

LIFE EXPECTANCY AND ECONOMIC DEVELOPMENT

A Dissertation

Presented to

the Faculty of the Department of Economics

University of Houston

In Partial Fulfillment

of the Requirements for the Degree

Doctor of Philosophy

By

Belgi Turan

May 2011

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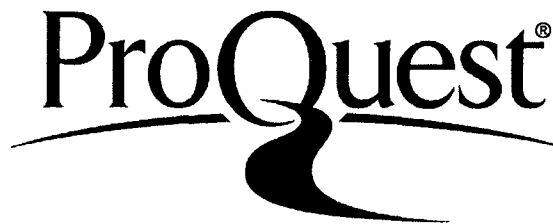
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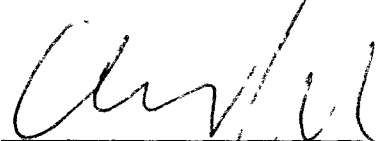


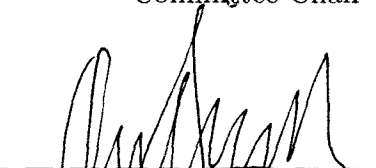
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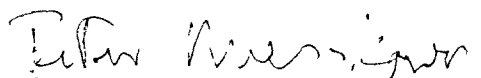

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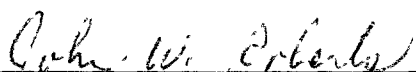
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Abstract

I study the relationship between life expectancy and economic development. In particular, I examine the effect of improvements in life expectancy on fertility, education and labor force participation.

In chapter 1, I exploit the shock to mortality caused by the Universal Immunization Program in India to study the impact of declining child mortality on fertility and human capital investment decisions of households. Between 1980 and 1990, immunization rates for the main childhood diseases in India increased 50 to 60 percentage points. Using country-level changes in immunization rates interacted with state-level initial mortality in 1980 as an instrument for child mortality changes, I find that a 1 percentage point decline in child mortality reduces births last year by about 5 percent, and increases female schooling by 0.4 years, an increase of about 7 percent. While previous papers have focused on the "horizon effect" where declines in adult mortality increase human capital accumulation, my findings point to another mechanism of the quantity/quality tradeoff that works through child mortality and fertility declines.

In chapter 2, using birth and sibling histories from Demographic Health Surveys conducted in sub-Saharan Africa I construct age-specific birth rates and age-specific mortality rates at the country-region level. I use this data to test the implications of a general equilibrium model linking life expectancy to fertility, education, and labor supply. I find that increased life expectancy lowers fertility, but the size of the effect is small. I find no difference between high HIV countries (those with greater than 5 percent prevalence) and low HIV countries, ruling out the possibility that fear of infection dramatically lowers fertility. I find a positive relationship between life expectancy and education when I pool all countries. Within high HIV countries, however, the relationship between life expectancy and education is less robust. Finally, I find a weak positive relationship between life expectancy and labor force participation for females, but no relationship among males. Overall the new data

suggests that in sub-Saharan Africa, increases in life expectancy will have a positive impact on growth through fertility and education but the effect will be small.

Acknowledgements

I give my sincere gratitude and appreciation to my advisors Chinhui Juhn and Sebnem Kalemli-Ozcan for being great mentors to me. They have supported and guided me to be an independent researcher. Working with them greatly improved my economic reasoning and empirical skills. Chinhui Juhn was the most incredibly supportive and committed advisor one could ever want. Her attention to detail and patience improved my papers immensely. She has been always very generous to me with her time and financial resources. I am indebted to Sebnem Kalemli-Ozcan for believing in me since the beginning of the graduate school, challenging me to be a better researcher and providing me with emotional support. She always offered me superb guidance, impressed me with her insights into economic issues and inspired me with her enthusiasm. I was very lucky to have her as my advisor and hope to have her as a friend in the future. I am particularly grateful to Aimee Chin who contributed immensely to my research with insightful comments and suggestions. Her classes greatly improved my empirical skills and discussions with her made me a critical researcher. I must also thank Bent Sorensen for his helpful comments and discussions.

I also thank to all faculty members of the Department of Economics for the stimulating discussions and friendly research environment. I am very grateful to Mevlude Akbulut-Yuksel for being the dearest friend and co-author. She always provided me with insightful comments and suggestions and made Houston fun to live in. I hope to continue working with her in the future as well. I also thank all my friends in the department for making the time at the University of Houston unforgettable.

I am very grateful to my husband, Burak Turan, for his unending love and encouragement. I am also indebted to him for providing me with technical support in

my papers. I want to thank my precious Duru for joining us along the way. I must also express my deepest gratitude to my father, Bekir Kilic, and my brother, Omer Kilic, for always being there for me. I dedicate this thesis to my mother, Sabiha Kilic, who was my best friend and also an economist. I wish you could have seen this.

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to my mother

Chapter 1

Mortality, Fertility and Human Capital Investment: Evidence from Immunization Program in India

1.1 Introduction

What is the effect of declining child mortality on fertility and human capital investment decisions? To answer this question, I exploit the large improvements in child mortality driven by Universal Immunization Program (UIP) in India. UIP started in 1985 aiming to immunize at least 85 percent of all infants (0-12 months) against six main childhood diseases.¹ Between 1980 and 1990, immunization rates for tuberculosis increased 63 percentage points, while immunization rates for the others increased about 50 percentage points on average. I estimate a large impact of the

¹The diseases are diphtheria, pertussis, tetanus, polio, typhoid and childhood tuberculosis.

program on child mortality, with the program potentially accounting for a 4 percentage point reduction in under-5 child mortality.² The program can be viewed as an exogenous shock to child mortality and provides an opportunity to understand the complex relationship between mortality, fertility and educational decisions.

The theoretical literature models the effect of adult mortality and child mortality on fertility and education decisions separately.³ A decline in the adult mortality affects fertility and education decisions through increased returns to education, the so-called "horizon effect." Decreases in adult mortality encourages educational investment as it increases the horizon over which returns to investments in human capital can be realized. Using household data from Sri Lanka and sub-Saharan Africa respectively, Jayachandran and Lleras-Muney (2009) and Fortson (2011) provide empirical evidence for this channel. Changes in child mortality, on the other hand, might work their way through fertility directly. There can be several mechanisms from child mortality to fertility. One is such where, at high levels of mortality parents engage in hoarding and bear more children relative to their desired number to ensure the survival. As the survival probability of each child increases, parents do not have to insure themselves by having more children.⁴ Another mechanism is about the returns to big family as highlighted by Soares (2005). If parents get utility from the number of offsprings surviving into adulthood and raising their own children, decreases in child mortality increases the probability to survival to adulthood, therefore, decreases the return to having a large family. Both adult and child mortality declines might induce a quality-quantity trade-off, but the underlying mechanism might differ. In the case of a decline in adult mortality, increases in educational investments driven by higher future return will cause a reduction in fertility. In the case of a decline

²Kumar (2009a) and Kumar(2009b) also evaluate the impact of the program on child mortality and find substantially smaller effects. I discuss possible reasons for this difference in the text.

³A notable exception is Soares (2005) that models both.

⁴See Kalemli-Ozcan (2002, 2003) and Tamura (2006).

in child mortality, decreases in fertility might cause parents to increase educational investments since now it is cheaper to invest in each surviving child. It is a daunting task to separate out these channels empirically. The literature focused on life expectancy at age 15 to pin down the adult mortality channel. The parallel literature that focuses on child mortality used mortality rates under 5. Both approaches remain vulnerable to the fact that simultaneous changes might drive mortality at any age together with fertility and education. I propose an alternative approach. By focusing on a program that solely targets child mortality, my goal is to provide evidence on the child mortality driven quality-quantity trade-off. To the best of my knowledge this has been a first in the literature.

I build child mortality rates by state and year and individual level fertility rates by year using birth histories from Demographic Health Surveys. I employ a difference in differences strategy where I compare mortality changes across states with high and low initial child mortality in 1980 (before the program). My identifying assumption is that the immunization program had disproportionately larger impact on states with high initial mortality. Without the program, the change in fertility for a one percentage change in mortality would be same in high and low mortality states. States with low initial child mortality may already have had access to vaccines or had health care services that could more adequately battle childhood diseases. My estimates indicate that the program reduced child mortality by approximately 4 percentage points (or approximately 36 percent) for states at mean initial mortality levels. To guard against the possibility that these differences were driven by prior trends, I show that prior to the program, there was no significant difference in changes in child mortality across states identified as high and low mortality states. After running reduced form specifications where I investigate the effect

of the program on fertility and education decisions, I also undertake an IV analysis. I instrument state-level child mortality with the initial mortality interacted with country level immunization rate. This allows me to gauge the size of child mortality effect on fertility and education. I also control for regional GDP differences because economically active and more developed states may have both lower mortality and lower fertility and higher education.

My results are as follows. Reduced form estimations show that the program had substantial effects on fertility and education. I estimate that relative to states with zero initial mortality the program reduced births last year by .032 in states with mean initial mortality. This corresponds to a decline of 14.7 percent. Next, I find that program led to .84 years increase in female years of schooling relative to states with zero initial mortality. Given that the average female schooling was about 6 years, the program effect corresponds to a 14 percent increase for the affected cohorts. Interestingly, however, the program did not have any statistically significant effect on male education. This is consistent with the literature that shows gender specific program effects.⁵ My findings suggest that girls may be the marginal children and they are provided with more family resources as they become available with the reductions in fertility. My IV estimates show that 1 percentage point decline in child mortality led to .010 reduction in births, which corresponds to a 5 percent decline. Finally, I show that 1 percentage point decrease in child mortality increased female schooling by 0.4 years, which translates to a 7 percent increase. Both of these effects are relative to the sample mean.

My results have important aggregate implications. The empirical literature on mortality and development mostly uses cross-country data and regresses countries' growth rates or per capita income levels on life expectancy. This literature faces

⁵See Chin (2005), Banerjee et al.(2004), Munshi and Rosenzweig (2004), Duflo (2003), among many others.

several challenges. First, it is difficult to establish causality since growth, income, and life expectancy are all simultaneously determined variables. Another challenge is the difficulty in identifying the exact mechanism through which increased life expectancy has a positive effect on development. The main channel according to the theoretical literature is the quality-quantity trade-off, on which there has not been robust evidence. The aforementioned papers such as Jayachandran and Lleras-Muney (2009) and Fortson (2009) provide credible evidence on one channel which works through the “horizon effect” and adult mortality. In this paper I provide systematic evidence on the quality-quantity trade-off that is driven by lower child mortality and accompanying declines in fertility.

The literature also focuses on the morbidity channel, where lower morbidity encourages higher investments in children. This literature generally focuses on the morbidity declines associated with hookworm eradication or malaria reductions (See Miguel and Kremer (2004), Bleakley (2007)). My initial mortality rates by state are not by disease and hence I cannot estimate the extent of initial morbidity and the associated decline afterwards. Although I cannot fully rule out the possibility that my results are affected by this, I believe that this is not a major concern. My reasoning is based on the fact that the six childhood disease that I use are the main killers of children aged 0-4 and UIP directly targets these diseases.

The rest of the paper is organized as follows. Section 2 provides a brief review of the related papers. Section 3 explains the program. Section 4 describes the data. Section 5 presents the empirical strategy and results. Section 6 presents robustness checks. Section 7 concludes.

1.2 Related Literature

Theoretically the effect of health and lower mortality on growth is ambiguous. On the one hand, lower mortality increases population growth and reduces income per capita. On the other hand, in response to lower mortality changes in fertility and human capital investment behaviors may increase TFP and the rate of human capital accumulation. Behavioral changes in fertility and education lead to quantity-quality trade-off. While the first-order effect of decreases in adult mortality is to increase human capital investment, first-order effect of child mortality is to decrease fertility. Both mechanisms shift quantity-quality trade-off towards fewer children with more education. (See Kalemli-Ozcan (2002, 2003), Tamura (2005), Soares (2005).) Most of the available evidence on the subject is from cross-country studies and they report positive effects of life expectancy on income growth (see Lorentzen, McMillan and Wacziarg (2008), Bloom and Sachs (1998), Gallup, Sachs, and Mellinger (1999), and Bloom et al. (2004).) However, since countries suffering from high mortality and ill health are also disadvantaged in other ways, most of these cross-country studies cannot suggest more than correlative evidence. Alternatively, Shastri and Weil (2003), Weil (2007), Ashraf et al. (2008) take a different approach and use calibrations based on microeconomic estimates to show the effect of health on growth. They find positive but small effects. In a recent influential paper, Acemoglu and Johnson (2007) instrument changes in life expectancy with dates of global health interventions to combat 15 major diseases and find that although these interventions increased life expectancy, they had a negative causal effect on income per capita.⁶

Several recent papers have turned to country-specific evidence, relying on natural

⁶See Cervellati and Sunde (2009) and Bloom, Canning and Fink (2009) for discussion of their findings.

experiment that provide plausible exogenous variation in life expectancy. Jayachandran and Lleras-Muney (2009) use exogenous declines in maternal mortality between 1946 and 1953 in Sri Lanka to show the effect of life expectancy on educational investment. They find that one year increase in life expectancy increased female literacy by 0.7 percentage point (2%) and years of schooling by 0.11 years (3%). Using household data from Brazil, Soares (2006) finds that a one-standard-deviation decrease in adult mortality decreases total number of births by 2.5-3.7% and increases years of schooling by 2.2-3.1%. Fortson (2011) uses individual data from sub-Saharan African countries and finds that children living in areas with 10 percent regional HIV prevalence complete 0.3 fewer years of schooling (7%) compared to children living in areas with zero HIV prevalence.

In addition to mortality, a number of papers have also investigated the impact of morbidity on educational outcomes. Miguel and Kremer (2004) and Bleakley (2007) show that deworming interventions improved educational outcomes in Kenya and American South, respectively. The fact that worm infections are rarely fatal but individuals who have these infections suffer substantial morbidity allow them to study the effect of morbidity on human capital accumulation. Bleakley (2007), employing a strategy similar to this paper, shows that areas with higher initial levels of hookworm infection benefited more from the eradication of this disease and experienced greater increases in school attendance and literacy.

