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AIDS, "REVERSAL" OF THE DEMOGRAPHIC TRANSITION AND ECONOMIC DEVELOPMENT: EVIDENCE FROM AFRICA

Sebnem Kalemli-Ozcan

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ABSTRACT

Theoretical models of demographic transition imply that fertility declines as a response to a decline in mortality. These models take their cue from the historical pattern of the demographic transition, which suggests that fertility declines follow mortality declines, followed by a rise in human capital accumulation and economic growth. The HIV/AIDS epidemic is a shock to mortality that threatens to reverse this path. Using country, and regional level data, this paper investigates the effect of HIV/AIDS on fertility rates from a panel of African countries during 1985-2000. Results differ depending on the estimation method. Cross-sectional estimates based on country and regional level data from Africa suggest a positive effect of HIV/AIDS on fertility both in OLS and in IV frameworks. Panel estimates show mixed results depending on the HIV/AIDS variable used, yielding a zero effect in most of the specifications. My results contrast with those of Young (2007), who find a strong negative effect of the epidemic on fertility using similar data from Africa and employing a panel estimation. I reconcile the different results by showing that his estimates also turns out to be statistically insignificant, once the standard errors are appropriately clustered.

Sebnem Kalemli-Ozcan
University of Houston
Department of Economics
Houston, TX 77204
and NBER
sebnem.kalemli-ozcan@mail.uh.edu

1 Introduction

"Economics is judged ultimately by how well it helps us understand the world, and how well we can help improve it." Gary Becker.

Nobody doubts that AIDS is the plaque of the 21th century. The impact of the epidemic on economic development is, on the other hand, a fiercely debated issue.[1] The epidemic has altered the patterns of both morbidity and mortality. In the 35 highly affected countries of Africa, life expectancy at birth dropped by 7 to 10 years in the last decade, bringing it down to 35 years left to live for a newborn in Botswana in 2007.[2] Thus, we should start by examining the large theoretical literature that links life expectancy to economic development.

Neoclassical growth models identify two effects. The first order effect of increased life expectancy is to increase population, which will be reversed in the case of HIV/AIDS. Absent behavioral responses in fertility, reductions in mortality increase population, thus reducing capital-labor and land-labor ratios and depressing per capita income. This effect is offset to some degree if increased life expectancy, and more generally, better health, raises TFP and the rate of human capital accumulation. Models in the tradition of Becker and Barro (1988) that endogenize fertility show that fertility may respond to reinforce this latter effect towards higher investment and growth.[3] Hence, declines in mortality could lead to a quantity-quality trade-off where parents have fewer children but invest more in each child. These models suggest that fertility and mortality are positively related and behavioral response in fertility

[1] While most of the researchers find negative effects of the epidemic on economic growth, some find no effect and some even find positive effects. Bloom and Mahal (1997) run cross-country regressions of growth of GDP per capita on HIV/AIDS prevalence and find no effect. Papageorgiou and Stoytcheva (2007) find a negative significant effect of AIDS on income per worker but the effect is small. Werker, Ahuja, and Wendell (2006) instrument HIV/AIDS prevalence by national circumcision rates and show that there is no effect of the epidemic on growth of the African countries. Corrigan, Gloom, and Mendez (2005) show calibration results that imply large negative effects of the epidemic on growth. The results of Lorentzen, McMillan, and Wacziarg (2007) imply significant long-run costs of AIDS on various outcome variables.

[2] A similar picture emerges if we look at life expectancy at age 20 instead of life expectancy at birth, where the latter might be affected from infant mortality.

[3] See Cervellati and Sunde (2007), Tamura (2006), Soares (2005), Kalemli-Ozcan (2002), Boldrin and Jones (2002), Lucas (2002), Galor and Weil (1999), and Ehrlich and Lui (1991) among many others.

can undo and even reverse the initial rise in population size.[4] The HIV/AIDS epidemic has generated a negative shock to life expectancy that threatens to reverse the path to growth laid out in these models. A key question, then, is the following: will fertility responses further reinforce, mitigate or even reverse the disease-induced population declines brought about by the HIV/AIDS crisis?[5]

The empirical literature so far has focused on micro data from a single country or from a small set of countries. For example, Young (2005) using household data on fertility from South Africa and relying on between cohort variation in country level HIV infection, estimates a large negative effect of HIV prevalence on fertility.[6] Young (2007) reaches a similar conclusion using similar survey data from a limited set of countries. On the other hand, recent studies using the newly available HIV data based on individual testing from population-based surveys find no significant effect of the disease on fertility behavior. Using data from 13 countries, for instance, Juhn, Kalemli-Ozcan, Turan (2008) find no significant effect of the community HIV prevalence on the fertility behavior of HIV negative women. Fortson (2008) and Fink and Linnemayr (2008) arrives at the same conclusion for the total

[4]While not directly related to HIV/AIDS, a recent paper by Acemoglu and Johnson (2007) find no effect of life expectancy on level and growth of per capita income. They instrument changes in life expectancy with dates of global interventions in disease prevention. Their results suggest that an increase in life expectancy leads to an increase in population and fertility responses are insufficient to compensate. It may be the case, however, that many of the countries in their sample have not yet completed the demographic transition. In fact, Ashraf et al. (2008) show that the effects of health improvements on income only emerge for half a century or more after the initial improvement in health.

[5]While the focus of this study is the fertility channel, an equally important question is the effect of HIV/AIDS on human capital investment. A large number of papers cover this topic and generally find substantial negative effects. Meltzer (1992) argues that AIDS raises mortality of young adults, which is going to have the biggest effect on the rate of return on educational investment. He claims for a 30 percent HIV positive population like Botswana, there would be a 6 percent reduction in the rate of return to education relative to no HIV. Bell, Shantayanan, and Gersbach (2003), using household survey data from South Africa argue that the long-term economic costs of AIDS could be devastating because of the cumulative weakening from generation to generation of human capital. Fortson (2007) shows children currently growing up in Africa, including non-orphans, will complete 0.3 fewer years of schooling compared to the case of zero HIV prevalence. Yet, another channel can be the effect of the disease on wages, which is harder to study empirically given the lack of wage data for Africa. See Boucekkine et al. (2008) for a model based on both fertility and wage channels.

[6]Kalemli-Ozcan (2009b) shows that this result for South Africa can be overturned if one focuses on the period 1990–1998, where actual HIV data are available as opposed to Young (2005), who focuses on 1961–1998, assuming zero HIV in the pre-1990 period.

fertility rate (TFR) and individual fertility using data from older surveys, respectively.

This paper takes a macroeconomic perspective based on the theoretical framework reviewed above. I investigate the effect of the epidemic on TFR, using country, and regional level data for fertility from a panel of 44 African countries during 1985–2004. The cross-time nature of the data allows me to exploit both between and within variation with the same data set. This is important since the general equilibrium effects might be overlooked by the above cited empirical papers as a result of using different types of variation from different data sets. Hence, I can exploit both cross-section and time series variations from a wide range of countries within the same data set.

I use four different indicators for HIV/AIDS, two of which are available both at the country and at the regional level. Results differ depending on the estimation method. Cross-sectional estimates based on country and regional level data from Africa suggest a positive effect of HIV/AIDS on fertility both in OLS and in IV frameworks. Panel estimates show mixed results depending on the HIV/AIDS variable used, yielding a zero effect in most of the specifications. My results contrast with those of Young (2007), who find a strong negative effect of the epidemic on fertility using similar data from a subset of African coutnries and employing a panel estimation. I reconcile the different results by showing that his estimates also turns out to be statistically insignificant, once the standard errors are clustered at the country level. This is the appropriate clustering since the treatment is at the country level given the country level HIV variable.

The rest of the paper is structured as follows. Section 2 outlines the conceptual framework and also discusses the multidisciplinary literature on HIV/AIDS. Section 3 examines the data. Section 4 presents the econometric framework, identification strategy and the empirical analysis. Section 5 replicates the Young (2007) analysis and reconciles the differences in the results. Section 6 concludes.

2 Conceptual Framework

In this section, I present a simple reformulation of the theoretical models that link fertility to an increase in mortality; specifically a simplified variant of Soares (2005).

2.1 Deterministic Survival

The models of Meltzer (1992) and Soares (2005) rely on the fact that the longer the adults live the more human capital investment they will undertake in themselves, which in turn will lead to a quality-quantity trade-off. To demonstrate their mechanism simply consider an economy inhabited by adult individuals who live for a deterministic amount of time and allocate their time to invest in their *own* education, work, consume, and have children.[7] A fraction β of children born die before reaching adulthood. Adults live for T periods and derive utility from their own consumption, c and from the human capital of their children, h, which is a linear function of their own human capital, H, given as $h = bH + d$.[8] Children have a time cost, b. Parents invest in their own education, e. Hence, adult human capital production is given as, $H = eh_0 + D$, where h_0 is the basic parental human capital inherited from own parents.[9] Parents also care about the number of children, n and how long they live combined in an altruism function, ρ, that multiplies the utility from children's human capital. Hence the utility function and the budget constraint are given as (ignoring the time subscript),

$$\begin{aligned} U &= T\frac{c^{\sigma}}{\sigma} + \rho(n, T, \beta)\frac{h^{\alpha}}{\alpha} \\ TH &= Tc + n + (bn + e)H \end{aligned} \tag{1}$$

To present the static implications of longevity losses in partial equilibrium we use the

[7] This section borrows heavily from Soares (2005).

[8] Note that only the partial equilibrium is being presented here. Economy wide production will be a function of adult human capital, H.

[9] d and D represent innate human capital in the absence of any investments.

first order conditions for maximization to show,

$$\frac{\partial \rho / \partial n}{\rho / n} = \alpha \tag{2}$$

Hence, the individual equates the elasticity of the altruism function with respect to the number of children to the constant elasticity of the utility from human capital of children. Combining above equation with the altruism function and using the implicit function theorem gives,

$$\frac{dn}{dT} = -\frac{\rho n - \frac{\partial \rho}{\partial T}\frac{\partial \rho}{\partial n} n}{\rho n[\frac{\partial^2 \rho}{\partial n^2} - \frac{\partial \rho / \partial n}{\rho}(\frac{\partial \rho}{\partial n} - \frac{\rho}{n})]} < 0 \tag{3}$$

The sign follows from the assumptions that a decrease in adult longevity, T, and an increase in child mortality, β, increase the marginal utility of fertility; and the elasticity of the altruism function is decreasing in the number of children.[10] The way I interpret this model in the context of HIV/AIDS is that the epidemic will cause a decrease in T. Given the representative agent framework, T should be declining as a result of the community HIV, which will lead a rise in fertility.

An alternative modeling strategy will rely on the uncertain survival of adolescents generated by the high mortality risk as argued by Sah (1991), Kalemli-Ozcan (2002), and Tamura (2006). The framework presented in section 2.1 abstracted from this type of uncertain survival in order to focus on the impact of *adult* longevity on the economic incentives faced by the individuals. However, in the context of HIV/AIDS, the uncertain survival of adolescents might have important consequences. Rising adult mortality will shorten the time horizon of parents leading to a quality-quantity trade-off as argued above. It is also plausible that, parents faced with a high mortality environment for young adults, may develop a precautionary demand for children due to uncertain survival and hence may choose to have more children and provide them with less education.

[10] See Soares (2005) for the justification of these assumptions.

2.2 Possible Fertility Responses in the Special Case of HIV/AIDS

I have so far considered HIV/AIDS as a shock to adult/child longevity. However, there are characteristics of HIV/AIDS which suggests that this formulation is overly simplified. First, field evidence suggests that there is a direct biological impact of the disease which lowers the fecundity of infected women, an effect which should be considered separately from the behavioral responses.[11] Fecundity is reduced by HIV infection due to higher rates of miscarriage and stillbirth and high rates of co-infection with other sexually transmitted infections, which may cause secondary infertility.[12]

Second, since it is a sexually transmitted disease, the impact on fertility can come through changes in sexual behavior, assuming individuals have accurate information about the disease. The impact of the disease on sexual behavior in Africa has proven to be much debated topic. Mwaluko et al. (2003), Bloom et al. (2000), Stoneburner and Low Beer (2004), Lagarde et al. (1996), Lindan et al. (1991), Ngwshemi et al. (1996), Williams et al. (2003), Caldwell et al. (1999) all find no change or very small change in sexual behavior. Oster (2005), using DHS data on sexual behavior from a subset of African countries finds that sexual behavior changed relatively little since the onset of the epidemic. Other researchers finds some evidence of risky behavior reductions in Zambia and Zimbabwe such as reductions in multiple partners; see Cheluget et al. (2006), Fylkesnes et al. (2001).

Oster (2005) suggests that the relatively little response in sexual behavior may be in part explained by low levels of knowledge about the disease. Data from DHS surveys show that the percentage of the female population that requests an HIV test, gets tested, and receives results is very small, the mean being 5.7 percent across 10 African countries with an average HIV prevalence of around 15 percent.[13] There is little systematic evidence that

[11] Many African studies, both clinic and cohort based, indicate lower fertility (around 40 percent) and childbearing odds among HIV positive woman. See Lewis (2007) for a recent review of these studies.

[12] It is hard to separate out the biological effect from the behavioral response without data on individual HIV status. In Juhn, Kalemli-Ozcan, and Turan (2008), we take a first step in separating these two effects by utilizing recent rounds of the Demographic Health Surveys which link an individual woman's fertility outcomes to her *own* HIV-status, based on testing.

