HIV and Fertility in Africa: First Evidence from Population Based Surveys*

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Abstract

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 HIV-infected women have significantly lower fertility. In contrast to Young (2005), however, we find that 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

The scope of the worldwide AIDS epidemic is staggering. As of 2008 there were an estimated 32 million people living with HIV/AIDS, with more than 90 percent of the infected people living in developing countries. Africa alone accounts for two-thirds of the world total and almost all of the infected children. The epidemic has altered the patterns of morbidity and mortality tremendously. What is the impact of HIV/AIDS epidemic on the future development of these affected countries?[1]

Drawing a parallel between AIDS and the "Black Death", Young (2005) suggests that population declines will lead to higher capital-labor ratios and eventually to higher per capita income in the affected countries. While the epidemic will have a detrimental impact on human capital accumulation, 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]

Our study uses newly available micro data from population based surveys to examine the fertility response to HIV/AIDS. 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. The advantages of this

[1]Recent empirical papers show results that vary extensively. While most of the researchers find negative effects of the epidemic on economic growth, some find no effect and some even find positive effects. Bloom and Mahal (1997) run cross-country regressions of growth of GDP per capita on HIV/AIDS prevalence and find no effect. Papageorgiou and Stoytcheva (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. Corrigan, Gloom, and Mendez (2005) show calibration results that imply large negative effects of the epidemic on growth.

[2]Using similar household data on fertility from other African countries and HIV prevalence rate by country and time, Young (2007) reaches a similar conclusion. Kalemli-Ozcan (2006), on the other hand, reaches a different conclusion. She finds that HIV/AIDS is positively related to fertility, in the cross-section of countries, and in the household level data from South Africa for 1990s. In panel specifications with country fixed effects, however, Kalemli-Ozcan (2006) finds no significant effect of HIV on fertility.

data are multi-fold. One clear 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. Since pregnant women are engaging in unprotected sex they are at higher risk and the estimates are likely to overstate the prevalence rate in the population as a whole. 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 21 percent. Our investigation of births prior to 1986 (prior to the on-set of the HIV/AIDS) using women's fertility histories suggests that unobserved heterogeneity is not driving our results. The impact of own positive HIV status remains even when we control for sexual behavior such as condom use and multiple partners, suggesting that the physiological impact of the disease is important and may play a predominant role. In contrast to Young (2005), however, we do not find a significant effect of local community HIV prevalence, our measure of mortality risk, on the fertility of non-infected women.

A major challenge is identification of the community mortality risk effect. Our data indicate that HIV infection is higher in urban areas with a more educated population and greater economic activity. Since these are areas that are likely to have higher wages and lower fertility rates, this may bias us towards finding a negative effect. For robustness, we use an alternative strategy and instrument the community level HIV prevalence with distance to the origin of the epidemic, the Democratic Republic of Congo, as suggested by Oster (2005). We also examine the relationship between fertility rates and community level prevalence rates in *changes* assuming zero prevalence of the disease before 1986 following Young (2005) and Fortson (2007). Neither of these alternative strategies point to a significant community-level risk effect on non-infected women's fertility. Our lack of a significant effect

of community prevalence is confirmed by a recent paper by Fortson (2008), which also uses fertility histories and performs a variety of robustness checks in examining the relationship. 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.[3]

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 investigates the role of education and knowledge as possible explanations for the lack of behavioral response. Section 7 concludes.

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

[3]In the follow-up paper that uses country by cohort variation, Young (2007) reports a smaller coefficient of –0.60 instead of –1.63. This coefficient translates into a reduction of approximately 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.

and growth.[4] 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.[5] The HIV/AIDS epidemic has generated a negative shock to life expectancy which, according to these models, should *increase* fertility.[6]

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 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.[7]

[4]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).

[5]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.

[6]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.

[7]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 im-

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 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

portantly, 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.

have children.

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 attempt to separate out the physiological and behavioral responses to the disease by distinguishing between the effect of *own* HIV/AIDS status versus the effect of mortality risk as measured by the community-level prevalence rate.

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.

One caveat is that not all women were asked to give blood samples so that sample sizes are reduced. In addition, testing was voluntary so that some women refused to be tested, with the average response rate being approximately 85%. Table 1 shows the response rates for the 13 countries in our sample. Altogether, 64,062 women have non-missing HIV status out of a total of 131,575 women in the main DHS surveys. Column (1) shows the number of women in households who were randomly selected for HIV testing from the main survey sample. Column (2) shows the percent of women who were actually tested. Column (3) shows the percent of women who refused to be tested and the final column refers to women who were not at home for testing, were never interviewed, or had technical problems with

the test. The data shown in this table comes from the DHS reports.

Since some women refused to be tested, there is concern that there may be selection into the tested group. Table 2 examines the possibility of selection bias by comparing observable characteristics such as age, education, marital status, residence type (urban/rural), and wealth quintile between women who refused to be tested and women who agreed to testing and thus have non-missing HIV status. The table reports the mean differences (refusers minus compliers). The comparison is conducted only for the 6 countries in which we can identify refusers and compliers in the micro data. The table shows that the refusers are more likely to be educated, less likely to live in rural areas and more likely to be in the wealthiest quintile. Since we find a similar pattern when we compare HIV-positive and HIV-negative women among women with HIV status, this suggests that women with missing data are more likely to be HIV positive and our estimates of HIV prevalence may be too low in some countries.

