Mortality versus Morbidity in the Demographic Transition

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Abstract

The link between the mortality and epidemiological transitions is used to identify the effect of the former on the fertility transition: a mortality transition that is not accompanied by improving morbidity causes slower demographic and economic change. In a model where children may die from infectious disease, childhood health affects human capital and noninfectious-disease-related adult mortality. When child mortality falls from lower prevalence, as it did in western Europe, labor productivity improves, fertility falls and the economy prospers. When it falls mainly from better cures, as it has in sub-Saharan Africa, survivors are less healthy and there is little economic growth. The model can quantitatively explain sub-Saharan Africa's experience. More generally it shows that the commonly used indicator, life expectancy at birth, is a poor predictor of population health and economic growth unless morbidity falls with mortality.

KEYWORDS: Economic Growth; Africa; Fertility; Mortality; Morbidity; Epidemiological Transition

JEL CLASSIFICATION: O40, O55, J10, J13

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1 Introduction

A leading view of the demographic transition links the fertility transition to prior reductions of child mortality. This pattern fits well several early industrializers of the nineteenth century and most developing countries in the twentieth century. Sub-Saharan Africa has been an exception to the pattern: despite significant improvement in child mortality, its fertility transition has been markedly slower than what we have come to expect from completed transitions elsewhere.

We argue that the health of Africa's population has improved much less than improvements in child mortality or life expectancy at birth suggest and this is why its fertility transition has been slower, yielded little economic benefit. Our explanation draws on the twin transitions of mortality and morbidity. During the epidemiological transition that underlies the mortality transition, infectious disease mortality among the young and elderly falls, followed by non-infectious disease mortality among older adults (Omran, 1971). Furthermore, falling infectious disease mortality is typically associated with falling morbidity as younger adults become healthier and fewer older adults suffer from non-infectious disease. It is this link between mortality and morbidity that has weakened during sub-Saharan Africa's mortality transition.

We develop a general equilibrium dynastic model that qualitatively and quantitatively accounts for sub-Saharan Africa's demographic and disease transitions. In the model, infectious disease affects susceptible children some of whom die. The health of surviving children is weakened, lowering the return on their human capital investment. Childhood infections also predispose people towards non-communicable illnesses like cardiovascular disease (CVD) later in life. The attendant childhood morbidity and adult mortality affect the value parents place on family size relative to child quality and their own future needs.

We differentiate between two ways child mortality can decline, from lower infectious disease prevalence or better therapy that attenuates infections.[1] Falling prevalence was behind the West's mortality transition and morbidity declined in tandem: child survival gains in late nineteenth century England and Wales were followed by secular gains in adult stature in successive cohorts (Szreter, 1988, Hinde, 2003, Arora, 2005, Bozzoli *et al.*, 2009). African countries, on the other hand, have reduced child mortality through better treatments like antibiotics and ORT (Guerrant *et al.*, 2003, Boschi-Pinto *et al.*, 2006). While vaccination has eradicated diseases like smallpox and polio, those have been the exceptions. This means decades of falling child mortality have not substantially lowered the prevalence of major infectious diseases in sub-Saharan Africa (SSA). Unlike other developing regions, adult stature and non-infectious disease mortality have improved little (Akachi and Canning, 2010, Leeder *et al.*, 2003).

[1] This distinction is made for conceptual clarity. While some therapeutic treatments can lower prevalence by reducing contagion, that effect is typically second-order. See also footnote 14.

In the model when child mortality falls from lower prevalence, more surviving children are of better quality (health), which raises the return to investing in their education as long as rearing sick children is not too time intensive. Altruistic parents substitute toward child quality; the total and net fertility rates fall. Since fewer children suffer from infections in early life, as adults they face a lower risk of premature death from CVDs. This further lowers the fertility rate and raises investment in physical capital. These forces facilitate epidemiological, demographic and economic transitions.

When child mortality falls from better cures, on the other hand, that they suffered through repeated infections means more surviving children are of worse quality. Parents have little incentive to invest in their human capital, and continue to choose large families. While the total fertility rate falls, net fertility may not. Moreover, due to infections in early life, a large share of the adult population is exposed to CVD-related premature mortality. The fertility or epidemiological transitions are slower in this case. Economic growth may even worsen if the morbidity effect is particularly severe. Calibrations of the model mimic the mortality transition in SSA versus the historical experience of England & Wales. The initial exogenous trigger for the CMR decline, lower disease transmission versus lower case fatalities, takes the economy from a Malthusian regime to modern growth and determines the path of mortality and population health. We show that the morbidity channel can account for the African experience and that child mortality alone is a poor predictor of national health and development.

Changes in child mortality, life expectancy at birth, adult mortality and health have been favorably linked in the literature to fertility behavior, human capital investment and growth.[2] Conversely, some newer papers, notably Acemoglu and Johnson (2007), question if the relationship between life expectancy at birth and economic development is strong or even positive. Acemoglu and Johnson, in particular, construct instruments for life expectancy changes using global public health interventions based on antibiotics (penicillin and others), some vaccines, and DDT. While DDT falls in the prevention category, the authors show that removing malaria from consideration does not substantially change the predicted life expectancy improvements. They also point out that the two main killers at the time were TB and pneumonia, both treated with antibiotics. Therefore Acemoglu and Johnson's work primarily demonstrates that treatment-based interventions increased population growth.

Similarly, Hansen's (2014) work finds that the sharp mortality decline in mid-twentieth century US did not lead to slower population growth. The author measures the effect of life ex-

[2]For example, see Bleakley and Lange (2009) and Fogel (1996) for country-specific evidence, Bleakley (2010) and Lorentzen *et al* (2008) for cross-country evidence. On the theoretical side, Bar and Leukhina (2010), Boldrin and Jones (2002), Cervellati and Sunde (2011), Kalemli-Ozcan (2002), Lagerlöf (2003), Soares (2005) and Strulik and Weisdorf (2014) are but a few of the recent contributions on the mortality-fertility link. Schultz (1997) provides a review of the earlier literature.

pectancy improvements caused primarily by antibiotics, focusing on the treatment-based phase of the mortality decline compared to the nineteenth century's prevention-based decline (Cutler *et al.*, 2006). Both paper's findings are consistent with our theoretical predictions of the effect of treatment-based mortality reduction on population growth and economic development. Furthermore, our work cautions against the use of mortality alone to gauge the relationship between population health and economic development.[3]

This paper deepens our understanding of the demographic transition based on several recent papers. Doepke (2005) points out that under CRRA preferences and certainty, improving child survival lowers the total fertility rate but leaves net fertility unaffected, contrary to historical demographic transitions. In our model, even with these two assumptions, net fertility responds to child mortality because child quality is heterogeneous and parents view healthy and unhealthy children as imperfect substitutes. The epidemiological transition receives no attention in the literature except for Morand (2002). Since Morand's model does not account for the shifting disease pattern observed during the transition nor its effect on fertility and child quality, it is more appropriately viewed as a health transition that enhances overall quality of life. We share Birchenall's (2007) view that infectious disease declines were instrumental in the transition towards modern economic growth. Birchenall distinguishes between infection and case fatality rates and allows for childhood health to influence adult mortality, but the transition itself occurs through parental investment in nutrition.

Closely related is de la Croix and Licandro's (2013) explanation for western Europe's fertility transition based on rising life expectancy and sustained physical development. Their theory does not explain though why a comparable physical development has not occurred in Africa. By providing a micro-foundation for the relationship between mortality and health and by isolating the scarring effect of childhood disease, we are able to account for Africa's slow fertility transition and its higher incidence of adult mortality and morbidity. Our prior work, Aksan and Chakraborty (2013), is the first to connect sub-Saharan Africa's morbidity change to its slow fertility transition. Using a partial equilibrium model and data on infectious disease, it identifies the effect of child survival uncertainty. While we ignore uncertainty here, modeling the general equilibrium effects of the mortality transition captures a broader canvas and time-series behavior of mortality and fertility.

The rest of this paper is organized as follows. Section 2 presents a set of stylized facts on the demographic and epidemiological transitions and health. Section 3 constructs a dynastic model whose implications are developed in Sections 4 and 5. Quantitative experiments in Sec-

[3]In the industrialized world, a significant portion of the prevention-based life expectancy improvement and fertility reduction had already occurred by the twentieth century, the period of study in Acemoglu and Johnson (2007) and Hansen (2014). Any positive effect from health improvements would, hence, be understated.

tion 6 highlight the cost of childhood morbidity and the model's ability to explain SSA.

2 Some Facts

We begin with some facts on the demographic and epidemiological transitions. England & Wales is taken to represent the historical transition since we have reliable time series data on mortality, fertility and cause of death going back to the mid-nineteenth century.

2.1 The Demographic Transition

The conventional view of the demographic transition posits that falling child mortality makes possible subsequent fertility declines. This is the pattern exhibited by early industrializers such as England, Germany and Sweden where fertility responded to mortality with a lag. Figure 1 illustrates the case of England & Wales: the sharp decline in child mortality that began around 1872 was followed by declines in the total and net fertility rates.[4]

There is some evidence based on crude death rates and life expectancy at birth that mortality started falling in England & Wales earlier, from the late eighteenth century. Since age-specific mortality rates are available only from the early nineteenth century – the basis for Figure 1 – it is unclear to what extent those earlier mortality changes were amplified or masked by changing age-structure and, particularly, urbanization (Woods, 2000). The view among demographers is that "the English mortality decline at the national level went through three phases: there was a definite, though rather modest, decline in mortality between 1780 and 1830; this decline was arrested for a period during the mid-nineteenth century; but a second, *decisive period* of decline began during the 1870s" (Hinde, 2003, p. 195, emphasis added).

We focus on the latter period for several reasons. First, of course, is data availability and clear evidence of a steep and secular decline in child mortality. Secondly, this decisive decline was preceded by radical public health reforms that contained, in some cases eradicated, several infectious diseases. McKeown's (1976) view that nutrition and overall improvement in living standards, rather than public health, played a vital role in Britain's mortality transition is now widely questioned (Preston, 1996, Fogel, 1997, Cutler *et al.*, 2006). While the earlier mortality decline in England may have occurred due to better nutrition, the emerging consensus is that the late nineteenth century decline came decisively from public health initiatives. Thirdly, as we show below, health of the English population improved in the late nineteenth century along

[4] Infant mortality did not start falling until the early 1900s. A succession of hot summers in the late nineteenth and early twentieth centuries reduced the availability of clean water, causing diarrhea outbreaks. It has been conjectured that infant mortality would have otherwise fallen earlier (Hinde, 2003). For comparability, we restrict the analysis to child mortality rates in both the UK and SSA.

with child mortality. Our theory connects disease prevention to health improvements and, in our view, those improvements made England's late nineteenth century fertility decline possible. Hence the English transition is an appropriate benchmark against which we can compare the effects of therapeutic interventions. Note also that while this link between child mortality and fertility does not fit all historical transitions (Galor, 2005, Guinnane, 2011), it does fit twentieth century declines: falling child mortality has been instrumental in worldwide fertility reductions during 1955 – 2005 (Angeles, 2010).

Child mortality in sub-Saharan Africa (SSA) has fallen dramatically over the past half-century. Fertility, in contrast, has not fallen as rapidly as we have come to expect from successful transitions (Figure 2). For example, while the child mortality rate (CMR) in Niger fell from 319.4 per 1,000 live births (1966) to 168.5 (2005), its total fertility rate (TFR) increased from 7.26 to 7.3. Uganda's CMR fell from 202.6 per 1,000 live births (1965) to 115.5 (2005), while its TFR declined slightly from 7.1 to 6.6. The elasticity of TFR response with respect to CMR since the 1960s is lowest for SSA, 0.48 versus 0.71 – 0.81 for other developing regions (World Bank). This response is also weaker compared to England & Wales. While CMR fell by similar magnitudes during the first forty years of transition, 46% in SSA (1965-2005) and 45% in England & Wales (1870-1910), SSA's TFR declined by 21% compared to 37% in the latter.

Of course what matters for the demographic transition is net fertility. Here the available evidence is somewhat mixed. Angeles (2010) finds, using data on TFR and NFR, that net fertility fell due to falling child mortality and income growth had little explanatory power. Using data on the crude birth rate, Herzer *et al.* (2012) find the reverse, that mortality declines alone were insufficient to reduce net fertility, economic growth was necessary. In our model income has no effect on fertility. But it is not hard to imagine how weak GDP growth that depresses investment in water and sanitation can keep child morbidity high – see section 2.3 below – even as CMR falls. This would keep net fertility high even as total fertility fell. In this sense, Africa's slow GDP growth would reinforce the mortality-morbidity margin that we identify.[5]

2.2 The Epidemiological Transition

The onset of the epidemiological transition – the shift in mortality and disease patterns identified by Omran (1971) – makes possible the demographic transition. Declining infectious disease mortality among children and the elderly is followed by declining non-infectious disease death, primarily from CVDs, among the middle-aged and elderly. Figure 3 shows how the transition unfolded in England & Wales: infectious disease mortality started falling around 1872 and non-

[5] On the other hand, since we are contrasting the African experience to that of England & Wales, it is also unclear that income growth was instrumental in driving down the English net fertility rate (Bhattacharya and Chakraborty, 2014).

infectious disease mortality followed after about twenty years (Arora, 2005).[6]

Here too the experience of SSA differs. Figure 4 compares infectious disease deaths for children and non-infectious disease deaths for the elderly between SSA and England & Wales. Statistics for the latter are reported at three levels of life expectancy at birth. English life expectancy of 53 during 1905-14 comes closest to the current life expectancy of $42-63$ in SSA. Using that number as a crude age-standardized measure, the infectious disease burden in the two regions looks comparable. The non-infectious disease burden, on the other hand, is more than four times higher in SSA. Underlying this is the earlier age at which CVD deaths have been occurring there. The Global Burden of Disease project (2004) reports that CVD deaths accounted for 30% of all non-HIV-related deaths among $30-69$ year olds in SSA, about four times that in the US (Leeder *et al.*, 2003).

This difference is picked up by data on adult mortality. During the past seventy years, life expectancy at birth has converged around the world due to improvements in child survival in developing countries (Soares, 2007). In contrast, the adult survival rate, or its complement, the probability of dying between the ages of 15 and 60 ($_{45}q_{15}$), shows no such tendency. In Figure 5 this non-convergence is driven by Africa's persistently high adult mortality pre-dating the HIV crisis.

