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

Table of Contents

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Foreword

This country economic report on HIV/AIDS, adult mortality and fertility in Malawi, is part of a series of studies, undertaken by various Swedish universities and academic research institutes in collaboration with Sida. The main purpose of the studies is to enhance our knowledge and understanding of current economic and political development processes and challenges in Sweden's main partner countries for development co-operation. It is also hoped that they will have a broader academic interest and that the collaboration will serve to strengthen the Swedish academic resource base in the field of development economics.

The study investigates fertility patterns across regions of differing HIV/AIDS prevalence in Malawi. The major finding is that HIV/AIDS reduces fertility, as uninfected women both give birth to and desire to have fewer children in districts where prime-age mortality and HIV prevalence is high. However, young women (age 15 to 19) in these districts, tend to have higher fertility, possibly because they wish to have children while being uninfected. This is likely to have negative effects on both educational attainment and child mortality. The study was undertaken by Dick Durevall and Annika Lindskog at the Department of Economic at Göteborg University.

Per Ronnås
Chief Economist

Introduction

Malawi's first AIDS case was diagnosed in 1985. The epidemic then spread rapidly, first in the major cities, and more recently in rural areas. Currently Malawi has one of the highest national HIV-prevalence rates in the world, about 14% among adults. This is twice as high as the average rate in Sub-Saharan Africa, but clearly less than in the worst hit countries where prevalence rates are over 20% (UNAIDS, 2007a).

There is no doubt that HIV/AIDS has wide-ranging consequences for households affected by the disease. It is less obvious what the economic effects are at the national level. A number of studies have evaluated the nation-wide effect of HIV/AIDS by testing its impact on economic growth, but they have obtained very mixed results (see for example, Bloom and Mahal, 1997; Bell et al., 2004; Corrigan et al., 2005; Young, 2005a; McDonald and Roberts, 2006; and Werker et al., 2006). The reason for the inconclusive results is probably that HIV/AIDS affects growth through many different channels, and that the relative importance of these varies across countries and over time. It thus makes sense to consider one channel at a time.

One response to HIV/AIDS that has been discussed in the recent literature is changes in fertility. In the economics literature, the emphasis has been on its influence on the dependency ratio, i.e., the ratio between children plus elderly and adults at working age (Young, 2005a). The dependency ratio is known to exert a strong impact on economic growth (Bloom and Williamson, 1998). However, as shown by Gregson et al. (2002), changes in fertility caused by an HIV epidemic would also affect the overall impact on several demographic and socioeconomic indicators including population growth, orphanhood and early childhood mortality.

The general view is that HIV/AIDS leads to lower fertility (Ntozi, 2002; Epstein, 2004; Arrehag et al, 2007). It is mainly based on results from studies that analyse the direct impact of HIV/AIDS on individuals and households, i.e., on fertility among HIV-positive women, even though women not directly affected also might respond to the epidemic by altering their behaviour. Fertility is believed to decline because it tends to be lower among infected women for physiological reasons, while it is unchanged for uninfected women.

There are some studies that acknowledge behavioural change. Young (2005a; 2005b) claims that women desire to have fewer children because of the risk of infection. Moreover, by simulating the impact of the decrease in fertility in Sub-Saharan countries due to high prevalence rates,

he shows that the resulting improvement in the dependency ratio is so great that it far outweighs other negative economic effects of the disease. Hence, HIV/AIDS leads to higher GDP per capita in the long run.

In contrast, Kalemli-Ozcan (2002; 2006) and Lorentzen et al. (2005) argue that the increase in prime-age adult mortality associated with HIV/AIDS should raise fertility. This is because families tend to have more children when there is greater uncertainty of survival of their offspring. They also find empirical support for this hypothesis: according to Kalemli-Ozcan (2006) the HIV/AIDS-induced increase in fertility worsens the dependency ratio, which in turn reduces future per capita growth rates. Hence, as Glick (2006) notes in his review of HIV/AIDS and behaviour, the extent and the direction of the feedback effects from HIV/AIDS on reproductive choices is an important area for research.

The purpose of this study is to evaluate the impact of HIV/AIDS on fertility in Malawi. We use data from the Malawi Demographic and Health Survey (MDHS) carried out in 2004, the most recent data available on fertility. Apart from information on fertility, it has information on a range of characteristics of the respondents and their households. In addition, the 2004 survey has information about HIV-status for a subsample, and it is the first nationally representative survey of HIV prevalence.

Fertility is measured as the number of births given during the last five years, using individual data. To measure the impact of HIV/AIDS, we exploit the large geographical differences in HIV prevalence and mortality rates, using population census data from 1987 and 1998, and HIV-data from the nationally representative sample of HIV-positive men and women reported in the 2004 MDHS, and data from Antenatal Clinic (ANC) sentinel surveys since 1990. Our main focus is on how mortality and prevalence rates at the communal level affect fertility, but models of the stated ideal number of children, which is a measure of fertility preferences, are also estimated. Since our dependent variable is a count variable, we use the Poisson estimation model.

During recent years the distribution of Anti Retroviral Therapy has been scaled up rapidly, and by the end of 2006 close to 70 000 people were receiving therapy, which is a substantial share of the roughly 190 000 people in need of it (UNAIDS, 2007b). The availability of treatment is likely to affect behaviour and could thus influence fertility. However, in 2004, when our data was collected, very few Malawians had access to Anti Retroviral Therapy and it is unlikely to have any impact on our results.

Our approach resembles Young (2005b) who analyses the impact of HIV on fertility in 25 Sub-Saharan countries using Demographic and Health Survey data, but there are several differences. Five of these are worth noting here. First, we use new data from Malawi, not included in his sample. Second, we model the communal effects at a much more disaggregated level, using data from 27 Districts, while Young treats each country as one community. Third, we use past adult mortality rates, in addition to current HIV prevalence. Fourth, we do a more focussed analysis by only modelling fertility in rural areas; population in urban areas is heterogeneous and changes due to urbanisation. Fifth, we investigate the possibility that HIV/AIDS affects fertility differently in different age-groups, focusing on women 15-19 years old, since anecdotal evidence points towards increased fertility in this age group. Changes in fertility among teenagers are known to affect both school attainment and child mortality: high fertility is associated with fewer years of schooling and higher child mortality.

The main finding is that HIV/AIDS reduces fertility: women in districts where adult mortality and HIV-prevalence are high give birth to fewer children, and vice versa. Moreover, they desire to have fewer children. However, very young women, aged 15–19, who live in districts with high adult mortality and HIV-prevalence give birth to more children. These results hold for both HIV-negative and HIV-positive women, but HIV-infected women give birth to fewer children than uninfected women. This is probably due to physiological factors, though not exclusively since HIV-infected women also desire to have fewer children than other women.

It seems reasonable to assume that district prime-age mortality in 1998 is an exogenous variable, i.e., that it is not influenced by individual fertility during 1999–2004. However, the use of current HIV-status and HIV-prevalence rates creates a potential endogeneity problem, since women might contract the virus while getting pregnant. Nevertheless, this is probably not a serious weakness of the analysis because HIV prevalence is a stock variable that changes relatively slowly due to new infections, because the bias would be in the opposite direction of our main findings. If there is a simultaneity bias, it makes the estimated negative impacts smaller than the correct ones.

The report is structured as follows. The section following this introduction describes how HIV/AIDS might affect fertility, and reviews findings from previous studies. The section thereafter gives a brief background to the HIV/AIDS epidemic in Malawi, followed by a section providing details about fertility in Malawi. The empirical results are then reported in the penultimate section. The final section concludes the report.

Previous Findings on HIV/AIDS and Fertility

Broadly speaking, HIV/AIDS affects fertility through two channels: the biological one, which is due to the physiological consequences of the disease on fecundity and its subsequent effect on the population structure, and the behaviour-response channel, which includes a change in fertility preferences of both those that are HIV positive and those that are not, as well as changed sexual behaviour to avoid infection.

The biological impact 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, and less frequency of intercourse because of illness, and of premature death of regular partner (Zaba and Gregson, 1998; Fabiani, et al 2006).

Such difference in fertility between infected and uninfected women have been shown by a number of studies (see Gray et al., 1998; Zaba and Gregson, 1998; Terceira, et al., 2003; Fabiani et al., 2006).[1] HIV-positive women have a lower fertility than HIV-negative women in all age groups, possibly except for girls aged 15–19,[2] and the difference is considerable. For example, in a study of 4813 sexually active women aged 15–49 years in a rural area in Uganda, Gray et al. (1998) found that the incidence rate of pregnancy, measured per 100 woman-years, was 23.5 for HIV-positive women compared to 30.1 for HIV-negative women. The difference was particularly large for HIV-positive women who had other sexually transmitted diseases such as syphilis: among the women surveyed, 21.4% of those without HIV-infection or syphilis were pregnant, while the pregnancy rate was 14.2% among the HIV-negative women with syphilis and only 8.5% among women infected with both HIV and syphilis. Interestingly, there was no evidence that infection in male partners reduced fertility for HIV-negative women. It is also noteworthy that HIV-positive women without HIV-associated symptoms also had reduced fertility, although the fertility reduction was larger for those with symptoms.

[1] See Glick (2006) for more references showing that HIV reduces fertility among HIV infected women.

[2] The reason young HIV-positive women have higher fertility 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 (see Ntozi, 2002 and Fabiani et al., 2006).

In spite of these findings, and widespread consensus on the role of biological factors, it is possible that the findings of sub-fertility are, at least partially, due to other factors than HIV infection. Young (2005b) uses Demographic and Health Survey data for 25 African countries to test if HIV affects the probability that pregnancy ends in miscarriage, but finds no relation. He argues that the results of previous studies might be due to reverse causality: sub-fertility can lead to marital problems which increase the risk of infection. And the high correlation between HIV and other sexually transmitted diseases can confound the impact of HIV on fertility. Moreover, a study of female intravenous drug users in New York, which avoided issues related to sexual activity and relationships between married couples, failed to find a difference in pregnancy and miscarriage between HIV-positive and HIV-negative women (Selwin et al., 1989).

There is much less consensus on the role of behavioural factors and how they impact on fertility. According to Fabiani et al. (2006), behavioural responses among infected women cannot explain the decreasing trend in fertility observed in various African countries with high HIV prevalence. Avoidance of pregnancy because of the fear of leaving orphans, break-up of partnerships because of disclosure of HIV status, and increased use of contraceptives, etc, only have a minor impact on fertility (see also Gray et al., 1998; Zaba and Gregson, 1998; Zaba et al., 2003). The main reason is that few women know their HIV status, limiting the possibility that HIV-positive women decide to have fewer children.

Nevertheless, the question if HIV-positive women who are informed about their HIV status actually have fewer children is interesting, since the number of women tested is increasing in many African countries. There are studies showing that women diagnosed with HIV state that they intend to reduce fertility, but a common finding is that fertility and contraceptive use do not change much (Gray et al., 1998; Glick, 2006). In a recent study Oladapo et al. (2005) analysed 147 HIV-positive patients receiving care at a suburban clinic in Nigeria. Only 4.3% of those who desired children before finding out about their status did not intend to have any afterwards. The majority had a compelling desire for parenthood, even those who already had several children.

