Behavioral or Biological: Taking a Closer Look at the Relationship between HIV and Fertility

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Abstract

This paper examines the relationship between HIV and fertility at the individual level, using data from eight countries in Sub-Saharan Africa. I utilize the two most recent rounds of the Demographic and Health Surveys, which links women to their HIV test results, to distinguish between potential mechanisms linking HIV and fertility. I find that HIV positive women have significantly lower fertility. I argue that this relationship predominantly reflects the biological consequences of the disease, rather than a behavioral response. The relationship between HIV status and fertility holds even after controlling for several indicators of risky sexual behavior, and after restricting the sample to women who have never been tested for HIV prior to the survey. While HIV positive women have smaller ideal family sizes and want fewer children in the future, this does not appear to be driving the relationship between HIV and fertility.

Keywords: fertility, HIV, Sub-Saharan Africa

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1 Introduction

The HIV/AIDS epidemic in Sub-Saharan Africa has undoubtedly had an effect on the demographic makeup of countries in that region. While the effect on mortality is unambiguous, there has been much debate over the impact of the epidemic on fertility. HIV is hypothesized to have an effect on fertility through both a biological pathway, for women who are infected, as well as a behavioral response pathway, for both infected and uninfected women. Estimates of the magnitude and direction of the effect of HIV on fertility has varied depending on the data and estimation strategy used. Furthermore, the cross-sectional nature of the available data has made causal inference extremely difficult, as HIV and fertility share some of the same risk factors.

Understanding the relationship between HIV and fertility is important for several reasons. At the regional or country level, the relationship between HIV prevalence and fertility will affect population projections, and may also have economic consequences that affect the standard of living (Young, 2005). At the individual level, a difference in fertility rates between infected and uninfected women also has important implications. First, there are implications for the estimates of HIV prevalence, since most estimates rely on studies of women who visit antenatal clinics. Unless the difference in fertility rates by HIV status are taken into account, antenatal surveillance may underestimate the actual prevalence of HIV in the population (Hunter et al., 2003, Zaba and Gregson, 1998, Fabiani et al., 2006). Second, individual level fertility differences between infected and uninfected women also has implications for programs aiming to reduce mother-to-child transmission (Hunter et al., 2003).

Evidence from several studies in Sub-Saharan Africa suggests that fertility is lower among HIV infected women (Hunter et al., 2003, Juhn et al., 2013, Terceira et al., 2003, Zaba and Gregson, 1998). Less is known about the pathways through which HIV status affects fertility. Young (2005) finds that the HIV infection rate has a strong negative effect on predicted fertility, after controlling for income, education, etc., in South Africa. In a follow up paper, Young (2007) finds evidence to suggest that the decline in fertility due to HIV "appears to reflect a fall in the demand for children, and not any adverse physiological consequences of the disease, as it is matched by changes in the expressed preference for children and the use of contraception, and is not significantly correlated with biological markers of sub-fecundity." On the other hand, other studies have argued for both a behavioral and biological pathway (Hunter et al., 2003, Sneeringer and Logan, 2009, Juhn et al., 2013).

This paper makes use of both rounds of the Demographic and Health Surveys (DHS) that collected HIV test results which can be linked to the women in these surveys. I use DHS data from eight countries in Africa - Cameroon, Cote d'Ivoire, Ethiopia, Kenya, Lesotho, Malawi, Zimbabwe, and Senegal - to take a closer look at the relationship between HIV status and fertility at the individual level. I find that HIV positive women have significantly lower fertility. This result holds after one-to-one exact matching which pairs infected women with non-infected women with the exact set of "pre-treatment" covariates. The magnitude of the association between HIV status and fertility is consistent for women over the entire child-bearing age and with different years of education.I argue that this relationship predominantly reflects the biological consequences of the disease, rather than a behavioral response. While HIV positive women desire fewer children compared to HIV negative women, the preference for smaller family sizes does not appear to be driving the relationship between HIV status and fertility. The relationship between HIV status and fertility holds even after controlling for several indicators of risky sexual behavior, and after restricting the sample to

women who have never been tested for HIV prior to the survey i.e. are presumed to be unaware of their HIV status.

The remainder of the paper is organized as follows. The background and theory is presented in Section 2. In Section 3, I describe the data. Section 4 describes the estimation strategy, and Section 5 presents the results. Finally, I discuss the results and conclude in Section 6.

2 Background

The debate on the impact of the HIV/AIDS epidemic on fertility is a long-standing one. This is partially driven by the fact that the impact of the epidemic on fertility is ambiguous, as predicted by traditional fertility models. There are several pathways through which the HIV epidemic is hypothesized to affect fertility. Firstly, HIV is believed to have a direct biological effect on the fertility of infected women. Evidence from clinical and cohort based studies suggests that HIV positive women may have significantly lower fecundity and odds of bearing children (Zaba and Gregson, 1998). The lower fecundity among HIV positive women is a result of higher rates of stillbirths, fetal wastage, spontaneous abortions, greater risk of coinfection with other sexually transmitted infections, and reduced coital frequency as a result of the illness (Juhn et al., 2013, Gregson, 1994, Lewis et al., 2004, Zaba and Gregson, 1998). The reduction in fertility as a result of lower fecundity among HIV positive women is estimated to be around 30 - 40% (Carpenter et al., 1997, Hunter et al., 2003, Gray et al., 1998, Terceira et al., 2003). Furthermore, fertility has been shown to decrease significantly with disease progression and decreasing CD4 cell counts (Ross et al., 2004, Loko et al., 2005).

Secondly, behavioral responses of both infected and uninfected women may affect fertility, although the direction of the effect is ambiguous. Infected and uninfected women may change their sexual behavior in response to an epidemic to stop the disease from spreading or to protect themselves. The evidence for the impact of the epidemic on sexual behavior in Sub-Saharan Africa has been mixed. Several studies have documented little or no effect of the epidemic on sexual behavior (Bloom et al., 2000, Oster, 2005). Other studies have suggested that there may be reductions in certain risky behaviors such as lack of condom use, having multiple sexual partners and early age at first sex (Cheluget et al., 2006, Fylkesnes et al., 2001). HIV positive women, who are aware of their status, may also be worried about mother-to-child transmission, and desire fewer children as a result. Both of these behavioral responses would have the effect of depressing fertility.

On the other hand, traditional fertility models imply that a rise in youth and adult mortality causes an increase in fertility by creating a precautionary demand for children. This is a "hoarding effect" whereby parents bear more children than their desired total number of children, in order to insure against future deaths. If fertility decisions are made sequentially, then there may also be a "replacement effect" i.e. parents make decisions about having more children based on the survival of previously born children (Palloni and Rafalimanana, 1999). For HIV negative women, this effect is likely to be small as HIV mostly affects youth and adults . The HIV/AIDS epidemic may also affect fertility via its effect on parental wages. Most fertility models imply a negative correlation between parental income and fertility, a phenomenon that has been empirically observed. HIV positive women may find it harder to keep working or find work. Alternately, HIV negative women may find it easier to find work in high prevalence regions.

A few studies have attempted to empirically clarify the ambiguity in the relationship between HIV and fertility suggested by theory. Using household data from South Africa, Young (2005) finds that the historical HIV infection rate for each woman's age group, as recorded in maternity clinic seroprevalence surveys, has a strong negative effect on predicted fertility after controlling for income, education, etc.[1] Young argues that "widespread community infection lowers fertility, both directly, through a reduction in the willingness to engage in unprotected sexual activity, and indirectly, by increasing the scarcity of labor and the value of a woman's time." In a follow up paper, Young (2007) finds similar results using a larger sample of 27 countries. To examine whether lower fecundity among HIV positive women could explain the lower fertility, Young (2007) explores the relationship between positive HIV status and the probability of a recent menstrual period and the probability of a recent pregnancy resulting in a stillbirth, miscarriage or abortion. He finds no evidence to suggest that the decline in fertility due to HIV is a result of lower fecundity among HIV positive women, and instead argues that it is due to a reduction in the desired number of children.

