Twin Transitions*

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Abstract

The epidemiologic transition offers a novel explanation for developing countries' sluggish demographic transition, their high non-infectious disease burden and slow growth. In a three-period overlapping generations model of endogenous mortality and fertility, infection in early childhood affects health human capital and late-life mortality from non-infectious disease. Child mortality is the product of disease prevalence and fatality from infections. When it falls due to lower prevalence of infectious disease, as it did in western Europe, a quantity-quality tradeoff lowers net fertility and raises human capital investment. Accompanied by higher adult longevity, an epidemiologic transition and economic growth follow. When child mortality falls mainly due to better survival from infections, as in developing countries, life expectancy at birth improves but adult mortality remains high and more surviving children are of lower quality. Demographic, epidemiologic and economic transitions are muted in this case.

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1 Introduction

Despite dramatic improvements in child survival, the fertility transition across the developing world, particularly sub-Saharan Africa, has been slow by historical standards.

We draw upon the historical epidemiologic transition and biomedical evidence to propose a novel explanation for this. During the epidemiologic transition, infectious disease mortality among the young and the elderly declines, followed by non-infectious disease mortality among older adults. After the transition is complete, more children survive into adulthood, more adults into old-age, and more retirees enjoy their potential lifespans. Biomedical evidence documents that falling infectious disease morbidity during childhood is accompanied by better health of young adults and lower non-infectious disease mortality among the elderly.

We develop a three-period overlapping generations model of endogenous mortality and fertility. Infectious diseases affect susceptible children some of whom do not survive. The health capital of those who survive from (recurrent) childhood infections is compromised, lowering the return on their education. In the model childhood infectious disease morbidity also affects late-life mortality from non-communicable diseases like cardiovascular disease (CVD).

Child mortality can decline in two ways. It can decline from a lower infectious disease prevalence or due to better survival from infections despite high prevalence rates. It was falling prevalence that caused child survival to improve in the West. Morbidity declined in tandem. We see this clearly for England and Wales where child survival gains in the late nineteenth century were followed by secular gains in adult stature in successive cohorts.

In contrast, the widespread availability of relatively inexpensive antibiotics and public health innovations since World War II has enabled developing countries to reduce child mortality by averting fatalities from infections. Even though vaccination has helped eradicate several diseases like smallpox and polio, prevention has generally lagged. In Africa prevention is not only more expensive in terms of institutional capacity and resources, it is also confounded by the continent's disease ecology. For example, vaccines for malaria and diarrhea, two of the biggest causes of morbidity among African children, are lacking. Despite sharp improvements in child survival, sub-Saharan Africa's infectious disease prevalence and childhood morbidity remain high. No comparable height gains have occurred (Akachi and Canning, 2008).

In the model when child mortality falls due to lower disease prevalence, more surviving children are of better health (quality), which raises the return to investing in their education. Altruistic parents realize these higher returns by opting for fewer children but investing more in their education than in the past. Child mortality lowers both the total and net fertility rates. Secondly, since fewer children would have suffered from infectious disease 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. A mortality transition in this case paves the way for epidemiologic and economic transitions. That improving child survival was associated with improving health (height) and falling non-infectious disease mortality tells us this was largely how Western Europe's demographic and epidemiologic transitions occurred.

Contrast this to the case where child mortality falls from lower case fatality. If infections are widely prevalent, a large proportion of children are exposed to them on a recurrent basis. Such chronic exposure implies that a higher proportion of surviving children will be of poorer intrinsic health for whom education yields low economic returns. Despite falling child mortality, parents have little incentive to substitute towards quality and continue to choose relatively large families. While the total fertility rate falls, the net fertility rate may not. Moreover, infectious disease exposure in early life implies that a large share of the adult population suffers from CVD-related premature mortality. Hence a mortality transition does not translate into a fertility transition or an epidemiologic one. The combined effects of low child quality and adult longevity stall economic progress despite the onset of a demographic transition.

This paper is related to several works on economic demography and development. Using CRRA preferences and ignoring child survival uncertainty, Doepke (2005) raises a puzzle: higher rates of child survival lower the total fertility rate, but leave the net fertility rate unaffected, a prediction at odds with historical demographic transitions. Even with these two assumptions the net fertility rate responds to child mortality in our model because child quality is heterogeneous and healthy and unhealthy children are imperfect substitutes in parental preferences. How strongly the net fertility rate responds to child mortality, however, depends on exactly how mortality rates fall. By distinguishing between child mortality and morbidity, we show that a decline in child mortality may even raise fertility if morbidity rises.

Strulik (2008) attributes stalled demographic transitions in tropical countries to an unfavorable geography prone to infectious disease. Both the exogenous disease environment and parental investment in health and nutrition affect child survival. Parental investment is less effective at improving child survival in low-disease environments, so the shadow cost of a child is higher there and parents have fewer children on net while shifting the bulk of human capital investment away from health and nutrition and towards education.

Our work shares Birchenall's (2007) view that the decline of infectious disease was instrumental in the transition to modern economic growth. Birchenall distinguishes between infection and case fatality rates, and improvements in childhood health outcomes reduce adult mortality. Parents affect their children's survival outcomes by investing in nutrition and health care,

so gains in income affect the demographic profile, generating an inverted-U path of population growth. Population growth slows once technological progress allows for sustained economic growth.

In our model, an exogenous change in the disease burden affects child health outcomes, triggering changes in adult mortality, fertility, and human capital investment. Rather than health depending on economic growth, the right kind of health improvement can foster economic development. Better public sanitation or a new vaccine reduces disease prevalence and moves the economy towards low disease and higher growth outcomes. If, on the other hand, a new antibiotic improves survival from existing disease, the consequences of that health improvement are potentially reversed.

While the epidemiologic transition has received scant attention in the literature, Morand (2002) is an exception. Since he does not take into account the shifting burden from infectious to non-infectious diseases that is typical of the epidemiologic transition, Morand's focus is more accurately described as a health transition. Health directly improves an individual's quality of life in his model. Achieving a threshold level of income triggers personal health investment which encourages capital accumulation in early adulthood to accommodate rising late-age consumption demand. There is, however, no demographic or epidemiologic response to changing health outcomes nor is a distinction made between child and adult mortality.

Our paper complements de la Croix and Licandro's (2010) analysis of the mortality transition and its associated health and human capital improvements. The authors argue that Western Europe's sustained improvement in physical development was behind its demographic transition. We provide a biological foundation for changes in physical development and its effect on human capital arising from the conquest of infectious disease. Our theory can reconcile child survival and longevity improvements with the weak demographic, epidemiologic and economic responses in developing countries.

Section 2 below presents some stylized facts on the demographic and epidemiologic transitions and on the importance of childhood health. Section 3 develops the theoretical model whose implications for the demographic, epidemiologic and economic transitions are developed in Sections 4 and 5. Preliminary results from a quantitative experiment are presented in Section 6.

2 Some Facts

We begin with some evidence on the demographic and epidemiologic transitions. Some of these facts motivate model assumptions, others we seek to explain using our theory.

2.1 The Demographic Transition

The conventional view of the demographic transition posits that falling child mortality made 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 on historical demographic transitions illustrates how falling infant/child mortality spurred rapid population growth before fertility rates adjusted downwards to thwart its momentum. A sharp decline in child mortality in England and Wales began around 1872, followed about five years later by declines in the total and net fertility rates (Figure 2).[1] France and the United States are exceptions to this pattern. In France, which led Europe's fertility decline, life expectancy at birth began a steady rise around 1872, but in many regions fertility had already begun to decline, probably due to recessions.

While there is room for debate whether or not a mortality transition preceded all nineteenth century fertility transitions (for further details see Galor, 2005), the evidence is clearer for twentieth century transitions in developing countries. Declines in child mortality have been instrumental, with a lag of about a decade, in worldwide fertility reductions during 1955−2005 (Angeles, 2010). Interestingly though, a mortality transition has not always triggered a commensurate fertility transition.

The African experience is particularly noteworthy (Figure 3). African fertility has not fallen as fast as we have come to expect from successful nineteenth and twentieth century transitions. For example, while infant mortality in Niger fell from 146.1 per 1,000 live births (1970) to 78.7 (2008), its total fertility rate (TFR) barely budged from 7.6 to 7.1. Uganda's infant mortality fell from 131.4 per 1,000 live births (1960) to 81 (2008), while its TFR declined slightly from 7.0 to 6.3 (World Bank).[2]

[1] Infant mortality did not start falling until the early 1900s. It has been conjectured that this was due to rapid urbanization and a succession of hot summers without which infant mortality would have fallen around the same time as child mortality (Hinde, 2003).

[2] One competing explanation to the one offered here is a persistent precautionary motive since the residual uncertainty of child survival remains high in Africa (Aksan and Chakraborty, 2011). Also note that HIV has extracted a high toll on the adult population but not had as much of an impact on child mortality rates.

2.2 The Epidemiologic Transition

Less commonly discussed in economic demography is the epidemiologic transition, the shift in mortality and disease patterns that accompanies the demographic transition (Omran, 1971). In pre-transition countries, infectious diseases are the prime cause of death especially among children and the elderly. Declining infectious disease mortality is what causes child mortality to fall during the transition. This is soon followed by declining deaths from non-infectious diseases, primarily CVDs, among the middle-aged and elderly. Figure 4 offers the simplest evidence of such a transition using age-standardized death rates from infectious and non-infectious diseases in England and Wales.

The experience of developing countries differs significantly despite rapid gains in child survival. Figure 5 compares infectious disease deaths for children (0−4 years) between sub-Saharan Africa and England and Wales (Arora, 2003). Statistics for the UK are reported at three levels of life expectancy at birth; life expectancy of 53 during $1905-14$ comes closest to the current life expectancy of $42-63$ in sub-Saharan Africa. Using that number as a crude age-standardized measure, the infectious disease burden in the two areas looks comparable. Figures 2 and 4, though, tell us that the English demographic and epidemiologic transitions were further along by the early twentieth century. In other words, sub-Saharan Africa's child mortality has improved significantly more than it did for the English population.

That improvement is by and large shared by the developing world. But it masks a key difference in developing countries' epidemiologic transitions: the earlier age at which CVD deaths have been occurring. For example, in 1990 the proportion of CVD deaths occurring below the age of 70 years was 46.7% in developing countries compared to 26.5% in developed countries (Reddy and Yusuf, 1998; Reddy, 2002). In Figure 6, a contrast is drawn between a country like the United States at one end of the epidemiologic transition with selected developing countries. The burden of CVD, a prime cause of adult and late-life mortality, is disproportionately high compared to what we would expect for a country still undergoing an epidemiologic transition. In sub-Saharan Africa, the Global Burden of Disease (2004) project reports that CVD deaths accounted for 30% of all deaths among $30-69$ year olds (19% if HIV mortalities are included), about four times that in the US (Leeder *et al.*, 2003).

What these data reveal is a high non-infectious disease burden affecting working-age and retired adults in developing countries, a burden that ought to be much lower given the spectacular decline in infectious disease mortality that they have experienced.[3]

[3]Death from non-infectious disease as a share of total deaths would obviously increase with the epidemiologic transition. What concerns us is their disproportionate share in developing countries that are lagging in their epi-

2.3 Childhood Morbidity

Infectious diseases account for much of childhood illness in developing countries. A simple way to assess their morbidity effect is to look at adult stature. Adult height is the cumulative product of net nutritional intake during childhood, that is, nutrition net of various claims on it including recurrent infections. Malnutrition and infections in early life can cause stunting which means height gains in successive cohorts are a good indication that the health of a population has been improving (Scrimshaw *et al.*, 1968; Fogel, 1993).

When infectious disease mortality started to fall in England and Wales around 1872, it was followed about fifteen years later by rising adult stature (Figure 7). Cohorts that experienced lower infectious disease mortality grew to be taller than their predecessors. Voth and Leunig's (1996) work further examines this connection: they find that smallpox outbreaks reduced heights by at least one inch in teenage boys who survived the disease. This occurred despite improving nutrition (Szreter, 1992). Height gains among English men, therefore, indicate that childhood disease morbidity, not just mortality, fell (Bozzoli *et al.*, 2009).

