

Does Better Health Care Lead to Higher AIDS Rates? Evidence from Sub-Saharan Africa.

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Abstract:

HIV/AIDS is a disease for which we ourselves are the vector. Consequently, a high prevalence of the disease in the population is likely to generate a high incidence of new infections. This paper argues that in Sub-Saharan Africa, where the prevalence of other fatal diseases is high, there is a counter-intuitive effect of health care spending: such spending increases the life expectancy of the infected, and so drives up the prevalence of HIV in the population. The link between prevalence and incidence implies that high-quality health care may thus increase the speed of spread of the disease and drive up the peak prevalence that will be observed in the course of an epidemic. This depressing conclusion is derived from a simple theoretical model, and is confirmed using both cross-country evidence and individual-level data from Kenya. The paper concludes by discussing the policy implications for programs which seek to expand the distribution of anti-retrovirals.

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1. INTRODUCTION

The HIV/AIDS epidemic in Africa has been unique in several respects. The disease has moved through a population (women) and a transmission mechanism (heterosexual sex) that differ from most other regions of the world. Moreover, the peak prevalence rates seen there have been an order of magnitude higher than those observed elsewhere. Africa is also uniquely burdened by other endemic diseases; malaria, yellow fever, tuberculosis, and many others scourges are widespread. Since it is virtually always secondary infections that ultimately prove fatal to those infected with HIV, the role of these other diseases in shaping the life expectancy of the infected is central.¹ As we look across Sub-Saharan Africa, we see wide variation in the prevalence of these other diseases, and the incidence of these secondary diseases is highly correlated with low life expectancies and high mortality rates. HIV/AIDS, on the other hand, displays the reverse correlation: where mortality is highest and health care systems are worst, the AIDS epidemic has been substantially more muted in its progress. Indeed, the list of the highest prevalence countries in Africa is interchangeable with the list of countries that, as of 1990, did the most to promote the public health of their citizens: Botswana, Lesotho, Swaziland, Zimbabwe, Zambia, and South Africa.

Why should AIDS display a relationship with indicators of public health that is so different from other diseases? This paper suggests that the answer lies in a few simple facts about the disease. First, the efficacy of health care spending on the retardation of AIDS transmission may be limited.² Several recent randomized studies have shown that efforts to increase condom use (Stanton et al 1998), combat HIV through treating STDs (Wawer et al. 1999), or to change behavior through testing (Thornton 2006) have been only moderately

¹ See Corbett et al (2002) for a discussion of the ways in which malaria, TB, STDs, pneumonia, salmonella, and other diseases have interacted with HIV in Africa to increase mortality and morbidity.

² Gauri & Lieberman (2006) argue that 'boundary institutions', the procedures through which specific subgroups are monitored for the disease, have contributed to a particularly ineffective response to the epidemic in South Africa.

successful, if at all. Only in mother-to-child transmission have interventions been unarguably efficacious. Hence we do not expect to see a better health care system delivering a dramatically lower rate of adult-to-adult transmission. Secondly, a simple epidemiological model shows that the rate of spread of a sexually transmitted disease is a function of the prevalence of the disease in the sexually active population. Because secondary diseases in Africa are likely to play a major role in increasing both morbidity and mortality from AIDS, they will have the effect of pushing the infected out of the sexually active population sooner than would have been the case in a disease-free environment.³ It is demonstrated below that the strongest correlate of peak female HIV prevalence, and the correlate most robust to the inclusion of other explanatory variables, is female life expectancy as of 1982. Figure 1 plots the average prevalence for the longest- and shortest-lived quartiles in 1982, and we see an enormous difference between the subsequent trajectories of the disease. Our statistical results suggest that good public health as of the beginning of the epidemic is a primary determinant of a severe subsequent AIDS epidemic.

This finding runs contrary to predictions from several strains of the economics literature on AIDS. Kremer (1996) argues that fatalism among sexually active individuals may cause them to reduce their activity by less than others, leading to an increase in the percentage of risky partners in the pool. He states that ‘early public health efforts may allow societies to reach more favorable steady states’. Oster (2005) focuses on the role that sexually transmitted diseases play in the spread of HIV, and using simulations based on a model of

³ Colebunders et al (1991), N’Galy et al (1988), and Whittle et al (1992) find that the progression of HIV is faster in Africa than in the industrialized world, although Morgan et al (2002) find the pace to be similar, roughly 10 years from seroconversion to death. Schwartlander et al (1999) give estimates of survival times in high-mortality and low-mortality environments, and suggest that the average adult survival time falls from ten years to eight in high-mortality environments. Hoffman et al (1999) find that the presence of falciparum malaria in the blood significantly increases the viral load of those suffering from HIV, indicating that whether or not the average progression of the disease differs in Africa, cross-sectional variation within the continent is likely to exist in the speed of progression of the disease.

sexual behavior and survey data from 14 African countries finds that ‘differences across countries in Africa (in transmission rates) can be fully attributed to differences in risky sexual behavior and epidemic timing’. We fail to find any significant cross-sectional correlations between seroprevalence and condom use, and Figure 2 in the appendix shows the scatterplot of the maximum prevalence observed against the year in which prevalence first went above 5%; again no clear relationship is observable. While Young (2005) holds that there is indeed a ‘gift of the dying’, in his setup this benefit is realized through a posited decrease in fertility and the increase in resources per capita that accompany AIDS mortality.

The mechanism suggested here is more mechanical, simple, and macabre: the ‘gift of the dying’ is the removal of the potential infections that would have been caused by that individual. While this is a result of most standard epidemiological models, I have found only a single direct reference to it; May & Anderson (1987) say that:

“The frequent assumption that the severity of the epidemic, in terms of cumulative mortality, will be greatest if all those infected eventually develop AIDS and subsequently die is not necessarily true. Mortality depends critically on the duration of infectiousness of both those infected who develop AIDS and those infected who do not. If the latter have a similar life expectancy to those not infected, but remain infectious for life, they may contribute more to the net transmission of the virus than those who die of AIDS”.

By extension, if a policy intervention has the effect of extending the lifespan of the infected, this intervention will drive down mortality in the short run, but assuming any transmission of the disease to the uninfected, will increase the total number of people who eventually contract the disease. In what follows, we derive this result in a closed-system transitional model of epidemics as well as in a steady-state model of endemic disease with population growth. The relationship is confirmed using data from 32 African countries as well as micro-data from Kenya. The paper concludes with a discussion of the implications for interventions currently underway to ease the enormous burden that HIV imposes on Africa.

2. THEORETICAL MODEL

The effect that changes in mortality have on the prevalence and incidence of HIV/AIDS can be illustrated through a model that describes the dynamic path of the disease. We use a simplified form of the setup found in Palloni (1996). These dynamic equations, which were fit to East African data by Hueveline (2003), feature the age- (a) and time- (t) specific numbers of individuals who are uninfected, who are infected but have yet to develop the disease, and then those with full-blown AIDS. For parsimony we drop the intermediate category, and so we examine only the healthy $H(a,t)$ and those with AIDS $A(a,t)$. The differential equations that govern the motion of these two populations through time are:

$$\partial H(a+v, t+v)/\partial v = -(\mu_1(a,t) + \lambda(a,t))H(a,t)$$

$$\partial A(a+v, t+v)/\partial v = -\mu_2(a,t)A(a,t) + \lambda(a,t)H(a,t)$$

Here μ_1 is the mortality rate among the healthy, μ_2 is the mortality rate among those infected with AIDS, and λ is the rate at which the healthy contract the disease.

Because this model is primarily intended for estimation, there is no explicit modeling of the process through which infection occurs. For this, we modify the model used by Sweat et al (2000) and Bouey, Saidel and Rehle (1998). They give the probability of contracting AIDS over a discrete time period as:

$$1 - \{P[1 - R(1 - F * E) * S]^N + (1 - P)\}^M$$

where P is the prevalence rate among the sexual partners of healthy individuals, R is the probability of transmission per sexual act, F is the fraction of acts that use a condom, E is the efficacy of condoms, S is the likelihood of having sex, N is the number of sex acts within the period, and M is the number of sexual partners.⁴

⁴ To see values of these parameters estimated using data from Malawi, see Thornton (2006), and for per-act transmission probabilities in Rakai, Uganda, see Gray et al (2001).

To simplify the problem we can think of following a single cohort over time (which allows us to drop the age/time cell notation). In order to fit this model of infection into our dynamic equations, we assume that healthy individuals randomly partner with a person of their own age. A fraction θ of the infected individuals in a given age cell continue to be sexually active with their partners, regardless of whether the partner is infected.⁵ The prevalence of sexually active infected individuals is thus given by θP . Denoting the effective probability of transmission per sex act as $\pi = R(1 - F * E) * S$, we assume that in each period each individual has one sex act with one randomly chosen partner (making $M=N=1$), leaving us with the following equations of motion:

$$\partial H / \partial v = -(\mu_1 + (1 - \{\theta P(1 - \pi) + (1 - \theta P)\}))H$$

$$\partial A / \partial v = -\mu_2 A + (1 - \{\theta P(1 - \pi) + (1 - \theta P)\})H$$

Denoting the time derivative as \dot{H} and collecting terms, we have

$$(1) \quad \dot{H} = -\mu_1 H - \pi \theta P H$$

$$(2) \quad \dot{A} = -\mu_2 A + \pi \theta P H.$$

We can call the product $\pi \theta$ the ‘riskiness’ of a population; for a given prevalence rate and a healthy population of a given size, this product shows the rate at which new infections will occur. All infection in this model comes from risky sexual activity among those who are currently infected.⁶ Should θ or π fall to zero, transmission stops completely.⁷ This is

⁵ It is a well-known feature of such models that random mixing leads to higher steady-state levels of infection than assortative matching models where individuals pair only with members of their own subgroup, see Garnet & Anderson, 1996.