Juhn et al. (2013) utilize the first round of the DHS that collected HIV test results, and find that HIV positive women are significantly less likely (17-20% lower probability) to give births in the year preceding the survey compared to uninfected women. Contrary to Young (2007), they argue that a large part of the relationship is driven by biological, as opposed to behavioral, factors. Sneeringer (2009) provides evidence for three countries - Uganda, Burkina Faso and Zimbabwe - and argues that in regions with high HIV prevalence, "women are attempting to avoid HIV while maintaining high fertility."

In this paper, I utilize the latest rounds of the DHS that collected HIV test results to differentiate between the biological and behavioral pathways. In my analysis, I refer to the direct effect of the disease on fertility as the biological pathway. I examine two potential behavioral pathways. Firstly, I look at the extent to which changes in risky sexual behavior may explain the relationship between HIV status and fertility. Secondly, I examine differences in the fertility preferences between HIV infected and uninfected women. This may reflect either the "hoarding" or "replacement" effect, or a desire to have fewer children due to fear of mother-to-child transmission.

3 Data

I use repeated cross-sections of the DHS for eight countries in Sub-Saharan Africa, to examine the response of fertility to the HIV/AIDS epidemic. The DHS are nationally-representative household surveys that collect data on a wide range of outcomes and indicators in the areas of population, health and nutrition. The standard DHS are cross-section surveys of households with sample sizes that vary from 5000 to 30,000. They are conducted about every five years. The sample is usually based on a stratified two-stage cluster design. I restrict my sample of countries to only those which have two waves of DHS HIV data and have reasonably consistent region definitions over the two survey waves. This includes Cote d'Ivoire, Cameroon, Ethiopia, Kenya, Lesotho,

[1] Kalemli-Ozcan and Turan (2010) recreate Young's results and argue that Young's results are driven by the fact that he assigns zero prevalence to all observations prior to 1990 due to lack of data on HIV prevalence. When the sample is restricted to observations between 1990 - 1998, a period for which data on HIV prevalence is available, the significant negative relationship disappears. In fact, Kalemli-Ozcan and Turan find that the effect of HIV prevalence on fertility in South Africa was positive over that time period.

Malawi, Zimbabwe, and Senegal. While it would be ideal to include more countries in the study, my sample has good geographic variation and covers a range of HIV prevalence and fertility rates. I divide each country into geographic locales that are defined by administrative divisions. My final sample has 64 regions, with two waves of data for each region. The survey waves were conducted in different years in different countries. The latest wave was conducted between 2008 and 2012, while the previous wave was conducted between 2003 and 2006. Table 1 in Appendix A shows the countries and survey years in my sample. Figure 7 in Appendix A shows the geographic location, HIV prevalence and total fertility rates for the eight countries in my sample.

3.1 HIV Status and Prevalence

DHS started collecting HIV testing data in 2001. My sample only includes surveys where the HIV test results can be linked to the full DHS survey record. From the individual level test results, I construct the prevalence, expressed as percentage points, for each region within a country. I calculate the prevalence using test results for men and women between the ages of 15 and 49, and the HIV weights provided by DHS, which adjust for individual sampling probabilities and test non-response rates. The regional HIV prevalence varies from 0.068 percentage points in Senegal in 2005 to 30.940 percentage points in Lesotho in 2009.

3.2 Fertility

Each woman, in a DHS household survey, is asked about her complete birth history, including the sex, month and year of birth, age, and survival status for each of the births. I define fertility, $births_{i,r,t}$, as the number of births to women i, living in region r, in the three years preceding the survey year t. Past literature has used number of births in the last year, number of births in the last three years, and number of births in the last five years as a measure of individual level fertility. The main challenge is that I cannot identify when an individual becomes HIV positive. The number of births in the last year, i.e. the year immediately preceding the survey may thus be most appropriate, but is likely to be a very noisy measure of individual level fertility. Using a five year window makes it a less accurate measure of period fertility and is more likely to be representative of an individual's lifetime or completed fertility. Thus, my preferred dependent variable is the number of births in the last three years.

Figure 1 shows the distribution of the number of children born to HIV positive and HIV negative women in the three years preceding the survey. HIV positive women are more likely to have had no births in the past three years, and are less likely to have more than one birth in the past three years compared to HIV negative women.

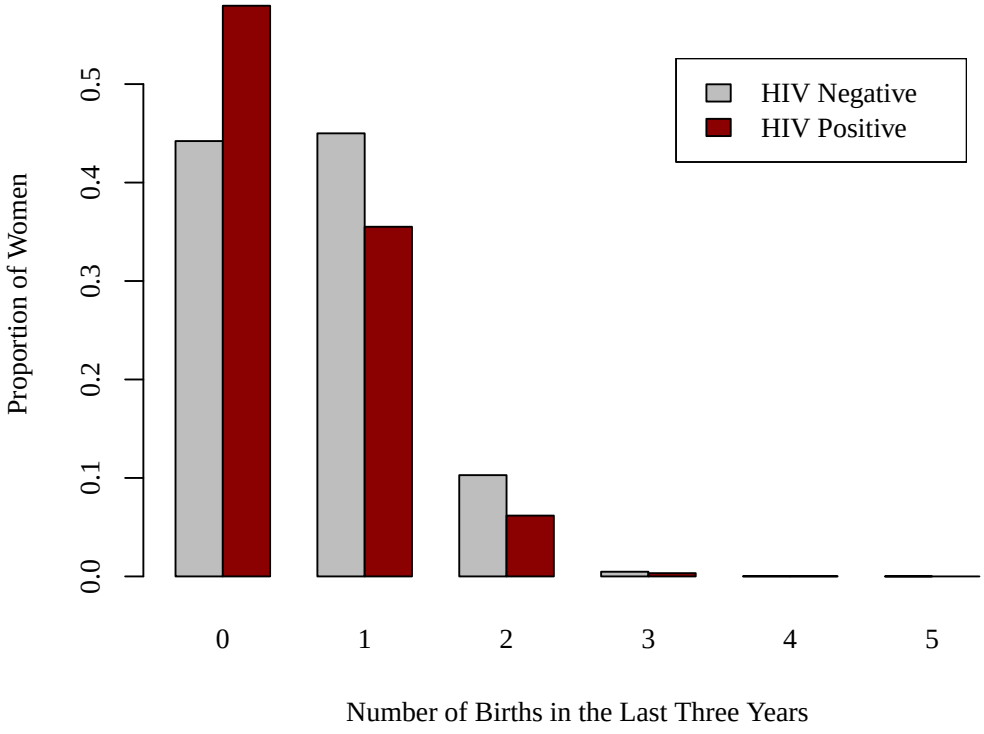


Fig. 1: Number of births by HIV status

The DHS collects data on a wide range of demographic and socioeconomic variables. Table 4 in Appendix A shows the mean and standard deviation for the variables I use in my analysis. HIV positive women are, on average, slightly older, more educated, and wealthier than HIV negative women, which confirms past research in this area. Furthermore, they are more likely to live in urban areas, less likely to be married, more likely to have used a condom during their last sexual encounter and have more partners on average compared to HIV negative women. To account for these observed differences, I include these variables as controls in my model.

I I use two variables as my indicator of risky sexual behavior - number of sexual partners (including husband) in the 12 months preceding the survey, and whether the woman reported using a condom during her last sexual encounter in the 12 months preceding the survey - as there is some evidence to suggest that the HIV epidemic in Sub-Saharan Africa is associated with changes in these risky sexual behaviors (Cheluget et al., 2006, Fylkesnes et al., 2001). I look at number of partners and condom use in the 12 month window preceding the survey as these are most likely to capture any behavioral change in response to becoming HIV positive. I also control for age at first sex as it has been shown to be a significant predictor of HIV infection (Pettifor et al. 2004), and may affect fertility.

