

Has Better Health Care Contributed to Higher HIV Prevalence in Sub-Saharan Africa?

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November 2007

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 inadvertently increase HIV transmission, leading to elevated HIV prevalence in the course of an epidemic.

JEL Classifications: I12, H51, O55.

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Thanks to Jean Ensminger, Takeo Hoshi, Ted Miguel, Tom Novotny, Dan Posner, Steffanie Strathdie, and seminar participants at Yale, the Working Group on African Political Economy, and the UCSD Medical Center Clinical Rounds for helpful suggestions, and to Christina Murray, Tara Ramanathan and Jacob Allen for excellent research assistance. All remaining errors are my own.

I. INTRODUCTION

Africa is uniquely burdened by endemic diseases; malaria, yellow fever, tuberculosis, and many other scourges are widespread. Since it is secondary infections that ultimately prove fatal to the victims of HIV, the role of these other diseases in shaping the life expectancy of those infected with HIV/AIDS is central.¹ As we look across Sub-Saharan Africa, we see wide variation in the prevalence of these other diseases, and the burden 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. Figure 1 plots prevalence rates among women at Sub-Saharan maternity clinics from 1987-2003, and shows that women in the highest quartile of 1982 life expectancy consistently have HIV rates which are roughly three times *higher* than rates among the lowest 1982 life expectancy quartile. 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 (Stanton et al 1998, Wawer et al. 1999, Thornton 2006, Gauri & Lieberman 2006). A simple epidemiological model shows that the rate of spread of a sexually transmitted disease is a function of the size of the sexually active infected population. Because secondary diseases in Africa are likely to play a major role in increasing

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.

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.² Therefore high mortality rates among the infected lead to lower rates of transmission of the disease to the uninfected.

The implications of the progress of the HIV/AIDS epidemic for African economic development cannot be overstated. Quite apart from the obvious effects of mortality on labor supply, prevalence rates are often highest among the urban educated elite, and so the impacts of the disease on the human capital stock of African countries has been catastrophic. Although Young (2005) simulates that HIV will lead to an increase in future per-capita consumption for South Africans, Thirumurthy et al (2005) illustrate substantial increases in labor supply as a result of anti-retroviral treatment (ARVs). Hence finding ways to contain the spread of the disease and lessen the burden of morbidity remains one of the major obstacles to African economic development. In what follows we develop a simple and intuitive but previously undiscussed link between health care infrastructure and the spread of the disease. We conclude with a simulation model which examines the epidemiological impacts of the introduction of ARVs in light of this link, and we find that despite the mechanism discussed in this paper, ARVs will have a net positive impact in the battle against HIV/AIDS.

² 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. 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.

II. THEORETICAL MODEL

We introduce a simplified version of the classic SIR model described in Hethcote (2000) and Brauer (1990) to illustrate the role that mortality among the infected and the uninfected plays in driving the dynamics of an epidemic. We denote the number of healthy individuals in the population by H , and the number of individuals infected with AIDS is given by A . There is a fraction θ of the infected individuals who continue to be sexually active with their partners, regardless of whether the partner is infected. If HIV prevalence is P , the prevalence of sexually active infected individuals is thus given by θP . μ^H is the mortality rate among the healthy, μ^A is the mortality rate among those infected with AIDS, and the time derivative is denoted as \dot{H} .³

We make the perfect mixing assumption that the probability of contracting HIV is proportional to the *number* of interactions between the healthy and infected, $\pi\theta AH$, with π representing the probability that an HIV-positive individual passes the disease to an uninfected sexual partner,⁴ and θ the share of infected who are sexually active. Writing population growth among the healthy in the absence of the disease as $f - \mu^H$ (where f is the fertility rate), we have

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

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

³ See Palloni (1996) and Hueveline (2003) for empirical estimations using similar models.

⁴ Specifically, $\pi = 1 - [1 - R(1 - F * E) * S]^N$ where P is the prevalence rate $\frac{A}{A+H}$ 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 (assumed equal to one) is the number of sexual partners. This approach is a simplification of that used in Sweat et al (2000).

This is a standard formulation of the predator-prey model, except that because healthy and infected are measured in the same units, it imposes that ‘one wolf equals one sheep’.

We can first use this model to examine transition dynamics: take a discrete change in mortality among the infected in the previous period, $\Delta\mu_{t-1}^A$. Its immediate effect is on \dot{A}_t ; which equals $\Delta\mu_{t-1}^A A_{t-1}$. This change in the number of infected feeds back into the rate of change of the healthy population in the next period; this effect is given by:

$$\Delta \frac{\dot{H}_t}{H_t} = \Delta\mu_{t-1}^A A_{t-1} \pi \theta, \text{ and so the rate of growth of the healthy population moves in the}$$

same direction as changes in mortality among the infected.

