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Twin Transitions

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Abstract

We provide a new explanation for sub-Saharan Africa's slow demographic and economic change. In a model where children die from infectious disease, childhood health affects human capital and noninfectious-disease-related adult mortality. When child mortality falls from lower prevalence, as in western Europe, labor productivity improves, fertility falls and the economy prospers. When it falls mainly from better cures, as in sub-Saharan Africa, survivors are less healthy and there is little economic payoff. The model quantitatively explains sub-Saharan Africa's experience. More generally it shows that life expectancy at birth is a poor indicator of population health unless morbidity falls with mortality.

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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 many 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 would suggest and that is why its fertility transition has been slower and yielded little economic benefit. Our explanation draws on the link between the demographic and epidemiological transitions. During the latter, infectious disease mortality among the young and elderly falls, followed by non-infectious disease mortality among older adults. Falling infectious disease mortality is typically associated with falling morbidity as younger adults become healthier and fewer older adults suffer from non-infectious disease. This link between mortality and morbidity has weakened for sub-Saharan Africa (SSA).

In this paper, we develop a general equilibrium dynastic model that qualitatively and quantitatively accounts for SSA'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. For example, child survival gains in late nineteenth century England and Wales were followed by secular gains in adult stature in successive cohorts. African countries, on the other hand, have reduced child mortality through better treatments like antibiotics. While vaccination has eradicated diseases like smallpox and polio, those have been the

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[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 12.

exceptions. This means decades of falling child mortality have not substantially lowered the prevalence of many major infectious diseases in SSA. Unlike other developing regions, adult stature and non-infectious disease mortality have improved little even as nutrition steadily improved.

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. 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, having suffered through repeated infections more surviving children are of worse quality. Parents have little incentive to invest in their human capital and continue to choose relatively 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. Here the mortality transition does not translate into a fertility or an epidemiological transition. Economic growth may even worsen if the morbidity effect is particularly severe. A calibrated version of the model mimics the mortality transition in SSA versus the historical experience of England & Wales. We show that the morbidity channel can plausibly explain the recent African experience and that child mortality alone can be a poor predictor of national health and development.

Our paper builds on the economic demography literature. Using CRRA preferences and ignoring child survival uncertainty, Doepke (2005) points to a puzzle: improving child survival lowers the total fertility rate but leaves net fertility unaffected, unlike historical demographic transitions. Even with these two assumptions net fertility responds to child mortality in our model because child quality is heterogeneous and healthy and unhealthy children are imperfect substitutes for parents.

Four papers in the health and demography literature are particularly relevant to this work.[2] Strulik (2008) attributes stalled demographic transitions in the tropics to diseases. In his model the shadow cost of a child is higher in low-disease environments

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[2]The epidemiological transition receives no attention in this 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.

similar to us, but the mechanism is different: parental investment in nutrition is assumed to be less effective at improving child survival in low-disease environments. In Birchenall (2007), the decline of infectious disease drives the transition to modern economic growth. He too distinguishes between infection and case fatality rates but the transition occurs through parental investment in nutrition. This paper is closely related to, and complements, de la Croix and Licandro (2012) who propose that rising life expectancy and sustained physical development made possible western Europe's fertility transition. Besides providing a micro-foundation for this mechanism, we emphasize the scarring effect of childhood morbidity. This furthers our understanding of how Europe's epidemiological transition was vital to its demographic and economic transitions and especially why similar dividends have not followed from Africa's rising life expectancy before the HIV crisis. By linking the demographic and epidemiological transitions, we also show how the same weak response explains Africa's higher incidence of adult mortality and morbidity.

Our earlier work, Aksan and Chakraborty (2013), is the first to connect the nature of sub-Saharan Africa's mortality transition to its slow fertility transition. The scope of that paper is more limited. It develops a partial equilibrium model of fertility choice under uncertain child survival and tests for its predictions using data on malaria, HIV, diarrheal and lower respiratory infections. While we ignore uncertainty in this paper, we capture a broader canvas. By explicitly formalizing the general equilibrium effects of Africa's mortality transition on fertility, epidemiology, adult mortality, morbidity and human capital, we are able to quantitatively assess how well transition paths explain Africa's population and economic changes.

Child mortality, life expectancy at birth and adult mortality have been variously linked to fertility behavior, human capital investment and growth in the literature. 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. Our work cautions against the use of mortality alone to gauge the relationship between population health and economic development.

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 Section 6 highlight the cost of childhood morbidity and the ability of the theory to explain SSA's experience.

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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 about five years later by declines in the total and net fertility rates.[3] While this pattern may not fit all transitions in the nineteenth century (Galor, 2005), it does in the twentieth century: falling child mortality has been instrumental in worldwide fertility reductions during $1955-2005$ (Angeles, 2010).

Despite large declines in child mortality sub-Saharan Africa's (SSA) fertility has, however, 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.

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[3]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 ourselves to child mortality rates in both the UK and SSA.

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-infectious disease mortality followed after about twenty years (Arora, 2005).[4]

Here too the experience of SSA differs fundamentally. 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. Figure 5 shows 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

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[4]Death rates are age-standardized. The epidemiological transition is usually stated in terms of increasing incidence of noninfectious disease deaths as 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, more importantly, non-infectious disease mortality *rates* among the middle-aged and elderly fell.

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).[5]

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 calorie intake (FAO) in Figure 7. Except for the late eighties, nutrition generally improved since 1960.[6]

Calorie supply data may, of course, 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 at best 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 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 not available for all countries, but it is available for over 30 countries, including 8 of the 10 most populous countries of sub-Saharan Africa. Exact country composition of height data for a particular year changes, but the countries that have height data have it for several decades. Akachi and Canning's (2010) more systematic

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[5]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, 2012).

[6]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.

analysis of the data affirm 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 in earlier 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 in developing countries and birthweight to higher adult schooling attainment, height and earnings. More recently, Case and Paxson (2010) 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 average educational attainment 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.

Frequent infections prevent the absorption of nutrients necessary for optimal cellular growth, affecting children's physical and cognitive development (Martorell and Habicht, 1986). The literature on the effect of specific infections on children is vast. We highlight a few papers that inform our modeling assumptions. Anemia is the most common type of malarial morbidity, 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).