2.3 Childhood Morbidity

One way to assess childhood morbidity is to look at adult stature, the cumulative product of childhood nutrition net of various claims on it including infections. The decline of infectious disease mortality in England & Wales was followed about fifteen years later by height gains in successive cohorts of 18-year old males (Figure 6). These gains occurred as childhood disease morbidity fell along with mortality (Bozzoli *et al.*, 2009; see also Voth and Leunig, 1996).[7]

Compared to this clear pattern, DHS survey data presented in Figure 7 shows that cohorts of African women enjoying better childhood survival did not experience pronounced height gains. In fact, after modest gains, average height has fallen in SSA since 1960. In principle this reflects the combined effect of childhood nutrition and disease exposure. Since nutrition in the first five years of life are crucial for physical development, we report 5-year moving averages of daily

[6] Death rates are age-standardized. The epidemiological transition is often stated as an increasing incidence of noninfectious disease deaths when infectious disease mortality falls. This is certainly true of England & Wales: the share of all disease-caused deaths due to noninfectious disease rose over time. Arora's work shows that this is because people were living longer and that noninfectious disease mortality *rates* among the middle-aged and elderly fell.

[7] The sharp fall in the decades preceding 1850 in Figure 6 has been linked to urbanization and a series of epidemics. It *preceded* the secular decline of child mortality. In fact it was the catalyst for aggressive public health initiatives that made possible the subsequent mortality transition (Szreter, 1988). Nineteenth century height gains were not unique to England (de la Croix and Licandro, 2013).

calorie intake (FAO) in Figure 7. Except for the late eighties, nutrition has improved since 1960.[8]

Calorie supply data may not identify disruptions in food supply, for example due to civil wars. Moradi (2006) finds that the latter explains little of the decline in stature. Likewise Africa's debt crisis and structural adjustment programs can only explain the decline in the late 1980s. Even there the evidence is unclear. Real public health expenditures generally rose or remained steady during structural adjustment due to increased World Bank adjustment lending. User fees instituted in some countries as part of the reforms were irregularly enforced, often accounting for a small percentage of national budgets. Where reforms may have had some impact was in shifting health care priorities from preventive to curative strategies (Sahn and Bernier, 1995), consistent with our theory.

Two further clarifications are in order. First, the trends that emerge from Figure 7 are unlikely to be driven by country composition. The nutrition data covers the same 44 countries throughout these years. We do have missing observations for CMR during 1960-75 (23 countries with data in 1960, 29 in 1965, 38 in 1970, 42 in 1975, 44 1980 onwards). The height data is available for over 30 countries, including 8 of the 10 most populous countries in SSA. Exact country composition for a particular year changes, but for those countries for which height data is reported in the DHS, the data is available for several decades. Akachi and Canning's (2010) systematic analysis, including restricting the sample to countries with complete data, affirms the overall trend of Figure 7. Secondly, selection is unlikely to explain Figure 7. If innately strong children survive to adulthood when CMR is very high, recent declines in stature throughout SSA may reflect a weakening of this selection effect. Yet this would have been also true of historical transitions. As seen by a clear increase in adult stature following mortality declines, morbidity must have declined enough in Europe to dominate any such selection effect.

We conclude that Figure 7 strongly indicates Africa's childhood disease exposure slowed physical improvements initially, possibly reversing them later, despite continuing improvements in childhood survival.

2.4 Child Quality

Next consider evidence on the long-term effects of childhood health. A well-established body of research links adult height to higher earning, and birthweight to higher schooling attainment, height and earnings. A recent example is Case and Paxson (2010) who find that taller individuals attain higher levels of education and that height is positively associated with better economic, health, and cognitive outcomes, associations only partly explained by the higher education lev-

[8]Figure 7 data are 1985 population weighted. Dropping Nigeria, which accounts for close to a quarter of SSA's population, takes the correlation between height and nutrition from 0.02 to a still weak 0.2. Protein intake shows similar improvements over the years.

els of taller individuals. Almond's (2006) study of the effect of the 1918 influenza epidemic on cohorts born in and around that time finds adverse lifetime socioeconomic and physical effects.

The process by which infectious diseases affect child quality is well understood: frequent infections prevent the absorption of nutrients necessary for optimal cellular growth, affecting children's physical and cognitive development (Martorell and Habicht, 1986). Anemia is the most common type of malarial morbidity in Africa, estimated to be 75% in areas where prevalence exceeds 25%. It is associated with poor school performance and lower earnings among working adults (Snow *et al.*, 2003; see Bleakley, 2007, for evidence on hookworm-related anemia and Bleakley and Lange, 2009, for its effect on fertility). Malarial infections are blamed for a 14-24% birthweight shortfall among slaves (Coelho and McGuire, 2000), a 1.1 inch height gap among Union Army veterans (Hong, 2007) and lower education and worse cognitive functions in the Taiwanese population (Chang *et al.*, 2011).

Diarrheal infections are the next highest cause of child mortality in Africa. Many children survive them but infections are frequent, averaging 9 median episodes during the first 4 years (Boschi-Pinto *et al.*, 2006). Checkley *et al.* (2008) report that the possibility of stunting at 24 months of age increases multiplicatively with each diarrheal episode. More direct evidence comes from Guerrant *et al.* (2003): diarrhea in the first 2 years of life is strongly associated with impaired cognitive function $4-7$ years later among Brazilian children even when disease symptoms did not manifest.

2.5 Late-Life Mortality

Childhood infections can increase susceptibility to non-infectious disease later in life. Measles, typhoid and malaria during childhood are known to be associated with cardiovascular problems, and pneumonia before age five to diminished respiratory function at ages 59-70 (Khosla, 1981, Barker 1994). Males who were *in utero* during the Spanish flu epidemic of 1918 had a 23% higher rate of heart disease in their $60-80$s regardless of whether or not their mothers had visible flu symptoms (Mazumder *et al.*, 2010). People who experienced higher malaria exposure risk in Taiwan had a higher incidence of CVDs and a higher mortality hazard in old age (Chang *et al.*, 2011).

In England, France, Sweden and Switzerland, improved longevity in the elderly, as indicated by declines in CVD, occurred among the same birth cohorts that experienced mortality reduction at younger ages. These cohorts were taller than their predecessors (Crimmins and Finch, 2006). Declining childhood and young adult infectious disease rates among Union Army veterans accounted for nearly 50% of the higher survival rates among 50–64 year old males (Costa, 2003). Jousilahti *et al.*'s (2000) study of Finnish men and women offers another set of evidence

linking childhood health to late-life mortality: height is inversely associated with CVD and total mortality, controlling for other known risk factors.

Figure 5 hints at this link between infectious and non-infectious diseases: non-infectious disease mortality followed infectious disease mortality by about a generation during the English epidemiological transition.

3 The Model

Our theory accounts for the facts outlined above by distinguishing between two ways child mortality can be lowered: lower disease prevalence (reduced exposure) or lower case fatality rate from an infection (better therapy or treatment). We argue that better therapy played a vital role in SSA's child mortality declines while historical declines, including England & Wales', came from the conquest of infectious diseases.[9]

The model economy is comprised of an infinite sequence of three-period lived overlapping generations of families. Not everyone lives for all three periods as some children die before reaching adulthood and some older adults die prematurely. Individuals are socially and economically active only in youth and old age. As active decision makers, young parents care about their own consumption and the number and human capital of their surviving children. Besides working, they allocate their time to raising surviving children and educating them.

Some children die in their early childhood from infectious disease. Define the survival rate of infected children by $p \equiv 1-d$ where d is the average case fatality rate from infections, that is, the mortality rate conditional on contracting infections. If i denotes the average childhood infection rate, then the (unconditional) child mortality rate is id. While the fatality rate is taken to be exogenously given by the state of medical knowhow and health care, the infection rate will be later determined endogenously.

All children are assumed to be equally healthy at birth. Of the n_t children born to a young adult in period t, $(1-i_t)n_t$ children are healthy (denoted by h), never having contracted infectious diseases. Another $pi_t n_t$ children are unhealthy (denoted by u), having experienced but survived from chronic infections. The total number of surviving children is then $(1-i_t)n_t + pi_t n_t = (1-di_t)n_t$.

[9] Since developing countries have all benefited from cures invented in the West, this also explains why the TFR response to CMR in other developing regions, while higher than SSA's, has been generally weaker than England & Wales'. That adult stature improved and adult mortality fell in these regions indicates that morbidity fell sufficiently with mortality unlike SSA (details available upon request).

3.1 Households and Preferences

A parent invests education time $e_t^h \in [0,1]$ and $e_t^u \in [0,1]$ towards each type of surviving child. The time cost of rearing an unhealthy child, τ_u, could be different from that of a healthy child, τ_h, although it need not be for the analysis. As an unhealthy child may need more parental care due to frequent illness, it is natural to think $\tau_u \geq \tau_h$.

Parents base their decisions on the expected number of survivors of each type. A young parent with human capital x_t maximizes expected lifetime utility[10]

$$U_t = \ln c_t^t + \beta\phi_t \ln c_{t+1}^t + \gamma\theta \ln\left[(1-di_t)n_t\right] + \gamma(1-\theta)\left[\frac{1-i_t}{1-di_t}\ln x_{t+1}^h + \frac{pi_t}{1-di_t}\ln x_{t+1}^u\right] \quad (1)$$

by choosing $\{c_t^t, c_{t+1}^t, s_t, n_t, e_t^h, e_t^u\}$ subject to the constraints

$$c_t^t = \left[1 - \left\{(1-i_t)(\tau_h + e_t^h) + pi_t(\tau_u + e_t^u)\right\} n_t - s_t\right] z_t \quad (2)$$

$$c_{t+1}^t = \frac{R_{t+1}}{\phi_t} s_t z_t \quad (3)$$

$$x_{t+1}^h = \lambda(\varepsilon + q^h e_t^h)^\nu x_t^\kappa \bar{x}_t^{1-\kappa}, \ e_t^h \geq 0 \quad (4)$$

$$x_{t+1}^u = \lambda(\varepsilon + q^u e_t^u)^\nu x_t^\kappa \bar{x}_t^{1-\kappa}, \ e_t^u \geq 0 \quad (5)$$

and taking the vector $(\phi_t, i_t, w_t, R_{t+1})$ as given. Here $\beta \in (0,1)$ is a subjective discount rate, $\phi_t \in (0,1]$ is the probability of surviving through old age and $\theta \in (0,1)$ is parental valuation of child quantity versus quality. There are decreasing returns to quality investment $\nu \in (0,1)$, the productivity parameter λ is positive and $\varepsilon > 0$ ensures positive human capital in the absence of quality investment. Wage per unit of human capital is represented by w while $s \in (0,1)$ denotes the propensity to save out of "full income" $z \equiv wx$. The second budget constraint incorporates the assumption of perfect annuities.

The last term in the utility function is a joy-of-giving bequest motive; $(1-i)/(1-di)$ is the fraction of surviving children who are healthy, $pi/(1-di)$ the fraction who are unhealthy. For $i = 0$ this term simplifies to $\ln x^h$, for $i = 1$ to $\ln x^u$. Hence the utility function nests a commonly used quantity-quality specification for homogeneous child quality, $\gamma[\theta \ln n + (1-\theta)\ln x]$. Embedded in parental altruism is an important assumption: healthy and unhealthy children are imperfect substitutes. As will be clear shortly, this is central to generating the appropriate fertility and investment responses from a decrease in child mortality.

The last two constraints in the parent's decision problem specify the human capital production function for each type of surviving child. Besides parental investment and human capital,

[10] Utility from premature death in old age is being normalized to a large negative number and parameter values are implicitly assumed to ensure old age consumption remains sufficiently above zero. That adults do not die from CVDs during their working life is a simplification.

they depend on the average human capital across working adults, $\bar{x}$, when $\kappa \in (0,1)$. The return to investment in child quality depends on health human capital q^j, $j \in \{h,u\}$, which is the outcome of a child's disease experience. Specifically it takes the value $q^h = 1$ should the child have experienced no (significant or recurrent) infectious disease and $q^u = \delta \in (0,1)$ otherwise. That illness from infectious disease depreciates intrinsic health and cognitive abilities through δ is relevant only when quality investment is positive. In other words, in the absence of educational investment, childhood morbidity has no effect on the quantity-quality tradeoff. Since the evidence on child quality suggests that the return on nutritional investment too depends on the childhood disease experience, in a more general setup child quality will include all forms of investment that augment a child's future labor productivity.

3.2 Old Age Mortality

Old age non-infectious diseases affect survival when people are retired and consume out of accumulated wealth. The survival probability depends on childhood health in line with the evidence presented in section 2.5. Specifically, an individual who survived from childhood infections in period $t-1$ to become a young adult in period t faces a lower survival probability, ϕ_u, as an elderly in period $t+1$ compared to ϕ_h for an individual who was a healthy child. That is,

$$\phi_t = \begin{cases} \phi_u, & \text{if } \mathscr{I}_{t-1} = 1 \\ \phi_h, & \text{if } \mathscr{I}_{t-1} = 0 \end{cases} \tag{6}$$

where $\mathscr{I}_{t-1}$ is an indicator function that takes the value 1 if the person was infected as a child in $t-1$ and zero otherwise, and $\phi_u < \phi_h = 1$.

3.3 Aggregate Production and Factor Prices

Let X denote the aggregate stock of human capital and K the aggregate stock of physical capital. Production of final output Y occurs according to the technology

$$Y_t = AK_t^{\alpha} X_t^{1-\alpha} \tag{7}$$

where $A > 0$ and $\alpha \in (0,1)$.

The depreciation rate on physical capital is hundred percent. Let R denote the rental on capital as well as the interest factor and $k_t \equiv K_t / X_t$ the physical-to-human capital ratio. In perfectly competitive output and inputs markets, (7) leads to the familiar pricing functions, $w_t = (1-\alpha) A k_t^{\alpha}$ and $R_t = \alpha A k_t^{\alpha-1}$ for labor and capital respectively.

