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Understanding the Southern African 'Anomaly'
Poverty, Endemic Disease, and HIV

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Abstract

Background: Adult HIV prevalence in the nine countries of southern Africa averages more than 16 times the prevalence in other low- and middle-income countries. Previous studies argue that the intensity of the HIV epidemic in southern Africa results from regional characteristics, such as apartheid labor regulations and regional mineral wealth, which contributed to circular migration patterns and highly skewed income distribution. The present study also emphasizes the importance of cofactor diseases, which are suspected of raising HIV prevalence by increasing HIV viral load in infected persons or by making uninfected persons more vulnerable to HIV infection through lower immunity or genital lesions and/or inflammation. *Method:* the study uses multiple regression analysis on country-level data with HIV prevalence as the dependent variable. Regressors are ten socio-economic variables used in most previous cross-national analyses of HIV, two measures of cross-border migration, and measures of six cofactor infections. *Results:* The 10 socio-economic variables "explain" statistically only 25% of the difference in HIV prevalence between southern Africa and other low- and middle-income countries, but adding the four cofactor infection variables to the model allows us to "explain" 80% of the southern Africa difference in HIV prevalence. *Conclusion:* The relative affluence of southern Africa and historical migration patterns have tended to mask the vulnerability of the majority of the population who are poor and who have very high prevalence of infectious and parasitic diseases. Those diseases replicate a cycle of poverty that can lead not just to social vulnerability to HIV through risky behaviors but also to biological vulnerability through coinfections. An important implication of this research is that integrating treatment of endemic diseases with other HIVprevention policies may be necessary to slow the spread of HIV. Treatment of cofactor infections is a lowcost, policy-sensitive, high-impact variable.

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Background: history, economics, and health in southern Africa

Some of southern Africa's historical features are unique, but many of its socio-economic, ecological, and health characteristics are similar to those in the rest of Africa and in other poor regions. The severity of the HIV epidemic in southern Africa is most probably the result of a combination of factors it shares with other poor countries, especially in Africa, and features unique to southern Africa. We examine here a number of factors in southern Africa that have been proposed in the literature as determinants in aggravating the spread of HIV.

Labor migration and apartheid: Perhaps the most obvious characteristic of southern Africa that sets it apart from the rest of the world is its history of apartheid and the system of internal and international labor migration that it reinforced. Even before the National Party decreed strict residential segregation in South Africa in 1948, cross-border and internal labor migration supplied the labor needed in the country's mines and factories. Long before the emergence of HIV, an extensive literature dating to the 1940s addressed the health effects of migration in southern Africa, including the spread of tuberculosis and sexually transmitted infections (STIs) from migrants to their home communities (see Horwitz 2001 for a summary of the early literature).

There is also a substantial literature that points to the role of migration in the spread of HIV as well, and many of those works focus on southern Africa in particular (Thahane 1991; International Labour Office 1998; Horwitz 2001; Brummer 2002; Hunter 2002; Lurie *et al.* 2002; Marks 2002; Kanyenze 2004; Lurie 2004; Crush *et al.* 2005; Clark *et al.* 2007; Hunter 2007). The assumption that long separation of spouses in circular migrant streams, single-sex hostels, and higher levels of commercial sex work around mines and factories would promote the spread of HIV is supported with prevalence rates among migrants that are higher than among others in the same communities (Lurie 2004; see also Brummer 2002; Clark *et al.* 2007; and Kanyenze 2004, although without data).

Migration has also been associated with HIV transmission in other parts of the world. In Ecuador, for example, over 80 percent of the people infected with HIV in the first decade of the epidemic had worked in the United States or were partners of migrants to the United States. Circular migration between the United States and Dominican Republic has also been associated with the spread of HIV in the Dominican Republic (Stillwaggon 2006). Not all studies, however, have found that labor migrants are more likely to be HIV-infected. In Southeast Asia, Oppenheimer and colleagues found that labor migrants were more risk averse and less likely to be infected (Oppenheimer *et al.* 1998, cited in Skeldon 2002, p. 3).

Labor migration has played a very important role in the economies of southern Africa and very likely has aggravated the HIV epidemic. But migration is not unique to the region and may be insufficient to explain the difference in prevalence between southern Africa and West Africa, the Caribbean, the Middle East, or Southeast Asia. In combination with other characteristics of southern Africa, however, migration may have been an important factor in the complex etiology of the epidemic and should be included in a model of HIV transmission.

