AIDS, Security, and Social Stability

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Abstract:

Policymakers and scholars frequently frame the HIV/AIDS epidemic in terms of "threats" to international peace and stability. It has been argued that HIV/AIDS contributes to the weakening of military capabilities, the dissolution of state institutions, and raises the risk of civil conflict. With the epidemic nearly three decades old, we are now in a position to ask (empirically) whether and how patterns of political and social stability have been shaped by the spread of HIV/AIDS. In this article, I explore whether greater HIV prevalence is associated with higher levels of crime and victimization—an internal metric that captures the decay of state institutions and social stability. Using sub-national data on HIV prevalence and crime in Uganda during the 1990s, I demonstrate that districts with the highest levels of HIV prevalence also tend to exhibit high levels of reported victimization. A one percentage point change in HIV prevalence is associated with a 1.5 percentage point change in the rate of victimization.

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Few would dispute the claim that HIV/AIDS is reshaping social and economic life across much of the developing world. The disease now afflicts approximately 40 million people; more than 20 million have already perished; nearly three-quarters of the global HIV burden resides in Sub-Saharan Africa; and conservative estimates suggest that at least 12 million children have been orphaned by the epidemic (UNAIDS 2006). In scale and scope, the toll taken by HIV/AIDS compares only to the Spanish Flu pandemic of 1918 and the Black Death in the fourteenth century (Garrett 2004). As with other epidemics, the demographic changes wrought by the disease are profound: gains in life expectancies and infant and child mortalities made over decades have been reversed almost overnight. The economic consequences are equally staggering. By some estimates, the worst-affected countries will experience declines in GDP of 1 to 1.5 percentage points (Haacker 2004); studies focused on South Africa specifically (one of Africa's hardest hit countries) predict a drop in GDP per capita of 8 percentage points by 2010, relative to a no-AIDS scenario (Arndt and Lewis 2000).

Significantly more disagreement marks debates about the consequences of HIV/AIDS for international security. Spurred by a U.S. National Intelligence Estimate on infectious diseases which concluded that "these diseases will endanger U.S. citizenry at home and abroad, threaten U.S. armed forces deployed overseas, and exacerbate social and political instability in key countries and regions," policymakers have frequently framed the HIV/AIDS epidemic in terms of "threats" to international peace and stability (National Intelligence Council 2000). Scholars and policy analysts have moved in lockstep, advancing a variety of claims linking AIDS to the weakening of military capabilities, state failure, and a higher risk of civil conflict driven in part by orphans as new "pools of combatants" (Singer 2002); to the rise of rape in civil war and the use of AIDS as a "novel psychological and biological weapon" (Elbe 2002); and to growing casualties in on-going conflicts as soldiers spread

HIV to previously unaffected areas and war strains already under-equipped medical facilities, exacerbating rates of mortality (Singer 2002; Elbe 2002). Critics suggest, however, that these links are overdrawn, only weakly supported by empirical evidence, and enumerated more for rhetorical purposes and advocacy than as part of serious scholarly investigation (Peterson 2002). In the words of one noted public health scholar, when it comes to the consequences of HIV/AIDS for security and social stability, "the picture is confusing." (Garrett 2004)

With the epidemic nearly three decades old, we are now in a position to ask (empirically) whether and how patterns of political and social stability have been shaped by the spread of HIV/AIDS. Scholars are beginning to pursue this approach at the macro-level, exploring whether rates of HIV prevalence correlate with the onset of civil conflict and the severity of human rights abuses (Peterson and Shellman 2005). Finding no direct correlation, the authors suggest that that the impact of disease is indirect—HIV/AIDS, they argue, undermines a state's "society, economy, and political institutions" thereby exacerbating the risk of conflict. A complementary strategy for identifying the impact of AIDS on stability focuses on these indirect effects by exploiting temporal and spatial variation within a single country. Here, I ask whether greater HIV prevalence is associated with higher levels of crime and victimization—an internal metric that captures the decay of state institutions and social stability. In doing so, my article contributes to the existing body of research in two ways. First, it enumerates a set of plausible channels through which HIV prevalence might undermine stability (and increase crime), clarifying a set of distinct logics through which disease might condition individual behavior and impact aggregate outcomes. Second, it employs a unique dataset that tracks patterns of HIV prevalence and crime in Uganda in the 1990s. Spatial and temporal variation across Ugandan districts allows for an identification strategy constructed around sub-national comparisons, a research design that enables the investigator to more easily control for

"cultural, historical, ecological, and socioeconomic dimensions" that may vary at the national level and make it difficult to identify causal effects in cross-country research (Snyder 2001).

My argument begins with the recognition that communities facing rising HIV prevalence are also those where life expectancies are shortening, orphan populations are expanding, and government bureaucracies (police and military included) are suffering the loss of skilled personnel and confronting pressures to redistribute budgetary resources into the health sector. Each may have an independent effect on social stability. At the individual level, shorter life expectancies lower the (present value) cost of crime; discounting the future heavily may lead individuals (on the margin) to exhibit a higher propensity to commit crime. At a more aggregate level, the growing burden of orphan care may tax familial and institutional coping mechanisms. Orphaned children that grow up without the affection of parents and lacking access to educational opportunities may be more likely to engage in illegal activities. Turning to institutions, it may also be the case that the deterrent capacity of government is undermined by the HIV/AIDS epidemic. With the loss of skilled personnel and the redistribution of resources toward health within cash-strapped governments, the credible threat of apprehension and punishment may recede, decreasing the (potential) costs of committing crime even further. I develop the logic of each of these arguments (and identify observable implications of each), drawing on an illustrative model of crime developed by Becker (1968).

I then test my hypotheses using data on HIV prevalence and crime from Uganda in the 1990s. Uganda represents an ideal case for investigating this relationship as it was the epicenter of the AIDS epidemic in Africa in the early 1980s. Spread at least initially through the movement of soldiers during and after Uganda's civil war, prevalence peaked in the early 1990s at nearly 20%, declining to under 6% today. Despite its success in combating HIV/AIDS, significant regional variation in prevalence remains with the central and northern regions exhibiting prevalence rates

nearly five percentage points higher than the national average (Ministry of Health 2004). The results show a strong and consistent relationship between HIV prevalence and crime. Cross-sectional regressions, for data from both 1992 and 1999, provide evidence that districts with the highest levels of HIV prevalence also tend to exhibit high levels of reported victimization. A one percentage point change in HIV prevalence is associated with a 1.5 percentage point change in the rate of victimization. Strikingly, there is some evidence that HIV is associated more with property crime (theft, robbery, etc.) than with violent crime (murder, assault, etc.)—a finding consistent with previous work that suggests that economic crimes are better explained by a rational model of criminal behavior than crimes of passion (Levitt 2004). My results are statistically significant in specifications that incorporate a range of control variables typically included in cross-sectional analyses of crime, including region fixed effects that, in this case, capture differing levels of social conflict and government capacity across Uganda.

Recognizing that high levels of crime and HIV prevalence might be jointly determined by another factor excluded from the model (or poorly measured) or that crime might exert a reciprocal effect on HIV prevalence (for example, if high crime areas attract less risk averse types who then engage in risky sexual behavior), I also explore the robustness of the findings to the use of instrumental variables estimation. The results here are disappointing, although the use of "weak" instruments (instruments not highly correlated with the independent variable) is likely to blame. In a final empirical section, I conduct a preliminary inquiry into the relative explanatory power of the three distinct logics outlined above.

The findings are encouraging in that they demonstrate the feasibility of rigorously evaluating claims made about the links between epidemic disease and political and social processes. While research in economics has made significant progress in enumerating the micro- and macro-level consequences of HIV/AIDS, political science has been mired in a sea of poorly specified causal

claims without much empirical testing. At the same time, this article makes apparent the challenges of linking disease to political processes. Sub-national comparison is one avenue for assessing the consequences of high prevalence, but it doesn't lend itself as readily to the examination of war outbreaks or the collapse of state institutions. Perhaps future research can better identify empirical tests that parse the distinct, micro-level channels through which disease conditions aggregate outcomes. But more cross-country work (either qualitative or quantitative) will likely be necessary to evaluate some of the claims advanced in the literature. In addition, this investigation illuminates the challenges posed for empirical work by the limited data availability (and perhaps poor data quality) on health outcomes in Africa. Better data collection tools and more systematic efforts to collect sub-national comparative data are essential if we are to better understand how epidemic disease and changing life expectancies shape political and social outcomes.

