Violent Conflict and the Spread of HIV/AIDS in Africa

Zaryab Iqbal Pennsylvania State University
Christopher Zorn Pennsylvania State University

It has been widely speculated that violent conflict acts as a key contributor to the transmission of human immunodeficiency virus (HIV). Yet to date no empirical examination of the conflict-HIV relationship has been conducted. Drawing on work in political science and public health, we set forth a theoretical framework for understanding this potential relationship and go on to present data on the spatio-temporal dispersion of HIV/AIDS in 43 African countries during the period from 1997 to 2005. We then assess the association between domestic and international conflict and levels of HIV/AIDS infection while controlling for a range of other influential factors. Our analyses support a clear positive relationship between both international and domestic conflict and climbing HIV/AIDS prevalence, as well as significant palliative effects for education and economic development on the incidence of HIV/AIDS.

Since the first cases of human immunodeficiency virus (HIV) and Acquired Immune Deficiency Syndrome (AIDS) were reported in 1981, over 25 million people have lost their lives to the disease. In the year 2007 alone, 2.7 million people around the world contracted HIV, 370,000 of whom were children (UNAIDS 2008). During the past three decades, the spread of the AIDS epidemic has exceeded nearly all predictions, and Africa has emerged as the region with the most harrowing HIV prevalence. Sub-Saharan Africa, in particular, has captured the world's attention due to its shockingly high HIV rates. In 2007, 35% of all new infections worldwide were recorded in sub-Saharan Africa; while the region holds just over 10% of the world's population, it is home to 67% of all people living with HIV/AIDS worldwide (UNAIDS 2008, 32).

Among the factors often cited as contributing to the international HIV/AIDS epidemic is armed conflict. Speaking at the United Nations General Assembly Special Session on HIV/AIDS, Atoki Ileka, Chairman of the Delegation from the Democratic Republic of Congo, noted that HIV/AIDS "will continue to worsen with the state of war that prevails in my country, the movement of people, such as refugees and terminally displaced persons, the chronic poverty of the population, food and security, (and) rape committed by men in uniform in the territories under occupation" (United Nations 2001, 62). As this statement suggests, it is commonly understood that violent conflict degrades the health of populations in a myriad of ways: in addition to combat-related casualties, collective violence can encourage population movements, disrupt access to health care services, destroy or damage critical infrastructure, limit access to food and safe drinking water, and leave populations vulnerable to infections and disease. Over and above these general influences, it is widely believed that, for a host of reasons, conflict within or between states results in conditions that are particularly favorable to the spread of HIV; as one commentator stated flatly, "(T)he HIV virus . . . spreads most rapidly and pervasively as a result of military conflict" (Copley 1999, 4).

But while the literature examining questions related to HIV and AIDS is vast, work addressing the relationship between violent conflict and HIV/AIDS has failed to take us very far in understanding that relationship. Far and away the most common analyses are descriptive case studies (e.g., Gorbach et al. 2002); also numerous are prescriptive papers addressing specific policy concerns (e.g., Miles 2003). Yet, to date, there have been no large-scale empirical analyses of the conflict-HIV relationship. In some respects, this is unsurprising, as the challenges inherent in such an undertaking are substantial; any accounting of the influence of violent conflict on HIV/AIDS must address both the numerous ways in which conflict can contribute to HIV's spread and the

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spatial and temporal aspects of the disease. An important consequence of this lack of empirical analysis has been the relatively uncritical acceptance of the "securitization" of HIV/AIDS; while important criticisms of that securitization exist (see, e.g., Elbe 2006; McInnes 2006), neither its proponents nor its critics have undertaken a broad-ranging empirical analysis of the HIV-security link.

Here, we examine the relationship between conflict and the spread of HIV/AIDS in Africa. We do so by assessing the prevalence of HIV in Africa in light of relevant demographic, political, and economic factors—including the presence of armed conflict—during the eight-year period from 1997 to 2005. In doing so, we seek to answer a series of questions. What are the patterns of HIV/AIDS infections in Africa, both across space and over time? What are the key influences on those infection rates? Most important, what is the net effect of violent conflict, both domestic and international, on HIV/AIDS prevalence?

The Etiology of HIV/AIDS in Africa

The dynamics of HIV/AIDS's diffusion in Africa differ significantly from those in the West. In the United States, for example, transmission of HIV occurs most frequently through homosexual contact and intravenous drug use (UNAIDS 2006, 53). As a result, HIV has historically been disproportionately prevalent among members of certain minority groups (such as the gay community) and among intravenous drug users. In Africa, by contrast, "the prime mode of transmission is through heterosexual contact, with minor parts played by homosexual contact, mother-child transmission, blood transfusions, and injecting drug use" (Akeroyd 2004, 90). HIV/AIDS in Africa is therefore both more widespread and less confined to members of particular groups, and its spread has thus been more strongly associated with broader economic, social, and political conditions.

Among these factors is armed conflict. As we noted at the outset, the conventional wisdom is that violent conflict contributes to the spread of HIV/AIDS, particularly in the developing world. A 2001 United States Institute of Peace report summarizes the consensus in stark terms, stating that "(N)o one denies the role of conflict in the spread of the virus" (USIP 2001, 8). Scholars agree; Jewkes notes that "(W)ar in Africa is generally assumed to be one of the factors fuelling the HIV epidemic" (2007, 2140). And Copley (1999) attributes substantial responsibility for the early spread of HIV to the wars in Angola during the 1970s.