This model has two steady-state solutions; an unstable equilibrium at (0,0) and a stable equilibrium at $(H, A) = \left(\frac{\mu^A}{\theta\pi}, \frac{f - \mu^H}{\theta\pi} \right)$. The steady state is the intersection where

$\dot{H} = \dot{A} = 0$, and healthy/infected numbers will move in a closed-orbit trajectory around this point. The steady-state prevalence rate will be $\frac{A^*}{A^* + H^*}$, which can be written as

$$P^* = \frac{f - \mu^H}{f - \mu^H + \mu^A}. \text{ Steady-state prevalence is decreasing in the mortality rate among the}$$

infected, μ^A . If we consider an underlying change which worsens the mortality rate for healthy and infected symmetrically, it cancels in the denominator, again indicating a decrease in equilibrium prevalence.

III. EMPIRICAL EVIDENCE.

Figure 2 shows that the relationship posited by the theory is present in variation across Sub-Saharan African countries. It gives the scatterplot of peak urban HIV rates (as measured from the UNAIDS STI Surveillance data) on the mortality rate among women in 1980.⁵ The UNAIDS prevalence rates are based on women visiting neonatal facilities, and thus this relationship shows how mortality among women prior to the beginning of the HIV epidemic relates to the severity of the epidemic among women of childbearing age. While the UNAIDS data has been criticized for overestimating prevalence rates in the population as a whole, it remains the only inter-comparable source of prevalence data in African which is not directly biased by the quality of the health care system itself.

The t-statistic on the overall relationship is strongly significant (-3.57). In order to show that this relationship is not driven by comparing the rest of Africa to the wealthier southern countries or the lower-HIV West Africa, Figure 1 shows the fitted line estimated within each of these regions. The slope of the relationship within Southern Africa is steeper and strongly significant, and the relationship within the 15 West African countries is also negative, although the t-statistic is only -1.5. If we remove the five low-mortality outlier countries (Swaziland, Botswana, Lesotho, Zimbabwe, and Liberia), the relationship is significant in the rest of Sub-Saharan Africa, with a t-statistic of -2.6. Thus the raw correlation is broadly negative in Sub-Saharan Africa.

The observation that wealthier countries in Africa have higher HIV rates is not new, and two previous explanations for this relationship have emerged. One is a 'wealth' story which says that individuals with higher disposable income are more prone to the behaviors which spread the disease. A second 'infrastructure' hypothesis posits that rich countries

⁵ The adult mortality rate gives the number (out of 1000) adults at age 16 who will die before their 60th birthday.

feature good transportation systems and are more urbanized, and therefore disseminate the virus more rapidly. While the cross-country sample size is very limited, we can use the 31 Sub-Saharan countries for which data exist to attempt to control for these other factors in a multivariate context.

Table 1 gives the results of this analysis, proxying for the ‘infrastructure’ story using population density and the percentage of roads which are paved, and for the ‘wealth’ story using per-capita PPP GDP. Since the STI data is partitioned into rural and urban clinics these regressions are run separately, and the dependent variable is either the maximum prevalence over the period 1987-2003 (columns 1 and 3) or the average change in prevalence over this period (columns 2 and 4). The infrastructure variables do appear to contribute to a more rapid increase in the disease in urban areas, but not in rural areas. GDP per capita is everywhere negative, indicating *lower* HIV rates in wealthy countries when other factors correlated with wealth are controlled for. In every specification, however, a high mortality rate among women in 1980 is conditionally correlated with lower HIV prevalence and slower growth in prevalence in both rural and urban areas. Cross-country evidence with so few data points cannot conclusively demonstrate causality, of course, but the variation across Sub-Saharan African countries is consistent with a direct effect wherein good health care drives up HIV rates.

Sub-national variation within Kenya provides an opportunity to hold more factors fixed when examining the correlation between HIV and pre-existing health conditions. The 2003 Kenyan Demographic and Health Survey (DHS) combines a health-focused household survey with blood testing data across 376 usable geographic clusters. We calculate prevalence at the cluster level and regress this on a battery of cluster-level average

characteristics. We attempt to use indicators of health and wealth which are sufficiently fundamental as to be exogenous to cross-sectional variations in the severity of the epidemic.