⁶ In countries where the epidemic is driven by transfusions, needle sharing, or homosexual sex, then θ should be thought of as the probability of the infected engaging in this activity with the uninfected, and π as the probability of transmission given that they do.

⁷ In practice this would require not only that all individuals who know they are infected remain totally abstinent with the uninfected, but that all infected individuals realize immediately that they have become infected. Given the very high infectiousness of individuals within their first few months of contracting HIV (Quinn et al, 2000), this is unlikely to be a realistic outcome with any level of testing and restraint.

similar to the classic ‘SIR’ epidemiological model except that there is no ‘recovered’ group when we model diseases that are always fatal, like HIV (Brauer, 1990).

What is the effect of the provision of health care in this environment? If high-quality health care were to alter sexual activity or the use or effectiveness of condoms, then we would see a corresponding decrease in π , the probability of infection per sex act. In a randomized study, however, Thornton (2006) finds that the impacts of testing & incentives on condom use are minimal. In the same study, while some evidence is found that condom use increases slightly when individuals learn their HIV status, there is no evidence that their amount of sexual activity changes. Hence while some decreases in π or θ may be observed as a consequence of extensive counseling and testing, these effects are likely to be much smaller than self-selected samples would lead us to believe. The more immediate and concrete effect of good health care is likely to be a decrease in mortality.

It is of interest to study how the mortality rate among the infected, μ_2 , influences the time path of prevalence. The instantaneous rate of change in the time derivative with respect to μ_2 is simply $-A$; meaning that as the rate of mortality changes, the change in the number of surviving infected individuals is proportional to the number of infected individuals. Once we examine changes over a discontinuous period of time, however, we see an additional effect enter the equation: a higher mortality rate leads to a lower prevalence rate, and assuming π and θ greater than zero, this leads to a lower *incidence* rate. This can be seen by examining the future change in prevalence in a discrete time model. In the first period, we would see only the direct effect of mortality on decreasing prevalence:

$$\frac{\Delta \dot{A}_t}{\Delta \mu_{t-1}} = -\Delta \mu_{t-1} A_{t-1}.$$

In the second period, however, we would begin to see the feedback effect of increased mortality:

$$(3) \frac{\Delta \dot{A}_t}{\Delta \mu_{t-2}} = -\Delta \mu_{t-2} A_{t-1} - \Delta \mu_{t-2} \pi \theta P_{t-2} H_{t-2}$$

This tells us that the change in the prevalence (which equals incidence minus mortality) will be affected in two different ways by an increase in the mortality rate. The first is the direct effect through which a one unit increase in the mortality rate removes a number of individuals from the infected population which is proportional to the population of infected people. The second effect arises because as mortality among the infected increases, the number of sexually active people who are HIV positive also decreases, and hence the probability of new infections, or the incidence, decreases as well. This means that when everything else is equal we expect more rapid increases (on the upward-sloping portion of the prevalence curve) and less rapid decreases (on the downward-sloping portion) in countries that provide long life spans and low mortality for their infected populations. These effects occur both because these individuals remain in the population to be counted for more years, and because the presence of any sexual activity among the infected ($\theta > 0$) caused a higher rate of infection among those who are currently healthy.

STEADY STATE.

Because the preceding approach uses transition dynamics it is not informative as to long-run equilibrium conditions. One way of writing down a model that generates a stable steady state is to introduce population growth into the healthy population, and then to make the ‘mass action’ or perfect mixing assumption; namely that the probability of contracting HIV is proportional to the *number* of interactions between the healthy and infected, which can be modeled as $\pi \theta A H$, with π modified to take the appropriate values for this new

formulation.⁸ The rationale for this assumption would be that large numbers in both populations increase opportunities to match with new partners and hence the number of individuals who contract the disease. The value of this simplification is that it allows us to write down a dynamic system which is a special case of the Volterra-Lotka predator-prey model. Writing population growth among the healthy in the absence of the disease as $f - \mu_1$ (where p is the fertility rate), we have

$$(4) \quad \frac{\dot{H}}{H} = f - \mu_1 - \pi\theta A$$

$$(5) \quad \frac{\dot{A}}{A} = -\mu_2 + \pi\theta H.$$

This is a standard formulation of the predator-prey model, except that because healthy and infected are measured in the same units, we impose that ‘one wolf equals one sheep’. This problem has two solutions; an unstable equilibrium at $(0,0)$ and a stable equilibrium at

$$(H, A) = \left(\frac{\mu_2}{\theta\pi}, \frac{f - \mu_1}{\theta\pi} \right).$$

Figure 4 shows the phase plane diagram of this system; the steady

state is the intersection of the two dotted lines where $\dot{H} = \dot{A} = 0$, and healthy/infected numbers will move in a closed-orbit trajectory around this point.⁹ From this picture it is clear that as μ_1 decreases, the steady state number of infected individuals increases. An increase in mortality among the infected has no effect on the steady state, and an increase in either π or θ leads to an initial increase in the number of infections but a decrease in the steady-state.

⁸ Specifically, divide the previous measure of π by $A+H$ to normalize the probability by the size of the healthy and infected population. See Hethcote (2000) for an excellent summary of mathematical models of infectious diseases.

⁹ If we return to modelling the probability of infection as being a function of the share of infected in the population, this model generates a steady state to which the model converges, rather than featuring perpetual oscillation.

Transition dynamics give us a mechanism through which high mortality among the *infected* will decrease prevalence. The steady-state analysis shows that high mortality among the *healthy* decreases prevalence. While we expect the variation in these two forms of mortality to be highly correlated, these models provide two conceptually separate mechanisms through which prevalence rates will be decreasing in mortality.

SIMULATIONS.

Figure 3 in the appendix uses parameter estimates taken from field studies in Africa to simulate how changes in the mortality rate among the infected alter the trajectory of the disease. We use the midpoint of the range given in Thornton (2006), and derive a probability of infection per sex act with an HIV-positive partner of $\pi = .006$.¹⁰ We calculate the overall

probability of infection per year as $P = 1 - \left[\frac{\theta A}{A + H} [(1 - \pi)^N - 1] + 1 \right]^M$, where $N=82.9$ is the

average number of sex acts per year and $M=1.2$ is the average number of partners. Since few reliable estimates of sexual activity among the infected exist, we use the Kenya DHS data to calculate θ : the percentage of respondents who test positive for HIV that report sexually activity (or pregnancy) in the past 4 weeks is 62%. We begin the simulations with a population of 1000 individuals where the initial prevalence rate is 5%, and there is 2% population growth in the absence of HIV. We see that when the mortality rate among the infected is 10%, the model converges rapidly to 100% infection. A mortality rate of 20% leads to a similar outcome but more slowly. Once the mortality rate has increased to 30% or 40%, however, die-off among the infected proceeds more rapidly than new infections, and so prevalence falls immediately from its initial level and asymptotes towards zero. We can solve

¹⁰ Specifically, we calculate $\pi = R(1 - FE)S$ with R (the transmission rate per sex act)=.01, F (the fraction of sex acts with a condom)=.21, E (the efficacy of condoms at preventing transmission)=.925, and S (the likelihood of having sex)=.77.

numerically for the mortality rate at which the prevalence rate reaches a steady-state; in our model this figure is a mortality rate of 27.2%, at which level the prevalence remains 5% as the population continues to grow.¹¹ Figure 4 shows the stock of healthy individuals over time, and only when mortality among the infected is *above* the low twenties does the healthy population continue to grow.

The mean U.S. life expectancy from infection, in the absence of ARVs, is estimated to be between 10 and 12 years, corresponding to a mortality rate among the infected of roughly 10%. The fact that decreasing life spans among the infected to 4 years can cause HIV to settle into a non-epidemic steady-state raises an intriguing possibility: it might be the case that HIV has been endemic in human populations for much longer than previously thought, but it was only when an outbreak occurred in a group whose mortality rates had been suppressed by modern medicine that the disease took on its epidemic form.

3. CROSS-COUNTRY ANALYSIS.

In examining the relationship between health care systems and AIDS rates there is a natural bias which would emerge if we used prevalence estimates based on data collected by the national health care systems. Those health care systems which are the most extensive and well funded would likely detect the disease in a higher proportion of cases and hence appear to have a higher prevalence level. For this reason, it is crucial that we use an independent, objective data source that is collected in a consistent way across countries. The UNAIDS/WHO Working Group on Global HIV/AIDS and STI Surveillance provides such a source: these data are based on blood tests at maternity clinics across SSA and reports

¹¹ Note that our simulation includes only a single cohort cell, and so is likely to overstate the speed of spread of the disease through all cells in the population. If sexual activity is strictly within-cell, then even the collapse of a specific cell may have a limited effect on overall population statistics.

statistics for each testing site for each year.¹² There are sophisticated epidemiological modeling tools that allow for the simulation of incidence and prevalence rates. These tools, however, use substantial additional demographic information in forming these projections, and so regression of such simulated data on explanatory characteristics may lead to a circular statistical relationship.