Cross-border and internal migration are socially disruptive and affect health in numerous ways, both positively and negatively. A problem with the HIV literature on migration, however, is the exclusive emphasis on the effect of migration on risky sexual behavior, often without data, to the exclusion of other health-related factors (Brummer 2002; Kanyenze 2004). While the literature of social medicine in South Africa of the last half century has always included some discussion of the miserable nutrition and living conditions of migrants (Horwitz 2001; Marks 2002), the sound bite that has emerged in AIDS policy discourse emphasizes "the constant movement of large numbers of sexually active men from town to countryside and back" (Marks 2002, p.18). Shula Marks, acknowledging both the migration and the deprivation under apartheid, argues that the spread of HIV in South Africa was "an epidemic waiting to happen" (Marks 2002, p.13). Certainly the behavioral implications of separating men from their families and communities were very important in the spread of STIs, and later HIV, in southern Africa. But the other important health effects of migration—resulting from poor sanitation and poor nutrition in single-sex barracks and squatter settlements and the unhealthy working conditions in the mines—are overlooked in emphasizing exclusively the effect of the southern African labor system on sexual partner change, as AIDS policy and discourse have done.

Post-apartheid migration and economic reform: In the post-apartheid years, there have been significant changes in the southern African economies. Migration within South Africa and to South Africa from most neighboring countries has increased, but the composition of the migrant labor force has changed significantly. Women are increasingly migrating, both with male partners and alone. Rising levels of male unemployment have changed the support relationships in the region (Posel and Casale 2003). Mark Hunter argues that the rapid increase and enormous scale of the HIV epidemic in South Africa (and one might extend that analysis to other countries in the region) was not an inevitable result of a history of apartheid, although he acknowledges that labor migration and the enforcement of family separation in apartheid did set the stage. Rather the decision to institute structural adjustment policies and austerity measures in the 1990s was disastrous for a large proportion of the population. Unemployment has risen to over 40 percent, marital rates have fallen to 30 percent, and inequality is substantially higher in the last two decades (Hunter 2007). Women's migration has increased and informal settlements have mushroomed around both large and small cities (Posel 2003; Posel and Casale 2003; Crush *et al.* 2005). Hunter draws much-needed attention to the gap in health care provision that mirrors the stark inequality in the economy. (On inequality in health care, see also Booysen 2002; 2003).

The earlier focus on mining, male migration, and apartheid and the recent emphasis on female migration and the social conditions of informal settlements are important. Some authors (Horwitz 2001; Marks 2002; Hunter 2007) allude to the miserable living and working conditions and poor nutrition of migrants and their families, but they do not make explicit the link between those conditions and susceptibility to infectious diseases, including HIV. Ultimately, analyses of the impact of migration on HIV rely on its effect on risky sexual behavior. They support, inadvertently or not, the standard explanation for high HIV prevalence in sub-Saharan Africa that focuses almost exclusively on risky sexual behavior, missing entirely the objective conditions of poverty that are inescapable for people living with poor nutrition, contaminated water supplies, inadequate housing, and little access to health services. Poverty may encourage risky sexual behavior, but it also has a direct impact on the health of poor people in ways that increase vulnerability to HIV, whether it is transmitted sexually, vertically, or through unsafe health care.

Inequality: Another striking characteristic of southern Africa is its inequality. All of the southern African countries except Malawi have above average income inequality, but Namibia, Lesotho, and Botswana have the highest income inequality in the world (measured by Gini coefficient) and South Africa, Swaziland, Zambia, and Zimbabwe are not far behind. In most previous cross-country studies of HIV prevalence, high income inequality is associated with higher prevalence of HIV (Over 1998; Stillwaggon 2000; Mahal 2002; Stillwaggon 2002; Tsafack Temah 2008). Inequality is important as a factor in the introduction of HIV into the region through international travel of the more affluent and migration of the poor to find work. At the most fundamental level, however, the stark inequality of southern Africa means that the relatively high per capita GDP masks the profound poverty of the majority of the population and the direct impact of that poverty on health.

Gender inequality: Another dimension of inequality is gender inequality. A number of authors have argued that the oppression of women, by reducing their incomes, assets, and power, makes it difficult, for example, to negotiate safe sexual practices and may encourage commercial sex work or transactional sex (Over 1998; Brummer 2002; Hunter 2002; Tsafack Temah 2008). In that case, a country with greater gender inequality could have higher rates of HIV. On the other hand, reducing the oppression of women might free them from traditional restrictions on sexual behavior and allow greater sexual activity, some of which might be risky sexual activity. In a statistical analysis using cross-national data, therefore, it is difficult to predict whether measures of gender inequality would be directly or inversely correlated with HIV prevalence.

Sexually transmitted infections (STIs): STIs that produce ulceration or inflammation of the genitals increase the likelihood of HIV transmission and are therefore considered HIV cofactors (Grosskurth *et al.* 1995; Fleming and Wasserheit 1999; Boulton and Gray-Owen 2002; Corbett *et al.* 2002). Ulcerations or genital lesions are open pathways for the HIV virus. Inflammation is associated with a high concentration of the cells ($CD4^+$) that are the target of the HIV virus. In poor populations, bacterial STIs are more likely to be untreated because of lack of access

to health care (Sturm *et al.* 1998). The health burden of STIs is far higher in Africa than in other low- and middle-income countries. (See Table 5.)

