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An association between neighborhood wealth inequality and HIV prevalence in sub-Saharan Africa

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Summary

This paper investigates whether community-level wealth inequality predicts HIV serostatus, using DHS household survey and HIV biomarker data for men and women ages 15-59 pooled from six sub-Saharan African countries with HIV prevalence rates exceeding five percent. The analysis relates the binary dependent variable HIV positive serostatus and two weighted aggregate predictors generated from the DHS Wealth Index: the Gini coefficient, and the ratio of the wealth of households in the top 20% wealth quintile to that of those in the bottom 20%. In separate multilevel logistic regression models, wealth inequality is used to predict HIV prevalence within each SEA, controlling for known individual-level demographic predictors of HIV serostatus. Potential individual-level sexual behavior mediating variables are added to assess attenuation, and ordered logit models investigate whether the effect is mediated through extramarital sexual partnerships. Both the cluster-level wealth Gini coefficient and wealth ratio significantly predict positive HIV serostatus: a 1 point increase in the cluster-level Gini coefficient and in the cluster-level wealth ratio is associated with a 2.35 and 1.3 times increased likelihood of being HIV positive, respectively, controlling for individual-level demographic predictors, and associations are stronger in models including only males. Adding sexual behavior variables attenuates the effects of both inequality measures. Reporting 11 plus lifetime sexual partners increases the odds of being HIV positive over five-fold. The likelihood of having more extramarital partners is significantly higher in clusters with greater wealth inequality measured by the wealth ratio. Disaggregating logit models by sex indicates important risk behavior differences. Household wealth inequality within DHS clusters predicts HIV serostatus, and the relationship is partially mediated by more extramarital partners. These results emphasize the importance of incorporating higher-level contextual factors, investigating behavioral mediators, and disaggregating by sex in assessing HIV risk in order to uncover potential mechanisms of action and points of preventive intervention

Introduction

The prevailing explanation for extraordinarily high HIV prevalence rates in parts of sub-Saharan Africa (SSA) employs a behavioral paradigm and emphasizes the high rate of concurrent sexual partnerships, although there are strongly opposing viewpoints in the

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literature regarding the role of the latter (Epstein, 2010; Epstein and Morris, 2011; Lurie and Rosenthal, 2010a; Lurie and Rosenthal, 2010b; Mah and Halperin, 2010a; Mah and Halperin, 2010b; Mah and Shelton, 2011; Morris, 2010; Sawers and Stillwaggon, 2010). Donor countries and international aid agencies have expended enormous effort to try to alter individual sexual behaviors, and only relatively recently has sexual concurrency *per se* been seriously addressed. Throughout the long history of the regional pandemic both donor and recipient countries have largely neglected the contexts and structural drivers of individual sexual behaviors—some have suggested, for political reasons (Hunsmann, 2009). As Paul Farmer notes in *Partner to the Poor*, "the failure to contemplate social and economic aspects of epidemics stunts our understanding of them," making it much more difficult to contain and defeat them (Farmer, 2010; Rosen, 2012).

Although behaviorally-focused prevention appears to have produced recent reductions in HIV incidence rates in the region (Joint United Nations Programme on HIV/AIDS, 2010), it is unclear which interventions have been most effective, nor to what extent. There is clearly a need to better understand the nature and role of network factors such as long-term sexual concurrency, which has been inadequately captured and underreported in sexual behavior surveys, and is at least partially structural in nature because it involves deeply entrenched social and cultural norms (Epstein and Morris, 2011). The heavy toll of the ongoing HIV pandemic in SSA has prompted renewed attention to the social and economic upstream contextual or structural factors, sometimes termed "the causes of the causes" of disease, which may facilitate viral transmission and undermine intervention effectiveness (Commission on Social Determinants of Health, 2008; Gupta *et al.*, 2008). In a recent supplement to the *Journal of the International AIDS Society* devoted entirely to structural drivers of HIV transmission, Seeley et al. (2012) note elimination of HIV will require "a comprehensive HIV response, that includes meaningful responses to the social, political, economic and environmental factors that affect HIV risk and vulnerability" (Seeley *et al.*, 2012).

Also, a prevailing view emphasizes the role of poverty in the spread of HIV, despite numerous studies demonstrating an inverse relationship between HIV serostatus and poverty status in SSA, which is opposite to the case in the developed world and contrary to common expectations about disease susceptibility and poverty status (Gillespie, Kadiyala and Greener, 2007; Mishra *et al.*, 2007; Parkhurst, 2010; Shelton, Cassell and Adetunji, 2005). Commenting in *the Lancet*, Shelton et al. (2005) suggested that both wealth and economic disadvantage may play pivotal roles in HIV transmission through sexual concurrency networks, with wealth being "associated with the mobility, time, and resources to maintain concurrent partnerships" and where women "might improve their economic situation by having more than one concurrent partner" (Shelton, Cassell and Adetunji, 2005) p 1058. Several investigators have attempted to help resolve the ongoing controversy about the relative importance of poverty, inequality, and sexual concurrency in explaining the severity of the SSA pandemic. For example, a review by Shandera (2007) identified several viral, host, transmission, and societal factors that might explain the higher rates of infection in the region (Shandera, 2007). A country-level empirical study by Nattrass (2009) identified a number of social factors associated with HIV prevalence rates, finding little effect of poverty but large and significant effects of the predominant religious affiliation of the country

(Nattrass, 2009). Within SSA countries, HIV prevalence rates are generally higher in urban compared to rural areas, but there is also much regional variation, with some poorer, rural areas, such as the Nyanza region of Kenya, having very high prevalence rates. Nattrass et al. (2012) provides an excellent review of the recent literature on the complex interrelationships among poverty, sexual behavior, and HIV in SSA and the methodological challenges inherent in studies attempting to shed light on them. The authors use a panel dataset on young men in Cape Town, South Africa to overcome problems of endogeneity and blunt indicator measurements of sexual behavior, finding important differences by sex (Nattrass *et al.*, 2012).

