

# Male Circumcision and the Impact of AIDS in Africa

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## Abstract

Theories abound on the possible impact of AIDS on economic growth and savings in Africa yet there have been surprisingly few empirical studies to test the mixed predictions. In this paper, we examine the impact of the AIDS epidemic on African nations through 2003 using the male circumcision rate to identify plausibly exogenous variation in HIV prevalence. Though medical researchers are still in debate over whether lack of male circumcision can lead to increased susceptibility of contracting HIV, the statistical correlation between the two is compelling. We assemble national circumcision rates for African nations and find that they are both a strong and robust predictor of HIV/AIDS prevalence and uncorrelated with other determinants of economic outcomes. Two-stage least squares regressions do not reveal that AIDS has had any measurable impact on economic growth in African nations, however we do find that AIDS has had considerable effect on the structure of the economy and on humanitarian outcomes such as undernutrition.

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## I. Introduction

The AIDS epidemic is a humanitarian disaster of massive proportions that has struck sub-Saharan Africa with particular severity. Africa accounts for nearly three quarters of the world's HIV infections, even though it is home to one eighth of the world's population and represents less than one twenty-fifth of worldwide GDP. Infection rates are frightening: in Botswana, HIV prevalence is 37 percent and in several other nations in the "AIDS belt" that runs down southern Africa, more than one quarter of the adult population is HIV positive. The human impact is staggering: life expectancy in Zimbabwe has fallen 18 years in less than a decade and a half to a depressing 39 years. Over the next ten years, AIDS-related illnesses will kill more Africans than all of the wars and natural disasters of the previous five decades (FAO, 2003).

The economic impact of AIDS is much less clear. Developmental economists have debated what, if any, effect HIV rates have on economic outcomes. Since HIV afflicts adults, unlike diseases that prey on children and the elderly, it may have severe effects on deteriorating human capital and impeding economic growth (Dixon et al, 2001). The United Nations predicts that the AIDS epidemic will cause a "growth drag" of between 1 and 1.5 percentage points in annual per capita GDP growth in sub-Saharan Africa for the next two decades (UNAIDS, 2000). Other economic studies hypothesize that the decrease in life-expectancy brought about by the high prevalence of HIV will reduce incentives for younger Africans to save and receive an education, decreasing the availability of both physical capital and human capital (Ferreira and Pessoa, 2003; Bell, Devarajan, and Gersbach, 2003). However, AIDS may also increase the capital to labor ratio if the reduction of population growth outweighs the reduced savings rate (Over, 1992). If it acts like the Black Death did in Europe, AIDS may actually increase the per capita income through positive pressure on wages that reduce fertility (Young, 2004). As it turns out, there is not a consensus among empirically-based studies on the link between HIV prevalence rate and economic growth (see Bloom and Mahal, 1997, and Bonnel, 2000).

While the economic literature remains inconclusive, other developmental studies have focused on the humanitarian costs. In particular, a number of studies examine the

role that AIDS plays on nutrition levels and undernourishment (FAO, 2003; FAO, 2001; Haddad and Gillespie, 2001). High AIDS prevalence not only causes a shortage of agricultural laborers to diminish food supply, but also can lead to entitlement failure through income loss. HIV infection often renders parents unable to earn enough income to purchase sufficient food, and since traditional support networks are also stricken, the AIDS epidemic can exacerbate a malnutrition crisis. AIDS can have an especially potent impact on women, as it forces them to adopt the burden of care for sick family members (Drimie, 2002), which can force them out of the labor force and exacerbate their poverty. Even without AIDS, portions of sub-Saharan Africa were rampant with malnutrition and poor agricultural infrastructure. The impact of HIV puts these regions at even greater risk, and may allow climatic crises that normally cause hunger to develop into full-blown famine conditions (de Waal and Whiteside, 2003).

A clear understanding of the real impact of AIDS is essential to formulating the correct policy response, as the optimal response may consist of more than simple prevention and treatment. If AIDS is an economic disaster, and the strength of the economy is instrumental in mitigating the impact of the disease, then programs targeting economic capabilities in afflicted countries should be a central part of developmental agencies' AIDS programming. If, on the other hand, AIDS is not an economic disaster, then research should be undertaken on which other outcomes are worst affected, and interventions targeting these channels should be considered—especially if indirect effects are involved, which could be alleviated with nontraditional strategies.

Measuring the impact of the AIDS epidemic is by no means straightforward. Cross-country comparisons are subject to omitted-variable bias since unobservable factors—such as the “responsibility” of the government or the “prudence” of the population—that affect AIDS may also have an independent impact on the dependent variable. Reverse causality is another valid concern as many dependent variables (income and nutrition levels, for example) may also influence national AIDS rates, as people change their behavior in response to the epidemic, which can lead to different infection rates (Kremer, 1996). Additionally, measurement error plagues most AIDS studies regardless of the dependent variable, an especially salient worry given the recent controversy over national HIV rates; classical measurement error can lead to attenuation

bias where the magnitude of the coefficient is underestimated. In order to combat these identification problems, we utilize first differences and instrument for HIV prevalence using the male circumcision rate.

Suggesting that male circumcision practices can be used to predict AIDS rates is hardly a novel idea. Indeed, the link between male circumcision and HIV prevalence has been debated in the medical literature for at least 15 years and was exploited as an instrument by Lucia Breirova (2002) in a recent paper examining the impact of AIDS on education in Kenya. Although the medical community has yet to establish with certainty that lack of male circumcision increases risk of HIV infection, statistical evidence demonstrates a strong association between low male circumcision rate and high HIV prevalence. Even if the jury is still out on whether circumcision protects against HIV infection, we can use male circumcision as an instrument for AIDS rates so long as it predicts exogenous variation in HIV/AIDS rates at the national level. In this paper, we argue that male circumcision in Africa does not co-vary with any salient omitted variables, such as initial income, initial life expectancy, risky sexual behavior, or modernity, and that the coefficient of male circumcision on HIV/AIDS remains large and significant even after controlling for religion and ethnicity. We then use a two-stage least-squares, first-differenced model on several outcome variables, testing AIDS effects on both economic and humanitarian measures between 1980 and 2003.

We find that AIDS has not had a measurable impact on several key economic and political variables in Africa. Specifically, HIV rates do not have a significant effect on GDP per capita, democracy, savings rates, school enrollment, or fertility. However, we do find that HIV has had a substantial impact on the structure of the economy, reducing female economic activity and hurting the growth of the agricultural sector. In addition, we find a large and negative impact of AIDS on humanitarian variables such as nutrition rates and of course life expectancy. Given the humanitarian damage inflicted by AIDS, one might hope to see a response by the international community, yet we do not find any evidence that foreign aid has been targeted to African countries worst hit by the disease.

This paper is organized as follows. In Section II, we estimate national male circumcision rates, examine the first stage of AIDS on circumcision, and subject the instrument to various robustness checks. In Section III, we use differenced regressions to

examine the impact of AIDS on economic, political, structural, and humanitarian outcomes. In Section IV, we conclude and discuss the implications of our results.

## II. Male Circumcision and AIDS in Africa

There is high variation in male circumcision practices both across and within countries in Africa. In primarily Muslim North Africa, male circumcision is treated as a religious ritual as prescribed by Islamic hygienic laws, while many sub-Saharan tribes incorporate male circumcision as a component on initiation rituals into manhood. Linguistic evidence indicates that male circumcision practices among the Bantu peoples of sub-Saharan Africa predate colonial influences (Marck, 1997). Moreover, available accounts suggest that male circumcision remains a powerful tradition in many African cultures (Mandela, 1994: 27; Saitoti, 1988: 19). Even so, it is possible that some tribes may have experienced a reduction in the proportion of their boys that are circumcised while others may have experienced an increase since tribal practices were measured. At any rate, we will use circumcision data by tribe from Murdock (1967) which predates the AIDS epidemic, so any effects that AIDS may have had on circumcision rates will not be tainting our data.

Although far from medically proven, the balance of the existing data suggests that it is not implausible to assume a causal relationship between male circumcision and HIV infection for Africa. In Appendix A we provide a summary of this literature. Yet our instrument does not *require* that circumcision protect against AIDS. Importantly, for the validity of male circumcision as an instrument for AIDS rates, we must only establish two conditions. First, we must show that our instrument (male circumcision) is a strong predictor of the independent variable (HIV prevalence). For example, circumcision may be correlated with other traditional practices, such as “wife cleansing” (sexual intercourse between a new widow and men of the village), that have an independent effect on the transmission of HIV. If circumcision has no direct effect on HIV transmission, but is picking up these practices, then we are still identifying plausibly exogenous variations in the AIDS rate. Second, we must establish that male circumcision does not covary with

important omitted variables that could impact the outcomes of the regression. If male circumcision rates satisfy these conditions, the instrument is acceptable for the purposes of this paper, irrespective of the debate over whether it is causal. The remainder of this section argues that these two conditions are met.

### *Measuring circumcision*

In order to implement male circumcision rate as an instrument in a cross-country two-stage regression model, we needed to formulate estimates of national male circumcision rates. Since data on male circumcision practices are in the form of ethnographic practices at the tribal level (largely from Murdock, 1967), we used demographic breakdowns of countries by tribe in order to convert the information on tribal practices to national male circumcision rate estimates.<sup>1</sup> With the binary assumption that male circumcision was either universal or absent for any given tribe, we calculated a male circumcision rate for each nation.<sup>2</sup> For example, for Kenya:

<b>KENYA'S TRIBAL BREAKDOWN</b>		
<b>Tribe</b>	<b>% of Population (Alesina, 2003)</b>	<b>Male Circumcision (Murdock 1967)</b>
Kikuyu	22%	Y
Luhya	14%	Y
Luo	13%	N
Kalenjin	12%	Y
Kamba	11%	Y
Kisii	6%	Y
Meru	6%	?
other African	15%	?
non-African	1%	?

