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

by

Dick Durevall and Annika Lindskog

Department of Economics,
School of Business, Economics
and Law, Göteborg University

June 2007

Sida Country Economic Report

Revised Preliminary version

Abstract: This paper analyses the impact of HIV/AIDS on fertility in Malawi. The future course of fertility will have an impact on both macroeconomic variables, such as GDP per capita, and various socioeconomic factors such as child mortality; mother-to-child-transmission of HIV, the number of orphans; and public expenditures on schooling. Data on both prime-age mortality and HIV prevalence rates at districts level are used to measure the impact of HIV/AIDS, exploiting the large time and geographical variation in the distribution of HIV/AIDS in Malawi. Fertility is estimated at the micro level of individual women, and fertility is measured as the number of births during the last five years. Estimations are also done of the stated ideal number of children, a closer measure of fertility preferences. The major finding is that HIV/AIDS reduces fertility. HIV-infected women give birth to fewer children, probably due to physiological reasons as well as changed fertility preferences. Moreover, in districts where adult mortality and HIV-prevalence is high, un-infected women give birth to fewer children, and they want to have fewer children.

1. 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 in Sub-Saharan Africa, but clearly less than in the worst hit countries where the prevalence rates are over 20% (UNAIDS Fact Sheet, 2007).

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. There are a number of studies that evaluate the nation-wide effect of HIV/AIDS by testing its impact on economic growth, but they get very mixed results (see 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. As shown by Gregson et al (2002), a change in fertility caused by an HIV epidemic would affect the overall impact of the epidemic on key demographic indicators including population growth, orphanhood and early childhood mortality. In the economics literature, the influence on the dependency ratio has been emphasised, since the dependency ratio is known to exert a strong impact on economic growth (Bloom and Williamson, 1998).

The general view is that HIV/AIDS leads to lower fertility (see Ntozi, 2002; Epstein, 2004; Arrehag et al, 2007). It is mainly based on studies that analyse the direct impact of HIV/AIDS on individuals and households, i.e., fertility among HIV positive women, even though women not directly affected might respond to the epidemic by altering their behaviour. Hence, fertility declines mainly because it tends to be lower for infected than for uninfected women. Net fertility[1] also delinces because increasing mortality rates among women reduces the total number of children they have in their lifetime; and because about one-third of the children born to infected

[1] The net fertility rate is the number of children a women gets that are subject to current age-specific mortality rates and age-specific mortality rates throughout the curse of her life, while the total fertility rate is the number of children a women gets that are subject only to current age-specific fertility rates.

mothers are themselves infected and unlikely to reach childbearing age. Some also acknowledge behaviour change, such as increased use condoms and the practice of safer sex.

By simulating the impact of a decrease in fertility in Sub-Saharan countries with high prevalence rates, Young (2005a; 2005b) shows that the resulting improvement in the dependency ratio far outweighs other negative economic effects of the disease. In contrast, Lorentzen et al. (2005) and Kalemli-Ozcan (2006) argue that the increase in prime-age adult mortality associated with HIV/AIDS raises 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. And 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 future 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 number of births, 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, 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 from the 2004 MDHS. To measure the impact of HIV/AIDS, we exploit the large geographical and time differences in HIV prevalence and mortality rates, using population census data from 1987 and 1998, and HIV-data from Antenatal Clinic (ANC) sentinel surveys since 1990. Moreover, we use the nationally representative sample of HIV positive men and women reported in the 2004 MDHS. Our main focus is on mortality and prevalence rates at the communal level affects fertility, but models with the stated ideal number of children are also estimated, which is measure of fertility preferences. 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 behavior 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 is similar to Young (2005b) who analyses the impact of HIV on fertility in 25 Sub-Saharan countries using Demographic and Health Survey data, but there are important differences. First, we use new data from Malawi, not included in his sample. Second, we model the communal effects at a much more disaggregate level, using data from the 27 Districts, while Young treats each country as one community. Third, we do a more focussed analysis by only modelling fertility in rural areas; the population in urban areas are heterogeneous. Fourth, by using information on HIV-status we can easily investigate whether fertility effects of HIV/AIDS are concentrated to HIV-infected women or if they can be found in the general public. Fifth, we investigate the possibility that HIV/AIDS affects fertility differently in different age-groups.

The main finding is that HIV/AIDS reduces fertility. Un-infected women in districts where adult mortality and HIV-prevalence are high give birth to fewer children, and vice versa, and they desire to have fewer children. Very young women, aged 15-19, however give birth to more children where adult mortality and HIV-prevalence is higher. HIV-infected women also give birth to fewer children than other women, which is in line with other studies. This is probably partially due to physiological changes, but not entirely as HIV-infected women also wants to have fewer children than other women.

The report is structured as follows. Section 2 describes how HIV/AIDS might affect fertility and reviews findings from previous studies. Section 3 gives a brief background to the HIV/AIDS epidemic in Malawi and Section 4 provides details about fertility in Malawi. The results are reported in Section 5. Section 6 concludes the report.

2. HIV/AIDS and fertility: channels and previous findings

Broadly speaking, HIV/AIDS affects fertility through two channels: the biological impact, which is due to the physiological consequences of the disease on fecundity and its subsequent effect on the population structure, and the behavioural response, which might include a change in fertility preferences of both those that are HIV positive and those that are not infected, 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. Several studies show a significant difference between infected and uninfected women (see Gray et al, 1998; Zaba and Gregson, 1998; Terceira, et al., 2003; Fabiani et al., 2006).[2] HIV-positive women have a lower fertility than HIV-negative women in all age groups, possibly except for girls aged 15-19[3], 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: the pregnancy rate was 21.4% among uninfected women, 14.2% among HIV-negative women without syphilis and 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.

The reduced fertility associated with HIV infection is attributed to a range of factors, but their relative importance is not known. 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 premature death of regular partner (Zaba and Gregson, 1998; 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 several African countries to test if HIV affects the probability that pregnancy results in live births, 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 increases 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

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

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

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 African countries with high 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 contraceptive methods, etc, only have a minor impact on fertility (see also Gray et al., 1998; Zaba and Gregson 1998; Ross et al. 1999; 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 that 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. Moreover, in a study on Malawian women, Thornton (2007) found little change in the purchases of condoms among women after their HIV-positive status had been revealed to them.

An implication of these findings is that the behavioural response of those that are HIV positive is unlikely to affect fertility much. Moreover, most Malawians are not tested until they have developed AIDS (Morah, 2007), so even if they would alter their behaviour and decide to stop having children, this would probably not have much impact on total fertility.

Young (2005b) argues that HIV/AIDS reduces fertility, but that it must be the result of behaviour response among people in general, irrespective of whether they are HIV positive or not; the decline cannot be explained by the physiological effects of the disease alone. 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 reducing fecundity to avoid having HIV-positive children. Increases in the use of both 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 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, 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 that stays at home taking care of children loose more income (see Todaro, pp. 280-85, 2003). 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.

There are a few studies that provide empirical support to the hypothesis that HIV/AIDS reduces fertility at a national level in SSA countries. The most comprehensive study is probably Young (2005b). He uses data from Demographic and Health Surveys from 25 nations of Sub-Saharan Africa to show that HIV/AIDS leads to lower fertility. 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 Terceira et al (2003) that analyze data from twelve communities in Zimbabwe. They find that HIV/AIDS explain 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. They find that, due to lower fertility among HIV-positive women, an increase of HIV prevalence by one percentage point in the general female population reduces the total fertility rate by 0.4%. (More studies?)

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 total fertility rates seem to have increased or stopped decreasing: for example, in Tanzania the total fertility rate increased from 5.6 in 1999 to 5.7 in 2004, and in Kenya it rose from 4.7 to 4.8 between 1998 and 2003, while in Uganda it was 6.9 in 1995 and 2000 (Measure DHS Statcomplier database). Westoff and Cross (2006) attributes the increase in Kenya to HIV/AIDS, but does not test the hypothesis. The most recent estimates of total fertility rate in Malawi also indicate an increase, from 6.0 (MDHS 2004) to 6.3 in 2006 (MICS 2006). 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 make decisions on fertility after considering the likelihood of the survival of their children as adults: in societies without insurance systems and pensions, the support of children is vital at old age. Since HIV/AIDS increases prime-age mortality, it reduces life expectancy and makes it less likely that a child will outlive its parents. This increase in uncertainty raises the demand for children (Lorentzen et al. 2005; Kalemli-Ozcan, 2006).

Lorentzen et al. (2005) use macro data for a large number of countries over the period 1960-2000 and show that adult mortality is positively associated with high fertility in cross-section regressions. They do not focus on HIV/AIDS explicitly but show 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 a 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; randomised experiments have shown that circumcision reduces the risk of becoming infected with HIV. Hence, it is treated as an exogenous factor that affects to the spread of the epidemic. However, Fortson (2007) argues that circumcision is a poor instrument, casting doubt on the findings of Werker et al. (2006).

3. The HIV/AIDS epidemic in Malawi: a brief background

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.