An implication of these findings is that the behavioural response of those who are HIV positive is unlikely to affect fertility much, even if they know their status. And, as pointed out by Morah (2007), most Malawians are not tested until they have developed AIDS. This implies that even if they would alter their behaviour and decide to not to have more children, the impact on fertility at a national level would be small.

Young (2005b) argues that HIV/AIDS reduces fertility, but that it is mainly the result of behavioural response among people in general, irrespective of whether they are HIV positive or not. The main explanation for the behavioural response is that women make risk assessments by observing AIDS-induced neonatal mortality of their own and other's children, and from adults dying in their community. And when they see the increased risk, they respond by having safe sex and reducing fertility to avoid getting infected and giving birth to HIV-positive babies. Increases both in the use of condoms and in non-viral-protective contraception in many Sub-Saharan countries provide indirect support of this view. For example, in Malawi condom use during last sexual intercourse with non-cohabiting partner increased from 38.9% to 47.1% among males and from 28.7% to 30.1% among females between 2000 and 2004 according to the MDHSs. By 2006, this percentage had increased to

59.6% and 39.6%, as reported by MICS (2007). The total use of all means of contraception among cohabiting partners has also increased, but not much. In 1992, 40.6% of all married women used any contraception, modern or traditional, while 44.9% used it in 2004 and 42% in 2006. However, these numbers hide a substantial increase in the use of modern methods, which increased from 19.1% in 1992 to 38.9% in 2006 (MDHS 1992, MDHS 2004, MICS, 2007).

Another explanation for reduced fertility, emphasised by Young (2005a), is increasing wages. According to economic theory, an increase in mortality rates for people at working age leads to more capital per worker, given savings, which should raise wages, and in cases where there is unemployment, improve job opportunities. This might reduce the number of children a household prefers to have since the opportunity costs for children increases, i.e., a woman who stays at home taking care of children looses more income (see Todaro, 2003, pp. 280-85). According to Young this mechanism is important in South Africa. But it might play a role in other parts of Sub-Saharan Africa as well, particularly in urban areas where increases in prime-age mortality are likely to improve female employment opportunities.

Glick (2006) points to yet another mechanism that might lead to lower fertility. The HIV/AIDS epidemic is turning many children into orphans; nearly 13% of the children aged 0–17 are orphans in Malawi, and as many 17% do not live with any of their biological parents (MICS, 2007). The vast majority of these are taken care of by relatives, very few are in orphanages. Families that take care of orphans, or other children who do not live with their parents, might decide to have fewer children of their own, both because of costs and because some of the children in their care might be substitutes for having more children of their own.

Even though there are good arguments and several studies pointing towards reduced fertility, there are few studies that provide empirical support to the hypothesis that HIV/AIDS causes fertility to decline at a national level in Sub-Saharan Africa. The most comprehensive study is probably Young (2005b). He finds that an increase in HIV prevalence from 0 to 100% reduces fertility by 88%, which is roughly of the same magnitude as moving from no education to tertiary education. Another study is by Terceira et al (2003) who analyze data from twelve communities in Zimbabwe. They find that HIV/AIDS explains about one quarter of the fertility decline observed since the 1980s. Moreover, in a study on Sub-Saharan Africa, Zaba and Gregson (1998) use data from case-control studies and theoretical predictions from a model of fertility and HIV incidence to obtain information about the overall impact of HIV on fertility. Their calculations show that an increase of HIV prevalence by one percentage point in the general female population reduces the total fertility rate[3] (TFR) by 0.4 percentage points, which is mainly due to lower fertility among HIV-positive women.

In spite of these arguments and findings, there is ample anecdotal evidence that HIV/AIDS increases fertility: some examples are stories about young women who state they would like to have children now, before they become HIV positive, or about grandparents and others who urge young women to have children while they are healthy. Moreover, in some countries TFRs seem to have increased or stopped decreasing recently. In Tanzania the TFR increased from 5.6 in 1999 to 5.7 in

[3] The total fertility rate (TFR) is the number of children a woman would have, who went through life and at every age gave birth to as many children as the average women in that age group currently does. It is a common measure of fertility because it is independent of the age- and sex-composition of the population, and of mortality among children and women.

2004, and in Kenya it rose from 4.7 to 4.8 between 1998 and 2003, while it remained at 6.9 in Uganda between 1995 and 2000.[4] Westoff and Cross (2006) attribute the increase in Kenya to HIV/AIDS, but do not test the hypothesis. The most recent estimates of TFR in Malawi also indicate an increase, from 6.0 in 2004 (NSO and OCR Macro, 2005) to 6.3 in 2006 (MICS 2007). However, the surveys have somewhat different coverage.

In addition, some recent studies claim that prime-age adult mortality, whether caused by AIDS or not, raises fertility. The argument is based on economic theory which predicts that families in societies without insurance systems and pensions, where the support of children is vital at old age, make decisions on fertility after considering the likelihood of the survival of their children as adults. Since HIV/AIDS increases prime-age mortality, it reduces life expectancy and makes it less likely that a child will outlive its parents. This raises the demand for children (Kalemli-Ozcan, 2002; 2006; Lorentzen et al., 2005).

Lorentzen et al. (2005) use macro data for a large number of countries over the period 1960–2000 to show that high adult mortality is positively associated with high fertility in cross-section regressions. They do not focus on HIV/AIDS explicitly but confirm that adult mortality is associated with AIDS deaths. Kalemli-Ozcan (2006) tests the association between HIV/AIDS and fertility directly on macro data, using both AIDS deaths and HIV prevalence as explanatory variables. She finds that HIV/AIDS increases fertility: a woman living in a high-HIV-prevalence country has two more children on average during here life time compared to a woman in a country with very low HIV prevalence. According to Kalemli-Ozcan, HIV/AIDS will reverse the demographic transition in several Sub-Saharan Africa countries.

There are also studies showing that HIV/AIDS has no affect on national fertility rates. Werker et al. (2006) use circumcision to identify the causal affect of HIV/AIDS on fertility, arguing that the number of circumcised males in a society can be used as an instrument for HIV prevalence to capture causality in the regression analysis. Randomised experiments have shown that circumcision reduces the risk of becoming infected with HIV, so it is treated as an exogenous factor that affects the spread of the epidemic. However, Fortson (2007) argues that circumcision is a poor instrument in a statistical sense, casting doubt on the findings of Werker et al. (2006).

[4] The data are from Measure DHS Statcompiler Database, available online http://www.statcompiler.com.

A Brief Background to the HIV/AIDS Epidemic in Malawi

In this section we describe the main characteristics of the HIV/AIDS epidemic in Malawi and the government's response to it. As this section is very concise readers are referred to Arrehag et al. (2006) for a more complete review.[5]

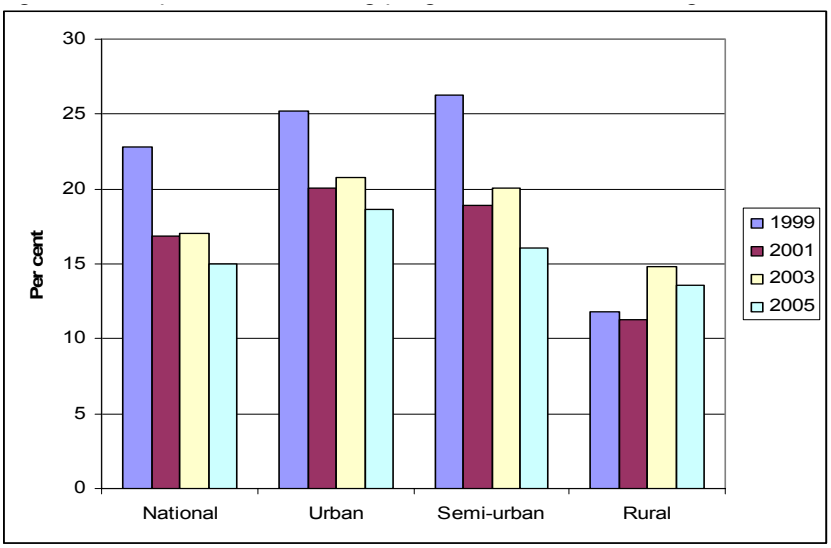
After the first AIDS case was diagnosed in 1985, HIV-prevalence rates increased dramatically and reached 30% by 1993 among women visiting ANC:s in the major cities (NAC, 2003). Currently, the national HIV-prevalence rate is estimated to be about 14% among adults. This means that approximately 940 000 Malawians are living with HIV/AIDS, out of which 57% are women, and that over 78 000 people die annually from the disease (UNAIDS, 2007a).

Since the end of the 1990s, national prevalence among women visiting ANCs has declined substantially and it is now 15%, as reported in Figure 1. The decline occurred in urban and semi-urban areas. In rural areas, where the majority of the people live, HIV-prevalence rates continued to increase from 11,8% in 1999 to 13,6% in 2005.

One of the striking features of the HIV/AIDS epidemic is its differential impact on men and women, as Figure 2 shows. HIV prevalence among women is 9 times higher than for men in the age group 15–19, and 3.4 times higher in the age group 20-24. There are several reasons for this: women become sexually active at an earlier age than men; it is common that women have relationships with older men; and women have greater sensitivity to contracting the disease.

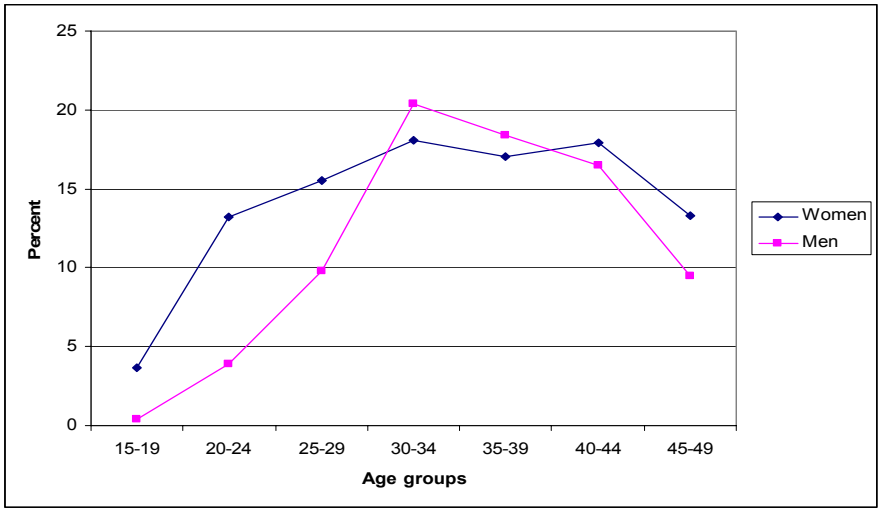
[5] This section relies on Arrehag et al. (2006) but it contains some updates and additional information.

Figure 1. HIV prevalence among pregnant women attending to ANCs



Source: Malawi Triangulation Project (GOM. 2006).

Figure 2. Percentage HIV positive among women and men age 15–49



Source: MDHS 2004 (NSO and ORC Macro, 2005).

In Malawi, as well as in other Sub-Saharan African countries, the virus is primarily spread through heterosexual contact, which accounts for about 90% of the infections (NAC, 2004). The other main channel is mother to child transmission. It is estimated that without any preventive measures approximately 30% of babies born to HIV-positive mothers are infected during pregnancy, birth, or through breastfeeding (NSO and ORC Macro, 2005).