Table 3 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. Timberg (2006) and many others argue that this method leads to an over-estimate of HIV prevalence because pregnant women, engaging in unprotected sex, have higher risk of HIV infection. 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. Given the concerns about selection bias we investigated whether the difference between DHS estimates and other country level estimates varied with the extent of refusal. A simple regression of the difference in the two measures on the refusal rate yielded an insignificant coefficient, lending credence to the view that these data provide the most reliable and representative estimates of HIV prevalence rates.

4 Empirical Results

4.1 Determinants of HIV Status

Before we report our results on HIV and fertility, we first explore determinants of positive HIV status using individual level data. Table 4 reports the marginal probabilities and the associated standard errors from a reduced-form probit regression. The table shows that the relationship between HIV infection and education 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". Married women are more likely to be infected than never married women."Formerly married" women, those who are widowed or divorced, have much higher infection rates. This most likely reflects the reverse causality with positive HIV status impacting marital status. Positive HIV status also varies by residence type with those in rural areas having lower infection rates. Wealth also matters with those who are in the "poorest" wealth quintile category having significantly lower propensity of being infected. One concern with using individual level data is that unobserved heterogeneity may influence both the propensity to contract the disease and fertility behavior. While our conditioning variables should take care of a large part of the problem, we return to this issue below.

4.2 Effects of Own HIV Status on Fertility

We begin by examining the effect of own HIV status on an individual woman's fertility. Our linear regression has the following form:

$$Fertility_i = \alpha + \beta OwnHIVStatus_i + \mathbf{X}_i'\gamma + D_c + D_{reg} + D_{res} + \epsilon_i, \quad (1)$$

where we use birth in the last year, birth in the last 3 years, and birth in the last 5 years as our fertility variables. Own HIV Status is a dummy variable that takes a value of 1 if individual i is HIV positive, $\mathbf{X}_i$ is a vector of other covariates, and ϵ_i is a random error term. We include as individual controls age, education, marital status, dummies for wealth quintile, and the number of living children. We also add dummies for country, region and residence

type (urban/rural) denoted as D_c, D_{reg}, D_{res} respectively. While we run linear regressions as a starting point, 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. We report estimates from both specifications below.[8]

Table 5 shows the effect of own HIV status on fertility. The effect of having positive HIV status is negative and significant in all specifications. Column (1) indicates that positive HIV status lowers births last year by -.034. Since the average is .163 births (indicated in the bottom row of the table), this translates into a reduction of 21 percent. The Poisson estimates are reported in columns (4)-(6). The coefficients can be transformed into incidence rate ratios, which are easier to interpret. These are reported in the bottom row of the table. According to column (4), positive HIV status reduces an infected woman's fertility last year by approximately 20 percent. The 3 and 5 year birth rates are reduced approximately 19 and 15 percents respectively. The table shows a sizeable negative impact of the disease on infected woman's own fertility. We also run this specification for each country, obtaining generally negative coefficient as shown in Appendix A.

It is not clear to what extent these estimates reflect the pure physiological impact of the disease versus behavioral response among the infected women. In table 6 we explore whether including various measures of sexual behavior impacts the coefficient on HIV status. We repeat the same regressions as in table 5 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. Our estimate in table 6, which implies a reduction of 23 percent, is smaller than the estimates reported in Gray et. al (1998). When we restrict our

[8]We therefore assume the number of births for a woman i, Y_i, follows a Poisson distribution, given the independent variables, X_1, $X_2, ...$

$$Prob(Y_i = y_i) = \frac{e^{-\mu_i} {\mu_i}^{y_i}}{y_i!}$$

where the log of the mean μ_i is assumed to be a linear function of the independent variables. That is,

$$ln\mu_i = \beta_0 + \beta_1 X_{1i} + \beta_2 X_{2i} + ...$$

sample to closely resemble theirs our estimate becomes larger to about 36 percent.[9]

Unobserved heterogeneity may also be biasing the results. If women who are more likely to engage in risky sexual behavior have both higher propensity of being infected and have lower desired fertility, this may cause a downward bias towards finding a negative effect. In table 7 we explore the role of unobserved heterogeneity and the question of whether women with positive HIV status are fundamentally different from those with negative status. Using the fertility histories of older women (aged 35-49) we examine the effect of current HIV 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 7 shows that there is no significant difference between HIV positive and negative women in births 20 or even 15 years ago. The difference in fertility of HIV positive and negative women, however, becomes more pronounced as the disease spreads over time.[10] Table 7 bolsters our confidence that the own HIV status effect reported in table 5 is not driven by unobserved heterogeneity.

4.3 Effects of Community HIV Prevalence on Fertility

Results from the previous section shows that that being infected with the HIV virus significantly lowers fertility. This effect however confounds the physiological impact of the disease which appears to be important with the behavioral impact. We are particularly interested in how the disease prevalence and the rise in mortality risk affect fertility behavior. To isolate the impact of the disease on behavior, we restrict our analysis in this section to women who are not infected. We run the following regression exclusively on women who are HIV negative:

[9]In results we do not report, we have run the same regression as in table 6 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.