A review by Fox (2010) identified a positive association between HIV prevalence at the country level and the Gini coefficient (a standard measure of economic inequality) among SSA countries (Fox, 2010). These findings suggested a potential association between HIV prevalence and rapid economic development affecting primarily the urban regions of poor developing countries and reflected in rising wealth inequalities, such that it is not poverty or wealth *per se*, but the level of inequality in a region that predicts HIV prevalence. However, cross-country aggregate-level comparisons are prone to problems such as ecologic fallacy or aggregation bias, and to omitted variable bias from the inability to control for many potentially important explanatory factors. Also, if absolute income (or wealth) affects health and there are diminishing returns to health, then a relationship between health and income is produced at the aggregate level in the absence of a direct effect of economic inequality—the so-called absolute income effect (Gravelle, Wildman and Sutton, 2002; Kawachi, 2011).

In contrast, the income inequality hypothesis argues that income inequality is an indicator of “social distance” and that greater distance causally leads to greater psychosocial stress and poorer health outcomes (Wilkinson and Pickett, 2009; Wilkinson, 2005; Wilkinson and Pickett, 2006). In the field of economics, this concept implies that “utility” from consumption depends on comparison of one's own income and consumption to that of others, a concept that has gained recent empirical support in behavioral economics (Fliessbach *et al.*, 2007; Luttmer, 2005). Yet a third “society-wide effects” hypothesis argues that the effects of inequality are related to social capital, trust and social cohesion, with increasing inequality causing reduced cohesion and increased crime and violence (Leigh, Jencks and Smeeding, 2009). Social heterogeneity, or a social context of varying and potentially competing population preferences and needs, has been linked to the under provision of public goods (Banerjee and Somanathan, 2007).

Using Demographic and Health Surveys (DHS) data from 170 regions across 16 countries, Fox (2012) extended her earlier work by employing multilevel modeling techniques to control for regional-level absolute wealth and a number of individual-level HIV risk factors and establishing an independent association between regional-level wealth inequality and HIV prevalence (Fox, 2012). It has also been noted that the geographic level of the community studied might affect the results of an evaluation of the association between wealth inequality and health outcomes, with support in the literature of a general pattern that the smaller the community, the less likely it is that you will observe the association (Wilkinson and Pickett, 2006). Nearly all studies of HIV/AIDS and wealth inequality have been cross-country or regional comparisons. However, one recent study examined two

regions (districts and DHS sampling clusters) simultaneously within one country, Malawi, using a multilevel framework (Durevall and Lindskog, 2012). Specifically, the authors evaluated the effect of district-level consumption inequality and cluster-level (neighborhood) wealth inequality on risk of HIV infection in Malawi women aged 15-24, finding a strong positive association between risk of HIV infection and inequality at both geographic levels, but no association for individual poverty.

The current study builds on these prior efforts by empirically investigating the relationship between wealth inequality at the statistical enumeration area (SEA) or cluster level within multiple countries in southeastern SSA using the most recent DHS data on HIV prevalence and several socio-economic and demographic factors. The advantages of this study are that all data within a given country are from the same survey; the number of data points is much larger than previous country-level studies; it examines the inequality-HIV association at a lower level of aggregation (i.e., at the cluster level, compared to the regional or district level) where it has been harder to detect; and two different measures of SEA wealth inequality are utilized as an internal validation of the key independent variable.

Conceptual model

This paper uses ecological systems theory applied to health (or the social ecological model of health). It views individual health status as determined by a broad array of factors operating at multiple levels, often termed macro-, exo-, meso-, and micro-, which describe influences as intercultural, community, organizational, and interpersonal or individual, and has been adopted by World Health Organization's Commission on the Social Determinants of Health. While this conceptual model applies to general health status, it is utilized here to examine potential influences on specific disease susceptibility. In the developing world the major threats to population health are infectious disease vulnerability and transmission. HIV is the leading cause of adult mortality in southern SSA and has been responsible for reversing a long-term trend of decreasing mortality rates there. Adult mortality (or, conversely, life expectancy) is a key indicator of population health and directly reflects the general health status of the population.

As a more direct mechanism of action, researchers theorize that rapid economic development is associated with rising wealth inequality and reduced social cohesion, leading to the breakdown of traditional family structures. For instance, new opportunities in urban regions may prompt economic migration by male or female household members. They, and those left behind in rural regions, may then take on informal, long-term partners, leading to higher prevalence of HIV in more unequal settings (Fox, 2012). Durevall et al. (2012) note several specific potential links between structural inequality and risk behaviors, particularly transactional sex providing young women and their families the means to remain above subsistence or to improve their economic status (Durevall and Lindskog, 2012). This paper investigates whether HIV prevalence rates are in part determined by such wealth inequities, which reflect differences in social position and levels of social cohesion within a given geographic region (in this case the DHS SEA or cluster), controlling for individual/household wealth and other key individual-level variables.

Methods

Data and sample

I conducted a pooled analysis using DHS household survey data collected since 2006 from six SSA countries with HIV prevalence rates exceeding five percent and HIV biomarker data and data on all covariates. The UNAIDS program classifies a national prevalence rate higher than 1% as a generalized epidemic (Joint United Nations Program on AIDS, 2011). Data are downloadable from the MEASURE DHS website at http://www.measuredhs.com/data/available-datasets.cfm. The six countries were: Kenya, Lesotho, Malawi, Swaziland, Zambia, and Zimbabwe. The countries are located in southeastern SSA and have among the highest HIV prevalence rates on the African continent (see Table 1).