Endemic cofactor diseases: Several diseases with high prevalence in southern Africa are associated with an increase in transmission of HIV. The relative affluence of the region and the emphasis on risky sexual behavior have led most policy makers to overlook the biological vulnerability of southern Africa's majority poor population. Between otherwise healthy adults in developed countries, HIV has very low rates of heterosexual transmission (World Bank 1997). In the developing world, including southern Africa, however, the majority of the population is not "otherwise healthy." Poor nutrition and the burden of other infectious and parasitic diseases suppress immune response in HIV-negative sexual partners and infants and increase viral load and viral shedding in HIV-infected partners and mothers (Semba *et al.* 1994; Landers 1996; John *et al.* 1997; Fawzi and Hunter 1998; Friis and Michaelsen 1998; Woodward 1998; Montresor *et al.* 2001; Nacher 2002; PPC (Partnership for Parasite Control) 2002; Wolday *et al.* 2002; Borkow and Bentwich 2006).

Mounting evidence also demonstrates the specific mechanisms whereby biological cofactors increase vulnerability to HIV infection and increase contagiousness of infected persons, and thus increase the probability of transmission. Certain parasites, common in tropical regions, produce inflammation and lesions in the genital tract. Lymphatic filariasis has been shown to suppress immune response in HIV-negative persons and increase viral load in HIV-infected persons, affecting individual transmission and population dynamics (Gopinath *et al.* 2000). Moreover, lymphatic filariasis (LF) affects the lymphatic immune system and also can result in infection of and damage to the genital organs.

Urinary schistosomiasis (*S. hematobium*), which afflicts over 200 million people in sub-Saharan Africa (and almost nowhere else), acts as a co-factor of HIV transmission much in the same way as do STIs. Worms and ova of *S. hematobium* infect the reproductive tract of both men and women and create lesions and inflammation of the genital area (Attili *et al.* 1983; Feldmeier *et al.* 1995; Marble and Key 1995; Leutscher *et al.* 1998). (For extensive bibliography on endemic cofactors of HIV, see Stillwaggon 2006). A recent trial in Zimbabwe found that genital lesions of schistosomiasis increased HIV risk in women three-fold compared to women in the same villages without genital lesions of schistosomiasis (Kjetland *et al.* 2006). The WHO Ad Hoc Strategic and Advisory Group on Neglected Tropical Diseases recently identified the interactions of endemic parasitic and infectious diseases with HIV as a priority for research.

Malaria, which is widespread in tropical areas, especially in sub-Saharan Africa, interacts with HIV, increasing viral load up to ten-fold. Viral load remains elevated in HIV-infected persons for six weeks after malarial episodes (Hoffman *et al.* 1999; Kublin *et al.* 2005; Abu-Raddad *et al.* 2006). Elevated viral load not only increases individual contagiousness of HIV-infected persons, it also affects the dynamics of the epidemic at a population level (Bloland *et al.* 1995; Xiao *et al.* 1998; Hoffman *et al.* 1999; Whitworth *et al.* 2000; Corbett *et al.* 2002; Abu-Raddad *et al.* 2006; for additional discussion and sources, see Stillwaggon 2006). Individuals in malaria-endemic areas have a higher probability of sexual contact with persons who have high viral load due to coinfection with malaria and who are therefore more contagious. The importance of newly infected persons as HIV transmitters because of their elevated viral loads is receiving warranted attention (Quinn *et al.* 2000; Pilcher *et al.* 2004; Cohen and Pilcher 2005; Brenner *et al.* 2007). Other causes of elevated viral load, including malaria, also merit inclusion in models of HIV transmission dynamics.

While all infectious and parasitic diseases have a probable impact on resistance to infection due to immune dysregulation, those diseases that have a substantial impact on viral load (malaria) and those that affect the genital organs (STIs, LF, and schistosomiasis) merit special attention as potential cofactors of HIV transmission, both sexually and vertically.

Trachoma might seem unrelated to HIV since it is a bacterial eye infection. But trachoma is caused by the same bacteria (*C. trachomatis*) as the STI, chlamydia. As much as 70 percent of women and 25 percent of men with chlamydia are asymptomatic (Webster *et al.* 1993). That suggests the possibility that trachoma sufferers are also

infected genitally without the patient or medical personnel being aware of the multiple presentation of the infection. Alternatively, high prevalence of trachoma may be merely a proxy for poor health care, since it is easily treated in its early stages and extracts a very high toll that would not be tolerated in a country with an adequate health system. Consequently, we include the trachoma variable in our model because we think it suggests some important factor worthy of further investigation.[1]

