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Abstract

Recently there has been a surge in interest on how HIV/AIDS affects fertility in countries hit by the disease. In this study, the effect of communal HIV/AIDS on fertility in rural Malawi is estimated using individual data from the 2004 Malawi Demographic and Health Survey on fertility and the ideal number of children. The survey includes individual HIV status, making it possible to distinguish between behavioural and physiological effects. The main indicator of communal HIV/AIDS is the district-level prime-age mortality rate, obtained from the 1998 Population Census. The paper first tests the overall behavioural fertility response due to the epidemic, and then tests for differences in response due to gender-specific communal mortality and HIV rates, as well as individual age and knowledge about mother-to-child HIV transmission. The main findings are: communal HIV/AIDS has a negative but small impact on fertility; actual fertility and women's ideal number of children is more negatively affected by HIV/AIDS among women than among men; and a woman's age and knowledge about mother-to-child transmission of HIV are important determinants of her fertility response to the disease.

Keywords: fertility, gender, HIV prevalence, mortality, prime-age adult mortality.
JEL classification: I10, J13, O12

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1. Introduction

Since the identification of HIV in the beginning of the 1980s, the virus has killed about 25 million people all over the world. The worst affected regions are Eastern and Southern Africa, where national infection rates among adults are well over 10% in several countries, and prime-age mortality has risen four- or five-fold (UNAIDS, 2008). HIV/AIDS is thus a human catastrophe, and will continue so for many years to come.

In the worst affected countries the epidemic is having wide-ranging consequences, especially for individuals and households directly affected by the disease, but also for people in general. One consequence that has attracted recent attention is the impact of HIV/AIDS on women's reproductive behaviour. According to Young (2005, 2007), who analyze individual data from various Sub-Saharan countries, HIV/AIDS reduces fertility greatly and thereby raise long run per capita growth. The epidemic decreases fertility because of behavioural changes, as women avoid giving birth to potentially HIV-positive babies, not because of HIV-related physiological consequences. Yet, economic models suggest that adult increases in mortality should increase fertility (Soares, 2005; Lorentzen et al., 2008; Kalemli-Ozcan, 2009a). In addition, some recent studies find that HIV/AIDS has a minor or no impact on fertility (Magadi and Agwanda, 2007; Juhn et al., 2008; Ahuja et al., 2009; Fortson, 2009; Kalemli-Ozcan, 2009a, 2009b).

Most of the studies that report no or very small effects implicitly point towards a lack of behavioural fertility response; those that find minor fertility-declines attribute them to lowered fecundity among HIV-positive women. In general, however, the studies make no attempt to explain why the disease has so little impact on behaviour.

HIV/AIDS can affect reproductive behaviour through various mechanisms, and it is possible that they counteract each other: For instance, risk reduction to avoid infection could reduce fertility, while AIDS mortality could raise it. Durevall and Lindskog (2009), using data from Malawi, show that young woman give birth to their first child sooner, while older women who has already started child-bearing, decrease their fertility. Fink and Linnemayr (2008), studying five other African countries, find that better educated women reduce fertility as a response to the HIV epidemic, while less educated women instead increase it. Hence both papers find substantial, but heterogeneous, behavioural changes, counteracting each other.

In this paper we study the impact of communal HIV/AIDS on the fertility of women in rural Malawi, HIV-positive and HIV-negative alike.[1] We estimate ordered probit models using individual data on actual fertility and ideal number of children from the 2004 Malawi Demographic and Health Survey (MDHS). The MDHS data includes individual HIV status, so we are able to distinguish between behavioural and physiological effects. Our main measure of the epidemic, i.e., our indicator of HIV/AIDS, is district prime-age mortality rates, obtained from the 1998 Population Census. However, we also use district HIV prevalence rates from the MDHS.

Our main purpose is to analyse specific channels through which HIV/AIDS affects fertility, going beyond the average effects. However, we first address the overall behavioural fertility response due to the epidemic. Then we test for differences in response due to gender-specific district mortality and HIV rates, as well as individual age and knowledge about mother-to-

[1] Women living in urban areas, about 17% of the sample, are excluded from the analysis since their behaviour is likely to differ substantially from those in rural areas. For instance, several of the explanatory variables, such as ethnicity, probably affect urban and rural women differently.

child HIV transmission.[2] By analyzing heterogeneous responses we can thus shed light on mechanisms whereby HIV/AIDS might affect fertility.

In line with some other studies, we find that HIV/AIDS has a small, but significant, negative impact on fertility. Overall, communal HIV/AIDS reduced fertility by about 5% during 1999-2004 in rural Malawi, with roughly half due to behavioural change in the general population, and half due to the physiological consequences of the disease. Hence, we fail to find support for the hypotheses that HIV/AIDS either sharply reduces fertility, or increases it. Furthermore, fertility response differs depending on gender-specific communal mortality and HIV rates: Actual fertility and women's ideal number of children are more negatively affected by communal HIV/AIDS among women than among men. And men's ideal number of children is more negatively affected by men's mortality and HIV rates. A woman's knowledge about mother-to-child HIV transmission is also an important determinant of her fertility response to the disease. We also confirm the age-specific pattern of the fertility response found by Durevall and Lindskog (2009) using different measurements of the HIV epidemic and different empirical method: The youngest women increase fertility, while older women decrease fertility. However, HIV/AIDS seems to reduce the ideal number of children slightly for women in all age groups.

There are a number of possible explanations to these findings. Two plausible ones are that parental altruism makes women give birth earlier or abstain from births to avoid having HIV-positive babies or leaving young children orphaned, and that women and men want fewer

[2] We also tested for different responses depending on the level of education, but could not confirm the findings of Fink and Linnemayr (2008). A possible explanation is that there are very few people with tertiary education in our sample and that it only includes the rural population. The results from the regressions are available on request.

children when gender-specific own mortality risks increase, since the need of care and support from children, and therefore their marginal benefit, is reduced.

The following section outlines various mechanisms through which HIV/AIDS might impact on fertility. Section 3 then briefly describes the recent development of HIV/AIDS and fertility in Malawi. Section 4 presents the empirical model, and Section 5 describes the data. Section 6 reports the results from the empirical analysis, while section 7 summarizes and draws conclusions.

2. Theory and Evidence on HIV/AIDS and Fertility

HIV/AIDS affects fertility in numerous ways with no single theory embracing them all. Broadly speaking, it can affect fertility through three channels: direct physiological effects; changes in desired number of children for both HIV-positive and HIV-negative women and men; and changes in sexual behaviour to avoid infection.

2.1 Physiological Effects of HIV/AIDS

The physiological channel works through several mechanisms that all seem to point towards reduced fertility among HIV-infected women, which is 20% to 40% less than for uninfected women, as shown by Gray et al. (1998), Zaba and Gregson (1998), Terceira, et al. (2003), Fabiani et al. (2006) and Juhn et al. (2008). The most important mechanisms are believed to be: higher rates of miscarriage and stillbirth; co-infection with other sexually transmitted diseases; weight loss leading to amenorrhoea; other menstrual dysfunctions; reduced frequency of intercourse because of illness; and premature death of regular partner (Zaba and Gregson, 1998; Fabiani, et al 2006). There is also an indirect impact on the net rate of fertility through premature deaths of women in their reproductive years (Lewis et al., 2004).

2.2 Changes in Desired Number of Children

The life expectancy of both children and adults can affect the desired number of children. And one of the most stunning effects of HIV/AIDS is the reduction in overall life expectancy, which dropped from an estimate at close to 50 in the beginning of the 1990s to 42 in the late 1990s Malawi (Government of Malawi, 2007), because prime-age adult mortality has quadrupled due to AIDS (Doctor and Weinreb, 2003). AIDS also raises child mortality, but under-five mortality has dropped sharply in the midst of the epidemic, from 211 per 1,0000 births in 1990 to 111 in 2007 (UNICEF, 2009). Hence, other factors dominated over AIDS, including increased immunization, vitamin A supplementation, and exclusive breastfeeding, and elimination of neonatal tetanus (NSO and UNICEF, 2008).

There is a sizeable literature on the effects of mortality on fertility, most pointing towards a positive association, but it mainly focuses on child mortality. Parents desire a certain number of surviving offspring, so with higher mortality more births are needed, either replacing deceased children ex post or giving birth to more to start with (Schultz, 1997). When parents are risk averse and there is uncertainty about child survival, they might even have more children than needed to reach their target (Kalemli-Ozcan 2003). However, Doepke (2005) notes that if parents are also risk averse towards consumption, the impact of child mortality is reduced. In any case, as noted, AIDS-induced child mortality does not seem to be large enough to have a substantial effect on fertility.