DHS surveys are nationally representative population-based surveys with large sample sizes (usually between 5,000 and 30,000 households). In all households, women age 15-49 are eligible to participate; in many surveys men age 15-54(59) from a sub-sample are also eligible to participate. There are three core questionnaires in DHS surveys: A Household Questionnaire, a Women's Questionnaire, and a Male questionnaire. HIV biomarker data complements self-reported household survey information by providing an objective profile of a HIV status in the population. The sample is usually based on a stratified two-stage cluster design. The first stage is the SEA (or cluster), generally drawn from Census files. In the second stage, within each SEA, a sample of households is drawn selected from an updated list of households. The sample is generally representative at the national level, residence (urban-rural), and regional (departments, states) levels. This paper evaluates regional or community-level factors (i.e., characteristics of the SEA) that may affect HIV prevalence. Admittedly, the SEA is an arbitrary geographic boundary used only for the purposes of the survey, but it is nevertheless based on Census data and can be aggregated proportionally using the DHS sampling weights so that it should remain representative of the populations under study.

I analyzed the binary dependent variable HIV positive serostatus. The DHS provides anonymous, voluntary testing using blood spots collected on filter paper from a finger prick. An initial ELISA test is performed in the laboratory, with retesting of all positive tests and 5-10 percent of the negative tests with a second ELISA. For those with discordant results on the two ELISA tests, a new ELISA or a Western Blot is performed (Measure DHS, 2012).

I created two key independent variables aggregated at the SEA level: 1) the Gini coeffcient, representing household wealth inequality, which was constructed using a transformation of the DHS wealth index score and a Stata user-provided program called Fast Gini for calculating a weighted Gini-coefficient; 2) a second inequality index using the categories of the DHS categorical wealth index variable: the ratio of the mean wealth of households in the top 20% wealth quintile to that of those in the bottom 20% quintile. I controlled for several key household- or individual-level characteristics, including household wealth quintile (using the DHS-provided household wealth index), frequency of multiple sexual partnerships during the past year and number of lifetime sexual partners, self-reported sexually transmitted infection in the past year, condom use at last intercourse, and several

demographic variables associated with HIV serostatus, though it should be recognized that the sexual behavior "controls" were recognized as potential mediators of the association.

Because they were constructed using principal components analysis, the wealth index scores in the DHS included negative values. Therefore, they had to be transformed in order to make all the values greater than zero in order to calculate a Gini coefficient. Although the most common method for doing so is additive transformation (adding the lowest negative value to make all scores positive), this method has been shown to have distortionary effects on the underlying distribution (Sahn and Stifel, 2003). Therefore, I used an alternative and more reliable exponential transformation of the wealth index scores (Fox, 2012; Wai-Poi, Spilerman and Torche, 2008).

Empirical model

The final empirical model regressed the dependent variable individual HIV serostatus on the two key community-level independent variables (mean cluster-level Gini coefficient and wealth ratio) separately, and included 12 individual-level control variables: number of sexual partners (other than husband/wife) in the past year (dummy-coded as 0, 1, 2, and 3 or more), lifetime number of sexual partners (coded as 1, 2, 3-5, 6-10, and >10), condom use at last intercourse, self-reported STD in the past year, wealth status, male sex, urban residence, age (in years), education level, employed (currently working, having worked in past year, or on leave in the past 7 days), married or living together, and religious affiliation (Catholic, Protestant/other Christian, Muslim, No/other religion).

I evaluated multicollinearity by examining the correlation matrix for potential control variables included in the final model. I initially evaluated two sexual risk behavior control variables which measured the number of sexual partnerships in the past year. These two variables were the number of sexual partners 1) other than the spouse and 2) including the spouse, during the past year. Because these two variables were correlated above 0.75, I chose a single measure—number of sexual partnerships (other than the spouse) during the past year—as the one providing the best measure of this construct. I also included a measure of number of lifetime sexual partners.

Analysis

Because the data were nested within clusters and within countries, the assumption of standard logistic regression that respondents are independent within each cluster and that there is equal variance among clusters did not hold. Therefore, a multilevel regression framework was needed to account for the hierarchical structure of the data. I used a final multilevel logistic regression model of the form:

$$\begin{gathered}\operatorname{logit}[\Pr(\mathrm{HIV}_{ijk}=1|X_{ijk},\zeta_{jk},\zeta_k)]=X'_{ijk}\beta+\zeta_{jk}+\zeta_k,\\ \text{where } \zeta_k|X_{ijk}\sim N(0,\psi)\text{at level 3, and}\\ \zeta_{jk}|X_{ijk},\zeta_k\sim N(0,\omega)\text{at level 2.}\end{gathered}$$

This model assumed random variation in the intercepts across clusters and countries (random intercepts model) but constant slopes for the beta coefficients.

The three levels consisted of 43,091 respondents clustered within 2,641 SEAs across six countries. The analysis proceeded in four steps. First, I ran a null or base model including only the dependent variable HIV prevalence to establish the degree of variance at each of the two higher levels in order to validate use of a multilevel framework. Next, I added the level-1 demographic control variables to the model in order to assess the improvement in model fit and presence of significant effects for individual-level predictors of HIV serostatus. Finally, in each of two separate models, I added the key independent variables wealth Gini coefficient and wealth ratio to test for significance of these two predictors, controlling for the individual-level demographic variables. Finally, I repeated these first three steps using a model that included individual-level measures of sexual risk behaviors serving as potential mediators of the association, in order to look for attenuation of the effects of the inequality measures. Also, because concurrent sexual partnerships are one mechanism linking HIV infection to increased economic inequality, in separate ordered logit models I assessed the pathway of extramarital relationships as the dependent variable to attempt to better understand the potential mechanisms by which inequality might increase HIV prevalence.

