Childhood Mortality and Morbidity in sub-Saharan Africa's Fertility Transition [1]

Anna-Maria Aksan [2]
Assistant Professor of Economics
Fairfield University

February 22, 2014

[1]I am grateful to two anonymous referees of this journal for valuable feedback. Thanks also to William Vasquez for his helpful suggestions.

[2]Author contact: Department of Economics, Fairfield University, 1073 North Benson Road, Fairfield, CT 08624-5195, USA, aaksan@fairfield.edu

Abstract

Child mortality rates have fallen substantially in developing countries since 1960. The expected fertility decline has followed only weakly in sub-Saharan Africa compared to other recent and historic demographic transitions. Disease and anthropometric data suggest that morbidity remains prevalent in Africa despite child survival improvements. The uniquely high infectious disease burden among children in Africa reduces population health and diminishes the returns to human capital investment, thwarting the quantity-quality tradeoff for children that typically accompanies the mortality transition. Individual-level data from the Demographic and Health Surveys are used to show persistent morbidity has weakened the positive relationship between child mortality and total fertility rates throughout the region, slowing Africa's demographic transition.

Keywords: fertility, morbidity, child mortality, sub-Saharan Africa, demographic transition
JEL Classification: I15, J11, J13

1 Introduction

A large body of theoretical and empirical literature establishes a positive relationship between child mortality rates and fertility rates (Boldrin and Jones 2002; Galor and Weil 1999; see Schultz (1997) for a review; Barro and Becker 1988). As long as child mortality rates are high, parents have more children as an insurance against expected child losses.[1] During the demographic transition parents begin to substitute quality for quantity of children, having smaller families but investing more in the human capital of each child (Kalemli-Ozcan 2008; Cervellati and Sunde 2007; Soares 2005). In countries lacking social security, children are a major source of financial support for the elderly, and parents aim to maximize the returns on their investments (Boldrin and Jones 2002; Oshomuvne 1990). If the expected returns to human capital investment are low, for example if high mortality rates make educating a single child risky, then parents continue to invest in quantity over quality of children.[2]

Child mortality has declined substantially in developing countries since 1960 with fertility rates following suit (Angeles 2010). But in sub-Saharan Africa (SSA), the fertility response has been weak. The elasticity of the total fertility rate (TFR) with respect to the child mortality rate (CMR) during 1975-2010 was 0.45, compared to at least 0.67 for every other developing region (World Bank Indicators). The TFR for SSA as a whole fell 23% from 6.8 to 5.2 while the CMR fell 51% from 215 to 106 per 1,000 live births. In The Gambia, Malawi, Togo, Cameroon and Burkina Faso, fertility *increased* between 1960 and 1980 even though infant and child mortality rates were falling, in contrast to the positive relationship usually observed in other regions. SSA has the fastest growing population in the world. Families remain large, which may deplete resources for human capital investment. Thus the factors hindering the region's demographic transition are important for understanding its slow economic growth.

Demographic transitions have historically started with an epidemiological transition whereby infectious diseases are combated and children grow up healthier (Omran 1971). In England deaths from infectious disease began a sustained decline around 1870 after the advent of the germ theory provoked widespread sanitation infrastructure reforms.[3] The first affected cohort of children experienced sharp increases in adult stature and reductions in adult pre-mature

[1] Sometimes the fertility decline has preceeded the mortality decline (Galor 2005), but generally fertility has followed mortality, especially recently (Angeles 2010).

[2] This applies even if children are not thought of as an investment for security in old age. For example, when facing high survival risk altruistic parents choose to have more children to ensure they do not end up childless, and resource constraints force a choice of quantity over quality.

[3] Mortality did decline modestly between 1780 and 1830, but this decline was arrested until a second decisive period of decline began during the 1870s (Hinde 2003, p. 195).

mortality, suggesting improving population health (Arora 2005).

The nature of SSA's mortality transition is different in that morbidity has not improved along with mortality. Today improvements in adult stature continue to lag that of other developing regions, and in some African countries adult stature has been declining (Akachi and Canning 2010). Figure 1 depicts the height of adult women throughout SSA grouped by birth year. Since child mortality has fallen since 1960 the downward trend in stature suggests the incidence of childhood disease remains high, that child nutrition has worsened, and/or that women are having children earlier and missing out on potential growth spurts in adolescence. Rising age at first childbirth (Figure 2) rules out the latter. While malnutrition remains a health challenge in many areas, an upward trend in caloric supply (Figure 3) suggests disease morbidity is important in explaining falling stature.

In SSA child survival has improved because of better nutrition, curative measures such as antibiotics and oral rehydration solutions (ORS), and increased vaccination coverage. Diarrheal infections are often successfully treated but inadequate water and sanitation systems facilitate reinfection. Health care is lacking in terms of access and appropriate technologies for common diseases (Millenium Development Goals). Protection from one disease by vaccination does not preclude infection by others. On average African children experience 15 episodes of diarrhea during the first 4 years of childhood and 1.6-5.4 malarial episodes per year (Boschi-Pinto *et al.* 2006; Murphy and Breman 2001). Incidence of most diseases is much higher than their fatality rate. Only 0.11% of diarrheal infections and 0.41% of malarial infections resulted in death among African children in 2004, even though these diseases accounted for almost 40% of deaths in children under 5 years of age. Malaria and diarrhea remain common experiences among children despite improvements in survival prospects.

Childhood infections have adverse effects on cognitive and physical development (Eppig, Fincher and Thornhill 2010; Martorell and Habicht 1986; Mata 1978), contributing to malnutrition and stunting (Checkley *et al.* 2008; Bates *et al.* 2004) and affecting education outcomes (Mendez and Adair 1999; Behrman 1996), earnings during adulthood, and adult health by contributing to non-infectious diseases later in life (Crimmins and Finch 2006; Arora 2005; Strauss and Thomas 1998; Barker 1994; Fogel 1993).[4] A school-aged child may have difficulty learning due to cognitive damage caused by early childhood illness, which is then exacerbated if new illness deters from school attendance and concentration while at school.

[4]More specifically, see Guerrant, Carneiro-Filho and Dillingham (2003) and Checkley *et al.* (2008) for effects of childhood diarrhea; see Miguel and Kremer (2004) and Coelho and McGuire (2000) for effects of hookworm infection; see Snow *et al.* (2003), Bleakley (2007), Holding and Snow (2001), Cutler *et al.* (2007) and Chang *et al.* (2011) for education effects of malaria and anemia; see Fogel (1993) and Almond and Currie (2011) for long run effects of childhood infection.

Less effective schooling combined with physiological scarring from childhood illness and increased susceptibility to adult illness may then dampen labor productivity in adulthood. For example, anemia, which is approximately 75% prevalent in malarious regions, reduces school performance and earnings among working adults (Breman *et al.* 2006; Snow *et al.* 2003). As more ill-stricken children survive infections in SSA, their average cohort health is diminished.

By depleting population health the morbidity burden in SSA dampens expected returns to human capital investment. Having many children remains relatively more attractive than investing in the quality of a few (Strauss and Thomas 1998; Behrman and Rosenzweig 2001; Case and Paxson 2010).[5] Childhood morbidity amplifies uncertainty about child survival, since parents perceive a high risk of their child becoming infected by one of Africa's many endemic diseases, and then once infected, uncertainty about the child's survival and health status. This paper distinguishes between child mortality and morbidity to identify the role the latter has played in slowing SSA's demographic transition.