Soares (2005) develops a model where adult mortality and fertility are also positively related, for which Soares (2006) provides empirical support. In this model there are two mechanisms at work. By reducing returns to education, adult mortality increases the relative attractiveness of childbearing, where the two are seen as alternatives: stay in school or get married and have babies. The second mechanism is that children's life expectancy enters directly into the

parents' utility function, since parents are assumed to care about the continued survival of their lineage, or at least evolution implies that they behave as if they do.

The quality of children is often interpreted in terms of their human capital, but it can also be understood as their welfare, which, since there is little doubt that altruism is important in families, must enter the parent's utility function (Becker, 1991: 277-78). Thus parents might wish to avoid giving birth to HIV-infected babies, or to leave their children orphaned at a young age. Young (2005, 2007) argues that this is an important reason for the fertility decline he finds. It is also possible that some women, instead of abstaining from having children, have them earlier, when they have a smaller probability of being HIV positive and dying of AIDS in the near future. In a qualitative study Zimbabwean women mention the possibility of both decreasing the number of births and giving birth earlier as responses to the risk of giving birth to HIV-infected babies (Grieser et al., 2001). Analysing data from Malawi, Durevall and Lindskog (2009) find that HIV/AIDS raised fertility among young HIV-negative women and decreased it among older ones.

In settings without social security and welfare programs, parents' need for support has been identified as an important motive for having children (Birdsall, 1987). In such settings, support and care are often organized around the extended family, to which reproduction and marriage of children contributes (Caldwell, 1976). If death rates of adult offspring increase, parents might decide to have more children, and the effect could be strong, because hoarding is the only alternative. However, HIV/AIDS affects parents' own mortality risk, possibly even more than that of their adult offspring since AIDS mortality can be expected to be lower by the time children reach adulthood. If parents risk dying at an early age, they have less need of old age support and care, reducing the marginal benefit of children. This effect might be

strong since there are indications that the HIV epidemic has raised the subjective mortality risk in Malawi even more than the objective risk (Delavande and Kohler, 2008). Ainsworth et al. (1998) mention this mechanism whereby HIV/AIDS could decrease fertility, but to our knowledge it has not been further explored in recent literature.

An aspect not previously addressed in the literature on HIV/AIDS is the impact of gender differences in infection rates. When parents' care about their own mortality, it might matter whether HIV/AIDS is more concentrated among women or men. For example, if the social security mechanism is important, and spouses care more about themselves than about each other, then the negative effect on desired fertility for women should be larger when the epidemic is more concentrated among women and smaller when concentrated among men.[3] For men's desired fertility, it should be the other way around. Also, if altruistic concerns about HIV mother-to-child transmission are important, female HIV/AIDS should matter more than male HIV/AIDS, and if women are more altruistic towards their children than men this difference should be more evident for women's desired fertility than for men's.

Yet another mechanism that might influence the desired number of children among women and men in Malawi is the increase in orphans. In Rutenberg et al.'s (2000) qualitative study in Zambia, the extra burden of caring for AIDS orphans was the only reason given by symptomless women for curtailing future childbearing. However, in Grieser et al.'s (2000) study in Zimbabwe many respondents said that they could not depend on fostered children for elderly care and support, and thus that taking care of orphans would not affect their own childbearing.

[3] Moreover, since husbands usually contribute economic resources and security to their wives, the risk that the husband dies increases the expected marginal value of future consumption and security, which can be provided by children. This raises the marginal benefit of children, and can thereby influence women's fertility preferences.

2.3 Changes in Sexual Behaviour to avoid HIV Infection

The risk of HIV infection increases the expected cost of sexual contact, particularly of risky sexual behaviour. Thus we should expect to see less risky sexual behaviour, which could mean increased abstinence, delayed age at sexual debut, increased condom use, fewer concurrent partners, and less extra marital sex.

There is an ongoing debate about the nature and extent of changes in sexual behavioural induced by the HIV epidemic, but there appears to have been little change in most African societies. Oster (2005) argues that the difference in response between the homosexual Americans and heterosexual Africans could be explained by the shorter life expectancy and lower incomes in Africa, which reduce the value of staying uninfected. However, the nature and extent varies widely, as documented by Glick and Sahn (2008), depending, for example, on HIV prevention campaigns and social stigmatization of the HIV infected (Epstein, 2007).

A response that would reduce the risk of infection is to marry and establish a (hopefully) monogamous relationship early. Ueyama and Yamauchi (2009) find that Malawian women marry earlier when district adult mortality is higher. Women might marry young voluntarily, but it could be that men have more bargaining power on the marriage market and decide to marry younger wives, less likely to be HIV positive. Another possible explanation is pressure on orphans to leave foster families. In any case, the fertility effect is that women start childbearing earlier.

3. Fertility and HIV/AIDS in Malawi

In the early 1960s the total fertility rate in Malawi was similar to those in other African and other less-developed countries. But while fertility in most other countries fell during 1960-1980, it grew in Malawi, 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). Fertility started to fall in the beginning of the 1980s at a rate similar to that in many other African countries. Nonetheless, it is still high compared to most countries, and there are indications that it has stopped falling. In the 2000s, the total fertility rate has been 6.0 to 6.3, implying that, on average, women in Malawi gave birth to one more child than women in the rest of Africa (NSO and ORC Macro, 2005; NSO and UNICEF, 2008).

Malawi's first AIDS case was diagnosed in 1985, when the national HIV prevalence rate should still have been very low. From then on the epidemic spread rapidly, first in the major cities, and then in rural areas. In the cities the HIV rate peaked in 1995 at 26% among women attending antenatal clinics, and then started to decline slowly. In the rural areas the rate was estimated to be 11.8% in 1999, and 10.8% in 2004 (National Aids Commission, 2004; NSO and OCR Macro, 2005). According to the most recent data, the national rate was 11.9% in 2007, when 68,000 people are estimated to have died from AIDS, out of a population of 13 million (UNAIDS, 2008).

HIV/AIDS has thus been prevalent in Malawi for over 25 years, and the epidemic has increased prime-age adult mortality about four times, i.e., three out of four deaths among prime-age adults are now due to AIDS (Doctor and Weinreb, 2003). As a result, knowledge about AIDS is widespread. In fact, already in the MDHS carried out in 1992, about 90% of respondents had heard about the disease, rising to 99% in the 2000 MDHS. So if HIV/AIDS affects decision-making about childbearing, this should be measurable.

One of the striking features of the epidemic is its differential impact on men and women: In 2004, close to 60% of the infected adults were women. Furthermore, male and female HIV rates vary widely across districts. For example, in Blantyre (with the most important commercial city), men and women have an equal probability of being HIV positive, while in Zomba (with an important university city), women have twice the probability of being affected as men (NSO and ORC Macro, 2005).

4. Empirical Model of Fertility

When analysing the effect of the HIV epidemic on fertility, we are ultimately interested in the effect on women's complete fertility, the total number of children given birth, and possibly the timing of those births. However, since the epidemic started in earnest in the mid-1980s, it is too early to study its effects on complete birth histories. Thus, using the approach of Soares (2006), we focus on fertility during the period 1999-2004. Soares studies childbearing up to the date of the survey, treated as a function of the woman's individual choice, factors not under her control, and her age. Since we are studying fertility during a limited period, we also use prior births to control for the stage of the reproductive life cycle the woman is in. Furthermore, fertility is allowed to depend on recent information on the HIV epidemic. There is also uncertainty in the model, which captures the fact that women cannot control their fertility perfectly, for biological reasons, such as fecundity, and for social reasons, such as their partner's attitudes.

We assume that the number of births during the study period is a function of *B*, a latent continuous variable that indicates the propensity to have a certain number of births, where $B = N + \varepsilon$, with $N = N(n(X), t, pb)$, and ε a random term representing uncertainty. Behaviour is determined by desired lifetime fertility, $n = n(X)$, where X includes individual

and communal factors; by the age of the woman, t; and the number of prior births, pb. The actual number of births, N, during a given period for a woman at a certain age is

$$\begin{aligned}
&0 \text{ if } B \le c_0;\\
&\text{k if } c_{k-1} < B \le c_k, \quad k = 1-3;\\
&4 \text{ if } c_3 < B;
\end{aligned}$$

where $c_0 - c_3$ are cut-off values and 4 is the maximum number of births observed during the period. We assume that ε is normally distributed and estimate this as an ordered probit model. The probability that a women will not give birth to any children during the period is then

$$\begin{aligned}
P(0) &= P(B \le c_0)\\
&= P(\varepsilon \le c_0 - N)\\
&= \Phi(c_0 - N)
\end{aligned} \qquad (1)$$

where $\Phi(.)$ indicates the standard normal distribution function. The other probabilities can be specified as

$$\begin{aligned}
P(1) &= \Phi(c_1 - N) - \Phi(c_0 - N)\\
P(2) &= \Phi(c_2 - N) - \Phi(c_1 - N)\\
P(3) &= \Phi(c_3 - N) - \Phi(c_2 - N)\\
P(4) &= 1 - \Phi(c_3 - N)
\end{aligned} \qquad (2)$$

The values of $c_0 - c_3$ are estimated as parameters in the model, together with the coefficients in $N = N(n(X), t, pb)$.