I first proceed by considering theoretical arguments about the links between HIV/AIDS and social stability. Then I present sub-national data on AIDS and crime, describing the imputation procedure I use to generate broad geographic coverage in my measure of HIV prevalence. Next, I present my estimates of the effect of HIV on crime, describing bivariate and multivariate results. I then explore the robustness of my results to an instrumental variables approach, probe the relative explanatory power of the distinct mechanisms enumerated above, and conclude.

HIV Prevalence and Crime

Claims linking HIV prevalence to political outcomes typically predict the collapse of state institutions, growing instability, and conflict. Because such concepts are easily employed but notoriously difficult to define, I begin from a narrower description of the outcome to be explained. Crime is a symptom often observed when state institutions erode or the rule of law is undermined; moreover, crime is something measured in a consistent and systematic way by government

bureaucracies (both within and across countries). It is thus a useful proxy for the social instability thought to accompany the spread of HIV. Crime is also something that has occupied the attention of political scientists and economists for decades. We therefore have ways of thinking about how individuals decide whether or not to commit a crime and know what factors predispose communities to experience high levels of crime—a base of knowledge that can be employed as we enumerate the plausible channels through which the HIV epidemic might increase aggregate levels of crime.

Shortening of Time Horizons

Beginning with Becker's seminal work (1968), social scientists have analyzed the determinants of crime in terms of an offender's rational decision to participate in illegal activities, on the basis of a cost-benefit calculation. Becker writes that this rationality implies that "some individuals become criminals because of the financial and other rewards from crime compared to legal work, taking account of the likelihood of apprehension and conviction, and the severity of punishment." (Becker 1993) One of the core insights of this approach is that criminal behavior responds to changes in expected punishment. This assertion has been the subject of a growing empirical literature that seeks to measure the impact of deterrence efforts, including the hiring of police, the building of prisons, and the lengthening of sentences (for example, see Levitt 1996, 1997, 1998). The impact of increases in the expected price of crime, however, depends crucially on how much potential offenders discount their future welfare (Lee and McCrary 2005). Discount rates thus matter a great deal for understanding the impact of policy initiatives designed to reduce crime, and for explaining aggregate levels of crime more generally.

For purposes of illustration, consider a dynamic model of the offender's decision (as given in Becker (1968), modified by Lee and McCrary 2005).[2] Throughout his lifetime, an individual chooses where or not to commit a crime in each period t during which he is free. If he chooses to commit a crime, with probability p_t he is apprehended and punished, leading to the payoff U_t^s (his lifetime utility discounted to period t). With probability 1 - p_t , the offense is not detected and he obtains a lifetime utility of U_t^{cr}. If he chooses not to commit the crime in period t, he receives lifetime utility of U_t^a. Thus, at each point in time t, the individual commits a crime if and only if

$$p_t U_t^s + (1- p_t) U_t^{cr} > U_t^a$$

where we assume $U_t^s < U_t^a < U_t^{cr}$. At the same time, he will commit a crime if and only if the probability of apprehension is less than a "reservation probability", given by

$$p_t^* = (U_t^{cr} - U_t^a) / (U_t^{cr} - U_t^s)$$

For any given p_t , if the rewards to crime rise (an increase in U_t^{cr}), the payoffs to legal activity fall (a decrease in U_t^a), or the criminal sanctions become less punitive (an increase in U_t^s), the individual is more likely to commit a crime, because it is more likely that p_t will fall below the reservation probability p_t^*.

Now, consider the impact of HIV/AIDS on individual decision-making. Perhaps the most staggering consequence of the disease is for life expectancies. According to the U.S. Census Bureau, by 2010 twenty-five nations in Sub-Saharan Africa will have significantly reduced life expectancies at birth, as compared to 1990 levels (Garrett 2004). In Malawi, for example, life expectancy in 2000 had fallen to the country's level in 1969. From 1970 to 2000, Zimbabwe saw a fall in life expectancy (at birth) from 56 years to 33 years. The numbers are equally grim in neighboring countries: Zambia (a loss of 32.4 years), Botswana (16 years), and Lesotho (14.4 years). For individuals living in the midst of a growing pandemic, time horizons are shrinking and discount factors are on the rise (objectively,

[2] This model is taken directly from Lee and McCrary (2005).

and perhaps even subjectively).[3] In the context of rational models of crime, the foreshortening of time horizons is equivalent to criminal sanctions becoming less punitive. As individuals are less likely to be around to bear the costs of punishment in the future, the deterrent effect of apprehension and punishment is reduced.

Moreover, with decreasing life expectancies, the payoffs to legal activity are also likely to decrease. Human capital accumulation—in particular, education—is spurred by the expanding time horizons that accompany societal decreases in mortality and morbidity (Becker 1993). The HIV epidemic reverses these demographic trends, decreasing the likelihood that individuals will be around to collect the returns from investments in skills and technologies made in the present. Finally, it might also be the case that rewards to crime rise in the context of high HIV prevalence.[4] Given the difficulty of accessing anti-retroviral drugs in most parts of Africa (high cost, limited supply, especially in the 1990s), life-saving medications might only be available to individuals willing to engage in illegal activities, as the return to licit occupations (for most in largely rural Africa) is quite limited. Taken together, these three factors suggest a relationship between HIV prevalence and crime—one that operates largely through the rational calculations of potential offenders that are HIV-positive or believe they might face a significant risk of contracting HIV.

Demographic Shifts

A second channel emphasizes how the HIV epidemic changes the membership and structure of populations within high-prevalence communities (Pharoah 2005). Consider first the impact of

[3] Indeed, recent research in Malawi suggests that, in a given population, significantly more individuals estimate their likelihood of having HIV as medium or high than actually have the disease (Anglewicz and Kohler 2005).

[4] An alternative framing might suggest that, in environments of high HIV prevalence, the inequality between those with and those without access to life-saving medications generates grievances. As a consequence, we might detect increases in the level of crime that follow from anger and frustration.

AIDS-related deaths on the number of orphans (UNAIDS/UNICEF 2003). In Botswana, for example, one out of every five children is an orphan, up from only 7% in 1990. South Africa's epidemic promises to double the percentage of orphans from 10% in1990 to 20% by 2010. The key question, from the perspective of political stability, centers on how afflicted communities manage the growing burden of dependents. To the extent that orphans face emotional trauma associated with AIDS-related losses, psychological difficulties owing to the loss of parental affection and positive role models, and socio-economic struggles because of inadequate nutrition, education, and health care, orphans may be at a disproportionately high risk of developing antisocial tendencies and engaging in criminal activities.

A growing empirical literature assesses the impact of parental death on socio-economic outcomes. Evans and Miguel (2004), for example, find that parental death significantly decreases children's participation in primary school in Kenya; moreover, it appears that these effects are most pronounced in poorer households. Yamano and Jayne (2004) provide evidence for a similar finding with respect to school participation, but also show that the death of a male household head is associated with a massive drop in household crop production and off-farm income, arguing that these income shocks likely have negative spillover effects on the general well-being of the remaining household members (2005). Gertler et al (2004) demonstrate that the loss of a parent doubles the risk that a child will drop out of school in Indonesia. Examining 10 Sub-Saharan African countries, Case et al (2004) find that orphans are significantly less likely to attend school than non-orphans living in the same household; moreover, orphans do worse the more distant the relative they are living with. Although this literature is still evolving, the early results suggest that orphans fare poorly and that the loss of a parent has measurable and significant impacts on a child's educational attainment. This puts orphans at a greater risk for participation in crime; without access to education, the opportunities lost when one engages in illegal activities are much less attractive.

Indeed, the empirical literature on crime in the United States finds that criminals are typically poorer and less educated than their counterparts in the general population (Freeman 1991).