From the above chart, we estimate that 65 percent of Kenyan males are circumcised while 13 percent are not circumcised. The remaining 22 percent are unknown, so we assume a 0.5 probability that any unknown male is circumcised, which is close to the continental average. Thus, mapping the ethnographic data on national demographic breakdowns, we estimate the male circumcision rate for Kenya to be

<sup>1</sup> The demographic breakdowns by tribe are from the dataset generated by Alesina and coauthors (2003), accessible at <http://www.stanford.edu/~wacziarg/downloads/fractionalization.xls>.

<sup>2</sup> We thank John Caldwell for this suggestion.

$(1*0.65) + (0.5*0.22) + (0*0.13) = 0.76$ . We assemble national male circumcision rates for all countries in continental Africa as well as Madagascar and Comoros using this methodology.

Although our estimates of African national male circumcision rates represent the most comprehensive and precise approximations available, there are earlier estimations of male circumcision rates of African nations. The first attempt to gauge national circumcision rates in Africa, using a methodology similar to ours, produced estimates for a subset of the countries (Bongaarts et al., 1989).<sup>3</sup> A decade later, Halperin and Bailey (1999) grouped African nations into broad groups of high (>80%) and low (<20%) male circumcision prevalence, which were expanded on by Shelton (2002) to cover more countries and add a middle group (20-80%). Appendix B.1 compares our estimates to those of Halperin and Bailey and Shelton, henceforth H/B. Ascribing values of 1 through 3 for low through high, there is a high correlation (0.82) between our approximations and those generated by H/B, and we use both estimates in our empirical work.

### *AIDS on circumcision*

In the first stage of the model, we test how well national male circumcision rates predict HIV prevalence levels in African nations. Our dependent variable, the national AIDS/HIV prevalence rate in 2003 (percent of adults age 15-49), is from the UNAIDS/WHO 2004 *Report on the global HIV/AIDS epidemic*. We only use the single year as the estimation methods have changed over time, and UNAIDS recommends against comparing estimates across time.<sup>4</sup> The average prevalence across the countries in Africa was 7.9 percent, ranging from less than 0.1 percent in Tunisia to 38.8 percent in Swaziland.<sup>5</sup> A data appendix for all the variables we use is included as Appendix B.2 with summary statistics in Table 1.

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<sup>3</sup> There is a correlation of 0.69 between the figures from Bongaarts et al and our estimates for those countries. Bongaarts et al used an ethnographic map overlaid on country borders to determine the tribal breakdown.

<sup>4</sup>[http://www.unaids.org/Unaid/EN/Resources/Epidemiology/How+do+UNAIDS\\_WHO+arrive+at+estimates.asp](http://www.unaids.org/Unaid/EN/Resources/Epidemiology/How+do+UNAIDS_WHO+arrive+at+estimates.asp), point 2.

<sup>5</sup> There has been controversy in recent news over whether the WHO numbers are overestimates (Donnelly, 2004). If the prevalence rates are systematically overestimated by a constant proportion, then (a) the signs and significance of the coefficients are correct, but (b) the size of the impact of the explanatory variables in the first stage will be overestimates while (c) the size of the impact of HIV/AIDS in the second stage will

Figure 1 plots the HIV/AIDS prevalence rate against the male circumcision rate, revealing an obvious negative correlation. The first column of Table 2 expresses this figure in regression coefficients. We find a large, negative effect of circumcision on AIDS that is statistically significant at the one percent level. To interpret the coefficient, we perform the following thought experiment: going from a totally uncircumcised country to a totally circumcised country predicts a decrease in the infection rate of HIV by over 18 percentage points. Cognizant of the criticism that the observed correlation between circumcision and AIDS may be driven by underlying factors, we include a number of key control variables in the two following regressions.<sup>6</sup>

Column 2 includes controls for the log of GDP per capita, the log of population density, the percentage of the population living in urban areas, and the Polity2 score that measures the level of democracy in the country (where -10 is a perfect autocracy and 10 is a perfect democracy). Certainly population density and urban residence may have effects on the HIV prevalence rate in the country independent of male circumcision, and GDP per capita and Polity2 may have important effects on later dependent variables such as economic growth and educational enrollment. The coefficient on male circumcision retains its significance and is still a large 14 percent. Of the controls, only GDP per capita has a significant effect on the HIV prevalence rate. An increase in GDP per capita of 10 percent predicts an increase in the HIV rate of 0.5 percent, perhaps due to the mechanisms of disease transmission (migrant labor, etc.) being facilitated in wealthier African economies. In column 3 we include three additional ethnic, religious, and linguistic controls: the ethnolinguistic fractionalization, the fraction Muslim, and the fraction of the population from a Bantu-speaking tribe. As expected, Muslim countries have lower AIDS rates, and including the fraction Muslim reduces some of the magnitude of the coefficient on circumcision.<sup>7</sup> Strikingly, the impact of circumcision on the AIDS rate is larger than the impact of the Muslim variable (which includes circumcision as well as behavioral norms). Controlling for these seven variables, going from an

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be an underestimate. If the prevalence rates are simply measured with a lot of error, then the need for instrumental variable regressions becomes all the more crucial.

<sup>6</sup> We do not control for health expenditures because it is higher in high-HIV countries perhaps due to medical expenses. We also do not control for other STIs which could obfuscate the relationship between circumcision and AIDS.

<sup>7</sup> Including the square of the fraction Muslim (allowing for more complex dynamics of culturally-specific sexual behavior) does not change the coefficients on circumcision.

uncircumcised population to a circumcised population predicts a decrease in the HIV prevalence rate by over 10 percentage points, and the result remains significant at the five percent level. Figure 2 plots the predicted HIV prevalence rate from this last regression (excluding the circumcision rate) against male circumcision, and the obvious negative relationship from Figure 1 remains.

To ensure that idiosyncrasies in our male circumcision rate estimates were not responsible for the strength of our results, we ran the same regression using the H/B estimates in column 4, where we converted the H/B circumcision estimates of medium (20-80 percent) and high (80-100 percent) into dummy variables. This also allows us to perform a non-parametric estimation.<sup>8</sup> Both coefficients on those estimates are strongly significant: countries with medium circumcision levels are associated with a decrease in the HIV prevalence rates of 11 percentage points and countries with high circumcision levels with a decrease of 16 percentage points relative to countries in the low circumcision group. This last coefficient is comparable to our thought experiment of moving from zero to total circumcision, and has a similar coefficient to the circumcision rates that we assembled.

Another potential critique might be that the nations of North Africa with low HIV prevalence and nearly complete male circumcision are driving these results. To test this possibility, we repeated regressions 3 and 4 excluding the North African nations. The coefficients on both our male circumcision estimates in column 5 and on the H/B dummies in column 6 remained statistically significant and close in magnitude to their counterparts in the full African sample. At first glance, then, the first-stage results demonstrate that male circumcision rates appear to be a powerful and robust predictor of HIV prevalence in 2003 for African nations.

#### *Validity of the instrument*

Of course, it remains possible that male circumcision is simply picking up omitted variables that may have an independent effect on the dependent variable in the two-stage regression. To address this concern, we will (1) examine whether the causal mechanism

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<sup>8</sup> We also ran some nonlinear approaches of our own circumcision rate estimates including a squared term and the log of circumcision, but found the simple linear version had the strongest fit.

proposed in the medical literature is supported by our cross-country data; (2) test whether circumcision is correlated with major potential omitted variables; and (3) run our two-stage regressions (in the following section) as differences in order to control for any time-invariant unobservable factors.

We can run a rudimentary test of the causal mechanism suggested in the medical literature that lack of male circumcision increases the prevalence of genital sores, which in turn increase fluid exchange during intercourse, making non-circumcised men more susceptible to both contract and spread HIV (Szabo and Short, 2000). Using data from the late 1990s Demographic and Health Surveys (DHS), we look at the impact of circumcision on self-reported sexually-transmitted infection (STI) rates and genital ulcer rates. Among sexually-active men, an average of 3.81 percent reported an STI in the previous year, while 2.27 percent reported a genital ulcer in the previous year. Among sexually active women, 2.69 percent reported an STI. Available medical evidence suggests that these numbers are gross underestimates (see Orroth et al, 2003), so the results should be taken only as suggestive.

We regressed the male and female STI rates on circumcision and the basic controls from column 2 of Table 2 and found a negative, though statistically insignificant, impact of circumcision on STIs (not reported). Regressing male genital ulcers on circumcision, we found that higher circumcision is associated with lower rates of genital sores, a result that is significant at the 5 percent level. Specifically, going from an uncircumcised population to a circumcised population would lower the prevalence of genital sores by 2.99 percent, a number larger than the mean level of genital sore prevalence. We repeated the regressions using the H/B dummies and found consistent results. The relationship revealed in the data, that male circumcision is associated with a lower prevalence of genital sores, is consistent with the proposed mechanism in the medical literature. It is also consistent with microdata from India: Reynolds et al (2004) report in the *Lancet* that HIV-negative circumcised men had a lower baseline of genital sores than uncircumcised men, were less likely to contract HIV, but developed the same incidence of other STIs as uncircumcised men. Still, in order to validate the use of male circumcision as an appropriate instrument, we must reject potential mechanisms through which it could bias the second stage. It is difficult to imagine a direct impact of

circumcision on economic growth, but perhaps the national circumcision rates may act as proxies for important omitted variables.

A possible concern is that the male circumcision rate is positively correlated with characteristics that are conducive to economic growth. To test this hypothesis, we measured the correlation between male circumcision rates and a variety of country characteristics from 1980, before the AIDS epidemic had taken off (first half of Table 3). We find that male circumcision is not correlated with initial GDP per capita and weakly negatively correlated with population density. There is a weak positive correlation between male circumcision and the domestic savings in 1980, suggesting that circumcised countries might be financially more forward-looking, however the result is not statistically significant. And there is an equally weak negative correlation between male circumcision and the level of democracy in 1980, suggesting that circumcised countries were not characterized by more democratic governments, but this result is also not statistically significant. Male circumcision is not correlated with life expectancy in 1980, indicating that it is not serving as a proxy for health.