Although UIP was an extensive program in terms of coverage and potential effects on the development, the repercussions of it has not been studied in detail. On exception of this is Kumar (2009a), which finds that immunization program decreased probability of primary school completion by 7.2% and increased secondary school completion by 11%. However, he does not find any program effect on literacy or average years of schooling. He explains the negative impact on primary education

with lower average health among marginal surviving children. Additionally, Kumar (2009b) shows that exposure of first-born child to the program is associated with lower cumulative fertility (1%) and larger birth intervals (2.5%).

1.3 Universal Immunization Program

Following smallpox eradication in the 1970s, India launched the Expanded Program on Immunization (EPI). Initially six childhood diseases were targeted: Diphtheria, pertussis, tetanus, polio, typhoid and childhood tuberculosis. However, the EPI was mostly limited to urban areas. To achieve national coverage, the program was universalized and renamed Universal Immunization Program (UIP) in 1985. Measles vaccine was included in the program and typhoid vaccine was discontinued. The program was introduced in a phased manner from 1985 to cover all districts in the country by 1990. The objective of UIP was to cover at least 85 percent of all infants (0-12 months) and immunize more than 90 million pregnant women and 83 million infants over a five year period. The program was given the status of 'National Technology Mission' in 1986 by the Indian government, accelerating the program and increasing coverage rapidly. UIP became a part of the Child Survival and State Motherhood (CSSM) Program in 1992 and Reproductive and Child Health (RCH) Program in 1997.

Figure 1.1 shows the national immunization coverage rates for 5 vaccines and diseases. The figure illustrates that the UIP was not only large in scale, leading to 50-60 percentage point increases in coverage, but the program was also implemented within a relatively short time period. For example, in the case of tuberculosis, the BCG vaccine coverage increased from 8 in 1985, at the inception of the UIP to 66 by 1990. According to UNICEF vaccine preventable diseases (VPDs) kill

approximately 2 million children every year. Increase in immunization coverage and declines in incidence of vaccine preventable diseases are expected to decrease the infant and under-five mortality in India, providing a unique opportunity to study the effect of child mortality on fertility and education. The large and discrete nature of the increase in coverage is likely to provide variation in child mortality that is less likely to be confounded with long run trends. I compare child mortality in 1980, before the program, to child mortality in 1990, the target date for near universal coverage. Additionally, I exploit the variation across states by comparing states with high and low initial child mortality. The assumption is that the near universal coverage mandate within a short time span resulted in differential treatments across states. Across 26 states in India, the realized declines in child mortality were larger in states with higher levels of initial child mortality, suggesting a convergence led by the immunization program. This pattern is shown in Figure 1.2. The relationship between initial child mortality and declines in child mortality between 1980 and 1990 is statistically significant at 1 percent level in a univariate regression.

1.4 Data

I use 1992-1993, 1998-1999 and 2005-2006 Demographic Health Surveys for India. The DHS surveys are nationally representative samples designed to gather detailed demographic and fertility information of women. In the survey, adult women (aged 15 to 49) answer retrospective questions about each birth, including year of birth, gender, and year of death, in case the child died. I use women's birth histories to construct the number of births in each year of their lives.

In addition to using retrospective birth histories, I construct child mortality rates by state and year. Under-five mortality rate is the probability of a child dying before

reaching the age of five, if subject to age-specific mortality rates of that state and year. Under-five mortality is calculated as follows:

$$MR_{0-5} = (1 - \prod_{a=0}^5 (1 - \frac{death_{a,t,s}}{children_{a,t,s}})) * 100 \quad (1.1)$$

where a refers to age, t refers to year and s refers to state. In my sample, average under-five mortality is 13.3 percent in 1980 and 10.1 percent in 1990.⁷ Child mortality in 1980 by state is taken as state initial mortality, which is the mortality rate before the immunization program.

I use immunization coverage estimations by WHO/UNICEF. Based on the official data reported by countries WHO/UNICEF provides its own estimations after correcting for potential biases. Estimations are available for BCG (tuberculosis vaccine), the third dose of diphtheria and tetanus toxoid and pertussis vaccine (DTP3), the third dose of polio vaccine (Pol3) - the first dose of measles vaccine (MCV). In my estimations, I use the average immunization rate for tuberculosis. However, in robustness section I show similar results for other vaccines. I also use state level GDP as additional controls. Data on state level GDP between 1960 and 2000 were obtained from CDs distributed by the EPW Research Foundation.

Table 1.1 reports the descriptive statistics. Panel A shows the means of the variables such as age, births last year, under-5 mortality, and immunization coverage rates that prevailed in 1980 and 1990. Panel B shows the means organized by birth cohort and in particular report separately the education levels of birth cohorts born prior to the program (1970-74, 1975-79, 1980-84) and after the program (1985-89, 1990-92). The bottom panel, Panel C, illustrates the variation in average number of births, under-5 mortality, and state GDP across states. I first calculate weighted

⁷The regression coefficient on the relationship between my country level estimations and official data between 1960-2005 is 1.03 (0.100).

averages by state and report the unweighted means and standard deviations of state averages. The table shows the dramatic increase in immunization rates as well as the decline in under-5 mortality which fell from 11.3 percent to 8.7 percent. The table also illustrates the large variation in mortality rates across states with the highest mortality state starting with under-5 mortality of 20.4 percent and lowest mortality state exhibiting a rate of 4.3 percent in 1980. By 1990, the gap between the highest and lowest had shrunk to 11.5 percentage points.

1.5 Empirical Framework and Results

1.5.1 Effect of Immunization on Child Mortality

I first examine the impact of the immunization program on child mortality rates. I begin with a simple difference in differences framework comparing states with high and low initial mortality rates. To examine the impact on child mortality, I created cell level data by state and year. The years I examine are 1980, the pre-treatment year, and 1990, the post-treatment year. I run the following regression to show the effect of immunization program on child mortality.

$$\begin{aligned}
 \text{Child Mortality}_{st} = & \alpha + \beta \text{Immunization Rate}_t \times \text{Initial Mortality}_s \quad (1.2) \\
 & + \theta \text{Immunization Rate}_t + \zeta \text{Initial Mortality}_s + \mathbf{X}'_{st} \gamma + \epsilon_{st}
 \end{aligned}$$

where s denotes state and t denotes year. Immunization rate is the country level immunization rate for tuberculosis in year t .⁸ State initial mortality is the under-5 mortality rate in the state in 1980. The interaction of the country-level immunization

⁸In the empirical analysis reported in the main body of the paper, I use country-level tuberculosis immunization rates. The results on mortality are the same regardless of the choice of vaccine. In Table 1.9 I report the results on fertility and education using other diseases.

rate with state initial mortality identifies the effect of the immunization program. The idea behind this specification is that the immunization program resulted in differential treatment across states, with those with the highest initial mortality getting the largest treatment. In addition to this simple basic specification, I also add state fixed effects and year fixed effects in columns (2) and (3) which more flexibly control for unobserved heterogeneity across states and time-varying factors. These fixed effects absorb the effect of state initial mortality and immunization rate, respectively. Additionally, in column (3) I control for GDP per capita which varies by state and year. It is important to control for economic activity when studying the effect of mortality on development. Richer and more developed states can have both lower mortality levels and also have lower fertility and higher human capital investment. Therefore, not controlling for economic activity may lead to omitted variable bias.

In all specifications in Table 1.2, there is a significant negative relationship between child mortality and the interaction between country level immunization rate and state initial mortality. It suggests that states with higher initial mortality benefited more from immunization program and experienced larger decreases in child mortality. According to column 3, a 10 percentage point increase in immunization rate reduces child mortality by .625 percentage points for states at the mean initial mortality (11.3 percent) relative to states with zero initial mortality. As mentioned in the previous section, Figure 1.2 presents the visual representation of these results. The figure shows that states with higher initial mortality experienced larger declines in child mortality.

One issue is whether this relationship reflects longer run convergence across states. I examine this issue by investigating the same relationship for 1970-1980. Figure 1.3 depicts the relationship between state initial mortality in 1980 and changes in

child mortality between 1970 and 1980, before the immunization program was in action. The figure reveals that prior to the immunization program, there was no significant difference in changes in child mortality across states identified as high and low mortality states in 1980. Another concern is that there may have been other health interventions that also impacted child mortality. To the best of my knowledge, there were no other major health interventions under way during the same time period. However, to check for this possibility, I examine whether adult mortality also exhibit similar patterns of improvement as child mortality. Since immunization should not have impacted adult mortality during this period, this provides a good falsification test. Figure 1.4 shows changes in adult mortality by initial state child mortality in 1980. Reassuringly, we see no systematic pattern for adult mortality changes.

1.5.2 Effects of Immunization on Fertility and Education

The previous section showed that states with higher initial mortality had larger declines in child mortality over the period 1980-1990. In this section, I examine whether the immunization program and the reduction in child mortality impacted fertility of mothers as well as the education level of the treated cohorts. To examine the impact on fertility of mothers, I specify the following reduced form regression that builds on the previous specification on child mortality.

$$Fertility_{ist} = \alpha + \beta Immunization Rate_t x Initial Mortality_s + \theta Immunization Rate_t + \zeta Initial Mortality_s + \mathbf{X}'_{ist} \gamma + \epsilon_{ist} \quad (1.3)$$

where i denotes the individual woman, s denotes state and t denotes year. The fertility variable is number of births last year for the individual woman. I examine fertility of women who are 15 to 49 years old, controlling for a quadratic function in

age.

Table 1.3 shows the results. I report the results of the basic specification above in column (1). The effect of the interaction between immunization rate and initial mortality, which I interpret as the program effect, is negative and significant in all specifications. In other words, states with higher initial child mortality realized larger declines in fertility relative to the states with lower initial mortality. Column 2 shows the results with state and year fixed effects. Column 3 is my preferred specification as it controls for both state and year fixed effects in addition to state GDP. According to the preferred specification in column (3), a 10 percentage point increase in immunization rate reduces births by .005 for states at mean initial mortality relative to states with zero initial mortality.⁹ Tuberculosis immunization rate increased approximately 63 percentage points so the decline in fertility due to the program was .032. Average births last year was .217 in 1980 so this translates into an approximate decline of 14.7 percent. Since the decline in fertility between 1980 and 1990 reported in Table 1.1 is approximately 33 percent $((.16-.24)/.24)$ the program can explain approximately 45 percent of the total decline in fertility over my sample period.

I next turn to the effect of the immunization program on education levels of the treated cohort.

$$Education_{isc} = \alpha + \beta Immunization Rate_c x Initial Mortality_s + \theta Immunization Rate_c + \zeta Initial Mortality_s + \mathbf{X}'_{isc} \gamma + \epsilon_{isc} \quad (1.4)$$

where i denotes the individual, s denotes state and c denotes birth cohorts. Education is measured as years of schooling. Included birth cohorts are 1980-1984, 1985-1989 and 1990-1992. Cohorts 1985-1989 and 1990-1992 are the treated cohorts since the

⁹I multiplied the coefficient -.004 by mean initial mortality of 11.3. The immunization rate variable ranges between 0 and 1 so a 10 percentage point increase translates into $.1 \times .05 = .005$.

immunization program began in 1985. For these regressions, I align the state GDP per capita to be that which prevailed while the cohort was of school-going age, 5 to 15. Since states with higher economic activity may have higher education and lower child mortality due to other underlying reasons, it is important to control for state GDP during the periods that the cohort is of school-going age.

Table 1.4 shows the results for females and males. Turning to the education results for females, the coefficient on “Immunization Rate” is positive indicating that years of schooling increased as immunization rate increased. The large negative coefficient on “State Initial Mortality” indicates that states with the highest initial mortality in 1980 have the lowest female schooling levels. The interaction terms which I interpret as the effect of immunization program on female years of schooling is positive and significant in all specifications. Again, in my preferred specification which includes state GDP reported in column (3), I find that a 10 percentage point increase in the immunization rate increases schooling for females by .134 years for states at mean initial mortality relative to states with zero initial mortality. The program resulted in a 63 percentage point increase in immunization rate from 1980 to 1990 so the total increase in female schooling due to the immunization program could be as large as 0.84 years. This translates into an approximate increase of 14 percent given that average schooling among females for the 1980-84 cohort was around 6 years.

1.5.3 Effect of Child Mortality on Fertility and Education - 2SLS Estimates

In the previous section, I examined the reduced form effects of the immunization program on child mortality, women’s fertility, and education levels of the treated cohorts. In this section, I exploit the large exogenous change in immunization rates

brought about by the program as an instrument for reductions in child mortality. This 2SLS specification allows me to gauge the size of the mortality effect on fertility and education. Before reporting the 2SLS estimates, I first report the estimates from simple OLS specifications relating state-specific child mortality rates to fertility as in the following:

$$Fertility_{ist} = \alpha + \beta Child\ Mortality_{st} + \mathbf{X}'_{ist}\gamma + D_s + D_t + GDP_{st} + \epsilon_{ist} \quad (1.5)$$

where child mortality is the mortality rate for children younger than 5 years old by state and year. D_s and D_t control for state and year fixed effects, respectively. \mathbf{X}'_{ist} includes age and age-squared; and ϵ_{ist} is the error term.

Results are reported in columns (1)-(3) in Table 1.5. The OLS coefficients are all positive but in specifications which include year and state fixed effects, the coefficients are no longer significant. The OLS coefficients, however, may suffer from omitted variables bias or reverse causality. Columns (4)-(6) report the instrumental variables estimates. I instrument child mortality with the interaction of county level immunization rate and state initial mortality. First stage estimates are negative and statistically significant at 1 percent level, suggesting that immunization program led to larger declines in child mortality in states with higher initial mortality. Second stage results show that there is a positive and significant relationship between child mortality and number of births per year. According to my preferred specification in column (6), 1 percentage point decrease in child mortality reduces births by .010, which is approximately a 5 percent decline in births.¹⁰

Similarly, I run the following OLS estimation for education:

¹⁰Basically, the IV estimate of child mortality on fertility is the ratio of reduced form effect to the first-stage effect of immunization program on child mortality.

$$Education_{isc} = \alpha + \beta Child\ Mortality_{sc} + \mathbf{X}'_{isc}\gamma + D_s + D_c + GDP_{sc} + \epsilon_{isc} \quad (1.6)$$

Table 1.6 displays the results for females. OLS estimations, in columns (1)-(3), show that declines in child mortality increases years of schooling for females. 2SLS estimates are reported in columns (4)-(6) and are considerably larger than the OLS estimates.¹¹ According to the coefficient reported in column (6), the size of the child mortality effect on schooling is substantial. A 1 percentage point decrease in child mortality increases female schooling by 0.4 years. Since average female schooling for the 1980-84 cohort was around 6 years, this translates into a 7 percent rise in female schooling. Table 1.7 shows the results for males. For males, the OLS coefficients are positive and significant, columns (2) and (3), once I control for year and state fixed effects. The 2SLS coefficients, however, reported in columns (4)-(6) are not significant.