For this reason, in this analysis we use the median seroprevalence level for rural and urban areas reported across the sentinel testing sites for each country and year. These data are somewhat noisy, and in some cases lack intermediate values, and so in order to create as sound and continuous a set of outcomes as possible, we first linearly interpolate missing years for any year in which the preceding and following data are not missing. Then we run a Lowess smoother over the resulting data points to reduce noise. These interpolated, smoothed data points are used for the analysis that follows, and in Figure 5 we see them, along with the original median levels, plotted for urban women for the countries used in the analysis. Any country that has fewer than 6 years of data is dropped from the analysis, leaving us with 32 countries total.

Table 1 gives the 1980 male and female life expectancies, the 1990 health care expenditures as a percentage of GDP, and the peak levels of rural and urban HIV prevalence observed for each country in the sample. Mortality and life expectancy are closely related, as we would expect, but the relationship with health care spending appears surprisingly weak. From this table it is visually clear that countries with longer life expectancies before the outbreak of the disease have higher maximum prevalence; the average urban HIV prevalence rate among the ten bottom-ranked countries in terms of life expectancies is 10, while in the

¹² http://www.who.int/GlobalAtlas/predefinedReports/EFS2004/EFS_PDFs/EFS2004_ET.pdf

ten top-ranked countries the average rate is 24. In what follows we undertake further examination of these correlations.

3.1 CORRELATES OF MORTALITY

The theoretical model focuses our attention on mortality rates as a key driver of the trajectory of the AIDS epidemic. Data on life expectancies or mortality rates for HIV-infected individuals do not exist in Sub-Saharan Africa. We do, however, observe male and female mortality rates in 1980 and 1990, and cross-country variation in mortality in the population prior to the onset of the epidemic is likely to be a reasonable proxy for variation in mortality among the infected once it begins. Table 2 in the Appendix shows the results of unconditional pairwise OLS regression of 1990 mortality rates on a variety of other national characteristics.

In general, these correlations hold no surprises. We see the strong, structural correlation between mortality rates and life expectancies that we would expect. Health care provision seems generally to have been efficacious, as expenditures, clean water, doctors, and immunizations are all associated with lower mortality rates. Hospital beds and population density are uncorrelated with mortality, but per capita GDP displays a very strong negative correlation.

The bottom of Table 2 shows how 1990 mortality rates correlate with the maximum late-90's prevalence of non-HIV TB and malaria, and the maximum annual number of cholera deaths per country. The first sign of an unusual relationship between mortality and HIV comes from the fact that non-HIV TB is the only covariate in the data which displays a counterintuitive relationship to mortality; TB is worst in the countries that had the lowest mortality rates in 1990. Given that the resurgence of non-HIV TB in Africa in the 1990s was predominantly caused by the spread of AIDS, the implication of this correlation is that the

spillover effects of HIV on other diseases were most muted in those countries with the highest mortality.

3.2 PREVALENCE OF OTHER DISEASES

We now examine in more detail the relationship between public health variables and the incidence of non-HIV diseases. There is potential endogeneity of placement and timing of health expenditures; if disease outbreaks cause the health infrastructure to ramp up operations then we have positive reverse causality. The same would be true for the direct effect of disease on mortality, again causing upward bias. To address this problem we lag our covariates; 1980 mortality rates are used in order to ensure that cross-sectional differences in HIV were not themselves driving mortality rates. The earliest high-quality health expenditure data available are from 1990; we use the 1990 public health expenditures as a percent of total government expenditure, as well as the 1995 expenditures as a percent of GDP.¹³

Table 3 shows the pairwise correlations that lagged mortality & health expenditures display with non-HIV TB, malaria, and cholera. In no case are these relationships significant, although we see that cholera outbreaks are somewhat more severe in countries where women had high mortality in 1980. One explanation for these weak relationships is that the long-term effects of health spending are minimal, whether because of a lack of duration in the effects or because the spending was ineffective in the first place. Another explanation is that the time path of these diseases did not fluctuate greatly, meaning that even the 1980 spending is endogenous to prevalence. This effect, if present, would increase spending in high-disease environments, pushing the sign of the relationship downwards and making spending appear less efficacious than it in fact is.

¹³ Data from World Health Report 1999, Statistical Annex, WHO

3.3 HIV/AIDS AND LAGGED PUBLIC HEALTH

When we move to examining correlations with HIV/AIDS, the picture changes dramatically. Table 4 gives the pairwise correlations using the same set of lagged public health outcomes and a variety of measures of the AIDS epidemic. We calculate mean and maximum prevalence and mean and maximum changes in prevalence separately for rural and urban areas for each country; mean prevalence is $E(A)$ from (2), and the mean change in prevalence is $E(\dot{A})$. 1980 Mortality displays a very strong negative correlation with subsequent AIDS prevalence, in both rural and urban areas. The lowest t-statistic for the relationship with prevalence levels is 4.4, and for prevalence changes is 3. While the relationships with health expenditures are less sharp, and not quite significant in explaining mean urban prevalence, in all other respects the relationship is significant, particularly in rural areas.

All of these correlations move in the opposite direction of what we should expect from an exogenous, efficacious health intervention, and all conform with the theory derived above. It is possible that the 1990s health care spending figures, being from years in which certain countries already had full-blown AIDS epidemics, are endogenous. While neither 1990 nor indeed 1995 had seen any large-scale response to the AIDS epidemic, the effect if present would explain the fact that those countries with the highest health care expenditures have the worst and fastest-spreading AIDS epidemics. The relationship with 1980 mortality, however, is both stronger and much harder to explain in terms of reverse causality. Because this is truly before the beginning of the epidemic, it is implausible that mortality was being driven directly by AIDS.

Changes in prevalence over time equal the incidence rate minus the mortality rate. Average changes in prevalence, then should display a negative structural relationship with

mortality that is not related to any possible effect of prevalence rates on incidence (the term on the right in (3)). Unfortunately, high quality panel data on incidence are lacking for countries in Sub-Saharan Africa, and so we are unable at this time to test this hypothesis directly. However, when we examine the relationship between the maximum increase in prevalence seen in a country and the contemporaneous prevalence, we see a very strong positive relationship (t-stats of 3.8 for urban women and 3.7 for rural women). This relationship is consistent with high infection levels leading to high transmission rates.

3.4 DIFFERENCE ESTIMATION

Because we have multiple observations for each country on several variables, panel identification can be used to examine the relationship between changes in health determinants and changes in HIV. We must be careful in setting up and interpreting these relationships, however, because once the epidemic had begun it exerted powerful causal forces on the time path of the health outcomes we have been using. In an attempt to construct a RHS variable that is truly exogenous to the subsequent trajectory of the disease, we calculate the change in life expectancy between 1972 and 1982. Correlations between HIV and this quantity are interesting because countries in the sample were experiencing almost linear increases in life expectancy over the decades prior to the onset of AIDS, whereupon they plummet by 20 years in most countries. Hence this linearized rate of growth over the decade prior to the onset of AIDS may provide a reasonable counterfactual for what would have obtained in the absence of the disease. We also use as a regressor the change in PPP GDP p/c from 1980 to 1990.

Table 5 reports the results of these changes on changes regressions. The top panel uses pairwise correlations, running a separate regression to estimate the correlation of each factor independently with changes in prevalence, while the bottom panel includes the three

regressors simultaneously. The pairwise correlations are consistent with the predictions of our theory; changes in life expectancy are associated with more rapid subsequent increases in seroprevalence, although these effects are insignificant when one uses changes in the UNAIDS discrete categories. The strongest effects in both panels are seen in the relationship between lagged increases in female life expectancy and subsequent increases in the rate at which HIV spreads in rural areas.

3.5 CONDITIONAL CORRELATIONS

In recent years increasing attention has been placed on the counterintuitive positive relationship between national income and HIV rates in Africa. There are two explanations commonly provided: that wealthier individuals are more able to engage in risky activities, and that rich countries have better transport networks and therefore disseminate the disease more widely. In a statistical sense, the relationship suggested in this paper is just a third explanation for this positive correlation. Teasing out the health channel from the infrastructure and wealth channels will be difficult for the same reason that cross-country growth analysis is difficult; a country that is 'good' under one definition is likely to be good under others as well. Indeed, many well-being indexes use precisely the kinds of health care measures (water & sewer infrastructure, local medical infrastructure, disease burden) that we wish to separately identify. In what follows we pursue several different strategies to shed some light on the presence of an independent health channel.

With data from only 32 countries and no natural experiment, our ability to ascribe causality to partial correlations is obviously limited. We use 1980 population density and 1990 road networks as infrastructure controls, and 1990 per-capita PPP GDP as the wealth control. Table 6 presents multivariate OLS results for urban and rural areas separately, and allows us to observe the partial correlation between female mortality and prevalence. There

is some evidence that population density pushes up urban prevalence while a good national system of roads pushes up prevalence in rural areas. In all regressions, however, it is 1980 female mortality rates that are the strongest conditional correlate of female seroprevalence after some two decades (except in urban areas where the relationship between income and the speed of spread of the disease is slightly stronger).