Fertility remains high if uncertainty about child survival *or* child quality remain high, and the two do not necessarily move together. If widespread water and sanitation infrastructure reforms reduce the incidence of infections, then child mortality declines because diseases such as diarrheal infections become less common. Both mortality and morbidity decline, reducing uncertainty about survival and health outcomes. A strong fertility decline is expected. If instead child survival improves because antibiotics become more accessible, then mortality declines but morbidity may persist. Even if treatment completely cures a child, the potential for reinfection remains high as transmission of pathogens is facilitated by inadequate water and sanitation infrastructure. More children survive because of the antibiotics, but recurrent infections compromise their health.[6] A weaker decline in fertility is expected as morbidity replaces mortality and returns to human capital investment remain low.

Child mortality and fertility rates vary considerably throughout SSA. The CMR ranges from 15.1 to 185.3 per 1,000 live births in Mauritius and Sierra Leone, respectively. The TFR ranges from 1.45 in Mauritius to 7.0 in Niger (World Bank Indicators 2011). Despite a clear positive relationship between the two, Figure 4 illustrates that the TFR varies substantially among countries for relatively moderate to high levels of child mortality. Childhood morbidity can explain some of this variation.

[5]Another role morbidity may play in the fertility transition is through its effect on pre-mature adult mortality. Shorter expected lifespans lead individuals to save less for the future, raising current consumption. Correspondingly, fertility is higher and investment in the human capital of children lower.

[6]Treatment with antibiotics may reduce the duration and severity of an infection, thereby mitigating some of the aggregate morbidity effects cause by recurrent infections but also diminishing the prevalence of disease by shortening the time during which a person is contagious. However overall a population avoiding infections in the first place will be healthier than a population successfully treating recurrent ones.

While the relationship between health and human capital is well established in the literature, little attention has been given to the connection between childhood morbidity and fertility. De la Croix and Licandro (2013) develop a theoretical model to show how childhood physical development and adult life expectancy played a fundamental role in the demographic transitions of western countries. Aksan and Chakraborty (2013) theoretically model the role of morbidity in the quantity-quality tradeoff for children by distinguishing between child survival improvements due to reduced disease prevalence versus reduced case fatality rates. An exception in the empirical literature is the work of Bleakley and Lange (2009) who find that a higher return to schooling generated by the eradication of hookworm in the US South was accompanied by significant fertility declines.

In the current paper individual survey data on women throughout SSA are used to show how childhood morbidity, captured by anthropometric measures, has weakened the positive relationship between child mortality and fertility in that region. The focus is on individual-level expectations about children's disease experience as determined by the prevalence of child mortality and morbidity in a woman's community. I find strong support for two channels of nonlinearity in the relationship between fertility and child mortality. One functions through uncertainty about child survival: fertility falls with child mortality but less at high levels of *mortality*. The other functions through uncertainty about child quality: fertility falls with child mortality but less where *morbidity* persists. The fertility-mortality relationship in SSA is strongest where both child mortality and morbidity are lower.

The following section presents the regression model. Section 3 describes the data used in estimation. Results are presented in Section 4 and discussed in Section 5. Section 6 concludes.

2 Model

If child mortality is replaced by morbidity, for example because better curative measures improve survival without a corresponding decrease in disease incidence, then parents face considerable uncertainty about their children's potential health status. Despite improved probability of survival, parents anticipate lower returns to human capital investment and continue to choose larger families.

Fertility decisions are made prior to knowing the survival and health outcomes of all of one's children, except in the case of replacement fertility. While parents cannot perfectly predict the survival and health outcomes of their children, they can form expectations based on rates of illness and mortality observed in the community about whether their children will become ill and their subsequent survival prospects.

The model below proposes a nonlinear relationship between fertility and child mortality. In line with previous demographic transitions, lower fertility is expected in communities with a lower mortality burden. Given SSA's persistent disease burden, however, I test whether this relationship is weaker in areas with more childhood morbidity, controlling for uncertainty about child survival.

$$Recent\ births_i = \beta_0 + \beta_1 Mortality_{c,t-5} + \beta_2 Mortality^2_{c,t-5} + \beta_3 Morbidity_c \quad (1)$$

$$+\beta_4 Mortality_{c,t-5} Morbidity_c + \beta_5 X_i + \beta_6 X_c + Year_i + Region_i + u_i$$

The subscript i indicates individual variables and c community variables, where a community is defined by survey clusters, or primary sampling units, in the data (see Section 3). The dependent variable is the number of children born to woman i in the past five years. The subscript $t-5$ indicates a value five years ago in order to avoid potential endogeneity between the dependent and the mortality variables. For example, child mortality may be higher in larger families because of constraints on nutriton and health care resources available for each child.

The focus of the paper is on community child mortality, but replacement fertility may play a role distinct from expectations about child mortality. Individual child mortality experienced by each woman is controlled for by including the number of deceased children as of five years ago, or $Deaths_{i,t-5}$, in X_i, a vector of individual-level control variables. A positive effect is expected. Since only women with many children born could report many deaths, the number of births up until five years ago, $Births_{i,t-5}$, is also included in X_i. All else equal, women with more children as of five years ago are likely to have fewer births since then.

The community child mortality rate is represented by the total number of respondents' children in the community who have died as a fraction of all those born, among the sampled women.

$$Mortality_{c,t-5} = \frac{\sum_i \text{Deaths}_{i,t-5} - \text{Deaths}_{i,t-5}}{\sum_i \text{Births}_{i,t-5} - \text{Births}_{i,t-5}}$$

Removing the individual woman's child mortality information from the aggregate measure distinguishes the effects of mortality expectations from replacement fertility. Note the child mortality variable differs from the standard definition for the CMR since some of the children may have been older than five years old when they died.

In order to achieve a desired family size, parents have more children if they expect many will not survive. Previous theoretical studies account for a precautionary motive for hoarding of children by explicitly modeling uncertainty about child survival (Kalemli-Ozcan 2003; Tamura 2006; Sah 1991). Child survival will be more uncertain in high mortality environments, and the square of $Mortality_c$ acts as a proxy for this uncertainty.[7] A positive coefficient

[7]This is also the approach used by Aksan and Chakraborty (2013) to test for a precautionary motive.

on $Mortality_c$ and a negative coefficient on its square are expected: parents lower their fertility in response to lower perceived child mortality, but where this perceived risk remains higher fertility responds only weakly. Not until uncertainty about child survival has declined sufficiently does the fertility response strengthen.

Expectations about child morbidity depend on the types of childhood diseases prevalent in the community, the degree to which they are fatal and their consequences for a child's health and development. Morbidity is difficult to measure because prevalence data on less fatal diseases such as diarrhea are limited and noisy. Focusing on a single disease does not necessarily capture the influence of others because of the ubiquity of infections in many areas of SSA and disease complementarities. Avoiding a particular disease, through vaccination for example, does not necessarily reduce the disease burden of children since they now survive one disease to face others. On the other hand, targeted disease eradication may have a magnifying improvement on the epidemiological environment. For example, insecticide-treated bednet programs result in a larger drop in all-cause mortality than can be accounted for by malaria alone (Shanks, Hay and Bradley 2008).

