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Abstract

In many Sub-Saharan countries the HIV epidemic has spread to over 10% of the working-age population, and is likely to affect economically relevant behaviour. We evaluate the impact of the HIV/AIDS epidemic on the reproductive behaviour for women in Malawi, allowing for a heterogeneous response depending on age and prior number of births. HIV/AIDS increases the probability that a young woman would give birth to her first child, while it decreases the probability to give birth of older women or of young women who have already given birth. The resulting change in the distribution of fertility across age groups is likely to be more demographically and economically important than changes in the total number of children a woman gives birth to.

Keywords: AIDS, Demographic Transition, HIV, Fertility, Malawi,

JEL classification: I10, J13, O12

1. Introduction

In many Sub-Saharan countries, HIV has spread to over 10% of the working-age population, making the epidemic a serious economic and social problem. There is no doubt that HIV/AIDS has wide-ranging consequences for households affected by the disease, but it is less obvious what the economic effects are at the national level. For instance, a number of studies have evaluated the nationwide economic effects of HIV/AIDS by testing its impact on growth, but with very mixed results (Bloom and Mahal, 1997; Bell et al., 2004; Corrigan et al., 2005; Young, 2005, 2007; McDonald and Roberts, 2006; Werker et al., 2006; Papageoriou and Stoytcheva, 2008; Santaeulalia-Llopis, 2008). One reason for the inconclusive results is probably that HIV/AIDS affects growth through many channels, whose relative importance is difficult to assess.

One channel that has received attention recently is how HIV/AIDS affects fertility. The course of fertility can have a decisive effect on the future development of GDP per capita in countries with high HIV prevalence (Young, 2005; Kalemli-Ozcan, 2009a). Moreover, fertility and its distribution across ages will have an impact on various socioeconomic problems, like child mortality, mother-to-child-transmission of HIV, the number of orphans, as well as demand for education. However, as Glick (2006) notes, there is no consensus on the extent and direction of the effects of HIV/AIDS on fertility.

The most common view is that HIV/AIDS leads to lower fertility (Ntozi, 2002; Epstein, 2004; Lewis et al., 2004). It is largely based on studies that analyse the fertility of HIV-positive women, whose fertility is believed to decline for mainly physiological reasons (Fabiani et al., 2006). However, recently a few studies have focussed on changes in fertility behaviour among all women, including those who are HIV negative. According to Young (2005; 2007), women desire to have fewer children because of the risk of infection, and the HIV epidemic should thus reduce fertility. Young supports his hypotheses with empirical evidence from Sub-Saharan countries. He also finds that the resulting improvement in the dependency ratio outweighs the negative economic effects of the disease, so that in the long run it leads to higher GDP per capita. Kalemli-Ozcan (2003; 2009a), Soares (2005) and Lorentzen

et al. (2008), on the other hand, argue for a positive association between mortality and fertility, so that an HIV-induced increase in mortality could raise fertility. Yet, Juhn et al. (2008), Fortson (2008), Fink and Linnemayr (2008) and Kalemli-Ozcan (2009a; 2009b) find the net effect on fertility to be very small or non-existent.

We evaluate the impact of communal HIV prevalence rates on realised fertility in Malawi – which is likely to be representative of at least several Eastern and Southern African countries – with focus on the behavioural response of the general female population, i.e. among non-infected as well infected women. In contrast to earlier studies, we include the possibility of a heterogeneous fertility response to the HIV epidemic, depending on the woman's age and number of children she has had.

Childbearing is an integral part of adulthood in most societies, and it is likely to be particularly important in societies where women have many children, such as in Malawi. This should make women respond differently depending on the stage of the reproductive life cycle. It is unlikely that women with no or few children would voluntarily abstain from giving birth. Instead of abstaining from births, women might give birth earlier when the risk of being HIV positive is lower. Using a sample from Malawi, Noël-Miller (2003) finds a positive association between the degree of worry regarding HIV-infection and fertility among young women, while the association is negative among older women. And there is evidence from qualitative studies that some women wish to have children quickly, before they become HIV positive, sometimes seeing this as unavoidable (Grieser et al., 2001). Women and men might also respond to the HIV epidemic by marrying and establishing supposedly monogamous relationships earlier, or men might try to marry younger women who are less likely to be HIV infected. Ueyama and Yamahuchi (2008) find that Malawian women marry earlier when district adult mortality is higher, which could affect fertility as the woman's child-bearing period is prolonged. These factors point to a difference in response between younger women, who have no or few children and a smaller probability of already being HIV infected, and older women, who probably have more children and a larger probability of being infected.

Our main source of data is the Malawi Demographic and Health Survey (MDHS) carried out in 2000 and 2004, which includes the entire birth history of nationally

representative samples of women. Using the retrospective birth information, we construct a panel of yearly observations from 1980 to the survey-year for each woman. The birth history is then modelled as a discrete time process with a binary birth no-birth outcome, allowing for dependence on recent communal HIV prevalence rates, the woman's earlier birth history and other individual and communal characteristics.

Communal HIV prevalence rates have varied greatly over both time and space in Malawi. To measure this variation we use district HIV rates obtained from women visiting antenatal clinics (ANCs). We have information from 19 of Malawi's 27 districts, which makes our measure of a communal effect different from other recent studies on HIV and fertility in Sub-Saharan Africa that use countries (Young, 2007, Kalemli-Ozcan, 2009a) or regions (Juhn et al., 2008; Kalemli-Ozcan, 2009a). There are three regions in Malawi, and HIV rates differ substantially within them.

To control for endogeneity of the spread of the HIV epidemic we include district dummy variables in our model. Year dummies are also included to capture any fertility changes over time that might otherwise have been attributed to communal HIV/AIDS. Furthermore we allow for unobserved individual effects. MDHS 2004 includes HIV status for a sub-sample of women. We use this information to verify that results are due to a behavioural response to the HIV epidemic, rather than being a biological difference in fertility between HIV-positive and HIV-negative women.

Our main findings are that, as a response to the HIV epidemic, young women tend to give birth to their first child sooner; an increase in district HIV from 0% to 15%, would raise the probability of a first birth for women aged 20-24 by five percentage points, but the probability of another birth would fall by almost 5 percentage points for those who have at least three children. For women over 29 years, the probability of giving birth to a first, second, or third child, would decrease by 7-15 percentage points, and the probability of women 35-39 giving birth to her fourth or fifth child would decrease by about 5 percentage points. The overall impact is small, and the consequent change in distribution of fertility across age groups is likely to be more important than any change in the total number of births per woman.

The next section discusses how HIV/AIDS might affect fertility in general, and reviews findings from previous studies. Section 3 gives a brief background to the HIV/AIDS epidemic and the evolution of fertility in Malawi. The empirical model and the data are described in Section 4, results are reported in Section 5, and their robustness is checked in Section 6. Section 7 discusses, summarizes and draws conclusions.

2. HIV/AIDS and Fertility: Theory and Evidence

HIV/AIDS seems to affect fertility in numerous ways with no single theory explaining them all. Broadly speaking, it could affect fertility directly in two ways: biologically, because of the physiological consequences of the disease on fecundity, and behaviourally, because of changes in fertility preferences of both HIV-positive and HIV-negative women, as well as of changes in sexual behaviour to avoid infection.

