# *Economic Inequality as an Underlying Cause of HIV in Africa? The HIV-Poverty Thesis Re-Examined*

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**Abstract:**
Contrary to theories that poverty acts as an underlying cause of HIV infection in sub-Saharan Africa, an increasing body of evidence at the national and individual levels indicates that wealthier countries and wealthier individuals within countries, are at heightened risk for HIV. This study poses the hypothesis that HIV infection rates increase as a result of conditions of socio-economic inequality rather than poverty. Examining demographic and health survey data from sixteen African countries, this study utilizes a multi-level model to assess the relationship between HIV infection, economic inequality and individual wealth. All multivariate models were run as a two-level, hierarchical random intercept and slope models in Stata 10 adjusted for clustering at the regional level. Results from the two-level random intercept model demonstrated that individual wealth status and regional gini coefficient positively predict HIV infection, with inequality producing a contextual effect even once the individual wealth composition of a region is taken into account. Thus, as hypothesized, wealthier individuals are at higher risk for HIV infection and the probability of infection increases with rising regional (within-country) inequality. Results from further analysis demonstrate that the heightened odds of infection amongst the wealthy and those living in more unequal regions appears to be driven by participation in increasingly informal sexual network structures. Implications of these findings for HIV prevention strategies in developing countries are discussed.

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## Introduction

Sub-Saharan Africa is home to only 10% of the world's population and yet carries over 64% of the world's HIV infections (UNAIDS, 2006). Explanations for sub-Saharan Africa's (SSA) disproportionate burden of HIV/AIDS have been linked directly or indirectly to the continent's endemic poverty and its corollaries (e.g., Coovadia & Hadingham, 2005; Barnett & Whiteside, 2002; Stillwaggon, 2006). However, contrary to theories of poverty as the underlying cause of HIV infection in Africa, an increasing body of evidence at the national and individual levels indicates that wealthier countries in Africa, and wealthier individuals within countries, are at heightened risk for HIV (Shelton, Cassell, & Adetunji, 2005; Wojcicki, 2005; Tomlinson, 2006; Mishra, et. al., 2007). This study tests the hypothesis that HIV infection increases under conditions of socio-economic inequality and aims to explain what has come to be known as the "positive wealth gradient in HIV infection" arguing that HIV is a disease associated with conditions of relative rather than absolute deprivation. Making use of sixteen Demographic and Health Surveys from sub-Saharan Africa with linked HIV test results, this study utilizes a multi-level model to examine the effects of the distribution of income at the national and sub-national levels, as well as the position of an individual within the economic hierarchy on HIV infection.

## Background

*The HIV-Poverty Thesis*. At least from an advocacy perspective (if not a scientific one) in explanations as to why SSA has taken the brunt of the epidemic, it is almost reflexively assumed that Africa's status as the poorest continent on earth is what has made it the most prone to HIV infection since HIV supposedly follows poverty and social

marginalization. HIV more than other global health afflictions, has been held up as the ultimate disease of poverty, used as an archetype of the harms of structural adjustment programs and social injustice generally.

The idea that poverty is an underlying cause of HIV in Africa was brought into sharp relief during the 13th International AIDS Conference in 2000 when President Mbeki, in an address at the opening ceremony of the conference, made various comments alluding to the notion that conditions of poverty in Africa constitute a primary reason that the virus has spread so rapidly across the continent. In his own words:

> "What I heard as that story [from the World Health Organization] was told, was that extreme poverty is the world's biggest killer and the greatest cause of ill health and suffering across the globe. As I listened longer, I heard stories being told about malaria, tuberculosis, hepatitis B, HIV-AIDS and other diseases. I heard also about micro-nutrient malnutrition, iodine and vitamin A deficiency. I heard of syphilis, gonorrhea, genital herpes and other sexually transmitted diseases as well as teenage pregnancies. I also heard of cholera, respiratory infections, anemia, bilharzias, river blindness, guinea worms and other illnesses with complicated Latin names. As I listened even longer to this tale of human woe, I heard the name recur with frightening frequency - Africa, Africa, Africa! As I listened and heard the whole story told about our own country, it seemed to me that we could not blame everything on a single virus. It seemed to me also that every living African, whether in good or ill health, is prey to many enemies of health that would interact one upon the other in many ways, within one human body."
> -(Mbeki, Speech at Opening Ceremony, 2000).

Regrettably, Mbeki's ideas regarding poverty as an underlying cause of HV became intermingled with his questioning of the virology of the HI virus and its connection to the syndrome of AIDS, and the questions he raised became framed as a form of AIDS denialism or dissident thinking. However, some social epidemiologists such as Paul Farmer defended the HIV-Poverty thesis arguing that there likely was something to the connection between Africa's poverty and high rates of infection (see e.g., Basu, Kedar &

Farmer, 2000). While Mbeki's comments were generally interpreted as a questioning of the physiological connection between HIV and the syndrome of AIDS, his logic could also be interpreted as questioning whether poverty acts as a distal or underlying cause of various health conditions in Africa, which interact to increase vulnerability to acquiring HIV and ultimately also to developing AIDS. This line of thinking has a basis in the field of social epidemiology where it has been argued that social conditions such as poverty act as a "fundamental cause" of disease in that "the association cannot be broken by merely addressing the proximal determinants" (Link & Phelan, 1995).

In fact, Mbeki's logic was further bolstered by the publication of a book entitled "The Ecology of AIDS in Africa," by Eileen Stillwaggon (2006), which built the case that the "ecobiology" of poverty was what made Africans so prone to contracting HIV/AIDS. Stillwaggon excoriated explanations for SSA's high HIV prevalence grounded in African sexual behavior in favor of "ecobiological determinants" such as "malaria, intestinal helminths, poor nutrition, untreated STDs, genital schistosomiasis, filariasis, and other endemic factors" characteristic of conditions of poverty experienced in the developing world that increase viral load and viral shedding. Stillwagon's line of reasoning makes an important contribution towards understanding that population characteristics not directly related to more proximal risk factors such as behavior can nevertheless exert powerful downstream influence on health outcomes- in this case, ecobiology affects vulnerability to infection with HIV by increasing the likelihood of infection at each exposure.

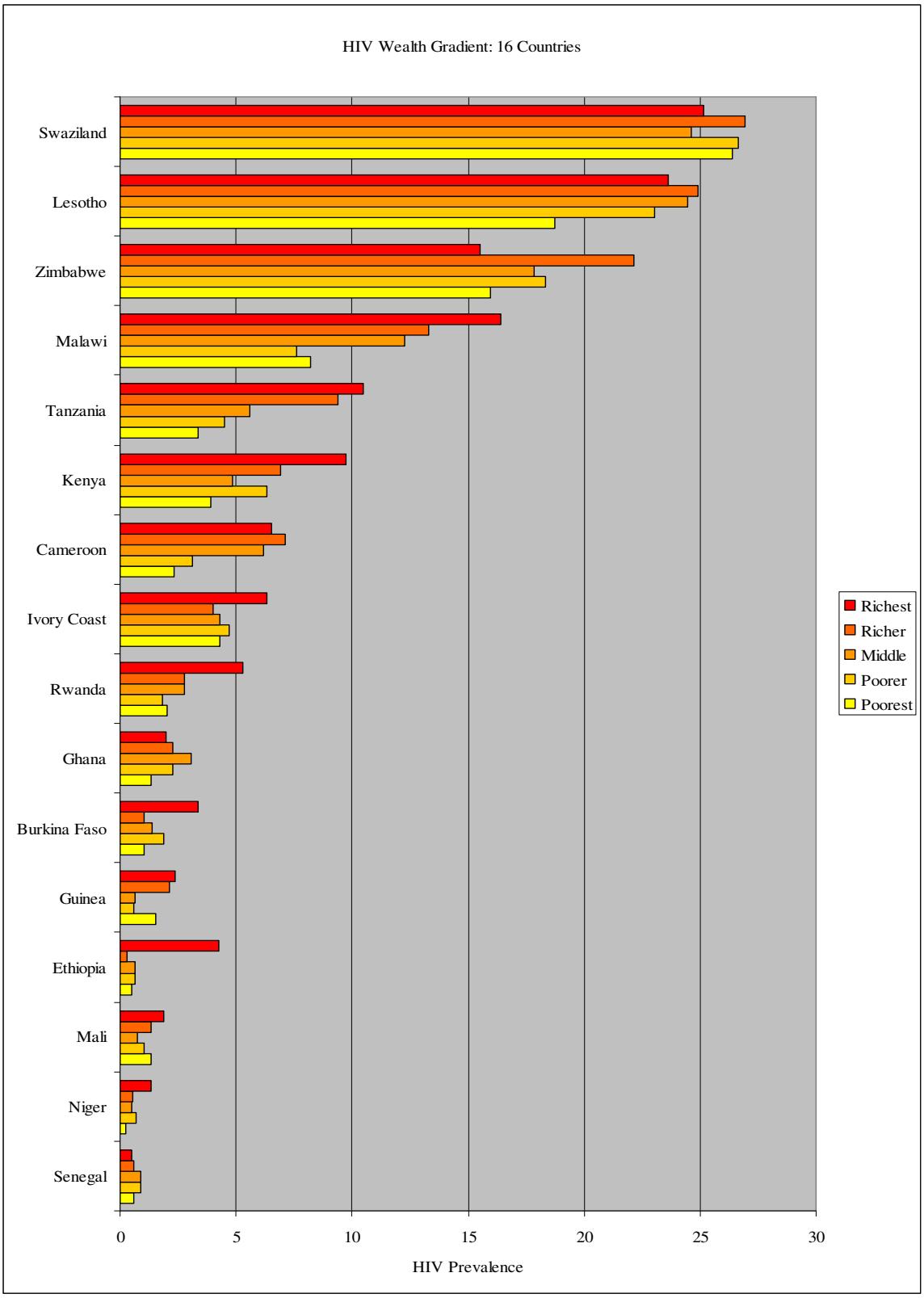
The only problem is, Mbeki and Stillwaggon got the diagnosis of who was most at risk wrong: it was not the poor that were the most likely to contract HIV, but the rich.

***The Social Gradient in HIV Infection***. Contrary to most health disparities where health outcomes tend to improve with rising wealth, an increasing body of evidence from actuarial, ethnographic and nationally representative probability studies of HIV infection in sub-Saharan Africa have demonstrated that wealthier and more educated individuals in Africa have higher rates of HIV infection (Shelton, Cassell, & Adetunji, 2005; Wojcicki, 2005; Tomlinson, 2006; Mishra, et. al., 2007; Forston, 2008). Researchers at MEASURE-DHS have conducted the most comprehensive analysis to date of their own data on the relationship between individual-level wealth and HIV/AIDS across eight African countries (Mishra, et. al., 2007). After controlling for an array of underlying and mediating factors (education, urban/rural residence, community wealth, sexual risk taking, condom use, and male circumcision) they find that wealthier individuals are still more likely to be HIV positive than poorer individuals, concluding that "these findings question the basis for poverty-driven programs for HIV/AIDS prevention in developing countries" (p. 45).

Below are the results from sixteen Demographic and Health Surveys with linked HIV biomarkers (see Figure 1). The bars represent HIV prevalence by wealth quintile. The red bar represents the wealthiest 20% of the population and the light yellow bar the poorest. What is evident from this graphic is that, more or less, across 16 African countries, HIV infection seems to rise with increasing wealth at the individual level, and it is certainly not the poorest individuals that are the most likely to be infected as has often been assumed. There is in fact a positive wealth gradient in HIV infection in most African countries.[1]

[1] Senegal, Ghana and Swaziland seem to be exceptions to this rule, at least at the national level. I will discuss potential reasons for this later.

**Figure 1: HIV Wealth Gradient, 16 Countries**



This finding is striking since there are a number of reasons to think that richer people (especially the richest people) should have lower infection rates, including higher educational attainment, and greater level of exposure to government messages and mass media. Other analyses (i.e., Forston, 2008) have also shown that this gradient in infection holds even more strongly for education than wealth, so it is truly an SES gradient in HIV infection and quite counterintuitive in many respects.