[13] Countries are Botswana, Burundi, Cameroon, Cote d'Ivoire, Gambia, Guinea-Bissau, Kenya, Lesotho, Mozambique, Niger, Nigeria, Senegal, Sierra Leone, Togo, Zambia. Sentinel surveillance programs (a form

countries with higher prevalence have better knowledge or perceptions of risk, as shown in figure 1. The percentage of 15-49 years old women who know that HIV can be transmitted from mother to child is 38 percent.[14]

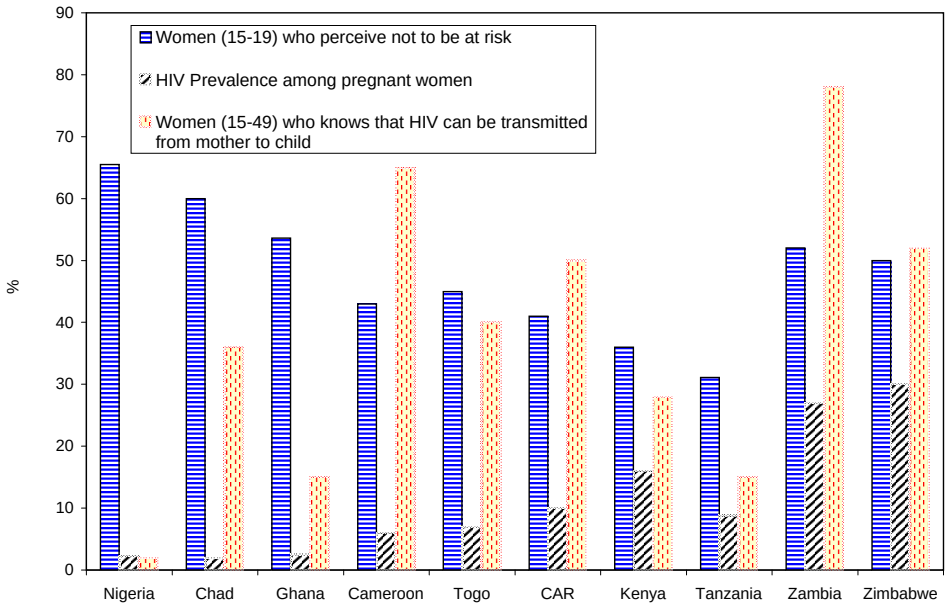
Thornton (2008), however, suggests that knowledge alone may not account for the limited response of sexual behavior in many African countries. Given a randomized experiment in Malawi in which individuals were given monetary incentives to get tested and learn about their HIV status and using randomized incentives as instrument for knowledge, she finds that those with positive HIV status were more likely to purchase condoms. This is a limited amount though, and there was no change in behavior among those with negative HIV status. Oster (2007) also argues along these lines suggesting that shorter life expectancy and lower income could account for the large differences in behavioral response between individuals in Africa and the gay population in the U.S.

Third, regardless of changes in sexual behavior, it may be the case that infected women who know their own status and have knowledge about mother-to-child transmission would want to reduce fertility rather than give birth to infected children. They might also behave in reverse if their desire to have children is high, given the transmission rate of 30% at birth. Again the field evidence on this channel is mixed. Temmerman et al. (1990) find that in Nairobi a single session of counseling—which is common in most African countries—has no effect on the subsequent reproductive behavior of HIV-positive women. Allen et al. (1993) using cohort data from Kigali, Rwanda, find that in the first 2 years of follow-up after HIV testing, HIV-negative women were more likely to become pregnant than HIV-positive women. However, among HIV-positive women, those with no children were more likely to become pregnant than those with children and married women are more likely to become pregnant than unmarried women. The desire to have children among HIV-positive women

of surveillance relates to a particular group) monitoring HIV/AIDS epidemic in Africa are not designed to detect and notify at-risk individuals. They are conducted using anonymous and unlinked blood samples from hospital blood donors, pregnant women attending antenatal clinics (ANC), or sexually transmitted disease (STD) clinic attenders. Thus, those with HIV who are tested will not receive a notification of their status.

[14]Mother-to-child transmission is 30 percent at birth and 3 percent with every month of breastfeeding. One must also note that the questions on knowledge and perceptions are typically asked to those who already heard about AIDS, which constitutes a high fraction.

Figure 1: Risk Perception for Selected African Countries, Demographic Health Surveys, 1994–2000



altogether was 45 percent. On the other hand, Noel-Miller (2003) using panel data from Malawi shows that women who have higher subjective HIV risk perceptions for themselves were less likely to have children.

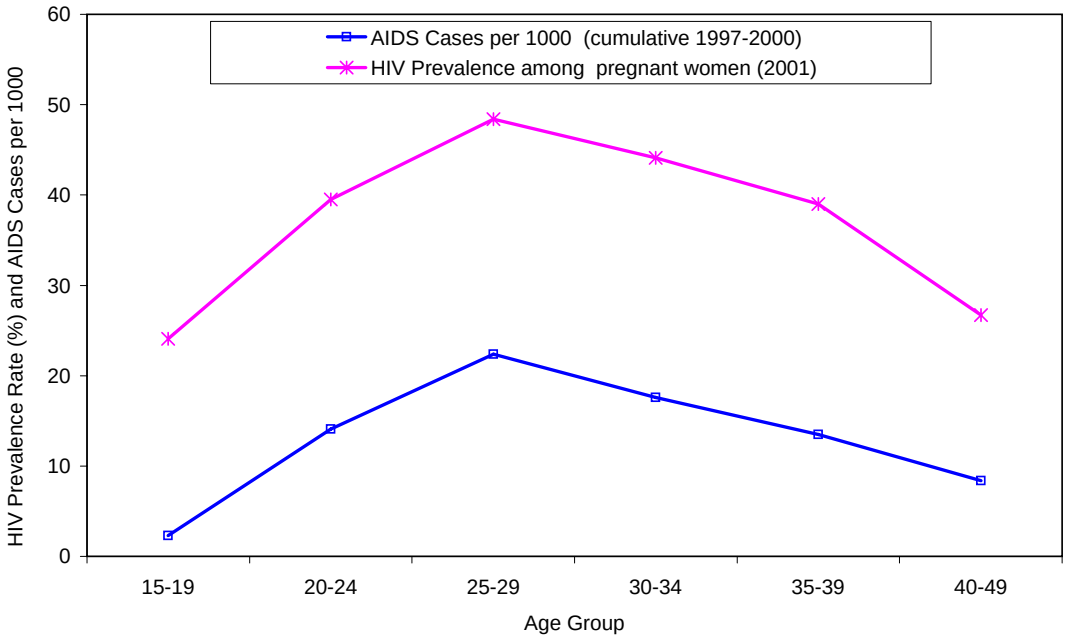
Uninfected people, and people who think they are not at risk, might behave differently. If they know that there is a high level of mortality in their surrounding population, they might reduce their risky sexual activity which will lead to lower fertility as a by-product, or they might increase their fertility along the lines of the outlined models above since the epidemic causes a rise in adult and youth mortality. HIV/AIDS prevalence peaks around age 25−30 in general as shown in figure 2. Figure 3 shows the mortality profile for adults and children as a function of time since infection. In the absence of antiretroviral therapy, the median survival time for adults is 9 years. The estimates also imply that all infected children die by age 12. Figure 3 also shows estimates from Feeney (2001) for Zimbabawe. The probability of a 15 year old dying before age 50 shows a sharp increase since late 1980s, implying high mortality for young adults due to the epidemic during this time period.[15] The higher probabilities (around 50 percent) implied by the household reports might reflect the rapidly rising mortality that is captured in those surveys which are undertaken in 1997 (top x-axis) relative to others that are done earlier. These also reflect the subjective probabilities of the family members who experienced the deaths due to AIDS very closely.

Overall there might be various responses of fertility to the HIV/AIDS epidemic, which might lead to a reduction in fertility or an increase. It is useful to summarize these effects in the table below:

A reduction in age-specific fertility rates among HIV positive woman due to the biological responses may serve to reduce total fertility in a high HIV prevalence country in the absence of any behavioral response from the uninfected woman. Behavioral response from the infected women (if they know their own status or have high risk perception) might also cause a

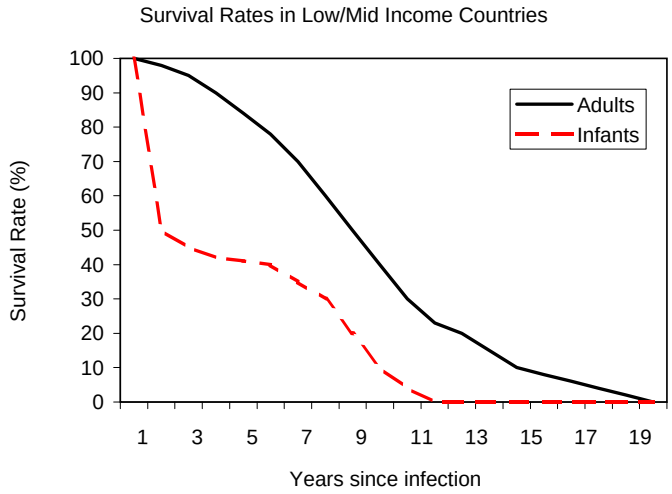
[15]This probability is defined as q_{15}^{35} in demographic terminology. Records from vital registration, reports from households and reports from surviving siblings all show an upward trend. Feeney (2001) argues the discrepancy between registered deaths and sibling reports comes from the fact that the former is adjusted for underreporting and the latter is not.

Figure 2: HIV/AIDS by age, Botswana

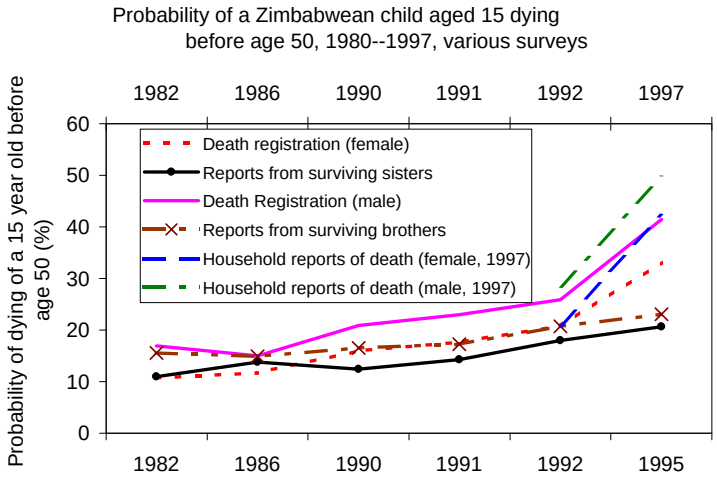


Data: Botswana 2001 HIV Sero-Prevalence Sentinel Survey among pregnant women.

Figure 3: Survival for Adolescents



Data: UNAIDS Reference Group, 2002.



Data: Feeney, 2001.

Table 1: Possible Fertility Responses (assuming individuals have a basic level of knowledge about the disease)

Behavioral Response:		
	HIV – women	HIV + women
Know own status and risk perception low	– or +	NA
Know own status and risk perception high	–	– (maybe +)
Do not know own status and risk perception low	– or +	– or +
Do not know own status and risk perception high	– or +	– or +
Biological Response:	0	–

reduction in fertility. Last but not least uninfected womens' fertility might also decrease due to a reduction in risky sexual behavior. Put it differently, for fertility to increase as a result of the epidemic any positive behavioral response of uninfected women have to overcome the negative biological and behavioral responses.

3 Data: Sources and Issues

3.1 Country Level

Fertility:

I use country level data on total fertility rates (TFR) both from World Bank, World Development Indicators and from Demographic Health Surveys (DHS).[16] WB data are available

[16]The World Bank uses UN World Population Prospects for every 2 years and update the UN data with the latest survey data such as DHS, MICS and so on. UN data comes from the countries vital registration system.

for 44 countries and ten years between 1985–2004. DHS data on fertility rates per woman ages 15–49 are available for 34 countries, where most countries has only 1 or 2 surveys. Only 3 out of 34 countries have 4 surveys, 10 countries have 3 surveys, 10 countries have 2 surveys and the remaining 11 countries has 1 survey. Survey years fall between 1986–2004. TFR is the sum of age-specific fertility rates; it is an approximation for the average lifetime fertility of women. I also use data on desired fertility rate per woman ages 15–49, available for 34 countries, from DHS. Details of the variables and a full list of countries and survey years are provided in the appendix.

Figure 4 plots TFR from DHS for Kenya, a high prevalence country, and shows that after more than a decade of rapid decline, the total fertility rate actually increased starting in the late 1990s. Westoff and Cross (2006) find the increase in fertility in Kenya is most pronounced for the least educated group of women. They also find a significant increase in the percentage of women who report wanting more children for each age group. Similar data from 10 other countries show either an uptick for fertility, such as in Nigeria and Mozambique, or a stall in fertility transition, such as Uganda and Cote D'Ivoire, as shown in figure 5.[17]

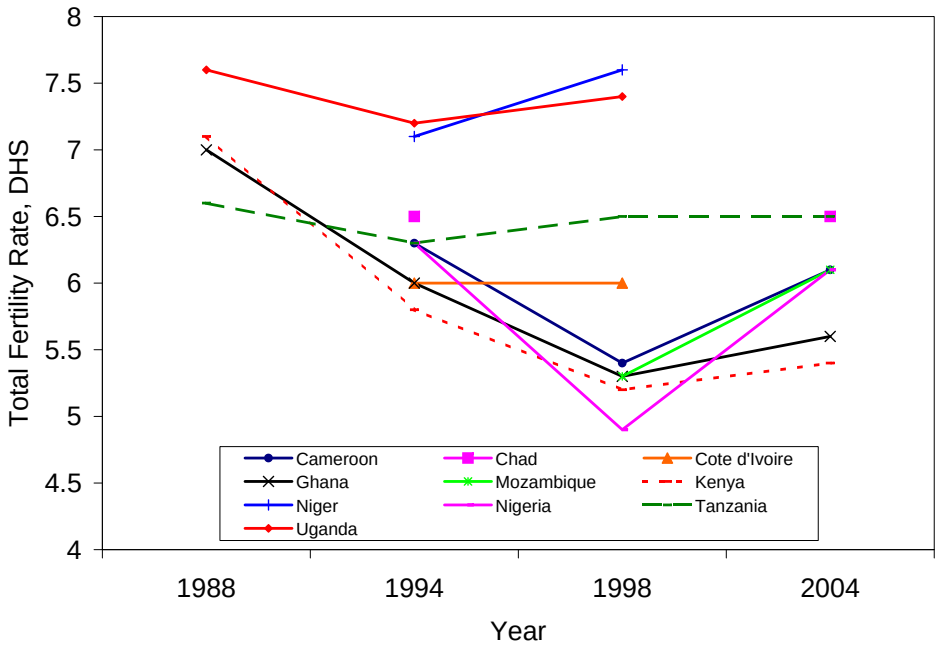
HIV/AIDS:

I use four different indicators for HIV/AIDS at the country level, none is perfect and all have different problems. For AIDS, I use data that come from UNAIDS/WHO, Epidemiological Fact Sheets (2003). These are the number of reported AIDS cases available for each country in every year between 1985–2004. I multiply the number of reported cases by 100,000 and divide by the country's population in each year, to obtain rate per 100,000 per country per year. According to UNAIDS, AIDS case reports come from surveillance systems of varying quality. Reporting rates vary substantially from country to country and low reporting rates are common in developing countries due to weaknesses in the health care systems. Hence there can be systematic biases such as in countries with worse medical institutions (which is probably correlated with other country characteristics) underreporting will be worse. AIDS case reporting provides information on transmission patterns and levels of

[17]Each countries survey year is on or around the dates shown on the x-axis.

