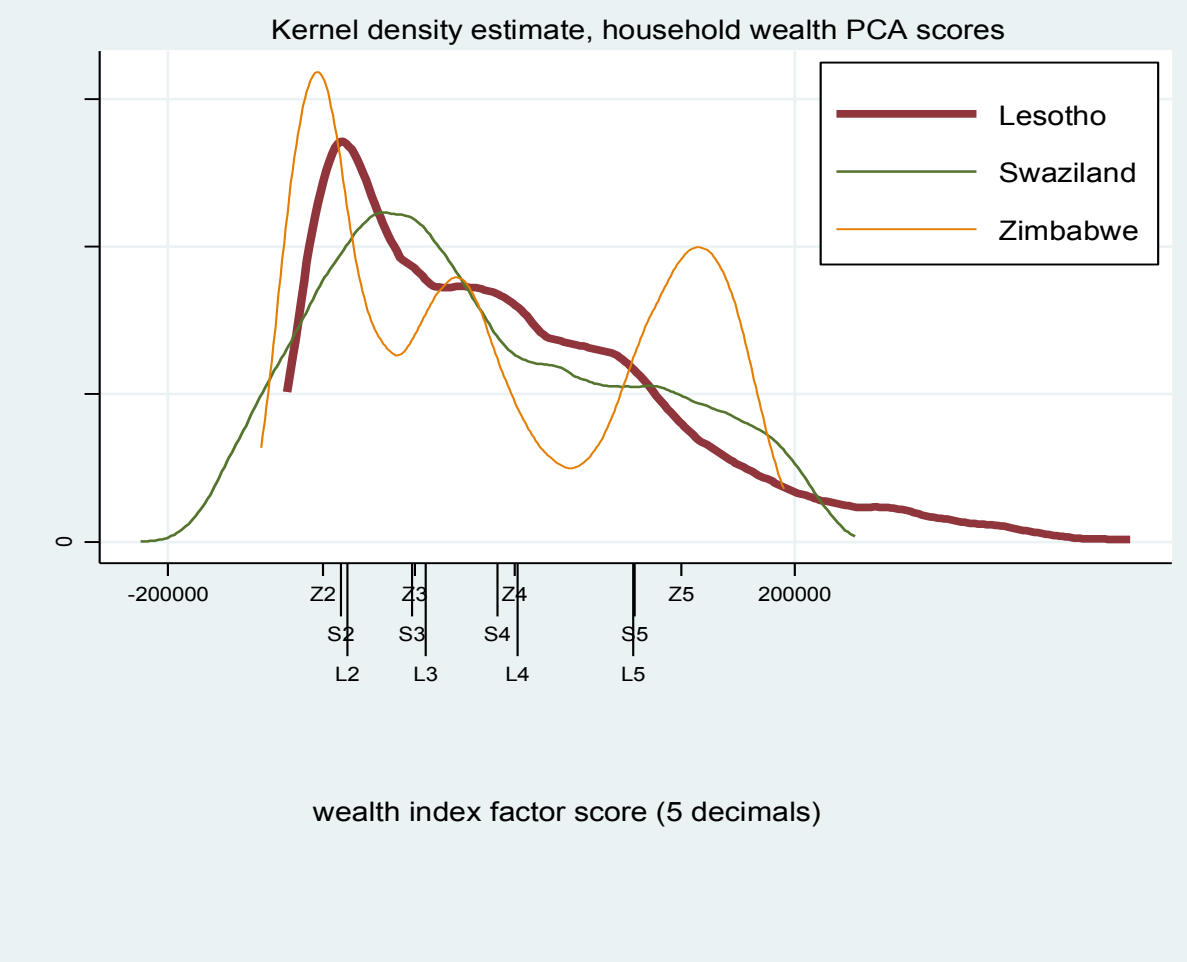

Figure 4.1 Kernel density estimates and quintile cut off points

On the x-axis, the markers L2 to L5 represent the lower bound cut-off points for household wealth quintiles two to five for Lesotho. Respondents to the left of L2 on the Lesotho kernel density function for example, are those Lesotho respondents in household wealth quintile 1. Those between L2 and L3 are in household wealth quintile 2 and so on up to those in quintile 5. Markers S2-S5 represent the same information for Swaziland (using the Swaziland kernel density function this time) while markers Z2 to Z5 represent Zimbabwe (using the Zimbabwe kernel density function).

The most important information from the graph is that although the three countries have different underlying wealth levels, the household wealth quintile cut-off points for the

respondents are nearly the same places for most of the range. The first wealth cut off points for each country (L2, S2 and Z2) are around the same region, and so are the third and fourth cut-off points. What this means then, is that for most of the time, the wealth quintiles for the different countries represent people with more or less the same wealth levels – in as far as housing building materials, ownership of household assets and access to basic amenities such as water, electricity, sanitation and health are concerned.

However, there are some overlaps in the quintiles. For example, the horizontal distance between Z2 and L2 represents individuals in Zimbabwe who are in wealth quintile 2 but would be in wealth quintile 1 if they were in Lesotho. Likewise, looking at the cut off points for the last quintile, the distance between L5 and Z5 shows those individuals considered wealthiest in Lesotho (quintile 5) that would fall in quintile 4 if they lived in Zimbabwe.

The kernel density estimates also give us an idea of the level of inequality across the countries. Inequality (and absolute wealth levels) are nearly identical for Swaziland and Lesotho. L2 and S2, L3 and S3 occur at nearly the same points on the x-axis while S5 and L5 (where quintile 4 ends and quintile 5 starts) coincide. Because the "spread" of the quintiles is the same for both countries across the scores, the level of inequality in these countries is about the same (however high or low it may be). Zimbabwe however, shows higher inequality than the other two countries. It has the lowest cut-off point for quintile 1 (Z2) and yet the highest lower bound cut-off for quintile 5 showing that the spread of wealth is much larger in Zimbabwe than it is in Lesotho and Swaziland.

4.5 A-priori expectations and potential issues

4.5.1 Endogeneity

One of the main assumptions of multivariate regression models is that there is no endogeneity. Endogeneity can occur when the outcome variable in the model (in this case HIV-status) also affects the independent variable(s). This particular case of endogeneity, called reverse causality, is where for example AIDS results in loss of employment and hence poverty; but then poverty could also lead to HIV. With cross-sectional data, it is difficult to separate the two effects. Two variables most likely to have reverse causality are employment status and household wealth. HIV-positive individuals may find it difficult to secure work because of the time required off work if they are AIDS sick. No employment could then diminish the capability to purchase household goods in the case of breadwinners or, in the event that other family members are sick, costs of treatment could lead to lower resources available to purchase household goods.

Endogeneity is likely to be present in some of the results reported, but, unfortunately, given that the data is cross-sectional, very little if anything can be done about that.

4.5.2 Selection effects

Another general analytical issue that could bias the regression coefficients that describe the relationships between explanatory variables and HIV status is the fact that HIV status (and sexual behavior variables) have high degrees of missingness in many of the DHS datasets (see, e.g., Barnighausen et al. Epidemiology 2011; Hogan et al. 2012). The potential limitations arising from selection effects are potentially biased coefficient estimates

4.6 Statistical Analysis

In the first set of regressions, we fit multivariate logistic regression models to the DHS data with HIV-status the dependent variable and selected variables for SES, together with control variables of age, age at first sex, age first marriage and sex independently for each country. This is because we strongly believe that the HIV epidemics in these countries, despite some similarities, are different.

However, owing to the small size of the respondents in the 15-19 age group that are HIV-positive (see table 4.5 below) we perform a second set of regression models in which we pool the data from the three countries together to boost sample size and improve coefficient robustness. In this second set of regressions, we use the same variables as above, but include and dummies for country. This will change the interpretation of some of the results, and this will be discussed with the results.

Multivariate logistic regressions of the form: $Y_i = \beta X_i + u_i$ are run, where: Y_i = logit of HIV-status (0 if negative and 1 if positive) for individual i, β_1 the coefficient of risk factor, X_i the respective risk factor and u the error term.

Analysis is done in STATA 11.1, applying the relevant statistical weights[29] to the survey data to enable population-level inferences and to adjust standard errors for sample clustering. We test for goodness of fit using a variant of the Hosmer-Lemeshow goodness-of-fit developed for use with logistic regression models when working with sample survey data[30] (Archer,

[29] We weight using the HIV weight, because not all individuals that were in the survey were tested for HIV. We then specify the primary sampling unit and sample strata.

Hosmer, & Lemeshow, 2007). The p-value for this test is reported in each regression under the label *lfit*, and a p-value less that 0.100 suggests that the model is not a good fit. We carry out additional post estimation checks for multicollinearity and influential observations, and report all results in odds ratios together with robust linearized standard errors. We also check for specification error and p-value for this test reported in each regression under the labels *_hat* and *_hat* squared. If the model is specified correctly, the _hat p-value should be less than 0.10 while the _hat square value greater than 0.10

We estimate 15 models, each varying in the number of variables and/or the sample size. These 15 models are split into two categories – regression models from the individual country samples and regression models from the pooled three-country sample. In the pooled sample, we combine the data from Lesotho, Swaziland and Zimbabwe, and estimate correlates of HIV-status for the total sample and then for women only. From the total sample, we perform the regression in three stages. First we regress age, age squared, country and sex on HIV-status. This is to observe the unadjusted effect of sex on HIV, while controlling for age and the country of residence. In the second model, we add socioeconomic indicators of place of residence, employment status, household wealth and education. In the last stage, we add to the second model the measures of individual behaviour. These three stages of modelling are reported under "Pooled sample, men and women" with the secondary headings "base" for the first, "SES" for the second and "behavioural" for the third.

As some of the literature shows, the relationship between the same indicators, e.g. employment and HIV, could be different between men and women. As one study in Tanzania showed decent employment among women predicted higher HIV risk while for men unemployment predicted higher HIV risk (Msisha et al., 2008). We assume that there may be

a gender differences in the relationship between all the indicators and HIV status and thus repeat the same regression processes above for the all-woman pooled sample. This generates three regression models under "All woman sample" and these are similarly labelled "Base", "SES" and "behavioural" for the first, second and third models respectively.

The above models make up the second table of our results. In the first table, we report HIV regression estimates from disaggregated data for the countries. The literature is rife with examples of how although the epidemic has some regional and even continental homogeneity, it still remains heterogeneous in many respects and may vary from country to country. The country-specific models also help us include more variables in the regression model, as for the pooled sample we can only use those variables that are present in all three country datasets. In these country-specific models, we run three models per country. In the first, we run a base regression, which includes both men and women with HIV-status the outcome variable and age, education, household wealth and place of residence as the independent variables. Next, using the same sample, we add the behavioural controls age at first sex, number of lifetime sexual partners and age at first marriage. In the third regression for each country, run the second regression again, but for women only.

4.7 Descriptive Statistics

The total pooled sample contained a total of 25 997 men and women between the ages of 15 and 49. Of these, 1 812 men (17%) and 3 779 women (25%) were HIV-positive. The corresponding figures for HIV-negative persons are 9 087 for men and 11 319 for women. Tables 4.4 to 4.7 below shows the age and HIV profile from the individual country data and from the pooled 3-country sample.

Table 4:4: Age group, Sex and HIV profile for adults (15-49) Lesotho

	Men			Women			Total
	HIV-negative	***HIV-positive***	***HIV-Prevalence***	***HIV-negative***	***HIV-positive***	***HIV-Prevalence***	***HIV prevalence***
15-19	612	12	2%	700	61	8%	5%
20-24	359	49	12%	469	150	24%	19%
25-29	214	73	25%	253	170	40%	34%
30-34	148	100	40%	218	151	41%	40%
35-39	109	69	38%	173	128	43%	41%
40-44	78	43	35%	207	84	29%	31%
45-49	97	28	22%	212	44	17%	19%

HIV prevalence in the sample is lowest for men and women in the 15-19 age-group. In chapter 2, we pointed out studies that argue that HIV prevalence for this age group is a proxy for HIV incidence as infection is most likely to be recent, and the data suggest that HIV incidence among young boys (15-19) in Lesotho is four times lower than that among 15-19 aged girls. Women from Lesotho in the sample exhibit a sustained peak HIV prevalence, with the prevalence hovering around 40 per cent for all the women between the ages of 25 and 39. Only at the age of 40 does HIV prevalence start to decline. Men on the other hand have a peak HIV prevalence between the ages of 30-34 (40%) from which age onwards HIV prevalence steadily declines.

Table 4:5: Age group, Sex and HIV profile for adults (15-49) Swaziland

	Men			Women			Total
Age-group	***HIV-negative***	***HIV-positive***	***HIV-Prevalence***	***HIV-negative***	***HIV-positive***	***HIV-Prevalence***	***HIV prevalence***
15-19	1 159	20	2%	1074	121	10%	6%
20-24	648	91	12%	579	365	39%	27%
25-29	396	141	26%	336	325	49%	39%
30-34	216	154	42%	309	256	45%	43%
35-39	185	142	43%	292	274	48%	40%
40-44	139	92	40%	282	117	29%	33%
45-49	155	64	29%	274	80	23%	25%

In the Swaziland sample, young women, compared to young men, also bear a much larger proportion of those living with HIV. HIV prevalence among young girls aged 15-19 is five times higher than that among similarly aged boys (10% vs 2%). While for both sexes HIV prevalence increases dramatically between the ages of 20 and 24, women in this age-group are still three times more likely to be HIV-positive than their male counterparts. HIV prevalence among women in the sample peaks at the ages of 25-29 where nearly half (49%) in the sample tested positive for HIV. Unlike in Lesotho, both men and women in Swaziland experience a sustained peak HIV prevalence across a number of age cohorts. For men, HIV prevalence peaks between the ages of 30 and 44, with all 5-year age groups in this range experiencing HIV prevalence rates of between 40 and 42 per cent. For women, peak HIV prevalence occurs in between the ages of 25 and 39.

Table 4:6: Age group, Sex and HIV profile for adults (15-49) Zimbabwe

	Men			**Women**			**Total**
Age-group	***HIV-negative***	***HIV-positive***	***HIV-Prevalence***	***HIV-negative***	***HIV-positive***	***HIV-Prevalence***	***HIV Prevalence***
15-19	1 573	46	3%	1 698	99	6%	4%
20-24	1 044	54	5%	1 350	265	16%	12%
25-29	664	111	14%	876	346	28%	23%
30-34	474	182	28%	675	342	34%	31%
35-39	331	148	31%	470	245	34%	33%
40-44	238	111	32%	440	158	26%	28%
45-49	248	82	25%	432	98	18%	21%

HIV prevalence patterns in Zimbabwe by age group mirror those from Lesotho and Swaziland. The lowest HIV prevalence is in the youngest age group and steadily rises to a peak between the ages of 35-39 for men and 30-39 for women.

Table 4:7: Age group, Sex and HIV profile for adults (15-49) Lesotho, Swaziland and Zimbabwe (pooled sample)

	Men			Women			Total
Age-group	***HIV-negative***	***HIV-positive***	***HIV-Prevalence***	***HIV-negative***	***HIV-positive***	***HIV-Prevalence***	***HIV Prevalence***
15-19	3 344	78	2%	3 472	281	7%	5%
20-24	2 051	194	9%	2 398	780	25%	18%
25-29	1 274	325	20%	1 465	841	36%	30%
30-34	838	436	34%	1 202	749	38%	37%
35-39	625	359	36%	935	547	37%	37%
40-44	455	246	35%	929	359	28%	30%
45-49	500	174	26%	918	222	19%	22%
Lesotho	1 617	374	19%	2232	788	26%	23%
Swaziland	2898	704	20%	3146	1438	31%	26%
Zimbabwe	4572	734	14%	5941	1553	21%	18%
Total	9087	1812	17%	11319	3779	25%	22%

Among 15-19 year olds in the pooled sample, there is a disproportionate HIV burden among young women, with young women twice as likely to be HIV-positive than similarly aged boys. HIV prevalence is very low among young men, with only 3 per cent prevalence between 15-19 and 7 per cent between the ages of 20 and 24. HIV prevalence peaks among 30-34 year old women, and these have the largest HIV prevalence rates in the full sample. HIV prevalence peaks slightly later for men, whose prevalence peaks in the mid-thirties to early forties. HIV prevalence drops by nearly ten per cent for women in their forties compared to those in the late thirties, and this suggest that either, HIV mortality increases drastically or the women in their forties made it through the earlier age-groups before HIV epidemic exploded in the sub-region. Among men, that drop is only evident as one moves from the early forties into the late forties.

Tables 4.8 to 4.11 show the summary statistics for the continuous data in the different countries and in the pooled sample.

Table 4:8 Summary statistics for continuous variables in Lesotho

Variable	Observations	Mean	Std. Dev	Min	Max
Age	5 020	26.4	9.5	15	49
Years of education	5 007	6.5	3.3	0	15
Age first sex	3 958	18	3.2	9	38
Lifetime number of partners	-	-	-	-	-
Age at first marriage	2475	20.63	4.3	8	40

The mean age in Lesotho about 26 years and the average number of years of education completed is 6.5 years. The average respondent in this sample, therefore, has not completed primary school.

Table 4:9 Summary statistics for continuous variables in Swaziland

Variable	Observations	Mean	Std. Dev	Min	Max
Age	8 186	27.04	9.7	15	49
Years of education	8 167	7.92	3.9	0	20
Age first sex	6 229	17.8	3.0	8	40
Lifetime number of partners (restricted to 15)	5 984 (5828)	3.9 (3.2)	5.3(2.8)	1(1)	80 (15)
Age at first marriage	3064	22.7	5.4	10	46

The mean age in the Swaziland sample is 27 years and the average years of education is 8 years, which roughly coincides with the number of years needed to complete primary school and have one year in high school. The average lifetime number of sexual partners in the sample is approximately 4, though there are significant outliers that affect the number of lifetime partners (2 of the respondents reported 80 lifetime partners, for example). We present two summary statistics for lifetime number of partners, for the full sample as is, and for a sub-sample restricted to a maximum of 15 lifetime partners. Restricting the sub-sample this

way only reduces the sample size by just over two per cent but significantly reduces the effect of outliers and nearly halves the standard deviation. In this restricted subsample, the average number of sexual partners is about three. For those ever married, 22 is the mean age of marriage, nearly two years higher than that in Lesotho

4:10 Summary statistics for continuous variables in Zimbabwe

Variable	**Observations**	**Mean**	**Std. Dev**	**Min**	**Max**
Age	12 817	27	9.4	15	49
Years of education	12 810	8.1	2.7	0	19
Age first sex	9 350	18	3.2	8	39
Lifetime number of partners	9 685 (9 506)	2.9(2.4)	4.4 (2.4)	1(1)	80(15)
Age at first marriage*	8 040	20	4.2	8	42

The mean age of the sample in Zimbabwe is 27 while the average respondent has 8.1 years of education, also corresponding to completed primary school and a single year in high school. We would have expected a higher average number of years in school for Zimbabwe, but table 4.6 above also shows that the Zimbabwe sample has a much larger proportion drawn from those between 15 and 19 than the other two samples. Some of these would most likely be in their early years of schooling. After restricting the number of sexual partners to 15 to minimise the effect of outliers, the average person in the sample who is sexually active has had about two partners in their life.

Table 4:11 Summary statistics for continuous variables in the three -country pooled sample

Variable	Observations	Mean	Std. Dev	Min	Max
Age	27 005	27.3	9.61	15	49
Years of education	26 966	7.7	3.13	0	20
Age first sex	19 537	18.1	3.16	8	40
Lifetime number of partners*	15 669(15 334)	3.33(2.79)	4.83(2.5)	1(1)	80(15)
Age at first marriage	14 552	20.1	4.62	8	46

*Lesotho does not have data on number of lifetime partners

Tables 4.12 to 4.15 below show the correlation coefficients between the continuous variables and the corresponding p-values. We do not report correlation coefficients between categorical variables as these are meaningless.

Table 4:12 Correlation coefficients for continuous data, adults (15-49) Lesotho

	Age	Education	Age first sex	Lifetime partners	Age first marriage	Education Squared	Age squared
Age	1						
Years of education (p-value)	-0.081 (0.000)	1					
Age first sex	0.405 (0.000)	-0.006 (0.623)	1				
Lifetime partners	-	-	-	-			
Age at first marriage	0.175 (0.000)	0.016 (0.344)	-0.210 (0.000)	-	1		
Education Squared	-0.026 (0.042)	0.942 (0.000)	-0.026 (0.042)	-	0.1154 (0.000)	1	
Age squared	0.990 (0.000)	-0.100 (0.000)	0.384 (0.000)	-	0.147 (0.000)	-0.048 (0.000)	1

The primary objective of presenting the correlation matrices is to check if any of our variables are highly collinear. Excluding the squared variables (education squared and age squared) which are closely related to their linear variables, the largest correlation coefficient

is between age at first sex and age. Other things to note are that the variable years of education is negatively related to age, and so is age at first marriage to age at first sex.

Table 4:13 Correlation coefficients for continuous data, adults (15-49) Swaziland

	Age	**Education**	**Age first sex**	**Lifetime partners**	**Age first marriage**	**Education Squared**	**Age squared**
Age	1.00						
Years of education (p-value)	-0.03 (0.01)	1.00					
Age first sex	0.51 (0.00)	0.06 (0.00)	1.00				
Lifetime partners	0.13 (0.00)	0.01 (0.39)	0.023 (0.08)	1.00			
Age at first marriage	0.24 (0.00)	0.28 (0.00)	0.11 (0.00)	0.15 (0.00)	1.00		
Education Squared	0.07 (0.00)	0.94 (0.00)	0.14 (0.00)	0.03 (0.00)	0.30 (0.00)	1.00	
Age squared	0.99 (0.00)	-0.06 (0.00)	0.46 (0.00)	0.13 (0.00)	0.22 (0.00)	0.04 (0.00)	1.00

None of the continuous variables are strongly collinear. With the exception of the relationship between age and age at first sex with a correlation coefficient of 0.51, all other linear variables have correlation coefficients below 0.3. Expectedly, the squared variables are strongly related to their corresponding linear variables, i.e. age is strongly related to age

squared and education strongly related to education squared. While all the other linear variables are positively related to each other, there is a negative relationship between age and years of education in Swaziland.

Table 4:14 Correlation coefficients for continuous data, Zimbabwe

	Age	**Education**	**Age first sex**	**Lifetime partners**	**Age first marriage**	**Education Squared**	**Age squared**
Age	1						
Years of education (p-value)	-0.222 (0.000)	1					
Age first sex	0.386 (0.000)	-0.066 (0.000)	1				
Lifetime partners	0.104 (0.000)	0.024 (0.017)	-0.051 (0.000)	1			
Age at first marriage	0.242 (0.000)	0.272 (0.000)	0.035 (0.002)	0.145 (0.000)	1		
Education Squared	-0.121 (0.000)	0.942 (0.000)	-0.026 (0.003)	0.033 (0.001)	0.290 (0.000)	1	
Age squared	0.989 (0.000)	-0.260 (0.000)	0.349 (0.000)	0.103 (0.000)	0.214 (0.000)	-0.157 (0.000)	1

Zimbabwe exhibits the strongest negative relationship between age and years of education. There is a weak negative relationship between age at first sex and education, and between the lifetime number of sexual partners and age at first sex.

Table 4:15 Correlation coefficients for continuous data 3-country pooled sample

	Age	**Education**	**Age first sex**	**Lifetime partners**	**Age first marriage**	**Education Squared**	**Age squared**
Age	1						

Years of education (p-value)	-0.117 (0.000)	1					
Age first sex	0.375 (0.000)	-0.060 (0.000)	1				
Lifetime partners	0.114 (0.000)	0.016 (0.045)	-0.036 (0.000)	1			
Age at first marriage	0.250 (0.000)	0.220 (0.000)	-0.080 (0.000)	0.177 (0.000)	1		
Education Squared	-0.030 (0.000)	0.940 (0.000)	-0.039 (0.000)	0.0434 (0.00)	0.280 (0.000)	1	
Age squared	0.989 (0.000)	-0.147 (0.000)	0.346 (0.000)	0.111 (0.000)	0.225 (0.000)	-0.061 (0.000)	1

There is a negative relationship between age and years of education in the pooled sample, showing that as age increases years of education decline. This is true for all three countries, although the most pronounced relationship of this kind is in Zimbabwe. A negative relationship between age and education is reflective of continuous improvements in access, for successive cohorts of Africans, to education over time. For all countries, age at first sex is positively correlated with age, and this suggests that the age at sexual debut in the sample has been falling over time.

Among those ever married, the age at first marriage is positively related to age, so for all countries, the average age of marriage has been going down over time (older respondents in the sample have higher age of first marriage). There is a negative correlation between the number of lifetime partners and age at first sex, suggesting that those with a high number of lifetime partners had a low age of sexual debut. This perhaps makes sense as these people would have been sexually active for longer and thus likely to have had a higher number of sexual partners.

There is a positive correlation between number of lifetime partners and education for all countries, suggesting that the more educated tend to engage in more sexual partnerships.

In light of the literature review in chapter 3, we expect a positive multivariate relationship between the lifetime number of partners and HIV-status. A large number of partners increases exposure to HIV and this should increase HIV risk. We also expect a negative relationship between age of marriage and HIV, with those who marry early more likely to be HIV-positive. The associations between education and HIV-status are not clear, but we would expect a positive linear relationship between years of education and HIV-status but up to some point in which increased education should result in higher HIV risk.

Next, we present descriptive statistics for the categorical variables. Tables 4.16 to 4.19 show the relationship between household wealth, place of residence and HIV status.

Table 4:16 Relationship between household wealth and place of residence for Lesotho, adults (15-49)

	Urban			**Rural**		
	HIV-negative	**HIV-positive**	**HIV-prevalence**	**HIV-negative**	**HIV-positive**	**HIV-prevalence**
1st quintile	8	6	43%	760	170	18%
2nd quintile	32	20	38%	781	223	22%
3rd quintile	101	35	26%	629	192	23%
4th quintile	227	106	32%	528	153	22%
5th quintile	463	170	27%	320	87	21%

Pearson chi2 (urban HIV prevalence vs rural HIV prevalence) = 27.82 (p-value=0.00)
Pearson chi2 (HIV prevalence vs wealth quintile) = 15.62 (p-value=0.00)

There are different HIV prevalence patterns by rural and urban areas and by wealth quintiles. In the urban areas, HIV is highest in the poorest quintile and it steadily decreases as household wealth increases. In the rural areas, HIV prevalence is relatively flat across the wealth quintiles, with nearly the same prevalence rates from the poorest to the wealthiest. Tests for independence show that the differences in HIV prevalence by rural or urban residence and those of HIV by wealth quintiles are statistically significant.

Table 4:17 Relationship between household wealth and place of residence for Swaziland, adults (15-49)

	Urban			Rural		
	HIV-negative	**HIV-positive**	**HIV-prevalence**	**HIV-negative**	**HIV-positive**	**HIV-prevalence**
1st quintile	10	9	47%	947	332	26%
2nd quintile	33	33	50%	982	344	26%
3rd quintile	165	94	36%	1 016	315	24%
4th quintile	391	237	38%	895	237	21%
5th quintile	1 100	404	27%	505	137	21%

Pearson chi2 (urban HIV prevalence vs rural HIV prevalence) = 49.96 (p-value=0.00)
Pearson chi2 (HIV prevalence vs wealth quintile) = 2.33 (p-value=0.68)

In Swaziland, the same relationship between HIV prevalence and sector or residence as in Lesotho is evident. The chi2 test for independence shows that HIV status is independent of wealth quintiles but it is not independent of rural or urban residence.

Table 4:18 Relationship between household wealth and place of residence for Zimbabwe, adults (15-49)

	Urban			Rural		Total
	HIV-negative	**HIV-positive**	**HIV-prevalence**	**HIV-negative**	**HIV-positive**	**HIV-prevalence**
1st quintile				2 008	405	37%
2nd quintile				2 051	446	18%
3rd quintile	47	15	24%	2 009	410	17%
4th quintile	1 195	363	23%	1 039	247	19%

5th quintile	2 057	380	16%	107	21	16%

Pearson chi2 (urban HIV prevalence vs rural HIV prevalence) = 2.7 (p-value=0.100)
Pearson chi2 (HIV prevalence vs wealth quintile) = 36.42 (p-value=0.00)

In Zimbabwe, there is a decline in HIV prevalence as household wealth increases, both in the rural areas and urban areas. HIV prevalence is highest in the poorest quintiles and lowest in the richest.

The table below explains some of the wealth differentials between rural residents and urban residents. More unemployed people live in the rural areas than in the urban areas and this, in part, probably explains the wealth differential between urban and rural areas.