Method

This research extends the work of authors who use cross-national data and multiple regression analysis to estimate the effect of factors thought to drive the HIV epidemic (Over 1998; Bonnel 2000; Stillwaggon 2000; Mahal 2002; Stillwaggon 2002; Drain *et al.* 2004; Deuchert and Brody 2007; Tsafack Temah 2008). All but one of those studies focuses on a specific issue that the author(s) believed especially important: risky sexual behaviors, gender discrimination, human development, income inequality, economic growth, iatrogenic transmission of HIV, and nutrition. Those authors all use the same or similar socio-economic control variables and then add other variables relevant to the specific issue addressed in their studies. The most common control variables used in those studies are income inequality, per capita income (level or growth rate), and urbanization (level or growth rate), proportion of the population that is Muslim, age of the epidemic, a measure of gender discrimination, literacy or school enrollment rates, and one or more binary variables for region. We add two variables that measure cross-border migration because of the importance of migration in the discussion of HIV in southern Africa and another variable that measures the burden of war on the population. In the present study and in our previous research (Sawers *et al.* 2008), we add to the socio-economic variables mentioned above measures of cofactor diseases that are thought to promote the transmission of HIV.

The regional binary variable in our model takes the value of one for the nine countries in southern Africa and zero for other countries. A positive and significant coefficient on the southern Africa variable tells us that HIV prevalence is higher in southern Africa than elsewhere but does not tell us why. Our objective is to explain differences in HIV prevalence among low- and middle-income countries (not just find statistical associations and a high R^2). Location, by itself, is not a policy-sensitive variable. While using a regional binary variable raises the predictive value of the model in a technical sense, it begs the question of what is causing high prevalence in the region. Accordingly, we measure our success in explaining the variations in HIV among countries by how far we can reduce the size of the regression coefficient on the southern Africa variable.

Using data for 85 developing and transition countries,[2] we estimate a number of ordinary least squares multivariate regressions on the log of adult HIV prevalence in 2006.[3] All of the regressions reported here include the socio-economic control variables: a binary variable for the nine countries of southern Africa, the Gini coefficient (the most widely used measure of income inequality), log of per capita income on a purchasing power parity basis, percent of adults who are literate,[4] percent of the population living in urban areas, age of the epidemic, a gender discrimination index that is the ratio of female economic participation to male economic participation, the effect of war measured by war-induced disability-adjusted life years (DALYs) per 100,000 population, percent of the population who are Muslim, workers' remittances as a percent of GDP, and percent of the population who are international migrants. Table 1 lists the source of each of the variables. The first step is to regress only the socio-economic variables on HIV prevalence. Next, we add each cofactor infection variable, one at a time, to a regression with the socio-economic variables. Lastly, we regress all of the cofactor infections together with the socio-economic variables. Estimations of all of the regressions are robust to heteroskedasticity.

Results

In the equation presented in Table 2, only the socio-economic variables are regressed on HIV prevalence. The R^2, that is, the percent of the variance in the log of adult HIV prevalence associated with the independent variables taken together, is 70.4. Six of the eleven independent variables are significant at the 96 percent level or better and two more are significant at the 90 percent level. As expected, the southern Africa variable, the Gini coefficient, the age of the epidemic, war burden, and migrant percent have positive coefficients. The Muslim percent, adult literacy,

and income per capita variables are negatively associated with HIV prevalence. The gender discrimination, urbanization, and remittances variables do not have a statistically significant association with HIV prevalence in the multiple regression.

The regression coefficient on each independent variable in the model tells us the change in the log of HIV prevalence—after accounting for the effects of the other independent variables—when the value of the independent variable changes by 1. Since the southern Africa variable takes the value of either 1 or 0, its coefficient tells us the difference in the log of HIV prevalence between southern Africa and elsewhere after taking into account the effects of the other socio-economic variables in the model. To find the difference in HIV prevalence (in percent) between southern Africa and elsewhere that is not explained by the socio-economic variables, one takes the antilog (with an adjustment for heteroskedasticity[5]) of the coefficient on the southern Africa variable in the multivariate regression.

Average HIV prevalence in southern Africa is 16.2 times the average prevalence in other low- and middle-income countries in the dataset. The regression coefficient on the southern Africa variable is 2.71, which tells us that HIV prevalence in the region not explained by the socio-economic variables is 12.0 times the level in other low- and middle-income countries. In other words, the socio-economic variables "explain" in a statistical sense about 25 percent—[(16.2 – 12.0)/16.2] x 100]—of the southern Africa differential in HIV prevalence.