[10]One possibility is that these women were too young 20 years ago to have pronounced differences in fertility behavior. We have also run the same regression using older women aged 40-49 and found very similar results.

$$Fertility_{ir} = \alpha' + \beta' CommunityHIV_r + \mathbf{X}'_{ir}\gamma' + D_c + D_{res} + \epsilon'_{ir}, \quad (2)$$

where r refers to country by region cell. Community HIV is defined as the fraction of all adults 15-49 (both men and women) with positive HIV status in the community.[11] Since we control for country dummies in the above regression, we are identifying the community HIV effect from cross-regional differences in HIV prevalence and fertility within countries. Before turning to the results, we report some descriptive statistics of communities in table 8. As table 8 shows, community level HIV prevalence ranges from 0 to 29 percent with the average being 5.7 percent.

We report the impact of community level HIV prevalence on non-infected women in table 9. Columns (1)-(3) report the effects on fertility when we specify the HIV prevalence rate in levels. Columns (4)-(6) report the results of an alternative specification when we specify the HIV prevalence rate in logs.[12] 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 3 years. The standard errors are large however, so that we cannot rule out either a positive or a negative effect. We conclude based on the OLS regression results in table 9 that we find little evidence of a significant positive or negative effect of community level HIV on non-infected women's fertility decisions.[13] In the following section we report results from two alternative estimation strategies.

Our examination of HIV status at the individual level shows that women who are somewhat educated and live in urban areas are more likely to be infected. There are compelling reasons to believe that HIV infection is higher in areas with greater population density and economic activity. Our community HIV regression may suffer from an omitted variables bias in that communities that are the most economically active may have both higher infection

[11] In a previous version we defined a community as a country by region by urban/rural residence cell. We thank Jane Fortson for pointing out that the DHS samples are not representative at this disaggregated level. We therefore use country by region cell to define communities in this version while still controlling for urban/rural residence. We have also used geographic sampling clusters to define communities. All definitions of a "community" yield qualitatively similar results.

[12] We lose only 1 community with zero prevalence when we use the log specification. We have also run the regression assigning a very small positive value to this community and the results were very similar.

[13] We also run this regression country by country finding mixed results as shown in Appendix A.

rates and lower fertility, the latter being due possibly to the higher cost of women's time. To address this issue, we instrument community-level HIV prevalence with distance to the origin of the epidemic, the Democratic Republic of Congo, as suggested by Oster (2005). For 4 of the 13 countries, the DHS reports the latitude and longitude of the geographic sampling cluster. Using this information we calculate distance to the center of the Democratic Republic of Congo.[14] Table 10 reports the IV results. The bottom panel of table 10 shows the significant first-stage results with distance having a negative effect on community HIV prevalence. In columns (1)-(3) we specify the community prevalence rate in levels while the specification is in logs in columns (4)-(6). Despite the significant first-stage results, however, we do not find significant effect of community HIV prevalence on non-infected women's fertility in the second stage.

We have thus far relied on cross-regional variation to identify the community level HIV effect. This was due to the fact that we have a single cross-section of HIV status based on testing data and we view the reliability of the time variation in HIV prevalence as suspect (see Kalemli-Ozcan (2006)). However, Young (2005) and Young (2007) rely on changes in HIV prevalence over time focusing on between-cohort changes in fertility with the on-set of the disease. In the following section, we follow Young's methodology and utilize the fertility histories of individual women to arrange the data by age and year at birth. We introduce time variation in community-level HIV prevalence by assuming zero prevalence in the years prior to 1986. This strategy is also followed by Fortson (2007) in estimating the impact of community-level HIV prevalence on educational outcomes. A recent paper by Fortson (2008) also uses fertility histories to examine changes in total fertility rate across regions with varying levels of HIV prevalence. More specifically, we run the following regression:

$$Fertility_{irt} = \alpha + \beta CommunityHIV_{rt} + \mathbf{X}'_{it}\gamma + \theta_r + \phi_t + \epsilon_{irt} \tag{3}$$

where t refers to year at birth and is specified as two periods, 1981–1985 and 2001–2005. The dependent variable is births last year among HIV negative women. We include education, ever married dummy at time of birth, and age at birth effects. We include community fixed

[14]To calculate the distance to the center of Democratic Republic of Congo of the community defined by region, we average longitudes and latitudes reported in the sampling clusters included in the community.

effects, 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. The results are reported in table 11. We examine women aged 15-49 at birth in columns (1) and (3) and examine women aged 20-49 at birth in columns (2) and (4) in an effort to isolate women with completed schooling. The table shows the declining trend in fertility due to demographic transition since fertility is much lower in the later period, 2001–2005, relative to the earlier period, 1981–1985. This suggests that these countries were likely completing the demographic transition over this period. Once again, none of the coefficients are significant at the 5 percent level.

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 relayed in table 5 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 12 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, marital status, and wealth since our earlier tables showed differences in these characteristics between the HIV positive and HIV negative populations.[15] Table 12 shows a

[15]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 (8), predict fertility by age group and add back residuals for HIV negative women.

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 12 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).

