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HIV and fertility in Africa: first evidence from population-based surveys

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Abstract The historical pattern of the demographic transition suggests that fertility declines follow mortality declines, followed by a rise in human capital accumulation and economic growth. The HIV/AIDS epidemic threatens to reverse this path. We utilize recent rounds of the demographic and health surveys that link an individual woman's fertility outcomes to her HIV status based on testing. The data allow us to distinguish the effect of own positive HIV status on fertility (which may be due to lower fecundity and other physiological reasons) from the behavioral response to higher mortality risk, as measured by the local community HIV prevalence. We show that although HIV-infected women have significantly lower fertility, local community HIV prevalence has no significant effect on noninfected women's fertility.

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1 Introduction

A fiercely debated question in the health and development literature is the impact of HIV/AIDS epidemic on economic growth. So far, there is no consensus. The calibration studies find big effects mainly due to the destruction of human capital (see Corrigan et al. 2005). The empirical studies using economic growth as an outcome show mixed results.[1] In an influential paper, Young (2005) suggests that population declines will lead to higher capital–labor ratios and eventually to higher per capita income in the affected countries. He postulates that widespread community infection will lower fertility, both directly through a reduction in the willingness to engage in unprotected sex, and indirectly, by increasing the scarcity of labor and the value of women's time. Using household data from South Africa and relying on between cohort variation in country-level HIV infection and number of births, he estimates a large negative effect of HIV prevalence on fertility. He concludes that even under the most pessimistic assumption for human capital destruction, the fertility effect dominates and hence future per capita income of South Africa improves.[2]

In this paper, we use newly available micro data from population-based surveys to examine the fertility response to HIV/AIDS. The question is important since we cannot answer the question of the effect of the disease on development without knowing the response of fertility to the disease. In the latest rounds of the demographic health surveys (DHS), HIV testing was administered in 13 African countries allowing us to link an individual woman's detailed fertility and health history to her own HIV status. One advantage of this newly available data is that it provides us with a more accurate estimate of HIV prevalence in the population. Previous researchers, including Young (2005), relied on estimates based on samples of pregnant women attending prenatal clinics which may have higher or lower prevalence rates relative to a more representative sample. Another advantage of the new data is that we can examine separately the impact of *own* HIV status from the impact of

[1]Bloom and Mahal (1997) run cross-country regressions of growth of GDP per capita on HIV/AIDS prevalence and find no effect. Papageorgiou C, Stoytcheva P (2008, What Is the Impact of AIDS on Cross-Country Income So Far? Evidence from Newly Reported AIDS Cases, unpublished) find negative effect on the level of income per capita in a similar framework. Werker, E D, Ahuja A, Wendell B (2006, Male Circumcision and AIDS: The Macroeconomic Impact of a Health Crisis, unpublished) instrument HIV/AIDS prevalence by national circumcision rates and show that there is no effect of the epidemic on growth of the African countries.

[2]Kalemli-Ozcan and Turan (2011) shows that Young's identification from time-series data may not be appropriate given the existing trends in South African data due to abolition of apartheid and the ongoing demographic transition.

community-wide prevalence. Women who are HIV positive may have lower fertility due to physiological reasons, i.e., the disease may lower fecundity or the individual may be too sick to be sexually active. By examining changes in fertility among *noninfected* women, we can focus on the behavioral response to increased risk of infection and death.

To preview our results, we find that the disease significantly lowers an infected woman's fertility. Being infected with HIV reduces births last year by approximately 20–25 %, depending on whether we control for marital status. Women who are infected are considerably more likely to be widowed, separated, or divorced, which are marital status categories also associated with lower birth rates.[3] The ordinary least squares (OLS) estimation assumes that HIV-positive and HIV-negative women are comparable once we control observable characteristics. This assumption may be violated if HIV-positive and HIV-negative women are systematically different in unobservable ways. While it is not possible for us to entirely rule out selection on unobservables given the cross-sectional nature of our data, we examine to what extent unobserved heterogeneity may be driving our results by exploiting fertility histories of older women who are currently observed to have positive or negative HIV status. We find little difference in birth outcomes of HIV-positive and HIV-negative women when we examine their fertility histories prior to 1986 (before the onset of the disease). This suggests that unobserved heterogeneity is not the major driving force behind our results. We also find similarly sized negative impact of HIV when we control for measures of sexual behavior such as condom use and multiple partners, which suggests that the physiological impact of the disease may play an important role.

We find little evidence, however, of a behavioral response in fertility to mortality risk, as proxied by community-level prevalence rates. In OLS regressions, we regress fertility of noninfected women on the local community HIV prevalence rate and find no significant effect. While our standard errors are large, we can nevertheless rule out the large negative fertility responses found in Young (2005). We also use earlier surveys to build community-level panel data. Assuming zero prevalence of the disease before 1986, we run community fixed effects regressions and find no significant effects. Our community-level results are consistent with Fortson (2009) who also uses fertility histories and performs a variety of robustness checks in examining the relationship between HIV and fertility. While the methodology and the results on community-level HIV rates are similar across the two papers, we also examine in this paper the effect of own HIV status on fertility which we believe is of interest in its own right.[4]

[3] Among HIV-positive women, 29 % are widowed, separated, or divorced as opposed to 7 % among HIV-negative women.