3.4 Household Decisions

Turn now to household behavior and denote the average cost per childbirth by

$$\chi_t \equiv (1-i_t)(\tau_h + e_t^h) + pi_t(\tau_u + e_t^u). \tag{8}$$

Substituting the production function for human capital into (1) and ignoring additive constant terms, the decision problem becomes

$$\begin{aligned}\text{Max}_{\{s_t, n_t, e_t^h, e_t^u\}} \ & \ln\left[\left(1-\chi_t n_t - s_t\right) z_t\right] + \beta\phi_t \ln\left[s_t z_t\right] + \gamma\theta \ln[(1-di_t)n_t] \\ & + \nu\gamma(1-\theta)\left[\frac{1-i_t}{1-di_t}\ln(\varepsilon + e_t^h) + \frac{pi_t}{1-di_t}\ln(\varepsilon + \delta e_t^u)\right]\end{aligned}$$

subject to e_t^h, $e_t^u \geq 0$. Inada conditions guarantee that inequality constraints for consumption in the two periods are satisfied. Besides the interior equilibrium, two types of corner equilibria are possible: $e_t^u = e_t^h = 0$ and $e_t^u = 0$, $e_t^h > 0$. As long as the average cost per childbirth is low, which occurs for relatively high prevalence rates, parents choose large families over investing in child quality.[11]

Interior equilibrium

In an interior equilibrium for quality investment, investment in unhealthy children is lower the higher is the morbidity effect of infections (lower δ)

$$e_t^u = e_t^h - \left(\frac{1-\delta}{\delta}\right)\varepsilon. \tag{9}$$

In turn this implies their human capital is less than that of healthy children, $x_{t+1}^u = \delta^\nu x_{t+1}^h$. Using (9), we express the average cost of fertility as

$$\chi_t = \tau_t + (1-di_t)e_t^h - \eta i_t \tag{10}$$

where $\tau_t \equiv (1-i_t)\tau_h + pi_t\tau_u$ is the average cost of child quantity and $\eta \equiv (1-\delta)\varepsilon p/\delta$ the cost saving from investing in unhealthy children relative to healthier ones. It follows that

$$s_t = \frac{\beta\phi_t}{1+\gamma\theta+\beta\phi_t}, \ n_t = \frac{\gamma\theta}{1+\gamma\theta+\beta\phi_t}\left(\frac{1}{\chi_t}\right),$$

[11] Here parents do not take any action to lower their children's exposure to infections. Since infectious disease transmission depends strongly on a negative externality, for high enough prevalence rates, parents would optimally choose not to invest in prevention (Chakraborty *et al.*, 2010). We focus on macro-level prevention when discussing the health transition. Prevention at the household level would reinforce the benefits of these macro-policies. Without the latter, prevention at the household level would be too weak to facilitate the transition.

and that quality investment in healthy children is

$$e_t^h = \frac{\nu(1-\theta)}{\theta-\nu(1-\theta)}\left[\frac{\tau_t-\eta i_t}{1-di_t}-\frac{\varepsilon\theta}{\nu(1-\theta)}\right]. \tag{11}$$

Note that quality investments are decreasing in i_t when $\psi(i_t)\equiv(\tau_t-\eta i_t)/(1-di_t)$ is decreasing in i_t. This requires that $p(\tau_u-\tau_h)-\eta<0$, or,

$$\tau_u < \tau_h + \left(\frac{1-\delta}{\delta}\right)\varepsilon \tag{A1}$$

which is satisfied for sure when $\tau_u=\tau_h$. Assumption (A1) requires that unhealthy children are not too costly; else, fewer sickly children would free up so much parental time that parents would choose to have more children. For (11) to be economically meaningful and to satisfy the second order conditions we assume that

$$\varepsilon < \nu(1-\theta)\tau_h/\theta \quad \text{and} \quad \nu<\theta/(1-\theta) \tag{A2}$$

respectively. The first restriction ensures that maximal human capital investment in healthy children is positive, that is $e_t^h>0$ when $i_t=0$. It implies that intrinsic human capital ε is not so high that parental investment is redundant. The second restriction $\theta>\nu(1-\theta)$ is related to the standard assumption in the literature that parents care more about the quantity of children than quality for the second order condition to be satisfied. The condition is actually less stringent: all we need is parents do not value quality "too much" since the restriction is satisfied even with $\theta<1/2$ (that is, quality is valued more than quantity) as long as $\nu<\theta/(1-\theta)$.

Collecting these results, equilibrium expressions for the average cost of childbearing and human capital are

$$\begin{aligned}
\chi_t &= \frac{\theta}{\theta-\nu(1-\theta)}\left[\tau_t-\eta i_t-(1-di_t)\varepsilon\right],\\
x_{t+1}^h &= \lambda x_t^{\kappa}\bar{x}_t^{1-\kappa}\left[\frac{\nu(1-\theta)}{\theta-\nu(1-\theta)}\right]^{\nu}\left[\frac{\tau_t-\eta i_t}{1-di_t}-\varepsilon\right]^{\nu},\\
x_{t+1}^u &= \delta^{\nu}x_{t+1}^h.
\end{aligned}$$

Unhealthy children receive less human capital investment and the return on that investment is lower. As adults they also face lower longevity that reduces their saving propensity. Differential human capital outcomes for healthy and unhealthy children arise in our model because we assume the two types are imperfect substitutes and parents do not take an egalitarian approach towards their offspring's human capital. If they did, they would have chosen $e_t^u=e_t^h/\delta>e_t^h$,

that is, more investment in unhealthy children to compensate for their health shock.[12] In not following this approach, we are relying on an established literature in development economics that consistently finds evidence of intrahousehold discrimination. For example, Akresh *et al.*'s (2012) study on Burkina Faso finds that lower ability children are typically less likely to be enrolled in school and that having higher ability siblings has at least as much effect on parental quality investment. While it is reasonable to think that some families would eschew such discrimination, the overall evidence indicates that such behavior is much too common in developing countries.

Corner equilibrium

Besides assumption (A2), positive quality investment in healthy children requires that

$$\Gamma^h(i_t) \equiv \psi(i_t) - \frac{\theta\varepsilon}{\nu(1-\theta)} \geq 0.$$

Likewise $e_t^u \geq 0$ requires that

$$\Gamma^u(i_t) \equiv \psi(i_t) - \frac{\theta\varepsilon}{\nu(1-\theta)} - \frac{\theta - \nu(1-\theta)}{\nu(1-\theta)}\left(\frac{1-\delta}{\delta}\right)\varepsilon \geq 0.$$

Evidently $\Gamma^h \geq \Gamma^u$ while (A1) ensures that ψ is decreasing in i_t. Let the threshold prevalence rates i^h and $i^u < i^h$ be defined implicitly by $\Gamma^h(i^h) = 0$ and $\Gamma^u(i^u) = 0$.

For $i_t \in (i^u, i^h)$, $\Gamma^h > 0$ but $\Gamma^u < 0$. Here parents invest in healthy children but not in unhealthy ones: $e_t^h > 0$, $e_t^u = 0$. While the saving propensity s_t is the same as in the interior equilibrium, other decisions change. Using the new expression for the average cost per childbirth, $\chi_t = \tau_t + (1-i_t)e_t^h$, we obtain

$$e_t^h = \frac{\nu(1-\theta)}{\theta - \nu(1-\theta)\left(\frac{1-i_t}{1-di_t}\right)}\left[\frac{\tau_t}{1-di_t} - \frac{\varepsilon\theta}{\nu(1-\theta)}\right], \quad n_t = \frac{\gamma\theta}{1+\gamma\theta+\beta\phi_t}\left(\frac{1}{\chi_t}\right).$$

For $i_t > i^h$, on the other hand, $\Gamma^h < 0$ and parents do not invest in either healthy or unhealthy children, $e_t^u = e_t^h = 0$. The saving decision is unchanged but since the average cost is lower still at $\chi_t = \tau_t$, fertility is the highest in this corner equilibrium

$$n_t = \frac{\gamma\theta}{1+\gamma\theta+\beta\phi_t}\left(\frac{1}{\chi_t}\right).$$

[12] Even so, healthy and unhealthy children would have different lifetime utility since their ϕ's differ.

The Effect of Mortality

Relevant to the demographic transition are how quantity and quality of children respond to child mortality. The latter depends on the prevalence rate i_t and the case fatality rate d, both of which are exogenous to a parent's decisions.

The household fertility rate n_t, which we shall call the total fertility rate (TFR) for convenience even though it differs across households due to adult longevity, responds negatively to fertility cost χ_t. Consider first interior quality equilibria where χ_t is a decreasing function of both i_t and d by assumption (A1). Here a decrease in child mortality either through the prevalence or case fatality rate lowers the TFR, consistent with the facts outlined earlier.

For the demographic transition, what is also relevant is the expected number of surviving children, $\hat{n}_t = (1 - d\,i_t)\,n_t$, or the net fertility rate (NFR). This is given by

$$\hat{n}_t = \frac{\gamma[\theta - \nu(1-\theta)]}{1 + \gamma\theta + \beta\phi_t}\left[\frac{1}{\psi_t - \varepsilon}\right]$$

where ψ_t was defined earlier. Under assumption (A1), ψ_t is decreasing in i_t but increasing in d. This means the NFR responds positively to i_t but negatively to d. When child mortality falls due to lower prevalence rates, the TFR decline is strong enough to ensure that NFR falls. In contrast, a decrease in the child mortality rate due to lower case fatality elicits a weak TFR response and the NFR increases despite fertility falling.[13]

Next consider the quantity-quality tradeoff. Quality investment in either type of child is increasing in ψ_t. Thus a reduction in child mortality through lower prevalence raises quality investments in both healthy and unhealthy children. The opposite is true for gains in child survival through lower d. A lower d implies that fewer sickly children succumb and a higher proportion of surviving children are of low quality. Parents have to devote more time towards raising unhealthy children on whom quality investment yields low returns.

Child mortality has a different effect on population growth in corner equilibria. For example, when parents do not invest in either type of child, the NFR responds negatively to i_t but positively to d unless $\tau_u = \tau_h$ in which case the NFR is unaffected, similar to Doepke (2005). Lower d shifts the childbearing cost towards unhealthy children who require more attention, lowering the demand for children. In contrast, lower i shifts the cost towards healthier children and demand rises. In the next generation, however, less healthy (healthier) adults will have more (fewer) children in response to their lower (greater) longevity, ϕ_t. Hence the overall NFR

[13] A lower prevalence or case fatality rate means fewer sickly children and, *ceteris paribus*, less time raising a given number of children. Assumption A1 is a robustness check to show that as long as unhealthy children do not require too much parental time commitment, total and net fertility rates behave this way when child mortality falls. Later, in the quantitative simulations, we restrict to $\tau_h = \tau_h$ for which these comparative statics results hold for sure.

response is ambiguous. Since we set $\tau_u = \tau_h$ in the quantitative work later, only the longevity margin is at play for SSA's simulated net fertility transition during the 1960s.

Beyond these, a continued decline in prevalence as it crosses first the threshold i^h, then i^u, has a general equilibrium effect: parents switch from valuing quantity alone to also valuing child quality. Furthermore, as a reduction in prevalence extends adult longevity, parents place a higher weight on their future consumption and fertility falls further. While the overall effect on fertility depends on equilibrium type and child morbidity, a reduction in case fatality alone does not generate a similar effect from adult survival to family size.

3.5 Disease Dynamics

While the prevalence rate i_t is exogenous to a household's decisions, it evolves endogenously in the aggregate. A pre-transition or transitional economy with high average mortality is best viewed as one where infectious diseases are endemic. We use a parsimonious version of the SIR epidemiological model for this. Infections can spread from infected to susceptible individuals directly or indirectly through non-human disease vectors. Tuberculosis, an airborne disease, largely spreads from close proximity to infected human. For malaria, the infection spreads through mosquitoes that take blood meal from an infective person and, after sporogony, release the parasite into the bloodstream of a susceptible individual. Diarrhea, on the other hand, can spread both directly and indirectly as the vector can be any number of possibilities including food, water, livestock and humans.

To account for these modes of transmission, the microbial load is assumed to be proportional to the fraction of currently working-age population who were infected in their childhood. This implicitly assumes that the ratio of vectors to the affected population is constant: if more children were infected, human and non-human vectors increase proportionately for future infections. For instance, mosquitoes become carriers when picking up the parasite from a human and more of them do when more people are infected. The current prevalence rate i_t then proxies for the density of disease microbes in $t+1$.

Susceptible newborns are exposed to $\mu > 1$ types of disease vectors and the transmission rate from a random match with a disease vector is $a > 0$. Then the number of newborns who contract infections in $t+1$ is $I_{t+1} = \pi_t \bar{n}_{t+1} L_{t+1}$ where $\pi_t = \mu a \left[p i_t / (1 - d i_t) \right]$ and the L_{t+1} young adults at $t+1$ each have $\bar{n}_{t+1}$ children on average. For high enough prevalence, π_t can exceed one. Hence we specify that the prevalence rate $i_t \equiv I_t / (\bar{n}_t L_t)$ follows

$$i_{t+1} = g(i_t) \equiv \min\{\pi(i_t), 1\} \tag{12}$$

given $i_0 > 0$. Equation (12) always entertains zero prevalence as a steady state whose stability

depends on $g'(0) = \mu ap$. The product μa is the so-called epidemiological threshold or *basic reproduction ratio*, the number of secondary cases per primary case of infection. We assume that in a pre-transition population

$$\mu a > 1, \tag{A4}$$

that is, each infection is capable of causing multiple others. Under some conditions this can cause the disease to become endemic over time. In addition to (A4) when we have $\mu ap > 1$, the zero-prevalence steady state is asymptotically unstable and $\pi(\hat{\imath}) > 1$ for $\hat{\imath} \equiv 1/[d + \mu ap]$. Full prevalence is the only asymptotically stable steady state in this scenario, as in Figure 8(a), and the disease is eventually endemic. Given the broad spectrum of infections that children are exposed to in developing countries, i represents the probability of contracting any number of diseases during childhood. Hence the full-prevalence steady state in Figure 8(a) is best thought of as an environment where these diseases, not all of them or the same ones, affect all at some point during their childhood.

Maintaining assumption (A4), suppose instead the survival rate is low enough that $\mu ap < 1$. This corresponds to Figure 8(b), where an intermediate steady state appears at $i^* = (1 - \mu ap)/d$ that is increasing (decreasing) in the case fatality (survival) rate d (p). For initial prevalence $i_0 > i^*$, infections spread rapidly until full prevalence is reached. Infections eventually abate, in contrast, for i_0 below i^*. Even though the basic reproduction ratio exceeds one, enough infected people must survive as vehicles of transmission in order for the disease to reach endemic proportions. Since $\hat{\imath}$ and i^* are decreasing in p, better therapy by raising (lowering) p (d) makes full prevalence more likely for a given i_0. Higher p ensures a larger pool of infective agents and faster transmission rates. In contrast, since i^* is decreasing in μa, improvements in prevention make full prevalence less likely.[14]

The last case, Figure 8(c), holds when (A4) is overturned which also implies $\mu ap < 1$. Zero prevalence is the unique asymptotically stable steady state: the basic reproduction ratio is too weak to forever sustain infections. This approximates the relatively low prevalence of infectious diseases observed in high-income countries. Malarial, helminth and many other types of infections common in developing countries are negligible in high-income countries. The disease burden of infectious and parasitic diseases, measured in DALYs, was 1% of the population under the age of five in high-income countries in 2011 compared to 67% in low-income countries (Global Burden of Disease, 2013). The burden of diarrheal infections specifically was 0.5%

[14]This distinction between the effects of p and μa is somewhat artificial. For pneumonia and pertussis, for example, antibiotics can reduce the time a person remains contagious. Prevention, however, reduces prevalence more than treatment by also removing the window of contagion. Our calibration allows for the possibility that a given intervention has both preventive and curative effects.

in high-income countries and 20% in low-income countries. The figure for childhood-cluster diseases (whooping cough, diptheria, measles and tetanus) was similarly 0.01% versus 9%, respectively.