There is no simple explanation to why HIV has spread to such a large part of the Malawian population. Many contributing factors interact and their relative importance is not known. However, one important factor is Malawi's location, i.e., its closeness to the region where HIV/AIDS seems to have started, Congo, and the fact that it is surrounded by other countries with high HIV-prevalence rates.

Another factor is the custom of having several relationships at the same time (Halperin and Epstein, 2007; SADCC, 2006). Since the virus load is very high during a month or so after infection, a newly infected person is very contagious compared to somebody who has had the disease for a couple of months. For a given number of life-time partners

the practice of having multiple partners simultaneously thus facilitates the spread of the virus compared to sequential monogamy. A recent study of Likoma Island in Lake Malawi is very suggestive of the role of multiple partners in spreading HIV. It found that 75% of the adult population were sexually interlinked (Helleringer and Kohler, 2006). In many Western countries people might have as many partners during their lifetime, but they usually have one stable partner at the time, and they tend to use condoms at casual encounters (Halperin and Epstein, 2004).

Another explanation to the high prevalence rates in Africa is the presence of untreated sexually transmitted diseases (STDs), such as syphilis or herpes. Oster (2005) shows that untreated STDs actually explain most of the difference in infection rates between the US and Sub-Saharan Africa. However, there is no study showing that STDs are a major factor in Malawi. In fact, the number of STD cases seems relatively small compared to HIV prevalence: in 2003 about 2.7% of the pregnant women visiting ANCs had syphilis (GOM, 2005).

Poverty is often blamed for causing the epidemic since it is only poor countries that have generalised epidemics.[6] However, within Sub-Saharan Africa some of the wealthiest countries have the highest prevalence (Botswana and South Africa) and some of the poorest have low prevalence rates (Niger). Moreover, in most countries prevalence is higher in high-income groups than in low-income groups (Gillespie and Greener, 2006). In Malawi, data from ANCs show that prevalence increases with level of education and with the skill level of partner's occupation (NAC, 2003), and nationally representative data show that better educated and wealthier men and women are relatively more likely to be HIV positive (NSO and OCR Macro, 2005). Nevertheless, it is obvious that poverty sometimes makes people take risks, contributing to the spread of HIV: women may sell sex for goods or money, and men may leave their families for extended periods to work far away from home. Moreover, poverty makes people more vulnerable to external shocks, such as drought, and the combined effect seems to increase risky behaviour (Bryceson and Fonseca, 2006).

Unequal income distribution is also likely to be an important driver of the epidemic. One reason is that women can only sell sex if there is a buyer with resources. Moreover, wealthy men have more partners, probably accounting for the high prevalence rates among relatively well-educated men and women. Other important factors are gender inequality and gender-based violence. For instance, female pupils are sometimes pressured to have sex with their teachers, and young girls are engaged in cross-generational relationships with so-called sugar daddies in exchange for gifts or money (Kadzamira et al., 2001; Weissman et al., 2006).

Another potential factor is cultural traditions involving sex. These practices are often based on deep-rooted associations between sex, health and illness and continue to influence sexual and reproductive health and health-seeking behaviour at least in the country-side (see Matinga and McConville, 2003; Bryceson et al., 2004; Arrehag et al., 2006).

The government's late response to the epidemic must also be considered a major factor behind the rapid spread of the virus. As evident from comparing the experiences of countries such as Uganda, Senegal and South Africa, a rapid and aggressive response seems to make a difference. Uganda has managed to reduce HIV prevalence substantially, and in Senegal it has hovered around 1% for pregnant women for the last 15

[6] A generalised epidemic is when adult HIV prevalence is at least 1% and not concentrated to specific groups according to UNAIDS.

years, which is attributed to forceful policy. In South Africa, in contrast, HIV prevalence among pregnant women in urban areas went from 0.6% in 1990 to over 28% in 2004 (Glick, 2006; UNAIDS, 2007a).

Malawi's first comprehensive HIV/AIDS policy was not formulated until 1999, the National HIV/AIDS Strategic Framework. It resulted in the establishment of the National AIDS Commission (NAC) 2001, which coordinates the multi-sectoral implementation and mainstreaming of national HIV/AIDS policies. A new HIV/AIDS strategy was launched in 2003.[7] The policy provided guidelines for all HIV/AIDS programmes in Malawi and had two main goals: (a) "to prevent the further spread of HIV infection" and (b) "to mitigate the impact of HIV/AIDS on the socio-economic status of individuals, families, communities and the nation". And in 2005, the current national HIV/AIDS strategy (National Action Framework) for 2005–2009 was formulated. It is based on the same fundamental principles as in the previous programme, but focuses on various "priority areas" of activity, which include a) the provision of an enabling environment; b) behaviour change interventions; c) mainstreaming HIV/AIDS into the public and private sectors; d) a prevention programme; e) a comprehensive HIV/AIDS care and support programme.

Recent programmes have strongly endorsed the use of condoms by ensuring that the government, through the NAC, undertakes to "ensure that affordable male and female condoms of good quality are made available to all those who need them". As a mentioned earlier, condom use appears to have increased significantly: 33% of the men used a condom when they last had sex with a non-cohabiting partner in 2000, and 59.6% used it in 2006 (GOM 2006, MICS, 2007). However, the usage of condoms is still far from satisfactory.

The government also has declared its commitment to "promote and provide high-quality, cost-effective, totally confidential, and accessible voluntary counselling and testing (VCT) services country-wide". HIV testing should also be "routinely offered to all pregnant women attending antenatal clinics unless they specifically choose to decline". Hence, the number of sites offering HIV counselling and testing grew from 14 in 2001 to 184 in 2005, but there is still a shortage of sites in the rural areas (GOM, 2005).

Anti Retroviral Therapy (ART) is provided free of charge (except in a few private clinics where a fairly low fee is charged) and is expected to be expanded considerably in the coming few years. By the year 2010, the number of patients who have ever started ART should, according to projections from the Ministry of Health, reach 245 000. The Global Fund is committed to financing the expansion up to 2008.

Free distribution of ARTs began a few years ago; in 2003 there were less than 20 sites and only a couple of percent of the eligible HIV-positive patients received ART (GOM, 2006). Since then the programme has been scaled up rapidly, and at the end of September 2006 close to 70 000 patients were receiving ART, which was about 40% of those entitled to treatment. On the other hand, prevention of mother to child transmission is lagging behind, partly because it requires more human resources, and Malawi has limited capacity in the health sector. At the end of 2005 only 6% of all HIV-infected pregnant women received treatment (UNAIDS, 2007b).

[7] Government of Malawi, Office of the President and Cabinet/National AIDS Commission, "National HIV/AIDS Policy. A Call for Renewed Action", October 2003

Fertility in Malawi

This section first describes the development of fertility and its characteristics, and then it discusses the relationship between the observed pattern of fertility and HIV/AIDS. The purpose is to give a background to the empirical analysis of how HIV/AIDS affects fertility.

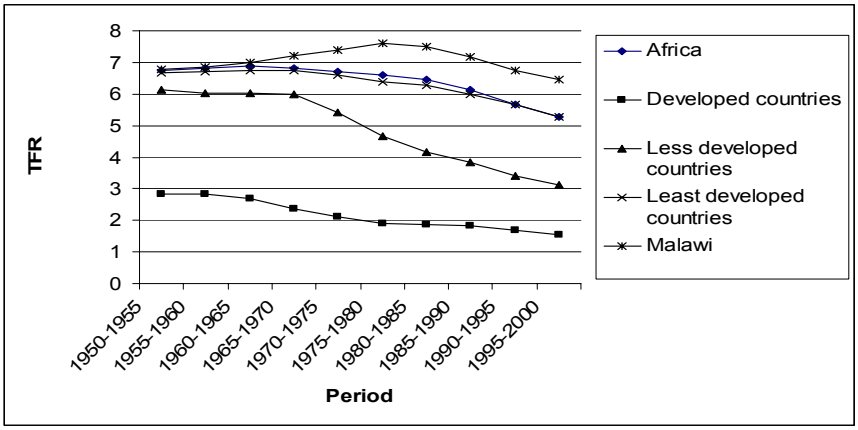
Development of Fertility

Figure 3 reports TFRs in Malawi and various regions in the world for the period 1955–2000. At the time of independence, in the early 1960s, the TFR in Malawi was similar to those in other African and developing countries. But while fertility in most other countries decreased, Malawi's TFR grew until 1980. This development was probably related to the ideology and policy of the Malawian government under President Banda. Birth control was seen as incompatible with the Malawian culture, and population growth was not recognized to be a problem. In fact, family planning was banned in the late 1960s (Chimbwete et al. 2005). In the beginning of the 1980s, the fertility trend changed and TFR started to decrease at a pace similar to that of other African and developing countries. The turning point seems to have coincided with the adoption of a child spacing program by the Banda government. Spacing, rather than limiting the number of births, was claimed to be in line with Malawian culture and the demand of Malawian women.

In 1994, when a democratically elected government got into power, the child spacing program was replaced by a family planning program with the explicit aim of reducing fertility (Chimbwete et al. 2005). Nonetheless, fertility is still very high in Malawi compared to most African countries. In 2000, the TFR was over 6.3, implying that women in Malawi on average gave birth to one child more than women in Africa. The most recent MDHS indicates that the TFR had fallen to 6.0 (MDHS, 2004). However, as already mentioned, there was a national survey in 2006 that showed a rise to 6.3 (MICS, 2007).[8]

[8] The data collected by MICS are not publicly available yet and are therefore not reported in the tables below.

Figure 3. Total fertility rates for Malawi and various regions 1950–2000

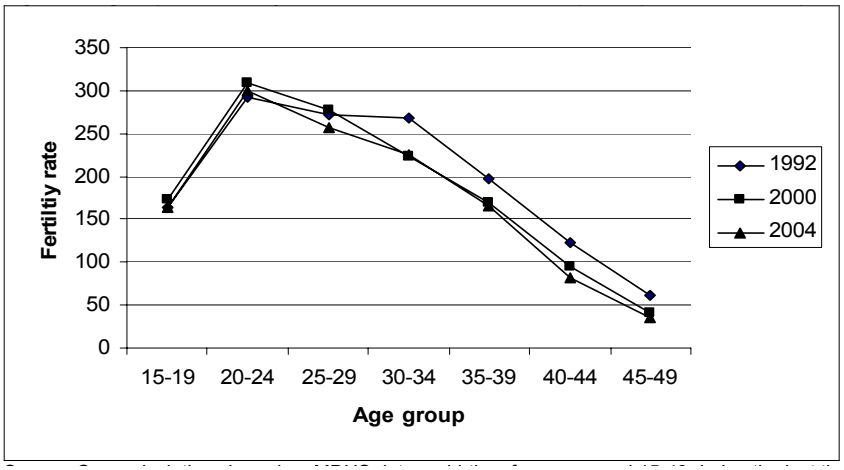


Source: Population Division of the Department of Economic and Social Affairs of the United Nations Secretariat. World Population Prospects: The 2006 Revision and World Urbanization Prospects (http://esa.un.org/unpp).