At the same time, however, empirical work supporting these assertions has been nearly nonexistent. The few descriptive analyses done so far offer, at best, contradictory conclusions about the link between HIV/AIDS and violent conflict. Dizolele, for example, notes that "Botswana—a stable, democratic, and rich nation—has the highest HIV prevalence rate among pregnant women. South Africa, arguably the most powerful country in Africa, is home to 5.3 million HIV-infected people—the world's largest" (2004). And a recent meta-anaysis (Spiegel et al. 2007) finds essentially no relationship between conflict and HIV prevalence in seven African countries. Reconciling these seemingly contradictory results requires first that we consider the potential causal linkages between HIV and violent conflict and that we do so mindful of the numerous other factors influencing the transmission of the disease.

Conflict and HIV

Students of international relations have recently begin paying greater attention to the nexus between conflict and health-related phenomena (Ghobarah, Huth, and Russett 2003, 2004a, 2004b; Iqbal 2006, 2010; Li and Wen 2005; McInnes 2009; Murray et al. 2002; Plümper and Neumeyer 2006). These scholars note that conflict is itself a significant public health problem and that conflict-related health externalities occur through a variety of mechanisms.[1] In considering the relationship between armed conflict and HIV/AIDS, we posit that violent conflict may contribute to rising HIV prevalence through two broad means. First, conflict increases the likelihood that uninfected populations will come into contact with infected ones. Second, and related, violent conflict raises the probability that HIV-transmitting events will occur.

As Decosas et al. note, "HIV, like any other infection spread from person to person, will follow the movement of people" (1995, 826). The movement of soldiers necessitated by large-scale armed conflict, as well as conflict's tendency to displace civilian populations, both serve to facilitate contact between infected and uninfected populations. Soldiers in wartime often act as vectors of disease (Ghobarah, Huth, and Russett 2003), and some estimates suggest that HIV prevalence in Africa is higher among both regular soldiers and insurgents than among civilians

[1]Among these, however, only Ghobarah, Huth, and Russett (2004b) directly examine the conflict-HIV relationship, finding that civil wars have a consistently positive impact on the loss of disability-adjusted life years due to HIV/AIDS during 1999.

(Miles 2003). In 1997, for example, U.N. estimates put the continent-wide HIV prevalence rate in Africa at 9.5% in the general population, but 27% among the armed forces (UNAIDS 2002).[2] The reasons for these elevated rates are manifold; Elbe notes that soldiers "are of a sexually active age; they are highly mobile and away from home for long periods of time; they often valorize violent and risky behavior; they have greater opportunities for casual sexual relations; and they may seek to relieve themselves from the stress of combat through sexual activity" (2002, 163).

Beyond the armies themselves, a signature trait of modern warfare is its displacing effect on local noncombatant populations. A number of recent studies have documented the strong relationship between violent conflict—particularly civil war—and refugee outflows (Davenport, Moore, and Poe 2003; Moore and Shellman 2004; Schmeidl 1997). Additional work has noted the more specific correlation between the presence of such migrant populations, including migrant labor (Lurie et al. 2003) and refugees (Decosas and Adrien 1997), and escalating HIV prevalence rates. The intuition is straightforward: the UNHCR has noted that "[r]efugees, asylum seekers and internally displaced persons are are [*sic*] vulnerable to HIV infection as conflict and displacement expose them to poverty, family disintegration, social disruption and increased sexual violence" (UNAIDS 2008, 6). Hence, in the same way that soldiers can act as vectors for transmission of the disease, so too can migrant and refugee populations carry the disease to formerly uninfected groups.

Thus, through both soldiers and refugees, the effect of armed conflict is to increase the intermingling of infected and noninfected populations. Over and above this direct effect, however, violent conflict may also alter social conditions in a number of interrelated ways that combine to increase the incidence of events that transmit the virus. Three of these changes stand out most starkly: violent conflict's negative effect on wealth and development, its negative influence on public health infrastructure, and its aggravation of both predatory and commercial sexual activity.

The adverse effects of conflict on economic development, growth, and wealth are well documented (e.g., Collier 1999), as is its impact on trade (Keshk, Pollins, and Reuveny 2004). Equally clear is the structural connection between poverty and both public health in general and HIV/AIDS in particular (Parker 2002). As a major indicator of development, higher levels of wealth and trade signal the presence of opportunities to limit HIV proliferation through better access to health care facilities, the availability of health and AIDS-prevention education, and a reduction in the poverty that compels women to become sex workers or engage in sexual activity in order to meet basic survival needs. Conversely, many of the effects of conflict that contribute to the spread of HIV/AIDS may be mitigated by higher national income levels. A similar dynamic holds for international trade; such activity, in addition to its wealth-creating potential, serves as a source of government revenue and helps build relations with the international community. Such relationships may in turn allow for greater levels of health-related involvement by international groups, and for higher levels of public health-related foreign assistance.[3]

The inevitable degradation of the public health system that accompanies large-scale violent conflict also contributes to conflict's influence on the spread of HIV. Recent work suggests that public health spending is among the first social programs to suffer in the face of the "guns vs. butter" trade-offs that inevitably result from war (Ghobarah, Huth, and Russett 2004a). More broadly, the social and political decline often caused by violent conflict makes prevention and treatment more costly and difficult; social institutions become less effective, law and order breaks down, and public health provisions make a steady move to the bottom of the government's priority list (Iqbal 2010). In the face of these changes, it would be surprising not to find an increasing prevalence of HIV/AIDS.

Finally, modern warfare is often associated with increases in high-risk sexual behavior among both armed forces and civilian populations—and by corresponding changes in the sexual status of men and women—which result in a higher incidence of HIV transmission. One such change is rape: the conflict-related breakdown of social control, together with wartime policies that encourage rape and other forms of sexual coercion as a tactic of war, increase the potential for military-civilian transmission of the

[2]Note, however, that more recent studies have questioned whether HIV prevalence is, in fact, higher among members of the military than in the (age- and gender-)comparable civilian population. Whiteside, de Waal, and Gebretensae (2006) offer a summary of this research; see also McInnes (2006).