The only significant results were a positive relationship between male circumcision and urban population as well as a negative relationship between male circumcision and adult literacy rates in 1980. Since low literacy rates and large urban populations have ambiguous—but likely negative—effects on an economy, these results do not reveal obvious weaknesses in the instrument. Even so, we run the gamut of regressions of Section III controlling for initial literacy and urban population (not reported), and the results do not change appreciably.

We also check whether male circumcision is correlated with the “modernity” of the population. After all, populations that adopt one new technology may be more likely to adopt other ones which could be conducive to economic growth. Table 3 shows the correlation between male circumcision and the number of radios and vehicles per 1000 population. The correlations are weak and insignificant, as they are using a variety of other technology-related variables that we do not report.

Even if male circumcision is correlated with neither typical preconditions for economic growth nor the adoption of newer technologies, it may be correlated with the level of risky sexual behavior of a given population. If circumcised tribes are also

sexually prudent, then we may observe a spurious correlation between circumcision and AIDS. Moreover, the prudence of the population may also be correlated with other characteristics that cannot be measured but may be conducive to good outcomes such as economic growth or high education. Thus we would like to be able to see whether circumcision is correlated with pre-AIDS sexual behavior. Unfortunately, such data are not available. The best available national data on risky sexual behavior for Africa rely on self-reported measures which may be systematically biased with respect to the AIDS environment. Moreover, they are only available since the AIDS epidemic got under way; clearly, the epidemic could lead to changes in sexual behavior, so these measures are far from perfect.

That having been said, the correlations between these measures of risky sexual behavior and male circumcision (Table 3, end) do not cast doubt on the validity of the instrument. We use two measures of risky sexual behavior, and have data for both men and women for around half of the countries. The first measure is whether adults report high-risk sexual activity, defined as sex with a non-regular, non-cohabiting partner, within the last year. Male circumcision is weakly negatively correlated with high-risk activity for both men and women, though the result is insignificant. On this dimension, circumcised populations appear slightly more prudent. The second measure is whether adults report using a condom at their last high-risk sexual activity. There is a negative relationship between circumcision and condom use at last high-risk sex among male respondents, which, though statistically insignificant, is stronger than the high-risk sex correlation. In other words, circumcised populations may be having slightly less high-risk sex, but they are also less likely to use condoms during those interactions.

Thus, the net association between circumcision and risky sexual behavior is statistically undetermined, and the signs of the correlations point to an ambiguous relationship. We do not interpret this as evidence that circumcised populations are less risky in their sexual behavior. In fact, we do not find any evidence that indicates that the use of male circumcision as an instrument for AIDS prevalence is worrisome in any discernible way.

### III. The Impact of AIDS

#### *Empirical strategy*

Despite the tests of our instrument in the previous section, any cross-country regression model is subject to the possibility of unobservable country-specific characteristics that may bias coefficient results. For example, a country's leader may select policies that impact the growth environment while also deciding how to deal with the AIDS crisis. Omitting the leadership variable will bias the estimate of the elasticity of growth with respect to AIDS. To combat this problem, we use differencing methods in our regressions. We assume that the level of the dependent variable in country  $i$ —for example, income per capita—in 2003 is a function of the AIDS rate of country  $i$  in 2003, political and economic controls  $X_i$  in 2003, plus some (unobservable) country-specific effect,  $\mu_i$ , that does not vary over time:

$$(1) \ y_{i, 2003} = \alpha + \beta * AIDS_{i, 2003} + \gamma * X_{i, 2003} + \mu_i + \varepsilon_{i, 2003}$$

Correspondingly, the level of income per capita in 1980, at the beginning of the AIDS epidemic being discovered, is:

$$(2) \ y_{i, 1980} = \alpha + \beta * AIDS_{i, 1980} + \gamma * X_{i, 1980} + \mu_i + \varepsilon_{i, 1980}$$

In this case, we assume  $AIDS_{i, 1980}$  to be zero. Clearly, there were already existing cases of AIDS in Africa, but the numbers are miniscule compared with 2003 and country-level data are simply not available. Thus we can subtract (2) from (1), and the country-specific effect  $\mu$  drops out:

$$(3) \ \Delta y_i = \alpha' + \beta * AIDS_{i, 2003} + \gamma * \Delta X_i + \varepsilon'_i$$

What (3) gives us is the regression of the change in GDP per capita from 1980-2003 on the AIDS rate and on the change in the control variables over the same time period. Any characteristic that does not change appreciably over a two-decade period, such as religion

or culture, does not need to be included in the estimations.<sup>9</sup> Empirically, we may be worried that the AIDS rate affects the evolution of the  $X$  variables, so we run the differences both (a) with no controls and (b) controlling for the change in the Polity2 score and the change in the log of GDP per capita (when they are not the left-hand side variable).

*Is AIDS an economic disaster?*

The bulk of economic literature on the impact of AIDS in Africa predicts a frightening set of scenarios.<sup>10</sup> The first channel focused on by economists was the reduction in the savings rate caused by AIDS-related expenditures (Over, 1992). With reasonable assumptions including a boon to growth from a reduction in the population growth rate, Over estimates a net negative effect on growth rates of around a third of a percentage point for African economies worst hit by the epidemic. This may be sensitive to whether the country is an open-economy, in which case reduced foreign direct investment and increased capital outflows can exacerbate the negative effects of AIDS (Haacker, 2002), or whether market inefficiencies such as unemployment are lessened, in which case the negative effects of AIDS are lessened (Cuddington, 1993). Similarly, savings may fall through the drastically lower life-expectancy rates. The shortened expected lifespan reduces incentives to save, which prohibits the capital accumulation central to models of economic growth (Arndt and Lewis, 2000). In a general equilibrium model, Arndt and Lewis predict that the income level in 2010 will be 7 percent lower in South Africa as a result of AIDS. Following the lines of that argument, the shortened lifespan also diminishes incentives for individuals to invest in education since the payoff to human capital investments decreases with falling life-expectancy (Ferreira and Pessoa, 2003). Ferreira and Pessoa predict a long-run drop in per-capita income of one quarter, driven in part by a reduction in schooling of one half. Existing literature on the impact of AIDS on democracy, though in early stages, is similarly pessimistic (Manning, 2002).

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<sup>9</sup> Of course, there may be time-invariant factors that have time-specific effects. For example, it may have been disadvantageous to be prudent during one period but advantageous during another. In this paper, we do not attempt to identify and control for such possibilities.

<sup>10</sup> See Gaffeo (2003) for a recent review of the literature.

This literature is not without its optimistic outliers. Studies by ING Barings (2000) and the Bureau for Economic Research (2000) predict a positive impact of AIDS on the South African economy. These have been criticized by Acott (2000) for their demographic projections and by Gaffeo (2003) for the primary channel of growth increase being through a higher demand for AIDS-related expenditures. Most recently, Alwyn Young (2004) has argued forcefully that AIDS may not be the economic disaster (in terms of per capita income) forecast as above. In his simulation of the South African economy, AIDS reduces the labor supply, putting positive pressure on the real wage which, in turn, lowers fertility (thus sustaining the rise in real wage). This effect should counteract the negative impact on the accumulation of human capital among orphans, leaving the economy with higher per capita output than in a no-AIDS scenario.

The negative impact of AIDS on population growth will be even stronger if fertility is reduced due to fear of mother-to-child transmission, longer abstinence, widowhood, reduced fecundity, increased condom use, and the desire to not leave progeny as orphans. However, AIDS could perversely increase fertility if families desire to have more children in response to a higher expected mortality per child, if women reduce breastfeeding in an effort to prevent transmission of the disease to a child, or if women reduce post-partum abstinence to discourage the husband from extra-marital sex that increases his risk of acquiring HIV (Ntozi, 2001). Evidence suggests that HIV-positive women have lower pregnancy rates than women who are not HIV-positive (Gray et al, 1998; Desgrees du Lou et al, 1998; Zaba and Gregson, 1998), though the net impact on fertility including the fertility-increasing behavioral response on the part of non HIV-positive women cannot be determined from these studies.

Surprisingly, there are few cross-country empirical studies on the macroeconomic effects of AIDS. The most widely-cited is Bloom and Mahal (1997), which looks at the impact from 1980-92 across 51 developing and industrial countries. Bloom and Mahal do not find a statistically significant relationship between AIDS and economic growth. Bonnel (2000) regresses growth from 1990-97 on AIDS in 1994-97 for approximately 80 developing countries and finds a negative impact of AIDS on growth that is statistically significant for the square of the log of the HIV/AIDS prevalence rate. Dixon, McDonald, and Roberts (2001) perform dynamic panel regressions from 1960-98 on African

economies where HIV enters the growth equation only through health capital. Their results suggest that the impact of HIV for low-AIDS countries is minimal, and for high-AIDS countries is unclear. In our view, it seems appropriate to worry about omitted variable bias and measurement error more than previous studies have done. For this, we now turn to our differenced and instrumented estimates.

Using both ordinary least squares (OLS) and instrumented two-stage least squares (2SLS) regressions, in Table 4 we test the hypothesis that AIDS has impacted economic outcomes in Africa from 1980 to 2003. Each row in the table represents a different dependent variable, and each column represents a different specification. The coefficients report the marginal impact of a one percentage point increase in the HIV prevalence rate on the outcome variable. Columns 1-3 do not include controls, and columns 4-5 control for the change in  $\ln(\text{GDP per capita})$  and in the Polity2 score. Column 1 is a simple OLS specification; in columns 2 and 4, AIDS is instrumented with the linear circumcision rate, and in columns 3 and 5, with the H/B dummies for medium and high circumcision.

The first row of Table 4 examines whether AIDS has had an impact on economic performance, specifically, on the change in per capita income from 1980 to 2003. The coefficients across all five specifications are insignificant and close to zero. Instrumenting for AIDS in columns 2 and 3, and adding the control for change in democracy does not impact the magnitude of the coefficient. The second row looks at the impact of AIDS on the change in the Polity2 score from 1980 to 2002, or level of democracy/autocracy. Again, the coefficients are insignificantly different from zero. The only substantive change is adding the controls for change in GDP per capita. Once that is factored in, AIDS appears to lead to a *higher* level of democracy, but the result is not statistically significant.