Table 4:19 Employment status and sector of residence for adults (15-49) three-country pooled sample

	Urban	**Rural**	**Total Sample**
Unemployed	1 580	6 028	7 608
Employed	4 796	9 009	13 805
% Employed	75%	60%	
Total	6 376	15 037	21 413

(Pearson chi2(1) = 458.02. P= 0.000)

In the urban areas, 25 per cent of the respondents are unemployed in the sample, compared to 40 per cent in the rural areas. The chi2 test for independence shows that the difference in employment rates in the urban and rural areas is statistically significant.

4.8 Results

Table 4.20 below shows the results from the individual country regressions. As discussed earlier, for each country, we perform three regressions. In the first regression for each country (labelled SES) we estimate the relationship between HIV-status and SES variables while controlling for age and sex. In the full model, we then add behavioural controls and in the last, we repeat the full model regression but only for women in that country.

Table 4:20: Individual country regressions, 15-49 year old men and women in Lesotho, Swaziland and Zimbabwe

	Lesotho			**Swaziland**			**Zimbabwe**		
	SES (1)	**Full sample(2)**	**All-woman sample (3)**	**SES (4)**	**Full sample(5)**	**All-woman sample (6)**	**SES (7)**	**Full Sample (8)**	**All-woman sample (9)**
Age ***(OR)*** ***(Std. Er.)*** ***(p-value)***	1.78*** (0.053) 0.000	1.50*** (0.073) 0.000	1.52*** (0.093) 0.000	1.74*** (0.039) 0.000	1.21*** (0.055) 0.000	1.18*** (0.064) 0.002	1.60*** (0.034) 0.000	1.43*** (0.042) 0.000	1.45*** (0.050) 0.000
Age Squared	0.99*** (0.000) 0.000	0.99*** (0.001) 0.000	0.99*** (0.007) 0.000	0.99*** (0.000) 0.000	0.997*** (0.001) 0.000	0.99*** (0.001) 0.000	0.99*** (0.000) 0.000	0.99*** (0.001) 0.000	0.99*** (0.001) 0.000
Female	1.50*** (0.121) 0.000	1.08 (0.137) 0.531		1.85*** (0.107) 0.000	1.31** (0.158) 0.026		1.51*** (0.082) 0.000	1.99*** (0.173) 0.000	
2nd quintile[31]	1.37*** (0.165) 0.010	1.30** (0.206) 0.096	1.53** (0.323) 0.042	1.09 (0.107) 0.388	1.29* (0.181) 0.069	1.35* (0.234) 0.080	1.14 (0.091) 0.104	1.17 (0.103) 0.081	1.24* (0.132) 0.046
3rd quintile	1.54*** (0.204) 0.001	1.88*** (0.321) 0.000	1.79** (0.415) 0.013	0.97 (0.095) 0.796	1.02 (0.149) 0.867	1.03 (0.184) 0.893	1.17** (0.096) 0.056	1.27** (0.116) 0.010	1.36** (0.149) 0.005
4th quintile	1.56*** (0.204) 0.001	1.84*** (0.324) 0.001	2.00** (0.460) 0.002	0.95 (0.095) 0.576	0.92 (0.137) 0.566	0.92 (0.172) 0.647	1.31** (0.125) 0.005	1.43*** (0.153) 0.001	1.59*** (0.208) 0.000
5th quintile	1.22 (0.179)	1.54** (0.308)	1.64* (0.423)	0.78** (0.088)	0.78 (0.133)	0.75 (0.166)	0.87 (0.110)	0.96 (0.140)	1.09 (0.192)

[31] I ran multiple regressions using the index in results that are note reported in the thesis. It was evident that the results did not vary greatly by changing the specification of the wealth variable. It was easier to interpret the wealth variable specified as the quintiles in the regressions, so for presentation purposes and clearer argument, I opted for the quintiles.

	0.170	0.029	0.054	0.026	0.139	0.189	0.271	0.754	0.642
Education	0.99 (0.035) 0.886	0.97 (0.043) 0.516	1.02 (0.075) 0.805	1.03 (0.023) 0.202	1.05 (0.031) 0.110	1.04 (0.041) 0.328	1.12*** (0.031) 0.000	1.11*** (0.034) 0.001	1.08 (0.040) 0.035
Education [32]Squared	1.00 (0.003) 0.523	1.00 (0.003) 0.863	1.00 (0.005) 0.861	0.99*** (0.001) 0.000	0.99*** (0.002) 0.004	0.99** (0.003) 0.036	0.99*** (0.002) 0.000	0.99*** (0.002) 0.000	0.99* (0.003) 0.010
Rural	0.71*** (0.069) 0.000	0.71** (0.097) 0.013	0.73** (0.127) 0.071	0.67*** (0.049) 0.000	0.72*** (0.083) 0.004	0.64*** (0.098) 0.003	0.80** (0.072) 0.014	0.87 (0.090) 0.186	0.92 (0.117) 0.516
Employed	1.16 (0.088) 0.054	1.04 (0.106) 0.731	1.06 (0.139) 0.675	1.16** (0.072) 0.018	1.05 (0.098) 0.633	1.11 (0.125) 0.353	0.95 (0.050) 0.368	0.91 (0.055) 0.114	0.97 (0.067) 0.642
Age at first marriage		1.01 (0.015) 0.445	1.03 (0.022) 0.120		1.05*** (0.010) 0.000	1.04*** (0.013) 0.001		1.04*** (0.010) 0.000	1.04*** (0.013) 0.003
Age first sex		0.95*** (0.017) 0.006	0.94** (0.025) 0.019		0.96*** (0.014) 0.003	0.98 (0.022) 0.324		0.96*** (0.011) 0.000	0.96** (0.016) 0.015
Number of lifetime partners					1.10*** (0.019) 0.000	1.20*** (0.048) 0.000		1.14*** (0.016) 0.000	1.38*** (0.058) 0.000
	N=4 995 Chi(2)P	**N=2 021 Chi(2)P=0**	**N=1 229 Chi(2)P=0**	**N=8 148 Chi(2)P=0**	**N=2 762 Chi(2)P=**	**N=1 757 Chi(2)P=0**	**N=12 758 Chi(2)P=0**	**N=7 369 Chi(2)P=0.0**	**N=5 059 Chi(2)P=0.**

[32] I considered the fact that the relationship between HIV and education may not necessarily be linear so also ran regressions specifying the education variable as "no education", "primary education", "secondary education" and "higher". I found that I lost a lot of explanatory power due to the fact that for some countries, there were disproportionately big clusters around some levels of education, especially no education and primary education. The regression models became more robust once I used the specific years of education per individual. Pertaining to the functional form, the decision to specify it as a quadratic function was informed by the cited research that had noted that this relationship exhibited at least one turning point in some countries

	=0.000 Pseudo R2=0.12	**.000 Pseudo R2=0.047**	**.000 Pseudo R2=0.06**	**.000 Pseudo R2=0.13**	**0.000 Pseudo R2=0.05**	**.000 Pseudo R2=0.06**	**.000 Pseudo R2=0.11**	**00 Pseudo R2=0.05**	**000 Pseudo R2=0.06**
_hat	0.000	0.017	0.106	0.001	0.000	0.000	0.000	0.000	0.000
_hat squared	0.001	0.137	0.072	0.000	0.477	0.455	0.026	0.249	0.000
lfit	0.517	0.464	0.511	0.180	0.428	0.385	0.0103	0.347	0.103

*** 1% significance level, ** 5% significance level, * 10% significance level

In Lesotho, (model 1), age has a very large coefficient (OR 1.78, p-value 0.000), after controlling for sex and SES variables. This coefficient is larger than that in any other model with just the SES variables controlled for. In Lesotho, age is picking up a lot of unexplained HIV risk that is not related to any of the SES variables. While still controlling for the above correlates, women are 50 per cent more likely to be HIV-positive than men (OR 1.50, p-value 0.000).

Compared to other wealth groups, and controlling for the effects of age, education and gender, the poorest and the wealthiest in Lesotho have the lowest odds of being HIV-positive, and HIV risk increases with wealth up to wealth quintile 4 (since the coefficients are positive and increasing). Individuals in the second quintile are 37 per cent more likely to be HIV-positive that those in quintile one (OR 1.37, p-value 0.009). We postulate that this is a relative wealth effect. Although respondents in quintile two are better off than those in quintile one, these are still poor households (as our earlier discussion of the composition of these quintiles would suggest). This result then suggests that for Lesotho, moving from being very poor to just poor comes with elevated HIV risk. While controlling for the same covariates, respondents in quintile 3 also have higher HIV odds than the poorest (OR 1.54, p-value 0.001) and those in quintile 4 have the highest HIV risk. Note that the difference in HIV prevalence between the poorest and the richest quintiles is statistically insignificant (OR 1.25, p-value 1.21). Controlling for age, sex and SES, HIV risk in Lesotho rises from the poorest up until quintile 4 where it the drops to levels not statistically different to quintile 1 levels.

The education variable is statistically insignificant in this model, while living in the rural areas has a protective effect (OR 0.71, p-value, 0.000).

Adding the behavioural controls of age at first marriage and age at first sex (model 2) drops the magnitude of the sex variable in Lesotho and renders it statistically insignificant (OR 1.08, p-value 0.492). Adding these behavioural variables accentuates the impact of wealth on HIV status. Wealth quintile 5 becomes statistically significant (OR 1.54, p-value 0.029), with the wealthiest showing about 54 per cent additional HIV risk than the poorest. Once these controls are added the age coefficient drops significantly in magnitude, from 1.78 in model 1 to 1.50 in model 2. This change in magnitude is partly because including the age at first marriage eliminates those respondents that have never been married (who tend to be very young) and it is the youngest age group in the sample that have the lowest HIV prevalence rates.

In the all-woman sample for Lesotho (model 3), there are no major differences in regression coefficients compared to the full Lesotho sample (2). In both these models, we control for age, household wealth, education, employment status, place of residence, age at first sex and age at first marriage. Women in the second quintile, however, have a much larger HIV risk level than those in the 1st quintile (OR 1.53, p-value 0.042) for the all-woman sample compared to 1.30 in the full (male and female) Lesotho sample. This suggests that for modest gains in household wealth among poor households, women have a larger increase in HIV risk. Such disparities are also notable for quintile 3, but then die out in quintile 5 where there is just a 2% difference in probability between the coefficient in model (2) and that in (3). Most notably, women in the 4th quintile are twice as likely to be HIV-positive compared to the poorest and this difference is after controlling for SES and other behavioural variables.

The rural coefficient remains more or less the same in Lesotho across all specifications of the model.

In Swaziland, the base regression (4) which controls for age, sex, household wealth and education shows that being a woman is the most powerful predictor of HIV status. Controlling for the above covariates, women are 85 per cent more likely to be HIV-positive than men (OR 1.85, p-value 0.000). There is no statistical difference in the odds of being HIV-positive between those in quintile 1 compared to quintiles 2 to 4, although the richest are about 20 per cent less likely to be HIV-positive compared to the poorest. This strongly suggests that relative wealth does not matter in Swaziland. The poorest and the poor show no differing HIV odds while the rich definitely show lower HIV risk than the poor.

There is no evidence of a relationship between HIV and education.

Adding behavioural controls of age at first marriage, age at first sex and the lifetime number of partners (model 5) changes the coefficients significantly. The magnitude of the age coefficient drops from OR 1.74 to OR 1.21, nearly a 50 per cent drop in the odds of being HIV-positive. As in the Lesotho regressions, age at first marriage cuts out those individuals that have never been married, so part of the reduction in the age coefficient reflects this. HIV in Swaziland is lowest in the 15-19 age-group, but peaks rapidly and maintains this peak for successive age cohorts. By removing the unmarried, who would mostly be young, this model then largely comprises of individuals in the older cohorts where age would thus have a smaller impact on HIV.

The female variable also drastically reduces in size, from OR1.85 to OR 1.31 while maintaining its statistical significance. This means that the difference in the odds of being HIV-positive is greater among married women than among unmarried women, controlling for the above covariates. Among the married, there is increased HIV risk as one moves from quintile 1 to quintile 2, but no other differences by wealth quintile.

Behavioural variables in Swaziland are a strong predictor of HIV-status. The 3 behavioural controls of age at first marriage, age at first sex and number of lifetime sexual partners are all statistically significant. An increase in the age of marriage by a year increases the odds of being HIV-positive by 5 per cent (OR 1.05, p-value 0.000). Delaying the onset of sexual debut has a small protective effect (OR 0.98, p-value 0.05): with every additional year reducing the chances of being HIV-positive by about 2 per cent. The number of lifetime sexual partners is the strongest predictor of HIV prevalence among the behavioural variables, with a 10 per cent increase in the probability of being HIV-positive with each additional sexual partner (OR 1.10, p-value 0.000).

In the last Swaziland model, there is one more notable change in the coefficients. Among women alone, number of lifetime sexual partners increases in size, to OR 1.20, suggesting that multiple partners carry increased HIV risk for women than for men.

Results from Swaziland strongly suggest that SES variables account for very little of the varying HIV risk levels while the addition of behavioural variables changes the regression coefficients notably.

In Zimbabwe, model (7) shows that there is no change in HIV risk among those in the first quintile and those in the second quintile, while controlling for age, sex, education and place of residence. Those respondents in the second and third quintiles are more likely to be HIV-positive than those in quintile 1, again controlling for the above correlates. Unlike any of the base models before, education has an effect on the odds of being HIV-positive, OR (1.12, p-value 0.000) in the base model. This model also shows that Zimbabwe has the smallest differential between rural and urban HIV prevalence rates in multivariate models, with those in the rural areas 20 per cent less likely to be HIV-positive than those in urban areas (compared to 30 and 35 per cent in identical models for Lesotho and Swaziland).

Sexual behaviour variables of age at first marriage, age at first sex and number of lifetime partners have more statistical significance in predicting HIV-status than in Lesotho, and Swaziland. A younger age at first sex, older age of marriage and a higher number of lifetime partners all increase HIV risk in the Zimbabwean sample of men and women. (ORs 1.04, 0.96 and 1.10 for model 8).

In the all-woman Zimbabwe full model, age at first sex and the number of partners variables do not change in size, suggesting that the effect of these variables on HIV status is the same for both men and women. This is different to what we observe for Swaziland. The effect of the number of sexual partners over a lifetime is smaller in the women-only sample (OR 1.14, p-value 0.00) compared to that in the combined sample. This means that higher numbers of sexual partners are associated with higher HIV risk for men than for women. This finding could be capturing the risk levels that come from paying for sex, as men are more likely than women to pay for sex.

Comparing the epidemics therefore, behavioural factors have more traction in the Swazi and Zimbabwean data, whereas in Lesotho, background SES factors matter more. We reflect on this more below

In the following table, we present regression models from the pooled data. In the following set of models, we pool all the data from the three countries. Pooling the data has some implications in the interpretation of the results. Take the wealth quintiles for example. Under quintile 1 in the pooled sample will be those respondents in quintile 1 in Lesotho, Swaziland and Zimbabwe. As we have shown earlier, these people have varying household wealth levels (as some of those classified under quintile 1 in Zimbabwe would be in quintile 2 if they lived in Lesotho). In principle then, we are eliminating all absolute household wealth differences and are only measuring how relative wealth, i.e. how being poorer or wealthier than other

people, affects HIV status. Since we are controlling for country, we can capture the country-level differences in this coefficient.

Table 4:21: Multivariate regression, pooled sample regressions 15-49 year old men and women in Lesotho, Swaziland and Zimbabwe

	Pooled sample, men and women			**Pooled sample, all woman Sample**		
	Base (10)	**SES (11)**	**Full (12)**	**Base (13)**	**SES (14)**	**Full (15)**
Age ***OR*** **(SE)** **P**	1.66*** (0.022) 0.000	1.69*** (0.023) 0.000	1.41*** (0.030) 0.000	1.63*** (0.026) 0.000	1.67*** (0.028) 0.000	1.45*** (0.036) 0.000
Age squared	0.99*** (0.000) 0.000	0.99*** (0.000) 0.000	0.99*** (0.000) 0.000	0.99*** (0.000) 0.000	0.99*** (0.000) 0.000	0.99*** (0.000) 0.000
Swaziland	1.25*** (0.056) 0.000	1.37*** (0.064) 0.000	1.09 (0.071) 0.169	1.33*** (0.073) 0.000	1.47*** (0.084) 0.000	0.99 (0.086) 0.932
Zimbabwe	0.69*** (0.030) 0.000	0.70*** (0.031) 0.000	0.70*** (0.040) 0.000	0.69*** (0.036) 0.000	0.70*** (0.038) 0.000	0.72*** (0.051) 0.000
Female	1.60*** (0.054) 0.000	1.60*** (0.056) 0.000	1.11** (0.054) 0.036			
2nd quintile		1.17** (0.064) 0.004	1.20*** (0.078) 0.005		1.23*** (0.083) 0.001	1.24** (0.101) 0.006
3rd quintile		1.19*** (0.067) 0.002	1.29*** (0.087) 0.000		1.27*** (0.088) 0.001	1.29*** (0.110) 0.002
4th quintile		1.22*** (0.073) 0.001	1.29*** (0.095) 0.001		1.32*** (0.096) 0.000	1.33*** (0.123) 0.002
5th quintile		0.91 (0.064) 0.163	0.96 (0.087) 0.640		0.96 (0.083) 0.598	0.98 (0.111) 0.645
Education		1.05*** (0.015) 0.003	1.05*** (0.018) 0.002		1.04 (0.021) 0.067	1.05 (0.025) 0.046
Education squared		0.99*** (0.001) 0.000	0.99*** (0.001) 0.000		0.99** (0.001) 0.000	0.99*** (0.002) 0.001
Rural		0.71*** (0.034) 0.000	0.75*** (0.047) 0.000		0.70*** (0.041) 0.000	0.72*** (0.056) 0.000
Employed		1.06* (0.037) 0.096	0.94 (0.041) 0.145		1.1** (0.047) 0.018	1.02 (0.053) 0.691

Age first Sex			0.94*** (0.007) 0.000			0.94*** (0.010) 0.000
Age first marriage			1.04*** (0.006) 0.000			1.05*** (0.008) 0.000
	N= 25 997 Chi(2)P=0.000 Pseudo R^2=0.113	**N=25 901 Chi(2)P=0.000 Pseudo R^2=0.12**	**N=12 641 Chi(2)P=0.000 Pseudo R^2=0.04**	**N=15 098 Chi(2)P=0.000 Pseudo R^2=0.09**	**N=15 067 Chi(2)P=0.000 Pseudo R^2=0.10**	**N=8 132 Chi(2)P=0.000 Pseudo R^2=0.044**
_hat	0.000	0.000	0.000	0.000	0.000	0.000
_hat squared	0.000	0.000	0.301	0.000	0.000	0.730
Lfit	0.000	0.0049	0.368	0.000	0.002	0.265

*** 1% level of significance, ** 5% level of significance, * 10% level of significance

In the first pooled model (10), we estimate the relationship between the outcome variable HIV-status and independent variables age, age squared, country and sex. All coefficients are as expected, with a positive relationship between age and HIV-status. An additional year of age, holding all else constant, increases the odds of HIV infection by 66% (OR 1.66). The age-squared variable is statistically significant, and we can use this to calculate the turning point in the relationship between HIV-status and age, adjusted for country and sex. The turning point of the relationship between age and HIV is about 25 years[33]. For the three countries therefore, HIV risk starts to decline once individuals are about 25 years of age. The country of residence is a powerful predictor of HIV-status, owing to the significant differences in national prevalence in the two countries. Expectedly, controlling for country and age, women have a much larger chance of being sero-positive (OR 1.60). Controlling for country of residence and age, women are 60 per cent more likely to be HIV positive than men.

[33] Calculated as – b(1)/2b(2) where b(1) is the **coefficient** (and not the odds ratio) of the linear term and b(2) the coefficient of the quadratic term

This base model confirms what we already know about the basic structure of HIV – that it is greater among women, lower as people get older beyond some point and that HIV risk varies from country to country. For all models, further diagnostic checks for correct specification and other assumptions of the logistic regression are carried out. As expected, this first regression model suffers from misspecification bias, as there are potentially other variables that affect HIV-status that are missing.

In model (11), we add the four variables of SES to the base model – education, sector of residence, employment status and relative household wealth. The signs in all the coefficients in the base model (10) do not change much but there is a notable reduction in the size of the country coefficients. After adding the SES variables, the odds of being HIV-positive in Swaziland are actually much higher than those in Lesotho assuming that SES variables bring out some of the country differences that were obscured in the previous model.

For men and women in the pooled sample, individuals in the second wealth quintile are 17 per cent more likely to be HIV-positive than those in the first quintile, while controlling for age, sex, education, employment status and rural and urban residence (OR 1.17, p-value 0.004). Those in wealth quintiles 3 and 4 have higher odds of being positive while in the pooled sample there are no differences in HIV risk between the poorest and the richest while controlling for the mentioned covariates. Notably, HIV risk is also increasing as household wealth increases. Controlling for age, country, education and sex, those in quintile two are 17 per cent more likely to be HIV-positive than those in quintile 1 whilst those in quintile 3 are 19 per cent more likely to be HIV-positive than those in quintile 1. There is thus an increase in HIV risk as an individual moves from quintile 2 to quintile 3.

Education is positively related to HIV-status, with each additional year of education, while controlling for age, household wealth, employment status, sex and country of residence

increasing the odds of being HIV-positive by five per cent (OR 1.05). This is quite a large coefficient as it shows the change in risk for each successive year of education.

Respondents in the rural areas, controlling for age, country, sex, household wealth, employment status and education are less likely to be HIV-positive than those in the urban areas (OR 0.71, p-value 0.000). Controlling for the same covariates, employment is linked to higher HIV odds (OR 1.06, p-value 0.096).

In the last of the pooled regression models, we include behavioural controls of age at first sex and age at first marriage. This results in two main changes in the coefficients. Firstly, the size of the age coefficient drops significantly from OR 1.69 to 1.41. Age at first sex and age at first marriage explain about a third of the contribution of age to HIV-status. Employment status also loses significance once these behavioural controls are added, suggesting that for the pooled sample, employment is proxied by these two variables.

From the first three models, we observe a notable impact of SES variables on the country coefficient and age coefficients. Secondly, we also observe that the variable age, carries a large coefficient in part due to age-related sexual behaviour. While the coefficients of age at first sex and age at first marriage are themselves statistically significant, they also explain away a large part of the age coefficient because years of sexual activity matter.

Between the first and the third model, there is a notable decline in the size of the sex coefficient. The coefficient changes from OR 1.60 in the first model to 1.11 in the third, and much of this difference stems from the behavioural variables of age at first sex and age at first marriage. For Lesotho, Swaziland and Zimbabwe therefore, the difference in the age at sexual debut in part accounts for the variation in the HIV prevalence rates between men and women if regional level inferences are made.

The regression coefficients in the base model for the all-woman sample (13) is almost identical to the base model for the combined sample (10). This suggests therefore, that the effect on country of residence and age on HIV-status is the same for both men and women in the three countries as the coefficients hardly change between the two models.

However, after adding SES variables not many notable changes in the coefficients occur. The effect of household wealth on HIV risk is about the same though the education coefficients become insignificant. What this could mean is that education, at a regional level only increases HIV risk among men.

Employment status drops in significance once sexual behaviour is added for the all-woman sample HIV risk. Among the employed (who are more likely to make independent life choices), individual sexual behaviour is possess more HIV risk than the risk brought on by structural factors (such as being poor).

In model 15, after adding the behavioural variables, the size of the age coefficient once again significantly drops (OR changes from 1.67 to 1.45). In this analysis, the coefficient for Swaziland falls away, showing that after controlling for those covariates, there is no elevated HIV risk among Swazi women compared to Basotho women. The coefficients for household wealth maintain the same trends in this model compared to (15), evidence that sexual behaviour differences are independent of age.

Diagnostic checks reveal that only the full models are a good fit and properly specified, with the other ones suffering a combination of from misspecification or lack of a good fit.

4.9 Limitations

These results should however be interpreted understanding the limitations of the nature of the study. Being cross sectional data, we are unable to test or control for potential reverse

causality between HIV and SES, i.e. how HIV affects SES. There is no way of chronologically determining whether the observed SES measures affected HIV-status first – it could well be that the individual got HIV infection first and this affected SES due to factors such as costs of treatment, required changes in diet, loss of job or a change in place of residence. Such assessments would require follow up studies that track HIV incidence (Gillespie, 2008: 3) but currently, there are only a handful of such studies in Southern Africa. These studies have found mixed results to the effect that no consistent conclusions may be formed either.

Other authors that have applied logistic regression to DHS data also point out other limitations of this methodology and these include age bias, cohort bias, year bias and selectivity bias (e.g. Warren, 2010; Magadi & Desta, 2011; Gersovitz, 2005).

Age bias may arise when younger people in a generation are silent or unforthcoming about sexual behaviour and sexual acts, but become more forthright as they get older. Such behaviour will tend to suggest that sexual activity, for example, is increasing across older cohorts, and yet it might not be. Cohort bias occurs when a particular cohort is unforthcoming about sexual practices or acts, and the cohort stays like that forever. Such a bias is difficult to control for, or even recognise, within the survey. Year bias may occur if in particular years people feel pressured to report safer sex behaviours (maybe due to a widely visible HIV/AIDS campaign).

Lastly, Gersovitz (2005) also talks of selectivity bias, where young people with highly risky sexual practices are more likely to die earlier (cause HIV is fatal), and thus are likely to be missing from older cohorts (Gersovitz, 2005:4).

4.10 Conclusions

This chapter's main purpose was to assess empirically the link between HIV-status and structural and behavioural potential determinants in hyper-epidemic countries through multivariate analysis using nationally representative data. Both structural factors and individual factors affect HIV risk, but behavioural factors appear to mitigate heavily the impact of structural factors. Given that there are country-level differences in HIV prevalence rates, broader, structural factors were likely to be statistically significant in multivariate regressions. However, once behavioural variables are added, significant changes in the structural coefficients can occur.

The cross-sectional analysis provides evidence of a highly homogenous HIV epidemic between the three countries in as far as structural factors are concerned. The coefficients in the base models for country, age and sex are highly comparable, for women only and for the combined sample. At a macro level then, there are factors that affect the HIV epidemics in these countries the same way.

The individual country level regressions show another dimension of the Southern African epidemic – that once we move from the homogenous regional level epidemic there are differences in the national epidemics. SES, as measured by place of residence, employment status, household wealth and education, controlling for country and age, starts determining different HIV risk probabilities by country.

The country-level regressions show that the structural factors get more traction than the individual behaviour variables in Lesotho. The impact of measures of individual behaviour on HIV-status is dwarfed by that of structural factors. With regards poverty, gender and HIV, modest gains in wealth are associated with increasing HIV risk for both men and women, but the difference is more acute for women. In Swaziland, individual factors affect HIV status

more than they do in Lesotho. It would appear that both individual factors and structural factors are equally as important. There is also a distinct difference in HIV risk between the poor and the wealthy. While in Lesotho HIV risk gradually increased with wealth, in Swaziland, the biggest differences are between the poorest and the richest, without much discernable differences in HIV risk between the poorest and other wealth quintiles. In Zimbabwe, behavioural factors affect HIV risk the most, with a muted impact of structural factors.

This chapter provides support for a key hypothesis of this dissertation – that the relationship between HIV and its various correlates is context-specific. While structural factors affect all three countries, each country exhibits individual epidemic traits. In Lesotho, the structural variables in the model are strongly related to individual HIV risk and there is a gradual increase in HIV risk as wealth increases. In Swaziland, the impact of structural factors is not as strong as in Lesotho and there is no stark difference in the way wealth status affects men and women. In Zimbabwe, the behavioural variables are strongly associated with HIV risk.

Chapter 5. Gender inequality as a correlate of HIV prevalence in sub-Saharan Africa: Explaining away the Southern African 'anomaly'

5.1 Introduction

In chapter 2, we discuss the various macro-level factors that may contribute to the HIV epidemic in Sub-Saharan Africa, and in Southern Africa more specifically and then in chapter 4, we conducted multiple HIV regressions using individual-level data. To continue with our analysis of what could be shaping the HIV epidemic under hyper-epidemic conditions for women, we direct analysis to cross-country data, as this will enable us to explore the impact of national-level or regional level determinants of HIV.

