HIV/AIDS, Adult Mortality and Fertility: Evidence from Malawi

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1. Introduction

On average 5 percent of the working-age population in Sub-Saharan Africa is HIV positive (UNAIDS, 2008), and AIDS has become a human catastrophe with no doubt about its wide-ranging consequences for individuals and households affected by the disease. However, it is less obvious what the effects are at the societal or national level.

One contentious issue is the impact on women's reproductive behaviour. Changes in fertility are important because they can determine the long run economic consequences of the disease. Young (2005; 2007), for example, argues that the HIV epidemic is decreasing fertility in the general female population, and that the decrease is so large that it will increase long-run economic growth in many Sub-Saharan countries. Other studies on the fertility impact of HIV/AIDS have, however, not confirmed his results. Instead they suggest that it either increases fertility slightly or has no impact or very little impact (Fortson, 2008; Juhn et al., 2008; Kalemli-Ozcan, 2009). The possibility of a heterogeneous fertility response to the HIV epidemic has also been recognized. In a study on Malawi, Durevall and Lindskog (2009) show that young women give birth to their first child sooner, while fertility decreases for older women who have already started child-bearing. Fink and Linnemayr (2008), using data from five other African countries, find that better educated women may decrease fertility as a response to the HIV epidemic, while less educated women instead increase fertility, resulting in a close to zero net impact on fertility.

Studies showing little or no fertility response indirectly point towards a lack of behavioural response. However, the HIV epidemic could affect reproductive behaviour through a wide range of mechanisms, some may counteract each other, and many are likely to vary with the context. Such heterogeneity is highlighted by the findings of Durevall and Lindskog (2009) and Fink and Linnemayer (2008), which imply substantial behavioural change. To increase our understanding about the mechanisms involved, we thus have to go beyond the average effect of HIV/AIDS on fertility, which has occupied most of the recent economics literature.

In this paper we study the impact of communal HIV/AIDS on both realised and desired fertility of women in general, HIV positive and HIV negative alike, in rural Malawi. Though we also study the aggregate effect, the focus is on heterogeneity. An important contribution concerns the analysis of a mechanism that has to date been neglected in the literature; that adult mortality could reduce the need of future social security and old-age support from children, and thereby fertility. If women are more in control of fertility than men this can lead to a differential fertility impact of female respectively male communal HIV/AIDS. We also investigate the possibility of a change in timing of births towards younger ages due to HIV/AIDS resulting in an age-heterogeneous fertility response, and the possibility of a differential response depending on the woman's education.

We use communal prime-age adult mortality and HIV prevalence as measures of the epidemic and individual data on actual and desired fertility from the 2004 Malawi Demographic and Health Survey (DHS). The survey includes tests of HIV status, so we are able to distinguish between behavioural and physiological effects.

We find a negative total behavioural fertility impact of HIV/AIDS. But this effect is small, and most likely not more important than the physiological fertility consequences on HIV positive women. More interesting, we find that the fertility response differs greatly between female and male communal adult mortality and HIV prevalence; women have a much stronger desire to have fewer children in communal areas with high female HIV and adult mortality rates than in communal areas with high male HIV and adult mortality rates, something and they also do give birth to fewer children in these areas. Using different measurements of the HIV epidemic data and empirical method, we also confirm the age specific pattern of the fertility response found by Durevall and Lindskog (2009): the youngest women increase fertility and older women decrease fertility. Communal HIV/AIDS lowers desired total number of children in all ages, suggesting this to be the result of earlier birth-giving. However, we fail to confirm the education specific pattern of the fertility response found by Fink and Linneamayr (2008) for some other African countries, though the negative impact on desired total number of children is stronger among better educated women.

In the next section we outline the different mechanisms through which HIV/AIDS may impact on fertility with special focus on the old-age support and social security benefit of children channel. Based on this, we formulate hypotheses that are tested in the empirical section. In section three we briefly mention the recent development of HIV/AIDS and fertility in Malawi. Section four treats the empirical model, and section five the data and variables that we use. Section six gives the results from our empirical analysis, and section seven discusses concludes.

2. Theoretical background

HIV/AIDS seems to affect fertility in numerous ways with no single theory embracing them all. Broadly speaking, fertility could be influenced through three channels: a biological one, because of the physiological consequences of the disease on fecundity; changes in reproductive behaviour due to changes in desired fertility of both HIV-positive and HIV-negative women; and changes in sexual behaviour to avoid infection. Economic theory on fertility is concerned with, and will have most to offer with regard to, the second channel.

2.1 Physiological consequences of the disease

The biological channel works through various mechanisms that all seem to point towards reduced fertility among HIV-infected women. The most important ones 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 less frequency of intercourse because of illness (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). The difference in fertility between infected and uninfected 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 for girls aged 15-19[1], HIV-positive women have a lower fertility than HIV-negative women in all age groups, and the difference is considerable, between 25 and 40 percent: amounting to 0.37 percent decline in total fertility for each percentage point of HIV prevalence (Lewis et al., 2004).

2.2 Changes in desired fertility

According to economic theory parents invest in children as long as the marginal benefit exceeds the marginal cost. In a simple model parents' utility depends on the number of children and their quality, and on other consumption. The cost of children is the opportunity cost, i.e. the foregone consumption as time and other resources are spent on children. In the optimum parents allocate resources to equate marginal utilities from spending them on more

[1] The reason young HIV-positive women are more fertile is probably because they are more sexually active than uninfected women, and thus more likely to become both HIV-positive and pregnant. Since they recently have become infected, they are still quite healthy. Several studies have found that fecundity among infected women relative to that of non-infected women declines with the age, which is attributed to the progress of the disease (Ntozi, 2002 and Fabiani et al., 2006).

children given child quality, on child quality given the number of children, and on consumption (Willis, 1973; Becker and Lewis, 1973; Becker, 1981).

Building on this basic set-up, there is a sizeable literature on the effects of child mortality on fertility, and some of the mechanisms at work may also apply to adult mortality. When parents demand a target level of surviving off-springs, with higher mortality more births are needed to reach it. Parents may replace deceased children ex post[2] or they may ex ante give birth to a sufficient number of children to expect to reach the target. If parents are risk averse and there is uncertainty about child survival, parents also have a pre-cautionary demand for children; i.e. they demand more children than what is needed to reach the target number of expected survivors (Schultz, 1997).

Mortality also has an impact on fertility if the demand of surviving off-springs is affected. In the words of Becker and Barro (1988), the cost of a surviving off-spring increase with mortality constituting a negative force on the demand for children. But returns to child quality investments also decrease, and through the quality-quantity trade-off the demand of surviving children could thus increase with mortality (O'Hara, 1972), a possibility more in line with the empirical evidence of the demographic transition.