Figure 4: Fertility in Kenya, Demographic Health Surveys: 1989, 1993, 1998, 2003

Figure 5: Fertility in Africa, Various Demographic Health Surveys



infection approximately 5-10 years in the past, limiting its usefulness for monitoring recent HIV infections. Despite these caveats, AIDS case reporting is useful in estimating the burden of HIV-related mortality.

For HIV, I use three different indicators. First, I use data on HIV prevalence rates among pregnant women that are from the U.S. Census Bureau, HIV Surveillance Database (2005). UNAIDS/WHO also provides similar data. This is the indicator that is used by most researchers. Both U.S. Census and UNAIDS databases collect regional estimates of HIV/AIDS prevalence since the early 1980s. The main indicator for the epidemic is the percent HIV-1 incidence among pregnant women for each country and year. However, these estimates are in general very high. Representativeness of these estimates for the general population is also debatable since they are based on pregnant women and high risk groups, which in turn is the main reason for these inflated estimates.[18] More recently, DHS started providing results from population-based HIV testing. These new estimates are much lower than the UNAIDS and U.S. Census estimates.[19] The new population based DHS estimates are only available for a limited set of countries for their latest survey though and hence do not provide enough information about variation over time, as the HIV estimates from the Surveillance Database. On the other hand, the time series variation in these prevalence rates from the Surveillance Database of U.S. Census and UNAIDS is far from perfect. UNAIDS (2006) notes that it is not possible to use previous reports to compare prevalence over time. Using the U.S. Census HIV surveillance database suggests that HIV rates are flat or falling over the 1990s in virtually all countries in Africa, which seems inconsistent with the casual observation. A close inspection of these estimates shows that there is considerable year to year variation which calls into question the reliability of the time variation in these data. It has been suggested by many that selection of locations that the estimates are collected are changing over time. Based on these problems, Oster (2007) develops a methodology to estimate HIV prevalence over time from mortality data. To avoid the problem of lack of

[18]See Timberg (2006) and McNeil (2007).

[19]See Juhn, Kalemli-Ozcan and Turan (2008) for a comparison of the various estimates.

official mortality statistics for Africa she takes advantage of sibling mortality histories in the DHS. She has HIV estimates only for 9 countries since mid-1980s though. I use her estimates as a second indicator for HIV.

As a third indicator for HIV, I use the *projected* HIV from the U.S. Census Bureau, International Programs Center. The International Programs Center uses Estimation and Projection Package (EPP) from WHO/UNAIDS to project adult HIV prevalence among 15–49 year old from U.S. Census Surveillance data between 1985–2004. While EPP can be used in all countries with sufficient surveillance data, it is specifically recommended for countries with "generalized epidemics." Generalized epidemics are those that have broken out into the general population or consistent HIV prevalence at over 1 percent in low risk individuals. The proxy for low risk individuals is women attending antenatal clinics. Thus, the inputs to EPP in countries with generalized epidemics are the same surveillance data on HIV prevalence among pregnant women. EPP estimates the trends over time of HIV prevalence by fitting an epidemiological model to data from urban and rural sites.[20] Although EPP model fits a somewhat flexible curve to a not so long time series, the modeling is still an issue of concern given the dynamic nature of the epidemic.

The correlation between different indicators is around 70 percent on average. The indicators in general suffer from different biases. Classical measurement error is one but there can also be other errors that are not classical. For example, since most of the indicators are based on estimates from antenatal clinics, the measurement error might be correlated with the population attending the clinics, which itself might be correlated with fertility. In case of AIDS the bias is in general downwards since estimated mortality is almost always lower than what is should be.

I also use data on *perceptions*, specifically the variable "know someone died of AIDS."

[20]It chooses a set minimizing least squares and projects future course based on fitted parameters, such as a parameter for the start year of the epidemic; one for the force of infection (how explosive the epidemic is in its initial stage); one for the fraction of new entrants to the population going into to the at-risk category (a parameter largely determines where the epidemic levels off); and one for the recruitment (a high value means people are brought into the at-risk population as people die of HIV, thus helping to sustain the epidemic at a higher level).

The data on the percent female who know someone personally who has the virus that causes AIDS or has died of AIDS are from DHS. This is the ideal measure for the purpose of this paper however since this question has only been asked in the most recent surveys the data are available only for 22 countries whose survey years fall between 1993–2004.

Other Controls:

All other controls such as female schooling, child mortality, contraception, and GDP per capita are taken from World Bank, WDI, and from DHS. The details of these data are provided in the appendix.

3.2 Regional Level

Fertility:

I use data on regional total fertility rates from DHS. They are available for 71 regions from 14 countries, whose surveys years fall between 1988–2004. A full list of regions is provided in the appendix.

HIV/AIDS:

The data for regional HIV rates come from U.S. Census Bureau, HIV Surveillance Database (2005) and available for 40 regions from 13 countries between 1985–1990. The overlap between the regional fertility rates and HIV rates give us 32 regions from 12 countries.

4 Econometric Framework, Identification Strategy and Empirical Analysis

4.1 Framework and Identification

Theoretical models of the demand for fertility have the following empirical predictions: 1) increased education of women raises the cost of childbearing and reduces fertility; 2) reduced child mortality, assuming the demand for surviving children is price inelastic, is associated

with a decline in fertility; 3) increased income per capita increases demand for children since they are normal goods. Thus, I control for these determinants, that are shown to be significant in the other empirical studies,[21] in a regression of total fertility rate on the indicators of HIV/AIDS. I estimate Ordinary Least Squares (OLS) regressions of the following form, using both country and regional level data:

$$TFR_i = \alpha + \beta HIV/AIDS_i + \mathbf{X}_i'\gamma + \epsilon_i, \tag{4}$$

where TFR_i is the total fertility rate for country i, $HIV/AIDS_i$ is the indicator for HIV or AIDS for country i, $\mathbf{X}_i$ is a vector of other covariates, and ϵ_i is a random error term.[22] The coefficient of interest is β, the effect of the epidemic on fertility. Recall that four different indicators for HIV/AIDS are used: I use AIDS cases per 100,000 per country per year from UNAIDS. I will call this variable "AIDS." Next, I use the HIV prevalence rates among pregnant women that are from the U.S. Census Bureau. I will call this "HIV." I also use Oster (2006) estimates, which I will call "HIV-Oster." Finally, I use the projections of the U.S. Census Bureau, which I will call "HIV-EPP."

Notice that the regression presented in equation (4) only exploits variation *between* countries, using averaged data over time, i.e., it is a "between regression." A "within regression" framework to identify the parameters using only *within* country variation over time is preferable since this framework controls unobserved country heterogeneity. However, as summarized above the information on the time variation is noisy hence I am hesitant to rely solely on within country time variation by using first differences or country fixed effects, which further exacerbates the measurement error. I will present results for both frameworks to get a better insight.

Total fertility rates were falling in almost all the African countries before the HIV/AIDS epidemic. Thus, I run a panel regression both with country and time fixed effects. I also

[21] See Schultz (1997) for an example.

[22] This regressions is also run at the regional level with country dummies included, i.e., for region r: $TFR_r = \alpha_i + \beta HIV/AIDS_r + \mathbf{X}_r'\gamma + \epsilon_r$, where α_i is the country dummy.

run the same regressions with a general time trend and country specific time trends. The "within regressions" are of the form:

$$TFR_{it} = \mu_i + \lambda_t + \psi HIV/AIDS_{it} + \mathbf{X}'_{it}\theta + \varepsilon_{it}, \tag{5}$$

where TFR_{it} is the total fertility rate for country i at time t, μ_i is the country fixed effect, λ_t is the time fixed effect, $HIV/AIDS_{it}$ is one of the four indicators for HIV/AIDS, $\mathbf{X}_{it}$ is a vector of other covariates, and ε_i is a random error term.

The econometric framework presented in equations (4) and (5) posits an endogeneity problem since HIV/AIDS is related to sexual behavior and marriage markets, both of which are independently related to fertility. Areas with initially higher levels of sexual behavior will have higher HIV rates and they may also have higher rates of fertility. Also there are compelling reasons to believe that HIV infection is higher in areas with greater population density and economic activity. Then, country level HIV rates suffer from an omitted variables bias since countries that are the most economically active may have both higher infection rates and lower fertility, the latter being due to possibly the higher cost of women's time. Failing to control any variable that is negatively correlated with the epidemic such as female education will cause a downward bias. There might also be a bias due to simultaneity that are not captured by the fixed effects.

The conditioning variables should take care of the large part of the effect of the differential development levels. The "within regressions" are immune to the unobservable factors that are time-invariant such as religion, climate and culture. However, individuals may start taking less risks as a result of the epidemic over time or across places, which will bias not only "between" but also "within" estimates. In the case of the "within" estimates the bias works against finding a positive effect of the disease on the fertility behavior though. If people start taking less risks (more condoms, fewer partners or abstaining) because of HIV/AIDS then fertility will decrease as a by-product, and hence a negative relation between fertility and HIV/AIDS will be the result. This would be true assuming that despite changes in sexual activity HIV rates remain high. Ultimately, it is plausible that, societies which lower

their level of risky sexual activity are likely to experience declines in HIV rates and in fertility levels. I do not expect this ultimate effect effect to be dominant for the time period that this paper is concerned with.[23]

For the "between" regressions, I will undertake a falsification exercise that investigates the relationship between pre-AIDS fertility and current HIV. To further deal with the problem of endogenity, I will follow Oster (2007) and instrument HIV/AIDS by the distance to the origin of the epidemic, which is Democratic Republic of Congo. Oster (2007) argues that two factors determine HIV prevalence within a given area are the speed at which the prevalence increases and the date at which the virus is introduced. The speed of increase, in turn, is determined by sexual behavior and the viral transmission rate. Hence the viral transmission rate or the arrival date of the virus, are potentially plausible instruments. She focuses on the virus arrival date. However, she also argues that using date directly is problematic since testing early in the epidemic is very limited and hence it is likely that the first date that the virus is observed is correlated with sexual behavior, which is also related to fertility for my case. She uses distance as an alternative since she argues that if the virus takes time to travel, moving from person to person, areas further from its origin should have lower prevalence on average. She uses the longitude and latitude of each DHS survey cluster to calculate the distance of cluster to the center of Congo (middle of the country) since the virus is originally observed on both sides. In a similar fashion, I use the distance from the capital city of each country in my sample to the capital city of the Democratic Republic of Congo, which is measured as the distance between the center of the capital cities.[24] In the regional regressions, I use the distance of each region to the center of the epidemic as calculated by Oster (2007).

For the instrument to be valid, it must be correlated with the HIV/AIDS but uncorrelated with the fertility rate, except through the variable of interest that is included in equation

[23]If sexual behavior declines for some other reason than HIV/AIDS, then this will lead a positive association between fertility and the epidemic since both will decline as a result. One cannot rule this out.

[24]I also use the alternative instrument of circumcision as used by Werker et al. (2006). Circumcision might be less appropriate for the case of fertility, since it is highly correlated with ethnic group, which is likely to be correlated in turn with the fertility behavior.

explaining fertility. The most obvious way in which distance to the origin of the epidemic might systematically affect the fertility rate—other than via HIV/AIDS— is through its correlation with geographic and/or socioeconomic variables. Having controlled for these factors, as will be shown in detail below, it seems plausible to argue that distance to the origin of the epidemic will be otherwise unrelated to fertility.

4.2 Descriptive Statistics

Table 2 shows the mean, maximum, minimum, and standard deviation of the dependent and independent variables. Fertility rates vary from 2 children to 8 children with a mean of 6 children. For AIDS, the most affected country has prevalence that is 160 times higher than that of the least affected country. The difference in the HIV prevalence between the highest and lowest prevalence country is 250 times in UNAIDS and U.S. Census data but only 10 times in the Oster (2006) estimates. GDP per capita moves between 100 and 6000 dollars. The remaining variables also show extensive variation.