Malawi's first AIDS case was diagnosed in 1985. The epidemic then spread rapidly, first in the major cities where the prevalence rate reached 30% among women visiting antenatal clinics (ANC) in 1993, and more recently in rural areas with large variations across the country (NAC, 2003). 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 in Sub-Saharan Africa, but clearly less than in the worst hit countries where the prevalence rates are over 20%. Recent estimates indicate that 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 Fact Sheet, 2007).

Since the end of the 1990s, national prevalence among women visiting ANCs has declined substantially, 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 however, from 11,8 % in 1999 to 13,6% in 2005 (GOM, 2006).

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.

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. Without any preventive measures, it is estimated that approximately 30% of babies born to HIV positive mothers are infected during pregnancy, birth, or through breastfeeding (DHS, 2004).

There is no simple explanation to why HIV has spread to such a large part of the Malawian population. Many contributing factors that interact and their relative importance are not known. One factor of importance should be Malawi's location, surrounded by other countries with high HIV-prevalence rates.

Apart from that the custom of having several relationships at the same time, at least among men, could be important. 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 (references). For a given number of life-time partners the practice of having multiple partners 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 other Western countries people might have as many partners during their lifetime, but they usually have one at the time (references)

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, and it is only poor countries that have generalised epidemics.[4] However, within Sub-Saharan Africa some of the wealthiest countries are the ones with 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). For instance, data from ANCs in Malawi shows 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: women may sell sex for goods or money (transactional sex), 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 that increases risky behaviour (Bryceson and Fonseca, 2006)

Unequal income distribution is also likely to be an important driver of the epidemic. One reason is that transactional sex and prostitution can only take place if there is a buyer with money. 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 students 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).

Yet another driver 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 Arrehag et al., 2006, Bryceson et al., 2004; Matinga and McConville, 2003).

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

[4] A generalised epidemic is when adult HIV prevalence is at least 1% according to UNAIDS.

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 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 Country Fact Sheets, 2007).

Malawi's first comprehensive HIV/AIDS policy was not formulated until 1999, the National HIV/AIDS Strategic Framework. It was developed through a participatory process involving representatives from all sectors and civil society, including faith organisations and people living with HIV/AIDS. 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.[5] 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 emphasised the need for information about HIV/AIDS. Moreover, they 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 result, 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 (Triangulisation 2006, MICS, 2007). However, the usage of condoms is still far from satisfactory.

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

Concerning voluntary HIV counselling and testing, the government declared its commitment to "promote and provide high-quality, cost-effective, totally confidential, and accessible 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 recently began; in 2003 there were less than 20 sites and only a couple of percent of the eligible HIV-positive patients received ART (Triangulaisation, 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, 2007).

4. Fertility in Malawi

In this section we look into fertility and its development in Malawi. The purpose is to give a background to the analysis on how the HIV/AIDS epidemic has affected fertility.

Figure 3 reports total fertility rates (TFR)[6] 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

[6] 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.

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 not seen as compatible 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, there is a turning point for the fertility trend and the TFR starts 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 Banda was replaced by the democratically elected government, the child spacing program was replaced with a family planning program with the explicit aim of reducing fertility (Chimbwete et. al. 2005). Nonetheless, fertility is still very high in Malawi, even when compared to other developing and 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 there was large national survey in 2006 (MIC, 2006) which showed rise to 6.3. However, MICS 2006 differs from MDHS somewhat and the estimates of TFR might not be comparable.[7]

Figure 3: Total fertility rates (TFR) over time across countries

Fertility at the national level hides large variations in fertility across different groups of women. For example, better educated women tend to give birth to fewer children than less educated women, as is the case in virtually every country in the world. It is therefore important to look at fertility and its evolution over time for different groups of women. Data on fertility is reported in Table 1 for various categories and 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. A more extensive table, including age-specific fertility rates and confidence intervals, can be found in the appendix.

[7] The data collected by MICS 2006 are not publicly available yet and are therefore not reported in the tables below.

As Table 1 shows, the difference between urban and rural fertility is substantial, 4.3 compared to 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.

Education and income also matter a great deal for fertility, although it is difficult to their relative importance. Higher education and income are associated with lower fertility. For women with no education, fertility remained stable in 1992-2000; but, as reported in the appendix, it increased for younger women with no education and decreased for older women with no education. In 2000-2004, however, 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. According to estimations using the DHS samples, 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%. Increased education has most likely contributed to the fertility decline 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[8]. There appears to be little difference in fertility between women from the two poorest wealth quintiles. And it is noteworthy that fertility 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, and especially women from the richest quintiles stand out.

There are also substantial differences across the three Regions. 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

[8] 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.

differences in urbanisation, income and education, but there are many other factors that vary across the Regions.

It is also interesting to distinguish between realised fertility (how many children the woman gave birth to during a certain time period) and desired fertility (how many children in total the women would want if she could choose freely). Realised and desired fertility could differ either because the women has difficulties in getting pregnant when she wants to, or because she does not have the access to means to prevent pregnancy when she does not want to become pregnant.

Data on desired fertility, available from 1992, 2000 and 2004, are reported in Table 2. They show the percentage distribution for desiring 0-2, 3-5 and 6 or more children. Overall, they provide the same information as the data on realised fertility. Nonetheless, it is interesting to note the small changes between 2000 and 2004 in many of the groups, a period when knowledge of HIV/AIDS became widespread, and practically everybody had personal experiences of the epidemic (NSO and OCR Macro, 2005).

The downward TFR trend shown in Figure 1 appears to be quite stable. However, looking at age-specific fertility rates and their changes over time, shown in Figure 4, 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, fertility among younger women decreased only marginally. Moreover, between 2000 and 2004 fertility only declined slightly or remained stable in all age groups.

Figure 5 shows age specific fertility in urban and rural areas for 1992 and 2004. During the period 1992-2000, fertility in urban areas decreased quite dramatically, while the decrease in 2000-2004 was modest. Desired fertility shows a similar pattern, a large decline in 1992-2000 and a smaller one in 2000-2004. In rural areas the fertility decline was smaller overall, and it appears to have been faster in 2000-2004 than in 1992-2000.

While fertility has decreased for all age groups in urban areas and women in age groups 25-29 and older in rural areas, as shown by Figure 5, it has increased in rural areas for the youngest

women, 15-19, 20-24. Desired fertility in rural areas decreased during 1992-2000, and remained stable thereafter. Hence changes in desired and realised fertility did not coincide in rural areas.

However, it should be recognised urbanization has changed the proportion of Malawians living in rural and urban areas. According to estimations using our DHS sample, 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.

When looking at age-specific fertility rates, shown in Figure 6, it becomes clear that there is not one wealth group that always has the lowest or the highest fertility rate. Yet, women in the wealthiest households start at a low TFR, and also reduce realised, and desired, fertility the most. In 2004, fertility rates are the lowest in this group for all age groups, as seen in Figure 7. On the other hand, for women in the poorest wealth quintile, fertility basically does not change at all, even though the proportion that wants six or more children decreases substantially. Comparing Figure 6 and 7 we also see that the age-profile of fertility changes towards younger women with a clear peak at the age 20-24.

4.1 HIV/AIDS and Fertility

Although fertility has declined during the spread of the epidemic, there are some signs of a stall in the fertility decline. This could be due to structural changes, such as urbanisation but it could also be due to the impact of HIV/AIDS. In Kenya, for instance, the recent rise in TFR is due to increased fertility among low income groups and it is associated with HIV/AIDS (Westoff and Cross, 2006).

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. It is also consistent with the fact that fertility declines have been concentrated among richer people and people in urban areas.

Whatever the reason behind this, the finding that fertility declines have been concentrated to older women and that fertility even 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 partners have before marriage. For a given number of desired children, they might also wish to have many children early to avoid the risk of turning ill or 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 amore 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 8 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 suggest that the HIV/AIDS epidemic decreases fertility. However, other factors may be associated with both higher adult mortality and lower total fertility rates. 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, even though the difference in HIV-prevalence between urban and rural areas is decreasing (NSO and OCR Macro, 2005). And we saw above that better educated, wealthier women living in urban areas have fewer children than others. It thus becomes important to control for these factors when comparing fertility in districts with differential adult mortality.

5. 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. For example, there are reasons to expect that adult mortality is higher in more urbanized districts, which have more educated and wealthier inhabitants as well as higher HIV prevalence, than in districts where most people live in rural areas. It is therefore necessary to use multivariate regression analysis to investigate if there is any relationship between district level adult mortality 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 presented

5.1 Data and variables

To investigate the impact of HIV/AIDS on fertility we primarily use data from the 2004 MDHS. The DHS-project was developed to primarily collect nationally representative samples 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 most recently available data are from the MDHS 2004. It was collected at a time when individuals should have adjusted fertility preferences and sexual behaviour to the new 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 2004 MDHS, giving us valuable information about their HIV-status. Altogether 11698 women aged 15 to 49 years were interviewed in the survey.

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, or future

children to contract HIV/AIDS, which may differ from assessed general risks in the community, but they should be related.