5. Data and Variables[4]

The main source of data is the nationally representative MDHS carried out in 2004, for which 10,058 women and 2,754 men living in rural areas were interviewed. As mentioned, we focus on rural areas since the determinants of fertility there probably differ substantially from those in urban areas. Apart from fertility-related information and data on a range of characteristics

[4] Tables A1 and A2 in Appendix report summary statistics for all variables used in the analysis.

of the respondents and their households, the 2004 MDHS contains HIV status for a subsample, the first nationally representative survey of HIV prevalence.

Our key dependent variable is realised fertility, which we measure as the number of births during the five–year period preceding data collection; i.e. from approximately mid-1999 to approximately mid-2004. The period was chosen to be after our preferred measure of HIV/AIDS, which is based on mortality data from the 1998 census. On average women gave birth to almost one child during the period (Table A1).

We also estimate models with desired fertility as the dependent variable. In the MDHS data, it is measured by asking about the ideal number of children a woman or man would have liked to have if it were possible to go back in time and choose freely. As with many survey questions of a subjective nature, it is not obvious that all persons understood the question equally, and as the researcher does. Still, we believe that estimations on the ideal number of children are a good complement to estimations on realised fertility, providing information about whether changes in realised fertility are related to changes in the desired number of children. On average both women and men want to have four children (Table A1).

We use two measures of HIV/AIDS, district prime-age adult mortality rates from the 1998 census, and the district HIV rate from the 2004 MDHS, both differentiated by gender.[5]

[5] The choice of indicator for HIV/AIDS varies in the literature: Young (2005, 2007) uses national HIV rates obtained from antenatal clinics; Kalemli-Ozcan (2009) uses national AIDS deaths, national HIV rates from antenatal clinics, and death rates based on data from DHSs; and Juhn et al. (2008) use regional HIV rates obtained from DHSs. In an earlier version Juhn at al. used DHS cluster HIV rates. We believe that our indicators strike a balance between 1) being sufficiently disaggregated to reflect a size of 'community' relevant for women's behavior (there are only three regions, but 27 districts, in Malawi) and 2) measurement problems from basing the indicators on too small samples, such as DHS clusters. Moreover, relatively few prime-age adults die each year, so the DHS estimates are less precise than the ones from population censuses. The drawback is that we only have cross-section data. In Durevall and Lindskog (2009) we use the Estimation and Projection package from WHO/UNAIDS and data from antenatal clinics in Malawi to estimate HIV prevalence over time for districts.

Prime-age adult mortality is the number of deaths per thousand individuals aged 30 to 49 years during 1997-1998 in rural areas of the district (provided by the Malawi National Statistical Office). As noted, AIDS causes three out of four deaths in this age group (Doctor and Weinreb, 2003). The mean district mortality rate is 17 deaths per 1000, varying from 7 to 36, and the mean district HIV rate is 11.5%, varying from 2.1% to 31.8% (Table A2).

In the 2004 MDHS, ten districts were oversampled to get statistically reliable estimates of HIV rates at district level: We use the data from these, but also from another sixteen districts since they are the best available measures of HIV prevalence in the general public.[6] Overall, in the sample of the rural population, 3,500 women aged 15-49, and 3,165 men aged 15-54, were eligible for HIV testing. The response rate was 71.2% for women and 64.8% for men: About 20 per cent of both women and men refused to take the test, and the rest were absent.

We prefer the 1998 prime-age adult mortality rates over the 2004 HIV rates as indicators of HIV/AIDS for three reasons. First, deaths are directly observable, as opposed to HIV status. Second, the mortality data should be of reasonably good quality as it comes from the population census. Finally, the mortality data predates the study period, so and any feed-back effects from fertility in 1999-2004 to mortality in 1998 are highly improbable.

Although HIV status is not directly observable, Young (2007) argues that women should be able to infer communal HIV rates from infants' deaths with AIDS symptoms, since the disease progresses rapidly in small children. But a potential drawback of using 2004 HIV rates is that women might have become infected while getting pregnant, which might cause a positive bias in the relationship between HIV rates and fertility. The expected survival time

[6] We exclude the smallest district, the island of Likoma, since there are too few observations on HIV status.

after infection is 10 to 12 years, so a rough estimate is that about half of the women who were HIV positive in 2004 became infected after 1999. To overcome this possible endogeneity, and to capture the biological consequences of the disease, we estimate models on a subsample of women with HIV status data, making it possible to distinguish between infected and uninfected women.

We also distinguish between effects of female and male district mortality and HIV rates. Differences in effects provide indirect information on such things as the role of children's support and care and parental altruism for fertility and ideal number of children, as described earlier. Another distinction regards the knowledge about mother-to-child HIV transmission, with women divided into three groups: those who did not know about it at all; those who knew about it but not how to prevent it; and those who knew about it and how to prevent it. This distinction can cast light on the importance of the worry of giving birth to potentially HIV-positive babies.

As noted earlier, we control for the woman's age and prior number of births. Age enters our model as seven dummy variables for the age groups 15-19, 20-24, etc., up to 45-49. Age at the time of the survey is used, with fertility measured during the previous five years. Prior number of births is the total up to the beginning of the five-year period.

Economic theory suggests that income and the opportunity costs of women's time should be important determinants of desired fertility. To capture these variables, household-wealth quintiles and the woman's educational level (no schooling or incomplete primary; complete primary; complete secondary; higher education) are included. If permanent income matters more than current income for fertility, wealth should be a good proxy (Bollen et al., 2007).

Information on wealth and education is available only from the survey year. Since less than 10% of the women had more than 8 years of schooling, most women have reached their completed level of schooling earlier than 1999, but endogeneity is a potential problem with the youngest group. There could also be endogeneity if there were systematic changes in relative wealth over the previous five years. However, we checked the robustness of our results by estimating models without wealth and education (not reported).[7]

Norms are likely to influence reproductive and sexual behaviour. Thus they might affect both the spread of HIV and fertility. We control for norms using dummies for ethnic and religious affiliation. It is hoped that these variables are sufficient to capture key differences in customs of the rural population under consideration.

Omitted variable bias is a possibility, so we include a number of district-level variables that could relate to fertility, adult mortality and HIV rates. Foremost, to ensure that adult mortality does not simply pick up child mortality effects, we control for child mortality (since adult and under-five mortality in 1998 are not correlated, this is not probable. Anyway it is important in principle to control for child mortality). Child mortality is measured by district under-five mortality in rural areas in the 1998 census. We also include measures of district poverty and economic inequality from the 1997/98 Malawi Integrated Household Survey. Moreover, since general health status is likely to influence fecundity as well as the spread of HIV (Stillwaggon, 2006), we use two district health variables. One is the share of women in the district with a Body Mass Index (BMI) below 20, calculated using the 2000 MDHS, included to control for the biological effects of bad health and poor nutrition on fertility. BMI below 20 is associated with significantly lower fertility rates (Nichols et al., 2003). The other is annual

[7] The regression results are available on request.

malaria incidence from Ministry of Health (2005). Malaria is widespread in Malawi, and it interacts with HIV as well as with fertility (Verhoeff et al., 1999; Van Geertruyden and D'Alessandro, 2007).

6. Empirical Results

In this section we first test for the effect of HIV/AIDS, measured by aggregated district-level mortality and HIV rates, on fertility. Then we test for differences in effects due to gender-specific district mortality and HIV rates (sub-section 6.2), individual knowledge about mother-to-child HIV transmission (sub-section 6.3), and individual age (sub-section 6.4).

6.1 Overall Fertility Effect

Table 1 reports eight specifications for fertility during 1999-2004. The first two specifications use the full rural sample of women, with prime-age adult mortality in (1) and HIV rates in (2). Specification (3) and (4) use the subsample of rural women with known HIV status, allowing for comparison with the remaining four specifications where the physiological consequences of the disease (BMI<20 and HIV status) are controlled for.

<<TABLE 1 ABOUT HERE>>

In specifications (1), (3), and (4), the null hypothesis of a zero effect of the HIV epidemic (adult mortality and HIV rate) on fertility can be rejected in favour of a negative effect. The results are stronger when we use adult mortality (specification 1) rather than HIV rates (specification 2), and when we use the HIV status subsample rather than the full sample (specifications 3 and 4). The stronger effect of adult mortality should be expected if the data is better (less measurement error attenuation) or if HIV rates in 2004 were not exogenous to fertility in 1999-2004, but partially the outcome of the sexual contacts that caused pregnancy.