[4] The community-level results were produced independently and at the same time in an earlier version of our paper, Juhn C, Kalemli-Ozcan S, Turan B (2008) HIV and Fertility in Africa: First Evidence from Population Based Surveys and a working paper version of Fortson (2009).

Overall, our estimate of the impact of HIV on total fertility rate is considerably smaller than reported in Young (2005). His estimates suggest that a community that has 100 % prevalence would have fertility that is approximately 80 % lower than a community with zero prevalence. Our estimate of the impact of HIV, working exclusively through the own effect, suggests that fertility would be approximately 20 % lower. Given that country-level prevalence rates fall well below 100 %, this translates into relatively small reductions in country-specific total fertility rates. For example, even in Lesotho, which has the highest prevalence rate in our sample (26.4 %), the total fertility rate would be 0.15–0.3 children, higher (approximately 4–8 %) in the absence of HIV/AIDS.[5]

The paper proceeds as follows. Section 2 lays out the conceptual framework. Section 3 describes the data. Section 4 presents the empirical results. Section 5 examines the impact of HIV on total fertility rates, and Section 6 concludes our study.

2 Conceptual framework

To begin, we can turn to the large theoretical literature that links life expectancy and economic development. Neoclassical growth models identify two effects. The first-order effect of increased life expectancy is to increase population. When there is no behavioral response in fertility, reductions in mortality increase population, thus reduce capital–labor and land–labor ratios and depress 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 (see, for example, (Cervelatti M, Sunde U (2007) Human Capital, Mortality, and Fertility: A Unified Theory of Economic and Demographic Transition, unpublished), Tamura (2006), Soares (2005), Kalemli-Ozcan (2003), Galor and Weil (2000), Lucas (2000), and Ehrlich and Lui (1991)). 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 can undo and even reverse the

[5]In the follow-up paper that uses country by cohort variation, Young (2007) reports a range of coefficients from −1.54 to −0.60 (high–low). These coefficients translates into a reduction in fertility of approximately 154–45 % as a country goes from 0 to 100 % prevalence. As discussed in Young (2007), the size of the coefficient appears to be sensitive to the inclusion of the country-specific time trends.

initial rise in population size.[6] The HIV/AIDS epidemic has generated a negative shock to life expectancy which, according to these models, should *increase* fertility.[7]

However, treating HIV/AIDS simply as a shock to adult longevity may be overly simplified. First, field evidence strongly suggests that there is a direct biological/physiological impact of the disease, which lowers the fecundity of infected women, an effect which should be considered separately from the behavioral responses, as we have argued in the introduction. Many African studies, both clinic and cohort based, indicate lower fertility (around 40 %) and childbearing odds among HIV-positive women. Gray et al. (1998), in a cross-sectional analysis of a Ugandan community, find that HIV reduced the pregnancy rate by 55 %. Carpenter et al. (1997) and Hunter et al. (2003), in cohort studies in Uganda and Tanzania, respectively, find a 30–40 % reduction in probability of becoming pregnant. Fecundity is reduced by HIV infection due to higher rates of miscarriage and stillbirth and high rates of coinfection with other sexually transmitted infections, which may cause secondary infertility.[8]

Second, since it is largely a sexually transmitted disease, we must consider how the disease impacts fertility through changes in sexual behavior, namely through the reduction in the willingness to engage in unprotected sex. The impact of the disease on sexual behavior in Africa has proven to be a much debated topic. Mwaluko et al. (2003), Bloom et al. (2000), Stoneburner and Low-Beer (2004), Lagarde et al. (1996), Lindan et al. (1991), Ng'weshemi et al. (1996), Williams et al. (2003), and Caldwell et al. (1999) all find no

[6]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. Ashraf et al. (2008) show that the effects of health improvements on income only emerge for half a century after the initial improvement in health.

[7]While the focus of our 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 that for a 30 % HIV-positive population like Botswana, there would be a 6 % reduction in the rate of return to education relative to no HIV. Bell et al. (2006), 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 (2011), using data similar to ours, shows that 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. Akbulut and Turan (2013) show that HIV prevalence in the community impairs the intergenerational human capital transfers even if the mother is HIV-negative.

[8]While their estimates are somewhat higher than other estimates, Gray et al. (1998) is often cited as the study that comes closest to identifying the effect on *fecundity*. The study interviewed a representative sample of women in their homes and obtained blood samples from 91 % of the women. Most importantly, women did not know their HIV status at baseline because access to testing prior to the survey was not available in the communities surveyed. Contraception and abstinence were also very rare in these communities.

change or very small change in sexual behavior. Luke and Munshi (2006) find that married men in AIDS-prevalent communities in Kenya have similar numbers of nonmarital partners as single men. One would expect the number of nonmarital partners to fall more for the married men if unprotected sexual activity is an issue or if wives could influence husband's extramarital sexual activity. 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. She shows that there has been a very small decrease in the share of single women having premarital sex. Other researchers find some evidence of risky behavior reductions in Zambia and Zimbabwe such as reductions in multiple partners; see Cheluget et al. (2006) and Fylkesnes et al. (2001).