What is even more striking is that, if anything, this positive association between wealth and HIV is even steeper for women than for men (see Figures 2 & 3). It is not the case that wealthy men are having sex with poor women, since wealthy women in some cases have an even steeper wealth gradient in infection than men. This finding is quite striking again given the amount of scholarship that has gone into linking women's vulnerability to HIV to their poverty.

**Figure 2: Female HIV-Wealth Gradient, 16 African Countries**

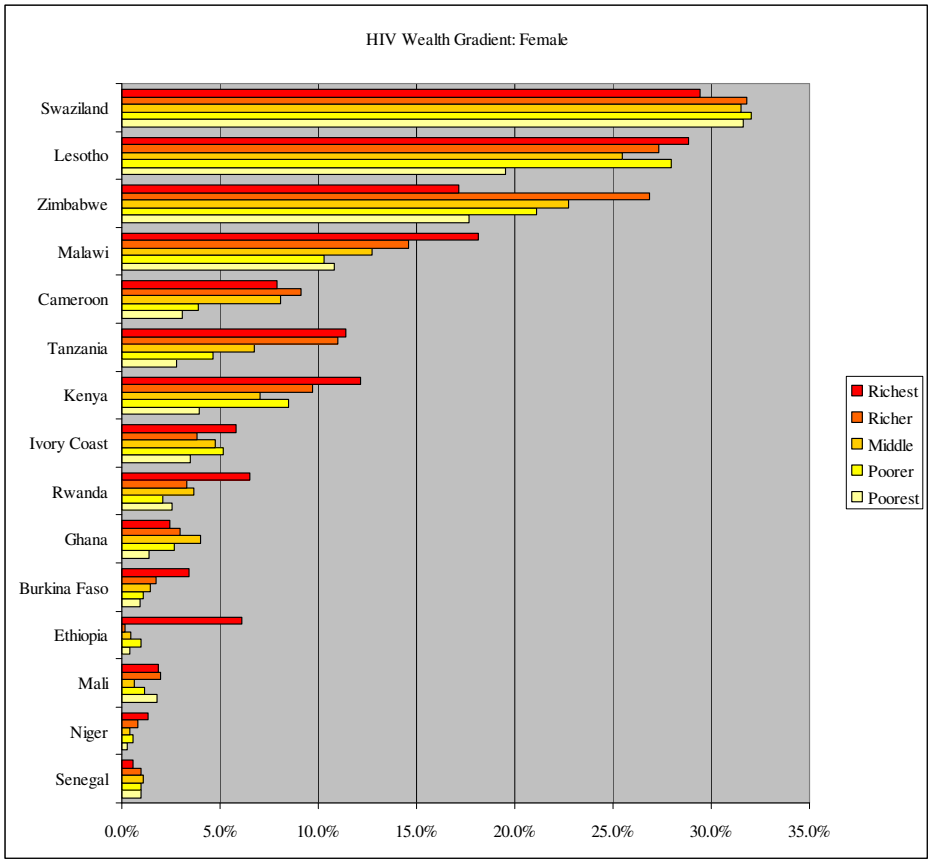
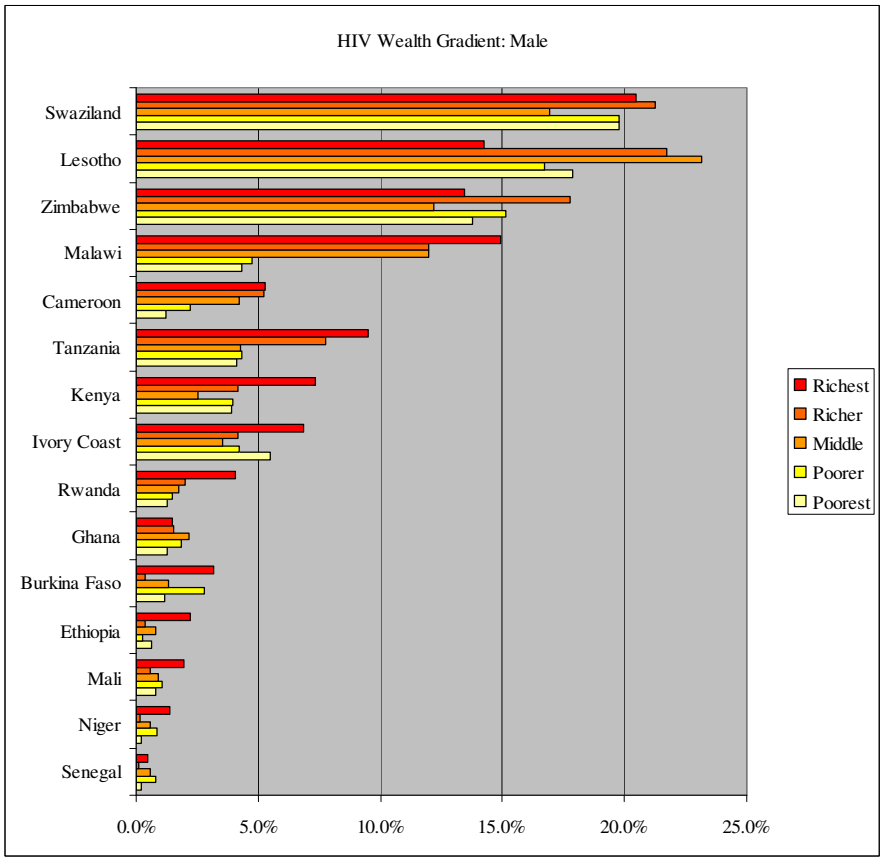



**Figure 3: Male HIV-Wealth Gradient, 16 African Countries**



The idea that poverty is an underlying cause of HIV in SSA appears to be largely derived from an ecologic fallacy arising out of an incorrect generalization from aggregate statistics on global HIV prevalence distribution to the national and individual level. Researchers and advocates have assumed that because Africa is the poorest continent on earth and has the highest HIV prevalence rates in the world that it is necessarily the poorest countries and individuals in SSA that are the most at risk for HIV. A second explanation is definitional and lies in a failure to distinguish between the concepts of relative poverty, as predominately experienced in industrialized countries, and absolute poverty, which persists largely in developing countries. Because HIV predominately affects the poor and marginalized in AICs, researchers have assumed that the same dynamic should hold in SSA. In reality, HIV in the developed world was previously more cross-cutting when it was first discovered in the 80s. Owing to the lack of screening in the blood supply and knowledge about the source of transmission, initially HIV/AIDS struck affluent homosexual men, hemophiliacs and others unfortunate enough to have become infected through a tainted blood supply and highly-exposed sexual networks, HIV was more "democratic" in who it infected (Baldwin, 2005). However, over time as research revealed the sources of transmission, how to protect oneself and access to tests and treatments became available, HIV began to descend the social gradient to mainly affect the poor, minority groups and the socially marginalized in AICs (Baldwin, 2005). In this way, HIV began to be associated with a disease of the poor. However, poverty in the context of AICs cannot be thought of as the same as poverty as experienced in the developing world. Poverty in the developing world context includes people who do not have their basic needs met and primarily live at a subsistence level. Conceptualized in

this absolute sense, poverty may not in fact be associated with HIV. Thus, if the relatively poor (as opposed to the absolutely poor) have higher infection rates in SSA, confusion about whether poverty is an underlying cause of HIV may be as simple as a conflation of the definition of absolute versus relative poverty.

In addition, qualitative and quantitative studies that do not utilize nationally representative probability samples and have tended to focus on economically disadvantaged populations may have furthered the idea that poverty is an underlying cause of HIV. Both quantitative and qualitative studies in public health tend to rely on convenience samples recruited from antenatal clinics, hospitals, high-risk groups, social service recipients, etc. As a result, even surveys with large sample sizes are unable to assess which demographic groups are most affected and because of a bias in assuming the poor are most at risk (as they often are for other diseases), tend to mainly be conducted with the poor or lower-income groups.

In fact, up until the introduction of the Demographic and Health Surveys with HIV biomarkers beginning in 2001, no national probability samples of HIV prevalence existed.[2] Instead, most estimates of HIV were either based on anonymous surveillance studies at antenatal clinics (modeling population prevalence from samples of pregnant women) or relied on some form of convenience sample. Further, because it was not possible to link these population estimates of HIV infection to other social and demographic variables, there was no way to know which social groups were most affected by HIV. With the introduction of Demographic and Health Surveys with HIV

[2] Probability samples are survey samples where each unit has a known, non-zero probability of selection. The advantage of a probability sample is that it allows researchers to estimate statistics for the whole population within a certain degree of measurable error (without actually having test everyone in the whole population).

biomarkers, which are based on national and sub-national probability samples it is possible to obtain HIV data linked with a series of demographic and social characteristics.

Previous research looking at the HIV-wealth gradient has primarily focused on efforts to explain away the positive relationship between wealth and HIV status as the product of confounding or measurement error. Opuni-Akuamoa (2008), for instance, argues that the positive wealth gradient is the result of confounding – since wealth is mainly concentrated in urban areas, as is HIV, it appears that wealthier individuals have higher infection rates, but in fact this is a product of urban residence rather than wealth per se. Once urbanicity is taken into account, she demonstrates that in fact poorer individuals in urban areas have a greater odds of infection. However, this explanation fails to explain why HIV infection rates should be higher in urban areas if not a product of the higher wealth of these regions and thereby fails to meet the requirements to adequately demonstrate confounding. If something about the greater wealth concentration in urban areas is contributing to the spread of HIV (which I argue it is), then wealth still remains a driver of the epidemic and the relationship between wealth and HIV is not the product of confounding, but rather is an artifact of a true relationship, though perhaps working through more proximal mechanisms to produce increased risk amongst the wealthy or those living in wealthier areas. Bingenheimer (2007), like Opuni-Akuamoa, questions whether something about the measurement of wealth contributes to confounding in the positive HIV-wealth gradient. He argues that the fact that traditional forms of wealth, like cattle ownership, is weighted negatively in the index, "leads to a situation in which some people may appear to be wealthy when one considers

their traditional holdings, yet appear to be poor according to their score on the DHS Wealth Index," (Bingenheimer, 2007). Bingenheimer gets closer than Opuni to identifying what I argue is the source of confusion around the HIV-wealth gradient, namely the idea that relative social position and integration into a market economy rather than a subsistence economy is a major driver of HIV infection. In other words, I believe that the mechanism linking wealth and HIV infection has to do with integration into market economies, which fundamentally changes ones relationship to the means of production and relative social position. People who would be considered rich in terms of their traditional holdings would still be considered poor by global or material standards. The issue for HIV risk is one's position in the social hierarchy and aspirations for modernity.

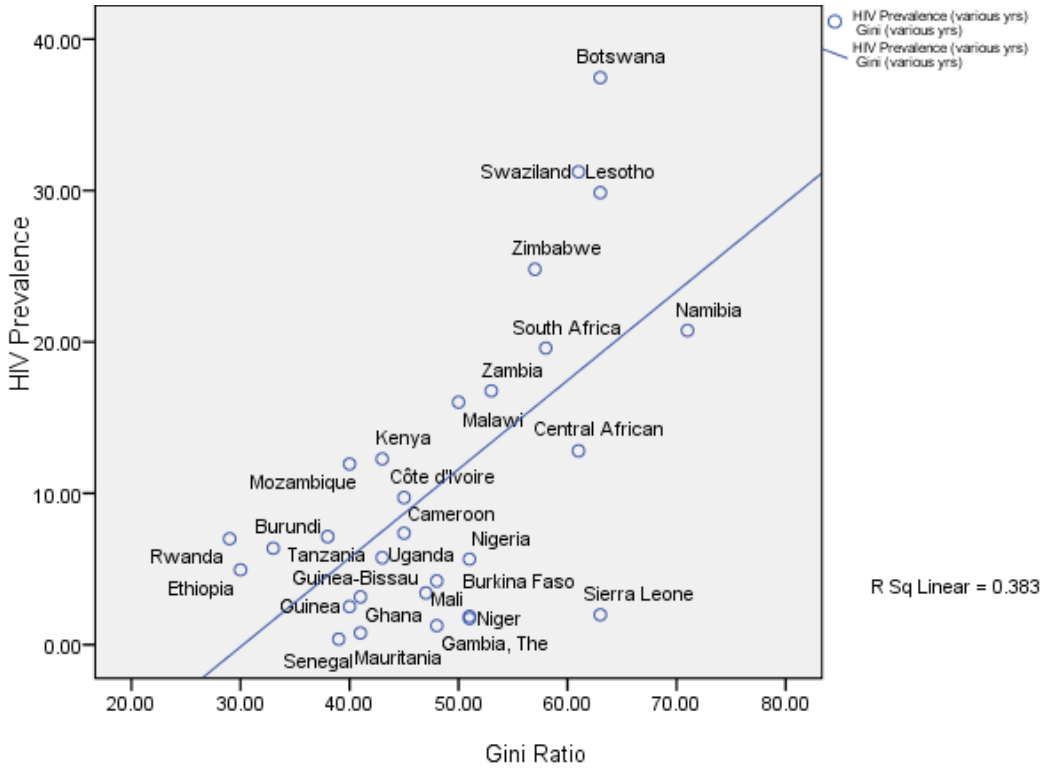
In another study looking exclusively at the case of Burkina Faso, Lachaud (2007) identifies the source of the HIV-wealth gradient as arising from the fact that richer people live in an area of Burkina Faso that is particularly tied into commerce with the Ivory Coast, a country with a high HIV prevalence rate. In this way he sees the positive wealth gradient in infection as arising from a compositional effect of richer people living in close proximity with and being economically linked to a higher prevalence country. Again, Lachaud's explanation does not explain why Ivory Coast, generally thought to be the economic hub of West Africa, itself has a higher HIV infection rate. He has also, somewhat unwittingly, identified more proximal causal mechanisms through which wealth generation at the aggregate level may be contributing to the spread of HIV through migration and economic linkages.

