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 allows 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 non-infected women's fertility.

JEL Codes: O12, I12, J13
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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.[1] The empirical studies using economic growth as an outcome show mixed results.[2] 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.[3]

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 without knowing the response of fertility to the disease we cannot answer the question of the effect of the disease on development. 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

[1] See Corrigan, Gloom, and Mendez (2005), Manuelli (2010).

[2] 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 (2008) find negative effect on the level of income per capita in a similar framework. 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.

[3] Kalemli-Ozcan and Turan (2010) 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.

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 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 *non-infected* 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 to 25 percent, 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.[4] The OLS estimation assumes that HIV positive and negative women are comparable once we control observable characteristics. This assumption may be violated if HIV positive and 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 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 non-infected women on the local community HIV prevalence rate and find no significant effect.

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

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.[5]

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 percent prevalence would have fertility that is approximately 80 percent 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 percent lower. Given that country level prevalence rates fall well below 100 percent, 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 percent), the total fertility rate would be 0.15 to 0.3 children higher (approximately 4-8 percent) in the absence of HIV/AIDS.[6]

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. Section 6 concludes.

[5]The community level results were produced independently and at the same time in an earlier version of our paper, Juhn, Kalemli-Ozcan, and Turan (2008) and a working paper version of Fortson (2009).

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

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. 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.[7] 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 initial rise in population size.[8] The HIV/AIDS epidemic has generated a negative shock to life expectancy which, according to these models, should *increase* fertility.[9]

Treating HIV/AIDS simply as a shock to adult longevity may be overly simplified, however. First, field evidence strongly suggests that there is a direct biological/physiological

[7]See, for example, Cervellati and Sunde (2007), Tamura (2006), Soares (2005), Kalemli-Ozcan (2003), Galor and Weil (2000), Lucas (2000), and Ehrlich and Lui (1991).

[8]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.

[9]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 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 (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 (2007), using data similar to ours, 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.

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 percent) 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 percent. Carpenter et al. (1997) and Hunter et al. (2003), in cohort studies in Uganda and Tanzania, respectively, find a 30–40 percent reduction in probability of becoming pregnant. 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.[10]

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), Caldwell et al. (1999) all find no 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 non-marital partners as single men. One would expect the number of non-marital partners to fall more for the married men if unprotected sexual activity is an issue or if wives could influence husband's extra-marital 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

[10]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 percent 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.

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 percent 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.[11]

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 prevalence rate. We believe it

[11]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 non-infected 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.

is important to differentiate the responses of the infected and non-infected women since the ultimate effect on growth through the fertility channel will be determined by the behavior of the non-infected women.

3 Data

We use data from Demographic and Health Surveys (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 percent of women in our sample are HIV positive. Average years of schooling is slightly over 4 years and 63 percent are currently married. Approximately one third of the sample lives in an urban area. We weight individual data with DHS provided HIV-weights which adjust for sampling probabilities and test non-response 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.[12] 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:

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

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.[13] 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 pre-determined, 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.[14] In the next two specifications, we successively add education and marital status variables. Columns (1) to (3) in Table 2, refer to births last year while columns (4) to (6) refer to births in the last 5 years. The effect of HIV is

[12] 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.

[13] 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.

[14] 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.

negative and significant in all specifications. Column (1) indicates that positive HIV status lowers births last year by -.043. Since the average is .167 births among non-infected women, this translates into a reduction of approximately 25-26 percent.[15] 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 -.032 which translates to a reduction of approximately 20 percent. 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) to (6) using number of births over 5 years as the dependent variable shows basically similar results with the negative impact of HIV status being approximately 24 and 20 percents respectively in specification with and without marital status controls.[16]

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 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

[15]The preponderance of zeros as well as the non-negative 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.