Testing the notion that the AIDS epidemic skews incentives to save, we examine how AIDS rates have impacted the change in gross domestic savings as a percent of GDP from 1980 to 2003. The third row of Table 4 reports our results. Again, the pessimistic theoretical predictions are not manifested in the data. The reduced-form results of columns 1-3 find a *positive* though statistically insignificant impact of AIDS on the savings rate. When controls for the change in income and democracy are included, the

sign reverses but the coefficients remain statistically insignificant. Thus, although the deterioration of incentives to save is an interesting economic insight that may yet come into play, empirical data do not find that the AIDS epidemic has had this effect as of 2003.

To measure the impact of AIDS on schooling, we use the change in primary, secondary, and tertiary enrollment as our dependent variables. Rows four through six of Table 4 report the results. We do not find evidence that AIDS has reduced school enrollments; indeed, the majority of coefficients are positive, though statistically insignificant. The one significant result in this exercise is a positive relationship between instrumented AIDS and tertiary education, controlling for changes in income per capita and democracy. However, this result should be taken with a grain of salt as the coverage of the tertiary education variable is very thin.

The AIDS epidemic also appears to have had no effect on fertility rates in Africa over 1980-2003. The final row of Table 4 repeats the analysis with the change in total fertility rate as the dependent variable. The measured coefficients are close to zero and insignificant. While this may mean that AIDS is not influencing fertility decisions, another possibility is that both the positive and the negative effects of AIDS on the fertility rate may be occurring with the net impact inconsistent across countries.

Despite a host of interesting and economically sound mechanisms through which the AIDS epidemic could severely hinder economic growth in Africa, the empirical data do not support any of the proposed hypotheses. However, it should be noted that the recent nature of the epidemic combined with the relatively lengthy incubation period of the virus could cause the effects to be delayed. As more and better data are produced, these theoretical predictions should be retested, and future empirics could be more consistent with the pessimistic models.

#### *AIDS and the structure of the economy*

As with models of economic performance, projections of the impact of AIDS on the structure of economic activity tend to emphasize the destructive power of the epidemic, depicting net out-migration from the cities, a reduction in the labor force, and shattered manufacturing and agricultural sectors. Dyson (2003: 433) notes anecdotal

evidence of town dwellers returning to their home rural areas when they fall ill as well as surviving household members leaving the cities following an AIDS death in the family. He also posits that, as AIDS is agreed to strike urban areas harder, higher AIDS-induced crude death rates and lower AIDS-induced crude birth rates in the cities should contribute to reduced urban population growth from AIDS.

Labor force participation is another structural outcome for which African data exist from the beginning of the epidemic. AIDS can affect participation in the work force through four main channels. One, it may lead to higher workforce participation as reduced population growth renders labor more scarce and unemployment falls. Two, it may lead to lower workforce participation as adult morbidity rises: a higher percentage of working-age people are simply too sick to participate in the job market. Three, it may reduce workforce participation by forcing families to sell off employment-generating capital such as livestock and land in order to pay for medical and funeral costs (Drimie, 2002: 16).<sup>11</sup> Four, AIDS may pull adults out of the workforce to care for sick relatives (Stokes, 2003), a burden that disproportionately falls on women (Drimie, 2002: 20). Since the first three effects should not disproportionately affect women, we can explore the depth of the “caregiving” effect by comparing the difference in the impact of HIV/AIDS on men and women.

Finally, AIDS is perceived to debilitate the agricultural and manufacturing sectors. Drimie (2002) surveys the potential impact on the agricultural sector, noting the depletion of human capital through AIDS-related mortality, a substitution of less labor-intensive crops, and the selling of agriculture-specific capital to cover AIDS-related costs. Stover and Bollinger (1999) survey the potential impact on firms and note absenteeism, health care costs, and low morale can reduce productivity. Decreased profit, in turn, can slow expansion. A recent survey in South Africa found that 40 percent of manufacturers surveyed reported lower profits as a result of the HIV/AIDS epidemic (Agence-France-Presse, 2004).

What some of these hypotheses fail to take into account is the possibility of substitution; if urban population growth is naturally lowered by AIDS, then that should

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<sup>11</sup> Loewenson and Whiteside (2001:10) report funeral expenditures to be up to 50 percent higher than medical care expenditures in Tanzania and Thailand.

create a premium of migration to the cities; migrants who in the absence of AIDS may have stayed in the countryside would then contemplate a move to the city. Likewise, when high-paying workers in the manufacturing sector drop out of the workforce, there could be a queue of healthy workers ready to take their place. Thus the hypotheses and micro evidence put forward in previous research do not imply that macroeconomic equilibria have been affected. To determine the equilibrium effect we must turn to the data and measure the impact of exogenous variation in the AIDS rate on sectoral outcomes.

Table 5 is organized the same way as Table 4: each row represents a different dependent variable and each column a different specification. In the first row we test the impact the change in AIDS has had on the change in urban population. The OLS result in the first column is weakly negative and statistically insignificant. When we instrument for HIV in the second and third columns, with the linear circumcision rate and the H/B dummies respectively, the magnitude of the negative effect increases but so does the standard error, and the results remain statistically insignificant. The magnitude suggested is that ten percentage points more in HIV prevalence predicts an urban population that is around two percentage points of the population lower. These results are little changed when controls for the change in GDP per capita and Polity2 are introduced in columns 4 and 5.

In the second and third rows we test whether AIDS has had an effect on the labor force participation of men and women. Looking at changes in the economic activity rate from 1980 to 2000 for both genders, we find that AIDS causes significant drops in female economic activity while not affecting male economic activity rates. The differencing method implies that in high-HIV prevalence nations, female labor participation declined relative to the “control” trend in low-HIV prevalence nations during the AIDS epidemic. The instrumental variable results confirm the OLS results, lending credibility to the plausible exogeneity of the impact of the epidemic on drops in female work force participation. According to the OLS specification (column 1), an increase in the HIV/AIDS prevalence rate by one percentage point decreases the percentage of females in the labor force by 0.1 percent. This may be underestimated due to measurement error in the HIV statistics or, indeed, biased due to some omitted variable. We repeat the

regression using male circumcision to instrument for HIV/AIDS, and the absolute value of the coefficient increases to 0.15 using the linear instrument and remains at 0.1 with the H/B dummies as the instrument. Controlling for changes in GDP per capita and Polity2 that may be unrelated to the AIDS epidemic, the absolute value of the coefficient rises to 0.18 and 0.15 with the two instrumental strategies. In other words, a one percent increase in the HIV prevalence rate leads to a decline in the female labor force participation rate of somewhere between one tenth and one fifth percent through 2003 in Africa.

A study in Uganda begun in 1990 among randomly-selected HIV-positive individuals reported a median time of 4.3 years before progression to full-blown AIDS (Morgan et al, 1997). Assuming a uniform distribution of progression to AIDS among the sample group, this implies that the median group member was already about halfway to AIDS. The median survival time among individuals who had AIDS was 9.3 months. Assuming HIV-positive individuals who do not have AIDS can still work, whereas AIDS sufferers cannot, this implies a ratio of employable to non-employable years of approximately 11. Hence a back-of-the-envelope calculation suggests that, if morbidity and care-giving were the only effects of AIDS on female workforce participation, an increase in the national HIV/AIDS prevalence rate of ten percentage points would remove almost one percent of adult women from the labor force through care-giving alone. And, if the other three avenues through which AIDS impacts labor force participation (labor scarcity, sale of assets, and morbidity) were equal between men and women, this would be an underestimate.

To examine the sectoral impact of the AIDS epidemic we look at the elasticity of value added in each of the agricultural, manufacturing, and services sector, with respect to the HIV prevalence rate. The dependent variable measures the net output of a sector after adding up all outputs and subtracting intermediate inputs. Rows 4-6 of Table 5 present the results.

In row 4 we observe a negative shock to the agricultural sector that is strikingly consistent across the various specifications. An increase in the HIV rate by 10 percentage points predicts a decrease in the growth of value added in agriculture between 1980 and 2003 of at least 10 percent. The estimate is slightly higher when changes in GDP per capita and Polity2 are factored in. Rows 5 and 6 reveal that the value added in

manufacturing and services are unchanged as a result of the AIDS epidemic. If anything, the OLS results in column 1 predict a small *increase* in the value added in these sectors, but that increase is wiped away once AIDS is instrumented for with circumcision. These results are in fact consistent with the possibility of substitution in high value-added jobs in manufacturing and services, where that substitution comes out of the agricultural sector. Such stylized facts are reminiscent of Lewis' (1954) model of labor surplus in agriculture, which may shine light on the absence of an impact on per capita economic growth even as the agricultural sector declines. Yet a reduction in the productivity in the agricultural sector and in female employment is sure to have human costs, particularly at the lower end of the income distribution.

#### *AIDS and humanitarian outcomes*

Though economists have (understandably) focused on the economic impact of AIDS, other social scientists have documented the humanitarian side of the epidemic.<sup>12</sup> Certainly, the two are interlinked. Absent unlimited, indefinite inflows of foreign aid, the ability of a nation to handle any disaster depends on its economic capabilities. In turn, temporary humanitarian crises can have long-run developmental impacts, especially if a young cohort is affected. Given the lack, thus far, of solid econometric evidence that there is a per capita economic disaster, it may make sense to turn at least some of our energies towards understanding the scope of the human costs associated with AIDS. And these costs appear to be many. A UNDP background paper prepared for the 2001 UN General Assembly Special Session on HIV/AIDS lists some of the humanitarian issues: “soaring adult and child mortality... poverty deepening... food security threatened... women severely affected... social cohesion disintegrating” (Loewenson and Whiteside, 2001: 7-11). In this section we investigate the impact of AIDS on undernutrition rates and food prices to understand poverty and food security, as well as on life expectancy to highlight the profound human costs of the epidemic.