Stunting is a lasting aggregate measure of morbidity, capturing net nutritional deficiencies caused by under or malnutrition and disease. Often the two causes are complementary. For example, diarrheal infection contributes to malnutrition through dehydration and malabsorption of nutrients, which in turn leaves children more susceptible to future diarrheal infections. Pneumonia, diarrhea and malaria, the most common childhood diseases in SSA, all contribute to stunting.[8]

The prevalence of child stunting in the community is used as an aggregate measure of childhood morbidity.

$$Morbidity_c = \frac{\sum_i \text{Stunted}_i - \text{Stunted}_i}{\sum_i \text{Recent births}_i - \text{Recent births}_i}$$

where $Stunted_i$ is the number of a woman's children born in the last five years who are currently stunted. A child is considered stunted if he or she falls at least two standard deviations below the Center for Disease Control reference median height, the definition used by the Demographic and Health Surveys. Removing the individual woman's children from the measure alleviates the problem that a child's current anthropometric status could be affected by the number of recent births in the family. For example, short birth intervals are associated

That paper uses country-level data and disease-specific measures of morbidity, while here the data are at the inidividual-level and the morbidity measure broader.

[8]Long-term morbidity can manifest in other ways also. For example, measles, typhoid and malaria during childhood have been linked to increased cardiovascular diseases in adulthood and childhood pneumonia to pulmonary diseases in adulthood (Barker 1994; Khosla 1981), but these diseases also affect height (Jousilahti *et al.* 2000).

with increased occurrence of low birth weight and size, among other adverse health outcomes (Zhu *et al.* 1999). $Morbidity_c$ represents current perceptions women have on the likelihood their children will be healthy upon reaching school age.

Child mortality has fallen in SSA while TFR remains stubbornly high. At the same time evidence on adult stature of women suggests morbidity is rising even as mortality recedes. To test whether the response of fertility to child mortality declines has been dampened by persistent or even rising morbidity, I include the interaction of $Mortality_c$ and $Morbidity_c$ in equation 1. This allows for perceived morbidity risk to affect the fertility response to perceived mortality risk. It is hypothesized that reductions in child mortality reduce fertility less where morbidity persists: $\beta_1 > 0$ and $\beta_4 < 0$. Moreover, $\beta_3 > 0$ is expected so that even in a low mortality community, morbidity sustains high fertility. The latter is consistent with the findings by Bleakley and Lange that widespread hookworm infection, a non-fatal disease that can cause anemia, hindered school achievement and was associated with higher fertility in the US South.

X_i and X_c include individual and community level control variables that may influence fertility and that are standard in the literature. The following variables have been chosen to maximize sample size while capturing the major factors influencing fertility decisions and outcomes.

More educated women may choose to have fewer children because the opportunity cost in terms of wages foregone is higher. They may be more knowledgable about and more likely to use contraceptives. The level of education of women in the community may have an effect on fertility in addition to that of the woman's own education. Education indicates greater autonomy of women in society. If women in the community are more educated, there may be greater emphasis on community investments in infrastructure (sanitation, health clinics) as well as spillover effects from one family to another regarding child and maternal health and contraception. Whether the individual woman has completed primary education and the prevalence of a primary education among women in the community are each included to account for these channels.

A woman's education may be interrupted if she has a child while attending school. The sample is restricted to women who have either not had any children or who were at least 18 years old at the time they first gave birth. Assuming women will have completed primary education by the age of 18 if they are ever to do so, this potential source of endogeneity is avoided.

Several variables (presence of electricity in the home, whether the household owns a car or truck) proxy for wealth effects on fertility.[9] Wealthier families can afford more children, but

[9]The Demographic and Health Survey data described in Section 3 provide a wealth index for only a subset of

also wealthier families may choose to have fewer children if their opportunity cost is higher. Previous studies have generally found a negative relationship between fertility and household wealth.

Electricity also proxies for availability of public services such as health care and schools. Where health care services are available, lower mortality and morbidity are expected and also lower fertility if contraceptives are more accessible. If there are no schools nearby, quality investment in children becomes more difficult and parents may continue to have large families instead.

Older women will be closer to their completed fertility. The log of age is included in X_i to account for a nonlinear relationship between a woman's age and the number of recent births.

Women who grew up in a larger family may have an inherent preference for larger families, so X_i includes the number of siblings a woman has had.

Including a control for whether a woman herself is stunted may capture intergenerational effects of child morbidity on fertility that are unique to contemporary effects. If a woman is stunted due to her own net nutritional deficiencies during childhood, it may physiologically influence her fertility. For example, stunting may affect whether she is physically strong enough to carry a pregnancy to term. In addition to affecting her fecundity, if a woman herself experienced morbidity in childhood substantial enough to persist into adulthood, then she may expect her own children to be vulnerable as well. Stunting would have occurred prior to adulthood and most likely in early childhood when nutritional needs are high due to rapid growth, faster than even peak height velocity in adolescence (Bozzoli, Deaton, and Quintana-Demoque 2009; Martorell, Khan, and Schroeder 1994). Stunting of women would then represent child morbidity lagged by a generation. A woman could also be stunted if she began having children as an adolescent, although all such women have been excluded from the current sample.[10]

The data set includes surveys conducted in different years. Both year and region (within country) dummy variables are included to control for unobserved regional or time heterogeneity. Cultural variables such as religion or marital customs (age of marriage, polygamy), which change only slowly over time, may affect fertility. A particular year may have brought

surveys. The index is a weighted measure of various consumption goods, including those used here as proxies for wealth. Since the aim is only to control for a wealth effect and not measure its magnitude, using an unweighted measure is sufficient.

[10]Adolescent mothers have a higher propensity for low birthweight, which could influence physical development of the child and thus contribute to child stunting. By restricting the sample to women who either have not yet had a child or whose first birth occurred at age 18 or older makes it more likely that $Morbidity_c$ is capturing environmentally-caused child morbidity and is not confounded by the effect of being born to an adolescent mother.

events that affected the entire region, which in turn may affect fertility, for example, if a global recession or a regional drought reduced incomes and thus marriages and fertility.

Since the dependent variable is a positive, discrete count variable with a low mean and a similar variance, a poisson model is used for estimation. The errors are clustered by regions within countries.

3 Data

Data is from the nationally representative Demographic and Health Surveys (DHS) at the individual level. The respondents are women ages 18-49 years old who either report no births or who were at least 18 years old at their first birth.

All available country and year surveys for SSA with complete data were included.[11] There are 30 countries represented in the final regression sample with 1-4 years of surveys for each, covering the years 1992-2008.[12]

Observations are identified by region within country (293 total regions) and at a more local level by clusters. For the variables $Mortality_c$ and $Morbidity_c$, "community" is defined as the survey cluster. On average a cluster contains 41 women, with a standard deviation of 33 and a range of 5-156 women per cluster. The sample is restricted to clusters comprised of at least five respondents so that they are a meaningful measure of community: a cluster of one or two households does not provide women with much "community" information about morbidity and mortality.

Since DHS oversamples some areas to ensure representative means, the DHS sampling weights are applied to each individual when aggregating data to the community, or cluster, level. The weighting is not explicitly shown in the community variable definitions in Section 2 in order to facilitate their exposition.

Descriptive statistics are presented in Table 1. There are a total of 174,936 women in the final sample. The average age of the women in the sample is 30 years. Approximately 40% of them have completed primary schooling, 64% reside in a rural area, 26% have electricity in their home, and 7% reside in a household owning a car or truck. Recent births (in the last

[11] Countries included in sample: Benin, Burkina Faso, Central African Republic, Cameroon, Chad, Congo, Cote d'Ivoire, Democratic Republic of Congo, Ethiopia, Gabon, Guinea, Kenya, Lesotho, Liberia, Madagascar, Malawi, Mali, Mozambique, Namibia, Niger, Nigeria, Rwanda, Senegal, Sierra Leone, Swaziland, Tanzania, Togo, Uganda, Zambia, Zimbabwe. Countries excluded due to survey or data unavailability: Angola, Botswana, Burundi, Comoros, Djibouti, Equatorial Guinea, Eritrea, Ghana, Guinea-Bissau, Mauritius, Mauritania, Sao Tome and Principe, Seychelles, Somalia, South Africa, South Sudan, Sudan.