Third, regardless of changes in sexual behavior and desire for unprotected sex, it may be the case that infected women who know their own status and have knowledge about mother–child transmission would want to reduce fertility rather than give birth to infected children. Again, the 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, even among HIV-positive women, 45 % expressed a desire to become pregnant. 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.[9]

A body of theoretical models imply that fertility responds positively to a rise in mortality risk by increasing the marginal utility of having more children. The special case of HIV/AIDS, however, suggests that fertility may decrease, first through direct physiological reasons, and second, through changes in sexual behavior and the reduction in willingness to engage in unprotected sex. In our empirical work below, we separate out the effect of own positive HIV status on fertility (which may be due to physiological factors) from the behavioral response to higher mortality risk as measured by the community-level

[9]In the 2000s, antiretroviral regimens to prevent mother to child transmission (MTCT) became more widely available even in resource poor countries in sub-Saharan Africa. While a full-scale analysis incorporating differences across regions and time in the availability of these drugs is beyond the scope of our paper, it is important to consider how the omission of this information may bias our results. The availability of drugs designed to reduces MTCT may encourage unprotected sex and higher fertility among HIV-infected women but it may also reduce the precautionary move towards having protected sex among noninfected women making it difficult to forecast a priori the bias in our individual-level regressions. In our community-level regressions, one possibility is that communities with higher infection rates also have more access to drugs (under the plausible scenario that health organizations concentrate their efforts in the most infected areas) and to the extent that the availability of these drugs reduces precautionary motive for protected sex; this would likely lead to a positive bias, confounding the true underlying negative effect of community-level HIV risk on individual behavior.

prevalence rate. We believe it is important to differentiate the responses of the infected and noninfected women since the ultimate effect on growth through the fertility channel will be determined by the behavior of the noninfected women.

3 Data

We use data from DHS, which are based on nationally representative samples. These surveys are designed to gather information on fertility and child mortality. Recent waves of these surveys have sought information on HIV/AIDS status by asking a subset of women who are interviewed to provide a few drops of blood for HIV testing. The collected blood specimens and the main surveys are linked by case identification numbers. The linked data are available for 13 out of the 16 countries who conducted the testing. Mali and Zambia have HIV data but cannot be linked to the main survey questions while Tanzanian survey does not include fertility questions. These countries were thus dropped from the analysis. While we can create individual-level panel data on fertility, we are limited in terms of information on HIV status since the testing was conducted only in one single year per country. Appendix Table A-1 summarizes the surveys used with those without asterisks denoting our main surveys containing information on HIV testing and those with asterisks denoting earlier surveys used in our community-level regressions. Table 1 provides summary statistics of our main data set which consists of women who are 15–49 years old from 13 countries with testing data. The table shows that 7 % of women in our sample are HIV-positive. Average years of schooling is slightly over 4 years and 63 % are currently married. Approximately one third of the sample lives in an urban

Table 1 Summary statistics

	No. of obs	Mean	SD.	Min	Max
Number of births last year	64,056	0.16	0.38	0	3
Number of births last five years	64,056	0.72	0.85	0	5
Number of children everborn	64,056	2.76	2.85	0	24
HIV status (1 = positive)	64,056	0.07	0.25	0	1
Age	64,056	28.12	9.49	15	49
Years of schooling	64,035	4.26	4.30	0	22
Never married	64,056	0.27	0.45	0	1
Currently married	64,056	0.63	0.48	0	1
Formerly married	64,056	0.09	0.29	0	1
Urban	64,056	0.34	0.47	0	1
Used condom in last intercourse	43965	0.09	0.29	0	1
Had more than one partner in last 12 months	48,016	0.08	0.27	0	1

Summary statistics are for women who are 15–49 years old from 13 countries with HIV testing data. HIV weights which adjust for individual sampling probabilities and test nonresponse rates are used in the calculations