I also examine differences in the fertility preferences of infected and uninfected women. Specifically, I use two measures of fertility preference - the ideal number of children reported by the woman, and whether or not she wants more children. The information for the ideal number of children

is collected via two possible questions. For women with living children, DHS asks questions such as "If you could go back to the time you did not have any children and could choose exactly the number of children to have in your whole life, how many would that be?". For women with no children, DHS asks questions such as "If you could choose exactly the number of children to have in your whole life, how many would that be?"(Integrated Demographic and Health Series, 2015). It is unclear whether women would change their answer to these questions after becoming HIV positive and learning about their infection status. Whether or not a woman reports wanting more children in the future may be more suited to capturing a change in fertility preferences as a result of becoming infected. Nonetheless, because I do not know when an individual becomes HIV positive, I cannot tell whether any association between HIV status and these measures of fertility preference is a behavioral response or simply a selection effect.

My final sample includes 32,759 women from the full sample of 1,489,959 women. I listwise delete all women for whom we have missing information on any of the variables of interest. It is important to note that there may be some selection bias both from the selection of women for testing as well as from the refusal of women to provide blood samples. DHS only collected blood samples from a subset of women interviewed for the main survey. In addition, the blood test was voluntary and women could refuse to provide a blood sample, which could introduce potential bias in my results. Table 3 in Appendix A shows the difference in means of my variables of interest by whether or not women were tested for HIV. I cannot distinguish between women who were asked for a blood sample but refused and women who were never asked for a blood sample. Women whose blood samples were collected for an HIV test were on average younger, had more births in the three years preceding the survey, less educated, less wealthy, more likely to be at a higher parity, less likely to live in an urban area, more likely to want more children, had a lower age at first sex, and had larger ideal family sizes. This suggests that generalizing my results to the entire population may be problematic due to potential selection bias. Future research will also need to explore the extent to which differences between women who were and were not tested could be affecting HIV prevalence estimates.

4 Methods

I assume that the main dependent variable, number of births in the three years preceding the survey, follows a poisson distribution. I also estimate linear models, but the poisson count model is a better fit, based on the Akaike Information Criterion (AIC). The poisson distribution requires that the mean of the dependent variable be equal to its variance, conditional on observables. Thus, overdispersion may be a problem. To deal with overdispersion, I use a negative binomial model as well, but the poisson count model has a lower AIC in all cases. I, therefore, do not include these results. All regressions are weighted by the HIV survey weights provided by DHS, and the standard errors are heteroscedasticity-consistent and clustered at the regional level. My full model specification takes the form:

$$Log[E(births_{i,rc,t}|\mathbf{X})] = Log[exposure_{i,rc,t}] + \beta HIV\,Positive_i + \gamma X_i + D_{rc} + D_t \qquad (1)$$

where $births_{i,rc,t}$ is the number of births to woman i, living in region rc, in the three years preceding the survey year t. $exposure_{i,rc,t}$ is the amount of exposure time (typically three years for

most women, unless their fifteenth birthday occurred during the interval). $HIVPositive_i$ is a dichotomous variable which takes the value of one if the individual has tested positive for HIV. X_i is a vector of covariates. I include country-region dummies, D_{rc} to capture differences across regions that are constant over time, and year dummies, D_t to capture differences over time that are constant across countries. The covariates in the model include the regional HIV prevalence, which would capture any community level behavioral response to the HIV epidemic. I control for age and age squared, since fertility has been shown to have a non-linear relationship with age. I control for parity, defined here as the number of births the woman already had prior to the births in the last three years. I also control for the number of years of education and the wealth quintile of the individual, since both of these have been shown to be related to fertility. I also include dummies for current marital status, urban/rural residence, and for never having been married. To examine the relative importance of biological versus behavioral factors, I estimate this model both with and without controls for risky sexual behavior, and separately for women who have and have not had an HIV test prior to the survey.

It is possible that the observed negative relationship between HIV infection and fertility may be due to selection effects. Unobserved, pre-existing differences between infected and uninfected women may be driving the observed differences in fertility. As a robustness check, I estimate the same model after one-to-one exact matching. One-to-one exact matching is the simplest way to obtain good matches for causal inference where the treatment was not randomized (Ho et al., 2007). Exact matching pairs each treated unit, i.e. an individual with positive HIV status, with a control unit, i.e. an uninfected individual with the same set of specified pre-treatment covariates. Individuals are matched on age, education, wealth quintile, urban/rural residence, age at first sex, year of survey, and country-region. It is important to note that because the data is cross-sectional it is impossible to determine whether any of these covariates are truly "pre-treatment". For instance, wealth and education may both change as a result of an individual getting infected i.e. due to the treatment. Thus, while I present the results as a robustness check, they should be interpreted with caution. Because there are sufficient one-to-one matches, the exact matching method does not need to balance the overall covariate distributions.

Finally, I examine the effect of HIV status and regional HIV prevalence on fertility preferences of women. I estimate the relationship between ideal number of children reported by women and their HIV status using a poisson count model. I estimate the relationship between whether women want more children and their HIV status using a logistic model. My model specifications are as follows:

$$Log[E(ideal_{i,rc,t}|\mathbf{X})] = \alpha + \beta HIVPositive_i + \gamma X_i + D_{rc} + D_t \tag{2}$$

$$Logit[E(want\_more_{i,rc,t}|\mathbf{X})] = \alpha + \beta HIVPositive_i + \gamma X_i + D_{rc} + D_t \tag{3}$$

where all variables are the same as Eq. 1; $ideal_{i,rc,t}$ is ideal number of children, and $want\_more_{i,rc,t}$ is a dichotomous variable that is equal to one if a woman wants more children.

5 Results and Discussion

5.1 HIV and Fertility

HIV positive women appear to have significantly fewer number of births in the last three years compared to HIV negative women. Figure 2 shows the estimated coefficients from the poisson model described by Eq. 1 (full results are presented in Table 5 in Appendix B). Model 1 presents estimates without controls for risky sexual behavior, year dummies, and country-region dummies; Model 2 includes controls for number of partners, condom use and age at first sex; Model 3 adds year dummies and country-region dummies. The estimated coefficient on HIV status is very similar in magnitude across all three model specifications, and remains significant at the 99 percent level.