While certain studies continue to look incredulously at the positive HIV-wealth relationship, others have found this relationship impossible to fully explain away. Gillespie, Kadiyala & Greenera (2007), for instance, conclude that despite the fact that cross-sectional studies such as the DHS suffer from the limitations that they are unable to establish a clear direction of causality (the effect of economic status on HIV infection versus the effect of HIV infection on economic status) and that they are unable to control for the fact that individuals from richer households may survive longer with HIV, they conclude that: "although it is true that poor individuals and households are likely to be hit harder by the downstream impacts of AIDS, their chances of being exposed to HIV in the first place are not necessarily greater than wealthier individuals or households," (Gillespie, Kadiyala & Greenera, 2007). Mishra et al (2007) also finally conclude after a series of tests that the positive HIV-wealth gradient appears to be a genuine artifact and that wealth seems to even supersede lack of male circumcision as a risk factor for HIV since his team finds that wealthier men are more likely to be circumcised and yet still have higher HIV infection rates. Daniel Halperin, a well known HIV prevention proponent, has added wealth among his list of important factors that figure into prevention strategies that the HIV community has relatively ignored (Potts et al, 2008).

To my knowledge, no previous study has looked at the full scope of the HIV wealth gradient across the sixteen countries where HIV biomarker data is available, nor has any prior study on this topic adopted a multi-level approach that allows for simultaneous testing of the ecologic and individual relationship between wealth and HIV. Finally, no other study has identified economic inequality and relative social status as the underlying theoretical construct driving the relationship between wealth and HIV.

***If Africa's poverty is not the problem, then what is?*** One commonality high prevalence countries do share is the fact that they are among the most unequal countries in the world. Whereas South Africa is typically held up as the most unequal country globally, in fact, Southern Africa on the whole is home to a host of highly unequal countries including Namibia, which tops the world charts with a gini ratio of 70.[3] Examining a scatterplot of HIV prevalence by gini ratio reveals a positive, upward sloping relationship between increasing inequality and HIV prevalence (see Figure 4).

**Figure 4: HIV Prevalence (2003) by Gini Ratio (various years). sub-Saharan African Countries**




Notes: Gini Source- Deininger & Squire Measuring Income Inequality Database, most recent year available. HIV Prevalence Source- UNAIDS 2006 HIV/AIDS estimates

[3] A gini ratio is a measure of dispersion often used to measure economic inequality. It is a measure of how far the per capita distribution of economic resources in a population diverges from what would be a perfectly equal distribution of resources and is expressed as a figure ranging between 0 and 100 with 0 representing perfect equality and 100 representing perfect inequality.

Economic inequality has been found to be significantly positively correlated with a number of health outcomes and health related behaviors including violence (Hsieh & Pugh, 1993), crime (Kawachi, et. al.,1998), overall life expectancy (Marmot, 2004), chronic disease (Wilkinson, 1999), and rates of tuberculosis (Harling, Ehrlich & Myer, 2007), as well as STDs and HIV in the developed world context (e.g., Hardwick & Patychuk, 1999). While all societies have been shown to have social gradients in health (generally such that wealthier individuals have better health outcomes), economic inequality has been demonstrated to worsen health outcomes across all economic strata in society (e.g., Marmot, 2004; Daniels, Kennedy, Kawachi, 1999). Economic inequality appears to have what social epidemiologists refer to as a "contextual" effect on health outcomes, such that everyone is made worse off as a result of living in unequal conditions (Kreiger, 2001). Ecologic studies of HIV risk have demonstrated that national income inequality is significantly positively correlated with HIV/AIDS levels (Drain, et. al., 2004; Talbott, 2007). However, the intersections of the distribution of economic wealth at the aggregate level and the effect of personal wealth status on HIV risk at the individual level are yet to be simultaneously evaluated and rectified.

Where inequality has been shown to produce higher rates of disease at the ecologic level, an individual's sense of relative deprivation, where she stands on the social ladder, has been shown to increase risk for a host of diseases (Marmot, 2004). Scholars working in the field of social epidemiology routinely distinguish between relative poverty, which has to do with an individual's relative economic position in society, and absolute poverty, which is considered to be a deprivation of basic needs such as running water, sanitation systems and adequate nutrition (Marmot & Wilkinson, 2005).

Whereas the HIV-Poverty thesis averred that conditions of absolute poverty were the driving force behind Africa's vulnerability to HIV infection, I argue that it is not absolute poverty, but relative poverty- economic inequality at the aggregate level and socio-economic position at the individual level- that drives HIV infection trends in SSA.

*Theory*: *Development, Distribution and Disease*. In the 1950s, during the heyday of development theory, Simon Kuznets, an economist, observed that economic inequality tended to form an inverted U-shape relationship with economic development. In other words, economic inequality appeared to rise in the early stages of economic development and then level off and decline over time. Though this idea has been contested and revised over time, especially in light of examples of successful development where economic growth has not come at the expense of equity (i.e., East Asia), in the case of Southern Africa, the apartheid-driven industrialization experienced in this sub-region has been particularly inequality-inducing, producing among the most unequal countries in the world. Likewise, Simon Szreter (1999), a demographic historian, argues that rapid economic growth may actually cause health to get worse before it gets better. He attributes this to what he calls the "four D's"- "disruption" of traditional ways of doing things, increasing relative "deprivation" followed by increases in "disease" and "death". As Szreter (1999) explains, in contrast with theories that posit a linear relationship between rising GDP and improvements in health, "economic growth should be understood as setting in train a socially and politically dangerous, destabilizing, and health-threatening set of forces." While Szreter poses his theory mainly in the historical context of the experience of Britain and Wales, referring to the modalities through which rapid urbanization and economic development increased the conditions rife for disease,

this model could be extended to explain HIV in SSA. It appears that HIV falls heaviest on incompletely industrialized countries, particularly those experiencing inequality-inducing economic growth.

In a similar vein to Szeter and Kuznets' U-shape relationship between development, inequality and health, the social epidemiology literature makes a distinction between diseases associated with absolute poverty and relative poverty. Examples of diseases associated with absolute poverty include malnutrition, diarrheal disease, and neglected "tropical" diseases. These diseases are subject to a threshold effect such that once a country overcomes the conditions that give rise to them (i.e., eradicates conditions of absolute poverty), these diseases tend to decline on their own. Though a great deal of debate exists about the relative contribution of different factors in explaining this decline including improved living standards (e.g., McKeown, 1979), social mobilization (Szreter, 1999) or access to life saving medicines and health services (e.g., Preston, 1975), most global health advocates would agree that eliminating conditions of extreme poverty and deprivation would go a long way towards reducing disease burden associated with infectious and poverty-driven diseases in developing countries.

Relative poverty or deprivation, on the other hand, refers to a situation where peoples' basic needs have been met, but relative to others are not doing so well. Diseases under these conditions tend to form a gradient such that the lower down the ladder one is, the more she is affected. This, at least, is the trend in rich countries, where poverty is best characterized as "relative" rather than absolute. However, in poor countries, people living under conditions of absolute poverty and those living in conditions of relative poverty may simultaneously exist. Richer people in poor countries may have their basic needs

met- electricity, a flush toilet, concrete floors- but they may not have as much as others. This same relationship between relative deprivation and health has been shown to exist at the country-level as well- past a certain GDP, cross-national studies have shown that there is a diminishing return to health with more GDP- what matters for health in a country becomes not the absolute amount of economic resources but the distribution of economic resources.

However, in addition to this dichotomy between diseases of absolute poverty and diseases of relative poverty, there appear to be certain diseases such as HIV/AIDS, that fall in between these two categories- diseases that tend to be exacerbated under conditions of industrialization or development when people are making a transition from a state of absolute poverty to one of relative poverty. This transition from absolute to relative poverty that accompanies economic "development" (or "marketization" to use a less loaded term) encourages the breakdown of traditional social controls on sexuality and their replacement with new and changing sexual mores, which are themselves the product of the interaction between changing material economic conditions and social and cultural value change.[4]

Clearly wealth or inequality is not a direct cause of HIV infection. Rather, ultimately some form of behavior or biology must mediate the relationship between

[4] A brief note on "transition theory" and its discontents. The field of demography has been criticized for its overreliance on modernist assumptions underlying its foundational theory of the demographic transition (Greenhalgh, 1996). Critics of transition theory have challenged the developmental paradigm associated with this modernist perspective for its linear, progressive and teleological bias. Post-modern theory has revealed the Eurocentric, ahistorical and apolitical evolutionary view of human history inherent in economic "development" and "modernization" theory. To clarify the position of this paper, while I reject the linear and teleological assumptions of transition theory - that all countries can expect to pass through a similar set of social and demographic changes that will progressively lead to a normatively superior state of being increasingly resembling the West. However, I do believe that it is both possible and useful to analyze how changing structural conditions and modes of production produce sets of social conditions that affect health in a predictable manner. When social science loses its ability to create generalizable theory, I believe that it loses its utility and power.

wealth or inequality and HIV infection. A plausible explanation for this relationship is that wealthier individuals or people living in more unequal regions should have more sexual partners or more high risk sexual networks, especially concurrent sexual partnerships which have found to be particularly risky, than poorer individuals (Epstein, 2008). Migration patterns and urbanization are especially disruptive to traditional controls on sexual behavior and encourage the establishment of multiple sexual partnerships. These economic transitions coupled with tipping points as HIV increases can explain individual and population risk for HIV (see Figure 5 in Appendix).

**Methods**

This research draws on multi-national data from Demographic and Health Surveys (DHS) with linked individual HIV test results from sixteen sub-Saharan African countries. Demographic and Health Surveys (DHS) are an international series of nationally representative household surveys conducted in middle- and lower-income countries (Fisher & Way, 1988; Mishra, et. al., 2006). Although the principal objective of the demographic and health surveys is to provide current and reliable data on fertility and family planning behavior, in recent years, the demographic and health survey program has become a major source of data on HIV prevalence in many countries. Sample sizes and sampling procedures in these surveys are adjusted to ensure statistically meaningful estimates can be reached for each region and for both urban and rural populations. MEASURE-DHS assists countries in calculating appropriate sample sizes to provide enough power to generate meaningful population estimates at the national and sub-national levels. Sample sizes within the sixteen countries vary depending on the size of the country's population, the number of administrative units and other factors.

Demographic and Health Survey data is available upon request from the MEASURE-DHS website (http://www.measuredhs.com/). The ethical review boards associated with ORC-Macro, a U.S.-based company that provides technical assistance to DHS worldwide, review all data collection and HIV testing procedures as well as the ethical review boards within the host country and any implementing partners.