A slightly broader framing of the demographic argument underlines how HIV/AIDS is reshaping the age pyramid in afflicted communities and societies. The death of members of the productive, working-age population not only produces orphans; it also gives rise to what demographers call the "chimney effect." (Garrett 2004) The demographic distribution in high-prevalence countries is transformed by HIV/AIDS—dramatic decreases in the percentage of the population aged 35-55 are accompanied by significant increases in share of the population aged 10-29, a so-called "youth bulge." Schonteich (1999) speculates that because young men are most likely to commit crime, a disproportionate number of young men in places severely affected by HIV may lead to higher levels of crime—"particularly violent crime and group-based aggression." His analysis is rooted in the empirical literature on crime which suggests that age and gender are two of the strongest predictors of a propensity to engage in illegal activities. Research on unrest and civil conflict also points to the impact of large populations of young men on the likelihood of violence (Mesquida and Warner 1999). Thus, the spread of HIV, by changing the demographics of affected societies, potentially increases the rate of crime and victimization.

Erosion of State Capacity

A third possibility is that the consequences of the HIV epidemic for instability operate through changes to institutions of governance (De Waal 2003). Modern organizations, whether in government, business, or even the security forces, depend on professional staffs with years of experience, substantial expertise, and extensive networks of personal contacts. HIV/AIDS attacks the human resources of these professional bureaucracies. Skilled and trained employees become sick and die, leaving critical posts unfilled or forcing the rapid promotion of people without the skills to

do the job. Illness, funerals, and the need to care for the sick result in high rates of absenteeism, reduced morale, and as a consequence, potentially declining institutional capacity. The pressure on employees to meet rising health expenditures and the decreasing likelihood that staff will be around to benefit from promotion and advancement also may distort the incentive structure within these bureaucracies, resulting in more opportunistic and corrupt behavior. Because these effects are not one-off shocks to the system, but rather deep, structural changes to the environment in which these institutions operate, it is likely that the effectiveness of government institutions and, perhaps, reform efforts designed to increase accountability and transparency, will be undermined.

Importantly, the consequences of epidemic disease for government capacity are likely to be long-lasting, in part because HIV/AIDS is devastating the educational system—a key driver of the development of future generations of trained professionals. In South Africa, it is estimated that one-third of teachers are HIV positive; observers speculate that the number is even higher in Zambia and Swaziland (Peterson 2002). Even without the death of teachers, school quality may still be adversely affected. Absenteeism is one result of HIV that has received substantial attention from scholars (Chaudhury and Hammer 2005). One study in Zambia found that a 5% increase in teacher absences is associated with a 4-8% drop in average gains for students over an academic year (Das et al 2005). 18% of teachers were recorded as absent on a given day, and teachers accumulated an average of 21 absences a year (60% of which were related to health problems)—rates far higher than those observed in the developed world.

Perhaps most importantly, for issues of crime and victimization, recent data suggest that police and military bureaucracies face a particularly high risk of suffering human resource losses owing to HIV/AIDS. Security forces tend to be dominated by young, active men. The combination of their age, long periods of deployment away from family, access to cash, tendency to purchase sex from prostitutes, likelihood to use alcohol and drugs when off duty, stressful (and often dangerous)

work environments, and participation in a generally "macho" culture renders policemen and soldiers especially susceptible to becoming infected with HIV (Garrett 2004). This does not necessarily imply that members of the security services will necessarily have a higher rate of infection than the general population; indeed, in many countries, the armed forces are subject to strict disciplinary guidelines and have access to better health care than most. Moreover, military leaders have strong incentives to bring HIV infection under control. But in Africa where militaries are notoriously undisciplined, the story is not so positive (Garrett 2004). The head of the Malawian Defense Forces reported publicly that his troop strength was down by more than 40 percent due to HIV death, a loss he said was felt most acutely in the command and control structure. In Mozambique, more than half of new recruits to the military tested positive for HIV when the national prevalence rate is only 13.6 percent. Indeed, the head of the Mozambican police explained that the country can no longer recruit and train police officers fast enough to replace those that are dying. Even in Uganda, the site of a generally successful campaign to fight HIV/AIDS, recent studies suggest that 13.3% of police are HIV-positive, when the national prevalence rate hovers around 6% (ICG 2004). To the extent that the effectiveness of militaries and police services is undermined by HIV/AIDS, their deterrent capacity wanes. Returning to the rational model of crime with which we began this investigation, we can interpret such changes as a decrease in the likelihood a criminal will be captured and punished—a shock that is likely to be associated with higher rates of crime and victimization.

Thus far, I have identified three plausible channels through which rising prevalence might be associated with crime—the foreshortening of individual time horizons, the growth of orphan and youth populations, and the decreasing deterrent capacity of state institutions.[5] Together, they suggest an empirical relationship between HIV prevalence and crime. The main hypothesis I therefore

[5] This is not an exhaustive list of the potential mechanisms linking HIV and crime. An additional mechanism might link HIV prevalence to negative economic shocks that reduce the relative returns of licit activity.

explore using data from Uganda predicts that districts with the highest levels of HIV will also experience greater crime.[6] But each channel suggests some unique observable implications as well, some of which are amenable to testing with my data. If the foreshortening of time horizons is causing individuals to make different decisions about the costs and benefits of committing a crime, we might expect that those that are or believe they are likely to be HIV-positive would be more likely to engage in illegal activities. If demographic changes are responsible for the relationship between HIV and crime, we would expect to find a larger burden of orphans or an age pyramid more heavily weighted toward young men in high prevalence communities. If institutional decline is the main driver of crime, districts with high HIV-prevalence should also exhibit decreases in governmental effectiveness in the security sector. While district-level data does not allow us to explore the individual-level mechanisms enumerated above, it does provide us with data useful for exploring the demographic and institutional channels. I turn first to the data and main empirical results, before exploring the evidence for these distinct causal mechanisms.

Data and the Empirical Approach

In order to test my core hypothesis, I use a new dataset that combines information about the incidence of crime in Uganda's districts in the 1990s, disaggregated data on HIV prevalence, and a set of standard control variables employed in models of crime. Because of the paucity of high-quality sub-national data in Africa, collecting this information and producing variables to be used in a cross-

[6] An alternative hypothesis suggests a negative relationship between HIV prevalence and crime. If it is the case that (1) risk-seeking individuals engage in risky sexual behavior and are more likely to commit crimes and (2) the lack of availability of medicine for HIV results in poor health outcomes and even death for those in the risk-seeking population, we would expect to find high HIV prevalence associated with lower levels of crime, as individuals become too sick to engage in illegal activities or because criminals disproportionately die off.

sectional analysis were not uncomplicated tasks. Before reporting the results of my analysis, I describe the data in greater detail.

Crime and Victimization

Empirical studies of crime tend to employ two main sources of data (Fajnzylber et al 2000). The first is statistics provided by the criminal justice system. The benefits of official crime statistics are obvious: they can be tabulated at different levels of aggregation and allow for both longitudinal and cross-sectional analysis. But the disadvantages are significant—most crimes are never actually reported and substantial deficiencies exist in the recording procedures of police and justice systems. These disadvantages are magnified in Sub-Saharan Africa where the weak administrative capacity of governments (especially outside of the capital) is likely to lead to significant underreporting of crimes; at the same time, the sub-national variation that exists in government capacity (and perhaps also, the faith that citizens have in government) renders suspect the cross-regional patterns we might observe in government statistics. The second key source is victimization surveys, which Glaeser calls the "primary workhorse" for measuring crime (Glaeser 1999). These surveys are generally collected from representative sample surveys of households and provide information about non-fatal crimes (such as assault and theft). Their main advantage lies in the accuracy with which they capture underlying levels of crime, because they capture the incidence of crime as reported by individuals (rather than that reported to the police). Unfortunately, however, victimization surveys have not been conducted with the frequency and geographic coverage required for significant longitudinal and cross-sectional analysis, especially in Africa.