4. INDIVIDUAL-LEVEL ANALYSIS.

The 2003 Kenya survey by Demographic and Health Surveys (DHS) gives us a unique opportunity to examine these correlations at the individual level, because it combines a health-focused household survey with blood testing data. The individual analogy to the preceding analysis is use individual- and locality-level data to predict the probability that a given individual is HIV-positive. The fact that these data contain 400 spatial clusters gives us substantially more power to separately identify the health channel. At the individual level we control for education, gender, age, ethnicity, and religion. The remaining variables are calculated as cluster-level averages, because we wish to explain how the prevailing local environment affects the spread of the disease. We attempt to avoid variables which suffer from obvious endogeneity in being themselves a function of the patterns of HIV.

In order to clarify the channels, we divide our explanatory variables as follows:

Health: % with no developed toilet facility, % drinking river water, % of deliveries for which there was no prenatal care, and % of children's fevers that remain untreated.

Wealth: % with electricity, % with radio, % who own their house, % own land.

Infrastructure: Distance by road to Trans-African Highway, distance by road to any paved road, average number of times men report being away from home in the last month, % in cluster who own cars.

We also include the following sets of cluster-level controls, both of which are potentially endogenous to the public health response to HIV:

Vaccinations: % children vaccinated for measles and DPT, % any additional vaccinations.

Circumcisions: % of men circumcised, % of women for whom FGM practiced.

Table 7 gives the results of this analysis for different control structures. All regressions are probits with clustered standard errors, and marginal effects are reported. Women have HIV rates 3 percentage points higher than men, and the relationship to age is strongly concave. Male circumcision at first appears to have a 10 percentage point effect on HIV rates, but the Luo group, who do not circumcise, have Kenya's highest infection rates. Once we include a dummy for this group the effect falls to roughly 3 percentage points, but still represents a significant decrease. Interestingly, the coefficient on female circumcision is consistently negative and comes near significance in some cases. Vaccines and religion are not found to have significant effects.

The effect of the health controls is generally consistent with the theory, with water and sanitation facilities showing a particularly strong correlation with HIV rates. The absence of prenatal care, and a high incidence of untreated fevers in the cluster have the expected signs but are in no cases significant. Among the infrastructure controls the number of times that the household head has traveled away from home has the strongest positive correlation with HIV rates, and distance to roads or the trans-African highway are surprisingly insignificant. Land and radio ownership are the most significant wealth controls. When we combine all of the explanatory variables and use ethnicity dummies, few of our disaggregated controls remain significant.

In order to investigate the effects of different channels in a more concise way, Table 8 combines all of the variables describing a given channel into a single index. Following the

Human Development Index, we calculate $Index_i = \frac{1}{4} \sum_{j=1}^4 \frac{X_{ji} - X_{j\min}}{X_{j\max} - X_{j\min}}$, where there are 4 variables in each index, and $X_{j\min}$ and $X_{j\max}$ represent the minimum and maximum values in the data for each variable. Each index thus calculates a relative rank, weighing each element in the index equally, with 0 being the ‘worst’ outcome and 4 the ‘best’ for each family of controls.¹⁴ Again we see the strong effects whereby good health care outcomes are correlated with high HIV rates, although the apparent magnitude of these effects is weakened when we simultaneously include wealth controls.

Causality is not rigorously established through these regressions. The use of contemporaneous controls opens up the possibility that our explanatory variables are being driven by the trajectory of HIV. Even if health care infrastructure is considered exogenous at a specific location, it is possible that infected individuals migrate to locations which offer the best services.¹⁵ Nonetheless, these micro-data strongly confirm the positive relationship between HIV prevalence and standard of living, measured in several different ways. Wealth and health controls have roughly equal explanatory power over HIV rates, however in an unequal, capitalist country such as Kenya, the large differences across economic lines in the quantity and quality of health care presents itself as a plausible channel for the otherwise surprising fact that the wealthy have substantially higher HIV rates than the poor. Overall, the results are suggestive of the presence of an independent channel through which good health care infrastructure is related to high HIV rates..

¹⁴ The vaccine index has only three elements, and thus ranges from 0 to 3.

¹⁵ Given the penury of many of those suffering from AIDS, however, it is also possible that the infected are forced to move back to the village where the cost of living is lower, causing the reverse effect.

5. CASE STUDIES.

We cannot interpret contemporaneous changes in public health as ‘shocks’ off of which to identify the effects on HIV, because they are strongly endogenous to the path of the disease. We can, however, examine cases in which radical changes in the public health environment occurred as a result of political shocks that we think were not caused by the AIDS epidemic.

4.1 ZIMBABWE & KENYA

These two countries provide case studies in the path of AIDS in economies that were relatively rich at the time the epidemic struck but which rapidly contracted thereafter. In Zimbabwe, Mugabe’s erratic grip on the country reduced access to imported medical supplies and food, and its people saw a huge drop in standards of living and have left the country in large numbers. A recent study on the subject (UNAIDS, 2005) concludes that “The decline in national HIV prevalence between 2000 and 2004 resulted from a combination of declining HIV incidence and rising adult mortality occurring from the mid- and early- 1990s, respectively.” Kenya’s institutional decline was less precipitous, but the PPP dollar value of the decline in per-capita GDP was roughly the same.

Figure 6 plots the trajectory over time of smoothed urban HIV prevalence for four countries. In the early years the shape of the curve for Zimbabwe resembles that of Botswana, whereas Kenya resembles South Africa. During the ‘90s, Kenya’s PPP GDP p/c fell by \$136, and Zimbabwe’s by \$144, while Botswana’s increased by \$1,795. In this way the subsequent trajectories of the disease provide a comparison between countries that were rapidly improving the standards of their people and those in which they were slipping backwards. When it comes to HIV, again, we see a worse-is-better response. The rapid increases in prevalence come to an end in contracting economies; both countries show a

slight fall in incidence around the mid-1990s, whereupon rates appear to plateau (at 15% in Kenya and 30% in Zimbabwe). Prevalence in the growing economies, on the other hand, continues to climb for roughly twice as long, eventually reaching levels which are 15% higher.¹⁶ The pattern is consistent with the ‘difference in differences’ intuition from our theoretical model.

4.2 UGANDA

Uganda provides a hopeful case study, although for a reason different from the one usually claimed. The countries of the Great Lakes region had similar female life expectancies in 1982, ranging from 48 in Rwanda to 52 in the DRC. The end of Uganda’s long civil war and Museveni’s ascension to power set the country on a path of stability and growth through the 1990s, during which it was one of the 10 fastest-growing economies in the world. Thus Uganda’s AIDS epidemic began in a poor country and ended in a richer one. Rwanda, Burundi, and the DRC, on the other hand, collapsed into chaos during this time. The DRC has by far the largest fall in PPP GDP p/c during the ‘90’s, \$877, and Rwanda fell by \$96 and Burundi by \$282 while Uganda increased by \$393. This means that, according to the relationships seen elsewhere in the data, for a given set of initial conditions we would expect prevalence to be higher and to fall more slowly in Uganda than in its neighbors. On the other hand, since the Great Lakes region was the first hit by HIV, and had very low levels of public health at that time, we would expect the epidemic to have peaked first there and fallen fastest there even in the absence of any effective AIDS policy.

Figure 7 displays the smoothed prevalence trajectories for these countries, and we see that Uganda is a success by both measures. It is indeed the case that the country sees sharp

¹⁶ Note that the link between economic collapse and the HIV epidemic introduces reverse causality into efforts such as Dixon et al (2002) to identify the impact of AIDS on economic growth using panel data. This positive feedback may explain why it has been hard to reach consensus on the causal effect of AIDS on growth (Wendell & Werker, 2004).

falls in prevalence rates, but then so do all of Great Lakes countries. What is more important is that the rate and total percentage of decrease is higher than in any Great Lakes countries despite the large relative increases in Uganda's standard of living.

The conclusion is that despite changes in variables that should otherwise have increased incidence, Uganda has successfully implemented other policies that have slowed the rate of new infections. Whether it was able to influence sexual behavior among the infected (θ) or to decrease the rate of transmission (π), Uganda would seem to have been unique in its ability to combine rapid growth with decreases in prevalence. Only one other country in Sub-Saharan Africa, Burkina Faso, combined increasing PPP GDP p/c with falling HIV prevalence during the '90's (the average fall in prevalence in Burkina Faso was a tenth of the average fall in Uganda).¹⁷ Since the A and B in Uganda's ABC policy speak to decreasing θ , while the C addresses decreasing π , so we cannot identify the channel through which it worked. What we can say is that Uganda is a success not because it brought down AIDS rates (many failing states achieved this) but because it did so while growing.

6. ANTI-RETROVIRALS.

In a 'risky' population where both π and θ are non-zero, a high prevalence rate leads directly to a high incidence rate by increasing the share of the sexually active who are infected. In this case, we see an exceedingly thorny policy problem emerge, in which actions taken to extend the lifespan of those suffering with HIV/AIDS lead in a direct causal sense to new infections. Such a policy, therefore, effectively trades off years of life among the currently infected against years of life among the currently uninfected. This indicates that there will exist a negative unintended consequence of the distribution of ARVs. If the only

¹⁷ Burkina Faso is another country that is credited with having implemented an aggressive, homegrown program to influence behavior among its citizens.

impact of the ARV intervention is to decrease mortality, this feedback is inevitable.