Table 2 Effect of own HIV status on fertility

	Number of births last year OLS (1)	Number of births last year OLS (2)	Number of births last year OLS (3)	Number of births last 5 years OLS (4)	Number of births last 5 years OLS (5)	Number of births last 5 years OLS (6)
Positive HIV status	−0.043*	−0.042*	−0.032*	−0.177*	−0.176*	−0.145*
	(0.007)	(0.007)	(0.007)	(0.015)	(0.015)	(0.014)
Age	0.051*	0.050*	0.020*	0.263*	0.263*	0.164*
	(0.001)	(0.001)	(0.001)	(0.002)	(0.002)	(0.003)
Age^2	−0.001*	−0.001*	−0.000*	−0.004*	−0.004*	−0.003*
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Rural	0.069*	0.053*	0.036*	0.242*	0.177*	0.124*
	(0.004)	(0.004)	(0.004)	(0.009)	(0.009)	(0.008)
Primary education		−0.032*	−0.015*		−0.123*	−0.069*
		(0.005)	(0.005)		(0.011)	(0.010)
Secondary education		−0.080*	−0.039*		−0.310*	−0.179*
		(0.005)	(0.005)		(0.012)	(0.011)
Tertiary education		−0.116*	−0.052*		−0.552*	−0.345*
		(0.010)	(0.010)		(0.022)	(0.022)
Currently married			0.242*			0.785*
			(0.005)			(0.010)
Formerly married			0.140*			0.458*
			(0.007)			(0.014)
Constant	−0.505*	−0.462*	−0.178*	−2.909*	−2.749*	−1.829*
	(0.025)	(0.025)	(0.026)	(0.052)	(0.052)	(0.052)
R^2	0.067	0.072	0.114	0.271	0.287	0.374
N	64,056	64,056	64,056	64,056	64,056	64,056
Mean HIV-Positive	0.127	0.127	0.127	0.616	0.616	0.616
Mean HIV-Negative	0.167	0.167	0.167	0.722	0.722	0.722

Women with non-missing HIV status are used in the regressions. All regressions include country by region dummies. The omitted categories are: "No Education", "Urban, " and "Never Married". HIV weights which adjust for individual sampling probabilities and test nonresponse rates are used in the regressions. Robust standard errors are in the parentheses.

$*p < 0.05$ (significant)

area. We weight individual data with DHS, provided HIV weights which adjust for sampling probabilities, and test nonresponse rates. A detailed discussion of these weights is in Appendix A. Appendix Table A-3 also reports probit estimates on the determinants of HIV status.

4 Empirical results

4.1 Effects of own HIV status on fertility

Table 2 reports the effect of own HIV status on an individual woman's fertility. While it is difficult to identify the causal impact of HIV on women's fertility given the limits of our data, we nevertheless believe it is useful to examine

the cross-sectional relationship between positive HIV status and fertility.[10] It is also useful to examine the impact of various correlates which are arguably more endogenous, such as education and marital status. We begin with the following individual-level regression:

$$\text{Fertility}_{irc} = \alpha + \beta \text{OwnHIVStatus}_{irc} + \mathbf{X}'_{irc}\gamma + D_{rc} + D_{\text{rural}} + \epsilon_{irc}, \quad (1)$$

where i denotes the individual and rc, the community, which is unique by country and region. We use number of births in the last year and number of births in the last 5 years as our fertility variables.[11] Own HIVStatus$_{irc}$ is a dummy variable that takes a value of 1 if individual i in region rc is HIV positive, $\mathbf{X}_{irc}$ is a vector of other covariates, and ϵ_{irc} is a random error term. To begin, we include covariates which are arguably more predetermined, such as age, region, and urban/rural residence. Region dummies and rural dummy are denoted as D_{rc} and D_{rural} in the above equation.[12] In the next two specifications, we successively add education and marital status variables. Columns 1–3 in Table 2, refer to births last year while columns 4–6 refer to births in the last 5 years. The effect of HIV is negative and significant in all specifications. Column 1 indicates that positive HIV status lowers births last year by −0.043. Since the average is 0.167 births among noninfected women, this translates into a reduction of approximately 25–26 %.[13] As shown in column (2), the effect of HIV status is virtually unchanged when we control for education level but drops significantly when we control for marital status. As illustrated in column 3, positive HIV status lowers births last year by −0.032 which translates to a reduction of approximately 20 %. One interpretation of the difference between columns 1 and 3 is that HIV infection leads to changes in marital status, and women are more likely to become divorced or widowed—marital status categories associated with lower fertility rates. Another possibility, of course, is that differences in fertility, marital status, and HIV infection rates are driven by unobserved heterogeneity, an issue we address further below. Columns 4–6 using number of births over 5 years as the dependent variable shows basically similar results with the negative impact of

[10] One important limit of our data is that we observe HIV status at time t while our fertility variables refer to births last year or earlier. One of the implicit assumptions is that infection at time t is a reasonable proxy for infection in previous years.

[11] Each fertility measure has advantages and disadvantages. On the one hand, since HIV status refers to the survey year, number of births last year provides the closest match between treatment and outcome variables. On the other hand, number of births last year is more subject to idiosyncratic noise, and cumulative birth measures may be better indicators of an individual woman's total fertility. We have also investigated the effects for older women aged 35–49 who may be close to their desired fertility levels except for the marginal child. These results are reported in Appendix Table B-1. The table shows that the results are similar for this group of women.

[12] Urban/rural residence is arguably more endogenous due to migration. In practice, however, we find that including or excluding urban/rural residence has little impact on the size of the HIV coefficient.

[13] The preponderance of zeros as well as the nonnegative and discrete nature of the dependent variable suggests a Poisson specification may be more appropriate. Our Poisson estimates yielded very similar results and are available upon request.