The contrast with sub-Saharan Africa here is stark. DHS survey data presented in Figure 8 shows that cohorts of African women who enjoyed higher survival in childhood experienced no such height gains. If anything, average height has fallen since 1960. In principle this reflects the combined effect of childhood nutrition and diseases (Akachi and Canning, 2008). The improvement in daily calorie intake that we see in Figure 9 from 1960 onwards, however, suggests infectious disease as the main culprit. The correlation between the two series is strongly negative, at -0.55. This pattern holds for protein intake data too (correlation of -0.27) since the two measures of nutrition strongly co-move (Figure 10).

2.4 The Economic Costs of Childhood Morbidity

Childhood morbidity concerns us because of its lifelong economic effects. The literature on childhood health is relevant here (Schultz, 2010; Almond and Currie, 2011).

Stunting in the first two years of life – an outcome of morbidity and diet – is significantly associated with poor cognitive performance in later childhood. A child's brain requires $44-87\%$ of the body's metabolic energy in the first few years after birth. Parasites and pathogens compete for this energy by feeding on the host's tissues, hijacking tissue growth to reproduce, and provoking the host's immune system. In a recent study, Eppig *et al.*, (2010) argue that the strong negative correlation between IQ scores and income levels across countries mainly reflects the

demiologic transition. The common explanation offered by public health professionals is "lifestyle factors".

toll of infectious disease on measured intelligence.

High infectious disease morbidity depresses returns to human capital (Behrman, 1996), while the cross-sectional evidence shows that adult height is associated with higher earning (Strauss and Thomas, 1998). Case and Paxson (2010) use five American and British surveys on height and various outcomes – including schooling, employment, earnings, health, and cognitive ability – from early to late adulthood. Across these samples, the authors 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.

Specific channels from infection to child quality can be identified for some diseases. Anemia is the most common effect of malaria and estimated to be 75% in areas where malaria prevalence exceeds 25%. Anemia has been directly linked to poor school performance and lower earnings among working adults (Snow *et al.*, 2003). Progressive iron and protein-deficiency anemia can also occur from hookworm infections which Bleakley's work (2007) identifies to be a leading cause of weak school performance in the American South. Remarkable improvements in children's scholastic performance were observed after the disease was eradicated in the early twentieth century. The higher return to schooling was accompanied by significant fertility declines (Bleakley and Lange, 2009).

Using randomized experiments, Miguel and Kremer (2004) study the educational effect of treatment for hookworm and other intestinal helminth infections on Kenyan primary school children. Reducing prevalence had large effects on school attendance (more effective learning) among the treated children, as well as among untreated children in treated schools (lower transmission due to externalities).

Diarrheal infections affect a large number of African children every year and are the second highest contributor to African child mortality. Many children also survive these infections. But infections are frequent: median episodes per child per year reaches a maximum of $4.1-4.3$ among $6-11$ month olds and drops down to $0.4-1.7$ by four years of age (Jamison *et al.*, 2006). Such frequent infections – as high as 9 median episodes during the first four years of life – cause permanent "scarring" because they prevent the absorption of nutrients necessary for optimal cellular growth in children (Martorell and Habicht, 1986; Mata, 1978). Guerrant *et al.* (2003) find that diarrhea in the first two years of life was strongly associated with impaired cognitive function $4-7$ years later among Brazilian children, surprisingly even when diarrheal symptoms did not manifest.

2.5 Late-Life Mortality

Another aspect of childhood morbidity bears upon the epidemiologic transition. Infectious disease early in life contributes to premature mortality from non-infectious causes later in life (Arora, 2005; Barker, 1994; Crimmins and Finch, 2006).

Measles, typhoid and malaria during childhood lead to cardiovascular problems (Khosla, 1981). Pneumonia before age 5 leads to diminished respiratory function at ages 59-70 (Barker 1992, 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).

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 a reduction in mortality at younger ages, and these cohorts also experienced increasing adult height (Crimmins and Finch, 2006). Data on Union Army veterans reveals that declining early life and young adult infectious disease rates accounted for close to 50% of the higher survival rates among 50–64 year old males, while reductions in infectious disease mortality accounted for the remaining (Costa, 2003). It is telling that England and Wales' decline in non-infectious disease mortality followed a generation after its infectious disease mortality began to fall (Figure 4).

Jousilahti *et al.*'s (2000) study of Finnish men and women offers another set of evidence linking childhood health to late-life mortality. In their sample, height was inversely associated with cardiovascular and total mortality. The negative relationship between height and mortality remained even after controlling for other known risk factors. Specifically, a 1 centimeter increase in height was associated with a 2 percent decrease in mortality.

2.6 Interpreting the Evidence

To explain the differential pattern of demographic, epidemiologic and economic transitions across the world, we propose a theoretical model below. The child mortality rate is a function of two factors, the infection rate and the case fatality rate from an infection. We propose that child mortality declines in developing countries have occurred mainly due to better survival from infections (lower case fatalities), while historical declines have occurred due to the conquest of infectious diseases.

This conquest was made possible by wide-ranging public health initiatives and innovations focused on prevention. In England and Wales the innovation of the germ theory coupled with sanitation reform triggered the transition. Improved sanitation and water management set up

in 1848 – 54 in England 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. With the establishment of the germ theory of disease, these public health reforms were complemented from 1880 onwards by immunization programs that tackled diptheria, smallpox, tuberculosis and whooping cough (Szreter, 1992; Easterlin, 1996). Between 1861 – 70 and 1891 – 1900, five diseases – smallpox, scarlet fever, whooping cough, diarrhea, and typhoid, all of which affected children in large numbers – accounted for more than 55% of the English mortality decline (Woods, 2000, Table 8.7). This decline was driven primarily by lower prevalence from weaker transmission and immunization. Scarlet fever deaths, for instance, could not have been lowered through therapy since antibiotics were to be invented later (Hinde, 2003).

That sub-Saharan Africa's infectious disease prevalence has not fallen as much as child mortality is clear.[4] The anthropometric evidence discussed above indicates a substantial morbidity burden pre-dating the HIV crisis. Progress has occurred in preventing some diseases. The fastest child mortality declines in developing countries occurred during 1975 – 85 as the WHO launched new vaccination programs. The eradication of polio in all but four countries remains a remarkable success for the WHO. Even though measles mortality remains high in sub-Saharan Africa, vaccination has evidently helped (Ewbank and Gribble, 1993).

But this progress is not widely shared across the spectrum of diseases afflicting sub-Saharan Africa. Prevention efforts have been constrained by moribund public health systems and lack of effective technologies. Consider diarrhea and malaria that together accounted for 34% of child deaths in Africa in 2005 (WHO). An efficient treatment, oral rehydration therapy (ORT), exists for diarrhea and the spread of its use has substantially cut down diarrheal mortality. But ORT usage rates remain below 50%, sometimes as low as 7% (Botswana; UNICEF). Compounding this glaring public health failure is the high prevalence of diarrhea among children. A review of 73 studies covering 23 countries during 1970 – 90, at the height of child mortality declines, found that children experienced on average 5 diarrheal episodes per year (ARCH Project Special Report, 1998). This reflects another kind of institutional failure, lack of adequate investment in sanitation and safe water that had radically transformed England's disease landscape (Guerrant *et al.*, 2003). Technological failures, on the other hand, are to blame for the continuing burden of malarial morbidity. The lack of an effective vaccine, the inability to control disease transmission

[4] 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 (Soares, 2007).

given existing technologies, and vectorial resistance to DDT mean that eradication efforts have floundered despite decades of intervention and investment.

3 The Model

An infinitely lived economy is populated by 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 each surviving child and educating him.

Some children die in their early childhood from infectious diseases. Define the survival rate of infected children by $p \equiv 1 - d$ where d is the case fatality rate (from an infection). If i denotes the childhood infection rate, then the child mortality rate is id. While the infection rate is endogenous in the model, we treat the fatality rate from an infection as an exogenous variable.

Of the n_t children born to each young adult in period t, $(1 - i_t)n_t$ children are healthy (denoted h), never having contracted infectious diseases. Another $pi_t n_t$ children are unhealthy (denoted u), having experienced but survived from chronic infectious diseases. 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 and e_t^u towards each type of surviving child.[5] The time cost of rearing an unhealthy child need not be the same as the cost of rearing a healthy one. In fact it is reasonable to think $\tau_u \geq \tau_h$ because an unhealthy child may need more care due to frequent bouts of infection and related ill health.

Following the literature we assume that parents base their decisions on the expected num-

[5] Children who die are costless. Adding a fixed birth cost only places an upper limit on the number of childbirths and does not qualitatively affect our results below.

ber of survivors of each type. A typical young parent maximizes expected lifetime utility[6]

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

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

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

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

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

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

taking as given the vector $(\phi_t, i_t, w_t, R_{t+1})$. Here $\beta \in (0,1)$ is a subjective discount rate and $\phi_t \in (0,1)$ is the probability of surviving to old-age. Wage per unit of human capital is represented by w, x represents the stock of human capital while s denotes the propensity to save out of potential income $z \equiv wx$. The second budget constraint incorporates the assumption of perfect annuities.

The last term in (1) is a joy-of-giving or warm glow bequest motive. $(1-i)/(1-di)$ is the fraction of surviving children who are healthy, $pi/(1-di)$ the fraction who are unhealthy. For $i = 0$ the joy-of-giving term simplifies to $\ln x^h$, while it becomes $\ln x^u$ for $i = 1$. Hence (1) nests the commonly used altruism specification for homogeneous child quality, $\gamma[\theta \ln n + (1-\theta)\ln x]$.

Embedded in the altruism specification are two assumptions. First, parents do not care about quantity and quality equally when $\theta \neq 1/2$. More importantly, quality investment in healthy and unhealthy children are imperfect substitutes. This latter assumption is central to generating an economic transition following (exogenous) declines in infectious disease prevalence.

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 parental investment in a child's human capital depends on his health human capital q_t^j, $j \in \{h,u\}$. This health is a product of childhood nutrition and disease experience. Specifically it takes the value $q_t^h = q_t$ should the child have experienced no (significant or recurrent) infectious disease and $q_t^u = \delta q_t$ otherwise.

Illness from infectious disease in early childhood depreciates a child's intrinsic health and cognitive abilities through the parameter $\delta \in (0,1)$. This effect is operative only when a child is

[6]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.

imparted human capital, a mechanism consistent with the recent evidence on infectious disease and IQ. In other words, absent human capital investment, healthy and unhealthy children are no different in their productivity. As δ rises (unhealthy children get healthier), e_t^u rises but investment in healthy children remains unaffected (see below). Historically health improvements, particularly those capitalized in anthropometric indicators like adult height, have been tied to nutritional and dietary gains. To incorporate this nutrition channel we posit that $q_t = q(y_t)$ depends on output per worker y_t. We assume that $q'(y) > 0$, $q'' < 0$, $q(0) = q_0 \geq 0$ and $\lim_{y\to\infty} q(y) = 1$.

3.2 Old Age Mortality

Old age diseases affect old age survival, when people are retired and consume out of accumulated wealth. This probability is endogenous with respect to past infectious disease experience. Specifically, 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 $\phi_h > \phi_u$.

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

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 0 otherwise. Alternatively we can write the survival probability as

$$\phi_t = [1 - (1-\phi) I_{t-2}] \text{ for } I_{t-2} \in \{0,1\} \tag{7}$$

that will be useful in constructing the general equilibrium of the model.

3.3 Production

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

$$Y_t = A K_t^{\alpha} X_t^{1-\alpha} \tag{8}$$

where $\alpha \in (0,1)$. Output and inputs markets are perfectly competitive and the depreciation rate on physical capital is assumed to be a hundred percent. CRS in two reproducible inputs opens up the possibility of endogenous growth under suitable parameterizations.