Results

There were significant differences by low and high values (above and below the median) for both key independent (predictor) variables considered in the model and for most demographic and sexual behavior variables. Table 2 indicates these differences, reporting means and standard errors for continuous variables and counts and percentages for categorical variables. Overall, the mean HIV prevalence rate was 17.3%. The mean percent of households in the lowest wealth quintile was 17.2%; the percent of respondents reporting multiple sexual partnerships in the past year was about 29%; the percent reporting condom use at last sex was 22.5%; and the percent reporting an STD in the past year was only 3.8%. Results were remarkably similar for the two measures of wealth inequality (Gini coefficient and wealth ratio), and most comparisons between low and high groups within these two separate inequality measures were statistically significant and largely in the anticipated direction. In these bivariate analyses, higher cluster-level Gini coefficients and wealth ratios were associated with higher HIV prevalence rates and generally with higher rates of risky sexual behaviors.

The final multilevel regression models are shown in Table 3 (for the Gini coefficient) and Table 4 (for the wealth ratio), both for women and men combined and separately for women and men. The coefficients and their patterns in the two tables are remarkably similar. Base models (data not shown) including only the dependent variable HIV prevalence rate showed significant variation by country as anticipated from Table 1. In Model 1of both Table 3 and Table 4, including the key independent variables cluster-level wealth Gini coefficient and wealth ratio, respectively, and individual-level demographic control variables, both inequality measures were associated with an increased likelihood of being HIV positive. Male sex was protective, while urban residence, age, and being employed were associated

with a slightly increased likelihood of being HIV positive. Compared to unmarried persons (the referent group), those who were married or living together were at higher risk and those not living together were at much higher risk of being HIV positive. Including the cluster-level wealth Gini coefficient and wealth ratio in Model 1 significantly improved the model fit (significant log likelihood ratio test) relative to base models. Both the cluster-level wealth Gini coefficient (OR = 2.35, $p < 0.05$) and the wealth ratio (OR = 1.32, $p < 0.01$) were associated with a significant increase in the likelihood of being HIV positive. The marginal effect of the Gini coefficient was that a 1 point increase in the Gini coefficient of an SEA cluster was associated with a 2.35 times increased likelihood of being HIV positive, controlling for all other variables in the model. Similarly, a 1 point increase in the wealth ratio was associated with a 1.3 times increased likelihood of being HIV positive, controlling for the other variables in the model.

Adding in the sexual behavior variables in Model 2 of Tables 3 and 4 attenuated the effects of both measures of wealth inequality. There was a dose-dependent increase in the odds of being HIV positive with more lifetime sexual partners such that reporting 11 or more partners increased the likelihood of being HIV positive over five-fold. Condom use at last intercourse and an STD in the past year increased this likelihood by two and almost two and-a-half times, respectively. (Note that because this analysis is correlational, endogeneity or reverse causality probably explains the former association, i.e., condom use is likely to be more frequent among those who know they are HIV positive and/or who engage in higher-risk sex.)

Looking at Tables 3 and 4, Models 3 and 4 for women only, some interesting findings are evident. In both tables the odds ratios decrease slightly but remain significant. Comparing Model 1 to Model 3 in both tables, the odds ratio for married/living together (compared to the referent group unmarried) reverses and becomes less than 1 for women, indicating a protective effect for women who are married to or living with their partner, controlling for age (which becomes non-significant in Table 3). More education among women appears to be slightly protective (decreased odds of HIV infection), whereas it appears to increase risk in the combined men-women models. Also, the odds of HIV infection associated with not living together reduces (from 4.3 to 2.9 times) compared to that for men and women combined. Comparing the full models incorporating the risk behaviors (Model 2 compared to Model 4), again age becomes non-significant and the odds ratio for married/living together again becomes greater than 1, suggesting that risk behaviors are mediating some of this effect and removing any protection associated with cohabitation with sexual partners for women.

Looking at Tables 3 and 4, Models 5 and 6 for men only, odds ratios for the Gini coefficient and wealth ratio are larger for men. Age is a significant predictor in both reduced and full models, and education appears to increase, rather than decrease risk, although it becomes non-significant in the full models (Model 6 in both tables). It is apparent that the risks associated with cohabitation and not living together (compared to the unmarried referent group) in the combined men-women model is driven by males. The coefficients increase from Model 5 to Model 6 for married/living together for males, suggesting that some other factors increasing risk are not being fully picked up by the risk behaviors.

Results from the ordered logit models predicting numbers of extramarital partners in the past year (Table 5) indicated that the likelihood of having more an extramarital partners was higher in clusters with more wealth inequality, although this relationship was significant only for the model including the wealth ratio predictor variable (OR = 1.27, $p < 0.001$). Not surprisingly, marriage/cohabitation appeared to be highly protective in all ordered logit models (OR = 0.01, $p < 0.001$), while being a younger male appeared to increase risk by over 4-fold. Looking at these models by sex indicated several interesting differences. The significant result for wealth ratio held for women (OR = 1.54, $p < 0.001$) but not for men (OR = 1.15); age was not a significant factor in the models for women but was significant and protective in the models for men; urban residence was a significant risk factor for women but not for men; among women, primary education appeared to be a significant risk factor, while a secondary or higher education was no longer significant (compared to models for women and men combined). The risk associated with secondary or higher education appeared to work in opposite directions for men compared to women, reducing risk for women and increasing it for men. Religion was not a significant factor among women but was among men. Among men, being of Protestant or other Christian faith (compared to Catholic) significantly reduced risk, while being of Muslin faith increased it.