Biologically HIV seems to reduce fertility among infected women through a variety of mechanisms. The most important are believed to be: higher rates of miscarriage and stillbirth; co-infection with other sexually transmitted diseases; menstrual dysfunctions; weight loss leading to amenorrhoea; premature death of regular partner; and reduced frequency of intercourse because of illness (Zaba and Gregson, 1998; Fabiani, et al., 2006). There is also an indirect impact on net fertility through premature deaths of women in their reproductive years (Lewis et al., 2004). The difference in fertility between HIV-positive and HIV-negative women has been shown by a number of studies (Gray et al, 1998; Zaba and Gregson, 1998; Terceira, et al., 2003; Fabiani et al., 2006). Except perhaps for girls aged 15-19, HIV-positive women have a lower fertility than HIV-negative women in all age groups, and the difference is considerable, 25%-40% (Lewis et al., 2004).[1]

There is much less consensus on the role of behavioural mechanisms and how they impact fertility. Naturally enough, most research has focused on behavioural changes

[1] The reason young HIV-positive women are more fertile is probably because they were more sexually active than uninfected women in the first place, and thus more likely to become both HIV-positive and pregnant. Since they became infected recently, they are still quite healthy (Ntozi, 2002; Fabiani et al., 2006).

among HIV-positive women. But according to Fabiani et al. (2006), these are negligible, not least because few women know their HIV status (Gray et al., 1998; Zaba and Gregson 1998; Terceira et al., 2003). This seems to be the case in Malawi, where most people are not tested until they have already developed AIDS (Morah, 2007). Moreover, a common finding in studies of women diagnosed with AIDS is that contraceptive use and fertility do not change much (Gray et al., 1998; Rutenberg et al., 2000; Oladapo et al., 2005), even though Yeatman (2007) finds that women who learn they are HIV positive desire fewer children than others.

Some recent research in economics has been concerned with changes in reproductive behaviour of women in general, not only among HIV-positive women. Young (2005; 2007) argues that women respond to increased risk of HIV infection by having safe sex, and by reducing childbearing, to avoid getting infected and giving birth to HIV-infected babies. Grieser et al. (2001) find similar arguments in a qualitative study on Zimbabweans. Young (2005) also emphasizes, theoretically, that fertility should decrease with the increasing opportunity costs of children, which result from increased job opportunities for women because of high mortality rates among prime-age adults.

Yet another behavioural mechanism that could lead to lower fertility relates to the increased number of orphans. In Malawi, nearly 13% of children aged 0-17 are orphaned, often due to AIDS (NSO and UNICEF, 2008). The vast majority of these are taken care of by relatives, very few are in orphanages. Families that take care of orphans might decide to have fewer children of their own, both because of the costs and because children in their care might be substitutes for having their own children. In a qualitative study in Zambia, the extra burden of caring for AIDS-orphans was the only reason given by symptomless women for curtailing future childbearing (Rutenberg et al., 2000). However, Zimbabweans told Grieser et al. (2000) that they could not depend on adopted children for old-age support and that taking care of orphans thus would not affect their own childbearing.

There are also reasons why HIV/AIDS might increase fertility among women in general. Some young are reported to have expressed a desire for children soon, and grandparents and others are reported to have urged young women to have children

while they are healthy, before they become HIV positive. And in countries with high HIV rates, such as Tanzania, Kenya, and Uganda, total fertility rates (TFRs) seem to have increased or to have stopped decreasing recently (Measure DHS, 2008). Estimates of the TFR in Malawi increased from 6.0 in 2004 (NSO and OCR Macro, 2005) to 6.3 in 2006 (NSO and UNICEF, 2008). Westoff and Cross (2006) attribute the increased TFR in Kenya to HIV/AIDS, but do not test the hypothesis.

It has long been accepted that child mortality increases fertility, both through replacement and by creating a precautionary demand for children. And economic theory suggests that adult mortality would be positively related to fertility as well (Soares, 2005; Lorentzen et al., 2008). Lower life expectancy in general leads to more children through the quantity-quality trade-off as returns to education decrease (Becker and Lewis, 1973). Soares (2005) also suggests that there is substitutability between the number of children and the lifetime of each child directly in the parent's utility function, and that parents care about the survival of their lineage which makes it important that enough children live through their reproductive period. Using macro data for 1960-2000, Lorentzen et al. (2008) find that adult mortality, which increases with HIV/AIDS, is positively associated with fertility. Soares (2006) also find that adult mortality increases fertility in a study on Brazilian households.

In his seminal paper, Young (2005) tests the behavioural fertility effect of HIV/AIDS directly on data from South Africa. He finds that the epidemic reduces fertility substantially. In a later paper, Young (2007), he obtains similar results using a larger sample of Sub-Saharan countries. However, recent studies do not confirm Young's results. Kalemli-Ozcan (2009a) tests the association between HIV/AIDS and fertility on both macro and micro data. Her estimates based on between country variation show that HIV/AIDS increases fertility, while her within country estimates show no behavioural effect on fertility. This latter result is confirmed by some other studies; Werker et al. (2006), who use macro data with circumcision as an instrument to identify the causal impact of HIV/AIDS on fertility;[2] Magadi and Agwanda (2007), who study the effect of communal HIV rates on fertility in Kenya; Juhn et al. (2008),

[2] Randomised experiments have shown that circumcision reduces the risk of becoming infected with HIV.

who use DHS data for thirteen Sub-Saharan countries;[3] and Fink and Linnemayr (2008) who use the latest DHS data for five African countries and data from the World Fertility Surveys from before the HIV epidemic;[4] and Fortson (2008) who use a panel of regional HIV and mortality rates and total fertility rates.

An issue not addressed in the studies described is age-specific fertility responses to the HIV epidemic, and the analyses could mask response heterogeneity across age groups. As noted earlier, there are a few studies that point in this direction: In a qualitative study, Grieser et al. (2001) find evidence that some young women want to have children quickly before getting HIV-infected; Ueyama and Yamahuchi (2008), using MDHS 2004 data, find that Malawian women marry earlier if district adult mortality is higher; and Noël-Miller (2003), using Malawian data from another survey, finds that the association between the degree of anxiety about becoming HIV-infected and the number of births is positive among young women but negative among older women.

3. HIV/AIDS and Fertility in Malawi

Malawi's first AIDS case was diagnosed in 1985, at a time when the national HIV prevalence rate was very low. From then and on, the HIV epidemic spread rapidly, first in the major cities, and then in rural areas. By the mid-1990s HIV prevalence reached 14%, where it stabilized. In urban areas it peaked at close to 25% in 1995, and then started to decline slowly. Current overall HIV prevalence rates are 15.6 in urban areas and 11.2% in rural areas (GoM, 2007). The actual variation is much greater, however; from 5% in some of Malawi's 27 districts to over 20% in others (NSO and OCR Macro, 2005).

HIV has thus been around in Malawi for over 25 years, raising prime-age adult mortality about four times; three out of four deaths among those aged 15-49 are due to AIDS (Doctor and Weinreb, 2003). As a result, knowledge about HIV/AIDS is widespread. Already in the MDHSs carried out in 1992 and 1996 about 90% of the respondents had heard about the disease, and in 2000 the number had risen to 99%.

[3] They find no effect among HIV negative women, but, in line with the literature referred to in the beginning of this section, HIV positive women have 20% lower fertility than HIV negative women.
[4] The countries are Cameron, Cote d'Ivoire, Ghana, Kenya, and Senegal.

Hence, if HIV/AIDS affects decision-making about childbearing, this should be visible in Malawi.

In the early 1960s, the fertility rates in Malawi were similar to those in other African and other developing countries. But while fertility in most other countries fell, Malawi's grew until 1980, probably because of the ideology and policy of the Malawian government under President Banda: birth control was seen as incompatible with Malawian culture (Chimbwete et al., 2005). Finally, in the beginning of the 1980s fertility started to fall as it had in many other developing countries. Nonetheless, it is still higher than in most African countries. In the 2000s, the TFR has been 6.0 to 6.3, implying that women in Malawi on average give birth to one child more than the average African woman (OCR and Macro, 2005; NSO and Unicef, 2007).

There are substantial differences in how age-specific fertility has evolved over time (see Figure 1). Fertility among women over 30 fell greatly during the 1990s, accounting for most of the downward trend during this period, while it only fell slightly among 20-24 and 25-29 year-olds and actually grew slightly among 15-19 year-olds. From 2000 to 2006, a period when HIV/AIDS knowledge was widespread and practically everybody had personal experiences of the epidemic, fertility appears to have remained mostly stable for older women, while still showing some increases among younger women.