Table 3 Effect of own HIV status on fertility history

	Survey year	5 years ago	10 years ago	15 years ago	20 years ago
Panel A: dependent variable: number of births last year					
Positive HIV Status	−0.017*	−0.027*	−0.007	−0.013	−0.008
	(0.008)	(0.011)	(0.014)	(0.015)	(0.013)
Mean	0.093	0.176	0.222	0.254	0.211
R^2	0.074	0.087	0.042	0.034	0.077
N	17696	17696	17696	17696	17696
Panel B: dependent variable: number of children ever born					
Positive HIV Status	−0.374*	−0.252*	−0.144*	−0.052	−0.014
	(0.085)	(0.078)	(0.066)	(0.051)	(0.036)
Mean	5.379	4.759	3.764	2.539	1.320
R^2	0.332	0.355	0.400	0.462	0.507
N	17696	17696	17696	17696	17696

Only women who are 35–49 and with HIV status are used in the regressions. In panel A, dependent variable is the births in previous year; in panel B, dependent variable is cumulative number of children born for each woman up to *N* years ago from the survey year. All regressions include country by region dummies. Other controls that are included are age, age squared, education, marital status, and urban/rural residence. The omitted categories are: "No Education," "Urban," and "Never Married". HIV weights which adjust for individual sampling probabilities and test nonresponse rates are used in the regressions. Robust standard errors are in the parentheses.

*$p < 0.05$ (significant)

HIV status being approximately 24 and 20 %s respectively in specification with and without marital status controls.[14]

The OLS estimation above assumes that controlling for observables, the error term ϵ_{irc} is uncorrelated with HIV status. This assumption may be violated if HIV-positive and HIV-negative women are systematically different in unobservable ways. While it is not possible for us to entirely rule out selection on unobservables given the limits of our data, we examine to what extent unobserved heterogeneity may be driving our results by exploiting fertility histories of women who are currently observed to have positive or negative HIV status. In Table 3 we use the fertility histories of older women (aged 35–49) and examine the effect of current HIV status on births 20, 15, 10, and 5 years ago as well as births last year. In the top panel, Panel A, the dependent variable is births last year in the indicated year. In the bottom panel, Panel B, the dependent variable is the cumulative number of births up to the indicated year. Since the spread of HIV/AIDS was negligible prior to 1986, we would not expect a significant difference in births 20 years ago as a function

[14] One concern is that there is insufficient overlap in the distribution of covariates. To investigate this issue, we estimated impact of own HIV status on births using propensity score matching. We use the propensity score from the probit estimation (results reported in Appendix Table A-3) and use one-to-one nearest neighbor matching without replacement. We implemented the STATA 9 procedure developed and described in Leuven and Sianesi (2003). We also conducted simple *t* test on differences in characteristics between HIV-positive and HIV-negative women in our matched sample and did not find statistically significant differences. The results are reported in Appendix Table B-2. The negative impact of HIV is slightly smaller ranging between 15 and 17 %.

Table 4 Effect of own HIV status on fertility, controlling for number of partners, and condom use

	Number of births last year (1)	Number of births last 5 years (2)
Positive HIV Status	−0.037*	−0.161*
	(0.008)	(0.017)
Condom Use	−0.032*	−0.070*
	(0.007)	(0.015)
More than one partner	−0.070*	−0.203*
	(0.008)	(0.018)
R^2	0.089	0.294
N	43965	43965

Regressions use all women with non-missing HIV status. All regressions include country by region dummies. Other controls that are included are age, age squared, education, marital status and urban/rural residence. The omitted categories are: "No Education", "Urban", "Never Married", "Did not use a condom during last intercourse", and "Did not have more than one partner in last 12 months". HIV weights which adjust for individual sampling probabilities and test nonresponse rates are used in the regressions. Robust standard errors are in the parentheses.

(* $= p < 0.05$ (significant)

of current HIV status. Table 3 shows that there is no significant difference between HIV- positive and HIV-negative women in births 20 years ago. The difference in fertility of HIV-positive and HIV-negative women, however, becomes more pronounced as the disease spreads over time. To address the concern that some women were too young 20 years ago to have pronounced differences in fertility behavior, we have also run the same regression using older women who are 20–29 years old 20 years ago. Among these women, the coefficient on current positive HIV status on children ever born 20 years ago is −0.038 with standard error of (0.060). Not only is the coefficient not significant, since the mean number of children ever born to these women is 1.97, the size of the coefficient signifies a trivial difference. These results suggest that unobserved heterogeneity is not the major driving force behind the negative effect of HIV on women's fertility.[15]

It is not clear to what extent the own effect reflects physiological impact of the disease versus behavioral response among the infected women. In Table 4 we explore whether including various measures of sexual behavior impacts the coefficient on HIV status. We repeat the same regressions as in Table 2 but include an indicator variable for using a condom during last intercourse

[15] Another concern is survivor bias. It may be the case that the HIV-positive women in Table 3 are not a representative sample of all women who ever contracted the virus and that women who contracted the disease earlier had already died. For there to be negative effect on fertility that is due to survivor bias, however, the HIV-positive women who died must have had *higher* fertility relative to even women who are HIV-negative. There is little to indicate that women who were early contractors of the disease would have had higher than average fertility. For example, those who are more likely to contract the disease are better educated and urban, characteristics that are associated with lower than average fertility.