However, our theoretical model gives us the machinery to derive the requirements on changes in π and θ in order for the net effect of an ARV intervention to be a decrease in the number of new infections.

The intervention potentially induces a change in all three of the parameters in Equation (2): the above arguments lead us to expect a strong decrease in mortality, some decrease in the rate of infection per sex act, and an ambiguous change in sexual activity among the infected. We can totally differentiate (2) and divide by A to get the following:

$$\frac{d\dot{A}}{A} = -d\mu + [d\pi \cdot \theta + d\theta \cdot \pi] \frac{H_{t-1}}{A_{t-1} + H_{t-1}}.$$

The expression $\frac{H_{t-1}}{A_{t-1} + H_{t-1}}$ gives the share of the population uninfected in the previous period, and so can be written $(1 - P_{t-1})$. The total change in the share newly infected will only be negative if the expression on the RHS is above is negative, that is if

$$(6) \quad -\left(\frac{d\pi}{\pi} + \frac{d\theta}{\theta}\right) > -\left(\frac{d\mu}{\theta\pi(1 - P_{t-1})}\right).$$

The left-hand side of (6) represents the behavioral change induced by the intervention, and the right side gives the perverse effect by which success at decreasing mortality among the infected causes new infections. This simple statement says that in order for the intervention to result in a decrease in future infections, the total percent fall in riskiness must be greater than the mortality effect, expressed as a percentage of the rate at which new infections are occurring. The medical literature tells us that viral loads, and hence the degree to which an individual is infectious, are depressed by ARVs, exerting a negative force on $\frac{d\pi}{\pi}$. Any

change in condom use is likely to be an increase, reinforcing this effect. In policy terms, this

implies that the distribution of ARVs must be made to have sufficiently strong effects on decreasing riskiness as to overwhelm the additional numbers of infected that have been ‘introduced’ into subsequent periods by the treatment.

The ambiguity of the sign of $\frac{d\theta}{\theta}$ will drive this inequality, because we have hard causal evidence demonstrating that both $\frac{d\pi}{\pi}$ and $\frac{d\mu}{\theta\pi(1-P_{t-1})}$ are negative. In other words, once we net out the immunological effects of the medication $\frac{d\pi}{\pi} - \frac{d\mu}{\theta\pi(1-P_{t-1})}$, is the change in behavior large enough to create a net decrease in new infections? If decreased sexual activity was arising from morbidity and ARVs improve the feeling of well-being, activity may increase. Similarly, if the availability of ARVs decrease the apparent costs of contracting HIV, a standard cost-benefit model would predict an increase in sexual activity as a result.

Perhaps the most powerful arguments for a decrease in sexual activity from an ARV intervention are that they provide an incentive for widespread HIV testing, and create very regular interactions between the infected and health care professionals.¹⁸ These interventions usually dramatically increase the percentage of the sexually active population who know their status. If we believe that behavioral change starts from knowing that one is infected, this is the key piece of groundwork in an effective response to the disease.¹⁹ Further, because the administration of ARVs requires ongoing, intense contact with health workers, a highly effective conduit is provided for dissemination of information by a health ministry.

¹⁸ See Philipson & Posner (1995) and Caplin & Eliaz (2003) for theoretical treatments of the demand for STD testing.

¹⁹ Booser and Philipson (2000) sound a note of caution on the impact of testing, suggesting that only those ‘surprised’ by the test results will change their behavior, and hence a population wherein numerous individuals who believed they were infected find out that they are not can in fact see an increase in sexual activity. In our setup there is no problem with this as long as the sexually riskiness of those who discover that they *are* infected decreases.

Sero-sorting, through which individuals who know their status pair with others of the same status, can render the same degree of overall sexual activity completely harmless in terms of incidence. While several studies of HIV testing have not found that it does not decrease sexual activity (Boozer & Philipson 2000, Thornton 2006), a population that does now know its status cannot sero-sort, and so this more subtle yet equally efficacious behavioral change may result from testing.

If such behavioral changes cannot be effected, then the distribution of ARVs introduces a macabre bargaining problem between the currently ill and those who will become infected in the future. Several factors lead us to expect this debate to be particularly difficult. First, discussion of the means through which riskiness may be changed are inevitably tied up with sexual behavior and birth control measures, both uncomfortable topics and ones lacking in consensus. The debate also suffers from at least two forms of agency problem; like trade debates, it pits a known, organized group (the currently infected) against a diffuse, unknown future group. Political processes naturally favor groups that can organize over those that cannot, but the fact that we cannot identify the specific individuals who will be infected as a result of decreased mortality makes their future suffering no less real. Thus, these 'future infected' are disenfranchised in the debate over current policy. Finally, in many cases foreign donors are playing a disproportionate role in providing ARVs, relative to the contribution of domestic governments. The decisions on how to address these tradeoffs would therefore be likely to fall on principals such as PEPFAR rather than being resolved through domestic political discourse.

7. CONCLUSION.

This paper connects two rather obvious propositions: that good public health policy reduces mortality, and that *ceteris paribus* high mortality hastens the course of an epidemic. By linking the two, the possibility is raised that Sub-Saharan Africa's high burden of endemic disease has created an unintended consequence of public health systems. The data show that nations which had previously done the best job of caring for their citizens are hit hardest by HIV/AIDS. Countries that had the worst prior public health see the epidemic roll through their populations more quickly, and with much lower peak prevalence rates. If African governments had more effective tools to combat AIDS directly, we might see that health care's direct negative effect on AIDS would be predominant; instead we see a positive relationship which is consistent with high background mortality suppressing the spread of AIDS. While this relationship is an immediate result of a simple epidemiological model, it is nonetheless strongly counterintuitive in a policy sense.

Seen through this bleak lens, Uganda provides a positive example in a manner somewhat different from the one usually advanced. Uganda is a Great Lakes country that was poor at the time the epidemic took off, and so it should come as no surprise that the country has seen a fall in prevalence rates (Rwanda, Burundi, and the DRC all saw large decreases in prevalence at the end of the '90's, all countries clearly preoccupied by issues other than AIDS prevention during that decade). What is impressive is the fact that Uganda is also a country that has seen an enormous relative increase in well-being during the course of the epidemic, and this improvement seems not to have shown up through higher eventual incidence. Uniquely among rapidly-growing economies, Uganda has seen falls in prevalence which appear almost linear; from 1991 to 2002 prevalence fell by 1.6 percentage points every year. The fact that this took place during a period of rapidly improving public health

provision suggests that, if the determinants of infection can be tackled head on, it is indeed possible to achieve improvements in health outcomes without provoking a corresponding increase in prevalence.

For a country battling a serious HIV epidemic, a successful response means maintaining a high prevalence rate in concert with a low incidence rate. The relationship suggested here suggests that this combination will be uniquely difficult to achieve because of a structural positive effect that the stock has on the flow in human-transmitted diseases. Indeed, *ceteris paribus*, the only way that we *could* observe this combination is in a country that has seen a recent decrease in the probability of transmission from infected individuals.

In the difficult debate over AIDS policy in Africa, perhaps no issue has been more contentious than the extent to which behavioral interventions should focus on abstinence versus condom use. For many rich-world governments it might appear attractive to sidestep this thorny cultural debate and instead to concentrate on the delivery of drugs to the needy. The argument laid out in this paper suggests that this is an exceedingly short-sighted policy. The moral imperative of distributing drugs as widely as possible is very real, but attached to this endeavor is a symmetric imperative to achieve a counterbalancing decrease in the transmission of the disease. Abstinence, condom use, sero-sorting, and suppression of viral loads are all reasonable means to achieving this end. No effort should be spared in conducting well-designed policy studies on the most effective use of these tools, because failing to do so will result in ‘solving’ the current wave of morbidity at the cost of a future wave of infection.

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Appendix.

Table 1: Summary Statistics.

Data Ranked by 1982 Female Life Expectancy.

Country	Female Life Expectancy, 1982	Female Mortality rate/10,000 1990	Health Exp as % of GDP, 1995	Peak Urban Prevalence	Peak Rural Prevalence
Comoros	51	.	0.9	0.1	0.0
Senegal	48	409	2.5	0.9	1.0
Niger	41	453	1.6	2.0	3.4
Benin	52	397	1.7	3.3	3.6
Ghana	56	279	1.4	3.7	3.7
Gabon	51	387	0.5	4.0	4.7
Nigeria	48	516	0.3	4.3	5.2
Guinea-Bissau	41	507	1.1	4.7	.
Guinea	41	517	1.2	5.0	2.4
Mali	44	458	1.2	5.6	5.2
Dem. Republic of Congo	52	375	0.2	6.6	5.4
Sierra Leone	37	527	4.3	6.9	.
Congo	54	.	3.2	7.1	10.0
Burkina Faso	46	349	2.3	7.9	7.9
Cameroon	53	415	1	8.5	7.9
Angola	44	401	4.1	8.6	8.0
Côte d'Ivoire	52	413	1.4	12.4	9.2
United Rep. of Tanzania	53	370	2.5	13.6	12.3
Central African Republic	50	424	1.9	15.0	15.0
Kenya	58	339	1.6	15.3	15.5
Togo	52	346	1.2	16.5	4.6
Mozambique	46	361	4.6	17.4	15.2
Ethiopia	44	362	1.6	19.2	15.5
Burundi	49	400	0.8	22.8	9.1
Malawi	46	362	2.3	25.2	18.7
Uganda	49	395	1.8	26.6	12.3
Namibia	55	232	3.7	27.4	21.9
South Africa	61	.	3.6	27.7	26.3
Rwanda	48	453	1.9	29.1	7.8
Zambia	52	.	2.6	29.6	17.6
Zimbabwe	58	321	2.2	32.0	33.5
Swaziland	55	366	2.8	36.5	38.5
Lesotho	56	334	4.1	38.2	27.0
Botswana	62	278	1.6	42.9	36.6

Table 2: Pairwise Correlates of 1990 Mortality.