<<FIGURE 1 ABOUT HERE>>

The observation that fertility fell among older women during the 90s, while it rose among younger women, is consistent with a heterogeneous response to the HIV epidemic across age groups, with younger women seeking to give birth earlier, and with older women, who already have children, reducing additional births.

4. Empirical Framework and Data

This section first outlines and explains the empirical model, then describes the data and variables in some detail.

4.1 Empirical model

When analysing the impact of the HIV epidemic on fertility we are ultimately interested in the effect on a woman's complete birth history, the total number of children she gives birth to and the timing of those births. However there are still no women whose whole reproductive life cycle has taken place during the HIV epidemic, which makes it impossible to study complete birth histories directly. Following Barmby and Cigno (1990), a suitable approach is thus to treat childbearing as a sequential choice where decisions are taken under uncertainty. The birth history is then modelled as a discrete time process with a binary birth no-birth outcome in each period. This approach allows fertility choice to depend on the most recent information on the HIV epidemic during each period, as well as on earlier birth history and other individual and communal characteristics. Even though we do not study complete birth histories, we can study the fertility of women at different stages in their reproductive life cycles (at different ages and with different number of earlier births), while allowing their response to the HIV epidemic to differ.

To illustrate the approach we outline a simple version of the model. Let $Y_{idt} = 1$ if woman *i* in district *d* gives birth in year *t*, and $Y_{idt} = 0$ if she does not. A woman decides whether to have a child or not during a given year by taking into account a number of individual and communal factors, particularly HIV rates, though of course, she cannot control the exact timing and number of births for both biological and behavioural reasons. Formally,

$$\begin{aligned} &Y_{idt} = 1 \quad if \quad \mathbf{X}'_{idt}\beta + \mathbf{Z}'_{idt}HIV'_{dt-1}\gamma + \varepsilon_{idt} > 0 \\ &Y_{idt} = 0 \quad otherwise \end{aligned} \tag{1}$$

where β and γ are coefficients, $\mathbf{X}_{idt}$ indicates individual characteristics of the woman; HIV_{dt-1} is last year's district HIV prevalence rate; and Z_{idt}, which is a sub-set of $\mathbf{X}_{idt}$, indicate the woman's age and her number of previous births. We allow for a differential impact of the district HIV rate depending on the factors in Z_{idt}. The

impossibility to exactly control fertility gives rise to an error term, ε_{idt}. In practice, the error term will also capture factors unobserved by the researcher.

Assuming ε_{idt} to be logistically distributed, we can use the logit estimator. The probability that woman *i*, in district *d*, has a child in year *t* is then

$$\begin{aligned}&\Pr\left(Y_{idt}=1\,|\,\mathrm{X}_{idt},HIV_{dt-1}\right)\\&=\exp\left(\mathrm{X}'_{idt}\beta+Z'_{idt}HIV'_{dt-1}\gamma\right)/1+\exp\left(\mathrm{X}'_{idt}\beta+Z'_{idt}HIV'_{dt-1}\gamma\right)\\&=\Delta\left(\mathrm{X}'_{idt}\beta+Z'_{idt}HIV'_{dt-1}\gamma\right),\end{aligned}\qquad(1)$$

where $\Delta(.)$ denotes the logistic cumulative distribution function.

This model has so far not taken into account the fact that the spread of the HIV epidemic across time and space is not exogenous. Norms of sexual and reproductive behaviour and other factors affecting them will have an impact on both fertility and the spread of HIV. We thus add district dummies, α_d, to control for district-level unobserved heterogeneity. Moreover, the number of people infected with HIV, and dying of AIDS, has increased over time. To capture unobserved time-varying effects we therefore add year-dummies, δ_t. As we have many observations in every year and district, this dummy-variable approach should yield consistent estimates, even though we use a non-linear model. Equation (2) then becomes

$$\Pr\left(Y_{idt}=1\,|\,\mathrm{X}_{idt},HIV_{dt-1},\delta_t,\alpha_d\right)=\Delta\left(\mathrm{X}'_{idt}\beta+Z'_{idt}HIV'_{dt-1}\gamma+\delta_t+\alpha_d\right).\qquad(2)$$

Estimations are carried out maximizing the likelihood function

$$\begin{aligned}L=\prod_{idt}&\left[\Delta\left(\mathrm{X}'_{idt}\beta+Z'_{idt}HIV'_{dt-1}\gamma+\delta_t+\alpha_d\right)\right]^{Y_{idt}}\\&\left[1-\Delta\left(\mathrm{X}'_{idt}\beta+Z'_{idt}HIV'_{dt-1}\gamma+\delta_t+\alpha_d\right)\right]^{1-Y_{idt}}\end{aligned}\qquad(3)$$

To draw inference we use standard errors clustered at the level of the DHS sampling clusters.[5]

In the robustness analysis we also allow for unobserved heterogeneity at the level of the individual, i.e., that women with the same observable characteristics might behave differently. To do so we divide the error term (ε_{idt}) into a time-constant individual unobserved effect, u_{id}, and time-varying component, v_{idt}. Given the large number of women and the small number of observations per woman, the simple dummy-variable approach would not be appropriate to model this unobserved individual heterogeneity. Instead a random effects estimator is used. The contribution of woman *i* to the likelihood function, L_i, is the joint probability of all her T_i observations, an integral of dimension T_i. With this binary choice random effects model, unobserved group effects, here u_{id}, are integrated out of the likelihood function,[6] which gives a conditional likelihood (on unobserved group effects) of the form

$$L_i = \int_{-\infty}^{\infty} \left[\prod_t \Pr\left(Y_{idt}=1 \mid \mathrm{X}_{idt}, HIV_{dt-1}, \delta_t, \alpha_d, u_{id}\right)^{Y_{idt}} \left(1-\Pr\left(Y_{idt}=0 \mid \mathrm{X}_{idt}, HIV_{dt-1}, \delta_t, \alpha_d, u_{id}\right)\right)^{1-Y_{idt}} \right] f\left(u_{id}\right) du_{id} \qquad (4)$$

where the term inside the brackets is the logit model. The solution now requires only one-dimensional integration. Most commonly the unobserved group effects are assumed to be normally distributed, and integration is done using numerical methods. This approach is also used here.[7]

4.2 Data and variables

The data used is from MDHS 2000 and 2004. In MDHS 2000, 13220 women were interviewed, and in MDHS 2004, 11698 women. The Demographic and Health

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[5] Clustering the standard errors at the district level does not significantly change our results, probably because district dummies capture most of the district correlation. Standard errors clustered by district are available from the authors.

[6] Conditional on unobserved group effects, the error terms ε_{idt} are independent, and their joint probability consequently equal to the product of the probability of each term.

[7] Integration is carried ou using adaptive Gaussian quadrature and Newton Raphton using the gllamm procedure in Stata. Standard errors are clustered at the level of the sampling cluster (which is the reason we use the gllamm procedure rather than the xtprobit procedure).

Surveys (DHS) project collects information about the entire birth history of interviewed women. Using this retrospective information we create a panel data set consisting of one observation for each woman and year. A woman enters the sample at age 15, or in 1980 if she was older than that then. She leaves the sample when she turns 40, or earlier if she is still not 40 in the survey year. The choice of 1980 as the start year is a compromise between the desire to include observations from before the onset of the HIV epidemic and the desire to include women from different age groups over time. This is also the reason to exclude women that are over 39 from the sample. Still, both the year distribution and the age distribution of the data are skewed with more observations for younger women and for later years (Figures 2 and 3). There are especially few observations for older women in the 1980s.[8] However, the skewed distribution should not be a problem since we use time and age dummies.