Generally, mortality decreases the utility payoff from investments in both child quality and child quantity. Depending on assumptions about the timing of costs, mortality and benefits, it is possible to generate results where the number of births increases and quality investments decreases, where the number of births decreases and quality investments increases, or where both child quantitative and child quality investments are reduced in favour of other consumption. Kalemli-Ozcan (2003) shows that with the plausible assumptions of uncertainty about survival and risk adverse parents, higher mortality will tend to lead to an increased number of births and less investment in child quality.

In early adulthood, child-bearing and investments in own education may be relevant alternatives for the woman. Since increased adult mortality reduces returns to education, women may invest less in own education and have more children, a mechanism explored in Soares (2005). He also argues that children's life expectancy and the number of children enter

[2] This is of course a less viable option if parents care about a possible death of off-springs after the first years of childhood.

as substitutes directly in the parent's utility function since parents care about the continued survival of their lineage, or at least evolution implies that they behave as if they do. To pass on one's genes throughout human history, it has been important not only that one's offspring survive the reproductive ages, but also that they do so in good shape. In Soares' model an exogenous increase in life expectancy will therefore decrease fertility.

Based on the discussion so far economic theory seems to suggest a positive relationship between fertility and mortality (of children and adults), and Kalemli-Ozcan 2009 argues that one could therefore expect a positive influence on fertility of the HIV epidemic. However, other effects of the epidemic are likely to affect fertility negatively.

The quality of children is often interpreted as their human capital but it can also be understood as the welfare of children. Becker (1981) explicitly acknowledges the role of altruism in the family whereby the utility (or welfare) of children enters the parent's utility function. This probably implies that parents wish to avoid giving birth to HIV-infected babies or leaving their children orphaned. Young (2005; 2007) argues that the HIV epidemic has decreased fertility and that this mechanism is an important reason for this. It is also possible that women, rather than abstaining from giving birth altogether, attempts to do so earlier when they have a smaller probability of being HIV positive and of dying in AIDS in the near future (Durevall and Lindskog, 2009). In a qualitative study on Zimbabweans (Grieser et al., 2001), women mention both the possibility of decreasing number of births and of giving birth earlier as a response to the risk of giving birth to HIV infected babies.

Yet another behavioural mechanism that might lead to lower fertility is the increase in orphans and children living with others than their parents. 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, in the study on Zimbabweans by Grieser et al. (2000), many respondents said that they could not depend on fostered children for old age support and that taking care of orphans does not affect their own childbearing.

2.3 Impacts on desired fertility through the old-age support and social security benefit of children

Children may of course not only draw on parent's resources, they could also contribute to them. An often proposed explanation for higher fertility in rural than urban areas in the developing countries is that children at young ages contribute to family income (Becker, 1981). And in settings without social security and pensions, parent's need for social security and support at old age has been identified as an important motive for having children. Caldwell (1976) writes extensively on the importance of children in African cultures where social and economic security is organized around the extended family. The routes to increasing ones network of relatives, and thereby the size of the security system and the cooperating group, is reproduction and marriage of children.

An increased risk of own early death means parents have less use of future social security and old age support from children, and the marginal benefit of children decreases. Furthermore, there are indications that the HIV epidemic has changed subjective mortality risks even more than the objective one in Malawi (Delavande and Kohler, 2008), which would decrease the marginal benefit of children even more.[3] Costs of children on the other hand tend to occur earlier and be more certain. Ainsworth et al. (1998) mention this mechanism whereby the HIV epidemic could reduce fertility, but it has not been acknowledged in the recent economics literature on how HIV/AIDS affects reproductive behaviour.

Furthermore, if life expectancy of off-springs and parents matter differently for fertility, responses to changes in general life expectancy might not be symmetric: during the demographic transition parents might have reduced fertility since they expected their children to live longer, while the current HIV/AIDS induced decrease in life expectancy may also decrease fertility, because parents expects to die sooner. The asymmetry could be worsened if subjective life expectancy changes do not correspond to the actual ones.

What's more, since the mechanism discussed here is related to parents' own mortality risks rather than their unborn children, gender-specific mortality rates may matter. If spouses care more about themselves than about each other, female mortality should be more important than male for women's desired fertility and vice verse for men. If women are more in control of fertility than their spouses, female mortality should matter most for realised fertility.[4]

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4 Moreover, since living with her husband is likely to increase the economic resources and security for the women, the risk that her husband dies decreases the marginal value of future consumption and security,

In general, men have most likely a larger bargaining power than women, but when it comes to fertility there is much room for the woman to manoeuvre it without asking about her partners' opinion. The DHS asks several questions about fertility regulation, and in the 2004 survey virtually all women that have ever had sex in Malawi knows about methods to regulate fertility. More importantly, the use of injectibles increased dramatically during the 1990s, and it has become the by far most popular method of birth control in Malawi, especially among married women (41% have used it and 18% currently used it) (NSO and OCR Macro, 2005). One feature of injectibles is that the women can use it without her husband's knowledge.

2.4 Changes in sexual behaviour to avoid HIV infection

The risk of HIV infection increases the expected cost of sexual contact, and particularly of risky sexual behaviour. Thus we should expect to see a decrease in risky sexual behaviour, which could for example materialise in increased condom use, delayed age at sexual debut, increased abstinence, less extra marital sex, or a decrease in concurrent partnership. There is a lively and ongoing debate about the extent and nature of sexual behaviour change induced by the HIV epidemic. However, it is probably fair to say that the degree of change has often been unexpectedly small in most African societies, and that both the extent and nature of the change varies widely depending on HIV prevention campaigns, social stigmatization of HIV infected, and other contextual factors. Oster (2005) argues that the difference in response between the homosexual community in the USA and heterosexual Africans could be explained by the shorter life expectancy and lower incomes in Africa, which reduces the value of staying uninfected. Irrespective of the degree of sexual behaviour change, people are probably unlikely to avoid unprotected sex with their partner when they want to have a child. But there could be a decrease in unwanted pregnancies.

Ueyama and Yamahuchi (2008) argue that that a possible response to decrease infection risk is to marry and establish a supposedly monogamous relationship earlier. They find that Malawian women do marry earlier when district adult mortality is higher. If men have more bargaining power on the marriage market, another explanation could be that men attempt to

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something that can be provided by children thus increasing the marginal benefit of children. So there can even be a positive influence of male mortality on women's fertility preferences.

marry younger wives that are less likely to be HIV positive. Irrespective of the underlying mechanism, the fertility effect is that women may start child bearing earlier.

2.5 Why education heterogeneity?

Fink and Linnemayr (2008) suggest three reasons to expect the fertility response to HIV/AIDS to differ between less- and better educated women; better educated women may easier take in information, understand risks, and thus change their behaviour appropriately; better educated women may have longer planning horizons; and better educated women tend to invest more, and for a longer time period, in child quality.

2.6 Empirical studies and formulation of testable hypotheses

To sum up the theoretical background, there are a number of mechanisms whereby mortality could influence fertility behaviour. And whether positive or negative forces dominate is an empirical question.