3.4 Household Decisions

Denote the average cost per childbirth by[7]

$$\chi_t \equiv (1-i_t)(\tau_h + e_t^h) + p i_t(\tau_u + e_t^u). \tag{9}$$

Rewriting the household's first period budget constraint as $c_t^t = (1-\chi_t n_t - s_t)z_t$, we obtain the following necessary and sufficient first order conditions in an interior optimum[8]

$$n_t \quad : \quad \frac{\chi_t z_t}{c_t^t} = \frac{\gamma\theta}{n_t} \tag{10}$$

$$s_t \quad : \quad \frac{1}{c_t^t} = \frac{\beta R_{t+1}}{c_{t+1}^t} \tag{11}$$

$$e_t^h \quad : \quad \frac{n_t z_t}{c_t^t} = \frac{\gamma(1-\theta)}{1-di_t}\left(\frac{\nu q_t}{\varepsilon + q_t e_t^h}\right) \tag{12}$$

$$e_t^u \quad : \quad \frac{n_t z_t}{c_t^t} = \frac{\gamma(1-\theta)}{1-di_t}\left(\frac{\nu\delta q_t}{\varepsilon + \delta q_t e_t^u}\right) \tag{13}$$

From (12) and (13), we see that investment in unhealthy children is lower the higher is the morbidity effect of infectious disease (lower δ)

$$e_t^u = e_t^h - \left(\frac{1-\delta}{\delta}\right)\frac{\varepsilon}{q_t}. \tag{14}$$

In turn this implies their human capital is a $\delta^\nu \in (0,1)$ proportion of that of healthy children

$$x_{t+1}^u = \delta^\nu \lambda(\varepsilon + e_t^h)^\nu x_t^\kappa \bar{x}_t^{1-\kappa} = \delta^\nu x_{t+1}^h.$$

Using (14), we express the average cost of fertility as

$$\chi_t = \tau_t + (1-di_t)e_t^h - \eta i_t / q_t \tag{15}$$

where $\tau_t \equiv (1-i_t)\tau_h + p i_t \tau_u$ is the average quantity cost, and $\eta \equiv (1-\delta)\varepsilon p/\delta$ is the effective cost saving from investing in unhealthy children relative to healthier ones. This average cost expression will be useful in establishing how child mortality affects the trade-off between quantity and quality of children.

Simplifying (10) to

$$n_t = \frac{\gamma\theta}{1+\gamma\theta}\left(\frac{1-s_t}{\chi_t}\right) \tag{16}$$

and combining with (11) we get

$$c_t = \frac{1}{1+\gamma\theta+\phi_t} z_t.$$

[7] We ignore the non-negativity constraint $0 \le \chi_t n_t \le 1$ for all t.

[8] Parametric assumptions below ensure this is always the case.

It follows that

$$\begin{aligned} s_t &= \frac{\phi_t}{1+\gamma\theta+\phi_t}, \\ n_t &= \frac{\gamma\theta}{1+\gamma\theta+\phi_t}\left(\frac{1}{\chi_t}\right). \end{aligned}$$

From (12) and (15), quality investment in healthy children is

$$e_t^h = \frac{\nu(1-\theta)}{\theta-\nu(1-\theta)}\left[\frac{\tau_t-\eta i_t}{1-di_t}-\frac{\varepsilon\theta}{\nu(1-\theta)}\right] \tag{17}$$

which requires

$$\nu < \theta/(1-\theta) \tag{A1}$$

for it to be a meaningful decision.

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

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

Since we are looking for an interior optimum where parents invest in both healthy and unhealthy children, it is necessary to impose parametric restrictions such that $e_t^h > e_t^u \geq 0$. First note that e_t^u and e_t^h are decreasing in the infection rate if $\psi_t \equiv (\tau_t-\eta i_t)/(1-di_t)$ is. This requires $p(\tau_u-\tau_h)-\eta/q_t<0$ for which it is sufficient to assume that

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

In other words unhealthy children cannot be too costly to raise. Else parental time freed up from lower infection rates would go towards raising more children instead of investing in child quality. The restriction above requires intrinsic child quality (ε) and the morbidity effect ($1-\delta$) to be large enough.

From the expression for optimal quality investment, $e_t^u \geq 0$ requires that

$$\begin{aligned} e_t^h &\geq \left(\frac{1-\delta}{\delta}\right)\frac{\varepsilon}{q_t} \\ &\Rightarrow \quad \psi_t \geq \frac{\varepsilon}{\nu(1-\theta)}\left[\theta+\{\theta-\nu(1-\theta)\}\left(\frac{1-\delta}{\delta}\right)\frac{1}{q_t}\right]. \end{aligned}$$

Since (A2) ensures that ψ_t is decreasing in i_t, a sufficient condition is

$$\tau_u \geq \frac{\varepsilon}{\nu(1-\theta)}\left[\theta+\{\theta-\nu(1-\theta)\}\left(\frac{1-\delta}{\delta}\right)\frac{1}{q_0}+\nu(1-\theta)\left(\frac{1-\delta}{\delta}\right)\right] \equiv \tau_L. \tag{A3}$$

The parameter space is henceforth restricted to satisfy assumption (A1) and $\tau_u \in [\tau_L, \tau_H]$.

The Effect of Child Mortality

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

The fertility rate n_t does not depend on household characteristics, specifically, parental human capital x_t. Consequently it represents the total fertility rate (TFR). The TFR responds negatively to the average fertility cost χ_t. Straightforward differentiation establishes that χ_t is a decreasing function of both i_t and d by assumptions (A2) and (A3). Hence a decrease in child mortality either through the infection or case fatality rate has the effect of lowering the TFR, consistent with the facts outlined earlier.

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

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

where the function ψ_t was defined earlier. Under assumption (A3), 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 infection rates, the drop in the TFR is strong enough to ensure the NFR falls. In contrast, a decrease in the child mortality rate due to lower case fatality elicits a weak TFR response: the NFR increases despite falling fertility.

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 infection rates raises quality investments in both healthy and unhealthy children. The opposite is true for gains in child survival through lower d. Since a higher proportion of surviving children are of low quality when fewer of the sickly children succumb, parents invest more time in raising a sickly brood on whom quality investment yields low returns.

3.5 Disease Dynamics

While the infection rate is exogenous to a household's decisions, the prevalence of infectious disease evolves endogenously in the aggregate.

We have been referring to i_t as the infection rate. Under the assumption that all children who survive from infection remain infective as adults, i_t also refers to the disease prevalence rate. Similar to John (1990), suppose each infectious person makes contact with $\mu > 1$ newborns

all of whom are susceptible. The probability that a susceptible child contracts infection is then

$$\pi_t = \mu a \left[\frac{p i_t}{1 - d i_t} \right], \tag{18}$$

an increasing convex function of i_t, where $\mu p i_t/(1 - d i_t)$ is the probability that a child encounters an infective adult and a is the transmission rate from that contact. This construction assumes that infective adults cannot infect non-infective adults, children cannot be infected by retired adults (that is all contact with children is limited to working-age adults such as parents, teachers and doctors), and that children do not infect other children. While μ represents the disease ecology like vectors, microbes and population density, it is helpful to think of a as determined by cultural and behavioral factors that are exogenous to the model and availability of preventive technologies such as vaccines.

For a high enough prevalence rate, the expression on the right in equation (18) can exceed one. Hence infectious disease prevalence follows the first-order non-linear difference equation

$$i_{t+1} = g(i_t) \equiv \min\{\pi(i_t), 1\} \tag{19}$$

By the law of large numbers, i_{t+1} represents the fraction of all children born in t who get infected.

Equation (19) always entertains zero as a steady state prevalence rate. Its behavior as illustrated in Figure 11 depends on parameter values. The slope at zero, $g'(0) = \mu a p$, governs the stability of the zero prevalence steady state. Suppose

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

that is, each infection is capable of generating multiple infections. When we also have

$$\mu a p > 1$$

as in Figure 11(a), zero is asymptotically unstable and $\pi(i) > 1$ for $i > \hat{\imath} \equiv 1/[d + \mu a p]$. In this case full prevalence is the only asymptotically stable steady state.

Maintaining assumption (A4), now suppose the survival rate is small enough that

$$\mu a p < 1.$$

This corresponds to Figure 11(b), where an intermediate steady state appears at

$$i^* = \frac{1 - \mu a p}{d}$$

that is increasing (decreasing) in the case fatality (survival) rate d ($p = 1 - d$). Initial conditions matter here: for initial prevalence exceeding i^*, infectious diseases replicate rapidly until full prevalence is reached. Infectious diseases dissipate in the long run, in contrast, for initial values below i^*.

Since $\partial \hat{\imath}/\partial p$, $\partial i^*/\partial p < 0$, better child survival makes it more likely the economy will reach full prevalence for a given initial value i_0. A higher p ensures that more of the infected population survives and hence, a larger pool of infective agents. This translates into faster transmission rates. In contrast, $\partial i^*/\partial(\mu a) < 0$: improvements in prevention make it less likely for the economy to end up with hundred percent prevalence.

The last case, Figure 11(c), holds when (A4) is overturned which also implies $\mu a p < 1$. Here zero is the unique steady state that is also asymptotically stable. The disease multiplier is not powerful enough to sustain infectious diseases in the long run.

4 General Equilibrium

4.1 Factor Prices

In competitive markets, the Cobb-Douglas production technology implies familiar pricing functions for capital and labor

$$\begin{aligned} w_t &= (1-\alpha) A \left(\frac{K_t}{X_t}\right)^{\alpha}, \\ R_t &= \alpha A \left(\frac{K_t}{X_t}\right)^{\alpha-1}, \end{aligned}$$

where w is the wage rate per efficiency unit of labor and R is the rental on capital (as well as the interest factor). Denote by $k_t \equiv K_t/X_t$ the physical-to-human capital ratio.

4.2 Demography and Labor Supply

Let $G_t(x)$ denote the measure of young adults with human capital below x in period t. First consider population size. This is determined by fertility behavior across the different types of households. Given the prevalence rate i_t, we assume a proportion i_t of children born to parents with $x \in [x-\epsilon, x+\epsilon]$ for arbitrarily small ϵ are infected and of them a d fraction die. Recall that fertility and human capital choices are independent of parental characteristics, that is, x_t. Hence 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^u_{t+1} units of human capital and adult longevity of $1+\phi_u$. Another $(1-i_t)n(i_t)$ grow up healthy with x^h_{t+1} units of human capital and adult longevity of $1+\phi_h$. The

remaining children succumb to infectious diseases.

Hence the size of the working population is

$$L_{t+1} = (1 - di_t)n(i_t)\int_0^\infty dG_t(x_t) = (1 - di_t)n(i_t)L_t. \tag{20}$$

The aggregate stock of human capital of the L_t workers is

$$X_t = \int_0^\infty x_t dG_t(x_t)$$

and the average stock

$$\bar{x}_t = \frac{X_t}{L_t}$$

which using output per worker $y_t \equiv Y_t/L_t$ can be expressed as

$$\frac{Y_t}{X_t} = \frac{Y_t/L_t}{X_t/L_t} = Ak_t^\alpha \Rightarrow y_t = Ak_t^\alpha \bar{x}_t. \tag{21}$$

The aggregate capital stock K_{t+1} comprises of the accumulated savings of both low (less healthy) and high (healthier) productivity workers. Suppose worker type is costlessly observed and hence, annuity sellers can calibrate their returns to the risk of premature mortality in each group. This means healthy workers earn the (gross) return R/ϕ_h on their saving, unhealthy workers earn the higher return R/ϕ_u. In any case, this is inconsequential for the supply of capital since preferences are logarithmic.