Fertility at the national level hides large variations across different groups of women, complicating the picture considerably. Data on various aspects of fertility are therefore reported in Table 1 and Figures 4. Table 1 shows data on fertility for various categories of women over the time periods 1989–1992, a period when HIV prevalence was fairly low; 1997–2000 when HIV-prevalence was still increasing; and 2001–2004, when HIV/AIDS was widespread and widely known. In the appendix, Table A1 shows more details, including age-specific fertility rates and confidence intervals, and Table A2 reports data on desired fertility, available from 1992, 2000 and 2004. It shows the percentage distribution for desiring 0–2, 3–5 and 6 or more children.

The downward TFR trend shown in Figure 3 above appears to be quite stable. However, looking at age-specific fertility rates and their changes over time, shown in Figure 4 (and Table A1), reveals that there was a large decline in the fertility among women 30 years and older during 1992–2000. It accounted for most of the downward trend, while fertility among younger women decreased only marginally. Moreover, between 2000 and 2004, a period when knowledge of HIV/AIDS was already widespread and practically everybody had personal experiences of the epidemic (MDHS, 2004), fertility only declined slightly or remained stable in all age groups.

Figure 4. Age-specific fertility rates in 1992. 2000 and 2004 (births per 1000 women)



Source: Own calculations based on MDHS data on births of women aged 15–49 during the last three years.

As Table 1 shows, there is also a substantial difference between urban and rural fertility, and it seems to be increasing. In 2004 the urban TFR was 4.3 compared to the rural one at 6.5. Since the majority of the population lives in rural areas, the development of rural fertility has a strong impact on TFR at a national level. During the period 1992–2000, fertility in urban areas decreased quite dramatically, while the decrease in 2000–2004 was modest.

Table 1. Total fertility rates for sub-groups of women in Malawi

	1989–1992	1997–2000	2001–2004
Total	6.9	6.4	6.1
Urban	5.6	4.5	4.3
Rural	7.1	6.8	6.5
No education	7.3	7.4	7.0
Primary education	6.6	6.4	6.3
Secondary education	4.4	3.1	3.9
Poorest quintile	7.4	7.4	7.3
Second poorest quintile	7.3	7.0	7.1
Middle quintile	7.1	6.4	6.6
Second richest quintile	6.5	6.1	5.9
Richest quintile	6.1	5.3	4.2
Northern region	6.9	6.3	5.7
Central region	7.6	6.9	6.5
Southern region	6.3	6.1	5.9

Source: Own calculations based on DHS data on births during the last 36 months for women in the age 15 to 49.

However, it should be recognised that urbanization has changed the proportion of Malawians living in rural and urban areas. According to estimations using the MDHS surveys, 12% were living in urban areas in 1992, while 18% were doing so in 2004. This change should also have contributed to the decline in fertility that has occurred at the national level since when rural migrants become urban dwellers they usually reduce fertility.

Education and income also matter a great deal for fertility. Table 1 reports that for women with no education, fertility remained stable in 1992–2000; but, as reported in Table A1, it increased for young women with no education and decreased for older women with no education. However, in 2000–2004 fertility of women with no education decreased substantially in all but the youngest age group.

When comparing fertility over time in different educational groups it must also be remembered that education has increased substantially over the period. The MDHS surveys indicate that the share of women with no education fell from 47% in 1992 to 23% in 2004, while the share with primary education increased from 48% to 62%, and the share with secondary education increased from only 4% to 15%. As for urbanisation, increased education has most likely reduced fertility in Malawi. Moreover, women that belong to a certain educational group in 2004 probably differ in many important ways from women that belonged to this same group in 1992.

Table 1 also reports five different income groups, depending on the wealth of the household to which they belong.[9] There appears to be little difference in fertility between women from the two poorest wealth quintiles. It is noteworthy that TFR is over 7 in both groups and that it hardly has declined since the beginning of the 1990s. Otherwise the relationship between household wealth and fertility seems to be negative; women from wealthier households give birth to fewer children. Especially women from the richest quintiles stand out both in terms of the low level of the TFR and in terms of the large fertility decline.

There are also substantial differences in fertility across the three Regions, as reported in Table 1. Northern region has the lowest TFR, 5.7; Central region has the highest, 6.5; while TFR is 5.9 in Southern region. The decline in fertility has been smallest in the Southern region. These differences are of course partly due to differences in urbanisation, income and education, but there are many other factors that vary across the Regions.

To sum up, the decline in fertility in Malawi between 1992 and 2004 is concentrated to the period 1992–2000, to older women, to women in urban areas, and to women in the wealthiest households.

HIV/AIDS and Fertility

TFR and desired fertility seem to have decreased for almost every subgroup, even though the decrease has been very modest in some cases, and fertility actually has increased among young women in some groups. The fact that fertility declined rapidly in urban areas in 1992–2000, while the decline in rural areas was faster during 2000–2004, is consistent with the hypothesis that the HIV/AIDS epidemic reduces fertility. This hypothesis is also consistent with the fact that fertility declines have been concentrated among richer people and people in urban areas.

The finding that fertility declines have been concentrated to older women and that fertility might have increased among young women in the rural areas is noteworthy. If young women in the country-side are more eager to have children this could be related to behavioural changes due to the HIV/AIDS epidemic. They may decide to marry and establish supposedly monogamous relationships early to reduce the number of partners they and their husbands have before marriage. For a given number of desired children, they might also wish to have many children early to avoid the risk of infecting the child or turning ill and die in AIDS while having very young children.

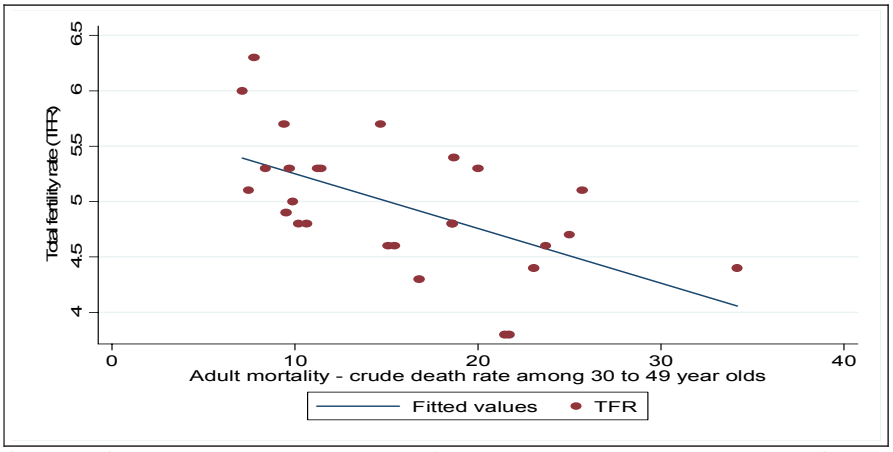
If the HIV/AIDS epidemic changes the desired number of children, one might expect fertility effects to be concentrated among older women who already have children. And this feature is evident in the data: fertility decreases are concentrated to older women. And if there are physiological reasons to why HIV/AIDS decreases fertility, these might be larger among older women among whom the disease is likely to have reached a more mature state.

Adult mortality has increased substantially due to HIV/AIDS, and is estimated to be the cause of three out of four deaths among adults (Doctor and Weinreb, 2003). The regional variation in adult mortality is thus likely to capture the regional variation in HIV/AIDS fairly well. Figure 5 depicts the relationship between the district level TFRs in 2004 and the number of deaths per 1000 persons in the age group 30–49 in 1998. There is a clear negative relationship. This could be taken to

[9] There is no data on total wealth, instead different indicators of wealth are used to create an index, and households are divided into five quintiles depending on their relative ranking according to this index. See in the variables section later.

suggest that the HIV/AIDS epidemic decreases fertility. However, other factors might be associated with both higher adult mortality and lower TFRs. For example, HIV/AIDS seems to, at least initially, have hit relatively well-educated and wealthy people living in urban areas more than less educated poor in the country-side (NSO and OCR Macro, 2005). And we saw above that better educated, wealthier women living in urban areas have fewer children than others. It is thus important to control for these factors when analysing how HIV/AIDS affects fertility.

Figure 5. District level fertility versus district level adult mortality in1998



Source: Own calculations using data from the Population and Housing Census 1998. NSO

Econometric Analysis

From the previous section we know that there is a negative association between adult mortality and fertility at the district level. However, this association might not imply a causal relationship, since other factors might determine both variables. It is therefore necessary to use multivariate regression analysis to investigate if there is any relationship between district level adult mortality, or alternatively HIV-prevalence, and fertility. In this section we first describe the different data sources and motivate the choice of variables. Then the results are reported.

Data and Variables

The main source of data is the MDHS 2004. The DHS-project was developed to collect nationally representative data on fertility and child- and maternal health, comparable across developing countries, but data on individual and household characteristics are also collected. Using the MDHS data we can look at fertility at the level of the individual women, controlling for individual level education, wealth, type of residence (urban or rural) and other potentially important factors. Additionally, the MDHS data contains information on both realised and desired fertility. The MDHS 2004 was collected at a time when individuals should have adjusted fertility preferences and sexual behaviour to the reality of HIV/AIDS and increased risk of prime-age death. Moreover, HIV tests were carried out on a sub-sample of respondents in the MDHS 2004, giving us valuable information about their HIV-status. Altogether 11698 women aged 15 to 49 years were interviewed in the survey and for 5357 women there is HIV-status information.

The dependent variables are realised or desired fertility. Realised fertility is the number of births the woman has had during the last five years; approximately from mid 1999 to mid 2004. Hence our fertility measure covers a five-year period starting just after the period when the district-level adult mortality was measured, i.e., in 1998 population census. Desired fertility is the number of children a woman says that she would have liked to have if she had the opportunity to choose freely.

Our most important explanatory variable is exposure to the HIV/AIDS epidemic. We are interested in potential effects on fertility in the general public, i.e. not only among HIV-positive women or women living in directly affected households. For this purpose we want to have a variable measuring the regional variation in assessed risks of HIV-infection and premature death. Among women in general, fertility could

be affected either if fertility preferences change or if sexual behaviour change, as one tries to decrease the risk of HIV-infection. These adjustments to the HIV-epidemic should depend on assessed risks for oneself, others in the family, and for future children. These may differ from actual risks in the community, but they should be related.

One potential indicator of perceived general risk of HIV-infection, and/or of premature death is district-level prime-age adult mortality. As opposed to HIV-prevalence, prime-age adult mortality is observable to people, and we have reliable data on district-level adult mortality from the population and housing censuses. The last two censuses were undertaken in 1987, when AIDS mortality was uncommon, and in 1998, when adult mortality was heavily influenced by AIDS. Using data from both these years we can compare the effect of district-level adult mortality before and after it increased due to the AIDS epidemic. The specific measure of adult mortality that we use in our analysis is the number of deaths per thousand individuals aged 30 to 49 years, a group in which AIDS was leading cause of death, in 1998.

In spite of HIV-prevalence rates being unobservable to people, we use estimates of them as an alternative measure in addition to prime-age mortality. We have data on current HIV-prevalence in the general male and female population from the MDHS 2004. If women did know about HIV-prevalence rates and infections, current HIV-prevalence is clearly the most obvious variable to use. Young (2005b) argues that women should possess this information as the disease progresses rapidly for small children and they could infer it from infants' deaths in AIDS symptoms.[10] Even though women cannot observe HIV-prevalence, current rates might still be informative about AIDS deaths and illness insofar as they are correlated with past prevalence rates.