After malaria, diarrheal infections are the next highest cause of child mortality in Africa. Many children survive them but infections are frequent, averaging 9 median

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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 also increase susceptibility to non-infectious disease later in life. Measles, typhoid and malaria during childhood are known to be associated with cardio-vascular 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. This link between infectious and non-infectious disease is evident in Figure 3: non-infectious disease mortality followed infectious disease mortality by about a generation during the English epidemiological transition.

3   The Model

Our theory accounts for these facts 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). We argue that better therapy played a vital role in SSA's child mortality declines while historical declines, including England & Wales',

occurred due to the conquest of infectious diseases.[7]

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.

We assume all children are homogeneous in health at birth. Of the n_t children born to each 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 $(1 - i_t)n_t + pi_t n_t = (1 - di_t)n_t$.

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 could be different from that of a healthy child. For example, if an unhealthy child needs more care due to frequent bouts of infection and related health problems, it is reasonable to assume $\tau_u > \tau_h$.

Parents base their decisions on the expected number of survivors of each type. A

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[7]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 indicates that morbidity fell sufficiently with mortality in these regions unlike SSA (details available upon request).

young parent with human capital x_t maximizes expected lifetime utility[8]

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

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

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

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

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

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

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 denotes the propensity to save out of "full income" $z \equiv w x$. The second budget constraint incorporates the assumption of perfect annuities.

The last term in the utility function is a joy-of-giving bequest motive; $(1 - i)/(1 - d i)$ is the fraction of surviving children who are healthy, $p i/(1 - d i)$ the fraction who are unhealthy.[9] Embedded in it is an important assumption: healthy and unhealthy children are imperfect substitutes. This assumption is central to generating the appropriate fertility and investment responses when child mortality falls.

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, 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 de-

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[8]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 in their working life is a simplification.

[9]For $i = 0$ this term simplifies to $\ln x^h$, for $i = 1$ to $\ln x^u$. Hence (1) nests the commonly used quantity-quality specification for homogeneous child quality, $\gamma\left[\theta \ln n + (1 - \theta) \ln x\right]$.

preciates intrinsic health and cognitive abilities through δ is relevant only when quality investment is positive.

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. The evidence presented in section 2.5 shows infectious diseases in childhood to be an important determinant of cardiovascular diseases and ill health for older adults. We assume that an individual who was exposed to infectious diseases as a child in period t but survived faces a lower survival probability $\phi_u < 1$ in period $t+2$. Otherwise his probability of survival is $\phi_h > \phi_u$. Hence

$$
\phi_t = \begin{cases} \phi_u, & \text{if } I_{t-2} = 1 \\ \phi_h, & \text{if } I_{t-2} = 0 \end{cases} \tag{6}
$$

where I_{t-2} is an indicator function that takes the value 1 if the person was infected as a child in $t-2$ and zero otherwise.

3.3 Production of Final Output

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 $\alpha \in (0,1)$. Output and inputs markets are perfectly competitive and the depreciation rate on physical capital is hundred percent.

3.4 Optimization

We start with household decisions. Denote the average cost per childbirth by

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

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

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

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.[10]

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 a δ proportion of that of healthy children, $x_{t+1}^u = \delta^v x_{t+1}^h$. 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 quality cost saving from investing in unhealthy children relative to healthier ones. It follows that

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

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[10]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 incentives at the household level would be lacking and hence, on their own, cannot facilitate the transition.

and that quality investment in healthy children is

$$e_t^h = \frac{v(1-\theta)}{\theta - v(1-\theta)} \left[\frac{\tau_t - \eta i_t}{1 - d i_t} - \frac{\varepsilon\theta}{v(1-\theta)} \right].$$

For the latter to be economically meaningful requires that

$$v < \theta/(1-\theta) \quad \text{and} \quad \varepsilon < v(1-\theta)\tau_h/\theta. \tag{A1}$$

The first restriction is guaranteed if $\theta > 1/2$, similar to other models of fertility. The second one ensures that maximal human capital investment in healthy children is positive.

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

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

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

$$\tau_u < \tau_h + \left(\frac{1-\delta}{\delta} \right) \varepsilon. \tag{A2}$$

In other words, unhealthy children cannot be too costly. Else parental time saved from having healthier children would be directed towards raising more children instead of quality investment.

Corner equilibrium

Besides (A1) positive quality investment in healthy children requires that

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

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Likewise $e_t^u \geq 0$ requires that

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

Evidently $\Gamma^h \geq \Gamma^u$ while (A2) 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

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

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

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

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.

Since the fertility rate n_t does not depend on household characteristics, specifically x_t, it corresponds to the total fertility rate (TFR) and 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 (A2). 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, though, what is relevant is the expected number of surviving children, $\hat{n}_t = (1-di_t)n_t$, or the net fertility rate (NFR). This is given by

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

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where ψ_t was defined earlier. Under assumption (A2), 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.

Turn now to 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.

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, in both interior and corner equilibria, a reduction in prevalence extends adult longevity. As parents place a higher weight on their future consumption, 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 simple version of the SIR epidemiological model for this.

Suppose that a fraction μ_0 of children who survive from infectious disease remain infective as adults. The remaining $1 - \mu_0$ fraction recover but not from the scarring effect

of their childhood illness. Suppose also that all newborns are susceptible to infection and that each of them is "matched" with $\mu_1 > 1$ adults, similar to John (1990).[11]

A proportion $\mu_0 p i_t/(1 - d i_t)$ of these adults is infective. Let a be the transmission rate from a match. Then the number of newborns who contract infections in $t + 1$ is $I_{t+1} = \pi_t n_{t+1} L_{t+1}$ where $\pi_t = \mu a \left[p i_t/(1 - d i_t) \right]$, $\mu \equiv \mu_0 \mu_1 > 1$ by assumption and each of the L_{t+1} young adults at $t + 1$ has n_{t+1} children. For high enough prevalence, π_t can exceed one. Hence we specify that the infection rate $i_t \equiv I_t/n_t L_t$ follows

$$i_{t+1} = g(i_t) \equiv \min\{\pi(i_t), 1\}. \tag{11}$$

Equation (11) always entertains zero prevalence as a steady state whose stability depends on $g'(0) = \mu a p$. 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

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

which means each infective person infects more than one. Under conditions we specify below this can cause the disease to become endemic over time.