4 Intertemporal Equilibrium

4.1 Population and Labor Supply

From here on we make the simplifying assumption that $\kappa = 0$ so that a child's human capital is determined by his health history and economic aggregates, not parental characteristics. This human capital takes one of two values that depends on the average stock of human capital and the prevalence rate.

Young adults in t are distinguished by their human capital x_t and old-age survival rate ϕ_t. While human capital can take values on a continuum, old-age survival can be either ϕ_u or $\phi_h = 1$. The cumulative distribution function $G_t(x,\phi)$ denotes the fraction of young adults at t with human capital at or below x and adult survival at or below ϕ. Let $g_t(x|\phi)$ be the conditional density function of x_t for a given value of ϕ_t. A young adult with human capital x_t and old-age survival ϕ_t has $n_t = n(i_t,\phi_t)$ children of whom $pi_t n(i_t,\phi_t)$ grow up to be unhealthy adults with x^u_{t+1} units of human capital and old-age survival rate $\phi_{t+1} = \phi_u$. The remaining $(1-i_t)n(i_t,\phi_t)$ surviving children grow up healthy with x^h_{t+1} units of human capital and old-age survival $\phi_{t+1} = \phi_h$. This determines the future working population as

$$L_{t+1} = (1-di_t)\left[\sum_{\phi}\left\{\int n(i_t,\phi)g_t(x|\phi)dx\right\}\Pr(\phi_t=\phi)\right]L_t \tag{13}$$

given i_t. The probability that ϕ_t takes the value ϕ_u is $\Pr(\phi_t = \phi_u) = pi_{t-1}/(1-di_{t-1})$. The aggregate human capital of L_t workers is $X_t = L_t\sum_\phi\left[\int xg_t(x|\phi)dx\right]\Pr(\phi)$ and human capital per worker $\bar{x}_t \equiv X_t/L_t$. Using the latter, output per worker can be expressed as $y_t = Ak_t^\alpha \bar{x}_t$.

Suppose worker type is costlessly observed and annuity sellers calibrate their returns to the mortality risk of each group. Healthy workers earn the (gross) return R/ϕ_h on their saving, unhealthy workers earn the higher return R/ϕ_u and aggregate saving is given by

$$S_t = \left[\sigma_u\left\{\int xg_t(x|\phi_u)dx\right\}\Pr(\phi_t=\phi_u) + \sigma_h\left\{\int xg_t(x|\phi_h)dx\right\}\Pr(\phi_t=\phi_h)\right]w_tL_t$$

where the saving propensities are

$$\sigma_u \equiv \frac{\beta\phi_u}{1+\beta\phi_u+\gamma\theta} < \frac{\beta\phi_h}{1+\beta\phi_h+\gamma\theta} \equiv \sigma_h.$$

Asset market clearing is ensured by the usual condition $K_{t+1} = S_t$.

4.2 Dynamics

The aggregate stock of human capital comprises of the human capital of healthy and unhealthy workers

$$X_{t+1} = \left[\frac{pi_t}{1-di_t} x^u_{t+1} + \frac{1-i_t}{1-di_t} x^h_{t+1} \right] L_{t+1}.$$

The human capital of each type, x^h_{t+1} and x^u_{t+1}, is linear in $\bar{x}_t$ where the proportionality functions ρ_{ht} and ρ_{ut} depend on the prevalence rate according to

$$\rho_{ht} = \begin{cases} \lambda \left[\frac{\nu(1-\theta)}{\theta - \nu(1-\theta)} \left\{ \frac{\tau_t - \eta i_t}{1-di_t} - \varepsilon \right\} \right]^\nu & \text{when } 0 \le i_t < i^u, \\ \lambda \left[\frac{\nu(1-\theta)}{\theta - \nu(1-\theta)(1-i_t)/(1-di_t)} \left\{ \frac{\tau_t}{1-di_t} - \varepsilon \frac{1-i_t}{1-di_t} \right\} \right]^\nu & \text{when } i^u \le i_t < i^h, \\ \lambda \varepsilon^\nu & \text{when } i^h \le i_t \le 1, \end{cases}$$

and $\rho_{ut} = \delta^\nu \rho_{ht}$ in the first case, $\lambda\varepsilon^\nu$ in the latter two. The evolution of human capital per worker can now be parsimoniously represented by

$$\bar{x}_{t+1} \equiv \frac{X_{t+1}}{L_{t+1}} = \left[\frac{pi_t}{1-di_t} \rho_{ut} + \frac{1-i_t}{1-di_t} \rho_{ht} \right] \bar{x}_t \equiv \rho(i_t) \bar{x}_t \tag{14}$$

and asset market clearing by

$$K_{t+1} = \left[\sigma_u \rho_{ut} \frac{pi_{t-1}}{1-di_{t-1}} + \sigma_h \rho_{ht} \frac{1-i_{t-1}}{1-di_{t-1}} \right] (1-\alpha) A k_t^\alpha \bar{x}_{t-1} L_t \tag{15}$$

given $K_0 > 0$ units of capital owned by the initial old generation and the distribution G_0 of the initial young adult population. Note that the disease history $i_{-1} > 0$ need not be separately specified as it is part of G_0. The evolution of the cumulative distribution is determined by

$$G_{t+1}(x', \phi') = \frac{L_t}{L_{t+1}} \left[\sum_\phi \left\{ \int (1-di_t) n(\phi, i_t) \mathscr{I}(x_{t+1} \le x') g_t(x|\phi) dx \right\} \Pr(\phi_t = \phi) \right] \Pr(\phi_{t+1} \le \phi') \tag{16}$$

where $\mathscr{I}$ is an indicator function.

Definition. *The general equilibrium of this economy consists of sequences of aggregate variables* $\{K_t, L_t, X_t, i_t\}$ *and cumulative distribution functions* $G_t(x, \phi)$ *that satisfy equations* (12) – (16)*, given* $K_0 > 0$*,* $i_0 > 0$ *and* G_0*.*

5 Transitions

Since disease prevalence does not depend on economic behavior, equation (12) evolves independently of equations (14) and (15). The long run trajectory of this economy is driven by disease dynamics, specifically the vector of parameters (i_{-1}, i_0, μ, a, p).

First note that steady-state growth of output per worker depends on whether or not human capital accumulation can be sustained on its own. That requires $\rho(i_t) > 1$ in (14) in which case a decrease in the prevalence rate unambiguously raises the rate of human, and thus, physical capital accumulation. When $\lim_{t\to\infty} i_t = 0$, the asymptotic growth factor of human capital and output per worker is

$$\rho(0) = \lambda \left[\frac{\nu(1-\theta)(\tau_h - \varepsilon)}{\theta - \nu(1-\theta)} \right]^{\nu}.$$

When $\lim_{t\to\infty} i_t = 1$, since no child receives quality investment, the long-run growth factor is

$$\rho(1) = p\lambda\varepsilon^{\nu} < \rho(0).$$

The saving propensities σ_h and σ_u have no effect on the growth rate, only level effect on the balanced growth path (BGP). Thus two BGPs are possible and the economy converges to only one of them for a given vector (i_{-1}, i_0, μ, a, p). The transition from one (Malthusian regime) to the other (modern growth regime) can only be exogenous, for example due to an exogenous reduction in child mortality.

5.1 Balanced Growth Paths

The first BGP, that we call a **Malthusian regime**, exists when (A4) holds and infectious disease fatalities are high enough that $\mu ap < 1$ (Figure 8b). For $i_0 > i^*$, full prevalence is the only stationary equilibrium. Along this BGP, fertility remains high and infections extract a high mortality toll on children. As all children are affected, survivors carry their morbidity burden in the form of low human capital and high risk of premature death from non-communicable disease. Labor income is low, and low adult longevity implies low rates of investment in physical capital. This BGP exhibits slow (if any) growth in income per capita of $\rho(1) - 1$ and persistent morbidity.

The second stationary equilibrium exhibits **modern economic growth** (MEG) and corresponds to $\mu a < 1$ (Figure 8c). Absent any threat of infections, child survival is ensured, fertility low and human capital relatively high. This economy is also in a post-epidemiological transition phase where adult longevity is maximal. High rates of investment in physical and human capital ensure a high growth rate of $\rho(0) - 1$.

Since the prevalence rate is either zero or hundred percent, we can easily compare child quantity and quality outcomes in the two steady states. We proceed with the assumption, validated later by our calibration, that $i^u < 1$ and $i^h > 0$ so that $e^u(i=1) = 0$ and $e^h(i=0) > 0$. Recall that the average fertility cost χ is decreasing in the prevalence rate. In the Malthusian BGP, this cost takes the lowest possible value $\chi_L = (1-d)\tau_u$, in the MEG BGP the highest possible value $\chi_H = \theta(\tau_h - \varepsilon)/[\theta - \nu(1-\theta)]$. It follows that the TFR in the Malthusian BGP takes the high value n_H and in the MEG BGP a lower value n_L where

$$n_H = \frac{\gamma\theta}{1+\gamma\theta+\phi_u}\left[\frac{1}{(1-d)\tau_u}\right],\ n_L = \frac{\gamma\theta}{1+\gamma\theta+\phi_h}\left(\frac{\theta-\nu(1-\theta)}{\theta}\right)\left[\frac{1}{\tau_h-\varepsilon}\right].$$

The fertility differential across these steady-state values is driven by several parameters. The parameters $(d, \tau_u, \tau_h, \delta)$ are related to the cost of fertility, parameters (ϕ_u, ϕ_h) determine parental willingness to substitute between personal consumption and altruistic behavior while $(\theta, \nu, \varepsilon)$ are related to the net benefit of quality investment. The corresponding net fertility rates in the two steady states are

$$\hat{n}_H = (1-d)n_H,\ \hat{n}_L = n_L$$

and human capital investment per child

$$e^u = 0,\ e^h = \frac{\nu(1-\theta)}{\theta-\nu(1-\theta)}\left(\tau_h - \frac{\theta\varepsilon}{\nu(1-\theta)}\right)$$

respectively, since all children are of homogeneous quality in either BGP.

5.2 Transitions Then

Start with assumption (A4) which is more likely when μa is relatively high. Prior to the public health and medical innovations of mid-to-late nineteenth century England, fatalities from childhood infections d would have been high too. Such a pre-transition economy corresponds to Figure 8(b) with $i_0 > i^*$.

John Snow's work in identifying the cause of London's 1854 cholera epidemic, the Public Health Acts of 1848 and 1875 and the commissioning of London's integrated sewerage system in 1865 paved the way for the eradication of infectious disease in England (Szreter, 1988). Improved sanitation and water management led to cleaner water supplies, food safety and effective sewage disposal. Supervision of water, food and pasteurization of milk drastically cut down the incidence of cholera, dysentry, typhoid, hookworm, diarrhea, measles and whooping cough. Immunization programs for diptheria, smallpox, tuberculosis and whooping cough

complemented these public health reforms 1880 onwards. Between 1861 and 1900, over 55% of England's mortality decline was accounted for by the control of five diseases – smallpox, scarlet fever, whooping cough, diarrhea and typhoid – all of which affected children in large numbers (Hinde, 2003). Therapy played a minor role; antibiotics for scarlet fever, for instance, were to be invented later. A similar prevention-based intervention also explains mortality reductions in nineteenth century US (Cutler *et al.*, 2006).

These preventive breakthroughs are akin to a reduction in μa. When the reduction is large enough to overturn (A4), zero becomes the unique asymptotically stable steady state as in Figure 8(c). But innovations do not have to be so drastic as to eliminate i^*. Modest continuing improvements in μa keep raising this threshold prevalence rate, that is i^* keeps falling in Figure 8(b), until an economy converging towards full prevalence falls below it. From then on, since the transmission rate has been brought sufficiently under control, infectious disease pathogens naturally die out. Falling infectious disease mortality *and* morbidity usher in a fertility transition. Both TFR and NFR decline, as parents substitute towards quality investment and late-life survival improves. Rapid demographic and disease transitions, in turn, drive the economy towards MEG.

5.3 Transitions Now

Twentieth century reductions in child mortality in developing countries have been facilitated by the transfer of public health innovations and medical technology, notably vaccines and antibiotics, from the West. In some cases, such as smallpox and polio, these transfers have wiped out major killers of children. Yet progress towards eradication has been uneven and nowhere is this more apparent than in sub-Saharan Africa (SSA). Despite a half-century of falling child mortality at a rate comparable to England & Wales, prevalence rates for Africa's major killer diseases remain high.

This is due to two reasons: deficiencies of best practice interventions and moribund public health systems. Take malaria and diarrhea which together accounted for 34% of child deaths in Africa in 2005 (WHO). The lack of an effective vaccine and inability of current technologies to drastically control disease transmission have made malaria eradication much harder in Africa than in other parts of the world. After some initial success, malaria control strategies have floundered. Eradication campaigns during the 1960-70s reduced malarial mortality by 18%, much of it due to better therapy (chloroquine), some due to vector control (DDT). Since the 1980s the disease has resurged to pre-1960 levels from chloroquine resistance and vectorial resistance to DDT. It now accounts for 18% of child deaths in SSA, as it did prior to 1960.[15]

[15]It is conceivable that this resurgence partly accounts for the drop in adult stature in the 1980s directly, and

Diarrheal mortality, on the other hand, has fallen in Africa by about 10.5% during 1990-2000. Most of this reduction is not due to lower incidence but from the spread of an effective treatment (ORT) which reduced diarrheal mortality by 9% (Ewbank and Gribble, 1993; Boschi-Pinto *et al.*, 2006). Moreover ORT usage rates remain below 50%, as low as 7% in Botswana (UNICEF). The problem is compounded by misplaced priorities: most interventions have been directed towards containing acute dehydrating diarrhea through ORT while ignoring antibiotic therapy for treating dysentery and persistent diarrhea. A review of 73 studies covering 23 countries during $1970-90$, at the height of child mortality declines, found that children experienced a high average diarrheal episodes of 5 per year (ARCH Project Special Report, 1998). Since as much as 88% of diarrheal deaths in Africa is water and sanitation related, this reflects an institutional failure to provide adequate sanitation and potable water, interventions that so radically transformed England's disease landscape (Guerrant *et al.*, 2003).