Earlier studies that have evaluated the fertility response to the HIV epidemic in the general female population have produced mixed results. Young (2005; 2007) find there to be a negative behavioural response to the epidemic in South-Africa and in a sample of Sub-Saharan countries, and Nöel-Miller (2003) finds that a high level of worrying regarding HIV/AIDS reduces fertility in Malawi. There are also studies that find a positive impact of adult mortality on fertility; Lorentzen et al. (2008) who use global macro data over the period 1960-2000, and Soares (2006) who use Brasilian micro data. Kalemli-Ozcan (2009) tests the association between HIV/AIDS and fertility, and her estimates based on between country variation shows that HIV/AIDS increases fertility. However, her within country estimates show no effect on fertility. This latter result is confirmed by some other studies; Magadi and Agwanda (2007), who study the effect of communal HIV rates on fertility in Kenya; Juhn et al. (2008), who use DHS data for thirteen Sub-Saharan countries;[5] Fink and Linnemayr (2008) who use the latest DHS data, and World Fertility Surveys, from before the HIV epidemic, for

[5] 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.

five African countries;[6] and Fortson (2008) who use a panel of regional mortality and total fertility rates.

A natural starting point when evaluating the fertility effect of the HIV epidemic in Malawi is thus to consider the sign of the effect; in doing so we attempt to differentiate between the total fertility impact, the behavioural fertility impact and the desired fertility impact. Our first hypotheses to test are:

Hypothesis 1: The total fertility impact is zero.
Hypothesis 2: The behavioural fertility impact is zero.
Hypothesis 3: The desired fertility impact is zero.

If impacts are different from zero it will be interesting to know whether positive or negative forces dominate, and the magnitude of effects.

From the preceding theoretical discussion more specific hypotheses can be formulated, that deals with gender, age and education specific effects. First, it was proposed that female and male HIV and adult mortality may have different impacts on desired fertility. Further, a number of mechanisms suggested that women may change the timing of fertility, and for a given number of children have them earlier. It was also suggested that the fertility response may differ between less- and better educated women. The following hypotheses are tested:

Hypothesis 4: Female HIV and adult mortality will have the same influence on women's desired fertility as male equivalents.
Hypothesis 5: Male HIV and adult mortality will have the same influence on men's desired fertility as female equivalents.
Hypothesis 6: Female HIV and adult mortality will have the same influence on realised fertility as male equivalents.
Hypothesis 7: Realised fertility of older women will be equally affected as fertility of younger women.
Hypothesis 8: There will be no difference in women and men's desired fertility by age.

[6] The countries are Cameron, Cote d'Ivoire, Ghana, Kenya, and Senegal.

Hypothesis 9: Realised fertility of better educated women will be equally affected as fertility of less educated women.

Hypothesis 10: There will be no difference in women and men's desired fertility by education.

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 should have been very low. From then on the epidemic, however, spread rapidly, first in the major cities, and then in rural areas. In urban areas the HIV prevalence disease peaked at 26 percent in 1995 among women attending antenatal clinics, but then started to decline slowly. In the rural areas the prevalence rate was estimated to be 11.8 percent in 1999 and 10.8 percent in 2004 (National Aids Commission, 2004; NSO and OCR Macro, 2005). According to the most recent data, the national rate was 11.9 percent in 2007, while 68,000 people died from AIDS during a year (UNAIDS, 2008).

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

One of the striking features of the HIV/AIDS epidemic is its differential impact on men and women: in 2004, close to 60 percent of the infected adults were women. Furthermore, the difference between male and female HIV prevalence rates differs widely between different 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) the female HIV prevalence rate is more than double the male one (NSO and ORC Macro, 2005).

In the early 1960s, the total fertility rate in Malawi was similar to those in other African and developing countries. But while fertility in most other countries fell, it grew in Malawi until 1980, probably because of the ideology and policy of the Malawian government under President Banda: birth control was seen as incompatible with the Malawian culture (Chimbwete et al., 2005). In the beginning of the 1980s fertility started to fall at a pace similar to that of many other developing countries. Nonetheless, it is still high compared to most African countries, and there are indications that fertility has stopped decreasing. In the 2000s, the total fertility rate has been 6.0 to 6.3, implying that women in Malawi on average gave birth to one child more than women in Africa (NSO and ORC Macro, 2005; MICS, 2007).

4. The Empirical Model of Fertility

When analysing the impact of the HIV epidemic on actual fertility we are ultimately interested in the effect on the woman's complete fertility, the total number of children she gives birth to, and possibly the timing of these births. However, the epidemic started in earnest in the mid-1980s and it is too early to study how it affects complete birth histories. We study fertility during the period 1999-2004, using the approach in Soares (2006). Soares studies child bearing for the woman up to the date of the survey, and child bearing is treated as a function of individual choice, factors not under the woman's control, and the woman's age. In addition to age we use prior births to control for the stage of the reproductive life cycle the woman is in, since we study only fertility in a limited time period. Using this approach desired fertility is allowed to depend on recent information on the HIV epidemic, the past birth history and other individual and communal characteristics. There is uncertainty since women cannot control their fertility perfectly for biological reasons, such as fecundity, and social reasons, such as the partner's attitudes.

Assume that the number of births during our study period is a function of *B*, a latent continuous variable that indicates the propensity to have a certain number of births. Moreover, $B = N + \varepsilon$, where $N = N(n(X), t, pb)$, and ε is a random term. Behaviour is determined by desired lifetime fertility, $n = n(X)$, where X includes individual and communal factors, the age of the woman, t, and the number of prior births, pb. The actual number of births during a given period for a woman at a certain age, N, is given by,

0 birth if $B \leq c_0$;

1 birth if $c_0 < B \leq c_1$;

2 birth if $c_1 < B \le c_2$;

3 birth if $c_2 < B \le c_3$;

4 birth if $c_4 < B$;

where the cut-offs, *c*, are parameters to be estimated and 4 is the maximum number of births observed during the period. If we assume that ε is normally distributed we can estimate our model as an ordered probit model. The probability that a women will not give birth to any children during the period is then

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

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

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

The values of the *c*s are estimated as parameters in the model, together with the coefficients in $N = N(n(X), t, pb)$. We assume *N* is linear, but allow for interaction between the HIV epidemic indicators and age and education, respectively, in some specifications.

6. Data and variables

The main source of data is the nationally representative Malawi Demographic and Health Survey (MDHS) carried out in 2004 where 10,058 women and 2,754 men living in rural areas were interviewed. Apart from fertility related information, data on a range of characteristics of the respondents and their households were collected. In addition, the 2004 survey has information about HIV status for a subsample, the first nationally representative survey of HIV prevalence.

The main dependent variable is realised fertility, which we measure as the number of births a woman has given during the five–year period preceding the data collection; approximately from mid-1999 to mid-2004. The time period was chosen because our preferred measure of the HIV epidemic, district-level prime-age adult mortality, is based on mortality in the twelve months period prior to the 1998 population census. Thus, households have had time to adjust behaviour to the mortality rates, and the HIV/AIDS epidemic.