Currently a total of twenty-two countries have collected biomarkers for HIV. This study includes sixteen of those twenty-two countries, representing diverse geographic regions in Africa- West, East and Southern (see Figure 6 for sample summary). Sampling within each of these countries is conducted as a two-stage process. First, enumerator areas (EA) are randomly selected with a probability proportional to the number of households in each EA. Second, urban or non-urban households are selected by systematic sampling. The use of multiple countries allows for comparisons both between and within countries to view the effects of the distribution of economic resources on the spread of HIV at multiple levels of analysis.

*Measures/Instrumentation*. This study uses a multi-level framework to model HIV serostatus linked to individual, regional and national levels of analysis.

*Dependent Variable: HIV Serostatus*. The principle dependent variable for this study, an individual's serostatus, is measured as the test result from the DHS rapid HIV test. Though precise methods of collection may vary slightly from country to country, in all countries, dried blood spots were collected from willing and informed participants to test for HIV using two Enzyme-Linked Immunosorbent Assay (ELISA) tests that would also allow for sero-typing. Both test results for HIV-1 and HIV-2 are available, but the

dependent variable for this study is overall HIV infection, regardless of the strain of the virus.

*Principle Explanatory Variable: Wealth Status and Inequality*. Typically, measures of inequality (such as the gini coefficient and Robin Hood Index) are derived from household surveys that collect information on self-reported household income and expenditure/consumption. However, Demographic and Health Surveys have historically not collected data on income and expenditure. They do, however, include a number of questions about possession of durable goods and utilities that are thought to be associated with health outcomes. From these variables, researchers at MEASURE-DHS developed a measure called the wealth index using principle components analysis to generate a factor score, which represents the composite wealth of a household (Rutstein & Johnson, 2004).

The constructs of individual wealth status and sub-national and national economic wealth distribution for this study will be assessed and constructed using the wealth index. The components of the wealth index are collected as part of the household component of the survey and not asked to each individual. Thus, individuals are coded according to household wealth. The wealth index has been judged to be as reliable and valid a proxy for economic well-being as income or consumption measures (Filmer & Pritchett, 2001). According to Rutstein & Johnson (2004), the use of quintile distribution performs well in comparisons of health outcomes across countries.

To examine inequality in this study, a measure of inequality was constructed using the wealth index at the national and sub-national levels. Because wealth index scores are calculated using principle components analysis, scores take on negative

numbers and have a mean value of zero. Consequently, it is not possible to calculate traditional measures of inequality unless the score is transformed to make all values positive and greater than zero (Mckenzie, 2005; Sahn & Stifel, 2003). As the most often utilized additive technique (adding the lowest negative value to make all scores positive) proposed by Sahn & Stifel (2003) has been demonstrated to have distortionary effects on the underlying distribution, this study utilizes an exponential transformation of the wealth index and finds a high correspondence between the standard deviation of regional wealth index scores and the gini coefficients generated from the exponentiated wealth index (Wai-Poi, 2008). Wealth ginis were calculated for every region within each country. Regions in this analysis represent the primary or secondary administrative units of the sixteen countries under analysis in this study. Ginis were also calculated for urban and rural areas within regions for each country.

Demographic and Health Surveys also include a series of questions on sexual behavior, self-reported STIs, male and female circumcision, and migration, which will be explored to determine mediation and moderation in the relationship between wealth, inequality and HIV infection. Demographic controls include age, education, sex, and marital status.

*Multi-level Modeling & Statistical Analysis*. Multilevel modeling works with the clustered or hierarchical nature of sample data utilizing sampling units at different levels of analysis to model inferences at the individual level (Goldstein, 1999). Data was analyzed using Stata version 10 (StataCorp; College Station, TX, USA). Bivariate relationships between the outcome measure (HIV serostatus) and economic inequality at the regional level were assessed within each of the sixteen countries. All multivariate

models were run as a two-level, hierarchical random coefficient and slope models in Stata adjusted for clustering at the regional level (Rabe-Hesketh, Pickles & Skrondal, 2004).

The analysis proceeded in six steps. First, an ecologic analysis of inequality at the sub-national level examined the relationship between regional economic inequality and HIV prevalence within the 16 countries. Next, a random-intercept model with country fixed effects was fit to examine the contextual effect of economic inequality on HIV infection across sub-national units within countries, controlling for country level variation. To test whether economic inequality has an effect on HIV risk independent of individual wealth, an individual wealth measure was added. A varying slope model was run to model slopes for the individual wealth gradient in order to assess whether there is evidence that the direction of the wealth-gradient varies systematically between rich and poor regions within countries and between urban and rural areas within regions. The final steps in the analysis involved conducting additional tests of mediation and moderation. Given the final specifications for wealth and inequality, the intervening effects of sexual behavior and risk occupations, circumcision and STI infection were modeled.

## Results

*Ecologic Association: Regional Inequality and HIV Prevalence within Countries.* At the ecologic level, in 14 out of 16 countries there is a positive correlation between regional inequality and the HIV prevalence of that region (see Figure 7). Although most of these associations do not reach statistical significance owing to the small number of regions within countries, the association is mainly in the hypothesized direction with the exception of Senegal and Guinea, two low-prevalence countries. The multi-level model allows for more accurate estimation of the average effect of regional inequality taking

advantage of the pooled data to estimate the effect of regional ginis much more powerfully than these ecologic associations with a small-N size. This is in fact the power of the random-intercept model- to estimate the degree to which the contextual effect of inequality matters for individual HIV infection.

*Random-Intercept Model: Contextual Effect of Inequality on HIV Infection*. Results from the two-level random intercept model demonstrated that as hypothesized, wealthier individuals, rather than poorer individuals as commonly thought, are on average at higher risk for HIV infection across regions (OR=1.06) and that the odds of HIV infection are higher in more unequal regions within countries (OR=2.75) controlling for regional fixed effects[5] (see Figure 9). In addition, in the random intercept model, regional inequality remained significant even after accounting for individual wealth, demonstrating a contextual effect of economic inequality on HIV infection over and above the compositional effect of individual wealth status. Specifically, even after controlling for individual wealth, economic inequality increased individual odds of infection by a factor of two. HIV infection, as hypothesized, was also more concentrated in urban areas where both wealth and inequality are systematically higher than in rural areas (see Figure 8). As wealth is highly concentrated in urban areas in SSA, when urban residence was entered into the model, the wealth measure reversed direction, with poorer individuals having an increased odds of infection. This is consistent with other findings and with the hypothesis that in richer (urban) areas, the relatively poor should have higher infection rates than the rich.

[5] Models using both country or regional (Western Eastern, Southern Africa) fixed effects produced similar results. To minimize variables entered in the model, entering regional fixed effects was the preferred method.

*Random-Slope Model: Wealth Gradients within Regions within Countries.* Similarly to the random-intercept model, as hypothesized, results from the random slope model uncovered an ecologic paradox- in poorer regions, wealthy individuals are at greater risk for HIV while in richer regions, poor individuals are at greater risk (see Figure 17). These results held for a majority of countries in the study, though in certain countries, the slopes of HIV-wealth gradient were all positive, and in Ivory Coast they were all negative except for the poorest region. The pattern appears to be that in lower prevalence countries, most of the regional HIV-wealth slopes were positive. Thus, in low-infection countries it appears that the wealthy are still very much driving the epidemic. However, in medium prevalence countries, which tend to be East African countries, in wealthy regions, the poor have higher infection rates. For instance, in Tanzania, the richest regions (Dar es Salam and Kilimanjaro) clearly have the highest HIV infection rates, but in these regions, it is the poor that are more affected than the rich, whereas in the poorest regions, Mtwara and Singida HIV infection is positively sloped in favor of the rich. This may also be the result of the region's longer experience with HIV/AIDS, giving the wealth gradient in these areas time to reverse as wealthier people in wealthy regions have adopted safer sex practices. Southern Africa was more mixed. Zimbabwe clearly fits the hypothesized direction of the slopes with the poor in rich regions (Harare & Bulawayo) having higher HIV infections rates. However, Lesotho shows only faint signs of reversal in its richest districts Berea and Maseru and Malawi's provinces are all positively sloped.

*Mechanisms: Sexual Behavior, Migration and Male Circumcision.* Clearly wealth is not a direct cause of HIV infection. HIV, like all infectious diseases is a communicable

illness and must be contracted through a certain delimited set of transmission pathways such as blood to blood or sexual fluid contact. Wealth, particularly in the context of SSA where IDU is not a major source of the spread of the epidemic, must therefore work through more proximal mechanisms such as patterns of sexual behavior or sexual networks configurations to heighten HIV risk. Yet, past studies have shown that traditional measures of sexual behavior fail to explain cross-national variations in HIV infection (Buve et al, 2001; Boerma et al, 2002), finding support instead for biosocial and economic factors such as the prevalence of male circumcision and various STIs and economic migration. Recent evidence suggests that traditional measures of sexual risk behavior such as overall number of sexual partners has failed to capture the particular risk conferred through being part of a network of concurrent sexual partnerships. Sexual concurrency, both informal and formalized in polygamous unions, has been found to be particularly common in SSA. Network analysis and modeling has shown these types of network patterns amplify risk for HIV infection relative to serial monogamy owing to the infectiousness of individuals in the acute infection phase just after contracting HIV, which allows the virus to spread rapidly through networks even when individuals within the network are themselves monogamous (Epstein, 2008; Thornton, 2008). These network patterns explain how it can be that populations that report the same number of sexual partners can have very different prevalence of HIV depending on the configuration sexual networks and the probability of those networks being exposed to the virus. Unfortunately, sexual behavior questions on the DHS do not specifically ask about whether sexual partners are concurrent. Instead, the principle question that measures the number of sexual partners is the number of partners respondents have had in the last year

or in their lifetime including non-spousal partners, which does not allow researchers to distinguish between sexual partners that were overlapping versus serially monogamous.

Drawing from this growing literature on sexual networks, this study adopts both traditional measures of HIV risk behaviors and also attempts to tap into the construct of sexual networks despite the limitation of the information available using number of non-spousal partners among married individuals in the past year as a proxy for concurrency.[6] In addition, this study introduces the importance of making a distinction between formal versus informal sexual network patterns and by doing so overcomes some of the limitations of the data by comparing the risk conferred by being in a polygamous union (formal marital relationship) versus having multiple non-spousal or extra-marital partners (informal partnerships).

On their own, all measures of sexual behavior were significant and mostly in the anticipated direction in their relationship to HIV infection. Total partners in the past year, number of wives, number of partners other than spouse had sex with in the past year and having paid for sex in the past year were all significantly positively predictive of HIV with and without country fixed effects. Age at first sex without country fixed effects was counterintuitively positively associated with HIV infection, but once country fixed effects were entered, age at first sex became negatively associated with HIV infection (lower age at first sex increases HIV risk). Having had symptoms of an STI in the past year was also positively associated with HIV infection, particularly having had a genital sore or ulcer (see Figure 10). This supports the literature noting the particular increased risk conferred by HSV-1. Counterintuitively, in the univariate analysis having tested for HIV was

[6] Presumably if someone is married and reports non-spousal sexual partners, these partnerships are concurrent.

positively associated with HIV infection as was having used a condom in the past year. However, having been tested for HIV was strongly correlated with wealth as was having used a condom. Those who had ever been tested for HIV were more likely to be infected, but in most countries only about 13% of people had ever been tested for HIV (see Figure 15).

In the fully specified regression model, however, the number of wives, number of other sexual partners in the past year and having paid for sex in the past year were not significantly associated with HIV infection. Having had a symptom of an STI (ulcer/sore) in the past year and having a lower age at first sex were the only variables that maintained significance in the fully specified model. Overall the findings around sexual behavior were weaker than one would expect, a perennial problem with the DHS where women particularly appear to underreport their number of sexual partners (Mishra, 2007). This confirms previous research efforts that have failed to explain significant differences in HIV infection patterns based on behavior (e.g., Buve, 2000; Boerma, 2001). Instead, biosocial factors such as male circumcision and STIs (especially HSV-1) are the strongest predictors of HIV.