It is important at this point to compare my educational outcomes to others in the literature as well as discuss possible channels. In their paper on the effect of maternal mortality reductions on female education, Jayachandran and Lleras-Muney (2009) report that an increase of a year in life expectancy at 15 results in .11 years of additional schooling, an increase of 3 percent.¹² Converting my reductions in child mortality to changes in life expectancy, I estimate that an increase of a year in life expectancy at birth translates into about a 1.5 percentage point reduction in under-5 mortality. According to the estimates in column (3) this would result in about 0.6

¹¹First stage results are slightly different in fertility regressions (Table 1.5) and education regressions. The difference is stemming from the fact that two datasets are structured differently and cover different years.

¹²Kumar (2009a) also provides estimates of UIP on education outcomes. He finds no impact on years of schooling but finds that the fraction who complete primary school decreases while the fraction who complete secondary school increases.

years of additional schooling for females, an increase of about 10 percent.¹³ While my estimates appear to be large, it is difficult to make a direct comparison since I focus on life expectancy at birth while Jayachandran and Lleras-Muney (2009) focus on life expectancy at age 15. They also point out that infant and child mortality should not affect human capital investments through the horizon effect since such investments do not begin until after age 5. While the horizon effect is not in operation, Table 1.3 and Table 1.5 demonstrated that the immunization program reduced fertility. Education of the treated cohort can increase if parents trade off the number of children for higher quality, i.e. better educated children. One thing we need to establish, however, is that the number of children per household did indeed decline over this period. In other words, taking into account reductions in both child mortality and fertility, is it the case that *net* fertility declined? I examine this question in Figure 1.5. The left axis (square symbol) refers to the average number of children born per woman each year who are expected to survive to age 5. The right axis (diamond symbol) refers to the net reproduction rate, which is defined as the number of daughters born per woman who are expected to survive. Unlike the previous measure, the net reproduction rate incorporates information on adult survival probability.¹⁴ Figure 1.5 shows that by both measures, net fertility declined in India over this period. The simultaneous reduction in net fertility and increase in education is consistent with a quantity-quality tradeoff which is triggered by shocks to child mortality which works its way through the fertility channel.

There are several caveats to the above interpretation. First, to the extent that the

¹³A 1 percentage point decrease in child mortality increases female schooling by 0.4 years, therefore, 1.5 percentage point decrease in child mortality leads to 1.5*0.4=0.6 years, which is 0.6/6=10%

¹⁴Net reproduction rate is calculated as:

$$NRR_{a,t} = \sum_{a=0}^{49} \frac{Female\ Offsprings_{a,t} * (1 - q_{a,t})}{Women_{a,t}}$$

where a, t denote to age and year, respectively. q is the probability of survival to age a. Adult mortality estimates by state and year is from Saikia et al. (2010).

reductions in fertility increase household resources that are used to educate older or younger children in the household, my estimates would tend to understate the impact on education. Second, another important channel is the impact of immunization on child morbidity. As cited in the introduction, a number of papers have found arguably causal impact of improvements in child health on educational outcomes (Miguel and Kremer (2004), Bleakley (2007)). To the extent that a direct morbidity channel exists, my estimates here would overstate the impact of child mortality changes on education.

The last issue to be addressed is my finding that the immunization program increased years of schooling for girls, but not boys. Gender-specific effects which resulted from programs have been documented in other studies (see Chin (2005), Banerjee et al.(2004), Munshi and Rosenzweig (2004), Duflo (2003), among many others). It may be the case that reduction in fertility frees up time for girls since they are more likely to have child care duties. Table 1.1 shows that there is substantial gender gap in schooling between males and females. My results suggest that female children are the marginal children who benefit from family resources which become available with the reductions in fertility.

1.6 Robustness

My identification strategy rested on the assumption that the immunization program had larger treatment effects on states with higher initial child mortality. I did indeed find that states with higher initial mortality in 1980 had larger declines in mortality from 1980 to 1990, as well as larger declines in fertility and increases in female schooling. One concern is that states with different initial child mortality also might

have different underlying trends in these outcome variables irrespective of the immunization program. I test for pre-trends before the immunization program by running the following regressions using the data between 1970 and 1980:

$$Fertility_{ist} = \alpha + \beta State\ Mortality_{s1980} X\ 1980_Dummy_t + \theta 1980_Dummy_t + \zeta State\ Mortality_{s1980} + \mathbf{X}'_{ist} \gamma + \epsilon_{ist} \quad (1.7)$$

and

$$Education_{isc} = \alpha + \beta State\ Mortality_{s1980} X\ 1980_Dummy_c + \theta 1980_Dummy_c + \zeta State\ Mortality_{s1980} + \mathbf{X}'_{isc} \gamma + \epsilon_{isc} \quad (1.8)$$

where *State Mortality*_{s1980} refers to state child mortality in 1980 and *1980_Dummy* takes value 1 in 1980, and 0 in 1970.

Results are shown in Table 1.8. Column (1) shows that there is indeed a pre-existing trend in fertility before the immunization program. However, the relationship has the opposite sign of the relationship found in Table 1.3. According to this table, states with higher initial mortality were experiencing smaller declines in fertility relative to the states with lower mortality. This behavior is consistent with a high child mortality environment in which the pre-cautionary motive is strong and parents continue to have children to guarantee a specific number of survivors (Kalemli-Ozcan 2002, 2003). However, this gap reverses sign with the program as shown in Table 1.3 and states with higher initial mortality start to experience larger decreases in fertility. This suggest that I may be understating the program effect and my estimates provide a lower bound for the program effect on fertility. In columns (2) and (3), I test

for pre-trends in female and male education. Cohorts born between 1970-1980 were not exposed to the program, and therefore, the increase in education across cohorts should not differ systematically across high and low mortality states. Indeed, the coefficients are not statistically significant indicating that there are no pre-existing trends in female/male years of schooling correlated with initial state mortality.

Next, I show the robustness of my results to the choice of vaccine included in the immunization and report these results in Table 1.9. In my analysis, I used BCG (vaccine for tuberculosis) coverage as country level immunization rate. In this section, I repeat the main specifications for fertility and education using DTP1, DTP3, Pol3 and MCV vaccines.¹⁵ As can be seen from the table, the results are quantitatively similar and robust to the choice of vaccine.

1.7 Conclusion

In 1985, India launched a large-scale immunization program which increased the country level immunization rates against basic childhood diseases by 50-60 percentage points. This major health intervention resulted in large declines in under-5 child mortality. Using fertility histories from Demographic Health Surveys for 1992-93, 1998-1999 and 2005-2006, I study the impact of this large shock in child mortality on fertility and human capital investment decisions of the household. Under the assumption that the program had a disproportionately large impact on states with high initial child mortality, I use the country level immunization rate interacted with state initial mortality as an instrument for child mortality. My estimates suggest that a 1 percentage point decline in child mortality reduces births last year by about

¹⁵DTP1 and DTP3 are first and third doses for diphtheria, tetanus and pertussis; Pol3 is the third dose for polio and MCV is the vaccine for measles.

5 percent. I check for pre-program trends and find no systematic relationship between initial child mortality in the state and child mortality changes prior to 1980. I also find no systematic pattern relating adult mortality changes during this period to state's initial child mortality in 1980, arguing against the relevance of other unobserved health improvements. While fertility was declining during the prior period, it was declining more slowly in high mortality states, suggesting that I may be understating the impact of child mortality on fertility. My estimates also suggest that a 1 percentage decline in child mortality increases female schooling by 0.4 years, an increase of about 7 percent. Interestingly, I find no significant effect on male schooling. While previous papers have focused on the "horizon effect" where declines in adult mortality increase human capital accumulation, my findings point to another mechanism of the quantity/quality tradeoff that works through child mortality and fertility declines.

Table 1.1: Descriptive Statistics

Panel A: Country Level Data:								
	<u>1980</u>	<u>1990</u>						
Age	22.5	24.8						
Number of Births Last Year	0.240	0.163						
Number of Births Last Year (15-35 years old)	0.243	0.186						
Under-five Mortality (%)	13.3	10.1						
Immunization Coverage (%)								
BCG	5.8	69						
DTP1	31.6	83						
DTP3	10.8	62.2						
Pol3	6.8	61.6						
MCV	10	55.2						
Panel B: Cohort Level Data:								
	<u>Female schooling</u>		<u>Male schooling</u>					
	At Age 15	At Age 20	At Age 15	At Age 20				
Birth Cohort 1970-74		4.2		7.2				
Birth Cohort 1975-79	4.5	5.1	6.0	7.8				
Birth Cohort 1980-84	5.3	5.9	6.4	8.0				
Birth Cohort 1985-89 (treated cohort)	5.9	6.3	7.0	8.2				
Birth Cohort 1990-92 (treated cohort)	6.4		7.2					
C. State Level Data:								
	<u>1980</u>				<u>1990</u>			
	Mean	Std. Dev.	Min	Max	Mean	Std. Dev.	Min	Max
Number of Births Last Year	0.217	0.039	0.153	0.304	0.157	0.029	0.098	0.208
Number of Births Last Year (15-35 years old)	0.220	0.039	0.156	0.308	0.178	0.030	0.118	0.234
Under-five Mortality (%)	11.3	4.1	4.3	20.4	8.7	2.9	3.3	14.8
State GDP	6402.7	2819.6	2959.9	15087.3	8831.2	4070.7	3829.4	20352.6

Notes: Females and males between ages 15 and 49 are included. State level means are taken and unweighted means and standard deviations across 26 states are reported in the table. State GDP per capita is in 1993 prices. Under-five mortality rate is the probability of a child dying before reaching the age of five, if subject to age-specific mortality rates of that state and year. Immunization rate for each disease between ages 0 and 1 is at country level. DTP1 and DTP3 are first and third doses for diphtheria, tetanus and pertussis; Pol3 is the third dose for polio and MCV is the vaccine for measles.

Table 1.2: Effect of Immunization Program on Child Mortality

	Under-five Mortality		
	(1)	(2)	(3)
Immunization Rate x State Initial Mortality	-0.607*** (0.123)	-0.607*** (0.174)	-0.553** (0.249)
State Initial Mortality	1.035*** (0.007)		
Immunization Rate	2.722 (1.601)		
Year FE	No	Yes	Yes
State FE	No	Yes	Yes
State GDP	No	No	Yes
R^2	0.920	0.960	0.962
N	52	52	44

Notes: 26 states are included in the regressions. Under-five mortality rate is the probability of a child dying before reaching the age of five, if subject to age-specific mortality rates of that state and year. Under-five mortality, immunization rate and initial mortality is aggregated to state by year level. Included years are 1980 and 1990. State initial mortality is the under-five mortality in 1980 by state. Immunization rate is the country level immunization rate for tuberculosis by year. State GDP per capita by year is in 1993 prices. Robust standard errors clustered at state level are in parentheses. Asterisks denote significance levels (* 0.1 ** 0.05 *** 0.01).

Table 1.3: Effect of Immunization Program on Fertility

	Dependent Variable: Number of Births		
	(1)	(2)	(3)
Immunization Rate x State Initial Mortality	-0.003*** (0.001)	-0.003*** (0.001)	-0.004*** (0.001)
State Initial Mortality	0.009*** (0.001)		
Immunization Rate	-0.042*** (0.013)		
Year FE	No	Yes	Yes
State FE	No	Yes	Yes
State GDP	No	No	Yes
R^2	0.040	0.043	0.043
N	325868	325868	304076

Notes: Women between ages 15 and 49 are used in the regressions. Included years are 1980 and 1990. State initial mortality is the under-five mortality in 1980 by state. Immunization rate is the country level immunization rate for tuberculosis by year. State GDP per capita by year is in 1993 prices. Regressions are weighted survey sampling weights. Robust standard errors clustered at state level are in parentheses. Asterisks denote significance levels (* 0.1 ** 0.05 *** 0.01).

Table 1.4: Effect of Immunization on Education

	(1)	(2)	(3)
Dependent Variable: Years of Schooling			
Panel A: FEMALES			
Immunization Rate x State Initial Mortality	0.094 (0.058)	0.121*** (0.037)	0.119*** (0.040)
State Initial Mortality	-1.052*** (0.260)		
Immunization Rate	0.437 (0.858)		
R^2	0.061	0.123	0.124
N	84728	84728	76672
MALES			
Immunization Rate x State Initial Mortality	0.028 (0.037)	0.040 (0.034)	-0.022 (0.030)
State Initial Mortality	-0.444** (0.183)		
Immunization Rate	0.185 (0.646)		
R^2	0.026	0.064	0.065
N	82719	82719	74797
Cohort FE	No	Yes	Yes
State FE	No	Yes	Yes
State GDP	No	No	Yes

Notes: Dependent variable is years of schooling. State initial mortality is the under-five mortality in 1980 by state. Immunization rate is the average country level immunization rate for tuberculosis when the cohort is younger than 12 months. Included birth cohorts are 1980-1984, 1985-1989 and 1990-1992. State GDP per capita when cohort is between ages 5 and 15 is in 1993 prices. Regressions are weighted by survey sampling weights. Robust standard errors clustered at state level are in parentheses. Asterisks denote significance levels (* 0.1 ** 0.05 *** 0.01).