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<sup>12</sup> See <http://www.odi.org.uk/hpg/aidsresources.html> as a reference point for non-economics literature on these topics. One humanitarian area where economists have delved is the impact of orphaning on child outcomes, with reference to HIV/AIDS. See Evans and Miguel (2004), Case, Paxson, and Ableidinger (2004), and Yamano and Jayne (2004).

A recent burgeoning literature highlights the complex relationship between AIDS prevalence and malnutrition in Africa. Since AIDS tends to attack young adults, it leaves behind a population disproportionately skewed toward the elderly and young children (FAO 2003). This diminishes the agricultural labor supply and affects food production as we observed in the previous section. The World Health Organization reports that while undernourishment rates fell for sub-Saharan Africa from 1980 to 2000, countries with high HIV prevalence (greater than 5 percent) actually experienced a dramatic *increase* in undernourishment over that time period (FAO, 2003: 11). Furthermore, the premature deaths due to AIDS may prohibit the normal intergenerational knowledge transfer through which agricultural skills are passed to the next generation (Haddad and Gillespie, 2001). AIDS has also been proposed to play a dangerous role during times of food emergencies to undermine traditional coping mechanisms and exacerbate nutritional crises (de Waal and Whiteside, 2003). Yet the causality may go in the opposite direction. Malnutrition increases the speed of progression to AIDS as well as the risk of HIV transmission from mother to child (Piwoz and Preble, 2000). It may also increase the transmission of the disease through behavioral mechanisms such as driving young women to enter the sex trade in an effort to escape destitution (de Waal and Whiteside, 2003) as observed in Zimbabwe by Save the Children researchers (O'Donnell, Khozombah, and Mudenda, 2002). Thus instrumental-variable regressions are crucial to pin down the impact of AIDS on food security.

To test the effect of AIDS on undernourishment, we look at the effect of HIV rates on changes in malnutrition rates in Africa from 1980 to 2000 in row 1 of Table 6. The World Food Program (WFP) calculates these data by comparing the total number of calories available in a region from local food production and trade with the minimum caloric requirements for the population. Since caloric requirements are a function of population demographics, specifically age and gender characteristics, the WFP calculates the minimum per capita caloric requirements uniquely across countries. Combining demographic characteristics of the population with estimates of the food distribution across income levels for each nation, the WFP estimates the percentage of the population whose food intake falls below the minimum threshold in each country (FAO, 2000).

Column 1 presents the reduced-form regression of change in malnutrition from 1980-2000 on the AIDS rate in 2001. The results are disquieting. An increase in the adult prevalence of HIV/AIDS by one percentage point predicts a rise in the malnutrition rate by 0.44 percentage points, significant at the  $p=0.1$  level. Measurement error in the independent variable suggests that this is an underestimation, while the most obvious omitted variables such as “responsibility of the government” would point to an overestimation: after all, irresponsible governments should both allow the AIDS epidemic to occur as well as undernourishment to occur. Furthermore, the possible reverse causality from malnutrition to AIDS also suggests an overestimation of the coefficient. To address these concerns we employ the instruments for HIV/AIDS. In column 2 we instrument for AIDS using the linear male circumcision rate. The coefficient rises to 0.74 though the statistical significance drops. In column 3 we use the H/B dummies for circumcision as the instruments for AIDS and the coefficient rises to 0.82 and regains its significance. It appears, then, that the coefficient in column 1 was underestimated due to measurement error and/or some omitted variable whose correlation with AIDS and malnutrition have opposite signs.

In columns 4 and 5 we reintroduce controls for the change in GDP per capita and Polity2. Once we have controlled for changes in socioeconomic conditions that may be unrelated to AIDS, a one percentage point increase in the adult HIV prevalence predicts an increase in the fraction of the population that is undernourished by 1.14-1.26 percentage points. Since undernourishment is not calculated using anthropometric methods (which could be inadvertently picking up AIDS-associated wasting), this suggests that AIDS may have a multiplier effect where it causes a humanitarian crisis not only in the afflicted population, but beyond.

Ideally, we would like to separate AIDS-induced rises in undernutrition into its components of agricultural output and poverty or inequality in order to understand which component of undernutrition is most vulnerable to the disease. Unfortunately, data on poverty and inequality do not exist in any cross-comparable form for more than a few African countries both in the vicinity of 1980 and the vicinity of 2000. We did, however, find that agricultural output suffers from AIDS in the previous section. We also have data on food prices for a number of countries through 2002, using an index that

normalizes 1995 to 100. Row 2 reports the impact of changes in the HIV prevalence rate on changes in the food price index. All the specifications are positive, and the instrumented results with linear circumcision (not controlling for GDP and democracy) and with both instrumental specifications (controlling for GDP and democracy) are statistically significant. It appears that an increase in the HIV prevalence rate of one percentage point increases the price of food by over 10 points in the normalized food price index. This number understates the dramatic difference in food price changes that have characterized high HIV countries. Of the 23 countries for which 1980 and 2002 food price indexes are available, 8 have an HIV prevalence rate in 2003 of at least 10 percent. Those same countries started with an average food price index of 20 and experienced an average rise in the index of 460 points. In contrast, the other 15 countries with low AIDS rate started with an average index of 31 and rose only 110 points on average. These results are consistent with AIDS leading to a reduction in domestic food production and an increase in the price of food, resulting in a reduction of people's entitlements and putting them at risk of not being able to purchase sufficient caloric input (i.e. Sen, 1983). This can be exacerbated if AIDS victims and caregivers drop out of the labor force.

Row 3 of Table 6 regresses the change in life expectancy on the change in the AIDS rate. Obviously, AIDS is driving a massive and tragic reduction in life expectancy, and the coefficients reflect that. In column 1, the basic OLS elasticity is -0.68. In other words, one extra percentage point of HIV prevalence is associated with a reduction of the average life expectancy of nearly 0.7 years. Columns 2 and 3 present the instrumented result, and the coefficient rises, probably due to a correction of the downward bias in the OLS resulting from measurement error in the HIV rates. Adding controls for GDP per capita and Polity2 do not appreciably change the estimates; one percentage point extra in HIV prevalence predicts a drop in life expectancy of around 0.9 years.

A simple mathematical example reveals how dramatic this estimate is. If we have a country with a life expectancy of 60 years, and assume that ten percent of the population will contract AIDS and die at age 25, then the average life expectancy of that population will fall to 56.5 years. In other words, if ten percent of the people will—in their lifetime—contract AIDS, the life expectancy only falls 3.5 years according to the

example. Yet we observe a drop of 9 years. This implies that the HIV prevalence rate is significantly lower than the proportion of people who, at some point in their lifetime, will contract the virus.

Finally, we examine the global humanitarian response. Given the tremendous human costs associated with AIDS, we should hope to see a response by the international community to help the distressed populations of Africa and to fund preventative programs. We regressed the change in foreign aid receipts as a percent of GDP on the HIV/AIDS prevalence rate in row 4 of Table 6. In both the OLS and the IV specifications, there was no impact of the AIDS rate on increases in foreign aid. In Africa by 2003, foreign aid does not appear to have been allocated in response to this massive epidemic. This suggests that despite the propaganda and promises of western leaders, the AIDS epidemic has remained a problem for Africa to address on its own.

*Robustness check: AIDS on earlier outcomes*

Even with the differenced regression, we are not able to control for time-variant country effects. For example, the advantageousness of unobservable characteristics that are correlated with male circumcision may have changed over time. However, it is unlikely that such effects change with high frequency. To check that our undernutrition and growth results are not being driven by time-variant country effects, we examine the statistical effect of the AIDS epidemic on humanitarian and economic outcomes over the decade 1970-80. If the post-1980 results were simply revealing an underlying trend unrelated to HIV/AIDS, we should expect that the 1970-80 results are identical to the later time period, even though AIDS could not have caused the outcomes. But if the 1970-80 results do not reveal the same movement, then AIDS remains a likely cause of the observed change through 2003.

In Appendix B.3, columns 1-4, we run AIDS on the change in undernutrition from 1970-80. Unfortunately, the % undernourished variable was not calculated prior to 1980, however the Food and Agriculture Organization did have an alternative variable: the per capita dietary energy supply, measured in kilocalories per day, that are available for both 1970 and 1980 (FAO, 1987). In columns 1 and 2 we run the instrumented specifications of HIV/AIDS in 2003 on the change in kilocalories per day over 1970-80. We use the

instrumental variable versions because we are chiefly worried that an omitted variable correlated with the instrument is proxying for the time-variant country effects. The coefficients are small and insignificantly different from zero. Concerned that the specification of the dependent variable could be responsible for the lack of impact, we rerun the regressions, this time using the percentage change in kilocalories per day over 1970-80. Again, the coefficients on AIDS are small and insignificantly different from zero. This is good news for our instrument, as it predicts changes in undernutrition that correspond to the period during which the AIDS epidemic took its terrible toll (Table 6) but does not predict any changes over the previous period, where the spread of AIDS was minimal. Of the control variables, only the change in GDP per capita over 1970-80 has any significant effect. As previously observed, countries that grew more also increased the caloric input of their citizens.

We also rerun the growth regressions of Table 4, this time on the period of 1970-80. Since there were no significant effects measured with our instrumented AIDS prevalence rates over the period of the epidemic, it would be worrying to see a significant effect of the 2003 prevalence rates on a period that was not characterized by the disease. Indeed, as columns 5 and 6 in Appendix B.3 show, we continue to find no significant impact of the instrumented AIDS rates on economic growth. The coefficients are small and close to zero. In both specifications, the change in the level of democracy has no measurable impact.