We also do estimations with desired fertility as the dependent variable. This is measured by answers to a question about the ideal number of children to have if the woman or man could go back in time and choose freely. As is the case with many survey question of a more subjective nature it is not obvious that all persons understand the question equally, and in the way the researcher does. And respondents may try to give what she perceives to be the 'right' answer rather than her own true answer. For example it could be difficult ex post to say that it would be ideal to have fewer children than you already have, and some women may answer how many children they would want in an ideal state of the world while others answer the question given her, or his, current situation. Still, we believe that estimations on the ideal number of children is a good complement to estimations on realised fertility, and that it can provide information about whether impacts on realised fertility is due to changes in the desired number of children.

Our explanatory variable of interest is presence of HIV/AIDS in the community. We use two different variables to measure this, district adult mortality rates from the 1998 population census and the district HIV prevalence rate in 2004, [7] both differentiated by gender. 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. As earlier mentioned, AIDS was the by far leading cause of death in this age group, causing three out of four deaths (Doctor and Weinreb, 2003). The district-level rural HIV rates are obtained from the 2004 MDHS. In the

[7] The choice of indicator for the HIV epidemic varies across authors: Young (2005, 2007) use national HIV rates obtained from antenatal clinics; Kalemli-Ozcan (2008) 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 HIV rates in the DHS clusters. We believe that our indicators strike a balance between being sufficiently disaggregated (there are only three regions but 27 districts in Malawi) and the measurement problems from basing the indicators on too small samples. 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 (2008) we use data from antenatal clinics in Malawi to estimate HIV prevalence over time for districts with the Estimation and Projection package from WHO/UNAIDS.

survey, ten districts were oversampled to get statistically reliable estimates of HIV prevalence rates at the district level. We use these, as well as data from another 16 districts[8] since they are the best available measures of HIV prevalence in the general public. In the sample of the rural population, 3,500 women aged 15-49 and 3165 men aged 15-54 were eligible for HIV testing. The response rate was 71.2 percent for women and 64.8 percent for men: about 20 per cent of both women and men refused to take the test, and the rest were absent. The response bias seems to be small according to an analysis carried out by NSO and ORC Macro (2005, Chap 12), particularly in the rural areas.

There are three reasons that make us prefer the adult mortality rates over the HIV rates. First, deaths are directly observable to people, as opposed to people's HIV status. Second, the data should be of good quality as it comes from the population census, and has been adjusted for underreporting. Finally the adult mortality information predates the period in which realised fertility is measured and any feed-back effects from fertility in 1999-2004 to adult mortality in 1998 are highly improbable. It should, however, be noted that although the HIV rates might not be directly observable to people, Young (2007) argues that women should be able to infer the HIV prevalence from infants' deaths with AIDS symptoms, since the disease progresses rapidly in small children.[9] One potential drawback of using HIV rates in 2004 is that women might actually have become infected while getting pregnant, and this may cause a positive bias in the relationship between HIV rates and fertility. The expected time to death after infection is about 10 to 12 year, so a rough estimate is that about half of the women HIV positive in 2004 were infected before 1998 and the rest after.

To overcome the endogeneity problem, and to capture the biological consequences of the disease, we estimate models with a sub-sample of women which has information on HIV status. Hence, in some specifications we control for HIV status.

Turning to our other explanatory variables, since our dependent variable is not complete fertility but fertility within a limited time period it becomes crucial to control for the woman's age and her prior number of births. Age enters as seven dummy variables for the age groups 15-19, 20-24, up to 45-49. Age at the time of the survey is used, so in the youngest age group

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[8] We exclude the smallest district, the island of Likoma, since there are too few observations on HIV status.
[9] According to this argument, mothers should also have a good idea of their own risk of being HIV positive.

fertility is measured around age 15 (for example age 10-15 for a 15 years old and age 14-19 for a 19 years old).

Economic theory on fertility suggests full income and the opportunity cost of women’s time to be important determinant of desired fertility. Household wealth quintile and the woman’s educational level (no schooling or incomplete primary, complete primary, complete secondary, or higher education) are included to capture this. Both education and wealth information is available only from the survey year. Since less than 10 percent of the women have more than 8 years of schooling most women ought to have reached their completed level of schooling earlier than five years ago, but endogeneity is a potential problem in the youngest age group. We assume that there have been no systematic changes in relative wealth over the last five years.

Norms and social interactions are likely to be important fertility determinants. Importantly, norms governing reproductive behaviour are likely to influence sexual behaviour in general, and may thus matter for the spread of the HIV epidemic. Hence, it is crucial to control for them. We do so by including dummies for ethnic belonging and religion.

Omitted variable bias is still an issue and we therefore include a number of district level variables that could be related to both fertility and the spread of the HIV epidemic or adult mortality. Foremost, we need to control for child mortality to ensure that adult mortality do not simply pick up child mortality effects (since adult mortality and child mortality in 1998 was not correlated this is not probable, but 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 population census. We also include measures of district poverty and economic inequality from the Malawi Integrated Household Survey 1997/98, since the socioeconomic characteristics of the community may be related to both the spread of HIV and fertility. Moreover, we use two district health variables since general health status is likely to influence fecundity as well as the spread of HIV (Stillwaggon, 2006). The first one is annual 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). The second is the share of women with a Body Mass Index (BMI) below 20, calculated using the MDHS 2000. BMI below 20 has been shown be associated with significantly lower fertility rates (Nichols et al., 2001). This variable is included to control for the biological effects of a bad health and nutritional status on fertility.

Table A1 and A2 in the Appendix show summary statistics for our variables and the district variables correlation matrix is displayed in table A3.

7. Empirical Results

Before discussing the impact of HIV on fertility, we describe the effects of the other explanatory variables. To save space we only present coefficients of these variables for the first set of estimations of realised fertility (Table 1) and desired fertility (Table 3). The basic results from these estimations do not change when we use gender-specific adult mortality and HIV rates or allow for age and education heterogeneity of the fertility response to the HIV epidemic. In the realised fertility estimations (Table 1), coefficient of all the age dummies are significant at the 1 percent level, and they show how age affects fertility non-linearly, peaking at the age groups 20-24 and 25-29. Women that had given birth to more children before 1999 gave birth to fewer children 1999-2004, controlling for other variables. As usual, education is associated with a decreased fertility. To have secondary education is associated with fewer births than not to have completed primary education, the difference between women with and without complete primary education is, however, not statistically significant. The lack of significance of the higher education coefficient in Specification 1 is probably because very few women have more than secondary education. Household wealth is negatively associated with fertility, although there does not seem to be any difference in fertility between women from the poorest and second poorest households. According to the economic theories of fertility this suggests that wealthier families substitute child quantity into child quality, or that the opportunity cost of women's time is higher in wealthier households, also when we control for their educational level. An alternative explanation is that norms differ between wealthier and poorer households.