In addition to (A4) when we have $\mu a p > 1$, the zero steady state is asymptotically unstable and $\pi(\hat{\imath}) > 1$ for $\hat{\imath} \equiv 1/[d + \mu a p]$. Full prevalence is the only asymptotically stable steady state, as in Figure 8(a), and the disease is eventually endemic. In other words, all children at some point in their childhood contract infections.

Maintaining assumption (A4), suppose instead the survival rate is low enough that $\mu a p < 1$. This corresponds to Figure 8(b), where an intermediate steady state appears at $i^* = (1 - \mu a p)/d$ 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 people have to be infective 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

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[11]Alternatively, infected people transmit the germs to non-human hosts that then assist in new infections through disease vectors. For a disease like malaria the matching occurs through mosquitoes that take blood meal from the infective person and, after sporogony, release the parasite into the bloodstream of an uninfected child. For diarrhea, on the other hand, the disease vector can be food, water or humans themselves.

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.[12]

The last case, Figure 8(c), holds when (A4) is overturned which also implies $\mu a p < 1$. Zero prevalence is the unique asymptotically stable steady state: the basic reproduction ratio is too weak to forever sustain infections. This scenario captures post-transition societies that have eradicated endemic infectious diseases.

4 General Equilibrium

4.1 Factor Prices

In competitive markets, the Cobb-Douglas production technology leads to the familiar pricing functions for labor and capital, $w_t = (1-\alpha)Ak_t^\alpha$ and, $R_t = \alpha Ak_t^{\alpha-1}$ respectively. Here w is the wage rate per efficiency unit of labor, R the rental on capital as well as the interest factor and $k_t \equiv K_t/X_t$ the physical-to-human capital ratio.

4.2 Population and Labor Supply

From here on we make the simplifying assumption that $\kappa = 0$ which means 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 depend on the average stock of human capital and the prevalence rate.

Let $G_t(x)$ denote the period-t measure of young adults with human capital below x. A young adult with human capital x_t has $n(i_t)$ children of whom $p i_t n(i_t)$ grow up to be unhealthy adults with x_{t+1}^u units of human capital and adult longevity $1 + \phi_u$. The remaining $(1 - i_t)n(i_t)$ surviving children grow up healthy with x_{t+1}^h units of human capital and adult longevity $1 + \phi_h$. This determines the future working population as

$$L_{t+1} = (1 - d i_t)n(i_t)\int_0^\infty dG_t(x_t) = (1 - d i_t)n(i_t)L_t. \tag{12}$$

The aggregate stock of human capital of L_t workers is $X_t = \int_0^\infty x_t dG_t(x_t)$ and human capital per worker $\bar{x}_t \equiv X_t/L_t$. Using this output per worker is $y_t = Ak_t^\alpha \bar{x}_t$.

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[12]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.

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. Aggregate saving is

$$S_t = \sigma_u \int_{\{x_t : I_{t-1} = 1\}} w_t x_t dG_t(x_t) + \sigma_h \int_{\{x_t : I_{t-1} = 0\}} w_t x_t dG_t(x_t)$$

where $\sigma_u \equiv \beta\phi_u/(1 + \beta\phi_u + \gamma) < \beta\phi_h/(1 + \beta\phi_h + \gamma\theta) \equiv \sigma_h$ and market clearing is determined by the usual $K_{t+1} = S_t$.

4.3 Dynamics

The aggregate stock of human capital is $X_{t+1} = \left[pi_t x_{t+1}^u + (1 - i_t)x_{t+1}^h \right] L_{t+1}$. The human capital of each child type, x_{t+1}^h and x_{t+1}^u, is linear in $\bar{x}_t$ where the proportionality functions ρ_{ht} and ρ_{ut} depend on the prevalence rate according to

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

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

$$\bar{x}_{t+1} \equiv \frac{X_{t+1}}{L_{t+1}} = \left[pi_t \rho_{ut} + (1 - i_t)\rho_{ht} \right] \bar{x}_t \equiv \rho(i_t)\bar{x}_t \tag{13}$$

and asset market clearing by

$$K_{t+1} = \left[\sigma_u \rho_{ut} pi_{t-1} + \sigma_h \rho_{ht}(1 - i_{t-1}) \right] (1 - \alpha) A k_t^\alpha \bar{x}_{t-1} \tag{14}$$

given $K_0 > 0$ units of capital owned by the initial old generation and the initial distribution G_0 among working households.

Definition. *The general equilibrium of this economy consists of sequences of $\{K_t, \bar{x}_t, i_t\}$ and cumulative density functions $G_t(x)$ that satisfy equations (11), (13) and (14), given $K_0 > 0$, $i_0 > 0$ and G_0.*

5 Transitions

Since disease prevalence does not depend on economic behavior, equation (11) evolves independently of equations (13) and (14). The long run trajectory of this economy is driven by disease dynamics, specifically the vector of parameters (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 (13) 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 $\rho(0) = \lambda [\nu(1-\theta)(\tau_h - \varepsilon)/\{\theta - \nu(1-\theta)\}]^\nu$. When $\lim_{t \to \infty} i_t = 1$, since no child receives quality investment, the long-run growth factor is $\rho(1) = p\lambda\varepsilon^\nu$, lower than $\rho(0)$. The saving propensities σ_h and σ_u have no effect on the growth rate, only level effect on the balanced growth path (BGP). Hence two BGPs are possible and the economy converges to only one of them for a given vector (i_0, μ, a, p).

5.1 Balanced Growth Paths

The first BGP, a **Malthusian regime**, exists when (A4) holds and infectious disease fatalities are high enough that $\mu a p < 1$ (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. Low labor income and adult longevity both imply 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 at zero or at hundred percent, we can easily compare quantity and quality decisions 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 $\chi_H = \theta(\tau_h - \varepsilon)/[\theta - v(1-\theta)]$. 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

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

The fertility differential across these steady-state values is driven by differences in these parameters $(d, \tau_u, \tau_h, \delta, \phi_u, \phi_h)$. The first four parameters are related to the cost of fertility, the last two determine parental willingness to substitute between personal consumption and altruistic behavior. 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

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

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 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 England's conquest of infectious disease (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 re-

forms 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).[13]

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: an economy converging towards full prevalence eventually falls below this threshold. At that point, the transmission rate has been brought under sufficient control that it is possible to gradually eradicate endemic infectious diseases. 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 SSA. Despite a half-century of falling child mortality at a rate comparable to England & Wales, prevalence rates for SSA's major killer diseases remain high.