Apart from pure chance, we can think of one explanation why the effects in the HIV status subsample would be larger than in the full sample: Those in the subsample, who accepted to be tested, are less inclined than others towards denial of the disease. Those who admit that the disease exists and might affect them are probably more likely to change behaviour, including childbearing.

Specifications (5) and (6) are the same as (3) and (4) but include individual HIV status and the district share of low-weight women (BMI<20). The addition of the control variables reduces the negative effect of both measures of district HIV/AIDS only marginally. HIV status has a statistically significant negative effect while BMI is not significant. In specifications (7) and (8) we use only the sample of women who were HIV negative in 2004, ruling out reduced fecundity due to HIV. Adult mortality still has a statistically significant negative effect, but the HIV rate is now not significant.

The magnitude of the effect of HIV/AIDS on fertility is illustrated in Table 2, which presents the predicted number of births per woman if prime-age adult mortality goes from its mean in 1987 (3.9 deaths per 1,000 aged 30-49) to its mean in 1998 (15.8).[8] Using the full sample and not controlling for physiological consequences (specification 1), women are predicted to give birth to 0.05 fewer children in a five-year period due to the HIV epidemic. If around 10% of the women were HIV positive, and HIV positive women give birth to about 30% fewer children (see Gray et al., 1998), and if women on average give birth to one child in five years without the HIV epidemic, then slightly more than half of the 0.05 decrease (0.03) would be explained by the physiological consequences of the disease on HIV-positive women. The effect of behavioural change is then roughly 0.02 fewer children per women.

[8] We also calculated the predicted number of births given changes in district HIV rate from 0 to 10.8% (the rural HIV rate estimated from the 2004 MDHS). The effect was weaker but the confidence intervals were in the negative region in all cases. The results are available upon request.

<<TABLE 2 ABOUT HERE>>

Restricting the sample to women with known HIV status (specification 3), however, leads to a substantially larger predicted decrease in births, 0.16 instead of 0.05. In this sample the effect decreases if we control for HIV status and district BMI<20 (specification 5), and if we further exclude HIV-positive women (specification 7), but it is always larger than the predicted effect in the full-sample estimations. However, the subsample of women who were tested for HIV should be less representative of women in rural Malawi than the full sample, so the behavioural effect is probably overestimated in the subsample. Nevertheless, using the subsample strengthen the findings of behavioural change, at least in a sub-group of the population. The behavioural effect thus seems to be negative but small, probably not more important than the negative effect of physiological consequences of the disease.

The coefficients of control variables are only presented in the first set of estimations of realised fertility (Table 1) and of ideal number of children (Table 3). The coefficients were not substantially different in other specifications. Moreover, to save space we do not report the coefficients of the ethnicity and religion dummies and the four cut-offs $c_0 - c_3$.

The coefficients of the age dummies show how age affects fertility non-linearly (Table 1), with a peak at 20-24 and 25-29, while giving birth to children before 1999 raises the probability of giving birth during 1999-2004. And, as usual, education is associated with lower fertility, with a statistically significant difference between those who have secondary education and those who have less than complete primary education.

Household wealth is also associated with lower fertility, with statistically significant effects for those in the third, fourth and fifth quintiles. This suggests that wealthier families substitute child quality for child quantity, or that the opportunity cost of women's time is higher in wealthier households, even after controlling for their educational level.

The district-level control-variable coefficients are mostly not significant except for wealth inequality, which is statistically significant in the regressions with the HIV-status subsamples. The coefficient is positive in all specifications, indicating that inequality is associated with higher fertility. Poverty, child mortality, malaria incidence and BMI<20 have no statistically significant associations with fertility.

Are reductions in fertility from district HIV/AIDS matched by corresponding decreases in the ideal number of children (Table 3)? As before, specifications (1) and (2) use the full sample of women, while specifications (3) and (4) use the HIV-status subsample controlling for individual HIV status, and specifications (5) to (8) are for men, first using the full sample, then the subsample with known HIV status. District adult mortality has a statistically significant negative effect on ideal number of children in the estimations on the full samples of women and men, and on the HIV-status sample of men, while district HIV rates have no statistically significant effects.

Age effects differ by gender; older women want fewer children than younger women, while older men want more children than younger men. The results of district poverty and health also differ by gender: Poverty and under five mortality are generally associated with a desire to have fewer children among women, while higher malaria incidence and the share of low-weight women are generally associated with a desire to have more children among men.

Women and men with more prior births also want more children. Finally, being HIV positive reduces the number of children wanted by men but not by women. Since many HIV-positive people do not know their HIV status we would not expect this result to be caused by that knowledge. But it could be that men who wish fewer children behave differently, which put them at higher risk of HIV infection.

Table 4 reports the predicted ideal number of children with and without the HIV epidemic, measured again by the change in adult mortality from 1987 to 1998. Since the predicted ideal number of children is just over 4, and the total fertility rate is about 6 children, we cannot really compare the size of the changes in ideal numbers of children with changes in realised fertility. Nevertheless, the average desired reduction in fertility was roughly 0.10 children for both sexes regardless of sample.

<<TABLE 4 ABOUT HERE>>

6.2 Gender-Differentiated Effects

Table 5 reports coefficients from regressions that distinguish between female and male district adult mortality rates, as well as predicted effects on fertility due to the increase in adult mortality from 1987 to 1998. To save space, the specifications with HIV rates and the coefficients of control variables are not reported.

<<TABLE 5 ABOUT HERE>>

The effect of female adult mortality on fertility is negative and statistically significant, both in the full sample (specification 1), and in the subsample with HIV-status control (specification

2). Male adult mortality has a statistically significant negative coefficient only in the HIV-status subsample (specification 4).

Male and female adult mortality rates are of course correlated, and thus likely to have similar coefficients when entered separately. However, the effects of female adult mortality rates are stronger in terms of both magnitude and statistical significance. In specifications (5) and (6), female and male rates enter simultaneously, resulting in statistically significant negative coefficients for female mortality in spite of the correlation, and positive, but statistically non-significant, coefficients for male mortality. The regressions control for the share of the district's women with BMI< 20, malaria incidence, under-five mortality, and poverty, making it unlikely that this result is due to less healthy women giving birth to fewer children.

To illustrate the importance of the gender effects, Table 6 presents results from simulations of three scenarios. 1) Female and male adult mortality rates increase in line with the country average from 1987 to 1998 (from 3.5 to 13.2 deaths per 1000 for females, from 4.5 to 18 deaths per 1000 for males). 2) The mortality increase is larger among women (3.5 to 15.9) and smaller for men (4.5 to 16.2). 3) The mortality increase is smaller among women (3.5 to 11.5) and larger among men (4.5 to 20.6).[9]

<<TABLE 6 ABOUT HERE>>

The negative impact of HIV/AIDS on fertility is clearly larger when it is more common among women than men. Using the full sample of women, women give birth to 0.060 fewer

[9] In the second and third scenarios, the proportions of the female to male mortality increases are the same as those in the districts with the largest increases in female and male mortality, respectively. The reason why male mortality has increased more than female mortality, in spite of higher HIV rates among women, is an interesting question, but not addressed in this paper.

children in scenario (2), where the mortality increase is relatively large for women, compared to 0.015 fewer children in scenario (3), when the mortality increase is relatively large for men. Using the HIV-status sample, the difference is also substantial: 0.175 compared to 0.083. Thus, there appears to be a behavioural response, which depends more on female than male HIV/AIDS.

The results from regressions with ideal number of children and, again, gender-differentiated mortality rates are presented in Table 7, women in the upper panel and men in the lower panel. For both, the predicted effects of the increase in adult mortality are negative. However, the effects are clearly stronger when female ideal number of children is regressed on female adult mortality, and when male is regressed on male, both in terms of the size of predicted decreases and the statistical significance. Furthermore, when female and male rates enter simultaneously, the effects of female mortality on women's ideal number of children are negative and those of male mortality are positive, whereas the reverse is true when men's ideal number of children is the dependent variable. However, in these cases the only statistically significant coefficient is the one for female adult mortality in the full sample regarding women's ideal number of children.

<<TABLE 7 ABOUT HERE>>

The finding that fertility and women's ideal number of children, but not men's ideal number of children, depends on gender-differentiated mortality in the same way suggests that women have a stronger influence on fertility than men. This probably seems natural too many, but contrast with some views of male dominance. Although men have more bargaining power than women in general, this is probably not the case for childbearing where a woman can use

contraceptives or even sterilization without asking her husbands' opinion. The use of injectable contraceptives in Malawi, which a woman can use without her husband's knowledge, in fact increased dramatically during the 1990s, and they are by far the most popular contraceptive, especially among married women: 18% used them at the time of the survey and 41% have used them at least once (NSO and OCR Macro, 2005).