One possible source of confounding that would prevent us from drawing meaningful insights from the sexual behavior data is if people who know their status (particularly if positive) have modified their sexual behavior since learning their status. This post-test risk-reduction could weaken the relationship between sexual behavior and HIV risk in a cross-sectional sample such as this one. In order to test for this possibility, I examined sexual behavior amongst those who reported having ever previously been tested for HIV. Hypothesis testing between tested and untested individuals across the 16

countries in the sample revealed that respondents who had been tested for HIV had on average either the same number of sexual partners as untested individuals or in some cases actually had more sexual partners than untested individuals. It is possible that testing negative for HIV may encourage more sexual license since individuals have confirmation of their negative status. However, individuals that have tested for HIV are also wealthier on average than respondents who have not tested and may have more partners on average as a result of their higher wealth (see Figure 11). However, reporting condom use in the past year was also higher amongst HIV positive individuals and wealthier individuals. Thus, respondents who know their status may be taking precautions against transmission through condom use, but not reducing their number of sexual partners. Based on this analysis, it is unlikely that there exists attenuation in the relationship between sexual risk behavior and having tested for HIV.

*Urban-Rural Sexual Behavior Differentials*. Wealth in SSA is highly concentrated in urban areas and the particularly pronounced urban-rural divide in development patterns is well-known to scholars of SSA (Sahn & Stifel, 2003). Because wealth in urban and rural areas may be expressed differently, I hypothesize that in urban areas, richer individuals will have more partners, but fewer wives and in rural areas richer individuals will have more wives, but fewer other partners.[7] Thus, at the national level, polygamy should be associated overall with low wealth and having other non-spousal partners with high wealth.

Comparison of sexual behavior between rural and urban areas within regions revealed that polygamy is indeed more common in rural areas and having another partner

[7] In rural areas, I would expect that having more wives should be a sign of wealth, whereas in urban areas, wealthier individuals may reject formalized polygamy as a "traditional" practice, but informally maintain multiple sexual partnerships or even secondary households.

besides a spouse and a higher total number of partners is more common in urban areas. Age at first sex is slightly higher in urban areas (see Figure 13). The results from a two-level random slope model predicting the number of other partners and polygamy by wealth confirm the hypothesis that at the national level wealth is on average negatively associated with number of wives and positively associated with having other partners across regions (see Figure 14). In the urban-specific model, as hypothesized richer individuals have more other partners but fewer wives. However, contrary to the hypothesized relationship, in rural areas, wealth is positively associated with having other partners but negatively associated with number of wives (richer people have fewer wives).

**Discussion**

The role of complex gender norms in explaining women's disproportionate vulnerability to HIV/AIDS became a popular field of investigation beginning in the late 1990s when researchers realized that unlike the concentrated epidemics in AICs and other parts of the world, SSA's generalized epidemic was resulting in higher infection rates amongst women rather than men. In fact, by 2003, 57% of the PLWHA in SSA were women (UNAIDS, 2006). Figure 6 demonstrates that in all countries except Burkina Faso, women comprise the majority of infections, in some cases being more than twice as likely as men to be infected with the virus. Initially, women's disproportionate vulnerability was explained as resulting from their economic reliance on men, which was argued to make women more open to coercion and place them in a position requiring an exchange of sex for money or housing (WHO, 2000; Gilbert, 1996; Gilbert & Walker, 2002). Variably termed "survival sex," or "sex-for-favors," researchers demonstrated

that many sexual relationships, even where not explicitly a commercial activity, contained a transactional element, placing women at risk for HIV.

However, a number of studies have now begun to question the theoretical and empirical basis for the notion of "survival sex," pointing out that women may engage in transactional sex not just to survive, but also to gain access to material possessions. For instance, Suzanne Leclerc Madlala (2004) identifies the notion of "consumption-sex," arguing that transactional sex occurs along a continuum of needs and wants. Mark Hunter (2002) points to the same phenomena in his essay "the Materiality of Everyday Sex", where he argues that it is the pursuit of modernity that puts women at risk through transactional sex rather than economic privation as such. Unlike the notion of survival sex which points to women's economic dependence or desperation as a source of HIV risk, the "sugar-daddy" phenomena stereotypically paints older men lavishing money and gifts on their younger partners (Luke, 2006). Further, the well-known phenomenon of the "3-C's boyfriend" that provide girls with much coveted "cash, cars and cell phones," have been identified in countries as far flung as Kenya and South Africa (Pisani, 2008).

Similarly, evidence from the collaborative ethnographic Love, Marriage and HIV project, finds that the pursuit of modern identities creates particular patterns of risk, especially in the form of the creation of informal secondary households and multiple concurrent sexual partnering as traditional polygyny has become stigmatized in SSA. For instance in the case of Uganda, Parikh (2007) finds that: "Increased stigma surrounding polygyny and infidelity combined with mobility and migration patterns have facilitated greater secrecy surrounding extramarital relationships and a pattern of informal secondary households." Smith (2007) finds similar though locally contextualized trends

in Nigeria. He observes that in Nigeria, even where Christian discourses exalting mutual monogamy are strong, historical polygyny and changing economic conditions have created "contradictory moralities" for men and women. Both point to the ways that changing economic conditions at least partially underlie what are seemingly ideological transformations. Through these ideological transformations, the aspirations of the burgeoning middle classes to demonstrate their "modernity" with the acquisition of material possessions leads to new sexual opportunity structures that heighten HIV risk.

In a synthesis of this project, Hirsch & Wardlow (2006) find in their book Modern Loves that that changing sexual opportunity structures associated with a global convergence towards a companionate marriage ideal increases women's risk for HIV, at least in the short term. Importantly, they argue that it is not cultural globalization that is causing this convergence in marital ideals, but also changing material structures of power:

> p. 2, "…we locate these ideologies of intimacy in relation to the material and demographic conditions of people's lives, looking at the ways in which the organization of production and consumption enables or impedes various kinds of conjugal ties, as well as the different strategic advantages men and women see (or don't) in their particular local version of companionate marriage."

To put this phenomena in the language of social epidemiology, the movement of people from conditions of absolute poverty to relative poverty is a particularly risky and fraught transition. as it turns out, it's not so much conditions of absolute or absolute deprivation- that poverty weakens peoples immune systems, etc- that makes people most at risk for HIV, but rather rising relative deprivation, especially in the context of urbanization and migration and the incorporation of people into the burgeoning middle classes that make them particularly vulnerable to HIV infection. For men, having multiple sexual partners

is a sign of social status and a performance of their masculinity- richer men are able to have more sexual partners. For women, the increasing materialism and consumerism of the aspiring middle classes makes them particularly prone towards transactional sex, or "consumption" sex. The social and cultural dislocations associated with economic transformations produce changing sexual opportunities structures, and, I argue, increasing informality of sexual concurrency as formalized polygamy is replaced with informal multiple partnerships. It is the rising relative deprivation or inequality that accompanies the marketization of the economy that is driving HIV.

Wojicki in 2005 conducting a systematic review of the literature of poverty and HIV infection in women in Africa and found that:

> "In low-income sub-Saharan African countries, where poverty is widespread, increasing access to resources for women may initially increase risk of HIV or have no effect on risk-taking behaviours. In some parts of Southern Africa where per capita income is higher and within-country inequalities in wealth are greater, studies suggest that increasing SES may decrease risk."

Here, Wojicki is emphasizing how the slopes in risk may change depending on the level of development of a country. Risk among wealthy women may be less in more economically developed settings, likely because these settings resemble advanced industrial countries, where women's basic needs are met and relative poverty has displaced absolute poverty. However, in very poor countries, where the majority of the population lives in conditions of absolute poverty, poorer women may experience less HIV risk than richer women.

It is not clear that the results of this study bear out Wojicki's hypothesis at least looking *between* countries. In both the lower income African countries and the relatively more prosperous Southern African countries, the rich seem to have higher infection rates

when looked at across the country sample (refer to Figure 17). However, *within* countries, this relationship is borne out in certain countries where in richer regions increasing SES is protective compared with poorer regions and it does appear that in poorer West African countries, there is little reversal in the wealth gradients in HIV infection- the rich are more affected in all regions, rich and poor, unlike East and Southern Africa where there are more signs of an inverse wealth-gradient. In the case of the female wealth gradient in infection, this phenomenon may also an issue of selection. Rich women may appear rich precisely because of their success at gaining access to goods through their partners.

So how do we understand these findings? It appears that the ongoing integration into market economies and burgeoning middle classes in developing countries generates risk for HIV. Booyson (2002), for instance, shows an increase in the presence of consumer goods (e.g., refrigerators, radios, telephones) since the 80s, but a reduction in the access to public goods (e.g., piped water, flush toilets). The resultant relative deprivation from the increasing availability of consumer goods vis-à-vis basic needs, may serve as a mediator between changing economic conditions and HIV risk. For instance, Ingelhart (1997), drawing on Maslow's theory of a hierarchy of needs notes that only after a certain level of wealth is obtained in a society can individuals begin thinking about issues other than their immediate material well-being, a state which he refers to as "post-materialism". Pointing the generation in AICs that lived through the Great Depression, prior to the era of widespread social protections, he notes that this generation was overwhelmingly concerned with economic issues. On the other hand, the generation that has grown up in post-materialist, decommodified labor markets, has begun to express concerns apart from economics about the environment and civil rights, for instance.

Relating this theory to forces influencing HIV risk, the increasing consumerism amongst individuals tied into market economies appears to encourage them to place their immediate material and consumption-based concerns above the calculated risk of being infected with a deadly disease.

The relationship between economic transitions and the diffusion of HIV is starker in countries where these transitions have been more rapid and abrupt than those that have occurred in SSA. For instance, it is easy to identify the mechanisms that caused HIV infection rates to surge upwards in the former USSR via the economic shock therapy applied during the overnight transformation from a centrally planned to a free market economy. As increasingly porous borders allowed cheap heroin to flood into the country from Afghanistan at a particularly vulnerable, anomic moment and social protections for women were eviscerated forcing many women to fall back on the "oldest profession in history" (sex work) to compensate for their increasingly re-commodified position in society, HIV infection rates soured upwards. In Asia, HIV risk appears to also be driven by economic transformation as increasing industrialization and economic prosperity allowed Asian men "possibly for the first time in their lives… [to] do what more well of [Asian] men have always done: go to a brothel" (Bales, 2004: 195). In SSA, these processes may be more subtle, occurring over a longer time horizon.

These findings raise a couple of important points. Fist, the increasing informality and fluidity of relationships may be what is particularly dangerous- both in terms of sex work and informal households. With regards to sex work, formalized sex work may be less of a risk factor for HIV than informal, yet nonetheless transactional relationships. One reason that HIV has been able to proliferate so rapidly in SSA despite the absence of

a high degree of formal sex work that comprises a major source of risk in other parts of the world are the presence of these informal yet transactional relationships, which are much more diffuse and difficult to identify and intervene with than formal sex work. Similarly, the proliferation of informal secondary households in lieu of formalized polygyny appear to be more risky than polygynous relationships as women in the absence of a formal marital bond may have more freedom to have additional partners. This observation points to a second issue, which is that the HIV community may need to start accepting the idea that it is not predominately men that are infecting women but often the reverse. Demographically, women are infected two-to-one in most African countries, so clearly serodiscordance will be titled in their favor, but also recent evidence from the DHS suggests that it is women that are bringing HIV into the relationship, even in ongoing relationships (Mishra, 2007).

In addition, another possible implication of this study is that poverty is not the problem, but development. Initially, the implication of the HIV-Poverty Thesis was that in order to address HIV it is necessary to reduce poverty as an indirect mechanism that is fueling the epidemic. This reasoning has been used as a justification for poverty reduction in SSA. However, the results of this study could raise the somewhat ridiculous question of whether poverty should actually be encouraged since it appears to be protective against HIV. Clearly this is not a recommendation I wish to make, but it does beg the question that if economic development is something one wants to encourage in developing countries, how can this be done in a manner that does not actually increase risk for HIV. I think that this is possible- growth that is inequality reducing rather than inequality-inducing can minimize the social dislocations that increase risk for HIV.