<<FIGURE 2 ABOUT HERE>>

<<FIGURE 3 ABOUT HERE>>

In total we have 296,067 woman-year observations for 24,915 women. However, we only use observations when we know where the woman lived during a particular year, where we have at least two observations for the woman, and from districts with HIV prevalence data, which reduces the number of woman-year observations to 148,166 for 14,241 women.[9]

As mentioned earlier, the dependent variable is binary, equalling 1 if the woman gave birth during a particular year, 0 otherwise.[10] A birth was recorded in about a fifth of the cases, 32,211 observations. Table 1 shows the distribution of number of prior births for different age groups, and the proportion (in brackets) that gives birth in each category. There are very few observations for 15 to 19 year-olds with three or more prior births, and for 20 to 24 year-olds with five or more births. And, as shown in the

[8] Women were sampled to be representative of 15-49 year old women in the survey years, resulting in a younger age-distribution as we go back in time.
[9] The sample was further reduced because of missing information regarding ethnicity for 6 women and regarding relative household wealth for another 6 women.
[10] The few cases where a woman gave birth to more than one child in a year are thereby not treated differently than cases where a woman gave birth to one child.

brackets, among the few women over 29 who have not given birth earlier, very few do it later. Still, it is clear that women in Malawi give birth to many children, and start childbearing early. The proportion of no-prior-births decreases rapidly with age, from 82% for those aged 15-19 to 27% for 20-24, and then 7%, 3% and 2% for 25-29, 30-34 and 35-39. By summing the proportions, we can also see that over 70% of women aged 20-24 have given birth at least once before, over 60% of women aged 25-29 and over 80% of women aged 30-34 have given birth at least three times, and almost 70% of women aged 35-39 have given birth to as many as five children or more.

<<TABLE 1 ABOUT HERE>>

We are interested in the effects of the HIV epidemic on the fertility of all women, not just the HIV-positive. For this purpose we need a variable measuring the geographic and time variation in risk of HIV infection. We use district-level HIV prevalence rates collected from pregnant women visiting antenatal clinics (ANCs).[11] The raw data consists of observations for selected years from 1985 to 2003 for a maximum of 18 clinics across Malawi. This is the only data on HIV prevalence rates that has been collected reasonably systematically over a longer time period.[12]

Worries are often expressed over how well such ANC-data represents HIV prevalence rates in the general population. However, our statistical identification of the HIV impact comes from relative levels of HIV prevalence rates over time and space. Thus, the HIV rates measured at the ANC do not have to be correct, as long as any bias is similar across clinics and over time. The use of HIV prevalence rates in empirical studies of human behaviour has also been questioned since they are not directly observable by people. But they might be indicative of observable AIDS illness and deaths. Young (2007) argues that women are able to infer the HIV rate from infants' deaths with AIDS symptoms, since the disease progresses rapidly in small children.[13]

[11] This data is provided by the U.S. Census Bureau.
[12] In a related study (Durevall and Lindskog, 2008) we instead use district adult mortality in 1998 and HIV prevalence in the general population in 2004. Though this data might be of better quality it only allows a cross-sectional analysis.
[13] According to this argument, mothers should also have a good idea of their own risk of being HIV-positive.

To obtain HIV prevalence rates for the 19 districts included in the analysis, and for all years from 1980 to 2004, we used the Estimation and Projections Package (EPP) of WHO/UNAIDS to estimate HIV trends. It fits a (nonlinear) epidemiological model to the data starting in 1980.[14] We assume a period of about one year between the decision to have a child and birth, so we use the one-year lag of district HIV rates in the regressions.

When modelling fertility as a sequential choice, it is crucial to control for the woman's age and number of earlier births. Age enters as five dummy variables for age groups, as seen in Table 1: 15-19 (the baseline in estimations); 20-24; 25-29; 30-34; and 35-39. Dummies indicating how many previous births the woman has had are also included; no prior births (the baseline in estimations); one or two prior births; three or four prior births; and five or more prior births. Since one purpose of this study is to allow the fertility response to the HIV epidemic to be conditional on age and number of previous births, we construct various interaction terms with HIV rates and the age and birth dummies.

According to economic theory, family income as well as the opportunity cost of having a child should influence fertility (Becker, 1960; Becker and Lewis, 1973). As a proxy for family income, dummies for the wealth quintile of the household are used (the middle quintile dummy is the baseline in estimations).[15] To capture the opportunity cost, the woman's education is included, indicated by dummies for no or incomplete primary school (the baseline in estimations); completed only primary school; and completed secondary school.[16] A problem with the education and wealth variables is that we have information from the survey year only. We thus have to assume that there have not been any systematic changes in the relative wealth of the households over time, and we assume that primary school was completed by age 15 and secondary school by age 20. These assumptions are checked by estimating the models without the education and wealth variables.

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[14] Young (2007) and Kalemli-Ozcan (2009a) also used the Estimation and Projections Package (EPP) to create their time series for HIV rates.

[15] The household wealth variable has been constructed using information on household assets. See Rutstein and Johnson (2004) for further information about the DHS wealth index.

[16] Education and household wealth might also indicate that they belong to different social groups with different norms.

Norms and social constraints are also important determinants of fertility. If ethnicity, through its influence on sexual and reproductive behaviour, also matters for the spread of HIV/AIDS, it is crucial to control for it. Nine dummies indicating ethnic group are thus included to capture variation in norms. Urban or rural residence is also controlled for with an urban-residence dummy, which is probably associated with both differences in opportunity costs of having children and with differences in norms.

Following Soares (2006), we also allow for intergenerational persistence in reproductive behaviour by including the woman's number of siblings, and the effects of child mortality in the woman's original family, as measured by the share of her siblings that died before age 10. Table 2 shows summery information of the main variables.

<<TABLE 2 ABOUT HERE>>

5. Empirical Results

We estimate four specification of our fertility model, with the degree of heterogeneity allowed for in the fertility response to communal HIV differing across them (Table 3). In the first specification, the fertility response to communal HIV is constrained to be equal for all women. In the second specification it is allowed to differ across age groups and in the third across prior number of births. In the fourth and most flexible specification, the response is allowed to differ for each combination of age and prior number of births.

<<TABLE 3 ABOUT HERE>>

In all four specifications, women older than 19 have a higher probability of giving birth than women aged 15 to 19, with child-bearing peaking in the early twenties. Women that have already had at least one child have a higher probability of giving birth than those with no prior children. This is in line with the findings of, for example, Barmby and Cigno (1990) and Angeles et al. (2005). Given at least one prior

birth, the probability of giving birth again seems to be decreasing, though there are small differences between 'three or four' and 'five or more' births.

Women in richer households give birth to fewer children than women in poor and middle-wealth households, which could suggest that the substitution effect is larger than the income effect. Women with more education give birth to fewer children, especially those few who have completed secondary school. This is in line with the hypothesis of lower fertility as the opportunity cost of having children rises, but could also be because better informed women, or women with a better bargaining position in the household, decide to have fewer children. As expected, women in cities have fewer children, which could be because of the higher opportunity costs of having children in cities, because of a larger need for (child) labour among farmers, or perhaps because of differences in norms. As in the analysis on Brazil by Soares (2006), Malawian women who had more siblings give birth to more children, as do those whose siblings died as children.[17]

So much for the control variables. In the first specification, the estimated coefficient of HIV prevalence is, as in Young (2007), negative but, in contrast to Young, not statistically significant.[18] The lack of significance could be because of heterogeneity in the fertility response to the HIV epidemic. Results from the remaining specifications support this. In the second specification there is a statistically significant negative effect of HIV rates on fertility for women over 24, and particularly for women over 29. In the third specification the probability of a first birth is positively related to HIV rates, while the probability of subsequent births is negatively related to HIV. In the fourth and most flexible specification, higher HIV rates increase the probability of a first birth for women aged 20-24 - the age at which fertility is highest for Malawian women - while there is a smaller and imprecisely measured change for women 15-19, and a decrease in the probability for women over 29. For women who have already had at least one child, the probability of another

[17] The coefficient on number of siblings, that is the number of children the mother of the woman has given birth to, is almost statistically significant at the 10% level in all specifications.
[18] There are several potential explanations for differences in results even though Young (2007) also use individual level data from the DHS project. For example, our measure of the communal effect is based on much smaller geographical areas, districts, whereas Young (2007) uses countries. Moreover, we use annual time dummies, while Young (2007) includes a linear time trend in some specifications.

birth falls with higher HIV rates, with the exception for women aged 15-19 who have a higher probability of another birth.