The finding that there is a more negative effect of female district HIV/AIDS on fertility and on women's ideal number of children, while men's ideal number of children is more negatively affected by male district HIV/AIDS, suggests that support and care from children to parents might be important for understanding the fertility response to HIV/AIDS. An alternative explanation could be altruism. Female HIV after all, is what matters most for the risk of giving birth to HIV-infected babies. A reason for men's results would then be that men are less altruistic towards their offspring than women.[10]

6.3 Knowledge About Mother-to-child Transmission and the Fertility Response

Table 8 reports predicted changes in number of births due to changes in adult mortality among three groups of women based on their knowledge of mother-to-child HIV transmission: those who did not know about mother-to-child HIV transmission at all; those who knew about it but not how to prevent it; and those who knew about it and how to prevent it.

<<TABLE 8 ABOUT HERE>>

The negative effect on fertility is substantially larger for women who knew about mother-to-child transmission but not how to prevent it, presumably indicating that parental altruism

[10] If parents are altruistic and want to avoid mother-to-child transmission, then less altruism among men would explain why men's ideal number of children is not, as women's, more negatively affected by female than male adult mortality. However, it does not explain why men's ideal number of children seems to be somewhat more negatively affected by male than female mortality.

should matter. An alternative explanation could be that selfish parents reduce births since the cost of a surviving child increases if its mortality risk increases. Either way, knowledge about the disease is important if women should respond to it efficiently in accordance with their own interests.

6.4 Age Heterogeneity of the Fertility Response to HIV/AIDS

Tables 9 and 10 report the predicted change in number of births and ideal number of children when adult mortality increase from its 1987 to its 1998 level, allowing the response to the HIV epidemic to differ by age group.[11] As also found by Durevall and Lindskog (2009), young women give birth to more children (Table 9) where district adult mortality (and HIV rates) are higher, while older women give birth to fewer children. In the full sample, women aged 15-19 give birth to about 0.08 more children during five years (34% more than without HIV/AIDS), while women aged 25-44 give birth to over 0.1 fewer children (about -10%) and women aged 45-49 give birth to 0.05 fewer children (about -14%). In the HIV-status sample, the declines in fertility are substantially larger. Nonetheless, young people in districts with high HIV rates do not want more children (Table 10). For women, more HIV/AIDS seems to be associated with a desire for fewer children irrespective of age, while for men it is difficult to discern any pattern. Again, the results are consistent with altruistic mothers who give birth earlier to reduce the risk of having HIV-infected babies or of leaving young children orphaned. Alternative explanations could be that earlier childbearing is an unintended consequence of women marrying earlier in an attempt by men (and women) to decrease the risk of HIV infection or of ending up childless (or because parents pressure them to marry early to avoid ending up without grandchildren).

<<TABLE 9 ABOUT HERE>>

[11] Results for predictions with HIV rates are qualitatively similar and available on request.

<<TABLE 10 ABOUT HERE>>

7. Summary and Conclusions

We analyzed the impact of district HIV/AIDS in rural Malawi on both women's fertility and women's and men's desired fertility, among HIV-positive and HIV-negative alike. The aim was to analyze how HIV/AIDS affects fertility rates and on the channels involved.

We find evidence of a negative impact on fertility. The impact seems to be partially due to behavioural changes, since the findings holds even in a sample of HIV-negative women, while controlling for district health indicators. Women living in districts with high prime-age adult mortality and HIV rates give birth to fewer children than women living in districts with low rates. We got similar results in estimations of women's and men's ideal number of children. Nonetheless the total (negative) fertility impact is small, and the contribution of behavioural change does not appear to be larger than the direct physiological contribution due to reduced fecundity among HIV-positive women, which is referred to as tiny in much of the literature, i.e., 2-4% fewer children per woman with an HIV rate at 10%, as in the rural areas of Malawi.

The small overall behavioural change is partly due to heterogeneity and we find larger responses to HIV/AIDS among certain groups of women. Understanding the reasons for the heterogeneity might shed light on why the behavioural effect of HIV/AIDS on fertility appears to be small and negative.

One source of heterogeneity is individual age. Young women (aged 15-19 at the time of the survey) are predicted to have given birth to 0.08 more children in five years, 34% more than

without HIV/AIDS. Older women (aged 25-44 at the time of the survey) have instead given birth to at least 0.11 fewer children due to the HIV epidemic, a decrease of about 10%. Since adult mortality and HIV rates are negatively associated with women's ideal number of children for all age groups, this indicates a shift in timing of births, not a desire by young women to have more children.

Another source of heterogeneity is women's knowledge about mother-to-child HIV transmission. The negative effect on fertility is substantially larger for women who knew about mother-to-child transmission, but not how to prevent it, than among women who either did not know about mother-to-child transmission or knew about it and how to prevent it. In the five-year period studied, women who feared mother-to-child transmission of HIV gave birth to 0.10 fewer children, while other women gave birth to 0.03 fewer children.

The last source of heterogeneity is due to gender differences in district mortality and HIV rates. Female mortality affects realised fertility and women's ideal number of children more than does male fertility, while the opposite is true for men's ideal number of children, indicating that women have more control over fertility than their male partner. The gender differences were illustrated by creating scenarios based on the actual distribution of district HIV/AIDS among women relative to men. In districts where HIV/AIDS is most common among women, the predicted number of births during five years decreases by 0.060, compared with a decrease of 0.015 in a scenario where it is most common among men.

There are several potential explanations for the heterogeneity, and they are not easy to disentangle. However, some are more credible than others. First, parental altruism could explain much of our results. Altruistic women probably do not want HIV-infected babies, and

the substantially stronger negative fertility impact among women with reason to fear mother-to-child transmission of HIV indicates an unwillingness to give birth to HIV-infected babies. Altruism could also explain the age heterogeneity: By giving birth earlier, women reduce the risk of giving birth to HIV-infected babies, or of leaving young children orphaned. Furthermore altruism could to some extent explain the heterogeneity due to gender differences in district mortality and HIV rates. Since female HIV is what matters most for the risk of having HIV-infected children, the fertility and women's ideal number of children should be, and are, more dependent on female than male mortality. Men's ideal number of children is less affected by female than male mortality, but it could be that men are less altruistic towards their children than women or less aware of the risks and consequences of transmission.

Self-interested motives could also explain some of the results. Our only candidate to fully explain heterogeneity due to gender differences in district mortality and HIV rates is self-interested women and men for whom children's support and care is an important reason for having children. If social security is a key motive for having children, parents own increased mortality risk reduces the marginal benefit of children, but there is a difference whether HIV/AIDS is more concentrated among women or men. If women have more power than men over fertility, then fertility and women's ideal number of children would depend more on female than male mortality, while men's ideal number of children would depend more on male than female mortality, which is the case in our data.

For selfish reasons, parents might also wish to avoid having children who might die soon anyway, if born HIV infected. This, however, would be contrary to how parents are usually believed to react to child mortality. Although Becker and Barro (1988) suggest that the

demand for surviving children should decrease with children's mortality, since the average cost of a surviving child increases, the fact that more births are needed to reach the target number of survivors makes it unlikely that this explains the result.

A self-interested concern over own health could also explain some of the results. A concern over getting infected by HIV could lead to less risky sexual behaviour, and, consistent with the age heterogeneity, to women marrying and starting childbearing earlier. Earlier marriage among women (reported by Yamauchi and Ueyama, 2009) could result from both men and women aiming at establishing stable monogamous relationships to decrease the risk of HIV infection, or from comparatively older men wanting to marry young women who are less likely to be HIV positive. However, early marriage and childbearing could also result from pressures on (AIDS) orphans to ease the burden on their foster families, or from the desire to start childbearing earlier for altruistic reasons as explained above.

Economic models of fertility and adult mortality predict that they covary positively (Soares, 2005; Lorentzen et al., 2008; Kalemli-Ozcan, 2009a). The finding of a negative behavioural fertility response to HIV/AIDS thus means that the mechanisms in those models do not dominate. Altruism towards children often does not enter economic models, but, as Becker (1991: pp. 8-9), argues, economists may be quite alone in considering it obvious that people like bananas, but not that parents are altruistic towards their children. Moreover, there is the social security mechanism, which, as opposed to most economic theory, depends on the parents' own mortality rather than that of their offspring. If it is important, fertility models should differentiate between parents' and offspring's mortality, or perhaps between parents' perceptions about their own and their offspring's mortality risks.