and an indicator variable for having more than one partner during previous 12 months. The significant negative effect of positive HIV status remains even when we control for these sexual behavior variables, suggesting that the physiological impact of the disease is important as suggested by Gray et. al (1998). Our estimate in Table 4, which implies a reduction of 23 %, is smaller than the estimates reported in Gray et. al (1998). However, when we restrict our sample to closely resemble theirs; our estimate becomes larger to about 36 %.[16]

4.2 Effects of community HIV prevalence on fertility

Results from the previous section showed that at the individual level, being infected with the HIV virus significantly lowers fertility. We are also interested in how fertility responds to increased mortality risk, a central concern in growth models. To gauge this response, we examine the impact of community-level HIV prevalence on fertility of noninfected women. A "community" in our analysis is a country by region cell.[17] We employ two alternative strategies. First, we run OLS regressions using only those surveys where actual HIV testing data is available. As an alternative strategy, we also use earlier waves of the DHS to build community-level panel data. More specifically, for the OLS specification, we run the following regression on women who are HIV negative:

$$\text{Fertility}_{irc} = \alpha' + \beta' \text{CommunityHIV}_{rc} + \mathbf{X}'_{irc}\gamma' + D_c + D_{\text{rural}} + \epsilon'_{irc}, \qquad (2)$$

Community HIV is defined as the fraction of all adults 15–49 (both men and women) with positive HIV status in the region. Since we control for country dummies in the above regression, D_c, we are identifying the community HIV effect from cross-regional differences in HIV prevalence and fertility within countries.

As an alternative strategy, we follow the methodology introduced by Young (2005) and utilize fertility histories to construct fertility by region and year. We introduce time variation in community-level HIV prevalence by assuming zero prevalence in the years prior to 1986. This strategy was used by Fortson (2011) to estimate the impact of community-level HIV prevalence on educational outcomes. In a recent paper, Fortson (2009) also utilizes the same strategy. More specifically, we run the following regression:

$$\text{Fertility}_{irct} = \alpha + \beta \text{CommunityHIV}_{rct} + \mathbf{X}'_{irct}\gamma + D_{rc} + \phi_t + \epsilon_{irct} \qquad (3)$$

[16] In results we do not report, we have run the same regression as in Table 4 but on a sample of women who reported positively to "ever had intercourse," who reported never being tested for HIV and who lived in rural areas where regional HIV prevalence exceeded 15 %.

[17] In a previous version, we defined a community as a country by region by urban/rural residence cell. However, DHS samples are not representative at the disaggregated level. We therefore use country by region cell to define communities in this version while still controlling for urban/rural residence. We thank Jane Fortson for pointing this out to us.

where t refers to year at birth and refers to two periods, 1981–1985 and 2001–2005. To obtain a more representative sample of women in the earlier period, we utilize earlier waves of the DHS that were conducted for the countries in the sample and build a community-level panel data set. Exact details on the countries and surveys used are outlined in Appendix Table A-1. Rather than making assumptions about the time path of HIV, we focus on the change from the 1981–1985 to 2001–2005 period and use data only from those years. While we include only HIV-negative women in the later years, testing data are not available in the earlier waves, and we are unable to distinguish HIV-positive and HIV-negative women. HIV prevalence rates are close to zero in years prior to 1985; however, this is not likely to seriously bias our results. We control for individual characteristics such as education, ever married dummy at time of birth, and age of the woman at birth. We include country–year fixed effects and community (region) fixed effects, D_{rc}, time effects, and age by time interactions in this specification. HIV prevalence varies by community and is assumed to be zero for all communities in 1981–1985. Controlling for other covariates, the coefficient β measures whether fertility increased or decreased in communities with larger increases in HIV prevalence.

Before turning to the results, we report some descriptive statistics of communities in Table 5. Panel A refers to the surveys with HIV testing data used in our cross-sectional regressions. Panel B refers to all the surveys used in our community fixed effects regressions. As Table 5 shows, community-level HIV prevalence ranges from 0 to 29 % with the average being 5.7 %. Note

Table 5 Descriptive statistics of communities

	Mean	Standard deviation	Min	Max
Panel A:	Number of Communities N=128			
Number of women	1,389.34	1,175.36	351	5,902
Number of births last year	0.17	0.05	0.05	0.30
Number of births last 5 years	0.74	0.22	0.23	1.28
HIV prevalence	0.057	0.069	0.000	0.288
Know someone with or died of AIDS	0.34	0.25	0.02	0.90
Number of communities per country	10.53	2.10	3.00	14.00
Panel B:	Number of Communities N=98			
Number of women	2,965.41	2,419.75	437	14,228
Number of births last year	0.17	0.04	0.05	0.28
Number of births last 5 years	0.72	0.19	0.23	1.23
HIV prevalence	0.080	0.087	0.000	0.314
Know someone with or died of AIDS	0.35	0.26	0.03	0.89
Number of communities per country	7.54	3.04	3.00	12.00
Number of year obs per community	9.26	1.26	3.00	10.00

Panel A reports the statistics for the single latest survey that includes the HIV testing. Panel B reports statistics from multiple surveys. "Community" refers to a country by region cell. For births, prevalence, and knowledge variables, we first calculated weighted community-level averages using the HIV weights and the table reports summary statistics across communities. HIV prevalence is based on both men and women while birth and knowledge variables refer to women with non-missing HIV status only.