Research that uses cross-country multivariate analysis to explore the determinants of HIV prevalence often reveals a statistically significant regional coefficient for Southern Africa[34] (e.g. Bonnel, 2000; Sawers, Stillwaggon & Hertz, 2008; Nattrass, 2008c; Tsafack 2007, 2008). This statistical feature persists despite controlling for a number of possible socio-economic, cultural and political drivers of HIV, suggesting that there is something unique about the epidemic in the sub-region. It is not yet clear what exactly is driving HIV in southern Africa and this has prompted UNAIDS and some authors to describe the epidemic as 'unique' or 'an anomaly' (UNAIDS, 2009; Halperin & Epstein, 2007; Gilbert & Walker, 2002). Other authors however argue that it is not in fact an anomaly, but can be adequately explained if focus is directed to the main or critical drivers (Stillwaggon & Sawers, 2010; Tsafack, 2008; Talbott, 2007). Opinions differ widely regards these 'critical' drivers of HIV, with poverty (Stillwaggon & Sawers, 2010), gender inequality (Tsafack, 2008) and a high

[34] This is either a dummy variable (separating Southern African countries from all other countries) or a categorical variable (including wider regional differentiations such as East or West Africa).

prevalence of commercial sex workers (Talbott, 2007) among some of them (see chapters 2 and 3).

UNAIDS cites as one of the main drivers of HIV in sub-Saharan Africa, the feminisation of the HIV/AIDS epidemic. In the 2008 epidemiological report, it states that:

> *"long-term success in responding to the HIV epidemic will require sustained progress in addressing human rights violations, gender inequality, stigma, and discrimination"* (UNAIDS, 2008:64)

In the same report, of the seven key findings, three relate to gender inequality and include investing in young girls' education; fostering gender equity norms between boys and girls and making governments and donors prioritize activities that improve women's economic independence – again showing the weight the organisation places on gender inequality as a driver of the HIV epidemic. As discussed in chapter 2, the view that gender inequality is the main driver of HIV infection among women in sub-Saharan Africa is also held by a number of other researchers (e.g. Tsafack, 2008; Gupta, 2000; 2002, Jewkes, Levin and Penn-Kekana, 2003; Rispel & Popay, 2009).

However, a number of shortcomings plague this literature, notably that many of the studies arguing that gender inequality is the main driver of HIV infection among women are qualitative (e.g. Brummer, 2002; Hunter, 2007; Over, 1998) and thus cannot quantify the impact.

One way of addressing this short-coming is to employ cross-country multivariate regression modelling of HIV status. The most common method in the literature is to model HIV prevalence as a function of the several covariates of HIV (as conducted in chapter 4). Many

authors create a 'base' model, that expresses the log of HIV prevalence as a function of GDP per capita, wealth inequality (Gini coefficient) and the proportion of population which is Muslim (e.g. Mahal, 2001; Sawers, Stillwaggon & Hertz, 2008, 2010, Holmqvist, 2007). To this model, various other potential determinants of interest are added, such as poverty (Stillwaggon, 2002; 2006; Nattrass, 2009; Holmqvist, 2007), infectious disease (Sawers, Stillwaggon & Hertz, 2008, 2010), gender inequality (Tsafack, 2008) and healthcare quality (Deuchert & Brody, 2007)

This chapter investigates, using cross-country multivariate analysis, whether the southern African anomaly is driven by the fact that southern African countries have greater gender inequality. It adds to the pool of studies that have modelled HIV prevalence across countries (such as Over, 1998; Stillwaggon 2000, 2002; Mahal, 2002; Drain et al, 2004, 2006; Deuchert & Brody, 2007; Sawers, Stillwaggon & Hertz, 2008; Tsafack, 2008 Stillwaggon & Sawers 2010). The chapter however aims at performing the most comprehensive test of the impact of gender inequality on HIV in southern Africa that has been done thus far. While we cannot claim to use necessarily better measures than those that have been used earlier, we certainly use a wider range of variables than anyone has attempted on the subject matter.

The chapter comprises four sections. The next section reviews and critiques all the recent econometric research that models HIV prevalence using cross-country multivariate regression analysis. Following this literature review, we perform our own cross-country multivariate analysis using data from a number of sub-Saharan African countries. The last section of the chapter concludes.

5.2 Literature review

We search relevant literature initially using Google scholar. A key word, title and abstract search including search words 'cross-country', 'regression', 'Southern Africa', 'women',

'multivariate' and 'HIV' is made. From the initial articles obtained, we then searched reference lists and citations. The inclusion criteria are that the article contains at least one multivariate regression model where HIV or a measure of sexual behaviour is the outcome variable and the sample, or sub-sample, includes sub-Saharan African countries. The search was limited to literature between 2000 and 2011. To verify that we had reasonably included all the relevant literature, subsequent database searches through Science Direct, Ovid and Alexis were carried out.

We identify fifteen relevant studies published between 2000 and 2010 that meet our inclusion criteria. While we do not claim to cover every single paper that has been published using the methodology (owing to unknown reasons for omission), we assert that these studies accurately represent the state of existing research in this subject matter. Table 5.1 summarises the findings from the relevant literature.

Table 5:1. Summary of cross-country multivariate HIV regressions and the gender related specific findings

Study	**Research interest**	**Outcome variable and sample**	**Independent variables**	**Main Results and specific findings concerning gender inequality**	**Limitations/shortcomings**
Bonnel (2000)	Determine the key determinants of HIV prevalence in developing countries.	Log HIV prevalence 1997 59 developing countries, mostly sub-Saharan Africa	Log of the average number of phones per person (1994) Growth rate of GDP per capita (1980-90) Share of female labour in industry (1990) Muslim (percentage of population) Ethnic fractionalisation (Easterly's ethnic fractionalisation index) Time since first HIV case was reported (years) Labour migration (share of factor receipts in exports, 1990) Secondary school enrolment rate (1990)	Income inequality and ethnic fractionalisation increase spread of AIDS. Age of epidemic the most important predictor of current prevalence in this model Share of female participation in labour force negatively related to HIV prevalence.	No measures for cofactor infections
Stillwaggon (2002)	Examines the biomedical effects of conditions in Africa that contribute to high HIV transmission rates	HIV prevalence 1999 44 African, Asian and Latin countries	Change in calories per capita 1970-95 Gini coefficient Change in urban population 1970-95 (%) Real GDP per capita 1995 Change in per capita GDP 1960-95	Nutrition, distribution of income and urbanisation correlated with HIV prevalence. The lower the calorie intake the higher HIV prevalence.	Ignores gender inequality and a wider range of correlates for HIV infection

Mahal (2001)	To show that human development has a strong effect on HIV prevalence (Uses multiple models (15), and these the are results from the main model)	Log HIV prevalence 1998 73 countries	Human Development Index, 1980 Real Income Per Capita, 1980 Share of population that is Muslim (%) Mean years of schooling 15+, 1980 Ratio of female to male literacy rates Ratio of female to male life expectancy at birth, 1980 (%) Ratio of females to males 15-49 years, 1980 (%) Ratio of exports + imports to GDP, 1980 (%) Gini coefficient, 1990 Annual average increase in migrant population, 1980-90 (%) Ratio of armed forces to urban population, circa 1990 (%) Freedom Index, 1995	Human development has a strong negative impact on HIV prevalence. Two measures of human development, the log of real GDP per capita and UNDP human development measure are negatively related to HIV prevalence. The age of the epidemic the most consistent predictor of HIV prevalence. It is significant in 6 of the models. Female participation rates marginally positively related to HIV prevalence	
Gray 2004	HIV and Islam: is HIV prevalence lower among Muslims	HIV prevalence 1999 38 sub-Saharan African countries	Percentage of Muslims in total population Population Density Percentage urban population Annual per capita purchasing power Years since first recorded AIDS case	Proportion of population that is Muslim negatively related to HIV prevalence	
Drain et al (2004)	Investigate the correlates of HIV infection in all	Natural log of HIV sero prevalence in 2000 (cases per 1 000 000	Males circumcised (%) (Categorical variable (Low <20%, medium 20-80%, high >80%)	Population-level behavioural approaches, such as delaying sexual debut and	Main study conclusions based on

	developing countries	adults) 89 developing countries	Age of the HIV/AIDS epidemic Geographic region Population younger than age 25(%) Female adult illiteracy rate (%) Children fully immunized for diphtheria, tetanus, and pertussis (%) No. of doctors per 1 000 000 people	discouraging casual sex partners, and reinforce biologic measures, such as controlling sexually transmitted infections and promoting male circumcision are necessary for HIV prevention In the multivariate regression, male circumcision and age of epidemic the most powerful determinants of HIV prevalence. The effects of these are negative and positive, respectively Negative relationship between female adult literacy rate and HIV(Coeff-0.020, p-value <0.001)	univariate regressions. Study offers virtually no interpretation for multivariate regression. Extensive analysis with a wider array of covariates only performs univariate analysis.
Drain et al (2006)	Investigate the impact of Male circumcision , religion and infectious disease on HIV	Natural log of HIV sero prevalence 2004 35 sub-Saharan countries	Percent population Muslim Percentage population Christian Male circumcision prevalence	MC significantly associated with lower HIV prevalence in sub-Saharan Africa, independent of Muslim and Christian religion	Ignores most correlates of HIV infection including gender inequality and cofactor infections
Lakhanpal	Judge the effect	Log HIV prevalence	Real GDP per capita, PPP dollars,	Negative relationship	

& Ram 2006	of adult educational attainment on HIV prevalence	2001 84 Developing countries	1997 Health expenditure per capita, PPP dollars, 1997 Mean years of school of population age 15+, 1990 Proportion of population that is Muslim	between education and HIV prevalence. An increase by 10 percent in education, entered by a 10 year lag, is associated with 12-17% reduction in HIV prevalence, holding other factors constant	
McIntosh 2007	Examine the relationship between HIV and healthcare	31 sub-Saharan African countries	1980 population density (people per square mile) Percentage of road network paved, 1990 1990 per capita GDP 1980 adult female mortality per 1000	Places with the poorest health best avoid HIV.	No clear explanation for relationship between the independent variables used and study question outlined in the abstract.
Tsafack (2008) PhD dissertation, University d'Auvergne	Asses the importance of gender inequality as a determinant of HIV prevalence	Logit HIV =ln[hiv/(C-hiv)] 29 Sub-Saharan African countries Panel data analysis from 1997-2005	Female/male school enrolment Maternal leave benefits Log GNI/capita Poverty (access to water and malnutrition) Access to healthcare Adult literacy Muslim % Ethnic fractionalisation Log TV sets Contraceptive prevalence Urbanisation rate Conflict Southern/Eastern Africa dummy	Gender inequality a determinant of HIV, either at its onset or in its evolution Female participation to economic life positively related to HIV prevalence, enrolment rate negatively and maternal leave benefits negatively	Ignores cofactor infections, age of epidemic and sexually risky behaviour

			Prevalence of border country		
Holmqvist (2009) Working paper, International Policy centre for inclusive growth	Explores the link between income inequality and HIV prevalence	Log HIV prevalence 2005	Gini coefficient[35] Log GDP per capita Adult literacy rate 1995-05 Urban growth 1990-95 % Muslim Southern African dummy	Study concludes (from all analyses) that there is a clear link between inequality and HIV prevalence but that interpretation of this link is, however, subject to an array of different interpretations. Female/male literacy rates, gender empowerment index HGI-GDI rank and contraceptive use among married women independently added to the base model in separate regressions. Female literacy	

[35] These are the variables in the "base" model for sub-Saharan Africa (N=33). It is to this model that other covariates of interest are independently added. The research paper contains 28 models in total, with varying sample sizes depending on data availability.

				rate and contraceptive prevalence both negatively associated with HIV prevalence in sub-Saharan Africa.	
Talbott (2007)	What is driving the HIV epidemic in sub-Saharan Africa	HIV prevalence 2005 77 countries	Gini coefficient Female illiteracy rate Muslim percentage Commercial sex workers prevalence GDP per capita	Cross-country regression does not support the theory that male circumcision is key to slowing down the HIV epidemic. Rather, it is the number of prostitutes in a country that is a robustly significant driver of the epidemic. Female literacy rate positively related with higher HIV prevalence	Has no discussion or control for other widely accepted correlates of HIV correlates such as cofactor infections and age of epidemic
Deuchert and Brody (2007)	Test the relationship between HIV prevalence and health care transmission using failure to use auto disable syringes and tetanus cover as main independent predictors of HIV prevalence	HIV prevalence 2005 64 low and middle-income countries and in a separate model, separately, for all sub-Saharan countries for which data exists.	GDP per capita, 1990 Tetanus coverage (%), 2003 Tetanus coverage (%), 2003 (squared) Antenatal care coverage (%), 1995–2003 Physicians (/100,000 people), 1990–2004 No use of autodisable syringes (binary variable), 2003 Urban population (%), 2003 Muslim (%), 2005 Female/male ratio of adult literacy	Healthcare indicators are robustly related to greater HIV prevalence. Lower coverage of tetanus and lower use of auto disable syringes associated with higher HIV prevalence. Variable, depending on model. In main model, female/male ratio of adult literacy rates and female economic activity negatively	No age of epidemic, no cofactor infections

			rates years), 2001 Female economic activity rate (%, ages >15 years) Age of the epidemic (years)	related to HIV prevalence. N=54. Coefficients -0.07 for literacy and -2.62 for participation in economic activity	
Nattrass (2009)	Explore the relative importance of poverty as a potential driver of HIV	Log HIV prevalence 2005 78 developing countries	Log GDP per Capita 2005 Log Calories per capita 2002/04 MSM/IDU epidemics Southern Africa dummy Rest of Sub-Saharan Africa Predominantly Muslim Predominantly protestant Log of ration female: male participation rates Log of income share bottom 20% Englebert's state legitimacy	No systematic relationship between poverty and HIV, although malnutrition and economic vulnerability may increase the risk of HIV infection in some settings. Southern African dummy strong predictor of prevalence Female participation rate not a significant determinant of HIV prevalence (p-value 0.423 in full model)	
Sawers Stillwaggon and Hertz (2008)	Investigate the determinants of HIV prevalence in low and middle income countries	Log of HIV prevalence 2006 80 developing and transition countries	Log GNI/cap (PPP) Adult literacy Urban % Age of epidemic Muslim % Southern Africa dummy Contraceptive use Cofactor infections (STIs, parasites)	Treating tropical diseases may be a cost effective add on to HIV prevention treatment programs thus slowing down the spread of HIV Contraceptive prevalence associated with lower HIV prevalence	Authors cite the omission of gender inequality variables

			Age first sex females		
Stillwaggon and Sawers (2010)	Comprehensively account for the Southern African anomaly and test it significantly	Log HIV prevalence 2007 91 developing and transition countries	Combined cofactors Malaria Prevalence Southern Africa dummy Gini coefficient Log of income per capita Urbanisation percentage Age of epidemic Adult literacy rate Female participation ratio Muslim % Remittances Autodisable syringe use	Relative affluence of Southern African countries mask the vulnerability of the majority of population who are poor and who have a high prevalence of infectious and parasitic diseases. Female participation in economic life marginally negatively related to HIV prevalence.	Completely leaves out gender inequality as a potential correlate of HIV infection.

Four studies investigate the determinants of HIV in general (Bonnel, 2000; Drain, 2004; Talbott, 2007 & Sawers, Stillwaggon and Hertz, 2008). In these studies, the question of interest is what drives HIV in sub-Saharan Africa. Nattrass (2009), Holmqvist (2009) and Mahal (2001) focus on poverty and income inequality as potential determinants of HIV prevalence. Two studies, (Deuchert & Brody, 2007 and McIntosh, 2007), look at the correlation between healthcare indicators (e.g. use of autodisable syringes, healthcare expenditure and vaccination coverage) and HIV prevalence. Three articles look at the relationship between biomedical conditions and HIV: Drain and colleagues (2006) specifically probe the link between the prevalence of male circumcision and HIV, Stillwaggon (2002) at poverty and Sawers and Stillwaggon (2010) at poverty and infectious disease. Single studies explore the role of gender inequality (Tsafack, 2008), educational attainment (Lakhanpal & Ram, 2006) and Islam (Gray, 2004), on HIV prevalence.

The sample sizes range from 29 sub-Saharan African countries (Tsafack, 2008) to 91developing countries (Stillwaggon & Sawers, 2010) and in 11 of the studies, the log of HIV prevalence is the outcome variable. In three studies, Stillwaggon (2002), Talbott (2007) and Lakhanpal & Ram (2008), the authors do not mention any transformation of the dependant variable and we assume that the dependent variable in use is HIV prevalence, i.e. not the log thereof. Tsafack (2008) uses the a logit transformation of HIV prevalence (defined as ln[HIV prevalence/1-HIV prevalence)]) as the independent variable. In the remaining study, McIntosh (2007) the construction of dependant variable is not clear but it is calculated from the "*annual median prevalence rate at UNAIDS/STI sentinal testing sites*" (McIntosh, 2007:17).

The most common practice in the literature is to develop a 'base' model that predicts HIV prevalence (or other dependant variable) first, and then to this base model add the specific

variables of interest to the authors. This 'base' model, in theory, should control for the basic or most common (or widely agreed upon) variables that are assumed to affect HIV prevalence. GDP per capita, the Gini coefficient, adult literacy rate and the percentage of population that is Muslim are the most widely used variables in the base model.

At a national level, per capita GDP is a sign of the level of development in a country. High per capita figures are associated with high rates of urbanisation, road network coverage and better healthcare facilities. Countries with low GDP per capita might therefore have higher HIV prevalence because the health infrastructure may not be as well developed, thus STIs may go untreated and this could exacerbate HIV transmission. Low GDP per capita may also infer that most people are poor (in an absolute sense) and to the extent that poverty drives risky behaviour, one might expect GDP to be positively correlated with HIV prevalence. High GDP per capita may also, however, be associated with higher HIV prevalence in that urbanisation and well developed transport networks enable a faster transmission of HIV. Furthermore, the degree of inequality is also important in structuring how many people are (absolutely) poor. Whether or not the level of development affects HIV prevalence is an empirical question.

The results from the relationship between national income and HIV prevalence are mixed. Four studies show a negative relationship between GDP per capita and HIV prevalence (Sawers and Stillwaggon, 2010; Deuchert and Brody, 2007; Mahal, 2001 and Sawers, Stillwaggon and Hertz, 2008). Countries with a lower GDP per capita are more likely to have big HIV epidemics. In four studies, national income is not correlated with HIV (Bonnel[36],

[36] Uses the growth rate of GDP and not GDP per capita.

2000 & Holmqvist, 2007; Tsafack[37], 2008; Nattrass, 2009). GDP per capita is widely in use as a predictor of HIV prevalence and yet it seems to perform poorly as a regressor.

A likely reason for this is that the relationship between GDP per capita and HIV is subdued by context-specific social and behavioural factors (Nattrass, 2009). While poor people may have limited access to treatment for STIs and engage in higher risk survival methods such as transactional sex, wealthier people may more likely move about more frequently, have more complex sexual patterns and this could increase their HIV risk. Nattrass (2009) specifically dwells on the relative importance of economic factors such as poverty and sexual behaviour in driving the African HIV epidemic. Although GDP per capita is initially (while controlling for type of epidemic and calorie intake) positively correlated to HIV prevalence, its significance falls away once the log of income share of the bottom quintile is added to the model (Nattrass, 2009:3).

While GDP per capita shows some information on the level of income, it does not reveal how this income is distributed. The Gini coefficient, a measure of how income is distributed, is a better proxy for income distribution. There are different possible reasons why the Gini coefficient correlates with HIV prevalence. Some argue that unequal societies have high levels of social friction and thus lack social capital – and social capital helps societies to mobilise and fight against disease (Barnett & Whiteside, 2006; Holtgrave & Crosby, 2003). Other authors argue that the Gini is a proxy for poverty, as very poor countries generally have high Gini coefficients (Stillwaggon & Sawers, 2010; Talbott, 2007). Stillwaggon and Sawers (2010) argue that the Gini coefficient simply highlights the proportion of those who are poor, and among the poor, treatment for STIs and other cofactor infections is low and calorie intake poor, and these conditions worsen HIV risk.

[37] Uses the log of GNI instead of per capita GDP

The Gini coefficient consistently predicts a positive relationship with HIV prevalence. All the studies (nine in total) that include the measure show this relationship. Holmqvist (2007), in addition to the Gini coefficient, also includes a measure of absolute poverty - the proportion of the population living under US$1 per day - and this variable, together with the Gini coefficient, is positively related to HIV prevalence. From this result, it would appear that the Gini is not just picking up a problem of poverty, but in part perhaps substantiates the suggestion that inequality contributes to worse HIV epidemics.

Nattrass (2009) shows the log of income share of the bottom 20 per cent of population to be negatively related to HIV prevalence – the larger the share of income the poorest have, the smaller the HIV epidemic. This significance however falls away once a regional variable (distinguishing Southern Africa from rest of sub-Saharan Africa), religion (Muslim and protestant) and the ratio of male to female participation rates are included in the model. It is not clear which of the three additional variables cause the significance of this poverty measure to drop in the model.

Change in per capita calorie intake is included in research as a possible measure of poverty. Stillwaggon (2002) argues that sub-Saharan Africa is the only region in the world that experienced a drop in food production in the 1980s leading to a drop in caloric intake, and this drop left many people undernourished and more vulnerable to HIV infection than populations elsewhere (Stillwaggon, 2002). Stillwaggon (2002) and Nattrass (2009) both show that the change in caloric intake per capita between 1970-1995 and caloric intake in 2002/2004, respectively, are both negatively related to HIV infection. Neither study is able to specify whether this relationship is because of better diet, or whether caloric intake is a proxy of some other underlying condition such as poverty.

Several studies reveal that the higher the proportion Muslim, the lower the HIV prevalence (Tsafack, 2008; Talbott, 2007; Sawers, Stillwaggon & Hertz, 2008; Mahal, 2001; Gray, 2004; Bonnel, 2000). This variable is the most consistent predictor of HIV prevalence in the literature, with the negative relationship between the percentage of population Muslim and HIV prevalence in all the studies that include the variable. The reasons for this correlation are not clear, but one possible reason is that in predominantly Muslim societies, circumcision is high – and male circumcision is often associated with lower HIV risk (Talbott, 2007, Drain et al, 2006). In the latter study, the authors find male circumcision prevalence and proportion of population that is Muslim highly collinear, and drop the Muslim variable because male circumcision was a 'better fit'[38] in their model. An alternative argument is that Muslim societies typically have restricted sexual behaviour – age of marriage is low and marriage rates are high, for example, potentially locking up women and men in confined sexual relationships (Gray, 2004). Other reasons could include personal hygiene and sexual network structures (Deuchert & Brody, 2007; Gray, 2004) but literature on cross-country determinants of HIV does not dwell much on uncovering the reason behind low HIV prevalence rates among predominantly Muslim populations.

Three studies use male circumcision as an explanatory variable (Drain et al., 2004, 2006; Holmqvist, 2007) and show a strong negative relationship between male circumcision and HIV prevalence. Although Drain and colleagues (2004) find male circumcision significantly associated with HIV prevalence, the methodology is unclear and the multivariate model only controls for a handful potential other covariates of HIV infection.

Talbott (2007) however argues that male circumcision is not an important factor in determining HIV epidemics in sub-Saharan Africa. The author argues that the proportion of

[38] No additional details about what made it better are given

population that is Muslim is in fact a proxy for the prevalence of male circumcision (a finding also noted by Drain et al., 2004:9), and that the proportion of population that is Muslim becomes a very poor explanatory variable once the prevalence of sex workers is added. Predominantly Muslim countries show a strong negative correlation with the prevalence of sex workers (correlation coefficient -1.897, standard error 0.396, t-stat -2.265). While Talbott (2007) claims that male circumcision is not important, none of the models presented in paper actually attempt to show the significance of the male circumcision variable falling away once the prevalence of sex workers is included. This interaction between male circumcision, proportion of population that is Muslim and HIV prevalence is a largely unexplored area of research.

Five of the studies include a regional coefficient and in all these studies, the Southern African coefficient is the most significant, after controlling for at least national income and income inequality. It is of widespread scholarly debate why the southern African anomaly exists. Possible reasons include gender inequality (Tsafack, 2008), poverty (Stillwaggon and Sawers, 2010; Sawers, Stillwaggon and Hertz, 2008), the high prevalence of commercial sex workers (Talbott, 2007) and the legacy of colonial history that necessitated the use of migrant labour and construction of good road transport networks (Nattrass, 2009; Holmqvist, 2007).

No single study using cross-country multivariate regression analysis has managed to explain away fully the effect of the Southern African dummy, although some have managed to reduce the size of the coefficient (e.g. Stillwaggon & Sawers 2010). Talbott, 2007 claims that Southern Africa is expected to have a higher HIV prevalence due to the high prevalence of commercial sex workers but does not test this correlation specifically in the regressions. Similarly, Stillwaggon and colleagues (2010) argue that it is poverty and not Southern Africa that makes HIV prevalence so severe in the sub-region. However, after including their

measures of poverty that the include the Gini coefficient[39], log of income per capita and a number of measures for STIs and cofactor infections such as malaria, they do not eliminate the statistical significance of the Southern African coefficient and the size remains large. The authors manage to reduce the size of the coefficient from 2.7 (p-value 0.00, using ordinary least squares regression, N = 91) in the base model to 1.8 (p-value 0.00, N=91) in the complete model.

Holmqvist (2007) presents a regression model in which he substitutes HIV prevalence with indicators of risk behaviour. In this model, the author regresses the Gini coefficient, adult literacy, the proportion of Muslim, the log of GDP and urban growth on female age at first sex. Then, in successive rounds, while keeping the same independent variables, uses the male age at first sex, young female premarital sex and commercial sex worker prevalence as the dependant variables. This specification of the model shows that the sub-Saharan Africa dummy is positively correlated with young female premarital sex and commercial sex worker prevalence and negatively related to a young age at first sex. The paper does not go on to discuss these findings in detail, but this suggests that sexual patterns are not the same everywhere.

Surprisingly, only one other study (Sawers, Stillwaggon & Hertz) includes sexual behaviour in the models of HIV prevalence. We would have expected more studies to include sexual behaviour variables in cross-country analyses given the widespread acknowledgement that sexual behaviour plays a significant role in shaping HIV epidemics. The authors find a negative relationship between the age at first sex and HIV prevalence.

[39] The authors argue that a high Gini coefficient is indicative of a large proportion of poor people and thus is also a measure of poverty.

Mobility, migration or internal movement are possible determinants of HIV prevalence. The one argument is that the demand for commercial sex and multiple sex partners depends on the need and opportunity for such interactions to occur. Thus, "*mobile populations with extra income such as migrant labourers, truck drivers, members of armed forces and possibly tourists – (along with a combination of separation, loneliness and anonymity) are especially likely to demand commercially available sex, or engage in multiple-partner sex*" (Mahal, 2001:2 as cited in Bloom & Mahal, 1995). Another argument is that migration masks the poor living conditions that migrants are often exposed to (such as poor sanitation, unsafe drinking water and a high prevalence of infectious disease), and these are associated with high HIV prevalence (Stillwaggon & Sawers, 2010).

In theory, migration therefore relates to higher HIV prevalence through two pathways. People moving from place to place can facilitate faster transmission of the virus, or, alternatively, the characteristics of these people make them more prone to HIV themselves, either due to risky behaviour, or due to living conditions surrounding them, or a combination of both. Only four studies however include measures of migration from this set of cross-country studies (Mahal, 2001; Bonnel, 2000; Stillwaggon & Sawers, 2010 and McIntosh, 2007)

Due to data constraints, different measures of migration are in use – for example remittances from abroad as a proportion of GDP (Stillwaggon, 2010), population growth 1980-1990 and average annual increase in migrant population 1980-1990 (Mahal, 2001), labour migration (Bonnel, 2000) and length of road network 1990 (McIntosh, 2007). The empirical evidence is weak though, with only one of these studies showing a statistically significant relationship between migration and HIV prevalence (Bonnel, 2000). This study shows a small positive relationship between labour migration and HIV prevalence.

The age of the epidemic is another potential determinant of HIV prevalence. Crudely, and regardless of other factors, HIV prevalence can be expected to rise sharply in the early stages of the epidemic. This is owing to the fact that relatively few people know about it and symptoms only develop much later. After some time, the epidemic peaks (as in heterosexually driven epidemics it can only affect sexually active people and there is a finite number of these in a population). After this peak, historical trends show that the progression is unpredictable: it will depend on awareness, sexual behaviour, and possibly treatment regimens (Low-beer & Stoneburner, 1997). It may remain constant around the peak prevalence rate (as in the case with many Southern African countries) or it may decline (as happened in Uganda) or drop, and then maintain a constant, albeit low prevalence level (as is the case with most Central, East and Western Countries).

All but one study that include the age of the HIV epidemic (measured by the number of years since first HIV or case was officially reported) show a positive relationship between the age of epidemic and HIV prevalence. Countries that reported HIV the earliest are most likely to have bigger epidemics (Drain et al., 2006, Sawers, Stillwaggon and Hertz, 2008; Stillwaggon & Sawers, 2010). Holmqvist (2007) finds no relationship between the age of the epidemic and HIV prevalence.