[16]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-tests on differences in characteristics between HIV positive and 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 15-17 percent.

status on births 20, 15, 10, 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 of current HIV status. Table 3 shows that there is no significant difference between HIV positive and negative women in births 20 years ago. The difference in fertility of HIV positive and 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 -.038 with standard error of (.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.[17]

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 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 percent, is smaller than the estimates reported in Gray et. al (1998).

[17]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.

However, when we restrict our sample to closely resemble theirs, our estimate becomes larger to about 36 percent.[18]

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 non-infected women. A "community" in our analysis is a country by region cell.[19] 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:

$$Fertility_{irc} = \alpha' + \beta' CommunityHIV_{rc} + \mathbf{X}'_{irc}\gamma' + D_c + D_{rural} + \epsilon'_{irc}, \tag{2}$$

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 (2007) to estimate the impact of community-level HIV

[18] 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 percent.

[19] 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.

prevalence on educational outcomes. A recent paper, Fortson (2009) also utilizes the same strategy. More specifically, we run the following regression:

$$Fertility_{irct} = \alpha + \beta CommunityHIV_{rct} + \mathbf{X}'_{irct}\gamma + D_{rc} + \phi_t + \epsilon_{irct} \quad (3)$$

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 make 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 is not available in the earlier waves and we are unable to distinguish HIV positive and negative women. Since, 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 percent with the average being 5.7 percent. Note 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 percent reported in table 1.[20]

[20]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.

We report the impact of community level HIV prevalence on non-infected 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 percent community prevalence would reduce fertility by 80 percent.[21] Three out of the four 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.[22]

The absence of a behavioral response among the non-infected 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 non-infected women, what is the impact of the infected women on the total fertility rate? The basic answer to this question was already

[21] See Young (2005), footnote 40.

[22] The one coefficient that is negative in sign (column 3) has a 95 percent confidence interval of -.489 and .069. Since the average number of births in last 5 years is .722, the largest negative effect we estimate is a reduction of approximately 67 percent (-.489/.722) which is still smaller than the coefficient in Young (2005).

relayed in tables 2 where we found that positive HIV status reduced births last year by approximately 20 percent. 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 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.[23] Table 7 shows a wide range for the total fertility rates among the countries in our sample with TFR ranging from 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 percent, births would increase by .31 if all women were HIV negative. As expected the correction for observable characteristics dampens the fertility differences between infected and non-infected women and TFR would be only .15 higher with the correction. Table 7 illustrates that without a large behavioral response among the non-infected women, the effect of HIV on aggregate fertility rate will be small and nowhere near the large negative impact reported in Young (2005).

[23]To calculate the total fertility rate (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 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 negative women as specified in equation (1), predict fertility by age-group and add back residuals for HIV negative women.

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 non-infected 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 non-infected women. The probability of giving births in the previous year is approximately 17 to 20 percent 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 non-infected 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) and (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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Table 1: Summary Statistics

	No. of Obs	Mean	Std. Dev.	Min	Max
Number of Births Last Year	64056	0.16	0.38	0	3
Number of Births Last 5 Years	64056	0.72	0.85	0	5
Number of Children Everborn	64056	2.76	2.85	0	24
HIV Status (1 = Positive)	64056	0.07	0.25	0	1
Age	64056	28.12	9.49	15	49
Years of Schooling	64035	4.26	4.30	0	22
Never Married	64056	0.27	0.45	0	1
Currently Married	64056	0.63	0.48	0	1
Formerly Married	64056	0.09	0.29	0	1
Urban	64056	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	48016	0.08	0.27	0	1

Notes: 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 non-response 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	64056	64056	64056	64056	64056	64056
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

Notes: 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 non-response rates are used in the regressions. Robust standard errors are in the parentheses. Asterisk denotes significance levels (* = p-value < .05).