In the survey, a sub-sample of respondents was asked for a blood sample to make a HIV-test. In ten over-sampled districts, enough tests were done to get statistically reliable prevalence rates at the district level. However, we use data from Malawi's other districts also to obtain more observations and more district-level variation.[11] These data are probably the best estimates of HIV-prevalence in the general public available, even for the districts that were not over-sampled in the MDHS.

In addition to the data on current HIV-prevalence, we have data on HIV-prevalence among pregnant women from the early 1990s for most districts. The advantage of these data is that they should provide information about prevalence rates of AIDS-related morbidity and mortality over the period 1999–2004. The disadvantage is the representativeness of the data: pregnant women may not be representative of the general population, and the samples were not chosen even to be representative of pregnant women.[12] To make figures more stable and relevant at the district level, the average HIV-prevalence rates was calculated using all studies that have been performed within a district during the 5-year period 1990–94.[13]

[10] According to this argument, mothers should also have a good idea of their own risk of being HIV-positive.

[11] The exception is the smallest district, the island Likoma, from which there were only 8 observations on HIV-status.

[12] For example, as ANCs are more common in urban and semi-urban areas the large group of women living in rural areas could be underrepresented.

[13] There are two different types of the human immunodeficiency virus, called HIV-1 and HIV-2. In Malawi and neighbouring countries HIV-1 is totally dominating, while HIV-2 is more common in West African countries. We therefore excluded one study that measured only HIV-2 (and found a prevalence rate on zero), and treated studies that measured both types or only HIV-1 as equivalent.

Table 2 reports descriptive statistics for prime-age adult mortality and the HIV-prevalence measures. Adult mortality was about four times larger in 1998 than in 1987, which mainly is due to the HIV/AIDS epidemic. This is consistent with Doctor and Weinreb (2003) who, using verbal autopsy, found that 75% of the deaths among adults are HIV/AIDS related. The relatively large standard deviations and the difference between the maximum and minimum values indicate substantial variation across districts in adult mortality. There are also large gender differences in mortality, the rates being clearly higher for males than females.

HIV-prevalence is higher among women than among men, as in most Sub-Saharan countries, and the standard deviations are large for both groups. The table also reports the change over time in HIV-prevalence among pregnant women. The prevalence increased during the 1990s, peaked around year 2000, and then decreased somewhat.

Table 2. Adult mortality and HIV-prevalence in districts of Malawi.

Variable	Obs	Mean	Std. Dev.	Median	Min	Max
Adult mortality 1998 (‰)	27	15.8	7.0	15.1	7.1	34.2
Female adult mortality 1998 (‰)	27	13.9	6.6	13.0	5.8	30.3
Male adult mortality 1998 (‰)	27	17.5	7.7	17.2	8.4	38.1
Adult mortality 1987 (‰)	24	3.9	1.4	3.9	2.1	7.6
Female adult mortality 1987 (‰)	24	3.6	1.4	3.4	1.2	8.0
Male adult mortality 1987 (‰)	24	4.4	1.8	4.0	1.9	8.6
HIV-prevalence 2004 (%)	26	12.7	7.2	12.4	2.1	33.4
Female HIV-prevalence 2004 (%)	26	13.4	6.8	14.3	2.6	31.2
Male HIV prevalence 2004 (%)	26	11.9	8.9	9.7	0.0	35.6
HIV prevalence among pregnant women 1990–94 (%)	19	14.8	7.1	15.4	2.7	27.6
HIV prevalence among pregnant women 1994–98 (%)	18	16.1	7.6	16.6	5.1	32.8
HIV prevalence among pregnant women 1998–2002 (%)	18	18.6	8.8	20.7	5.4	35.5
HIV prevalence among pregnant women 2000–04 (%)	18	17.4	8.3	15.9	5.9	34.9

Sources: Mortality data from NSO. 1987 and 1998 Population and Housing Census. HIV-prevalence in general public from MDHS 2004. HIV-prevalence data among pregnant women are from the U.S. Census Bureau HIV/AIDS Surveillance Data Base.

Table 3 reports correlations between district-level adult mortality and HIV-prevalence measures. The variation in adult mortality in 1998 across districts is positively correlated both with the variation in adult mortality across districts in 1987, and with the variation of HIV prevalence rates across districts in 2004, the correlations are 0.59 and 0.47, respectively. The correlation between adult mortality 1998 and HIV-prevalence among pregnant women 1990–1994 is positive but low, 0.36, and not significant. However, the variation in adult mortality in 1987 is clearly not correlated with HIV prevalence rates. This indicates that the change in mortality is due to HIV/AIDS.

Table 3. Correlation matrix – adult mortality and HIV prevalence

	Adult mortality 1998	Adult mortality 1987	HIV-prevalence 2004
Adult mortality 1987	0.588		
	(0.003)		
HIV-prevalence 2004	0.471	0.171	
	(0.015)	(0.424)	
HIV-prevalence pregnant women 1990–1994	0.357	0.041	0.643
	(0.134)	(0.868)	(0.003)

Note: p-values in parenthesis

If district level adult mortality or HIV-prevalence rates determine fertility in the econometric analysis, this could be due to women directly affected by the HIV/AIDS epidemic: in districts with high adult mortality and high HIV-prevalence rates, there are of course more women that are HIV positive. To distinguish between fertility effects among the general public and among directly affected women, we control for those individuals that are HIV positive, and evaluate whether any fertility effects from district level adult mortality or HIV-prevalence rates remain. Additional information is obtained by comparing if realised or desired fertility is different among infected and non-infected women.

Another group of women that could be considered directly affected by the disease are those that live in households hosting AIDS-orphans. It is quite possible that they decide to have fewer children of their own. In the MDHS there is information about the orphanhood status of every child in interviewed households. Hence, the number of orphans in the household is used to evaluate if it affects fertility among women, and to investigate whether a possible fertility effect from district-level adult mortality remains when including this control. However, there is a potentially important endogeneity problem: couples who have few children of their own might be more willing to host orphans.

As already described, changes in fertility in 1992–2004 were not uniform across age-groups. In particular, fertility declines have been concentrated to older women and fertility has even increased among young women in rural areas. This suggests that the HIV/AIDS epidemic could affect fertility differently in different age-groups. If women desire to have fewer children in total, it is also reasonable that fertility declines should be larger among older women who already have children. To test for this possibility we split the sample to evaluate whether the effect of HIV is different for the youngest age-group, 15–19.

The advantage of using individual level data is that we can control for various individual characteristics. Age is entered non-linearly into the model as the relationship between age and fertility should be increasing first and then decreasing. Education is measured as years of schooling. There is no information on income in MDHS. Instead the economic status variable, reported in MDHS, is used. It is calculated by first computing a wealth index based on a range of different wealth indicators. Households are then ranked and classified into a wealth quintile. In addition to these variables, the number of children the women had given birth to five years ago is also included in all estimations.

The Malawian population consist of people from a wide range of different ethnic groups, the two largest being Chewa and Yao. Different ethnic groups have different norms, traditions and cultural practises that

might affect fertility. Hence, we control for ethnicity of the women in the regressions.

Fertility is also likely to be influenced by other communal characteristics which could vary geographically (see Matinga and McConville, 2003; Munthali et al., 2006). The ethnicity dummies should capture some of this variation in prevailing norms. However, other characteristics might be influenced by poverty and income levels at the district level. Moreover, Malawi consists of three regions, and norms might also differ between these. To give an example, in the Northern region the patrilineal system is dominant, while the matrilineal system, and combinations of the two are common in the other regions, something that may impact on fertility, as for example found by Kalipeni (1997). Hence, regional dummies, district poverty rates and district median per capita consumption are included in some regressions.

Since our dependent variables are count variables, we use the Poisson estimation model. The Poisson estimation model assumes that the conditional variance of the dependent variable is equal to the conditional mean. If this assumption is not true estimated standard errors will be incorrect. The most common case is when the variance is larger than the mean implying that estimated standard errors are too small, i.e., there is over-dispersion. We tested our model for over-dispersion but instead found a small degree of under-dispersion. As under-dispersion leads to too large standard errors, the risk of committing a type I error[14] is smaller than in the ideal case. We therefore proceed with the Poisson estimation model.

Results

We first estimated the model of fertility separately for women residing in urban and rural areas, since fertility might be governed by different factors in the two groups. It turned out that there was no association between fertility and district adult mortality in urban areas. This could be due to the fact that 80% of the Malawians live in rural areas, and that district-level adult mortality thus is dominated by rural mortality. Hence, below we only report the results from the analysis of rural areas, the ones for urban areas can be obtained from the authors upon request.

Table 4 reports the results from our basic estimations of realised fertility for 1999–2004 among rural women. Most of the control variables are clearly significant. Age affects fertility non-linearly as expected, but the effect from already having more children is not statistically significant, probably because it is closely related to the women's age. As expected, more educated women give birth to fewer children. Household wealth also impacts on fertility as predicted, although there does not seem to be any difference between women from average-wealth households and those from poor households, but women in the two richest quintiles give birth to fewer children than the others.

The coefficient on adult mortality is negative and clearly significant, showing that women in districts with high adult mortality give birth to fewer children. An increase in district adult mortality from the average in 1987 to the average in 1998 is associated with about two percent larger probability that a woman has no children, and 0.017 reduced probability of having two or more children. This might sound as a small effect, but it is considerably larger than that of increasing education from the average in 1992 to the average in 2004. However, from this we

[14] A type I error is committed when the null hypothesis is rejected even though it is true.

cannot say that adult mortality is more important than education in determining fertility, since the increase in adult mortality has been quite dramatic (it has quadrupled), while the increase in education has been relatively modest. The effect is comparable in size to that of a woman having four years instead of zero years of education.

Table 5 reports estimations with various control variables. Model 1 is the basic model expanded with ethnicity dummies. The dummies only have a minor effect on the coefficient of district adult mortality. In Model 2 control variables capturing other sources of variation across space are added. These are regional dummies, district level poverty rates and median consumption per capita. The inclusion of the variables reduces the statistical significance of the coefficient on district adult mortality (p=0.089). This is probably due to multicollinearity. However, district adult mortality is the only variable capturing geographical variation that has a reasonably low p-value, while the other variables are clearly insignificant.

Table 4. Poisson regression results
Dependent variable: Number of births given during the past five years

	Coefficient	**Effect of a discrete change in the explanatory variable on the probability of zero, one, or two or more births**[1]			
		Change	No births	One birth	Two or more births
Age in years	0.401*** (0.012)				
Age in years squared	-0.007*** (0.000)				
Births 5 years ago	0.007 (0.007)				
Education in years	-0.016*** (0.003)	Mean 1992 to mean 2004	0.012	-0.002	-0.010
Household in poorest quintile	0.019 (0.023)	Middle to poorest quintile	-0.007	0.001	0.006
Household in second poorest quintile	0.021 (0.024)	Middle to second poorest quintile	-0.008	0.001	0.007
Household in second richest quintile	-0.084*** (0.026)	Middle to second richest quintile	0.030	-0.006	-0.024
Household in richest quintile	-0.268*** (0.038)	Middle to richest quintile	0.095	-0.023	-0.072
District adult mortality	**-0.005*** (0.002)**	**Mean 1987 to mean 1998**	**0.020**	**-0.003**	**-0.017**
Constant	Yes				
Observations	10058				

*Note: Linearized standard errors taking into account the sample design are reported in parenthesis. *** indicates statistical significance at the 1% level. ** indicates statistical significance at the 5% level. and * indicates statistical significance at the 10% level*

[1] Effects of discrete changes are calculated holding all other variables constant at their mean values. For the dummy variables household wealth quintile this is not realistic but for ease of exposition we prefer this to reporting a larger number of different effects for different values on these variables

In Model 3 HIV status and the number of orphans in the household are included.[15] It is likely, though not certain, that HIV-infected women have lower fertility. Moreover, it is also probable that women who take care of many orphans want to have fewer children of their own. If there is an independent effect from increased risk of HIV-infection and premature deaths in society in the general population, then district adult mortality should have an impact on fertility also when controlling for these two variables. It turns out that HIV-positive women have substantially lower fertility than other women, a result also obtained by Gray et al. (1998). Moreover, women in households that host orphans give birth to fewer children than other women.