One striking feature in the desired fertility estimations is that impacts seem to differ widely by gender. Older women state they want fewer children than younger women, while older men state that they want more children than younger men. Though highly speculative, one possibility is that older women have realised that it can be exhausting with many pregnancies and children, and since they spend more time with their children they may have come to the conclusion that in a poor setting it is better to spend more of the limited resources on each child than to spread it on more children. For men the age effect could pick up a shift in norms away from traditional norms of having many children. Also, district poverty and under five mortality in Specifications (1) (2) and (4) are associated with a desire to have fewer children among women, while a worse health situation – measured by the share of underweight women and a higher malaria incidence, in Specifications (7) and (8), – are associated with a desire to have more children for men. Again it is possible that women who spend more time with the children are more affected by lower wellbeing of their children. Women and men with more living children also state a higher ideal number of children.

Our first hypothesis deals with the total effect of the HIV epidemic on fertility. We evaluate it using estimations of realised fertility in 1999-2004 (presented in Table 1) among all women, not controlling for HIV status, and also not controlling for the share of women with a BMI below 20 since weight loss is a symptom of AIDS. The first two columns of Table 1 uses the full rural sample of women, where adult mortality is used to measure the communal presence of HIV in Specification (1), and HIV rates in Specification (2). In Specifications (3) and (4), only the sub-sample of rural women with HIV status information is used, to allow for fairer comparison with the remaining four specifications where the physiological consequences of the disease are controlled for.

In Specifications (1), (3) and (4) the null hypothesis of a zero total effect of the HIV epidemic on fertility can be rejected in favour of a negative fertility impact. The results are stronger when we use adult mortality rather than HIV rates, and when we use the HIV status sub-sample rather than the full sample. The stronger effects from using adult mortality should be expected if this data is of better quality (less measurement error attenuation bias) or if HIV prevalence in 2004 is not exogenous to fertility in 1999-2004, but at least partially the outcome of the same sexual contacts that cause pregnancy.

In addition to pure chance we can think of one possible explanation for the stronger effect in the HIV status sub-sample; sample selection into the HIV status sub-sample of people that are less inclined towards denial of the disease, as have been argued, leading to a stronger behavioural response to the epidemic among those who took the test. HIV/AIDS is often surrounded by much stigmatization and someone more likely to admit the disease and that it may concern oneself is probably also more likely to change behaviour as a response to the disease.

In specifications (5) to (8) the second hypothesis, about the behavioural fertility impact of the HIV epidemic, is evaluated. Specifications (5) and (6) add the individual HIV status and district share of underweight women controls to Specifications (3) and (4), something which somewhat weakens the still negative effects of communal HIV. In Specifications (7) and (8) only the sample of women HIV negative in 2004 are used, ruling out any impacts of biological consequences of HIV on the estimated communal HIV coefficient. When adult mortality is used to measure communal HIV there is still a statistically significant negative impact on fertility, but when district HIV rates are used the coefficient loses its significance.

The magnitude of the impact of the HIV epidemic on fertility is illustrated in, Table 2 that presents the predicted number of births per woman if district prime-age adult mortality goes from its mean in 1987 to its mean in 1998 (from 3.9 to 15.8 deaths per 1000 people aged 30-49) or if the district HIV prevalence rate goes from 0 to 15 percent. Using estimation results from Specification 1 in Table 1 (the full sample, not controlling for individual HIV status or share of district's women with BMI<20, prime-age adult mortality to capture communal HIV) women are predicted to give birth to 0.05 fewer children in a five year period due to the HIV epidemic. If around 15% of women are HIV positive, HIV positive women give birth to about 25% fewer children, and women give birth to around 1 child in five years without the HIV epidemic, this decrease could almost be explained by the physiological consequences of the disease on HIV positive women (the number of births during five years should decrease with 0.0375). 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, the share of district's women with BMI<20, and if we exclude HIV positive women, but it is always larger than the predicted effect from the full sample estimations. It thus appears that fertility decrease is not only due to physiological consequences among HIV positive women. But the effect, for some reason, appears to be larger among the women that were tested for HIV than among other women. Given that this smaller sample should be less representative of women in rural Malawi than the full sample, the behavioural impact is probably overestimated when using it. In our view the most reasonable conclusion is that the behavioural impact is negative but small, probably not more important than the negative impact from physiological consequences of the disease.

Table 1: Ordered probit estimations of realised fertility among rural women in Malawi

Dependent variable is births during the last five years

	(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		-0.480		-0.991**		-0.777*		-0.390
prevalence		(0.300)		(0.484)		(0.465)		(0.491)
Under 5	-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)
Asset	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	9945	9945	2379	2379	9964	9964

Note: All estimations also include ethnicity dummies, religion dummies and four cut-offs.
Standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 2: Impact of the HIV epidemic on realised fertility

Change in predicted number of births during five years when either district adult mortality goes from the 1987 to the 1998 mean (from 3.9 to 15.8 per 1000 people age 30-49) or the district HIV rate goes from 0 to 15%.

Table 1 specif.	Sample	HIV epidemic variable	HIV status control	District share BMI<20 control	Predicted number of births: Without HIV epidemic	With HIV epidemic	Change	95% CI for Change	
(1)	Full	Adult mortality	No	No	0,924	0,974	-0,050	[-0,052,	-0,048]
(2)	Full	HIV rate	No	No	0,921	0,957	-0,037	[-0,038,	-0,035]
(3)	HIV status	Adult mortality	No	No	0,970	1,134	-0,164	[-0,177,	-0,151]
(4)	HIV status	HIV rate	No	No	0,961	1,039	-0,077	[-0,083,	-0,072]
(5)	HIV status	Adult mortality	Yes	Yes	0,987	1,128	-0,141	[-0,152,	-0,130]
(6)	HIV status	HIV rate	Yes	Yes	0,977	1,038	-0,060	[-0,065,	-0,056]
(7)	HIV negative	Adult mortality		Yes	1,012	1,107	-0,096	[-0,104,	-0,088]
(8)	HIV negative	HIV rate		Yes	1,007	1,037	-0,030	[-0,033,	-0,028]

The third hypothesis deals with the impact of communal HIV on desired fertility. Estimations of desired fertility are presented in Table 3. The first two columns present coefficient from estimations of women's ideal number of children using the full sample of women, and estimations in the next two columns use the HIV status sub-sample and control for individual HIV status. Columns (5) to (8) show coefficients from estimations of men's ideal number of children. We find a negative effect of communal adult mortality from estimations on the full sample of women and on men. The coefficient of communal HIV rates is never statistically significant. Being HIV positive is associated with a desire of having fewer children for men but not for women. Since many HIV positive people do not know about their HIV status we should not expect this necessarily to be caused by knowledge of being HIV positive. It could also be that men who wish fewer children behave differently and that this put them at higher risk of HIV infection.