#### **IV. Conclusion**

The empirical results presented in this paper are consistent with early studies that showed the AIDS epidemic having little impact on economic growth in Africa. Although future data may align with the various predictions of economic disaster that some theoretical papers have put forward, those fears do not seem to have taken root as of 2003. Hopefully, this paper has demonstrated that economists have overlooked the structural and humanitarian side of the disaster; as it turns out, data in this realm reveal statistically significant and practically important trends. In particular, the impact of

AIDS on the agricultural sector, on women, and on malnourishment deserves closer scrutiny by economists. Additionally, we put forth the notion of using male circumcision rates as an instrument for HIV prevalence in econometric models. Although the medical community is still debating the causal mechanisms, the ability of national male circumcision rates to predict AIDS levels in African nations is strong and robust. Therefore it is not implausible to suggest that male circumcision rate can be used as an instrumental variable for economists exploring the impact of AIDS in Africa when concerns of causality and measurement error arise.

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**Table 1: Summary Statistics**

Variable	Obs	Mean	Std. Dev.
% of adults 15-49 living with HIV/AIDS in 2003	44	7.90	9.56
Male circumcision rate	49	0.65	0.33
H/B circumcision 20%-80%	48	0.19	0.39
H/B circumcision 80%-100%	48	0.63	0.49
GDP per capita, \$1995, 2003	50	1082.55	1523.28
Population per sq km, 2003	54	81.58	114.13
Urban population (% of total), 2003	53	40.07	17.18
Polity2 score, 2002	50	0.64	5.31
Ethnolinguistic fractionalization, 1985	51	0.62	0.27
Fraction Muslim	54	0.39	0.40
Fraction population from Bantu-speaking tribe	49	0.32	0.38
GDP per capita, \$1995, 1980	41	951.44	1235.87
Gross domestic savings (% of GDP), 1980	42	10.14	20.08
Population per sq km, 1980	51	45.85	75.83
Urban population (% of total), 1980	52	27.10	15.05
Polity2 score, 1980	48	-4.96	5.33
Literacy rate, adult total (% age 15+), 1980	46	39.86	17.83
Life expectancy at birth, 1980	51	48.95	6.66
Radios (per 1,000 people), 1997	53	183.86	110.82
Vehicles (per 1,000 people), 1996	51	29.47	42.46
% self-reported high risk sex in year, adult males, 1990-2000	32	32.01	21.28
% self-reported high risk sex in year, adult females, 1990-2000	31	12.77	12.48
% condom use at last high risk sex, male, 1996-2001	27	35.89	23.07
% condom use at last high risk sex, female, 1996-2001	27	18.71	16.67
Change in ln(GDP per capita, \$1995), 1980-2003	40	0.01	0.48
Change in Polity2 score, 1980-2002	48	5.65	6.46
Change in gross domestic savings (% of GDP), 1980-2003	37	0.91	12.94
Change in primary school enrollment, (% gross), 1980-2001	38	12.37	25.30
Change in secondary school enrollment, (% gross), 1980-2001	23	46.43	28.09
Change in tertiary school enrollment, (% gross), 1980-2001	18	9.90	12.37
Change in total fertility rate, 1980-2002	51	-1.60	1.02
Change in urban population (% of total), 1980-2003	52	13.13	8.29
Change in economic activity rate, %, women age 15+, 1980-2000	50	-0.16	4.03
Change in economic activity rate, %, men age 15+, 1980-2000	50	-2.61	1.59
Change in ln(agriculture, value added, \$1995), 1980-2003	38	0.51	0.29
Change in ln(manufacturing, value added, \$1995), 1980-2003	27	0.78	0.56
Change in ln(services, value added, \$1995), 1980-2003	38	0.57	0.57
Change in % undernourished, 1980-2000	42	-3.40	18.81
Change in food price index (1995=100), 1980-2002	23	231.33	395.95
Change in life expectancy at birth, 1980-2002	51	0.60	8.15
Change in foreign aid (% GDP), 1980-2002	40	2.85	14.58
Change in daily kcal per capita dietary energy supplies, 1970-80	42	132.62	343.46
% change in per capita dietary energy supplies, 1970-80	42	6.12	15.63
Change in ln(GDP per capita, \$1995), 1970-80	37	0.14	0.29
Change in Polity2 score, 1970-80	43	-0.05	4.25

**Table 2: Predicting AIDS Rates with Male Circumcision**

	(1)	(2)	(3)	(4)	(5)	(6)
	OLS	OLS	OLS	OLS	OLS	OLS
	AIDS03	AIDS03	AIDS03	AIDS03	AIDS03	AIDS03
Male circumcision rate	-18.813 [3.950]***	-13.903 [2.612]***	-10.211 [4.283]**		-8.125 [4.603]*	
H/B circumcision 20%-80%				-11.111 [4.124]**		-8.620 [3.717]**
H/B circumcision 80%-100%				-15.822 [3.851]***		-13.586 [3.350]***
ln(GDP per capita, \$1995), 2003		5.195 [2.104]**	4.344 [1.754]**	3.534 [1.295]**	5.042 [1.884]**	4.211 [1.359]***
ln(Population per sq km), 2003		-1.215 [0.769]	-1.429 [0.722]*	-1.053 [0.673]	-1.069 [0.850]	-0.822 [0.771]
Urban population (% of total), 2003		-0.173 [0.111]	-0.138 [0.103]	-0.039 [0.062]	-0.125 [0.086]	-0.037 [0.058]
Polity2 score, 2002		0.265 [0.260]	0.255 [0.266]	0.185 [0.255]	0.244 [0.245]	0.184 [0.241]
Ethnolinguistic fractionalization, 1985			-5.540 [4.870]	1.742 [4.806]	-11.957 [7.460]	-4.564 [6.915]
Fraction Muslim			-8.644 [3.930]**	-6.790 [2.261]***	-7.317 [3.250]**	-5.784 [2.041]***
Fraction population from Bantu-speaking tribe			-2.943 [3.014]	-4.557 [3.030]	-3.556 [2.879]	-4.694 [2.851]
Constant	19.545 [3.383]***	-5.259 [9.635]	5.085 [8.162]	5.437 [7.213]	2.646 [10.029]	3.068 [7.670]
Observations	44	42	42	41	38	37
R-squared	0.43	0.61	0.68	0.77	0.7	0.78

NOTE: Dependent variable AIDS03 is % of adults 15-49 living with HIV/AIDS in 2003. Regressions (5) and (6) exclude North Africa. Robust standard errors in brackets. \* significant at 10%; \*\* significant at 5%; \*\*\* significant at 1%.

**Table 3: Correlatives with Male Circumcision**

	Correlation with male circumcision rate	p-value
ln(GDP per capita, \$1995), 1980	0.001	0.998
Gross domestic savings (% of GDP), 1980	0.245	0.133
ln(Population density (per sq km)), 1980	-0.240	0.104
Urban population (% of total), 1980	0.390	0.006
Polity2 score, 1980	-0.238	0.108
Literacy rate, adult total (% age 15+), 1980	-0.441	0.003
Life expectancy at birth, 1980	-0.142	0.330
Radios (per 1,000 people), 1997	0.200	0.168
Vehicles (per 1,000 people), 1996	0.019	0.898
% self-reported high risk sex in year, adult males, 1990-2000	-0.133	0.475
% self-reported high risk sex in year, adult females, 1990-2000	-0.094	0.616
% condom use at last high risk sex, male, 1996-2001	-0.280	0.166
% condom use at last high risk sex, female, 1996-2001	0.001	0.997

**Table 4: The Impact of AIDS on Economic Outcomes**

<i>Dependent Variable</i>	(1) OLS	(2) IV-2SLS	(3) IV-2SLS	(4) IV-2SLS	(5) IV-2SLS
Change in ln(GDP per capita, \$1995), 1980-2003	0.012 [0.007] 35	0.010 [0.010] 35	0.010 [0.009] 35	0.014 [0.011] 34	0.015 [0.009] 34
Change in Polity2 score, 1980-2002	-0.008 [0.121] 42	-0.027 [0.205] 42	0.015 [0.173] 41	0.227 [0.169] 34	0.214 [0.158] 34
Change in gross domestic savings (% of GDP), 1980-2003	0.116 [0.245] 31	0.185 [0.401] 31	0.089 [0.348] 31	-0.086 [0.300] 30	-0.177 [0.267] 30
Change in primary school enrollment (% gross), 1980-2001	-0.229 [0.260] 31	0.892 [0.809] 30	0.689 [0.626] 29	0.421 [0.763] 26	0.674 [0.789] 26
Change in secondary school enrollment, (% gross), 1980-2001	0.667 [0.541] 20	1.416 [1.290] 20	0.320 [0.972] 20	1.146 [1.201] 17	0.365 [1.245] 17
Change in tertiary school enrollment (% gross), 1980-2001	-0.164 [0.281] 17	0.814 [0.567] 16	-0.138 [0.285] 16	1.108 [0.484]** 14	-0.026 [0.494] 14
Change in total fertility rate, 1980-2002	-0.001 [0.012] 43	0.008 [0.022] 43	-0.004 [0.017] 42	0.013 [0.026] 34	0.008 [0.021] 34

NOTE: Coefficient is the impact of the % of adults 15-49 living with HIV/AIDS in 2003 (or 2001) on the dependent variable.

HIV/AIDS is instrumented in regressions (2) and (4) with the male circumcision rate, and in (3) and (5) with the H/B dummies for circumcision.

Regressions (4) and (5) control for the change in ln(GDP per capita, \$1995) and the change in Polity2.

When the most recent data were not available, the previous year's value was used.

Robust standard errors in brackets. \* significant at 10%; \*\* significant at 5%; \*\*\* significant at 1%. Number of observations below.