Table 5. Number of births given during the past five years

	Model 1	**Model 2**	**Model 3**
Age in years	0.401*** (0.012)	0.401*** (0.012)	0.386*** (0.019)
Age in years squared	-0.007*** (0.000)	-0.007*** (0.000)	-0.007*** (0.000)
Births 5 years ago	0.007 (0.007)	0.007 (0.007)	0.005 (0.015)
Education in years	-0.016*** (0.003)	-0.016*** (0.003)	-0.012** (0.006)
Household in poorest quintile	0.020 (0.023)	0.020 (0.023)	0.091** (0.045)
Household in second poorest quintile	0.023 (0.024)	0.022 (0.024)	0.033 (0.050)
Household in second richest quintile	-0.088*** (0.026)	-0.091*** (0.026)	-0.065 (0.050)
Household in richest quintile	-0.270*** (0.038)	-0.279*** (0.038)	-0.276*** (0.082)
District Adult mortality	**-0.005** (0.002)**	**-0.004 * (0.003)**	**-0.011*** (0.004)**
District poverty rate		-0.004 (0.005)	0.000 (0.007)
District median expenditure per capita		0.000 (0.000)	0.000 (0.000)
Northern region		0.025 (0.040)	-0.018 (0.081)
Southern region		-0.025 (0.041)	0.031 (0.070)
Orphans in household			-0.067** (0.026)
HIV-status			-0.322*** (0.060)
Constant	Yes	Yes	Yes
Ethnicity dummies	Yes	Yes	Yes
Observations	10057	10035	2482

Note: Linearized standard errors taking into account the sample design, in parenthesis.
**** indicates statistical significance at the 1% level. ** indicates statistical significance at the 5% level. and * indicates statistical significance at the 10% level*

[15] Initially we entered maternal orphans, paternal orphans and double orphans separately, but since the estimated effects were very similar both in size and statistical significance we only report results for the aggregate.

District adult mortality has an impact even when we are controlling for HIV status and orphans.[16] However, the effects of different wealth quintiles change, making the relationship between economic status and fertility clearer. It should be noted that splitting the sample into HIV-positive and HIV-negative women does not alter the results; there is a statistically significant effect of district adult mortality in both samples (the results of these estimations are not reported, but are available upon request).

Table 6 shows the effects of discrete changes in the explanatory variables on the probability of having given birth to none, one, or two or more children in the last five years in the model with all controls. The estimated effects of an increase in district adult mortality from its 1987 mean value to its 1998 mean value are to increase the probability of having no children by 4.9%, and decrease the probability of having two or more children by 4.4%. These effects are similar to having two orphans in the household, and falls in-between the effects of having completed primary school and of having completed secondary school. There is also a substantial difference between HIV-positive and HIV-negative women. HIV-positive women have more than 11% higher probability than HIV-negative women of not having given birth to any children in the past five years, and -8.6% probability of having two or more children.

Next we investigate the relationship between communal prevalence rates and fertility by considering alternative variables to district adult mortality in 1998. Moreover, we differentiate between mortality and HIV-prevalence of men and women. The variables included in addition to mortality in 1998 are prime-age adult mortality in 1987, which should not be affected by AIDS, district HIV-prevalence rates in 2004, and HIV-prevalence rates among pregnant women visiting antenatal clinics in the beginning of the 1990s.

Table 7 shows that for adult mortality in 1998, the effect on fertility is somewhat stronger for female mortality than male mortality (model 1 and 2).[17] This is not due to women dying before having completed their reproductive life, since we study the fertility of women who are alive.

There is also a negative and statistically significant relationship between district adult mortality in 1987 and fertility in 1999–2004 (model 3), but only for male adult mortality (model 4 and 5). The coefficient is only significant at the 10% level, but it is puzzling. A possible explanation is that the spread of the HIV-epidemic is mainly due to men, and that male behaviour is influenced by adult mortality, i.e., high mortality makes people myopic and care less about the future, as suggested by Lorentzen (2005).[18] This explanation is supported by the fact that the correlation between male district adult mortality in 1987 and district HIV prevalence rates in 2004 is 0.34, almost significant at the 10% level, while the correlation coefficient between district female adult mortality in 1987 and district HIV-prevalence in 2004 is only 0.02 (not reported).

[16] It is actually strengthened, but this appears to be mainly due to a stronger effect in the smaller sample of women with HIV-status information.

[17] The coefficient of female district-HIV prevalence in 2004 is also stronger than that of male district HIV prevalence. The results of these estimations are available from the authors upon request.

[18] An alternative explanation is that high adult male mortality is related to working outside the village, for instance in commercial farms, where the risk of becoming HIV positive is greater than in the village.

Table 6. Effect of a discrete change in the explanatory variable on the probability of zero, one, or two or more births [1]

	Change	No births	One birth	Two or more births
Education in years	Mean 1992 to mean 2004 (+2.09 years)	0.009	-0.001	-0.008
Education in years	No school to completed secondary (+12 years)	0.051	-0.009	-0.042
Education in years	No school to completed primary (+8 years)	0.034	-0.005	-0.029
Household in poorest quintile	Middle to poorest quintile	-0.033	0.004	0.029
Household in second poorest quintile	Middle to second poorest quintile	-0.012	0.002	0.011
Household in second richest quintile	Middle to second richest quintile	0.024	-0.004	-0.020
Household in richest quintile	Middle to richest quintile	0.098	-0.022	-0.076
District adult mortality	**Mean 1987 to mean 1998 (+11.9 persons per 1000 surviving)**	**0.049**	**-0.005**	**-0.044**
Northern region	Central to Northern region	0.007	0.001	-0.008
Southern region	Central to Southern region	-0.011	0.002	0.009
Chewa	Other to Chewa	-0.060	0.008	0.052
Tumbuka	Other to Tumbuka	-0.072	0.006	0.066
Lomwe	Other to Lomwe	-0.043	0.005	0.038
Tonga	Other to Tonga	-0.114	0.002	0.112
Yao	Other to Yao	-0.061	0.006	0.055
Sena	Other to Sena	0.009	-0.002	-0.007
Nkonde	Other to Nkonde	-0.022	0.003	0.019
Ngoni	Other to Ngoni	-0.041	0.005	0.035
Orphans in household	Zero to two orphans	0.049	-0.009	-0.040
HIV-status	HIV-negative to HIV-positive	0.114	-0.028	-0.086

Note: Additional variables in the model are age, age squared, births given five years ago, district poverty rate, district median expenditure per capita and a constant.

[1] Effects of discrete changes are calculated holding all other variables constant at their mean values. For the dummy variables household wealth quintile this is not realistic, but for ease of exposition we prefer this to reporting a larger number of different effects for different values on these variables.

Table 7. Poisson regression results

Dependent variable: Number of births given during the past five years

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Age in years	0.386***	0.387***	0.387***	0.388***	0.386***	0.391***	0.408***
	(0.019)	(0.019)	(0.019)	(0.019)	(0.019)	(0.020)	(0.021)
Age in years squared	-0.007***	-0.007***	-0.007***	-0.007***	-0.007***	-0.007***	-0.007***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Births 5 years ago	0.006	0.004	0.004	0.004	0.004	0.005	0.005
	(0.014)	(0.015)	(0.015)	(0.015)	(0.015)	(0.014)	(0.017)
Education in years	-0.011*	-0.012**	-0.013**	-0.013**	-0.013**	-0.014**	-0.014**
	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.007)
Household in poorest quintile	0.088**	0.092**	0.097**	0.095**	0.097**	0.80*	0.094**
	(0.045)	(0.045)	(0.045)	(0.045)	(0.045)	(0.045)	(0.052)
Household in second poorest quintile	0.033	0.034	0.037	0.036	0.037	0.024	0.037
	(0.051)	(0.050)	(0.050)	(0.050)	(0.050)	(0.052)	(0.059)
Household in second richest quintile	-0.066	-0.067	-0.071	-0.071	-0.071	-0.069	-0.052
	(0.050)	(0.050)	(0.050)	(0.050)	(0.050)	(0.050)	(0.055)
Household in richest quintile	-0.276***	-0.278***	-0.285***	-0.283***	-0.286***	-0.294***	-0.293***
	(0.081)	(0.082)	(0.082)	(0.082)	(0.082)	(0.082)	(0.097)
District female adult mortality 1998	-0.014***						
	(0.004)						
District male adult mortality 1998		-0.008**					
		(0.004)					
District adult mortality 1987			-0.036*				
			(0.020)				
District female adult mortality 1987				-0.022			
				(0.023)			
District male adult mortality 1987					-0.032**		
					(0.013)		
District HIV-prevalence 2004						-0.590*	
						(0.323)	
District HIV-prevalence pregnant 1990–94							-0.004
							(0.004)
Orphans in household	-0.066**	-0.068***	-0.069***	-0.070***	-0.069***	-0.064**	-0.064**
	(0.026)	(0.027)	(0.027)	(0.026)	(0.027)	(0.026)	(0.032)
HIV-status	-0.318***	-0.326***	-0.327***	-0.329***	-0.324***	-0.321***	-0.373***
	(0.060)	(0.060)	(0.060)	(0.060)	(0.060)	(0.061)	(0.072)
Constant	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Ethnicity dummies	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Additional regional variation controls[1]	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	2482	2482	2482	2482	2482	2451	1934

Note: Linearized standard errors taking into account the sample design, in parenthesis.

[1] Regional dummies, district poverty rate and district median per capita consumption..