Outcome:	Female 1990 Mortality	Male 1990 Mortality	nobs
Male Life Expectancy, 1982	-10.951 -(5.37)	-12.442 -(6.83)	28
Female Life Expectancy, 1982	-10.551 -(6.09)	-11.628 -(7.35)	28
Health exp as % of GDP, 1990	-28.620 -(1.62)	-33.901 -(2.01)	23
% with good drinking water, 1990	-1.668 -(1.83)	-2.143 -(2.25)	23
% immunized for DPT, 1990	-1.327 -(2.16)	-1.510 -(2.58)	26
% immunized for measles, 1990	-1.640 -(2.27)	-1.815 -(2.62)	26
hospital beds per 1000, 1990	-0.282 -(0.01)	-4.005 -(0.19)	20
doctors per 1000, 1990	-441.810 -(0.90)	-874.069 -(1.80)	16
% with improved sanitation, 1990	-0.924 -(0.98)	-1.471 -(1.49)	23
Population density, 1980	0.251 (0.79)	0.285 (0.87)	27
p/c GDP PPP, 1990	-0.022 -(2.31)	-0.025 -(2.68)	28
Non-HIV TB, max prevalence	-115.406 -(1.59)	-117.425 -(1.57)	28
Malaria, max prevalence	0.548 (0.79)	0.317 (0.44)	28
Cholera deaths, max prevalence	2621.293 (2.62)	1674.099 (1.52)	27

(t-statistics in parentheses)

Table 3: Relationship between Lagged Health Outcomes and Other Diseases.

Outcome:	Non-HIV TB prevalence, max	Malaria prevalence, max	Annual Cholera deaths, max	nobs:
Female Mortality, 1980	-0.001 (-1.10)	-0.032 (-0.56)	0.000 (1.84)	30
Male Mortality, 1980	-0.001 (-1.38)	-0.039 (-0.65)	0.000 (0.74)	30
Health care as % of gov expenditures, 1990	0.048 (1.06)	2.939 (0.63)	-0.001 (-0.38)	27
Health Expenditures as % of GDP, 1995	0.017 (1.06)	1.632 (0.63)	-0.001 (-0.38)	33

(t-statistics in parentheses)

Table 4: Relationship between Lagged Health Outcomes and HIV/AIDS.

	HIV prevalence, average	HIV prevalence, maximum	Annual change in prevalence, average	Annual change in prevalence, maximum	nobs:
Urban Prevalence					
Female mortality, 1980	-0.086 (-4.46)	-0.113 (-4.66)	-0.007 (-3.02)	-0.016 (-3.88)	30
Male mortality, 1980	-0.093 (-4.81)	-0.121 (-4.97)	-0.008 (-3.44)	-0.018 (-4.31)	30
Health care as % of gov expenditures, 1990	3.892 (1.88)	6.384 (2.42)	0.632 (2.98)	0.895 (2.09)	28
Health Expenditures as % of GDP, 1995	1.906 (1.42)	3.585 (2.10)	0.417 (3.10)	0.566 (2.11)	34
Rural Prevalence					
Female mortality, 1980	-0.082 (-5.19)	-0.121 (-5.72)	-0.010 (-4.97)	-0.020 (-4.89)	28
Male mortality, 1980	-0.077 (-5.29)	-0.113 (-5.74)	-0.009 (-4.70)	-0.017 (-4.26)	28
Health care as % of gov expenditures, 1990	3.900 (2.44)	5.877 (2.56)	0.558 (2.56)	0.881 (2.27)	27
Health Expenditures as % of GDP, 1995	2.771 (2.67)	4.213 (2.85)	0.312 (2.09)	0.880 (3.63)	32

(t-statistics in parentheses)

Table 5: Estimation using Changes on Changes.

Pairwise Correlations, Changes on Changes:

Outcome:	Ordered Probit on UNAIDS discrete prevalence categories	Average Change in Urban Prevalence	Average Change in Rural Prevalence
Change in GDP p/c ppp, 1980-90	0.0001 (0.34)	0.0004 (1.75)	0.0004 (1.71)
Change in male life expectancy, 1972-82	0.131 (0.78)	0.195 (1.84)	0.216 (1.94)
Change in female life expectancy, 1972-82	0.089 (0.64)	0.207 (2.30)	0.220 (2.38)
nobs:	25	34	32
	(z-statistics)	(t-statistics in parentheses)	

Multivariate OLS, Changes on Changes:

Outcome:	Ordered Probit on UNAIDS discrete prevalence categories	Average Change in Urban Prevalence	Average Change in Rural Prevalence
Change in GDP p/c ppp, 1980-90	0.0002 (0.74)	0.0003 (1.35)	0.0002 (1.15)
Change in male life expectancy, 1972-82	-0.490 -(1.24)	-0.197 -(0.86)	-0.194 -(0.87)
Change in female life expectancy, 1972-82	-0.143 -(0.50)	0.316 (1.55)	0.409 (2.18)
nobs:	20	25	23
	(z-statistics)	(t-statistics in parentheses)	

Table 6: Multivariate Regression.

	Urban Prevalence		Rural Prevalence	
	Maximum prevalence	Maximum change in prevalence	Maximum prevalence	Maximum change in prevalence
Population density, 1980	0.079 (1.72)	-0.006 -(0.88)	-0.014 -(0.41)	0.002 (0.28)
Road network, 1990	0.111 (0.58)	0.036 (1.33)	0.232 (1.60)	0.033 (1.17)
p/c GDP 1990	0.002 (1.36)	0.000 (2.01)	0.001 (1.21)	0.000 (0.80)
Female mortality, 1980	-0.085 -(2.85)	-0.008 -(1.91)	-0.091 -(3.55)	-0.016 -(3.20)
nobs:	27	27	25	25
	(t-statistics in parentheses)			

Table 7: Individual-level analysis using all controls.

Cluster-Level Averages:		Probit Controls:				Ethnicity Dummies:		
		Basic	Transport	Wealth	Vaccines	All	Basic	All
Health:	No flush toilet	-0.032 (2.82)**	-0.034 (3.12)**	-0.024 (1.23)	-0.036 (3.02)**	-0.021 (1.14)	-0.026 (2.37)*	-0.007 (0.42)
	Drink riverwater	-0.031 (2.39)*	-0.03 (2.47)*	-0.022 (1.78)	-0.03 (2.29)*	-0.02 (1.65)	-0.03 (2.49)*	-0.023 (1.98)*
	Children with untreated fevers	-0.017 (1.20)	-0.01 (0.72)	-0.014 (1.06)	-0.024 (1.60)	-0.018 (1.19)	-0.004 (0.32)	-0.005 (0.34)
	No prenatal care	-0.016 (0.44)	-0.013 (0.33)	-0.007 (0.21)	-0.033 (0.89)	-0.029 (0.82)	0.001 (0.03)	-0.025 (0.74)
	Transport:	Distance to TA Highway		-0.001 (0.42)		0 (0.32)		0.002 (1.52)
	Distance to paved road		0 (0.02)		0 (0.04)		0.002 (0.60)	
	Number of times hh head away from home		0.002 (2.38)*		0.001 (1.76)		0.001 (1.50)	
	Own car or truck		-0.048 (1.69)		-0.021 (0.68)		-0.013 (0.43)	
Wealth:	Own house			0.009 (0.65)	0.01 (0.67)		0.001 (0.05)	
	Own land			0.018 (1.54)	0.019 (1.56)		0.023 (2.11)*	
	Electricity			-0.025 (1.42)	-0.019 (1.10)		-0.005 (0.29)	
	Own radio			0.037 (1.76)	0.051 (2.14)*		0.042 (1.64)	
Vaccines:	DPT vaccine				-0.024 (0.57)	-0.035 (0.86)	-0.023 (0.55)	
	Measles vaccine				-0.025 (1.11)	-0.025 (1.20)	-0.016 (0.78)	
	Any other vaccine				0.014 (0.51)	0.009 (0.35)	-0.001 (0.06)	
Individual-Level Controls								
	Education	0 (0.01)	0 (0.03)	0 (0.31)	0 (0.14)	0 (0.26)	0 (0.57)	0 (0.57)
	Female	0.032 (6.15)**	0.032 (6.19)**	0.033 (6.30)**	0.032 (6.05)**	0.033 (6.22)**	0.031 (6.16)**	0.032 (6.52)**
	Age	0.018 (7.83)**	0.018 (7.74)**	0.018 (7.72)**	0.018 (7.84)**	0.018 (7.68)**	0.018 (8.21)**	0.018 (8.01)**
	Age squared	-0.0003 (7.04)**	-0.0003 (6.96)**	-0.0003 (6.93)**	-0.0003 (7.06)**	-0.0003 (6.91)**	-0.0003 (7.52)**	-0.0003 (7.31)**
	Circumcized, male	-0.102 (8.72)**	-0.098 (8.55)**	-0.105 (9.43)**	-0.093 (8.08)**	-0.091 (8.76)**	-0.038 (2.50)*	-0.034 (2.34)*
	FGM practiced	-0.017 (1.50)	-0.015 (1.26)	-0.019 (1.69)	-0.019 (1.61)	-0.02 (1.70)	-0.005 (0.34)	-0.017 (1.14)
	Muslim						-0.005 (0.37)	-0.006 (0.55)
	Catholic						-0.003 (0.40)	-0.004 (0.69)
Observations		6211	6200	6211	6098	6087	6047	5924

Robust z statistics in parentheses, * significant at 5%; ** significant at 1%

Table 8: Individual-level analysis using indexes.