## Conclusions

Despite the vast popular and rhetorical attention that has been given the role that poverty plays as an underlying cause of HIV in SSA, this research argues that the HIV-poverty thesis essentially got it wrong. It is not so much conditions of absolute poverty or absolute deprivation- i.e., that poverty weakens peoples immune systems or makes them vulnerable in other ways- that places people at risk for contracting HIV, but rather rising relative deprivation, especially in the context of urbanization and migration and the incorporation of individuals into the burgeoning middle classes that make them particularly vulnerable to HIV infection. This research finds that economic inequality acts as a contextual driver of HIV infection. In more unequal regions within countries, and more unequal countries in SSA, HIV infection rates are higher than more equal areas, even controlling for the individual wealth composition of the region. The effect of economic inequality appears to work through an increasing deregulation of "traditional" social norms surrounding sexuality in more unequal regions as increasing incorporation into market-based economies promotes consumerism and the potential for increasing informality in sexual network patterns that have been shown to have a particularly profound affect on HIV risk.

It is important here to note some limitations and caveats to this study. Since the data presented here is cross-sectional, it is only possible to speak about the affect of inequality on HIV risk at a given moment in time. Also, this study has only dealt with the upstream relationship between wealth and initial infection with HIV and has not dealt with the downstream effect of HIV infection on wealth. While wealthier individuals may become infected with HIV at higher rates, subsequently their infection may result in a loss of

household wealth, and poorer individuals that become infected with HIV will likely progress to AIDS and expire at a faster pace than the wealthy. Notwithstanding these limitations, I believe that these findings contribute an important new approach to understanding the mechanisms underlying varying trends in HIV infection between and within countries in SSA and globally.

# APPENDIX

**Figure 5: Theoretical Model: Economic Development and HIV Risk**

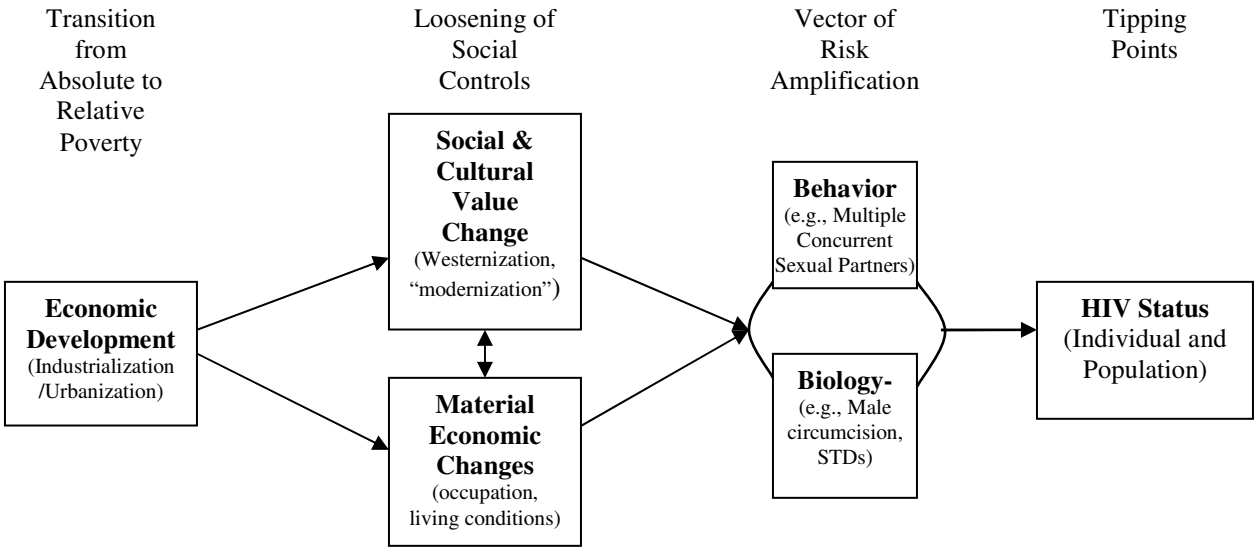



**Figure 6: Country Sample and HIV Prevalence**

| Country | Prevalence | Tested Sample | Sex Ratio | Year of Survey | Refusal Rate |
|---|---|---|---|---|---|
| **Low Prevalence (<3%)** | | | | | |
| Senegal | 0.7 (0.4, 1.0) | 7,823 | 2.3 | 2005 | 12.5% |
| Niger | 0.7 (0.5, 0.9) | 7,283 | 1 | 2006 | 4.0% |
| Ethiopia | 1.4 (1.2- 1.6) | 11,383 | 2.1 | 2005 | 15.2% |
| Guinea | 1.6 (1.2-1.9) | 6,912 | 2.1 | 2005 | 6.6% |
| Mali | 1.7 (1.5- 1.9) | 8,629 | 1.5 | 2006 | 12% |
| Burkina Faso | 1.8 (1.6- 2.2) | 7,790 | 0.9 | 2003 | 5.4% |
| Ghana | 2.2 (1.8-2.4) | 9,779 | 1.8 | 2003 | 8.1% |
| **Medium Prevalence (3%-10%)** | | | | | |
| Rwanda | 3.0 (2.9-3.5) | 10,592 | 1.6 | 2005 | 1.5% |
| Ivory Coast | 4.7 (4.5- 5.4) | 8,570 | 2.2 | 2005 | 11% |
| Cameroon | 5.5 (5.0- 6.0) | 10,682 | 1.7 | 2004 | 5.5% |
| Kenya | 6.8 (6.0-6.9) | 6,360 | 1.8 | 2003 | 14% |
| Tanzania | 7.0 (5.9-7.2) | 10,957 | 1.2 | 2003 | 13% |
| **High Prevalence (10%+)** | | | | | |
| Malawi | 11.7 (10.7-12.7) | 5,357 | 1.3 | 2004 | 22.2% |
| Zambia | 15.6 (13.4-17.3) | 3,949 | 1.4 | 2001/2 | 15.3% |
| Zimbabwe | 18.1 (16.9-19.3) | 13,069 | 1.5 | 2005/6 | 15.2% |
| Lesotho | 23.2 (21.7-24.5) | 5,364 | 1.1 | 2004 | 14.0% |
| Swaziland | 25.9 (25.2-27.1) | 8,187 | 1.6 | 2006/7 | 12.8% |

**Figure 7: Ecologic Association- Regional Inequality by HIV Prevalence, 17 Countries**

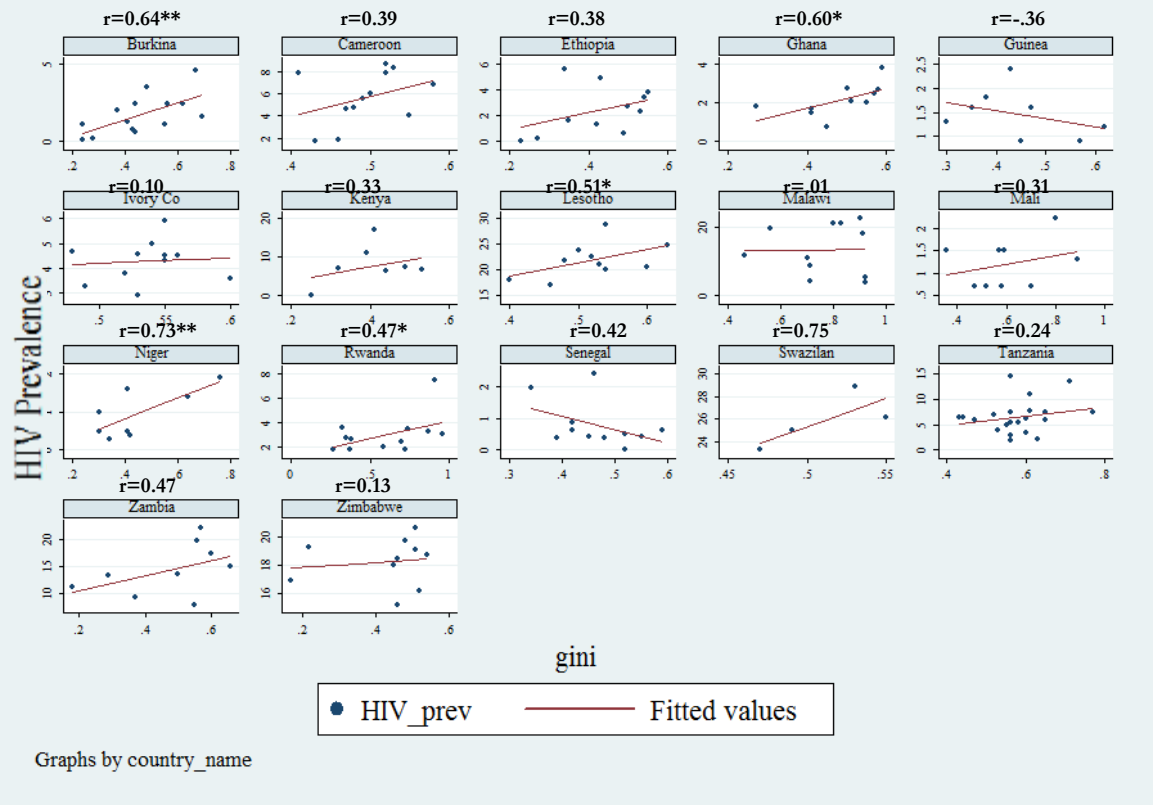



**Figure 8: Ecologic Association- Regional Inequality by HIV Prevalence in Urban vs Rural Areas of Regions, 16 Countries**

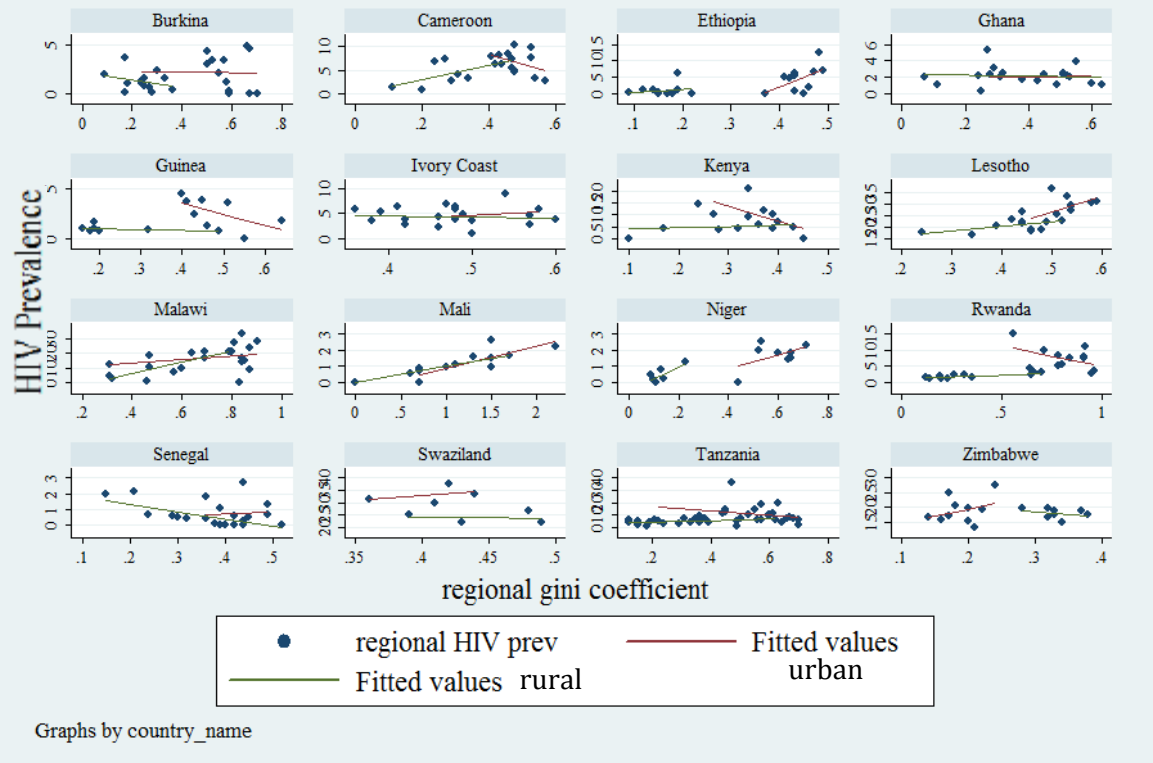


**Figure 9: Inequality, Individual Wealth, Urbanicity and HIV: Results from Random Intercept and Random Slope Models with and without Covariates**

| | Model 1 (Within-country Inequality w/ regional fixed effects) | Model 2 (Within-country Inequality & Individual Wealth w/ regional fixed effects) | Model 3 (Regional Inequality, Individual Wealth & Urban w/ regional fixed effects) |
|---|---|---|---|
| *Level 2 Indicator* | | | |
| Regional Gini Coefficient | 2.87*** (.253) | 2.75*** (.253) | 2.21*** (.254) |
| *Level 1 Indicators* | | | |
| Individual Wealth | - | 1.06*** (.014) | .914*** (.018) |
| Urban | - | - | 1.86*** (.037) |
| *Regional Fixed Effects* | | | |
| West Africa | .298*** (.115) | .309*** (.119) | .292*** (.119) |
| East Africa (referent) | - | - | - |
| Southern Africa | 7.14*** (.176) | 7.47*** (.177) | 7.47*** (.177) |
| log likelihood | -22578.326 | -20460.088 | -20322.271 |
| Wald chi2(2) | 347.95*** | 367.82*** | 651.62*** |