Africa's fastest child mortality declines occurred during 1975-85 as the WHO launched new vaccination programs. Under the Expanded Program on Immunization (EPI), vaccination for measles, diphtheria-pertussis-tetanus, poliomyelitis, and tuberculosis improved significantly. Prior to eradication in 1979, smallpox killed 30% of those infected and left 65-80% of survivors with visible scars, in many cases blind. Eradication had a strong impact not just on child mortality but on long-term morbidity too. Similarly, complicated by malnutrition and parasitic infections, measles fatality rates used to be as high as 25% in Africa. In this case, however, the reduction in measles cases from the introduction of the vaccine in 1964 would have made little difference to overall childhood morbidity since measles does not usually have long-term health effects (Ewbank and Gribble, 1993).[16]

Ultimately successful disease prevention rests on both private (e.g. boiling water, getting vaccinated, investing in insecticide-sprayed bed nets) and public interventions (e.g. provision of clean water, accessible and well-equipped health clinics, regular draining of stagnant water).[17] Private health investment exhibits positive externalities, so without public interven-

indirectly from co-morbidity with other diseases.

[16]Measles remains a leading cause of African child mortality, a lesson in the limitations of technology transfer. Introduction of the vaccine in West Africa was subject to political and business pressure as the US government advocated a US-produced vaccine over a French one despite sparse evidence of its effectiveness. Besides miscalculating the target population and under-providing vaccines, USAID failed to anticipate ground realities. The vaccines required careful storage in cold temperatures, out of the sun. Even with refrigerators this proved to be challenging in West Africa's hot climate. Lawrence K. Altman provides an entertaining account in "How Tiny Errors in Africa Led to a Global Triumph", *New York Times*, Sept. 26, 2011, and explains how this paradoxically led to a successful smallpox campaign. Subsequent vaccination programs have not always proved to be as effective as clinical trials due to the low level of health services, inappropriate age at vaccination and incorrect vaccination procedures (Ewbank and Gribble, 1993).

[17]Prevention seems to have been vital in eliminating malaria, cholera and smallpox during Sri Lanka, Costa Rica, Jamaica and Kerala's (India) successful demographic transitions. This was made possible by improvements in public health and schooling as well as the relatively higher status enjoyed by women in the two South Asian cases

tion there is too little of it. Effective public intervention, on the other hand, requires credible macroeconomic policies and institutions that are costlier to implement than importing (subsidized) therapeutic interventions. Low levels of public health funding in sub-Saharan Africa have shifted focus to a handful of cost effective treatments. In some cases a curative strategy has been prioritized over prevention (Ewbank and Gribble, 1993; Sahn and Bernier, 1995).

In terms of our theory, these examples demonstrate that child mortality has fallen in sub-Saharan Africa through a combination of better cures (lower d) and weaker transmission (lower μa), the first of which would have had a weak effect on fertility choice and human capital investment. Starting with $i > i^*$ in Figure 8(b), a decrease in μa will raise i^* while a decrease in d will lower it. The former makes it easier to escape full prevalence, the latter makes it harder. It requires a much stronger decline in μa for the economy to get all the way to Figure 8(c) which happens when it has declined by enough to overturn (A4) and eliminate i^*. There is also a composition effect from lower d: a higher proportion of surviving children are of low health, depressing returns on their human capital investment. Mortality falls but morbidity may rise on the whole: the resulting slower demographic and disease transitions lead to slower economic growth. We proceed to examine the quantitative significance of such a mortality transition.

6 Quantitative Experiments

We calibrate the model and present three experiments. The first experiment simulates England's transitions and compares them to slower counterfactuals relevant for developing countries generally where only the trigger for the mortality transition differs. The second is specifically tailored to account for transitions in sub-Saharan Africa (SSA) and compares them to a faster alternative had morbidity fallen rapidly. Finally we make a general point, that child mortality improvements which have contributed globally to life expectancy improvements have little effect on economic growth unless accompanied by improving morbidity.

6.1 Parameter Values

Table 1 reports benchmark parameter values. Childhood is assumed to last 15 years and each period of adulthood potentially 30 years long. Maximum longevity is then 75 years, commensurate with life expectancy in post-transition UK in the 1950s and 1960s.

A pre-transition economy in our model is at full prevalence. Assuming pre-transition UK (1860 – 69) is close to this, we start at $i_{-1} = i_0 = 1$. In the model parents implicitly choose quality investment in surviving children after the first five years of life. The value for d is picked to

(Soares, 2007).

Preference	Technology	Disease Ecology
$\theta = 0.36$	$\lambda = 2.85$	$\mu = 1.5$
$\beta = (0.99)^{120} = 0.30$	$\varepsilon = 0.12$	$a = 0.87$
$\phi_h = 1$	$\nu = 0.48$	$d = 0.28$
$\phi_u = 0.46$	$A = 10$	$\delta = 0.85$
$\gamma = 1.13$	$\alpha = 1/3$	
$\tau_u = \tau_u = 0.15$		

Table 1: Parameter Values

be 0.28 so that $i_0 d$ matches the child mortality rate of 0.28 (probability of dying between ages 0-5) in the UK between $1860-69$ (mortality.org).[18] With $\phi_h = 1$, we set $\phi_u = 0.46$ to match life expectancy (LE) at age 15 of 43.8 in the UK during the same period (mortality.org). In the absence of information on the differential cost of raising sickly children, child rearing costs (τ_h, τ_u) are both set equal to 0.15 (Haveman and Wolf, 1995) which satisfies (A1). Since we do not have resource cost of raising children this caps the fertility of a household of two parents at a little above 13. For the quantity-quality tradeoff we set θ to 0.36 which, given the value for ν reported in Table 1, satisfies (A2). We set $\gamma = 1.13$ such that pre-transition TFR is 4.9 children per woman, consistent with data for the UK.

For the human capital technology, since externalities can yield growth in human capital even in the absence of quality investment when $\kappa = 1$, we set ε so that there is no growth in human capital (and output) per worker in this scenario. We set $\lambda = 2.85$ so that post-transition economic growth converges to 1% annual growth of GDP per worker, as in the case of the UK (Broadberry and Klein, 2011). Then $\nu = 0.48$ implies that post-transition TFR reaches replacement level. For the aggregate technology, α is set to 1/3 and A normalized to 10.

Since the effects of μ and a are not separately identified in the model, we arbitrarily set $\mu = 1.5$ which requires $a > 0.67$ for $\mu a > 1$. We set $a = 0.87$ which ensures that (A4) is satisfied and Figure 8(b) applies ($p\mu a < 1$). The morbidity parameter δ is calibrated from evidence on labor earnings. Recall that malaria is the main cause of child mortality in sub-Saharan Africa. Cutler *et al.* (2007) estimate that moving from the most to least malarious district in India raised income of male workers by 3-13% though no effect on educational attainment is observed. Bleakley (2010), on the other hand, finds malaria eradication affected both school attainment and earnings in Brazil, Colombia, Mexico and the US. According to his estimates, income between the least and most malarious regions differed by 12-40%, persistent malarial morbidity lowering income by as much as 50%. Arguably early-life exposure to malaria has a greater health effect in

[18] *Human Mortality Database*, University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). Downloaded from www.mortality.org in July, 2011.

Africa than elsewhere. On the other hand, our model does not distinguish between chronically and occasionally infected children and morbidity from other infections like diarrhea could well be smaller. Hence δ is best viewed as the average morbidity effect on all children who have survived various types of childhood infections. We set its value to 0.85. The implied earnings gap of 8% between healthy and unhealthy workers matches the mid-point estimate of Cutler *et al.*[19]

6.2 The Historical Transition

An exogenous one-time change in child mortality causes a dynamic mortality transition in our model. This happens because the incidence of infectious disease and mortality from it are endogenous. Starting with an initial parameter vector $(a_0, d_0) = (0.87, 0.28)$ for an economy in the Malthusian regime, consider two scenarios. In the first one – relevant for developed countries (DC) that underwent demographic transitions in the nineteenth century – the economy experiences an exogenous reduction in child mortality as the prevalence rate falls. Maintaining the value of d, a large decline in a from $a_0 = 0.87$ to $a_1 = 0.37$ pushes i^* above 1 and a transition towards full eradication begins as the economy switches from panel (b) to (c) in Figure 8.

The second scenario broadly replicates those less developed countries (LDC) where child mortality fell in the twentieth century from a combination of lower prevalence and better cures. These interventions could be different types of preventive and curative interventions, or those that are primarily curative but also have an indirect effect in lowering prevalence. We choose a new set of values (a_1, d_1) so that the immediate improvement in child survival is equal to that in the first scenario. We lower a to $a_1 = 0.58$, just enough to eliminate the full prevalence steady state. The residual improvement in child survival is absorbed by the fatality rate falling to $d_1 = 0.18$. The combined effect also starts a transition towards full eradication, but at a slower pace. The two scenarios differ only in how the mortality transition is triggered; the magnitude of the initial mortality shock is identical as are all parameter values besides a_1 and d_1. We start both economies (DC and LDC) at $(x_0, k_0) = (1, 0.17)$, the steady-state values of human and physical capital per worker in the pre-transition economy. Figure 9 illustrates the demographic, epidemiological and economic responses.

The English transition began around 1872 and by 1950 child survival had improved to 96.5%, LE at age 15 to 57 years, and the TFR had fallen to 2.1. The simulated DC transition matches these trends closely. Within three generations (90 years), child survival is 96.7%, LE at age 15 is 56.6 years and TFR 2.1 per woman, the latter specifically targeted in our calibration. In contrast, the LDC transition takes almost three times as long. Due to higher childhood morbidity in the first few generations, the TFR is generally higher which causes a population explosion. During

[19] In the pre-transition economy where investment in human capital is nil, childhood infections matter only for late-life mortality and δ is irrelevant.

the first forty years of the UK and SSA transitions, CMR fell by similar magnitudes (about 46%) but TFR responded more strongly in the UK, an elasticity of 0.82 compared to 0.46 for SSA.[20] By construction child mortality initially falls by the same magnitude in the two cases reported in Figure 9. The TFR response is stronger in the DC transition: its elasticity with respect to CMR in the first period is 0.64 relative to 0.34 for the LDC transition. While this under-predicts the fertility transition – presumably other factors also facilitated the transition – the relative speed of the two regions is comparable to the data. Due to the slower transition, worker productivity and GDP per worker are permanently lower in the LDC scenario.

Noninfectious disease mortality among the elderly fluctuated around 40% in the UK prior to the transition, began to decline in 1900, about one generation after infectious disease mortality started to fall, and dropped to 9% by 1980 (Arora, 2005). Our simulations slightly over-predict late-life mortality for the DC transition. The noninfectious mortality rate begins at 54% of the elderly and, after lagging the infectious disease mortality decline as in the data, falls to 11% in four generations or 120 years. In the LDC scenario, in contrast, noninfectious mortality falls only to 38% four generations into the transition, about four times higher than the DC scenario similar to the facts outlined in section 2.2.

The nature of the LDC mortality transition extracts a cost relative to the DC transition. Since the prevalence rate initially falls by less, childhood morbidity and late-life mortality remain high several generations into the transition. This mutes the fertility response through the quantity-quality and adult longevity margins.

6.3 The Transition in Sub-Saharan Africa

Beginning in the 1960s, child mortality improved in SSA from the availability of antibiotics, the use of DDT in malaria eradication efforts and the establishment of the WHO which facilitated technology transfer and public health campaigns. Later in the 1980s child vaccination further lowered mortality. The LDC experiment above was inspired by this combination of prevalence- and therapy-based efforts.

On two margins, however, SSA significantly differs from the UK: its pre-transition (1950-60) CMR was actually *lower* than the UK's (1850-70) while its pre-transition TFR was significantly *higher*. It is plausible that even if SSA had benefited from a prevalence-only mortality reduction, its transition to replacement fertility would have been slower. To identify how much slower SSA's experience has been relative to such a prevalence-only scenario, we recalibrate the model to match SSA's pre-transition levels of CMR and TFR and its observed fertility transition since

[20] This elasticity gap is larger than that for other developing regions. Outside SSA, the elasticity ranges from 0.69 for MENA to 0.81 for East Asia. Child mortality fell, however, by a larger magnitude in these regions relative to SSA or the UK during comparable periods.

the 1960s.

Compared to the UK benchmark, the parameter γ is adjusted to 2.69 to match the higher pre-transition TFR in SSA which fluctuated around 6.6-6.7 during 1950-1960. We then set θ = 0.25 to maintain post-transition fertility at replacement level. To satisfy (A2) we also lower ν to 0.30. We lower the value of δ to 0.75 (earnings gap of 8% as before) in order to match the slower rate of transition in SSA during 1960 - 2010 relative to the UK. In fact, SSA's TFR barely moved during the first thirty years of its CMR decline. The new values of ν and θ ensure that quality investment is nil and fertility relatively high during the first generation of the transition. The value for d is picked to be 0.25 so that $i_0 d$ matches the mortality rate for children under age 5 in SSA in 1960 (World Bank). To reflect lower pre-transition LE at age 15 in SSA of 40.2, we set ϕ_u = 0.34. As in the UK experiment, there is no pre-transition growth in human capital (and output) per worker, and post-transition economic growth converges to 1% annual growth of GDP per worker. The remaining parameter values are as in Table 1.

We start with the initial configuration $(a_0, d_0) = (0.87, 0.25)$ for the pre-transition economy. To simulate the SSA transition, child mortality falls due to a combination of exogenous reduction in prevalence and case fatalities. Specifically, a falls from 0.87 to 0.52 so that the economy can start transitioning towards zero prevalence while d falls from 0.25 to 0.2. Together these changes replicate the two-period $(1950/60 - 2010)$ changes in CMR and TFR observed for SSA: two generations after the mortality shock, TFR falls to just under 5 children per woman (Figure 10), similar to what we observe in the data (Figure 2).

The alternative scenario in Figure 10 shows what would have happened had the initial mortality decline been solely from lower prevalence. Holding the case fatality rate constant at 0.25, a declines from 0.87 to 0.41 to match the initial drop in CMR for SSA. Not surprisingly the demographic transition would have progressed further by now with fertility significantly lower at 3.2, lagging MENA and South Asia by about a decade. At its current pace, SSA will reach that level by 2040.

Life expectancy at birth rose in SSA from 41 to 54 years during 1960-2010 (World Development Indicators), some of the progress undone by the HIV crisis since the late 1980s. In comparison, life expectancy at age 15 changed little in the decades leading up to the HIV crisis, from 40.2 years in 1975 to 41.2 years in 1980 to 40.6 years in 1990, falling to 38.3 years by 2000 due to the AIDS epidemic. The third panel of Figure 10 shows how life expectancy might have improved with child survival in the absence of the AIDS epidemic, where ϕ_u has been chosen to match LE at age 15 in SSA in 1975.[21]

[21] While the transition began earlier in SSA, data availability restricts us to 1975. If life expectancy at 15 responds to CMR with a lag, then it would have been similar between 1950 and 1975.