Discussion

The relationships between HIV prevalence and the control variables were all in the anticipated direction based on previous studies and expectations about HIV risk and demographic and sexual variables operating at the individual level. Both the cluster-level Gini coefficient for household wealth and the wealth ratio were significant predictors of HIV serostatus, controlling for all other variables in the models, including household wealth and several known behavioral and demographic predictors of positive serostatus. This is the second known study to produce empirical evidence of these effects using multiple countries and regions in SSA, and the second to demonstrate this effect at the DHS cluster level by utilizing its inherent population-based survey sampling strategy. Although a large literature suggests that economic inequality increases the risk for a variety of diseases after controlling for absolute levels of wealth or income (Subramanian and Kawachi, 2004; Wilkinson and Pickett, 2009; Wilkinson, 2005), very few have demonstrated it in the context of infectious disease in developing countries. Similarly to these two prior studies (Durevall and Lindskog, 2012; Fox, 2012), there is a persistent association between regional/district- and/or neighborhood/cluster-level wealth inequality and HIV serostatus after controlling for household-level wealth. Also consistent with these two prior studies, results from models with extramarital partners as the dependent variable suggest that the mechanism is at least in part mediated by an increase in risky sexual behavior.

Consistent with the one prior study of this association at the DHS cluster level in Malawi (Durevall and Lindskog, 2012) but contrary to findings from national-level studies, household wealth was not significantly associated with HIV positive serostatus. This result could be explained by more recent evidence pointing to a complex, dynamic association between wealth and HIV serostatus in SSA. Parkhurst (2010) found that, at the country level, as per capita GDP increased, the confirmed trend for the prevalence of HIV infection to increase with increasing wealth quintile dissipated. He identified a threshold of

approximately US$ 2,000 above which this tracking with wealth becomes inconsistent. Half of the countries in the current sample exceeded this GDP threshold. Furthermore, Parkhurst's analysis of trend data from two Tanzania DHS studies suggested that HIV has become more prevalent in poorer individuals as development has progressed (Parkhurst, 2010). Similarly, Fox (2012) found that in poorer regions/countries, individuals with more wealth were more likely to be infected, whereas in wealthier regions/countries, individuals with less wealth were more likely to be infected with HIV. She noted that these results, combined with those of prior supportive studies, suggest a changing social gradient with increasing wealth and development in which, due to increasing knowledge, prestige, and power, wealthier segments of the population are better able to adapt to new health threats, socially reproducing health inequalities via social stratification mechanisms (Fox, 2012; Phelan, Link and Tehranifar, 2010).

The null results for the major religious affiliations could be explained in that most national studies have looked at national-level religious affiliation, and most African Muslim nations are supra-Saharan, where HIV prevalence rates are much lower. Muslims comprised a very small segment of this pooled sample (less than 5%) so the power to detect a significant effect was reduced. However, the direction of the odds ratios suggested a slight protective effect for Muslim religion. The significant effect for “none/other” religious affiliation, which is attenuated with the addition of the sexual behavior variables to the models, is consistent with the possibility of increased risky-taking behaviors in this minority group in association with lower social status or differential treatment based on religion.

Disaggregation by sex indicated that the relationships between community wealth inequality and HIV positive status were stronger for males. However, in the ordered logit models predicting risk behavior (number of extramarital partners in the past year), the wealth ratio was a significant predictor only in models for women. Primary education was a risk, and age was not a risk in models for women (these two reversed in the models for males). Although causality cannot be inferred from these results, taken as a whole, they could be interpreted as suggesting a situation in which neighborhood wealth inequalities, particularly in urbanized areas, are associated with, and possibly promoting, increased extramarital sexual relationships by women of childbearing age, who are able to trade sex for money or resources provided by men in households with greater wealth, thus escalating HIV risk within sexual networks. The positive relationship for women with a primary education could reflect increased contact in urbanized areas, through the educational system, with men seeking extramarital partnerships, with women increasingly engaging in such relationships in contexts of high wealth inequalities but less so as they achieve higher levels of education and a trajectory leading to greater autonomy and financial independence. These results point to the importance of disaggregating by sex in such analyses and exploring potential mechanisms of action/causal pathways through modeling behavioral mediators. They are also consistent with the Nattrass et al, 2012 panel study, in which household assets were negatively correlated with HIV status for women but not for men, and in which HIV-positive women were much less likely to have made the transition from (primary) school to tertiary education, a transition which would place them on a trajectory of lowered risk (Nattrass et al., 2012).

It is important to note several study limitations. One methodological limitation is the assumption of constant slopes on the beta coefficients for the two key independent variables. However, adding random slopes to the models would have introduced greater complexity and was not necessary for the purposes of the analysis, and the limited number of countries (six) at level 3 precluded adding this feature at that level. Another likely limitation is violation of basic ordinary least squares logistic regression assumptions through inadequate model specification and the presence of omitted variables, which would bias the beta estimate for the key independent variables in the direction of a type-1 error. Clearly the final regression model did not capture all variables affecting HIV prevalence at multiple levels and may conflate to some degree mediation and confounding, but it did capture important available individual-level predictors, and by adding potential behavioral mediators last, I was able to assess the extent to which some variables might mediate the observed effects of the independent variables. Endogeneity in the independent variables or omission of a "left-out common cause" of both wealth inequality and HIV prevalence in which an omitted variable is associated with both cluster-level wealth inequality and with HIV prevalence, or other forms of omitted variable bias, are also a potential sources of bias in this analysis. Also, the DHS Wealth Index used to construct these independent variables has limitations which may lead to erroneous conclusions regarding the direct effect of wealth on HIV status. Because different assets are used in each country to construct the index (although a basic set of assets, such as type of flooring, water supply, sanitation facilities, appliances, transportation, etc. are included in every survey), it is not directly comparable across countries. Also, the index is not the best proxy for consumption expenditure, the SES measure preferred by economists (Howe *et al.*, 2009). It also tends to negatively weight assets from traditional forms of subsistence production and over-weight assets obtained in the modern cash economy, and thus tends to capture involvement in the modern, cash-oriented economy, which is also highly correlated with both urbanization and education level (Bingenheimer, 2007). This property may help explain its consistent positive association with HIV status among poorer developing African countries. However, the DHS index is considered a reasonable measure of economic well-being, it is the measure that was available in the datasets, and its major purpose was to construct within-cluster relative measures of economic inequality and to control for absolute measures of individual wealth status rather than to compare wealth status across countries.