To formally compare the four specifications we re-estimated them without clustered standard errors and performed log-likelihood ratio tests. The second or third specification is strongly favoured (at the one percent level of significance) over the first one, and the fourth specification is strongly favoured over the second or third ones.[19]

These results support the hypothesis that young women start child-bearing earlier when HIV rates are high, while older women are less likely to give birth even if they have no children. The results come out even though we control for the district in which the woman lives and her ethnicity, which means that results probably are not due to any initial differences in sexual and reproductive behaviour across districts or ethnic groups.

To illustrate the magnitude of the effects of HIV on fertility, predicted probabilities of women giving birth with a district HIV rate at 0% and 15% are calculated (Table 4). Among those aged 15 to 19 HIV rates do not make much difference. But for women aged 20-24 the probability of a first birth is five percentage points higher when HIV rates are 15%, whereas the probability of another birth is almost 5 percentage points lower for women that have at least three children already. The other large effects are among women over 29, whose probability of giving birth to a first, second, or third child, are 7-15 percentage points lower with higher HIV rates. The probability of women 35-39 giving birth to her fourth or fifth child is about 5 percentage points lower. It thus seems that the HIV epidemic changes the distribution of fertility across age groups, leading to more births for younger women and fewer for older women.

<<TABLE 4 ABOUT HERE>>

[19] The LR test statistics are 38.99 and 55.71 when the first specification is tested against the second and third ones, and 501.79 and 485.02 when the second and third specifications are tested against the fourth one.

Simple simulations of total fertility rates for women between age 15 to 39, based on the predicted probabilities in Table 4, indicate virtually no difference between districts with zero HIV (5.40 children), and district with 15% HIV (5.31 children).[20] The changed distribution of fertility across age groups because of the HIV epidemic, i.e., changes in the timing of births, is thus likely to be more important than any impact on the total number of births.

6. Robustness Analysis

How can we know that the effects of HIV on fertility are due to changes in reproductive behaviour and not just to biological differences in fertility between HIV-positive and HIV-negative women? For a sub-sample of the respondents in MDHS 2004 there is information about HIV status. We re-estimate the fourth specification in Table 3, while controlling for HIV status (Table 5, column 1), and using only the sub-sample of HIV-negative women (Table 5, column 2). Any remaining effects of HIV rates in these specifications must indicate that women, regardless of their HIV status, change their reproductive behaviour. Of course we do not know when the HIV-positive women got infected. The HIV-status variable therefore captures the difference in realised fertility between women who were HIV negative in 2004 and those who were HIV positive in 2004. Similarly, the sample of HIV-negative women excludes women who were HIV positive in 2004 but might have been HIV negative in earlier years.

<<TABLE 5 ABOUT HERE>>

Consistent with the findings in various other studies, women who were HIV-positive in year 2004 are less likely to give birth in any given year than HIV-negative women (specification 1). The odds ratio of giving birth for women who were HIV positive in 2004 compared to HIV-negative women is 0.81. So, as in Juhn et al. (2008), HIV-infected women on average had about 20% lower probability to give birth.[21] For the district HIV prevalence interaction terms the general pattern is the same as in Table 3.

[20] Usually the TFR is calculated for women 15- 49, which of course gives a larger number.
[21] We expect a lower value than Juhn et al. (2008) if physiological consequences of the disease are to blame for the difference in fertility, since our regression includes a longer period before several of the women became HIV positive.

Young women with no prior births are more likely to give birth where HIV prevalence is higher, whereas the oldest women are less likely to give birth to their first child. Women who already have children generally have a lower probability of giving birth where the district HIV prevalence is higher. The differences in estimated coefficients in these two specifications, compared to specification (4) in Table 3, appear to be due to restricting the sample to women with HIV-status information rather than to controlling for HIV status (column 1) or excluding HIV-positive women (column 2). Estimations of specification (4) in Table 3 only on the sub-sample of women with HIV-status information, i.e., without controlling for HIV status, give results almost identical to those in specification (1) in Table 5 (available from the authors upon request).

Table 5 also includes a specification where the relative household-wealth variables have been excluded (specification 3), because, strictly speaking, we only know the relative wealth of the woman's household in the survey year. The specification (4) also excludes education variables as education could be endogenous if girls make joint decisions on fertility and education early in life. Excluding the wealth and schooling variables has no substantial effect on estimated coefficients of other variables.

It could also be that it is not the number of children the woman has already given birth to that matters for her will to give birth, but the number of surviving children. Specification (5) therefore uses information about the number of surviving children, rather than the number the woman has earlier given birth to (both for dummies and for HIV prevalence interaction terms). Again, this change has no substantial effect on the estimated coefficients.

Finally, we allowed for unobserved individual heterogeneity: results from estimations with the random effects logit estimator are in specification (6) in Table 5. As seen, the inclusion of woman unobserved effects is unimportant. The variance of unobserved individual effects is not significantly different from zero, and again the change has no substantial impact on the estimated coefficients.

7. Discussion and Conclusions

We evaluated the impact of HIV/AIDS on reproductive behaviour in the general female population in Malawi, i.e. HIV-negative as well as HIV-positive women. In contrast to earlier studies on changes in reproductive behaviour due to HIV/AIDS, we allowed for a differential response to HIV/AIDS depending on the woman's age and number of prior births.

Using retrospective birth information in the MDHS 2000 and 2004 surveys we constructed a panel of yearly observations from 1980 to the survey year for each woman. The birth history was then modelled as a discrete time process, allowing for dependence on recent district HIV prevalence rates, the woman's earlier birth history and other individual and communal characteristics. There is a possibility that sexual behaviour affects both the spread of the HIV epidemic and child-bearing, so we controlled for unobserved heterogeneity across districts and ethnic groups. Hence we conclude that the results are not due to differences in sexual and reproductive norms or behaviour across districts and ethnic groups. Nor are they due to changes in reproductive behaviour over time, as we included year dummies. Furthermore, we did a random-effects estimation to ensure that the results are not confounded by unobserved individual heterogeneity. To make sure that our results are due to behaviour changes in the general female population, and not due to biological differences in fertility between HIV-positive and HIV-negative women, we controlled for the HIV status in 2004 of a subsample of women, and we re-estimated our model only on women who were HIV negative in 2004.

The probability that a young woman would give birth to her first child increases with the district HIV prevalence rate, whereas the probability that older women would give birth decreases. An increase in district HIV from 0% to 15%, would raise the probability of a first birth for women aged 20-24 by five percentage points, whereas the probability of another birth would fall by almost 5 percentage points for those who have at least three children. For women over 29 years, the probability of giving birth to a first, second, or third child, would decrease by 7-15 percentage points, and the probability of women 35-39 giving birth to her fourth or fifth child would decrease by about 5 percentage points. This suggest that young women may seek to give birth

earlier, when the risk of being HIV infected is lower, or that women give birth earlier as a consequence of marrying and establishing supposedly monogamous relationships earlier, as both women and men attempt to reduce the risk of becoming infected.