**** indicates statistical significance at the 1% level. ** indicates statistical significance at the 5% level. and * indicates statistical significance at the 10% level*

Table 8. Poisson regression results
Dependent variable: Ideal number of children

	Model 1	**Model 2**	**Model 3**	**Model 4**
Age in years	0.027*** (0.003)	0.027*** (0.003)	0.027*** (0.003)	0.024*** (0.006)
Age in years squared	0.000*** (0.000)	0.000*** (0.000)	0.000*** (0.000)	0.000*** (0.000)
Children currently alive	0.042*** (0.003)	0.042*** (0.003)	0.041*** (0.003)	0.041*** (0.005)
Education in years	-0.008*** (0.001)	-0.008*** (0.001)	-0.008*** (0.001)	-0.010*** (0.002)
Household in poorest quintile	-0.010 (0.010)	-0.009 (0.010)	-0.007 (0.010)	0.016 (0.018)
Household in second poorest quintile	0.001 (0.011)	0.002 (0.011)	0.003 (0.011)	-0.024 (0.020)
Household in second richest quintile	-0.025** (0.011)	-0.028** (0.011)	-0.029*** (0.011)	-0.017 (0.018)
Household in richest quintile	-0.078*** (0.014)	-0.080*** (0.014)	-0.077*** (0.014)	-0.063*** (0.024)
District Adult mortality	**-0.002*** (0.001)**	**-0.003*** (0.001)**	**-0.004*** (0.001)**	**-0.003** (0.002)**
District poverty rate			0.002 (0.002)	-0.002 (0.003)
District median expenditure per capita			0.000 (0.000)	0.000 (0.000)
Northern region			0.085*** (0.019)	0.055 (0.37)
Southern region			0.005 (0.019)	-0.004 (0.032)
Orphans in household				0.001 (0.007)
HIV-status				-0.034* (0.020)
Constant	Yes	Yes	Yes	Yes
Ethnicity dummies	Yes	Yes	Yes	Yes
Observations	9688	9687	9665	2399

Table 9. Poisson regression results
Dependent variable: Ideal number of children

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Age in years	0.024***	0.024***	0.024***			0.024***	
	(0.006)	(0.006)	(0.006)			(0.006)	
Age in years squared							
Births 5 years ago	0.041***	0.041***	0.041***	0.041***	0.041***		
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)		
Education in years							
Household in poorest quintile	0.015	0.017	0.016	0.016	0.016	0.016	0.019
	(0.018)	0.018)	(0.018)	(0.018)	(0.017)	(0.018)	(0.020)
Household in second poorest quintile	-0.024	-0.024	-0.024	-0.024	-0.024	-0.024	-0.009
	(0.020)	(0.020)	(0.020)	(0.020)	(0.020)	(0.020)	(0.021)
Household in second richest quintile	-0.017	-0.017	-0.018	-0.019	-0.019	-0.014	-0.014
	(0.018)	(0.018)	(0.018)	(0.018)	(0.018)	(0.018)	(0.021)
Household in richest quintile							-0.053**
							(0.027)
District female adult mortality 1998	-0.004**						
	(0.002)						
District male adult mortality 1998		-0.002					
		(0.001)					
District adult mortality 1987			0.007				
			(0.007)				
District female adult mortality 1987				0.006			
				(0.007)			
District male adult mortality 1987					0.005		
					(0.005)		
District HIV-prevalence 2004						-0.282**	
						(0.133)	
Orphans in household	0.001	0.007	-0.003	-0.000	-0.000	0.004	-0.004
	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.009)
HIV-status	-0.033*	-0.035*	-0.036*	-0.040**	-0.040**	-0.032*	-0.031
	(0.020)	(0.020)	(0.020)	(0.020)	(0.020)	(0.020)	(0.022)
Constant	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Ethnicity dummies	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Additional regional variation controls[1]	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	2399	2399	2407	2407	2407	2369	1866

[1] *Regional dummies, district infant mortality, district poverty rate and district median per capita consumption.*
Linearized standard errors taking into account the sample design are reported in parenthesis.
**** indicates statistical significance at the 1% level. ** indicates statistical significance at the 5% level. and * indicates statistical significance at the 10% level.*

The coefficient for district HIV-prevalence in 2004 is negative and significant, as expected. This is not surprising since it was shown to be correlated with 1998 adult mortality (see Table 3). HIV-prevalence rates among pregnant women in the first half of the 1990s are not significant, although the coefficient is negative as expected. The lack of significance might be due to the concentration of ANCs in urban areas, making the estimates less relevant for rural areas.

The estimations with the stated ideal number of children (desired fertility) as the dependent variable are reported in Table 8.[19] The same explanatory variables are used as in the previous regressions with the exception that the variable births given five years ago is replaced with the number of children currently alive.

When using only the explanatory variables from the basic model, there is a clear negative relationship between district adult mortality and desired number of children. This model is very similar to the one in Young (2005b), who uses DHS data from several Sub-Saharan countries, but not from MDHS 2004. And the results are also similar: women living in districts with higher adult mortality want to have fewer children (Model 1). Adding ethnicity dummies, the set of district level variation controls, and HIV-status and orphans only have minor effects (see Models 2–4).

Table 10. Poisson estimation of realised fertility for teenagers and others
Dependent variable: Number of births given during the past five years

	Sample of 15-19 years old		**Sample of 20-49 years old**	
	Model 1	**Model 2**	**Model 3**	**Model 4**
Age in years	6.876*** (1.261)	6.908*** (1.302)	0.172*** (0.012)	0.173*** (0.013)
Age in years squared	-0.177*** (0.036)	-0.178*** (0.037)	-0.003*** (0.000)	-0.004*** (0.000)
Births 5 years ago	0.117 (0.316)	0.154 (0.328)	0.044*** (0.007)	0.044*** (0.007)
Education in years	-0.097*** (0.014)	-0.100*** (0.014)	-0.016*** (0.003)	-0.016*** (0.003)
Household in poorest quintile	0.215* (0.119)	0.153 (0.117)	-0.002 (0.023)	-0.002 (0.023)
Household in second poorest quintile	0.304** (0.119)	0.252** (0.115)	-0.004 (0.024)	0.007 (0.024)
Household in second richest quintile	0.029 (0.135)	-0.079 (0.131)	-0.067*** (0.025)	-0.068*** (0.026)
Household in richest quintile	-0.145 (0.164)	-0.178 (0.165)	-0.204*** (0.039)	-0.219*** (0.039)
District Adult mortality	**0.028*** (0.009)**		**-0.007*** (0.002)**	
District HIV-prevalence 2004		**1.476** (0.731)**		**-0.451*** (0.186)**
Constant	Yes	Yes	Yes	Yes
Ethnicity dummies	Yes	Yes	Yes	Yes
Observation	2028	2003	8029	7908

**** indicates statistical significance at the 1% level. ** indicates statistical significance at the 5% level. and * indicates statistical significance at the 10% level.*
A constant is included in all estimations.

Although there is a negative relationship between HIV-status and desired number of children, this relationship is much weaker than that with realised fertility, suggesting that the lower fertility among HIV-infected is partially due to physiological reasons and partially due to behavioural reasons. Women living in households with orphans do not express a

[19] For the variable ideal number of children we do not have complete information. The exact number of children the woman wants is recorded for up to five children. When the woman wants to have six or more children this is recorded as six children.

desire to have fewer children than other women. As we do not know the extent to which orphans are included among the desired number of children this result is difficult to interpret.

Results from regressions with the alternative communal measures of HIV/AIDS are presented in Table 9. Interestingly, only 1998 female adult mortality reduces the desire to have children, male mortality is clearly insignificant (Models 1, 2 and 3). As expected, there is no association whatsoever between adult mortality in 1987, neither for men nor for women, and the expressed desired fertility in 2004 (Model 4 and 5). The negative association between male non-AIDS adult mortality and realised fertility is accordingly not accompanied by a similar relationship with desired fertility. Not surprisingly, the coefficient for district HIV-prevalence rates in 2004 is negative and statistically significant (Model 6).

Last we investigate the possibility that the spread of the HIV/AIDS epidemic is affecting fertility differently, depending on the age group. As seen in Table 10, the youngest women, aged 15–19, have more children when district prime-age adult mortality and HIV-prevalence are high, while other women, including the 20–24 age-group, have fewer children. The higher fertility among young women is, however, not accompanied by a desire to have more children in total, as shown by using ideal number of children as dependent variable (results available upon request). It thus seems as if though young women are more eager to have their children early before they become HIV positive.

Conclusions

The purpose of this study was to analyse the impact of HIV/AIDS on fertility in Malawi. Data on fertility, and most other variables, were taken from the Malawi Demographic and Health Surveys carried out in 2004. The impact of HIV/AIDS was estimated using the district-level variation in prime-age adult mortality rates and HIV prevalence, obtained from population censuses in 1987 and 1998, the 2004 Demographic and Health Survey and Antenatal Clinic sentinel surveys. Fertility was measured as the number of births given during the five years, 1999–2004. Estimations were also carried out with the stated ideal number of children, a measure of fertility preferences. Since our dependent variables are count variables, we used the Poisson estimation model.

The main finding is that HIV/AIDS reduces fertility in general. One way of illustrating the size of the impact is to calculate the decline in the total fertility rate due to the increase in prime-age mortality between 1987 and 1998: it appears to have reduced total fertility rate by roughly by 0.5 children. This decrease appears to be due to its impact both on behaviour and on health. In districts where adult mortality and HIV-prevalence is high, both uninfected and infected women give birth to fewer children, and they desire to have fewer children. HIV-infected women give birth to fewer children than non-infected women. They also desire to have fewer children than other women, but the effect is not as strong as that of HIV-infection on actual births. This suggests that HIV-infected women have fewer children both because of physiological reasons and because of changed fertility preferences.

The preferred number of children is negatively associated with district HIV-prevalence rates and with adult mortality in 1998, but not with adult mortality in 1987. This shows that there is a negative effect of the spread of HIV/AIDS on the preferred number of children, and that part of the negative relationship between realised fertility and district HIV-prevalence rates and adult mortality is attributable to a change in fertility preferences because of the HIV/AIDS epidemic.

One important result is that the HIV/AIDS epidemic has different effects on the fertility of young women, 15–19 years old, and other women. Young women give birth to more children in districts where adult mortality and HIV-prevalence is high. However, this is not accompanied by a desire to have more children in total. These results suggest that young women feel that they should have children early because of the epidemic, probably to avoid giving birth to HIV-positive babies. An

increasing teenage fertility is likely to have negative effects on several socioeconomic indicators, including schooling and child mortality.

Our results stand in sharp contrast to those obtained by Lorentzen et al. (2005) and Kalemli-Ozcan (2006), who find that fertility rates increase with adult mortality. In fact, we find the opposite, adult mortality reduces fertility. The reasons for why the results differ merit further exploration. Nevertheless, our findings are in line with those found by Young (2005b), although he only uses HIV prevalence and does not address the issue of adult mortality.

According to Young (2005a; 2005b) HIV/AIDS improves the dependency ratio substantially, contributing strongly to economic growth in per capita terms. However, our estimates indicate that the decline in fertility so far is small, about 0.5 children. It thus seems unlikely that HIV/AIDS will reduce Malawi's dependency ratio much since it also increases prime–age mortality. Nevertheless, a detailed analysis of demographic interactions overtime is required to evaluate the evolution of the dependency ratio in the medium to long run, i.e., over the next 20 to 40 years.

We also find an interesting gender difference in the impact of adult mortality. Female mortality in 1998 is clearly more important for fertility 1999–2004, than male mortality. However, male adult mortality in 1987, but not female mortality, has a negative effect on fertility 1999–2004. This could be because high male mortality has led to high HIV prevalence in 2004, and thus lower fertility. High adult mortality could increase the risk of infection, possibly because of its effect on behaviour, as suggested by Lorentzen et al (2005), or because high mortality simply is associated with higher HIV-prevalence due to, for example, men working far away from their villages (as fishermen, farm labourers, etc.).

Another finding is that HIV/AIDS seems to reduce fertility by causing many children to become orphans, since households that have orphans tend to have lower fertility. However, there is an obvious problem with the estimate of orphans, since couples with few children might be more willing to host orphans than others.