Aggregate saving at t 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

$$\sigma_u \equiv \frac{\beta\phi_u}{1 + \beta\phi_u + \gamma\theta} \text{ and } \sigma_h \equiv \frac{\beta\phi_h}{1 + \beta\phi_h + \gamma\theta}$$

and market clearing is determined by

$$K_{t+1} = S_t.$$

4.3 Simplification

Henceforth we make two simplifying assumptions. First, we shut down the effect of nutrition on human capital investment by assuming $q(y_t) = q_0 = 1$ for all t. Since the model's central

mechanism does not rely on the complementarity between nutrition and educational investment, this is a minor simplification. This also simplifies assumptions (A2) – (A3) to

$$\tau_h + \left(\frac{1-\delta}{\delta}\right)\varepsilon > \tau_u > \tau_h > \left(\frac{\theta}{\nu(1-\theta)}\right)\frac{\varepsilon}{\delta} \tag{A5}$$

under which parental investment in both low and high quality children is always positive.

The second simplification we make is to assume $\kappa = 0$, that is, the future productivity of children is determined by their health history and economic aggregates, not parental characteristics. In this case, their human capital takes one of two values that depend on the average stock of human capital and the prevalence rate

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

Noting that (x^h_{t+1}, x^u_{t+1}) are linear in $\bar{x}_t$ and denoting the proportionality constants by ρ_h and $\rho_u \equiv \delta^\nu \rho_h$ respectively, the average human capital stock evolves according to

$$\bar{x}_{t+1} \equiv \frac{X_{t+1}}{L_{t+1}} = \left[pi_t\rho_u + (1-i_t)\rho_h\right]\bar{x}_t \equiv \rho(i_t)\bar{x}_t.$$

Combining this with asset market clearing leads to the law of motion for physical capital

$$\begin{aligned} K_{t+1} = S_t &= \sigma_u \int_{\{x_t: I_{t-1}=1\}} w_t x_t dG_t(x_t) + \sigma_h \int_{\{x_t: I_{t-1}=0\}} w_t x_t dG_t(x_t) \\ &= \sigma_u(1-d)i_{t-1}\rho_u w_t \bar{x}_{t-1} + \sigma_h(1-i_{t-1})\rho_h w_t \bar{x}_{t-1} \\ &= \left[\sigma_u\rho_u(1-d)i_{t-1} + \sigma_h\rho_h(1-i_{t-1})\right] w(k_t)\,\bar{x}_{t-1} \end{aligned}$$

given $K_0 > 0$ units of the initial capital stock owned by the old generation at $t = 0$.

Since parents invest in their children's human capital and parental preferences are logarithmic in children's human capital, higher returns to human capital do not directly affect human capital accumulation. Instead long run growth hinges on whether or not human capital accumulation can be sustained on its own. In other words, it requires $\rho(i_t) > 1$. If that holds, a decrease in the prevalence rate will unambiguously raise the rate of human capital accumulation, and hence, physical capital accumulation. If not, marginal reductions in i_t raise the steady-state output per worker alone, while large-scale reductions can trigger a growth take-off.

When $\lim_{t\to\infty} i_t = 0$, the growth rate asymptotes to $\rho(0) = \rho_h$. When $\lim_{t\to\infty} i_t = 1$, in contrast, the long-run growth rate is $\rho(1) = p\rho_u$ which may or may not exceed 1. The saving propensities σ_h and σ_u have no effect in this economy on the long run growth rate. They only have a level effect on the balanced growth path.

Finally, given initial conditions and parameter values in Figure 11, only one of these BGPs is possible at a time. The higher growth path is feasible only for values of μ and a that overturn (A4).

5 Transitions

The long run behavior of this economy is driven entirely by disease dynamics, the vector of parameters (i_0, μ, a, p). Specifically two balanced growth paths (BGPs) are possible and the economy converges to only one of them for a given vector.

5.1 Steady States

The first BGP is of **Malthusian Stagnation** and exists when (A4) holds and infectious disease fatalities are high enough that $\mu ap < 1$ (Figure 11b). For $i_0 > i^*$, full prevalence is the only stationary equilibrium. Along this BPG, fertility rates remain high and infectious diseases extract a high mortality toll on children. Since all individuals are infected, survivors from childhood illness all carry their morbidity burden in the form of low health human capital, low labor productivity, and higher propensity for non-communicable disease later in life. Low labor income and adult longevity both imply low rates of investment in physical capital. This BGP then exhibits very slow (if any) growth in income per capita of $p\rho_u$ and a persistent morbidity burden from infectious disease.

The second stationary equilibrium exhibits **Modern Economic Growth** (MEG) and corresponds to $\mu a < 1$ (Figure 11c). Zero prevalence of infectious diseases is the only stationary equilibrium here. Absent any possibility of infection, child survival is ensured: fertility is low in this BGP and both forms of human capital relatively high. This economy is also in a post-epidemiologic transition phase where adult longevity is maximal due to the absence of infectious disease caused non-communicable diseases. High rates of investment in physical and human capital ensure a high growth rate of ρ_h.

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. Recall that the average fertility cost χ is decreasing in the prevalence rate. In the Malthusian BGP, this cost takes the lowest possible value ($i = 1$)

$$\chi_L = \frac{\theta}{\theta - \nu(1-\theta)}(\tau_h - \varepsilon).$$

In the MEG BGP, on the other hand, χ takes the highest possible value ($i = 0$)

$$\chi_H = \frac{\theta}{\theta - \nu(1-\theta)}(1-d)\left(\tau_u - \frac{\varepsilon}{\delta}\right).$$

It follows that the TFR in the Malthusian BGP takes the highest value

$$n_H = \frac{\gamma\theta}{1+\gamma\theta+\phi_u}\left(\frac{\theta - \nu(1-\theta)}{\theta}\right)\left[\frac{1}{(1-d)(\tau_u - \varepsilon/\delta)}\right]$$

while it takes the lowest value

$$n_L = \frac{\gamma\theta}{1+\gamma\theta+\phi_h}\left(\frac{\theta-\nu(1-\theta)}{\theta}\right)\left[\frac{1}{\tau_h-\varepsilon}\right]$$

in the MEG BGP. 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 drive the cost of fertility. The last two determine parental willingness to substitute between personal consumption and altruistic behavior. The value of future consumption rises with a longer lifespan, causing parents to substitute away from family size.

The corresponding net fertility rates in the two steady states are

$$\begin{aligned}\hat{n}_H &= (1-d)n_H \\ \hat{n}_L &= n_L\end{aligned}$$

while (average) human capital investments are

$$e^u = \frac{\nu(1-\theta)}{\theta-\nu(1-\theta)}\left(\tau_u - \frac{\theta\varepsilon}{\nu(1-\theta)}\right) - \frac{\theta}{\theta-\nu(1-\theta)}\left(\frac{1-\delta}{\delta}\right)\varepsilon$$

and

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

respectively.

5.2 Transitions Then

Start with assumption (A4) which is more likely when a and μ are relatively high. Before the arrival of public health and medical innovations of the late nineteenth and early twentieth centuries, it is also plausible to assume high fatalities from childhood infectious disease. In other words, suppose the world looked like Figure 11(b) with $i_0 > i^*$. Such an economy would have been mired in a Malthusian steady state.

Suppose next dramatic public health and medical innovations lower a by enough (a medical innovation would be vaccines, but potentially antibiotics can also help by reducing the time a person remains infective) to overturn (A4). In that case, i^* will cease to be a steady state, only zero remains one and becomes asymptotically stable. This suggests the potential gains from health improvements that work through a are not just a marginal reduction in i^* but eradication of diseases in the long run. Indeed, innovations do not have to be drastic enough to eliminate i^*. Modest but continuing improvements in a (or equivalently, μ) keep raising the threshold prevalence rate i^* – an economy with less than full prevalence but converging towards it can find itself below this threshold after sufficient improvements have occurred. At

that point, the disease multiplier has been brought sufficiently under control that the existing prevalence rate cannot be sustained: the economy switches from converging to the Malthusian BGP to converging to the MEG one.

We argued earlier that the conquest of infectious diseases that occurred in nineteenth century England and Wales and elsewhere in the West was made possible by large-scale public health efforts and innovations. In the model these preventive gains are akin to a reduction in μa that lowers susceptibility to infections. A sufficiently large reduction in prevention tips the prevalence rate below the threshold i^* in Figure 11(b). Thereafter, the forces of convergence propel the economy towards the MEG path. As the economy converges to this growth path, the decline of infectious disease mortality *and* morbidity paves the way for a fertility transition. The TFR and NFR both decline, parents substitute towards quality investment as child health capital improves and late-life survival improves. Successful demographic and epidemiologic transitions drive the economy towards rapid economic growth.

5.3 Transitions Now

Twentieth century reductions in child mortality in the developing world have been possible due to the adoption of some nineteenth century public health innovations. Medical technologies like antibiotics and vaccines have, however, been the main drivers. Though vaccines can be a successful preventive strategy, they are lacking for several developing countries' infectious disease. Child mortality reductions in developing countries have thus occurred through a combination of lower case fatalities, d, and lower incidence rates (μa).

Disease dynamics can respond perversely to a decrease in d. In Figure 11(b), it lowers i^* making it less likely for the economy to escape the disease burden. More generally, tackling case fatalities alone does not address the central problem, the high prevalence of infectious disease and disease vectors. Despite improving child survival, the infectious disease morbidity burden remains high as most children suffer through childhood. There is also a composition effect: a higher proportion of surviving children are of low health, depressing returns on their human capital investment.

If improvements in child survival have occurred mainly through lower case fatalities and, in for some diseases, through lower prevalence, then the combined effect on fertility behavior is unclear. The NFR may or may not fall, but it will for sure fall by less than if the entire increase in child survival were to occur through lower prevalence. Consequently falling child mortality does not move the demographic transition as far along. The high disease burden in turn suspends an epidemiologic transition: more of the population is susceptible to non-infectious

Preference	Technology	Disease Ecology
$\theta = 0.5$	$\lambda = 2$	$\mu = 1.9$
$\gamma = 10$	$\varepsilon = 0.001$	$a = 0.7$
$\beta = (0.99)^{30} = 0.740$	$\nu = 0.5$	$d = 0.33$
$\phi_h = 1$	$A = 10$	$\delta = 0.7$
$\phi_u = 0.367$	$\alpha = 1/3$	

Table 1: Parameter Values

disease later in life and faces high premature mortality, and non-infectious diseases account for a disproportionate fraction of early deaths. The absence of a fertility transition and low health capital also means the economy remains mired in slow growth even if that growth rate exceeds that under Malthusian stagnation.

6 A Computational Experiment

The simple numerical experiment presented below highlights the morbidity channel. Specifically it shows that demographic, epidemiologic and economic transitions are more muted when child mortality declines are not accompanied by morbidity declines.[9]

6.1 Parameter Values

We start by calibrating parameter values. Suppose childhood lasts for 20 years while each period of adulthood is potentially 30 years long. Maximum longevity is then 80 years. Table 1 reports values for some of the parameters

A pre-transition economy in our model is at full prevalence ($i = 1$). Assuming pre-transition UK ($1860-69$) is close to this, we start at $i_0 = 1$. The value for d is picked to be 0.33 so that $i_0 d$ matches the probability of dying between ages $0-20$ in the UK between $1860-69$ (mortality.org). Normalizing $\phi_h = 1$, we set $\phi_u = 0.367$ to match life expectancy at age 20 of 41.01 in the UK during the same period (mortality.org). On the preference side, child rearing cost parameters (τ_h, τ_u) are calibrated to satisfy the modified assumption (A5).

Since we do not have much guidance on μ and a, we arbitrarily set $\mu = 1.9$. This requires $a > 0.52$ so that $i^* < 1$. We set $a = 0.7$. The morbidity parameter δ is set at 0.7, relatively close to 1. It implies that the human capital of unhealthy children is about 84% of that of healthy

[9] Preliminary results, not all parameters are tightly calibrated.

children.

6.2 Simulating a Transition

Starting with an initial configuration of parameter values $(a_0, d_0) = (0.7, 0.33)$ for an economy stuck in Malthusian stagnation, consider two experiments. In one experiment relevant for developed countries (DC) that underwent demographic transitions in the nineteenth century, a pre-transition economy undergoes an exogenous reduction in child mortality purely from infectious disease prevalence. Specifically, maintaining the value of d, a substantial decline in a from $a_0 = 0.7$ to $a' = 0.3038$ pushes i^* above 1 and starts a transition towards zero disease prevalence.