One potential measure 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, we use them as an alternative measure of district variation in HIV/AIDS prevalence. Past HIV-prevalence rates may be quite informative about current AIDS morbidity and mortality. We have data on HIV-prevalence among pregnant women from the early 1990s for most districts (from U.S. Census Bureau HIV/AIDS Surveillance Data Base). 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.[9]

In addition to data on HIV-prevalence among samples of pregnant women we have data on current HIV- prevalence in the general male and female population from the 2004 MDHS. 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 progress rapidly for small children and they could infer it from infants' deaths in AIDS symptoms.[10] Even though women could not observe HIV-prevalence, current rates might still be informative about AIDS deaths and illness insofar as they are correlated with past prevalence rates.

[9] 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.

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

In MDHS 2004, 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 used to obtain more observations and more district- level variation. These data are probably the best measures of HIV-prevalence in the general public available, even for the districts that were not over-sampled in the MDHS.

Table 3 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.

HIV-prevalence is higher among women than among men, as in most Sun-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 4 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 correlation between adult mortality 1998 and HIV-prevalence among pregnant women 1990-1994 is 0.36, but 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.

If district level adult mortality or HIV-prevalence rates determine fertility is affected in the econometric analysis, there is a possibility that this is due to women directly affected by the HIV/AIDS epidemic only. In districts with high adult mortality and high HIV-prevalence rates, there are of course more women that are directly affected. 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 test 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.

We know that changes in fertility in 1992-2004 were not uniform across age-groups (see Sub-Section on fertility). 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. Young women might feel that they would like to have their children early, so that children have time to grow up before she or her partner becomes sick and dies. If women want to have fewer children in total, it is also reasonable that fertility declines should be larger among the group of older women that already have children. To test for this possibility we split the sample to evaluate whether the effect of HIV is different across age-groups.

The dependent variables are realised or desired fertility. Realised fertility is the number of births the woman has given 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. 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.

The advantage of using individual level data is that we can control for various individual characteristics as mentioned earlier. Age is entered non-linearly into the model as the relationship between age and fertility should be first increasing 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 will also be 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. Ethnicity is likely to be particularly important in rural areas. Hence, we control for ethnicity of the women in the regressions on fertility in rural areas.

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 across space in prevailing norms. However, other characteristics might be influenced by poverty and income levels, so that, independently of own income. 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 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 is the case of over-dispersion, that the variance is larger than the mean implying that estimated standard errors are too small. 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 1 error[11] is smaller rather than larger. We therefore proceed with the Poisson estimation model.

[11] A Type 1 error is committed when the null hypothesis is rejected even though it is true.

5.2 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, while there was so in rural 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 5 reports the results from our basic estimations of realised fertility for 1999-2004 among rural women. Most of the [i]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 the women's age. As expected more educated women have given birth to fewer children. Household wealth 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 the women has no children, and a reduced probability mainly 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 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) compared with a more modest increase in education.

Column 2 in Table 6 shows the basic model with ethnicity dummies. The dummies only have a minor effect on the coefficient of district adult mortality. Column 3 reports the model with a number of control variables that are added to capture the variation across space in fertility caused by other factors that potentially are correlated with adult mortality. 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=0.104). 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.

The fourth column shows the results when the woman's HIV-status and the number of orphans[12] in the household in are included. 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.

Adding HIV-status to the explanatory variables changes the estimated coefficients of a number of other variables considerably.[13] First, it weakens the negative relationship between education and fertility, suggesting that part of it is due to the fact that HIV-prevalence is higher among better educated, and that HIV-positive women have fewer children. Second, it strengthens the positive relationship between belonging to the poorest wealth quintile and fertility, and weakens that between belonging to the second richest wealth quintile and fertility, making the relationship between economic status and fertility more linear (though the negative effect on fertility on belonging to the richest households is still larger than the positive fertility effect from being from the poorest households). Third, the associations between fertility and ethnic groups change, and it does now appear as if though Chewa, Yao and Tumbuka have higher fertility than other ethnic groups. Finally, the negative relationship between district adult mortality and individual fertility is strengthened, the coefficient decreases from -0.004 to -0.014 and becomes significant at the 1% level..

[12] 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.
[13] These changes do not occur if only orphans are added, but do so if only HIV-status is added.

Table 7 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 model with HIV prevalence and orphans. The estimated effect of an increase in district adult mortality from its 1987 mean value to its 1998 mean value is to increase the probability of having no children by 6%, and decrease the probability of having two or more children by 5.3%. This effect is somewhat larger than that of having two orphans in the household. HIV-negative women have more than 11% higher probability than HIV-negative women of not having given birth to any children in the past five years.

Next we investigate the relationship between the geographical spread of HIV/AIDS and fertility by considering alternative variables to district adult mortality in 1998. Moreover, we test for differences in the impact on fertility between females and males. The variables included are HIV-prevalence rates among pregnant women visiting antenatal clinics in the beginning of the 1990s; district HIV-prevalence rates in 2004; and adult mortality in 1987, which should not be affected by AIDS.

Table 8 shows that for adult mortality in 1998, the effect on fertility is somewhat stronger for female mortality on than for male mortality (model 1 and 2).[14] 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). A possible explanation of this pattern 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).[15] This explanation is supported 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

[14] 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.
[15] 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.

coefficient between district female adult mortality in 1987 and district HIV-prevalence in 2004 is only 0.02.

The estimations with the stated ideal number of children (desired fertility) as the dependent variable are reported in Table 9.[16] 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 in the basic set-up, 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. Adding the set of district level variation controls only strengthens this result, while adding ethnicity dummies somewhat weakens it, rendering the coefficient to be insignificant.

In the analysis of realised fertility, the HIV-status control strengthened the negative association with district adult mortality. But for desired fertility it weakens the relationship. As seen in Table 10, a statistically significant relationship however remains when district HIV-prevalence rates are used instead of district adult mortality rates. Moreover, 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 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 measures of HIV/AIDS are presented in Table 10. Desired fertility seems to be more affected by HIV-prevalence rates, both from 2004 and early

[16] 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.

1990s and both for males and females[17], than by adult mortality rates. There is no association whatsoever between adult mortality in 1987, neither for men nor for women, and the expressed desired fertility in 2004. The negative association between male non-AIDS adult mortality and realised fertility is accordingly not accompanied by a similar relationship with desired fertility.

Last we investigated the possibility that the spread of the HIV/AIDS epidemic is affecting fertility differently, depending on the age group. As seen in Table 11, the youngest women, aged 15-19, have more children where adult mortality and HIV-prevalence are higher, while other women have fewer children. The higher fertility among young women in districts with higher HIV-prevalence and adult mortality is however not accompanied by a desire to have more children in total. It thus seems as if though young women are more eager to have their children fast before they become HIV positive.

7. Conclusions

The purpose of this study was to analyse the impact of HIV/AIDS on fertility in Malawi. Data on fertility, and most of the other variables were taken from the Malawi Demographic and Health Surveys carried out in 2004. Fertility is measured as the number of births given during the last five years, 1999-2004. 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, Antenatal Clinic sentinel surveys, and the 2004 Demographic and Health Survey. Estimations were also carried out with the stated ideal number of children, a closer 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 districts where adult mortality and HIV-prevalence is high, un-infected women give birth to fewer children, and desire to have fewer children. There is an interesting gender difference in the impact of adult mortality. Female mortality in 1998 is clearly more important for fertility 199-2004, than male fertility. However, male adult mortality in 1987, but not female mortality in 1977, has a negative effect on mortality 1999-2004. This could be because high male mortality has led to high HIV prevalence in 2004,

[17] Results of estimations on male respectively female HIV-prevalence rates in 2004 are available from the authors on request.

and thus lower fertility. High adult mortality thus increases 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.).

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 suggests that there is a negative effect of the spread of HIV/AIDS on preferred number of children, and that part of the negative relationship between realised fertility and district adult mortality respectively HIV-prevalence rates is attributable to a change in fertility preferences because of the HIV/AIDS epidemic.

It was moreover found that the HIV/AIDS epidemic affected fertility of young women very different than fertility of older women. Young women had given birth to more children where adult mortality and HIV-prevalence was higher, but this was not accompanied by a desire to have more children in total. This result suggests that young women may feel that they want to have children quickly because of the epidemic, probably because they wish to avoid giving birth to HIV-positive babies.

In addition to the effect of HIV/AIDS on fertility in the general public, HIV-infected women give birth to fewer children than non-infected women. They also desire to have fewer children than other women, but this 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 effect of district level measures is thus to lower the fertility of HIV-positive women somewhat more than for HIV-negative women.

Our results stand in sharp contrast to those obtained by Lorentzen et al. (2005) and Kalemli-Ozcan (2006), where national total fertility rates increase with adult mortality. In fact, we find the opposite, adult mortality reduces mortality. The mechanisms that generate this result merit further exploration. Nevertheless, the results are in line with those found by Young (2005b), using micro-data from various Sub-Saharan African countries, but here the interpretation is that HIV/AIDS reduces fertility.

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 endogeneity problem with the estimate of orphans, since households with few children might have more 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 can be improved. The next step should 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 variation in data. Then the impact of the recent scale-up of the distribution of Anti Retroviral Therapy (ART) should be considered. However currently there are no available data.