Only one study specifically looks at the relationship between gender inequality and HIV (Tsafack, 2008). The study uses panel data (looking at the countries from 1995 to 2000 for 29 sub-Saharan African countries). In the main model, gender inequality is measured by the female school enrolment rate, maternal leave benefits (in weeks) and the female labour force participation rate. While controlling for income (log GNI per capita and Gini coefficient), access to healthcare, education (adult literacy), access to information (log of TV sets per capita), contraceptive prevalence, ethnic fractionalisation, proportion Muslim and region

(East and Southern Africa dummy), the author concludes that gender inequality variables are "*critical*" determinants of the epidemic (Tsafack, 2008:22). The study concludes that inequality is a determinant of HIV either for its onset or in its evolution. From the regressions, the proportion of women in the labour force is positively related to HIV prevalence (Coefficient 0.00, t=2.01) but the female school enrolment rate and maternal leave benefits are negatively related to the log of HIV prevalence (Coefficients -0.011 (t=2.19) and -0.001 (t=1.76), respectively.

Tsafack (2008) is the most comprehensive test of the relationship between HIV and gender inequality thus far. However, it has some limitations. Firstly, the variables for gender inequality of choice are not necessarily reflective of gender inequality, but rather more generally, the living standards of women. Differences in the length of maternity leave for example might not necessarily measure inequality, or gender specific disadvantage for that matter – it could simply be a measure of how the differences in business practice between regions affect HIV prevalence, for example. Secondly, the study does not control for other potentially important determinants of health that have been cited elsewhere such as cofactor infections, the age of the epidemic and sexually risky behaviour. These variables could prove instrumental in predicting HIV prevalence or reducing the impact of gender variables on HIV prevalence.

One of the limitations of literature in this genre is that it typically does not distinguish general disadvantage from gender inequality. Female labour participation rates, female literacy rates and maternity leave benefits are some of the more common variables used to denote gender inequality (e.g. in Bonnel, 2000, Drain et al., 2004, Tsafack, 2008) and yet these measures do not measure inequality per se but rather point to disadvantages experienced by women. These variables assume then, than any sort of disadvantage accruing to women, is because of gender

inequality – and this is not true. A low female labour participation rate, on its own, may be indicative of a general lack of employment opportunities in a population, rather than an unequal distribution of employment, for example.

Results from these measures of female disadvantage/inequality are mixed. Bonnel, 2000; Mahal, 2001; Holmqvist (2007) and Stillwaggon and Sawers (2010) find that low female participation in the labour market and low female literacy rates predict higher HIV prevalence. Nattrass (2009) finds no relationship between female participation and HIV prevalence while Mahal (2001) and Talbott (2007) find that higher participation rates are in-fact linked with higher HIV prevalence.

At an individual level, education is widely linked to lower HIV risk, and although the discussion is open, it seems widely accepted that an educated population is better able to deal with the progression of HIV than an uneducated one (Gregson et al., 2006). Educated people are more likely to respond better to calls for behaviour change and are able to access prevention and protection information.

A few key revelations become apparent after addressing the literature on cross-country multivariate analysis. Firstly, a number of studies use GDP per capita as a regressor and yet it consistently performs poorly. Gender inequality as a determinant of HIV is inadequately explored. Only one study performs an in-depth analysis and despite its strengths, it has some shortcomings. Some literature leaves out the age of the epidemic in the base model, and yet this performs powerfully in most of the models it is included. The relationship or interaction between commercial sex work, proportion Muslim and male circumcision needs to be explored further. Lastly, there is a tendency for gender inequality and absolute disadvantage among women to be conflated. The literature is not clear whether this is a data availability problem, or whether authors believe the two to be synonymous.

5.3 Empirical Analysis

This section conducts a cross-country regression analysis that presents comprehensive models to test whether gender inequality may be responsible for the disproportionate nature of the southern African HIV epidemic. While in chapter 4 we test the impact of various individual characteristics such as sexual behaviour and SES on HIV, individual-level data makes it difficult to assess the influence of macro-level factors that may be operating across different sub-regions within sub-Saharan Africa. Using cross-country data, we are able to explore the effect of various variables (such as inequality, poverty and access to healthcare) on adult HIV prevalence across different countries.

We apply a simple methodological approach. Firstly, and in line with most of the literature cited earlier that uses cross-country regression analysis to probe some of the determinants of HIV prevalence, we construct a base model with widely accepted determinants of HIV prevalence. In this base model and all other models, the log of HIV prevalence in 2007 is the outcome variable. To this base model, we then successively add more explanatory variables and track the evolution of the coefficients and their level of significance. The following section provides a detailed description of the data and the theoretical hypotheses underlying our model.

5.3.1 Independent variables

5.3.1.1 Base variables

At a cross-national level, income could drive national prevalence in two main ways. Firstly, poor countries may have bad health infrastructure, and this could lead to a higher transmission of HIV through cofactor infections. Secondly, in poor countries, individual-level poverty may drive individuals to higher risk sexual practices such as transactional sex. To capture the impact of inequality, we use the Gini coefficient and the log of the income share

of the poorest quintile. If wealth differentials encourage transactional sex and other risky behaviours at all levels of income (for example, relatively poorer women engaging in transactional or intergenerational sex to obtain status and luxury items) then this will be picked up by the Gini coefficient. If, however, the key driver of risky sexual behaviour is poverty, (where poor women engage in transactional sex for survival, or there is a persistence of cofactor infections due to a large proportion of poor people) then the proportion of the population in the bottom quintile will pick this up.

A more educated population may be able to respond to an epidemic faster than a less educated population. The level of literacy in a population may thus be correlated to HIV prevalence and in the base model, we include the adult literacy rate.

Ordinary least squares regression assumes a linear relationship between the explanatory variables and the outcome variable. The progression of HIV through a population over time is however not linear owing to the time between HIV infection and death, which in the absence of ARV treatment is between 6 and 9 years (Leclerc, Mathews & Garenne, 2009:1). Without any intervention, HIV prevalence in a population can be expected to rise in the early years of an epidemic but then have a tipping point at some time as mortality rises from AIDS. To control for these epidemic dynamics, we include the age of the epidemic in the base model.

Age of epidemic is measured by the number of years up to 2007 since the first case of HIV was reported. The number of years since HIV was first reported is the most widely used measure in the literature (see table 5.1). This is not a very accurate measure as it completely ignores the rate of HIV spread in the early years and thus assumes that for all countries, HIV progressed at the same rate from the time the first HIV diagnosis was made. It also implies that the year of first diagnosis occurs, for all countries, when the HIV epidemic in each

country is at the same stage[40]. Possible alternative measures include the distance from Kinshasa to the country in question as a measure of the spread or the time since HIV prevalence exceeded some objective measure e.g. 1 per cent. We argue that using an "objective" measure has inconsistencies too, as the progression of HIV both within and between countries in the early years of the epidemic is not known. By the time the first HIV antibody test was approved in 1985, WHO already suspected that in at least 11 African countries, adult HIV prevalence exceeded 5 per cent.

Distance from Kinshasa primarily assumes that HIV spread as a function of road networks linking Kinshasa to the rest of East and Southern Africa. While this may have been the case, the rate of spread would have more likely been determined by the relative economic strength of the towns and cities along the chain. In the light of the limitations of the other methods in the literature that measure the age of the epidemic, we reason that age since first HIV case was reported at least has the advantage of comparability to other studies that have used it. We also include the square of the age of epidemic to test whether the variable has a turning point.

The proportion of population that is Muslim is used as a proxy for religion. The proportion of population Muslim has been discussed earlier in this chapter as a viable proxy for male circumcision as well, due to near universal circumcision among Muslims. Since data on circumcision prevalence is not easily accessible and when it is, estimating circumcision prevalence using point estimates is likely to include estimation errors, we deliberately use proportion of population Muslim as a measure that could show the impact of religion on HIV prevalence, or the impact of male circumcision on HIV prevalence. We reflect on this some more when we discuss the results.

[40] The first diagnosis in country A could have happened when national prevalence in that country was, for example 2 % while the first diagnosis in country B could have happened when national prevalence was 5%.

Internal and external migration results in movement of people within and across national boundaries. Such movement may be accompanied sexual mixing and sexual mixing may spread HIV. Stillwaggon and Sawers (2010) also argue that the state of sanitation and general health conditions many migrants face are poor, and that they are at higher risk of HIV infection because their immune systems are accordingly compromised. Measures of migration may capture this effect of how mobile a population is, and the corresponding elevation in HIV risk.

Migration is problematic to measure. Some studies have used urban growth as a proxy measure for rural-urban migration while others have used remittances from abroad as a proxy variable (table 5.1). We opt to use remittances from abroad as a percentage of GDP as remittances per capita represent those people living in other countries who may not have been accounted for using formal migration methods (e.g. illegal immigrants). An inherent assumption in using this measure is that amount of remittances received in a country is a measure of the number of people who migrate in and out of that country. Remittances from abroad also accounts for those individuals migrating that are not officially included in migration statistics, e.g. illegal immigrants, and is therefore more likely to be a closer approximation of the proportion of population that may be shuttling back and forth between countries.

We include a secondary migration measure, proportion of population that is urbanised. A high proportion of urbanisation is not in itself a direct cause of higher HIV prevalence, but is indicative of internal migration rates (Dyson, 2003).

To control for the type of epidemic, we control for Sub-Saharan Africa and Southern Africa with dummy variables. This method assumes therefore, that there are 3 epidemics we are controlling for, a southern African epidemic, a sub-Saharan African epidemic and an "other"

epidemic for the rest of the world. While the rest of the world does not have a single epidemic, for the purposes of this study, distinguishing the sub-Saharan epidemic from the rest of the world and further distinguishing the southern African epidemic from this will suffice.

5.3.1.2 Cofactor infections and health quality

At an individual level, STIs can cause higher HIV risk as some of them are cofactors to HIV (discussion in chapter 3) and this possibly facilitates HIV transmission. Faster HIV transmission at an individual level in a country could then lead to higher national HIV prevalence. Nutritional deficiencies may also exacerbate individual rates of susceptibility (as discussed in chapter 2) and over a sustained period of nutritional deficiencies, this could result in higher HIV prevalence.

To capture the impact of STIs and nutritional deficiencies, we include as regressors the log of the disability-adjusted life year (DALY) measure of the burden of disease caused by classical STIs (syphilis, chlamydia and gonorrhoea) and nutritional deficiencies. The DALY is a WHO measure that quantifies the burden of a disease in a country by estimating years of life lost due to individuals prematurely dying and time lost due to individuals living in a state of less than perfect health. A detailed explanation of how the measure is computed, and its weaknesses are extensively discussed by Murray (1994) as well as a main critique by Anand & Hanson (1997). In short , the measure computes the number of lives that are lost due to a disease and the equivalence of life lost if a person is living in a state of disability or less than perfect health all weighted by the age at death or of disability. In appendix III, we compare the logged and unlogged DALYs for these two measures and the kernel density plots show that the logged measures provide a more normal fit than the unlogged measures.

The overall state of a country's healthcare system is measured by the corresponding UNHDR index. The UNDR 'health care system' index rates the overall healthcare system in a country by assessing the average healthcare quality of health centres and the proportion of population with access to the health services. Where healthcare systems are poor or sanitation conditions low, a population may have higher HIV prevalence due to parasitic and cofactor infections (see chapter two).

5.3.1.3 Gender variables

We classify gender variables into two categories. Those that measure general gender disadvantage and those that measure gender inequality. Inequality variables are those that measure the comparative difference between men and women, such as the male/female literacy rate, as compared to those that just measure disadvantage such as the female literacy rate.

Different ways to measure gender inequality exist and a number of them focus on the dimensions of education, political representation and economic control of resources (e.g. UN, 2004; Dollar & Gati; Dorius 2010). The United Nations develops a gender empowerment measure and uses the ratio of literate women to men, ratio of girls to boys in primary, secondary and tertiary education, proportion of seats in parliament and proportion of wage earners in the agricultural sector who are women (UN, 2004).

Measures of general disadvantage are those that represent absolute disadvantage among women without any comparison to men. In the literature, educational attainment among women, economic activity and life expectancy are used (e.g. Dorius, 2010). Household specific indicators that include the right of women to refuse sex (or lack of) perceptions about the acceptability of wife beating and withdrawal or withholding of financial support by men have also found themselves in the literature (e.g. Aparna & Dipanwita, 2011).

To capture general disadvantage among women, we experiment with an index of polygamy. Polygamy is measured using a categorical variable, low (for countries in which less than 5 per cent of the men are in polygamous marriages), medium (5-15 %) and high (15 %+). The prevalence of modern contraceptive use may also represent general disadvantage among women as it may represent how much of a priority, at a national level, women's reproductive health is. Average maternity leave benefits are measured in weeks of average maternity leave granted with partial or full remuneration.

Jensen, (2002) (unpublished), in a discussion of how fertility practices can affect socio-economic outcomes, argues interestingly that in a society where there is a preference for male children over female children[41], parents need not treat their children differently, in order to have worse outcomes for girls than for boys (Jensen, 2002). In societies where a preference for boy over girl children such preferences prevail, couples tend to have more and more children until they have their desired number of boy children. Girls therefore, on average, will tend to have more siblings than boys, hence come from larger families. Assuming that the household income is for the most part independent of the number of children in the household, girls will thus tend to come from poorer families than boys do, as their families tend to be larger. This worsens their socio-economic background, not a result of any direct discrimination but because of the underlying preference for boys over girls.

The UN index has a measurement for preference for sons. This is measure calculates a preference of sons due to missing women – which measures gender bias in deaths among baby girls due to selective abortion or insufficient care (http://genderindex.org/content/social-institutions-variables). Son preference is categorised as a binary variable separating those

[41] This preference can actually be rational, given the socio-cultural set up of the society. Southern and East African societies are patriarchal, and due to this, boys may be preferred to girls in order to keep the lineage going.

countries with a low son preference from those with a high son preference. We experiment with this measure and attempt to see if a prevalence for sons (which is a gender bias) accounts for some of the variance in HIV prevalence between countries.

5.4 Potential limitations

5.4.1 Robustness checks

We carry out a number of post estimation techniques to check if our regressions are robust. First, for all regressions, we apply the 'robust' option when using Stata. This option estimates standard errors using the Huber-White estimators and can deal with a number of minor issues surrounding failure to meet assumptions such as small problems about normality, heteroscedasticity and outliers (http://www.ats.ucla.edu/stat/stata/webbooks/reg/chapter4/statareg4.htm).

Secondly, for every regression, we carry out a goodness of fit test. Goodness of fit is measured using the 'linktest' command. Third, we check for collinearity using the variance inflation factors (VIF). Fourth, we test for normality by plotting the kernel density estimates of the residuals against a normally distributed function. The other test we carry out are for strong heteroscedasticity, and we use the Breusch-Pagan test. Lastly, we perform the Wald test for every successive model after the Base model. The regression models we run are nested models, where every other model is the "base model" plus a group of other variables (such as cofactor infections in the second model, or gender inequality measures in the third). The Wald test tests whether the variables that are added to the base model result in a better fit of the model, i.e. that the new coefficients are jointly not equal to zero.

Over and above these post estimation techniques, we also run alternative regressions to check if the coefficients are sensitive to the variables included in the model. As discussed earlier,

the Gini coefficient could well be picking up the effects of poverty and or the broader income distribution on HIV. We thus include as regressors the Gini, and the wealth share of the bottom 20 per cent of the population. None of the studies we review adopt this method, and for comparability, as well as checking the sensitivity of our models to a change in variable specification, we run, alternative regressions while excluding the Gini measure. These are presented in the appendix, although where appropriate, we will discuss them along with our main results.

5.4.2 Practical data limitations

Missing data is a significant problem. In all regression analysis, we rely on STATA's list wise deletion function. STATA omits any observation for which the entire regression model cannot be specified, i.e. if for example contraceptive prevalence data in a country is not available, then that country is left out of that regression model altogether. In table 5.2 where we include all developing and transition countries, the regression sample sizes vary from 50 to 62 countries, a 20 per cent change in sample size. This can cause coefficients to change between different model specifications not because of a robust change in the coefficient estimates, but simply because the underlying data set has changed. To assess the impact of changing data points, we replicate all the regressions from table 5.2 in the appendix, restricting the sample for all regressions to include only those countries available for all regressions.

Regressions with the sample size restricted to sub-Saharan countries typically suffer from low sample sizes (see for example Tsafack, 2007). In our case, the samples vary from 19 to 25. While these are small sample sizes, we have the necessary degrees of freedom necessary for running valid regression models that produce unbiased estimators.

In the appendix, we also present two tables with list for the specific countries that are used in each regression.

5.5 Results

Table 5:2 Multivariate regressions, developing and transition countries

Log HIV 2007	**Base (16)**	**Cofactor infections and health (17)**	**Gender inequality (18)**	**General disadvantage for women (19)**
Southern Africa **Coeff. (t) P**	1.24*** (3.91) 0.000	1.08*** (2.65) 0.011	1.55*** (6.56) 0.000	1.22*** (4.06) 0.000
Sub-Saharan Africa	2.46*** (9.21) 0.000	2.25*** (6.44) 0.000	2.26*** (6.65) 0.000	2.52*** (5.51) 0.000
Gini	0.05 (1.63) 0.110	0.03 (1.00) 0.323	0.05 (1.68) 0.101	0.04 (1.31 0.197
Log of share of income bottom 20% of population	0.91 (1.31) 0.197	0.62 (0.78) 0.441	0.86 (1.25) 0.219	0.79 (1.39) 0.173
Proportion urbanised	-0.05 (-0.81) 0.422	0.00 (0.06) 0.951	0.00 (-0.09) 0.927	-0.01 (-0.79) 0.432
Adult literacy	0.01** (1.70) 0.095	0.01 (1.16) 0.251	0.02 (2.07) 0.045	0.00 (0.52) 0.251
Age of epidemic	-0.68** (-2.49) 0.016	-0.75** (-2.17) 0.035	-0.63** (-2.31) 0.026	-0.51** (-2.34) 0.025
Age of Epidemic squared	0.02*** (2.79) 0.007	0.02*** (2.39) 0.021	0.02*** (2.45) 0.019	0.01*** (2.58) 0.014
Proportion Muslim	-0.01** (-2.49) 0.016	-0.01** (-2.30) 0.026	-0.01** (-2.16) 0.037	-0.01** (-1.93) 0.061
Log of remittances received per capita	0.10** (2.16) 0.035	0.10** (1.97) 0.055	0.09** (1.82) 0.077	0.13** (2.55) 0.015
Log of STDs DALY		0.31** (1.12) (0.268)		

Log of Nutritional deficiencies DALY		0.00 (0.02) 0.988		
Healthcare quality		0.01 (1.27) 0.210		
Proportion of women seats in parliament			-0.12 (-1.52) 0.136	
Female labour force participation rate			0.00 (-0.22) 0.828	
Proportion women secondary school relative to men			-0.01 (-1.31) 0.199	
Maternal leave length (Weeks)				-0.7* (-1.93) 0.062
Contraceptive prevalence percentage				0.02* (1.78) 0.084
Medium rates of polygamy				0.19 (0.70) 0.488
High rates of polygamy				0.43 (1.56) 0.128
High son preference				-0.66* (-1.99) 0.055
N	62	57	53	50
F-test (P-value)	0.00	0.00	0.00	0.00
R2	0.84	0.71	0.68	0.91

Controlling for sub-Saharan Africa and holding the Gini coefficient, income share of the bottom 20 per cent, proportion of population that is urbanised, the adult literacy, age of epidemic, proportion of population Muslim and remittances per capita constant, southern African countries have, on average, a prevalence rate 15 per cent higher HIV prevalence than other developing and transition countries. A change from a non-southern African country in sub-Saharan Africa to a southern African country increases the natural log of HIV by 1.24

units, i.e. by $e^{1.24} = 3.45$ per cent. In the base model, the southern African coefficient is the second strongest predictor of HIV prevalence, after sub-Saharan Africa.

In this base model, the age of epidemic, and proportion of population that is Muslim are negatively related to HIV prevalence. A one per cent increase in the proportion of the population that is Muslim decreases HIV prevalence by approximately one per cent, holding the other covariates constant. Holding the same covariates of HIV prevalence constant, an increase in adult literacy by one per cent predicts that HIV will be lower by a single percentage point. The age of the epidemic coefficient is much smaller though, with the model suggesting that an increase in the age of epidemic by a year, all-else equal, increases the expected HIV prevalence by 0.5 per cent.

The proportion of population that is urbanised, the Gini coefficient and the log of the income share that the poorest 20 per cent of the population hold are all not significantly related to HIV prevalence in this model. Lastly, the measure of migration, remittances from abroad per capita (logged) are related to HIV prevalence with an increase in the remittances of 1 per cent increases HIV prevalence by $1.01^{\wedge 0.1} = 1$ per cent, holding everything else constant.

All the regression coefficients in this model carry expected signs in line with other research that adopts the same methodology. Sub-Saharan African countries have higher prevalence rates that the rest of the world, and among them, those in Southern Africa an even higher prevalence rate while controlling for a number of HIV prevalence correlates. The tests do not find evidence of misspecification, heteroscedasticity or multicollinearity.

In the second stage of the modelling, (17) we add to the base model measures of poor health. The DALY for the classical STIs (logged) is not related to HIV prevalence with the coefficient not statistically significant while controlling for nutritional deficiencies,

healthcare quality and the base model. Nutritional deficiencies in any country are also not related to HIV prevalence and neither is the measure of healthcare quality. None of the three variables of cofactor infections and health are statistically significant, and the Wald test (Appendix V) shows that the three variables do not provide a better fit and we cannot reject the hypothesis that the three new coefficients are jointly equal to zero.

Since these three variables do not add anything new to the model, if the model is correctly specified we do not expect the coefficients in the base model to change by much – and they do not. All the estimators that make up the base model keep their signs and with the exception of the Southern African coefficient that drops, all the others remain more or less the same. The magnitude of the coefficient change is the Southern African coefficient is not large, with the new coefficient predicting and increase in HIV prevalence by 2.9% this time, compared to 3.45 per cent in the base model alone.

Model 17 has fewer observations than the base model, and to rule out the potential effect of this reduction in sample size we replicated both regressions using only the countries common to both. These are reported in Appendix VIII. As the appendix shows, when the models are replicated on the same sample, the cofactor and health measures are not significant. In the replica model, keeping the sample fixed, there is no notable change in any of the coefficients from the base variable. Appendix (VIII) also reveals that the countries Lesotho, Swaziland and Zambia are excluded from regression 17, and this would most likely explain the drop in the southern African coefficient. In another regression, this time excluding the Gini coefficient (Appendix XI) these variables remain statistically insignificant.

This model, suggests therefore, that STI infections, the state of the healthcare system and nutritional deficiencies are not strong predictors of HIV prevalence once the above covariates are controlled for.

In model 18, we add to the base model measures of gender inequality. Measures of gender inequality are not strongly related to HIV prevalence. The female labour force participation rate, proportion of women (compared to men) in secondary school and proportion of women seats in parliament are all not statistically significant at conventionally reported significance levels (p-values (0.82, 0.19 and 0.13), respectively. The Wald test for model fit in Appendix V shows that we cannot reject the hypothesis that these three coefficients are jointly equal to zero. The base coefficients, in theory, should thus not change by much and this is confirmed in regression model 18.

Measures of general disadvantage among women however are stronger predictors of HIV status than the other classes of explanatory variables we add. Longer maternal leave length predicts lower HIV prevalence, with a one-week increase in maternal leave length reducing HIV prevalence by 0.5 per cent. If the length of maternity leave is a proxy for the emphasis a country may have on women's health, then this would make sense as women who are afforded more leave are potentially likely to be afforded other health-enhancing benefits.

Controlling for the base model, the prevalence of polygamy does not appear to be related to HIV prevalence. Countries that have moderate or high rates of polygamy do not show a statistically different HIV prevalence rate that those with low polygamy.

The preference for sons is also associated with HIV, with those countries in which the preference for sons is high reporting lower HIV prevalence rates. A-priori, we would have expected a positive relationship between a high son preference and the prevalence rate, perhaps because in places where sons were preferred to girl children, girl children would tend to be less schooled, come from larger families and probably poorer. However, this is clearly not the case. Upon scrutiny of the countries in regression (19) classified as those with a "high" son prevalence, a combination of religious and cultural factors appear to affect this

preference of sons. Of the 11 countries with a high son preference (Angola, Namibia, Nigeria, Niger, Morocco, Tunisia, Pakistan, Iran, Nepal, Angola and China) a number of them are Muslim states while China and India have a unique socio historical background that advocated for one child per family. It is not clear why Namibia and Angola are in the list, though we speculate that the prolonged civil war in Angola could have cause a preference for sons perhaps for protection or from the fear that girl children could be vulnerable. In any case, although the measure is not entirely a proxy for religion, (because there are other, Muslim countries not in this list) we cannot be confident that this is a measure of disadvantage among women as there are plausible alternative explanations as to why a preference for sons may be related to HIV prevalence.

None of the grouped variables we successively add to the base model result in significant changes in the coefficients. In the case of cofactor infections and health, the model actually becomes mis-specified and regression diagnostics suggest that only the measures for general disadvantage among women improve the base model. There is no evidence from the regressions to support the proposition that gender inequality is a significant driver of the HIV epidemic.

Next, we restrict the sample size to sub-Saharan African countries and rerun the above regressions. The table below presents the results.

Table 5:3: Multivariate regression, sub-Saharan African countries

Loghiv2007	**Base (20)**	**Cofactor infections and health (21)**	**Gender inequality (22)**	**General disadvantage for women (23)**
Southern Africa **Coefficient** **(t)** **P**	1.56*** (5.94) 0.00	1.88** (4.64) 0.02	1.65*** (12.35) 0.00	1.47** (3.88) 0.003
Gini	0.07* (1.72) 0.10	0.13** (3.18) 0.013	0.12*** (4.27) 0.004	0.01 (1.15) 0.275
Log of share of income bottom 20% of population	2.34** (2.32) 0.03	3.64** (3.83) 0.05	2.81*** (4.81) 0.002	2.13 (1.57) 0.147
Proportion urbanised	0.00 (0.08) 0.94	-0.00 (-0.09) 0.92	0.01 (3.18) 0.016	0.00 (-0.12) 0.906
Adult literacy	0.03*** (2.97) 0.01	0.04** (3.07) 0.015	0.04*** (3.87) 0.006	0.03* (1.80) 0.10
Age of epidemic	-0.79** (-2.41) 0.03	-1.27** (-2.3) 0.050	-0.98*** (-3.94) 0.011	-0.71 (-1.52) 0.159
Age of Epidemic squared	0.02** (2.38) 0.03	0.03* (1.95) 0.086	0.02*** (3.45) 0.011	0.02 (1.50) 0.164
Proportion Muslim	0.00 (-0.18) 0.86	-0.01 (-0.87) 0.411	-0.01 (-1.10) 0.306	-0.01 (-0.01) 0.994
Log of remittances received per capita	0.07 (1.33) 0.20	0.04 (0.49) 0.639	0.12** (3.77) 0.07	0.05 (0.58) 0.578
Log STDs DALY		0.84 (1.17) 0.274		
Log Nutritional deficiencies		-0.38 (-1.09) 0.308		
Healthcare quality		-0.02* (-2.07) 0.072		
Proportion women seats in parliament compared to men			-0.01 (-1.1) 0.30	
Female labour force participation rate			-0.01 (0.88)	

			0.408	
Proportion women secondary school relative to men			-0.02*** (-3.40) 0.011	
Maternal leave length in weeks				-0.03 (-0.35) 0.733
Contraceptive prevalence				0.01 (0.30) 0.767
Low polygamy				-0.62 (-1.69) 0.122
High polygamy				-0.57 (-1.30) 0.223
High son preference				0.11 (-0.18) 0.863
N	25	20	19	24
F-test (p-value)	0.000	0.00	0.00	0.001
R2	0.95	0.97	0.98	0.92

*** 1%, ** 5%, *10% significance levels

Although the sample sizes in these models are smaller, ranging from N=19 to 25[42], in general, these models restricting the sample to sub-Saharan African countries are a better fit than those including developing and other transition countries. In the base model, the Gini coefficient, share of income available to the bottom 20 per cent and the adult literacy rate are all positively correlated with the log of HIV, while controlling for the age of epidemic, urbanisation, proportion Muslim and remittances from abroad. An increase in the Gini coefficient by one unit, controlling for the above covariates increases the log of HIV prevalence in a country by 0.07 units, i.e. by exp(0.07)= 1.07% (p-value 0.10).

In the base model, the log of income share held by the bottom 20 per cent is the largest determinant of HIV prevalence, even more powerful than the southern African coefficient.

[42] For a list of all the countries in each of the models in table 4.3, please see appendix (IX).