Level 1 units: 120,519
Level 2 units: 148
Obs per group avg: 814.3
† Only coefficients reported for Random Intercept Model

**Figure 10: Univariate Random Slope Models**

| | **(Univariate, w/country fixed effects)** |
|---|---|
| ***Sexual Behavior*** | |
| Total Partners Past Year (including spouse) | 1.013*** (.002.) |
| Number of Wives | 1.003*** (.001) |
| Polygamous union | 1.11*** (.034) |
| # other partners had sex w/ past year (besides spouse) | 1.01*** (.002) |
| Age first sex (amongst those reporting ever having had sex) | 0.995*** (.0004) |
| Paid Sex last 12 months | 1.10*** (.039) |
| Last intercourse used condom | 1.13*** (.021) |
| ***Demographic*** | |
| Age | 1.03*** (.001) |
| Sex (Male) | .628*** (.023) |
| Education | 1.01* (.003) |
| Ever Tested for HIV | 2.03*** (.027) |
| Married | 1.03 (018) |
| Works away from home | 1.09** (.043) |
| ***Biosocial*** | |
| Male Circumcision | 1.15*** (.05) |
| Female Circumcision | .912* (.051) |
| Self-reported symptom of an STI (any) | 2.89*** (.052) |
| Self-reported symptom of an STI (sores) | 3.33*** (.044) |
| Self-reported symptom of an STI (discharge) | 2.32*** (.041) |
| Injections (only Zimbabwe, Kenya, Ivory Coast, Mali) | 1.02*** (.004) |

**Figure 11: Comparison of Means: Sexual Behavior of Respondents Tested and Untested for HIV**

| | HIV Tested (mean) | HIV Untested | % Tested HIV | Received Results |
|---|---|---|---|---|
| **Total partners past year (mean)** | 1.1*** | .93*** | | |
| **Senegal** | 1.67*** | .90*** | 3.9% | 90.8% |
| **Niger** | 1.05 | 1.49 | 5.1% | 84.8% |
| **Mali** | 2.13*** | 1.25*** | No data | 90.8% |
| **Guinea** | 1.23 | .98 | 4.2% | 89.9% |
| **Burkina Faso** | 1.23 | .99 | 7.4% | 83.5% |
| **Ghana** | .89 | .78 | 9.6% | 78.2% |
| **Ivory Coast** | 1.21 | 1.23 | 7.7% | 86.2% |
| **Ethiopia** | .74 | .74 | 4.5% | 92.8% |
| **Rwanda** | .74*** | .50*** | 22.3% | 94.7% |
| **Cameroon** | 1.67** | 1.36** | 18.3% | 89.6% |
| **Tanzania** | 1.04 | .97 | 15.5% | 86.3% |
| **Kenya** | .96 | .92 | 15.3% | 90.6% |
| **Malawi** | .92 | .92 | 16.2% | 91.6% |
| **Zimbabwe** | .98*** | .71*** | 22.6% | 85.9% |
| **Lesotho** | .95*** | .78*** | 13.2% | 83.0% |
| **Swaziland** | 1.22*** | .77*** | 30.2% | 89.9% |
| **Wealth (mean)** | .12*** | .09*** | | |

**Figure 12: Multivariate Random Intercept Models with Covariates**

| | Model 1: Multivariate, with regional fixed effects (OR, SE in parentheses) |
|---|---|
| *Level 2 Indicator* | |
| Regional Gini Coefficient | 2.16* (.531) |
| *Level 1 Indicators* | |
| Individual Wealth | .98 (.049) |
| Urban | 1.48*** (.105) |
| *Sexual Behavior* | |
| Polygamous Union | .79* (.155) |
| Had sex w/ other partners past year (besides spouse) | 1.08 (.202) |
| Age 1st Sex (ever had sex) | .96*** (.011) |
| Last Intercourse Used Condom | 1.26*** (.067) |
| *Biosocial* | |
| Male Circumcision | .68*** (.111) |
| Self-reported symptom of an STI (sores) | 2.66*** (.166) |
| Ever Tested for HIV | 1.30*** (.092) |
| *Regional Fixed Effects* | |
| West Africa | .463*** (.185) |
| East Africa (referent) | |
| Southern Africa | 9.02*** (.272) |
| log likelihood | -2607.3 |
| Wald chi2(2) | 270.34 |

Level 1 units: 13,462
Level 2 units: 99

**Figure 13: Urban versus Rural Sexual Behavior Comparison of Means: Urban vs Rural Sexual Behavior**

| | Urban | Rural |
|---|---|---|
| **Total partners past year (mean)** | **1.02***** | .93*** |
| **# of other partners past year (mean)** | **.57***** | .33*** |
| **# of wives (mean)** | .76*** | **1.11***** |
| **Age First Sex (mean)** | **17.81***** | 17.45*** |

**Figure 14: Random Slope Models of Other Partners and Number of Wives by Individual Wealth, national and urban-rural specific models w/ regional fixed effects**

| | National<br>Coef , SE | Urban-Specific<br>Coef , SE | Rural-Specific<br>Coef, SE |
|---|---|---|---|
| **Wealth coefficient (other partners)** | .030, .003*** | .022, .007*** | .041, .008*** |
| **Wealth coefficient (number of wives)** | -.037, .004*** | -.058, .008*** | -.067, .012*** |
| Number of obs | 111,690 | 32,424 | 76,037 |
| Number of groups | 136 | 134 | 129 |
| Obs per group: min | 188 | 26 | 49 |
| avg | 821.3 | 242 | 589.5 |
| max | 2564 | 1161 | 2205 |
| Wald chi2(1) | 69.37 | 31.97 | 49.63 |
| Prob > chi2 | 0.0000 | 0.0000 | 0.0000 |

**Figure 15: Comparative Sexual Behavior Across 16 Countries***

| | | Never had Sex | Total Lifetime Sexual Partners (mean, sexually active) | Had sex w/ partners other than husband/wife past year | % Polygamous Union (2+ wives) | # of Wives (mean) | Mean Age 1st sex (of those who have ever had sex) | % Genital Sore past 12 months | Paid Sex (% ever, men) |
|---|---|---|---|---|---|---|---|---|---|
| West Africa | **Senegal** | 14.5% | 1.31 | 13.4% | 62.9% | 2.19 | 17.9 | 2.1% | 1.4% |
| | **Niger** | 15.6% | 2.21 | 3.4% | 58.9% | 1.94 | 18.0 | 1.9% | 1.6% |
| | **Mali** | 10.4% | 3.09 | 11.1% | 42.3% | 1.48 | 15.8 | 4.8% | 1.9% |
| | **Guinea** | 19.5% | 1.47 | 21.6% | 70.5% | 2.83 | 17.4 | 3.9% | 1.0% |
| | **Burkina Faso** | 10.4% | 1.35 | 14.1% | 68.9% | 2.49 | 18.2 | 1.5% | No Data |
| | **Ghana** | 12.2% | 1.59 | 18.0% | 55.7% | 1.72 | 18.2 | 1.9% | 8.1% |
| | **Ivory Coast** | 18.3% | 2.84 | 44.1% | 19.6% | 1.27 | 16.6 | 4.2% | 3.1% |
| East/Cent Africa | **Ethiopia** | 29.8% | 1.42 | 3.4% | 52.5% | 2.05 | 17.9 | .5% | .8% |
| | **Rwanda** | 10.8% | 1.07 | 6.3% | 53.9% | 1.96 | 18.8 | 1.6% | 1.0% |
| | **Cameroon** | 28.2% | No Data | 33.7% | 31.4% | 1.47 | 15.9 | 2.5% | 27.0% |
| Southern Africa | **Tanzania** | 16.7% | No Data | 26.1% | 10.0% | 1.12 | 17.3 | 2.4% | 1.5%* |
| | **Kenya** | 22.9% | 1.38 | 19.9% | 54.7% | 1.98 | 16.5 | 1.6% | 15.6% |
| | **Malawi** | 24.1% | 1.16 | 12.7% | 56.1% | 2.08 | 17.0 | 4.9% | 21.2% |
| | **Zimbabwe** | 15.7% | No Data | 15.3% | 48.9% | 1.51 | 18.4 | 4.1% | 3.5% |
| | **Lesotho** | 9.8% | 1.05 | 25.9% | No Data | 3.07 | 18.1 | 5.9% | 5.7% |
| | **Swaziland** | 28.4% | 3.09 | 32.8% | 53.9% | 1.62 | 17.9 | 6.4% | No Data |

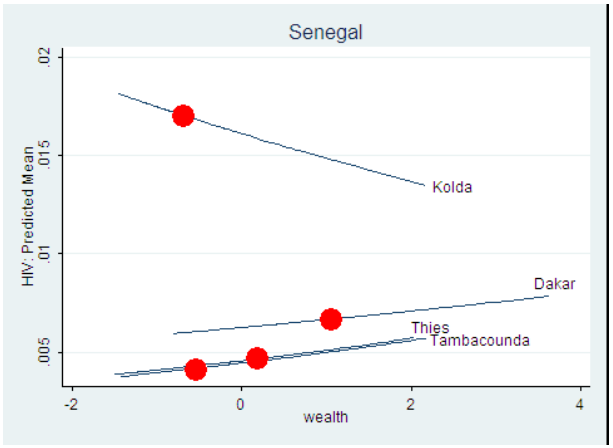
* weighted estimates

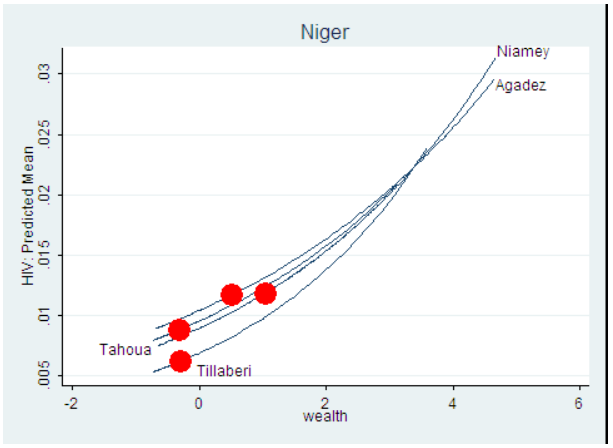
**Figure 16: Hypothetical Country, Odds of HIV Infection by Wealth in Rich and Poor Regions within Countries- Expected Random Slopes and Intercepts**