6 Why No Behavioral Response? The Role of Education and Knowledge

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. Recent DHS survey reports show that among 34 African countries, only 6.4 percent of the female population requested an HIV test, got tested, and received results. Moreover, only 23 percent of the women in these countries had comprehensive knowledge about the disease.[16] In this section we investigate whether lack of knowledge about the disease can at least

[16]See DHS country reports at www.macrointernational.org. "Comprehensive knowledge" consists of correctly identifying the two major ways of preventing sexual transmission of HIV and rejecting the two most common misconceptions about HIV transmission. Recent evidence in Thornton (2008), however, suggests that knowledge alone may not account for the limited response of sexual behavior in many African countries. The paper is based on a randomized experiment in Malawi in which individuals were given monetary incentives to get tested and learn about their HIV status. Using randomized incentives as instrument for knowledge, she finds that those with positive HIV status were more likely to purchase condoms but buy at most 2 condoms over a period of two months, and there was no change in behavior among those with negative HIV status. A recent paper by Oster (2007) also argues along these lines suggesting that shorter life expectancy and lower income could account for the large differences in behavioral response between individuals in Africa and the gay population in the U.S.

partially explain the absence of behavioral response in fertility. In table 13 we repeat the cross sectional and community fixed effects regressions presented in our earlier table 9 and table 11, interacting community HIV prevalence with two variables that may proxy for knowledge and salience of the disease– education and knowing someone who has AIDS. Again we only include women with negative HIV status in our regressions. Columns (1) and (2) use the cross sectional variation as in our table 9 while columns (3) and (4) report results from the community fixed effects regressions as in our table 11. None of the interactions using cross-sectional variation in columns (1) and (2) produce significant results at the 5% level. However, the interactions in the community fixed effects regressions in column (3) and column (4) suggest that education and knowledge may play a role. In column (3) HIV prevalence interacted with education shows that women with more education generally have greater negative response to community prevalence compared to women with no education although the interaction terms are not significant.[17] Column (4) shows that women who know someone with or died of AIDS respond more negatively to community HIV prevalence than those without knowledge. The direct effect of knowing someone with AIDS is about zero at the mean level of HIV prevalence, which is 5.7 percent in our sample. Interestingly, women who live in low prevalence communities have higher fertility if they know someone with or died of AIDS, consistent with the neoclassical models discussed before. Living in both high prevalence community and knowing someone with AIDS, however, lowers women's fertility.[18] In this section we find some suggestive, although not always robust, evidence that lack of education and knowledge about the disease may partially account for the lack of behavioral response.

[17]A recent paper by Fink and Linnemayr (2008) find a strong negative interaction effect between community HIV prevalence and education using the earlier World Fertility Surveys and the more recent DHS for five countries, Cameroon, Cote d'Ivoire, Ghana, Kenya, and Senegal. While we do not consistently find a negative interaction effect with respect to education, we find a more robust negative effect consistent with their results when we restrict the sample to younger aged women, 15-29.

[18]The variable "know someone with or died of AIDS" is available only in the 2000–2005 period. We make the same assumption as we did with HIV prevalence and set this variable equal to zero in 1980–1985.

7 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. To this date, there has not been robust evidence on this issue. The effect of HIV on fertility will be the 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/AIDS 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 can further reinforce or possibly mitigate the population declines brought on by the disease. We undertake this exercise for the first time by making use of the 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 20 percent lower. Robustness checks imply that these results are not driven by unobserved heterogeneity or different sexual behavior among the HIV positive women, suggesting that the disease significantly lowers the fecundity of the infected women. 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: HIV Testing Response Rates

	Testing Sample	Actually Tested	Refused	Other
Burkina Faso	4575	92.3	4.4	3.3
Cameroon	5703	92.1	5.4	2.5
Cote d'Ivoire	5772	79.1	10.6	10.3
Ethiopia	7142	83.2	13.4	3.4
Ghana	5949	89.3	5.7	5.0
Guinea	4189	92.5	5.0	2.5
Kenya	4303	76.3	14.4	9.3
Lesotho	3758	80.7	12.0	7.3
Malawi	4071	70.4	22.5	7.1
Niger	4899	90.7	3.5	5.8
Rwanda	5837	97.3	1.1	1.6
Senegal	5350	84.5	9.9	5.6
Zimbabwe	9870	75.9	13.2	10.9

Notes: The data are based on the following DHS surveys and from DHS Country Reports: Burkina Faso (2003), Cameroon (2004), Cote d'Ivoire (2005), Ethiopia (2005), Ghana (2003), Guinea (2005), Kenya (2003), Lesotho (2004), Malawi (2004), Niger (2006), Rwanda (2005), Senegal (2005), Zimbabwe (2005/2006)."Testing Sample" refers to the number of women in households who were randomly selected for HIV testing from the main survey sample. "Actually Tested" refers to women who were tested. Based on the reason for non-response, individuals who were not tested are divided into four categories: those who refused testing, those who were interviewed in the survey but who were absent when the health worker arrived for testing, those who were never interviewed, and those who had missing test results due to technical problems. Column (3) reports the percent of women who refused to be tested. Column (4) reports the percent who do not have HIV test results for other reasons.