Table 3: Effect of Own HIV Status on Fertility History

	Panel A: Dependent Variable: Number of Births Last Year				
	Survey Year	5 Years Ago	10 Years Ago	15 Years Ago	20 Years Ago
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				
	Survey Year	5 Years Ago	10 Years Ago	15 Years Ago	20 Years Ago
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

Notes: 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 non-response rates are used in the regressions. Robust standard errors are in the parentheses. Asterisk denotes significance levels (* = p-value $< .05$).

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

Notes: 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 non-response rates are used in the regressions. Robust standard errors are in the parentheses. Asterisk denotes significance levels (* = p-value $< .05$).

Table 5: Descriptive Statistics of Communities

	Mean	Standard Deviation	Min	Max
Panel A:	Number of Communities N=128			
Number of Women	1389.34	1175.36	351	5902
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	2965.41	2419.75	437	14228
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

Notes: 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

Notes: 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 non-response rates are used in the regressions. Robust standard errors clustered at the country level are in the parentheses. Asterisk denotes significance levels (* = p-value $< .05$).

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

Notes: The total fertility rate (TFR) 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.

Appendix A

Table A-1 summaries the countries and years used in our analysis. For the most part our analysis uses the surveys where HIV testing data are available. For regional analysis in table 6, however, we also include surveys from earlier years in order to construct a panel of regional data. These surveys are marked with an asterisk. Table A-2 compares the country-level HIV prevalence rates among 15–49 year old women from the DHS with HIV prevalence rates from other data sources. Column (1) presents the DHS data. Rates in column (3) are from UNAIDS and rates in column (4) are from the U.S. Census Bureau's HIV Surveillance Database. Column (5) presents U.S. Census Bureau's projections using the Estimation and Projections Package (EPP) from WHO/UNAIDS. EPP estimates HIV trends by fitting an epidemiological model to the surveillance data. The other sources largely rely on HIV prevalence among pregnant women attending pre-natal clinics, a sample which is unlikely to be geographically or demographically representative of the population (Timberg (2006)). The table shows that country level prevalence from other data sources are generally higher than those we estimate from the population based samples in the DHS. In our analysis we use HIV weights provided by the DHS which adjust for individual sampling probabilities and test non-response rates. According to DHS reports, the sample is selected with unequal probability to expand the number of cases available for certain areas or subgroups for which statistics are needed. When weights are calculated because of sample design, corrections for differential response rates are also made. There are two main sampling weights in the DHS surveys: household weights and individual weights. The household weight for a particular household is the inverse of its household selection probability multiplied by the inverse of the household response rate of its household response rate group. The individual weight of a respondent's case is the household weight multiplied by the inverse of the individual response rate of the individual response rate group. Additional sampling weights such as HIV testing are included when there is a differential probability in selecting subsamples.[24] There is some concern that

[24]Source: DHS reports, www.measureddhs.com.

prevalence rates from the DHS may be too low since some women refused to be tested or were missing during the time of the survey. In comparing the observable characteristics of respondents and non-respondents, we found that non-respondents were more likely to be educated and less likely to be living in rural areas, suggesting the importance of controlling for these observable characteristics in our analysis (Juhn, Kalemli-Ozcan, Turan (2008)). Mishra et al. (2006), however, report that the non-response bias is not significant for estimating national HIV prevalence rates. Finally table A-3 explores the determinants of HIV status. The table reports the marginal probabilites and associated standard errors from a probit regression with HIV status as the dependent variable. The table shows that education is strongly related to HIV status but the relationship is non-linear with those with "No Education" having the lowest infection rates. The relationship has an inverted-U shape with infection rates turning negative again at the highest education level, "Tertiary Education." "Formerly Married" women who are widowed or divorced have higher infection rates relative to "Never Married" and "Currently Married" women. Positive HIV status also varies by residence type with those in rural areas having lower infection rates.