We include controls to try to account for the competing hypotheses. For the ‘wealth’ story, we use % with electricity, % with radio, % who own their house, % own land. For ‘infrastructure’ and ‘density’, we use proximity to Trans-African Highway (1/distance by road), proximity to any paved road, average number of times men report being away from home in the last month, % in cluster who own cars. The variables which proxy for the quality of local health care are: % 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. Demographic cluster-level controls such as % female, % circumcised, average age and education, and rural/urban status are included.

Table 2 presents the cluster-level results. The dominant effect is that male circumcision is strongly correlated with lower HIV. We must take some care in interpreting this result, however, as groups such as the Luo who do not circumcise are anyway closer to the high-HIV Great Lakes region, and are more likely to be involved in high-HIV activities like fishing. Transport characteristics and infant vaccination rates appear to bear no direct relationship with HIV rates. Attributes of wealth are important, but the effects across them is contradictory: land ownership is positively correlated with HIV, but the remainder of the wealth effects appear negative. The health indicators, on the other hand, all indicate that bad sanitation and health are correlated with low HIV.

In order to test the aggregate effects of these variables in explaining a positive relationship between standard of living and HIV rates, the third column of Table 2 presents F-tests over the sum of the coefficients within each channel (all the variables are given signs such that a higher number is an improvement). Because the wealth effects are in

contradictory directions, the summed explanatory power of the wealth variables is low. The collective effect of the health variables is, however, significant, and this result is robust to the exclusion of the wealth, transport, and vaccine effects.

A variety of endogeneity problems are present in an effort to identify causal effects from this kind of cross-section. The obvious endogeneity story for the relationship between health care and HIV would seem to be that health care systems become overwhelmed by the burden of the disease, leading to an overall deterioration in health in areas of high prevalence. Instead, we find the reverse relationship: it is actually those places with the poorest health environment which best avoid HIV. A different source of potential endogeneity in this relationship is internal migration; if HIV sufferers move towards health care and sanitation, this would cause bias in the direction of the effects observed. This story cannot be ruled out in cross-sectional data, but given the penury of many of those with HIV it may be just as likely that urban individuals who become infected move home to the village where they can survive on less. Overall, these empirical results are consistent with an independent causal channel from good health care to high HIV.

IV. ANTI-RETROVIRALS.

The influence of mortality on HIV rates introduces a perverse effect of implementing widespread ARV treatment. If the only effect of ARVs were to extend lifespan, their distribution would lead in a direct causal sense to new infections. In this case we would see an exceedingly thorny policy problem emerge wherein years of life among the currently infected are traded off against years of life among the currently uninfected. However, recent epidemiological research suggests that the decrease in viral loads induced by the distribution of ARVs induces a substantial decrease in the probability of transmission

per sex act, and thereby a change in π . Further, we may expect the mortality effects of ARVs to phase in slowly (because they increase lifespan more for those who were relatively healthy when they began taking them) while the effects on transmission will be immediate. Thus the immediate impact is a decrease in $\pi\theta$, and hence the discontinuous effect is an increase in the rate of growth of the healthy, and a decrease in the rate of growth of the infected. Over the longer term, however, there is a mechanical effect by which decreases in μ^A leave more infected individuals in the population each period, and therefore drive up prevalence. The net effect of ARVs on incidence, even ignoring the complex behavioral response to treatment, is ambiguous because of these competing effects.

In order to get some idea of the relative magnitude of these effects, we use estimates of the parameters of our model from recent field tests in Africa and run a simulation. The term $\pi\theta$ falls out of the steady-state prevalence rate in the predator-prey specification, and so we switch to a somewhat more realistic model in which

$$\begin{aligned}\dot{H} &= -\mu^H H - \pi\theta PH \\ \dot{A} &= -\mu^A A + \pi\theta PH.\end{aligned}$$

This implies that the number of new infections is given by the probability of infection multiplied times the number of sero-discordant couples in the population. It has been widely noted that purely epidemiological models of HIV tend to over-predict the spread of the disease relative to more nuanced behavioral models. The behavioral response to ARVs and the testing that accompanies them is ambiguous (Kremer 1996, Boozer & Philipson 2000, Caplin & Eliaz 2003, Oster 2005, Lackdawalla et al 2006), and so we stick with this epidemiological specification because it allows us to understand the mechanical influence of ARVs prior to the more subtle and unpredictable behavioral responses.