The next step in the analysis is to add each of the six cofactor infection variables—one at a time—to a multiple regression in which the other independent variables are the same as those used in the regression reported above. Schistosomiasis, lymphatic filariasis, trachoma, chlamydia, and gonorrhea are measured by DALYs per 100,000 population, and malaria is measured by the number of annual cases as a percent of the population.[6] The results of those six regressions are reported in Table 3. All of the cofactor infection variables are positively correlated with HIV prevalence. Five of the cofactor infection variables are significant at the 99.8 percent level or better and one is significant at the 98 percent level. The variables with the most significant correlation with HIV prevalence are schistosomiasis and trachoma.[7]

The six regressions reported in Table 3 do not measure the true importance of each cofactor infection since they are subject to missing variable bias, that is, they do not include the other cofactor infections that are correlated with HIV prevalence. On the other hand, if all of the cofactor infections are put into a regression at the same time, only two remain statistically significant, which is the classic indicator of multicollinearity. (The simple correlation, R, between each pair of the six cofactor diseases ranges between 0.50 and 0.93.) We take two steps to address the collinearity. First, similar diseases are grouped into summary variables. We sum DALYs for gonorrhea and chlamydia to form a new variable, sexually transmitted infections (STIs). Similarly, the sum of DALYs for schistosomiasis and lymphatic filariasis constitutes the variable, worms. This helps but does not eliminate the problem of multicollinearity. For example, the simple correlation between the two composite variables is 0.77. Hence, we also address collinearity among the cofactor infections by measuring their joint statistical significance using the F-test.

The last step of our analysis is to estimate a regression with socio-economic variables and all of the cofactor infections together. Adding the cofactor infections to the equation raises the R^2 to 80.8 percent from 70.4 percent and raises the F statistic for the equation to 78 from 33. Two of the cofactor infection variables (STIs and trachoma) are significant at the 99.9 percent level, but the worms and malaria prevalence variables are not significant. Nevertheless, the four variables are highly collinear and thus competing over the same variance in explaining the log of HIV prevalence. The F statistic for the four cofactor infection variables taken together is 9.6, significant at the 99.99 percent level. That test confirms our hypothesis that the cofactor infections are significantly correlated with HIV prevalence.

The regression coefficient on the southern Africa variable in the equation including all cofactor infection variables is 1.38, which tells us the level of HIV prevalence that is not "explained" by the equation is 3.2 times that of other low- and middle-income countries. In other words, adding the cofactor infections to the model allows us to "explain," in a statistical sense, 80 percent—[(16.2 – 3.2)/16.2] x 100—of the southern Africa differential in HIV

prevalence, substantially more than the model without the cofactor diseases (25 percent).

One can compare the regression coefficient and *t* statistic on the log of per capita income in the regression with only the socio-economic variables in Table 2 to those in the regression that includes the cofactor infections in Table 4. The coefficient on per capita income falls sharply when the cofactor infections are added to the equation. Without the cofactor infections in the model, per capita income is significantly correlated with HIV prevalence at the 96 percent level, but adding the cofactor infections makes the coefficient on per capita income insignificant. That is consistent with our argument that what is important about the association between HIV prevalence and per capita income is the high disease burden of the poor. That the cofactor infections variables cause the per capita income variable to become insignificant is not a statistical artifact produced by collinearity. Per capita income and the cofactor infections are highly collinear (the lowest R between per capita income and each of the six cofactor infections is 0.50), but a multiple regression produces unbiased coefficients even with collinearity.

Limitations

The statistical analysis presented above shows that location by itself accounts for only a fifth of the more than 16-fold difference in HIV prevalence between southern Africa and the rest of the world. Two points should be emphasized. The fact that we have not explained all of the southern Africa differential means that our model suffers from missing variable bias. The southern Africa variable is without causal content. If it remains statistically significant in the regression equations, it means that something else sets southern Africa apart from the rest of the world that we have not yet discovered or found a way to measure correctly. Examples of variables that one would like to add to the equation include condom use, a measure of injection safety practices in medical settings, various measures of risky sexual behavior[8], prevalence of circumcision (not just percent Muslim), and so on.

A second specification error is perhaps more serious. All of the independent variables when regressed on HIV prevalence in a bivariate regression have statistically significant coefficients and seven of them remain statistically significant in a multiple regression using all of the variables together. While there are grounds for believing that each one of those seven variables is causally related to the spread of HIV, the statistically significant correlation with HIV prevalence does not prove that causal relationship. The correlation of any of the independent variables with HIV prevalence may be spurious in the sense that a better specified model would eliminate its correlation with HIV prevalence. An illustration of how that works is what happened to per capita income when the cofactor infections are added. Low income at first seemed to raise HIV prevalence, but when the model was more fully specified by adding cofactor infections, it was seen that income by itself is unimportant. What happened to per capita income points to the principal weakness of ecological studies of this sort. The variables that are significant in the equations presented in this paper may be only standing in for what is really driving the HIV epidemics. Biomedical studies have demonstrated the mechanisms that increase HIV transmission for most of the cofactors used here. Ecological studies, however, can suggest lines of inquiry that might result in additional evidence through clinical trials.