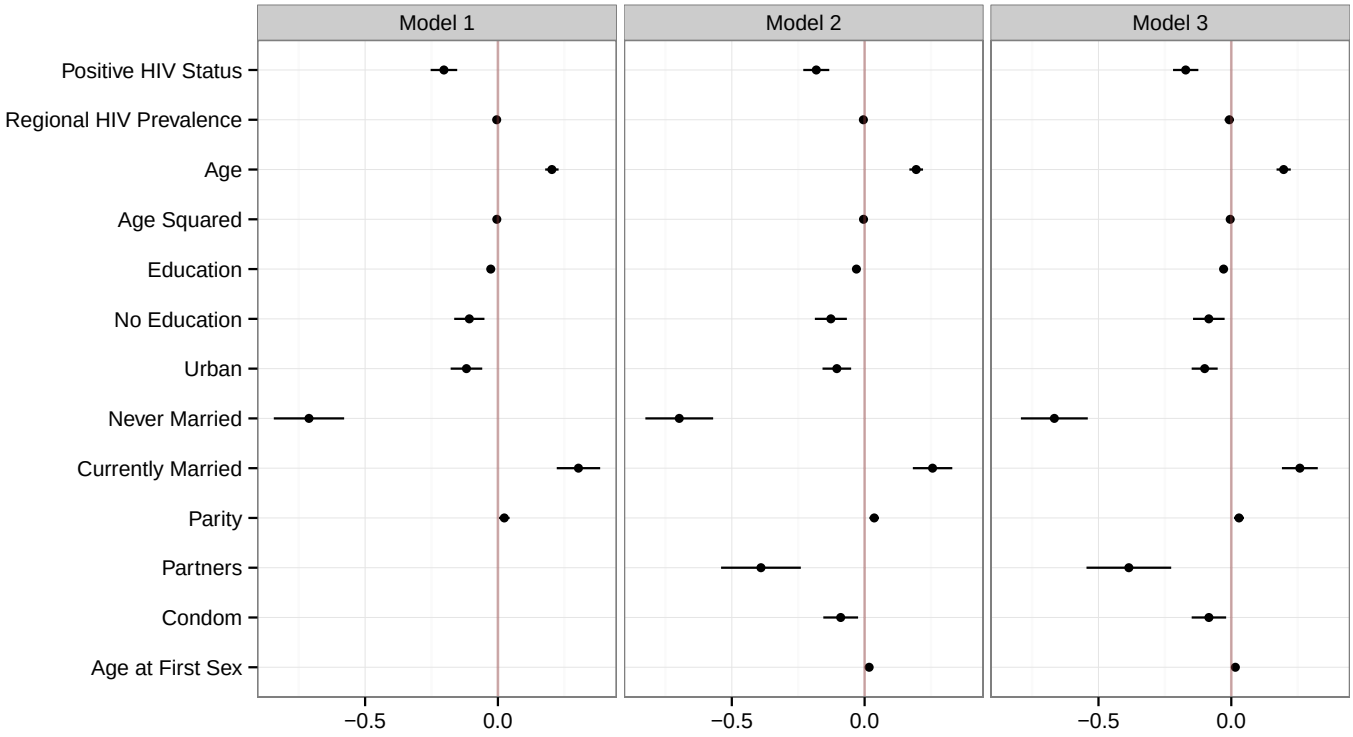


Fig. 2: Relationship between individual's positive HIV status and the number of births in the last three years. Estimated coefficients from a poisson regression are plotted along with the 95% confidence intervals. Women with non-missing HIV status are used in the regressions. Education is measured in years. Regressions also include dummies for wealth quintiles. Models (2) and (3) control for number of partners, condom use and age at first sex. Model (3) includes country by region dummies and year dummies. HIV weights which adjust for individual sampling probabilities and test non-response rates are used in the regressions. Heteroscedasticity-consistent standard errors are clustered at the regional level.

The estimated coefficient on $HIVPositive_i$ from the full specification (Model 3) is -0.172 (95% CI: -0.219 to -0.124) which translates to an incident rate ratio of 0.84. Thus, HIV positive women appear to have 0.84 times the number of births that HIV negative women have in the three years preceding the survey (about 16% fewer births). For instance, for every two children that an HIV negative woman has, an HIV positive woman with the exact same characteristics will have, on average, 1.68 children. The results hold after one-to-one exact matching on covariates (matching

results are presented in Table 9 in Appendix C). These results do not appear to be driven by any particular country, as the estimated coefficients on HIV status and its standard errors are very similar when the analysis is conducted separately for each country (results not shown). Age, living in a rural area, being married at the time of the survey, parity, and age at first sex are positively associated with the number of births in the last three years. Years of education, never having married, number of partners, wealth and condom use are negatively associated with the number of children in the last three years. These associations are consistent with findings from studies looking at the determinants of fertility in Sub-Saharan Africa.

The estimates from Model 3 suggest that higher regional HIV prevalence is associated with lower fertility. The coefficient on regional HIV prevalence may be biased due to endogeneity. For instance, it is possible that HIV prevalence in a region is higher because women in that region are having more births i.e. engaging in riskier sexual behavior. Thus, reverse causality would cause the coefficient to be positive. However, my analysis yields a negative coefficient although it is statistically insignificant in the full model. I also restrict the sample to uninfected women only and examine whether there is an association between regional HIV prevalence and fertility. If there is a behavioral response among uninfected women to the HIV epidemic, then we would expect the regional HIV prevalence to be a predictor of fertility. The estimated coefficient on regional HIV prevalence, when I restrict the sample to uninfected women only, is small in magnitude and not statistically significant (results not shown). Thus, I find no evidence of a behavioral response to the HIV epidemic among uninfected women.

Next, I estimate the analogue of Eq. 1 separately by single year of age and by single year of education, leaving out the age and education variables where appropriate. Figure 3 shows the estimated coefficient, and the 95% confidence interval, on positive HIV status by age and years of education.

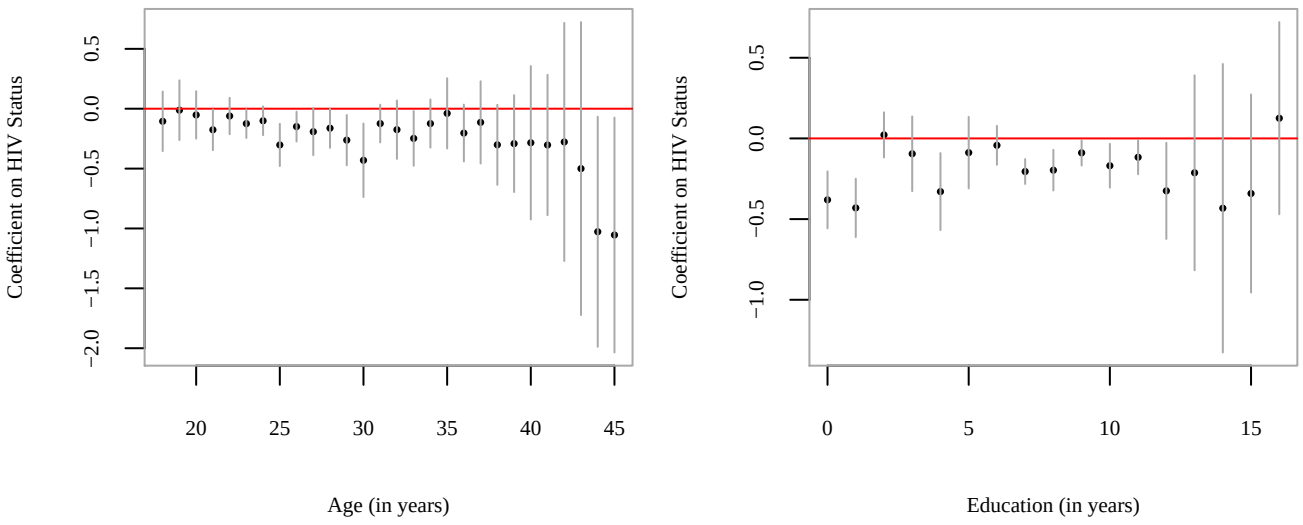


Fig. 3: Relationship between HIV positive status and fertility by age and education. Estimated coefficients on $HIVPositive_i$ from estimating the analogue of Eq. 1 separately by single year of age and by single year of education are plotted along with the 95% confidence intervals. Women with non-missing HIV status are used in the regressions. HIV weights which adjust for individual sampling probabilities and test non-response rates are used in the regressions. Heteroscedasticity-consistent standard errors are clustered at the regional level.

Restricting the sample to specific ages or years of education increases the standard error for the estimates because of the greatly reduced sample sizes. However, the interesting feature is that the estimated associations between HIV and fertility remain very similar and fairly constant over age and years of education. This suggests that the pathway through which HIV is influencing fertility is likely to be unaffected by age and years of education.

5.2 Behavioral Pathway: Risky Sexual Behavior

The estimated coefficient on HIV status barely changes when controls are included for indicators of risky sexual behavior (Model 3 in Figure 2). When controls are added for number of partners in the last 12 months, whether or not the respondent reported using a condom during their most recent sexual encounter in the last 12 months, and age at first sex, the estimated coefficient on $HIVPositive_i$ changes from -0.182 to -0.172 (Models 2 and 3 in Figure 2 above and Table 5 in Appendix B). To the extent that the number of partners and condom use in the last 12 months are capturing the behavioral response of women to either their own status or the regional HIV prevalence, this suggests that the relationship between HIV status and fertility is not operating primarily through changes in risky sexual behavior.

It is possible that these variables are poor indicators of risky sexual behavior, perhaps because of potential social desirability bias. It is also possible that HIV positive women are changing their sexual behavior in other ways, which in turn could be affecting their fertility. However, recent

literature has provided little evidence for a shift in sexual behavior patterns in response to the HIV epidemic. For instance, Oster (2005) uses DHS data to show that sexual behavior in a sample of African countries has changed very little over the course of the epidemic. Thus, the impact of any changes in sexual behavior on fertility is also likely to be small.

5.3 Behavioral Pathway: Fertility Preference

Next, I estimate the impact of being HIV positive on women's fertility preferences. The results from estimating Eq. 2 and Eq. 3 are presented in Figures 4 and 5 (full results are presented in Tables 6 and 7 in Appendix B).