4.3 AIDS, HIV and Fertility: Between Regressions

Table 3 reports the results of the OLS estimation of equation (4). I match the years of TFR data, i.e., 1985, 1987, 1990, 1992, 1995, 1997, 2000 to that of HIV/AIDS indicators before averaging. Columns (1)-(3) of table 3 uses the average values of dependent and independent variables over $1985-2000$ and show that the first two indicators of the epidemic, namely, AIDS and HIV, are positively significant at 1 percent and at 5 percent level respectively, whereas the other indicator, i.e., the HIV-EPP is not statistically significant. Using both indicators AIDS and HIV in a horse race, leads a positive significant coefficient on AIDS (0.23 with a standard error of 0.09) and a negative insignificant one on HIV. Female schooling measured as secondary school enrollment is negative and significant at 1 percent level, while GDP per capita is insignificant, a result which is probably due to the high correlation between

GDP per capita and female schooling.[25] Another important control is infant and child mortality, which is positive and significant at 1 percent level. This control partly accounts for the variation in fertility rates due to youth mortality risk from other competing diseases such as Malaria.

To test for outliers, figure 6 shows the partial correlation plot for the regression shown in column (1), hence the slope of the solid blue line is 0.14. If I omit Congo, Rep. the coefficient goes down to 0.10 but stays statistically significant at 5 percent level as shown by the dashed red line.

Columns (4)-(6) shows similar regressions using data on fertility from DHS. The fertility observations are averaged over the survey years, which change between 1987–2004 and from country to country.[26] The point estimates for HIV and AIDS are larger and significant at 1 percent level for fertility rate. The coefficient estimates for HIV-EPP are insignificant as before and hence not reported. I also use Oster (2006) HIV estimates (not shown due to space limitations) that deliver a positive but borderline significant coefficient. However there is only 9 countries in this estimation and hence the large standard errors are not surprising.[27]

Finally column (6) uses data on perceptions about the epidemic instead of the actual prevalence rates. Women who know someone who died of AIDS, are the ones who should react most by changing their fertility behavior. As shown this is indeed the case. The data on the percent female who know someone personally who has the virus that causes AIDS or has died of AIDS are from DHS and averaged according to the available survey years. In spite of the limited number of countries there is a strong positive association between perceptions about the epidemic and the fertility behavior as also shown in figure 7.[28] The variable "know someone who died of AIDS" can by itself explain 20 percent of the cross-country variation

[25] Using other measures of female schooling yield similar results.

[26] See Appendix for details on survey years.

[27] I also used desired fertility from DHS obtaining very similar results and hence I do not report them but they are available upon request.

[28] I have also tried interacting the perception variables with the actual prevalence rates. However due to the high correlation between the HIV/AIDS prevalence rates and the perception variables and also due to the fact that I have limited number of countries the results of those interaction regressions are weaker.

Figure 6: Partial Correlation Plot for AIDS and Fertility

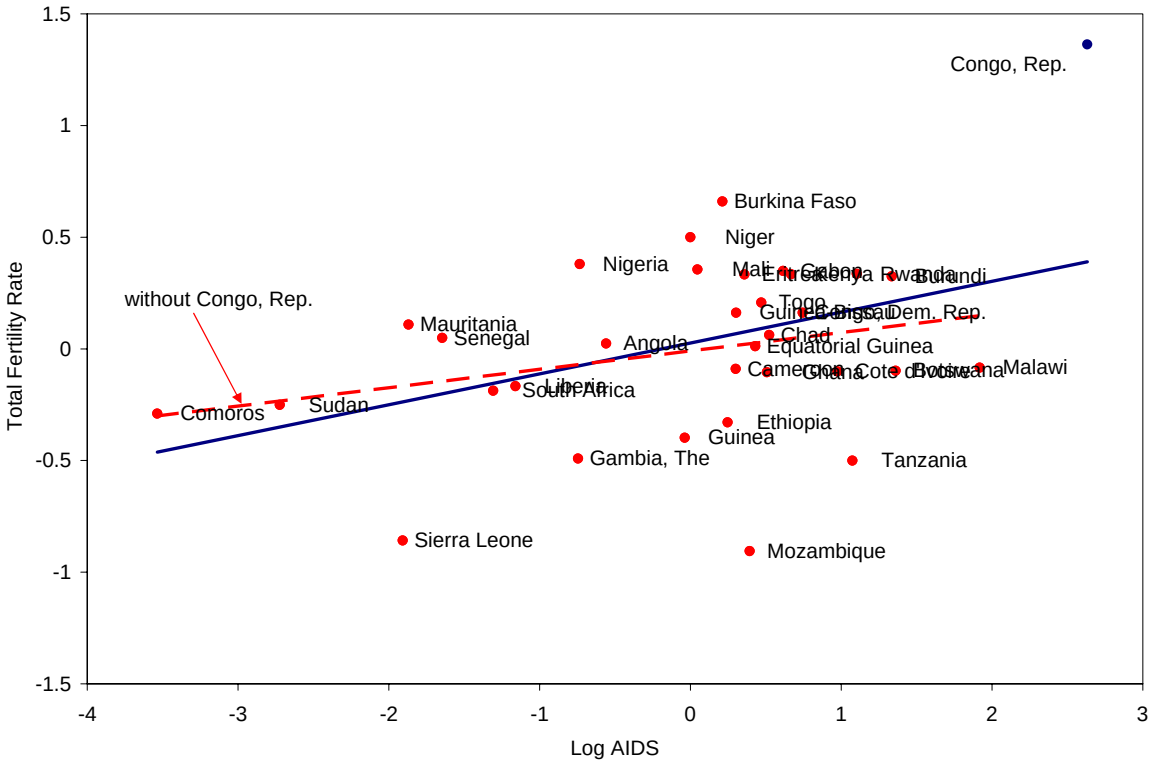
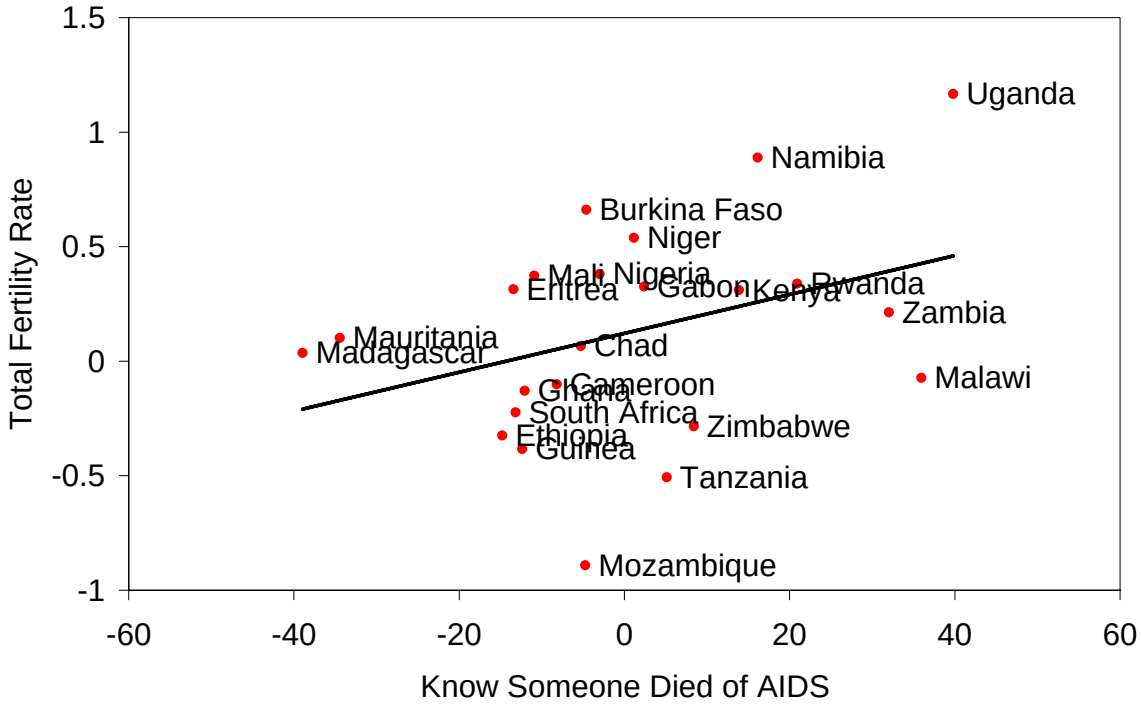


Figure 7: Partial Correlation Plot for Perceptions of the Epidemic and Fertility



in the fertility behavior.[29]

The indicators of HIV/AIDS are used in logs following Oster (2007). She argues that in her first stage estimations, where she regresses HIV on distance, log HIV prevalence on linear distance provides the best fit (most linear) as shown by simulations. I run similar first stage regressions, where log HIV provides a better fit. Hence the non-IV OLS is also used in this functional form for comparison. Although using the log of HIV/AIDS makes the quantitative

[29]This is the partial R^2.

interpretation harder, it has several econometric advantages such as dampening the outliers and making the estimated coefficient immune to the scale effect due to underreporting, assuming underreporting is similar across countries. There might be a concern in using the log form though since log specification in principle compares the countries that have any AIDS to those that don't. I would argue that this is not a serious concern in the case of Africa. First of all due to averaging over time, I do not have any zeros in HIV/AIDS; the only zeros for the initial years of epidemic for few countries are averaged out. Second of all, the sample I am using are composed of countries that are classified as "generalized epidemic countries" with the exception of Comoros, Madagascar, Mauritania, and Sudan. The results are robust to excluding these four countries. The results are also robust to, even stronger, using the non-logged proxies for HIV/AIDS and available from the author upon request.

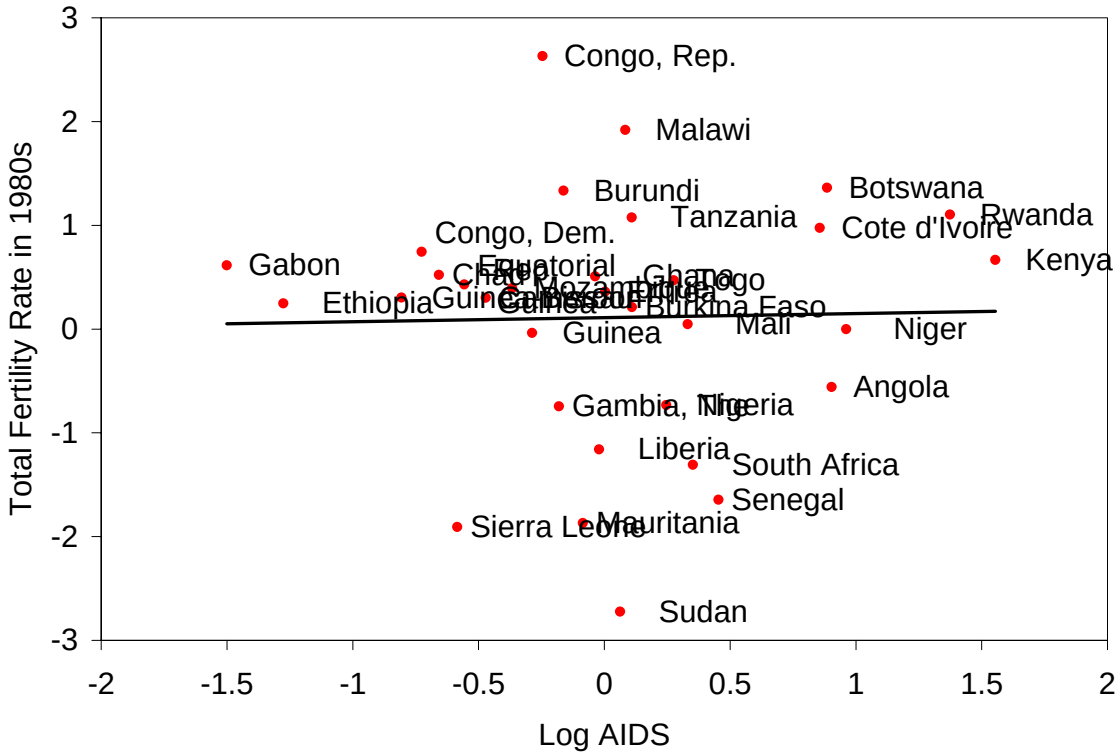
To deal with the possibility that unobserved country heterogeneity are driving the results, I will undertake a falsification exercise. Table 4 represents the results from a regression, where I regress fertility rates from 1980s, on the current HIV/AIDS, averaged over 1995–2004 and the other controls. Fertility in 1980s is the average of rates in 1980, 1982, 1987. There is no statistically significant relationship between current HIV/AIDS and fertility in 1980s as shown in columns (1) and (2) and further in figure 8. The 95% confidence interval implied by the estimate and the standard error in column (1) does not include the estimates from table 3, however this is not the case for the column (2). Thus, this exercise suggests that time invariant unobserved country heterogeneity is not driving the results, at least in the case of AIDS indicator.

4.4 Country Level HIV and Fertility: Within Regressions

Table 5 reports results of the OLS estimation of equation (5) using country level data. Standard errors are clustered by country to deal with the possible serial correlation among residuals.[30] For each of the three indicator of HIV/AIDS both pooled regression results

[30]I also perform Weighted Least Squares (WLS) panel regressions; where all observations are weighted in the second step with the inverse of the estimated standard deviations from the first step. Weighting by

Figure 8: Falsification Plot for AIDS and Fertility



with a common time trend (that captures the declining trend of fertility in the absence of HIV/AIDS) and "within" regression results with both country and time fixed effects are shown in columns (1)-(6).[31] AIDS is positively significant both in the pooled and in the fixed effects regressions, HIV is only significant in the pooled regressions, and as before HIV-EPP is not significant. All other control variables yield similar results as before.

I also use country-specific time trends as shown in the last 3 columns of table 5. The first two indicators gave insignificant results, however the third indicator, that is HIV-EPP yields a negative significant result. Recall that this variable never turned out to be statistically significant up until this specification. I also run a regression similar to a diff-in-diff specification such as I regress change in fertility from 1990 to 2004 on the change in HIV/AIDS from 1990 to 2004, obtaining a positive significant result for AIDS and a negative insignificant result for HIV.