The second experiment is relevant for less developed countries (LDC) where child mortality reductions have occurred partly from decline in prevalence, partly from lower case fatalities. The new set of parameters (a_1, d_1) is chosen so that the first period improvement in child survival is equal to that in the first scenario. This requires the following relationship to hold

$$a' = \frac{a_1 d_1 (1 - d_1)(1 - d_0 i_0)}{d_0 (1 - d_0)(1 - d_1 i_0)}.$$

We lower a to $a_1 = 0.5$, just enough to eliminate the full prevalence steady state. The remaining drop is child mortality is absorbed by the case fatality rate falling to $d_1 = 0.2$. The combined effect pushes $i^* > 1$ and the transition towards zero disease prevalence begins, but at a slower rate.

We start both economies (DC and LDC) with $(x_0, k_0) = (0.001, 0.0001)$ and a workforce comprised entirely of unhealthy workers consistent with the Malthusian BGP. Figures 12–14 illustrate the results from these two experiments for the demographic, epidemiologic and economic transitions respectively. In each figure the top panel corresponds to the DC transition, the bottom one to the LDC transition.

It is clear from these trajectories that in each case the nature of LDC mortality transition extracts a demographic and economic cost relative to the DC transition. LDC transitions are slower for two reasons. First because the initial decline in prevalence is smaller so that childhood morbidity and late-life mortality are higher. Secondly, the decline in case fatality means a higher survival rate of infective agents. This facilitates disease transmission and tends to move the economy towards a higher morbidity burden. Thus in the LDC transition, disease prevalence and fertility fall more slowly than in the DC transition. Life expectancy rises more slowly with adult longevity ($LE20$) lagging longevity at birth ($LE0$) by more. The non-infectious disease burden, which follows the infectious disease burden with a lag, initially rises in the LDC

transition and then declines more slowly.

Between periods 0 and 1, when a (and d) decline, more children who became infected in period 0 survive into period 1. They become working adults in period 1 and retire in period 2. This initial improvement in survival probability from period 0 to 1 causes a jump in the unhealthy retired population during period 2. Thus we see a rise in the fraction of the population dying from non-infectious disease in period 2. This is particularly apparent in the LDC transition, since a decline in the case fatality rate allows more unhealthy children born in period 0 to survive to adulthood, in contrast to the DC transition where fewer children become infected in period 0 and thus fewer retired adults are unhealthy. In the LDC scenario, parental investment in children's human capital rises more slowly, and the economy grows more slowly. A persistent income gap opens up purely due to the differential nature of child survival improvements.

7 Conclusion

Understanding the morbidity effect of infectious disease is vital to understanding demographic, epidemiologic and economic changes in the long run. We showed this using a three period overlapping generations model where fertility as well as childhood mortality are endogenous. The theory is able to reconcile conflicting evidence on the effect of child mortality on the fertility transition and population growth. It can also explain low human capital investments in developing countries as well as their disproportionate incidence of non-infectious disease mortality.

A notable implication of our analysis is that not all health improvements are equal. Specifically, child mortality (or life expectancy at birth) is a weak index of a nation's underlying health and health transition. Higher life expectancy at birth does not necessarily lead to faster growth. While lowering sub-Saharan Africa's remaining child mortality burden is important, lowering its morbidity burden is more so.

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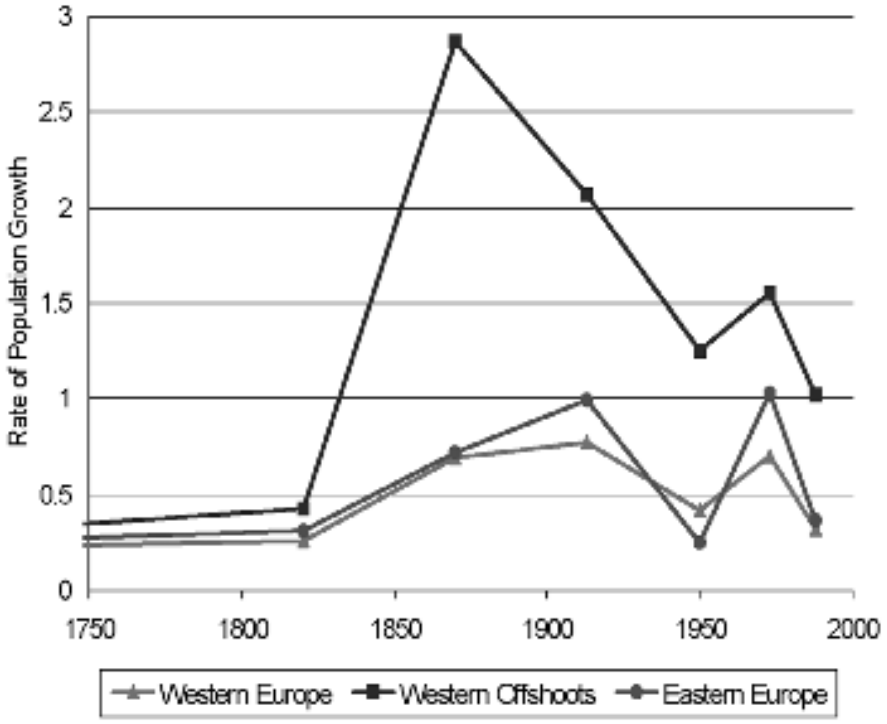


Figure 1 Early Demographic Transitions (Galor, 2005)