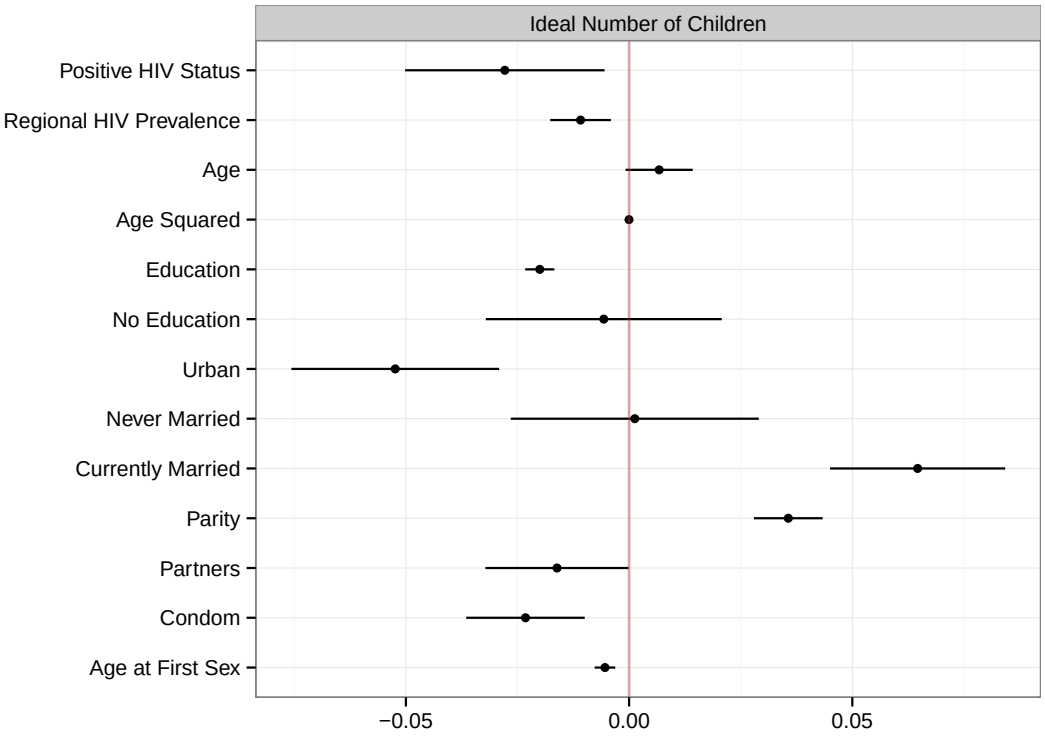


Fig. 4: Relationship between individual's positive HIV status and the ideal number of children. Estimated coefficients from a poisson regression are plotted along with the 95% confidence intervals. Women with non-missing HIV status are used in the regressions. Education is measured in years. Regressions also include dummies for wealth quintiles, country by region dummies and year dummies. HIV weights which adjust for individual sampling probabilities and test non-response rates are used in the regressions. Heteroscedasticity-consistent standard errors are clustered at the regional level.

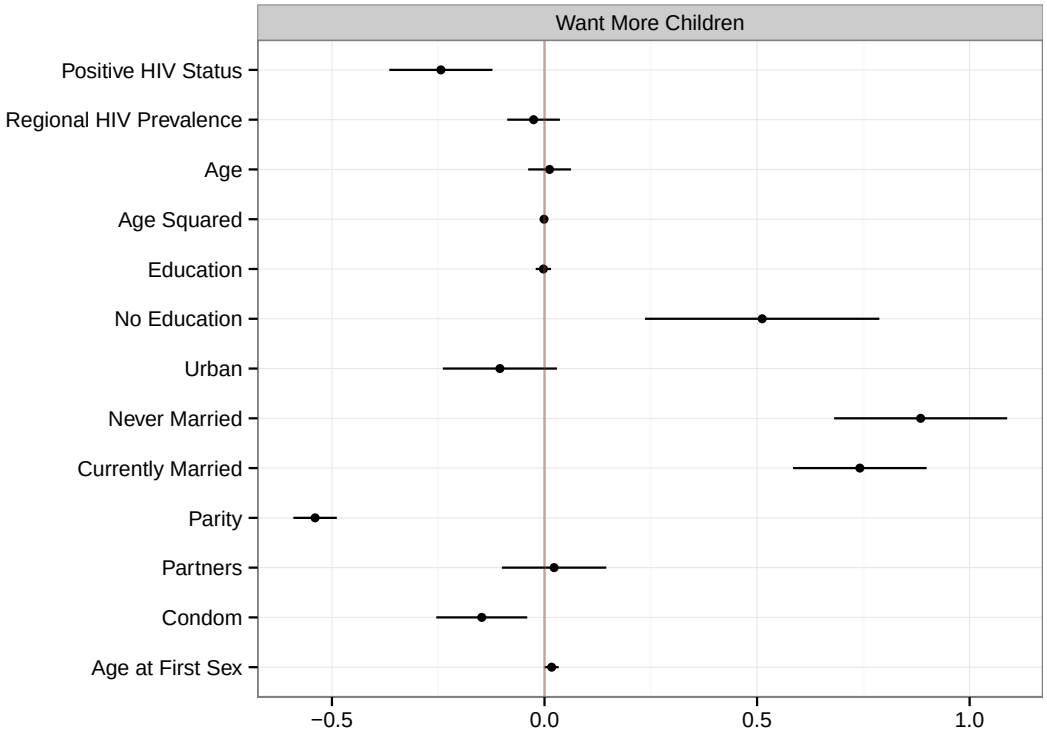


Fig. 5: Relationship between individual's positive HIV status and whether or not she wants more children. Estimated coefficients from a logistic regression are plotted along with the 95% confidence intervals. Women with non-missing HIV status are used in the regressions. Education is measured in years. Regressions also include dummies for wealth quintiles, country by region dummies and year dummies. HIV weights which adjust for individual sampling probabilities and test non-response rates are used in the regressions. Heteroscedasticity-consistent standard errors are clustered at the regional level.

HIV positive women have significantly smaller ideal family sizes. The estimated coefficient on $HIVPositive_i$ from estimating Eq. 2 is -0.028 (95% CI: -0.050 to -0.005) which corresponds to an incident rate ratio of 0.97. In other words, the ideal number of births for HIV positive women is 0.97 times the ideal number of births for HIV negative women. This is a very small effect, but nonetheless suggests that there is some heterogeneity in fertility preferences between HIV positive and HIV negative women. Women living in high prevalences region also appear to have significantly lower ideal number of children compared to women living in lower prevalence regions. The interaction between regional HIV prevalence and an individual's HIV status is small in magnitude and not significant (results not shown). HIV positive women are also significantly less likely to want more children compared to women who are HIV negative. The coefficient on $HIVPositive_i$ from estimating Eq. 3 is -0.244 (95% CI: -0.365 to -0.122) which translates to an odds ratio of 0.78. This suggests that the odds for HIV positive women to want more children is 0.78 times the odds for HIV negative women, holding all else equal.

Given that HIV positive women have significantly smaller ideal family sizes, it is possible that the negative relationship between HIV status and number of children in the last three years is working primarily through HIV positive women wanting smaller families. In order to explore this possibility, I examine how the ideal family size influences fertility and the extent to which this might differ for HIV positive versus HIV negative women. Table 1 shows the results of controlling for ideal family size in Eq. 1 (Model 2), and including an interaction term between HIV status and

the ideal family size (Model 3).

Table 1: Relationship between fertility and the ideal number of children

	Dependent variable:		
	Number of births in the last three years		
	(1)	(2)	(3)
Positive HIV Status	−0.172***	−0.169***	−0.073
	(0.024)	(0.024)	(0.064)
Ideal Number of Children		0.019***	0.021***
		(0.007)	(0.006)
Ideal × Positive HIV Status			−0.025*
			(0.014)
Observations	32,759	32,759	32,759
Akaike Inf. Crit.	57,353.390	57,334.140	57,332.210
Note:			*p<0.1; **p<0.05; ***p<0.01

Table 1: Estimates from a poisson regression. Women with non-missing HIV status are used in the regressions. All regressions control for age, age squared, years of education, no education, urban/rural status, marital status, never been married, parity, number of partners, condom use, age at first sex, and include dummies for wealth quintiles, country by region dummies and year dummies. HIV weights which adjust for individual sampling probabilities and test non-response rates are used in the regressions. Heteroscedasticity-consistent standard errors are clustered at the regional level.