6.4 The Role of Morbidity

Several effects are at work in these experiments: how childhood morbidity affects human capital, how it affects adult mortality, and the dynamic path child mortality follows from a reduction in the prevalence rate.

To clearly identify the role of morbidity, we construct "impulse responses". Starting from full disease prevalence and 28% case fatalities (CMR of 28% as in the UK experiment), we hold the prevalence rate at a level where an exogenous reduction in i does not tip the economy towards zero prevalence. This switches off the dynamic response due to changing prevalence rates. We then compare the growth effects of a reduction in child mortality due to an exogenous reduction in case fatalities from 28% to 18% versus the same decrease when the prevalence rate falls to 64%. In the two cases, the initial increase in life expectancy at birth is identical.

Figure 11 plots the annualized growth rate of GDP per capita for the i-decline and d-decline scenarios when $\delta = 0.85$ (UK experiment) and $\delta = 1$ (no scarring effect). To maintain positive human capital investment in each of the following experiments, θ is lowered to 0.35. Parameter values are otherwise identical to the UK experiment. The initial increase in life expectancy at birth is 1.5 years, from 54.6 to 56.1. In Figure 11 economic growth is permanently faster in the i-decline scenario when $\delta < 1$ (left panel), because the NFR declines and human capital investment in each child increases, while in the d-decline scenario these remain unchanged. For the $\delta = 1$ case, the difference in growth rates between the i-decline and d-decline scenarios is temporary and results from old-age mortality differences. Since $\phi_u < \phi_h = 1$, children who suffer from infections grow up predisposed towards non-infectious disease in old age. These individuals save less of their income and have more children, thereby slowing human and physical capital accumulation.

To isolate the effect of childhood morbidity we next set $\phi_u = \phi_h = 1$. Childhood infections now affect fertility through the quantity-quality margin alone. Initially life expectancy at birth rises by 1.5 years, from 70.8 to 72.3. As in the previous case, a permanent growth gap opens up between the two cases when $\delta < 1$ (Figure 11, right panel). If $\delta = 1$, all children whether infected or not grow up to be healthy, so fertility, quality investment and saving decisions are identical in the two economies. The long-run growth effects are identical in the two panels because only childhood morbidity affects human capital accumulation, the engine of growth in this model. Of course, as we have shown earlier, if both economies were to converge to zero prevalence in the long run, eventually growth rates will converge but income levels will not.[22] In fact for $\delta < 1$ the resulting income gap would be even greater because morbidity persists in the d-decline scenario.

[22] In Figure 11, a lagged response occurs because lower morbidity translates into higher physical and human capital among the following generation of working-age adults.

When $i = 1$, all surviving children are unhealthy and a decline in d does not change this. When $i < 1$, some surviving children are healthy while others are unhealthy, so a decline in d raises the proportion of unhealthy children relative to healthy ones. When $i = 1$ human capital investment per child and the NFR are unaffected by changes in d, but if initial $i < 1$, then a reduction in the case fatality rate generates an *increase* in the NFR and a *decrease* in human capital investment as long as $\delta < 1$. We illustrate this scenario by starting with $i = 0.9$ and $d = 0.31$ so that initial CMR = 28%, the same as before. We then compare the growth effects when case fatalities decline from 31% to 20% versus the same decrease in child mortality from the prevalence falling from 90% to 58%. CMR falls from 28% to 18% in all of the scenarios in Figure 11, but economic growth decreases initially only in the d-decline scenario when initial $i < 1$. For $\delta = 1$ growth dips initially with the d-decline only if $\phi_u < \phi_h$ due to the longevity effect. For $\delta < 1$ the longevity effect contributes to the dip in growth when $\phi_u < \phi_h$, while human capital investment declines with a fall in d regardless of the values for ϕ_u and ϕ_h.

We conclude that when childhood infections have a scarring effect, a reduction in child mortality (or increase in life expectancy at birth) does not increase economic growth unless childhood morbidity also falls, as in the i-decline scenario. Indeed, the d-decline scenario shows that growth may temporarily fall when reductions in child mortality dilute average child quality and increase fertility.

7 Conclusion

This paper shows that the morbidity effect of infectious disease is an important element of demographic and epidemiological changes in the long run. We constructed a dynastic model of endogenous mortality and fertility where childhood infections depreciate child quality and predispose people towards non-infectious disease in late life. A calibrated version of the model is able to explain why sub-Saharan Africa's mortality transition has not generated the kind of fertility, epidemiological and economic responses that we have seen historically or in other developing regions.

Mortality, particularly among children, has been widely used as a parsimonious metric of economic development. A notable implication of our analysis is that child mortality (or life expectancy at birth) is a weak proxy of a population's underlying health since mortality and morbidity do not always move in the same direction, a point made earlier by Murray and Chen (1992). Higher life expectancy at birth facilitates a fertility transition and economic development only when accompanied by lower childhood morbidity and adult mortality.

In a promising development, some African countries have recently reduced infant and child mortality through prevalence efforts. Kenya's infant mortality rate fell from 8.1 to 6.0% between

2003 and 2008-09 driven wholly by reductions in postneonatal mortality. This was made possible by new public health efforts targeting the transmission of infectious diseases, for instance increased usage of insecticide-sprayed bednets and better access to clean water and sanitation (Demombynes and Trommlerova, 2012). In our view, replicating this experience across the continent would facilitate sub-Saharan Africa's demographic and epidemiological transitions and contribute to its economic development.

Bibliography

1. Acemoglu, D. and S. Johnson (2007), "Disease and Development: The Effect of Life Expectancy on Economic Growth", *Journal of Political Economy*, 115 (6), 925-985.
2. Akachi, Y. and D. Canning (2010), "Health trends in Sub-Saharan Africa: Conflicting evidence from infant mortality rates and adult heights", *Economics & Human Biology*, 8: 273-288.
3. Aksan, A. and S. Chakraborty (2013), "Childhood Disease Burden and the Precautionary Demand for Children", *Journal of Population Economics*, 26 (3): 855-885.
4. Almond D. (2006), "Is the 1918 Influenza pandemic over? Long-term effects of in utero influenza exposure in the post-1940 US population", *Journal of Political Economy*, 114: 672-712.
5. Angeles, L. (2010), "Demographic Transitions: Analyzing the Effects of Mortality on Fertility", *Journal of Population Economics*, 23: 99-120.
6. Arora, S. (2005), "On epidemiologicalal and economic transitions: a historical view", in *Health and Economic Growth: Findings and Policy Implications*, edited by G. Lopez Casasnovas, B. Rivera and L. Currais, MIT Press, Cambridge, MA.
7. Bar, Michael and Oksana Leukhina (2010), "Demographic transition and industrial revolution: A macroeconomic investigation", *Review of Economic Dynamics,* 13(2), 424-451.
8. Barker, D.J.P. (1994), *Mothers, babies, and disease in later life*. London: British Medical Journal Publishing Group.
9. Bhattacharya, J. and S. Chakraborty (2014), "Contraception and the Fertility Transition", MPRA Paper No. 53129.
10. Birchenall, J. (2007), "Escaping High Mortality", *Journal of Economic Growth*, 12: 351-387.
11. Bleakley, H. (2007), "Disease and development: Evidence from hookworm eradication in the American South", *Quarterly Journal of Economics*, 122 (1): 73-117.

12. Bleakley, H. (2010), "Malaria Eradication in the Americas: A Retrospective Analysis of Childhood Exposure", *American Economic Journal: Applied Economics*, Vol. 2, pp. 1-45.
13. Bleakley, H. and F. Lange (2009), "Chronic disease burden and the interaction of education, fertility and growth", *Review of Economics and Statistics*, Vol. 91, pp. 52-65.
14. Boldrin, Michele and Larry E. Jones (2002), "Mortality, fertility, and saving in a Malthusian economy," *Review of Economic Dynamics*, 5(4): 775-814.
15. Boschi-Pinto, C., Lanata, C.F., Mendoza, W., and Habte, D. (2006), "Diarrheal Diseases", in *Disease and Mortality in sub-Saharan Africa*, edited by D.T. Jamison, R.G. Feachem, M.W. Makgoba, E.R. Bos, F.K. Baingana, K.J. Hofman, and K.O. Rogo, World Bank, Washington D.C.
16. Bozzoli, C., A. Deaton, C. Quintana-Domeque (2009), "Adult Height and Childhood Disease", *Demography*, 46 (4): 647-669.
17. Broadberry, S. and A. Klein (2011), "Aggregate and per capita GDP in Europe, 1870-2000: Continental, Regional and National Data with Changing Boundaries", *working paper*, University of Warwick.
18. Case, A. and C. Paxson (2010), "Causes and Consequences of Early-Life Health", *Demography*, 47 (Supplement): S65.
19. Cervellati, M. and Sunde U. (2011), "Life Expectancy and Economic Growth: The Role of the Demographic Transition," *Journal of Economic Growth*, 16, 99-133.
20. Chakraborty, S., C. Papageorgiou and F. Pérez-Sebastián (2010), "Diseases, Infection Dynamics and Development", *Journal of Monetary Economics*, 57 (7): 859-872.
21. Chang, S., Fleisher, B., Kim, S. and S. Liu (2011), "Long-term Effects of Early Childhood Malaria Exposure on Education and Health: Evidence from Colonial Taiwan", IZA Discussion Paper No. 5526.
22. Checkley, W. *et al.* (2008), "Multi-country analysis of the effects of diarrhoea on childhood stunting", *International Journal of Epidemiology*, 37 (4): 816-830.
23. Coelho, P. and R. McGuire (2000), "Diets Versus Diseases: The Anthropometrics of Slave Children", *The Journal of Economic History*, 60: 232-246.
24. Costa, D.L. (2003), "Understanding mid-life and older age mortality declines: evidence from Union Army veterans", *Journal of Econometrics*, 112 (1): 175-192.
25. Crimmins, E.M. and C.E. Finch (2006), "Infection, Inflammation, Height, and Longevity", *Proceedings of the National Academy of Sciences*, 103: 498-503.
26. Cutler, D., Deaton, A. and A. Lleras-Muney (2006), "The Determinants of Mortality", *Journal of Economic Perspectives*, 20, 97-120.

27. Cutler, D., Fung, W., Kremer, M., Singhal, M. and T. Vogl (2007), "Mosquitoes: The Long-term Effects of Malaria Eradication in India", NBER Working Paper No. 13539.

28. de la Croix, D. and O. Licandro (2013), "The Child is Father of the Man: Implications for the Demographic Transition", *Economic Journal*, 123: 236-261.

29. Demombynes, G. and S. F. Trommlerova (2012), "What has driven the decline of infant mortality in Kenya?", *World Bank Policy Research Working Paper*, No. 6057.

30. Doepke, M. (2005), "Child Mortality and Fertility Decline: Does the Barro-Becker Model Fit the Facts?" *Journal of Population Economics*, 18: 337-366.

31. Ewbank, D. C. and J. N. Gribble (eds.) (1993), *Effects of Health Programs on Child Mortality in sub-Saharan Africa*, National Academic Press, Washington D.C.

32. Fogel, R. (1997), "New Findings on Secular Trends in Nutrition and Mortality: Some implications for population theory", in M. Rosenzwig and O. Stark (eds.) *Handbook of Population and Family Economics*, vol. 1A, Elsevier Science, The Netherlands.

33. Galor, O. (2005), "From Stagnation to Growth: Unified Growth Theory", in *Handbook of Economic Growth*, Vol. 1A, P. Aghion and S. N. Durlauf (eds). North Holland: Elsevier, 171-293.

34. Guerrant, R.L., B. Carneiro-Filho, and Dillingham, R.A. (2003), "Cholera, Diarrhea, and Oral Rehydration Therapy: Triumph and Indictment", *Clinical Infectious Diseases*, 37: 398-405.

35. Guinnane, T. W. (2011), "The historical fertility transition: A guide for economists", *Journal of Economic Literature*, 49(3), 589-614.

36. Hansen, C.W. (2014), "Cause of death and development in the US", *Journal of Development Economics*, forthcoming.

37. Haveman, R. and B. Wolfe (1995), "The Determinants of Children's Attainments: A Review of Methods and Findings," *Journal of Economic Literature*, vol. 33(4): 1829-1878.

38. Herzer, D., Strulik, H., and Vollmer, S. (2012), "The long-run determinants of fertility: one century of demographic change 1900–1999", *Journal of Economic Growth*, 17(4), 357-385.

39. Hinde, A. (2003), *England's Population: A History since the Domesday Survey*, Hodder Arnold, London.

40. Hong, S. C. (2007), "The Burden of Early Exposure to Malaria in the United States, 1850-1860: Malnutrition and Immune Disorders", *Journal of Economic History*, 67: 1001-1035.

41. John, A. M. (1990), "Transmission and Control of Childhood Infectious Diseases: Does Demography Matter?" *Population Studies*, 44: 195-215.

42. Jousilahti, P., J. Tuomilehto, E. Vartiainen, J. Eriksson, and P. 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): 1112-1120.

43. Kalemli-Ozcan, S. (2002), "Does the Mortality Decline Promote Economic Growth?", *Journal of Economic Growth*, 7(4), 411-439.

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

45. Lagerlöf, Nils-Petter (2003), "From Malthus to Modern Growth: Can Epidemics Explain the Three Regimes?", *International Economic Review*, 44: 755-777.

46. Lorentzen, P., McMillan, J., Wacziarg, R. (2008), "Death and development", *Journal of Economic Growth*, 13(2), 81-124.

47. Leeder, S., S. Raymond and H. Greenberg (2003), "A Race Against Time: The Challenge of Cardiovascular Disease in Developing Economies", *Center for Global Health and Economic Development*, Columbia University and University of Sydney.

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

49. Mazumder, B., D. Almond, K. Parka, E. M. Crimmins and C. E. Finch (2010), "Lingering prenatal effects of the 1918 influenza pandemic on cardiovascular disease", *Journal of Developmental Origins of Health and Disease*, 1 (1): 26-34.

50. McKeown, Thomas (1976), *The Modern Rise of Population*, Academic Press, New York.

51. Moradi, A. (2006), "The nutritional status of women in Sub-Saharan Africa, 1950-1980", *mimeo*, Department of Economics, University of Oxford.

52. Morand, O. (2004), "Economic growth, longevity and the epidemiological transition", *European Journal of Health Economics*, 5 (2): 1-9.

53. Murray, C. J. L. and L. C. Chen (1992), "Understanding Morbidity Change", *Population and Development Review*, 18 (3): 481-503.

54. Omran, A. R. (1971), "The Epidemiological Transition: A Theory of the Epidemiology of Population Change", *The Milbank Memorial Fund Quarterly*, 49 (4): 509-38.