Discussion

In retrospect, it is clear that, in southern Africa, HIV was an epidemic "waiting to happen" (Marks, 2002). The factors that made southern Africa an ideal setting for HIV to flourish—that perfect storm—are numerous. In many ways, southern Africa has much in common with the rest of sub-Saharan Africa. Table 5 presents means of the variables used in this analysis for four groups of countries—three regions within sub-Saharan Africa and a fourth group of low- and middle-income countries outside sub-Saharan Africa. The burdens of war are highest in mid Africa, but southern Africa and the rest of sub-Saharan Africa have two and three times, respectively, the level in developing and transition countries outside sub-Saharan Africa. Excluding South Africa, the proportion of the population who live on less than two dollars a day (not shown in Table 5) is the same in southern Africa (71 percent) as elsewhere in sub-Saharan Africa. Other low- and middle-income countries average 40 percent of people living on less than two dollars a day. It should come as no surprise, then, to find that the cofactor diseases analyzed in this study (most of them diseases of poverty) are found at roughly similar levels throughout sub-Saharan Africa, but at far lower levels outside Africa. STIs are epidemic around the world, but their impact in sub-Saharan Africa is nearly

triple the level in other low- and middle-income countries. There is an even greater difference for the other cofactor diseases. To the extent that war, poverty, and the diseases that poverty promotes predispose one to HIV, then southern Africa is similar to the rest of sub-Saharan Africa and much more vulnerable than other low- and middle-income countries.

Other factors that arguably promote the spread of HIV set southern Africa apart from the rest of sub-Saharan Africa but make it more similar to other developing and transition countries. A number of authors have noted the positive correlation of educational attainment and literacy with HIV (Brent 2006). With regard to adult literacy and the ratio of female to male literacy (not shown in Table 5), southern Africa is more like low- and middle-income countries outside of sub-Saharan Africa than within. In addition, the two measures of migration used in this study show that southern Africa is more like non-African countries than the two other regions of sub-Saharan Africa.

Income inequality puts southern Africa in a category by itself. Gini coefficients in southern Africa are the highest in the world, but Gini coefficients in the rest of sub-Saharan Africa average about the same as in low- and middle-income countries elsewhere. Furthermore, southern Africa on average has the lowest percentage of the population who are Muslim than any of the four groups of developing and transition countries in Table 5.

So why does the perfect storm occur in southern Africa? Why has HIV reached its highest levels there—one-quarter of the adult population in the most gravely affected countries? Perhaps what is unique about southern Africa is that it scores badly on so many of the factors that promote HIV epidemics. It is the interaction of all of those factors that produced the crisis. HIV is opportunist, but it is a relatively weak pathogen. HIV prevention policy is founded on the assumption that risky sexual behavior, untreated STIs, and lack of circumcision drive the epidemic. Nevertheless, survey data show that by almost all measures of risky sexual behavior, people in the United States and other affluent countries far outdo the average African, including in southern Africa. (See Stillwaggon 2006 and Wellings *et al.* 2006 for extensive data on sexual behavior worldwide.) Recent explanations of the high rates of HIV in southern Africa emphasize the importance *concurrent* sexual relationships, but adding the adjective does not change the fact that risky sex, concurrent or otherwise, is more prevalent in developed countries than in the poor countries of Africa. What might be most important about concurrent sexual relationships in Africa are the elevated levels of viral load in co-infected persons in long-term concurrent relationships. In affluent countries, however, the impact of risky sex and consequent STI epidemics is dampened by early diagnosis and treatment. Affluent populations are not burdened by untreated, multiple bacterial, viral, and parasitic diseases that undermine the immune system and open pathways for the HIV virus. They are relatively well nourished and thus less susceptible to most infectious diseases, including sexually transmitted ones. Public and private health systems keep the population far more healthy than in poor countries.

Conclusions

AIDS policy has failed to stem the spread of HIV in southern Africa and neighboring countries. Policy makers have targeted too few factors in their attempts to stem the epidemic. In countries with few interacting variables, such as in North America and Europe, behavioral interventions have been quite successful in reducing new infections. Nevertheless, North America, Western Europe, and in fact, most of the rest of the world give us limited information about the true nature of HIV. We get the most information about this opportunist pathogen from southern Africa, where the intersection of social, economic, and ecological factors has allowed the explosive advance of HIV.

If that analysis is correct, then HIV prevention policy must address the multitude of health issues that plague Africa. Experimentation with deworming and other interventions for endemic diseases is a pragmatic, ethically sound, and relatively inexpensive strategy. Such interventions have substantial beneficial outcomes on their own, in better health, better school performance, and higher productivity. And they allow for structured learning about disease interactions at very low cost, using medicines that are safe and effective. Moreover, the improvement in overall wellbeing in itself promotes healthier behaviors in healthier people.

AIDS policy is paralyzed by its crisis mode. Policy makers need to step out and try something in addition to the

behavioral interventions ("ABC" plus circumcision). That means some HIV prevention funds should be spent on deworming, sanitation, and safe water. Antiretroviral treatment protocols should include deworming and nutrition, just as they already include treatment for tuberculosis. Of the numerous economic, biological, and social determinants of HIV epidemics, treating cofactors infections may be the most policy-sensitive and least expensive interventions and have the most immediate return on investment.