References

Arndt, Channing (2002) "HIV/AIDS, Human Capital and Economic Prospects for Mozambique" TMD Discussion paper No. 48, IFPRI, Washington.

Arndt, Channing (2006) "Book Review, The Macroeconomics of HIV/AIDS" *Journal of African Economies*, Vol. 16, No. 1, pp. 166-171.

Arrehag, L., de Vylder, S., Durevall, D. and Sjöblom, M. (2006) *The impact of HIV/AIDS on the livelihoods, poverty and the economy of Malawi*, Sida Studies, No. 18, Stockholm.

Bell, C., Devarajan, S. and Gersbach, H. (2004) "Thinking About the Long-Run Economic Costs of AIDS", Chap. 3 in Haacker, M. (ed.) *The Macroeconomics of HIV/AIDS*, IMF, Washington, D.C.

Bloom, D. and J. Williamson (1998) "Demographic Transition and Economic Miracles in Emerging Asia" *World Bank Economic Review*, Vol. 12, No 1. pp.

Bloom, David and Ajay Mahal (1997) "Does the AIDS Epidemic threaten Economic Growth" *Journal of Econometrics*, Vol 77. pp.105-124.

Bryceson, D, Fonseca, J. and Kadzandira, J., (2004) "Social Pathways from the HIV/AIDS Deadlock of Disease, Denial and Desperation in Rural Malawi", CARE, Lilongwe.

Brysecon D. F. and J. Fonseca (2006) "Risking Death for Survival: Peasant Responses to Hunger and HIV/AIDS in Malawi", *World Development*, Vol. 34, No. 8, pp. 1654-1666.

Bulatao, R. (2006) "Population and Mortality in after AIDS", Chap. 6 in Eds. D. T. Jamison et al. *Disease and Mortality in Sub-Saharan Africa*, World Bank

Chimbwete, C., S. C. Watkins and Msiyaphazi Zulu, E. (2005) "The evolution of population policies in Kenya and Malawi", *Population Research and policy review*, Vol. 24, pp. 85-106

Corrigan, P., G. Glomm, and F Mendez (2005) "AIDS Crisis and Growth", *Journal of Development Economics* Volume 77, Issue 1, Pages 107-124.

Dercon, S. (2006) "Fate and Fear: Risk and its Consequences in Africa" paper prepared for the AERC, University of Oxford.

Doctor, Henry V. and Alexander A. Weinreb (2003) "Estimation od AIDS adult mortality by verbal autopsy in rural Malawi" *AIDS*, 17(17) pp. 2509-2513.

Epstein, B. G. (2004) "The Demographic Impact of HIV/AIDS", Chapter 1 in Haacker, M. (ed.) *The Macroeconomics of HIV/AIDS*, IMF, Washington, D.C.

Fabiani, M., B. Nattabi, E. Ayella, M. Ogwang, and S. Declich (2006),"Differences in fertility by HIV serostatus and adjusted HIV prevalence data from an antenatal clinic in northern Uganda" *Tropical Medicine and International Healt*h, 11(2) pp. 182-187..

Fortson, J. (2006) "Mortality Risk and Human Capital Investment: The Impact of HIV/AIDS in Sub-Saharan Africa" Mimeo, Dept. of Economics, Princeton University.

Gillespie, Stuart and Robert Greener (2006) "Is Poverty or Wealth driving HIV Transmission" Working Paper for the UNAIDS Technical Consultation on Prevention of Sexual Transmission of HIV.

Glick, P. (2006) "Reproductive Health and Behaviour, HIV/AIDS, and Poverty in Africa" paper prepared for the AERC, Cornell University.

Gray RH, et al. (1998) "Population-based study of fertility in women with HIV-1 infection in Uganda" *Lancet*, Jan 10;351 (9096): 98-103.

Gregson, S., Zaba, B., Hunter, S. (2002) "Modelling the relationship between the HIV epidemic, reproductive behaviour and fertility outcomes" United Nations Population Division.

GoM (2005) "Malawi HIV and AIDS Monitoring and Evaluation Report 2005" Government of Malawi, Lilongwe.

GoM (2006) "Report on the Malawi Triangulation Project" Government of Malawi, Lilongwe.

Haacker, M. editor, (2004) *The Macroeconomics of HIV/AIDS*, IMF, Washington, D.C.

Helleringer, S. and Hans-Peter Kohler (2006) "The structure of sexual networks and the spread of HIV in Sub-Saharan Africa: evidence from Likoma island (Malawi)" Mimeo, Department of Sociology, Population Studies Center, University of Pennsylvania.

Kadzamira E.C, Maluwa-Banda, D., Kamlonger, A. and Swainson, N. (2001) "The Impact of HIV/AIDS on Primary and Secondary Schooling in Malawi: Developing a Comprehensive Strategic Response", Centre for Educational Research and Training (CERT).

Kalemli-Ozcan, S. (2006) "AIDS, Reversal of the Demographic Transition and Economic Development: Evidence from Africa", Working Paper 12181, National Bureau of Economic Research.

Kalipeni, E. (1997) "Population pressure, social change, culture and Malawi's pattern of fertility transition", *African studies review*, Vol. 40(2), pp. 173-208

Kohler, H..P. J. R. Behrman and S. C. Watkins (2006). "Social Networks, HIV/AIDS and Risk Perceptions." *Demography* 44(1), 1-33.

Lewis, J. C., C. Ronsmans, A. Ezeh, and S. Gregson (2004) "The population impact of HIV on fertility in Sub-Saharan Africa" *AIDS*, 18 (suppl 2).

Lorentzen, P., J. McMillan and R. Wacziarg (2005) “Death and Development”, NBER Working Paper 11620, Cambridge, MA

Matinga, P. and F. McConville (2003) “A Review of Cultural Beliefs and Practices Influencing Sexual and Reproductive Health, and Health-Seeking behaviour in Malawi”, Department for International Development (DFID).

MDHS (various years), Malawi *Demographic and Health Survey*; National Statistical Office and ORC Macro. Available at http://www.measuredhs.com/

MICS (2007) “Malawi, Multiple Cluster Survey 2006”, NSO, Zomba and UNICEF, Lilongwe.

Morah, E. (2007) “Are people Aware of Their HIV-positive Status responsible for Driving the Epidemic in Sub-Saharan Africa: The Case of Malawi” *Development Policy Review*, 25(2) 215-242.

Munthali, A., P. Mvula and C. Chilimampunga (2006) “Determinants of Adolescent Fertility in Malawi: A Review of the Literature” Draft Report submitted to UNFPA, Lilongwe.

NAC (National AIDS Commission) (2001) “Estimating the HIV Prevalence in Malawi from Sentinel Surveillance Data” The Policy Project, Lilongwe, 2001.

NAC (National AIDS Commission) (2003) “HIV Sentinel Surveillance Report 2003” Lilongwe.

NSO (National Statistical Office) (2007). Population statistics, website: http://www.nso.malawi.net/

NSO (Natioal statistical Office) and OCR Macro (2005) “Malawi: DHS 2004 – final report”

Ntozi, James, (2002) ”Impact of HIVAIDS on Fertility in Sub-Saharan Africa” Economic Commission for Africa in collaboration with UNFPA.

Oster, E. (2005) “Sexually Transmitted Infections, Sexual Behavior and the HIV/AIDS Epidemic” *Quarterly Journal of Economics*, May, pp. 467-515.

Selwin, P., et al. (1989) “Prospective Study on Human Immunodeficiency Virus Infection and Pregnancy Outcomes in Intravenous Drug Users” JAMA 262, 1289-1294.

Terceira, N, Gregson, S., Zaba, B., et al. (2003) “The contribution of HIV to fertility decline in rural Zimbabwe, 1985-2000” *Population Studies* Vol. 57, No. 2/July, 149-64.

Thornton, R. (2007) “The Demand for and Impact of HIV Testing: Evidence from a Field Experiment” Working Paper, Harvard University.

UNAIDS (2006) “2006 Report on the global AIDS epidemic”.

United Nations and the Joint United Nations Programme on HIV/AIDS (UNAIDS), Geneva, Switzerland

UNAIDS (2007a) "Country Situation Analysis: Malawi" available at www.unaids.org.

UNAIDS (2007b) *Towards Universal Access: Scaling up priority HIV/AIDS interventions in the health sector*, WHO, UNAIDS, UNICEF progress report, April, 2007.

UN Population Division (2005) "The 2004 Revision Population Database", United Nations, New York

UN Population Statistics (2006) Data online, http://www.un.org/esa/population/unpop.htm

Weissman, A., Cocker, J. Sherburne, L. Powers M.B., Lovich, R. and Mukaka, M. (2006) "Cross-generational relationships: using a 'Continuum of Volition' in HIV prevention work among young people", *Gender & Development* Vol. 14, No. 1.

Werker, Eric. Amrita Ahuja, and Brian Wendell (2006) "Male Circumcison and the Economic Impact of AIDS in Africa" mimeo Harvard Business School.