Several studies have been done which examine the discontinuous decrease in prevalence that occurs with the introduction of Highly Active ARV Therapy, or HAART. Work in Taiwan (Fang et al, 2004) and British Columbia (Montander et al, 2006) has found this effect to be on the order of a 50% drop. The mortality effects are not instantaneously experienced, and it seems reasonable to assume that the discontinuous shifts are not driven by behavioral changes, and so we ascribe this change entirely to π^e (specifically to R, the probability of transmission per unprotected sex act). From here we can make the additional assumption that a successful HAART regimen pushes mortality rates back to levels seen among the uninfected, and use our simulation model to study the effects of ARV treatment.

For the probability of transmission we use the midpoint of the ranges given in Thornton (2006), and calculate the overall probability of infection per year in a sero-discordant couple as $1 - \left[\theta P \left[(1 - R(1 - FE)S)^N - 1 \right] + 1 \right]^M$, where $N=82.9$ is the average number of sex acts per year and $M=1.2$ is the average number of partners. 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. 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%. Population growth in the healthy group is 4%. Mortality rates among the uninfected are .0075, and the baseline mortality rate among the infected is .071 (these are the rates measured in Zimbabwe by Lopman et al (2006)). We begin the simulations with a population of 1000 individuals wherein the initial prevalence is 5%.

Figure 3 shows the results of this simulation. Consistent with the experience that such epidemiological model over-predict epidemics, in the baseline scenario this model predicts rapid increases in prevalence over time and predicts almost 100% prevalence after 50 periods. The red dashed line holds the probability of transmission at its baseline values, and introduces only the mortality-reducing effects of ARVs. In this scenario we see a more rapid spread of the disease, with 100% prevalence approached ten periods earlier. The green dashed line carries out the reverse exercise, holding the mortality rate at its baseline value and simulating only the incidence-reducing impacts of ARVs. This induces a dramatic decrease in the spread of the disease, with prevalence remaining below 50% after 50 periods.

The dot-dashed blue line shows the simulation which includes both effects of ARVs simultaneously. We see that, even using a possibly underestimated measure of the impact of ARVs on the probability of transmission (because it is based on aggregate decrease in incidence when HAART is introduced) and using a simulation model which over-predicts the spread of the disease, ARVs slow the spread of the epidemic. This indicates that the mortality effect introduced in this paper is not sufficiently strong as to overwhelm the role that ARVs play in decreasing incidence, and hence ARVs remain net reducers of prevalence.

A note of caution is sounded, however, by asking the following question: what is the behavioral change in θ which would be required to hold prevalence constant at 5% in the ARV and the non-ARV scenario? We can solve for this value numerically, and we find that that without ARVs θ must fall from the 62% measured in the DHS data to 22%. When ARVs are being used, however, the fall must be even larger, to 15%. Why would an intervention that lowered prevalence require a greater behavioral change? This arises because, without ARVs, prevalence is being lowered by mortality among the infected. With the fall in mortality brought about by treatment, the incidence rate required to hold

prevalence steady under an ARV treatment (.18%) is less than half what it was without treatment (.47%). Hence if our criterion of success is holding prevalence constant, we require an even larger behavioral change with ARVs than without. While a low incidence rate is easier to maintain with ARV treatment, lowered mortality implies that for any given incidence, prevalence will be higher under the ARV treatment.

V. 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.

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 illustrated 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. One of the basic points of broadening access to ARVs is to extend lifespan among the infected, which has the mechanical effect of increasing prevalence rates. We should therefore be careful in interpreting high prevalence rates as an indicator of an ineffective response to an epidemic; in some sense it is just the reverse. This ambiguity makes it all the more imperative that we have good data on HIV incidence in Sub-Saharan Africa, which is the true indicator of success in winning the longer-term battle against the disease.

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Table 1: Cross-country regression.

	Urban Prevalence		Rural Prevalence	
	Maximum prevalence	Maximum change in prevalence	Maximum prevalence	Maximum change in prevalence
1980 Population density (people per square mile)	0.090 -(1.67)	0.057 (3.33)**	-0.085 (3.35)**	-0.020 -(1.65)
% of road network paved, 1990	0.177 -(0.90)	0.153 (2.25)*	0.195 -(1.33)	0.035 -(0.50)
1990 p/c GDP, PPP	0.002 -(1.49)	0.001 -(1.80)	0.000 -(0.41)	-0.001 -(1.05)
1980 Adult female mortality per 1000	-0.062 (2.25)*	-0.019 (2.09)*	-0.068 (3.05)**	-0.029 (2.56)*
nobs:	31	28	30	26
R-Squared:	0.38	0.61	0.47	0.29

(t-statistics in parentheses)

Dependent variable calculated from annual median prevalence rate at UNAIDS/STI sentinel testing sites.
All explanatory variables taken from World Development Indicators

Table 2: Sub-national analysis using Kenya DHS.