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Tables

Table 1. **Sources of Data**

Variable	*Definition*	*Source*
Adult HIV prevalence 2006	Percent of persons aged 15 to 49 with HIV	UNAIDS reports, 2006 and 2007
Gini coefficient	Measure of income inequality, 2004	World Development Indicators
Income per capita	Per capita GDP (PPP basis), 2003	World Development Indicators
Urbanization	Percent of population in urban areas, 2003	World Development Indicators
Age of the epidemic	Years since first diagnosis of HIV	World Bank, *Confronting AIDS*, 1997, 318–24
Adult literacy	Percent of adult population who are literate, 2003	Human Development Indicators, 2005
Female participation ratio	Ratio of female economic activity rate to male economic activity rate, 2005	Human Development Indicators, 2007
War burden	War-induced DALYs per 100,000 population, 2002	WHO, Global Health Atlas, 2004
Muslim percent	Percent of the population who are Muslim	CIA World Factbook
Migrants	International migrants as a percent of population	World Development Indicators
Remittances	Workers' remittances as a percent of GDP	World Development Indicators
STI, worms, trachoma	DALYs (Disability-adjusted life years) for each disease per 100,000 people, 2002	WHO, Global Burden of Disease Estimates, 2004
Malaria prevalence	Cases as a percent of population	WHO, Global Health Atlas, 2008

Table 2. **Multiple Regression Analysis with socio-economic variables and without cofactor diseases.**

Variable	*coefficient*	t *statistic*	*probability*
Southern Africa	2.7076	7.37	0.000
Gini Coefficient	.0336	2.36	0.021
Income per capita (log)	–.5467	– 2.10	0.039
Urbanization	.0023	0.28	0.779
Age of epidemic	.1404	3.37	0.001
Adult literacy	–.0136	–1.71	0.091
Female participation ratio	.0109	1.52	0.133
Muslim	–.0082	– 2.66	0.010
War burden	.0003	1.67	0.100
Remittances	.0200	1.14	0.259
Migrants	.0862	3.92	0.000
Constant	–.2586	– 0.14	0.889

Table 3. **Multiple Regression Analysis: each cofactor infection with socio-economic variables**

Variable	*coefficient*	t *statistic*	*probability*
STI (log)	1.2568	4.71	0.000
Chlamydia	.0266	5.29	0.000
Gonorrhea	.0212	5.94	0.000
Worms	.0055	5.46	0.000
Schistosomiasis	.0101	6.74	0.000
Lymphatic Filariasis	.0047	2.39	0.019
Trachoma	.0117	7.02	0.000
Malaria	.0675	2.75	0.008

Table 4. **Multiple Regression Analysis with socio-economic and cofactor diseases.**

Variable	*coefficient*	t *statistic*	*probability*
Southern Africa	1.3801	4.32	0.000
Gini Coefficient	.0371	2.87	0.005
Income per capita (log)	.1179	0.54	0.590
Urbanization	–.0011	–0.13	0.895
Age of epidemic	.0557	1.36	0.179
Adult literacy	.0081	0.98	0.329
Female participation ratio	.0044	0.57	0.570
Muslim	–.0111	–4.15	0.000
War burden	–.0000	–0.31	0.754
Remittances	.0590	3.36	0.001
Migrants	.0774	3.09	0.003
STI (log)	.6329	2.99	0.004
Worms	.0013	1.21	0.231
Trachoma	.0053	3.58	0.001
Malaria prevalence	.0125	1.10	0.277
Constant	–8.8583	–4.37	0.000

Table 5. **Means of variables by region**

Variable	*southern African countries*	*mid African countries*[a]	*other sub-Saharan African countries*	*other low- and middle- income countries*
HIV prevalence in percent of adult population	19.3	5.3	1.8	0.6
Gini Coefficient	.553	.429	.431	.424
Income per capita PPP in U$S	4391	1171	1321	5205
Urbanization, percent of population	33.8	30.5	36.0	52.4
Age of epidemic in years	19.6	19.7	18.2	17.8
Adult literacy, percent of population	73.2	67.1	35.5	85.0
Female economic participation ratio	.721	.824	.727	.663
War burden DALYs per 100k population	133	787	187	60
Muslim, percent of population	9.8	20.2	61.9	19.8
Remittances as percent of GDP	5.9	2.8	2.8	5.2
Migrants (international), percent of population	3.1	1.3	4.5	3.3
STI DALYs per 100k population	275	284	287	100
Worms DALYs per 100k population	363	589	604	46
Trachoma DALYs per 100k population	148	192	189	10
Malaria cases as percent of population	11.8	16.7	8.4	0.4