[3]We note, however, that an alternative view suggests a positive relationship between economic openness and HIV. To the extent that openness leads to greater interaction of HIV-infected individuals across national borders, one might in fact find higher levels of infections in more open countries. In one example of this, a number of small-scale studies point to the importance of long-haul truck drivers in the spread of HIV in southern and eastern Africa (e.g., Bwayo et al. 1994; Gysels, Pool, and Bwanika 2001).

disease. A second, related phenomenon is the sex industry; as one commentator has noted, "(E)verywhere the military goes, in wartime or in peacetime, the CSI (commercial sex industry) is sure to follow" (Jordan 2000, 536).[4] Such individuals constitute an important vector for the disease; one U.N. report estimates that HIV prevalence rates for sex workers in sub-Saharan Africa are as high as 73% in some nations (UNAIDS 2003). As a result of these dynamics, estimates by UNAIDS suggest that deployment in conflict-ridden areas can result in HIV/AIDS rates in military personnel that are two to five times higher than those among civilians in areas that do not have violent conflict (Fleshman 2001).

Taken together, these influences paint a picture of the effect of violent conflict on HIV/AIDS that is as grim as it is multifaceted. Armed conflict brings together soldiers and noncombatants in a context in which adequate health care and preventive education are likely to be sorely lacking. Faced with a breakdown of the rule of law—and, in some cases, encouraged by their superiors—soldiers both victimize unwilling women and make liberal use of the services of commercial sex workers. At the same time, the poverty and desperation that accompanies conflict forces women and girls to become prostitutes or engage in sexual relations out of a need for food or shelter. The latter phenomenon may be especially prevalent among refugees, whose conflict-related poverty and insecurity are even higher than the indigenous population, and for whom the unavailability of preventive health care is often even more pronounced.

International and Domestic Conflict

Our discussion of the direct and indirect effects of conflict on HIV further suggests that the influence of armed conflict on HIV/AIDS will vary as a function of the nature of the conflict itself. From our perspective, the most important such distinction is between violent conflicts of a domestic nature (including civil wars) and international conflicts. On the one hand, we might expect the direct effects of conflict on HIV to be greater for international conflicts. As we noted above, transmission due to the intermingling of soldiers and civilians is often more common when soldiers are away from their home countries for long periods of time.

Conversely, the indirect influences of conflict on HIV/AIDS—in particular, its effects through wealth, health spending, and so forth—ought to be more pronounced in instances of civil war. Such wars are often characterized by localized combat and widespread destruction of infrastructure; in addition, because all the fighting occurs in the territory of one state, the incidence of conflict-related externalities such as HIV is likely to be more prevalent than in wars that are at least partially fought abroad.[5] At the same time, the fighting in most civil wars is characterized by irregular troops, guerilla actions, and substantial interaction between rebel combatants and civilians, with the result that such conflicts' direct effects on HIV transmission will be substantial as well. Accordingly, we expect that the relative impact of domestic conflict on HIV/AIDS will be greater than that for international conflicts; in our models below, we assess these relative effects empirically.

Taken as a whole, our discussion of conflict and HIV/AIDS suggests a number of expectations about the relationships among armed conflict, HIV/AIDS, and other state-level factors; these are outlined in Figure 1. For example, we expect that, all else equal, *wealthier* and more *open* states will experience lower HIV prevalence than poorer ones, as will those with greater *health care expenditures*. Similarly, we expect that states hosting large numbers of *refugees* are likely to experience higher levels of HIV/AIDS. Finally, our model suggests that, over and above the indirect effects of conflict through these other mechanisms, the occurrence of violent conflict itself will lead to higher HIV prevalence rates, and that this effect will be more pronounced for domestic violence than for international conflicts.

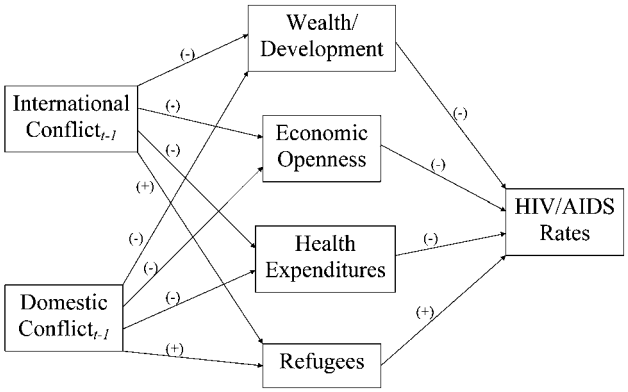
Other Influences

In addition to conflict-related influences on HIV/AIDS, existing studies outline a range of other factors that influence levels of HIV/AIDS. Population is one; existing research has shown that higher levels of *population density* are likely to raise the prevalence of HIV infection. Since the most common mode of HIV transmission is through sexual contact, more densely populated areas are likely to experience higher spread of HIV, as the levels of interaction among people in such areas typically exceed those in more sparsely populated regions. In fact, all else equal, HIV/AIDS prevalence rates tend to be higher in urban areas (Buvé, Bishikwabo-Nsarhaza, and Mutangadura 2002), and the most densely populated

[4]For example, a 2004 survey revealed that over 32% of soldiers in the Nigerian navy had engaged in sexual contact with a sex worker, and nearly half of them had not used condoms (UNAIDS 2005).

[5]In contrast, international wars are not by definition fought on the home territory of all nations involved, which means that the indirect effects discussed above may be less pronounced in such conflicts.

FIGURE 1 War and HIV/AIDS: A Conceptual Framework



states of sub-Saharan Africa suffer from the most severe rates of HIV prevalence.