Also, because these data are cross-sectional, we can only observe the relationship between wealth inequality and HIV prevalence at a single point in time. Reverse causality, in which HIV infection affects household wealth and cluster-level wealth distribution, is undoubtedly present. Although the sexual behavior variables were somewhat weak measures of HIV risk behavior (with floor effects and apparent underreporting), a dose-response relationship for lifetime sexual partners suggested that variance associated with this risk factor was captured. Future studies should try to identify more valid measures of behavioral risk and to assess these factors as potential individual-level mediators in a multi-level modeling framework. Also, these results point to some of the difficulties in doing empirical work in the field of social/structural determinants of health. It is often difficult to relate macro-level social factors to individual health status due to unavailability of accurate measures of both micro- and macro- level factors, and to the complexity of methodologies needed to adequately

control for factors operating at multiple levels. Nevertheless, neglect of these higher-level factors moderating individual behaviors risks ascribing too much predictive power to micro-level factors and may lead to missed opportunities to modify social environments and create structural changes which induce more health-supportive behaviors.

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Table 1

Country sample

Country	HIV prevalence (CI)[a]	Tested sample	Year of survey	Refusal rate (%)	GNI per capita (2010)[b]	GDP growth rate (2010)[c]	Gini ratio (most recent year)[d]
Kenya	6.4 (5.4– 7.4)	6,906	2008/9	10.8	1640	5.6	41.9
Malawi	10.7 (9.8–11.7)	13,927	2010	6.5	860	7.1	39.0
Zambia	14.2 (13.1–15.4)	10,873	2007	20.0	1380	7.6	50.8
Zimbabwe	15.3 (14.4–16.2)	13,897	2010/11	14.1	—	9.0	56.6
Lesotho	23.0 (21.5–24.5)	6,924	2009	4.7	1970	5.6	58.1
Swaziland	25.9 (24.5–27.5)	8,187	2006/07	12.8	5600	2.0	50.4

[a] Countries ordered from lowest to highest prevalence. Figures weighted for probability of selection into the sample.

[b] GNI per capita, PPP (purchasing power parity, current international $), 2010. Zimbabwe's data missing due to the ongoing political and economic crisis; data from World Bank, World Development Indicators Database.

[c] Annual GDP growth rate for 2010. Data from World Bank, World Development Indicators Database.

[d] Gini data from UNU-WIDER World Income Inequality Database, Version 2.0c, May 2008.

Table 2

Summary statistics for variables included in final model, by low and high Gini coefficient and wealth ratio

		Gini coefficient		Wealth ratio	
	Total	**Low**	**High**	**Low**	**High**
Count	43,032[a]	21,520	21,512	21,522	21,509
Dependent variable					
HIV positive, n (%)	7,444 (17.3)	3,395 (15.8)	4,048 (18.8) ***	3,347 (15.6)	4,097 (19.0) ***
Independent variables					
Gini coefficient, mean (SE)	0.42 (0.004)	0.28 (0.003)	0.56 (0.003) ***	0.34 (0.006)	0.50 (0.004) ***
Wealth ratio, mean (SE)	1.49 (0.008)	1.35 (0.009)	1.62 (0.012) ***	1.23 (0.003)	1.74 (0.011) ***
Demographic variables					
Age in years, mean (SE)	30.8 (0.06)	31.1 (0.09)	30.5 (0.08) ***	31.1 (0.09)	30.5 (0.08) ***
Male, n (%)	20,403 (47.4)	10,337 (48.0)	10,066 (46.8) *	10,421 (48.4)	9,982 (46.4) ***
Urban residence	12,273 (28.5)	7,808 (36.3)	4,465 (20.8) ***	8,540 (40.0)	3,733 (17.4) ***
Working	30,916 (71.8)	15,893 (73.8)	15,023 (69.8) ***	15,954 (74.1)	14,962 (69.6) ***
Education					
None	3,348 (7.8)	1,993 (9.3)	1,355 (6.3) ***	1,591 (7.4)	1,757 (8.2)
Primary	20,418 (47.4)	10,105 (47.0)	10,313 (47.9)	9,611 (44.6)	10,807 (50.2) ***
Secondary+	16,650 (38.7)	7,699 (35.8)	8,951 (41.6) ***	8,651 (40.2)	7,998 (37.2) **
Wealth quintile					
Lowest	7,380 (17.2)	4,819 (22.4)	2,561 (11.9) ***	3,504 (16.3)	3,876 (18.0)
Second	7,940 (18.4)	3,953 (18.4)	3,988 (18.5)	3,739 (17.4)	4,201 (19.5) *
Middle	8,386 (19.5)	2,892 (13.4)	5,494 (25.5) ***	3,826 (17.8)	4,560 (21.2) ***
Fourth	9,284 (21.6)	3,429 (15.9)	5,855 (27.2) ***	4,352 (20.2)	4,932 (22.9) *
Highest	10,041 (23.3)	6,428 (29.9)	3,613 (16.8) ***	6,101(28.4)	3,940 (18.3) ***
Marital status					
Unmarried	8,363 (19.4)	3,935 (18.3)	4,428 (20.6) **	3,624 (16.8)	4,739 (22.0) ***
Married/living together	31,888 (74.1)	16,260 (75.6)	15,628 (72.6) ***	16,504 (76.7)	15,384 (71.5) ***