Westoff, C. F. and A. R. and Cross (2006) "The Stall of the Fertility Transition in Kenya" DHS Analytical Studies No. 9, ORC Macro, Calverton, Maryland, USA

Young, A. (2005a) "The Gift of Dying: The Tragedy of AIDS and the Welfare of Future African Generations", Quarterly Journal of Economics, vol. 120, no. 2, May pp. 423-466.

Young, A. (2005b) "In Sorrow to Bring Forth Children: Fertility amidst the Plague of HIV" Mimeo, University of Chicago

Zaba B, Gregson S. (1998) "Measuring the impact of HIV on fertility in Africa" *AIDS*, 1998;12 Suppl 1, 41-50

Appendix

Table AX: 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]		Coeff.	[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

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
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 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.

Table 2: 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

Table 3: Adult mortality and HIV-prevalence in districts of Malawi.

Variable	Obs	Mean	Std, Dev,	Median	Min	Max
Adult mortality 1998 (‰)	24	15,2	6,9	13,1	7,1	33,6
Male adult mortality 1998 (‰)	24	16,8	7,4	15,1	8,4	37,4
Female adult mortality 1998 (‰)	24	13,4	6,7	11,1	5,8	29,8
Adult mortality 1987 (‰)	24	3,9	1,4	3,9	2,1	7,6
Male adult mortality 1987 (‰)	24	4,4	1,8	4,0	1,9	8,6
Female adult mortality 1987 (‰)	24	3,6	1,4	3,4	1,2	8,0
HIV-prevalence 2004 (%)	24	12,5	7,2	12,0	2,1	33,4
Male HIV-prevalence 2004 (%)	24	11,5	8,8	9,4	0,0	35,6
Female HIV prevalence 2004 (%)	24	13,1	6,8	14,2	2,6	31,2
HIV prevalence among pregnant women 1990-94 (%)	19	14,8	7,1	15,4	2,7	27,6
HIV prevalence among pregnant women 1992-96 (%)	19	15,3	7,5	15,9	3,5	31,1
HIV prevalence among pregnant women 1994-98 (%)	18	16,1	7,6	16,6	5,1	32,8
HIV prevalence among pregnant women 1996-2000 (%)	18	17,7	8,0	18,3	5,4	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 2004 MDHS (NSO and MACRO, 2005). HIV-prevalence data among pregnant women are from the U.S. Census Bureau HIV/AIDS Surveillance Data Base.

Table 4: 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	0,485 (0,010)	0,171 (0,424)	
HIV-prevalence pregnant women 1990-1994	0,357 (0,134)	0,041 (0,868)	0,643 (0,003)

Note*: p*-values in parenthesis

Table 5: 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***	Mean 1992 to	0,011	-0,002	-0,009
	(0,003)	mean 2004			
Household in poorest	0,019	Middle to poorest	-0,007	0,001	0,006
quintile	(0,023)	quintile			
Household in second	0,021	Middle to second	-0,008	0,001	0,006
poorest quintile	(0,024)	poorest quintile			
Household in second	-0,084***	Middle to second	0,030	-0,006	-0,024
richest quintile	(0,026)	richest quintile			
Household in richest	-0,268***	Middle to richest	0,095	-0,023	-0,071
quintile	(0,033)	quintile			
District adult mortality	-0,005***	Mean 1987 to	0,020	-0,003	-0,017
	(0,002)	mean 1998			
Constant	-5,354***				
	(0,171)				
Observations	10058				
Model F	171,15				

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
[1] Effects of discrete changes are calculated holding all other variables 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 6: Poisson regression results: Dependent variable: Number of births given during the past five years

	Model 1:	Model 2:	Model 3:
Age in years	0,400***	0,400***	0,386***
	(0,012)	(0,012)	(0,019)
Age in years squared	-0,007***	-0,007***	-0,007***
	(0,000)	(0,000)	(0,000)
Births 5 years ago	0,007	0,007	0,005
	(0,007)	(0,007)	(0,015)
Education in years	-0,015***	-0,015***	-0,011* (0,006)
	(0,003)	(0,003)	
Household in poorest quintile	0,019	0,019	0,088*
	(0,023)	(0,023)	(0,045)
Household in second poorest quintile	0,022	0,021	0,030
	(0,024)	(0,024)	(0,051)
Household in second richest quintile	-0,090***	-0,093***	-0,069
	(0,026)	(0,026)	(0,051)
Household in richest quintile	-0,277***	-0,287***	-0,291***
	(0,039)	(0,039)	(0,083)
District Adult mortality	**-0,005****	**-0,004**	**-0,014*****
	(0,002)	**(0,003)**	**(0,004)**
Chewa	0,053	0,043	0,171*
	(0,043)	(0,046)	(0,090)
Tumbuka	0,088 *	0,046	0,229** (0,112)
	(0,050)	(0,054)	
Lomwe	0,032	0,044	0,128
	(0,044)	(0,045)	(0,088)
Tonga	0,117	0,060	0,089
	(0,072)	(0,085)	(0,162)
Yao	0,079 *	0,085*	0,184** (0,087)
	(0,043)	(0,044)	
Sena	0,068	0,095	-0,037
	(0,062)	(0,063)	(0,112)
Nkonde	0,194 **	0,142	0,118
	(0,088)	(0,094)	(0,228)
Ngoni	0,015	0,006	0,127
	(0,052)	(0,053)	(0,099)
District poverty rate		-0,004	0,002
		(0,005)	(0,008)
District median expenditure per capita		0,000	0,000
		(0,000)	(0,000)
Northern region		0,028	-0,005
		(0,041)	(0,084)
Southern region		-0,024	0,047
		(0,041)	(0,069)
Orphans in household			-0,065** (0,027)
HIV-status			-0,315***
			(0,061)
Constant	-5,405***	-4,854***	-4,930***
	(0,000)	(0,561)	(0,940)
Observations	9920	9898	25,75
Model F	89,39	72,68	2451

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

Table 7: 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	0,009	-0,001	-0,008
Household in poorest quintile	Middle to poorest quintile	-0,032	0,004	0,028
Household in second poorest quintile	Middle to second poorest quintile	-0,011	0,002	0,009
Household in second richest quintile	Middle to second richest quintile	0,025	-0,004	-0,021
Household in richest quintile	Middle to richest quintile	0,104	-0,024	-0,080
District adult mortality	Mean 1987 to mean 1998	0,058	-0,005	-0,053
Northern region	Central to Northern region	0,002	0,000	-0,002
Southern region	Central to Southern region	-0,018	0,003	0,015
Chewa	Other ethnicity to Chewa	-0,062	0,008	0,055
Tumbuka	Other ethnicity to Tumbuka	-0,084	0,006	0,077
Lomwe	Other ethnicity to Lomwe	-0,047	0,006	0,041
Tonga	Other ethnicity to Tonga	-0,033	0,004	0,029
Yao	Other ethnicity to Yao	-0,067	0,007	0,060
Sena	Other ethnicity to Sena	0,014	-0,002	-0,011
Nkonde	Other ethnicity to Nkonde	-0,043	0,005	0,038
Ngoni	Other ethnicity to Ngoni	-0,046	0,005	0,041
Orphans in household	Zero to two orphans	0,047	-0,009	-0,038
HIV-status	HIV-negative to HIV-positive	0,112	-0,027	-0,085

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 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 8: 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	0,378***	0,387***	0,388***	0,390***	0,388***	0,390***	0,388***
	(0,020)	(0,019)	(0,020)	(0,020)	(0,020)	(0,020)	(0,020)
Age in years	-0,006***	-0,007***	-0,007***	-0,007***	-0,007***	-0,007***	-0,007***
squared	(0,000)	(0,000)	(0,000)	(0,000)	(0,000)	(0,000)	(0,000)
Births 5 years ago	0,013	0,004	0,006	0,006	0,007	0,008	0,006
	(0,014)	(0,015)	(0,015)	(0,015)	(0,015)	(0,015)	(0,015)
Education in years	-0,011*	-0,011**	-0,013**	-0,014**	-0,014**	-0,013**	-0,013**
	(0,006)	(0,006)	(0,006)	(0,006)	(0,006)	(0,006)	(0,006)
Household in	0,091*	0,091**	0,113**	0,110**	0,112**	0,106**	0,113**
poorest quintile	(0,045)	(0,045)	(0,048)	(0,047)	(0,047)	(0,048)	(0,048)
Household in	0,038	0,031	0,038	0,041	0,042	0,034	0,038
second poorest	(0,050)	(0,051)	(0,052)	(0,052)	(0,052)	(0,053)	(0,052)
quintile							
Household in	-0,069	-0,069	-0,077	-0,073	-0,073	-0,073	-0,077
second richest	(0,050)	(0,051)	(0,052)	(0,052)	(0,052)	(0,052)	(0,052)
quintile							
Household in richest	-0,304***	-0,293***	-0,295***	-0,274***	-0,276***	-0,284***	-0,295***
quintile	(0,081)	(0,083)	(0,087)	(0,086)	(0,086)	(0,086)	(0,087)
District female adult	-0,018***						
mortality	(0,005)						
District male adult		-0,010**					
mortality		(0,004)					
District adult			-0,045**				
mortality 1987			(0,020)				
District female adult				-0,024			
mortality 1987				(0,023)			
District male adult					-0,036***		
mortality 1987					(0,013)		
District HIV-						-0,732*	
prevalence 2004						(0,377)	
District HIV-							-0,004
prevalence pregnant							(0,003)
1990-94							
Orphans in	-0,064**	-0,066**	-0,068**	-0,072**	-0,071**	-0,070**	-0,068**
household	(0,027)	(0,027)	(0,029)	(0,028)	(0,029)	(0,028)	(0,029)
HIV-status	-0,311***	-0,319***	-0,329***	-0,340***	-0,335***	-0,326***	-0,329***
	(0,061)	(0,061)	(0,064)	(0,063)	(0,063)	(0,064)	(0,064)
Constant	-4,792***	-4,969***	-4,447***	-4,960***	-4,918***	-4,216***	-4,447***
	(0,958)	(0,961)	(1,012)	(0,977)	(0,954)	(0,990)	(1,012)
Ethnicity dummies	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Additional regional variation controls[1]	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	2451	2451	2335	2366	2366	2335	2335
Model F	25,29	24,60	24,01	24,90	26,11	24,90	24,01