This is due to two reasons: deficiencies of best practice interventions and moribund public health systems. Consider malaria and diarrhea which together accounted for 34% of child deaths in Africa in 2005 (WHO). 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.[14] Diarrheal mortality, on the other hand, has fallen in Africa by about 10.5% during 1990-2000. Most of this reduction is not

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[13]Therapy played a minor role. Antibiotics for scarlet fever, for instance, were to be invented later.

[14]It is conceivable that this resurgence partly accounts for the drop in adult stature in the 1980s directly, and indirectly from co-morbidity with other diseases.

due to lower incidence but from the spread of an effective treatment (ORT) which re-
duced diarrheal mortality by 9% (Ewbank and Gribble, 1993; Boschi-Pinto *et al.*, 2006).
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, in-
terventions that so radically transformed England's disease landscape (Guerrant *et al.*,
2003).

Successful disease prevention comes from both private and public interventions.
Private health investment exhibits positive externalities, so without public intervention
there is too little investment. 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 SSA 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, this means child mortality has fallen in SSA through a combi-
nation 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. We
proceed to examine the quantitative significance of such a mortality transition.

6 Quantitative Experiments

We start by calibrating the model and then present three experiments. The first experi-
ment simulates England's transitions and compares them to slower counterfactuals rel-
evant for developing countries generally. The second is specifically tailored to account
for transitions in SSA. Finally we show that child mortality improvements, which have
contributed globally to improvements in life expectancy at birth, have little effect on
economic growth unless accompanied by improvements in 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

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

UK ($1860 - 69$) is close to this, we start at $i_0 = 1$. In the model parents implicitly choose quality investment in surviving children after the first 5 years of life. The value for d is picked to 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).[15] Normalizing $\phi_h = 1$, 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 (A2). 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 (A1). Consistent with data for the UK, we set $\gamma = 1.13$ such that pre-transition TFR is 4.9 children per woman.

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, consistent with data for the UK (Broadberry and Klein, 2011). Then $\nu = 0.48$ implies 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

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

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 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.*[16]

6.2 The Historical Transition

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 $a' = 0.37$ 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. We start both economies (DC and LDC) with $(x_0, k_0) = (1, 0.17)$, the steady-state values of human and

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[16]In pre-transition, childhood infections matter only for late-life mortality, δ is irrelevant.

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, TFR is generally higher resulting in a population explosion. During 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, with an elasticity of 0.82 relative to 0.46 in SSA. By construction child mortality falls by the same magnitude initially 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.[17]

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.

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[17]The simulated 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.

6.3 The Transition in Sub-Saharan Africa

Beginning in the 1960s, child mortality improved in SSA due to a combination of more widely available 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. But two features distinguish SSA. First, its pre-transition (1950-60) CMR was actually lower than the UK's (1850-70). Pre-transition TFR, on the other hand, 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 slow. 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 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 $\nu = 0.30$ and $\theta = 0.25$ to maintain post-transition fertility at replacement level. 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). Remaining parameter values are same 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 from 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 contrast 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.[18]

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 trajectory of child mortality from a falling 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. 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.

We plot 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

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[18]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.

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.[19] In fact for $\delta < 1$ the resulting income gap would be even greater because morbidity persists in the d-decline scenario.

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

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[19]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.

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.

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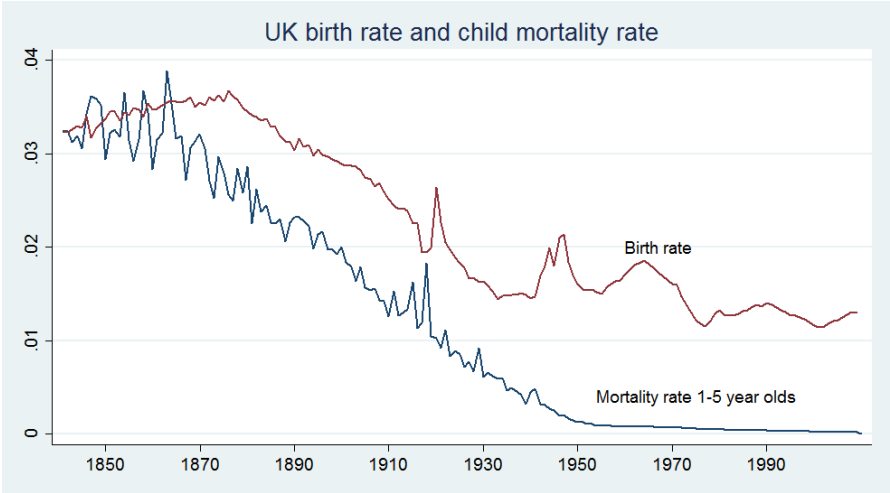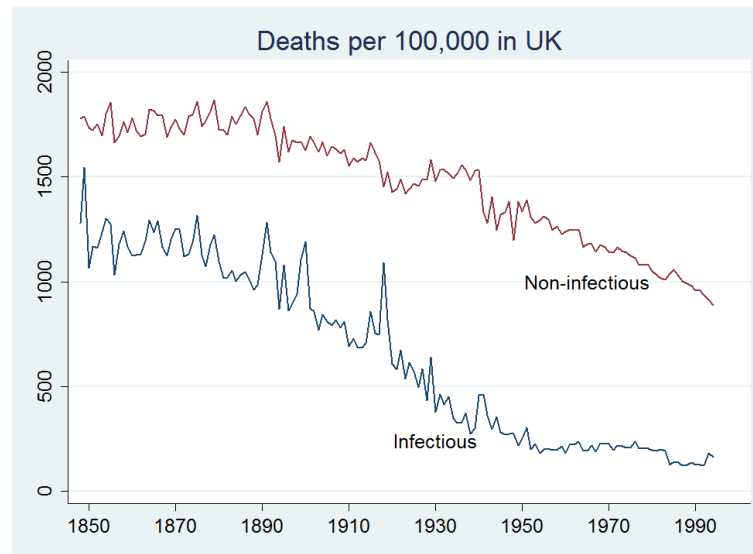
Source: mortality.org