Table 3: Ordered probit estimations of desired fertility among rural women and men in Malawi

Dependent variable is ideal number of children

	Women				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 prevalence		-0.471		-0.140		0.0908		0.0963
		(0.299)		(0.605)		(0.410)		(0.446)
Under 5 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)
Asset 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

Note: All estimations also include ethnicity dummies, religion dummies and four cut-offs.
Standard errors in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Predictions on ideal number of children….

7.2 Gender-differentiated effects

In Table 4 communal adult mortality and HIV rate coefficients from estimations differentiating between female and male mortality and HIV are reported together with the predicted fertility change of the HIV epidemic. The effect of female adult mortality and female HIV rates is negative and statistically significant, both in the full sample, and in the HIV status sub-sample with the HIV status control. Male adult mortality has a statistically significant negative coefficient only in the HIV status sub-sample and male HIV rates in none of the samples. Male and female adult mortality and HIV prevalence rates are of course correlated, and thus likely to get similar coefficients when entered separately. The effects of female adult mortality and HIV rates, however, seem to be stronger both in terms of statistical significance and in terms of size. In Specifications (9) to (12) female and male rates enter simultaneously, resulting in a negative statistically significant coefficient of female mortality and HIV in spite of the correlation, but a positive statistically insignificant of male mortality and HIV. Our hypothesis thus seems to be confirmed. Female adult mortality and HIV rates have a more negative impact on fertility than the male equivalents. We control for various measures related to the health situation in the district – the share of the district's women with BMI below 20, malaria incidence, and under five mortality and poverty - making it unlikely that this result is due to less healthy women giving birth to fewer children.

Table 4: Ordered probit estimations of realised fertility among rural women in Malawi – differentiating between female and male communal mortality and HIV

Dependent variable is births during the last five years

	Sample	HIV status control	Coefficients Female adult mortality	Female HIV rate	Male adult mortality	Male HIV rate	Predicted change of HIV epidemic on births per woman during 5 years
(1)	Full	No	-6.549** (3.208)				-0.0449 [-0.047, -0.043]
(2)	Full	No		-0.743** (0.343)			-0.0624 [-0.065, -0.059]
(3)	HIV status	Yes	-19.04*** (5.305)				-0.1335 [-0.144, -0.123]
(4)	HIV status	Yes		-1.114** (0.550)			-0.0943 [-0.101, -0.087]
(5)	Full	No			-3.576 (2.349)		-0.034 [-0.036, -0.033]
(6)	Full	No				-0.268 (0.230)	-0.0199 [-0.021, -0.019]
(7)	HIV status	Yes			-11.95*** (4.189)		-0.1162 [-0.125, -0.107]
(8)	HIV status	Yes				-0.403 (0.364)	-0.0309 [0.033, -0.028]
(9)	Full	No	-10.62* (5.834)		3.954 (4.139)		-0.0354 [-0.037, -0.034]
(10)	Full	No		-1.541** (0.602)		0.663 (0.403)	-0.0786 [-0.082, -0.075]
(11)	HIV status	Yes	-24.32** (9.672)		5.006 (7.507)		-0.1218 [-0.132, -0.112]
(12)	HIV status	Yes		-2.219** (1.073)		0.935 (0.726)	-0.1167 [-0.126, -0.107]

Note: All estimations include age group dummies, prior number of births, educational level dummies, household wealth quintile dummies, ethnicity dummies, religion dummies, district under five mortality, share of district's women with BMI<20, district annual malaria incidence, district poverty, district asset inequality, and four cut-offs.
Standard errors in parentheses
95% confidence intervals in brackets
*** p<0,01, ** p<0,05, * p<0,1

Turning to desired fertility, results are presented in Table 5. For women effects are statistically significant only in the full sample, and for men only when using adult mortality. But there is a more negative effect of HIV and adult mortality among people with the same sex as the respondent than of the equivalents among people of the opposite sex.

Table 5: Ordered probit estimations of desired fertility among rural women and men in Malawi – differentiating between female and male communal mortality and HIV

Dependent variable is ideal number of children

	Sample	HIV status control	Coefficients		Predicted change of HIV epidemic on ideal number of children
			Female adult mortality	Male adult mortality	
(1)	Women: Full	No	-8.126** (3.174)		-0.1233 [-0.1776, -0.0708]
(3)	Women: HIV status	Yes	-5.226 (5.816)		-0.0776 [-0.146, -0.0074]
(5)	Women: Full	No		-4.734* (2.439)	-0.0993 [-0.1505, -0.0497]
(7)	Women: HIV status	Yes		-2.588 (4.035)	-0.0536 [-0.1092, 0.0053]
(9)	Women: Full	No	-12.42** (5.951)	4.222 (4.525)	-0.0997 [-0.1505, -0.0505]
(11)	Women: HIV status	Yes	-9.590 (11.04)	4.160 (7.596)	-0.0567 [-0.1164, 0.0032]
(13)	Men: Full	No	-7.093* (4.199)		-0.0807 [-0.1292, -0.0332]
(15)	Men: HIV status	Yes	-7.119 (4.706)		-0.0781 [-0.1317, -0.0257]
(17)	Men: Full	No		-7.078** (3.281)	-0.1109 [-0.1751, -0.0479]
(19)	Men: HIV status	Yes		-7.968** (3.579)	-0.1218 [-0.1998, -0.0445]
(21)	Men: Full	No	0.612 (8.573)	-7.522 (6.683)	-0.1106 [-0.1748, -0.0479]
(23)	Men: HIV status	Yes	4.360 (9.218)	-11.15 (6.994)	-0.1233 [-0.2009, -0.0453]

Note: All estimations include age group dummies, number of living children, educational level dummies, household wealth quintile dummies, ethnicity dummies, religion dummies, district under five mortality, share of district's women with BMI<20, district annual malaria incidence, district poverty, district asset inequality, and four cut-offs.
Standard errors in parentheses
95% confidence intervals in brackets
*** p<0,01, ** p<0,05, * p<0,1

7.3 Age and education heterogeneity of the fertility response

In Tables 6 and 7 results from estimations of realised respectively desired fertility where the response to the HIV epidemic was allowed to differ by age group are reported. For realised fertility, the pattern found in Durevall and Lindskog (2009) is confirmed. Young women give birth to more children where communal adult mortality and HIV rates are higher, while older women instead give birth to fewer children in these places. Earlier we argued that the magnitudes obtained from the full sample estimations with adult mortality to measure communal HIV were probably most sensible. Women aged 15-19 then gave birth to about 0.8 more children during five years, while women aged 25-44 gave birth to over 0.1 fewer children, and women aged 45-49 0.5 fewer children. Young people do not seem to want to have more children in total in districts with higher HIV rates. For women higher HIV rates generally seem to be associated with a desire of having fewer children, while for men it is difficult to discern any pattern.