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

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

Age in years	0,027***	0,028***	0,028***	0,024***
	(0,003)	(0,003)	(0,003)	(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***	0,041***	0,041***	0,041***
	(0,003)	(0,003)	(0,003)	(0,005)
Education in years	-0,008***	-0,009***	-0,008***	-0,010***
	(0,001)	(0,001)	(0,001)	(0,002)
Household in poorest quintile				0,017
	-0,010 (0,011)	-0,006 (0,011)	-0,006 (0,010)	(0,018)
Household in second poorest quintile	0,001	0,005	0,004	
	(0,011)	(0,011)	(0,011)	-0,023 (0,020)
Household in second richest quintile	-0,025**	-0,027**	-0,028**	
	(0,012)	(0,011)	(0,011)	-0,016 (0,018)
Household in richest quintile	-0,077***	-0,075***	-0,075***	-0,063***
	(0,014)	(0,014)	(0,014)	(0,024)
District Adult mortality	-0,003***	-0,005***	-0,004***	
	(0,001)	(0,001)	(0,001)	-0,003 (0,002)
District infant mortality		0,000	0,000	0,000
		(0,000)	(0,000)	(0,000)
District poverty rate		0,002	0,002	
		(0,002)	(0,002)	-0,002 (0,003)
District median expenditure per capita		0,000	0,000	0,000
		(0,000)	(0,000)	(0,000)
Northern region		0,066***	0,093***	0,059
		(0,015)	(0,020)	(0,40)
Southern region		0,011	0,000	
		(0,018)	(0,020)	-0,001 (0,034)
Chewa			-0,005 (0,018)	-0,028 (0,032)
Tumbuka			-0,058***	
			(0,021)	-0,054 (0,037)
Lomwe			-0,023 (0,014)	-0,005 (0,026)
Tonga			0,056	0,021
			(0,042)	(0,070)
Yao			-0,010 (0,017)	-0,008 (0,027)
Sena			0,061***	0,093***
			(0,022)	(0,035)
Nkonde			0,028	0,033
			(0,039)	(0,061)
Ngoni			-0,048**	
			(0,021)	-0,035 (0,042)
Orphans in household				0,000
				(0,008)
HIV-status				-0,036* (0,020)
Constant	0,926***	0,778***	0,797***	1,311***
	(0,054)	(0,210)	(0,205)	(0,316)
Observations	9565	9543	9542	2369
Model F	184,63	128,33	88,90	21,00

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

Table 10: 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***	0,026***	0,026***	0,026***	0,026***	0,026***
	(0,006)	(0,006)	(0,006)	(0,006)	(0,006)	(0,006)	(0,006)
Age in years squared	0,000***	0,000***	0,000***	0,000***	0,000***	0,000***	0,000***
	(0,000)	(0,000)	(0,000)	(0,000)	(0,000)	(0,000)	(0,000)
Births 5 years ago	0,041***	0,041***	0,041***	0,041***	0,041***	0,041***	0,043***
	(0,005)	(0,005)	(0,005)	(0,005)	(0,005)	(0,005)	(0,006)
Education in years	-0,010***	-0,010***	-0,011***	-0,011***	-0,011***	-0,011***	-0,010***
	(0,002)	(0,002)	(0,002)	(0,002)	(0,002)	(0,002)	(0,003)
Household in poorest quintile	0,017	0,018	0,016	0,014	0,015	0,015	0,021
	(0,018)	0,018)	(0,018)	(0,018)	(0,018)	(0,018)	(0,020)
Household in second poorest	-0,024	-0,023	-0,019	-0,019	-0,019	-0,020	-0,008
quintile	(0,020)	(0,020)	(0,020)	(0,019)	(0,019)	(0,019)	(0,021)
Household in second richest	-0,016	-0,016	-0,017	-0,017	-0,017	-0,015	-0,013
quintile	(0,018)	(0,018)	(0,019)	(0,019)	(0,019)	(0,019)	(0,021)
Household in richest quintile	-0,063***	-0,064***	-0,073***	-0,072***	-0,072***	-0,071***	-0,053*
	(0,024)	(0,024)	(0,025)	(0,025)	(0,025)	(0,025)	(0,027)
District female adult	-0,004*						
mortality	(0,002)						
District male adult mortality		-0,002					
		(0,002)					
District adult mortality 1987			-0,003				
			(0,007)				
District female adult				0,005			
mortality 1987				(0,007)			
District male adult mortality					0,003		
1987					(0,005)		
District HIV-prevalence 2004						-0,338**	
						(0,141)	
District HIV-prevalence							-0,003**
pregnant women early 90s							(0,001)
Orphans in household	0,001	0,000	-0,001	-0,002	-0,001	0,002	-0,002
	(0,008)	(0,008)	(0,008)	(0,008)	(0,008)	(0,008)	(0,009)
HIV-status	-0,035*	-0,036*	-0,038*	-0,038*	-0,039*	-0,036*	-0,031*
	(0,050)	(0,020)	(0,021)	(0,050)	(0,050)	(0,020)	(0,022)
Constant	1,337***	1,300***	1,128***	0,798***	0,829***	1,499***	1,105***
	(0,316)	(0,316)	(0,316)	(0,282)	(0,274)	(0,327)	(0,322)
Ethnicity dummies	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Additional regional variation controls[1]	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	2369	2369	2602	2289	2289	2259	1836
Model F	21,02	20,99	21,48	22,40	22,43	21,95	17,58

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

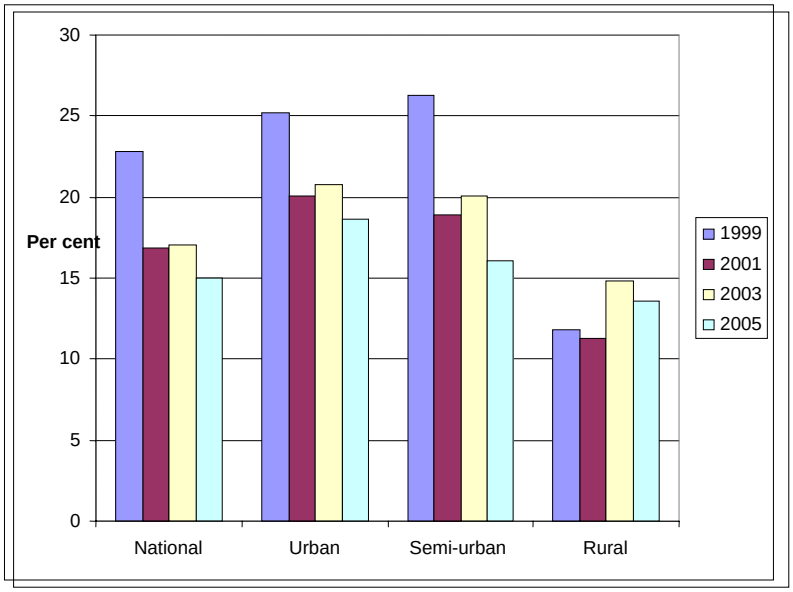
Table 11: Poisson estimation of realised fertility for teenagers and others

Dependent variable: Number of births given during the past five years				
	Sample of 15-19 years old		Sample of 20-49 years old	
	(1)	(2)	(3)	(4)
Age in years	7,072***	6,908***	0,172***	0,174***
	(1,295)	(1,302)	(0,013)	(0,013)
Age in years squared	-0,182***	-0,178***	-0,003***	-0,004***
	(0,037)	(0,037)	(0,000)	(0,000)
Births 5 years ago	0,386***	0,154	0,043***	0,045***
	(0,141)	(0,328)	(0,007)	(0,007)
Education in years	-0,098***	-0,100***	-0,015***	-0,017***
	(0,014)	(0,014)	(0,003)	(0,003)
Household in poorest quintile	0,202*	0,153	-0,003	-0,001
	(0,118)	(0,117)	(0,023)	(0,023)
Household in second poorest quintile	0,290**	0,252**	-0,005	0,002
	(0,117)	(0,115)	(0,024)	(0,025)
Household in second richest quintile	0,017	-0,079	-0,067***	-0,066***
	(0,137)	(0,131)	(0,026)	(0,025)
Household in richest quintile	-0,163	-0,178	-0,210***	-0,206***
	(0,162)	(0,165)	(0,040)	(0,039)
District Adult mortality	**0,029*** **		**-0,007*** **	
	(0,008)		**(0,002)**	
District HIV-prevalence 2004		**1,476** **		**-0,529*** **
		(0,731)		**(0,200)**
Ethnicity dummies	Yes	Yes	Yes	Yes
Constant	-69,04***	-67,05	-1,70***	-1,749***
	(0,000)		(0,201)	(0,203)
Observation	1997	1941	7923	7702
Model F	23,00	21,41	44,33	43,13