We would like to stress two additional findings: first, women with reason to fear mother-to-child transmission of HIV reduces fertility much more than other women, clearly showing the importance of information and knowledge for the ability to make decisions in line with one's interests and preferences; second, women in the HIV-tested subsample reduced fertility much more than those in the full sample, indicating some systematic differences in the samples.

As with all observational studies, the interpretation of causality is tenuous, but detailed analysis of each specific explanation for the behavioural response could be carried out, perhaps using instruments. Furthermore, some questions that should be addressed in future studies are how the behavioural responses affect socioeconomic variables, such as demand for schooling, child mortality, and female labour supply, and what policies governments should implement to mitigate negative effects.

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Appendix

Table A1: Individual-level summary statistics

	Women		Men	
	Mean	Std. Err.	Mean	Std. Err.
Births last 5 years	0.974	0.870		
Ideal number of children	4.056	1.345	4.096	1.523
Age 15-19	0.200	0.400	0.200	0.400
Age 20-24	0.236	0.425	0.179	0.384
Age 25-29	0.181	0.385	0.191	0.393
Age 30-34	0.133	0.339	0.144	0.351
Age 35-39	0.099	0.298	0.087	0.283
Age 40-44	0.083	0.276	0.091	0.288
Age 45-49	0.068	0.252	0.052	0.223
Age 50-54			0.054	0.225
Prior births (5 years ago)	2.220	2.611		
Total number of births	3.194	2.740	2.994	3.328
No or incomplete primary education	0.260	0.439	0.117	0.321
Primary education	0.641	0.480	0.678	0.467
Secondary education	0.097	0.296	0.195	0.396
Higher education	0.002	0.043	0.010	0.099
1st wealth quintile	0.203	0.402	0.150	0.357
2nd wealth quintile	0.227	0.419	0.236	0.425
3rd wealth quitile	0.238	0.426	0.256	0.437
4th wealth quintile	0.217	0.412	0.233	0.423
5th wealth quintile	0.115	0.319	0.124	0.330
Catholic	0.779	0.415	0.786	0.410
Central African Presbyterian Church	0.164	0.371	0.175	0.380
Anglican	0.016	0.127	0.017	0.130
Seventh day adventist/baptist	0.061	0.239	0.058	0.234
Other christian	0.365	0.481	0.379	0.485
Muslim	0.161	0.367	0.132	0.339
No religion	0.012	0.107	0.026	0.159
Chewa	0.314	0.464	0.323	0.468
Tumbuka	0.098	0.297	0.098	0.297
Lomwe	0.201	0.400	0.204	0.403
Tonga	0.019	0.135	0.020	0.140
Yao	0.160	0.366	0.134	0.341
Sena	0.031	0.174	0.035	0.185
Nkonde	0.008	0.088	0.010	0.099
Ngoni	0.091	0.287	0.095	0.293
Other ethnicity	0.079	0.269	0.082	0.274

Table A2: District-level variables summary statistics

	Obs.	Mean	Std. dev.	Min	Max
Adult mortality	27	0.017	0.008	0.007	0.036
Female adult mortality	27	0.014	0.007	0.006	0.031
Male adult mortality	27	0.019	0.010	0.007	0.041
HIV rate	26	0.115	0.075	0.021	0.318
Female HIV rate	26	0.122	0.068	0.022	0.301
Male HIV rate	26	0.108	0.091	0.000	0.336
Under-five mortality	27	0.161	0.043	0.053	0.238
Malaria incidence	26	0.308	0.111	0.150	0.630
Share of women with BMI<20	26	0.256	0.050	0.148	0.365
Poverty (headcount)	25	0.656	0.108	0.421	0.840
Wealth inequality (Gini)	24	0.671	0.098	0.450	0.820

Table 1: Realised fertility among rural women in Malawi 1999-2004: Ordered probit coefficients with dependent variable births during the previous five years (0 to 4)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Age 20-24	1.550***	1.552***	1.431***	1.435***	1.494***	1.500***	1.495***	1.499***
	(0.0471)	(0.0472)	(0.0908)	(0.0910)	(0.0906)	(0.0907)	(0.0922)	(0.0923)
Age 25-29	1.569***	1.568***	1.404***	1.407***	1.481***	1.487***	1.529***	1.537***
	(0.0560)	(0.0558)	(0.101)	(0.100)	(0.102)	(0.101)	(0.108)	(0.107)
Age 30-34	1.182***	1.182***	1.049***	1.046***	1.160***	1.162***	1.227***	1.234***
	(0.0601)	(0.0602)	(0.126)	(0.126)	(0.126)	(0.126)	(0.138)	(0.139)
Age 35-39	0.860***	0.860***	0.870***	0.861***	0.987***	0.984***	1.032***	1.033***
	(0.0748)	(0.0749)	(0.153)	(0.154)	(0.156)	(0.156)	(0.169)	(0.169)
Age 40-44	0.161*	0.158*	0.00217	-0.0220	0.125	0.110	0.145	0.137
	(0.0878)	(0.0879)	(0.168)	(0.169)	(0.169)	(0.170)	(0.187)	(0.187)
Age 45-49	-0.507***	-0.509***	-0.616***	-0.617***	-0.517***	-0.513***	-0.484**	-0.476**
	(0.0972)	(0.0974)	(0.194)	(0.195)	(0.195)	(0.197)	(0.218)	(0.218)
Prior births	0.0668***	0.0669***	0.0543***	0.0543***	0.0456**	0.0450**	0.0373	0.0366
	(0.00984)	(0.00982)	(0.0205)	(0.0204)	(0.0205)	(0.0204)	(0.0232)	(0.0232)
Primary	-0.0130	-0.0175	0.0323	0.00962	0.0273	0.00716	0.00574	-0.00505
education	(0.0394)	(0.0393)	(0.0702)	(0.0703)	(0.0700)	(0.0701)	(0.0770)	(0.0770)
Secondary	-0.339***	-0.345***	-0.304***	-0.329***	-0.314***	-0.334***	-0.342***	-0.351***
education	(0.0597)	(0.0599)	(0.113)	(0.114)	(0.113)	(0.114)	(0.124)	(0.125)
Higher	-0.684	-0.694*	-1.557**	-1.602***	-1.715***	-1.770***	-1.722***	-1.779***
education	(0.420)	(0.418)	(0.619)	(0.614)	(0.626)	(0.626)	(0.604)	(0.604)
2nd wealth	-0.00242	-0.00374	-0.108	-0.104	-0.118	-0.115	-0.139	-0.138
quintile	(0.0426)	(0.0426)	(0.0885)	(0.0874)	(0.0891)	(0.0880)	(0.0980)	(0.0974)
3rd wealth	-0.0405	-0.0424	-0.168**	-0.166**	-0.170**	-0.170**	-0.219**	-0.220**
quitile	(0.0370)	(0.0368)	(0.0785)	(0.0777)	(0.0794)	(0.0787)	(0.0887)	(0.0886)
4th wealth	-0.145***	-0.147***	-0.229***	-0.240***	-0.221**	-0.232***	-0.248**	-0.256***
quintile	(0.0448)	(0.0449)	(0.0873)	(0.0869)	(0.0880)	(0.0875)	(0.0959)	(0.0955)
5th wealth	-0.324***	-0.323***	-0.441***	-0.439***	-0.401***	-0.400***	-0.484***	-0.487***
quintile	(0.0514)	(0.0513)	(0.103)	(0.104)	(0.105)	(0.106)	(0.115)	(0.117)
HIV positive					-0.506***	-0.515***		
					(0.0769)	(0.0761)		
Adult	-5.909**		-18.88***		-16.34***		-11.18**	
mortality	(2.911)		(4.959)		(4.920)		(5.382)	
HIV rate		-0.480		-0.991**		-0.777*		-0.390
		(0.300)		(0.484)		(0.465)		(0.491)
Under-five	-0.430	-0.730	1.493	0.536	1.983**	1.223	1.621	1.086
mortality	(0.482)	(0.458)	(0.939)	(0.871)	(0.973)	(0.900)	(1.027)	(0.950)
Malaria	-0.0636	-0.182	-0.136	-0.403	-0.133	-0.327	-0.160	-0.281
incidence	(0.167)	(0.178)	(0.275)	(0.285)	(0.296)	(0.304)	(0.310)	(0.310)
Share					-0.492	-0.716	-0.268	-0.397
BMI<20					(0.668)	(0.709)	(0.700)	(0.727)
Poverty	0.0571	0.136	0.183	0.299	0.198	0.267	0.159	0.165
	(0.191)	(0.204)	(0.320)	(0.349)	(0.326)	(0.350)	(0.352)	(0.376)
Wealth	0.218	0.126	0.994***	0.742*	1.051***	0.838**	0.865**	0.745*
inequality	(0.204)	(0.204)	(0.377)	(0.400)	(0.378)	(0.400)	(0.413)	(0.422)
Observations	9645	9645	2379	2379	2379	2379	2057	2057

Notes: All estimations also include ethnicity dummies. religion dummies, and four ordered probit cut-off values. Standard errors in parentheses.