Controlling for ideal family size barely changes the estimate for the coefficient on HIV status. The coefficient on the interaction term is small in magnitude and not significant at the 95% level. Thus, even though HIV positive women appear to want smaller family sizes, this result suggests that this alone cannot fully explain why HIV positive women are having fewer children.

Somewhat puzzlingly, the relationship between HIV status and fertility preferences holds even after restricting the sample to women who have never been tested for HIV, although the estimated coefficient on $HIVPositive_i$ is smaller in magnitude and is significant at the 90% level (for both Eq. 2 and Eq. 3). This suggests that there may be a selection effect at play; women who are more likely to be HIV positive may also be more likely to want smaller families, for unobserved reasons, regardless of whether or not they are aware of their HIV status.

5.4 Biological Pathway

To examine the relative importance of the biological pathway versus the behavioral pathway, I estimate the full model described by Eq. 1 for the sample of women who have never been tested for HIV prior to the survey. Here, I make the assumption that women who report never having been tested for HIV are unaware of their HIV status. Figure 6 shows the coefficients from estimating the full model for the sample of women who report never having had an HIV test prior to the survey (Model 1), and the sample of women who report having had at least one HIV test prior to the survey (Model 2). Full results are presented in Table 8 in Appendix B.

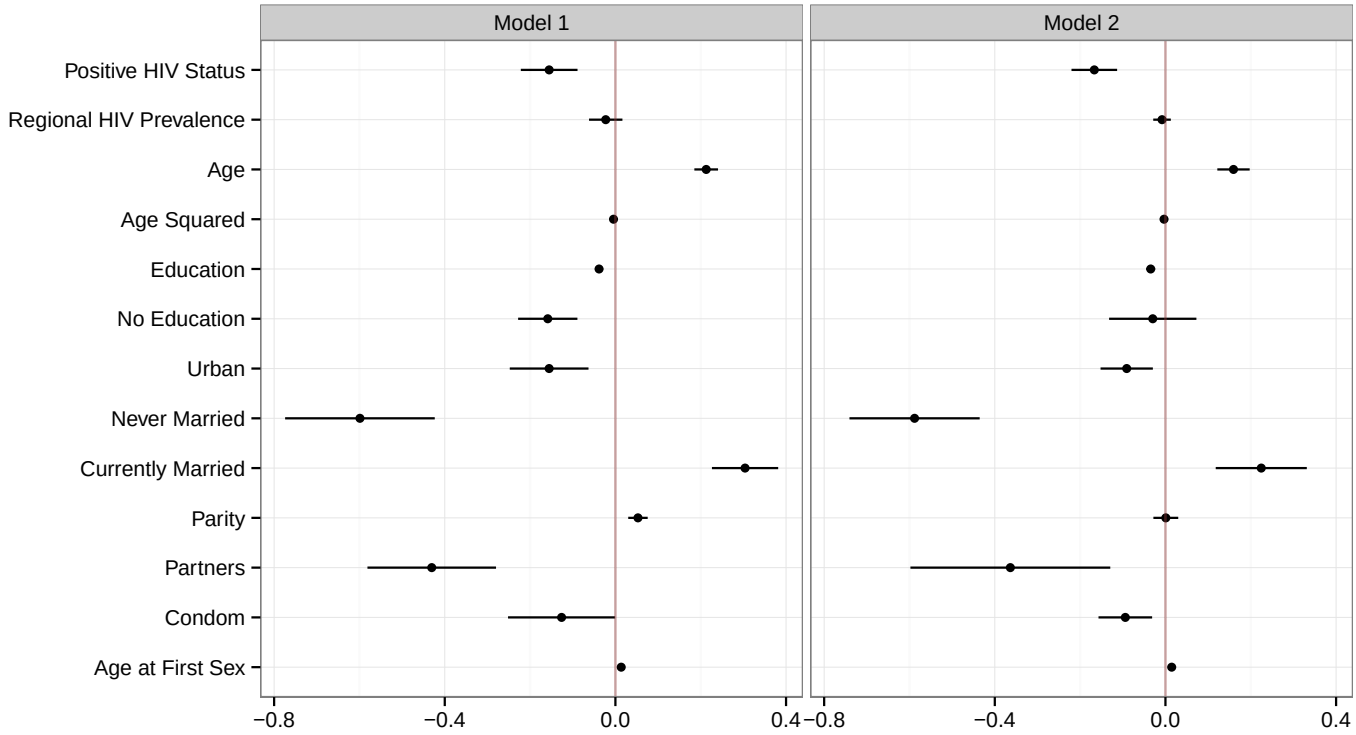


Fig. 6: Relationship between individual's positive HIV status and the number of births in the last three years. Estimated coefficients from a poisson regression are plotted along with the 95% confidence intervals. Model 1 restricts the sample to women who have never had an HIV test. Model 2 restricts the sample to women who have had at least one HIV test in the past. Education is measured in years. Regressions also include dummies for wealth quintiles, country by region dummies and year dummies. HIV weights which adjust for individual sampling probabilities and test non-response rates are used in the regressions. Heteroscedasticity-consistent standard errors are clustered at the regional level.

It is possible that women who have never had an HIV test may still be aware of their HIV status, and may change their fertility preferences or sexual behavior as a response. However, to the extent that this variable captures actual knowledge of one's HIV status, we would expect to see no effect of being HIV positive on fertility in the sample of women who have never been tested prior to the survey if there was no physiological effect of the disease on fertility, and the sole mechanism was an indirect behavioral response. On the other hand, if there is a physiological mechanism underlying the relationship, then we would expect to see an association between HIV status and fertility regardless of whether women are aware of their HIV status. Similarly, if a behavioral mechanism is present we would expect to see a larger effect of being HIV positive on fertility for women who have knowledge of their HIV status. The estimated coefficient on $HIVPositive_i$ is statistically significant and similar in magnitude for the two samples (-0.155 in Model 1 versus -0.167 in Model 2). The estimated coefficient is about seven percent smaller when the sample is restricted to women who have never been tested. This suggests that the biological pathway is likely to be the dominant mechanism underlying the observed relationship between HIV status and fertility.

6 Conclusion

This paper confirms past findings of lower fertility among HIV positive women compared to uninfected women, and attempts to distinguish between the various hypothesized pathways underlying this empirical relationship. I find that this relationship is consistent over all ages and for women with different years of education. Similar to Young (2007), I attempt to distinguish between a direct biological link between HIV and fertility and an indirect behavioral response that could explain the relationship. Unlike Young (2007), which examines the relationship between community HIV prevalence and fertility, I utilize the most recent rounds of the DHS, which allows me to link birth histories of women to their HIV test status.

Young (2007) argues that the relationship between the HIV epidemic and reduced fertility, "reflects broad communal responses, rather than the physiological or behavioral response of infected women alone." I find no evidence for a community response to the epidemic. I argue that the biological pathway is the dominant mechanism underlying the observed differences in fertility among infected and uninfected women. While HIV positive women desire fewer children and are less likely to want more children, this difference in fertility preferences is small in magnitude and cannot fully explain the observed relationship. Furthermore, when I restrict the sample to women who have never been tested for HIV prior to the survey, being HIV positive remains significantly associated with reduced fertility. Assuming that these women were unaware of their HIV status, this suggests that the adverse biological effects of the disease is likely to be the dominant mechanism underlying the relationship between HIV and fertility at the individual level.

There are several limitations to this study. First, there may be selection bias due to the sampling of women for HIV testing and the refusal of women to give blood. Previous work has concluded that HIV prevalence estimates based on the DHS data are not biased by nonresponse (Mishra et al., 2006). Nonetheless, this is a concern that future work will need to address. Second, the cross-sectional nature of the DHS data makes it impossible to know when a woman who tested positive for HIV during the survey actually became HIV positive. Furthermore, it is also not possible to know for certain whether women are aware of their HIV status. Thus, I use number of births in the three years preceding the survey as my measure of fertility, and examine the relationship between HIV status and fertility for both women who have and have not had an HIV test prior to the survey. Third, the cross-sectional data also makes causal inference difficult. It is impossible to rule out reverse causality or omitted variables bias. As a robustness check, I use exact matching to pair infected and uninfected women based on several covariates. However, it is impossible to determine whether these covariates are truly "pre-treatment", a necessary condition for causal inference.