Table 6 Effect of community HIV prevalence on fertility

	Number of births last year OLS (1)	Number of births last 5 years OLS (2)	Number of births last year fixed effects (3)	Number of births last 5 years fixed effects (4)
Community HIV prev.	0.113	−0.210	0.120	0.299
	(0.088)	(0.279)	(0.078)	(0.376)
Country fixed effects	Yes	Yes	–	–
Country-year fixed effects	No	No	Yes	Yes
Region fixed effects	No	No	Yes	Yes
R^2	0.116	0.382	0.071	0.304
N	59579	59579	576172	576172

Women with negative HIV status are included in the regressions."Community HIV Prevalence" refers to the fraction of men and women with positive HIV status in the region, excluding the woman herself. Columns 1 and 2 control for age, age-squared, education, marital status, urban/rural residence, wealth quintile category and country dummies. In columns 3 and 4 Burkina Faso (2003, 1998, 1992), Cameroon (2004, 1998, 1991), Cote d'Ivoire (2005, 1994), Ethiopia (2005, 2000), Ghana (2003, 1998, 1993, 1988), Guinea (2005, 1999), Kenya (2003, 1998, 1993, 1989), Malawi (2004, 2000, 1992), Niger (2006, 1998, 1992), Rwanda (2005, 2000), Senegal (2005, 1997, 1992), and Zimbabwe(2005, 1999, 1994, 1988) are used; Lesotho is not used since it does not have an earlier cross section. In columns 3 and 4, surveys are used to construct birth histories for two periods, 1981–1985 and 2001-2005; dependent variable is the number of births last year or last 5 years; HIV Prevalence is assumed to be zero before 1985; omitted categories are "1981–1985", "Ages 25–29", "No education", "Not married"; and education, marital status at birth, age-group dummies, period dummies, age group by time interactions, year by country fixed effects, country by region dummies, and rural dummy are included in the regressions. HIV weights which adjust for individual sampling probabilities and test nonresponse rates are used in the regressions. Robust standard errors clustered at the country level are in the parentheses. ($^*p < 0.05$)

that we also include men in measuring community-level prevalence, and since men's infection rates are lower than women's, we end up with lower average prevalence rate than the 7.0 % reported in Table 1.[18]

We report the impact of community-level HIV prevalence on noninfected women in Table 6. Columns 1 and 2 refer to our OLS estimates, while columns 3 and 4 refer to community-level fixed-effect regressions. As reported in columns 1 and 2, the sign on the community HIV effect switches from being positive for birth last year to being negative for births last 5 years. The standard errors are large, however, so that we cannot rule out either a positive or a negative effect. The estimates based on fixed effects regressions in column (3) and (4) are positive but not statistically significant. Overall, our estimate of the impact of HIV on total fertility rate is considerably smaller than reported in Young (2005). His estimates suggest that a 100 % community prevalence would reduce fertility by 80 % (see Young (2005)). Three out of the four

[18]The numbers in Table 5 may also differ from those in Table 1 due to the fact that we report unweighted averages across communities in Table 5.

estimates reported in Table 6 are positive in sign and given the standard errors, we can rule out negative effects and particularly large negative effects.[19]

The absence of a behavioral response among the noninfected women is consistent with recent findings in Oster (2005), among others, who document relatively little change in sexual behavior in response to HIV. Oster (2005) suggests that the relatively little response in sexual behavior may be in part explained by low levels of knowledge about the disease. To investigate whether an alternative measure which better captures knowledge and *perceived* risk produces different results, we use the share of individuals who report knowing someone with AIDS or someone who died of AIDS as our independent variable. These results are reported in Appendix Table B-3. The coefficient on this knowledge variable is not significantly different from zero.

5 The impact of HIV on the total fertility rate

Assuming that HIV has a zero impact on noninfected women, what is the impact of the infected women on the total fertility rate? The basic answer to this question was already relayed in Table 2 where we found that positive-HIV status reduced births last year by approximately 20 %. However, in the following table, we put this in the context of the fertility levels and HIV prevalence rates of each country. The top row of Table 7 reports the HIV prevalence rate for each country based on the HIV testing sample. The second row reports the total fertility rate (TFR) calculated from age-specific birth rates of all women with HIV status. The third row calculates the TFR using age-specific birth rates of HIV-negative women only. Finally, the last row corrects for differences in observable characteristics such as age, education, and marital status since our earlier tables showed differences in these characteristics between the HIV-positive and HIV-negative populations.[20] Table 7 shows a wide range for the total fertility rates among the countries in our sample with TFR ranging from

[19]The one coefficient that is negative in sign (column 3) has a 95 % confidence interval of −0.489 and 0.069. Since the average number of births in the last 5 years is 0.722, the largest negative effect we estimate is a reduction of approximately 67 % (−0.489/0.722) which is still smaller than the coefficient in Young (2005).