Table 12: Poisson estimation of desired fertility for teenagers and others

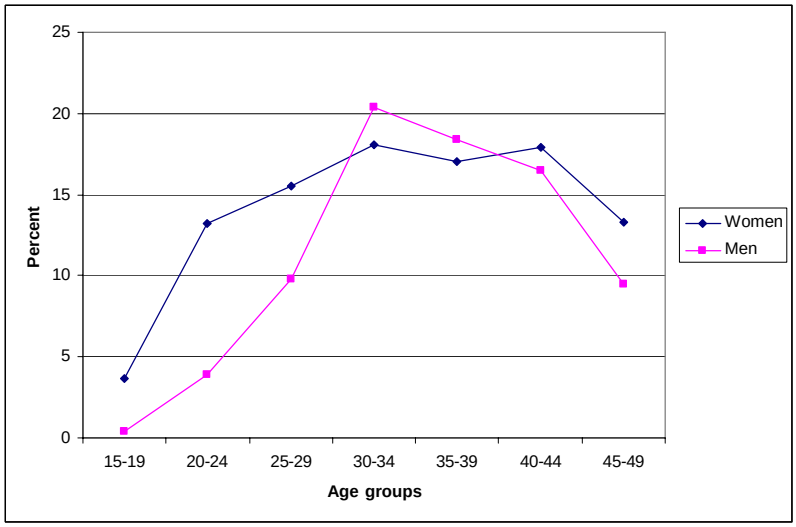
Dependent variable: Ideal number of children				
	15-19 years old		20-49 years old	
	(5)	(6)	(7)	(8)
Age in years	0,220	0,138	0,031***	0,029***
	(0,211)	(0,212)	(0,004)	(0,004)
Age in years squared	-0,006	-0,004	0,000***	0,000***
	(0,006)	(0,006)	(0,000)	(0,000)
Number of living children	0,040**	0,041**	0,042***	0,042***
	(0,20)	(0,020)	(0,003)	(0,003)
Education in years	-0,001	0,000	-0,009***	-0,009***
	(0,004)	(0,004)	(0,001)	(0,001)
Household in poorest quintile	-0,018	-0,031	-0,006	-0,010
	(0,026)	(0,026)	(0,011)	(0,011)
Household in second poorest quintile	0,034	0,032	-0,003	-0,003
	(0,032)	(0,033)	(0,011)	(0,010)
Household in second richest quintile	-0,031	-0,042	-0,027**	-0,025**
	(0,031)	(0,031)	(0,012)	(0,012)
Household in richest quintile	-0,074**	-0,079**	-0,083***	-0,082***
	(0,003)	(0,033)	(0,014)	(0,014)
District Adult mortality	**-0,002**		**-0,003*** **	
	(0,002)		**(0,001)**	
District HIV-prevalence 2004		**-0,236**		**-0,363*** **
		(0,180)		**(0,058)**
Ethnicity dummies	Yes	Yes	Yes	Yes
Constant	-0,725		0,901***	0,930***
	(1,797)		(0,075)	(0,072)
Observations	1944	1886	7620	7401
Model F-test	4,00	4,10	83,30	91,28

Figure 1: HIV prevalence among pregnant women attending to ANCs



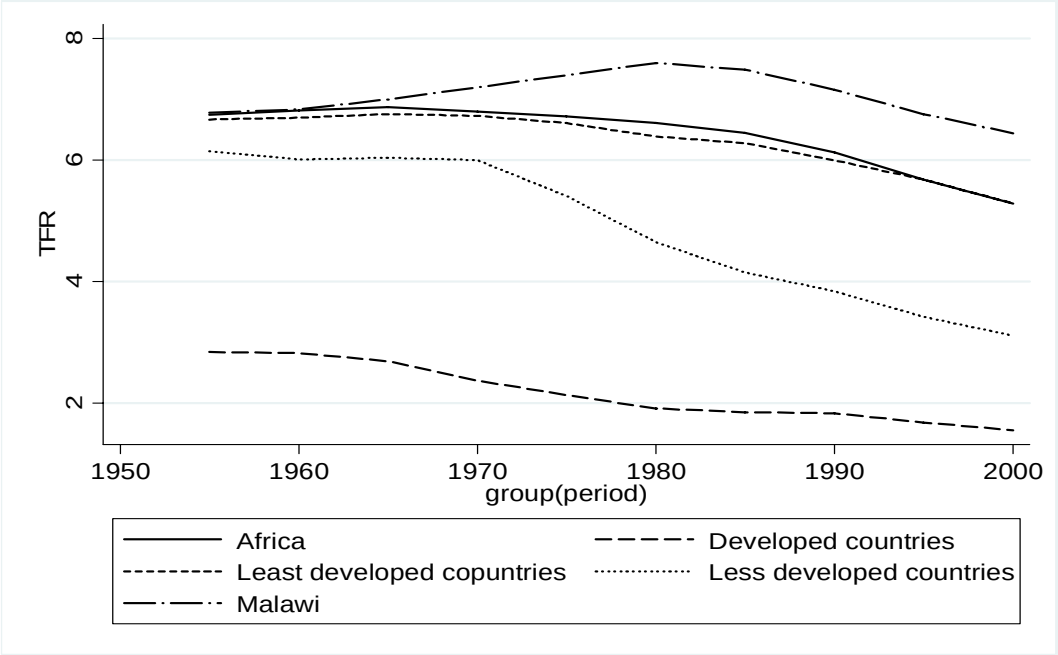
Source: Malawi Triangulation Project (GOM, 2006)

Figure 2: Percentage HIV positive among women and men age 15-49



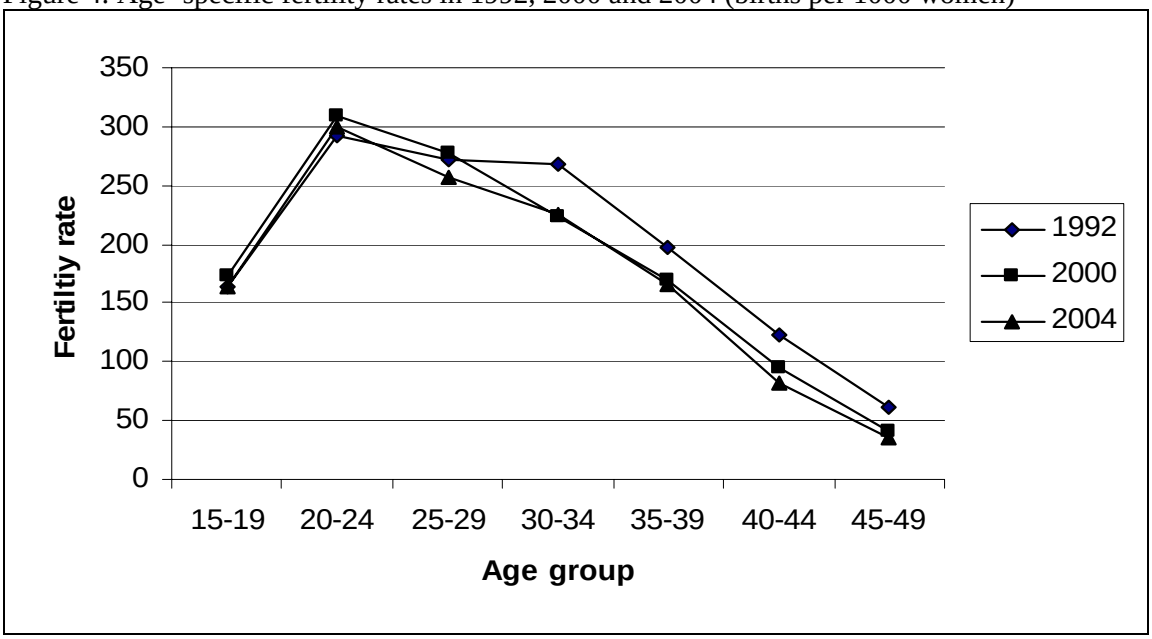
Source: Demographic and Health Survey 2004 (ORC Macro and NSO, 2005)