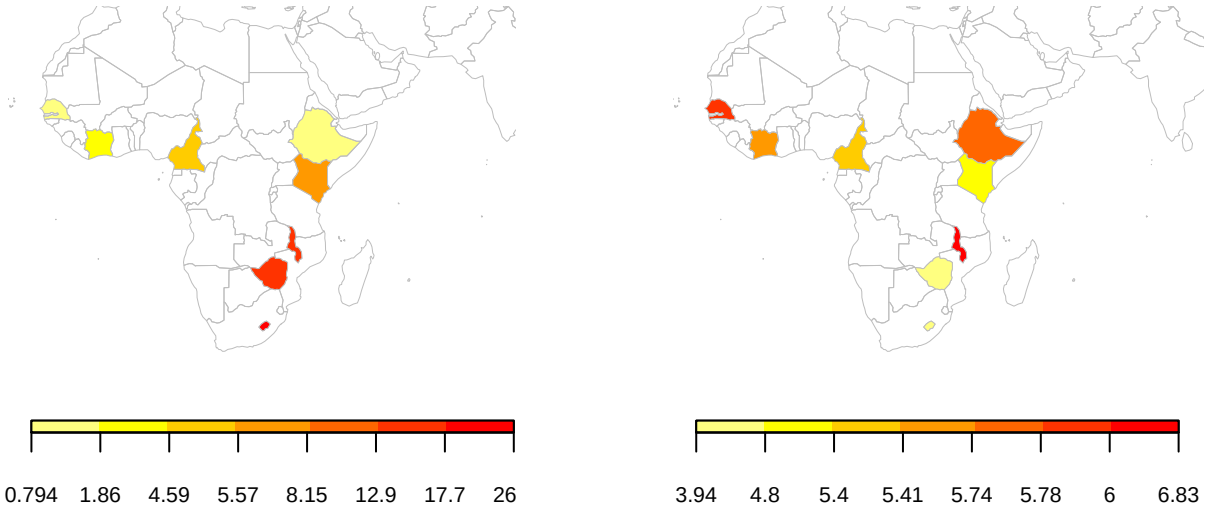
Finally, my analysis does not account for the widespread introduction of antiretroviral therapy (ART) in Africa in the 2000s. Kaida et al. (2006) propose several possible ways in which the use of ART could impact the fertility of infected women. They speculate that, "as antiretroviral therapy becomes increasingly accessible in Sub-Saharan Africa, the associated improvements in health, quality of life, and survival are anticipated to influence both the biological and behavioral fertility determinants of infected women." ART may increase the fecundity of HIV positive women. Drugs that reduce the probability of mother-to-child transmission may alter the fertility preferences of infected women. The availability of drugs that improve health and quality of life, while suppressing symptoms, may also encourage riskier sexual behavior (Juhn et al., 2013). However, there has been little empirical work to examine these hypotheses. My results are similar across both survey waves, although this may be due to the fact that ART was already in use by the time my first survey

round was conducted (between 2003 and 2006). Examining the role of ART in mediating the relationship between HIV and fertility is a potential direction for future research in this area.

A Data Description

Table 2: Datasets

Cote d'Ivoire	2011	2005
Cameroon	2011	2004
Ethiopia	2011	2005
Kenya	2008	2003
Lesotho	2009	2004
Malawi	2010	2004
Senegal	2010	2005
Zimbabwe	2011	2006



(a) HIV Prevalence (b) Total Fertility Rate

Fig. 7: HIV Prevalence and Total Fertility Rates (TFR) by Country: HIV prevalence is measured in percentage points and calculated using test results for men and women between the ages of 15 and 49, and the HIV weights provided by DHS. The period TFR, which is a sum of the period age-specific fertility rates, is defined as the average number of children a women would bear if she were to survive to the end of her childbearing period, and at each age experience the ASFR observed in that period. I calculate the period TFR using the number of children born to women in each age group in the three year interval preceding the survey.

Table 3: Descriptive statistics by HIV test status

	Not Tested for HIV	Tested for HIV
n	1406	32759
Number of children born in past three years	0.56	0.64*
Age	28.67	27.65*
Education	8.56	6.54*
No education (1 = Zero years of education)	0.08	0.17*
Wealth quintile (1 = Poorest, 5 = Richest)	3.79	3.18*
Never married (1 = Never been married)	0.11	0.12
Currently married (1 = Currently married)	0.82	0.80
Parity	1.95	2.19*
Urban (1 = Urban residence status)	0.55	0.35*
Number of partners	1.06	1.05
Used condoms during last intercourse (1 = Used condoms during last intercourse)	0.15	0.14
Age at First Sex	18.21	17.13*
Wants more children (1 = Wants more children)	0.55	0.62*
Ideal number of children	3.71	4.33*

Table 3: Column 1 is the sample of women who were not tested for HIV. Column 2 is the sample of women who were tested for HIV. * indicates whether the means are significantly different at the 95% level.

Table 4: Descriptive statistics by HIV status

	HIV Positive	HIV Negative
n	4807	27952
Number of children born in past three years	0.49	0.67
	(0.63)	(0.68)
Age	28.81	27.42
	(7.21)	(8.09)
Education	7.48	6.45
	(3.30)	(4.28)
No education	0.06	0.19
(1 = Zero years of education)	(0.24)	(0.39)
Wealth quintile	3.42	3.24
(1 = Poorest, 5 = Richest)	(1.35)	(1.41)
Never married	0.10	0.12
(1 = Never been married)	(0.31)	(0.32)
Currently married	0.69	0.82
(1 = Currently married)	(0.46)	(0.38)
Parity	2.06	2.18
	(1.88)	(2.36)
Urban	0.41	0.37
(1 = Urban residence status)	(0.49)	(0.48)
Number of partners	1.10	1.05
	(0.96)	(0.42)
Used condoms during last intercourse	0.26	0.12
(1 = Used condoms during last intercourse)	(0.44)	(0.33)
Age at First Sex	17.29	17.18
	(2.82)	(2.93)
Wants more children	0.48	0.64
(1 = Wants more children)	(0.50)	(0.48)
Ideal number of children	3.70	4.36
	(1.8)	(2.16)

Table 4: Means and standard deviations (in parentheses) of main variables. Column 1 is the sample of HIV positive women; column 2 is the sample of HIV negative women.

B Main Results

Table 5: Relationship between individual's positive HIV status and the number of children born

	Dependent variable:		
	Number of births in the last three years		
	(1)	(2)	(3)
Positive HIV Status	-0.203^{***}	-0.182^{***}	-0.172^{***}
	(0.026)	(0.025)	(0.024)
Regional HIV Prevalence	-0.005^{*}	-0.005^{**}	-0.007
	(0.002)	(0.002)	(0.009)
Age	0.203^{***}	0.194^{***}	0.197^{***}
	(0.013)	(0.013)	(0.014)
Age Squared	-0.004^{***}	-0.004^{***}	-0.004^{***}
	(0.0002)	(0.0002)	(0.0002)
Education	-0.027^{***}	-0.031^{***}	-0.029^{***}
	(0.003)	(0.003)	(0.003)
No Education	-0.108^{***}	-0.127^{***}	-0.085^{***}
	(0.029)	(0.031)	(0.030)
Urban	-0.118^{***}	-0.105^{***}	-0.100^{***}
	(0.030)	(0.027)	(0.025)
Never Married	-0.711^{***}	-0.698^{***}	-0.666^{***}
	(0.068)	(0.065)	(0.064)
Currently Married	0.303^{***}	0.256^{***}	0.258^{***}
	(0.042)	(0.038)	(0.034)
Parity	0.024^{**}	0.036^{***}	0.029^{***}
	(0.010)	(0.009)	(0.010)
Partners		-0.390^{***}	-0.386^{***}
		(0.077)	(0.081)
Condom		-0.090^{***}	-0.084^{**}
		(0.033)	(0.033)
Age at First Sex		0.017^{***}	0.015^{***}
		(0.003)	(0.002)
Year Dummies	*No*	*No*	*Yes*
Country-Region Dummies	*No*	*No*	*Yes*
Observations	32,759	32,759	32,759
Akaike Inf. Crit.	57,668.620	57,520.750	57,353.390
Note:			*p<0.1; **p<0.05; ***p<0.01

Table 5: Estimates from a poisson regression. Women with non-missing HIV status are used in the regressions. Education is measured in years. Regressions also include dummies for wealth quintiles. Regression (3) includes country by region dummies and year dummies. Regressions (2) and (3) control for number of partners, condom use and age at first sex. HIV weights which adjust for individual sampling probabilities and test non-response rates are used in the regressions. Heteroscedasticity-consistent standard errors are clustered at the regional level.