Table A-1: Summary of Surveys Used

Country	Number of Survey Years	Survey Years
Burkina Faso	3	2003, 1998*, 1992*
Cameroon	3	2004, 1998*, 1991*
Cote d'Ivoire	2	2005, 1994*
Ethiopia	2	2005, 2000*
Ghana	4	2003, 1998*, 1993*, 1988*
Guinea	2	2005, 1999*
Kenya	4	2003, 1998*, 1993*, 1989*
Lesotho	1	2004
Malawi	3	2004, 2000*, 1992*
Niger	3	2006, 1998*, 1992*
Rwanda	2	2005, 2000*
Senegal	3	2005, 1996*, 1992*
Zimbabwe	4	2005, 1999*, 1994*, 1988*

Notes: Our dataset consists of 36 surveys from 13 countries. * indicates that the survey is used for regional analysis in table 10 to construct a panel of regional data.

Table A-2: HIV Prevalence Rates Across Countries: Different Sources

Country	DHS (1)	Survey Year (2)	UNAIDS/WHO (3)	US Census (4)	EPP (5)
Burkina Faso	1.8	2003	2.9	4.1	4.2
Cameroon	6.6	2004	9.1	8.6	6.9
Cote d'Ivoire	4.7	2005	9.1	3.0	7.0
Ethiopia	1.9	2005	9.6	8.6	4.7
Ghana	2.7	2003	3.7	1.7	2.2
Guinea	1.9	2005	4.2	4.4	3.6
Kenya	8.7	2003	12.0	11.1	6.7
Lesotho	26.4	2004	31.0	28.0	28.7
Malawi	13.3	2004	18.0	18.0	14.1
Mali	1.8	2001	5.8	5.8	2.0
Niger	0.7	2005	2.3	2.9	1.4
Rwanda	3.6	2005	8.3	5.4	5.1
Senegal	0.9	2005	1.7	0.8	0.9
Tanzania	6.3	2003	8.1	17.5	8.6
Zambia	19.7	2001/2002	25.6	19.6	15.8
Zimbabwe	21.1	2005/2006	21.1	21.6	24.6

Notes: Rates shown in column (1) are calculated using DHS HIV data including women ages 15–49 and weighted using HIV survey sample weights. Survey years are for Burkina Faso (2003), Cameroon (2004), Cote d'Ivoire (2005), Ethiopia (2005), Ghana (2003), Guinea (2005), Kenya (2003), Lesotho (2004), Malawi (2004), Mali (2001), Niger (2005), Rwanda (2005), Senegal (2005), Tanzania (2003) and Zambia (2001/2002), Zimbabwe (2005/2006). Column 2 reports the survey years. In columns (3)-(5), prevalence rates among pregnant women are reported and survey years are matched when available, otherwise the rates for nearby years are reported. Rates in column (3) are from UNAIDS/WHO Epidemiological Fact Sheets. In column (3), for Niger 2000, for Ghana 2002, for Cameroon, Ethiopia, Lesotho and Rwanda 2003, and for Cote d'Ivoire, Guinea and Zimbabwe 2004 HIV prevalence rates are reported. Column (4) is from US Census Bureau's HIV Surveillance Database (2006). In column (4), for Niger reported rate is for 2000, for Cote d'Ivoire, Cameroon, Ethiopia, Lesotho and Rwanda reported rates are for 2003, for Zimbabwe reported rate is for 2004. Column (5) presents US Census Bureau's projections using the Estimation and Projections Package (EPP) from WHO/UNAIDS. In column (5) all survey years are matched. Since HIV data for Mali and Zambia cannot be linked to main survey, and Tanzania survey does not contain fertility variables they are not used in the regressions, but prevalence rates are presented here for comparison purposes.