The HIV epidemic is thus likely to change the timing of births, while effects on a woman's total number of children may be very small. All else equal, this change in distribution of fertility across age groups should have a positive impact on net fertility,[22] which is what matters for population growth and the evolution of the dependency ratio. However, as there is a negative impact on net fertility from increased mortality due to HIV/AIDS, the total effect on net fertility is uncertain. But, perhaps more worrying is that women giving birth earlier could negatively impact female education. The effects on child welfare are hard to predict. As younger women have a smaller risk of being HIV infected, a shift towards more births in younger ages should imply less mother-to-child transmission of HIV than what would otherwise have been the case. However, traditionally infant and child mortality traditionally tended to be higher when the mother is younger.

Our results for the overall impact of HIV/AIDS on fertility thus differ from Kalemli-Ozcan (2009a) and Young (2007). We do not find that HIV/AIDS increases fertility, as Kalemli-Ozcan (2009a) does using macro data. And our results do not support Young's (2007) finding of a strong negative effect on fertility in Sub-Saharan countries. Our results are, however, consistent with Juhn, et al. (2008). They report that HIV/AIDS has no or a small effect on aggregate fertility, but that it reduces fertility among HIV-positive women by 20% for direct physiological reasons. We estimate that women who were HIV positive in 2004 had 20% lower probability of giving birth. However, in contrast to Juhn et al. (2008), as well as to Fortson (2008), Fink and Linnemayr (2008) and Kalemli-Ozcan (2009b), we find a behavioural response in the general female population by conditioning the fertility response on the woman's age and number of prior births.

[22] Net fertility is the number of births of a woman if she was subject to current age specific fertility and -mortality rates throughout her life.

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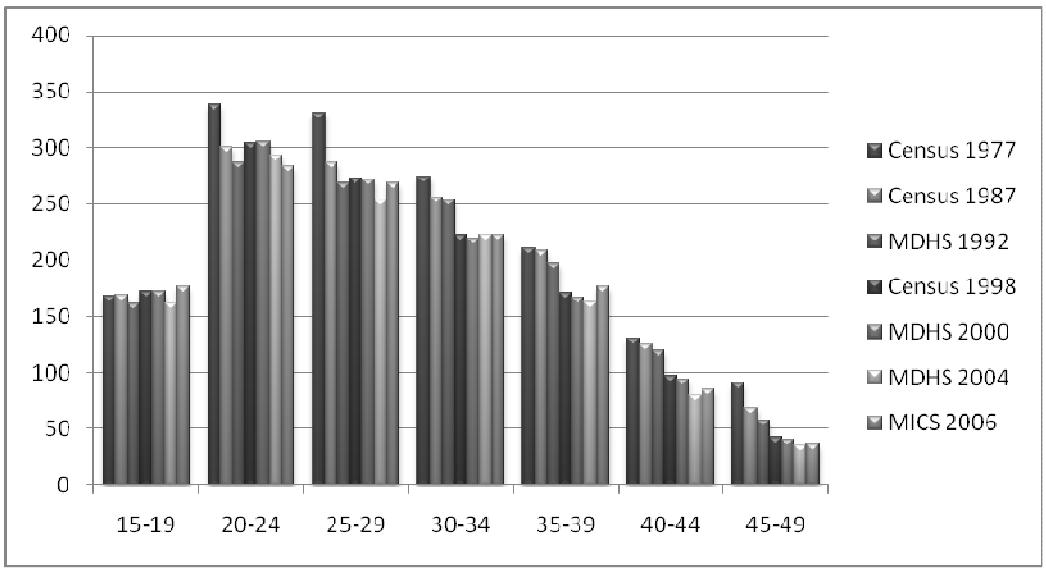
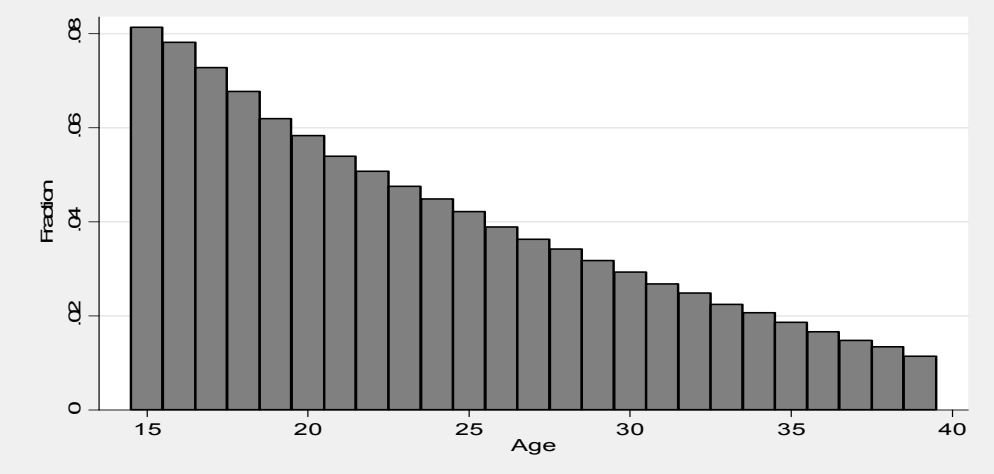
Figures and Tables

Figure 1: Age-specific fertility rates (births per 1000 women) for various years



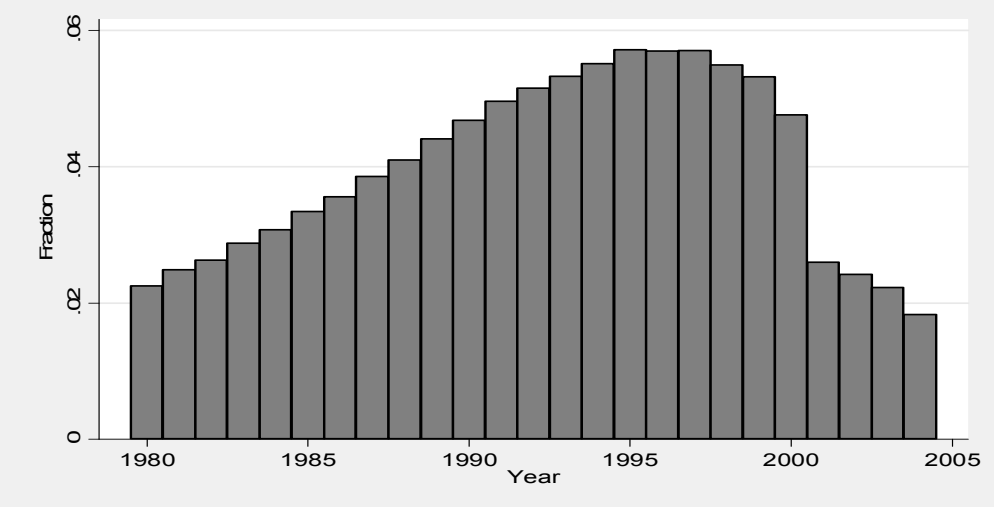
Sources: Population censuses 1977, 1987 and 1998 (NSO, 1993; 2002), MDHS 1992, 2000 and 2004 (NSO and ORC Macro, 2001; 2005) and MICS 2006 (NSO and UNICEF, 2008).

Figure 2: Age distribution of the estimation sample



Source: Own calculations with Malawi Demographic and Health Survey Data 2000 and 2004.

Figure 3: The distribution per year of the estimation sample



Source: see Figure 2.