[12] The countries with multiple survey years do not constitute panels since different households were interviewed each year and it is impossible to identify if any particular individual appears in multiple surveys.

five years) range from zero to six children with a mean of 0.93. At five years preceeding the survey, woman had between zero and 16 children born, and a range of zero to 13 children deceased. Twenty-five percent of the surviving children born in the past five years were stunted at the time of interview, as were 15% of the women. If woman's stunting reflects child morbidity when the woman was young, then the higher prevalence of stunting among children suggests rising child morbidity over time. However, it could also reflect the impact of "catch-up" growth during adolescence, which is substantial in SSA (Moradi 2010).

4 Results

Column 1 of Table 2 presents the regression results for the model when all the community mortality and morbidity variables are omitted, included as a baseline. The coefficient on individual child mortality is positive and statistically significant. Adding $Mortality_c$ in Column 2 leaves the coefficient on $Deaths_i$ unchanged but is itself insignificant. Further adding $Mortality_c^2$ in Column 3 makes the coefficient on $Mortality_c$ significant and is itself negative, consistent with the theory of a precautionary motive. If child mortality remains high, the uncertainty that parents face about their children's survival dampens their fertility response to declining child mortality. These results also support distinct effects of replacement fertility and parents' child mortality expectations.

The coefficient on $Morbidity_c$ in Column 4 is positive and significant. Child morbidity is associated with higher fertility, holding constant the level of child mortality. Further adding the interaction of $Mortality_c$ and $Morbidity_c$ in Column 5 illustrates a nonlinear relationship between morbidity and fertility as well as an additional channel of nonlinearity between mortality and fertility. The coefficient on $Morbidity_c$ increases in magnitude, while the interaction term has a negative, statistically significant coefficient. While the positive relationship between child morbidity and fertility remains, it is weaker in communities with low child survival. Morbidity is less relevant in the family planning process in high mortality environments since fewer children survive long enough for the consequences of morbidity on schooling to matter: the focus is on survival.

If morbidity is simply proxying for "high mortality" and the results are picking up a nonlinear relationship between fertility and child mortality, then including $Mortality_c^2$ should remove the statistical significance of the interaction term, $Mortality_c * Morbidity_c$, but that is not the case in Column 5. Lower community child mortality is associated with fewer births, but the relationship is weaker where child morbidity is prevalent. Uncertainty about child quality and lower expected returns to human capital investment replace the uncertainty about child survival as mortality rates decline.

Coefficients on the control variables have their expected signs and are statistically significant in each specification. Women who grew up in larger families and women living in rural areas tend to have more births. Presence of electricity or a car/truck in the household are each associated with fewer births. More educated women and women living in communities where women tend to be educated have fewer births. Women who are stunted tend to have fewer births, possibly because of physiological effects on fertility.

5 Discussion

While some African countries have at times exhibited a negative correlation between child mortality and fertility rates, the regression results here support the historically typical positive relationship, albeit a weak one in high mortality or high morbidity communities. Child mortality remains a driver of high fertility in SSA, but as health interventions continue to reduce child deaths, the morbidity burden emerges as a primary health policy challenge. Improvements in child survival without corresponding improvements in the epidemiological environment worsen population health. One consequence is that depleted health capital of children lowers expected returns to human capital invesment, impeding a quantity-quality tradeoff for children.

Consistent with a precautionary motive, fertility declines with community mortality only weakly at high levels of mortality. Moreover, not only does fertility respond to child mortality more weakly in high mortality communities, but even if mortality falls decisively, the fertility response remains weak unless the larger surviving cohort of children is healthy. If morbidity replaces mortality, then fertility rates remain high.

Using the final specification in Column 5, the mean net effect of a percentage point increase in community child mortality is $0.01 * exp(0.39 - 2 * 0.56 * Mortality_{c,t-5} - 0.49 * Morbidity_c) = 0.22\%$ more births. If child stunting were eliminated, all else equal, this figure increases to 0.37%, a response which is 68% stronger. During 1975-2010 the elasticity of TFR with respect to CMR was 0.67 in the Middle East and North Africa, 0.68 in Latin America and the Caribbean, 0.71 in East Asia, and 0.76 in South Asia (World Bank Indicators). In other words the response was 49-69% stronger in all other developing regions compared to the 0.45 observed for SSA. The relative difference in the estimated mean effect of community child mortality when child stunting is 0% versus 25%, the sample mean, falls within the range of these observed regional differences. The lower levels of CMR in all of these regions relative to SSA may account for some of the stronger response since the precautionary motive holding back the TFR decline would be relatively weaker in the other regions.[13] But also the highest

[13]During 1975-2010 CMR (under-5 mortality per 1,000 live births) fell from 215 to 106 in SSA, 165 to 27 in the Middle East and North Africa, 102 to 23 in Latin America and the Caribbean, 88 to 22 in East Asia, and

prevalence of stunting occurs in SSA.

The effect of community child mortality on a woman's fertility is relatively stronger than that of personal child losses. The sample mean value of $Mortality_c$ is 7%, so community child mortality is contributing on average $7\% * 0.22\% = 1.54\%$ more births. In the sample women have experienced on average 2.03 children born by five years prior to the interview, and of those an average of 0.38 children had died. Replacement fertility is contributing approximately $0.38 * 0.01\% = 0.0038\%$ more births in the sample. Perceived mortality risk seems to be a more important driver of high fertility in SSA, consistent with previous studies of insurance fertility that find a stronger effect of community child mortality on fertility than of replacement fertility (see Schultz 1997 for a review). It may also be that the replacement effect is deflated here because the individual mortality variable includes child deaths at all ages, and replacement will be more difficult or undesired if a woman is approaching the end of her reproductive life.

The model distinguishes between the effects on fertility of current child stunting and a woman's own stunting status, or child morbidity lagged by one generation. The estimates indicate a negative influence of maternal stunting on fertility. The effects of morbidity on fertility are thus multifaceted. Expected morbidity raises fertility by lowering parental expectations of child quality. But also child morbidity lingers on into adulthood and may reduce fecundity. In the sample this negative fecundity effect dominates any positive effect of fertility associated with a woman's expectations that her children are more likely to be stunted if she was. Women may move away from their childhood communities, especially if marriage implies relocation to her husband's community, and thus the morbidity profile of her childhood differs from that of her current environment. Moreover, maternal stunting has been associated with offspring mortality, low birthweight and stunting, presenting another channel by which morbidity persists from one generation to the next (Özaltin, Hill and Subramanian 2010). The relationship between maternal stunting and fertility warrants further investigation but is beyond the focus of this paper.

6 Conclusion

Despite substantial declines in child mortality throughout SSA, the fertility response has been weak and net fertility remains near its level in the 1960s (Figure 5). The results of this paper suggest the cause for this stagnation is a combination of continued uncertainty about child survival, since child mortality remains relatively high, and poor expected health for those surviving.

193 to 65 in South Asia.

In SSA vaccination coverage for some childhood diseases such as measles, pertussis, diptheria and tetanus has improved dramatically since 1980. Yet treatments and vaccines for a number of prevalent diseases, such as malaria, still do not exist or are unavailable because of poverty and inadequate health care. Children survive one disease to face another, sometimes with compromised defences. Poor sanitation and water infrastructure exasperate the problem by allowing infectious diseases to proliferate. When ORS treatment is available more children survive diarrheal infections, but infections are recurrent. Uncertainty about child survival and health of surviving children remains high due to the persistent infectious disease burden.