For the purposes of this project, I began by compiling government statistics on the sub-national incidence of crime in Uganda. Practically, this involved working with the Central Investigative Division of the Ugandan National Police to organize paper records submitted by police

stations around the country. A police officer was assigned to record, for each district and each year between 1992 and 1998, the total number of crimes committed, arrests made, and prosecutions successfully undertaken for a set of 17 different crimes (including homicide, rape, assault, theft, and so on). To complement this data, I obtained a nationally-representative household survey conducted by the Uganda Bureau of Statistics in 1999. In the survey, completed with 10,696 households, respondents were asked to report the number of incidents of theft they experienced in the previous 12 months (and in 1992) and the number of incidents of physical assault experienced in the last year (and in 1992). These are the types of questions employed in detailed victimization surveys. I aggregate responses to these questions across all households in a district in order to produce reliable measures of the incidence of (or share of households that have experienced) property crime, assault, and overall crime for each district in 1992 and 1999.[7]

(Table 1)

Table 1 reports a correlation matrix of district level measures of crime in 1992 drawn from the two different sources. The results are striking. The measures drawn from the victimization survey are highly correlated with one another; two aggregate measures of economic crime and violence crime constructed from the government statistics are also highly correlated. Yet the correspondence between victimization data and government statistics is very low. The correlation is, in fact, negative. Areas with higher crime as reported by individuals in a household survey do not tend to have higher levels of crime as captured in government statistics. In the remainder of the paper, I proceed with dependent variables drawn from the victimization survey—following the norm emerging among social scientists studying crime. I am more confident that the data collected

[7] It is worth noting that because the number of districts in Uganda increased markedly between 1992 and 1999 (from 38 to 45), data is aggregated according to the district in which populations resided in 1992. Because new districts (with only one exception) have been carved entirely out of old districts, resorting police station statistics/household responses into old districts is a relatively simple task.

in the nationally representative sample survey provides an accurate barometer of crime at the district level; the cost, however, is the loss of substantial temporal variation contained in the government statistics, but lacking in the household survey (because questions on crime were not asked in previous iterations of the survey).

HIV Prevalence

To measure HIV prevalence, I begin with the information contained in the HIV Surveillance Reports produced by the Ugandan Ministry of Health. These reports document reported HIV prevalence at a set of sentinel sites around the country—clinics or hospitals where HIV testing is conducted on pregnant women. The testing of pregnant women as a source of generating reliable estimates of HIV prevalence continues to be common practice; although, recently, nationally representative random sample Demographic and Health Surveys (DHS) have begun to administer HIV tests to selected households allowing for better estimates of prevalence. The number of sentinel sites has increased significantly over time; only six were in operation in 1992, 13 in 1993, and 15 by 1999. However, with such a small number of sentinel sites, data on HIV prevalence is missing for 24 districts that never housed an antenatal clinic. Without data for these missing districts, the overall sample size is simply too small to identify statistical relationships (and control for relevant omitted variables) with great confidence.

For this reason, I employ an imputation procedure drawing on a natural experiment in Uganda—the demobilization of 20,000 soldiers from the Uganda People's Defense Forces (UPDF) in 1992. Rakai, in southwestern Uganda, was the main launching point for the HIV epidemic in the country. Scholars and practitioners engaged in HIV research in the region often trace the rise in HIV cases to violence and instability in the area as anti-Amin rebels entered Uganda from Tanzania in 1979. Following a brief interlude of peace after Amin was overthrown, the civil war resumed in

1980, and the same soldiers that passed through Rakai in 1979 established insurgent bases in the center and western parts of Uganda. It is commonly argued in the Ugandan case, and has been demonstrated empirically elsewhere, that violence and the concomitant movement of people speed the spread of HIV. Although no actual statistics are available, it is widely believed that a significant share of now-President Museveni's army was HIV positive when they won the guerrilla struggle and took power in 1986. The demobilization—which took place six years later—is useful for our purposes because it provided serving UPDF soldiers with financial incentives to retire from the army and return to their home communities. In doing so, the demobilization contributed to the spread of HIV/AIDS around the country, as young men returned home, reengaged with their families or created new ones, and passed on the disease to wives and other sexual partners.[8]

Thus, to impute data for districts without antenatal clinics, I take two steps. First, I regress actual prevalence rates (from sentinel sites) on the number of soldiers demobilized to each district. With an estimated coefficient, I then compute the predicted value of prevalence for the districts with missing data using information about the number of soldiers demobilized to the area.[9] The second step involves creating longitudinal data on prevalence for districts without antenatal clinics. Instead of computing a national rate of change, I instead partition the country into its four component regions; for each region, I calculate a linear rate of change in HIV prevalence using actual antenatal

[8] It is possible that HIV was passed to home communities even before the demobilization as many soldiers kept wives in their home communities while being posted to the other areas.

[9] I produce two intercepts for each district in 1992. The first intercept ("the 1992 intercept") employs coefficients drawn from a regression of prevalence data on the number of demobilized soldiers for five sentinel sites in 1992 (statistically significant coefficient, R-squared of 0.57). The second intercept ("the 1993 intercept") employs coefficients drawn from the same regression, but using data on prevalence from the ten sentinel sites with information in 1993 (statistically significant coefficient, R-squared of 0.38). It is not immediately clear which estimate is more valid. I present results in the remainder of the paper using the intercept computed with the 1993 data (more observations), but the results are robust to specifications using the alternative version.

clinic data. I then apply this rate of change to the imputed measures of each district without a sentinel site to produce estimates of prevalence for each district in each year.

This approach is obviously not ideal. Significant measurement error is likely introduced in the imputation process. But, in general, the more noise that exists in the data, the more difficult it will be to identify statistically significant effects—so this would simply lead us to underestimate the causal impact of HIV prevalence. The larger concern is whether this approach introduces bias in the measurement of HIV prevalence—bias that might account for any relationships that are observed. For example, if the imputed measures systematically overstate HIV prevalence and imputation is necessary for regions with higher levels of crime, than we would observe a relationship between prevalence and crime when no such relationship exists. However, there is no statistically significant difference in crime rates between those districts for which prevalence data is available and those for which imputation was necessary.

It is natural to wonder, as well, whether the demobilization of soldiers might be associated with increases in crime. If this were the case, using patterns of demobilization to predict HIV prevalence might also generate a spurious correlation between HIV and crime. Importantly, however, we know from previous empirical research in Uganda that demobilization had no direct, long-lasting impact on crime (Collier 1994). The demobilization of landless soldiers (about 10% of total fighters) was found to correlate with crime in the first quarter of 1993, but not subsequently. Moreover, no relationship between demobilization and crime was observed for the vast majority of soldiers (90%) that had access to land. We can be reasonably confident, then, that any observed relationship between HIV prevalence and crime in the data from 1999 is not due to spurious

correlation; for our analysis of the 1992 data, we must be more cautious, but Collier's research suggests that, if anything, the direct link between demobilization and crime is weak and short-lived.[10]

Control Variables

Concerned about the possible impact of omitted variables on my estimate of the causal impact of HIV prevalence, I also introduce a set of control variables typically included in empirical studies of the determinants of crime. I begin with variables proposed in the literature on the payoffs to and opportunity costs of crime. Fleisher (1966), for example, suggests that the theoretical effect of average income levels on crime is ambiguous: on the one hand, it increases the opportunity cost of engaging in illegal activities (leading to reduction in crime), while on the other increasing the payoffs to crime (leading to an increase). Empirical work in the United States has identified contradictory results (Fleisher 1966; Ehrlich 1973). Recent research in developing societies is also mixed: Demobynes and Ozler (2002) find that burglary rates are 25-43% higher in South African neighborhoods that are wealthier than their neighbors, while Fafchamps and Moser (2002) find that crop theft increases with poverty in Madagascar. Inequality is also thought to have an effect on crime, distinct from that of poverty. Controlling for poverty, more inequality means that there is more wealth to be stolen. Inequality may also create conflict leading to envy and a greater willingness to commit crime. A large literature has identified a crime-inducing impact of inequality measured in various ways (Fleisher 1966; Ehrlich 1973; Fajnzylber et al 2000; Demobynes and Ozler 2002; Fafchamps and Moser 2002).