Table 1.5: Effect of Child Mortality on Fertility

	Dependent Variable: Number of Births						
	(1)	(2)	(3)		(4)	(5)	(6)
	OLS				2SLS		
Under-five Mortality	0.010*** (0.001)	0.001 (0.003)	0.001 (0.003)		0.008*** (0.002)	0.008*** (0.002)	0.010** (0.004)
R^2	0.040	0.043	0.043		0.040	0.042	0.042
N	325868	325868	304076		325868	325868	304076
					First Stage		
Immunization Rate x State Initial Mortality					-0.377*** (0.061)	-0.376*** (0.060)	-0.380*** (0.087)
R^2					0.950	0.983	0.983
N					325868	325868	304076
Year FE	No	Yes	Yes		No	Yes	Yes
State FE	No	Yes	Yes		No	Yes	Yes
State GDP	No	No	Yes		No	No	Yes

Notes: Women between ages 15 and 49 are used in the regressions. Included years are 1980 and 1990. Under-five mortality rate is the probability of a child dying before reaching the age of five, if subject to age-specific mortality rates of that state and year. Under-five mortality is instrumented by the interaction of initial mortality in 1980 and average country level immunization rate. Initial mortality is the under-five mortality in 1980 by state. Immunization rate is the country level immunization rate for tuberculosis. State GDP per capita by year is in 1993 prices. Regressions are weighted by survey sampling weights. Robust standard errors clustered at state level are in parentheses. Asterisks denote significance levels (* 0.1 ** 0.05 *** 0.01).

Table 1.6: Effect of Child Mortality on Female Education

Dependent Variable: Years of Schooling							
	(1)	(2)	(3)		(4)	(5)	(6)
	OLS				2SLS		
Under-five Mortality	-0.454*** (0.078)	-0.185*** (0.047)	-0.185*** (0.049)		-0.365*** (0.108)	-0.392*** (0.038)	-0.396*** (0.037)
R^2	0.084	0.122	0.122		0.082	0.121	0.121
N	84728	84728	83447		84728	84728	83447
					First Stage		
Immunization Rate x State Initial Mortality					-0.415*** (0.041)	-0.387*** (0.021)	-0.388*** (0.020)
R^2					0.877	0.991	0.991
N					84728	84728	83447
Year FE	No	Yes	Yes		No	Yes	Yes
State FE	No	Yes	Yes		No	Yes	Yes
State GDP	No	No	Yes		No	No	Yes

Notes: Dependent variable is female years of schooling. Under-five mortality rate is the average probability of a child dying before reaching the age of five, if subject to age-specific mortality rates of that state when the cohort is between ages 0 and 5. Included birth cohorts are 1980-1984, 1985-1989 and 1990-1992. State GDP per capita is the average GDP per capita when cohort is between ages 5 and 15. State GDP per capita is in 1993 prices. Under-five mortality is instrumented by the interaction of initial mortality in 1980 and average country level immunization rate. Initial mortality is the under-five mortality in 1980 by state. Immunization rate is the average country level immunization rate for tuberculosis when the cohort is younger than 12 months. Regressions are weighted by survey sampling weights. Robust standard errors clustered at state level are in parentheses. Asterisks denote significance levels (* 0.1 ** 0.05 *** 0.01).

Table 1.7: Effect of Child Mortality on Male Education

Dependent Variable: Years of Schooling							
	(1)	(2)	(3)		(4)	(5)	(6)
	OLS				2SLS		
Under-five Mortality	-0.196*** (0.059)	0.182*** (0.055)	0.188*** (0.050)		-0.134 (2.905)	-0.063 (0.073)	-0.042 (0.039)
R^2	0.032	0.064	0.065		0.031	0.063	0.064
N	82719	82719	81545		82719	82719	81545
					First Stage		
Immunization Rate x State Initial Mortality					-0.424*** (0.042)	-0.388*** (0.023)	-0.389*** (0.021)
R^2					0.878	0.991	0.991
N					82719	82719	81545
Year FE	No	Yes	Yes		No	Yes	Yes
State FE	No	Yes	Yes		No	Yes	Yes
State GDP	No	No	Yes		No	No	Yes

Notes: Dependent variable is male years of schooling. Under-five mortality rate is the average probability of a child dying before reaching the age of five, if subject to age-specific mortality rates of that state when the cohort is between ages 0 and 5. Included birth cohorts are 1980-1984, 1985-1989 and 1990-1992. State GDP per capita is the average GDP per capita when cohort is between ages 5 and 15. State GDP per capita is in 1993 prices. Under-five mortality is instrumented by the interaction of initial mortality in 1980 and average country level immunization rate. Initial mortality is the under-five mortality in 1980 by state. Immunization rate is the average country level immunization rate for tuberculosis when the cohort is younger than 12 months. Regressions are weighted by survey sampling weights. Robust standard errors clustered at state level are in parentheses. Asterisks denote significance levels (* 0.1 ** 0.05 *** 0.01).

Table 1.8: Robustness: Pre-trend Tests using 1970-1980 Data

	(1)	(2)	(3)
	Fertility	Education	
	Number of Births	Years of Schooling	
		Females	Males
<i>State Mortality</i> ₁₉₈₀ X 1980_Dummy	0.004*** (0.001)	0.005 (0.017)	-0.014* (0.008)
1980_Dummy	-0.058*** (0.020)	0.959*** (0.304)	0.544*** (0.123)
<i>State Mortality</i> ₁₉₈₀	0.004*** (0.001)	-0.306*** (0.070)	-0.116** (0.046)
R^2	0.040	0.073	0.022
N	151558	184185	175473

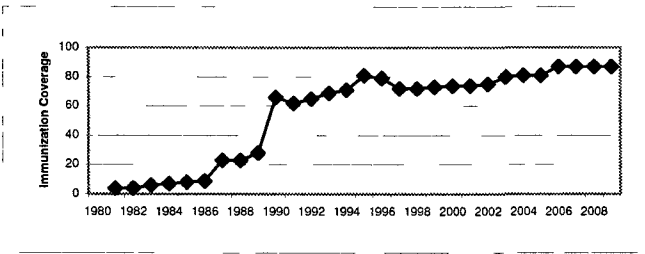
Notes: *State Mortality*₁₉₈₀ is the under-five mortality in 1980 by state. Included years are 1970 and 1980. 1980_Dummy takes value 1 in 1980, and 0 in 1970. Regressions are weighted by survey sampling weights. Robust standard errors clustered at state level are in parentheses. Asterisks denote significance levels (* 0.1 ** 0.05 *** 0.01).

Table 1.9: Robustness: Other Diseases

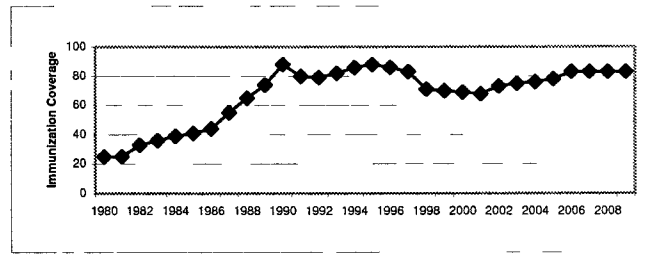
Vaccines	DTP1			DTP3			Pol3			MCV		
	Fertility	Education		Fertility	Education		Fertility	Education		Fertility	Education	
		Female	Male		Female	Male		Female	Male		Female	Male
Initial Mortality x Immunization Rate	-0.005*** (0.001)	0.128*** (0.020)	-0.037** (0.017)	-0.005*** (0.001)	0.140*** (0.019)	-0.015 (0.017)	-0.016*** (0.001)	0.136*** (0.017)	-0.008 (0.015)	-0.002* (0.001)	0.131*** (0.016)	0.003 (0.018)
Year/Cohort FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State GDP	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R^2	0.043	0.123	0.064	0.043	0.123	0.064	0.043	0.123	0.064	0.038	0.127	0.071
N	304076	76672	74797	304076	76672	74797	304076	76672	74797	359184	68716	67146

Notes: In fertility regressions women between ages 15 and 49 are used. Initial mortality is the under-five mortality at 1980 by state. Immunization rate is the country level immunization rate for each disease between ages 0 and 1. Included years are 1980 and 1990. Since measles information available since 1986, 1986 and 1990 are used in fertility regressions for measles vaccine. State GDP per capita by year is at constant 1993 prices. In education regressions included birth cohorts are 1980-1984, 1985-1989 and 1990-1992. Initial mortality is the under-five mortality per 100 children at 1980 by state. State GDP per capita when cohort is between ages 5 and 15 is at constant 1993 prices. DTP1 and DTP3 are first and third doses for diphtheria, tetanus and pertussis, Pol3 is the third dose for polio and MCV is the vaccine for measles. Regressions are weighted by survey sampling weights. Robust standard errors clustered at state level are in parentheses. Asterisks denote significance levels (* 0.1 ** 0.05 *** 0.01).

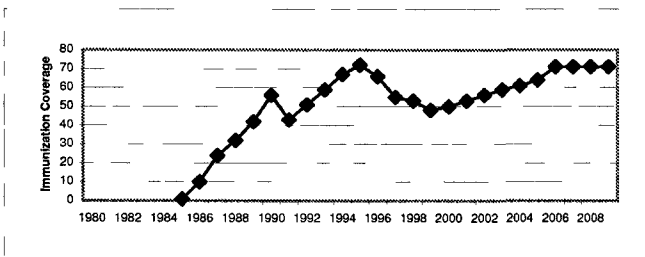
Figure 1.1. National Immunization Coverage in India



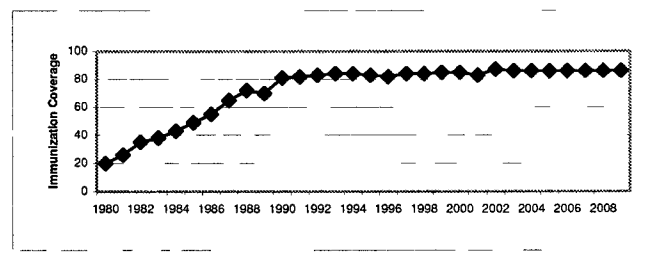
a- BCG (Tuberculosis)



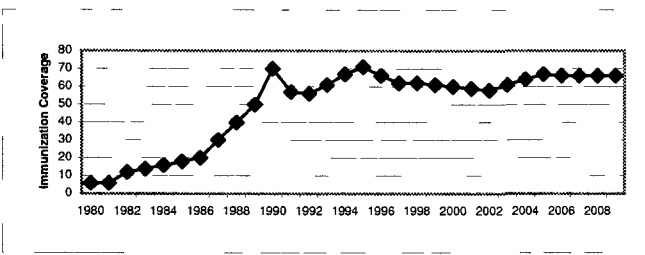
b- DTP1 (Diphtheria, pertussis, tetanus)



c- MCV (Measles)



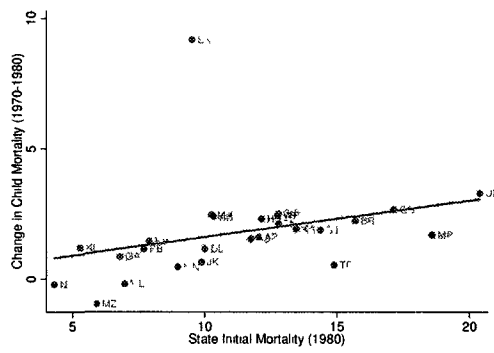
d- Pol3 (Polio)



e- DTP3 (Diphtheria, pertussis, tetanus)

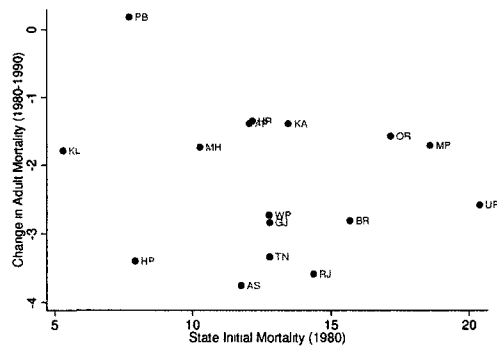
Source: WHO/UNICEF estimates of national immunization coverage.

Figure 1.3: Robustness: Pre-trend Tests using 1970-1980 Data



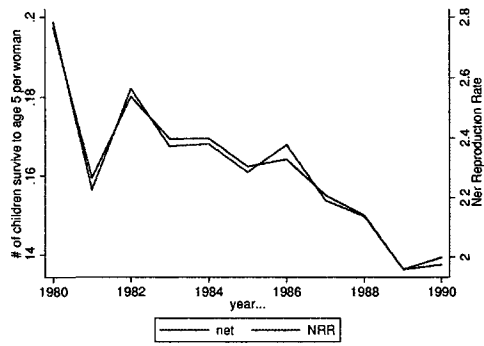
Notes: Each dot represents a state. Mortality rates are the probability of a child dying before reaching the age of five, if subject to age-specific mortality rates of that state and year. The coefficient on initial mortality is not statistically significant at the 5% level.

Figure 1.4: Robustness: Declines in Adult Mortality across States: 1980-1990



Notes: Each dot represents a state. Mortality is the number of deaths per 100 adults. The coefficient on initial mortality is not statistically significant at the 5% level.

Figure 1.5: Decline in Net Reproduction Rate: 1980-1990



Notes: The difference between “number of children survive to age 5 per woman” and “net reproduction rate” is that latter also takes into account the female mortality rates after age 5 including adult mortality.

Chapter 2

Life Expectancy and Economic Development: Evidence from Micro Data

2.1 Introduction

A large literature studies the effect of improving health and life expectancy on economic development and growth. However, as of yet, there is little consensus on this subject either on the theoretical or the empirical front. On the theory side, the standard theory predicts that increased life expectancy increases population which reduces capital-labor ratios and depresses per capita income. On the other hand, endogenous growth models in the tradition of Becker and Barro (1988) suggest that human capital investment and fertility responses may offset the grim predictions of the neoclassical model. In particular, increased life expectancy could lead to a quantity-quality trade-off where parents have fewer children but invest more in the education of their children and their own education (see Kalemli-Ozcan (2003),

Soares (2005), Cervellati and Sunde (2007), Tamura (2006) among others). This suggests that behavioral responses in fertility and human capital investment can offset the decline in capital-labor ratios and productivity.