We expect *religion* to be another significant influence on the incidence of HIV/AIDS. In general, communities in which sexual behaviors are generally governed by strict religious standards that prohibit casual sexual relationships are likely to experience lower rates of sexually transmitted infections (e.g., Allain et al. 2004). In Africa, this dynamic is most prevalent among followers of Islam, among whom religious and cultural norms against premarital and extramarital sexual relationships are strong. Numerous studies at both the aggregate (e.g., Drain et al. 2004) and individual (Rakwar et al. 1999) levels have shown a negative association between Islam and HIV prevalence. Accordingly, we expect HIV/AIDS levels to be lower in predominantly Muslim countries.

Yet another important factor is *education*; over and above the generalized effects of wealth and development, education plays a key role in HIV/AIDS prevention by enabling people at risk of acquiring the disease to obtain relevant information and resources. Educated men and women are better able to make informed decisions regarding safer sexual behavior and contraception, and the effectiveness of HIV/AIDS prevention programs is significantly enhanced by literacy (Peterman, Lindsey, and Selik 2005). Education also negatively affects the spread of HIV through an increase in economic opportunity: women with higher levels of education, for instance, are likely to delay pregnancy or have fewer children in order to engage in gainful employment outside the home. Education, therefore, has the combined effect of reducing high-risk sexual behavior and lowering fertility rates (Appleton 2000), which is important since a growing number of children are born HIV-positive in Africa. Finally, education is also likely to influence men's behavior in terms of safer sexual practices; for example, Ukwuani, Tsui, and Suchindran (2003) find a positive relationship between education and condom use among men in Uganda and Tanzania, and analyses by the Global Campaign for Education suggest that 700,000 cases of HIV/AIDS in young adults could be prevented each year by universal primary school education (Global Campaign for Education 2004).

We also consider the possible effects of regime type, for several reasons. A number of studies have linked the presence of democratic government to more positive health outcomes (e.g., Przeworski, Cheibub, and Limongi 2000; Sen 1981), and Ghobarah, Huth, and Russett's (2004a) work demonstrates a clear link between levels of democratic governance and health spending. More broadly, it is widely acknowledged that democratic states tend to be more responsive to the needs of their populations (e.g., Lake and Baum 2001; Olson 1993). For example, Bueno de Mesquita et al. (2003) posit and demonstrate that as the size of the winning coalition for leadership selection increases, the effectiveness of private goods as a means of retaining power decreases; since democratic leaders are brought into power by a larger number of voters, they tend to focus on provision of public goods that benefit the entire society. All of these points suggest that democracies will, *ceteris paribus*, be both better positioned to combat the factors that facilitate the spread of HIV and more likely to do so given the opportunity.[6] Thus, we expect that more *democratic* states will generally exhibit lower levels of HIV/AIDS infection than their autocratic counterparts.

The Spatial Dispersion of HIV/AIDS in Africa

As we noted in the introduction, Africa—and particularly sub-Saharan Africa—has been at the epicenter of the international HIV/AIDS epidemic for more than two decades. In considering questions about the disease in general, and the nexus between conflict and HIV more specifically, a key factor is space. At the individual level, direct transmission of HIV requires proximity; this in turn suggests that individual cases of HIV/AIDS will tend to cluster geographically (e.g.,

[6]Note, however, that we do not mean to suggest that democracy is either necessary or sufficient to ensure an effective government response to HIV/AIDS. In fact, scholars have long recognized that the higher incidence of HIV infection among members of historically marginalized groups (including homosexuals and racial and religious minorities) has limited the responsiveness of even democratic governments to the disease (Altman 1988).

Tanser et al. 2000). Similarly, aggregate-level studies of transmission dynamics confirm a strong spatial character to the disease (Radkhoshnoud 2002) and indicate that factors relating to the spread of the disease have a distinctly spatial character (Cliff and Smallman-Raynor 1992). These traits lead us to expect that aggregate HIV/AIDS levels will tend to be spatially homogenous as well. It is well-understood that spatial dependence poses potentially serious threats to inference (Cressie 1993); as a result, it is important to begin our analysis with an initial assessment of the spatial distribution of HIV/AIDS prevalence rates in Africa during the period we study.

Our data on HIV/AIDS prevalence are provided by the United Nations UNAIDS/WHO Working Group on Global HIV/AIDS and STI Surveillance, a joint initiative of the United Nations Programme on HIV/AIDS (UNAIDS) and the World Health Organization. The UNAIDS/WHO *Report on the Global HIV/AIDS Epidemic 2002, 2008* (UNAIDS/WHO 2002) provides data on national adult HIV prevalence rates—that is, the percentage of the adult population living with HIV—for odd-numbered years between 1997 and 2005; these data constitute the primary focus of our study. In spite of continuous efforts by national and international agencies to improve the quality of data on HIV/AIDS, there are currently no exact numbers available for the incidence of the disease in most countries (see Walker et al. 2004). HIV/AIDS prevalence rates compiled by UNAIDS/WHO are therefore estimates of the number of people living with HIV/AIDS in specific countries. These estimates are based on a number of surveillance methods, including data from both national surveys and on pregnant women attending antenatal clinics in urban and non-urban areas. Data from antenatal clinics avoid some of the biases inherent in population-based surveys due to factors such as nonresponse and absenteeism (Boerma, Ghys, and Walker 2003; Schwartlander et al. 1999), though they also raise issues regarding re-weighting to account for age- and gender-based selection. The *Report on the Global HIV/AIDS Epidemic* provides low- and high-prevalence estimates; we use the mean of these estimates in our analyses.[7]

Overall, HIV infection levels in Africa are significantly higher than those for the rest of the world; the average rate of 7.4% in sub-Saharan Africa is sharply higher than that for North America (0.6%), Latin America (0.6%), or South and Southeast Asia (0.6%; UNAIDS 2004). Over the eight-year period that we examine, HIV rates in Africa show an initial upward trend, followed by a leveling off; moreover, the extent of this increase varies significantly among countries (Asamoah-Odei, Garcia-Calleja, and Boerma 2004). Even more important, the pattern of that variation has a significant spatial component, as seen in the left panel of Figure 2. In particular, the southern part of the continent suffers from significantly higher rates of HIV infection than states in the north. In fact, southern Africa is the region most severely affected by the AIDS epidemic in the world; to take one example, in South Africa, approximately 5.7 million people—one of every nine persons in the country—are living with HIV (UNAIDS 2008, 40). Other sub-Saharan countries—including Botswana, Zimbabwe, Namibia, and Swaziland—continue to experience high HIV/AIDS prevalence rates. In contrast, countries in north Africa exhibit both low and generally stable HIV/AIDS rates, while countries in central and west Africa vary significantly in both prevalence rates and changes over time.