Table 1: Distribution of prior births for different age groups (proportion of positive birth-outcomes in brackets)

	Age 15-19	Age 20-24	Age 25-29	Age 30-34	Age 35-39
No prior births	0.82	0.27	0.07	0.03	0.02
	[0.12]	[0.26]	[0.19]	[0.11]	[0.04]
One or two prior births	0.17	0.56	0.31	0.15	0.09
	[0.23]	[0.31]	[0.30]	[0.19]	[0.09]
Three or four prior births	0.01	0.17	0.46	0.33	0.20
	[0.21]	[0.24]	[0.28]	[0.26]	[0.17]
Five or more prior births	0.00	0.01	0.16	0.49	0.68
	[0.25]	[0.22]	[0.24]	[0.25]	[0.20]
Total	1.000	1.000	1.000	1.000	1.000
	[0.14]	[0.28]	[0.28]	[0.24]	[0.18]

Table 2: Summary statistics

Variable	Obs	Mean	Std. Dev.	Min	Max
Gave birth last 12 months	148020	0.217	0.412	0.000	1.000
Age 15-19	148020	0.358	0.479	0.000	1.000
Age 20-24	148020	0.257	0.437	0.000	1.000
Age 25-29	148020	0.184	0.388	0.000	1.000
Age 30-34	148020	0.125	0.331	0.000	1.000
Age 35-40	148020	0.076	0.264	0.000	1.000
No prior births	148020	0.380	0.486	0.000	1.000
One or two prior births	148020	0.289	0.453	0.000	1.000
Three or four prior births	148020	0.186	0.389	0.000	1.000
Five or more prior births	148020	0.145	0.352	0.000	1.000
Poorest household wealth quintile	14230	0.170	0.376	0.000	1.000
Second poorest household wealth quintile	14230	0.187	0.390	0.000	1.000
Middle household wealth quintile	14230	0.200	0.400	0.000	1.000
Second richest household wealth quintile	14230	0.206	0.404	0.000	1.000
Richest household wealth quintile	14230	0.236	0.425	0.000	1.000
Child mortality among siblings	14230	0.151	0.214	0.000	1.000
Number of siblings	14230	5.760	2.733	0.000	18.000
Urban residence	14230	0.205	0.372	0.000	1.000
Completed primary school, only	14230	0.685	0.440	0.000	1.000
Completed secondary school	148020	0.054	0.226	0.000	1.000
Last year's district HIV rate	701	10.250	9.086	0.000	32.390
Chewa	14230	0.303	0.460	0.000	1.000
Tumbuka	14230	0.127	0.333	0.000	1.000
Lomwe	14230	0.144	0.351	0.000	1.000
Tonga	14230	0.026	0.158	0.000	1.000
Yao	14230	0.157	0.364	0.000	1.000
Sena	14230	0.029	0.168	0.000	1.000
Nkonde	14230	0.028	0.165	0.000	1.000
Ngoni	14230	0.123	0.328	0.000	1.000
Other ethnicity	14230	0.045	0.207	0.000	1.000
Blantyre	14230	0.097	0.296	0.000	1.000
Kasungu	14230	0.091	0.287	0.000	1.000
machinga	14230	0.089	0.284	0.000	1.000
Mangochi	14230	0.086	0.280	0.000	1.000
Mzimba	14230	0.093	0.291	0.000	1.000
Lilongwe	14230	0.092	0.289	0.000	1.000
Mulanje	14230	0.102	0.303	0.000	1.000
Karonga	14230	0.064	0.244	0.000	1.000
Nkhata bay	14230	0.018	0.132	0.000	1.000
Rumphi	14230	0.012	0.107	0.000	1.000
Nkhota kota	14230	0.022	0.147	0.000	1.000
Dowa	14230	0.050	0.219	0.000	1.000
Mchinji	14230	0.037	0.190	0.000	1.000
Dedza	14230	0.055	0.229	0.000	1.000
Ntcheu	14230	0.048	0.214	0.000	1.000
Chiradzulu	14230	0.023	0.149	0.000	1.000
Nsanje	14230	0.020	0.140	0.000	1.000
Mzuzu city	14230	0.001	0.027	0.000	1.000

Summary statistics are based on; all observations for time varying individual variables, one observation per woman for time constant individual variables, and one observation per district and year for 'Last year's district HIV rate'.

Table 3: Logit estimates of the fertility effect of the HIV epidemic (dependent variable is birth/no-birth)

	(1)	(2)	(3)	(4)
Age 20-24	0.691***	0.716***	0.692***	0.671***
	(0.023)	(0.029)	(0.023)	(0.031)
Age 25-29	0.613***	0.679***	0.616***	0.641***
	(0.028)	(0.034)	(0.028)	(0.038)
Age 30-34	0.434***	0.538***	0.441***	0.528***
	(0.034)	(0.041)	(0.034)	(0.047)
Age 35-39	0.098**	0.327***	0.109***	0.345***
	(0.042)	(0.057)	(0.042)	(0.062)
One or two prior births	0.492***	0.492***	0.615***	0.611***
	(0.019)	(0.019)	(0.026)	(0.028)
Three or four prior births	0.329***	0.330***	0.445***	0.408***
	(0.025)	(0.025)	(0.032)	(0.036)
Five or more prior births	0.360***	0.358***	0.445***	0.346***
	(0.031)	(0.031)	(0.040)	(0.046)
Poorest quintile	0.003	0.001	0.002	-0.003
	(0.020)	(0.020)	(0.020)	(0.020)
Second poorest quintile	0.011	0.011	0.011	0.013
	(0.020)	(0.020)	(0.020)	(0.020)
Second richest quintile	-0.062***	-0.062***	-0.061***	-0.061***
	(0.020)	(0.020)	(0.020)	(0.020)
Richest quintile	-0.101***	-0.102***	-0.103***	-0.101***
	(0.025)	(0.025)	(0.025)	(0.025)
Child mortality among siblings	0.087***	0.083***	0.083***	0.084***
	(0.029)	(0.030)	(0.030)	(0.029)
Number of siblings	0.003	0.004	0.004	0.004
	(0.002)	(0.002)	(0.002)	(0.002)
Urban residence	-0.071***	-0.074***	-0.074***	-0.073***
	(0.026)	(0.026)	(0.026)	(0.026)
Completed primary school	-0.064***	-0.067***	-0.066***	-0.071***
	(0.015)	(0.015)	(0.015)	(0.015)
Completed secondary school	-0.337***	-0.337***	-0.341***	-0.397***
	(0.035)	(0.035)	(0.035)	(0.036)
District HIV prevalence	-0.002			
	(0.001)			
District HIV prevalence interaction terms				
Age 15-19		0.003		
		(0.002)		
Age 20-24		0.000		
		(0.002)		
Age 25-29		-0.003*		
		(0.002)		
Age 30-34		-0.006***		
		(0.002)		
Age 35-39		-0.014***		
		(0.003)		
No prior births			0.005***	
			(0.002)	
One or two prior births			-0.006***	
			(0.002)	
Three or four prior births			-0.005***	

			(0.002)	

Table 3 (continued)

Five or more prior births			-0.003	
			(0.002)	
No prior births				0.002
*Age 15-19				(0.002)
No prior births				0.020***
*Age 20-24				(0.003)
No prior births				-0.005
*Age 25-29				(0.005)
No prior births				-0.043***
*Age 30-34				(0.011)
No prior births				-0.107***
*Age 35-39				(0.033)
One or two prior births				0.004*
*Age 15-19				(0.002)
One or two prior births				-0.004**
*Age 20-24				(0.002)
One or two prior births				-0.004
*Age 25-29				(0.002)
One or two prior births				-0.036***
*Age 30-34				(0.004)
One or two prior births				-0.071***
*Age 35-39				(0.009)
Three or four prior births				0.007
*Age 15-19				(0.009)
Three or four prior births				-0.016***
*Age 20-24				(0.003)
Three or four prior births				-0.001
*Age 25-29				(0.002)
Three or four prior births				0.000
*Age 30-34				(0.003)
Three or four prior births				-0.022***
*Age 35-39				(0.004)
Five or more prior births				-0.832***
*Age 15-19				(0.230)
Five or more prior births				-0.019
*Age 20-24				(0.013)
Five or more prior births				-0.012***
*Age 25-29				(0.003)
Five or more prior births				-0.000
*Age 30-34				(0.002)
Five or more prior births				-0.005
*Age 35-39				(0.003)
Observations	148020	148020	148020	148020
Log pseudo likelihood	-74735.89	-74716.39	-74708.03	-74465.53
Pseudo R2	0.0354	0.0356	0.0357	0.0388

Notes: All estimations include a constant, ethnicity dummies, year dummies, and district dummies. Robust standard errors, clustered at the level of the sampling cluster, are in parentheses. Significance is indicated as, *=significant at 10%; **=significant at 5%; ***=significant at 1%.