**Table 5: The Impact of AIDS on the Structure of the Economy**

<i>Dependent Variable</i>	(1) OLS	(2) IV-2SLS	(3) IV-2SLS	(4) IV-2SLS	(5) IV-2SLS
Change in urban population (% of total), 1980-2003	-0.011 [0.187] 43	-0.139 [0.243] 43	-0.25 [0.217] 42	-0.19 [0.304] 33	-0.385 [0.269] 33
Change in economic activity rate, % women age 15+, 1980-2000	-0.105 [0.061]* 42	-0.15 [0.073]** 41	-0.101 [0.058]* 40	-0.183 [0.086]** 32	-0.148 [0.067]** 32
Change in economic activity rate, % men age 15+, 1980-2000	-0.004 [0.023] 42	0.039 [0.039] 41	0.019 [0.034] 40	0.039 [0.035] 32	0.028 [0.032] 32
Change in ln(agriculture, value added, \$1995), 1980-2003	-0.011 [0.002]*** 33	-0.011 [0.004]** 33	-0.01 [0.004]** 33	-0.014 [0.006]** 31	-0.014 [0.005]** 31
Change in ln(manufacturing, value added, \$1995), 1980-2003	0.019 [0.010]* 24	-0.004 [0.016] 24	0.002 [0.015] 24	-0.004 [0.016] 22	0 [0.015] 22
Change in ln(services, value added, \$1995), 1980-2003	0.013 [0.010] 33	0.01 [0.011] 33	0.005 [0.010] 33	0.006 [0.007] 31	-0.003 [0.006] 31

NOTE: Coefficient is the impact of the % of adults 15-49 living with HIV/AIDS in 2003 (or 2001) on the dependent variable.

HIV/AIDS is instrumented in regressions (2) and (4) with the male circumcision rate, and in (3) and (5) with the H/B dummies for circumcision.

Regressions (4) and (5) control for the change in ln(GDP per capita, \$1995) and the change in Polity2.

When the most recent data were not available, the previous year's value was used.

Robust standard errors in brackets. \* significant at 10%; \*\* significant at 5%; \*\*\* significant at 1%. Number of observations below.

**Table 6: The Impact of AIDS on Humanitarian Outcomes**

<i>Dependent Variable</i>	(1) OLS	(2) IV-2SLS	(3) IV-2SLS	(4) IV-2SLS	(5) IV-2SLS
Change in % undernourished, 1980-2000	0.442 [0.237]* 36	0.744 [0.478] 35	0.817 [0.432]* 34	1.142 [0.526]** 29	1.258 [0.414]*** 29
Change in food price index (1995=100), 1980-2002	15.926 [12.865] 21	29.225 [16.139]* 21	24.476 [15.688] 20	13.651 [6.091]** 18	10.452 [5.861]* 18
Change in life expectancy at birth, 1980-2002	-0.684 [0.120]*** 44	-1.026 [0.142]*** 44	-0.857 [0.149]*** 43	-0.989 [0.151]*** 34	-0.827 [0.184]*** 34
Change in foreign aid (% GDP), 1980-2002	-0.18 [0.146] 34	0.537 [0.548] 34	-0.028 [0.205] 34	0.547 [0.468] 33	-0.059 [0.223] 33

NOTE: Coefficient is the impact of the % of adults 15-49 living with HIV/AIDS in 2003 (or 2001) on the dependent variable.

HIV/AIDS is instrumented in regressions (2) and (4) with the male circumcision rate, and in (3) and (5) with the H/B dummies for circumcision.

Regressions (4) and (5) control for the change in ln(GDP per capita, \$1995) and the change in Polity2.

When the most recent data were not available, the previous year's value was used.

Robust standard errors in brackets. \* significant at 10%; \*\* significant at 5%; \*\*\* significant at 1%. Number of observations below.

Figure 1: AIDS and Male Circumcision

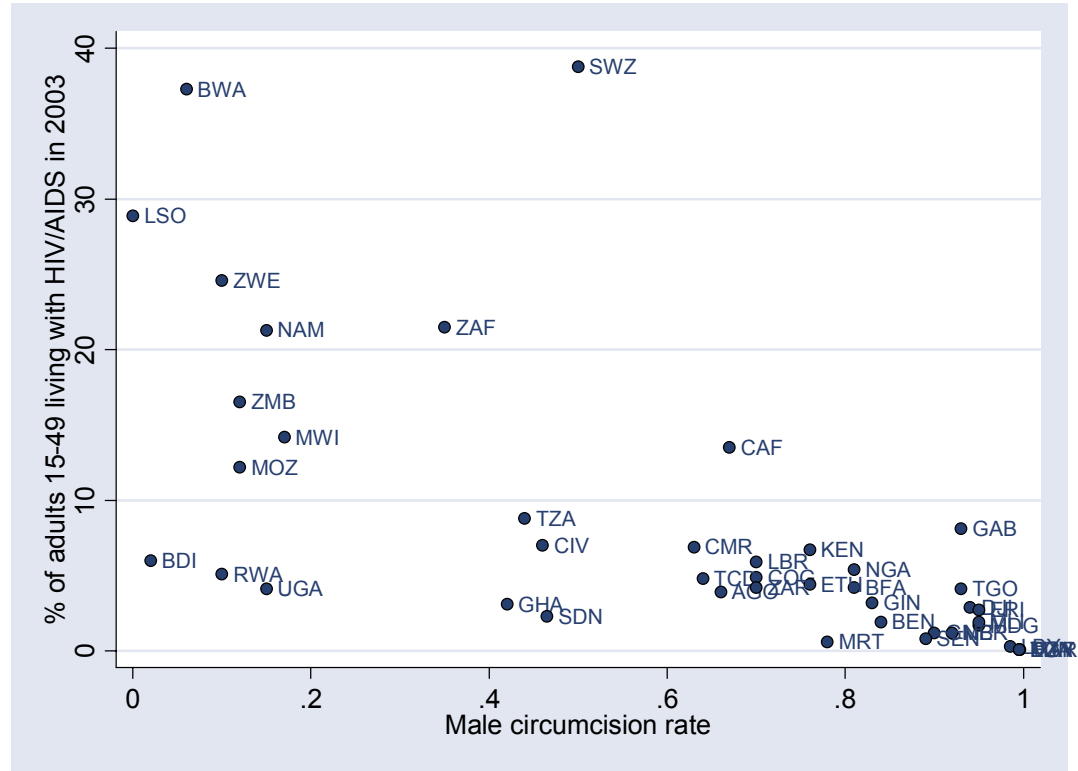
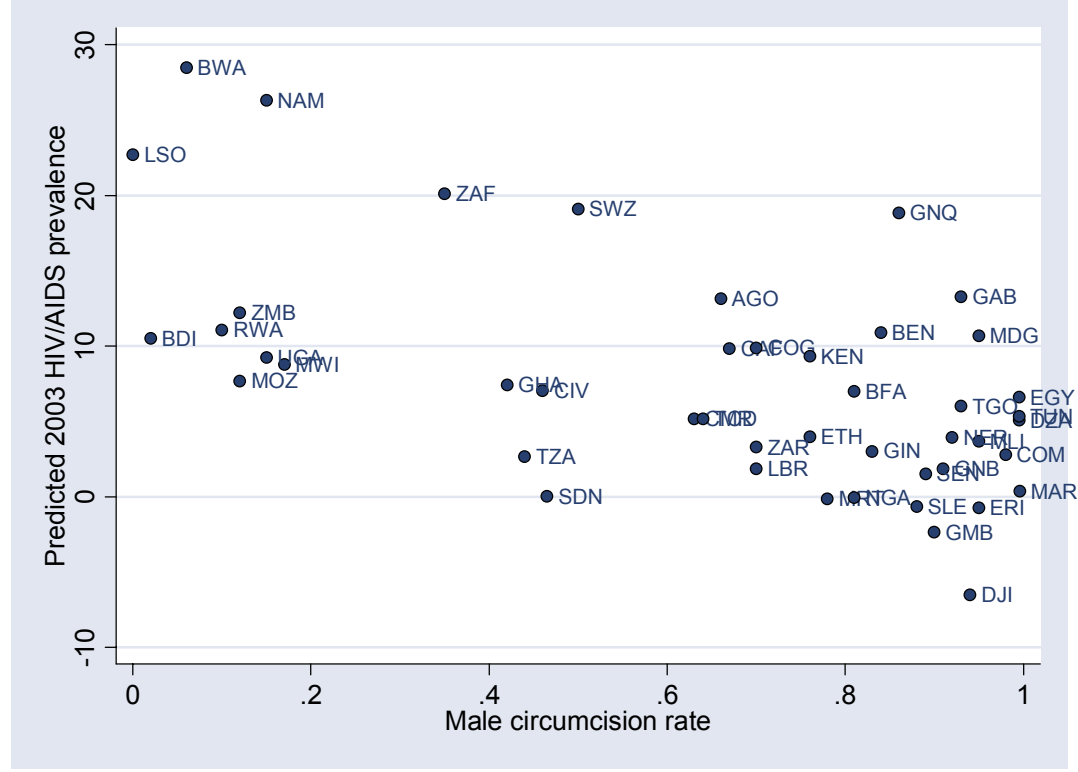


Figure 2: AIDS and Male Circumcision, controlling for economic, political, linguistic, and religious factors



## **Appendix A. Circumcision and HIV Transmission.**

### *Does a lack of circumcision lead to higher AIDS rates?*

Extreme differences in AIDS prevalence in countries with seemingly similar risk factors prompted the World Health Organization to admit that it was “not fully understood why HIV infection rates take off in some countries while remaining stable in neighboring countries over many years” (UNAIDS/WHO 1998). Trying to account for the drastically different AIDS infection rates in sub-Saharan Africa, Cameron et al (1989) found that uncircumcised men faced an eight fold risk of AIDS infection over their circumcised counterparts. Five years later, Moses and colleagues (1994) published a paper summarizing the growing quantity of evidence suggesting a link between uncircumcised men and HIV prevalence. John and Pat Caldwell’s 1996 *Scientific America* article suggested that male circumcision rates were among the strongest predictors of AIDS prevalence in central and southern Africa. In their conclusion, Caldwell and Caldwell stated that “the link between lack of [male] circumcision and elevated levels of HIV infection rates appears robust...in some parts of the AIDS belt, nearly all men are uncircumcised, a situation unlike almost anywhere else in Africa” (1996: 68).