The cycle of malnutrition and disease hinders cognitive and physical development, reducing returns to quality investment in children. In the sample analyzed here, this was reflected in a 25% prevalence of stunting in children aged five and under. This high morbidity burden has thwarted the quantity-quality tradeoff associated with mortality transitions in other developing regions recently and in western countries historically. Parents continue to have many children in the communities where morbidity persists even if child mortality has fallen.

Morbidity can be measured in a variety of ways. This paper uses an aggregate measure of morbidity, stunting. Focusing on a single disease would be less appropriate for SSA which hosts an array of endemic diseases. A more geographically concentrated analysis could allow for more precise measures such as prevalence of a particular disease or of a condition such as anemia, a major symptom of malaria, hookworm and iron deficiency, all common afflictions in SSA.

Recently child mortality declines have accelerated in many countries in SSA. In Kenya half the decline in infant mortality in endemic malarial regions has been attributed to an increase in household ownership of insecticide treated bednets from 8% to 60% between 2003 and 2008. The recent uptick in the rate of mortality decline throughout much of SSA should reduce survival uncertainty and accelerate the fertlity decline as the precautionary motive ($Mortality_c^2$ in the model) weakens. But also the nature of the mortality decline, reduced prevalence of malaria, will reduce the morbidity burden associated with this disease, in particular malnutrition, anemia, and permanent damage to cardiovascular health. This should further hasten the fertility decline as mortality and morbidity both decline ($Mortality_c * Morbidity_c$ in the model). Through widespread improvements in water and sanitation infrastructure, increased malarial vector control, innovation of appropriate technologies, and improvements in nutrition to reduce susceptibility to infections, morbidity and mortality will decline in tandem and contribute to a strong demographic transition.

7 References

Akachi, Yoko and David Canning. 2010. "Health trends in Sub-Saharan Africa: Conflicting evidence from infant mortality rates and adult heights," *Economics and Human Biology* 8: 273-288.

Aksan, Anna-Maria and Shankha Chakraborty. 2013. "Childhood disease and the precautionary demand for children," *Journal of Population Economics* 26(3): 855-885.

Almond, Douglas and Janet Currie. 2011. "Killing me softly: The fetal origins hypothesis," *Journal of Economic Perspectives* 25(3): 153-172.

Angeles, Luis. 2010. "Demographic transitions: Analyzing the effects of mortality on fertility," *Journal of Population Economics* 23: 99-120.

Arora, Suchit. 2005 "On epidemiological and economic transitions: A historical view," in Guillem Lopez-Casasnovas, Berta Rivera and Luis Currais (eds.), *Health and Economic Growth: Findings and Policy Implications*, Cambridge, MA: MIT Press.

Barker, David J.P. 1994. *Mothers, babies, and disease in later life.* London: British Medical Journal Publishing Group.

Barro, Robert J. and Gary S. Becker. 1988. "A reformulation of the economic theory of fertility," *Quarterly Journal of Economics* 103(1): 1-25 (February).

Bates, Imelda *et al.* 2004. "Vulnerability to malaria, tuberculosis, and HIV/AIDS infection and disease," *The Lancet Infectious Diseases* Part 1 4(5): 267-277, Part 2 4(6): 368-375.

Behrman, Jere and Mark R. Rosenzweig. 2001. "The returns to increasing body weight," Penn Institute for Economic Research Working Paper No 01-052.

Behrman, Jere. 1996. "The impact of health and nutrition on education," *The World Bank Research Observer* 11: 23-37.

Black, Robert E. *et al.* 2008. "Maternal and child undernutrition: Global and regional exposures and health consequences," *The Lancet* 371(9608): 243-260.

Bleakley, Hoyt. 2007. "Disease and development: Evidence from hookworm eradication in the American South," *The Quarterly Journal of Economics* 122(1): 73-117.

Bleakley, Hoyt and Fabian Lange. 2009. "Chronic disease burden and the interaction of education, fertility, and growth," *The Review of Economics and Statistics* 91(1): 52-65.

Boldrin, Michele and Larry E. Jones. 2002. "Mortality, fertility, and saving in a Malthusian economy," *Review of Economic Dynamics* 5(4): 775-814.

Boschi-Pinto, Cynthia, Claudio F. Lanata, Walter Mendoza, and Demissie Habte. 2006. "Diarrheal diseases," in Dean T. Jamison, Richard G. Feachem, Malegapuru W. Makgoba, Eduard R. Bos, Florence K. Baingana, Karen J. Hofman and Khama O. Rogo (eds.), *Disease and Mortality in sub-Saharan Africa*, World Bank, Washington D.C.

Bozzoli, Carlos, Angus Deaton, and Climent Quintana-Domeque. 2009. "Adult height and childhood disease," *Demography* 46(4): 647-669.

Breman, Joel G. *et al.* 2006. "Conquering malaria," in Dean T. Jamison, Joel G. Breman, Athony R. Measham, George Alleyne, Mariam Claeson, David B. Evans, Prabhat Jha, Anne Mills and Philip Musgrove (eds.), *Disease Control Priorities in Developing Countries*, 2nd ed. New York: Oxford University Press, 413-432.

Case, Anne and Christina Paxson. 2010. "Causes and consequences of early-life health," *Demography* 47 (Supplement): S65.

Cervellati, Matteo and Uwe Sunde. 2007. "Human capital, mortality, and fertility: A unified theory of demographic transition," IZA Working Paper 2905.

Checkley, William *et al.* 2008. "Multi-country analysis of the effects of diarrhoea on childhood stunting," *International Journal of Epidemiology* 37(4): 816-830.

Chang, Simon, Belton Fleisher, Seonghoon Kim, and Shi-yung Liu. 2011. "Long-term effects of early childhood malaria exposure on education and health: Evidence from colonial Taiwan," IZA Discussion Paper No. 5526.

Coelho, Phillip R.P. and Robert A. McGuire. 2000. "Diets versus diseases: The anthropometrics of slave children," *The Journal of Economic History* 60: 232-246.

Crimmins, Eileen M. and Caleb E. Finch. 2006. "Infection, inflammation, height, and longevity," *Proceedings of the National Academy of Sciences of the United States of America* 103(2): 498-503.

Cutler, David, Winnie Fung, Michael Kremer, Monica Singhal and Tom Vogl. 2007. "Mosquitoes: The long-term effects of malaria eradication in India," NBER Working Paper No. 13539.

De la Croix, David and Omar Licandro. 2013. "The child is father of the man: Implications for the demographic transition," *Economic Journal* 123(567): 236-261.

Demombynes, Gabriel and Sofia Karina Trommlerova. 2012 "What has driven the decline of infant mortality in Kenya? World Bank Policy Research Working Paper, No. 6057.

Eppig, Christopher, Corey L. Fincher and Randy Thornhill. 2010. "Parasite prevalence and the worldwide distribution of cognitive ability," *Proceedings of the Royal Society B* 277(1701): 3801-8 (December).

Fogel, Robert W. 1993. "New sources and new techniques for the study of secular trends in nutritional status, health, mortality, and the process of aging," *Historical Methods* 26(1): 5-43.

Galor, Oded. 2005. "From stagnation to growth: Unified growth theory," in Philippe Aghion and Steven N. Durlauf (eds.), *Handbook of Economic Growth*, Vol. 1A, North Holland: Elsevier, 171-293.

Galor, Oded and David N. Weil. 1999. "From Malthusian stagnation to modern growth," *American Economic Review* 89: 150-154.