Overall, the mixed results can be explained by the use of within country time variation as the main identifier. Figures 9, 10, and 11 show time series path of HIV data from three countries, where there is a lot of noise. Exploiting noisy variation can lead to different results depending on different specifications.

4.5 Alternative Explanations

The finding of a positive effect of AIDS on fertility and a negative, though insignificant, effect of HIV on fertility constitutes a puzzle. This section tries to address some alternative explanations for the positive association between AIDS and fertility. One story that comes into mind is a shift in the population age distribution. If older women are dying because of the epidemic, the total fertility rate will increase simply due to the fact that younger women have more children. Figure 12 takes a stab at this by looking at the data for Kenya—a high prevalence country. The data show that, in spite of the high mortality, the population age distribution has not shifted much. This is not surprising since AIDS is mostly kills prime-age

country's population or log population yields similar results.

[31] I also experimented with a common non-linear quadratic and cubic trend obtaining similar results.

Figure 9: Ethopia HIV

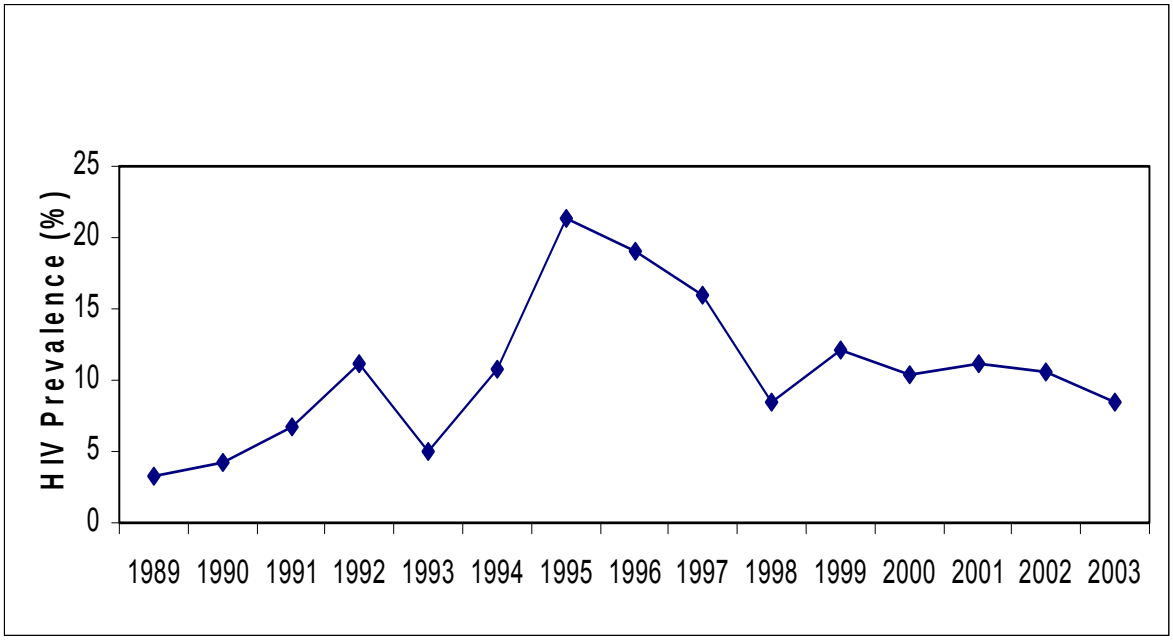


Figure 10: Kenya HIV

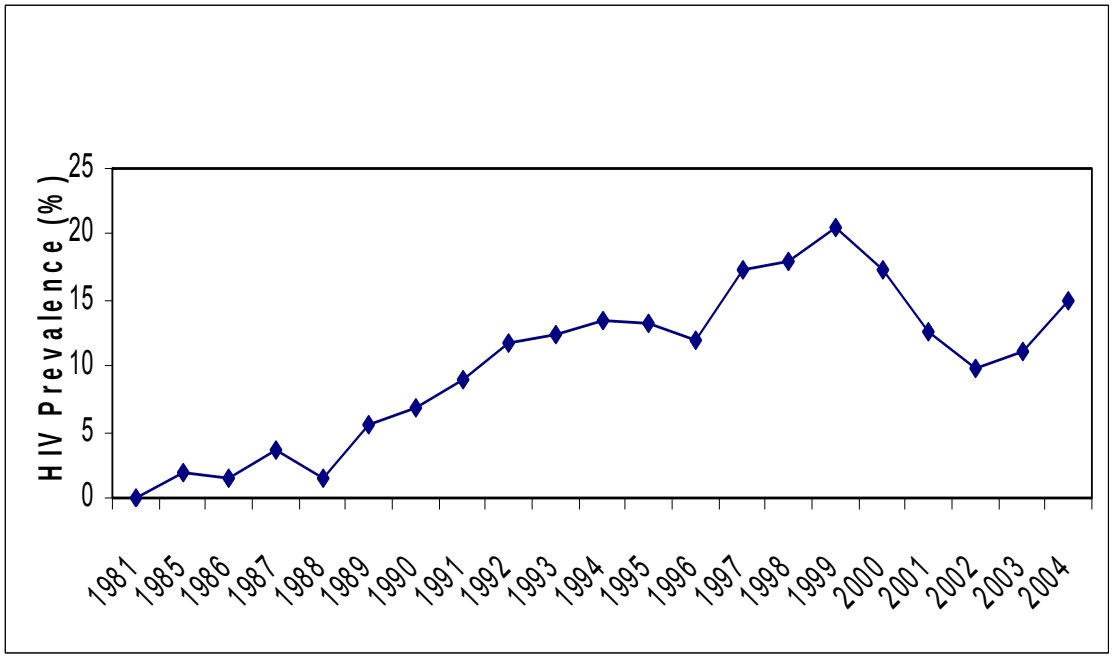


Figure 11: Malawi HIV

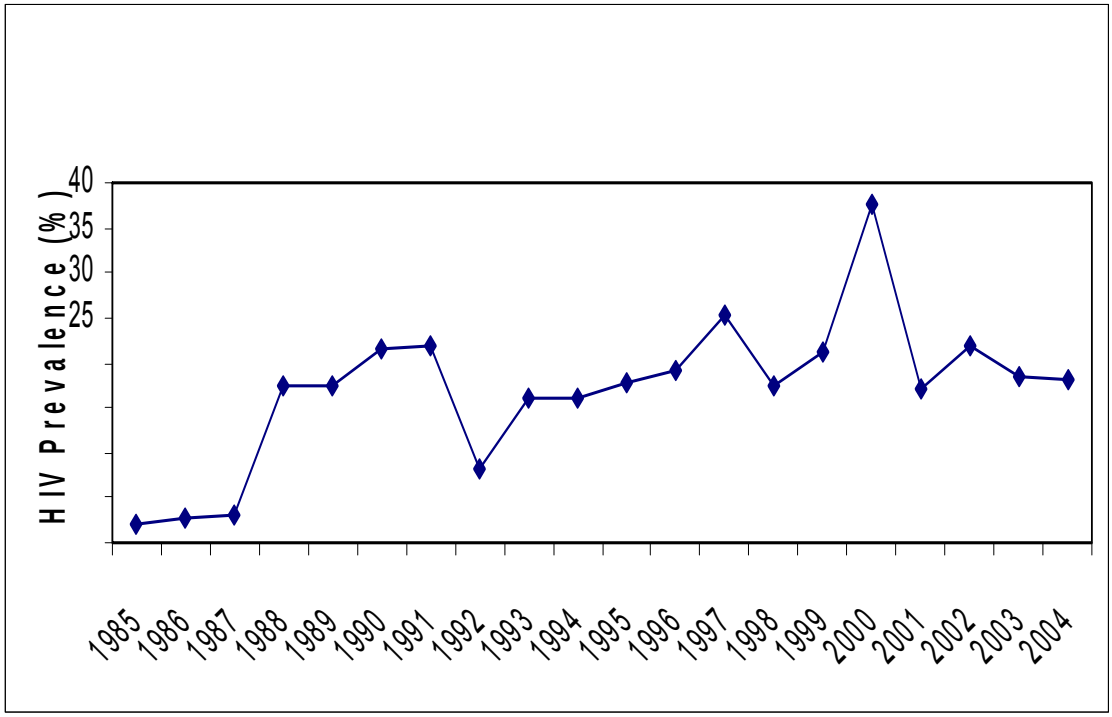
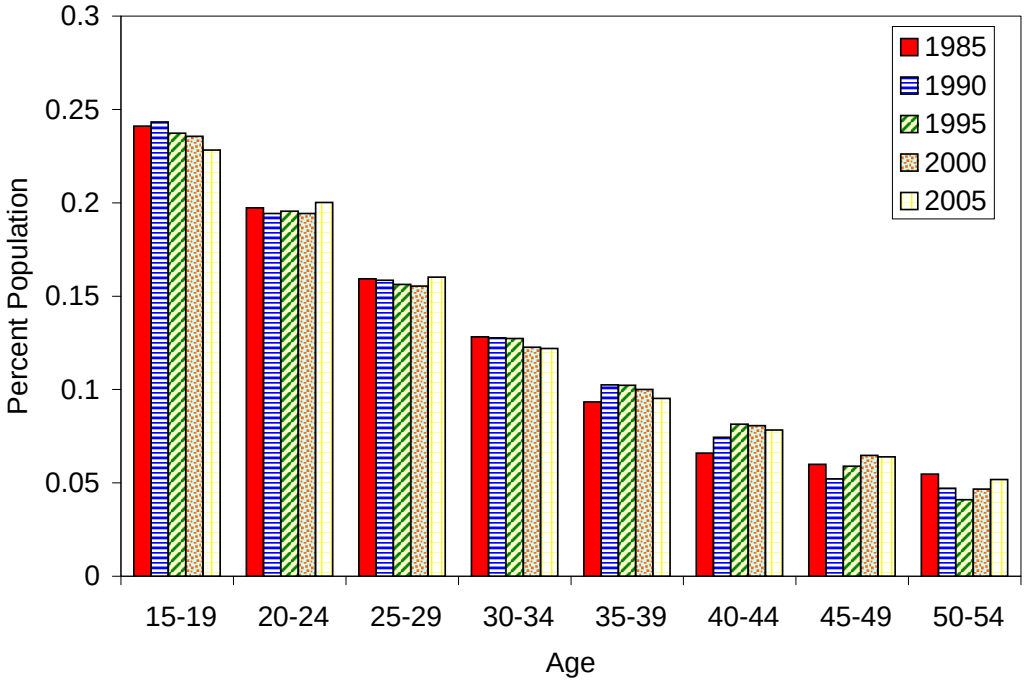


Figure 12: Population age Distribution in Kenya, 1985–2005



adults and hence population-age distribution should not change in favor of younger women.

Another alternative story rests on the question that what is driving the results in terms of sub-groups. Given the fact that total fertility rates is a summation over the age-specific fertility rates, the following scenario is also plausible. People might marry early, settle down sooner and start having children, due to the epidemic. This would lead to a shift in the timing of fertility, where people have children at younger ages. Thus even if each woman were to have no more children with HIV/AIDS than without HIV/AIDS, one might observe a gain in measured total fertility rates because in a given period two generations of women

would be bearing children, a previous generation whose schedule of childbearing had not been affected by AIDS and a new generation who had decided to have children earlier.

A first step would be looking at age-specific fertility rates from Kenya as shown in figure 13. These rates show that it does not seem to be the case that age-specific fertility rates are changing disproportionately, if anything they all have increased in the last survey consistent with the increase in the total fertility rate in Kenya, shown before. One must caution though since there is also ample evidence—as reviewed in section 2.3—from clinic and cohort based studies that HIV positive woman have lower fertility and childbearing odds. Hence age-specific fertility rates can also be lower for high HIV countries as a result of the epidemic. Indeed, for Kenya the decline in the age-specific fertility rates from 1993 to 1998 survey can easily be due to this biological effect of the epidemic. The point I am trying to make is that it seems not to be the case that there is a disproportionate change in age-specific fertility rates, at least for Kenya.

I also have investigated the effect of other control variables such as the use of contraception. The contraception data (defined as any form but mostly constitutes condom use) are available for 34 DHS survey countries for few years at most from World Bank and DHS. The data on condom use does not capture consistency of use; the DHS question is such that it involves reporting condom use during the last sexual encounter. Oster (2007) finds no statistically significant effects of HIV on condom usage.