The higher the share of income going to the bottom 20 per cent, the much higher the prevalence rate, again controlling for the covariates in the base model. Increasing the log of share of income held by the poorest 20 per cent by one unit (2.7%) increases the size of the epidemic by approximately 10 per cent. Since this model controls for the Gini coefficient i.e. the effect of large wealth differentials across the population, the income share the bottom 20 per cent can be interpreted as capturing the effect of poverty on HIV prevalence. This result is not affected by the choice of countries selected and remains strong in the three models above. In the 4th model in the table, the log of share of wealth variable loses significance but ordinary least squares diagnostics show that the model is not specified correctly.

In chapter 3, we presented a qualitative snap-shot of some of the households that lie in the bottom 20 per cent using case examples from Zimbabwe, Lesotho and Swaziland. These household are for the most part, very rural and very poor. Given that southern Africa as a region has some of the wealthiest countries, we can reasonably suppose that the poorest 20 per cent of population in other parts of sub-Saharan Africa are also likely to be absolutely poor. In terms of our regression results then, we interpret the positive log share variable as follows: holding the underlying wealth distribution constant (Gini) the greater the share of income going to the bottom 20 percent, the worse the HIV epidemic. In short, the idea that poverty is driving the HIV epidemic is not supported by these results.

If the poorest live in largely rural communities and under dire poverty, small increases in living standards might enable them to travel more frequently to trading centres and towns, and because they are still poor, could find themselves exposed to higher HIV risk. If this is happening at a very large scale, then this could result in a larger national HIV prevalence. In a separate regression, we run the base model without the Gini coefficient. There is still a positive relationship between the income share going to the poorest 20 per cent and HIV

prevalence, although the coefficient size is much smaller. These results are presented in appendix XI.

The southern African coefficient remains large and compared to other sub-Saharan African countries, this model predicts that moving from a country not in southern Africa to a southern African country only increases the predicted HIV prevalence by about 5 per cent (exp 1.56 = 4.75%, p-value 0.00). This is while controlling for the Gini coefficient, income share of the bottom 20 per cent, the proportion of the population urbanised, age of epidemic, percentage Muslim and remittances from abroad.

Measures of cofactor infections and health perform better as regressors in the sub-Saharan Africa only model compared to the earlier model with all countries. While the burden of disease caused by nutritional deficiencies and STDs (excluding HIV) are statistically insignificant, there is a statistically significant negative relationship between healthcare quality and HIV prevalence. Controlling for the base model covariates, the higher the healthcare quality in a country, the smaller the HIV epidemic.

The proportion of female seats in parliament and the female labour force participation rate are not correlated with HIV prevalence, controlling for the base covariates. The difference between men and women's secondary school enrolment rates however predicts that the higher the number of women that make it to secondary school compared to men, the smaller the HIV epidemic.

Measures of general disadvantage for women, after controlling for base variables, suggest that these are not strongly related to HIV prevalence. A preference for sons, maternal leave length and an index for polygamy all are statistically insignificant. These variables actually add noise to the model, rendering all but the Southern African coefficient statistically

insignificant and yet the model has a higher R^2 value – an indication that the model is mis-specified. The Wald test results confirm that these variables do not improve the model and the test fails to reject the hypothesis that these new coefficients are jointly equal to zero.

5.6 Discussion

Our cross-country multivariate regressions produced some expected and other unexpected results. Using simple OLS regression, we confirmed that the HIV epidemic is worse in Sub-Saharan Africa than elsewhere – and that Southern Africa has exceptionally high HIV prevalence rates. In sub-Saharan Africa, the share of income going to the bottom 20 per cent, controlling for the Gini coefficient and the age of the epidemic, shows a positive relationship with HIV prevalence. Thus, while there are reasons to believe that poverty can worsen HIV risk for individuals, these national-level regressions suggest that redistributing income to the poor may have the counter-intuitive result of worsening the epidemic. This may be because increasing living standards at the bottom end of the income distribution facilitates the engagement in sexual networks, but this remains speculative.

Our analysis also does not support the proposition that gender inequality is a significant driver of the HIV epidemic. While it is likely that HIV prevalence is a function of some structural underlying factors peculiar to sub-Saharan Africa and Southern Africa, that those structural factors include gender cannot be substantiated.

We also fail to find any evidence that southern Africa has higher HIV rates due to high levels of poverty. Controlling for the Gini coefficient, which some authors hypothesize is a measure of the prevalence of poverty, as well as the share of income going to the poorest quintile, the Southern African coefficient remains statistically significant. This is probably unsurprising, however, given that there is a large gap between the average HIV prevalence in Southern Africa (about 15 per cent) and in the rest of Africa (about 4 per cent). It is thus noteworthy

that in multivariate analysis, the base model predicts that the difference from moving to a Southern African country increases HIV prevalence by only about 4.75% on average. Including the other covariates thus reduces substantially the difference in HIV prevalence attributable to regional factors.

Chapter 6. Intimate Partner Violence (IPV) and HIV in southern Africa

6.1 Background

There is a substantial body of literature showing that IPV is responsible for negative health outcomes among women and it is not surprising that it is now recognised as a significant public health problem around the world (Sareen, Pagura, & Grant, 2010: 2; Affifi et al., 2008; Garcia-Moreno et al., 2006; Mitchel, 2008; Norman, Bradshaw, Sneider et al., 2000). In sub-Saharan Africa, one of the main areas of concern is the association between IPV and sexually transmitted diseases such as HIV (Raj et al., 2006; Townsend, Jewkes, Mathews et al., 2010:1; Sareen et al., 2009).

So far, this discussion has not taken into account IPV and its associations with gender inequality and HIV in southern Africa. This will be the focal interest of this and the next chapter. Continuing with our discussion of the determinants of high HIV prevalence among women in the region, we attempt to bring to the fore, and analyse, the salient features of this hypothesised relationship between IPV and HIV. There is a lot of contradictory evidence on the associations between HIV and IPV in the sub-region and in what follows, we weigh and contrast the evidence.

While no universal definitions for IPV exist (Anderson, Foster, Scheepeers & Goldstein, 2007:1; Hamberger & Guse, 2005), it is broadly understood as the use of physical, sexual and/or psychological violence among current or former partners (Arias & Corso, 2005:1). Physical violence includes slaps, punches, kicks and assaults with a weapon while sexual violence includes rape, coercion, use of physical force, verbal threats and harassment to have sex (WHO, 2005: 2). Psychological violence includes threats to harm the woman or her relatives/friends, withholding resources, preventing her from working or confiscating her

earnings (loc. cit). Coincidentally, the most prevalent form of gender violence is the abuse of women by an intimate partner (Tang & Lai, 2008:2; Heise, Ellsberg, & Gottmoeller, 2002:2).

There are many studies within southern Africa (discussed below) arguing that gender inequity manifests itself through abuse, some of which is culturally and legally endorsed and through some of the mechanisms highlighted in chapter two, leaves women more vulnerable to HIV. Wood and Jewkes (2001), for example, though without specifying the countries, state that in a number of African countries, there are very few disincentives for men to abuse or even rape women and go on to argue that this inadequate protection by law makes them more vulnerable to violence.

While there are studies that directly link IPV to HIV among women, men who perpetrate IPV have been shown in some cases to have higher sexually risky behaviour (Dunkle et al., 2004a; Dunkle et al., 2004b; Jewkes et al., 2006; Pettifor et al., 2006). In light of this, it could be that IPV in itself is not a risk factor for HIV, but just a proxy of partner characteristics. If men who perpetrate IPV are more likely to be sexual risk takers as some studies have shown, then IPV in itself may not be a risk factor for HIV among women, as stopping that violence will not change the underlying sexual risk nature of those men (and the inherent HIV risk that comes with sleeping with those men). It merely becomes an indicator of partner characteristics as the men who would likely abuse their partners pose a high HIV risk not because of the violence itself but because of their sexual behaviour.

We argue that the relationship between IPV and HIV is complex as it involves a combination of social, individual and perhaps economic interactions that govern how people meet and have sex with each other and the underlying power differentials within those relationships. It equally involves men as it involves women, and yet, many studies take a simplistic view and focus on the effects that IPV may have or not have on HIV among and women.

There is evidence that gender differences exist with regard to the motivations for (and acceptance of) violent behaviour (Archer, 2000; Swan, Gambone, Caldwell et al., 2008). A nuanced understanding of the interaction between IPV and HIV ideally requires an understanding of the men who perpetrate it, the women who experience it and how experiences of IPV can then translate into increased HIV risk. A very limited body of literature speaks to these aspects of the link between IPV and HIV. Furthermore, in order to evaluate the impact of IPV on HIV among women, we argue that it does not make sense to probe only the statistical link between women's experience of IPV and HIV status. We need also to take into account partner characteristics, notably their sexual risk profile.

The pathways between IPV and HIV are likely to be complicated and emphasis should be placed not only on the direct associations between IPV and HIV, but also, on the reasons for IPV, attitudes of the perpetrators and the context within which it happens. IPV potentially has a two-way relationship with HIV. It may increase HIV among women because forced sex increases HIV transmission and those abused at a younger age may have psychological scars that predispose them to engage in higher risk behaviour. It could also be that IPV is primarily a strong indicator of partner type, and in that case, it merely confirms what is known about high-risk sexual partners – that they increase their partners' chances of contracting the disease. Depending on the reasons why IPV happens, it could be that women who are sexually risky are more likely to be abused by their partners maybe as a form of punishment for their "wrong" actions. If these women happen to be more likely to be HIV-positive, IPV will be strongly correlated to this heightened HIV risk, but only because it also acts as a proxy of their underlying sexual behaviour.

To understand fully the associations between IPV and HIV researchers ought to be able to consider those that perpetrate IPV, what their reasons are and what sexual characteristics they

exhibit. Once we understand these for both sexes, we may then synthesise the results and draw conclusions that are potentially more illuminating about the associations between IPV and HIV among women in southern Africa.

6.2 Literature review

Scholarship on gender abuse and its health effects among women reveals that in many parts of the world, a number of health problems are associated with experiences of abuse. In America, women who experience IPV are more likely to report negative health outcomes that include joint disease, asthma, STIs, vaginal problems and digestive problems (Fletcher, 2010; Breiding, Michele, Black et al., 2005; Coker, Smith & Fadden, 2005; Campbell, 2002).

Campbell (2002), for example, reviews the studies on IPV among women from different settings in the USA and concludes that IPV victims suffer from increased chronic pain, STIs, depression, post-traumatic stress disorder and a higher likelihood of mortality resulting from IPV. In a cross-sectional survey of 1152 American women, Coker and colleagues (2005) corroborate the above findings, concluding that women who experienced IPV were more likely to report poor physical and mental health on average (Coker, Smith & Fadden, 2005). These women had higher incidence of arthritis, chronic pain, stammering, STIs, stomach ulcers among other infections in multivariate models controlling for education and employment (Coker et al., 2005:1).

In another study in America, covering 16 states with over 70 000 respondents, Breiding and colleagues find that joint disease, current asthma, heavy drinking or binging and not checking up with the doctor over the past year were all linked to IPV, while controlling for age, sex, race/ethnicity, income and education (Breiding, Michele, Black et al., 2005:1). Also in America, studies have shown that women who experience IPV are more likely to exhibit

higher health risk behaviours such as smoking, alcohol and drug abuse and higher sexual risk taking (El-Bassel, Gilbert, Witte et al., 2003; Lemon, Verhoek-Oftendahl, 2002).

Although the majority of the studies on IPV and its health consequences on women are from America, there are some studies from sub-Saharan Africa indicating that IPV is detrimental to women's health in the region. In Rwanda, Dude (2009), using the 2005 Rwanda DHS survey, finds spousal IPV to be positively associated with HIV and other STIs among married women (Dude, 2009). Married women subjected to IPV by their spouses were more likely to test positive for HIV, and more likely to test positive for a secondary STI while controlling for household wealth, religion and other socioeconomic characteristics (Dude, 2009).

In South Africa, research shows that adolescents (15-24) from communities that have high levels of sexual violence are more likely to be HIV-positive than those from communities with lower sexual violence, while individual violence was negatively associated with condom use (Speizer, Pettifor, Cummings et al., 2009). In Mali and Kenya, IPV at a community level was associated with higher premarital sex (Gomez & Speizer, 2010). The authors did not link this to higher HIV rates, but hypothesised that premarital sex would likely lead to higher HIV risk.

In a cross-sectional survey covering eight Southern African countries where 18 per cent of the women (2 032 women from a total sample of 11 063 women) reported experiencing IPV (defined as being beaten, kicked or slapped by the current partner in the past year), reporting multiple partners among women was a consistent risk factor for experiencing IPV (Anderson et al., 2007). Women who had multiple partners were more likely to experience IPV than women who reported fewer sexual partners. The study also compared rates of IPV victimisation between men and women and found no differences in the reported rates of IPV

by gender in Botswana, Lesotho, Namibia and Zimbabwe. In Malawi, Mozambique, Swaziland and Zambia however, women were more likely to report IPV.

Note that while Anderson et al (2007) found that women with multiple partners were more likely to experience IPV, it was not possible to ascertain whether this relationship was causal (as it was a cross-sectional study). They merely probe the correlates of HIV and asses the link between various demographic characteristics and IPV. Respondents aged between 30 and 39 (unweighted sample) reported more violent encounters than younger respondents while those with incomplete primary education appeared more violent than those who had completed primary school, though this difference (for both men and women) became insignificant after stratifying the samples by country. There was no relationship between household size and IPV, nor were there any differences in the rates of IPV between urban and rural residence. This study also found no convincing relationship between household income and IPV and the authors conclude that multiple partners and subjective norms and attitudes among men are the main correlates of IPV in southern Africa, and not poverty, overcrowding and education.

In the last step of their analysis, Anderson and colleagues attempted to link IPV to HIV risk. Those individuals who experienced IPV were more likely to believe that they were at risk of getting HIV, but the study did not test for HIV. However, whether or not the added perceived HIV risk was due to IPV or due to higher sexual risk taking was beyond the scope of the study. The main limitation of Anderson et al (2007) was that it did not include HIV testing. However, after exploring the profile of respondents, they suggest that HIV risk has less to do with poverty, education and overcrowding, and more to do with individual sexual behaviour.

In South Africa, a cluster randomised trial in rural Limpopo that investigated the impact of microfinance among over 400 women showed progressive declines in IPV among women assisted with microfinance, but no changes in HIV risk and safer sex behaviour (Proynk,

Hargreaves, Kim et al, 2006). This study aimed at evaluating the effect of a structural intervention (IMAGE) that included microfinance and an HIV and gender awareness program for women. The authors postulated that "*underdevelopment, lack of economic opportunities for both sexes, and entrenched inequalities in the distribution of power, resources, and responsibilities between men and women*" fostered an environment that promoted HIV infection and IPV (Proynk, Hargreaves, Kim et al., 2006:1). The underlying associations between access to microfinance and IPV are not explicitly outlined in the study, though the authors state that structural interventions (such as microfinance) alter the conditions under which ill-health occurs and "*address upstream determinants of health and have the potential to affect multiple endpoints*" (Proynk et al, 2006:1).

Over a two-year follow up period, Proynk and colleagues reported declines in IPV among women. They estimate that over that period, IPV experienced by women was 55 per cent lower for women in the microfinance intervention compared to those in the control group. Owing to the lack of specification of the direct pathways through which the microfinance was seen to be linked to IPV, it is not exactly clear how this decline was achieved. For example, was it that there was a more harmonious atmosphere in some of the households owing to the presence of more resources so couples were less likely to be stressed or angry with each and therefore not fight or, alternatively, financially empowered women were able and more likely to leave abusive husbands and therefore suffer less incidences of abuse? The study does not explore this further.

While women who benefited from the actual IMAGE intervention were less likely to suffer from IPV and more likely to have "*progressive*" beliefs on gender norms, the follow up study observed no change in HIV risk behaviour (Proynk, Hargreaves, Kim et al., 2006). It also showed no lowered HIV incidence among the beneficiaries relative to non-beneficiaries.

What this study (Proynk et al (2006)) suggests is that it is not that IPV leads to HIV; it is that the relationship between IPV and HIV is likely to reflect the sexual risk profile of men who perpetrate IPV and the women who experience it. Micro finance as a means to empower women has been widely suggested as sustainable means to help women fight gender abuse and militate against HIV infection, but this study suggests that as long as they remain with high-risk partners, regardless of a lower likelihood of abuse, they remain at a high risk of contracting HIV.

In Soweto, South Africa, Dunkle and colleagues find that among 1 366 women attending antenatal clinics in Soweto, those that experienced IPV at some point in their lives were nearly 55 per cent more likely to be HIV-positive than those who did not (Dunkle et al., 2004). In this study, physical IPV was measured as ever being shoved, pushed, slapped, hit with a fist, kicked, beaten up, dragged, strangled, burnt, hurt/threatened with a weapon or have something thrown at one that could hurt(ibid: 2). The questionnaire probed three questions pertaining to sexual violence – being forced to have sex, having sex because one is afraid to refuse and being forced to do something degrading or humiliating. Although women with multiple partners were more likely to experience IPV, the increased HIV infection odds remained statistically significant in multivariate analysis after controlling for women's own risk factors such as multiple partners. The authors then conclude that although IPV is linked to increased likelihood of HIV risk through multiple partners, engaging in transactional sex and substance abuse, these heightened HIV risk factors did not fully account for the association between IPV and HIV. The study suggests that controlling for women's own sexual behaviour, IPV increases the odds of being HIV-positive.

In a randomised control trial (RCT) among rural men in the Eastern Cape, a study investigated rape perpetration by young men (15-26) towards partners and non-partners and

the link between perpetration and sexual behaviour (Dunkle et al., 2006). Rape of a non-intimate partner was measured by using the questions. "*Was there a time when you made a woman or girl, other than your girlfriend at the time: (1) Have sex with you when she did not want to? (2) When she was too drunk to say whether she wanted it*" (ibid: 3). The study probed gang rape using two gang rape questions: '*Have you ever done streamlining? And, 'Was there ever an occasion when you and other men had sex with a woman against her will or she was too drunk to stop you?*" (loc. cit). Twenty one per cent of 1 275 sexually experienced men reported raping a non-partner, participating in a form of gang rape or had been sexually violent towards an intimate partner. Men who were likely to perpetrate violence against their partners were more likely to engage in a host of other risky sexual behaviours. They were more likely to have higher numbers of sexual partners, have a greater chance of reporting casual sex partners, transactional sex and even more likely to report sexual violence against women other than their primary partner (Dunkle et al., 2006).

This observation, in part, would give possible insights as to why the IMAGE study intervention managed to reduce IPV but did not reduce HIV risk. It seems consistent that among men, IPV correlates with sexual risk taking and other violence measures. Women with partners who are violent seem to have compounded risk – direct risk from the partner in the form of probable sexual abuse and risk from the fact that the partner himself is more likely to be HIV-positive or contract HIV in the future. It is still unclear from the literature which type of risk is greater, and by what margin.

Studies have also linked a history of abuse among women to HIV through women's own sexual risk taking behaviours (e.g. Jewkes, Dunkle, Nduna et al., 2010:2; Maman et al.,2000). Abused women, especially those who experienced child sexual abuse, may themselves exhibit higher risk sexual practices later in life, such as multiple partners, inconsistent

condom use, and a young age of sexual debut, perhaps due to reasons of perceived low self-worth (Maman et al., 2000). This link has been widely flagged in the literature, but the most comprehensive research comes from the United States (e.g. Hrens, Katon McCarthy et al., 2012; Senn, Carey & Coury-Doniger, 2011; Senn, Carey & Vanable, 2007). These studies all find childhood and adolescent sexual abuse linked to riskier sexual practices such as transactional sexual trading and more sexual partners (Senn, Carey & Vanable, 2007; Ahrens et al., 2012) as well an early age of first intercourse (Senn et al., 2007).

Jewkes and colleagues (2010) depict the relationship between IPV and HIV in sub-Saharan Africa (largely based on studies from South Africa though), through figure 6.1. The figure suggests that child sexual abuse may directly lead to higher risk sexual patterns such as multiple partners and low condom use, or the resultant stress from child abuse will trigger these same behaviours.

In a study using 1370 male volunteers from 70 rural villages in the Eastern Cape, South Africa, a study found that young men who perpetrate rape were more likely to be physically violent to their partner, have transactional sex with a casual partner and have more sexual partners than non-offenders (Jewkes, Dunkle, Koss, Levinn et al., 2006). This RCT recruited men between the ages of 15-26 with the aim of evaluating an HIV behavioural campaign (Stepping Stones). The respondents filled in questionnaires, participated in face-face interviews and tested for HIV every two years. The authors found strong correlations between men who perpetrate rape and higher risk sexual behaviour, with men that perpetrated rape, in addition to more transactional sex and casual partners, also likely to be associated with gang membership, have peer pressure to have sex and be drug users (Jewkes et al., 2006). For many of the perpetrators, unstable family backgrounds such as child abuse was more common.

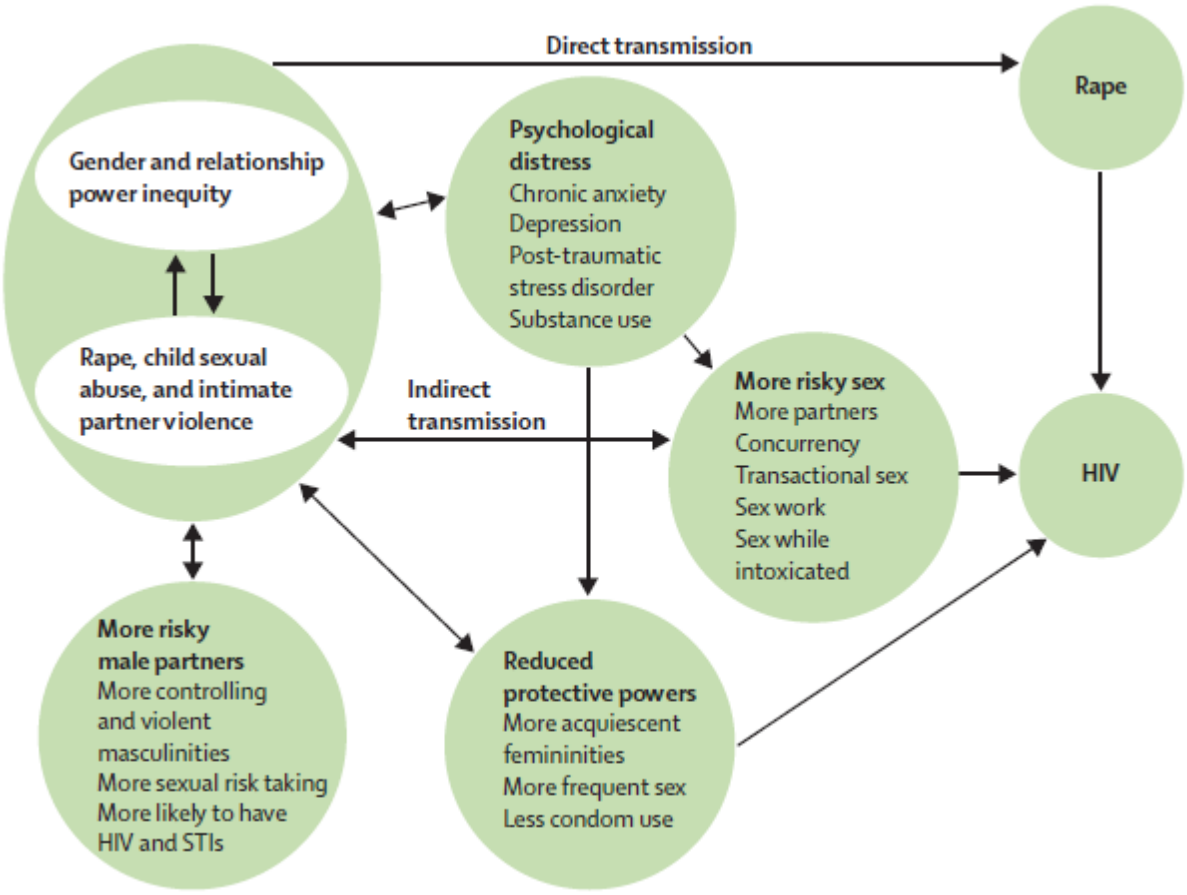

Figure 6.1 Relationship between IPV and HIV.

Source: Jewkes, Dunkle, Nduna et al., 2010:2

Another study, using the same sample but this time looking at physical abuse and not rape, reports that physically abusive men were also more likely to have casual sexual partners and more sexual partners than non-abusive young men (Dunkle, Jewkes, Nduna, Levin et al., 2006). Using 1 275 sexually experienced men of ages 15-26, this study finds that young men who reported perpetration of IPV were associated with higher numbers of lifetime sexual partners, transactional sex, substance abuse and more recent intercourse. More severe violence was also linked to higher levels of risky behaviour, i.e. among perpetrators, those that severely assaulted/abused their partners also exhibited higher levels of risk.

In Cape Town, a study among working-men showed that after adjusting for socio-demographic and socio-economic characteristics of education level and job level, sexually abusive men had more than one current partner and were more likely to be associated with "*particular types of conflict stemming from ideas of male sexual dominance and entitlement*" (Abrahams, Jewkes, Hoffman & Laubsher, 2004:6). This cross-sectional study was conducted on 1 368 randomly selected men from 3 Cape Town municipalities. The men were asked about perpetration of IPV over the past ten years and of the 15.3 per cent that reported perpetrating IPV, violence outside the home, problematic alcohol abuse and having more than1 current partner was more common (Abrahams et al., 2004).

Elsewhere, physically abusive men in India were found to more likely report unplanned pregnancies among their wives and, compared to married non-abusive men, also reported higher prevalence of STDs (Martin, Kilgallen, Tsui et al., 1999). There is a growing body of evidence from South Africa and elsewhere that men who perpetrate IPV are more likely to exhibit higher sexual risk practices than non-perpetrators.

This last connection between the characteristics of men who perpetrate IPV and their sexual behaviour is very important, but in our view, has not received adequate attention in research. If IPV is a risk factor of HIV (due to IPV exposing women to higher HIV odds in the pathways that directly increase transmission of HIV, or prevents women from seeking treatment or insisting on protection) then the implications for prevention are to stop men abusing their intimate partners. If however the men who perpetrate IPV are just riskier sexual individuals, then stopping them from perpetrating violence will not change risk levels among their female partners. These men are still likely to have many partners and unlikely to use condoms with these other partners. In this case, IPV stops being a risk factor for HIV and

merely becomes an indicator of the quality of partner that one has, in this case a sexually risky one. If the latter case is common, then interventions targeting IPV itself will have very small impact on reducing HIV risk among women.

Some literature suggests that the attitudes of women towards IPV are among some of the most powerful predictors of actually experiencing or perpetrating IPV (e.g. Hanson, Cadsky, Harris, & Lalonde, 1997; Faramarzi, Esmailzadeh, & Mosavi, 2005). Attitudes towards IPV have thus received widespread scholarly interest in Southern Africa, with researchers looking for links between attitudes towards IPV and experiencing IPV itself. If attitudes towards IPV form the basis of IPV, and this relationship is causal, then the implications for research are that intervention effort towards reducing IPV should include changing these among men and women.

One of the earliest studies on the risk factors to experiencing IPV among women was conducted in South Africa, using a cross-sectional survey of 1 306 women from 3 South African provinces (Northern province, Eastern Cape and Mpumalanga) (Jewkes, Levin & Kekana 2002). The main aim of the study was to identify the main causes of domestic violence in South African households. Using logistic multivariate analysis, the study tested for various correlates of domestic violence using a backward stepwise regression method. An extensive data trawl of potential correlates of domestic violence including measures such as the experience of childhood violence, perceptions towards violence, partner's age, employment status, possessions, marital status, as well as tests of whether these differed by province was conducted.

The authors found that women who had experienced childhood violence, were not well educated, drank alcohol, were often in conflict with the partner over drinking habits, had another partner during the year of the survey and had liberal views on women's roles were

more likely to have experienced domestic violence over the past year. A majority of the partner's own demographic characteristics such as age, employment, marital status and having other partners were not correlated with domestic violence. The authors subsequently conclude that domestic violence was most strongly related to the social status of women in society and to the discretionary use of violence to resolve domestic conflict.

Shortly afterwards, Maman, Mbwando Nora Hogan et al (2002) investigated the link between being HIV-positive and the likelihood of experiencing partner violence. The study tracked 340 women for 3 months after they had tested for HIV (whether positive or not). Using 'experiencing at least one episode of violence over the past three months' as the outcome variable, those women who tested HIV-positive for the first time after the three month period were 2.6 times more likely to have experienced violence. This violence was also most pronounced among those younger than 30, compared to older women.