55. Preston, S. H. (1996), "Population Studies of Mortality", *Population Studies*, 50, 525-536.

56. Rajaratnam, J., Marcus, J., Levin-Rector, A., Chalupka, A., Wang, H., Dwyer, L., Costa, M., Lopez, A. D., and C. J. L. Murray (2010), "Worldwide mortality in men and women aged 15-59 years from 1970 to 2010: a systematic analysis", *The Lancet*, 375 (9727): 1704-1720.

57. Sahn, D. and R. Bernier (1995), "Have structural adjustments led to health sector reform in Africa?", *Health Policy*, 32: 193-214.

58. 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.

59. Soares, R. (2005), "Mortality Reductions, Educational Attainment, and Fertility Choice", *American Economic Review*, 95(3), 580-601.

60. Soares, R. (2007), "On the Determinants of Mortality Reductions in the Developing World", *Population and Development Review*, 33 (2): 247-287.

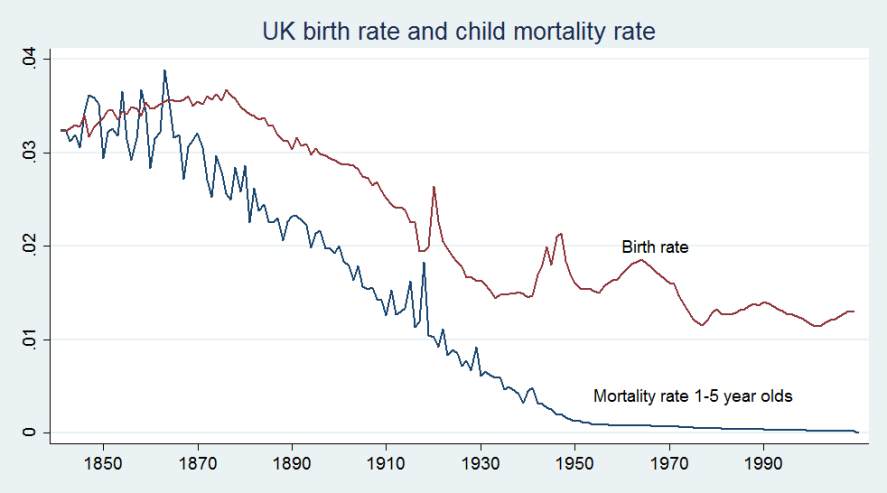
61. Snow, R.W., Craig, M.H., Newton, C.R.J.C., and Steketee, R.W. (2003), "The public health burden of *Plasmodium falciparum* malaria, in Africa: Deriving the numbers", Washington DC: The Disease Control Priorities Project (DCPP) Working Paper Number 11, vol 75.

62. Strulik, Holger and Jacob Weisdorf (2014), "How Child Costs and Survival Shaped the Industrial Revolution and the Demographic Transition", *Macroeconomic Dynamics*, 18 (1): 114-144.

63. Szreter, S. (1988), "The importance of social intervention in Britain's mortality decline c.1850-1914: a reinterpretation of the role of Public Health", *Social History of Medicine*, 1: 1-38.

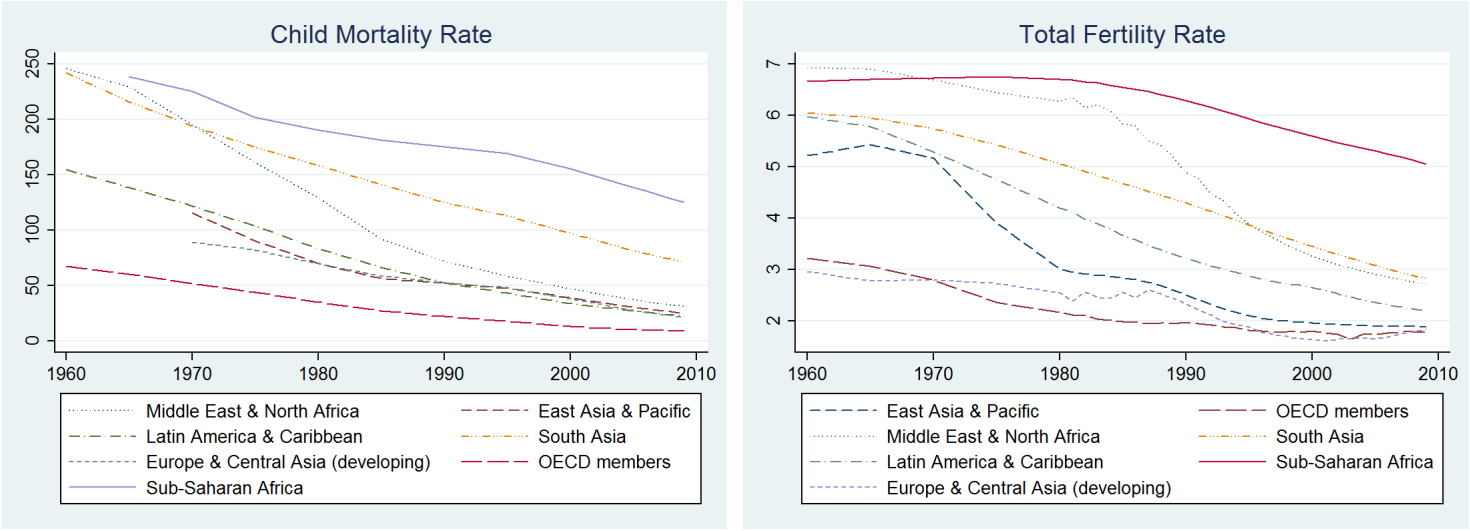
64. Voth, H. and T. Leunig (1996), "Did Smallpox reduce Height? Stature and the Standard of Living in London, 1770-1873", *Economic History Review*, XLIX (3): 541-560.

65. Woods, R. E. 2000, *The Demography of Victorian England and Wales*, Cambridge University Press.



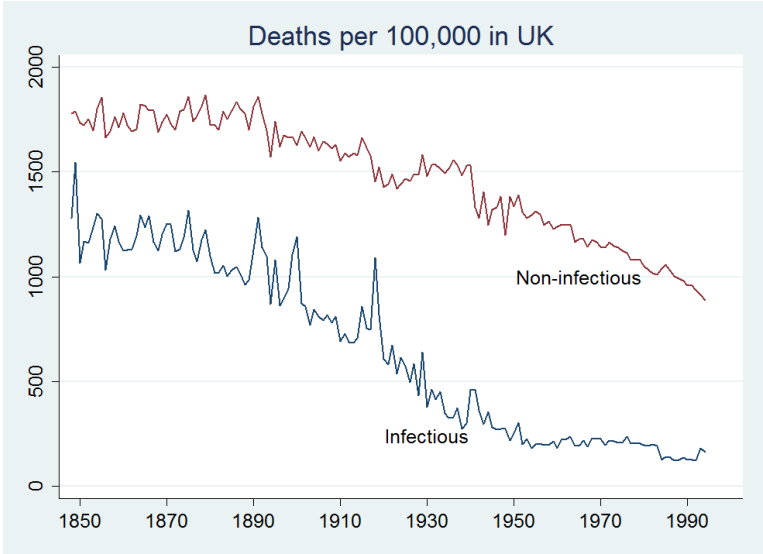
Source: mortality.org

Figure 1 Demographic Transition in England & Wales



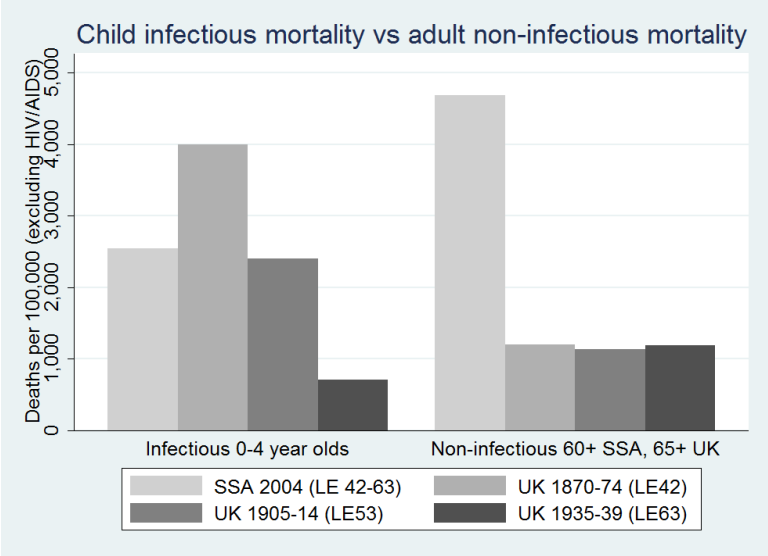
Source: World Development Indicators

Figure 2 Demographic Transitions since 1960



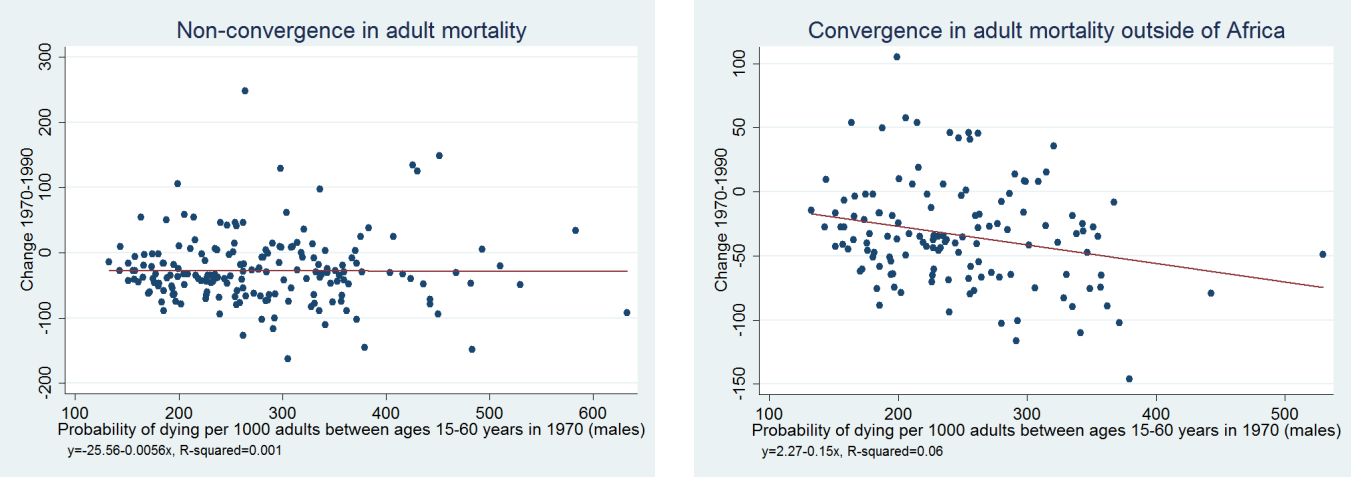
Source: Arora (2005)

Figure 3 Epidemiological Transition in England & Wales 1848-1994



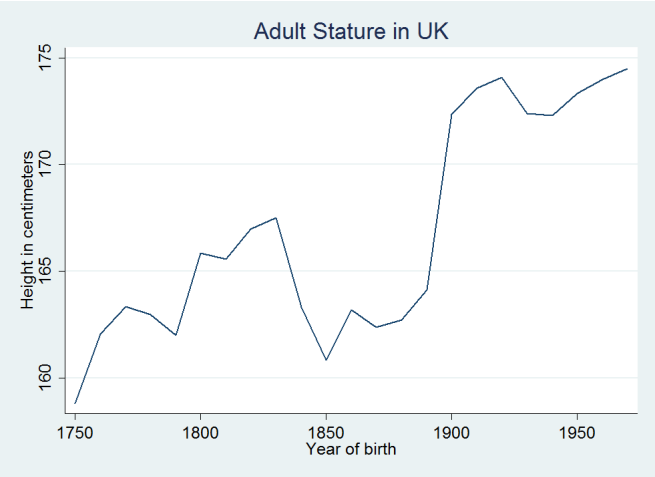
Source: Global Burden of Disease (2004), Arora (2005)

Figure 4 Disease Patterns in sub-Saharan Africa and the UK



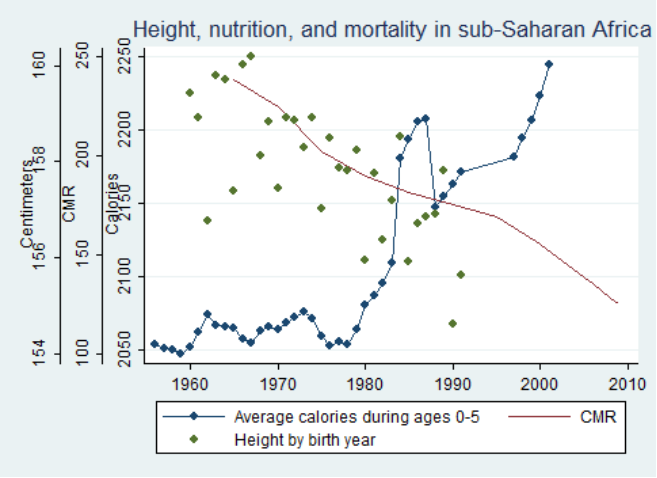
Source: World Development Indicators, Rajaratnam *et al*. (2010)

Figure 5 Convergence Patterns in Life Expectancy at Birth and Adult Mortality across Countries



Source: Arora (2005)