		Gini coefficient		Wealth ratio	
	Total	**Low**	**High**	**Low**	**High**
Not living together	2,780 (6.5)	1,325 (6.2)	1,455 (6.8) *	1,394 (6.5)	1,386 (6.4)
Religion					
Roman Catholic	8,369 (19.4)	4,452 (20.7)	3,917 (18.2) **	4,149 (19.3)	4,220 (19.6)
Protestant/other Christian	27,131 (63.0)	13,961 (64.9)	13,170 (61.2) ***	14,578 (67.7)	12,553 (58.4) ***
Muslim	1,793 (4.2)	947 (4.4)	846 (3.9)	818 (3.8)	975 (4.5)
None/other	5,739 (13.3)	2,160 (10.0)	3,579 (16.6) ***	1,978 (9.2)	3,762 (17.5) ***
Sexual behavior variables					
# extramarital sex partners past year					
0	30,684 (71.3)	15,661 (72.8)	15,023 (69.8) **	15,979 (74.2)	14,705 (68.4) ***
1	10,463 (24.3)	5,007 (23.3)	5,456 (25.4) **	4,722 (21.9)	5,741 (26.7) ***
2	1,531 (3.6)	669 (3.1)	862 (4.0) ***	645 (3.0)	886 (4.1) ***
3+	354 (0.8)	183 (0.9)	171 (0.8)	177 (0.8)	177 (0.8)
# lifetime sex partners					
1	13,653 (31.7)	6,809 (31.6)	6,844 (31.8)	6,918 (32.1)	6,735 (31.3)
2	9,946 (23.1)	4,960 (23.0)	4,986 (23.2)	4,888 (22.7)	5,058 (23.5)
3 - 5	12,339 (28.7)	6,083 (28.3)	6,256 (29.1)	6,018 (28.0)	6,321 (29.4) *
6 - 10	3,864 (9.0)	2,027 (9.4)	1,837 (8.5) *	2,028 (9.4)	1,836 (8.5) *
11+	3,229 (7.5)	1,641 (7.6)	1,588 (7.4)	1,670 (7.8)	1,559 (7.2)
Condom use last sex	9,697 (22.5)	4,477 (20.8)	5,220 (24.3) ***	4,315 (20.0)	5,382 (25.0) ***
STD past year	1,624 (3.8)	738 (3.4)	887 (4.1) **	717 (3.3)	907 (4.2) ***

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$

[a] estimated population size adjusted for survey sampling frame

Table 3

Parameter estimates[a] (odds ratios) for three-level models predicting HIV positive status

	Men and women		Women only		Men only	
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**
Fixed effects						
Community (cluster) charac.						
Gini coefficient	2.35*	2.04*	2.19*	1.88*	2.49*	2.27*
Individual demographic char.						
Age (years)	1.03***	1.02***	1.02	1.01	1.04***	1.03***
Sex (male)	0.69***	0.39***	—	—	—	—
Residence (urban)	1.85***	1.64***	1.91***	1.67***	1.60***	1.50**
Working	1.19**	1.06	1.18**	1.05	1.13	1.06
Education						
None (Ref.)						
Primary	1.25**	1.22*	1.32*	1.32*	1.15	1.09
Secondary+	1.31**	1.25*	1.28*	1.27	1.28*	1.17
Marital status						
Unmarried (Ref.)						
Married/living together	1.36*	2.16***	0.82**	1.25*	2.42***	3.71***
Not living together	4.31***	3.73***	2.90***	2.36***	6.57***	6.19***
Wealth quintile						
Lowest (Ref.)						
Second	1.07	1.07	1.06	1.05	1.11	1.10
Middle	1.08	1.08	1.10	1.08	1.08	1.09
Fourth	1.12	1.12	1.19	1.21	1.12	1.07
Highest	1.01	1.01	1.03	1.03	1.12	1.06
Religion						
Roman Catholic (Ref.)						
Protestant/other Christian	1.02	1.02	1.03	1.01	1.02	1.04

	Men and women		Women only		Men only	
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**
Muslim	0.93	0.89	1.02	0.95	0.95	0.90
None/other	1.22***	1.13	1.28*	1.22	1.22***	1.12
Individual sexual behaviors						
# extramarital part. past year						
0 (Ref.)						
1	—	1.15	—	1.00	—	1.14
2	—	0.84	—	0.75	—	1.06
3+	—	0.81	—	1.33	—	0.96
# lifetime sex partners						
1 (Ref.)						
2	—	2.39***	—	2.53***	—	2.24***
3 – 5	—	3.64***	—	3.95***	—	3.31***
6 – 10	—	4.47***	—	5.11***	—	4.00***
11+	—	5.38***	—	4.49***	—	4.92***
Condom use last intercourse	—	2.06***	—	2.21***	—	2.09***
Any STD past year	—	2.41***	—	2.46***	—	2.57***
Random effects						
Cluster-level (level 2) variance	0.50	0.45	0.38	0.37	0.55	0.53
Country-level (level 3) var.[b]	2.04***	1.84***	0.56***	0.28***	0.51***	0.41***
Log likelihood	-17545.25	-16636.01	-9990.919	-9344.24	-7607.446	-7297.367
AIC	35128.51	33328.03	20017.84	18742.48	15250.89	14648.73
Log likelihood Ratio Test (X^2)[c]	34.69***	27.21***	24.18***	14.44***	22.24***	17.64***

[a] Parameters for predictors (fixed effects) are reported as odds ratio; for random effects, the parameter is the variance

[b] Significance of random effects evaluated by comparing model with a similar one in which random effects have been constrained to be zero.