Figure 14 presents the data on contraception prevalence from WB and DHS. Each countries survey year is on or around the dates shown on the x-axis. After an initial increase the use of contraception came to a halt in the latest surveys, as shown for Kenya and Cameroon. For some countries it has been constant throughout such as South Africa and Eritrea. And for some others, the use of contraception seems to have decreased in the latest years such as Chad, Nigeria, and Rwanda. There are yet other Africa countries where the use of contraception is on a steady rise. The bottomline is that it is hard to conclude sexual behavior and/or fertility is changing in any direction based on the available contraception data since there seems to be no definite pattern. Including the use of contraception in the between regres-

Figure 13: Age-Specific Fertility in Kenya, 1989, 1993, 1998, 2003

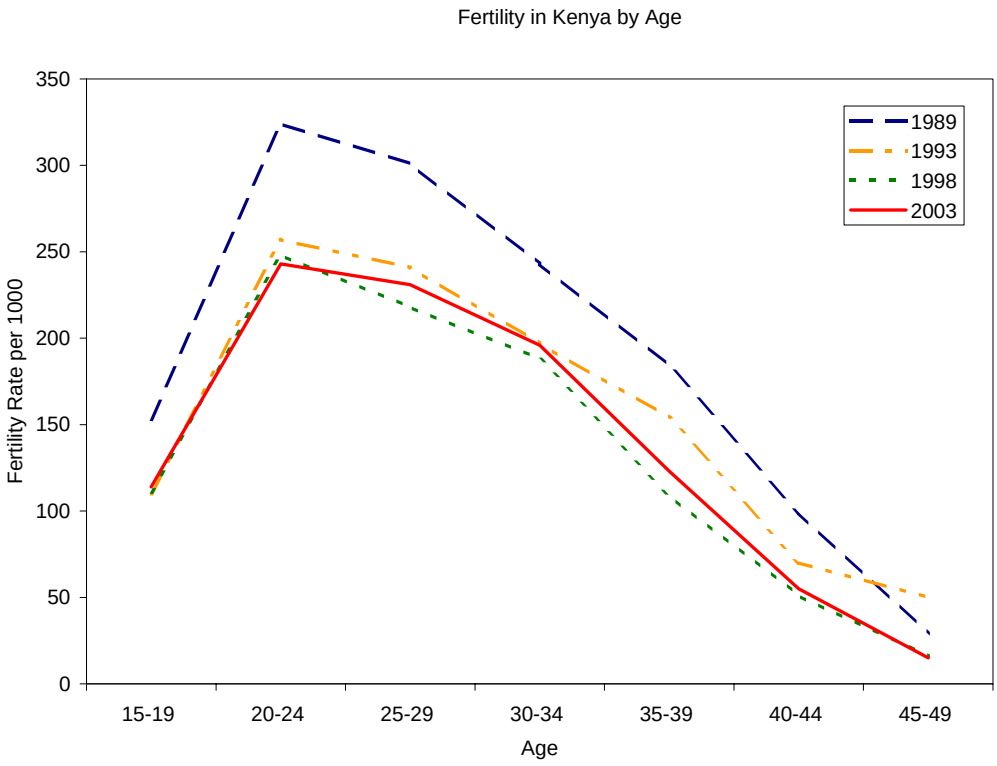
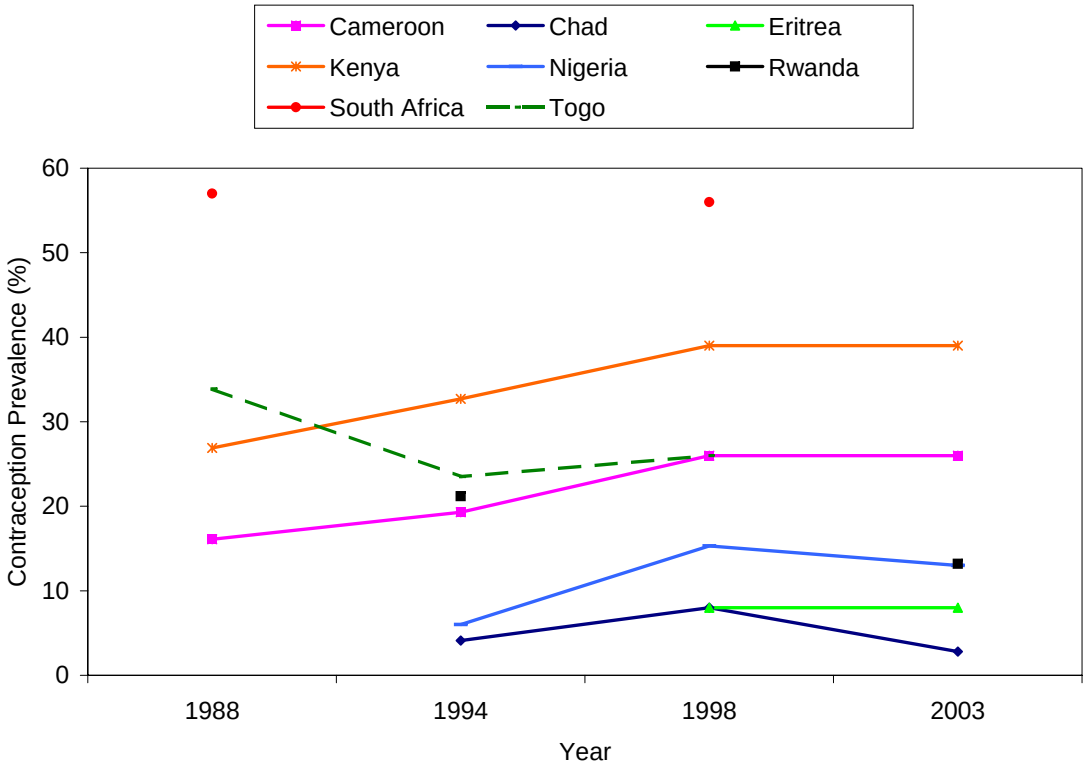


Figure 14: Contraception Prevalence in Africa



sions does not change the main result: HIV/AIDS prevalence is still positive and significant. Contraception use is negative and significant at 10 percent level.

I have also included the population age structure, male schooling, urbanization, and regional dummies for East, West and Southern Africa, as controls, and they mostly turn out to be insignificant.

4.6 IV Regressions

As discussed before to deal with the issue of endogeneity I will pursue an instrumental variable strategy. Following Oster (2007), I instrument HIV/AIDS by the distance to the origin of the epidemic, which is Democratic Republic of Congo. Given the time invariant nature of the instrument I will focus on the between estimates. For the instrument to be valid, it must be correlated with the HIV/AIDS but uncorrelated with the fertility rate, except through variable of interest that is included in the equation explaining fertility. The most obvious way in which distance to the origin of the epidemic might systematically affect the fertility rate—other than via HIV/AIDS— is through its correlation with geographic and/or socioeconomic variables. Hence these factors will be controlled in various ways as detailed out below.

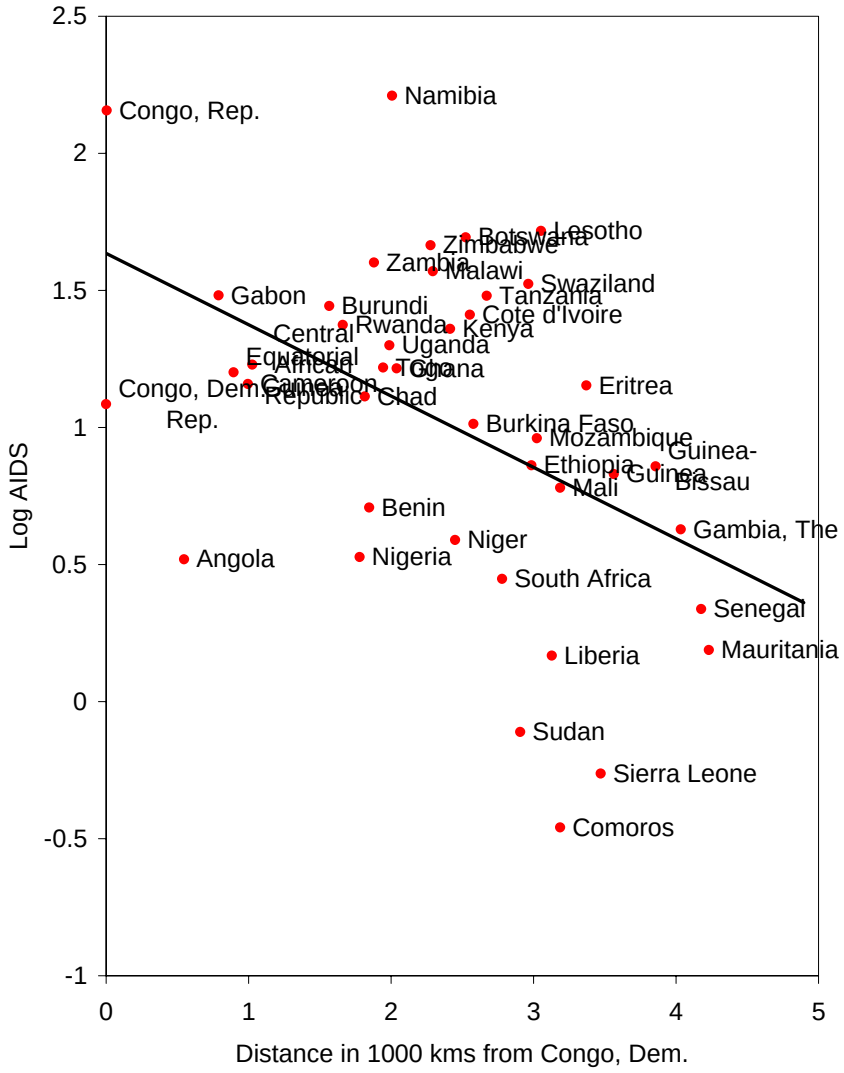
The first stage regression will be of the form:

$$HIV/AIDS_i = \varsigma + \chi Distance_i + \mathbf{X}_i'\pi + \xi, \tag{6}$$

where distance is measured as the distance from the capital city of each country to the capital city of the Democratic Republic of Congo, which is measured as the distance between the center of the capital cities. $\mathbf{X}$ matrix represents the controls that are used in the second stage regression. Figure 15 shows the relationship between log HIV prevalence and distance to the center of the Democratic Republic of the Congo. The relationship is clearly downward sloping and approximately linear as in Oster (2007).

Table 6 estimates the relationship between HIV/AIDS and distance adding the controls instead of the simple scatter plot shown in figure 15. Column (1) and column (3) show the relationship between distance and AIDS and HIV without any controls, respectively. However if there are regional differences among fertility rates and this is correlated with distance to Congo, then this will bias the results. Hence I include regional dummy variables for East, West and Southern Africa in column (2) and (4). Columns (2) and (4) also include the other socioeconomic controls such as female schooling, GDP per capita, and infant

Figure 15: Distance to the Center of the Epidemic and HIV/AIDS



mortality, which are the control variables in the second stage. The coefficient on distance stays negative significant and similar across all these specifications.[32]

Table 7 reports the 2SLS estimates for the effect of HIV/AIDS on fertility. The coefficients in columns (1) and (2) is positive and significant at 1 percent level. The coefficient for the projected HIV-EPP is insignificant as before and hence not reported. Notice that the coefficients for HIV and AIDS are higher than the corresponding OLS coefficients. This is probably due to the fact that the IV regression corrects for the measurement error which leads to attenuation bias in the OLS regression.

It is unlikely that distance drives fertility directly. There might still be a concern that distance might be correlated with fertility in the pre-HIV/AIDS period. Table 8 shows the falsification exercise for the exclusion restriction. Column (1) regresses fertility in 1980s on distance, obtaining an insignificant coefficient. The same result follows for fertility in 1970s. Columns (2)-(4) checks whether or not distance is directly correlated with socioeconomic factors such as GDP per capita, infant mortality, and child mortality.[33] Overall these results suggest that distance seems to be unrelated to pre-epidemic fertility and to various socioeconomic factors. These exercises provide confidence in the validity of instrument and it seems plausible to argue that distance to the origin of the epidemic will be unrelated to fertility.

4.7 Regional Evidence

This section uses regional data on fertility and HIV from 12 African countries. I have data on 32 regions. These are the regions with overlapping data on regional total fertility rates and HIV prevalence rates. Each country's survey year falls between 1998–2004. If there is more than 1 survey year during this period, than the data on the total fertility rate are averaged. I regress the regional total fertility rates on the logarithm of regional HIV prevalence rates among pregnant women averaged over 1990–1995, including country

[32]Oster's (2007) first stage estimates vary from –0.3 to –0.7 depending on the controls and country fixed effects.

[33]Oster (2007) further shows that there is no correlation between distance and malaria and life expectancy.

dummies. Unfortunately, the other controls are not available at the regional level.

Table 9 shows the results of the OLS regressions. Both columns show that results are positive and significant at 1 percent level. To deal with the potential serial correlation across residuals given the regional data, I cluster at the country level, which raises the standard errors as shown in column (2). Though the results are still significant at 1 percent level. I also tried a "Weighted Least Sqaures (WLS)" specification, where in order to limit the influence of small regions, I weighted by the population and also alternatively by the logarithm of regional population from DHS, averaged over the survey years. Results were similar and hence not reported. The results requires some caution since there might still be heterogeneity in spite of the country effects and clustering the standard errors, due to variation in proximity to road networks and urban-rural differences.[34]

5 Reconciling the results with those of Young (2007)

Instead of TFR, Young (2007) uses data on individual level fertility from Demographic Health Surveys (DHS) and the country level HIV projections, that is "HIV-EPP" and exploits within country variation over time. For his twenty-seven country sample there are only two countries have four surveys, ten countries have three surveys, eight countries have two surveys, and the remaining seven countries have only one survey. Thus the identification rests on limited observations. The quality of the time variation in HIV observations are also of suspect, as shown before in figures 9, 10, and 11.

Table 10 shows the replication of Young's (2007) results. Every regression has controls for age and education and also country and year effects. Columns (1)-(4) undertake a Poisson estimation of past year births following Young (2007) and columns (5)-(8) perform an OLS exercise. The difference between column (1) and (2) and similarly the other column pairs is the inclusion of control variables, which are marital status, urban/rural location, the number

[34]I also run IV regressions for a smaller sub-sample. In spite of a strong first stage the second stage regressions gave statistically insignificant results.

and square of born and living children, and the presence of a radio, television, refrigerator or bicycle (each entered separately) in the household.

Columns (1) and (2) replicates regressions of Young (2007) by clustering at individual level (on case-id). Not clustering at all give very close results. Columns (1) is an exact match to Young (2007) and column (2) is very close.[35] However, when I cluster by country in columns (3) and (4), the standard errors for the HIV coefficient get large and the coefficient ceases to be significant. The same story holds for the OLS estimation as shown in columns (4)-(8). Given the fact that the treatment is at the country-time level, it is preferable to cluster either by country-time or by country, to deal with possible serial correlation. Since the autocorrelations can be positive or negative it is possible for the non-clustered standard error to under or over-estimate the true standard error. In the case of HIV, positively serially correlated residuals lead to underestimated standard errors and hence false significance.