Guerrant, Richard L., Benedito A. Carneiro-Filho and Rebecca A. Dillingham. 2003. "Cholera, diarrhea, and oral rehydration therapy: Triumph and indictment," *Clinical Infectious Diseases* 37: 398-405.

Hinde, Andrew. 2003. *England's Population: A History since the Domesday Survey*, London:

Hodder Arnold.

Holding, Penny A. and Robert W. Snow. 2001. "Impact of *Plasmodium falciparum* malaria on performance and learning: Review of the evidence," *American Journal of Tropical Medicine and Hygiene* 64: 68-75.

Jousilahti, Pekka, Jaakko Tuomilehto, Erkki Vartiainen, Johan Eriksson and Pekka Puska. 2000. "Relation of adult height to cause-specific and total mortality: A prospective follow-up study of 31,199 middle-aged men and women in Finland," *American Journal of Epidemiology* 151(11): 1112-120.

Kalemli-Ozcan, Sebnem. 2008. "The uncertain lifetime and the timing of human capital investment," *Journal of Population Economics* 21(3): 557-572.

Kalemli-Ozcan, Sebnem. 2003. "A stochastic model of mortality, fertility, and human capital investment," *Journal of Development Economics* 70(1): 103-118.

Khosla, S. N. 1981. "The heart in enteric (typhoid) fever," *Journal of Tropical Medicine and Hygiene* 84(3): 125-131.

Martorell R., Khan L.K., Schroeder D.G. 1994. "Reversibility of stunting: Epidemiological findings in children from developing countries," *European Journal of Clinical Nutrition* 48: S45-57.

Martorell R. and Habicht J.P. 1986. "Growth in early childhood in developing countries," in Frank Falkner and J.M. Tanner (eds.), *Human Growth: A Comprehensive Treatise*, Vol.3, New York and London: Plenum Press, 241-263.

Mata, Leonardo J. 1978. *The Children of Santa Maria Cauque: A Prospective Field Study of Health and Growth*, Cambridge, MA: The MIT Press.

Mendez, Michelle A. and Linda S. Adair. 1999. "Severity and timing of stunting in the first two years of life affect performance on cognitive tests in late childhood," *Journal of Nutrition* 129: 1555-1562.

Miguel, Edward and Michael Kremer. 2004. "Worms: Identifying impacts on education

and health in the presence of treatment externalities," *Econometrica* 72(1): 159-217.

Moradi, Alexander. 2010. "Selective mortality or growth after childhood? What really is key to understand the puzzlingly tall adult heights in sub-Saharan Africa," Working paper.

Murphy, Sean C. and Joel G. Breman. 2001. "Gaps in the childhood malaria burden in Africa: Cerebral malaria, neurological sequelae, anemia, respiratory distress, hypoglycemia, and complications of pregnancy," *The Intolerable Burden of Malaria: A New Look at the Numbers*: Supplement to *American Journal of Tropical Medicine and Hygiene* 64 (1): 57-67.

Omran, Abdel R. 1971. "The epidemiological transition: A theory of the epidemiology of population change," *The Milbank Memorial Fund Quarterly* 49(4): 509-538.

Oshomuvwe, J.O. 1990. "Health services for the aged in sub-Saharan Africa," *Social Science and Medicine* 31(6): 661-665.

Özaltin E., Hill K. and Subramania S.V. 2010. "Association of maternal stature with offspring mortality, underweight, and stunting in low-to-middle-income countries," *JAMA* 303(15): 1507-1516.

Sah, Raaj K. 1991. "The effects of child mortality changes on fertility choice and parental welfare," *The Journal of Political Economy* 99(3): 582-606.

Schultz, T. Paul. 1997. "Demand for children in low income countries," in Mark R. Rosenzweig and Oded Stark (eds.), *Handbook of Population and Family Economics* 1: 349-430.

Shanks, G. Dennis, Simon I. Hay and David J. Bradley. 2008. "Malaria's indirect contribution to all-cause mortality in the Andaman Islands during the colonial era," *The Lancet Infectious Diseases* 8: 564-570.

Snow, Robert W., Marlies H. Craig, Charles R.J.C. Newton and Richard W. Steketee. 2003. "The public health burden of *Plasmodium falciparum* malaria in Africa: Deriving the numbers," Washington DC: The Disease Control Priorities Project (DCPP) Working Paper Number 11.

Soares, Rodrigo. 2005. "Mortality reduction, educational attainment, and fertility choice,"

American Economic Review 95(3): 580-601 (June).

Strauss, John and Duncan Thomas. 1998. "Health, nutrition and economic development," *Journal of Economic Literature* 36(2): 766-817.

Tamura, Robert. 2006. "Human capital and economic development," *Journal of Development Economics* 79: 26-72.

Zhu, Bao-Ping, Robert T. Rolfs, Barry E. Nangle and John M. Horan. 1999. "Effect of the interval between pregnancies on perinatal outcomes," *New England Journal of Medicine* 340(8): 589-594.