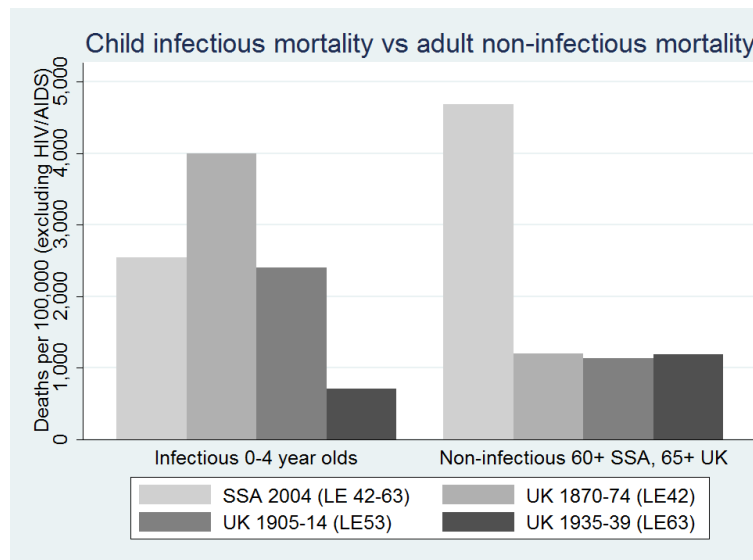**Figure 1** Demographic Transition in England and Wales
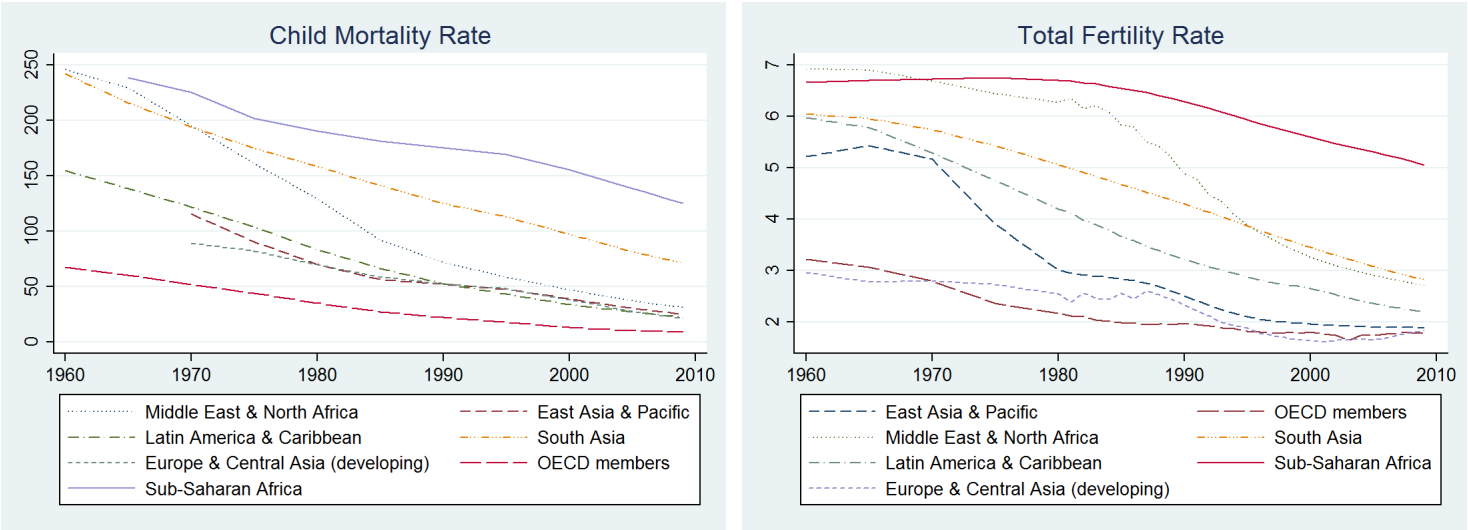
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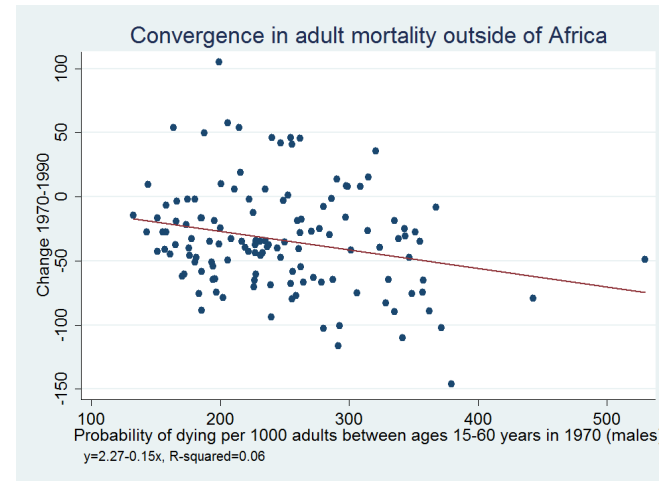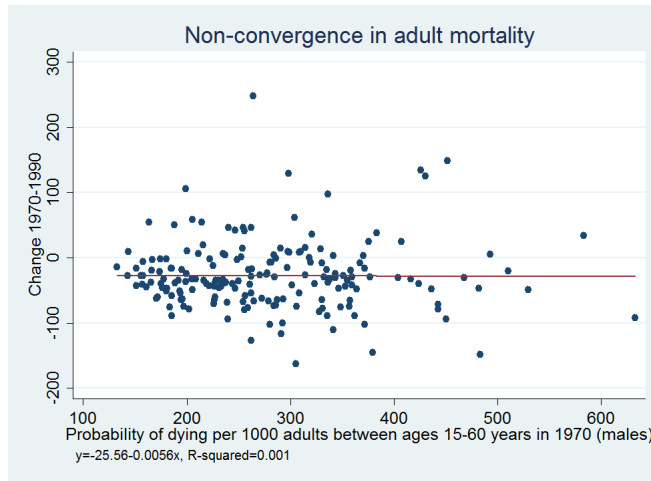**Figure 2** Demographic Transitions since 1960