Table 6: Age-specific changes in realised fertility due to the HIV epidemic

Change in number of births during five years when either district adult mortality goes from the 1987 to the 1998 mean (from 3.9 to 15.8 per 1000 people age 30-49) or the district HIV rate goes from 0 to 15%.

Sample	Full sample	Full sample	HIV-status sub-sample	HIV-status sub-sample
Communal HIV variable	Adult mortality	HIV rate	Adult mortality	HIV rate
Age 15-19	0.079 [0.078. 0.080]	0.063 [0.062. 0.064]	0.074 [0.072. 0.077]	0.126 [0.121. 0.129]
Age 20-24	-0.007 [-0.010. -0.003]	-0.063 [-0.071. -0.057]	-0.112 [-0.131. -0.092]	-0.152 [-0.177. -0.127]
Age 25-29	-0.118 [-0.136. -0.101]	-0.048 [-0.057. -0.039]	-0.189 [-0.226. -0.151]	-0.045 [-0.054. -0.035]
Age 30-34	-0.130 [-0.141. -0.118]	-0.109 [-0.118. -0.100]	-0.266 [-0.302. -0.230]	-0.073 [-0.083. -0.063]
Age 35-39	-0.111 [-0.119. -0.104]	-0.048 [-0.051. -0.044]	-0.178 [-0.201. -0.155]	-0.106 [-0.119. -0.093]
Age 40-44	-0.122 [-0.125. -0.118]	-0.094 [-0.097. -0.091]	-0.244 [-0.258. -0.231]	-0.103 [-0.108. -0.098]
Age 45-49	-0.047 [-0.048. -0.046]	-0.010 [-0.010. -0.010]	-0.201 [-0.208. -0.194]	-0.095 [-0.099. -0.092]
HIV status control in estimation	No	No	Yes	Yes

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

Table 7: Age-specific changes in desired fertility due to the HIV epidemic

Change in ideal number of children when district adult mortality goes from the 1987 to the 1998 mean (from 3.9 to 15.8 per 1000 people age 30-49).

Sample	Women		Men	
	Full sample	HIV-status sub-sample	Full sample	HIV-status sub-sample
Age 15-19	-0.217 [-0.316. -0.118]	-0.2317 [-0.4061. -0.0584]	-0.285 [-0.399. -0.172]	-0.372 [-0.558. -0.188]
Age 20-24	-0.071 [-0.127. -0.016]	-0.0264 [-0.0891. 0.0378]	-0.016 [-0.030. 0.000]	-0.024 [-0.043. -0.006]
Age 25-29	-0.091 [-0.156. -0.027]	0.0379 [-0.038. 0.114]	0.023 [-0.006. 0.049]	-0.015 [-0.043. 0.011]

Age 30-34	-0.223 [-0.328. -0.119]	-0.1292 [-0.2724. 0.0137]	-0.255 [-0.422. -0.089]	-0.300 [-0.525. -0.077]
Age 35-39	-0.128 [-0.230. -0.024]	0.0448 [-0.0585. 0.1476]	-0.155 [-0.365. 0.056]	-0.045 [-0.195. 0.105]
Age 40-44	0.047 [-0.030. 0.124]	0.0257 [-0.0803. 0.1326]	0.187 [-0.028. 0.403]	0.302 [0.043. 0.562]
Age 45-49	-0.176 [-0.312. -0.041]	-0.4336 [-0.7916. -0.0765]	-0.252 [-0.711. 0.206]	-0.063 [-0.361. 0.236]
HIV status control in estimation	No	Yes	No	Yes

Note: 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 annual malaria incidence, district poverty, and district asset inequality.
95% confidence intervals in brackets.

In Tables 8 and 9 we allow the response in realised and desired fertility to the HIV epidemic to vary by educational groups. Starting with realised fertility we cannot confirm the pattern of positive fertility responses among less educated and negative among more educated from Fink and Linnemayr (2008). The fertility effect appear to be negative for all educational groups, and it appears to be largest among the least and the most educated and somewhat smaller among those with intermediate levels of education. For desired fertility a pattern more compatible with Fink and Linnemayr's findings is found; men and women without primary education do not want to have fewer children where adult mortality (and HIV rates) are higher, and for women the negative desired fertility response to the HIV epidemic increases with educational level.

Table 8: Education-specific changes in realised fertility due to the HIV epidemic

Change in number of births during five years when either district adult mortality goes from the 1987 to the 1998 mean (from 3.9 to 15.8 per 1000 people age 30-49) or the district HIV rate goes from 0 to 15%.

Sample	Full sample	Full sample	HIV status sub-sample	HIV status sub-sample
Communal HIV variable	Adult mort	Hiv rates	Adult mort	HIV rates
No educ	-0.124 [-0.131. -0.117]	-0.124 [-0.131. -0.117]	-0.228 [-0.251 -0.206]	-0.060 [-0.066 -0.054]
Priamry	-0.024 [-0.025. -0.023]	-0.024 [-0.025. -0.023]	-0.122 [-0.132 -0.112]	-0.064 [-0.070 -0.059]
Secondary	-0.020 [-0.021. -0.019]	-0.020 [-0.021. -0.019]	-0.067 [-0.071 -0.063]	-0.020 [-0.021 -0.018]
Higher educ	-0.157 [-0.164. -0.150]	-0.157 [-0.164. -0.150]	-0.669 [-0.727 -0.610]	-0.473 [-0.506 -0.439]
HIV status control in estimation	No	Yes	No	Yes

Note: Predictions are based on ordered probit estimations with controls for age group, prior number of births, educational level, household wealth quintile, ethnicity, religion, district under five mortality, share

of district's women with BMI<20, district annual malaria incidence, district poverty, and district asset inequality.
95% confidence intervals in brackets.

Table 9: Education-specific changes in desired fertility due to the HIV epidemic

Change in ideal number of children when district adult mortality goes from the 1987 to the 1998 mean (from 3.9 to 15.8 per 1000 people age 30-49).				
Sample	Women		Men	
	Full sample	HIV-status sub-sample	Full sample	HIV-status sub-sample
No educ	-0.048 [-0.111. 0.018]	0.057 [-0.033. 0.148]	0.019 [-0.060. 0.097]	0.048 [-0.053. 0.148]
Primary	-0.124 [-0.183. -0.066]	-0.057 [-0.125. 0.011]	-0.114 [-0.189. -0.041]	-0.154 [-0.260. -0.047]
Secondary	-0.255 [-0.366. -0.143]	-0.365 [-0.564. -0.167]	-0.162 [-0.228. -0.096]	-0.074 [-0.111. -0.037]
Higher educ	-1.202 [-4.020. 1.615]	-3.010 [-14.147. 8.126]	0.146 [0.084. 0.206]	-0.144 [-0.254. -0.033]
HIV status control in estimation	No	Yes	No	Yes

Note: 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 annual malaria incidence, district poverty, and district asset inequality.
95% confidence intervals in brackets.