	Probit Controls:				
	Basic	Transport	Wealth	Vacc	All
Health Index	0.023 (3.86)**	0.024 (3.93)**	0.013 (1.74)	0.026 (4.13)**	0.017 (2.18)*
Transportation Index		-0.003 (0.22)			-0.009 (0.74)
Wealth Index			0.013 (2.06)*		0.016 (2.26)*
Vaccine Index				-0.012 (1.36)	-0.013 (1.51)
Education	0 (0.50)	0 (0.45)	-0.001 (0.85)	0 (0.31)	-0.001 (0.58)
Female	0.032 (6.14)**	0.032 (6.14)**	0.032 (6.13)**	0.032 (6.19)**	0.032 (6.19)**
Age	0.019 (8.18)**	0.019 (8.17)**	0.018 (8.11)**	0.019 (8.12)**	0.019 (8.04)**
Age squared	-0.0003 (7.51)**	-0.0003 (7.50)**	-0.0003 (7.45)**	-0.0003 (7.46)**	-0.0003 (7.38)**
Circumcized, male	-0.033 (2.37)*	-0.033 (2.38)*	-0.038 (2.65)**	-0.028 (1.98)*	-0.033 (2.29)*
FGM practiced	-0.016 (1.53)	-0.016 (1.53)	-0.016 (1.58)	-0.018 (1.69)	-0.019 (1.81)
Luo	0.105 (6.76)**	0.106 (6.77)**	0.098 (6.25)**	0.103 (6.72)**	0.096 (6.23)**
Muslim	-0.016 (1.85)	-0.016 (1.89)	-0.016 (1.90)	-0.018 (2.06)*	-0.02 (2.24)*
Catholic	-0.003 (0.47)	-0.003 (0.49)	-0.003 (0.37)	-0.004 (0.59)	-0.003 (0.51)
Observations	6047	6036	6047	5935	5924

Robust z statistics in parentheses

* significant at 5%; ** significant at 1%

Figure 1

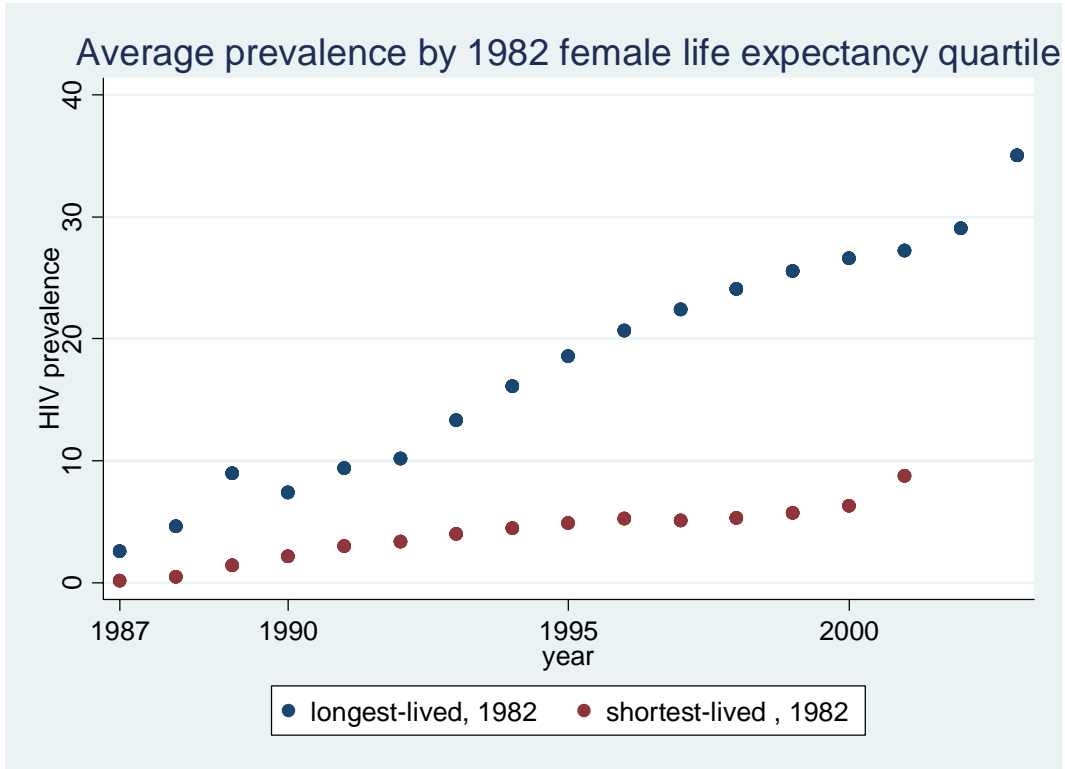


Figure 2.

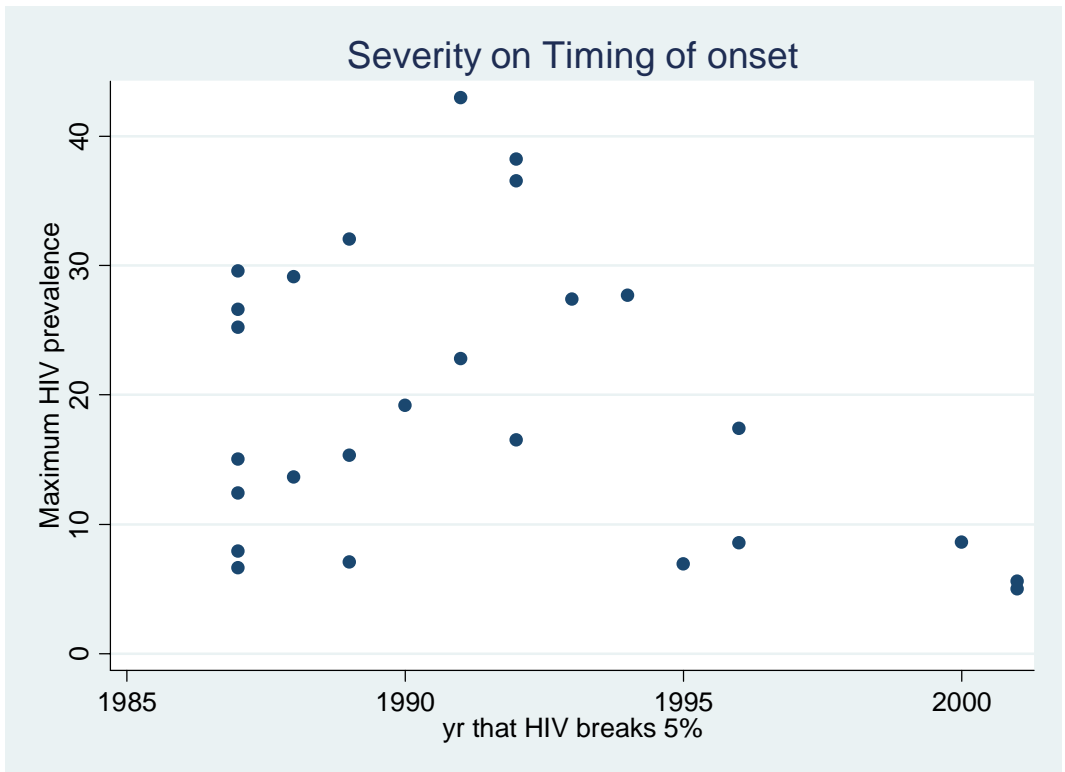


Figure 3.