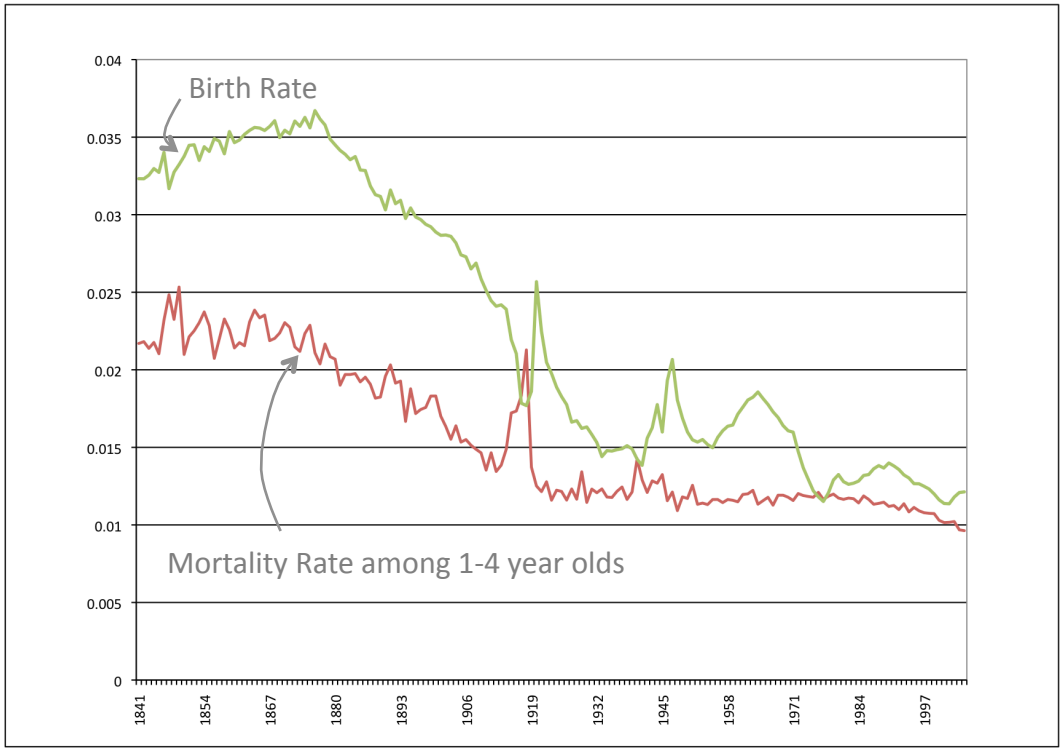


Figure 2 Demographic Transition in England and Wales

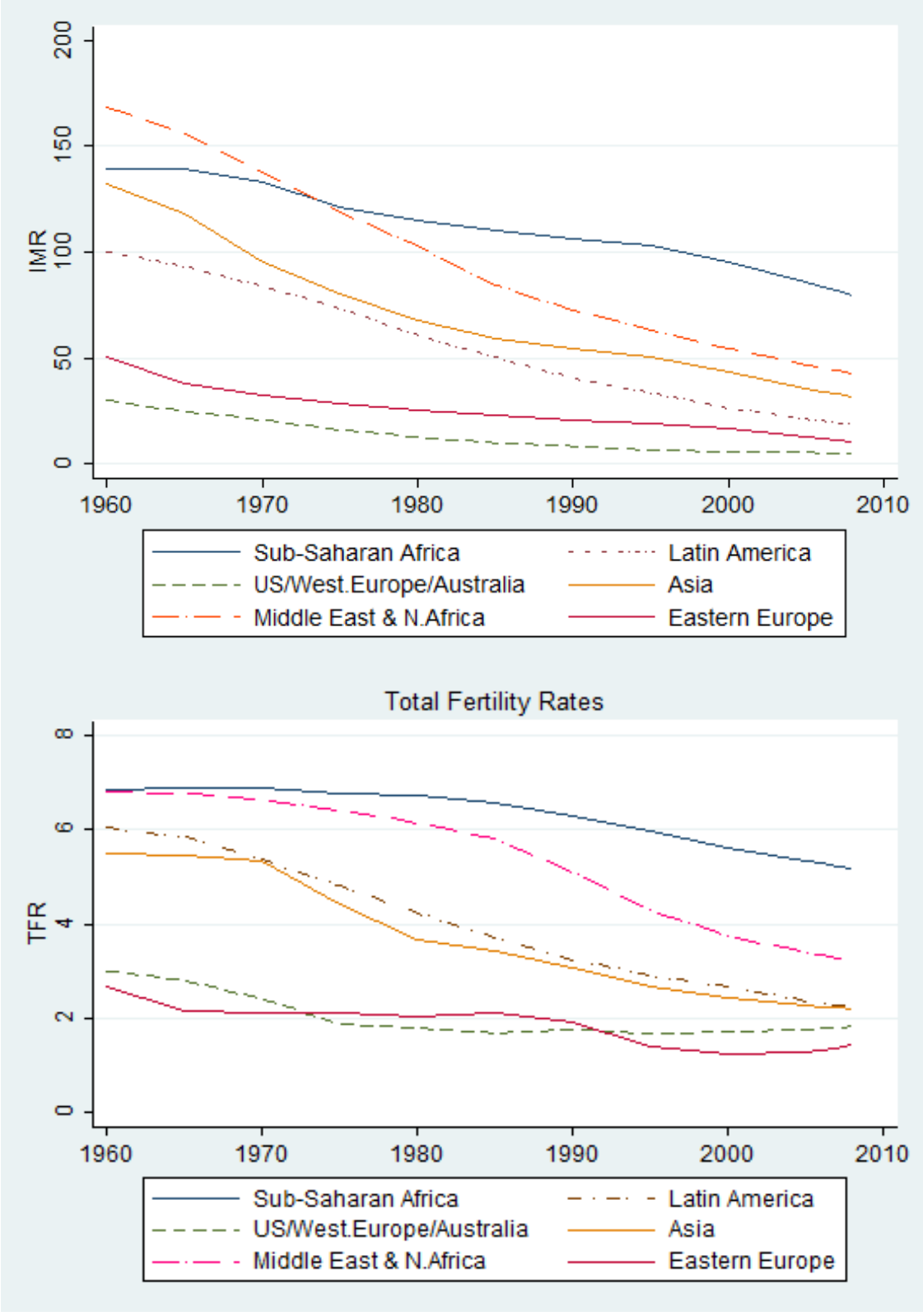


Figure 3 Demographic Transition: Africa versus the Rest

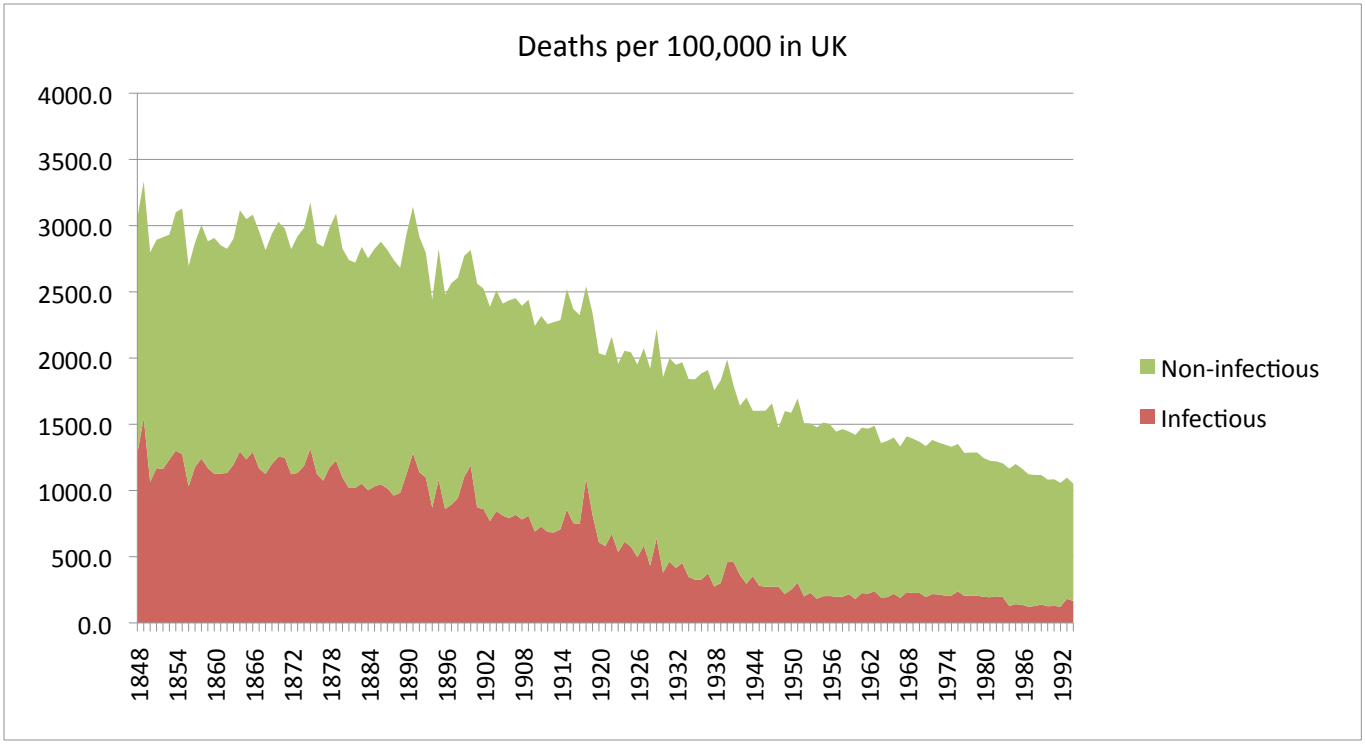


Figure 4 Epidemiologic Transition in England & Wales 1848-1994 (Arora, 2005)

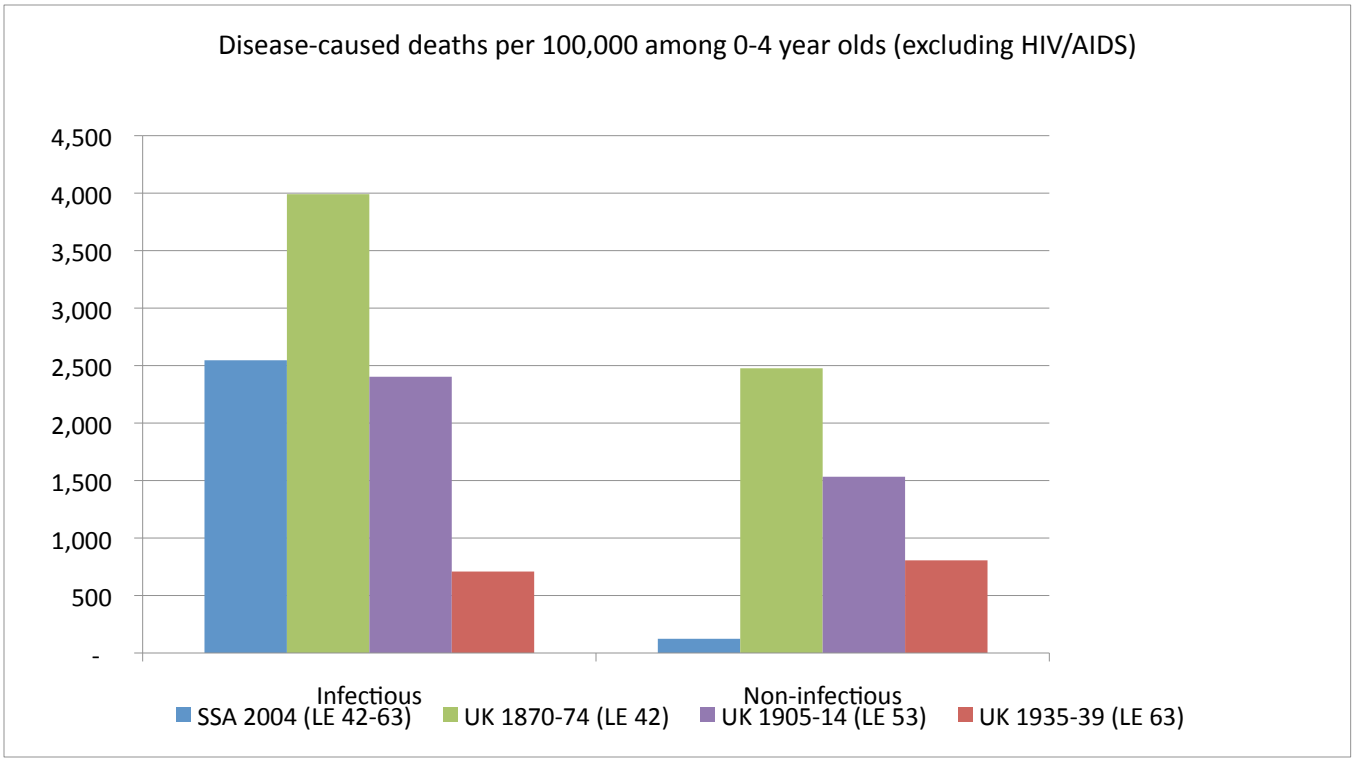
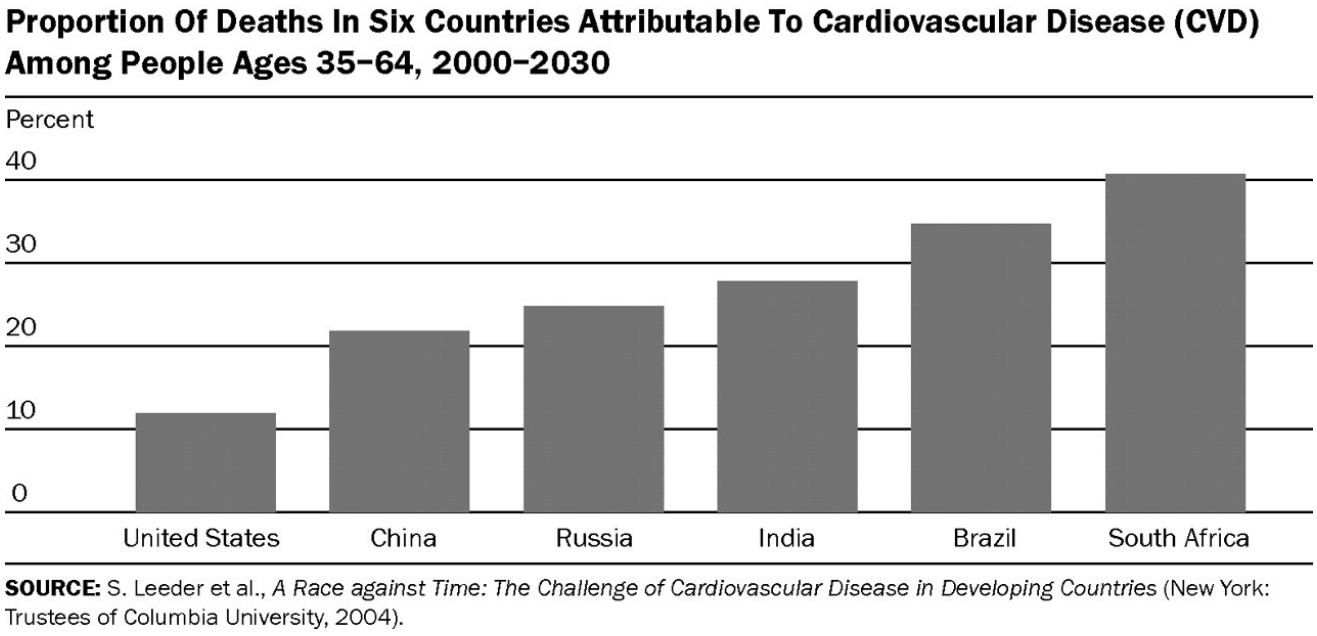


Figure 5 Disease Patterns SSA vs UK



SOURCE: S. Leeder et al., *A Race against Time: The Challenge of Cardiovascular Disease in Developing Countries* (New York: Trustees of Columbia University, 2004).

Figure 6 Burden of Noninfectious Disease in Selected Countries

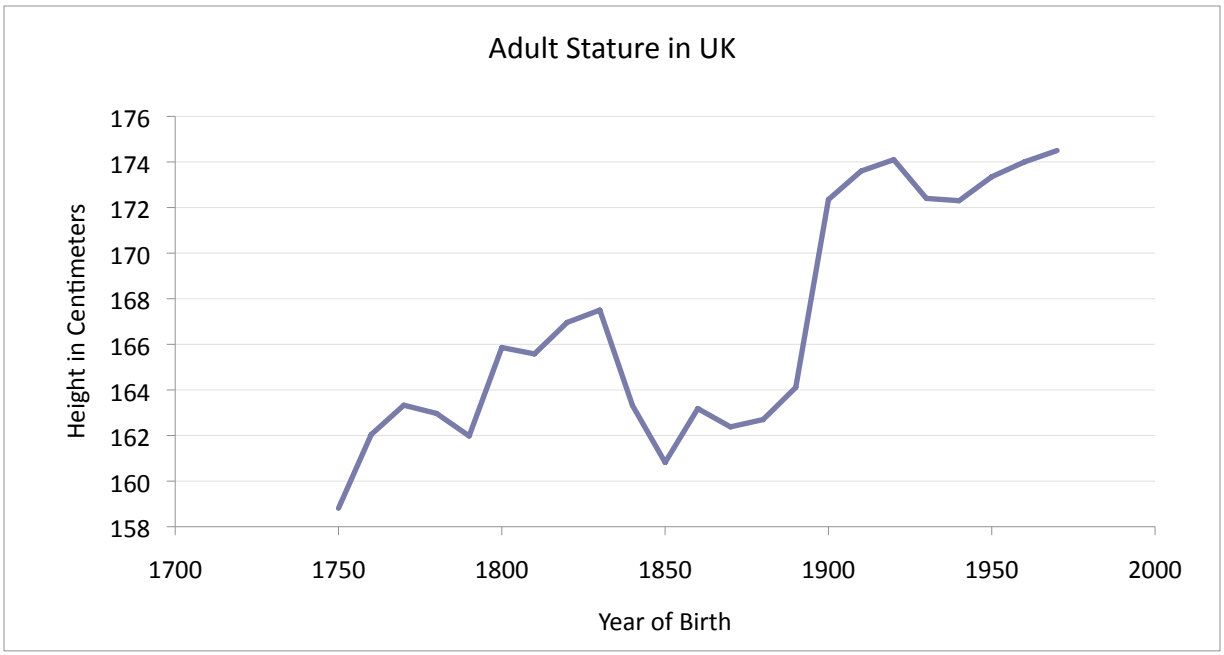


Figure 7 Stature of 18-year Old Males in the UK, 1800-1950 (Arora, 2005)