Table 2: Differences in Characteristics of Refusers and Compliers

	Cameroon	Lesotho	Niger	Rwanda	Senegal	Zimbabwe
Age	–0.773	1.120	–2.433*	–2.686*	–0.096	–0.170
	(0.600)	(0.623)	(0.993)	(1.094)	(1.119)	(0.311)
Highest Education	0.299*	0.236*	0.235*	0.199	0.239	0.100*
	(0.056)	(0.036)	(0.068)	(0.110)	(0.142)	(0.021)
Ever Married	0.059*	–0.019	0.018	0.053	0.080	–0.005
	(0.026)	(0.021)	(0.019)	(0.065)	(0.051)	(0.012)
Rural	–0.283*	–0.338*	–0.322*	–0.211*	–0.115	–0.234*
	(0.027)	(0.031)	(0.049)	(0.069)	(0.062)	(0.017)
Poorest	–0.108*	–0.100*	–0.103*	–0.108*	–0.083*	–0.071*
	(0.020)	(0.012)	(0.033)	(0.050)	(0.031)	(0.012)
Richest	0.252*	0.235*	0.268*	0.365*	–0.056	0.168*
	(0.036)	(0.031)	(0.048)	(0.075)	(0.061)	(0.016)

Notes: The table reports the mean differences in characteristics of women who refused to be tested relative to women who agreed to be tested. Main survey sample weights are used in the specifications and standard errors are in the parentheses. The variables identifying women who are in the testing sample, women who refused or do not have valid testing data for other reasons are available in the micro data only for the above six countries. Asterisk denotes significance levels (* = p-value $< .05$).

Table 3: 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 4: Determinants of HIV Status: Probit Regression

Age	0.014*
	(0.001)
Age^2	–0.000*
	(0.000)
Primary Education	0.011*
	(0.003)
Secondary Education	0.009*
	(0.003)
Higher Education	–0.010*
	(0.004)
Currently Married	0.004
	(0.002)
Formerly Married	0.080*
	(0.007)
Rural	–0.013*
	(0.003)
Poorer	0.011*
	(0.003)
Middle	0.017*
	(0.003)
Richer	0.020*
	(0.004)
Richest	0.018*
	(0.004)
Pseudo R^2	0.241
N	63904

Notes: Country and region dummies are included in the regression. The omitted categories are: "No Education", "Urban ", "Never Married", and "Poorest Wealth". The table reports marginal probabilities and associated standard errors. Asterisk denotes significance levels (* = p-value $< .05$).

Table 5: Effect of Own HIV Status on Fertility

	Last Year Birth OLS (1)	Last 3 Year Birth OLS (2)	Last 5 Year Birth OLS (3)	Last Year Birth Poisson (4)	Last 3 Year Birth Poisson (5)	Last 5 Year Birth Poisson (6)
Positive HIV Status	–0.034*	–0.092*	–0.136*	–0.225*	–0.209*	–0.168*
	(0.007)	(0.010)	(0.014)	(0.051)	(0.028)	(0.022)
Age	0.021*	0.075*	0.163*	0.227*	0.247*	0.294*
	(0.001)	(0.002)	(0.003)	(0.011)	(0.006)	(0.005)
Age^2	0.000*	–0.002*	–0.003*	–0.005*	–0.005*	–0.005*
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Primary Education	–0.012*	–0.029*	–0.055*	–0.063*	–0.061*	–0.071*
	(0.005)	(0.008)	(0.010)	(0.030)	(0.017)	(0.014)
Secondary Education	–0.034*	–0.079*	–0.145*	–0.249*	–0.217*	–0.245*
	(0.005)	(0.008)	(0.011)	(0.040)	(0.022)	(0.019
Higher Education	–0.048*	–0.142*	–0.290*	–0.530*	–0.540*	–0.595*
	(0.010)	(0.016)	(0.022)	(0.128)	(0.076)	(0.060)
Currently Married	0.245*	0.559*	0.782*	2.125*	2.113*	2.091*
	(0.005)	(0.008)	(0.010)	(0.060)	(0.040)	(0.038)
Formerly Married	0.138*	0.339*	0.456*	1.355*	1.567*	1.627*
	(0.007)	(0.011)	(0.014)	(0.080)	(0.048)	(0.043)
Rural	0.021*	0.040*	0.077*	0.141*	0.090*	0.101*
	(0.005)	(0.007)	(0.010)	(0.036)	(0.019)	(0.016)
Poorer	–0.016*	–0.021*	–0.046*	–0.077*	–0.037*	–0.049*
	(0.006)	(0.008)	(0.011)	(0.029)	(0.016)	(0.013)
Middle	–0.022*	–0.041*	–0.067*	–0.103*	–0.073*	–0.070*
	(0.006)	(0.008)	(0.012)	(0.031)	(0.017)	(0.014)
Richer	–0.034*	–0.064*	–0.105*	–0.161*	–0.112*	–0.111*
	(0.007)	(0.009)	(0.012)	(0.035)	(0.019)	(0.015)
Richest	–0.046*	–0.078*	–0.129*	–0.301*	–0.182*	–0.182*
	(0.007)	(0.010)	(0.014)	(0.047)	(0.025)	(0.020)
R^2	0.116	0.297	0.377	–	–	–
N	64056	64056	64056	64056	64056	64056
Mean	0.163	0.430	0.710	–	–	–
	(0.001)	(0.001)	(0.002)	–	–	–
Incidence Rate Ratio	–	–	–	0.798	0.812	0.845
	–	–	–	(0.040)	(0.023)	(0.019)

Notes: Women with non-missing HIV status are used in the regressions. All regressions include country and region dummies. The omitted categories are: "No Education", "Urban, " "Never Married", and "Poorest Wealth" quintile. Columns (1) and (4) control for number of living children last year, columns (2) and (5) control for number of living children 3 years ago, columns (3) and (6) control for number of living children 5 years ago. HIV survey sample weights are used in the regressions, and robust standard errors are in the parentheses. Asterisk denotes significance levels (* = p-value $< .05$).