There is likewise little agreement on the empirical front. While Bloom and Sachs (1998), Gallup, Sachs and Mellinger (1999), Bloom, Canning and Sevilla (2002), Lorentzen, McMillan and Wacziarg (2008) find large effects of increasing life expectancy on growth, a recent paper by Acemoglu and Johnson (2007) find little effect. They instrument changes in life expectancy with dates of global health interventions to combat 15 major diseases. While health interventions increase life expectancy, they find little impact on per capita GDP.¹

I develop a general equilibrium model linking life expectancy to important behavioral variables such as fertility, human capital investment, and labor supply. I extend the models introduced by Zhang and Zhang (2005), who solve education and fertility as endogenous but labor supply as exogenous, and Boucekine et al. (2009), who model fertility and labor supply as endogenous but education as exogenous. My model treats all three as endogenous variables. There are two offsetting effects in the model. On the one hand, an increase in life expectancy leads to a quantity-quality tradeoff where parents have fewer children but invest more in their own education as well as the education of the children.² Individuals also work and save more since they have a higher probability of surviving to old age. This is called the horizon effect. On the other hand, there is a countervailing effect in which increased life expectancy

¹See Cervellati and Sunde (2009) and Bloom, Canning and Fink (2009) for arguments against their results. See also calibration studies by Shastry and Weil (2003), Weil (2007), Ashraf et al. (2008) that conclude that while positive, the effect of improved health on growth is likely to be small.

²Alternatively, parents may have precautionary demand for children and respond by giving fewer births when child mortality falls (Kalemli-Ozcan, 2003). Cervellati and Sunde (2007) identify yet another possible channel where increases in life expectancy raise the returns to human capital for the parents and the opportunity cost of raising children thereby leading to a negative relationship between longevity and fertility.

expands the size of the adult population and lowers wages. Under certain parameter configurations, this general equilibrium effect, which works through lower wages, can increase fertility (since the opportunity cost of bearing and rearing children is lower) and reduce education and labor supply, leading to overall ambiguous effects.

The ambiguous theoretical predictions suggest that the proper understanding of the link between life expectancy and fertility, education, and labor supply, rests on empirical work. The empirical work to this point, however, has been limited by the lack of data. Researchers in this area have largely relied on country-level data (Shastri and Weil (2003), Acemoglu and Johnson (2007), Lorentzen, McMillan and Wacziarg (2008)).³ The empirical work has also largely focused on per capita GDP as the outcome variable.⁴

In this paper, I fill this gap by constructing a new panel data set from Demographic Health Surveys and testing the implications of endogenous growth models that relate life expectancy to fertility, human capital, and labor supply. More specifically, I use 67 Demographic Health Surveys of 28 countries from sub-Saharan Africa taken between the years 1987 and 2007. I calculate fertility rates and child mortality rates from birth histories and adult mortality rates and life expectancy from sibling histories. Based on this information I construct birth and death rates by region, year and age of the mother.⁵

There are several advantages to this data. First, by building mortality rates from sibling histories, I obtain more accurate measures of mortality for some countries. Country level life expectancy measures for developing countries, especially for sub-Saharan Africa, are often not accurate since reliable vital registration data are not available. Estimated infant mortality and under-five mortality rates and an assumed

³See Bloom, Canning, Sevilla (2004) for review of such studies.

⁴Jayachandran and Lleras-Muney (2009) and Soares (2006) are notable exceptions using micro-level data.

⁵Countries and the years included are explained in the Appendix II.

age pattern of mortality are used to calculate life expectancy. My mortality data set is based on actual reported deaths. Secondly, since my data are at the individual level, I can construct age-specific birth and mortality rates by region and exploit variations in the data beyond the usual cross-country variation. Finally, the data set I have constructed is unique in terms of the number of countries covered (28) and the period covered (1975 to 2007).⁶ Most of the related studies do not cover sub-Saharan Africa due to lack of reliable data. However my approach enables me to extend my analysis to sub-Saharan Africa. Africa is also an interesting testing ground for these models since adult mortality suffered a large negative shock with the introduction of HIV/AIDS in the mid 1980s. How fertility, education, and labor supply have responded in this environment has been the topic of special interest in a recent set of papers. Young (2005) suggests that similar to the "Black Death" plague in Europe, HIV/AIDS will reduce fertility and population and eventually enhance growth among the affected African nations. Recent papers by Juhn et al. (2009), Fortson (2009) and Fink and Linnemayer (2009) find little impact on fertility and negative impact on education.

To preview my empirical results, I find that adult life expectancy is negatively related to fertility suggesting that the horizon effect dominates the general equilibrium effect and behavioral changes in fertility counter-act the rise in population. While the relationship is negative the size of the effect is small. I find no systematic difference between high HIV countries, which I define as those with greater than 5 percent prevalence rate, and low HIV countries. I find little evidence that fertility declines rapidly through fear of infection as suggested by Young (2005). I also find that life expectancy is positively related to education which again suggests that the horizon

⁶Oster (2010) covers 12 countries and years between 1981 and 2005. Soares (2006) covers Brazil since mid-60s but aggregates the information up to 1996. While they do not examine mortality, Juhn et al. (2009), Fortson (2009) and Fink and Linnemayer (2009) use fertility histories. Coverage is much smaller however.

effect dominates. Finally, I find a weak positive relationship between life expectancy and labor force participation for females, but no relationship among males. Overall, results using this newly constructed data suggests that in the context of sub-Saharan Africa, increases in life expectancy will have a positive impact on growth through fertility and education, but the size of effect will be small.

The rest of the paper is organized as follows. Section 2 lays out the conceptual framework. Section 3 describes the data. Section 4 presents the empirical results. Section 5 concludes.

2.2 The Model

Assume a 3 period over-lapping generations (OLG) model. In period t , individual invests in education, e_t . Human capital h_{t+1} accumulation is given by:

$$h_{t+1} = e_t h_p \tag{2.1}$$

where h_p denotes parental human capital.

In period $t+1$, 1 unit time endowment is allocated among raising children, working and leisure. Individual gives birth to n_{t+1} children, consumes c_{t+1} , saves s_{t+1} , works l_{t+1} and earns w_{t+1} per unit time based on his human capital. Income is given by:

$$y_{t+1} = h_{t+1} l_{t+1} w_{t+1} \tag{2.2}$$

The time cost of child bearing is v , where $v > 0$.⁷ Young adults have p_{t+1} probability of survival to period $t + 2$, where $p \in (0, 1)$. In period $t + 2$, survivors do not work but only consume c_{t+2} out of their savings s_{t+1} .

⁷There is no uncertainty about child survival since my focus is on adult longevity.

The maximization of the utility function subject to the intertemporal budget constraint can be written as follows:

$$\max U(e_t, c_{t+1}, n_{t+1}, l_{t+1}, c_{t+2}) = \frac{(1 - e_t)^{1-\sigma}}{1 - \sigma} + \frac{c_{t+1}^{1-\sigma}}{1 - \sigma} + \frac{(n_{t+1})^{1-\sigma}}{1 - \sigma} + \frac{(1 - vn_{t+1} - l_{t+1})^{1-\sigma}}{1 - \sigma} \quad (2.3)$$

$$+ p_{t+1} \frac{c_{t+2}^{1-\sigma}}{1 - \sigma} \quad (2.4)$$

$$s.t. \quad c_{t+1} + p_{t+1}c_{t+2} = h_{t+1}l_{t+1}w_{t+1}$$

where σ governs risk aversion and intertemporal elasticity of substitution. The first order conditions with respect to e_t , n_{t+1} , l_{t+1} , and will yield the following equations:

$$1 - (1 - n_{t+1}v - n_{t+1}v^{\frac{1}{\sigma}})[1 + [w_{t+1}^{\frac{\sigma-1}{\sigma}} \frac{1}{1 + p_{t+1}} (1 + w_{t+1}^{\frac{\sigma-1}{\sigma}} (1 - n_{t+1}v - n_{t+1}v^{\frac{1}{\sigma}})^{\frac{\sigma-1}{\sigma}} \frac{1}{1 + p_{t+1}})^{\frac{1-\sigma}{\sigma}} (1 + v^{\frac{\sigma-1}{\sigma}})]] = 0 \quad (2.5)$$

$$1 - \left[\frac{(1 - e_t)(1 + p_{t+1})}{e_t w_{t+1}^{\frac{\sigma-1}{\sigma}} h_p^{\frac{\sigma-1}{\sigma}}} \right]^{\frac{\sigma}{\sigma-1}} [1 + [(1 + v^{\frac{\sigma-1}{\sigma}})(w_{t+1}^{\frac{\sigma-1}{\sigma}} h_p^{\frac{\sigma-1}{\sigma}} \frac{1}{1 + p_{t+1}} e_t^{\frac{\sigma-1}{\sigma}})]] = 0 \quad (2.6)$$

$$1 - l_{t+1} - l_{t+1}[(1 + v^{\frac{\sigma-1}{\sigma}})(1 + w_{t+1}^{\frac{\sigma-1}{\sigma}} h_p^{\frac{\sigma-1}{\sigma}} l_{t+1}^{\frac{\sigma-1}{\sigma}} \frac{1}{1 + p_{t+1}})^{\frac{1-\sigma}{\sigma}} w_{t+1}^{\frac{\sigma-1}{\sigma}} h_p^{\frac{\sigma-1}{\sigma}} \frac{1}{1 + p_{t+1}}] = 0 \quad (2.7)$$

Proposition 1: An exogenous increase in adult survival probability p_{t+1} lowers fertility n_{t+1} and increases education e_t and labor supply l_{t+1} , for any $\sigma > 0.5$, as shown in Figures 2.1, 2.2 and 2.3.⁸ The intuition is as follows. In a low mortality

⁸See Appendix I for derivations.

environment, parents have more incentive to invest in themselves and their children, since the horizon over which human capital investments can be realized by them and by their children is longer. This is called the “horizon effect.” Labor supply is also increasing due to this horizon effect, since with a longer life span there are more years to save and consume for retirement, leading to an increase in the labor supply.⁹

Proposition 2: The effect of an increase in wages on fertility, education and labor supply decision is ambiguous. Fertility increases with wages only if $\sigma > 1$. Education decreases with wages when $\sigma > 1$, and labor supply also decreases with wages when $\sigma > 1$. Figures 2.4, 2.5, and 2.6 summarizes the different effects of wages on fertility, education and labor supply given the σ parameter.

The intuition for this ambiguity is straightforward. In order for higher wages to decrease fertility, the substitution effect, which lowers fertility through increasing the opportunity cost of rearing children, needs to dominate the income effect which increases fertility through a higher amount of resources available to bring up more children. And this is the case only if $\sigma < 1$. As returns to education increases due to higher wages individuals have more incentive to invest in themselves. Likewise, with higher wages opportunity cost of leisure is higher, therefore more people would prefer to work. But again for these effects to be prevalent, the substitution effect needs to be dominating. If $\sigma < 1$, elasticity of intertemporal substitution is high enough that substitution effect dominates income effect and labor supply increases.

In general, the empirical estimates suggest that σ is around 3.¹⁰

⁹There also can be an indirect effect of mortality on labor force participation stemming from an increase in human capital investment as in Soares (2008).

¹⁰Hansen and Singleton (1983) reports relative risk aversion estimates between 0.68 and 0.97, Szpiro (1986) shows that it is between 1.2 and 1.8, Mankiw (1985) shows even larger estimates in the range of 2.44 to 5.26, Halek and Eisnehauer (2001) finds 3.75. On the theoretical front, assumptions both below or above 1 are common. While Becker, Philipson and Soares (2005), Doepke (2005), Boucekkine et al. (2009), Chakraborty and Das (2009) among many others, assume that $\sigma < 1$, Cordoba and Ripoll (2009), Fonseca et al. (2009), to name a few, assume that $\sigma > 1$.

2.2.1 General Equilibrium Implications:

As outlined above, exogenous shocks to mortality and wages may induce opposing effects on fertility, education and labor supply. To show the general equilibrium effect, I assume a Cobb-Douglas production function:

$$Y_t = K_t^\alpha (l_t L_t h_p)^{1-\alpha} \quad (2.8)$$

where K_t is the capital stock, L_t is the active population, l_t is the labor supply, and α is the capital share. Here, I assume no technological progress and full capital depreciation in one period. The factors get paid their marginal products and population growth is given by n_t . The capital accumulation and wages are given by:

$$K_{t+1} = L_t s_t \quad (2.9)$$

$$w_{t+1} = (1 - \alpha) \left(\frac{s_t}{n_t l_{t+1}} \right)^\alpha \quad (2.10)$$

In Proposition 1, I showed that an increase in adult survival probability reduces fertility and increases labor supply and education. However, from the above equation, one can see that an increase in labor supply leads to a decrease in wages. So, in general equilibrium a reduction in adult mortality can lead to labor surplus and lower wages.¹¹ If $\sigma > 1$, lower wages induced by higher labor supply due to reduced mortality will also reduce fertility. Combined with the direct positive effect of reduced mortality on fertility the net effect of mortality and wage changes on fertility will be a reduction of fertility. However, if $0.5 < \sigma < 1$, horizon effect and wage effect work

¹¹A well-researched example in the history is the Black Death in the late fourteenth century. Black Death is estimated to have reduced Britain's population to about half of its pre-plague level in three decades. With a declining labor force, real wages rose rapidly during the plague years, and then remained high throughout the fifteenth century (Herlihy (1997), Young (2005)).

in the opposite directions giving an ambiguous effect on fertility. While longer time horizon shifts quantity-quality trade-off towards less children, lower wages increase fertility due to lower opportunity cost. Similarly, while longer time horizon increases incentive to get more schooling and work more, lower wages reduce incentive to do so. On the other hand, if $\sigma > 1$, both lower wages and higher survival probability reduce fertility and increase education and labor supply.

Following table summarizes the theoretical predictions of the model:

Panel A: $0.5 < \sigma < 1$			
	Fertility	Education	Labor Supply
Longer Horizon	-	+	+
Lower Wages	+	-	-
Net Effect	+/-/0	+/-/0	+/-/0
Panel B: $\sigma > 1$			
	Fertility	Education	Labor Supply
Longer Horizon	-	+	+
Lower Wages	-	+	+
Net Effect	-	+	+

2.3 Data

I use 67 Demographic Health Surveys (DHS) from 28 countries in sub-Saharan Africa with survey years spanning between 1987 and 2007.¹² The DHS surveys are nationally representative samples designed to gather detailed demographic and fertility information of women. Similar to Young (2005), I use birth histories to construct a time-series of birth rates by region and age of the mother. In the survey, adult

¹²Details about the surveys used are in Appendix II.

women (aged 15 to 49) answer retrospective questions about each birth, including year of birth, gender, and year of death, in case the child died. I divide the number of births in the region and year and age of mother by the total number of women in the region and year and age category. Infant and child mortality rates are also constructed using birth histories. Using birth and death years, I calculate the fraction of children who were alive at the beginning of each year and died during that year. By this method, I obtain a time-series of regional child mortality rates between 1975 and 2007.