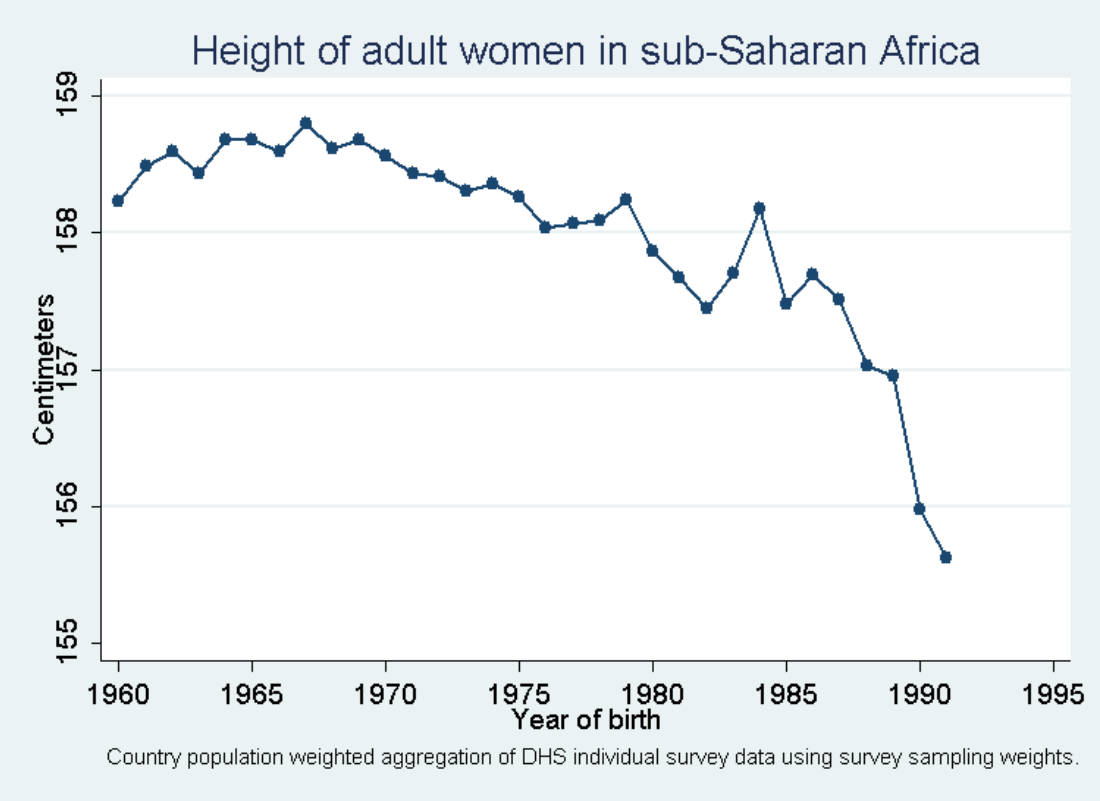

Figure 1: Falling height in sub-Saharan Africa

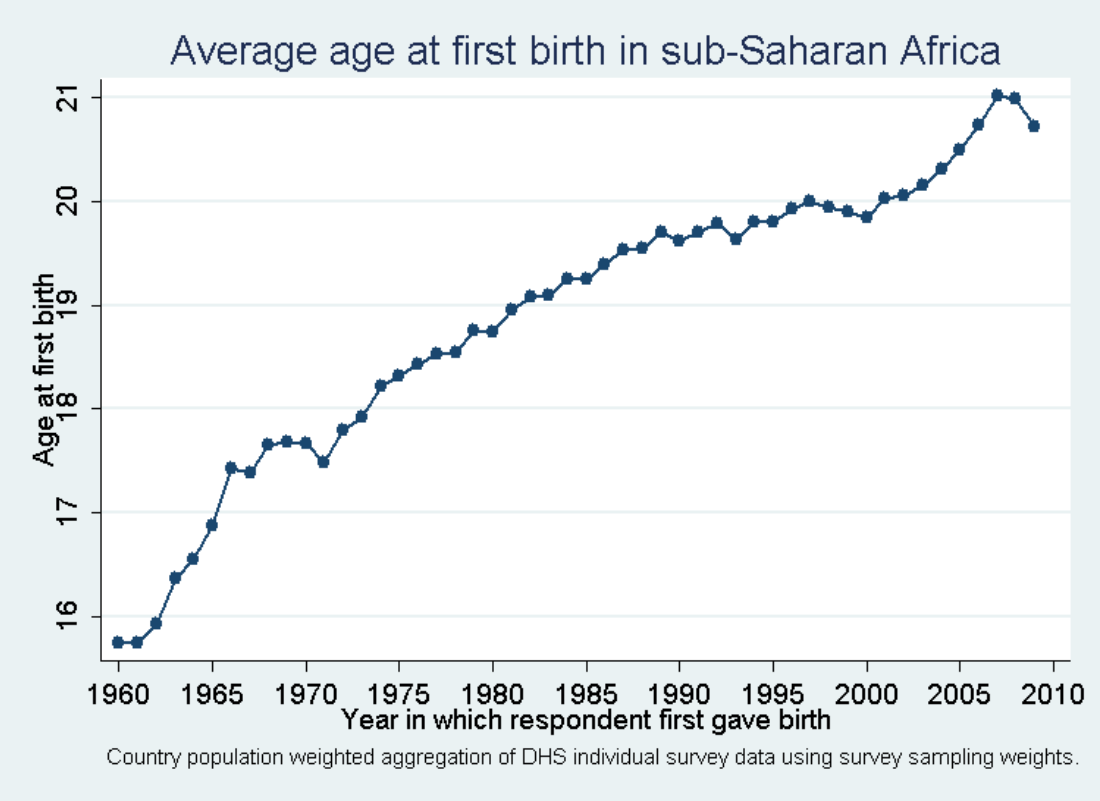

Figure 2: Rising age at first birth in sub-Saharan Africa

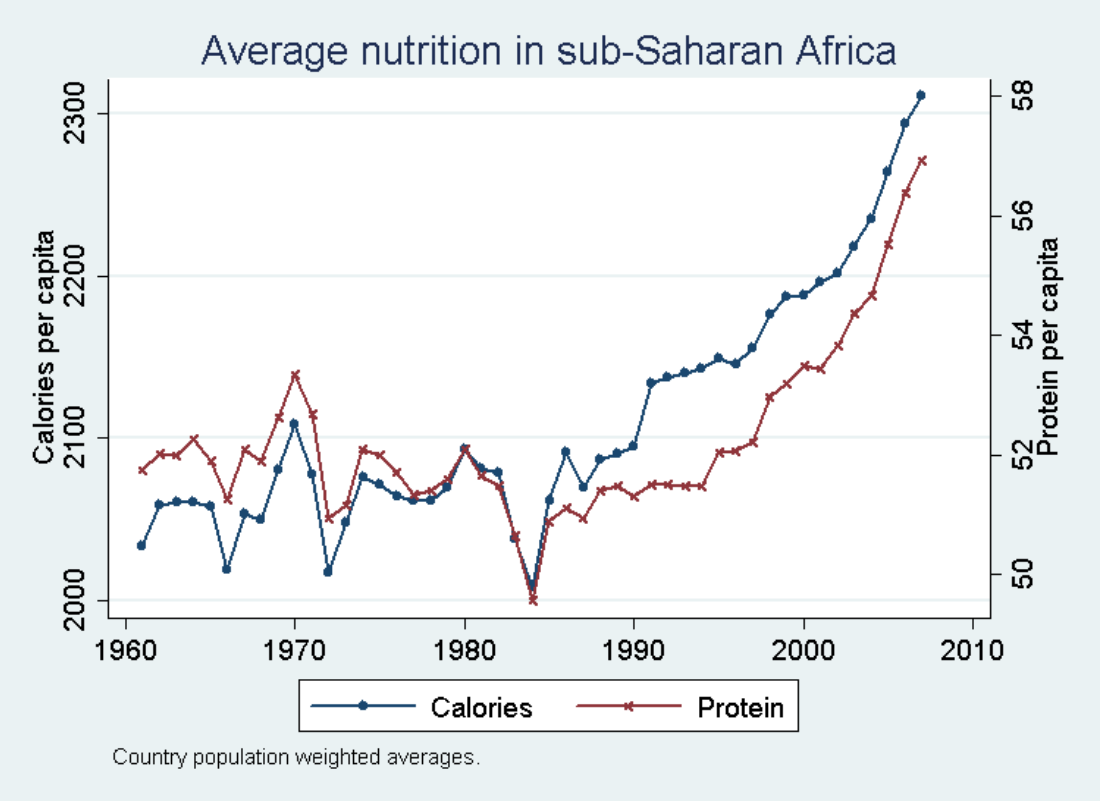

Figure 3: Rising caloric and protein intake in sub-Saharan Africa (FAOSTAT data)