Table 6: Effect of Own HIV Status on Fertility, Controlling for Number of Partners and Condom Use

	Last Year Birth OLS (1)	Last 3 Year Birth OLS (2)	Last 5 Year Birth OLS (3)	Last Year Birth Poisson (4)	Last 3 Year Birth Poisson (5)	Last 5 Year Birth Poisson (6)
Positive HIV Status	–0.038*	–0.101*	–0.152*	–0.228*	–0.212*	–0.177*
	(0.008)	(0.013)	(0.017)	(0.053)	(0.031)	(0.024)
Condom Use	–0.030*	–0.043*	–0.065*	–0.292*	–0.167*	–0.175*
	(0.007)	(0.011)	(0.015)	(0.069)	(0.039)	(0.032)
More Than One Partner	–0.073*	–0.153*	–0.203*	–0.522*	–0.413*	–0.321*
	(0.008)	(0.012)	(0.018)	(0.079)	(0.043)	(0.036)
R^2	0.092	0.234	0.297	–	–	–
N	43965	43965	43965	43965	43965	43965

Notes: Women with non-missing HIV status are used in the regressions. All regressions include country and region dummies. Other controls that are included are age, age squared, education, marital status, number of living children, wealth quintile. The omitted categories are: "No Education", "Urban", "Never Married", and "Poorest Wealth" quintile, "Did not use a condom during last intercourse", and "Did not have more than one partner in last 12 months". HIV survey sample weights are used in the regressions, and robust standard errors are in the parentheses. Asterisks denote significance levels (* = p-value $< .05$).

Table 7: 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: Last Year Births				
Positive HIV Status	–0.017*	–0.027*	–0.007	–0.013	–0.008
	(0.008)	(0.011)	(0.014)	(0.015)	(0.013)
Mean	0.109	0.177	0.220	0.251	0.206
	(0.002)	(0.003)	(0.003)	(0.003)	(0.003)
R^2	0.053	0.087	0.042	0.034	0.077
N	17696	17696	17696	17696	17696
	Panel B: Dependent Variable: Cumulative 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	5.367	4.748	3.746	2.531	1.315
	(0.022)	(0.021)	(0.019)	(0.016)	(0.012)
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 are used in the regressions. In panel A dependent variable is the births previous year, in panel B dependent variable is cumulative number of children born for each woman up to N years ago from survey year. All women with HIV status are used in the regressions. All regressions include country and region dummies. The omitted categories are: "No Education", "Urban", "Never Married", and "Poorest Wealth" quintile. HIV survey sample weights are used in the regressions, and robust standard errors are in the parentheses. Robust standard errors are in the parentheses. Asterisk denotes significance levels (* = p-value $< .05$).

Table 8: Descriptive Statistics of Communities

	Mean	Standard Deviation	Min	Max
	Number of Communities N=128			
Number of Women	1389.34	1175.36	351	5902
Last Year Birth	0.17	0.05	0.05	0.30
Last 3 Year Births	0.45	0.13	0.13	0.75
Last 5 Year Births	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

Notes: "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 the 128 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 9: Effect of Community HIV Prevalence on Fertility: OLS

	Last Year Birth (1)	Last 3 Year Birth (2)	Last 5 Year Birth (3)	Last Year Birth (4)	Last 3 Year Birth (5)	Last 5 Year Birth (6)
Community HIV Prevalence	0.111	–0.052	–0.205	–	–	–
	(0.089)	(0.157)	(0.272)	–	–	–
Log Community HIV Prevalence	–	–	–	–0.002	–0.005	–0.018
	–	–	–	(0.005)	(0.008)	(0.013)
R^2	0.116	0.304	0.384	0.116	0.304	0.384
N	59579	59579	59579	59427	59427	59427

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 country by region cell, excluding the woman herself. Controls are age, age squared, education, marital status, number of living children, wealth quintile, country dummies and rural dummy. HIV survey sample weights are used in the regressions, and robust standard errors clustered at the country by region level are in the parentheses.

Table 10: Effect of Community HIV Prevalence on Fertility: IV

	Last Year Birth (1)	Last 3 Year Birth (2)	Last 5 Year Birth (3)	Last Year Birth (4)	Last 3 Year Birth (5)	Last 5 Year Birth (6)
Community HIV Prevalence	0.406	–0.294	–0.965	–	–	–
	(0.375)	(0.928)	(1.521)	–	–	–
Log Community HIV	–	–	–	0.083	0.037	–0.008
Prevalence	–	–	–	(0.121)	(0.161)	(0.209)
Latitude	–0.001	–0.006	–0.012	0.012	0.000	–0.012
	(0.003)	(0.005)	(0.008)	(0.017)	(0.026)	(0.035)
Longitude	–0.003	–0.016*	–0.032*	0.012	–0.008	–0.030
	(0.003)	(0.007)	(0.009)	(0.024)	(0.033)	(0.041)
R^2	0.115	0.300	0.393	0.108	0.299	0.393
N	14484	14484	14484	14332	14332	14332