Figure 3: Total fertility rates (TFR) over time across countries



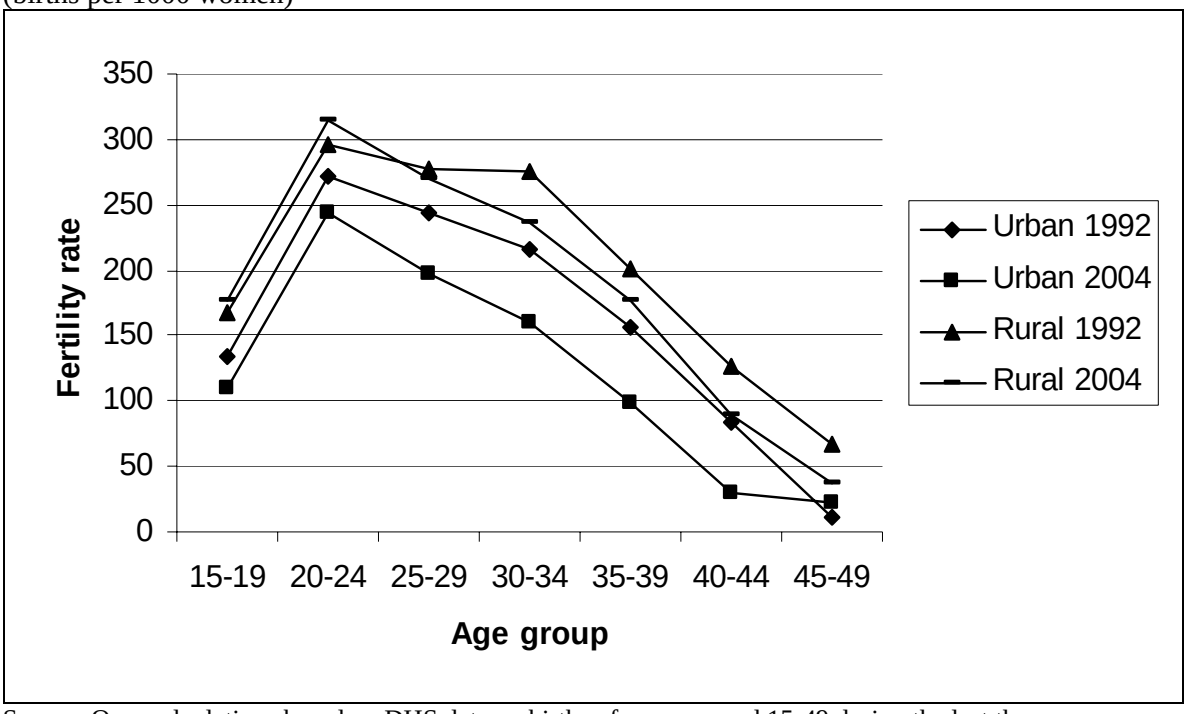
Source: UN Population ::::::::

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



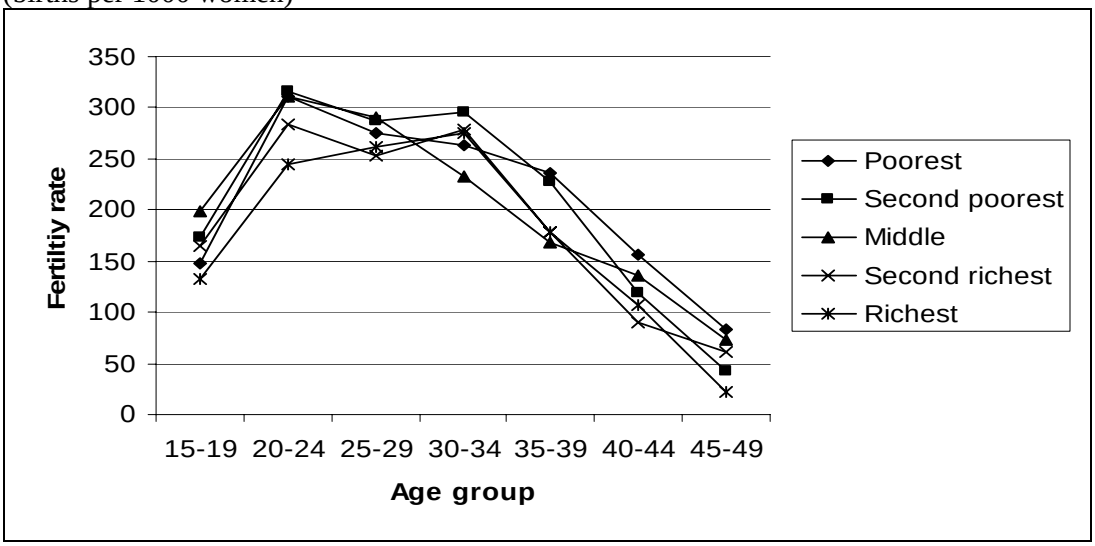
Source: Own calculations based on DHS data on births of women aged 15-49 during the last three years.

Figure 5: Age- specific fertility rates in urban and rural areas 1992 and 2004
(births per 1000 women)



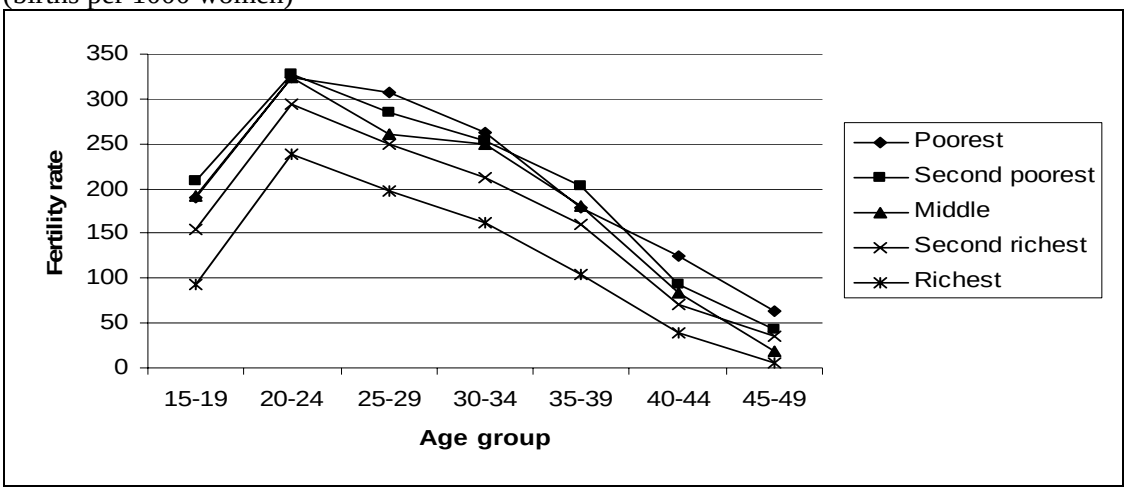
Source: Own calculations based on DHS data on births of women aged 15-49 during the last three years.

Figure 6: Age- specific fertility rates for women in different wealth quintiles in 1992
(births per 1000 women)



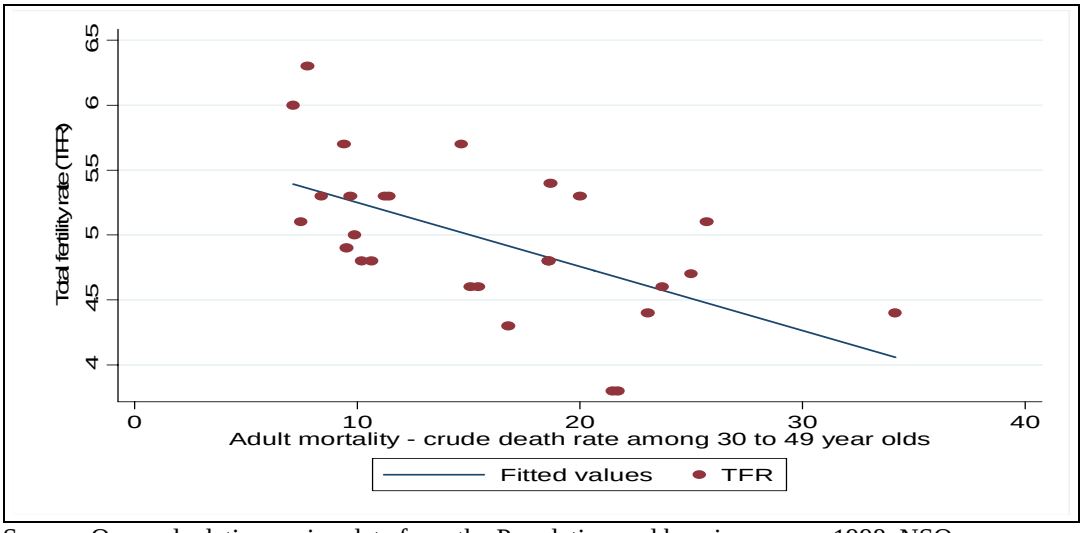
Source: Own calculations based on DHS data on births of women aged 15-49 during the last three years.

Figure 7: Age- specific fertility rates for women in different wealth quintiles in 2004 (births per 1000 women)



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

Figure 8: District level fertility versus district level adult mortality in1998



Source: Own calculations using data from the Population and housing census 1998, NSO