*** p<0.01, ** p<0.05, * p<0.1.

Specifications (1) and (2) use the full rural sample of women; (3), (4), (5), and (6) use the subsample with known HIV-status; (7) and (8) use only the subsample of women knowingly HIV-negative in 2004.

Table 2: Effect of HIV/AIDS on realised fertility among rural women in Malawi

Change in predicted number of births during five years when district adult mortality goes from its 1987 to its 1998 mean (from 3.9 to 15.8 deaths per 1000 aged 30-49)								
				Predicted number of births				
Table 1 specification	Sample	HIV-status control	District share BMI<20	Without HIV/AIDS	With HIV/AIDS	Change	95% confidence interval for change	
(1)	Full	No	No	0.974	0.924	-0.050	[-0.052.	-0.048]
(3)	HIV status	No	No	1.134	0.970	-0.164	[-0.177.	-0.151]
(5)	HIV status	Yes	Yes	1.128	0.987	-0.141	[-0.152.	-0.130]
(7)	HIV neg.		Yes	1.107	1.012	-0.096	[-0.104.	-0.088]

Table 3: Ideal number of children among rural women and men in Malawi: Ordered probit coefficients with dependent variable ideal number of children

	Panel A: Women				Panel B: Men			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Age 20-24	-0.130***	-0.128***	-0.179**	-0.177**	-0.0167	-0.0225	-0.0697	-0.0730
	(0.0381)	(0.0382)	(0.0811)	(0.0812)	(0.0793)	(0.0796)	(0.0863)	(0.0868)
Age 25-29	-0.158***	-0.157***	-0.139	-0.137	0.0473	0.0471	0.0530	0.0553
	(0.0453)	(0.0453)	(0.0918)	(0.0916)	(0.0954)	(0.0955)	(0.101)	(0.101)
Age 30-34	-0.142***	-0.141***	-0.211*	-0.209*	0.157	0.151	0.198*	0.194*
	(0.0519)	(0.0520)	(0.112)	(0.112)	(0.0965)	(0.0969)	(0.114)	(0.114)
Age 35-39	-0.113*	-0.112*	-0.270*	-0.270*	0.257**	0.253**	0.331**	0.330**
	(0.0631)	(0.0632)	(0.139)	(0.139)	(0.120)	(0.120)	(0.141)	(0.141)
Age 40-44	-0.163**	-0.166**	-0.288**	-0.292**	0.248**	0.242*	0.293**	0.289**
	(0.0658)	(0.0660)	(0.141)	(0.140)	(0.124)	(0.124)	(0.144)	(0.144)
Age 45-49	-0.153**	-0.152**	-0.343**	-0.343**	0.241	0.233	0.199	0.193
	(0.0681)	(0.0681)	(0.145)	(0.145)	(0.165)	(0.165)	(0.199)	(0.199)
Age 50-54					0.251	0.246	0.244	0.240
					(0.170)	(0.169)	(0.201)	(0.200)
Prior births	0.0420***	0.0418***	0.0696***	0.0695***	0.150***	0.151***	0.133***	0.134***
	(0.00966)	(0.00966)	(0.0194)	(0.0194)	(0.0177)	(0.0176)	(0.0202)	(0.0200)
Primary education	-0.0510	-0.0558*	-0.0230	-0.0274	-0.0338	-0.0447	-0.0703	-0.0834
	(0.0319)	(0.0317)	(0.0612)	(0.0610)	(0.0783)	(0.0785)	(0.0991)	(0.0993)
Secondary education	-0.0924	-0.0988	-0.109	-0.113	-0.314***	-0.324***	-0.378***	-0.390***
	(0.0640)	(0.0642)	(0.116)	(0.116)	(0.0961)	(0.0962)	(0.116)	(0.116)
Higher education	0.195	0.183	-0.933*	-0.952**	-0.319	-0.315	-0.356	-0.360
	(0.431)	(0.430)	(0.496)	(0.483)	(0.291)	(0.292)	(0.343)	(0.344)
2nd wealth quintile	-0.0450	-0.0467	0.0310	0.0316	-0.149*	-0.149*	-0.182**	-0.179*
	(0.0394)	(0.0392)	(0.0797)	(0.0793)	(0.0762)	(0.0761)	(0.0924)	(0.0922)
3rd wealth quitile	-0.0687*	-0.0719*	-0.0455	-0.0457	-0.162**	-0.163**	-0.132	-0.130
	(0.0404)	(0.0402)	(0.0778)	(0.0772)	(0.0739)	(0.0740)	(0.0860)	(0.0862)
4th wealth quintile	-0.0240	-0.0268	0.0339	0.0316	-0.309***	-0.314***	-0.321***	-0.322***
	(0.0444)	(0.0444)	(0.0855)	(0.0849)	(0.0807)	(0.0803)	(0.0980)	(0.0976)
5th wealth quintile	-0.161***	-0.162***	-0.193*	-0.194*	-0.492***	-0.509***	-0.555***	-0.567***
	(0.0621)	(0.0616)	(0.103)	(0.103)	(0.0976)	(0.0987)	(0.117)	(0.118)
HIV positive			0.00304	-0.000801			-0.221**	-0.233**
			(0.0832)	(0.0830)			(0.0896)	(0.0903)
Adult mortality	-6.765**		-4.009		-7.736**		-8.263*	
	(2.937)		(5.100)		(3.867)		(4.288)	
HIV rate		-0.471		-0.140		0.0908		0.0963
		(0.299)		(0.605)		(0.410)		(0.446)
Under-five mortality	-1.274**	-1.615***	-1.368	-1.581*	-0.313	-0.739	-0.685	-1.154
	(0.625)	(0.574)	(0.948)	(0.885)	(1.023)	(0.991)	(1.132)	(1.111)
Malaria incidence	0.450	0.338	0.166	0.119	1.550**	1.456**	1.938**	1.828**
	(0.452)	(0.448)	(0.719)	(0.704)	(0.706)	(0.705)	(0.857)	(0.852)
Share BMI<20	0.106	-0.0123	0.209	0.165	0.676**	0.637*	0.520	0.494
	(0.201)	(0.207)	(0.279)	(0.294)	(0.329)	(0.333)	(0.360)	(0.362)
Poverty	-0.486**	-0.442**	-0.651*	-0.649*	-0.291	-0.382	-0.0723	-0.159
	(0.192)	(0.212)	(0.342)	(0.381)	(0.301)	(0.329)	(0.326)	(0.355)
Wealth inequality	0.451*	0.349	0.233	0.181	-0.0226	-0.0786	-0.315	-0.342
	(0.248)	(0.252)	(0.384)	(0.379)	(0.424)	(0.432)	(0.471)	(0.490)
Observations	8572	8572	2138	2138	2515	2515	1887	1887

Notes: All estimations also include ethnicity dummies, religion dummies, and four ordered probit cut-off values. Standard errors in parentheses.

*** p<0.01, ** p<0.05, * p<0.1.

Specifications (1), (2), (5), and (6) use the full rural sample of women or men; (3), (4), (7), and (8) use the subsamples with known HIV-status.

Table 4: Effect of HIV/AIDS on ideal number of children among rural women and men in Malawi

Change in predicted ideal number of children when district adult mortality goes from its 1987 to its 1998 mean (from 3.9 to 15.8 deaths per 1000 aged 30-49)

Table 3 specification	Sample	HIV-status control	Predicted ideal number of children Without HIV/AIDS	With HIV/AIDS	Change	95% confidence interval for change	
Panel A: Women							
(1)	Full	No	4.206	4.332	-0.127	[-0.184.	-0.069]
(3)	HIV status	Yes	4.180	4.252	-0.072	[-0.142.	-0.003]
Panel B: Men							
(5)	Full	No	4.046	4.153	-0.107	[-0.171.	-0.046]
(7)	HIV status	Yes	4.050	4.163	-0.112	[-0.185.	-0.040]

Table 5: Realised fertility among rural women in Malawi – differentiating between female and male district mortality and HIV: Ordered probit coefficients and predicted effects of HIV/AIDS with dependent variable births during the previous five years

	Sample	HIV-status control	Coefficients: Female adult mortality		Male adult mortality		Predicted effect of an increase in adult mortality from its 1987 to its 1998 mean		
(1)	Full	No	-6.549**	(3.208)			-0.0449	[-0.047	0.043]
(2)	HIV status	Yes	-19.04***	(5.305)			-0.1335	[-0.144	-0.123]
(3)	Full	No			-3.576	(2.349)	-0.034	[-0.036	-0.033]
(4)	HIV status	Yes			-11.95***	(4.189)	-0.1162	[-0.125	-0.107]
(5)	Full	No	-10.62*	(5.834)	3.954	(4.139)	-0.035	[-0.037	-0.034]
(6)	HIV status	Yes	-24.32**	(9.672)	5.006	(7.507)	-0.122	[-0.132	-0.112]

Notes: All estimations include age group, prior births, educational level, household wealth quintile, ethnicity, religion, district under-five mortality, share of district's women with BMI<20, district malaria incidence, district poverty, district wealth inequality, and four ordered probit cut-off values.
Standard errors in parentheses.
95% confidence intervals in brackets.
*** p<0.01, ** p<0.05, * p<0.1.