Analysis of Cluster-Level Prevalence

Cluster-Level Averages:		OLS	Robust t- stat	F-test that sum of coefficients=0	P-score for F-test
Health:	No flush toilet	-0.0530	(1.53)	4.42	.0363*
	Drink riverwater	-0.0245	(1.53)		
	Children with untreated fevers	-0.0347	(2.12)*		
	No prenatal care	-0.0221	(0.52)		
Transport:	Proximity to TA Highway (inverse of distance)	-0.0003	(0.25)	0.53	0.53
	Proximity to paved road (inverse of distance)	-0.0008	(0.25)		
	Number of times hh head away from home	0.0023	(1.37)		
	Own car or truck	-0.0453	(0.76)		
Wealth:	Own house	-0.0044	(0.21)	0.24	0.24
	Own land	0.0397	(2.29)*		
	Electricity	-0.0546	(1.85)		
	Own radio	0.0423	(1.23)		
Vaccines:	DPT vaccine	-0.0396	(0.68)	0.74	0.74
	Measles vaccine	-0.0425	(1.36)		
	Any other vaccine	0.0308	(0.89)		
Controls:	Fraction female	0.2938	(2.54)*		
	Average age	0.0002	(0.11)		
	Fraction circumcised	-0.1703	(6.39)**		
	Average education	0.0008	(0.18)		
	Rural Village	-0.0094	(0.49)		
Observations		376			

Heteroskedasticity-robust regression of cluster-level prevalence on cluster average characteristics, using DHS survey weights.

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

Figure 1. UNAIDS/STI prevalence at maternity clinics against 1982 life expectancy.

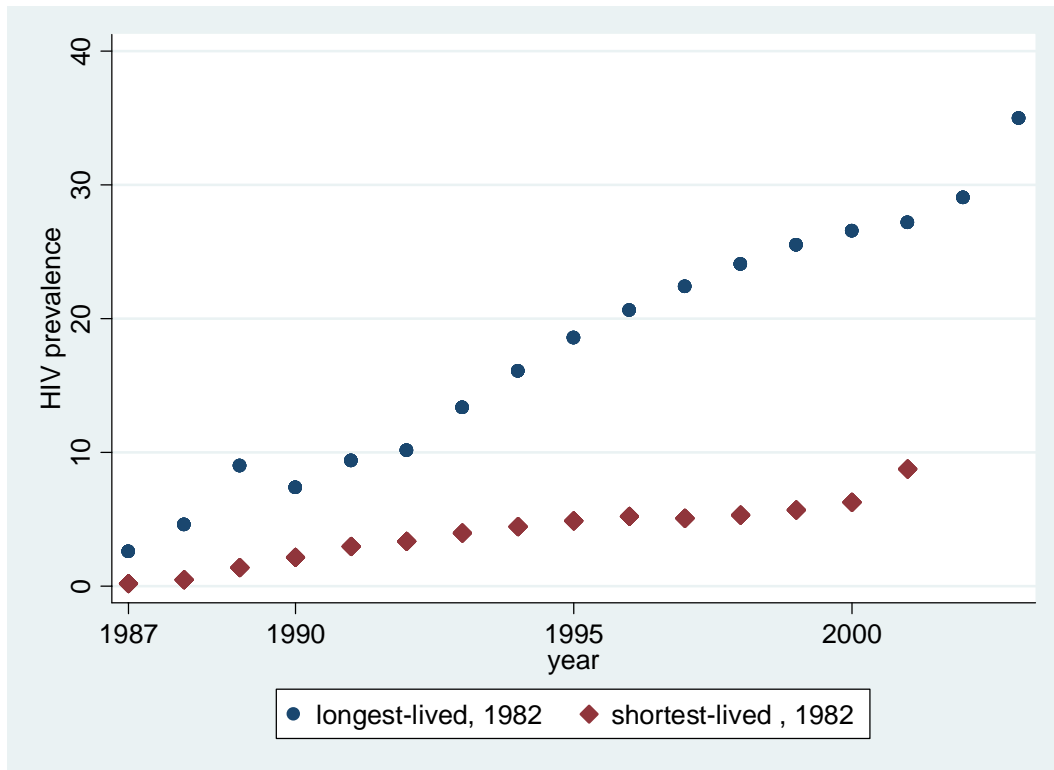


Figure 3. Anti-retroviral simulations.