Source: Arora (2005)

Figure 3 Epidemiological Transition in England & Wales 1848-1994
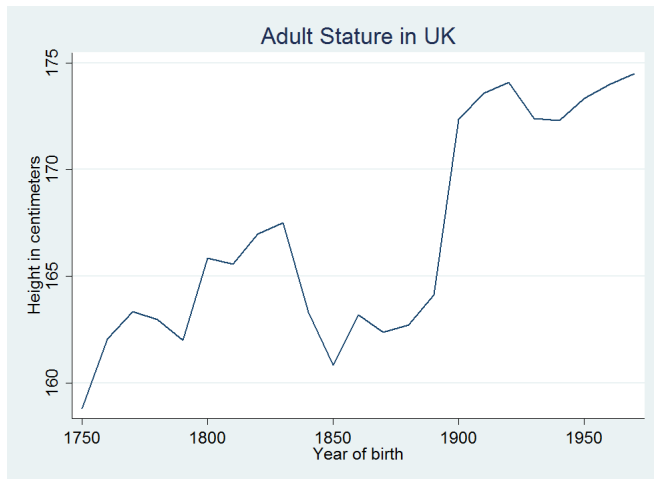

Source: Global Burden of Disease (2004), Arora (2005)

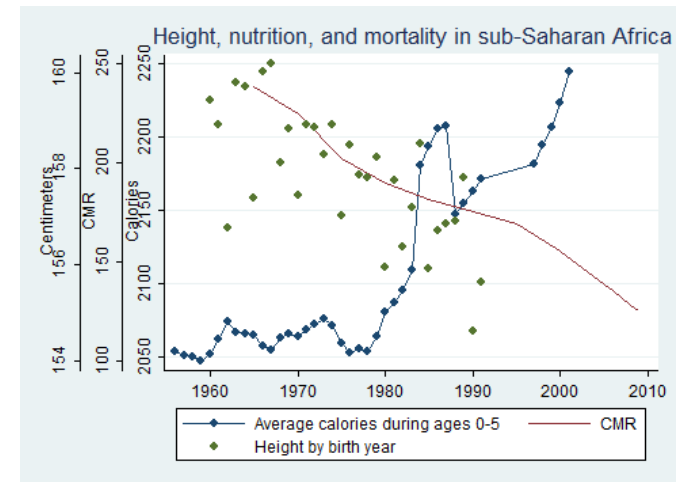**Figure 4** Disease Patterns in sub-Saharan Africa and the UK