8. Conclusions and discussion

In this paper we study the impact of communal HIV/AIDS on both realised and desired fertility of women in general, HIV positive and HIV negative alike, in rural Malawi. Though we also study the aggregate effect, the focus is on heterogeneity.

There is clear evidence of a negative behavioural fertility impact, when we control for individual HIV status and the share of the district's women with a BMI below 20, or even restrict the sample to knowingly HIV negative women, we find that women give birth to fewer children where district adult mortality and HIV rates are higher. Still the total (negative) fertility impact appears to be small, and the contribution of behavioural change does not appear to be larger than the contribution from the direct impact of lowered fecundity among HIV positive women.

Interestingly, we find that women have a stronger desire to have fewer children in communal areas with high female HIV and adult mortality rates than in communal areas with high male HIV and adult mortality rates. For men communal male adult mortality and HIV rates appear to be more important. Actual realised fertility is more strongly influenced by female than male communal adult mortality and HIV rates. This result is consistent with parents desiring fewer children if their own risk of dying is higher, and with women being more in control of fertility than men. In a setting where children are an important source of future social security and old-age support the marginal benefit of having a child decreases when own mortality risks increase. It could also be that women are more worried about their children being maternal than paternal orphans, while men are relatively more concerned about their children being paternal orphans. The fact that we control for a number of health related variables – district under five mortality, district poverty, district share of women with BMI <20 – makes it unlikely that the results are only due to lower fecundity among women in a worse health situation.

Moreover, it seem to be the case that women try to give birth earlier: the youngest women give birth to more children where communal adult mortality and HIV rates are higher and older women to fewer children, while woman in all age groups desire somewhat fewer children in total where adult mortality and HIV rates are higher. If women believe they might become HIV infected and die at a relatively younger age, giving birth to children earlier decreases the risk of giving birth to HIV infected babies, of leaving children orphaned, or of ending up childless. Women may also start child-bearing earlier as a consequence of marrying earlier, either because both men and women try to establish monogamous relationships earlier or because men try to marry younger wives that are less likely to be HIV positive. Another study suggests that earlier marriage may be part of the explanation.

We did not find any strong evidence of a differential fertility response to HIV/AIDS among better educated than less educated women. Though the negative impact on desired total number of children is stronger among better educated women, this did not carry through to actual fertility.

For some reason women the negative behavioural impact is much stronger among women that took the HIV test than among other women. A possible explanation is that women that agreed to do the test are less prone to deny the disease and its potential concern to them, and that this makes them respond stronger to it.

A final comment is that this study shows that the fertility response to HIV/AIDS is likely to depend on specific circumstances, in our case in Malawi up to 2004. For example the improved access to ARTs in Malawi, and other countries, decrease parents' risk of dying early, possibly implying that it is not as important to give birth early and that the future social security and old age support value of children becomes more important again.

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Appendix

Table A1: Individaul level varaible summary statistics

	Women		Men	
	Mean	Std, Err,	Mean	Std, Err,
Births last 5 years	0,971	0,012		
Ideal number of children	4,177	0,028	4,058	0,039
Age 15-19	0,201	0,005	0,204	0,010
Age 20-24	0,235	0,005	0,176	0,008
Age 25-29	0,182	0,005	0,195	0,010
Age 30-34	0,133	0,004	0,143	0,008
Age 35-39	0,098	0,003	0,091	0,007
Age 40-44	0,084	0,003	0,090	0,006
Age 45-49	0,067	0,003	0,052	0,005
Age 50-54			0,048	0,005
Total number of births 5 years ago	2,223	0,033		
Currently living children	2,600	0,027	2,359	0,059
No or incomplete priamry education	0,257	0,008	0,118	0,009
Primary education	0,642	0,008	0,675	0,012
Secondary education	0,100	0,006	0,195	0,010
Higher education	0,002	0,000	0,012	0,003
Prior births	0,204	0,007	0,146	0,010
1st wealth quintile	0,228	0,008	0,238	0,013
2nd wealth quintile	0,236	0,007	0,252	0,011
3rd wealth quitile	0,215	0,007	0,234	0,013
4th wealth quintile	0,117	0,009	0,129	0,015
5th wealth quintile	0,232	0,011	0,212	0,013
Catholic	0,172	0,009	0,180	0,013
Rel2	0,024	0,004	0,023	0,004
Rel3	0,059	0,005	0,066	0,008
Rel4	0,380	0,012	0,377	0,015
Rel5	0,120	0,011	0,105	0,011
Rel6	0,012	0,002	0,034	0,006
Rel7	0,352	0,022	0,347	0,024
Chewa	0,094	0,013	0,093	0,015
Eth2	0,176	0,014	0,178	0,015
Eth3	0,018	0,006	0,018	0,006
Eth4	0,127	0,011	0,118	0,012
Eth5	0,041	0,009	0,046	0,011
Eth6	0,010	0,004	0,012	0,006
Ngoni	0,107	0,012	0,109	0,012
Other ethnicity	0,075	0,010	0,079	0,011

Table A2: District level varaibles summary statistics

	Obs.	Mean	Std. dev.	Min	Max
cdr_3049rur	27	0,017	0,008	0,007	0,036
cdr_3049frur	27	0,014	0,007	0,006	0,031
cdr_3049mrur	27	0,019	0,010	0,007	0,041
hiv_rural	26	0,115	0,075	0,021	0,318
hiv_rurwomen	26	0,122	0,068	0,022	0,301
hiv_rurmen	26	0,108	0,091	0,000	0,336
cdr_u5yrur	27	0,161	0,043	0,053	0,238
Mala	26	0,308	0,111	0,150	0,630

lowbmi_20_d	26	0,256	0,050	0,148	0,365
pov98	25	0,656	0,108	0,421	0,840
assets	24	0,671	0,098	0,450	0,820

Table A3: District level varaibles correaltion matrix

	Adult mortality	Female ad. mort	Male ad mortality	HIV rate	Female HIV rate	Male HIV rate	Under 5 mortality	Malaria	BMI<20	Poverty
Female ad.mort.	0,977									
Male ad. mortality	0,983	0,922								
HIV rate	0,424	0,499	0,350							
Female HIV rate	0,481	0,555	0,407	0,965						
Male HIV rate	0,349	0,421	0,282	0,969	0,871					
Under 5 mortality	0,365	0,346	0,365	0,300	0,335	0,243				
Malaria	0,191	0,146	0,216	-0,232	-0,179	-0,265	0,073			
BMI<20	0,246	0,208	0,269	0,104	0,149	0,064	0,267	0,279		
Poverty	0,046	0,039	0,055	0,415	0,406	0,383	0,245	-0,241	-0,130	
Asset inequalit	0,144	0,111	0,158	-0,035	-0,110	0,049	0,327	0,041	0,133	0,058