Figure 6 Stature of 18-year Old Males in the UK, 1800-1950



Source: FAO, DHS, World Development Indicators

Figure 7 Nutrition, Average Height and Child Mortality

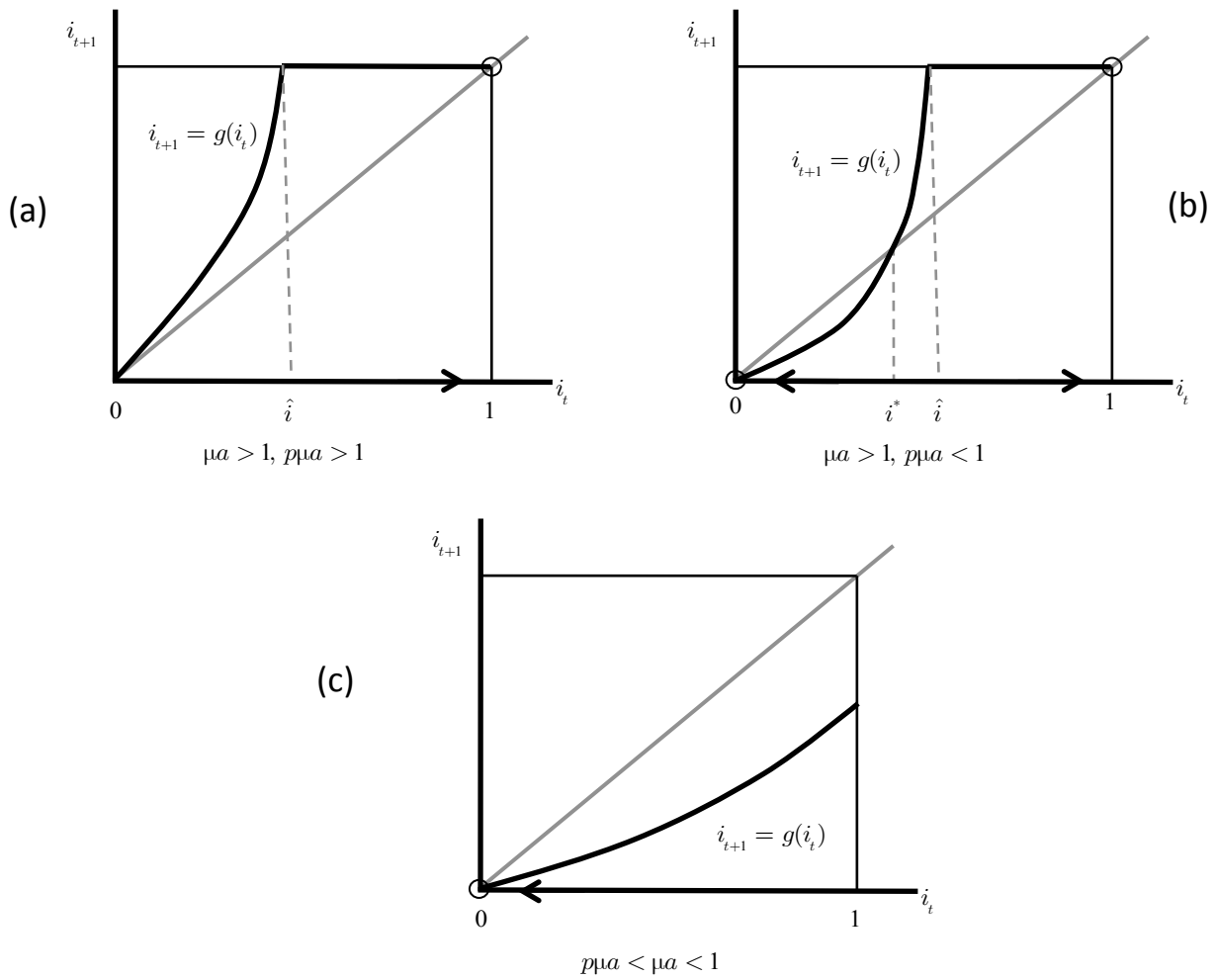


Figure 8 Dynamics of the Prevalence Rate

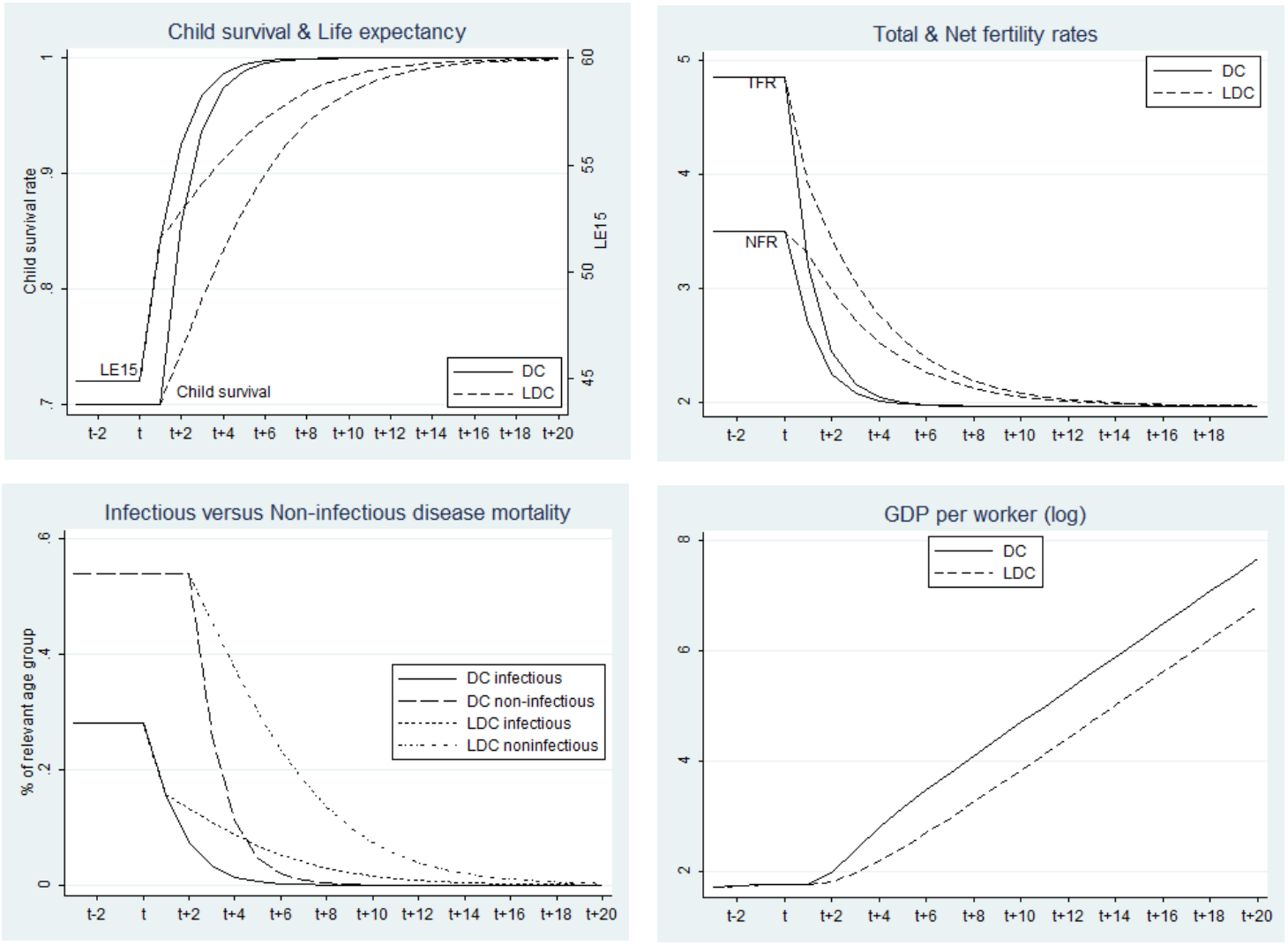


Figure 9 Simulated Demographic, Epidemiological and Economic Transitions

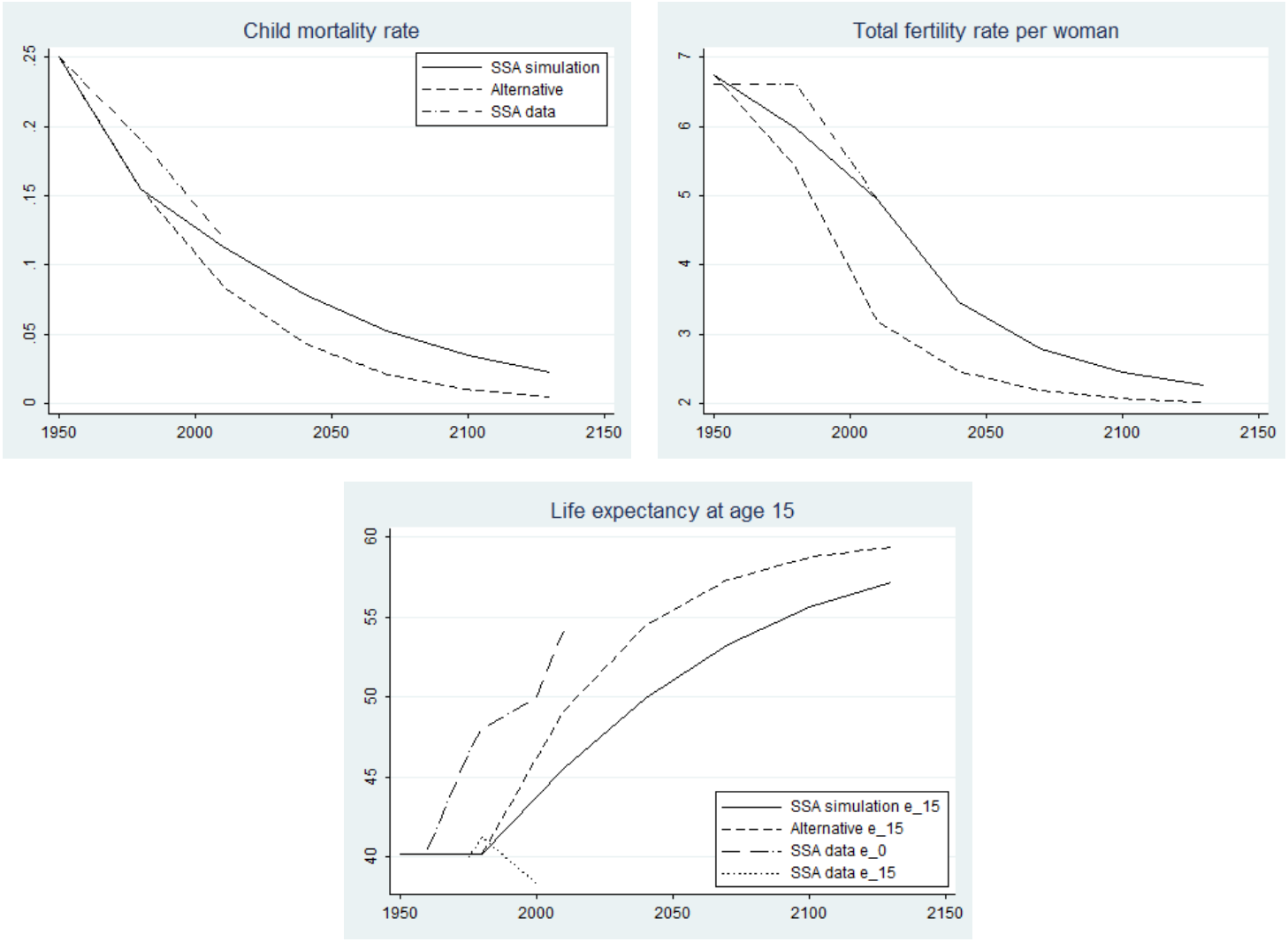


Figure 10 Simulated Demographic and Epidemiological Transitions in sub-Saharan Africa

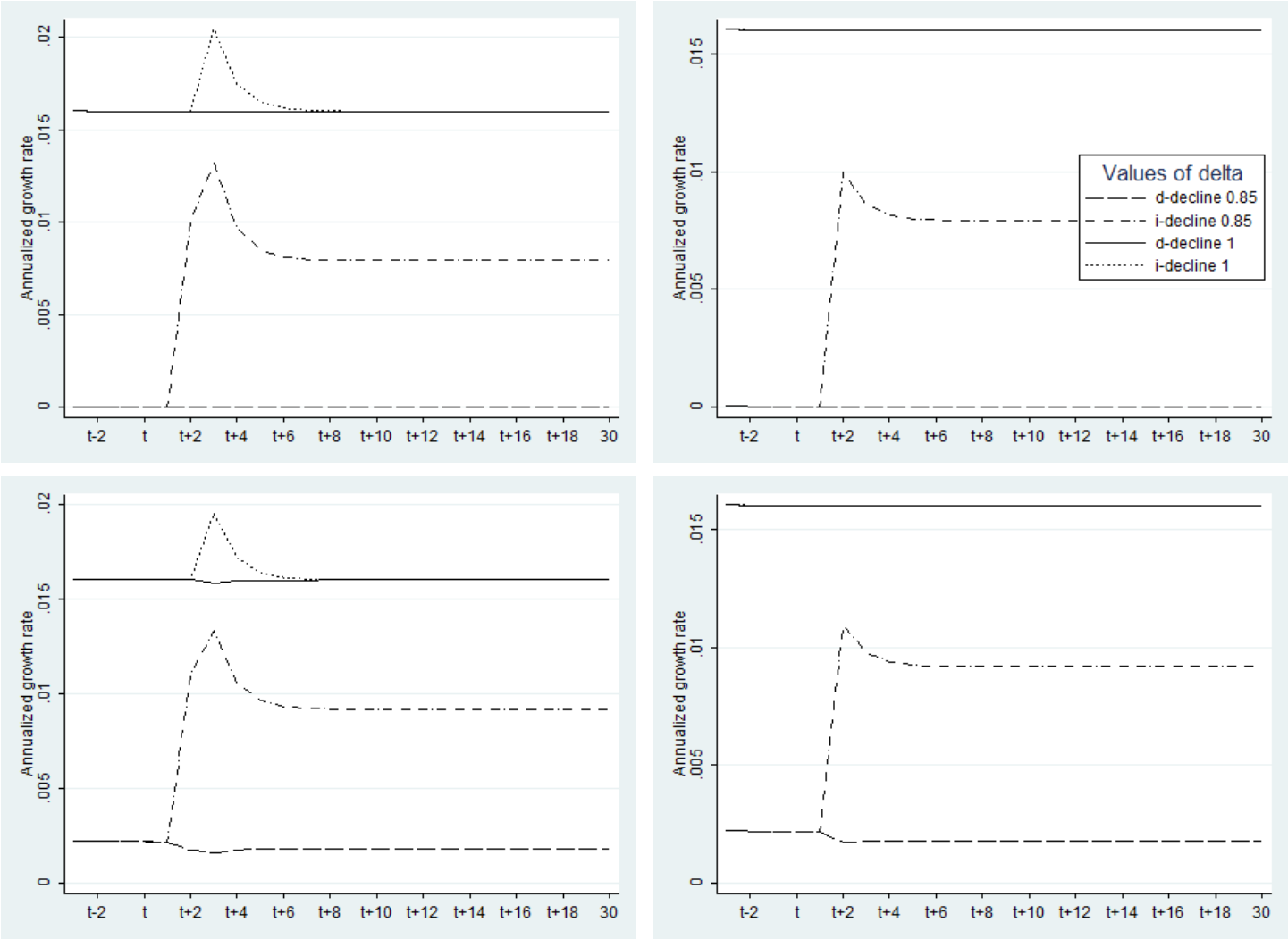


Figure 11 Growth effects of *i* versus *d* decline when CMR falls from 28% to 18%

Upper two panels correspond to initial prevalence rate of 100%, lower panels 90%.
In each case, the left panel uses $\phi_u = 0.46$, $\phi_h = 1$, the right panel $\phi_u = \phi_h = 1$