Using cross-sectional household surveys from Botswana, Lesotho, Malawi, Mozambique, Namibia, Swaziland, Zambia and Zimbabwe, Anderson, Ho-Foster, Mitchell Sheepers and Goldstein (2007) identify multiple partners as the most consistent risk factor to IPV. About 20 639 adults were interviewed about their experience of IPV over the past year (without any classifications about the frequency and severity of the violence). Fourteen per cent of men and 18 per cent of women reported being beaten, kicked or slapped in the past year, but the authors did not find any association with demographic characteristics of age, education, household size and employment. Instead, having multiple partners was strongly associated with physical violence in the households. Among women, those who had two or more partners over the past year were almost twice as likely to have been a victim of IPV over the year, while men with more than two partners over the past year were twice as likely to have been beaten, kicked or slapped by their main partners over the past year.

In a more recent study, (Abramsky, Watts, Garcia-Moreno, Devries et al (2011) the link between IPV and possible risk factors was explored, using data from the WHO multicentre study (one randomly selected woman from each of 24 097 sampled households from Bangladesh, Brazil, Ethiopia, Japan, Namibia, Peru, Tanzania, Samoa, Serbia, and Montenegro and Thailand). The study finds secondary education, high SES (as measured by household wealth) and formal marriage to offer protection among women. On the contrary, alcohol abuse, cohabitation, young age, attitudes supportive of wife beating, other partners, childhood sexual abuse and experiencing or perpetrating other forms of violence were all associated with higher IPV risks.

Producing similar results to Abramsky et al (2011), Shamu et al (2011) finds a history of abuse, low SES, risky sexual behaviour, age and partners' drinking habits to predict the experience of IPV among pregnant women in sub-Saharan Africa. In a review of 13 studies investigating the prevalence of IPV among pregnant women in the region, women with more than five lifetime partners were more likely to have experienced IPV in the past year (OR 3.2 95% CI 1.21-2.34). Those whose partners had more than one current partner were also at a higher IPV risk (OR 1.53 95% CI 1.15-2.20). Younger women were at a higher risk of IPV than older women and women who reported alcohol use (either occasionally or frequently by either the woman or the partner) were more likely to report IPV while pregnant.

Assuming that attitudes towards IPV are the greatest determinant of IPV, Rani and colleagues (2004) attempt to examine the size and correlates of acceptance of wife beating among both men and women using DHSs conducted between 1999 and 2001 from Benin, Ethiopia, Malawi, Mali, Rwanda, Uganda and Zimbabwe (Rani, Bonu &Diop-Sidibe, 2004). The authors hypothesize that attitudes towards IPV are rooted in background characteristics such as patriarchy, gender roles, prevalence of wife beating in community as well as the social,

political and legal environment (ibid: 5). These background characteristics affect present attitudes towards IPV through three "*enablers*" – "*conflict between reality and myth of male superiority, exposure to more egalitarian authority structures and exposure to non-conformist ideas*" (ibid: 6).

They hypothesise that the belief that men are superior, is widespread in African cultures, but as gender roles change and women partake in more, traditionally 'male' roles such as providing for the family, some women begin to question the notions that men are superior. Among men, this creates a conflict between reality and myth of the roles of dominance where for some men, they believe themselves to be superior to women, but in practice, their partners or wives dominate or outperform them on many of these perceived dimensions or superiority. This, the authors hypothesize, may have two effects on men. For some, and most likely those that cannot find work or provide for their families, it creates a need to express dominance in other ways, such as violence. For others, it results in a revaluation of roles and acceptance of more liberal behaviour.

According to Rani et al (2004), exposure to more liberal and egalitarian structures, through travel, education and media, can also lead men and women to revise their attitudes towards IPV within the boundaries prescribed by background sociocultural factors. Women and men in societies with a high prevalence of wife beating may thus advocate or lobby towards the outlawing of those practices. Closely related to exposure to more egalitarian structures is the exposure to non-conformist ideas. This exposure, mainly through education, enables men and women to break away from the socially acceptable ways of behaviour, and in societies where wife beating is accepted, such people fight it.

Rani and colleagues hypothesize that culturally, men have a privileged social position that benefits them, and thus are less likely to give this up or want to change their view as they

stand to lose more. Building on this premise, they argue that the effect of these three enablers then, should be stronger among women than among men (ibid: 6). If schooling, for example, exposes men and women to more egalitarian norms, the effect it has on attitudes towards IPV should be greater among women than among men because women would benefit more from the change in attitudes (or cultural practices) that would follow.

However, these authors find a much higher rate of acceptance of IPV among women than men, regardless of education, employment and wealth status – quite a stark contrast to their hypothesis. They expected more educated women to justify wife beating less than less educated women, but this was not the case. This leads the authors to conclude that their finding, "*goes against basic instincts of survival*" and, "*is indeed perplexing, and it may be explained only by entrenched social and cultural learning processes that subjugate the position of women in the society, socially and collectively undermine their self-esteem and facilitate romanticisation of the "ideal" gender role of women, which also includes docile acceptance of husband's tyranny, etc*" (Rani et al., 2004:22).

Speizer (2009), using DHS data from Uganda (2006), finds that men and women who witnessed IPV in their childhood were more likely to be perpetrators of IPV later on in life. They also find positive attitudes towards IPV among those who witnessed it in their childhood to be strong correlates of determinants of IPV perpetration among men. The study does not clearly define the ways in which attitudes towards IPV and perpetration of IPV are actually linked. There is also no clarification of whether attitudes towards IPV still predicted the incidence of IPV independent of childhood abuse or if childhood abuse predicted both the attitudes and the actual perpetration of violence.

Using DHS surveys from Liberia, Kenya, Malawi, Rwanda, Zambia and Zimbabwe, Alio and colleagues (2011) find concordant attitudes towards IPV among couples a risk factor to

experiencing IPV (Alio, Clayton, Garba, et al., 2011). Those couples who concordantly held positive attitudes towards IPV were more likely to report IPV occurring within the union in the past year. The authors admit that the pathways through which concordant attitudes towards IPV affect IPV itself are unclear, but theystate that despite this, it is important for researchers to know of these connections. In other results, the same authors find household wealth, low level of education and rural residence positively associated with the experience of IPV among women.

Using pooled DHS data from 17 countries in sub-Saharan Africa, Uthman and Lawoko (2009) also look at the attitudes towards IPV. Among other findings, this study observed that women were 34 per cent more likely to justify intimate partner violence against women than men. The study also observed that the kind of community one resided in affected justifying attitudes about IPV. Men and women in disadvantaged neighbourhoods were more likely to justify it than men and women in more affluent neighbourhoods, regardless of individual socioeconomic position (Uthman & Lawoko, 2009).

Although Uthman and Lawoko do not look for the association between IPV and HIV, their study shows that the motivations and acceptance of IPV are probably complex. The study fails to categorise the women who think IPV is acceptable and those who think it is not by their actual experience of IPV. Such information could be vital, as, although more women think IPV is acceptable, many of these women may not necessarily be victims of IPV.

The studies that we reviewed on the risk factors for experiencing IPV are all indicative of IPV being associated with the normative perception of women's social standing in society, with those women perceived to be among the lower ranks in society more likely to experience IPV. Generally, young, uneducated women who also believe IPV to be justified are at the most risk of IPV whereas older women with higher SES and tend to be less at risk.

There are, however, notable differences in these associations between studies. Although many of the studies find a link between multiple partners and IPV, no single study specifically tries to tie the IPV to having multiple partners.

6.2.1 The prevalence of IPV

One of the main problems in the study of how IPV links with HIV is that the extent of IPV among women is unclear. One of the early investigations into the prevalence of IPV was conducted by WHO before 1999, and this study reviews over 50 population-based surveys around the world (Heise, Ellsberg & Gottmoeller, 2002). The study reports that between 10 and 52 per cent of women in different countries had been abused at some point in their lives and 10-30 per cent encountered IPV within the previous year (Garcia-Moreno et al, 2002:1). This pioneering study gave useful markers to the extent of IPV, but the measures of IPV used were very broad and the population-based surveys reviewed differed from each other and were hard to compare.

In a subsequent refinement of the same study, conducted across fifteen sites in ten countries, (Bangladesh, Brazil, Ethiopia, Japan, Namibia, Peru, Samoa, Serbia and Montenegro, Thailand, and Tanzania), and among ever partnered women, the reported lifetime prevalence of violence by an intimate partner ranged from 15-71 per cent (Garcia-Moreno et al, 2006). The lowest incidence of IPV (both physical and sexual) was in Japan (15.4 per cent) while the highest rates of IPV were in Ethiopia, Bangladesh and Peru, with incidence rates of 71, 62 and 69 per cent respectively. While the main objective of the study was not to evaluate causes of IPV but mainly measure its prevalence, in subsidiary pooled multivariate logistic regressions the authors find that age, partnership status and educational attainment differences across the sites did not explain the differences in IPV prevalence among the sites.

In southern Africa, a 2002 survey covering Botswana, Lesotho, Malawi, Mozambique, Namibia, Swaziland, Zambia and Zimbabwe, recorded 18 per cent of women between the ages of 16 and 60 reporting IPV, (measured by asking the respondents if they had been beaten, kicked or slapped by their partner in the past year (Anderson et al., 2007)).

According to the 1998 South Africa DHS, 13 per cent of the women surveyed reported suffering from IPV during their lifetime. In a cross-sectional study conducted in the Eastern Cape, Mpumalanga and the Northern Province, 24.6 per cent of the women reported ever-experiencing domestic violence in their lifetime, and 9.5 per cent reported experiencing that violence in the past year (Jewkes, Levin & Penn-Kekana, 2002). In a differently angled study in the Eastern Cape, Dunkle and colleagues investigate the prevalence of reported IPV among perpetrators, and 31.8 per cent of the men, in a 1 275 sample across 70 rural villages, report perpetrating physical or sexual violence against their female main partners (Dunkle, Jewkes, Nduna et al., 2006). Compared to other regions in Africa, the data on prevalence of IPV in South Africa, reported either as experiencing at least an episode of physical or sexual violence during the lifetime or over the past year, is closely comparable and is between 24 and 32 per cent of women.

In Tanzania, Prabhu and colleagues find prevalence of IPV to be 17.7 per cent among 2 436 women at a VCT testing centre in Moshi Tanzania (Prabhu et al, 2010). In this study, only physical violence and sexual violence was measured, with respondents answering questions about a lifetime experience of physical violence and sexual violence from their husband or current partner. In 2010, the first systematic review of quantitative studies that measure IPV or risk factors for IPV among pregnant women found IPV among pregnant women to range from 2 per cent to 57 per cent in 13 studies conducted in Nigeria, South Africa, Zimbabwe, Uganda and Rwanda (Shamu, Abrahams, Temmerman, et al., 2011). However, as the table

below shows, there were significant differences in the rates of IPV across sites even within the same countries.

Table 6:1 : Differences in IPV prevalence within and across sites among pregnant women in sub-Saharan Africa

Study	**Country**	**Sample**	**Overall IPV during entire pregnancy**
Ameh et al., (2009)	South Africa	340	31%
Hoque et al., (2009)	Nigeria	652	48.6%
Ezechi et al., (2004)	Nigeria	418	28.7%
Ameh & Abdul (2004)	Nigeria	178	28%
Chandisarewa et al., 2007	Zimbabwe	221	8%
Gyuse & Ushie 2009	Nigeria	340	11.6%
Olabuji et al., 2009	Nigeria	502	28.3%
Mbokota & Moodley 2003	South Africa	570	35%
Kaye et al., 2006	Uganda	612	27.7%
Fawole et al., 2008	Nigeria	534	2.3%
Kaye et al., 2002	Uganda	379	57.1%
Umeora et al., 2008	Nigeria	500	13.6%

Adapted from: Shamu, Abrahams, Temmerman, Musekiwa & Zarowsky, (2010: 4)

Although this study focuses on pregnant women only and is heavily weighted towards Nigerian studies, it highlights the disparities in IPV prevalence across studies even within the same countries. In Nigeria for example, the range of IPV is between 2 and 49 per cent while in Uganda it is between 27 and 57 per cent. This variation is in part due to the variation in IPV across places and due to "*differences in research methods, definitions of violence, sampling techniques, interviewer training and skills, and cultural differences that affect respondents' willingness to reveal intimate experiences*" (Watts & Zimmerman, 2002:3). Although it is clear that that a substantial proportion of women in Africa (and Southern Africa) experience IPV, the extent and scope of the IPV is difficult to ascertain. The range in many of the studies, except in South Africa, is still wide, and in the three global studies cited earlier, this range is anything between 2 and 71 per cent.

Numerous other studies in East and southern Africa investigate the association between IPV and HIV, and in these, the prevalence of IPV among women greatly varies (e.g. Grieg & Koopman, 2003 (Botswana); Mills et al., 2002 (Ghana); Mtika, 2001 (Malawi); Maman et al., 2002 (Tanzania); Wallman, 2000 and Ukwkuani et al., 2002 (Uganda).

6.3 Concluding remarks on IPV and HIV

From the above literature, a few pointers to research on HIV are clear. The first is that no new information is likely to come from studies that just investigate the relationship between the incidence of IPV and HIV risk. Depending on research design, available data and setting, this association could be positive, negative or non-existent. Studies that are able to show the risk factors pertaining to IPV are likely to help us more in determining the relationships between IPV and HIV as they outline the context in which IPV occurs. Not many such studies, especially in southern Africa, exist.

Secondly, if studies could compare the profile of men who perpetrate IPV to that of women who experience IPV and then link these to the risk factors of IPV, it would be possible to outline the relationships between HIV and IPV better. However, the data available has severe limitations in this regard. The most widely available nationally representative data do not include the timeline of when the abuse happened. Thirdly, there is a growing realisation that attitudes towards IPV form a crucial part of any link between IPV and HIV, but no research seems to have been able to pinpoint what exactly it is about attitudes that makes them important to HIV prevention. In the following chapter, we attempt to address some of these limitations in the literature.

Chapter 7. An empirical analysis of the association between attitudes towards IPV, risk behaviours associated with IPV and IPV as a determinant of HIV among women

7.1 Introduction

This chapter uses DHS data to explore the measurement and analysis of the different types of IPV and their associations with HIV risk in Lesotho, Swaziland and Zimbabwe. Firstly, we analyse the men who perpetrate IPV and test whether their sexual behaviour differs from those men who do not. In this part of the chapter, we attempt to understand men's attitudes towards IPV and how this affects their sexual behaviour and statistically test whether in Lesotho, Swaziland and Zimbabwe there is a difference in sexual behaviour between those men who perpetrate IPV and those who do not.

Next, we assess the women experience IPV. Here, we look at the determinants of IPV itself, and attempt to see if these are correlated with factors that influence HIV-status. In this part of the chapter, we run logistic regressions where IPV is the outcome variable and we investigate whether there is a connection between predictors of IPV and those of HIV.

In the third and last empirical part of the chapter, we investigate the relationship between IPV and HIV risk among women. We run logistic regressions with HIV-status as the outcome variable and IPV the independent variable, while controlling for other potential determinants of HIV that we have highlighted in preceding chapters such as risky sexual behaviour and low SES.

7.2 Statistical analysis

7.2.1 Sample

We use Zimbabwe DHS 2005/06, Lesotho DHS 2004 and Swaziland DHS 2006 data. The characteristics of the datasets are described in chapter four and more information on the type of questionnaire, sample design, and other technical information is available in the individual country reports see MOHSW [Zimbabwe], 2006; MOHSW [Lesotho], 2006 ; CSO [Swaziland] 2006-07.

7.2.2 Measures of IPV

The physical violence questions were prefaced as follows: "I am going to ask you about some situations which happen to some women. Please tell me if these apply to your relationship with your (last) partner? (*Does/did) your (last) husband/partner ever: a) Slap you?* b) *Punch, kick, drag or beat you up?* c) *Attack you with a knife, gun or any other weapon*?" Respondents had three possible responses: 1) *Often*, 2) *Sometimes* and 3) *Not at all*. We create a dichotomous variable which measures if any of the above 3 events happened, versus none of the 3events occurring. Table 6.1 below gives more detail on how the variable is constructed.

Sexual violence was measured using one question: "*(Does/did) your (last) husband/partner ever physically force you to have sexual intercourse with him*." We generate a dummy variable distinguishing those who have been physically forced to have sex from those who have not.

The last variable we use to measure IPV is a composite IPV measure that combines physical and sexual violence. This is a dummy variable, distinguishing between those women who

have experienced any physical or sexual violence from those who have not experienced any of the three measures of IPV.

Table 7:1 Specification of gender abuse variables

Variable	DHS question	Responses	Coding
Sexual violence	(Does/did) your (last) husband/partner ever physically force you to have sexual intercourse with him?	1. No 2. Sometimes 3. Often	Response 1 coded "No sexual violence" 2 and 3 coded "Sexual violence"
Physical violence	(Does/did) your (last) husband/partner ever: 1. Attack you with a knife, gun or any other weapon? 2. Slap You? 3. Punched you with a fist or something harmful	1. No 2. Sometimes 3. Often	A negative response for ALL 3 questions coded "No physical violence", else coded "Physical violence"
IPV	Combination of both measures above. Affirmative responses for sexual and physical violence coded "IPV", else coded "No IPV".		

A substantial body of research attests that violence that can predispose women or men to negative health outcomes may not only be limited to physical and sexual violence, but also includes emotional or psychological violence (Anderson, Cockroft & Shea, 2008; Alio et al., 2010; Jina, Jewkes, Hoffman, Dunkle et al, 2011). Psychological violence however is difficult to measure and understanding the association between threats, for example, and HIV outcomes can be problematic. The DHS surveys that have modules on domestic violence ask a number of questions pertaining to psychological violence – some of which are very broad, such as asking if the partner has shouted or embarrassed the respondent over the past year.

These questions are however difficult to relate to HIV risk as the scales of being "*embarrassed*" or "*made to feel bad*" are very subjective. There are other, more direct

psychological measures, in which the respondents are asked the question: *"(Does/did) your (last) husband/partner ever threaten to harm you, or someone close to you?*" While we do not doubt that threats to one's family or violence could affect sexual behaviour, threats may only work if they are serious, and the severity of the threat probably also affects how influential it is. There are no questions that probe the severity of the threats and we feel that including psychological violence from this dataset does not add value to this analysis.

On the attitudes towards abuse, respondents were asked whether they think it would be justified for a man to beat their wife in a number of different situation. The IPV attitudes question was asked as follows: "*Do you think that is justified for a husband to beat up his wife in the following circumstance: a)*
If she goes out without telling him. B) If she neglects the children. C) If she argues with him. D) If she refuses to have sex and E) If she burns the food. To these questions, the respondents could answer "*yes*", "*no*" or "*I don't know*".

A-priori, we suspected each of these five variables (A-E) to be strongly correlated with each other, because they measure the same underlying variable – perceptions towards wife beating. To check the validity of this suspicion, we performed factor analysis for all the five variables, to check whether they co-factored. Factor analysis was performed at a country level, i.e. we performed factor analysis for the same five variables by country. Detailed results from the factor analysis as well as methodology used and the interpretation are in appendix II. In short, for all three countries, the same underlying factors explained the variability in the five variables. That means that intrinsically, there was no difference in the statistical comparisons between any of the wife beating variables for each country. Since there was no difference between the underlying determinants of these variables, we arbitrarily picked one variable for

the analysis. The choice variable we use is variable d, "*if she refuses to have sex*". This is a dichotomous variable, zero if the respondent does not think wife beating is justified in that case, and one if they think it is justified.

We measure education by using the highest education level attained in years. This is a continuous variable, measured from zero to the highest number of years successfully completed in school. We use the DHS provided wealth index as the wealth variable. The index is divided into five population quintiles; with the poorest 20 per cent of the households in the first quintile and the richest households in quintile 5 (in chapter 3, we provide more detail as to how these population quintiles are constructed and offer a discussion of who is in these quintiles).

While IPV may have an impact on HIV, some sexual behaviours or practices confound this relationship. These behaviours and practices increase vulnerability to HIV infection, either through increasing the likelihood of contact with infected people (when the risk behaviour entails multiple partners), or through increasing the transmission probability once the individual is exposed to infection e.g. by not using a condom. Without controlling for the sexual behaviour of the respondents, there is no way to ascertain whether the HIV risk levels we observe are largely due to the effect of IPV, or are due to other individual risk factors.

7.2.3 Methodology

For each country (separately), we perform statistical analyses in three stages. First, we compare the attitudes towards IPV between men and women and determine whether they vary by age and socioeconomic background. Here, we investigate if perceptions about

whether IPV is justifiable or not vary by age, household wealth, employment status and urban or rural residence. We juxtapose observed sexual behaviour with the corresponding attitudes of IPV among men in the three countries. As shown earlier, the literature suggests that men who perpetrate IPV are more likely not to use condoms and are generally high risk. We compare attitudes towards IPV to condom use at last sex, ever testing for HIV and with the number of lifetime partners for both men and women. For those men and women who actually think IPV is justified, we then proceed to see if the attitudes about IPV are statistically linked to the actual perpetration of IPV.

We then investigate the risk factors for experiencing IPV among women. We use the literature to identify possible determinants of IPV among women. We run logistic regressions where the experience of IPV is the outcome variable, coded one if the woman has experienced sexual, physical or both forms of violence in the past year and zero otherwise. These regressions apply to the Zimbabwe 2005/6 DHS and Swaziland 2006 DHS data on female respondents only, with IPV the dependent variable and selected risk factors, together with control variables for wealth, education, age at first intercourse and number of sex partners as independent variables. We cannot extend this specific analysis to DHS Lesotho 2004 and Swaziland data as these surveys did not include a domestic violence module. In the main results presented in this chapter, we do not distinguish between the various kinds of IPV. There are two justifications for this.

The first is that there is sufficient evidence that there is an overlap of perpetration of the different types of IPV with those likely to perpetrate rape, for example, also more likely to physically abuse partners or non-partners. Secondly, using aggregated cross-sectional data in which the timing of abuse and the HIV-status cannot be differentiated, a detailed categorical

analysis by type and frequency of violence is more likely to generate a lot of noise in multivariate analysis, brought up by the increased chances of bias in finer specifications of the IPV variables. (Such finer categorisation of frequency, severity and timing of violence would be more useful if we had longitudinal data, in which HIV incidence could be tracked). However, in the appendix, we show the results that distinguish between the different kinds of IPV.

In the last stage of the analysis, we probe whether IPV is a risk factor for HIV. Multivariate logistic regressions for women respondents are run with HIV-status the dependant variable now, while controlling for a wide range of women's own risk factors to HIV.

All analysis is done in STATA 11.1, applying the relevant statistical weights to the survey data to enable population level inferences and standard errors adjusted for sample clustering. We test for goodness of fit is using a variant of the Hosmer-Lemeshow goodness-of-fit developed for use with logistic regression models when working with sample survey data[43] (Archer, Hosmer, & Lemeshow, 2007). We carry out additional post estimation checks for multicollinearity and goodness of fit, and report all results in odds ratios together with the linearized standard errors.

[43] The "*svylogitgof*" command is used. This command tests the independence of the independent variables from the dependant variable.

7.3 Results

7.3.1 Attitudes about the acceptability of IPV by demographic and socioeconomic characteristics in Lesotho, Swaziland and Zimbabwe for men and women

Table 7:2. Acceptance of IPV by age and sex (15-49) in Lesotho

	Sample Size		Proportion who believe wife beating is justified if the woman refuses to have sex	
Age-group	**Men**	**women**	**Men**	**Women**
15-19	682	805	22%	23%
20-24	482	694	20%	22%
25-29	357	497	19%	20%
30-34	295	407	17%	25%
35-39	216	353	24%	21%
40-44	159	353	26%	22%
45-49	169	298	25%	25%

Pearson chi(2) (**6**) (age-group vs perceptions of wife beating) = 8.9735. P-value = 0.175
Pearson chi(2) (**1**) (Sex vs perceptions of wife beating) = 1.3974. P-value = 0.237

In Lesotho, perceptions of wife beating if the wife refuses sex are independent of age group and independent of sex. There is no statistical evidence that perceptions of wife beating vary by age, thus we can conclude that these perceptions have not been changing over time.

Table 7:3. Acceptance of IPV by age and sex (15-49) in Swaziland

	Sample Size		Proportion who believe wife beating is justified if the woman refuses to have sex	
Age-group	**Men**	**women**	**Men**	**Women**
15-19	1 128	1 143	6%	4%
20-24	731	929	3%	3%
25-29	532	648	3%	3%
30-34	363	561	2%	4%
35-39	324	455	2%	2%
40-44	227	386	3%	4%
45-49	215	342	6%	7%

Pearson chi(2) (6) (age-group vs perceptions of wife beating) = 41.59. P-value = 0.000
Pearson chi(2) (1) (Sex vs perceptions of wife beating) = 0.1850 . P-value = 0.667

Positive perceptions towards wife beating if the wife refuses to have sex are much lower in Swaziland than in Lesotho, with less than five per cent of the respondents in most age-groups in Swaziland believing that wife beating is justifiable. While the positive perceptions do not vary by gender, they vary by age, with the oldest members in the sample exhibiting the largest proportions of those who believe wife beating is justified. This may indicate that there has been a change in perceptions over wife beating in Swaziland over time, with younger generations less likely to condone IPV.

Table 7:4. Acceptance of IPV by age and sex (15-49) in Zimbabwe

	Sample Size		**Proportion who believe wife beating is justified if the woman refuses to have sex**	
Age-group	**Men**	**women**	**Men**	**Women**
15-19	1 555	1 697	13%	21%
20-24	1 069	1 584	8%	26%
25-29	735	1 215	6%	25%
30-34	626	1 009	6%	26%
35-39	463	712	7%	26%
40-44	335	591	6%	30%
45-49	320	527	6%	33%

Pearson chi(2) (**6**) (age-group vs perceptions of wife beating) = 20.497. P-value = 0.002
Pearson chi(2) (**1**) (Sex vs perceptions of wife beating) = 5620.51. P-value = 0.000

Women are much more likely to report that wife beating is acceptable than men in Zimbabwe, with nearly three times more women likely to believe that wife beating if the wife refuses sex is acceptable. There are also contrasting trends by age between men and women, with older women more likely to believe that it is justified than younger women while among men, it is the youngest who believe that wife beating if a wife refuses sex is justifiable.

Table 7:5. Attitudes towards wife beating by household wealth, and sex (15-49) Lesotho,

	Sample Size		**Proportion who believe wife beating is justified if woman refuses to have sex with husband**		**P-values Pearson chi(2) by wealth quintile (men vs women per wealth level)**
Wealth	**Men**	**Women**	**Men**	**Women**	
1st quintile	453	552	35%	39%	0.115
2nd quintile	451	685	26%	30%	0.107
3rd quintile	464	599	21%	20%	0.602
4th quintile	495	697	16%	16%	0.907
5th quintile	497	874	9%	12%	0.131

Pearson Chi(2) (**4**) (wealth index vs attitudes) = 286. 2183. P-value =0.000

Table 7.5 shows the attitudes towards wife beating for men and women in Lesotho, by household wealth. Column six computes the p-value for the chi squared test for independence between men and women in each wealth quintile.

Within each of the wealth quintiles, there are no gender differences in the proportions that believe wife beating is acceptable in Lesotho. All the tests for independence by wealth quintile show that there are no gender differences per wealth quintile.

Across the wealth quintiles however, those that are wealthiest are less likely to believe that wife beating is justified. Among men, those from the poorest 20 per cent households are about four times more likely to believe that wife beating is justified than those in the wealthiest 20 per cent households (35% and 9% respectively). The same trend is observed for women, with the corresponding figures at 39 and 12 per cent for those in the poorest and wealthiest households, respectively.

While wife-beating acceptance among wealthy women is low, it is nearly double that of similarly endowed men, and this relationship is statistically significant.

Table 7:6. Attitudes towards wife beating by household wealth, men and women (15-49) Swaziland

	Sample Size		**Proportion who believe wife beating is justified if woman refuses to have sex with husband**		**P-values Pearson chi(2) by wealth quintile (men vs women per wealth level)**
Wealth	**Men**	**Women**	**Men**	**Women**	
1st quintile	519	711	6%	7%	0.287
2nd quintile	557	793	5%	4%	0.825
3rd quintile	690	864	5%	3%	0.038
4th quintile	765	961	3%	3%	0.506
5th quintile	989	1 135	2%	2%	0.730

Pearson Chi(2) (**4**) (wealth index vs attitudes) = 55.704. P-value =0.000

In comparison to Lesotho, the same trends between attitudes towards wife beating and household wealth exist, albeit at lower levels in Swaziland (due to the fact that Swaziland has lower proportions of people who hold positive attitudes towards wife beating). These perceptions generally do not vary by gender within each wealth quintile and vary by household wealth with the poorest more likely to believe that wife beating is justified if the wife refuses to have sex.