[10] Moreover, it is possible to argue that if there were a spurious relationship between HIV prevalence and crime, it would appear in the 1993 data, but not the 1992 data, as the demobilization took place late in 1992. Yet the high rate of HIV among soldiers might have shaped prevalence far earlier than the actual demobilization, as patterns of demobilization tell us not only where soldiers went, but generally where they came from. Thus, HIV might have been spread as instability in the country decreased when soldiers reconnected with their families and communities, even if they were still based elsewhere.

Drawing on two nationally representative household surveys conducted by the Uganda Bureau of Statistics, one in 1992 and one in 1999, I compute an average measure of household wealth for each district. The measure of wealth (for each household) is an asset index, aggregating individual questions about home ownership; the material with which the roof, walls, and floor are constructed; the source of water that households use; the type of toilet they access; what source of fuel they use for cooking; and whether households have electricity. The weights are generated using factor analysis with household information in the 1992 survey; these 1992 weights are then used with household information from the 1992 survey to create a measure of average district wealth for 1992, and with data from the 1999 survey to produce a second measure of average district wealth for 1999. The standard deviation of the asset index within each district is employed as a proxy for inequality.

A number of studies also propose that the education level of the population may matter, as it shapes the expected rewards from both criminal and legal activities. Evidence suggests that criminals tend to be less educated and from poorer backgrounds than non-criminals, which some interpret as evidence that the ratio of benefits to costs of crime improves as the opportunity cost of participating in crime diminishes (Freeman 1991). I proxy for the education level of the population by computing the mean level of education in each household (for all household members above 15) and then averaging across the district. More generally, some have suggested that men have a higher innate tendency to commit crime (Clinard & Abbott 1973; Grogger 1997). I include a control variable that captures the imbalance between men and women in a district, computed as the average share of men in each household by district.

Crime incidence depends (as may HIV prevalence) on the intensity and nature of social interaction as well. An increasing number of studies focus on "social capital," arguing that baseline levels of trust and norms of reciprocity may affect the level of crime in a community. For example,

Freeman (1986) demonstrates that church attendance is associated with a lower likelihood of arrests among youth. Glaeser and Sacerdote (1999) focus on the incidence of female-headed households, showing empirically that weaknesses in the structure of families can explain a great deal of the incidence of crime in cities. More broadly, we can imagine that communities with strong networks and norms may be better able to prevent crime by raising the costs of crime (through increases in the likelihood of detection or in the punitive (social) sanctions that are experienced). I proxy for low levels of "social capital" with a measure of ethnic diversity drawn from the 1991 census—the probability that two randomly selected individuals in a district are from the same ethnic group. It has been shown repeatedly that ethnic diversity is associated with lower levels of collective action (for a review, see Alesina and La Ferrara 2005), and there is some evidence as well that ethnic diversity is positively associated with crime (Fajnzylber et al 2000). A second way of thinking about the intensity of social interaction focuses on geographic factors rather than social ones. Isolated communities, it has been argued, face a much lower incidence of crime because human interaction is much more constrained. Within isolated communities, it may be the case that strong norms deter crime (as above); it may also be the case that isolation reduces crime because those wishing to engage in illegal activities face high costs in reaching the area. For this reason, and consistent with Rephann (1999), I include a proxy for the road density of each district, anticipating that higher road density is likely to be associated with more crime. The measure of road density is taken from the Uganda Statistical Abstract (2003).

Estimates of the Determinants of Crime

Before presenting the results of the multivariate specification, I begin by exploring the bivariate relationship using data drawn from a cross-section of districts in 1999.[11] Figure 1 presents

[11] HIV prevalence is actually measured in 1998, as 1999 data were unavailable.

scatter-plots showing the correlation between HIV prevalence and victimization for each of three measures: the incidence of theft, assault, and overall crime. As one can see in the figures, HIV prevalence is positively associated with the incidence of theft and overall crime, but not physical assault. In both bivariate regressions, we can reject the null hypothesis of no effect at the 99% level (for theft) and the 95% level (for overall crime). Substantively, the correlation suggests that an increase in the prevalence rate of a district by one percentage point is associated with an increase in crime of 1.5 percentage points. Importantly, the bivariate results are robust to the exclusion of Apac, Lira, and Mbale—three districts with levels of crime far higher than other districts in the country.

(Figure 1)

To ensure that the overall results are not driven by the imputation process, I examine the bivariate relationship between HIV prevalence and crime using *only* data on prevalence gathered from the 11 sentinel sites that reported in 1999. We can reject the null hypothesis of no effect at the 99% level (for theft) and the 90% level (for overall crime). Substantively, the magnitude of the coefficients is unchanged.

The multivariate OLS regressions reported in Table 1 test the core hypothesis by considering determinants of the incidence of crime in Uganda's districts. Table 1(a) reports results using the measure of HIV prevalence imputed using the 1993 intercept; Table 1(b) employs the 1992 intercept (see footnote 6 for a more detailed explanation). Columns (1)-(3) in each table report results for a single cross-section of districts in 1999; columns (4)-(6) show the findings for a cross-section of districts in 1992. The results suggest that HIV prevalence is positively and significantly associated with the incidence of crime in both 1999 and 1992. The substantive size of the effect appears to increase over time. A one percentage point rise in HIV prevalence is associated with 0.5 percentage point increase in the incidence of crime in 1992; in 1999, the impact increases to 1.5 percentage points. The results employing the 1992 intercept suggests a similar increase in the size of the

estimated impact. Consistent with what we saw in the bivariate scatter-plots, there is also some evidence in the data from 1999 that the impact of HIV prevalence on crime operates largely through changes in the incidence of property crime, rather than violent crime.

(Table 1)

Although the core set of variables explain a great deal of variation in district crime rates (R-squared ranging from 0.36 to 0.61), the results are often contradictory when it comes to other variables hypothesized to impact the incidence of crime. Measures of wealth enter positively in explaining crime in 1999, while entering negatively for 1992 data (in both specifications). Inequality enters positively in both cross-sections, although the coefficients are statistically significant only in the analysis of 1992 data. Perhaps, surprisingly, measures of ethnic diversity enter negatively and significantly in most specifications—a result opposite to that proposed by the literature. Further, there is some evidence, in the 1992 data, that the relationship between diversity and crime is non-linear, with diversity decreasing crime up to a moderate level of diversity, after which greater diversity increases the incidence of crime.

An important test of the robustness of the results involves the introduction of controls for the region in which districts are located. A number of unobserved district attributes are not adequately controlled for in our models; without significant additional data, however, district fixed effects cannot be easily employed given the small sample size. Region dummies are a second best approach, allowing us to control for region-specific characteristics that may jointly determine HIV prevalence and the incidence of crime. For example, districts in the North remain enmeshed in a nearly 20-year long civil war with the Lord's Resistance Army, a war that has forced large segments of the population into internally displaced persons camps. This level of violence and instability is likely to be associated with greater transmission of HIV and higher levels of crime. Yet the inclusion of fixed effects for the regions in which districts are located—in effect, asking whether HIV

prevalence is associated with higher levels of crime *within* each region—does not change our core results. HIV prevalence is associated with a higher incidence of overall crime in cross-sections using both 1999 and 1992 data and the magnitude of the coefficient is basically unchanged, although the standard errors increase slightly.[12] The coefficient on HIV prevalence in models of the determinants of theft (for 1999) remains unchanged, but drops just below conventional levels of significance.

(Table 2)

It is also important to explore the implications of the fact that many of the variables I employ are district level aggregates of household survey responses. Because the number of respondents in each district varies, some district level averages may be more precise than others. This introduces a concern about measurement error. In general, measurement error in right-hand side variables leads to attenuation bias; the fact that we find strong results given measurement error adds confidence that the underlying relationship is strong. But it may be the case that measurement error in the dependent variable causes heteroskedasticity, leading to a bias in the estimates of the standard errors. I correct for this with the use of robust standard errors throughout. I also explore the data to see whether the measurement error is correlated with an omitted variable, district population (as sample size at the district level is a function of population). Finding that it is in some specifications, I add population to the core regression models as an additional control. The coefficients on HIV prevalence are largely unchanged with the inclusion of a control for population, although population is correlated with higher levels of crime.