	First Stage	
Dependent Variable:	Community HIV Prevalence	Log Community HIV Prevalence
Distance to Congo	–0.060*	–0.348*
	(0.002)	(0.027)
Latitude	–0.001*	–0.165*
	(0.000)	(0.004)
Longitude	–0.008*	–0.212*
	(0.000)	(0.003)
R^2	0.946	0.873

Notes: Women with negative HIV status are included in the regressions."Community HIV Prevalence" and its log are instrumented with distance between the community and the center of the Democratic Republic of Congo. First stage results are reported at the bottom of the table. Regressions include controls for age, age squared, education, marital status, number of living children, wealth quintile, country dummies and rural dummy. HIV survey sample weights are used in the regressions, and robust standard errors clustered at the country by region level are in the parentheses. Asterisk denotes significance levels (* = p-value $< .05$).

Table 11: Effect of Community HIV Prevalence on Last Year Birth: OLS

	Ages 15-49 (1)	Ages 20-49 (2)	Ages 15-49 (3)	Ages 20-49 (4)
Community HIV Prevalence	0.111	0.020	–	–
	(0.082)	(0.077)	–	–
Log Community HIV Prevalence	–	–	0.008	0.003
	–	–	(0.007)	(0.006)
2001-2005	–0.104*	–0.096*	–0.163*	–0.120*
	(0.011)	(0.010)	(0.058)	(0.050)
Community Fixed Effects	Yes	Yes	Yes	Yes
R^2	0.091	0.082	0.092	0.082
N	309406	218862	309406	218862

Notes: Regressions only include HIV negative women. Dependent variable is the number of births last year. HIV Prevalence is assumed to be zero before 1985. Omitted categories are "1981-1985", "Ages 25-29", "No education", "Not married". In columns (3) and (4) prevalence rate is taken as 0.00001 at regions with 0 prevalence. All regressions include controls for education, marital status at birth, age group dummies, period dummies, age group by time interactions, community dummies, country dummies, and rural dummy. Robust standard errors are clustered at country by region level and in parentheses. Asterisk denotes significance levels (* = p-value $< .05$).

Table 12: 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.

Table 13: Effect of Community HIV Prevalence Interacted with Education and Knowledge

	Last Year Birth (1)	Last Year Birth (2)	Last Year Birth (3)	Last Year Birth (4)
HIV Prevalence	0.023	0.113	0.132	0.146
	(0.101)	(0.091)	(0.101)	(0.091)
Primary Education	–0.013	–0.008	–0.003	–0.008*
	(0.010)	(0.006)	(0.003)	(0.002)
Secondary Education	–0.039*	–0.032*	–0.015*	–0.029*
	(0.013)	(0.006)	(0.005)	(0.003)
Higher Education	–0.037*	–0.048*	–0.015*	–0.039*
	(0.022)	(0.010)	(0.009)	(0.005)
HIV Prevalence * Primary Education	0.114	–	–0.009	–
	(0.105)	–	(0.044)	–
HIV Prevalence * Secondary Education	0.123	–	–0.031	–
	(0.101)	–	(0.049)	–
HIV Prevalence * Higher Education	–0.107	–	–0.086	–
	(0.151)	–	(0.078)	–
Know Someone with or Died of AIDS	–	–0.004	–	0.014*
	–	(0.006)	–	(0.006)
HIV Prevalence *Know Someone with or Died of AIDS	–	–0.061	–	–0.108*
	–	(0.052)	–	(0.045)
Community Fixed Effects	No	No	Yes	Yes
R^2	0.116	0.120	0.092	0.094
N	59561	46332	309343	251425

Notes: HIV negative women are used in the regressions. Columns (1) and (2) are cross-sectional regressions. Column (3) and (4) are community fixed effect regressions. "No education" is the omitted education category. Columns (1) and (2) also controls for age, age squared, marital status, number of living children, wealth quintile, country dummies and rural dummy. Columns (3) and (4) control for marital status at birth, age group dummies, period dummies, age group by time interactions, community dummies, country dummies, and rural dummy. In columns (3) and (4), HIV Prevalence is assumed to be zero before 1986. In column (4) knowledge of someone with AIDS is also set to zero before 1986. HIV survey sample weights are used in the regressions, and robust standard errors clustered at the country by region level are in the parentheses. Asterisk denotes significance levels (* = p-value $< .05$).