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
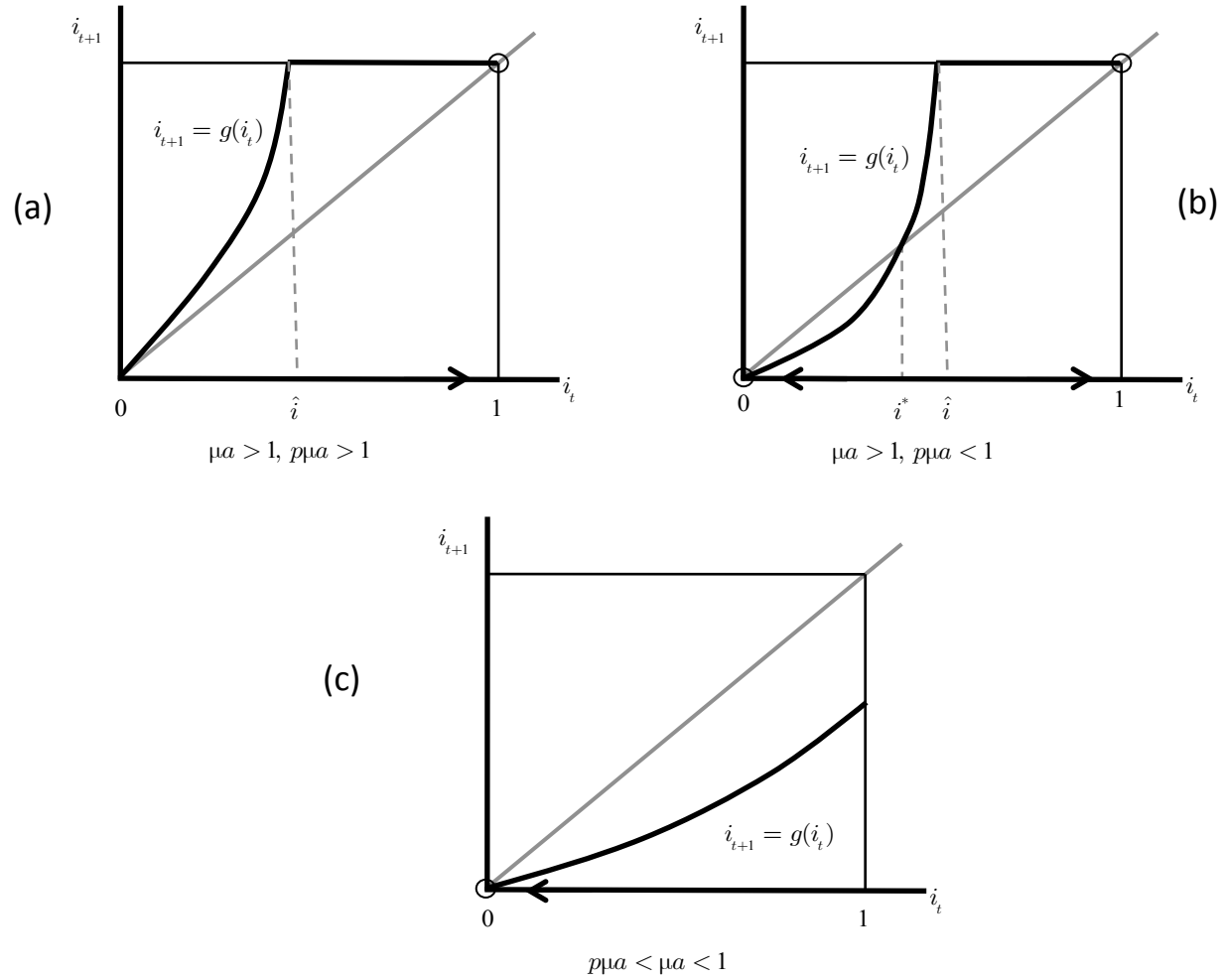
Figure 8 Dynamics of the Prevalence Rate
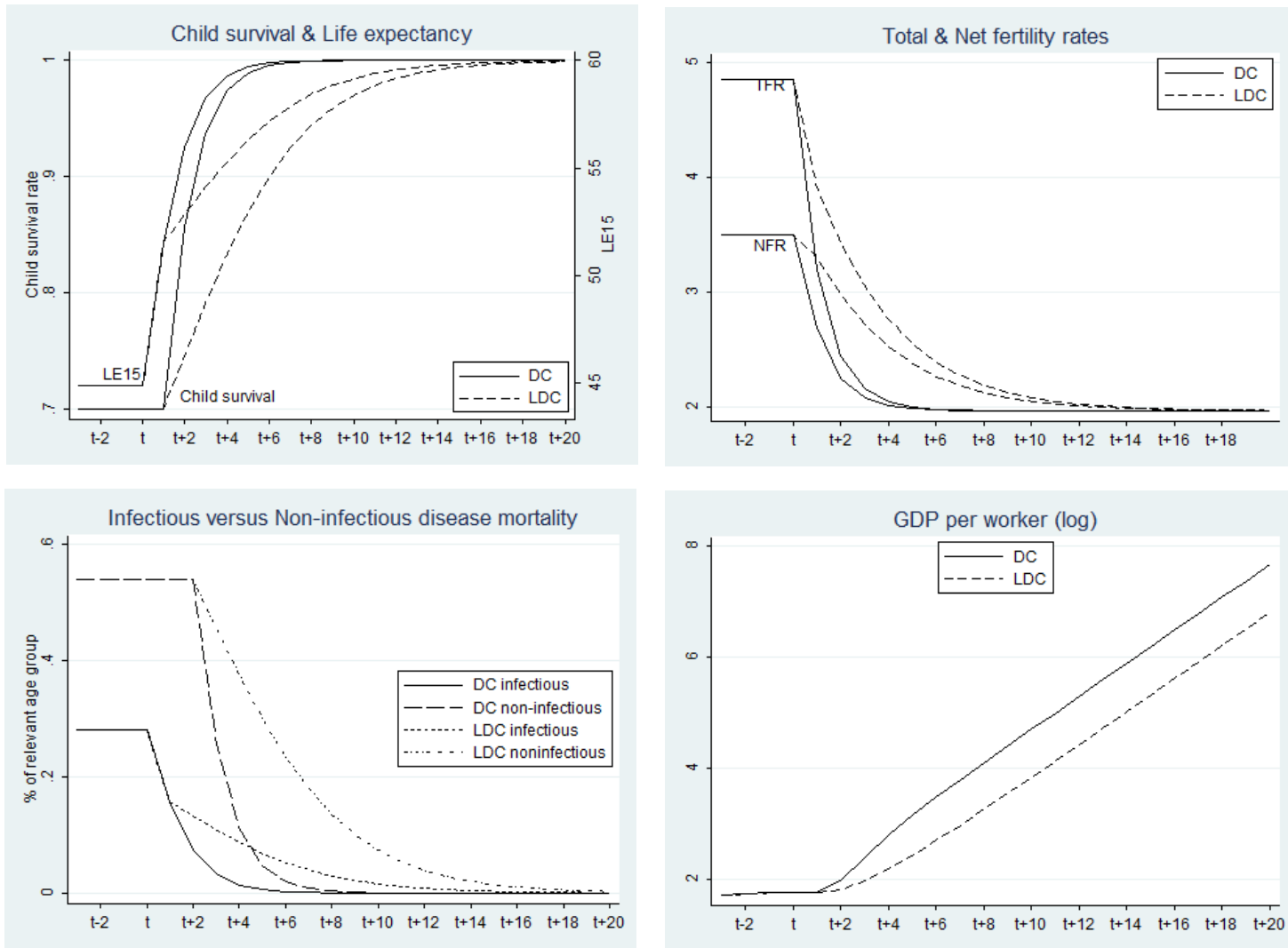
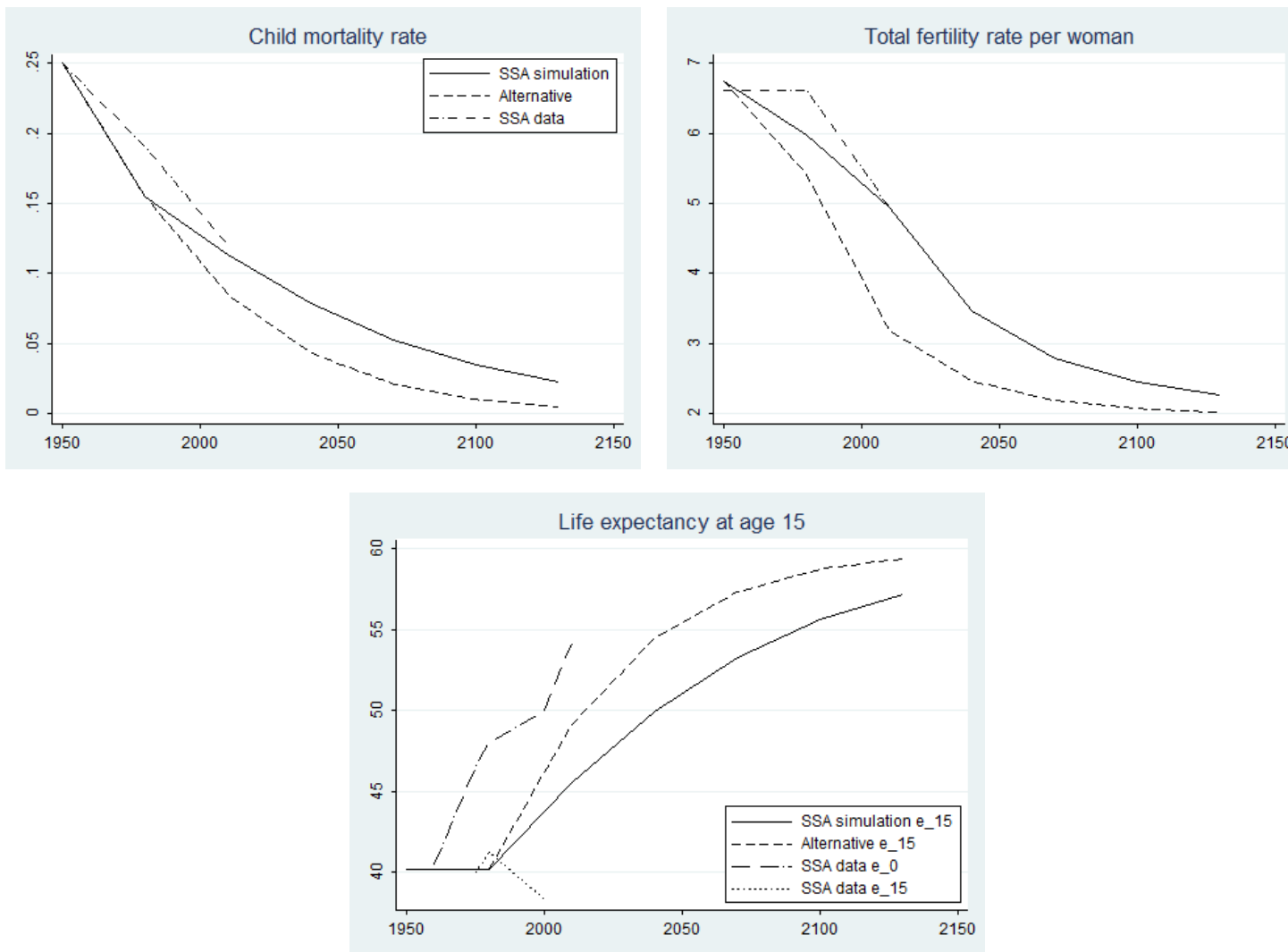**Figure 9** Simulated Demographic, Epidemiological and Economic Transitions