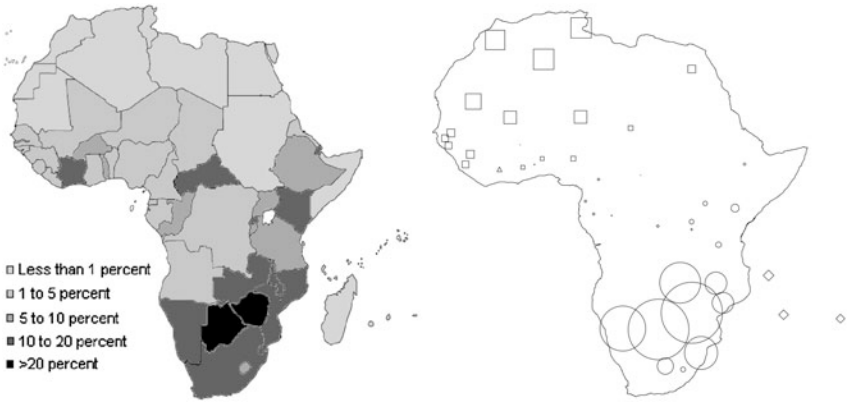
In spatial analyses, the extent of spatial correlation in a variable is often summarized by Moran's I (Anselin 1995). Of particular interest here is the degree of "neighborhood" correlation in AIDS rates across nations (Fotheringham, Brunsdon, and Charlton 2000); reexamination of the first panel in Figure 2, for example, suggests that spatial correlation tends to be highest in the north and the south of the continent (where we find strong consistency in infection rates from one nation to the next) and lower in central and western Africa, where cross-country rates are more variable.

In panel two of Figure 2, we plot the values of local Moran's I_i for HIV/AIDS rates for the various nations in Africa, based on data from 1997.[8] Both circles and squares represent neighborhoods of positive spatial correlation, while triangles and diamonds indicate regions of negative spatial correlation. The symbols also indicate absolute levels of infection: circles thus indicate (relatively) high-infection rate countries in high-infection rate "neighborhoods," while squares indicate low-infection rate countries in low-infection rate "neighborhoods." Conversely, diamonds correspond to low-infection nations in

[7]For a more detailed discussion of the methods used in calculating these estimates, as well as a discussion of the limitations of these data, see Schwartlander et al. (1999). More generally, all existing seroprevalence data suffer from a range of possible biases, including sample selection, testing bias, and uncertainty about the optimal means of combining survey-based and clinical surveillance estimates; Rutherford and Diaz (2004) and Davenport and Loyle (2009) provide good overviews of these issues.

[8]Distances are based on degrees of latitude and longitude between countries' geographic centroids; since the spatial approaches we adopt are all invariant to affine transformations of the distance metric, our results are the same as those we would obtain had we measured distance in miles, kilometers, etc. Plots using data from other years exhibit similar patterns.

FIGURE 2 **Adult HIV/AIDS Infection Rates in Africa and Geoplot of Local Moran's I_i for HIV/AIDS Rates, 1997**



high-infection areas, while triangles indicate high-infection countries in low-infection subregions. Finally, for all symbols, size indicates the absolute magnitude of the local correlation; smaller symbols indicate values of I_i closer to zero.

The second panel of Figure 2 reinforces the intuition of the first: as a subregion, southern Africa exhibits consistently high rates of infection, with the island nations of Madagascar and Comoros, and Angola, being significant exceptions. Conversely, north and west Africa are regions of relatively (and consistently) low infection rates, with Ivory Coast being an outlier in this regard. Across central Africa, by contrast, spatial correlations are relatively low; that subregion is largely heterogeneous with respect to infection rates.

From these initial analyses, two clear conclusions may be drawn. First, despite overall high levels of HIV/AIDS infection, nations in Africa also exhibit significant cross-national variability in those rates. Second, the patterns of that variation demonstrate a clear spatial component, with nations in southern Africa demonstrating consistently high rates, those in north Africa consistently low ones, and those in central and west Africa being largely heterogenous. These spatial considerations play an important role in our analysis and understanding of the conflict-HIV relationship.

Data, Operationalization, and Methods

We next turn to our empirical analysis of the dynamics of conflict and HIV. Our central variable of interest is the UNAIDS/WHO measure of adult HIV/AIDS infection rates in 43 African countries for the years 1997–2005. Figure 1 suggests the need for variables measuring wealth and development, economic openness, healthcare expenditures, and the presence and number of refugees, as well as indicators for both domestic and international conflict. In addition, we include control variables for population density, religion, education, and regime type.