Table 7:7. Attitudes towards wife beating by household wealth, men and women (15-49)

Zimbabwe

	Sample Size		**Proportion who believe wife beating is justified if woman refuses to have sex with husband**		**P-values Pearson chi(2) by wealth quintile (men vs women per wealth level)**
Wealth	**Men**	**Women**	**Men**	**Women**	
1st quintile	962	1 404	13%	37%	0.000
2nd quintile	1 009	1 398	11%	34%	0.000
3rd quintile	991	1 401	11%	28%	0.000
4th quintile	1 225	1 538	6%	19%	0.000
5th quintile	916	1 594	3%	11%	0.000

Pearson Chi(2) (**4**) (wealth index vs attitudes) = 401. 403. P-value =0.000

In Zimbabwe however, positive perceptions towards wife beating vary by sex within each age-group and also vary by household wealth. Within each age band, women are more likely to believe that wife beating is justified than men, while across wealth quintiles those at the bottom are more likely to endorse wife beating.

The same variation in perceptions towards wife beating across wealth quintiles therefore exists in the three countries. Wife beating is mostly endorsed by the poorest. In Zimbabwe, unlike in the other two countries, these perceptions are different between men and women in each wealth quintile.

Next, we compare positive perceptions of wife beating across education levels, place of residence and employment status.

Table 7:8 Acceptance of wife beating by education, place of residence and employment status, men and women (15-49) Lesotho

	Sample size		Proportion who think wife beating justified if wife refuses to have sex		Pearson chi(2) for independence (p-values)
Education	**Men**	**Women**	**Men**	**Women**	
No education	419	88	27%	43%	No education vs some education 0.000
Primary only	1 266	2 036	26%	28%	
Secondary only	615	1 234	8%	13%	
Higher education	60	49	7%	4%	
Unemployed	1 247	1 905	19%	26%	Employed vs unemployed 0.029
Employed	1 113	1 502	24%	18%	
Urban residence	601	935	14%	12%	Rural v urban residence 0.000
Rural residence	1 759	2 472	24%	26%	

There are clear differentials in perceptions of wife beating by education, employment and place of residence in Lesotho, all of which are statistically significant. For both men and women, those who have no education are more likely to think that wife beating is justified than those who have some. By employment status, men and women that are unemployed are more likely to believe that wife beating where the woman refuses sex, is justified, than those who are unemployed. Respondents in the rural areas are more likely to endorse perceptions of wife beating than those in the urban areas.

As discussed in chapter 6, Rani et al (2004) talks of the varying effect of 'enablers' on men and women, with the enablers being those events that affect people's perceptions about the various social and cultural practices that affect them. They hypothesize that enablers such as education and employment should have a greater effect among women because men stand to lose more social privileges by changing norms. The results in the above table are consistent with this hypothesis. The proportion of those believing that wife beating is justified declines

as education level rises – and this is much more pronounced for women than for men. The same trend is observed with employment status: positive perceptions towards wife beating are lower for the employed compared to the unemployed – especially so for women. (This however can be confounded by the fact that educated women are more likely to be employed.)

Table 7:9 Acceptance of wife beating by education, place of residence and employment status, men and women (15-49) Swaziland

	Sample size		**Proportion who think wife beating justified if wife refuses to have sex**		**Pearson chi(2) for independence**
Education	**Men**	**Women**	**Men**	**Women**	
No education	282	372	7%	8%	No education vs some education 0.000
Primary only	1 246	1 495	6%	6%	
Secondary only	1 715	2 283	2%	2%	
Higher education	277	314	1%	0%	
Unemployed	1 545	2 487	5%	4%	Employed vs unemployed 0.051
Employed	1 975	1 977	3%	3%	
Urban residence	1 147	1 305	2%	2%	Rural v urban residence 0.000
Rural residence	2 373	3 159	5%	4%	

Table 7:10 Acceptance of wife beating by education, place of residence and employment status, men and women (15-49) Zimbabwe

	Sample size		**Proportion who think wife beating justified if wife refuses to have sex**		**Pearson chi(2) for independence**
Education	**Men**	**Women**	**Men**	**Women**	
No education	66	323	11%	49%	No education vs some education 0.000
Primary only	1 493	2 491	13%	35%	
Secondary only	3 312	4 331	7%	19%	
Higher education	232	190	1%	5%	
Unemployed	1 672	4 241	9%	25%	Employed vs unemployed 0.000
Employed	3 431	3 094	8%	26%	
Urban residence	1 565	2 407	4%	13%	Rural v urban residence 0.000
Rural residence	3 538	4 928	10%	31%	

Tables 7.9 and 7.10 above show the same trends as those from Lesotho. Positive perceptions towards wife beating vary by education, but more so for men than for women. They also vary by employment status, with those unemployed more likely to believe that wife beating is justified.

There is no literature that we know of that offers plausible explanations with regards the inter country differentials observed in tables 7.6 and 7.7. We do not know why there is a huge gap by gender, in the proportions that believe wife beating is justified in Zimbabwe, compared to Lesotho and Swaziland nor why the levels of acceptance is much lower in Swaziland. Other research has also failed to explain these differentials in perceptions towards wife beating by gender.

However, we can conclude that attitudes towards wife beating vary according to gender, country, employment, education and sector of residence. Attitudes in themselves however, are only important in as much as they affect the sexual behaviour and the actual experience of IPV. Next, we then compare the attitudes towards wife beating to actual sexual behaviour.

7.3.2 Attitudes towards wife beating and actual sexual behaviour among men and women in Lesotho, Swaziland and Zimbabwe

In this sub-section, we compare attitudes towards wife beating and sexual behaviour patterns that may be risky. We investigate the link between attitudes towards wife beating and sexual behaviour variables of using condoms during last sex, ever testing for HIV and multiple partners.

Among women in Lesotho, those that believe that wife beating is justified were less likely to have used condoms during their last sexual encounter. Of the women that reported using condoms during the last sexual encounter, 13 per cent thought wife beating was justified compared to 25 per cent who held the same views and did not use condoms in their last sexual encounter. Among women, perceptions towards wife beating had a smaller bearing on ever testing for AIDS. There is no statistically significant difference in ever testing for AIDS and perceptions towards wife beating among women.

Among men, there is also no statistical difference in testing for AIDS and perceptions towards wife beating. Men in Lesotho were not asked about condom use during their last sexual encounter.

Table 7:11. Attitudes on violence, condom use and testing for AIDS among women (15-49) in Lesotho

	Sample size		Proportion who think wife beating is justified if wife refuses sex	
Behaviour	**Men**	**Women**	**Men**	**Women**
Did not use condom last sex		2 018		25%
Used condom last sex		430		13%
Has never been tested for HIV	2 170	2 668	21%	21%
Has tested for HIV	278	496	16%	22%

Pearson chi2(1) (Condom use vs Attitudes wife beating) = 26.3. P-value = 0.000
Pearson chi2(1) (Testing for AIDS vs Attitudes wife beating) = 0.88. P-value = 0.349

In table 7.12 below, we present the same data for Swaziland.

Table 7:12. Attitudes on violence, condom use and testing for AIDS among men and women (15-49) in Swaziland

	Sample size		Proportion of those that believe wife beating is justified if wife refuses sex	
Behaviour	**Men**	**Women**	**Men**	**Women**
Did not use condom last sex	1 108	1 879	4%	4%
Used condom last sex	981	1 124	2%	3%
Never been tested for HIV	2 837	2 518	4%	4%
Has been tested for HIV	659	1 826	2%	4%

Pearson chi2(1) (Condom use vs Attitudes wife beating) = 9.4 P-value = 0.009
Pearson chi2(1) (Testing for AIDS vs Attitudes wife beating) = 2.5 P-value = 0.114

In Swaziland, there is no difference between HIV testing rates and perceptions towards wife beating for both men and women. However, as with the Lesotho sample, there are differences in condom use during last the last sexual encounter and positive perceptions towards wife

beating if the wife refuses to have sex. Those who used condoms during the last sexual encounters are less likely to justify wife beating.

Table 7:13. Attitudes on violence, condom use and testing for AIDS among men and women (15-49) in Zimbabwe

	Sample size		**Proportion who think wife beating is justified if wife refuses sex**	
Behaviour	**Men**	**Women**	**Men**	**Women**
Did not use condom last sex	2 553	4 518	7%	28%
Used condom last sex	784	397	8%	22%
Has never been tested for HIV	4 421	5 386	9%	26%
Has tested for HIV	895	1 901	6%	22%

Pearson chi2(1) (Condom use vs Attitudes wife beating) = 45.7 Pr = 0.000
Pearson chi2(1) (Testing for AIDS vs Attitudes wife beating) = 3.39 Pr = 0.066

In Zimbabwe, there is a very small difference between condom use and justifying wife beating. Among men for example, of those that used condoms during the last sexual encounter, eight per cent believe that wife beating is justified and of those that did not use condoms, seven per cent justify it.

We explore the distribution of multiple partners between men and women and if that differs according to their attitudes towards IPV. Owing to the influence of very large reported numbers of sexual partners that can change the mean significantly we restrict this analysis to those men and women who report 15 or fewer lifetime sexual partners.

Table 7:14. Multiple partners and perceptions about IPV men and women (15-49) Swaziland

Category	N	Mean number of lifetime partners	Std. Dev	Min-Max
All men	2 169	4.9	3.5	1-15
Men reporting beating justified	62	5.0	3.9	1-15
Men reporting beating not justified	2 075	4.9	3.5	1-15
All Women	3 569	2.3	1.6	1-15
Women reporting beating not justified	3 460	2.3	1.7	1-15
Women reporting beating justified	130	2.3	1.2	1-8

Overall, men report higher numbers of lifetime partners than women. The average number of lifetime partners among men in Swaziland is 4.9 partners, while among women it is 2.3 partners. There are no significant variations in the means of lifetime sexual partners and justifying wife beating among either men or women in Lesotho.

In Zimbabwe, men also report a higher average number of lifetime partners than women. Table 7.15 below compares multiple partners and perceptions towards IPV. We also include a comparison of lifetime sexual partners between those justify wife beating and those women that have actually experienced intimate partner violence. This is not included for Swaziland and Lesotho because this question was in the domestic violence module, and the surveys for these two countries do not have this module.

Table 7:15. Multiple partners and perceptions about IPV among men and women (15-49) Zimbabwe

Category	N	Mean number of lifetime partners	Std. Dev	Min-Max
All men	3 791	4.0	3.1	1-15
Men reporting beating not justified	3 406	4.0	3.1	1-15
Men reporting beating justified	258	3.8	3.0	1-15
All Women	5 926	1.6	1.1	1-15
Women reporting beating not justified	4 247	1.6	1.1	1-15
Women reporting beating justified	1 614	1.5	1.0	1-15
Women that have been physically and sexually abused	1 513	1.6	1.2	1-15

There is no evidence that justifying wife beating results in a higher number of partners among men and women in Zimbabwe. For men, those that believe wife beating is justified actually have a lower average number of sexual partners than those who do not justify it.

We do not find a relationship between average number of partners and experiencing abuse. The mean number of lifetime partners for women who have experienced abuse is nearly the same as that of those who have not experienced abuse.

7.3.3 Attitudes towards IPV and the actual experience of IPV

We have discussed the distribution of attitudes towards violence and a number of demographic, socioeconomic and behavioural variables among men and women in Lesotho, Swaziland and Zimbabwe. In this sub-section, we investigate whether these attitudes towards IPV are actually related to the experience of IPV. DHS Lesotho and DHS Swaziland, however do not have data on actual experiences of domestic violence. This analysis therefore

is restricted to Zimbabwean women between the ages of 15-49 who were included in the domestic violence questionnaire.

We assess association between partner violence and attitudes towards violence using three variables: Experiencing less severe violence, severe violence or sexual violence in the past year. Less severe violence is measured by an affirmative response to any of these scenarios: "*has your partner in the past year, pushed, shook, threw something at you, slapped or punched you*"? Severe violence is measured by affirmative responses to the questions: *has your partner strangled, burned, attacked you with a knife, attacked you with a gun in the past 12 months*. Sexual violence is measured by asking if the respondents if they had been forced to have sex or perform sexual acts by their partners.

Table 7:16. Experience of sexual violence and perceptions towards wife beating among women (15-49) in Zimbabwe

Wife beating justified if wife refuses to have sex	**Experienced any sexual violence**		**Total**
	No	**Yes**	
No	2 613 88%	362 12%	2 975 100%
Yes	933 85%	239 14%	1 172 100%
Total	3 546	601	4 147

Pearson chi2(1) (Attitudes wife beating vs sexual violence)=45.89. P-value = 0.000

Among Zimbabwean women, perceptions towards IPV are not linked to experiencing sexual violence. Twelve per cent of those that justify wife beating experienced sexual violence in the past year compared to 14 per cent who experienced sexual violence and did not justify IPV.

Table 7:17. Experience of less severe violence and perceptions towards wife beating among women (15-49) in Zimbabwe

Wife beating justified if wife refuses to have sex	Experienced any less severe physical violence		Total
	No	Yes	
No	2 124 71%	854 29%	2 978 100%
Yes	752 64%	420 36%	1 172 100%
Total	2 876	1 274	4 150

Pearson chi2(1) (Attitudes wife beating vs Less severe physical violence) =20.2. Pr = 0.000

Perceptions towards wife beating are however related to experiencing less severe violence. Nearly 36 per cent of those that justify wife beating experienced less severe violence, compared to 29 per cent who experienced it but did not justify it.

Table 7:18. Experience of less severe violence and perceptions towards wife beating among women (15-49) in Zimbabwe

Wife beating justified if wife refuses to have sex	Experienced severe physical violence		Total
	No	Yes	
No	2 698 91%	277 9%	2 975 100%
Yes	1 034 88%	138 12%	1 172 100%
Total	3 732	415	4 147

Pearson chi2(1) (Attitudes wife beating vs Severe physical violence) =5.7. Pr = 0.000

The relationship between justifying IPV and experiencing severe violence is small.Overall, among Zimbabwean women, justifying wife beating is associated with a higher likelihood of experiencing less severe violence.

7.4 Risk factors for experiencing of IPV among women (15-49) in Zimbabwe

We estimate logistic regressions with IPV as the dependant variable and variables identified in the literature as possible determinants of IPV as independent variables. To keep the regressions as simple as possible, we test for the impact of age, partner's education, other partners, household wealth, having children, rural and urban residence and perceptions of wife beating on experiencing IPV over the past year. Age, education and partner's education are continuous variables, in years, reflective of the individual's age or education level. Sex with other partners is measured using a binary variable, one if the respondent had sex with another man other than the husband over the past year, and zero otherwise. Perceptions towards wife beating is a binary variable, one if the respondent believes wife beating is justified if the wife refuses sex and zero otherwise. We include another binary variable distinguishing between women who have children and those that do not to explore whether women's role as a mother has any bearing on IPV. Household wealth is measured using the DHS-provided wealth indices while we also identify those that live in the rural areas compared to those that live in urban areas.

With these variables, we aim to control for the dimensions or social standing, fulfilment of parental duties, as well as own sexual behaviour. This analysis applies to the sample of Zimbabwean women only, as this is the only dataset which provides the necessary variables.

(DHS Lesotho (2005/06) and Swaziland (2004) do not have the domestic violence module for women questions).

To verify that our coefficients are robust, we run the same regression model on an unweighted sample, the sample weighted by HIV selection and testing and then we also reweight by the selection for inclusion in the domestic violence questionnaire. Among the female respondents selected in the DHS questionnaire, not all were selected for HIV testing, so weighting by HIV selection corrects for bias that may have been involved. Among those tested, not all were selected to be in the domestic violence model as well, so re-weighting the sample by the domestic questionnaire weights adjusts for any selection bias in the household selection. The main results are presented below, with additional results in the appendix, but the coefficients are not sensitive to the weights applied nor are they to the specific type of IPV.

Table 7.19 below shows the results from the multivariate logistic regression, weighted by the selection probability of being in the domestic violence module. There are three regression models, the first one with sexual violence as the dependant variable, then physical violence as the dependant variable and in the last model, the composite IPV variable (have you ever experience sexual or physical violence over the past year) is the outcome variable.

Table 7:19. Risk factors to experiencing sexual and physical IPV over the past year among Zimbabwean women (15-49)

Variable		Sexual violence	Physical violence	Sexual and Physical violence
Age	(**coef.**)	-0.011*	-0.02***	-0.02***
	(**std. err**)	(-0.006)	(-0.005)	(-0.005)
Education		-0.010	-0.05***	-0.04**
		-0.020	-0.014	-0.014
Husband's education		-0.001	-0.003	-0.00507*
		-0.004	-0.003	-0.003
Had sex with man other than husband over the past year		0.286	0.438***	0.401***
		-0.186	-0.144	-0.143
Wife beating justified		0.515***	0.194**	0.311***
		-0.095	-0.076	-0.074
Has children		0.189	0.700***	0.650***
		-0.182	-0.152	-0.143
Household wealth quintile 2		0.061	0.144	0.116
		-0.129	-0.100	-0.097
Household wealth quintile 3		0.149	-0.131	-0.094
		-0.135	-0.109	-0.104
Household wealth quintile 4		0.030	-0.046	-0.132
		-0.166	-0.131	-0.127
Household wealth quintile 5		-0.267	-0.47**	-0.58***
		-0.252	-0.187	-0.180
Rural residence		0.292	-0.022	-0.010
		-0.185	-0.134	-0.130
Constant		-1.92***	-0.384	-0.291
		-0.364	-0.262	-0.255
N		4127	4129	4126

*** p<0.01, ** p<0.05, * p<0.1

Reporting sexual encounters with a man other than husband in the past year increases the probability of experiencing physical violence but not sexual violence. Those that have had

sex with another man, controlling for age, husband's education and SES are 44 per cent more likely to experience physical violence (coeff 0.438, $p<0.01$). The same respondents do not show any increased vulnerability to sexual violence.

In multivariate analysis, women's perceptions towards IPV are strongly linked to whether or not they actually experience it. Zimbabwean women who report that IPV is justified if the woman refuses to have sex with the husband are nearly 50 per cent more likely to have been sexually assaulted over the past year (coeff 0.515, $p<0.01$) and about 20 per cent more likely to have been physically assaulted (coeff 0.194, $p<0.05$) over the same period. This is controlling for the effects of age, own education, husband's education, having children and SES.

Women from the wealthiest 20 per cent of the households are much less likely to report experiencing IPV, when controlling for age, education, urban or rural residence, number of children and sex with other men than women from the poorest 20 per cent of the population. The model predicts that those from the wealthiest households are nearly half as likely to be physically assaulted (coeff -0.47. $p<0.05$).

The strongest predictor of IPV in the model is having children. Controlling for household wealth, perceptions of wife beating, household residence and place of residence, having children is a huge IPV risk factor among women. Those that have children are 70 per cent more likely to experience IPV. Although the outcome variable in this set of regressions was wife beating when the wife refuses to have sex, the same variable strongly cofactors with wife beating if the wife neglects the children. This would most likely explain the link between having children and experiencing physical violence.

These regressions suggest that the strongest reasons for experiencing IPV are having sex with other men, having children and believing that wife beating is justified. If IPV leads to higher HIV odds, it is important to note that this relationship would be in part confounded by women's own risk behaviour.

7.5 IPV as a risk factor for HIV

We have explored some of the characteristics of men that are likely to perpetrate IPV and women likely to experience violence from their partners but we still need to test the association of IPV with HIV-status.

In this section, we investigate the relationship between experiencing IPV in the past year and HIV-status amongst women in Zimbabwe (the only country for which we have both HIV status and IPV data). We run logistic regressions with HIV-status as the outcome variable. We use the "full" model we developed to model HIV in chapter 4, where the other correlates of HIV status are age, age squared, education, education squared, household wealth, urban or rural residence, age first sex, age first marriage, and the number of lifetime partners. To this full model, we alternatively add measures of IPV (sexual violence only, physical violence only and the composite IPV variable)

Table 7.20 presents the summary statistics for the variables used in this regression. These descriptive statistics are restricted to the sub-sample of women between the ages of 15-49 who were included in the domestic violence module and were tested for HIV.

Table 7:20.Descriptive statistics for continuous data

Variable	Observations	Mean	Std. Dev	Min	Max
Age first Sex	4569	17.88	2.89	9	35
Age first marriage	4569	18.66	3.54	8	44
Number of lifetime partners	4569	1.60	1.21	1	15
Education	4568	7.57	2.92	0	19
Age	4569	30.53	8.30	15	49
IPV	4951	0.34	0.47	0	1
Sexual violence	4913	0.10	0.30	0	1
Physical violence	4965	0.30	0.46	0	1
HIV-Status	3851	0.26	0.44	0	1

The mean age of this sample is 30.5 years. Thirty four per cent of the women report experiencing either sexual or physical violence or both. Ten per cent report experiencing sexual violence only and 30 per cent physical violence only. The lifetime average number of partners in this sample is 1.6 and HIV prevalence is 26 per cent. Mean education is seven years and this implies that on average, the women completed primary school.

Table 7:21 Linear correlations for variables

	HIV-status	Age first Sex	Age first marriage	Number of lifetime partners	Education	Age
HIV-status	1					
Age first Sex	-0.0095	1				
Age first marriage	0.0706	0.6715	1			
Number of lifetime partners	0.1883	-0.1682	0.0423	1		
Education	-0.0007	0.3685	0.2776	-0.0478	1	
Age	0.095	0.0796	0.189	0.1193	-0.3019	1

There is a negative relationship between education and HIV-status. Education is also negatively related to the number of lifetime partners. There is a strong positive correlation between the age of first marriage and age at first sex.

Table 7:22 Multivariate Regression Results. IPV as a risk factor to HIV among Zimbabwean women (15-49)

Variable	**Base**	**SES**	**Full**
Composite IPV **(OR)** **(Std. Err.)**	1.19** (0.925)	1.19** (0.094)	1.16** (0.094)
Age	1.45*** (0.057)	1.47*** (0.059)	1.44*** (0.0588)
Age Squared	0.99*** (0.000)	0.99*** (0.000)	0.99*** (0.001)
Education		1.08** (0.044)	1.07*** (0.045)
Education squared		0.99** (0.003)	0.99*** (0.002)
Household wealth quintile 2		1.10 (0.131)	1.13 (0.135)
Household wealth quintile 3		1.27** (0.155)	1.34** (0.165)
Household wealth quintile 4		1.42** (0.204)	1.45*** (0.211)
Household wealth quintile 5		0.92 (0.182)	0.98 (0.199)
Rural residence		0.87 (0.123)	0.90 (0.129)
Employed		0.98 (0.075)	0.97 (0.076)
Age first sex			0.97* (0.018)
Age first marriage			1.04*** (0.151)
Number of partners			1.35*** (0.062)
N	3857	3856	3 856
Chi(2)P-value	0.000	0.000	0.000
Pseudo R2	0.0296	0.0356	0.0584

We run three regressions testing for the impact of IPV on HIV status. In the first regression, we run IPV on HIV while controlling for age only. Experiencing IPV over the past year is associated with higher odds of HIV, with those that have experienced IPV about 19 per cent more likely to experience partner violence (OR 1.19, $p<0.05$) while controlling for age. In appendix XII, we repeat these regressions for sexual violence and physical violence separately, and these additional regressions confirm that this effect is largely due to physical violence. Sexual violence alone shows no association with HIV status.

Next, we add SES variables of household wealth, place of residence, education and employment status. In this model, the SES measures improve the specification of the model and the model has more explanatory power. The coefficient for IPV is still the same, (OR 1.19, $p<0.05$) strongly showing that the impact of IPV on HIV is independent of SES. Regardless of age and other SES variables, those that have experienced IPV over the past year are nearly 20 per cent more likely to test positive for HIV.

In the last model, we add behavioural variables to the SES variables. Behavioural variables reduce the size of the IPV coefficient (OR 1.16, $p<0.05$). This reduced coefficient size captures the effect of own sexual behaviour on IPV, i.e. this model predicts that approximately three per cent of the higher HIV risk that IPV victims have is explained by their own sexual behaviour. This confirms our earlier finding that men who perpetrate IPV in Zimbabwe do not appear to behave any riskier than those that do not perpetrate IPV. Overall, IPV predicts higher HIV odds for victims, while controlling for own sexual behaviour and SES.

7.6 Conclusions

The main purpose of this chapter was to explain the link between IPV and HIV specifically within the Southern African context. We noted that this relationship between IPV and HIV is not well understood and the causal pathways between IPV and HIV are possibly confounded by many factors. On the one hand, substantial research implicates IPV as a strong determinant of HIV among women, yet it is also widely documented that men who perpetrate IPV are more likely to have higher risk sexual practices themselves. In order to understand the link between IPV and HIV better, we argued that researchers should consider a holistic approach in understanding IPV and HIV, starting from the motivations of IPV, who is most at risk of IPV and the eventual associations with HIV. To our knowledge, this study is the first that attempts to apply this approach using nationally representative data.

The first finding we make is that women tend to be more supportive of wife beating than men do and this higher level of acceptance by gender is independent of age, household wealth, employment status and place of residence. As to why this is the case, the data does not allow us to follow this up any further, and no other literature has clear hypotheses as to why this may be the case. More research on the reasons as to why women are more likely to be in support of wife beating than men is required. However, among women themselves, perceptions of wife beating notably vary by country and by demographic characteristics. Educated, employed women who live in towns are much less likely to endorse wife beating than those without these attributes (although they themselves have higher acceptance levels than similarly endowed men). This finding suggests that the perceptions towards wife beating are in part a function of one's social status, with those higher up the social ladder less likely to endorse IPV.

After noting the differences in perception of wife beating by gender as well as demographic characteristics, we then explored the actual sexual behaviour of those men and women who believed wife beating was acceptable in certain circumstances. We observe that men's attitudes towards IPV do not provide evidence of higher risk sexual behaviour, as shown by condom use during the last sexual encounter, ever testing for HIV and the lifetime number of sexual partners. Data limitations hamstring our efforts to test against a wider range of sexual behaviours, but there is no evidence from this sample that among men, perceptions of wife beating in themselves are associated with higher risk sexual behaviour. Among women, there is a small positive relationship between holding the belief that wife beating is acceptable if the woman refuses sex and having a higher average number of sexual partners than other women.

Comparing the actual IPV experiences to reported sexual behaviour, both men and women who reported experiencing IPV within the past year had higher risk sexual behaviour, although the effects were much stronger among women. Women who reported IPV were less likely to have used condoms and less likely to have tested for HIV.

We then conduct multivariate regressions to explore which women are at the most risk for experiencing IPV. In contrast to the results from perceptions on wife beating among women, we find no evidence in multivariate logistic regressions of the impact of background SES on IPV. Whether or not a woman in the sample experienced IPV in the past year was not strongly related to their age, place of residence and household wealth. Rather, multiple partnerships, having children and endorsing wife beating were the strongest at risk factors. We also note, however, the protective effect of being in the highest wealth group.

Finally, we test for the link between having experienced IPV in the past year and the probability of being HIV-positive, controlling for a number of other covariates of HIV infection. IPV does appear to be a powerful risk factor of HIV among women in Zimbabwe in multivariate analysis.

Chapter 8. Summary of key findings and concluding remarks

The main purpose of this dissertation was to critique and add to the understanding of the relationship between gender, poverty, IPV and HIV in Southern Africa. Using a raft of quantitative and qualitative evidence, we presented detailed reviews of the relevant literature and conducted multiple models of HIV-status and HIV prevalence at individual and cross-country levels.

We began by contesting the common perception of a monotonic relationship between 'gender' and the 'African HIV epidemic' – that gender inequality leads to higher HIV risk among women. We posited that there is no reason why this should necessary be the case and that this relationship is likely to vary from country to country as it depends on the specific conditions in different settings. Just because limiting women's options can exacerbate HIV risk (for example, by forcing them into transactional sex) does not mean that more egalitarian environments are protective for women as these might facilitate their participation in risky sexual networks.

In discussing the connection between gender and HIV, the dissertation was drawn into exploring the associations between poverty, inequality and HIV. In chapters 2 and 3, we review the broad literature on the key drivers of HIV in sub-Saharan Africa. We discuss and weigh the evidence of the association between HIV and structural and behavioural determinants, and conclude that while there is some evidence of positive associations between poverty and HIV, there is also evidence that people higher up the income distribution have an elevated HIV risk. Furthermore, the precise links (transmission pathways) are rarely spelled out adequately. We recommend the wider use of detailed case

specific studies that thoroughly investigate the associations between specific measures of poverty and HIV risk and elaborate the specific pathways through which HIV risk is increased or decreased in particular contexts.