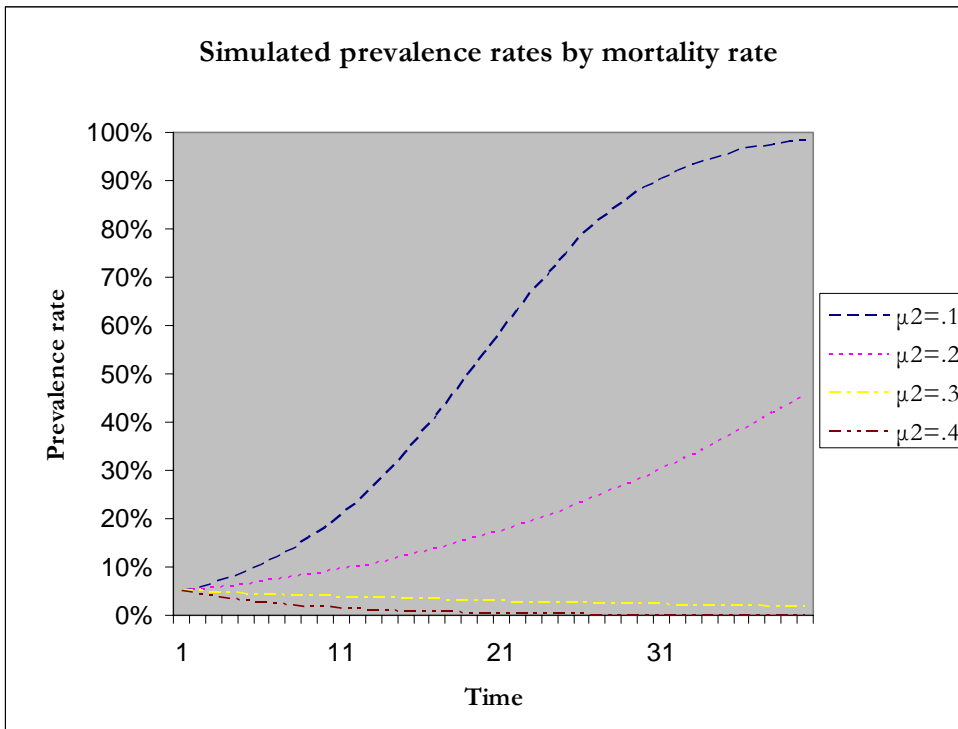


Figure 4. Phase plane diagram of predator-prey model.

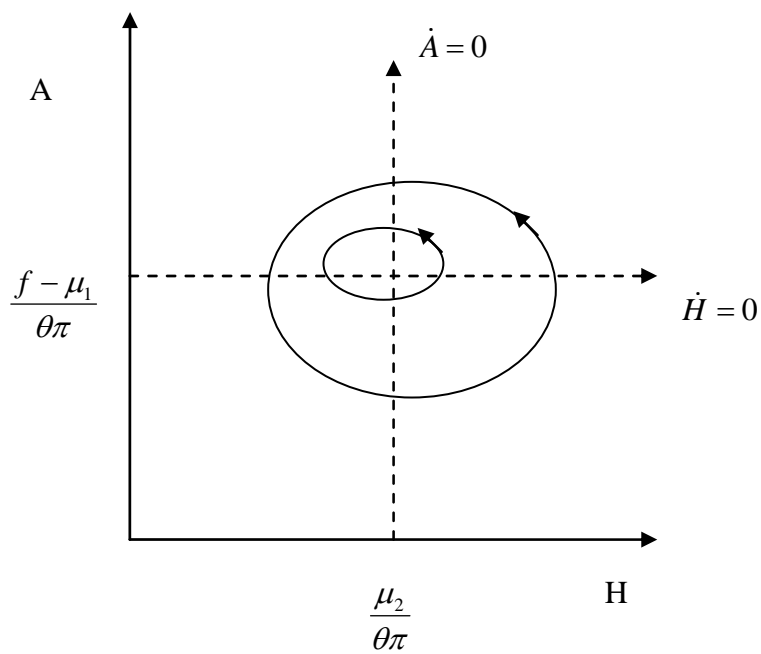
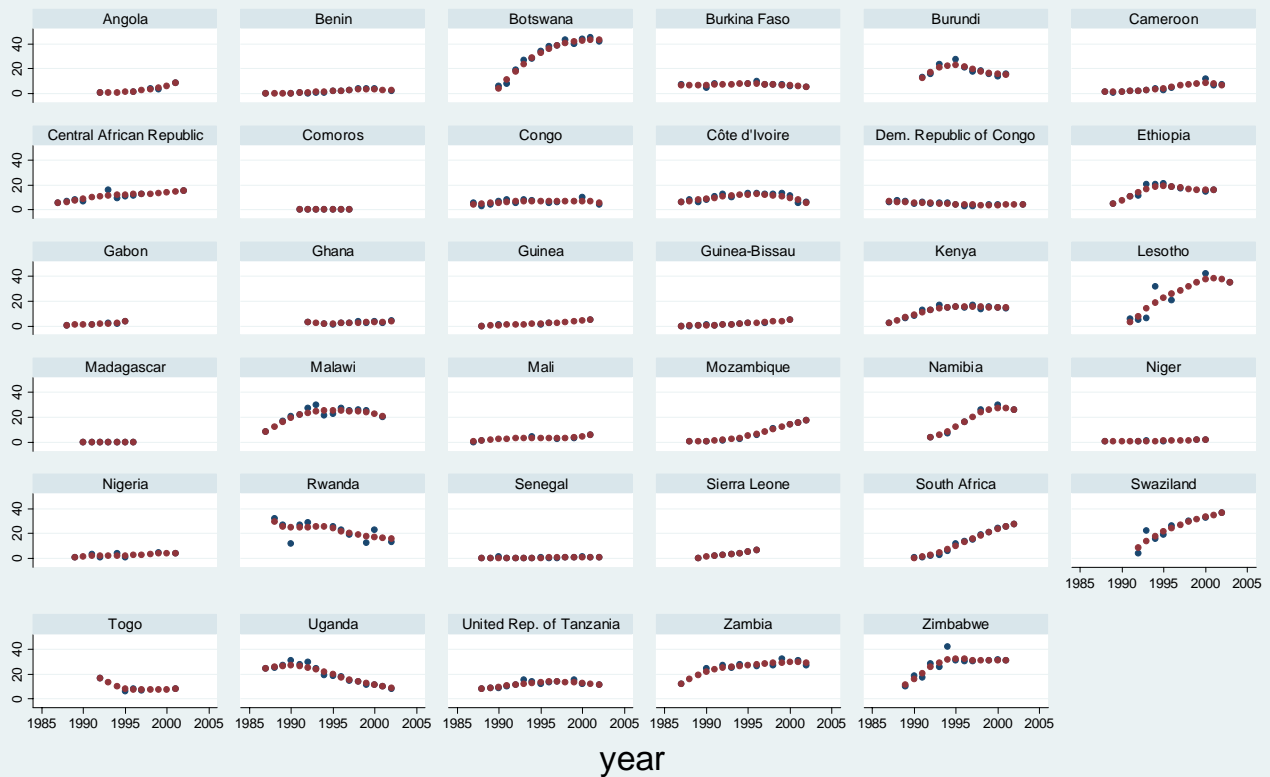


Figure 5. Median & smoothed seroprevalence rates from UNAIDS, by country/year.



● Urban female seroprevalence ● Smoothed prevalence

Graphs by country

Figure 6.

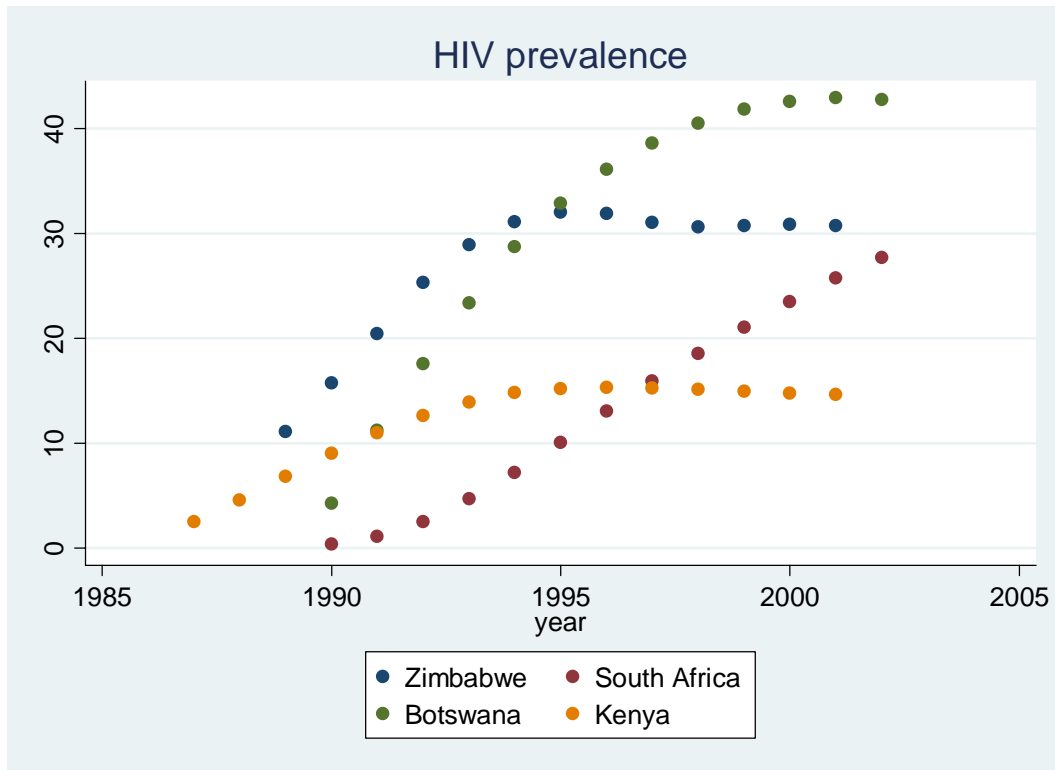


Figure 7.