Table 6: Effect of HIV/AIDS on realised fertility depending on whether the disease is more concentrated among women or men: Effect on predicted number of births 1999-2004 of increased adult mortality.

Table 5 specification	Sample	Scenario 1: HIV/AIDS hit men and women as today[1]	Scenario 2: HIV/AIDS hit women more[2]	Scenario 3: HIV/AIDS hit men more[3]
(5)	Full	-0.035 [-0.037, -0.034]	-0.060 [-0.063, -0.058]	-0.015 [-0.017, -0.014]
(6)	HIV status	-0.122 [-0.132, -0.112]	-0.175 [-0.188, -0.162]	-0.083 [-0.090, -0.076]

[1] Female adult mortality increase from 3.5 to 13.2 annual deaths per 1000 women, and male from 4.5 to 18 (from 1987 to 1998 census district averages).
[2] Female adult mortality increase from 3.5 to 16 annual deaths per 1000 women and male from 4.5 to 16.2 (increase distributed as in district where it is largest among women).
[3] Female adult mortality increase from 3.5 to 11.5 annual deaths per 1000 women and male from 4.5 to 20.5 (increase distributed as in district where it is largest among men).
95% confidence intervals in brackets.

Table 7: Ideal number of children among rural women and men in Malawi – differentiating between female and male district adult mortality: Ordered probit coefficients and predicted effects of HIV/AIDS

	Sample	HIV-status control	Coefficients				Predicted effect of an increase in adult mortality from its 1987 to its 1998 mean
			Female adult mortality		Male adult mortality		
Panel A: Women							
(1)	Full	No	-8.126**	(3.174)			-0.1233 [-0.1776. -0.0708]
(2)	HIV status	Yes	-5.226	(5.816)			-0.0776 [-0.146. -0.0074]
(3)	Full	No			-4.734*	(2.439)	-0.0993 [-0.1505. -0.0497]
(4)	HIV status	Yes			-2.588	(4.035)	-0.0536 [-0.1092. 0.0053]
(5)	Full	No	-12.42**	(5.951)	4.222	(4.525)	-0.164 [-0.230. -0.100]
(6)	HIV status	Yes	-9.590	(11.04)	4.160	(7.596)	-0.0567 [-0.1164. 0.0032]
Panel B: Men							
(7)	Full	No	-7.093*	(4.199)			-0.0807 [-0.1292. -0.0332]
(8)	HIV status	Yes	-7.119	(4.706)			-0.0781 [-0.1317. -0.0257]
(9)	Full	No			-7.078**	(3.281)	-0.1109 [-0.1751. -0.0479]
(10)	HIV status	Yes			-7.968**	(3.579)	-0.1218 [-0.1998. -0.0445]
(11)	Full	No	0.612	(8.573)	-7.522	(6.683)	-0.1106 [-0.1748. -0.0479]
(12)	HIV status	Yes	4.360	(9.218)	-11.15	(6.994)	-0.1233 [-0.2009. -0.0453]

Notes: All estimations include age group, prior births, educational level, household wealth quintile, ethnicity, religion, district under-five mortality, share of district's women with BMI<20, district malaria incidence, district poverty, district wealth inequality, and four ordered probit cut-off values.
Standard errors in parentheses.
95% confidence intervals in brackets.
*** p<0.01, ** p<0.05, * p<0.1.

Table 8: Effect of district HIV/AIDS on realised fertility depending on knowledge about mother-to-child transmission: Effect on predicted number of births 1999-2004 of increased adult mortality from its 1987 (3.9 deaths per 1000 aged 30-49) to its 1998 mean (15.8).

	Do not know about mother-to-child transmission of HIV		Know about mother-to-child transmission of HIV but not how to prevent it		Know about mother-to-child transmission of HIV and how to prevent it	
Sample	Predicted change	Obs.	Predicted change	Obs.	Predicted change	Obs.
Full	-0.036 [-0.044. -0.028]	1226	-0.101 [-0.108. -0.095]	2718	-0.029 [-0.031. -0.027]	3452
HIV status	-0.041 [-0.053. -0.029]	299	-0.223 [-0.286. -0.160]	637	-0.143 [-0.160. -0.125]	888
HIV neg.	0.040 [0.029. 0.051]	258	-0.270 [-0.351. -0.188]	567	-0.055 [-0.062. -0.048]	747

Notes: Predictions are based on ordered probit estimations with controls for age group, prior births, educational level, household wealth quintile, ethnicity, religion, district under-five mortality, share of district's women with BMI<20, district malaria incidence, district poverty, and district wealth inequality.
95% confidence intervals in brackets.

Table 9: Age-specific predicted changes in number of births 1999-2004 when district adult mortality goes from its 1987 to its 1998 mean (from 3.9 to 15.8 deaths per 1000 aged 30-49)

	Full sample		HIV-status subsample	
Age 15-19	0.079	[0.078. 0.080]	0.074	[0.072. 0.077]
Age 20-24	-0.007	[-0.010. -0.003]	-0.112	[-0.131. -0.092]
Age 25-29	-0.118	[-0.136. -0.101]	-0.189	[-0.226. -0.151]
Age 30-34	-0.130	[-0.141. -0.118]	-0.266	[-0.302. -0.230]
Age 35-39	-0.111	[-0.119. -0.104]	-0.178	[-0.201. -0.155]
Age 40-44	-0.122	[-0.125. -0.118]	-0.244	[-0.258. -0.231]
Age 45-49	-0.047	[-0.048. -0.046]	-0.201	[-0.208. -0.194]
HIV-status control	No		Yes	
Observations	9645		2379	

Notes: Predictions are based on ordered probit estimations with controls for age group, prior births, educational level, household wealth quintile, ethnicity, religion, district under-five mortality, share of district's women with BMI<20, district malaria incidence, district poverty, and district wealth inequality.
95% confidence intervals in brackets.

Table 10: Age-specific predicted changes in ideal number of children when district adult mortality goes from its 1987 to its 1998 mean (from 3.9 to 15.8 deaths per 1000 aged 30-49).

	Panel A: Women		Panel B: Men	
	Full sample	HIV-status subsample	Full sample	HIV-status subsample
Age 15-19	-0.217	-0.2317	-0.285	-0.372
	[-0.316. -0.118]	[-0.4061. -0.0584]	[-0.399. -0.172]	[-0.558. -0.188]
Age 20-24	-0.071	-0.0264	-0.016	-0.024
	[-0.127. -0.016]	[-0.0891. 0.0378]	[-0.030. 0.000]	[-0.043. -0.006]
Age 25-29	-0.091	0.0379	0.023	-0.015
	[-0.156. -0.027]	[-0.038. 0.114]	[-0.006. 0.049]	[-0.043. 0.011]
Age 30-34	-0.223	-0.1292	-0.255	-0.300
	[-0.328. -0.119]	[-0.2724. 0.0137]	[-0.422. -0.089]	[-0.525. -0.077]
Age 35-39	-0.128	0.0448	-0.155	-0.045
	[-0.230. -0.024]	[-0.0585. 0.1476]	[-0.365. 0.056]	[-0.195. 0.105]
Age 40-44	0.047	0.0257	0.187	0.302
	[-0.030. 0.124]	[-0.0803. 0.1326]	[-0.028. 0.403]	[0.043. 0.562]
Age 45-49	-0.176	-0.4336	-0.252	-0.063
	[-0.312. -0.041]	[-0.7916. -0.0765]	[-0.711. 0.206]	[-0.361. 0.236]
HIV-status control	No	Yes	No	Yes
Observations	8572	2138	2515	1887

Notes: Predictions are based on ordered probit estimations with controls for age group, number of living children, educational level, household wealth quintile, ethnicity, religion, district under five mortality, share of district's women with BMI<20, district malaria incidence, district poverty, and district wealth inequality.
95% confidence intervals in brackets.