In order to accurately estimate adult mortality rates, complete vital registration system through which all deaths are reported to a government agency is crucial. However, vital registration systems in developing countries, particularly in sub-Saharan Africa, are underdeveloped. Therefore, reliable mortality data for these countries are not available. For this reason, I use sibling history modules of the DHS to construct time-series of adult mortality rates. In most of the DHS surveys, adult women are asked about their siblings. Respondents give information on sibling's gender, date of birth and death. Using this information, I construct age-specific mortality rates that vary by year, region, and gender. Again, I divide the number of siblings who died at each age in each region in each year by the number siblings alive at each age at the beginning of the year in each region.¹³ The constructed average age-specific death rates are shown in Tables 2.1 and 2.2. Numbers accord with the usual age profile with high infant mortality rates followed by U-shaped death rates. Furthermore, as established in the literature, female mortality rates are lower than male mortality rates for all age groups in my sample.

Although Bicego (1997), Timaeus and Jasseh (2004) show that sibling histories

¹³While my data technically go back to the 1960s for some age groups, for comparability across years I start my analysis in 1975 when the oldest women in the sample are 39. I examine fertility for 15-39 year old women. While the changing age distribution is also of concern for mortality calculations, robustness checks using more recent data led to similar qualitative results.

usually estimate mortality rates fairly accurately, there might still be concerns about potential biases. Estimations can be downward biased since families in which all adult members died are not represented. Dead people with no siblings are also not included since there is no one to report on them. Additionally, families with favorable mortality rates are misrepresented in the sample since experience of sisters might be counted several times. On the other hand, this methodology may overestimate the overall death rate since respondents, who are obviously alive, are not taken into consideration in the mortality calculation. Trussell and Rodriguez (1990) show that with a random sample from a population, these potential biases cancel each other and this method gives unbiased mortality estimates if mortality rates are not correlated with the number of siblings. Of course, this assumption does not hold if people in high mortality areas have more children or if children in crowded households are less likely to survive because of the limited resources. However, when I incorporate the adjustments proposed in Gadikou and King (2006) to eliminate these biases my results still hold.¹⁴ Additionally, as stated in Oster (2010), this correlation is more of a concern for child mortality, not for adult mortality. To examine further the potential biases, in Figure 2.7 I compare mortality rates calculated from sibling histories to the official death data available for Zimbabwe, which is the only country in sub-Saharan Africa in my sample that has official vital registration data. The figure shows that the match between the two sources is very strong, meaning that calculating mortality rates from sibling histories provides quite accurate estimates when the official data is not available.¹⁵

The theory calls for life expectancy measures since I model forward looking behavior by individuals who incorporate the remaining life span. I convert the age-specific mortality rates to life expectancy measures. Life expectancy is the number of years

¹⁴Results are available from author upon request.

¹⁵See Oster(2010) for more evidence on the reliability of sibling mortality histories.

an individual in a region can be expected to live if he/she experienced the current age-specific death rates of the region throughout his/her life. The exact formulas used for calculation are in Appendix II. Since survivor bias becomes a bigger problem at older ages, I censor life expectancy at age 60.

Constructed life expectancy measures are shown in Table 2.3 and 2.4. The tables show that life expectancy both at birth and at age 15 have declined on average among countries in sub-Saharan Africa. Indeed among high HIV/AIDS countries, life expectancy is lower than 25 years ago.

2.4 Empirical Analysis and Results

2.4.1 Effect of Life Expectancy on Fertility

I examine the relationship between fertility and life expectancy as follows:

$$Fertility_{art} = \alpha + \beta LE_{art} + \gamma_a + \lambda_r + \eta_t + \theta_{rt} + \epsilon_{art}, \quad (2.11)$$

where a refers to age group, r refers to region and t refers to year. Fertility is the number of children born per 1000 women by age group, region, and year. Since in the earlier years of my data I only observe relatively young women (the oldest woman being 39 in 1975) I only include women who are 15-39 years old in the fertility regressions. LE_{art} is the number of average years an individual is expected to live conditional on surviving to age-group a in year t in region r . For a woman of a particular age group, mortality shocks that affect older individuals impact her life expectancy but not mortality shocks that affect younger individuals. While in principle I have annual observations, serial correlation is likely to lead me to understate the standard errors. I therefore use data in five-year intervals

corresponding to 1975, 1980, 1985, 1990, 1995, 2000, and 2005. I also aggregate single-year age categories into five 5-year age groups. The regression controls for age group dummies, region fixed effects, year fixed effects as well as region- specific trends.¹⁶

Results are reported in Table 2.5. The table shows that for the overall sample, the relationship between adult life expectancy and fertility is negative, which suggests that the horizon effect dominates the general equilibrium wage effect and women respond to increases in life expectancy by having fewer children. How large is the effect? While the relationship is negative, the size of the effect is small. A one year increase in adult life expectancy reduces the annual birth rate by 0.244 percentage points. Over 25 years from ages 15-39, a woman will have 0.061 ($=25 \cdot .00244$) fewer children. Table 2.4 showed that life expectancy at 15 decreased by an average of two years in my sample. This suggests that total fertility rate may have risen by 0.12 children as a result.

Recently, several papers have examined the effect of HIV/AIDS on fertility. While Young (2005, 2007) finds that HIV/AIDS decreases fertility, papers using newly-available individual level testing data find that regional HIV prevalence representing mortality risk has little measurable effect on fertility (Juhn et al. (2009) and Fortson(2009)). Although I do not directly attempt to test the effect of HIV/AIDS on fertility, in the following section I divide the countries in sub-Saharan Africa by their country-level prevalence using data from UNAIDS/WHO. My two groups are "high" (5 percent or greater) and "low" (less than 5 percent) HIV prevalence countries. The results are reported in columns (2) and (3) of table 2.5. For both high and low HIV countries, I find a negative significant effect of increasing life expectancy on fertility. In the context of high HIV countries which are experiencing *reductions* in adult life

¹⁶I report results where each cell is weighted by number of observations in the cell. Results which do not weight by cell size are qualitatively similar and are not reported.

expectancy, this means that women are *increasing* fertility in response to mortality shocks.

My findings on fertility for high HIV countries seem somewhat at odds with the lack of fertility effects documented in the previous papers. There are two potential explanations. The first point is that while I do find a significant effect, the impact of adult life expectancy on fertility is economically small. Second, while previous papers have examined the impact of HIV infection, my measure here is mortality, which is likely to be more correlated with AIDS and advanced stages of the disease. Some researchers have found that HIV prevalence in the community has little impact on sexual behavior suggesting that individuals may lack knowledge of the disease and their own infected status.¹⁷ Uncertainty or lack of knowledge about the consequences of the disease is not likely to be a factor with mortality.

My results differ from Young (2005) which finds a large decline in fertility due to HIV related deaths in South Africa. The results here also differ from Boucekkine et al. (2009) which documents a large negative effect of adult mortality on fertility. The difference in my results from the latter paper may stem from the fact that I use within-country cross-region variation in fertility and mortality rates, while Boucekkine et al. (2009) uses country level data.

2.4.2 Effect of Life Expectancy on Education

I run the following regression covering the years 1975 to 2007 to examine the relationship between education and life expectancy:

¹⁷For example, Oster (2005), using DHS data find little change in sexual behavior since the onset of the epidemic. Luke and Munshi (2006) find that married men in highly infected communities in Kenya have similar numbers of non-marital partners as single men.

$$Education_{cr} = \alpha + \beta LE_{15} + \lambda_r + \gamma_c + \zeta_{cr} + \epsilon_{cr}, \quad (2.12)$$

where c refers 5-year birth cohorts and r refers to region. Education is the average years of schooling by cohort by region. LE_{15} is the average number of years a 15 years-old is expected to live when the cohort is 5 to 15 years old. The choice of average life expectancy between ages 5 to 15 is to account for the fact that education decision is made during school-going age and not afterwards. Thus, a 30 year old woman who experiences an increase in life expectancy will not increase her own education because she is beyond the age where investments are made. The change in life expectancy will however impact educational investment for her children. Additionally, the above regression includes region and cohort fixed effects and region-specific cohort trends.

Results are shown in Table 2.6. Columns (1) and (2) show the results for all countries in my sample. Both females and males increase education as life expectancy increases with the size of the effect being larger for males. Intuitively, as the horizon to reap the benefits of the investment lengthens, returns to schooling increases and, on average, individuals go to school for a longer period of time. The coefficients of 0.054 and 0.110 imply that 1 year increase in life expectancy increases education by 0.05 years for females and 0.1 years for males, which approximately correspond to 1% and 2% increases relative to the sample means. It is plausible to find a bigger effect for males as it has been established in the literature that under unfavorable conditions and scarcity of resources, household resources are distributed in favor of boys.

In a paper that examines the impact of HIV/AIDS on education, Fortson (2011) finds that regions with higher HIV prevalence experienced relatively larger declines in education over time. My results above are consistent with these findings. In columns

(3)-(6), I examine whether the positive impact of life expectancy on education remains even when I look within high and low HIV countries. Columns (3) and (4) show that in low HIV countries, the robust and positive impact of life expectancy on education remains. However, when I look within high HIV countries, the relationship between life expectancy and education remains although no longer significant.

Among high HIV countries, life expectancy at age 15 declined by approximately 4 years from 40.7 in 1990 to 36.9 in 2000. The size of the education effect suggests that average years of schooling could decline by as much as 0.1-0.4 years in these countries. Given that average years of schooling is slightly over 6 years in these countries, and had not increased much since 1975, this is a sizable effect.

2.4.3 Effect of Life Expectancy on Labor Force Participation

In this section I examine the relationship between labor force participation and adult life expectancy. On the one hand, the horizon effect encourages work and savings as the probability of surviving to old age increases. On the other, the general equilibrium effect of increased population size will reduce wages and decrease the incentive to work. In the case of sub-Saharan Africa where life expectancy is falling over time, this would lead to a rise in wages and individuals will increase labor force participation as long as the substitution effect dominates, i.e. $\sigma < 1$. While examining the impact of life expectancy on wages directly would be the ideal first step, wage data is not available in the DHS. I test the effect of increased life expectancy on the participation decision as follows:

$$LFPR_{art} = \alpha + \beta LE_{15rt} + \eta_r + \lambda_t + \epsilon_{art}, \quad (2.13)$$

where a refers to age group, r refers to region and t refers to year. Labor force

participation is the fraction of people who report to be working. LE_{15rt} is life expectancy at 15 in region r and year t . The regression also includes year and region fixed effects. Since work histories are not available labor force participation is observed only during the survey year. In order to control for region fixed effects, I include in the regressions only those countries where at least two surveys are available. This significantly reduces my sample size relative to fertility and education regressions reported in the previous tables.

Table 2.7 shows the results for both females and males. As the table shows, life expectancy increases labor force participation among females but not males. The coefficient is marginally significant. When I divide the sample into low and high HIV countries, the results remain for women in high HIV countries but not for women in low HIV countries.

2.5 Conclusion

There is an on-going debate in the literature on the impact of health improvements and increased life expectancy on development and growth. The current paper contributes to this debate on several grounds. First, I write down a comprehensive general equilibrium model which incorporates endogenous fertility, education, and labor supply. Second, I construct a new panel data set to test the implications of this model. More specifically, I use birth and sibling histories to construct age-specific birth rates and age-specific mortality rates at the country region level in sub-Saharan Africa. Since reliable vital statistics are often not available, the data I construct may be the most accurate data on fertility and mortality available for many of these countries. Since I build the panel from micro data, I can exploit within country regional variation which is not often used in this literature. The data

is also unique in terms of the number of surveys (67) and countries (28) covered and span of years it encompasses, 1975-2007. My results suggest that increases in life expectancy reduce fertility, increase education, and increase labor force participation of women (but not men). Overall, my empirical results suggest that in sub-Saharan Africa, increases in life expectancy will have a positive impact on growth through fertility and education and possibly labor supply, but the effect will be small. On the other hand, my results rule out the possibility that recent shocks to adult mortality in high HIV countries will reduce fertility, increase labor productivity, and lead to faster growth.