[20]To calculate the TFR for our sample of women with HIV status instead of all the women in DHS survey sample, we follow the method used by the DHS, which uses information on births over the last 36 months for each woman based on the fertility histories. The numerator of each age-specific birth rate is the total number of births over the previous 36 months for women in each 5-year age category based on age at birth. The denominator is the total number of women-years in each 5-year age category. Then we summed up all the age-specific fertility rates and multiply it by 5 (since each woman is present in each age group for 5 years) to end up with the TFR as done by DHS. To adjust TFR for differences in observable characteristics between all and negative HIV women, we run the fertility regression pooling HIV-positive and HIV-negative women as specified in Eq. 1, predict fertility by age group, and add back residuals for HIV-negative women.

Table 7 HIV/AIDS impact on total fertility rate

	Burkina Faso	Cameroon	Cote d'Ivoire	Ethiopia	Ghana	Guinea	Kenya	Lesotho	Malawi	Niger	Rwanda	Senegal	Zimbabwe	AVERAGE
HIV prevalence (%)	1.8	6.6	4.7	1.9	2.7	1.9	8.7	26.4	13.3	0.7	3.6	0.9	21.1	7.3
All women	5.903	5.119	2.416	5.719	4.558	5.758	4.759	3.624	6.249	7.065	6.062	5.101	3.823	5.089
HIV negative women	5.943	5.289	2.407	5.751	4.593	5.825	4.887	3.932	6.634	7.089	6.168	5.126	4.084	5.210
HIV negative women correcting for observables	5.922	5.207	2.369	5.718	4.580	5.788	4.825	3.777	6.522	7.079	6.112	5.121	3.925	5.150

The total fertility rate is an age-period fertility rate for a synthetic cohort of women. It gives the average number of births that women in the sample would have by the time they reach age 49 if they were to give birth at the current age-specific fertility rates. It is the sum of the age-specific fertility rates for all women multiplied by five. These rates are calculated using birth histories of each women in last 36 months and weighted using HIV sample weights. Row 1 gives the HIV prevalence rates in the survey year. Row 2 reports the TFR for women with HIV status and row 3 reports rates for HIV-negative women. Row 4 is TFR for HIV-negative women after correcting for their observable characteristics that may be associated with higher fertility. Retrospective fertilities are regressed on Xs using both HIV-negative and HIV-positive women, and predicted values are obtained by age group at the time of birth, and each HIV-negative woman's residual is added to her age group's predicted value

the low of 2.4 for Cote d'Ivoire to 7.1 for Niger. Comparing rows 2 and 3, we see that there is virtually no impact on the aggregate fertility rate for countries with very low HIV prevalence rates. Even for high prevalence countries, such as Lesotho, Malawi, Zimbabwe, and Kenya, the total impact is relatively small. For example, for the highest prevalence country, Lesotho, which has a prevalence rate of 26.4 %, births would increase by 0.31 if all women were HIV-negative. As expected, the correction for observable characteristics dampens the fertility differences between infected and noninfected women and TFR would be only 0.15 higher with the correction. Table 7 illustrates that without a large behavioral response among the noninfected women, the effect of HIV on aggregate fertility rate will be small and nowhere near the large negative impact reported in Young (2005).

6 Conclusion

A body of theoretical models imply that fertility responds positively to a rise in mortality risk, either by reducing the returns to adult human capital or by inducing a precautionary demand for children. The special case of HIV/AIDS however suggests that fertility may decrease, first through direct physiological reasons, and second, through changes in sexual behavior and the reduction in willingness to engage in unprotected sex. The effect of HIV on fertility is a key to evaluating the aggregate impact of the disease on economic development.

In our empirical work, we attempt to separate out the physiological and behavioral responses to the disease by distinguishing between the effect of *own* HIV status versus the effect of mortality risk as measured by the community-level prevalence rate. We argue that it is important to distinguish these two effects since behavioral responses of noninfected women can further reinforce or possibly mitigate the population declines brought on by the disease. We undertake this exercise using individual-level HIV testing data that have recently become available.

Our results show that infected women are significantly less likely to give births than noninfected women. The probability of giving births in the previous year is approximately 17–20 % lower. Robustness checks imply that these results are not driven by unobserved heterogeneity or different sexual behavior among the HIV-positive women. Our interpretation is that the disease has a significant negative effect on infected women's fertility, a large part of which may be physiological. In contrast to Young (2005, 2007), however, we find no significant impact of community-level infection rates on fertility of noninfected women. Will the fertility responses to HIV reinforce or offset the declines in population due to mortality? Our results suggest that only fertility of infected women will decline, and hence the total impact of HIV on the aggregate economy is much smaller than the effect implied by Young (2005, 2007). Together with the results from other papers that document substantial declines in human capital accumulation, the results here suggest that HIV/AIDS is likely to decrease rather than increase future per capita incomes in Africa.

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