Given the imputation procedure employed to produce measures of HIV prevalence, one still might be concerned that the relationship we observe is driven by the direct impact of demobilization

[12] The results are robust to the inclusion of region dummies for measures of HIV prevalence using either the 1993 intercept or the 1992 intercept. Moreover, region dummies eliminate the non-linearity observed in the data, as northern regions with moderately high prevalence experience high rates of crime due to on-going civil conflict. Regional fixed effects capture these distinct patterns across Ugandan districts.

on levels of crime. The evidence suggests, however, that HIV prevalence exerts an independent effect on the incidence of crime in 1999, when we control for the number of soldiers demobilized to a district. These results hold in the sample of districts with measured prevalence rates and in the broader sample combining districts with actual and imputed measures of HIV.

Instrumental Variables Estimation

I described earlier two possible ways in which the OLS regressions might not accurately capture the impact of HIV prevalence on crime. It may be the case that other, omitted variables jointly determine prevalence and the incidence of crime; it is also possible that crime exerts a reciprocal effect on HIV prevalence, perhaps because areas where crimes can be easily committed attract more risk-seeking types that tend to be HIV positive. For both of these reasons, I employ instrumental variables estimation to explore the robustness of my findings.

An appropriate instrumental variable must meet two conditions: it must be correlated with the independent variable (in this case, HIV prevalence) and correlated with the dependent variable (crime) only through the independent variable of interest. Building on recent work on the impact of HIV on economic growth, I employ an instrument for HIV prevalence based on male circumcision rates (Wendell and Werker 2004). The link between circumcision and the prevalence of HIV has been debated in the medical literature for more than a decade (Caldwell and Caldwell 1996). Although it has not yet been established with certainty that circumcision reduces the likelihood of transmission, a recent randomized trial of circumcision in South Africa was suspended when the early data showed overwhelmingly that circumcised males were less likely to contract the disease.

To formulate district level estimates of male circumcision, I follow first the strategy pursued by Wendell and Werker (2004). I used the demographic breakdown of districts by tribe (from the 1991 census) to convert information on tribal circumcision practices (in Murdock 1967) into

estimates of the circumcision rate. With the assumption that circumcision was either present or absent for a given tribe, I calculated a district level circumcision rate. Only two major ethnic groups in Uganda (the Bagisu and the Lendu) are coded as having traditional circumcision practices; consequently, more than 50% of districts have a circumcision rate of less than 7%. The mean circumcision rate is 9% and the maximum is just under 50%. In addition to the rate of circumcision, I used census data to compute the fraction of each district that is Muslim; because Muslims practice circumcision and the data on tribal practices may introduce some measurement error (because it does not cover all groups and is based on ethnographic work decades ago), I employ the fraction of Muslims as a second instrumental variable.

Disappointingly, both circumcision rates and the percent Muslim are weak instruments in the first stage of the model.[13] Neither is a strong and statistically significant predictor of HIV prevalence. Moreover, they enter positively in predicting HIV prevalence using the measure with the 1993 intercept—opposite to what the literature predicts. So it is not surprising that, in two-stage least squares estimations with circumcision or percent Muslim used as an instrument, I find no significant impact of HIV prevalence on the incidence of crime (results not shown). Because of concerns associated with omitted variables and reciprocal causation, the rationale for two-stages least squares estimation is strong if we are to identify precisely the causal impact of HIV on crime. However, these instruments are simply not up to the task.

Mechanisms Linking HIV to Crime

[13] The absence of a relationship between HIV prevalence and circumcision/Muslim holds in the reduced sample of districts in which sentinel site data on prevalence is available. While surprising, the lack of a relationship might be due to measurement error in the measure of circumcision or the fact that circumcision may be poorly correlated with the percentage of Muslims in some countries given broader circumcision practices.

Overall, the results suggest a positive association between HIV prevalence and crime. These findings are robust to the inclusion of fixed effects for region, thereby eliminating some concern that regional patterns of violence and instability in Uganda might be driving the results. The results are stronger and substantively larger for 1999 than 1992, suggesting that the impact of high HIV prevalence may accumulate over time. Moreover, there is some evidence that the impact of HIV prevalence is primarily on the incidence of property crime rather than violent crime. Earlier, I suggested that this relationship might operate through three plausible mechanisms: the foreshortening of time horizons, the growth of orphan and youth populations, and the decreasing deterrent capacity of state institutions. While the first mechanism predicts that HIV-positive individuals, or those that believe they are likely to contract the virus, will be more likely to commit crime, I lack the data required to evaluate the relative explanatory power of this mechanism.[14] The other two, however, can be investigated with the data that has been compiled. My results are presented in Table 3.

(Table 3)

The argument that emphasizes the demographic consequences of HIV suggests that high prevalence communities will also be those with large orphan populations and an age pyramid weighted heavily toward young men. Columns (1) and (2) examine whether HIV prevalence is positively associated with the percentage of households having a two-parent orphan (as measured in the 1997 UBOS Household Survey). Column (3) explores the impact of HIV prevalence on the share of the community that is composed of young men aged 15-29 (as measured in the 1999 UBOS

[14] One testable prediction that might follow from the first mechanism is that we would expect contemporaneous movements of prevalence and crime. Thus, with the decrease in prevalence between 1992 and 1999 in Uganda, we might anticipate a contemporaneous decrease in crime (while for the other mechanisms, the effects are expected to accumulate). There is no evidence to support this temporal pattern in the data, but because the model relies on expectations about life expectancies rather than actual prevalence, it is not a direct test. Indeed, as prevalence was falling in Uganda in the 1990s, the number of people aware of their status was likely on the rise.

Household Survey). In both cases, we cannot reject the null hypothesis that district-level variation in HIV prevalence does not account for variation in the orphan population or fraction of young men. Moreover, in OLS regressions with crime as the dependent variable, the inclusion of a control for the share of households with an orphan or the proportion of young men does not weaken the estimated coefficient of HIV prevalence.[15] While it is possible that this mechanism does not account for patterns of crime, it is nonetheless surprising that HIV prevalence does not predict the share of orphans or the age pyramid of the population. This might be because AIDS orphans are not necessarily the dominant part of orphan populations in Africa (although they are often a substantial share), or it might reflect the weakness of our imputed measures of HIV prevalence.

Turning to the mechanism highlighting processes of institutional decline, I look for evidence that districts with high HIV-prevalence exhibit diminished government capacity, particularly in the security sector. Here, I employ the crime data collected by the Ugandan government to compute a measure of arrest rates for each district, for two types of crime (murder and theft). Although the actual number of crimes is likely underreported, police stations are also asked to record the number of arrests they make for each crime; taking the ratio of arrests to crimes provides us with a proxy for government effectiveness in the security sector. The sorts of caveats identified earlier are of slightly less concern in our analysis of arrest rates. While differences in administrative capacity (rather than the incidence of actual crime) might drive the government statistics, rendering those variables less useful for our analysis of the relationship between HIV and crime, these differences in administrative capacity (and in particular, the capacity to make arrests for crimes reported) are exactly what we care about here. Yet, as we see in columns (4) and (5), there is no statistically

[15] When the sample is constrained to those districts with sentinel sites, there is a strong, statistically significant relationship between prevalence and the burden of orphans and the percentage of young men in the population (as we would expect). But including measures of orphan burden and the percentage of young men still does not weaken the relationship between HIV prevalence and crime among the sentinel sites.

significant relationship between HIV prevalence and the reported arrest rates for murder and theft.[16] Again, it is difficult to know whether this evidence against the plausibility of this mechanism or simply a reflection of the weakness of the data.