This study is a first attempt to evaluate the impact of HIV/AIDS and fertility in Malawi, and there are several ways in which it can be improved or complemented. The most obvious next step would be to exploit the longitudinal information available in the Demographic and Health Surveys to ascertain the causal effects. However, preliminary results show that the basic results are the same when discrete hierarchical panel data models are estimated. Nonetheless, it could be used to evaluate teenage fertility in more detail. Another way to check the results is using data from the most recent survey, MICS 2006, when it becomes available. It contains many more observations than MDHS 2004, but lack information about HIV status. Yet another improvement would be to use adult mortality rates for Traditional Authorities (TAs), the administrative level under districts in Malawi. There are over 80 TAs in Malawi, allowing for more variation in the data. Moreover, a study of a series of cases would also improve our understanding of how fertility decisions are taken. It is also desirable to evaluate the impact on fertility of the recent scale-up of the distribution of Anti Retroviral Therapy (ART). However currently there are no available data.

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Appendix

Table A1. Fertility rates for subgroups of Malawian women (Births per 1000 women in age group and the total fertility rate)

	1989–1992			1997–2000			2001–2004		
Total	**Coeff.**	**[95% Conf, Interval]**		**Co-eff.**	**[95% Conf, Interval]**		**Coeff.**	**[95% Conf, Interval]**	
Age 15–19	164	148	180	174	165	183	164	154	174
Age 20–24	292	274	311	309	298	320	300	288	311
Age 25–29	272	251	293	277	265	289	258	245	270
Age 30–34	267	245	289	223	209	237	226	211	241
Age 35–39	197	178	217	168	155	182	165	149	181
Age 40–44	123	100	147	95	82	109	82	70	94
Age 45–49	62	38	86	42	32	51	35	25	45
TFR	6,9	6,6	7,2	6,4	6,3	6,6	6,1	5,9	6,3
Urban									
Age 15–19	135	107	163	134	116	151	109	88	131
Age 20–24	272	242	302	245	222	268	243	206	280
Age 25–29	244	216	272	227	186	268	196	172	221
Age 30–34	215	172	259	147	110	184	161	128	193
Age 35–39	156	114	198	104	73	136	99	64	135
Age 40–44	84	45	122	51	19	84	29	7	51
Age 45–49	12	-10	34	1	-1	3	22	-3	46
TFR	5,6	5,1	6,1	4,5	4,1	5,0	4,3	3,8	4,8
Rural									
Age 15–19	168	150	186	182	172	192	177	167	187
Age 20–24	296	275	318	324	313	336	315	304	326
Age 25–29	277	253	300	286	274	298	270	257	284
Age 30–34	275	251	299	236	222	250	237	221	253
Age 35–39	202	181	223	178	163	192	177	160	193
Age 40–44	127	101	153	102	87	116	90	77	103
Age 45–49	66	41	92	46	35	57	37	26	48
TFR	7,1	6,7	7,4	6,8	6,6	6,9	6,5	6,3	6,7

No education									
Age 15–19	217	188	245	243	211	275	245	211	279
Age 20–24	285	255	315	321	302	341	315	288	342
Age 25–29	264	234	293	305	286	324	274	250	298
Age 30–34	256	227	286	236	216	255	251	228	275
Age 35–39	230	201	260	203	182	224	171	148	193
Age 40–44	143	109	177	117	97	137	96	76	115
Age 45–49	73	44	103	54	39	69	43	28	58
TFR	7,3	6,9	7,7	7,4	7,1	7,7	7,0	6,6	7,3
Primary education									
Age 15–19	145	126	164	184	174	195	184	173	195
Age 20–24	313	288	337	332	319	344	319	306	332
Age 25–29	285	258	312	273	257	288	264	248	280
Age 30–34	287	249	325	231	211	250	220	201	238
Age 35–39	156	129	184	155	136	173	169	148	190
Age 40–44	96	69	122	84	65	103	76	60	93
Age 45–49	32	-3	67	31	19	43	29	15	43
TFR	6,6	6,2	6,9	6,4	6,2	6,6	6,3	6,1	6,5

Table A1 continued

Secondary education									
Age 15–19	53	19	88	83	67	99	77	64	91
Age 20–24	185	134	236	208	185	230	235	211	259
Age 25–29	225	157	293	195	161	228	198	168	227
Age 30–34	255	168	342	92	55	129	155	116	194
Age 35–39	107	47	167	43	15	72	89	53	125
Age 40–44	57	-28	142	0	0	0	23	-4	50
Age 45–49	0	0	0	0	0	0	0	0	0
TFR	4,4	3,6	5,3	3,1	2,8	3,4	3,9	3,5	4,2
Poorest quintile									
Age 15–19	148	113	183	187	167	208	190	170	211
Age 20–24	310	269	351	333	308	357	324	298	349
Age 25–29	275	233	316	297	273	321	308	272	343
Age 30–34	264	219	309	256	224	288	263	228	299
Age 35–39	236	186	286	209	182	236	179	147	211
Age 40–44	156	104	209	129	99	159	125	87	163
Age 45–49	83	40	126	61	37	86	63	37	89
TFR	7,4	6,7	8,0	7,4	7,0	7,7	7,3	6,9	7,7
Second poorest quintile									
Age 15–19	173	143	204	171	151	191	209	187	231
Age 20–24	316	273	358	319	295	342	328	305	351
Age 25–29	287	241	333	312	287	337	286	259	313
Age 30–34	296	244	349	260	233	288	253	217	290
Age 35–39	228	178	278	178	148	207	202	170	234
Age 40–44	119	61	176	117	83	152	93	64	122
Age 45–49	43	-1	88	40	18	61	43	16	70
TFR	7,3	6,7	7,9	7,0	6,7	7,3	7,1	6,7	7,4
Middle quintile									
Age 15–19	199	163	234	179	157	201	191	171	212
Age 20–24	311	273	350	319	294	344	324	303	345

Age 25–29	291	248	335	282	256	307	261	237	286
Age 30–34	232	189	275	241	214	269	249	221	277
Age 35–39	169	123	214	160	131	188	181	151	212
Age 40–44	136	88	183	69	40	97	85	59	111
Age 45–49	74	14	134	39	20	58	18	4	32
TFR	7,1	6,4	7,7	6,4	6,1	6,8	6,6	6,3	6,9
Second richest quintile									
Age 15–19	164	135	194	174	156	192	155	135	174
Age 20–24	284	247	322	323	304	342	295	273	317
Age 25–29	253	207	299	262	238	286	249	228	271
Age 30–34	278	220	337	195	160	230	211	184	238
Age 35–39	178	130	227	149	118	181	161	126	195
Age 40–44	91	52	129	77	48	105	70	48	93
Age 45–49	61	11	111	34	14	54	35	13	57
TFR	6,5	6,0	7,1	6,1	5,7	6,4	5,9	5,6	6,2

Table A1 continued

Richest quintile									
Age 15–19	133	102	164	161	142	181	93	76	109
Age 20–24	245	214	277	266	244	287	239	212	266
Age 25–29	262	224	300	238	211	264	197	175	219
Age 30–34	275	233	317	163	138	188	162	137	187
Age 35–39	178	136	221	138	110	167	105	78	132
Age 40–44	108	65	151	65	38	93	38	19	58
Age 45–49	22	-3	47	20	3	38	6	-3	15
TFR	6,1	5,6	6,6	5,3	4,8	5,7	4,2	3,9	4,5
Nortern region									
Age 15–19	132	110	154	164	142	186	168	144	191
Age 20–24	294	269	319	326	299	353	304	271	336
Age 25–29	311	280	341	300	270	329	251	222	281
Age 30–34	280	249	312	236	199	273	185	154	217
Age 35–39	204	168	240	157	125	188	163	117	209
Age 40–44	108	75	142	75	29	121	60	35	85
Age 45–49	41	7	76	12	-3	27	6	-5	17
TFR	6,9	6,5	7,2	6,3	5,9	6,8	5,7	5,3	6,1
Central region									
Age 15–19	148	125	171	164	148	179	142	126	158
Age 20–24	316	286	345	318	300	336	315	296	335
Age 25–29	304	270	338	301	283	319	275	253	297
Age 30–34	275	241	309	259	236	282	244	216	271
Age 35–39	228	196	260	183	160	206	185	156	213
Age 40–44	150	113	187	103	81	126	99	76	123
Age 45–49	97	50	144	54	34	74	49	30	68
TFR	7,6	7,1	8,1	6,9	6,6	7,2	6,5	6,2	6,9
Southern region									
Age 15–19	186	161	211	183	171	196	182	168	196
Age 20–24	273	244	302	298	283	314	285	270	300
Age 25–29	238	207	268	248	232	265	244	228	261
Age 30–34	258	224	293	190	172	207	221	202	240

Age 35–39	173	147	199	159	139	178	149	130	167
Age 40–44	107	72	143	94	76	112	75	59	90
Age 45–49	35	12	57	38	27	50	30	17	43
TFR	6,3	5,9	6,8	6,1	5,8	6,3	5,9	5,7	6,2

Source: Own calculations based on DHS data on births of women aged 15–49 during the last three years.

Table A2. Ideal number of children for subgroups of women in Malawi (Percentage wanting 0-2. 3-5. and 6 or more children)

	Ideal number of children	1992	2000	2004
Total	0–2	8.4	16.3	17.8
	3–5	58.6	65.4	65.9
	6+	33.0	18.4	16.3
Urban	0–2	10.9	27.3	33.0
	3–5	66.3	63.0	58.3
	6+	22.8	9.8	8.6
Rural	0–2	8.0	14.1	14.5
	3–5	57.5	65.9	67.6
	6+	34.5	20.0	18.0
Northern region	0–2	7.4	12.0	14.2
	3–5	55.5	62.5	66.4
	6+	37.1	25.5	19.4
Central region	0–2	9.8	15.1	17.6
	3–5	60.4	67.7	67.8
	6+	29.8	17.2	14.6
Southern region	0–2	7.6	18.2	18.9
	3–5	58.0	64.2	64.2
	6+	34.3	17.6	16.9
No education	0–2	7.9	9.5	9.2
	3–5	52.7	60.7	60.6
	6+	39.4	29.7	30.1
Primary education	0–2	8.1	15.7	16.1
	3–5	63.0	68.3	69.3
	6+	28.8	16.0	14.6
Secondary education	0–2	13.1	34.5	34.8
	3–5	72.0	60.8	61.8
	6+	14.9	4.7	3.4
Poorest quintile	0–2	10.4	12.8	13.6
	3–5	53.4	65.4	65.7
	6+	36.2	21.8	20.6
Second poorest quintile	0–2	8.8	13.0	11.3
	3–5	56.3	64.4	69.1
	6+	34.9	22.5	19.6
Middle quintile	0–2	6.9	13.9	13.2
	3–5	60.4	66.1	68.3
	6+	32.7	20.0	18.5

Second richest quintile	0–2	6.3	15.1	16.9
	3–5	60.4	67.4	67.3
	6+	33.3	17.6	15.9
Richest quintile	0–2	10.0	25.5	31.3
	3–5	61.9	63.8	60.1
	6+	28.1	10.7	8.6

Source: Own calculations using DHS data on women aged 15 to 49.

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