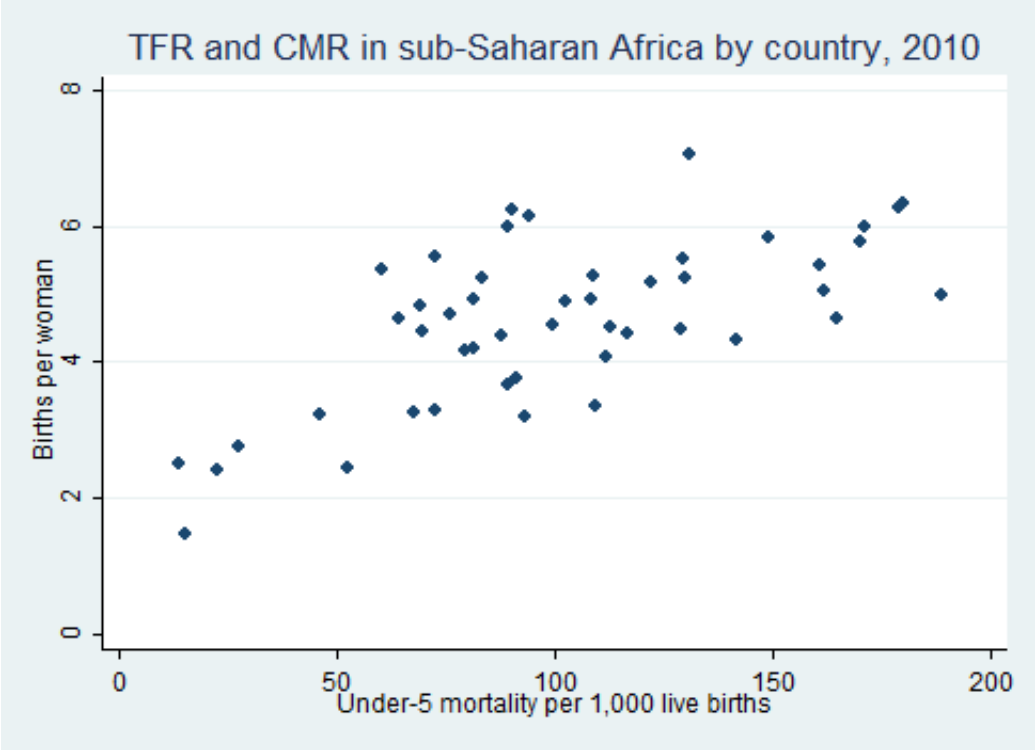

Figure 4: TFR and CMR by country in sub-Saharan Africa (World Bank data)

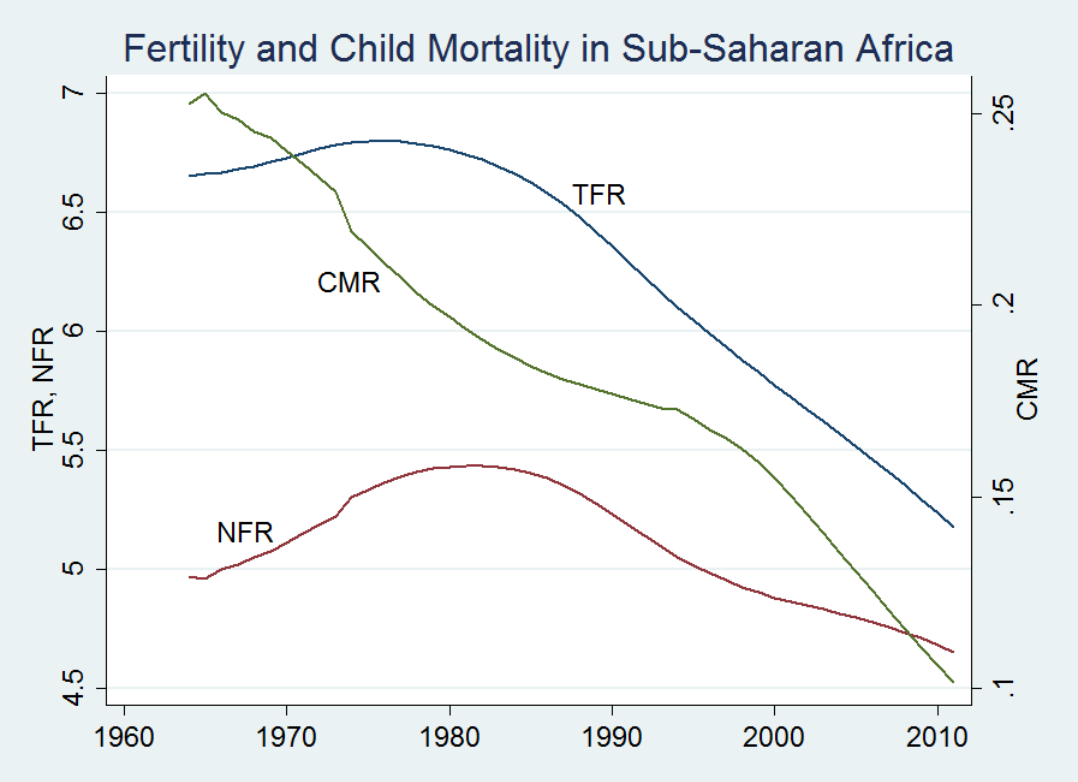

Figure 5: Fertility and CMR in sub-Saharan Africa (World Bank data)

Table 1: Descriptive Statistics

Variable	Mean	Std. Dev.	Min	Max
Individual-level variables				
Births in past 5 years	0.93	0.90	0	6
Total births$_{t-5}$	2.03	2.48	0	16
Deaths$_{t-5}$	0.38	0.88	0	13
Primary education	0.41	0.49	0	1
Electricity	0.26	0.44	0	1
Car/Truck	0.07	0.25	0	1
Age	30	8.58	18	49
Rural	0.64	0.48	0	1
Siblings	5.90	2.60	1	20
Woman stunted	0.15	0.36	0	1
Community-level variables				
Stunting prevalence of children	0.25	0.17	0	1
Mortality$_{t-5}$	0.07	0.06	0	1
Primary education	0.40	0.34	0	1
Households per cluster[a]	41	33.26	5	156

Observations = 174,936 women who did not give birth prior to age 18.

a. Number of households without sampling weights applied.

Table 2: Poisson Regression Results of *Births in the last 5 years*

	1	2	3	4	5
Community mortality		0.07	0.28	0.26	0.39
		(0.06)	(0.10)***	(0.10)**	(0.11)***
Community mortality squared			-0.62	-0.61	-0.56
			(0.23)***	(0.23)***	(0.23)**
Community morbidity				0.12	0.17
				(0.02)***	(0.03)***
Interaction of community mortality and morbidity					-0.49
					(0.21)**
Deaths_{t-5}	0.01	0.01	0.01	0.01	0.01
	(0.005)*	(0.005)*	(0.005)*	(0.005)*	(0.005)*
$\text{Total births}_{t-5}$	-0.02	-0.02	-0.02	-0.02	-0.02
	(0.005)***	(0.005)***	(0.005)***	(0.005)***	(0.005)***
Electricity	-0.16	-0.16	-0.16	-0.16	-0.16
	(0.02)***	(0.02)***	(0.02)***	(0.02)***	(0.02)***
Car/Truck	-0.11	-0.11	-0.11	-0.11	-0.11
	(0.02)***	(0.02)***	(0.02)***	(0.02)***	(0.02)***
Log(age)	-0.29	0.29	0.29	0.29	0.29
	(0.05)***	(0.05)***	(0.05)***	(0.05)***	(0.05)***
Rural	0.06	0.06	0.06	0.05	0.05
	(0.01)***	(0.01)***	(0.01)***	(0.01)***	(0.01)***
Siblings	0.02	0.02	0.02	0.02	0.02
	(0.001)***	(0.001)***	(0.001)***	(0.001)***	(0.001)***
Woman stunted	-0.04	-0.04	-0.04	-0.04	-0.04
	(0.01)***	(0.01)***	(0.01)***	(0.01)***	(0.01)***
Individual primary education	-0.11	-0.11	-0.11	-0.11	-0.11
	(0.02)***	(0.02)***	(0.02)***	(0.02)***	(0.02)***
Community primary education	-0.36	-0.36	-0.35	-0.34	-0.34
	(0.03)***	(0.03)***	(0.03)***	(0.03)***	(0.03)***
Constant	-1.58	-1.60	-1.61	-1.66	-1.67
	(0.18)***	(0.18)***	(0.18)***	(0.19)***	(0.19)***

Observations = 174,936

All regressions include region (within country) and year fixed effects.

Robust standard errors clustered on region are in parentheses.

* significant at 10%; ** significant at 5%; *** significant at 1%