We measure population density as the natural logarithm of population per square kilometer; these data are drawn from the *World Development Indicators* of the World Bank (2008). We also include in our model a variable that accounts for the Muslim percentage of each country's population; values for this variable, acquired from the CIA World Factbook (2008), range from zero (for example, in Bostwana and Namibia) to over 95% (e.g., in Mauratania, Morocco, and Libya). Education is measured as the percentage of adults (age 15+) in the population who are literate; these data were drawn from the *World Development Indicators* (World Bank 2008), with additional data from UNESCO (2005). To operationalize regime type, we adopt the widely used 21-point POLITY IV score (Marshall and Jaggers 2004), with higher values indicating greater levels of democracy and lower values higher levels of autocracy. We used one-year lagged values of this variable, which ranges from a low of -9 (for Swaziland in all five years in our data) to a high of 10 (for Mauritius in 2001, 2003, and 2005).

For our measure of wealth, we use the natural logarithm of per capita gross national product, in constant U.S. dollars; these data, along with those for economic openness and health expenditures, are drawn from the *World Development Indicators* database (World Bank 2008). For openness, we follow convention and calculate the natural logarithm of the sum of total imports plus exports, as a percentage of total GDP. Health expenditures are measured as the

TABLE 1 Variables and Expectations

Variable	Operationalization (Source)	Mean	Standard Deviation	Expected Influence
HIV/AIDS Rate*	Adult HIV incidence rate (UNAIDS)	7.45	7.85	—
Year	Recoded to 1997 = 1	5.18	2.84	Negative
Population Density*	Population per km^2 (World Bank)	74.8	102.2	Positive
Muslim Population	Muslim percent of the population (CIA World Factbook 2008)	36.4	33.8	Negative
Education	Adult literacy rate (WDI, UNESCO 2005)	58.6	18.1	Negative
$Democracy_{t-1}$	Lagged POLITY score (Marshall and Jaggers 2004)	0.70	5.32	Negative
Wealth/Development*	Per capita GDP, in US $s (World Development Indicators)	2380	3096	Negative
Economic Openness*	$\frac{\text{Imports+Exports}}{\text{GDP}} \times 100$ (World Development Indicators)	68.9	31.4	Negative
Health Expenditures	Public and private health expenditures, % of GDP (WDI)	4.81	1.75	Negative
Refugees*	Refugees present, in thousands (UNHCR *Statistical Yearbook*)	57.8	105.7	Positive
$International\ Conflict_{t-1}$	Lagged Armed Conflict Score (Strand et. al. 2003)	0.03	0.16	Positive
$Domestic\ Conflict_{t-1}$	Lagged Armed Conflict Score (Strand et. al. 2003)	0.19	0.46	Positive

Note: $NT = 181$. Asterisks indicate variables that are logged in the analyses below. *Expected Influence* denotes the predicted effect of that variable on HIV/AIDS prevalence rates. See text for details.

sum of public and private expenditures as a percentage of total GDP, measured in constant U.S. dollars; these range from a low of 1.8% (Madagascar in 1997) to a high of 12.8% (Malawi in 2003). Our data on refugees are drawn from the UNHCR's *Statistical Yearbook* (various years). We consider the natural logarithm of stocks of refugees present in the country and year in question. While numerous countries register values of zero on this variable, the highest value occurs for Tanzania in 2003, when the country was host to some 689,000 refugees.

Finally, we operationalize conflict using measures from the Peace Research Institute of Oslo (Strand, Wilhelmsen, and Gleditsch 2003). These 4-point ordinal scales reflect both the presence and the intensity of violent conflict, both internal (that is, civil wars and insurrections) and external. A value of zero indicates no conflict, while a one indicates a minor conflict (that is, one with more than 25 battle deaths in each year of the conflict, but fewer than 1,000 total). A value of two denotes intermediate conflict (greater than 1,000 battle deaths total) while a value of three indicates full-scale war (more than 1,000 battle deaths in every year of the conflict).[9] In our analyses, we use one-year lagged values, recognizing (as in Figure 1) that armed conflict is a causal antecedent to HIV, and that the full effects of such conflicts are likely to take time to be felt.[10] In Africa during this period, international wars were relatively less common than domestic conflicts; we record 31 instances of the latter, but only five of the former. Summary statistics for our measures, as well as expectations for their influences on HIV/AIDS infection rates, are presented in Table 1.[11]

As we discuss above, HIV/AIDS exhibits strong spatial and temporal patterns; infection rates within a particular nation tend to be both similar to those of its neighbors and strongly correlated over time. We are thus forced to contend with both spatial and temporal dependencies. At the outset, we consider three different approaches for dealing with spatial correlation and compare our findings as a check on their robustness. Standard regression models assume conditional independence across observations and thus represent a special case of more general models which incorporate spatial dependence. *Spatial lag* models allow the values of the response variable to influence those of its "neighbors" directly, through a process akin to including a (temporally) lagged dependent variable in one's model. In

[9]While this measure thus reflects conflict intensity, we recognize that it fails to capture other potentially relevant aspects of conflict, such as the occurrence of genocide or indiscriminate massacres.

[10]Note that the indicators for international and domestic conflict are only moderately related to one another ($\gamma = 0.56$).

[11]All data and syntax necessary to replicate these results are available at http://journalofpolitics.org.

TABLE 2 Influences on HIV/AIDS Rates in Africa, 1997–2005

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

Note: $NT = 181$. Numbers in parentheses are robust (White 1980) standard errors. One asterisk indicates $p < 0.05$, two indicate $p < 0.01$ (one-tailed, except for *Economic Openness*, where tests are two-tailed). See text for details.

contrast, *spatial error* models allow for spatial dependence in the error terms; this allows for the possibility of a local or regional dimension to shocks that are otherwise omitted from the model (Anselin 1988). As a practical matter, it can be difficult to ascertain a priori which of these two approaches is more appropriate for the data at hand; accordingly, we present results using both methods, as well as OLS results for comparison.