Most quantitative evidence is as stark as the case-based and anecdotal reports. Halperin and Bailey (1999) estimated male circumcision rates into high, medium, and low for many sub-Saharan African countries and showed a high correlation between low circumcision and high HIV prevalence. They also argued that the relationship seems to hold for other regions of the world such as Southeast Asia. Weiss, Quigley, and Hayes (2000) conduct a random-effects meta-analysis of 21 independent studies to calculate a pooled relative risk coefficient and confidence interval for determining if male circumcision was a significant determinant of HIV infection. They found a highly significant relative risk coefficient of 0.52, implying that circumcised men were half as likely to be HIV positive as their uncircumcised counterparts.

Many of these articles emphasize the biological mechanism through which uncircumcised men may be more susceptible to HIV infection. A leading theory is that uncircumcised males in less sanitary environments are more likely to get chancroid

infections that cause genital sores. Since these chancroid sores catalyze the transmission of the HIV virus through increased blood exposure, uncircumcised men would have a higher likelihood of transmitting and receiving the AIDS virus (Caldwell and Caldwell, 1996). Steen (2001) notes that chancroids are particularly important in catalyzing HIV transmissions in countries with high AIDS prevalence and that male circumcision reduces chancroid infections. A more direct theory states that the vascular prepuce (foreskin removed in male circumcision) contains a high concentration of Langerhans cells which are known target cells for HIV transmission (Hussain and Lehner, 1995). Other sexually transmitted infections that can increase the risk for HIV infection are also more prevalent among uncircumcised males, strengthening the biological argument for the relationship between male circumcision and AIDS (Moses, Bailey, and Ronald, 1998).

Though extensive medical literature supports the direct link between lack of male circumcision and increased HIV infection rates, these studies are subject to criticism.<sup>13</sup> Since existing evidence takes the form of natural experiments rather than randomized controlled trials, cultural effects correlated with circumcision cannot be differentiated from the impact of male circumcision (Siegfried et al, 2003). One specific critique of the existing literature explores the degree to which religion is confounded with male circumcision. In many studies, the non-circumcised participants are non-Muslim while the circumcised males tend to be disproportionately Muslim (Oster, 2004). Oster notes that in a few of these studies, the significant result of circumcision preventing HIV spread disappears when only non-Muslims are included in the data. She performs simulations that can generate an observed “circumcision effect” when the circumcised and uncircumcised populations have the same transmission rates but differ in other respects that may not be observable to the researcher.

Aside from the omitted-variables critique, there are studies that have not found any significant differences in HIV infection by status of circumcision. Grulich et al (2001) look at HIV transmission among homosexual men in Australia and do not find that circumcision plays an important role. Van Howe (1999) performs a meta-analysis of peer-reviewed papers on the relationship between circumcision and HIV transmission and

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<sup>13</sup> Indeed, there is a non-academic website dedicated to this body of criticism:  
<http://www.circumstitutions.com/HIV.html>

argues that the body of papers in fact predicts higher transmission from circumcision. Laumann, Massey, and Zuckerman (1999) do not find any differences in sexually-transmitted disease rate between circumcised and uncircumcised men in the United States, though they do find differences in sexual practice. Finally, and also in the developed world, it is well noted that the United States has much higher circumcision than Europe, yet also a higher AIDS rate.

### Appendix B.1: Male Circumcision Rate Estimates

Country	Male Circumcision Rate	Halperin/Bailey
Algeria	1.00	>80%
Angola	0.66	>80%
Benin	0.84	>80%
Botswana	0.06	<20%
Burkina Faso	0.81	20%-80%
Burundi	0.02	<20%
Cameroon	0.63	>80%
Central African Rep.	0.67	20%-80%
Chad	0.64	>80%
Comoros	0.98	>80%
Congo	0.70	>80%
Cote d'Ivoire	0.46	20%-80%
Djibouti	0.94	>80%
Egypt	1.00	>80%
Equat. Guinea	0.86	>80%
Eritrea	0.95	>80%
Ethiopia	0.76	-
Gabon	0.93	>80%
Gambia	0.90	>80%
Ghana	0.42	>80%
Guinea	0.83	>80%
Guinea-Bissau	0.91	>80%
Kenya	0.76	>80%
Lesotho	0.00	<20%
Liberia	0.70	>80%
Libya	0.99	>80%
Madagascar	0.95	>80%
Malawi	0.17	<20%
Mali	0.95	>80%
Mauritania	0.78	>80%
Morocco	1.00	>80%
Mozambique	0.12	20%-80%
Namibia	0.15	<20%
Niger	0.92	>80%
Nigeria	0.81	>80%
Rwanda	0.10	<20%
Senegal	0.89	>80%
Sierra Leone	0.88	>80%
Somalia	0.93	>80%
South Africa	0.35	20%-80%
Sudan	0.47	20%-80%
Swaziland	0.50	<20%
Tanzania	0.44	20%-80%
Togo	0.93	>80%
Tunisia	1.00	>80%
Uganda	0.15	20%-80%
Zaire	0.70	20%-80%
Zambia	0.12	<20%
Zimbabwe	0.10	<20%

## Appendix B.2: Data Sources

Variable	Source
% of adults 15-49 living with HIV/AIDS in 2003	UNAIDS/WHO (2004)
Male circumcision rate	Murdock (1967)*
H/B circumcision 20%-80%	Halperin and Bailey (1999)
H/B circumcision 80%-100%	Halperin and Bailey (1999)
GDP per capita, \$1995, 2003	WDI
Population per sq km, 2003	WDI
Urban population (% of total), 2003	WDI
Polity2 score, 2002	Marshall and Jagers (2002)
Ethnolinguistic fractionalization, 1985	Roeder (2001)
Fraction Muslim	Alesina et al (2003)
Fraction population from Bantu-speaking tribe	Murdock (1967)*
GDP per capita, \$1995, 1980	WDI
Gross domestic savings (% of GDP), 1980	WDI
Population per sq km, 1980	WDI
Urban population (% of total), 1980	WDI
Polity2 score, 1980	Marshall and Jagers (2002)
Literacy rate, adult total (% age 15+), 1980	WDI
Life expectancy at birth, 1980	WDI
Radios (per 1,000 people), 1997	WDI
Vehicles (per 1,000 people), 1996	WDI
% self-reported high risk sex in year, adult males, 1990-2000	UNAIDS (2002)
% self-reported high risk sex in year, adult females, 1990-2000	UNAIDS (2002)
% condom use at last high risk sex, male, 1996-2001	UNAIDS (2002)
% condom use at last high risk sex, female, 1996-2001	UNAIDS (2002)
Change in ln(GDP per capita, \$1995), 1980-2003	WDI
Change in Polity2 score, 1980-2002	WDI
Change in gross domestic savings (% of GDP), 1980-2003	WDI
Change in primary school enrollment, (% gross), 1980-2001	WDI
Change in secondary school enrollment, (% gross), 1980-2001	WDI
Change in tertiary school enrollment, (% gross), 1980-2001	WDI
Change in total fertility rate, 1980-2002	WDI
Change in urban population (% of total), 1980-2003	WDI
Change in economic activity rate, %, women age 15+, 1980-2000	WISTAT (1999)
Change in economic activity rate, %, men age 15+, 1980-2000	WISTAT (1999)
Change in ln(agriculture, value added, \$1995), 1980-2003	WDI
Change in ln(manufacturing, value added, \$1995), 1980-2003	WDI
Change in ln(services, value added, \$1995), 1980-2003	WDI
Change in % undernourished, 1980-2000	FAO (2000), UN Com. D.base
Change in food price index (1995=100), 1980-2002	WDI
Change in life expectancy at birth, 1980-2002	WDI
Change in foreign aid (% GDP), 1980-2002	WDI
Change in daily kcal per capita dietary energy supplies, 1970-80	FAO (1987)
% change in per capita dietary energy supplies, 1970-80	FAO (1987)
Change in ln(GDP per capita, \$1995), 1970-80	WDI
Change in Polity2 score, 1970-80	Marshall and Jagers (2002)

\*compiled by authors. WDI either 2003 or 2004 edition for latest variables.

### Appendix B.3: Robustness Check: AIDS on Earlier Outcomes

	(1)	(2)	(3)	(4)	(5)	(6)
	IV-2SLS	IV-2SLS	IV-2SLS	IV-2SLS	IV-2SLS	IV-2SLS
	$\Delta$ KCAL	$\Delta$ KCAL	$\Delta$ KCAL%	$\Delta$ KCAL%	$\Delta$ LN(y)	$\Delta$ LN(y)
% of adults 15-49 living with HIV/AIDS in 2003	-8.951 [7.669]	-3.115 [5.773]	-0.397 [0.376]	-0.134 [0.277]	0.007 [0.009]	0.010 [0.007]
Change in ln(GDP per capita, \$1995), 1970-80	524.487 [219.410]**	419.276 [194.113]**	23.667 [10.324]**	18.927 [8.983]**		
Change in Polity2 score, 1970-80	-10.755 [11.371]	-6.494 [7.741]	-0.490 [0.515]	-0.298 [0.348]	0.001 [0.013]	0.002 [0.012]
Constant	99.825 [67.747]	71.377 [65.377]	4.660 [3.344]	3.379 [3.154]	0.089 [0.074]	0.062 [0.068]
Observations	31	31	31	31	34	34
R-squared	0.18	0.19	0.17	0.18	0.15	0.18

NOTE: dependent variable definitions.  $\Delta$ KCAL is Change in daily kcal per capita dietary energy supplies, 1970-80.

$\Delta$ KCAL% is % change in per capita dietary energy supplies, 1970-80.  $\Delta$ LN(y) is Change in ln(GDP per capita, \$1995), 1970-80.

HIV/AIDS is instrumented in regressions (1), (3), and (5) with the male circumcision rate, and in (2), (4), and (6) with the H/B dummies for circumcision.

Robust standard errors in brackets. \* significant at 10%; \*\* significant at 5%; \*\*\* significant at 1%.