[c] Compared to model excluding the independent variable

* p<0.05

** p<0.01

***p<0.001

Table 4

Parameter estimates[a] (odds ratios) for three-level models predicting HIV positive status

	Men and women		Women only		Men only	
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**
Fixed effects						
Community (cluster) charac.						
Wealth ratio	1.32**	1.30***	1.25**	1.24**	1.36**	1.35***
Individual demographic char.						
Age (years)	1.03***	1.02***	1.02***	1.01	1.04***	1.03***
Sex (male)	0.69***	0.39***	—	—	—	—
Residence (urban)	1.83***	1.63***	1.88***	1.66***	1.59***	1.49***
Working	1.19**	1.07	1.18**	1.06	1.13	1.06
Education						
None (Ref.)						
Primary	1.26**	1.23**	1.33*	1.33*	1.17	1.11
Secondary+	1.32**	1.26*	1.29*	1.28*	1.30**	1.19
Marital status						
Unmarried (Ref.)						
Married/living together	1.36*	2.15***	0.82**	1.24*	2.42***	3.72***
Not living together	4.32***	3.74***	2.90***	2.36***	6.59***	6.22***
Wealth quintile						
Lowest (Ref.)						
Second	1.10	1.10	1.10	1.09	1.17	1.15
Middle	1.15	1.14	1.18	1.15	1.19	1.19
Fourth	1.18	1.18	1.28	1.28	1.22	1.16
Highest	1.04	1.04	1.07	1.06	1.17	1.10
Religion						
Roman Catholic (Ref.)						
Protestant/other Christian	1.03	1.03	1.04	1.01	1.02	1.04

	Men and women		Women only		Men only	
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**
Muslim	0.93	0.89	1.02	0.95	0.94	0.89
None/other	1.22***	1.13*	1.28*	1.22	1.23***	1.13
Individual sexual behaviors						
# extramarital part. past yr.						
0 (Ref.)						
1	—	1.14	—	1.00	—	1.14
2	—	0.84	—	0.74	—	1.07
3+	—	0.81	—	1.29	—	0.96
# lifetime sex partners						
1 (Ref.)						
2	—	2.39***	—	2.53***	—	2.25***
3 – 5	—	3.66***	—	3.97***	—	3.33***
6 – 10	—	4.49***	—	5.13***	—	4.02***
11+	—	5.40***	—	4.51***	—	4.96***
Condom use last intercourse	—	2.06***	—	2.22***	—	2.11***
Any STD past year	—	2.41***	—	2.47***	—	2.56***
Random effects						
Cluster-level (level 2) variance	0.51	0.45	0.39	0.37	0.56	0.53
Country-level (level 3) var.[b]	1.93***	2.13***	0.77***	0.33***	0.50***	0.41***
Log likelihood	-17553.54	-16640.29	-9998.201	-9346.904	-7612.116	-7299.98
AIC	35145.08	33336.57	20032.4	18747.81	15260.23	14653.96
Log likelihood Ratio Test (X^2)[c]	18.12***	18.67***	9.62**	9.12**	12.90***	12.41***

[a] Parameters for predictors (fixed effects) are reported as odds ratio; for random effects, the parameter is the variance

[b] Significance of random effects evaluated by comparing model with a similar one in which random effects have been constrained to be zero.

[c] Compared to model excluding the independent variable

* p<0.05

** p<0.01

***p<0.001

Table 5

Parameter estimates[a] (odds ratios) for three-level ordered logit models predicting number of extramarital partners in the past year

	Men and women		Women only		Men only	
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**
Explanatory variables						
Fixed effects						
Community (cluster) charac.						
Gini coefficient	1.41	—	1.29	—	1.48	—
Wealth ratio	—	1.27***	—	1.54***	—	1.15
Individual characteristics						
Demographics						
Age (years)	0.99**	0.99**	1.01	1.01	0.97***	0.97***
Sex (male)	4.41***	4.41***	—	—	—	—
Residence (urban)	1.27***	1.29***	1.68**	1.80**	1.12	1.12
Working	1.44***	1.45***	1.49**	1.51***	1.39***	1.39***
Education						
None (Ref.)						
Primary	1.06	1.07	1.39***	1.37***	0.95	0.96
Secondary+	1.14*	1.14*	1.08	1.06	1.11	1.12
Marital status						
Unmarried (Ref.)						
Married/living together	0.01***	0.01***	0.01***	0.01***	0.01***	0.01***
Not living together	0.25***	0.25***	0.08***	0.08***	0.41***	0.41***
Wealth quintile						
Lowest (Ref.)						
Second	0.93	0.95	1.22	1.25	0.85	0.87
Middle	1.00	1.04	1.28	1.33	0.93	0.98
Fourth	0.99	1.02	1.23	1.27	0.92	0.96
Highest	1.07	1.09	1.10	1.12	1.10	1.12

	Men and women		Women only		Men only	
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**
Religion						
Roman Catholic (Ref.)						
Protest./other Christian	0.91**	0.91**	1.00	1.01	0.87***	0.87***
Muslim	1.17	1.16	1.15	1.15	1.15**	1.14**
None/other	1.10	1.10	1.11	1.11	1.10	1.11
Random effects						
Cluster-level (level 2) var.	0.24	0.24	0.50	0.49	0.32	0.32
Country-level (level 3) var.[b]	0.30***	0.33***	0.61***	0.57***	0.26***	0.32***
Log likelihood	-15059.09	-15054.39	-4139.389	-4131.821	-10607.86	-10608.94
AIC	30160.19	30150.79	8318.779	8303.643	21255.73	21257.88

[a] Parameters for predictors (fixed effects) are reported as odds ratio; for random effects, the parameter is the variance

[b] Significance of random effects evaluated by comparing model with a similar one in which random effects have been constrained to be zero.

* p<0.05

** p<0.01

*** p<0.001