Figure 2.1: Response of Fertility to Life Expectancy

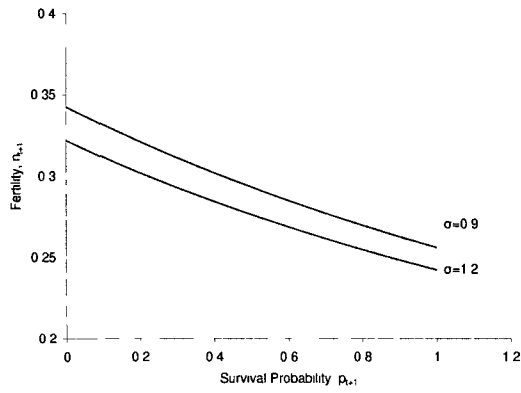


Figure 2.2: Response of Education to Life Expectancy

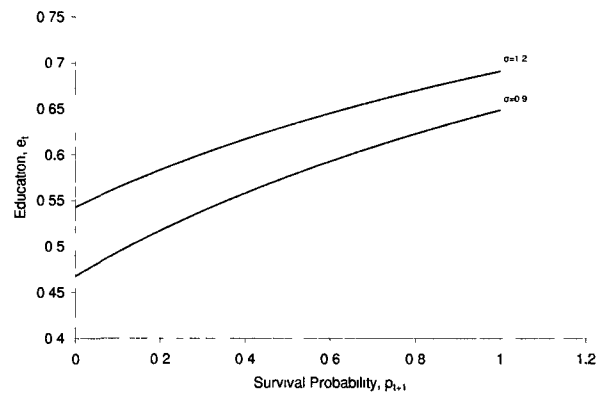


Figure 2.3: Response of Labor Supply to Life Expectancy

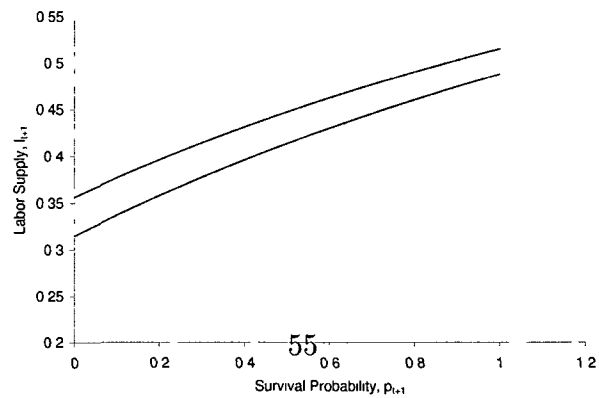


Figure 2.4: Response of Fertility to Wages

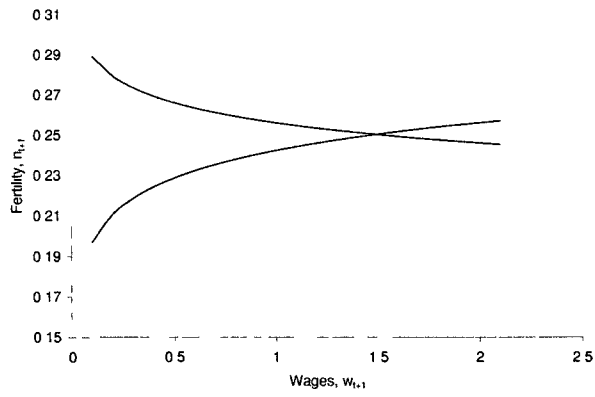


Figure 2.5: Response of Education to Wages

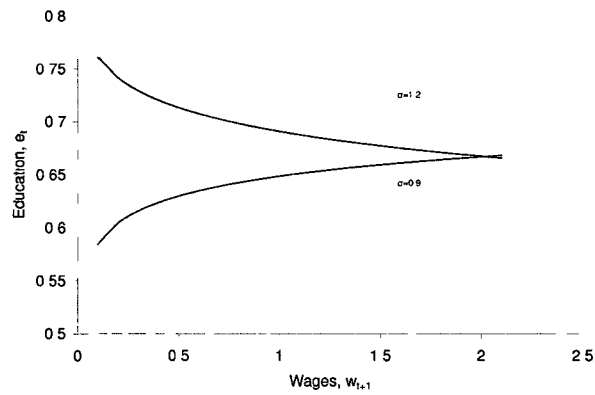


Figure 2.6: Response of Labor Supply to Wages

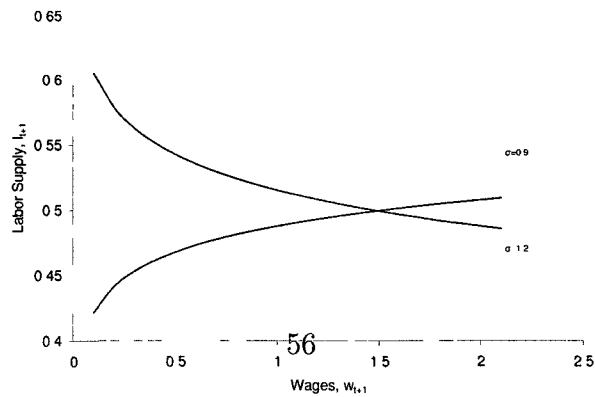
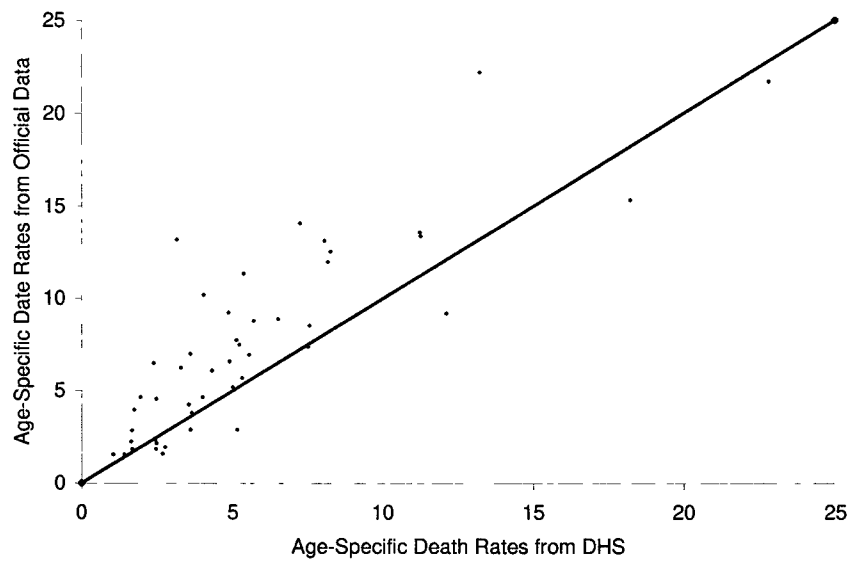


Figure 2.7: Reliability of Sibling Histories: Zimbabwe



Notes: Notes: Official death rates for Zimbabwe for years 1981, 1985, 1989, 1990, 1991 and 1994 from Feeney(2001) are compared to death rates calculated from sibling histories. Age-specific date rates for men and women are calculated for ages 15-24, 25-34, 35-44 and 45-54 and rates are per 1000 population. Line represents the 45-Degree Line.

Table 2.1: Mean Age-Specific Death Rates

	1975	1980	1990	2000
Ages 0-4	136.67	138.50	150.01	152.25
Ages 5-9	24.38	21.74	19.74	18.27
Ages 10-14	15.63	13.68	12.29	12.56
Ages 15-19	15.10	13.09	13.60	13.57
Ages 20-24	17.00	14.65	16.28	20.72
Ages 25-29	18.73	15.32	19.52	31.45
Ages 30-34	24.23	21.14	27.16	45.24
Ages 35-39	24.69	23.18	27.75	54.50
Ages 40-44	39.49	22.65	36.51	65.56
Ages 45-49	29.73	33.42	39.05	65.44
Average Mortality (0-49)	34.57	31.74	36.19	47.96
Average Adult Mortality (15-49)	24.14	20.49	25.70	42.36

Notes: Average mortality rates across 52 surveys from 28 countries are shown. Death rates are per 1000 population.

Table 2.2: Mean Age-Specific Death Rates by Gender

Panel A: Female Age-Specific Death Rates				
	1975	1980	1990	2000
Ages 0-4	126.46	128.68	141.84	146.62
Ages 5-9	23.54	21.33	18.90	17.44
Ages 10-14	14.42	12.29	11.80	12.32
Ages 15-19	14.63	12.69	13.96	14.13
Ages 20-24	15.02	13.04	16.52	22.44
Ages 25-29	16.50	13.76	18.15	34.72
Ages 30-34	26.56	17.68	24.12	43.99
Ages 35-39	18.43	17.01	25.46	50.06
Ages 40-44	33.86	17.97	31.01	54.27
Ages 45-49	44.81	28.60	34.75	56.39
Average Female Mortality (0-49)	33.42	28.31	33.65	45.24
Average Female Adult Mortality (15-49)	24.26	17.25	23.42	39.43
Panel B: Male Age-Specific Death Rates				
	1975	1980	1990	2000
Ages 0-4	146.07	147.64	157.73	157.53
Ages 5-9	25.14	22.02	20.58	19.11
Ages 10-14	16.69	15.00	12.77	12.81
Ages 15-19	15.48	13.53	13.27	12.99
Ages 20-24	18.63	16.20	16.05	19.04
Ages 25-29	20.83	16.79	21.02	28.19
Ages 30-34	23.37	24.44	30.28	46.51
Ages 35-39	30.74	27.95	29.92	59.24
Ages 40-44	39.81	27.42	42.31	77.26
Ages 45-49	17.02	40.75	43.33	75.13
Average Male Mortality (0-49)	35.38	35.18	38.73	50.78
Average Male Adult Mortality (15-49)	23.70	23.87	28.02	45.48

Notes: Average mortality rates across 52 surveys from 28 countries are shown. Death rates are per 1000 population. Panel A displays the rates for females and panel B displays male mortality rates.

Table 2.3: Life Expectancy at Birth

	1975	1980	1990	2000
Benin	44.7	46.5	46.5	47.0
Burkina Faso	45.6	46.0	46.3	45.7
Cameroon	44.9	48.4	48.7	43.3
CAR	43.9	47.0	42.6	
CDR	47.8	48.8	46.2	42.6
Chad	45.0	43.5	43.6	43.4
Congo	39.5	51.8	49.4	44.0
Cote d'Ivoire	47.6	49.7	46.4	47.2
Ethiopia	43.1	43.9	43.5	
Gabon	49.1	50.0	51.0	50.8
Guinea	42.3	42.5	42.8	44.4
Kenya	52.1	52.4	51.4	46.5
Lesotho	46.8	48.5	51.2	43.9
Liberia	54.1	53.5	41.3	47.5
Malawi	44.8	46.3	43.1	40.9
Mali	42.1	41.7	42.2	44.9
Mozambique	47.3	46.0	44.7	44.7
Namibia	51.8	52.4	51.1	48.1
Niger	41.2	41.7	40.9	43.9
Rwanda	47.4	45.3	42.0	36.8
Senegal	44.0	45.8	48.4	46.9
South Africa	51.4	52.1	53.0	
Swaziland	52.2	51.5	54.6	44.7
Tanzania	49.1	47.9	47.0	46.1
Togo	45.6	46.1	46.8	
Uganda	45.4	44.2	43.8	43.7
Zambia	50.5	49.2	43.6	39.7
Zimbabwe	52.6	51.9	53.0	46.1
Average	46.8	47.7	46.6	44.7
Low HIV Countries(HIV _i =%5)	45.1	46.2	44.7	44.5
High HIV Countries (HIV _i =%5)	48.8	49.4	48.8	44.9

Notes: Life Expectancy measures are calculated from 52 surveys from 28 countries.

Table 2.4: Life Expectancy at 15

	1975	1980	1990	2000
Benin	40.5	41.6	41.8	41.0
Burkina Faso	41.0	41.0	41.6	40.7
Cameroon	38.2	42.8	41.7	38.2
CAR	36.8	40.6	37.3	
CDR	41.4	42.1	41.0	38.8
Chad	41.0	39.8	40.7	38.8
Congo	28.8	42.6	39.9	37.7
Cote d'Ivoire	40.7	42.5	39.9	39.4
Ethiopia	38.6	40.0	38.0	
Gabon	40.8	41.5	41.3	40.9
Guinea	39.6	39.9	41.3	40.2
Kenya	42.6	43.0	42.3	38.7
Lesotho	38.4	38.9	41.1	35.3
Liberia	44.3	43.2	36.8	41.3
Malawi	41.3	42.1	39.7	34.2
Mali	40.3	40.2	40.7	41.4
Mozambique	40.6	40.4	41.6	39.0
Namibia	40.8	41.7	40.5	37.4
Niger	40.3	40.3	41.6	41.7
Rwanda	42.0	39.4	37.7	37.2
Senegal	41.1	41.3	41.7	41.4
South Africa	42.2	42.3	42.1	
Swaziland	41.6	40.3	42.0	35.4
Tanzania	42.2	41.3	41.3	38.2
Togo	41.5	42.3	41.8	
Uganda	39.5	39.5	38.3	36.7
Zambia	42.3	42.4	38.7	33.4
Zimbabwe	42.1	41.6	41.7	35.1
Average	40.2	41.2	40.6	38.3
Low HIV Countries(HIV _i =%5)	40.1	41.2	40.3	40.0
High HIV Countries (HIV _i %5)	40.7	41.3	40.7	36.9

Notes: Life Expectancy measures are calculated from 52 surveys from 28 countries. Life expectancy at 15 is the average expected years of life individuals are expected to live between 15 and 60 conditional on surviving to 15.

Table 2.5: Effect of Life Expectancy on Fertility: 1975-2005

	<u>All Countries</u>	<u>Low HIV Countries</u>	<u>High HIV Countries</u>
	(1)	(2)	(3)
Dependent Variable: Age-Specific Birth Rates by Region by Year			
Life Expectancy	-2.442*** (0.335)	-2.202*** (0.362)	-1.544** (0.707)
Controls	Region FEs, Year FEs, Region-Specific Time Trends, Age Group FE		
Mean	216	217	215
R ²	0.814	0.822	0.817
N (Region x Year x Age Group)	5971	3096	2875

Notes: Dependent variable is the number of children per 1000 women by age-group by region and by year. Life Expectancy is the average number of years individuals in an age-group are expected to live conditional on surviving to age a. Years used in the regressions are 1975, 1980, 1985, 1990, 1995, 2000 and 2005. Women are grouped into 5 groups according to their ages: 15-19, 20-24, 25-29, 30-34, 35-39. Column (1) includes 28 countries, column (2) includes 15 countries in sub-Saharan Africa with less than 5 % HIV prevalence, and column (3) includes 13 countries in sub-Saharan Africa with more than 5% HIV prevalence. HIV prevalence is as of 2008 and taken from UNAIDS/WHO database. Regressions have region, year and age group fixed effects and region-specific time trends. Regressions are weighted by the number of women by age group by region by year. Standard errors are clustered at region by year level. Asterisks denote significance levels (*=.10, **=.05, ***=.01).

Table 2.6: Effect of Life Expectancy on Education: 1975-2007

	<u>All Countries</u>		<u>Low HIV Countries</u>		<u>High HIV Countries</u>	
	Female	Male	Female	Male	Female	Male
	(1)	(2)	(3)	(4)	(5)	(6)
Dependent Variable: Years of Schooling by Cohort by Region						
LE at 15 when cohort was 5-15	0.054*** (0.021)	0.110*** (0.024)	0.044*** (0.022)	0.074*** (0.027)	0.091* (0.048)	0.075 (0.052)
Controls	Region FEs, Cohort FEs, Region-Specific Cohort Trends					
Mean	4.6	5.6	3.3	4.8	6.1	6.5
R^2	0.986	0.970	0.981	0.965	0.980	0.960
N (Cohort x Region)	1110	1042	583	553	527	489

Notes: Dependent variable is the average years of schooling by region by cohort. Life expectancy variable is the number of years a 15 years-old is expected to live when the cohort is 5 to 15 years old. Columns (1) and (2) include 28 countries, columns (3) and (4) include 15 countries in sub-Saharan Africa with less than 5 % HIV prevalence, columns (5) and (6) include 13 countries in sub-Saharan Africa with more than 5% HIV prevalence. HIV prevalence is as of 2008 and taken from UNAIDS/WHO database. 5-year birth cohorts are 1970-1974, 1975-1979, 1980-1984, 1985-1989, and 1990-1994. Regressions are weighted by the population in year-region-sex cell. Standard errors are clustered at region by year level. Asterisks denote significance levels (*=.10, **=.05, ***=.01).