Peterson (2007) and Bertrand et al. (2004) report that, it had been common practice that researchers does not adjust the standard errors for possible dependence in residuals in the panel data sets. Peterson (2007) reports that 42 percent of the papers published in the last five years in finance using panel data by firm and by time does not adjust standard errors at all. He shows that the true standard error is 11 times the estimated and 81 percent of the time t-statistics are falsely significant at 1 percent. Bertrand et al. (2004) have drawn attention to robust standard error estimation in the context of a special fixed effect model, that is "Differences-in-Differences (DD)," where they show 65 percent of the time, there is false significance because of non-clustering. Out of 92 DD papers only 36 deal with the issue. Peterson (2007) and Bertrand et al. (2004) both show using simulations that clustered standard errors adequately account for the residual dependence created by the state (or firm or country) effect and thus provide unbiased estimates.[36] Peterson (2007) argues that if

[35]Column (1) is an exact match to the working paper version of Young (2007). Differences might be due to different controls. Although I tried to match the controls in Young (2007), some of his specifications do not detail the set of controls used.

[36]Peterson (2007) shows this for the standard OLS regression but he reports that his results generalizes to non-linear models too. Bertrand et al. (2004) focuses on a DD model such as; $Y_{ist} = A_s + B_t + cX_{ist} + \beta I_{st} + \epsilon_{ist}$, for individual i, state s, and time t. They also show simple parametric corrections, such as fitting an

there are both country and time fixed effects the best practice is to cluster at both levels or if the number of clusters is small in one dimension, like the time dimension, then use a fixed effect for that dimension and cluster on the other dimension, where more clusters are available.[37] As a result the cross-country finding of the negative significant effect of HIV on fertility is not robust.

6 Conclusion

Theoretical models of demographic transition imply a positive association between fertility and mortality. Should we expect then an increase in fertility as a response to the HIV/AIDS epidemic? The answer is not clear, since in the case of HIV/AIDS there might be various responses of fertility since this is a sexually transmitted disease.

Using country and regional level data from a panel of African countries during 1985−2000, I show a positive effect of the epidemic on fertility in between country and between region comparisons. I find no robust effect of the disease on fertility in within country comparisons, however. The within estimates range from positive to negative significant, yielding an insignificant effect in most of the specifications. Replicating Young (2007) also suggests an insignificant effect, once the standard errors are clustered by country. These results are consistent with the recent micro studies that use HIV data based on blood-testing from DHS surveys and find no effect of the disease on the fertility behavior.[38]

AR1 process for the error structure, or non parametric corrections, such as block bootstrap, only works with large number of states/cross-sectional units. They show that clustering at state level not just at state-year cell is the best solution.

[37]Kezdi (2004) shows clustered standard errors can be too large in a fixed effects model but he also shows only clustered standard errors are unbiased irrespective of having a country effect, as also shown by Peterson (2007). Peterson (2007) also shows the generalization of the results for the GLS case. Kezdi (2004) shows that the general robust standard error estimator known as the cluster estimator is not only consistent in general but it behaves well in finite samples. His Monte Carlo simulations shows that only cluster estimator gives unbiased results even in small cross-sectional samples. He shows in a fixed effect model with short time series (as here), serial correlation in the error process and the right hand side variables induce severe bias in conventional standard errors. Clustered estimator applied to mean-differenced data is consistent and behaves well in finite sample and it does not get biased with high T or small N.

[38]See Juhn et al. (2008), Fink and Linnemayr (2008), and Fortson (2008).

Appendix

Country Level Data:

Countries: Angola, Benin**, Botswana, Burkina Faso**, Burundi*, Cameroon**, Central African Republic*, Chad**, Comoros, Congo Democratic Republic, Congo Republic, Cote D'Ivoire**, Equatorial Guinea, Eritrea**, Ethiopia*, Gabon*, Gambia, Ghana**, Guinea*, Guinea-Bissau, Kenya**, Lesotho, Liberia*, Madagascar**, Malawi**, Mali**, Mauritania, Mauritius, Mozambique**, Namibia**, Niger**, Nigeria**, Rwanda** Senegal**, Seychelles, Sierra Leone, South Africa*, Sudan, Swaziland, Tanzania**, Togo**, Uganda**, Zambia**, Zimbabwe**.

Countries with a * has at least one DHS survey, and countries with ** has more than one DHS survey. The survey years are: Benin (1996, 2001), Burkina Faso (1992/1993, 1998/1999, 2003), Burundi (1987), Cameroon (1991, 1998), Central Republic of Africa(1994/1995), Chad (1996/1997), Cote D'Ivoire (1994, 1998), Ethiopia (2000), Gabon (2000), Ghana (1988, 1993, 1998, 2003), Guinea (1999), Kenya (1989, 1993, 1998, 2003), Liberia (1986), Malawi (1992, 2000), Mali (1987, 1995/1996, 2001), Mozambique (1997), Namibia (1992, 2000), Niger (1992, 1998), Nigeria (1990, 1999, 2003), Rwanda (1992, 2000), Senegal (1986, 1992/1993, 1997), South Africa (1998), Tanzania (1992, 1996, 1999), Togo (1988, 1998), Uganda (1988, 1995, 2000/2001), Zambia (1992, 1996, 2001/2002), Zimbabwe (1988, 1994, 1999).

- *AIDS*: The AIDS data come from UNAIDS/WHO, Epidemiological Fact Sheets (2003) and US Census Bureau HIV/AIDS Surveillance Database (2005). These are the number of reported AIDS cases for each country in every year and available for 44 African countries for 1985–2004. I multiply these number of reported incidents by 100,000 and divide by the country's population in each year, converting them to incidence per 100,000 per country per year. WHO-UNAIDS definition of AIDS (Acquired Immunodeficiency Syndrome) is that AIDS is the most severe manifestation of infection with the HIV (human immunodeficiency virus). The Centers for Disease Control and Preven-

tion (CDC) lists numerous opportunistic infections and neoplasms (cancers) that, in the presence of HIV infection, constitute an AIDS diagnosis. In 1993, CDC expanded the criteria for an AIDS diagnosis to include CD4+ T-cell count at or below 200 cells per microliter in the presence of HIV infection. In persons (aged 5 and older) with normally functioning immune systems, CD4+ T-cell counts usually range from 500 to 1500 cells per microliter. Persons living with AIDS often have infections of the lungs, brain, eyes and other organs, and frequently suffer debilitating weight loss, diarrhoea, and a type of cancer called Kaposi's sarcoma.

- *Contraceptive Prevalence*: Data on the percentage of women aged 15-49 who are using, or whose partners are using, any form of contraception, whether modern or traditional are available from World Bank, World Development Indicators (2006) and from DHS. The data are available only for 34 countries and few years between 1985–2004.

- *Distance to Democratic Republic of Congo in kms:* Pair-wise distance is taken from Arcview 3.x software, where each country's distance to Congo is measured as the distance from its capital to the capital of Congo.

- *Enrollment Rates*: Gross school enrollment rates are from World Bank, Word Development Indicators (2006). They are available for 35 countries and years between 1985–2004.

- *GDP per capita*: GDP per capita (PPP 2000 $s) is from World Bank, World Development Indicators (2006).

- *HIV*: HIV prevalence rates among pregnant women are from the U.S. Census Bureau, HIV Surveillance Database (2003). UNAIDS/WHO also provides similar data. Both Census and UNAIDS databases collect all studies and estimates of HIV/AIDS prevalence since the early 1980s. They provide information on prevalence, population and other factors and also provide regional estimates. The main indicator for the epidemic is the percent HIV-1 incidence among pregnant women for each country and year. HIV

is the retrovirus isolated and recognized as the etiologic (i.e. causing or contributing to the cause of a disease) agent of AIDS. HIV-1 is classified as a lentivirus in a subgroup of retroviruses. Most viruses and all bacteria, plants, and animals have genetic codes made up of DNA, which uses RNA to build specific proteins. The genetic material of a retrovirus such as HIV is the RNA itself. HIV inserts its own RNA into the host cell's DNA, preventing the host cell from carrying out its natural functions and turning it into an HIV factory. HIV-2 is a virus closely related to HIV-1 that has also been found to cause AIDS. It was first isolated in West Africa. Although HIV-1 and HIV-2 are similar in their viral structure, modes of transmission, and resulting opportunistic infections, they have differed in their geographical patterns of infection.

- *HIV-EPP*: The International Programs Center of the Census Bureau uses Estimation and Projection Package (EPP) from WHO/UNAIDS to estimate and project adult HIV prevalence among 15–49 year old from surveillance data between 1985–2004. While EPP can be used in all countries with sufficient surveillance data, it is specifically recommended for countries with generalized epidemics. Generalized epidemics are those that have broken out into the general population or consistent HIV prevalence at over 1 percent in low risk individuals. The proxy for low risk individuals is women attending antenatal clinics. The input to EPP in countries with generalized epidemics is surveillance data from various sites and years showing HIV prevalence among pregnant women, as well as data from national population-based surveys. EPP estimates the trends over time of HIV prevalence by fitting an epidemiological model to data from urban and rural sites. It tests possible epidemiological parameters, chooses a set minimizing least squares and projects future course based on fitted parameters, such as a parameter for the start year of the epidemic; one for the force of infection (how explosive the epidemic is in its initial stage); one for the fraction of new entrants to the population going into to the at-risk category (a parameter largely determines where the epidemic levels off); and one for the recruitment (a high value means people are brought into the at-risk population as people die of HIV, thus helping to sustain the

epidemic at a higher level).

- *HIV-Oster*: Oster (2006) estimates HIV rates for 9 countries between 1985–2000 for 15–35 year old individuals of both genders. She develops a methodology to estimate HIV prevalence over time from mortality data. To avoid the problem of lack of official mortality statistics for Africa she takes advantage of sibling mortality histories in the DHS.

- *Know Someone Died of AIDS*: The data on the percent female who know someone personally who has the virus that causes AIDS or has died of AIDS are from DHS, www.measuredhs.com, MEASURE DHS, Macro International Inc. The data are available for 23 countries whose survey years fall between 1993–2004.

- *Mortality*:
  Infant Mortality: Infant mortality is the rate per 1000 live births and from World Bank, World Development Indicators (2006). The data are available for 8 years (1985, 1987, 1990, 1992, 1995, 1997, 2000,2004).
  Age 5 Mortality: Age 5 mortality is the rate per 1000 children under age 5 and from World Bank, World Development Indicators (2006). The data are available for 5 years (1985, 1990, 1995, 2000, 2004).

- *Total Fertility Rate*: Data on total fertility rates are from World Bank, World Development Indicators (2006) and available for 10 years (1985, 1987, 1990, 1992, 1995, 1997, 2000, 2002, 2003, 2004) and 44 countries. DHS data on total fertility rate per woman ages 15–49 are from DHS, www.measuredhs.com, MEASURE DHS, Macro International Inc. The data are available for 34 countries whose survey years fall between 1986–2004.

Regional Level Data:

Regions:

Benin: Atacora Province, Atlantique Province, Borgou Province, Mono Province, Oueme Province, Zou Province.

Ethiopia: Addis Ababa, Dire Dawa, Gambella, Harari.

Ghana: Accra, Northern region, Upper East region, Upper West region.

Lesotho: Maseru, Leribe district, Mafeteng district, Quthing district, Mokhotlong.

Madagascar: Antananarivo, Antsiranana, Fianarantsoa, Mahajanga, Toamasina, Toliary.

Malawi: Lilongwe, Blantyre, Mangochi, Mulanje, Mzimba, Thyolo.

Mali:Bamako, Koulikoro, Mopti, Sikasso.

Niger:Dosso, Maradi, Niamey, Tahoua, Zinder.

Nigeria: North East zone, North West zone, South East zone, South West zone.

Rwanda: Butare, Byumba, Gisenyi, Kigali, Ruhengeri.

South Africa: Eastern Cape Province, Free State Province, Gauteng Province, Mpumalanga Province, Northern Cape Province, Northern Province, North-West Province, Western Cape Province.

Tanzania: Dar es Salaam, Rukwa region, Arusha region, Zanzibar area.

Togo: Kara, Plateaux, Savanes.

Zimbabwe: Harare, Bulawayo, Manicaland, Masvingo, Mashonaland West Province, Matabeleland South.

- *Fertility Rates*: Regional fertility rates are from DHS, www.measuredhs.com, MEASURE DHS, Macro International Inc., and available for 14 countries, whose surveys years fall between 1988–2004.

- *Distance to Democratic Republic of Congo in kms:* Distance from center of every region to the center of Congo is provided by Emily Oster.

- *HIV Rates-US Census*: Regional HIV data come from U.S. Census Bureau, HIV Surveillance Database (2005) and available for 14 African countries. The data are available for 1985–1990 and also for later years for a smaller number of regions.

Individual Level Data:

Individual level data are used for 27 countries from 57 Demographic Health Surveys: Benin (1996, 2001), Burkina Faso (1992/1993, 1998/1999, 2003), Burundi (1987), Cameroon (1991, 1998), Central Republic of Africa(1994/1995), Chad (1996/1997), Cote D'Ivoire (1994, 1998), Ethiopia (2000), Gabon (2000), Ghana (1988, 1993, 1998, 2003), Guinea (1999), Kenya (1989, 1993, 1998, 2003), Liberia (1986), Malawi (1992, 2000), Mali (1987, 1995/1996, 2001), Mozambique (1997), Namibia (1992, 2000), Niger (1992, 1998), Nigeria (1990, 1999, 2003), Rwanda (1992, 2000), Senegal (1986, 1992/1993, 1997), South Africa (1998), Tanzania (1992, 1996, 1999), Togo (1988, 1998), Uganda (1988, 1995, 2000/2001), Zambia (1992, 1996, 2001/2002), Zimbabwe (1988, 1994, 1999).

- *Educational Attainment*: This is a categorical variable for woman's educational attainment level. Categories are "No Education", "Primary Education", "Secondary Education", "Tertiary Education" (v106).