In chapter 4, we probe individual level data from southern Africa to investigate two aspects of the Southern African HIV epidemic. The first is the relative importance of individual level or structural factors in driving the HIV epidemic. The second evolved around whether there was a difference in HIV risk between men and women, emanating from these structural or behavioural determinants. The modelling in this chapter provided evidence of a varied relationship between structural and behavioural factors. In Lesotho, structural factors appeared to be far more important than individual behaviour variables. The importance of the structural context was underlined by the brief commentary provided about Lesotho's limited healthcare system (both with regard to human resources and infrastructure). While structural factors were a contributory factor to HIV risk levels in Swaziland, the behavioural variables also weighed in significantly. This is consistent with the brief macro-economic and socio-demographic review of the country which suggested that the country as a whole faces some structural and behavioural challenges especially regards to multiple partners and a persistently high HIV prevalence.

In Zimbabwe, where the healthcare system is better developed and where per capita incomes exceed those in Lesotho and Swaziland, behavioural factors were the strongest determinants of HIV status.

Among the three countries, the individual level relationship between the various correlates of HIV and HIV-status among women turned out to be quite different. As examples, while poor women in quintile two for all the countries had higher HIV risk levels than those in quintile one (table 4.21, models 3,6 and 9), this magnitude varied by country with the largest

differential risk between women in the lowest two quintiles in Lesotho. In this country, the richest women were more likely to be HIV-positive than the poorest, but this relationship was not evident in Swaziland and Zimbabwe. The findings in chapter 4 serve to reinforce our argument that the relationship between HIV and some of its touted correlates that include gender and poverty is context-specific and varies from country to country.

In chapter 5, we use cross-country analysis to investigate whether southern African countries have larger HIV epidemics because of gender inequality. We do not find any evidence in support of this claim. The literature generally bundles gender inequality measures and those that measure general disadvantage. We separate measures of general disadvantage and those that measure gender inequality and we observe two things. First, measures of gender inequality that compare the relative access to social amenities and resources between men and women perform poorly as determinants of HIV prevalence, and so do measures of general disadvantage, such as limited contraceptive prevalence.

The second and subsidiary observation from this chapter is that HIV dynamics in sub-Saharan Africa are distinctly different from those outside Africa. Cross-country research on the determinants of HIV should avoid, wherever possible, modelling correlates of HIV prevalence for countries within Africa and those outside Africa within the same multivariate models as this accentuates the difference between the southern African epidemics from that in other parts of the world. Just by restricting the sample to sub-Saharan data, the southern African dummy coefficient reduces to a significantly lower size than that reported by many studies.

Chapter 6 explores the relationship between IPV and HIV. We investigate the association between IPV and HIV by tracking and linking attitudes towards IPV to sexual behaviour associated with such attitudes, then to risk factors to experiencing IPV, and then the eventual

link to HIV-status. We come across a number of expected and unexpected results. One of the expected findings is that women in southern Africa endorse IPV more than men do. This is in line with what other researchers have found in other parts of sub-Saharan Africa. Why this is the case is still unclear.

Positive attitudes towards violence were, for both men and women, strongly correlated with social status, with those higher up less likely to endorse IPV, but again the gender differences were stark. Among the most educated and employed, women were more likely to endorse IPV.

Among men, experiencing IPV was not associated with sexual behaviour deviating from the mean, in terms of condom use, number of sexual partners and testing for HIV. Among women however, those that experienced IPV were likely to have more sexual partners and use condoms less – highly suggestive of individual behaviour attracting violence (and potentially heightening HIV risk) than the reverse, where IPV leads to HIV.

Upon investigation of whether IPV could be linked to HIV-status, data from Zimbabwean women does show evidence for the claim. Women that experienced IPV in the full model were about 16 per cent more likely to be HIV-positive. Although some of this heightened risk may come from their own behaviour, our model suggests that this effect is small, only accounting for about 20 per cent of this increased risk. So while IPV was associated with HIV risk, this was not because of women's own sexual behaviour and there was no evidence that men who perpetrated IPV were any sexually riskier than the other men in the sample. More research is thus needed to elucidate the specific pathways between IPV and HIV.

Our concluding observation is that the empirical literature and available survey data does not provide support for the proposition that empowering women will help combat the HIV

epidemic. The connections between various gender inequities and HIV are not understood adequately at the different levels in which they operate (national, communal and inter-personal). We are not saying that women do not suffer from oppression nor are we saying that this is not a problem. What we argue is that to be able to protect women from HIV, blanket approaches that assume unidirectional relationships between different forms of inequality and abuse and HIV risk are inappropriate. Aligning HIV prevention objectives with gender equality objectives is not necessarily the best way to prevent HIV among women. In some cases, it could work. In others, it may not be effective – it may actually create worse HIV consequences for women.

Secondly, there is no evidence that higher gender inequality in southern Africa is responsible for the higher HIV prevalence rates in the region. In the sub region and in greater sub-Saharan Africa, prevalence rates are more a function the age of the epidemic and income share among the poorest, than they are of any other factors.

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Appendixes

Appendix I. Notes on logistic regression

Logistic regression analysis comes with a number of assumptions. The main assumptions for logistic regression are:

1. There is not specification error

No specification error implies that the true conditional probabilities are a logistic function of the independent variables (the specification of the model is correct, i.e. it is reasonable to model the probability of the outcome variable in terms of the prescribed independent variables).

To test that there is no specification error, we use the linktest command.

"*The idea behind linktest is that if the model is properly specified, one should not be able to find any additional predictors that are statistically significant except by chance. After the regression command (in our case, logit or logistic), linktest uses the linear predicted value (_hat) and linear predicted value squared (_hatsq) as the predictors to rebuild the model. The variable _hat should be a statistically significant predictor, since it is the predicted value from the model. This will be the case unless the model is completely misspecified. On the other hand, if our model is properly specified, variable _hatsq shouldn't have much predictive power except by chance. Therefore, if _hatsq is significant, then the linktest is significant. This usually means that either we have omitted relevant variable(s) or our link function is not correctly specified*." http://www.ats.ucla.edu/stat/stata/webbooks/logistic/chapter3/statalog3.htm Accessed on: 2012/07/02

2. That there is Goodness of fit

Goodness of fit implies that no important variables are omitted, no extraneous variables are included and the independent variables are measured without error.

To assess goodness of fit, we use the lfit command in stata.

3. There is no collinearity and multicollinearity

This assumption implies that the observations are independent and thus the independent variables are not linear combinations of each other.

For each continuous variable, we compute the correlation coefficients as well as their p-values

Appendix II: Factor analysis for categorical variables

Although factor analysis is normally used with continuous variables, stata has an add-on command, polychloric, that can perform factor analysis with dichotomous variables. When using this command, the output from the factor analysis for categorical data is interpreted the same way as that from continuous data. (For more information on this command, please see http://www.ats.ucla.edu/stat/stata/faq/efa_categorical.htm [Accessed on 29/05/2012]). Below are the results from country-level factor analyses for the attitudes towards wife beating variables.

Please note, for all countries:

v744a: wife beating justified if she (wife) goes out without telling him (husband)
v744b: wife beating justified if she neglects the children
v744c: wife beating justified if she argues with him
v744d: wife beating justified if she refuses to have sex
v744e: wife beating justified if she burns the food

Factor analysis results, Lesotho

Factor analysis/correlation	Number of observations	=	5670
Method: principal factors	Retained factors	=	2
Rotation: orthogonal varimax (Kaiser off)	Number of parameters	=	9

Factor	**Variance**	**Difference**	**Proportion**	**Cumulative**
Factor1	2.45902	1.06971	0.6720	0.6720
Factor2	1.38931	.	0.3797	1.0517

LR test: independent vs. saturated: chi2(10) = 2.6e+04 Prob>chi2 = 0.0000

Rotated factor loadings (pattern matrix) and unique variances

Variable	**Factor1**	**Factor2**	**Uniqueness**
v744a	0.8017	0.3926	0.2032
v744b	0.8101	0.4611	0.1311
v744c	0.7177	0.5428	0.1903
v744d	0.5799	0.5975	0.3068
v744e	0.5557	0.6090	0.3203

Factor analysis results, Swaziland

Factor analysis/correlation	Number of observations	=	7746
Method: principal factors	Retained factors	=	3
Rotation: orthogonal varimax (Kaiser off)	Number of parameters	=	10

Factor	Variance	Difference	Proportion	Cumulative
Factor1	2.34929	1.19190	0.7151	0.7151
Factor2	1.15740	1.15701	0.3523	1.0674
Factor3	0.00039	.	0.0001	1.0675

LR test: independent vs. saturated: chi2(10) = 2.8e+04 Prob>chi2 = 0.0000

Rotated factor loadings (pattern matrix) and unique variances

Variable	Factor1	Factor2	Factor3	Uniqueness
v744a	0.7833	0.3796	-0.0034	0.2424
v744b	0.8446	0.3211	0.0079	0.1836
v744c	0.6990	0.5062	-0.0073	0.2550
v744d	0.4385	0.6162	-0.0006	0.4281
v744e	0.5845	0.5237	0.0161	0.3838

Factor analysis results, Zimbabwe

Factor analysis/correlation	Number of observations	=	12 119
Method: principal factors	Retained factors	=	2
Rotation: orthogonal varimax (Kaiser off)	Number of parameters	=	9

Factor	Variance	Difference	Proportion	Cumulative
Factor1	**2.32626**	**1.08333**	**0.7000**	**0.7000**
Factor2	1.24293	.	0.3740	1.0740

LR test: independent vs. saturated: chi2(10) = 4.6e+04 Prob>chi2 = 0.0000

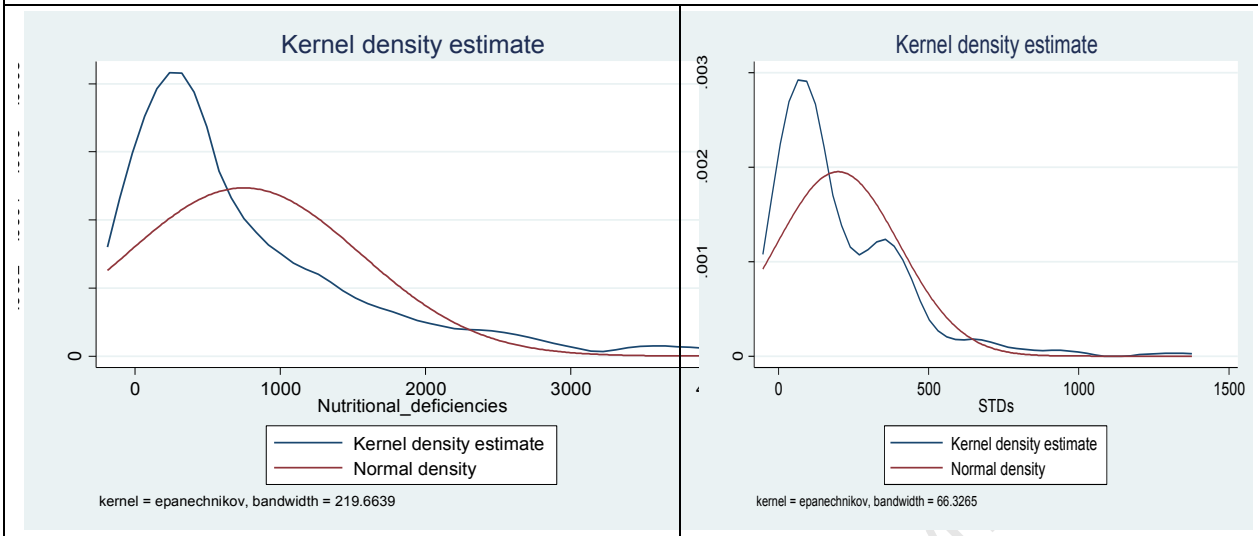
Rotated factor loadings (pattern matrix) and unique variances

Variable	Factor1	Factor2	Uniqueness
v744a	0.7427	0.4382	0.2564
v744b	0.7595	0.4238	0.2436
v744c	0.7130	0.5010	0.2407
v744d	0.5902	0.5652	0.3323
v744e	0.5841	0.5486	0.3578

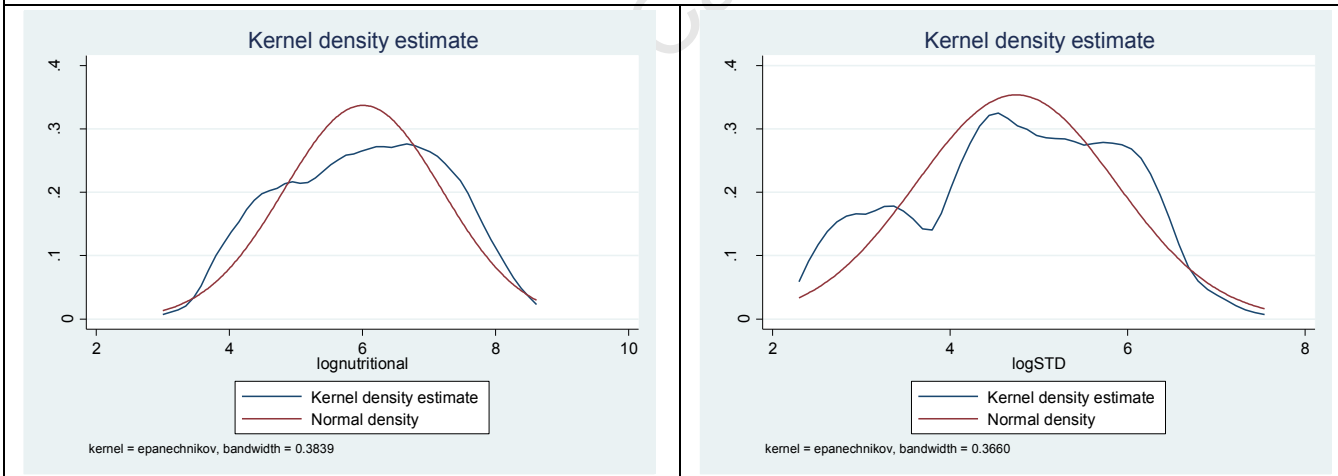
Appendix III. Logged and unlogged kernel density functions for DALY variables

Normality of data, STD DALY and Nutritional deficiency DALY

Kernel density estimates, unlogged DALYs



Kernel density estimates for logged DALYs



The logged DALY's for nutritional deficiencies and classical STDs have a more 'normal' distribution compared to the unlogged variables

Appendix IV. List of countries in OLS regressions, table 5.2

Base	Cofactor infections and health	Gender inequality	General disadvantage	Constant Sample
Angola	Angola	Botswana	Angola	Botswana
Botswana	Botswana	Lesotho	Botswana	Malawi
Lesotho	Malawi	Malawi	Lesotho	Mozambique
Malawi	Mozambique	Mozambique	Malawi	Namibia
Mozambique	Namibia	Namibia	Mozambique	Zambia
Namibia	Zambia	Swaziland	Namibia	Burundi
Swaziland	Burundi	Zambia	Swaziland	Rwanda
Zambia	Ethiopia	Burundi	Zambia	Uganda
Burundi	Rwanda	Rwanda	Burundi	Benin
Ethiopia	Tanzania	Uganda	Ethiopia	Cameroon
Rwanda	Uganda	Benin	Rwanda	Côte d'Ivoire
Tanzania	Benin	Cameroon	Tanzania	Ghana
Uganda	Burkina Faso	Côte d'Ivoire	Uganda	Niger
Benin	Cameroon	Gabon	Benin	Senegal
Burkina Faso	Côte d'Ivoire	Gambia	Burkina Faso	Argentina
Cameroon	Ghana	Ghana	Cameroon	Armenia
Côte d'Ivoire	Niger	Niger	Côte d'Ivoire	Azerbaijan
Gabon	Nigeria	Senegal	Gabon	Bolivia
Gambia	Senegal	Sierra Leone	Gambia	Cambodia
Ghana	Sierra Leone	Argentina	Ghana	China
Guinea-Bissau	Argentina	Armenia	Guinea-Bissau	Colombia
Niger	Armenia	Azerbaijan	Niger	Dominican Republic
Nigeria	Azerbaijan	Bolivia	Nigeria	El Salvador
Senegal	Belarus	Brazil	Senegal	Guatemala
Sierra Leone	Bolivia	Cambodia	Argentina	Honduras
Argentina	Brazil	Chile	Armenia	India
Armenia	Cambodia	China	Azerbaijan	Indonesia
Azerbaijan	Chile	Colombia	Belarus	Iran
Belarus	China	Costa Rica	Bolivia	Jamaica
Bolivia	Colombia	Croatia	Cambodia	Kazakhstan
Brazil	Comoros	Dominican Republic	China	Kyrgyzstan
Cambodia	Costa Rica	El Salvador	Colombia	Morocco
Chile	Croatia	Georgia	Dominican Republic	Nepal
China	Dominican Republic	Guatemala	El Salvador	Pakistan

Colombia	El Salvador	Honduras	Guatemala	Paraguay
Comoros	Georgia	India	Honduras	Thailand
Costa Rica	Guatemala	Indonesia	India	Tunisia
Croatia	Honduras	Iran	Indonesia	Viet Nam
Dominican Republic	India	Jamaica	Iran	
El Salvador	Indonesia	Kazakhstan	Jamaica	
Georgia	Iran	Kyrgyzstan	Kazakhstan	
Guatemala	Jamaica	Latvia	Kyrgyzstan	
Honduras	Kazakhstan	Lithuania	Madagascar	
India	Kyrgyzstan	Malaysia	Morocco	
Indonesia	Latvia	Morocco	Nepal	
Iran	Lithuania	Nepal	Pakistan	
Jamaica	Madagascar	Pakistan	Paraguay	
Kazakhstan	Malaysia	Panama	Thailand	
Kyrgyzstan	Morocco	Paraguay	Tunisia	
Latvia	Nepal	Russian Federation	Viet Nam	
Lithuania	Pakistan	Thailand		
Madagascar	Panama	Tunisia		
Malaysia	Paraguay	Viet Nam		
Morocco	Russian Federation			
Nepal	Thailand			
Pakistan	Tunisia			
Panama	Viet Nam			
Paraguay				
Russian Federation				
Thailand				
Tunisia				
Viet Nam				

Appendix V. OLS regression diagnostics: Goodness of fit, heteroskedasticity and Wald test of better fit in nested models for regression table 5.2

Associated p-values for the outlined tests

	Base	Cofactor infections and health	Gender inequality	General disadvantage for women
_hat	0.000	0.000	0.000	0.000
hat2	0.888	0.511	0.872	0.89
White test	0.4047	0.4377	0.4357	0.4334
Wald test		0.452	0.201	0.0614

Appendix VI. Multivariate regression, all regression models in table 4.2 replicated without including the GINI variable

Log HIV	**Base**	**Cofactor infections and health**	**Gender inequality**	**General disadvantage**
Southern Africa (***coef.)***	1.27***	1.13***	1.65***	1.24***
(***Std. Err.)***	-0.332	-0.418	-0.258	-0.305
Sub-Saharan Africa	2.371***	2.131***	2.197***	2.461***
	-0.259	-0.339	-0.385	-0.47
Log of share of income bottom 20% of population	-0.0656	-0.0542	-0.137	0.125
	-0.244	-0.223	-0.384	-0.286
Proportion urbanised	-0.00738	-0.00112	-0.0017	-0.00632
	-0.00658	-0.00855	-0.00952	-0.0067
Adult literacy	0.00909	0.00827	0.0143	0.00233
	-0.0059	-0.00687	-0.00948	-0.00701
Age of Epidemic	-0.282***	-0.503**	-0.218*	-0.231**

	-0.0942	-0.221	-0.11	-0.111
Age of Epidemic squared	0.00961*** -0.00304	0.0145** -0.00541	0.00728** -0.00346	0.00717** -0.00348
Proportion Muslim	-0.0085*** -0.00265	-0.0088*** -0.00311	-0.0098*** -0.00349	-0.00862** -0.00321
Log of remittances received per capita	0.0833* -0.0457	0.0925* -0.049	0.0717 -0.0507	0.141*** -0.0494
Log of STDs DALY		0.4 -0.249		
Log of Nutritional deficiencies DALY		-0.0351 -0.262		
Healthcare quality		0.00913 -0.0073		
Proportion of women seats in parliament			-0.0131 -0.00827	
Female labour force participation rate			-0.000489 -0.00767	
Proportion women secondary school relative to men			-0.0104 -0.00813	
Maternal leave length (Weeks)				-0.0698* -0.0362
Contraceptive prevalence percentage				0.0163* -0.00917
Medium rates of polygamy				0.356 -0.278
High rates of polygamy				0.532* -0.273
High son preference				-0.632* -0.334
Observations	62	57	53	50
R-squared	0.83	0.831	0.858	0.906

Appendix VII. Multivariate regression, replica of table 5.2 restricting sample to 38 countries for which no data is missing

Variables	Base	Cofactor infections and health	Gender inequality	General disadvantage
Southern Africa (***Coef***)	1.650***	1.555***	1.849***	1.590***
(Std error)	-0.281	-0.343	-0.243	-0.373
Sub-Saharan Africa	1.910***	1.849***	2.248***	1.572***
	-0.339	-0.421	-0.46	-0.515
Gini coefficient	0.0466	0.0164	0.0373	0.0652
	-0.0386	-0.041	-0.0354	-0.044
Log of share of wealth bottom 20 % share	0.738	0.146	0.761	0.971
	-0.829	-0.876	-0.785	-0.864
Proportion of population urbanised	-0.00653	-0.00148	-0.00268	-0.00631
	-0.00982	-0.0101	-0.0108	-0.0083
Adult literacy rate	0.00472	0.00574	0.0229**	-0.00517
	-0.00645	-0.00876	-0.00943	-0.0105
Epidemic age	-0.556*	-0.662	-0.616**	-0.537
	-0.306	-0.409	-0.282	-0.314
Age of epidemic squared	0.0160**	0.0185*	0.0174**	0.0144*
	-0.00749	-0.0102	-0.00717	-0.00773
Proportion of population Muslim	-0.0089**	-0.00850*	-0.00890*	-0.00489
	-0.00385	-0.00432	-0.00456	-0.00413
Log of remittances received per capita	0.0829	0.104	0.170**	0.063
	-0.0637	-0.0698	-0.0693	-0.0677
Log of STD DALY		0.353		
		-0.241		
Log nutritional deficiency DALY		0.089		
		-0.302		
Healthcare quality		0.0123		
		-0.0119		
Proportion seats parliament female			-0.0172*	
			-0.0092	
Female labour force participation rate			-0.00132	
			-0.00831	
Proportion of women in with secondary education			-0.0168**	

			-0.0074	
Length of maternal leave (weeks)				-0.0421 -0.0361
Modern contraceptive prevalence				0.0109 -0.0109
Medium rates of polygamy				0.236 -0.306
High rates of polygamy				0.518* -0.297
High son preference				-1.003** -0.37
Observations	38	38	38	38
R-squared	0.891	0.91	0.916	0.928

Appendix VIII. List of countries in regression table 5.3

Base	Cofactor infections and health	Gender inequality	General disadvantage	Constant sample
Angola	Angola	Botswana	Angola	Botswana
Botswana	Botswana	Lesotho	Botswana	Malawi
Lesotho	Malawi	Malawi	Lesotho	Mozambique
Malawi	Mozambique	Mozambique	Malawi	Namibia
Mozambique	Namibia	Namibia	Mozambique	Zambia
Namibia	Zambia	Swaziland	Namibia	Burundi
Swaziland	Burundi	Zambia	Swaziland	Rwanda
Zambia	Ethiopia	Burundi	Zambia	Uganda
Burundi	Rwanda	Rwanda	Burundi	Benin
Ethiopia	Tanzania	Uganda	Ethiopia	Cameroon
Rwanda	Uganda	Benin	Rwanda	Côte d'Ivoire
Tanzania	Benin	Cameroon	Tanzania	Ghana
Uganda	Burkina Faso	Côte d'Ivoire	Uganda	Niger
Benin	Cameroon	Gabon	Benin	Senegal
Burkina Faso	Côte d'Ivoire	Gambia	Burkina Faso	
Cameroon	Ghana	Ghana	Cameroon	
Côte d'Ivoire	Niger	Niger	Côte d'Ivoire	
Gabon	Nigeria	Senegal	Gabon	

Gambia Ghana Guinea-Bissau Niger Nigeria Senegal Sierra Leone	Senegal Sierra Leone	Sierra Leone	Gambia Ghana Guinea-Bissau Niger Nigeria Senegal	

Appendix IX. Diagnostics for regressions in table 5.3

P-values for respective tests

	Base	**Cofactor infections and health**	**Gender inequality**	**General disadvantage for women**
_hat	0.002	0.000	0.000	0.000
hat2	0.861	0.238	0.747	0.611
White test	0.4058	0.3496	0.3918	0.4038
Wald test		0.0226	0.041	0.323

Appendix X. Replicating base model regression in table 5.3, using the constant sample

Log HIV prevalence	**Coefficient**	**Std. Err.**	**t**	**P>t**
Southern Africa	1.62***	0.275	5.89	0.002
Gini	0.11	0.066	1.61	0.168
Log of share of income bottom 20% of population	2.51	1.526	1.65	0.161
Proportion urbanised	0.02	0.017	1.13	0.311
Adult literacy	0.02	0.015	1.63	0.165
Age of epidemic	-1.08*	0.535	-2.02	0.1
Age of Epidemic squared	0.03	0.014	1.99	0.103
Proportion Muslim	0.00	0.006	0.13	0.902
Log of remittances received per capita	-0.02	0.138	-0.15	0.885
N=14. F (9, 15)= 94.14. R squared=0.98				

Appendix XI. Regression (20) excluding Gini coefficient

Log HIV	Coef.	Robust Std. Err.	t	P>t
Southern Africa	1.58	0.307	5.140	0.00
Log share of income bottom 20 %	0.87	0.527	1.650	0.12
Proportion urbanised	0.00	0.007	-0.130	0.90
Adult literacy	0.03	0.011	2.950	0.01
Epidemic age	-0.27	0.176	-1.550	0.14
Age of epidemic squared	0.01	0.005	1.200	0.25
Proportion Muslim	0.00	0.006	-0.070	0.95
Log of remittances per capita	0.08	0.058	1.400	0.18

Appendix XII: Multiple regression: Sexual violence as dependant variable

	Base	**SES**	**Full**
Sexual violence ***Coeff***	0.0411	0.0466	0.0509
Std Err.	(0.106)	(0.107)	(0.108)
Age	0.374***	0.385***	0.362***
	(0.0393)	(0.0400)	(0.0408)
Age Squared	-0.00531***	-0.00545***	-0.00522***
	(0.000595)	(0.000611)	(0.000624)
Education		0.0791*	0.0684
		(0.0410)	(0.0419)
Education Squared		-0.00677**	-0.00614**
		(0.00287)	(0.00292)
Household Wealth Quintile 2		0.101	0.122
		(0.118)	(0.119)
Household Wealth Quintile 3		0.234*	0.288**
		(0.122)	(0.123)
Household Wealth Quintile 4		0.335**	0.360**
		(0.144)	(0.146)
Household Wealth Quintile 5		-0.114	-0.0376
		(0.199)	(0.203)
Rural residence		-0.135	-0.105

		(0.141)	(0.143)
Age first sex			-0.0348* (0.0187)
Age first marriage			0.0371** (0.0145)
Lifetime sex partners			0.302*** (0.0462)
Constant	-7.238*** (0.626)	-7.614*** (0.653)	-7.729*** (0.686)
Observations	3 855	3 854	3 854
Pseudo R2	0.0285	0.0343	0.057
P	0.000	0.000	0.000

Appendix XIII : Multiple regression Physical violence as independent variable

	Base	**SES**	**Full**
Physical violence ***Coeff*** ***Std Err.***	0.235*** (0.0797)	0.230*** (0.0806)	0.191** (0.0824)
Age	0.379*** (0.039)	0.388*** (0.040)	0.363*** (0.041)
Age Squared	-0.005*** (0.001)	-0.005*** (0.001)	-0.005*** (0.001)
Education		0.0820** (0.0410)	0.0702* (0.0419)
Education Squared		-0.00680** (0.00287)	-0.00622** (0.00292)
Household Wealth Quintile 2		0.0979 (0.118)	0.119 (0.120)
Household Wealth Quintile 3		0.245** (0.123)	0.297** (0.123)
Household Wealth Quintile 4		0.350** (0.144)	0.371** (0.145)
Household Wealth Quintile 5		-0.0888 (0.199)	-0.0203 (0.203)
Rural residence		-0.129 (0.140)	-0.103 (0.142)
Age first sex			-0.0340* (0.0187)
Age first marriage			0.0391***

			(0.0146)
Lifetime sex partners			0.298*** (0.0462)
Constant	-7.388*** (0.627)	-7.767*** (0.655)	-7.869*** (0.688)
Observations	3 857	3 856	3 856
Pseudo R2	0.030	0.036	0.058
Chi(2) P-value	0.000	0.000	0.000