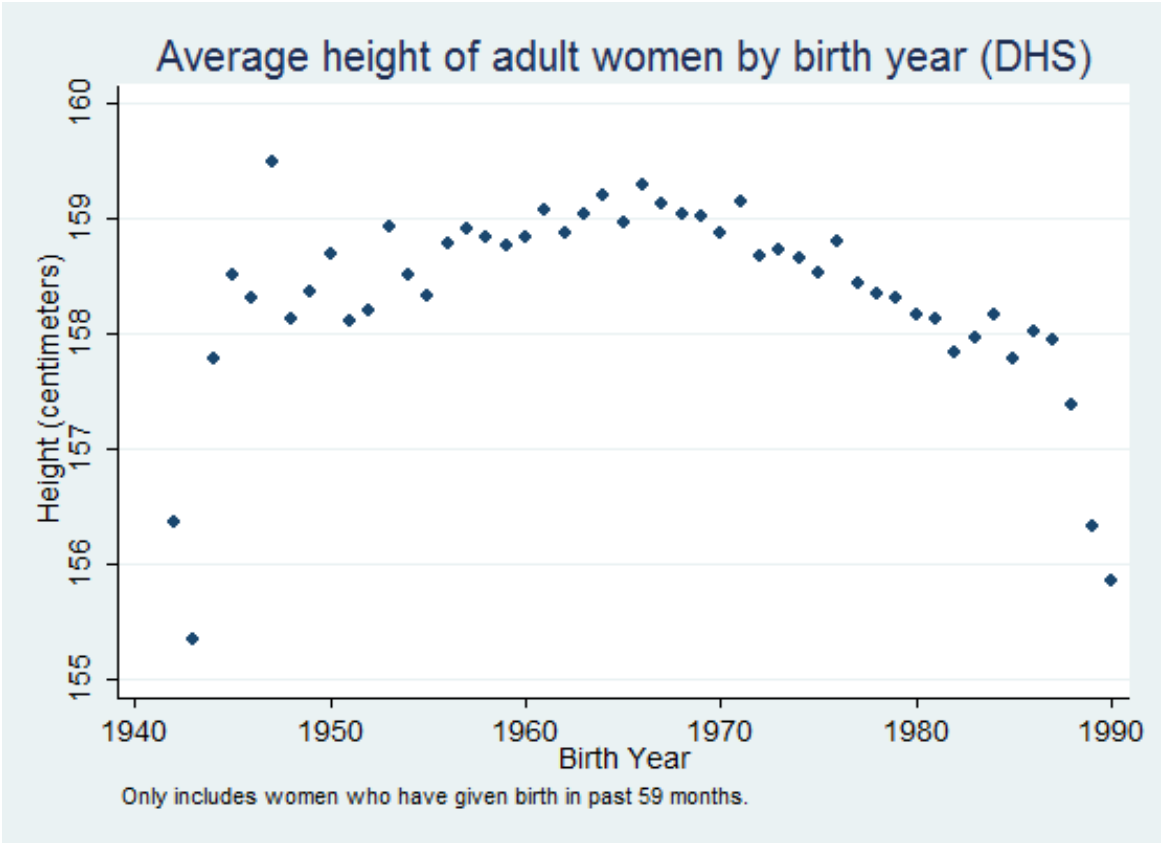


Figure 8 Stature of Adult Women in sub-Saharan Africa (DHS)