Conclusion

It is increasingly common to find arguments suggesting that the spread of HIV/AIDS will give rise to an increased incidence of civil war and the collapse of state institutions. Yet most of these hypotheses are advanced with little in the way of empirical evidence. In this article, I have put these conjectures to an empirical test by focusing attention on patterns of crime and victimization in Uganda, a country hit hard by the epidemic over the past two decades. I have argued that patterns of crime are a good proxy for the types of instability proposed in the literature; that the extensive literature on crime provides a way of identifying distinct causal mechanisms that might link HIV to victimization; and that the availability of sub-national data on patterns of crime allows one to investigate causal effects while controlling for many of the unobserved country attributes that might bias estimates in cross-national analysis. The evidence is strongly suggestive. I have found that districts with a high prevalence of HIV also tend to experience higher rates of criminal victimization. It appears as well that HIV tends to increase the level of property crime, but not violent crime, consistent with previous work suggesting that rational models of criminal behavior better explain economic crime. Though the evidence presented here is robust to the inclusion of standard control variables, there are several important issues that merit exploration in future research.

First, issues of data availability and quality, particularly at the sub-national level, severely hamper our ability to precisely estimate the causal impact of health conditions on social and political

[16] An alternative measure of institutional weakness draws on the residuals from a regression of reported crime (police statistics) on actual crime, with larger residuals acting as a proxy for administrative incompetence. There is no relationship, however, between prevalence and these residuals either.

outcomes. While the imputation procedure used in this article is defensible, better sub-national measures of prevalence would add to our confidence in the patterns identified here. More importantly, access to longitudinal data on both HIV prevalence and victimization would make it possible to generate reliable estimates of impact based on both temporal and cross-sectional variation.

Second, further investigation is needed to examine the mechanisms through which HIV/AIDS shapes patterns of social stability. This article makes a start in enumerating a set of plausible channels. The empirical tests, however, are disappointing in their ability to help us parse these mechanisms. Future work, in countries with better data, might shed some additional light on the relative explanatory power of these different stories linking HIV and crime. More broadly, it would seem important for political scientists concerned with the consequences of epidemic disease to spell out more precisely the micro-level pathways through which HIV/AIDS might change individual behavior and social outcomes. Identifying and providing evidence for distinct mechanisms is not simply an exercise of academic interest. Knowing how and why HIV/AIDS undermines stability is essential for thinking about policy responses to minimize its negative social and political consequences.

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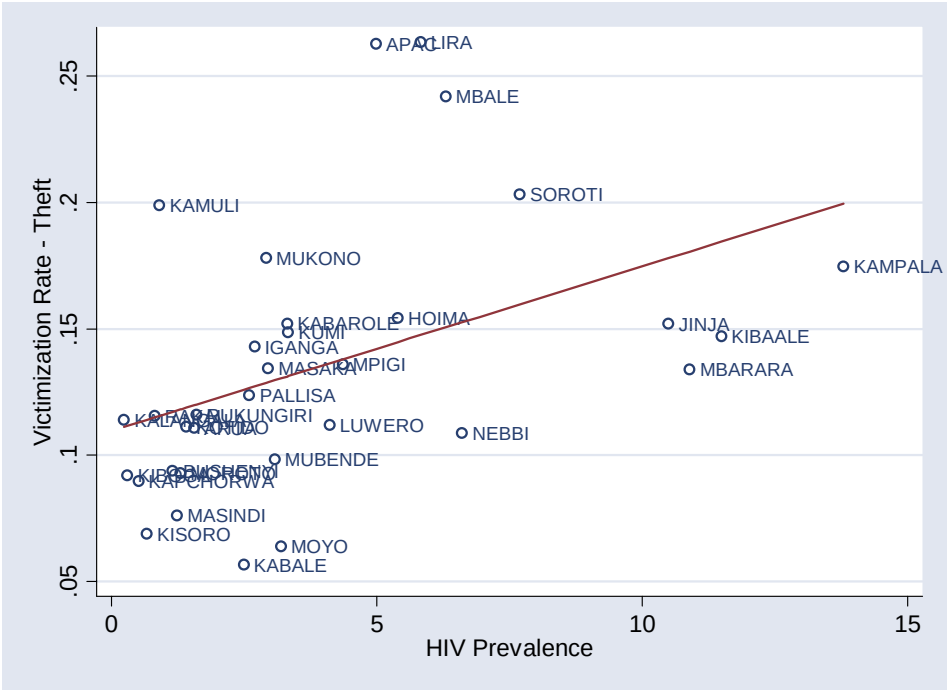
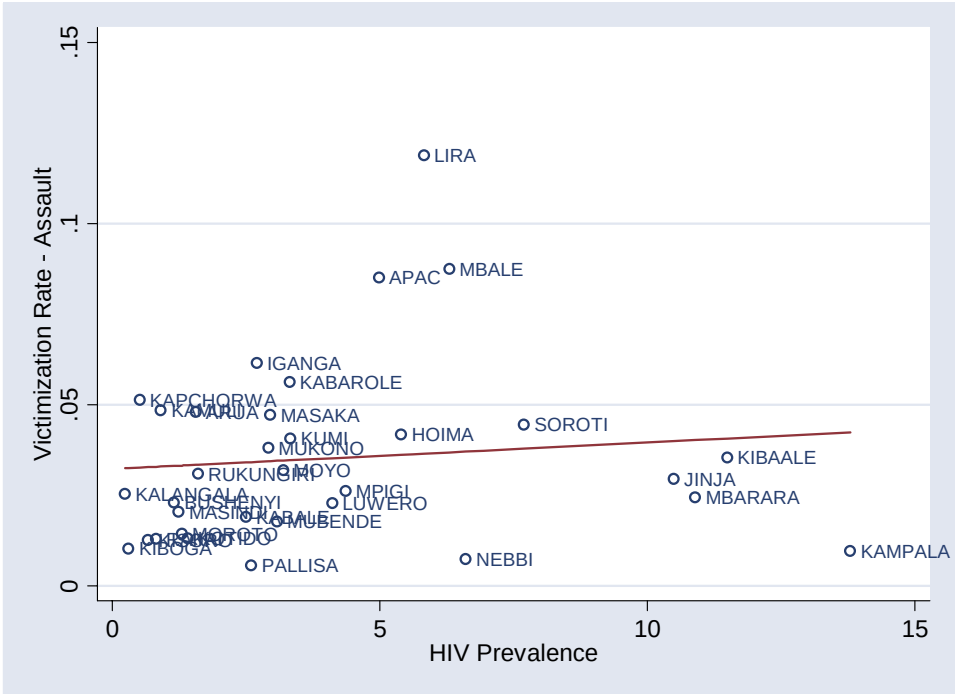
Figure 1: Bivariate Relationship between HIV Prevalence and Crime

(a) Theft



(b) Assault



(c) Crime

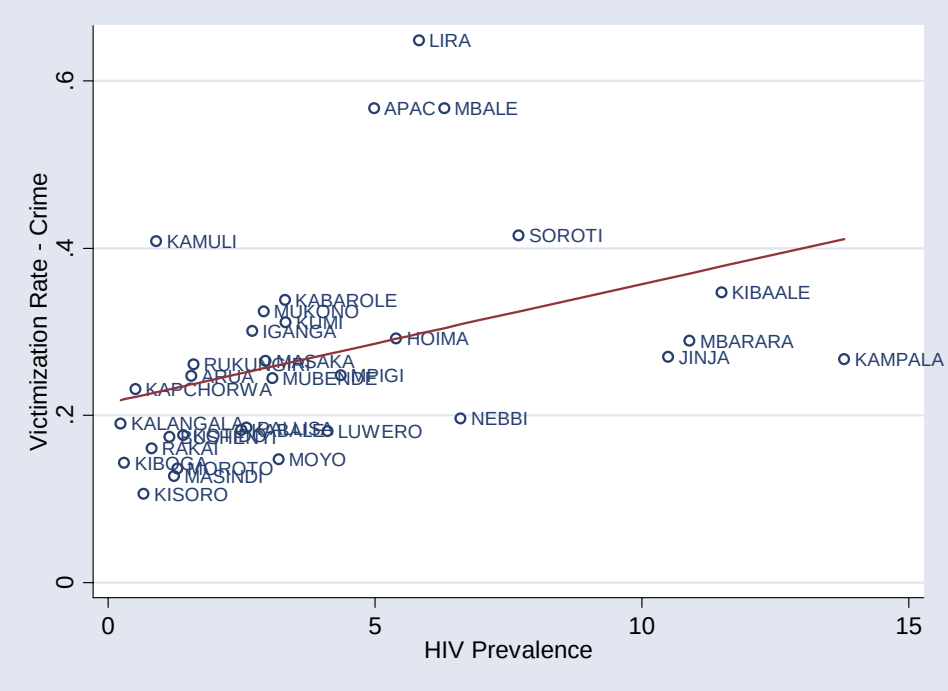

LIRA
APAC
MBALE
KAMULI
SOROTI
KABAROLE
KIBAALE
MUKONO
KUMI
IGANGA
HOIMA
MBARARA
JINJA
KAMPALA
MASAKA
MUBENDE
KAPCHORWA
NEBBI
LUWERO
MOYO
KISORO
Victimization Rate - Crime
0
.2
.4
.6
0
5
10
15
HIV Prevalence