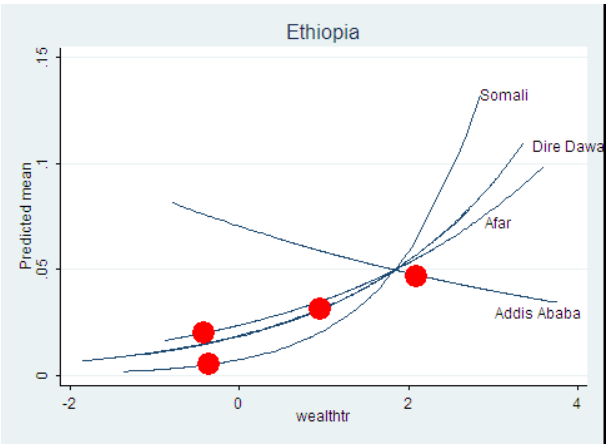

*Notes: "Ideal type" graph of regional wealth gradients in HIV infection for a hypothetical country showing that, theoretically, richer regions (higher mean wealth) should have higher HIV infection rates than poorer regions (random intercepts), and that the slopes of the wealth-gradient in HIV infection should be positive for poorer regions and negative for richer regions (random slopes). In other words, in richer regions the relatively poor should have higher HIV infection rates and poorer regions the relatively rich should have higher infection rates. The gradients should further be steeper or shallower depending on the wealth of the region.*

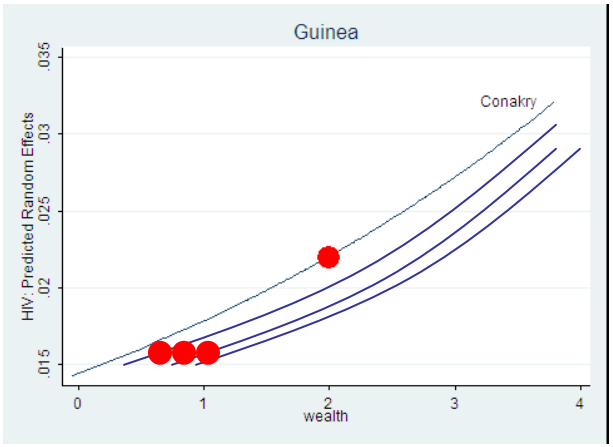
**Figure 17: Post-estimation from Country-level Random Slope Models. Regional-HIV Wealth Gradient by Regional Wealth**

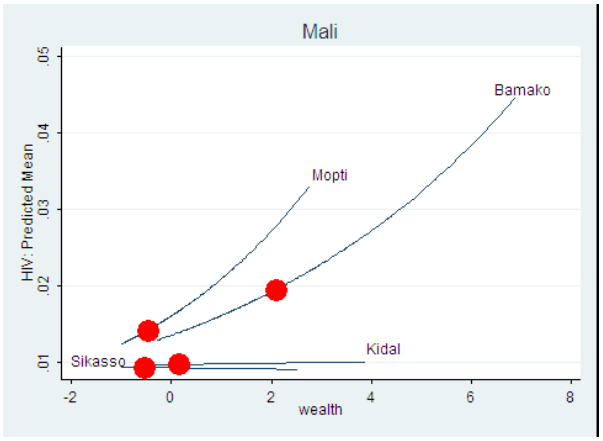
Low Prevalence/ Sahelian West Africa & The Horn

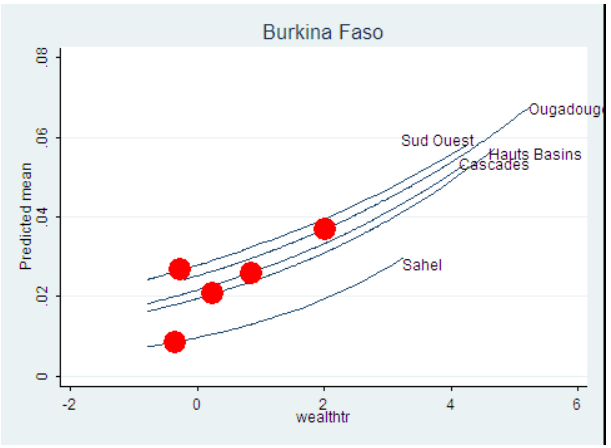


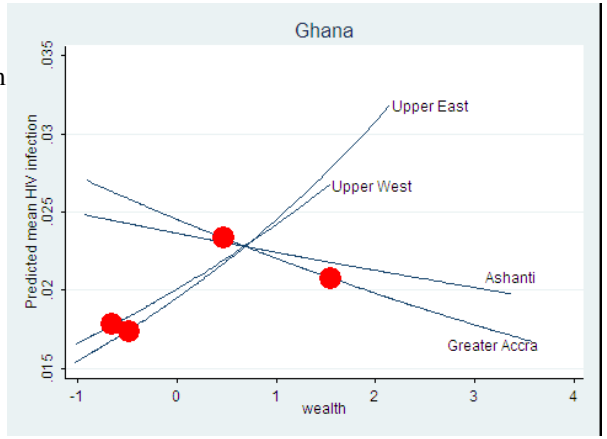



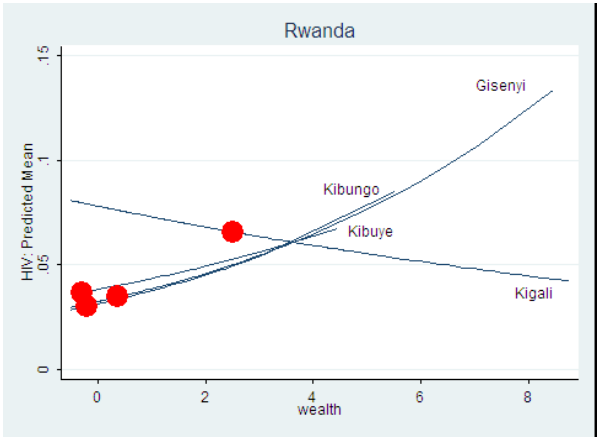



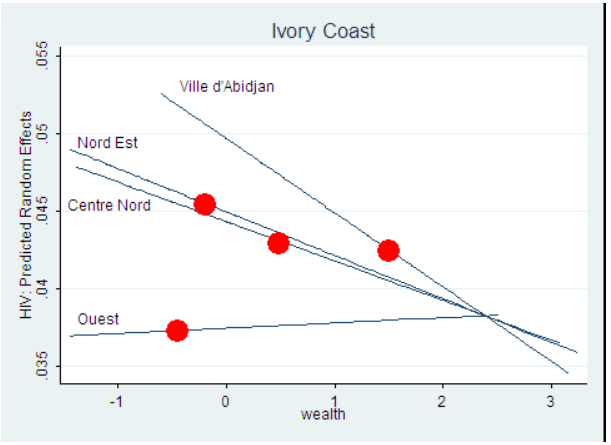



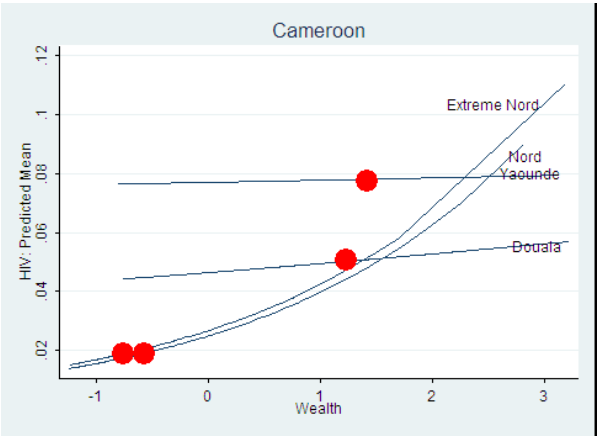

Low/Medium Prevalence/ Lower West & East Africa

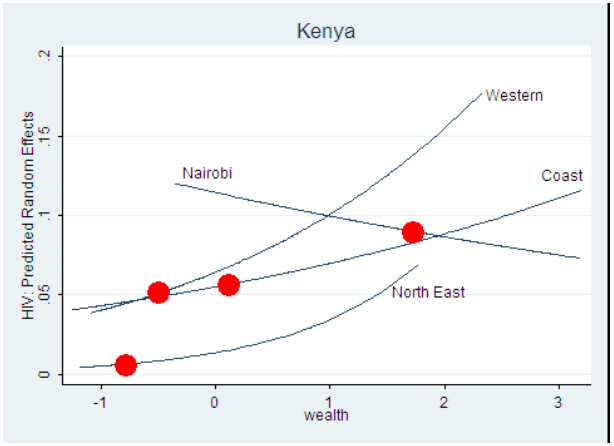


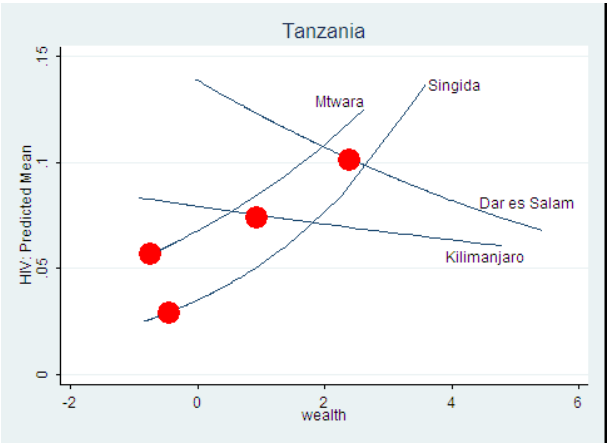



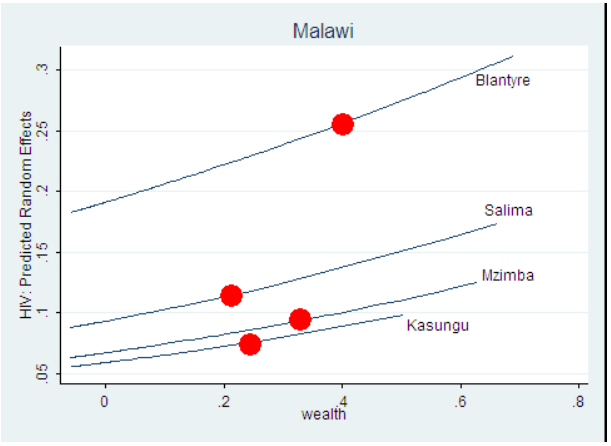
Medium Prevalence/ Lower West & East Africa

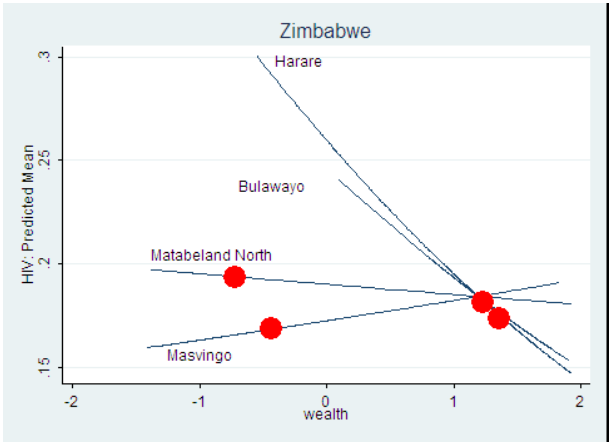


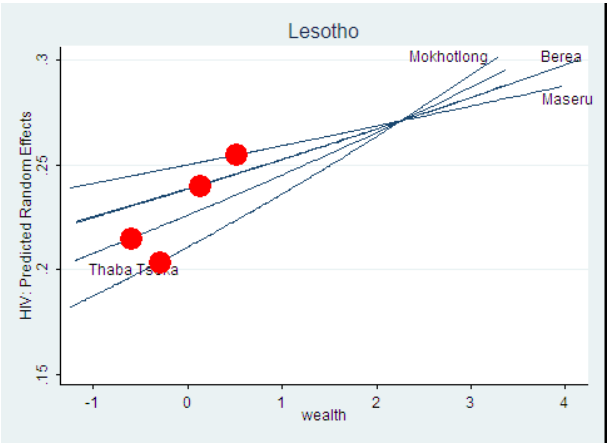




High Prevalence/ Southern Africa




*Notes: Swaziland is missing from this analysis as a result of its small regional N-size. In eight out of the fifteen countries in this analysis there is evidence of an ecologic paradox with the poor experiencing higher odds of infection in rich regions and the rich experiencing higher odds of infection in poor regions. These results appear to mainly hold in medium prevalence, particularly in East Africa, and some high prevalence countries. In most countries, as predicted, richer regions have higher HIV infection rates than poorer regions and inverse wealth-slopes are steeper or shallower depending on the mean wealth of the region.*

● = Regional Mean Wealth

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