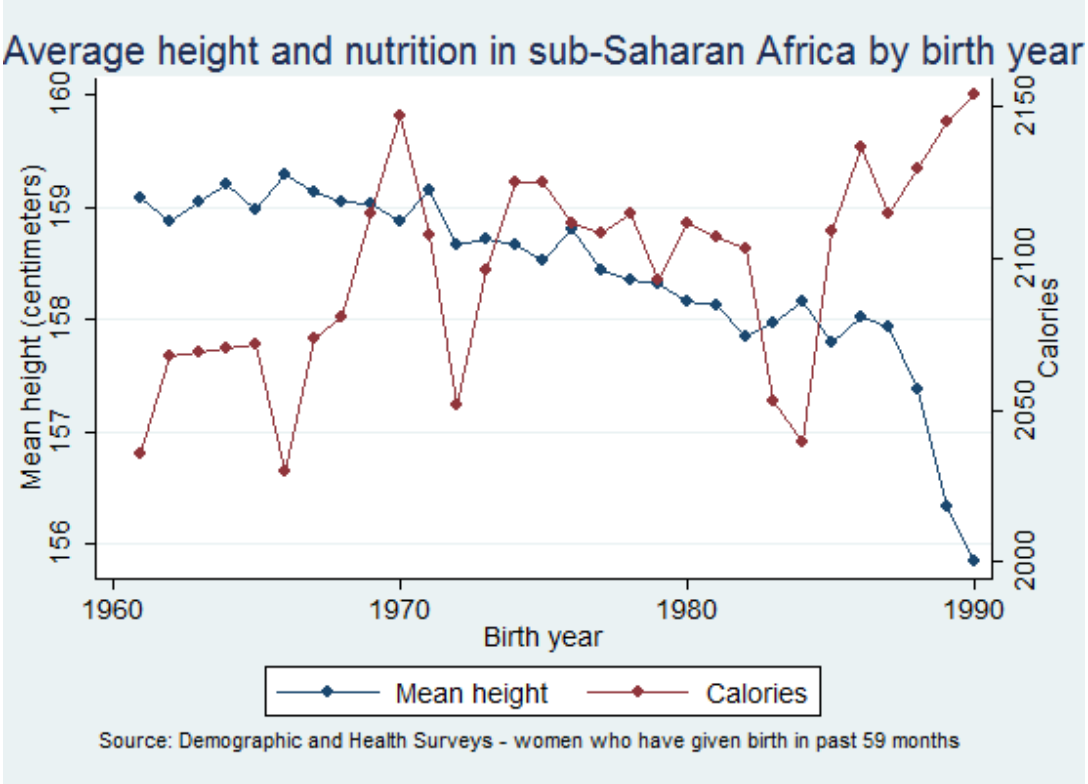


Figure 9 Nutrition versus Average Height

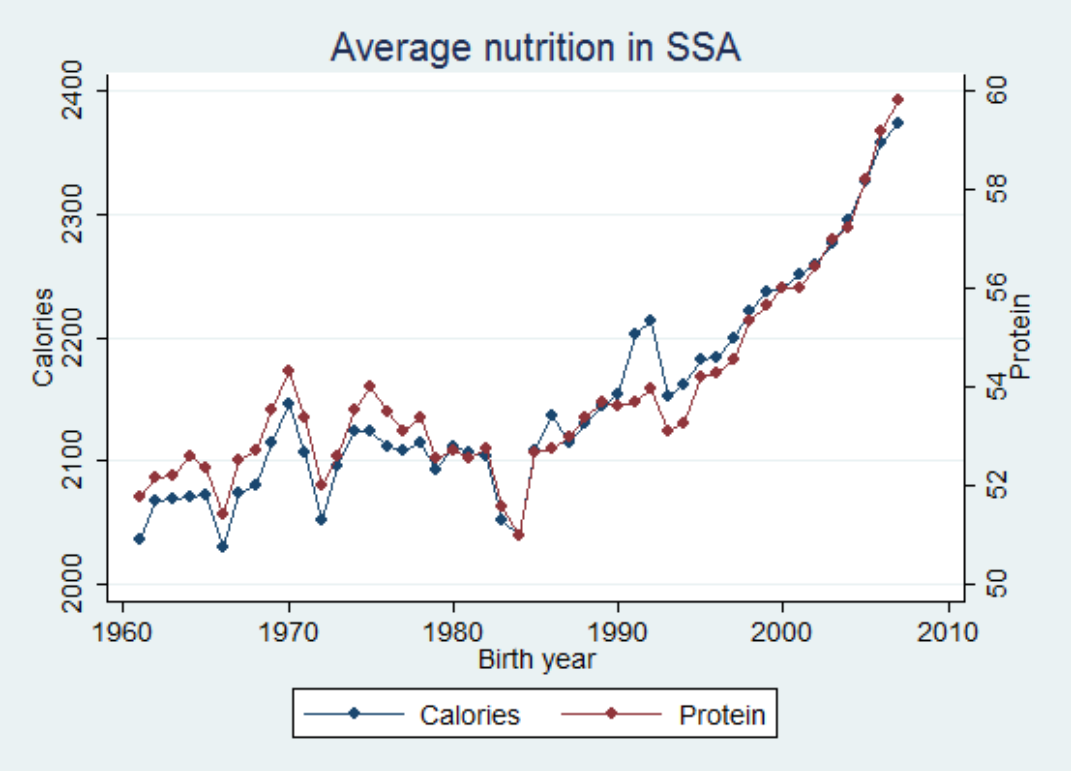


Figure 10 Protein and Calories

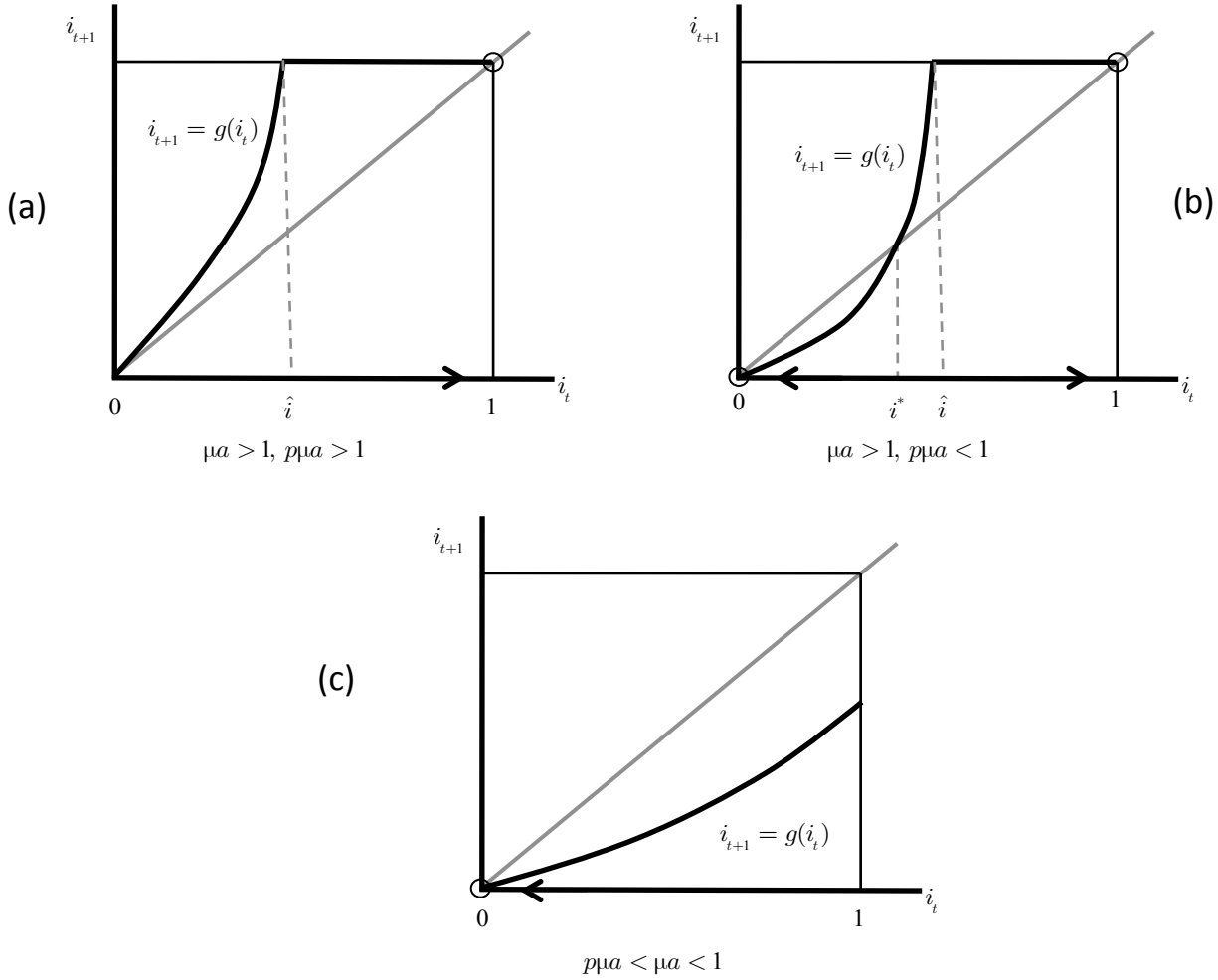


Figure 11 Dynamics of Disease Prevalence

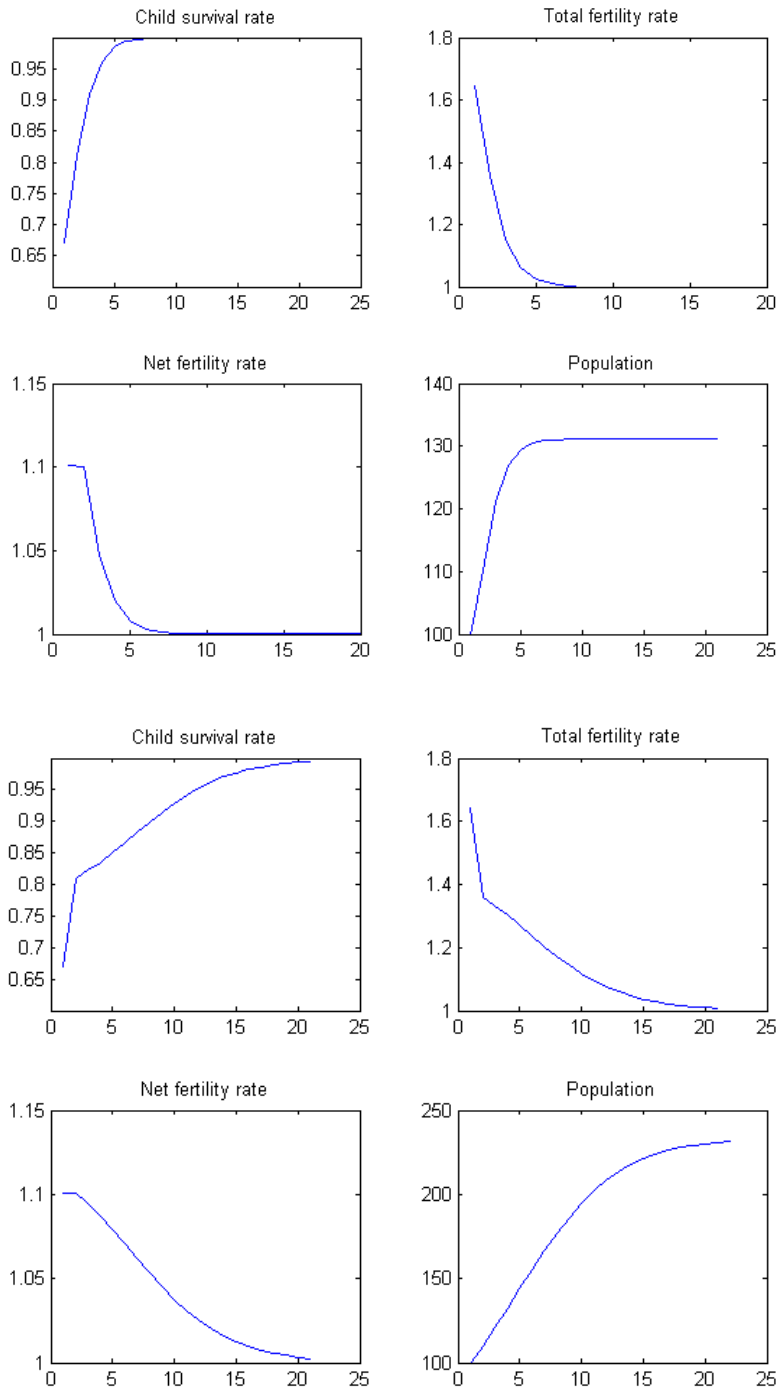


Figure 12 Demographic Transition (top panel DC, bottom LDC)

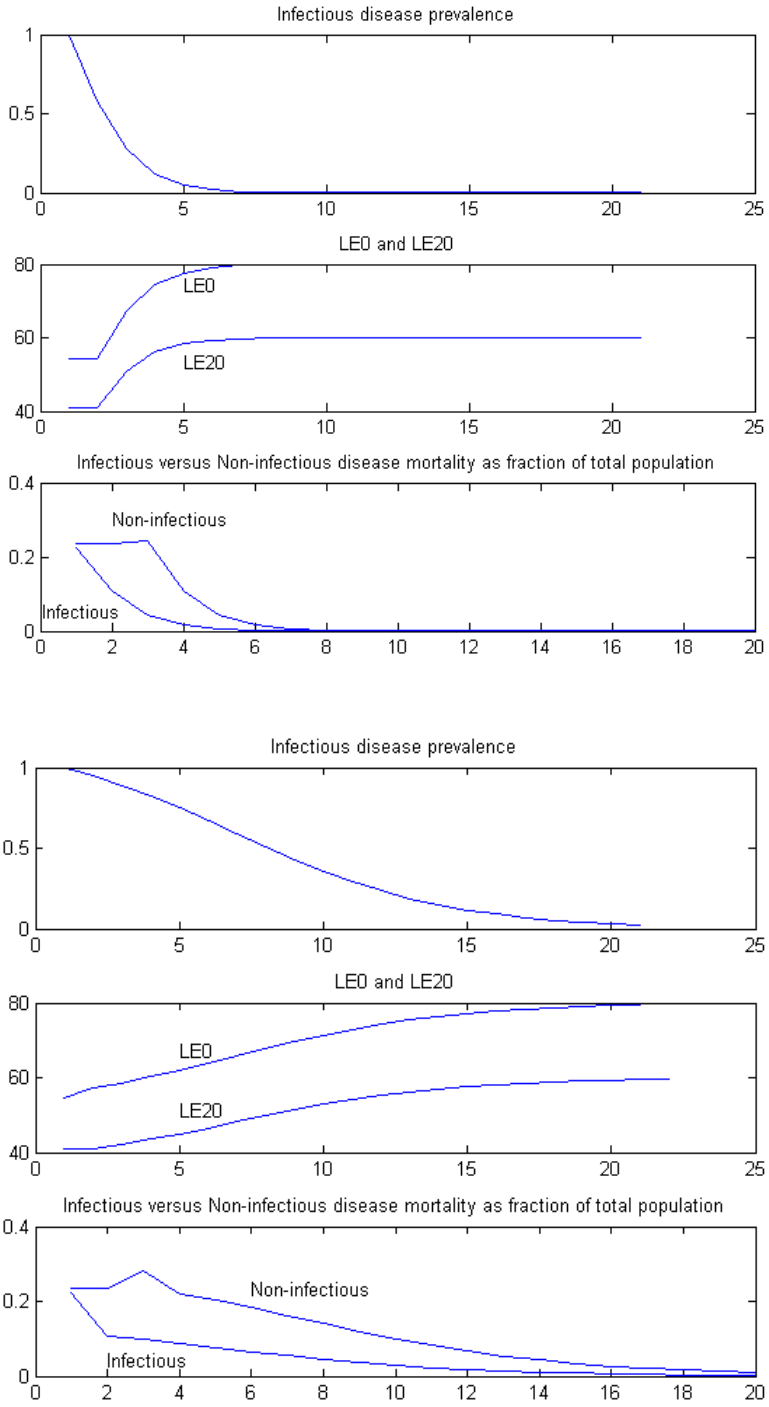


Figure 13 Epidemiologic Transition (top panel DC, bottom LDC)

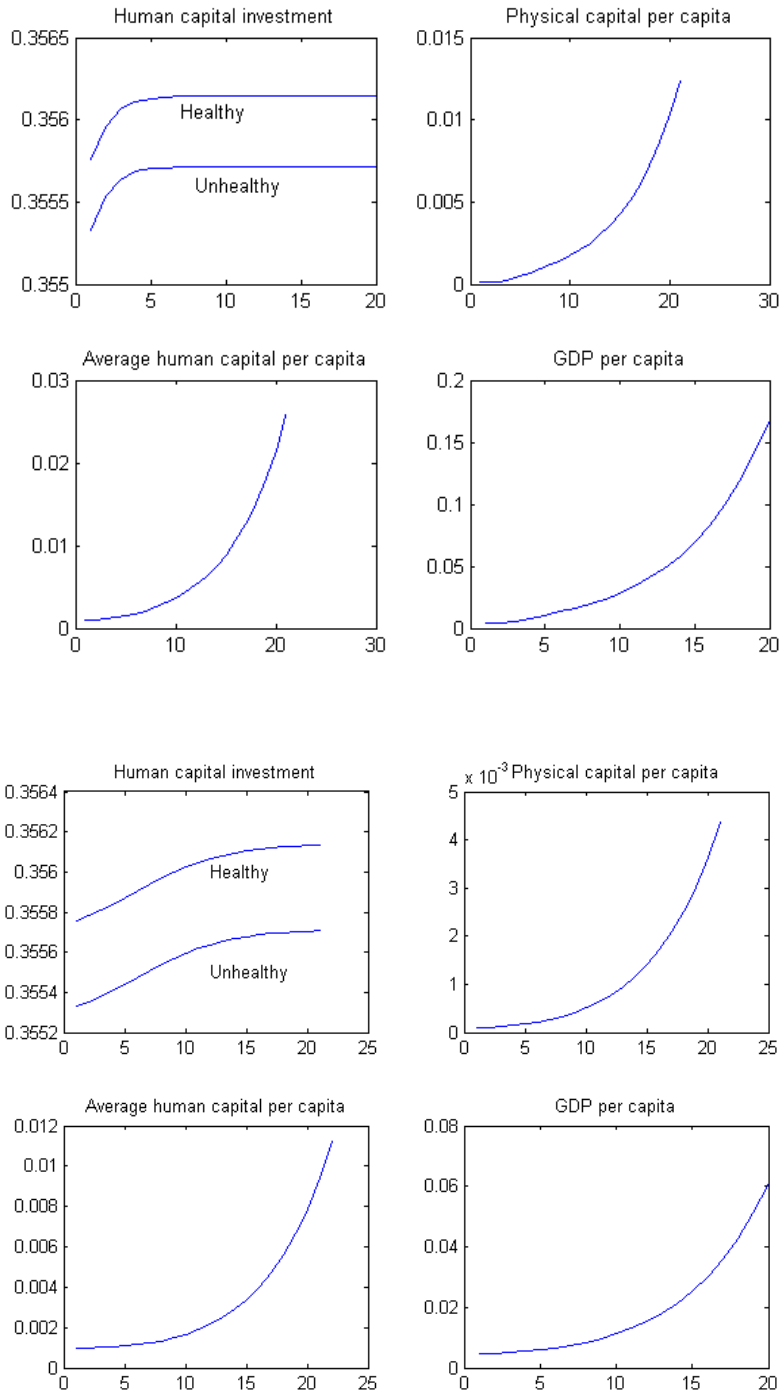


Figure 14 Economic Transition (top panel DC, bottom LDC)