Table 1: Measures of Crime and Victimization in 1992, Correlation Matrix

	Theft	Assault	Overall	Reported Violent Crime	Reported Economic Crime
Theft	1.0000				
Assault	0.7262	1.0000			
Overall	0.9246	0.8106	1.0000		
Reported Violent Crime	-0.0129	-0.0106	-0.0017	1.0000	
Reported Economic Crime	-0.1572	-0.1449	-0.1632	0.6598	1.0000

Table 2: Impact of HIV Prevalence on Crime, Multivariate Results

(a) Measure 1, 1993 Intercept

	1999			1992		
	Theft	Assault	Overall	Theft	Assault	Overall
	(1)	(2)	(3)	(4)	(5)	(6)
HIV Prevalence	0.0054	0.0011	0.0151	0.0007	0.0008	0.0041
	[1.76]*	[0.77]	[2.23]**	[0.76]	[1.44]	[1.73]*
Males	0.4374	0.2222	1.3333	-0.1060	-0.1204	0.1120
	[1.23]	[0.95]	[1.45]	[0.33]	[0.79]	[0.16]
Education	-0.0039	0.0043	0.0036	0.0074	0.0112	0.0214
	[0.40]	[0.76]	[0.14]	[0.85]	[3.35]***	[1.20]
Ethnic Diversity	-0.1356	-0.0688	-0.3601	-0.0799	-0.0341	-0.2655
	[1.70]	[1.88]*	[1.99]*	[2.42]**	[2.30]**	[3.51]***
Wealth Index	0.0604	0.0150	0.1130	-0.0677	-0.0510	-0.2104
	[2.00]*	[0.73]	[1.41]	[2.49]**	[4.32]***	[3.00]***
St. Dev. of Wealth Index	0.0888	0.0221	0.1309	0.0727	0.0589	0.3167
	[0.76]	[0.50]	[0.50]	[1.80]*	[3.60]***	[3.38]***
Road Density	-0.0404	-0.0482	-0.1905	0.0575	0.0116	0.1425
	[0.48]	[1.42]	[1.08]	[1.33]	[0.51]	[1.28]
Constant	-0.0449	-0.0662	-0.2931	-0.0198	-0.0609	-0.3888
	[0.24]	[0.61]	[0.63]	[0.14]	[0.82]	[1.24]
Observations	33	33	33	34	34	34
R-squared	0.42	0.34	0.46	0.36	0.56	0.49

Notes: Robust t-statistics in brackets. * significant at 10%; ** significant at 5%; *** significant at 1%.

(b) Measure 2, 1992 Intercept

	1999			1992		
	Theft	Assault	Overall	Theft	Assault	Overall
	(1)	(2)	(3)	(4)	(5)	(6)
HIV Prevalence	0.0042	0.0017	0.0106	0.0016	0.0009	0.0063
	[2.70]**	[1.63]	[2.89]***	[1.50]	[2.01]*	[2.52]**
Males	0.5062	0.2426	1.5167	-0.0292	-0.0925	0.3505
	[1.68]	[1.17]	[1.78]*	[0.09]	[0.60]	[0.46]
Education	-0.0075	0.0023	-0.0050	0.0046	0.0101	0.0124
	[0.94]	[0.45]	[0.24]	[0.52]	[2.84]***	[0.63]
Ethnic Diversity	-0.1147	-0.0582	-0.3090	-0.0848	-0.0361	-0.2815
	[1.72]*	[2.05]*	[2.13]**	[2.70]**	[2.63]**	[3.85]***
Wealth Index	0.0769	0.0204	0.1562	-0.0676	-0.0483	-0.1999
	[2.19]**	[0.78]	[1.58]	[2.28]**	[4.32]***	[2.65]**
St. Dev. of Wealth Index	0.1234	0.0344	0.2209	0.0838	0.0636	0.3536
	[1.12]	[0.85]	[0.88]	[2.12]**	[3.86]***	[3.89]***
Road Density	-0.0230	-0.0478	-0.1391	0.0594	0.0123	0.1485
	[0.30]	[1.42]	[0.83]	[1.58]	[0.63]	[1.65]
Constant	-0.1000	-0.0853	-0.4371	-0.0705	-0.0773	-0.5384
	[0.62]	[0.85]	[0.99]	[0.51]	[1.10]	[1.72]*
Observations	33	33	33	34	34	34
R-squared	0.49	0.43	0.51	0.42	0.61	0.58

Notes: Robust t-statistics in brackets. * significant at 10%; ** significant at 5%; *** significant at 1%.

Table 3: Mechanisms Linking HIV to Crime

	Demographic Structure			Government Capacity	
	% of Households with Orphan (15 and below), 1997	% of Households with Orphan (18 and below), 1997	% of Population between 15-29 (Male), 1999	Arrest Rate, Murder, 1997	Arrest Rate, Theft, 1997
	(1)	(2)	(3)	(4)	(5)
HIV Prevalence	-0.0011	0.0002	0.0006	-0.0037	0.0039
	[0.36]	[0.06]	[0.68]	[0.24]	[0.33]
Males	-0.0611	-0.0582	0.1904	4.3131	0.9942
	[0.19]	[0.18]	[1.73]*	[1.82]*	[0.68]
Education	0.0097	0.0115	0.0054	-0.0721	-0.0135
	[0.78]	[0.95]	[1.60]	[0.60]	[0.29]
Ethnic Diversity	0.0798	0.0958	-0.0016	0.1988	-0.2034
	[1.37]	[1.73]*	[0.08]	[0.56]	[1.24]
Wealth Index	0.0809	0.0876	-0.0107	0.2042	-0.1878
	[1.37]	[1.45]	[1.18]	[0.63]	[1.00]
St. Dev. of Wealth Index	-0.1532	-0.1655	0.0169	-0.4211	-0.0960
	[1.28]	[1.37]	[0.50]	[0.68]	[0.32]
Road Density	-0.1410	-0.1651	0.0230	-0.4313	0.1653
	[1.32]	[1.51]	[0.94]	[1.08]	[0.54]
Constant	0.2103	0.2171	0.0356	-0.5924	0.6650
	[1.90]*	[2.10]**	[0.66]	[0.84]	[1.08]
Observations	35	35	33	35	35
R-squared	0.18	0.24	0.45	0.22	0.21

Notes: Robust t-statistics in brackets. * significant at 10%; ** significant at 5%; *** significant at 1%.

Appendix A: Summary Statistics

Variable	Obs	Mean	Std. Dev.	Min	Max
Theft, 1999	34	.1353022	.0520164	.0566038	.2634033
Assault, 1999	34	.0355416	.0250926	.005618	.1188811
Crime, 1999	34	.2704412	.1280916	.106	.648
HIV Prevalence, 1998	37	4.180769	3.561114	.23655	13.8
% Males	34	.4878313	.0226882	.4404332	.5639344
Average Education of HH	34	4.847582	1.175636	1.497126	8.378451
Ethnic Diversity	38	.4476703	.2185918	.0402737	.8171253
Wealth Index	34	-.5036521	.3839687	-1.215324	.8299089
Standard Deviation of Wealth	34	.7189157	.1270925	.4595641	.9875126
Road Density	38	.1862272	.2097436	0	1.294261
% Circumcised	38	.0984839	.0986017	.0031395	.4833742
% Muslim	38	.0850614	.0746759	.0020449	.2841949