Table 6: Relationship between HIV status and the ideal number of children

	Dependent variable:	
	Ideal Number of Children	
	(1)	(2)
Positive HIV Status	−0.028**	−0.024*
	(0.011)	(0.014)
Regional HIV Prevalence	−0.011***	−0.009*
	(0.003)	(0.005)
Age	0.007*	0.009*
	(0.004)	(0.005)
Age Squared	−0.00003	−0.0001
	(0.0001)	(0.0001)
Education	−0.020***	−0.023***
	(0.002)	(0.003)
No Education	−0.006	−0.015
	(0.013)	(0.016)
Urban	−0.052***	−0.064***
	(0.012)	(0.016)
Never Married	0.001	−0.001
	(0.014)	(0.019)
Currently Married	0.065***	0.055***
	(0.010)	(0.014)
Parity	0.036***	0.031***
	(0.004)	(0.005)
Partners	−0.016**	−0.035***
	(0.008)	(0.006)
Condom	−0.023***	−0.017
	(0.007)	(0.012)
Age at First Sex	−0.005***	−0.006***
	(0.001)	(0.002)
Observations	32,759	15,635
Akaike Inf. Crit.	121,443.700	61,406.200
Note:	*p<0.1; **p<0.05; ***p<0.01	

Table 6: Estimates from a poisson regression. Women with non-missing HIV status are used in model 1. Model 2 is restricted to women who have never been tested for HIV. Education is measured in years. Regressions also include dummies for wealth quintiles, country by region dummies and year dummies. HIV weights which adjust for individual sampling probabilities and test non-response rates are used in the regressions. Heteroscedasticity-consistent standard errors are clustered at the regional level.

Table 7: Relationship between HIV status and the desire to have more children

	Dependent variable:	
	Want more children	
	(1)	(2)
Positive HIV Status	−0.244***	−0.157*
	(0.062)	(0.089)
Regional HIV Prevalence	−0.026	−0.025
	(0.032)	(0.032)
Age	0.012	0.043
	(0.026)	(0.033)
Age Squared	−0.001***	−0.002***
	(0.0004)	(0.0005)
Education	−0.003	−0.011
	(0.009)	(0.013)
No Education	0.512***	0.423***
	(0.141)	(0.120)
Urban	−0.105	−0.113
	(0.069)	(0.079)
Never Married	0.885***	1.130***
	(0.104)	(0.147)
Currently Married	0.742***	0.880***
	(0.080)	(0.109)
Parity	−0.540***	−0.493***
	(0.026)	(0.027)
Partners	0.023	−0.042
	(0.063)	(0.084)
Condom	−0.148***	−0.035
	(0.055)	(0.071)
Age at First Sex	0.017**	0.010
	(0.008)	(0.013)
Observations	32,759	15,635
Akaike Inf. Crit.	28,865.060	13,726.080
Note:	*p<0.1; **p<0.05; ***p<0.01	

Table 7: Estimates from a logistic regression. Women with non-missing HIV status are used in model 1. Model 2 is restricted to women who have never been tested for HIV. Education is measured in years. Regressions also include dummies for wealth quintiles, country by region dummies and year dummies. HIV weights which adjust for individual sampling probabilities and test non-response rates are used in the regressions. Heteroscedasticity-consistent standard errors are clustered at the regional level.

Table 8: Relationship between HIV status and the number of children born by whether or not women were tested prior to the survey

	Dependent variable:	
	Number of births in the last three years	
	(1)	(2)
Positive HIV Status	-0.155^{***}	-0.167^{***}
	(0.034)	(0.027)
Regional HIV Prevalence	-0.023	-0.008
	(0.020)	(0.011)
Age	0.213^{***}	0.160^{***}
	(0.014)	(0.019)
Age Squared	-0.004^{***}	-0.003^{***}
	(0.0003)	(0.0003)
Education	-0.038^{***}	-0.035^{***}
	(0.005)	(0.005)
No Education	-0.159^{***}	-0.030
	(0.035)	(0.052)
Urban	-0.155^{***}	-0.091^{***}
	(0.047)	(0.031)
Never Married	-0.599^{***}	-0.588^{***}
	(0.089)	(0.078)
Currently Married	0.304^{***}	0.225^{***}
	(0.040)	(0.054)
Parity	0.053^{***}	0.001
	(0.012)	(0.015)
Partners	-0.431^{***}	-0.364^{***}
	(0.077)	(0.120)
Condom	-0.126^{**}	-0.094^{***}
	(0.064)	(0.032)
Age at First Sex	0.014^{***}	0.015^{***}
	(0.004)	(0.004)
Ever had and HIV Test	*No*	*Yes*
Observations	15,635	17,124
Akaike Inf. Crit.	26,067.780	30,818.340
Note:		*p<0.1; **p<0.05; ***p<0.01

Table 8: Estimates from a poisson regression. Model 1 restricts the sample to women who have never had an HIV test. Model 2 restricts the sample to women who have had at least one HIV test in the past. Education is measured in years. Regressions also include dummies for wealth quintiles, country by region dummies and year dummies. HIV weights which adjust for individual sampling probabilities and test non-response rates are used in the regressions. Heteroscedasticity-consistent standard errors are clustered at the regional level.

C Additional Results

Table 9: Relationship between individual's positive HIV status and the number of children born after matching

	Dependent variable:		
	Number of births in the last three years		
	(1)	(2)	(3)
Positive HIV Status	−0.189**	−0.178**	−0.177**
	(0.084)	(0.084)	(0.084)
Regional HIV Prevalence	0.004	0.002	0.016
	(0.007)	(0.008)	(0.048)
Age	0.129**	0.117*	0.119*
	(0.062)	(0.063)	(0.068)
Age Squared	−0.003***	−0.003**	−0.003**
	(0.001)	(0.001)	(0.001)
Education	−0.025	−0.029	−0.046
	(0.021)	(0.022)	(0.029)
No Education	0.065	0.031	−0.061
	(0.194)	(0.197)	(0.232)
Urban	−0.059	−0.050	−0.184
	(0.161)	(0.164)	(0.213)
Never Married	−0.300	−0.283	−0.250
	(0.218)	(0.218)	(0.224)
Currently Married	0.382***	0.300**	0.300**
	(0.141)	(0.145)	(0.148)
Parity	−0.007	−0.004	−0.007
	(0.037)	(0.038)	(0.040)
Partners		−0.208	−0.193
		(0.209)	(0.216)
Condom		−0.292**	−0.236
		(0.149)	(0.153)
Age at First Sex		0.013	0.027
		(0.024)	(0.027)
Year Dummies	*No*	*No*	*Yes*
Country-Region Dummies	*No*	*No*	*Yes*
Observations	939	939	939
Akaike Inf. Crit.	1,819.204	1,819.396	1,878.798
Note:			*p<0.1; **p<0.05; ***p<0.01

Table 9: Estimates from a poisson regression. Individuals with positive HIV status are matched to a control unit using exact matching. The individuals are matched on age, education, wealth quintile, rural/urban residence, age at first sex, year of survey, and country-region. Education is measured in years. Regressions also include dummies for wealth quintiles.

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