- *Fertility:* Measured as number of births or pregnancies in last year for each woman (v209).

- *Controls*: Other control variables from are: Age (v121), year of survey (v007), presence of radio in the household (v120), presence of television in the household (v121), presence of refrigerator in the household (v122), presence of bicycle in the household (v123), urban/rural (v102), number of born children (v201), number of living children (v201-v206-v207).

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Table 2: **Descriptive Statistics**

	No. of Countries	Mean	Std.dev.	Max	Min
Total Fertility Rate, WB	44	5.71	1.22	8.06	2.08
Total Fertility Rate, DHS	34	6.07	0.87	7.40	3.90
AIDS (per 100,000)	42	22.38	32.84	162.16	0.02
HIV	44	0.08	0.07	0.25	0.001
HIV-EPP	38	0.06	0.05	0.21	0.004
HIV-Oster	9	0.04	0.03	0.09	0.009
Know Someone Died of AIDS (%)	23	40.81	21.03	88.45	6.90
Secondary School for Female (%)	38	24.55	23.62	112.82	4.24
GDP per capita (PPP 1996 $s)	44	798.24	1217.78	6168.33	95.92
Infant Mortality (per 1000)	44	100.30	36.06	176.75	14.99
Mortality Under 5 (per 1000)	44	159.11	63.80	295.76	17.60
Contraceptive Use (%)	27	20.16	14.38	63.75	4.00

Notes: All variables are averaged over 1985−2000 and 44 countries depending on the availability. Total Fertility Rate is the sum of age-specific fertility rates (number of children that a woman would have if she lived through all of her child-bearing years and experienced the current age-specific fertility rates at each age); from World Bank (WB), World Development Indicators (WDI) and from DHS, www.measuredhs.com, MEASURE DHS, Macro International Inc., respectively. The survey years for the data from DHS fall between 1986–2004. Data Appendix reports the survey years for each country. AIDS represents the number of officially reported AIDS cases per 100,000 per country per year, calculated as multiplying the officially reported AIDS cases by 100,000 and dividing by population; from WHO/UNAIDS, Epidemiological Fact Sheets. HIV represents percent HIV-1 sero-prevalence infection rate among pregnant women attending antenatal clinics; from U.S. Census Bureau, HIV Surveillance Database. HIV-EPP represents *estimated* national HIV prevalence among 15 to 49 year olds calculated by fitting an epidemiological model to data (Estimation and Projection Package-EPP) from urban and rural surveillance sites; from U.S. Census Bureau, International Programs Center. HIV-Oster represents Oster (2006) estimates that are based on mortality data from sibling histories in the DHS. Know Someone Died of AIDS, represents the percent female who know someone personally who has the virus that causes AIDS or has died of AIDS; from DHS. Secondary Schooling is the gross enrollment rates from WDI. GDP per capita is the Gross Domestic Product (PPP 1996 $) divided by population; from WDI. Infant Mortality is the infant mortality rate per 1000 births; from WDI. Mortality under 5 is the age 5 and under mortality per 1000 births; from WDI. Contraceptive Use represents the percent women aged 15-49 who are using, or whose partners are using, any form of contraception; from WDI. See Appendix for more information on the variables.

Table 3: **AIDS, HIV and Fertility: Between Regressions**

Dependent variable: Total Fertility Rate (TFR)

Source for TFR:	WB	WB	WB	DHS	DHS	DHS
	(1)	(2)	(3)	(4)	(5)	(6)
Log AIDS	0.14***	–	–	0.20***	–	–
	(0.05)	–	–	(0.08)	–	–
Log HIV	–	0.14**	–	–	0.20***	–
	–	(0.07)	–	–	(0.09)	–
Log HIV-EPP	–	–	–0.04	–	–	–
	–	–	(0.11)	–	–	–
Know Someone Died	–	–	–	–	–	0.20***
of AIDS	–	–	–	–	–	(0.07)
Female Schooling	–0.02***	–0.02***	–0.02***	–0.02***	–0.03***	–0.02***
	(0.006)	(0.006)	(0.006)	(0.009)	(0.01)	(0.009)
Log GDP per capita	–0.11	–0.10	–0.11	–0.01	–0.12	–0.13
	(0.06)	(0.08)	(0.07)	(0.17)	(0.20)	(0.19)
Infant Mortality	0.02***	0.02***	0.01***	0.01***	0.01***	0.01***
	(0.003)	(0.003	(0.003))	(0.004)	(0.01)	(0.007)
R^2	0.84	0.88	0.80	0.63	0.61	0.67
Observations	33	35	30	26	26	22

Notes: Robust standard errors (White correction) are in parentheses. The Between Regressions report the results using country averages depending on availability, and including a constant. See table 2 for the detailed explanation of the variables. ***, **, * denote 1, 5, 10 percent significance respectively.

Table 4: **AIDS, HIV and Fertility: Falsification Exercise**

Dependent variable: Total Fertility Rate in 1980s		
	(1)	(2)
Log AIDS	0.01	–
	(0.06)	–
Log HIV	–	0.08
	–	(0.10)
Controls	Yes	Yes
R^2	0.70	0.72
Observations	32	33

Notes: Robust standard errors (White correction) are in parentheses. The Between Regressions report the results using country averages depending on availability, and including a constant. See table 3 for the set of controls. See table 2 for the detailed explanation of the variables.

Table 5: **AIDS, HIV and Fertility: Pooled and Within Regressions**

Dependent variable: Total Fertility Rate (TFR)

	Pooled (1)	Pooled (2)	Pooled (3)	Within (4)	Within (5)	Within (6)	Within (8)	Within (9)	Within (10)
Log AIDS	0.09**	–	–	0.09**	–	–	–0.06	–	–
	(0.04)	–	–	(0.04)	–	–	(0.05)	–	–
Log HIV	–	0.12**	–	–	–0.01	–	–	–0.08	–
	–	(0.06)	–	–	(0.05)	–	–	(0.05)	–
Log	–	–	–0.01	–	–	0.10	–	–	–0.15***
HIV-EPP	–	–	(0.07)	–	–	(0.10)	–	–	(0.05)
Female	–0.02***	–0.03***	–0.02***	–0.004***	–0.001***	–0.002***	–0.03***	–0.003***	–0.03***
Schooling	(0.005)	(0.005)	(0.005)	(0.01)	(0.01)	(0.01)	(0.02)	(0.008)	(0.01)
Log GDP	–0.10	–0.12	–0.12	–0.27	–0.29	–0.43	–0.27	–0.29	–0.13
per capita	(0.06)	(0.08)	(0.07)	(0.31)	(0.27)	(0.29)	(0.27)	(0.27)	(0.24)
Infant	0.01***	0.01***	0.01***	0.003***	0.006***	0.004***	0.007***	0.01***	0.005***
Mortality	(0.002)	(0.032)	(0.002)	(0.01)	(0.004)	(0.01)	(0.007)	(0.005)	(0.004)
Common Trend	–0.05***	–0.04***	–0.03***	–	–	–	–	–	–
	(0.02)	(0.01)	(0.01)	–	–	–	–	–	–
Country Effects	No	No	No	Yes	Yes	Yes	Yes	Yes	Yes
Year Effects	No	No	No	Yes	Yes	Yes	No	No	No
Country Trends	No	No	No	No	No	No	Yes	Yes	Yes
R^2	0.76	0.82	0.76	0.87	0.87	0.87	0.95	0.95	0.95
Observations	111	153	139	111	153	139	111	153	139
Countries	33	35	30	33	35	30	33	35	30

Notes: Robust standard errors (White correction; clustered on countries) are in parentheses. The Within Regressions report results using country fixed effects. See table 2 for the detailed explanation of the variables. ***, **, * denote 1, 5, 10 percent significance respectively.

Table 6: **Instrumental Variables Regression: First Stage**

Dependent variable:	Log AIDS (1)	Log AIDS (2)	Log HIV (3)	Log HIV (4)
Distance (1000km)	–0.6***	–0.7***	–0.5***	–0.6***
	(0.2)	(0.2)	(0.2)	(0.2)
Female Schooling	–	–0.01	–	0.05
	–	(0.01)	–	(0.01)
Log GDP per capita	–	–0.13	–	–0.67
	–	(0.26)	–	(0.27)
Infant Mortality	–	0.01	–	0.01
	–	(0.01)	–	(0.01)
Regional Dummies	No	Yes	No	Yes
R^2	0.28	0.34	0.20	0.36
Observations	33	33	35	35

Notes: Robust standard errors (White correction) are in parentheses. The Between Regressions report the results using country averages of the variables, and including a constant. Distance to Dem. Congo is in 1000km. See table 2 for the detailed explanation of the variables. ***, **, * denote 1, 5, 10 percent significance respectively.

Table 7: **Instrumental Variable Regressions: Second Stage**

Dependent variable: Total Fertility Rate (TFR)

	(1)	(2)
Log AIDS	0.28***	–
	(0.09)	–
Log HIV	–	0.37***
	–	(0.14)
Female Schooling	–0.02***	–0.03***
	(0.006)	(0.007)
Log GDP per capita	–0.15	–0.02
	(0.08)	(0.11)
Infant Mortality	0.02***	0.01***
	(0.003)	(0.004)
R^2	0.80	0.82
Observations	33	35

Notes: Robust standard errors (White correction) are in parentheses. The 2SLS Regressions report the results using country averages, and including a constant. Distance to Dem. Congo is in 1000km. See table 2 for the detailed explanation of the variables. ***, **, * denote 1, 5, 10 percent significance respectively.

Table 8: **Falsification on Exclusion Restriction**

Dependent var.:	TFR in 1980s (1)	Log GDP (2)	Infant Mort. (3)	Child Mort. (4)
Distance (1000km)	0.03	0.01	5.83	–12.1
	(0.1)	(0.1)	(5.98)	(11.4)
R^2	0.00	0.00	0.03	0.04
Observations	33	35	35	35

Notes: Robust standard errors (White correction) are in parentheses. The Between Regressions report the results using country averages of the variables, and including a constant. Distance to Dem. Congo is in 1000km. See table 2 for the detailed explanation of the variables.

Table 9: **HIV and Fertility: Between Regressions at the Regional Level**

Dependent variable: TFR in 1998–2004		
	(1)	(2)
Log HIV in 1990–1995	0.29***	0.29***
	(0.05)	(0.08)
Country Dummies	Yes	Yes
Cluster	Region	Country
R^2	0.79	0.79
Observations	32	32
Countries	12	12

Notes: Robust standard errors (column 1: clustered on regions, column 2: clustered on countries) are in parentheses. All regressions report results using country fixed effects. Regional TFRs are from DHS, various survey years (mean: 5.07, std dev.: 1.60, max: 8.7, min: 1.9). Each country's survey year falls between 1987–2004. The data are averaged over the survey years. Regional HIV rates (percent HIV-1 sero-prevalence among pregnant women) are from the U.S. Census Bureau, HIV Surveillance Database (2003) (mean: 0.047, std dev.: 0.079, max: 0.3094, min: 0). HIV prevalence rates are averaged over 1990–1995 or used as a single year depending on the availability.

Table 10: **HIV and Individual Fertility in a Panel of African Countries**

Dependent variable is Last Year Births

Estimation	Poisson	Poisson	Poisson	Poisson	OLS	OLS	OLS	OLS
Cluster:	Individual (1)	Individual (2)	Country (3)	Country (4)	Individual (5)	Individual (6)	Country (7)	Country (8)
Projected HIV	–1.260***	–0.963***	–1.260	–0.963	–0.218***	–0.234***	–0.218	–0.234
	(0.380)	(0.385)	(1.17)	(0.908)	(0.057)	(0.054)	(0.201)	(0.190)
Primary Education	–0.294***	0.071***	–0.294***	0.071***	0.060***	0.010***	0.060***	0.010***
	(0.054)	(0.057)	(0.005)	(0.001)	(0.009)	(0.010)	(0.001)	(0.000)
Secondary Education	–0.798***	0.252***	–0.798***	0.252**	-0.132***	0.029***	–0.132***	0.029***
	(0.091)	(0.102)	(0.005)	(0.012)	(0.010)	(0.012)	(0.001)	(0.002)
Tertiary Education	–1.190***	0.759**	–1.190***	0.759***	–0.155***	0.116***	–0.155***	0.116***
	(0.445)	(0.461)	(0.007)	(0.036)	(0.030)	(0.033)	(0.001)	(0.007)
Age	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Age^2	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Controls	No	Yes	No	Yes	No	Yes	No	Yes
Country Effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Country Trends	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	403100	350586	403100	350586	403100	350586	403100	350586

Notes: Countries and survey years are Benin (1996, 2001), Burkina Faso (1992/1993, 1998/1999, 2003), Burundi (1987), Cameroon (1991, 1998), Central Republic of Africa(1994/1995), Chad (1996/1997), Cote D'Ivoire (1998/1999), Ethiopia (2000), Gabon (2000), Ghana (1988, 1993, 1998, 2003), Guinea (1999), Kenya (1989, 1993, 1998, 2003), Liberia (1986), Malawi (1992, 2000), Mali (1987, 1995/1996, 2001), Mozambique (1997), Namibia (1992, 2000), Niger (1992, 1998), Nigeria (1990, 1999, 2003), Rwanda (1992, 2000), Senegal (1986, 1992/1993, 1997), South Africa (1998), Tanzania (1992, 1996, 1999), Togo (1988, 1998), Uganda (1988, 1995, 2000/2001), Zambia (1992, 1996, 2001/2002), Zimbabwe (1988, 1994, 1999). Other controls in the regressions are marital status (never, currently or formerly married), urban/rural location, the number and square of born and living children, and the presence of a radio, television, refrigerator or bicycle (each entered separately) in the household. Robust standard errors (clustered as indicated) are in parentheses. $***$ denotes 1% significance.