Table A-1: Effect of Own HIV Status on Fertility Country by Country: OLS

	Last Year Birth OLS (1)	Last 3 Year Birth OLS (2)	Last 5 Year Birth OLS (3)	Last Year Birth Poisson (4)	Last 3 Year Birth Poisson (5)	Last 5 Year Birth Poisson (6)
Burkina Faso	–0.071	–0.095	–0.214*	–0.466	–0.152	–0.214
	(0.043)	(0.078)	(0.095)	(0.362)	(0.183)	(0.131)
Cameroon	–0.027	–0.146*	–0.186*	–0.185	–0.348*	–0.244*
	(0.024)	(0.034)	(0.047)	(0.147)	(0.087)	(0.066)
Cote d'Ivoire	0.006	–0.006	–0.015	0.099	0.014	–0.015
	(0.024)	(0.038)	(0.060)	(0.276)	(0.177)	(0.157)
Ethiopia	–0.053	0.011	–0.115	–0.231	0.138	–0.051
	(0.041)	(0.068)	(0.083)	(0.353)	(0.148)	(0.127)
Ghana	–0.024	–0.111*	–0.256*	–0.157	–0.270	–0.392*
	(0.035)	(0.050)	(0.063)	(0.244)	(0.143)	(0.118)
Guinea	–0.091*	–0.157*	–0.310*	–0.838*	–0.479*	–0.562*
	(0.034)	(0.072)	(0.102)	(0.371)	(0.241)	(0.202)
Kenya	–0.029	–0.079	–0.101	–0.174	–0.162	–0.096
	(0.027)	(0.041)	(0.052)	(0.156)	(0.095)	(0.066)
Lesotho	–0.031	–0.085*	–0.109*	–0.219	–0.229*	–0.163*
	(0.017)	(0.024)	(0.032)	(0.127)	(0.074)	(0.056)
Malawi	–0.068*	–0.197*	–0.293*	–0.375*	–0.371*	–0.313*
	(0.023)	(0.033)	(0.047)	(0.128)	(0.069)	(0.056)
Niger	–0.121*	–0.282*	–0.413*	–0.634	–0.477*	–0.375
	(0.061)	(0.094)	(0.154)	(0.477)	(0.234)	(0.199)
Rwanda	–0.063*	–0.077	–0.074	–0.398*	–0.148	–0.082
	(0.023)	(0.040)	(0.059)	(0.179)	(0.088)	(0.074)
Senegal	–0.038	–0.183*	–0.298*	–0.314	–0.548*	–0.509*
	(0.064)	(0.072)	(0.091)	(0.488)	(0.257)	(0.181)
Zimbabwe	–0.031*	–0.082*	–0.107*	–0.271*	–0.222*	–0.172*
	(0.012)	(0.018)	(0.023)	(0.096)	(0.048)	(0.035)

Notes: Women with non–missing HIV status are used in the regressions. All regressions include controls for age, education, marital status, number of living children, wealth quintile, region dummies and rural dummy. HIV survey sample weights are used in the regressions, and robust standard errors are in the parentheses. Asterisk denotes significance levels (* = p-value $< .05$).

Table A-2: Effect of Community HIV Prevalence on Fertility Country by Country: OLS

	Last Year Birth	Last 3 Year Birth	Last 5 Year Birth	Last Year Birth	Last 3 Year Birth	Last 5 Year Birth
	HIV Prevalence			Log HIV Prevalence		
Burkina Faso	0.050	0.614	0.753	0.001	0.004	–0.011
	(0.516)	(0.882)	(2.021)	(0.007)	(0.014)	(0.039)
Cameroon	–0.002	–0.052	–0.865	0.000	–0.002	–0.042
	(0.200)	(0.523)	(0.749)	(0.010)	(0.022)	(0.030)
Cote d'Ivoire	–0.093	1.428	1.259	0.006	0.072	0.074
	(0.539)	(0.937)	(1.558)	(0.024)	(0.039)	(0.068)
Ethiopia	–1.045	–1.230	–1.517	–0.017	–0.015	–0.018
	(1.074)	(1.308)	(1.911)	(0.013)	(0.023)	(0.034)
Ghana	–0.074	0.784	1.342	-0.004	0.010	0.020
	(0.395)	(0.446)	(1.230)	(0.006)	(0.009)	(0.024)
Guinea	0.326	0.904	–2.764	0.008	0.026	–0.026
	(1.655)	(3.244)	(5.450)	(0.030)	(0.058)	(0.099)
Kenya	0.451*	0.349*	0.466	0.038*	0.031*	0.045
	(0.077)	(0.132)	(0.304)	(0.006)	(0.012)	(0.030)
Lesotho	–0.293	–0.686*	–0.595	–0.075	–0.166*	–0.148
	(0.181)	(0.210)	(0.392)	(0.043)	(0.044)	(0.085)
Malawi	0.021	–0.206	–0.425*	0.003	–0.025	–0.051*
	(0.045)	(0.088)	(0.078)	(0.005)	(0.010)	(0.009)
Niger	1.814*	–2.809	–9.140	0.017*	-0.018	-0.075
	(0.661)	(1.998)	(4.555)	(0.005)	(0.017)	(0.038)
Rwanda	–0.403	–0.678	–1.626	–0.018	–0.024	–0.051
	(0.354)	(0.589)	(0.947)	(0.016)	(0.025)	(0.042)
Senegal	–2.326*	–4.156*	–5.821*	–0.015	–0.032*	–0.045*
	(0.870)	(1.595)	(2.383)	(0.007)	(0.010)	(0.014)
Zimbabwe	0.841*	–0.071	0.416	0.155*	–0.015	0.071
	(0.339)	(0.426)	(0.617)	(0.063)	(0.076)	(0.111)

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 region, excluding the woman herself. In columns (1)–(3) HIV prevalence is used, in columns (4)–(6) log HIV prevalence is used. Other controls are age, age squared, education, marital status, number of living children, wealth quintile and rural dummy. HIV survey sample weights are used in the regressions, and robust standard errors clustered at the region level are in the parentheses. Asterisk denotes significance levels (* = p-value $< .05$).