Beyond questions of spatial dependence, there are important reasons to believe that overall levels of HIV/AIDS rates may have changed substantially over the study period. On one hand, the period from 1997 to 2005 saw significant increases in HIV/AIDS rates on the African continent. At the same time, that period also marked the beginning of aggressive national and international efforts to rein in the virus. Beginning with the establishment of UNAIDS in 1996, international spending on HIV/AIDS worldwide rose from roughly $500 million in 1997 to nearly $10 billion in 2007 (UNAIDS 2004, 2008). That response, along with the availability of more advanced antiretroviral treatments and improved infrastructure for their delivery, raises the possibility that HIV/AIDS rates have declined over time.

Results

Results of our models for HIV/AIDS rates during the period under study are presented in Table 2. We estimate both "full" and "reduced form" models; the former omit the intervening variables in Figure 1 and include only the four control variables as well as the direct effects of conflict, while the latter include all the variables outlined in Figure 1.[12]

From the estimates in Table 2, several findings are apparent. In general, the models provide a reasonable fit to the data; the spatial-regression

[12]King, Keohane, and Verba note that "in general, we should not control for an explanatory variable that is in part a consequence of our key causal variable" (1994, 173). Here, as Figure 1 indicates, we have reason to believe that the variables omitted in our "reduced form" models are likely also to be consequences of the occurrence of conflict in those countries. An alternative approach would be to estimate a system of equations for the five endogenous variables in Figure 1; to do so here, however, would require that we specify a full causal model for each of the components in Figure 1 and that we ignore the spatial dependencies in the data. Accordingly, we leave such analyses for future work, though we note that estimating such a system (which ignores spatial dependence and endogenizes two of the four variables in Figure 1) yields very similar results to the OLS models presented in Table 2.

analogues to R^2 statistics are 0.72 and 0.67 for the "full" spatial lag and spatial error models, respectively, and 0.57 and 0.51 for the two "reduced form" models. In addition, across all sets of models we see substantial differences between the results which ignore spatial dependence and those that take such contagion into account. In fact, robust Wald tests for the presence of spatial lags clearly support the use of the spatial models.[13] Relatedly, our estimates of $\hat{\rho}$ and $\hat{\lambda}$ consistently support the presence of high levels of positive spatial contagion, even after controlling for a range of important covariates. Substantively, these figures bear out what Figure 2 suggests: that HIV/AIDS rates exhibit substantial geographic dependence, even after controlling for factors likely to influence those rates at the country level.

Turning to the covariate effects, we note a number of consistent results.[14] Countries with significant Muslim populations show significantly lower HIV prevalence rates; for a typical country, each 10% increase in the Muslim population corresponds to a predicted decrease in the HIV/AIDS rate of roughly 1%. The effect of education is almost equally significant; each 10% increase in adult literacy decreases the predicted prevalence rate by roughly three-quarters of 1%. At the same time, the influence of democracy—while strongest in the fully-specified models—is relatively small and imprecisely estimated, with a 10-unit change on the POLITY scale equating to an expected drop of roughly 0.3% in the overall adult HIV rate.

The results for our measures of international and domestic conflict are also relatively consistent, though they also exhibit slight differences both between specifications and across the various types of models. Most starkly, the influence of international conflict is large and significant in almost every case, whereas that for domestic conflict is consistently smaller (roughly one-half the size of that for international conflict in the full models) and, in the reduced-form models, undifferentiable from zero.[15] In a median case, a one-unit increase in international conflict increases expected HIV/AIDS rates by 5.7% in the spatial lag model and 5.9% in the spatial error model.[16]

In the fully specified models, we note a mild palliative effect of wealth: a one percentage increase in GDP lowers expected HIV/AIDS rates by nearly 1%. Health expenditures, by contrast, vary positively with HIV/AIDS rates, suggesting that such expenditures are likely responding to, rather than driving, high levels of HIV/AIDS; note that the highly aggregated nature of this measure does not allow us to distinguish between curative and preventive health care expenditures. We also note that the expected, positive effect of refugee influxes is consistent across all three models, though relatively small in substantive terms.[17] Finally, while the direction of change over time is negative as expected, it fails to be statistically differentiable from zero in four of our six models. In substantive terms, we see little decrease in HIV/AIDS rates across time, once we control for other factors.

[13]Those test statistics, which are distributed as $\chi^2(1)$, equal 17.72 and 8.09 ($p < 0.001$ and $p = 0.004$) for the fully specified spatial lag and spatial error models, respectively. The tests indicate that the spatial lag model is somewhat more appropriate for our data, a suggestion borne out by the relative fits of the two models.

[14]We report robust (White 1980) standard error estimates; however, our inferences are identical if we use standard (Hessian-based) variance-covariance estimates instead. Note that because the dependent variable is logged, the standard interpretation of the coefficient estimates is as elasticities—that is, as percentage changes in *Y*. Here, *Y* is itself a percentage; accordingly, to avoid confusion we focus on changes in predicted HIV/AIDS rates in a "typical" (median) country where the prevalence rate is 5%.

[15]An alternative specification of this variable, using an indicator for the presence of a civil war, yielded substantively identical results as well. In addition, to evaluate the possibility of reciprocal causality in the conflict-HIV/AIDS relationship, we estimated a series of empirical models assessing the effect of HIV/AIDS rates—as well as deaths from the disease—on the incidence and intensity of civil conflict. Our results offered no empirical support for the assertion that HIV/AIDS prevalence rates are positively associated with the likelihood of violent conflict, though those findings must necessarily be considered highly preliminary. We return to the question of a reciprocal HIV-conflict link in the conclusion.