Table A-3: Determinants of HIV Status: Probit Regression

Age	0.014*
	(0.001)
Age^2	–0.000*
	(0.000)
Primary Education	0.013*
	(0.003)
Secondary Education	0.012*
	(0.003)
Tertiary Education	–0.009*
	(0.004)
Currently Married	0.004
	(0.002)
Formerly Married	0.078*
	(0.007)
Rural	–0.018*
	(0.002)
Pseudo R^2	0.239
N	63904

Notes: Country and region dummies are included in the regression. The omitted categories are: "No Education", "Urban " and "Never Married". The table reports marginal probabilities and associated standard errors. HIV weights which adjust for individual sampling probabilities and test non-response rates are used in the regressions. Robust standard errors are in the parentheses. Asterisk denotes significance levels (* = p-value $< .05$).

Appendix B

Table B-1: Effect of Own HIV Status on Fertility: Women aged 35–49

	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	-0.038*	-0.037*	-0.017*	-0.199*	-0.194*	-0.109*
	(0.008)	(0.008)	(0.008)	(0.021)	(0.021)	(0.021)
Age	-0.058*	-0.058*	-0.057*	-0.072*	-0.073*	-0.070*
	(0.014)	(0.014)	(0.014)	(0.031)	(0.031)	(0.031)
Age^2	0.001*	0.001*	0.000*	0.000	0.000	0.000
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Rural	0.046*	0.039*	0.035*	0.203*	0.169*	0.151*
	(0.007)	(0.007)	(0.007)	(0.017)	(0.017)	(0.017)
Primary Education		-0.008	-0.007		-0.049*	-0.043*
		(0.008)	(0.008)		(0.019)	(0.019)
Secondary Education		-0.043*	-0.040*		-0.204*	-0.192*
		(0.009)	(0.009)		(0.021)	(0.021)
Tertiary Education		-0.058*	-0.059*		-0.227*	-0.229*
		(0.013)	(0.013)		(0.039)	(0.040)
Currently Married			0.081*			0.428*
			(0.011)			(0.032)
Formerly Married			0.010			0.132*
			(0.011)			(0.033)
Constant	1.588*	1.611*	1.513*	3.301*	3.412*	2.917*
	(0.301)	(0.300)	(0.299)	(0.657)	(0.653)	(0.647)
R^2	0.080	0.082	0.089	0.219	0.225	0.246
N	17696	17696	17696	17696	17696	17696
Mean HIV-Positive	0.057	0.057	0.057	0.347	0.347	0.347
Mean HIV-Negative	0.111	0.111	0.111	0.632	0.632	0.632

Notes: Women aged 35–49 and 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 non-response rates are used in the regressions. Robust standard errors are in the parentheses. Asterisk denotes significance levels (* = p-value $< .05$).

Table B-2: Effect of Own HIV Status on Fertility: Propensity Score Matching

	Number of Births Last Year (1)	Number of Births Last 3 Years (2)	Number of Births Last 5 Years (3)
Positive HIV Status	–0.027*	–0.069*	–0.105*
	(0.008)	(0.012)	(0.017)
Treatment Group	0.133	0.359	0.633
Control Group	0.160	0.427	0.737
Number of Matched Pairs	4474	4474	4474

Notes: Women are matched (one-to-one, without replacement) to their nearest-neighbor based on age, age-squared, education, marital status, wealth index, residence type and number of living children, country and region. Asterisk denotes significance levels (* = p-value $< .05$).

Table B-3: Perceived HIV Risk: Share of Individuals Knowing Someone with AIDS

	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)
Knowing Someone with AIDS	0.012	0.022	0.042	0.062
	(0.022)	(0.091)	(0.044)	(0.200)
Country fixed effects	Yes	Yes	-	-
Country-year fixed effects	No	No	Yes	Yes
Region fixed effects	No	No	Yes	Yes
R^2	0.115	0.380	0.071	0.304
N	59579	59579	576172	576172

Notes: Women with negative HIV status are included in the regressions."Knowing Someone with AIDS" refers to the fraction of women know someone with or died of AIDS in the region. 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; knowing someone with AIDS 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 non-response rates are used in the regressions. Robust standard errors clustered at the country level are in the parentheses. Asterisk denotes significance levels (* = p-value $< .05$).