Figure 10 Simulated Demographic and Epidemiological Transitions in sub-Saharan Africa
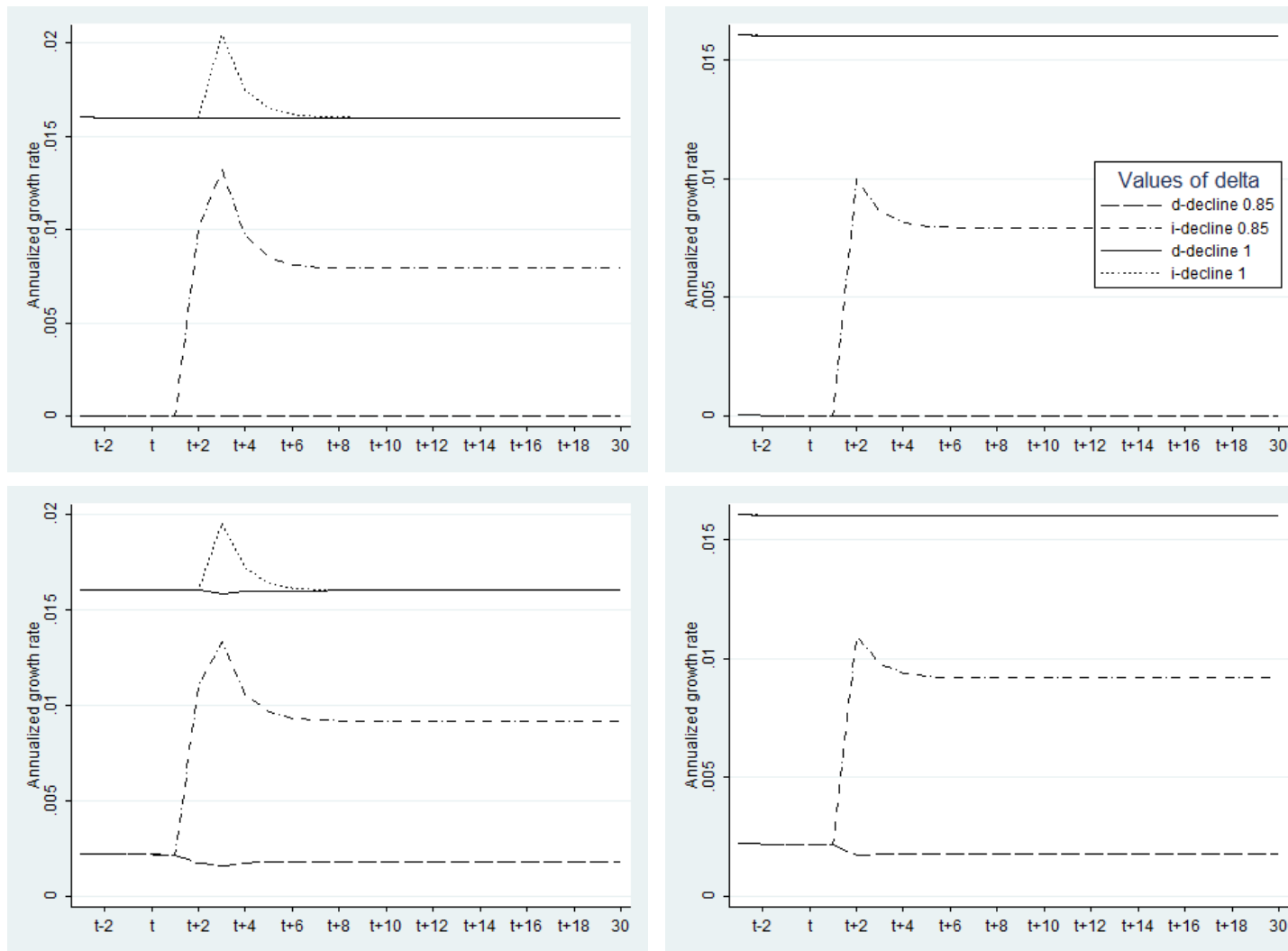
Figure 11 Differences in growth rates for *i* versus *d* decline economies when CMR falls from 28% to 18%

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 $\phi_u = \phi_h = 1$