[16]Ancillary models regressing wealth, trade, health expenditures, and refugees on the other variables in the model each demonstrate strong negative correlations between at least one type of conflict and each of these save the last; this suggests that conflict influences HIV rates both directly and indirectly. Additionally, we also estimated a series of models using different lags of the two conflict variables; in no case did we uncover evidence that such longer lag effects were present. Finally, because of the potential differences in the measurement of HIV/AIDS from year to year, we estimated models including a separate intercept (baseline HIV/AIDS rate) for each year; the inclusion of year dummies revealed no substantial year-to-year differences, and left our other findings substantively unchanged. All of these results are available from the authors upon request.

[17]We also estimated "full" models that included a separate variable measuring the (logged) number of internally displaced people in each country-year, using data from the UNHCR. The estimated effects of that variable were consistently small and statistically insignificant; we therefore omit it from the models in Table 2.

Conclusion

Scholars, policy makers, and the general population are well aware of the tremendous importance the HIV epidemic has for the developed as well as the developing world. Our work here has examined, for what we believe to be the first time, the link between HIV/AIDS and large-scale violent conflict. We have done so for a place and a period in which both armed conflict and HIV were prevalent. In one sense, then, ours is an easy test: the absence of a clear conflict-HIV link for the data studied would be strong evidence against the proposition that conflict contributes to rising HIV infection rates. In fact, our findings demonstrate that, even after addressing difficult issues of spatial contagion and controlling for a host of other related factors, both international and domestic conflict are consistently associated with substantially higher adult HIV infection rates. Our findings also reinforce empirically what has become the conventional wisdom regarding HIV/AIDS in the developing world: that education, wealth, and development retard the progress of the epidemic, while large influxes of migrant populations accelerate it.

But despite these grim facts, we believe our research also suggests some reasons for optimism. For one, the incidence of conflict, both in Africa and worldwide, has been decreasing since the end of the Cold War; our findings suggest that one of the potential effects of this reduction will be a lessening of upward pressure on HIV/AIDS rates. A related finding is the slight but notable decline in African HIV/AIDS rates over the period examined; while it is tempting to interpret those changes as a consequence of burgeoning levels of international assistance to fight the epidemic, clearly additional work is needed before such links can be drawn.

Our research also raises a number of other important questions for future work in this area. Chief among these is the extent to which our findings generalize beyond the African case; while we believe our framework is a general one, additional empirical analyses would be informative in this regard. Particularly important in this regard are additional investigations into the relative impact of international and domestic conflicts; while our findings support the notion that international conflicts contribute to the spread of HIV to a greater extent than domestic ones, additional work to confirm or refute this result is clearly needed.[18] Likewise, there is a pressing need to unpack in greater detail the causal linkages among the various factors we discuss above, including potential variation in conflict's influence by duration, severity, and geographic scope. Moreover, although we have demonstrated an association between conflict and the prevalence of HIV/AIDS, the manner in which states recover from violence is likely to have an impact as well; accordingly, another useful direction for future research would be to explore the effect of the speed and extent of postconflict reconstruction on the incidence on the disease. The complexity of these issues suggests that the most fruitful approaches to this work would balance large-scale quantitative analyses with carefully selected case studies in conflict-ridden areas.

Finally, while we focus on the extent to which violent conflict leads to higher HIV/AIDS prevalence rates, a critical topic for future empirical work is the possibility of reciprocal causality: whether, and to what extent, higher HIV/AIDS rates lead to conflict, particularly civil conflict. While the potential security implications of the disease have been recognized for some time (e.g., Elbe 2002; Peterson 2002), scholars have only recently begun to consider the potential strategic implications of the HIV epidemic, including its potential impact on such crucial phenomena as state capacity and intrastate militarized conflict. Some arguments suggest a positive influence of higher HIV/AIDS rates on state propensity for violent conflict, particularly civil wars. For example, Price-Smith's (2007) investigation of Zimbabwe suggests that AIDS acts as a "stressor" variable, simultaneously eroding aspects of both human capital and state capacity, which may in turn lead to an increase in domestic violence or even state failure. Similarly, Singer asserts that "AIDS not only threatens to heighten the risks of conflict, but also multiply its impact" (2002, 4). This effect, he argues, results from weakening of the military institution as personnel fall victim to the disease and subsequent domestic instability, including coups and revolts; a manifestly weak military may also invite foreign invasion. Whether and to what extent this reciprocal link exists, however, remains an open empirical question.[19]

More broadly, our focus on HIV/AIDS as a consequence of violent conflict is consistent with a wider movement away from traditional conceptualizations

[18]This is particularly true since, as we note above, international conflicts were relatively uncommon in Africa during the period studied here.

[19]For example, Peterson and Shellman (2006) provide an initial assessment of this link. Their findings are consistent with an indirect link between HIV/AIDS rates and aspects of security, including the incidence of civil conflict, with the bulk of that influence occurring through the disease's debilitating effects on the institutions of government and society; see also de Waal (2003) and Ostergard (2007). In contrast, Sato's (2008) analysis finds no association between a series of measures of state fragility and HIV/AIDS rates; see also Barnett and Dutta (2008).

of national security and toward what the United Nations and others have termed *human security*: ''protection from the threat of disease, hunger, unemployment, crime, social conflict, political repression, and environmental hazards'' (UNDP 1994, 25; see also King and Murray 2001–02). This broader focus dovetails with the scholarly community's recent recognition of the consequences of conflict as key to understanding its onset, most notably in the context of bargaining models of war (Reiter 2003). Crucial to the development of those models is a thorough understanding of the likely costs and benefits of violent conflict, including its long-term impact on nations' stability, development, and wealth. By noting, both conceptually and empirically, the key link between conflict and HIV/AIDS, we thus highlight a phenomenon of increasing importance on the international scene and contribute to broadening scholars' conceptions of legitimate factors in the study of international politics.

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Zaryab Iqbal is Assistant Professor of Political Science at Pennsylvania State University, University Park, PA 16802.

Christopher Zorn is Professor of Political Science at Pennsylvania State University, University Park, PA 16802.