[a] Congo, Rwanda, Kenya, Gabon, Nigeria, Cameroon, Burundi, Democratic Republic of the Congo, Angola, Uganda, Central African Republic, Tanzania

Endnotes

[1] We have provided a rationale for a long list of independent variables in our model, but we should point out a variable that we are not including. Numerous colleagues have suggested we add clade to our analysis. The subtype of HIV 1 that is dominant in southern Africa is clade C, but that subtype is relatively rare is most other countries. That fact suggests to many observers that clade C is more fit for transmission than other clades and several contributors to the virological literature have speculated just that (Ping *et al.* 1999; Spira *et al.* 2003; Rainwater *et al.* 2005; Tebit *et al.* 2007). There is, however, little evidence to confirm that suspicion. One study finds that vaginal shedding in pregnant women is greater with clade C (John-Stewart *et al.* 2005), but another study found no difference in MTC transmissibility of the virus among different clades (Tapia *et al.* 2003). A third study finds no evidence of different fitness for heterosexual transmission of the different clades (Morison *et al.* 2001). When we add a binary variable for clade C to our regression, the coefficient is insignificant and negative, not positive as

has been speculated. Nevertheless, except for three countries (China, India, and Nepal), the clade C variable and the southern Africa variable are the same. The negative sign on the clade C coefficient is most likely a result of spurious interaction with the southern Africa variable and is not an indication that clade C is less fit for transmission. Hence, we do not include the clade C variable in our model.

[2] Countries with per capita income in 2003 of less than U$S12000 on a purchasing power parity basis were included in the analysis. Rather than imputing missing values, countries with missing data were dropped from the analysis, leaving a data set of 85 countries. The present study follows established practice by using the log form of HIV prevalence, per capita income, and STIs, which reduces the influence of outliers and generally leads to more robust and efficient estimates when analyzing highly skewed variables such as these.

[3] It would have been preferable to use HIV incidence to describe how the epidemic is unfolding. Prevalence includes historical information and is confounded by greater survival in countries with effective antiretroviral delivery programs. But incidence data are generally not available.

[4] All measures of literacy and school enrollment for our sample are highly correlated and perform similarly as regressors. In our sample, the adult literacy rate and the female-to-male literacy ratio are virtually identical in a statistical sense (with a simple correlation of 0.93). Using the latter variable, however, gives the misleading impression that the variable can measure gender discrimination; hence the present study uses the more straightforward variable, adult literacy, to avoid that confusion.

[5] The coefficient on the southern Africa variable estimates the difference between the mean log HIV prevalence in southern Africa compared to other countries in the data set. In order to convert this to a statement about the difference in mean HIV prevalence (as opposed to mean log prevalence), one must take the antilog of that coefficient, but also correct for the non-zero mean of the antilogged error terms. That correction takes into account the heteroskedasticity in error terms between southern Africa and the other countries and yields a close approximation of the actual ratio of means. The ratio of the predicted means is the antilog of [the regression coefficient on the southern Africa variable plus (half the difference between the variance of the regression residuals in southern Africa and the variance of the regression residuals outside of southern Africa)]. Thomas Hertz devised this calculation.

[6] Because the vast majority of deaths from malaria are in infants, the DALYs associated with malaria are extremely high. That measure, however, does not reflect the burden of malaria among sexually active adults, unlike the DALYs from other diseases. We have therefore use malaria prevalence as our measure.

[7] We approached this research aware of evidence linking HIV transmission with ulcerative or inflammatory STIs, schistosomiasis, lymphatic filariasis, and malaria and we found those infections to be strongly correlated with HIV prevalence. If

many other infections without plausible connection to HIV were also highly correlated with HIV, we would suspect that HIV's statistical correlation with our hypothesized cofactor infections was spurious. Accordingly, we tested a total of 18 separate infectious diseases (excluding childhood diseases and the opportunistic infections associated with AIDS) by regressing each one on HIV prevalence without other potential cofactors but with the socio-economic variables. In that test, all of the diseases on our original list were correlated with HIV prevalence at the 98 percent or better confidence level. Note also that, at the 95 percent confidence level, we would expect 5 percent of the 18 infections we tested (or no more than one) to be significantly correlated with HIV prevalence by chance alone. Nevertheless, seven of the 18 potential cofactors are significant at the 98 percent level, meaning that they are unlikely to have found their way onto our list purely by chance.

[8] The most widely available measure of risky sexual behavior is age at first sex for females, but that variable has not been measured for 39 of the 85 countries in our dataset. Twenty-two of the 46 countries for which there are such data are in Africa, so those 46 countries are not a representative sample of low- and middle-income countries. Sexual behavior surveys reflect a bias in data collection common in AIDS research. The assumption that the AIDS epidemics in sub-Saharan Africa result from something distinctive about African sexuality influences the decision to collect data on sexual behavior in African countries more than in other parts of the world. For a discussion of bias in data selection in Africa, see Stillwaggon 2003.