Table 4: Predicted probabilities of giving birth during any given year

	No HIV	15% HIV prevalence rate	Difference	95% confidence interval of difference	
No prior births					
Age 15-19	0.118	0.121	0.003	[-0.003,	0.010]
Age 20-24	0.195	0.246	0.051	[0.0380,	0.064]
Age 25-29	0.198	0.188	-0.011	[-0.031,	0.010]
Age 30-34	0.185	0.107	-0.079	[-0.110,	-0.047]
Age 35-39	0.158	0.037	-0.122	[-0.159,	-0.084]
One or two prior births					
Age 15-19	0.202	0.212	0.010	[-0.002,	0.022]
Age 20-24	0.323	0.310	-0.012	[-0.025,	-0.000]
Age 25-29	0.312	0.301	-0.011	[-0.026,	0.004]
Age 30-34	0.290	0.193	-0.098	[-0.119,	-0.076]
Age 35-39	0.251	0.103	-0.148	[-0.178,	-0.117]
Three or four prior births					
Age 15-19	0.176	0.192	0.016	[-0.027,	0.059]
Age 20-24	0.289	0.242	-0.047	[-0.063,	-0.032]
Age 25-29	0.279	0.275	-0.003	[-0.016,	0.009]
Age 30-34	0.253	0.253	0.000	[-0.015,	0.016]
Age 35-39	0.215	0.164	-0.051	[-0.071,	-0.031]
Five or more prior births					
Age 20-24	0.287	0.232	-0.055	[-0.122,	0.011]
Age 25-29	0.273	0.238	-0.035	[-0.053,	-0.017]
Age 30-34	0.248	0.247	-0.001	[-0.014,	0.012]
Age 35-39	0.212	0.200	-0.011	[-0.026,	0.004]

Notes: Predicted probabilities are based on parameter estimates in specification (4) in Table 3. The value of characteristics other than age, prior births, and district HIV prevalence are set to the mean for women in each age and prior births group. Predicted probabilities for 15-19 year-olds with five or more births are not reported as there are only four observations and one woman in this category.

Table 5: Logit estimates of the fertility effect of the HIV epidemic: Robustness analysis (dependent variable is birth/no-birth)

	(1)	(2)	(3)	(4)	(5)	(6)
	HIV-status control	HIV-negative only	No hh wealth controls	No hh wealth or education	Children alive (not born)[1]	Random effects estimator[2]
Wealth quintile dummies	Yes	Yes	No	No	Yes	Yes
Primary and secondary school dummies	Yes	Yes	Yes	No	Yes	Yes
HIV-positive in 2004	-0.193***					
	(0.054)					
District HIV interaction terms						
No prior births	0.010*	0.009	0.002	0.003	0.002	0.002
*Age 15-19	(0.005)	(0.006)	(0.002)	(0.002)	(0.002)	(0.002)
No prior births	0.026***	0.032***	0.020***	0.017***	0.015***	0.020***
*Age 20-24	(0.008)	(0.009)	(0.003)	(0.003)	(0.002)	(0.003)
No prior births	-0.023	-0.022	-0.005	-0.006	-0.007*	-0.005
*Age 25-29	(0.016)	(0.020)	(0.005)	(0.004)	(0.004)	(0.005)
No prior births	-0.046*	-0.061*	-0.043***	-0.043***	-0.043***	-0.043***
*Age 30-34	(0.028)	(0.033)	(0.011)	(0.011)	(0.008)	(0.011)
No prior births			-0.107***	-0.105***	-0.089***	-0.107***
*Age 35-39			(0.033)	(0.033)	(0.019)	(0.033)
One or two prior births	0.003	0.006	0.004*	0.004*	0.005*	0.004*
*Age 15-19	(0.007)	(0.007)	(0.002)	(0.002)	(0.003)	(0.002)
One or two prior births	-0.001	0.003	-0.004*	-0.004*	-0.005**	-0.004**
*Age 20-24	(0.005)	(0.006)	(0.002)	(0.002)	(0.002)	(0.002)
One or two prior births	0.006	0.008	-0.004	-0.004*	-0.000	-0.004
*Age 25-29	(0.007)	(0.007)	(0.002)	(0.002)	(0.002)	(0.002)
One or two prior births	-0.038**	-0.051***	-0.036***	-0.036***	-0.022***	-0.036***
*Age 30-34	(0.016)	(0.019)	(0.004)	(0.004)	(0.003)	(0.004)
One or two prior births	-0.093**	-0.124***	-0.071***	-0.070***	-0.046***	-0.071***
*Age 35-39	(0.038)	(0.048)	(0.009)	(0.009)	(0.005)	(0.009)
Three or four prior births	0.039**	0.039**	0.007	0.007	0.007	0.007
*Age 15-19	(0.015)	(0.017)	(0.009)	(0.009)	(0.018)	(0.009)
Three or four prior births	-0.019**	-0.016*	-0.016***	-0.015***	-0.020***	-0.016***
*Age 20-24	(0.008)	(0.009)	(0.003)	(0.003)	(0.004)	(0.003)
Three or four prior births	-0.007	-0.003	-0.001	-0.000	-0.005**	-0.001
*Age 25-29	(0.006)	(0.006)	(0.002)	(0.002)	(0.002)	(0.002)
Three or four prior births	0.002	0.003	0.000	0.000	0.005**	0.000
*Age 30-34	(0.009)	(0.010)	(0.003)	(0.003)	(0.003)	(0.003)
Three or four prior births	-0.038***	-0.053***	-0.022***	-0.022***	-0.008**	-0.022***
*Age 35-39	(0.014)	(0.015)	(0.004)	(0.004)	(0.004)	(0.004)

Table 5 (continued)

Five or more prior births *Age 15-19			-0.830*** (0.230)	-0.825*** (0.230)	-0.731*** (0.242)	-0.854*** (0.231)
Five or more prior births *Age 20-24	0.018 (0.021)	0.037* (0.023)	-0.019 (0.013)	-0.017 (0.013)	-0.029 (0.024)	-0.019* (0.011)
Five or more prior births *Age 25-29	0.001 (0.009)	0.001 (0.011)	-0.012*** (0.003)	-0.011*** (0.003)	-0.018*** (0.005)	-0.012*** (0.003)
Five or more prior births *Age 30-34	0.001 (0.007)	0.001 (0.007)	-0.000 (0.002)	0.000 (0.002)	-0.005 (0.003)	-0.000 (0.002)
Five or more prior births *Age 35-39	-0.020* (0.011)	-0.022** (0.011)	-0.005 (0.003)	-0.004 (0.003)	-0.006* (0.003)	-0.005 (0.003)
Variance of unobserved individual effects						0.000 (0.000)
Observations	17,185	14,233	148,085	148,085	148,020	148,020
Log pseudo-likelihood	-8,825.20	-7,369.41	-7,4505.5	-7,4590.6	-7,4737.9	-7,4465.5

Notes: All estimations include a constant, age group dummies, prior-births dummies, an urban-residence dummy, the number of siblings, child mortality among siblings, ethnicity dummies, year dummies, and district dummies. Robust standard errors, clustered at the level of the sampling cluster, are in parentheses. Significance levels are indicated as *=significant at 10%; **=significant at 5%; ***=significant at 1%.

[1] In (5), dummies for number of surviving children are used instead of dummies for number of children born, both separately and when interacted with district HIV prevalence and age dummies.

[2] Random effects are at the level of the individual.