Table 2.7: Effect of Life Expectancy on Labor Force Participation

	<u>All Countries</u>		<u>Low HIV Countries</u>		<u>High HIV Countries</u>	
	Female (1)	Male (2)	Female (3)	Male (4)	Female (5)	Male (6)
Dependent Variable: Percentage Working by Age Group by Region by Year						
Life Expectancy at 15	0.010* (0.005)	0.003 (0.005)	0.008 (0.008)	-0.003 (0.005)	0.017* (0.008)	-0.018 (0.012)
Controls	Region FEs, Year FEs, Age Group FE					
Mean	0.566	0.710	0.586	0.739	0.540	0.681
R^2	0.819	0.821	0.867	0.829	0.838	0.871
N (Region x Year x Age Group)	2926	2246	1407	1154	1519	1092

Notes: Dependent variable is the proportion of females/males that are working by age group by region by year. Life expectancy at 15 is the number of years a 15 years old is expected to live conditional on surviving to 15. Only countries with more than 1 survey are used in the regressions. Columns (1) and (2) include 20 countries, columns (3) and (4) include 12 countries in sub-Saharan Africa with less than 5 % HIV prevalence, columns (5) and (6) include 8 countries in sub-Saharan Africa with more than 5% HIV prevalence. HIV prevalence is as of 2008 and taken from UNAIDS/WHO database. Regressions are weighted by the population in year-region-age group and sex cell. Standard errors are clustered at region by year level. Asterisks denote significance levels (*=.10, **=.05, ***=.01).

A-1 Appendix I

Using intertemporal budget constraint and considering $C_{t+1} = C_{t+2}$

$$c_{t+1} + p_{t+1}c_{t+2} = e_t h_p l_{t+1} w_{t+1} \quad (\text{A-1})$$

$$c_{t+1} = \frac{e_t h_p l_{t+1} w_{t+1}}{1 + p_{t+1}} \quad (\text{A-2})$$

Using first-order conditions for n_{t+1} and l_{t+1}

$$(1 - v n_{t+1} - l_{t+1})^{-\sigma} = \lambda e_t h_p w_{t+1} \quad (\text{A-3})$$

$$n_{t+1}^{-\sigma} = v \lambda_{t+1} e_t h_p w_{t+1} \quad (\text{A-4})$$

$$n_{t+1}^{-\sigma} = v \left(\frac{e_t h_p l_{t+1} w_{t+1}}{1 + p_{t+1}} \right)^{-\sigma} e_t h_p w_{t+1} \quad (\text{A-5})$$

$$n = e_t^{\frac{\sigma-1}{\sigma}} w_{t+1}^{\frac{\sigma-1}{\sigma}} l_{t+1} h_p^{\frac{\sigma-1}{\sigma}} v^{-1/\sigma} (p_{t+1} + 1)^{-1} \quad (\text{A-6})$$

Using first-order conditions for e_t and l_{t+1}

$$(1 - e_t)^{-\sigma} = \lambda_{t+1} h_p l_{t+1} w_{t+1} \quad (\text{A-7})$$

$$(1 - e_t)^{-\sigma} = \left(\frac{e_t h_p l_{t+1} w_{t+1}}{1 + p_{t+1}} \right)^{-\sigma} h_p l_{t+1} w_{t+1} \quad (\text{A-8})$$

$$e_t = (1 + h_p^{\frac{\sigma-1}{\sigma}} l_{t+1}^{\frac{\sigma-1}{\sigma}} w_{t+1}^{\frac{\sigma-1}{\sigma}} (p_{t+1} + 1)^{-1})^{-1} \quad (\text{A-9})$$

Combining Eq.(A-3) and (A-6) and (A-9) yields a single equation for l_{t+1} :

$$1 - l_{t+1} - l_{t+1} \left[(1 + v^{\frac{\sigma-1}{\sigma}}) \left(1 + w_{t+1}^{\frac{\sigma-1}{\sigma}} h_p^{\frac{\sigma-1}{\sigma}} l_{t+1}^{\frac{\sigma-1}{\sigma}} \frac{1}{1 + p_{t+1}} \right)^{\frac{1-\sigma}{\sigma}} w_{t+1}^{\frac{\sigma-1}{\sigma}} h_p^{\frac{\sigma-1}{\sigma}} \frac{1}{1 + p_{t+1}} \right] = 0 \quad (\text{A-10})$$

Using Newton-Raphson iteration method it can be numerically proofed that:

$$\frac{dl_{t+1}}{dp_{t+1}} > 0 \text{ if } \sigma > 0.5 \quad (\text{A-11})$$

and

$$\frac{dl_{t+1}}{dw_{t+1}} < 0 \text{ if } \sigma > 1 \quad (\text{A-12})$$

$$\frac{dl_{t+1}}{dw_{t+1}} > 0 \text{ if } 0.5 < \sigma < 1 \quad (\text{A-13})$$

To find an equation for fertility:

$$n_{t+1}^{-\sigma} = v(1 - vn_{t+1} - l_{t+1})^{-\sigma} \quad (\text{A-14})$$

$$l_{t+1} = 1 - n_{t+1}v - n_{t+1}v^{1/\sigma} \quad (\text{A-15})$$

Combining Eq.(A-10) and (A-15) gives a single equation for n_{t+1} in terms of exogenous variables:

$$1 - (1 - n_{t+1}v - n_{t+1}v^{1/\sigma}) \left[\left[w_{t+1}^{\frac{\sigma-1}{\sigma}} h_p^{\frac{\sigma-1}{\sigma}} \frac{1}{1+p_{t+1}} \left(1 + w_{t+1}^{\frac{\sigma-1}{\sigma}} h_p^{\frac{\sigma-1}{\sigma}} (1 - n_{t+1}v - n_{t+1}v^{1/\sigma})^{\frac{\sigma-1}{\sigma}} \frac{1}{1+p_{t+1}} \right)^{\frac{1-\sigma}{\sigma}} \right. \right. \\ \left. \left. (1 + v^{\frac{\sigma-1}{\sigma}}) \right] + 1 \right] = 0 \quad (\text{A-16})$$

Numerical iteration method yields

$$\frac{dn_{t+1}}{dp_{t+1}} < 0 \text{ if } \sigma > 0.5 \quad (\text{A-17})$$

Similarly

$$\frac{dn_{t+1}}{dw_{t+1}} < 0 \text{ if } 0.5 < \sigma < 1 \quad (\text{A-18})$$

$$\frac{dn_{t+1}}{dw_{t+1}} > 0 \text{ if } \sigma > 1 \quad (\text{A-19})$$

Finally to find an equation for education:

$$(1 - e_t)^{-\sigma} = \lambda_{t+1} h_p l_{t+1} w_{t+1} \quad (\text{A-20})$$

$$(1 - e_t)^{-\sigma} = \left(\frac{e_t h_p l_{t+1} w_{t+1}}{1 + p_{t+1}} \right)^{-\sigma} h_p l_{t+1} w_{t+1} \quad (\text{A-21})$$

$$l = \left[\frac{(1 - e_t)(1 + p_{t+1})}{e_t w_{t+1}^{\frac{\sigma-1}{\sigma}} h_p^{\frac{\sigma-1}{\sigma}}} \right]^{\frac{\sigma}{\sigma-1}} \quad (\text{A-22})$$

Combining Eq.(A-6), (A-10) and (A-22) one can obtain the corresponding equation for schooling decision e_t in terms of exogenous variables:

$$1 - \left[\frac{(1 - e_t)(1 + p_{t+1})}{e_t w_{t+1}^{\frac{\sigma-1}{\sigma}} h_p^{\frac{\sigma-1}{\sigma}}} \right]^{\frac{\sigma}{\sigma-1}} [1 + [(1 + v^{\frac{\sigma-1}{\sigma}})(w_{t+1}^{\frac{\sigma-1}{\sigma}} h_p^{\frac{\sigma-1}{\sigma}} \frac{1}{1 + p_{t+1}} e_t^{\frac{\sigma-1}{\sigma}})]] = 0 \quad (\text{A-23})$$

It can be numerically proofed that:

$$\frac{de_t}{dp_{t+1}} > 0 \text{ if } \sigma > 0.5 \quad (\text{A-24})$$

and

$$\frac{de_t}{dw_{t+1}} < 0 \text{ if } \sigma > 1 \quad (\text{A-25})$$

$$\frac{de_t}{dw_{t+1}} > 0 \text{ if } 0.5 < \sigma < 1 \quad (\text{A-26})$$

A-2 Appendix II

A-2.1 Surveys Used:

Table A1 lists the 67 Demographic Health Surveys (DHS) from 28 countries used in the regressions. Datasets are available from Measure DHS, ICF Macro, at www.measuredhs.com.

A-2.2 Age-Specific Death Rate Calculation:

Age-specific death rate is the total number of deaths of a specified age in a region divided by the population of the same age in the same geographic area and multiplied by 1000. For example, to find the mortality rate of 30 years old in 1994 in region A, I divide the number of 30 years-old siblings died in 1994 in region A by the number of 30 years-old siblings alive at the beginning of 1994, and multiply by 1000.

When annual numbers of deaths for specific ages are small (< 10 or 20), calculated age-specific mortality rates may be too unstable or unreliable for analysis. To eliminate this noise and volatility across years, I calculate death rates by taking three-year averages. For example, to find the mortality rate of 30 years old in 1994 in region A, I divide the number of 30 years-old siblings died between 1993 and 1995 in region A by the number of 30 years-old siblings alive between 1993 and 1995. Usage of multiple surveys matched on the regions increases the sibling history information, thus increases the robustness of death rate computations.

Table A-1: Surveys Used in the Paper

Country	Number of Survey Years	Survey Years
Benin	3	2006*, 2001, 1996*
Burkina Faso	3	2003*, 1998*, 1992
Cameroon	3	2004*, 1998*, 1991
Central African Republic	1	1994/1995*
Chad	2	2004*, 1996*
Congo	1	2005*
Cote d'Ivoire	2	2005*, 1994*
Democratic Republic of the Congo	1	2007*
Ethiopia	2	2005*, 2000*
Gabon	1	2000*
Guinea	2	2005*, 1999*
Kenya	4	2003*, 1998*, 1993, 1989
Lesotho	1	2004*
Liberia	1	2007*
Malawi	3	2004*, 2000*, 1992*
Mali	4	2006*, 2001*, 1995/1996*, 1987
Mozambique	2	2003*, 1997*
Namibia	3	2006/2007*, 2000*, 1992*
Niger	3	2006*, 1998, 1992*
Rwanda	2	2005*, 2000*
Senegal	4	2006, 2005*, 1996, 1992*
South Africa	1	1998*
Swaziland	1	2006*
Togo	2	1998*, 1988
Uganda	4	2006*, 2000*, 1995*, 1988
United Republic of Tanzania	3	2004*, 1999, 1996*
Zambia	4	2007*, 2001*, 1996*, 1992
Zimbabwe	4	2005*, 1999*, 1994*, 1988

Notes: In total my dataset consists of 67 surveys from 28 countries. * indicates that the survey has sibling histories and is used in mortality calculations. Although Nigeria 1999 has sibling histories, Nigeria is not used in the study since the quality of the sibling history module is questionable. In all of the surveys listed here sibling histories are over 98% complete, whereas for Nigeria only 68% of the sibling information is complete.

A-2.3 Life Expectancy Calculation:

Life expectancy is the number of years an individual is expected to live if he/she experienced the current age-specific death rates throughout his/her life. After age-specific death rates are calculated as explained above, they are used to calculate a life table from which one can calculate the probability of surviving to each age. For example, if 10% of a group of siblings alive at age 30 die before reaching to age 31 in 1994, then the age-specific death probability at age 30 in 1994 would be 10%. Life expectancy at age a is then calculated by adding up the survival probabilities at each age. $e(a-b)$ shows the expected years of life between ages a and b conditional on survival to age a . If data is believed to be noisy after an age it can be censored at age b . My results are not sensitive to censoring age. Life expectancy formula is as follows:

$$e(a-b) = \left(\sum_{t=a, a+1, \dots}^b (t+1/2) * \left(\prod_{\tau=a}^{t-1} (1-q_{\tau}) \right) * q_t \right) + b * \left(\prod_{\tau=a}^{b-1} (1-q_{\tau}) \right) * (1-q_b) - a \quad (A-27)$$

where q_t mortality rate for age t , ${}_t p_a$ is the probability of survival from age a to age t . $1/2$ is added to each year assuming, in average, people live half a year at their final age. The term after summation a is subtracted because formula gives the life expectancy beyond age a .

A-2.4 Fertility:

Age-Specific Birth Rates: I use women's retrospective reports of their children in the DHS to reconstruct the number of births per 1000 women at each age in each region in each year. More specifically, to find birth rate of 30 years-old women in region A in year 1994, I divide the total number of births to 30 years-old women in region A

in 1994 by the total number of women, regardless of having birth or not, at age 30 in region A in 1994 and multiply by 1000.

Total Fertility Rate: Calculated age-specific birth rates are used to calculate total fertility rate by region by year. More specifically, total fertility rate (TFR) is the average number of births that women in the sample would have by the time they reach age 49 if they were to give birth at the current age-specific fertility rates. It is the sum of the age-specific fertility rates multiplied by five. TFR formula is as follows:

$$TFR_{r,t} = \sum_{a=1}^{49} \frac{Births_{r,t,a}}{Women_{r,t,a}} \quad (A-28)$$

where r is region, t is year and a is age. $Births_{r,t,a}$ is the total number of births to women at age a in region r in year t. Similarly, $Women_{r,t,a}$ is the number of women at age a in region r in year t.

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