THREE ESSAYS ON DISEASE AND ECONOMIC DEVELOPMENT

by

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This dissertation addresses the high disease burden in developing countries today by examining the role of disease in economic development through its impact on productivity, fertility and human capital investment.

In the second chapter of this dissertation, I model the impact on labor productivity of a change in disease susceptibility that results from intellectual property rights (IPR) reform. I develop a North-South model in which the disease environments differ between the rich and poor countries, and individuals consume innovated health goods to avoid the cost (labor time lost) of getting a disease. Southern welfare is shown to increase with the imposition of IPR protection when health needs in the South differ sufficiently from those in the North, and when health goods are accessible (in terms of adequate health care infrastructure) and effective (in counteracting disease).

In the third chapter of this dissertation, I model the impact of child disease burden on fertility and human capital investment. The fertility response to a decline in child mortality depends on the morbidity effect of the disease, the level of disease burden, and whether prevalence rates or case fatalities decline. Fertility rates follow mortality and morbidity, but since mortality and morbidity do not always move in the same direction, the fertility response may be dampened or non-monotonic. Using a 20-year panel data set on malaria prevalence for 44 countries in sub-Saharan Africa, I find empirical support for the cases defined by the model; changes in malaria prevalence affect fertility more in non-endemic areas, where cases are more severe and more fatal relative to endemic areas.

Historical and biological evidence suggest a link between (infectious) diseases early in life and (non-infectious) diseases later in life. In Chapter IV I model this link using a three-period overlapping

generations model in which childhood disease outcomes affect longevity. Simulations in a general equilibrium framework duplicate the defining characteristics of the epidemiological-demographic transition as it occurred in many industrialized countries: as disease declines parents engage in a quantity-quality tradeoff for children, longevity rises and population declines after an initial jump.

This dissertation includes unpublished co-authored material.

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CHAPTER I

INTRODUCTION

This dissertation addresses the high disease burden in developing countries today, highlighting the role of morbidity. In countries where infectious disease remains widespread, economic development is slow or stagnant.[1] Mortality and morbidity from disease affect labor productivity, fertility and human capital; illness keeps productivity low, high mortality and morbidity keep birth rates high, depleting resources for education, and mortality risk and morbidity reduce the returns to education. Economic growth improves health to some degree, but directly improving health promotes economic growth also.[2] Gallup and Sachs (2001) estimate that in regions heavily burdened by malaria, growth in GDP per person is 1.3% less per year, and a 10% reduction in malaria would raise growth by 0.3%. Many policies aim to reduce the disease burden in developing countries, either directly through public health initiatives such as the World Health Organization's successful campaign to eradicate polio, or less directly, through law reforms, for example. Yet the precise nature of the relationship between disease and economic growth remains unclear.

Chapters II, III and IV of this dissertation examine the relationship between disease and economic development. In Chapter II I focus on the difference in disease environments between rich and poor countries and analyze the impact of patent policy reform on innovation of health goods. In Chapters III and IV I focus on the disease environment itself and how it may evolve from a high to a low disease burden environment; I consider both disease-caused mortality and morbidity and their consequences for fertility, human capital investment and economic growth. By focusing on morbidity, I gain unique insights that contribute to the mortality-fertility literature.[3]

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[1]Studies analyzing the relationship between health and growth include Acemoglu and Johnson (2006), Cole and Neumayer (2006), Kalemli-Ozcan (2002), Lorentzen, McMillan and Wacziarg (2005), Qureshi and Mohyuddin (2006) and Shastry and Weil (2003).

[2]See Arora (2003) and Soares (2005) for evidence.

[3]See, for example, Barro and Becker (1988, 1989), Boldrin and Jones (2002), Boucekkine et al. (2008), Doepke (2005),

In Chapter II I show the influence of reforming intellectual property rights (IPR) on innovation of medicine geared at developing country needs. Many studies find enforcement of IPR in developing countries to be welfare reducing. However when disease environments differ between developed and developing countries, free-riding by the latter on the innovation incentives of industrialized countries deters innovation of medicine for diseases, such as malaria, that are a large burden only on developing countries. The few studies that do account for different needs in developed and developing countries exogenously impose demand for innovated goods, thus ignoring budget constraints and consumption trade-offs faced by consumers. I model this demand for health goods as a function of a country's particular disease environment, where disease imposes a cost to agents in terms of a reduction in available labor supply.

The analysis in Chapter II evokes the question of how the disease environment might evolve in response to IPR reform. A shock to the infectious disease burden, such as a new antibiotic or vaccine, could trigger an epidemiological transition whereby the disease burden declines considerably. Today's industrialized countries have completed such a transition, but disease in many developing countries, particularly infectious disease, remains both prevalent and virulent. In Chapters III and IV I examine the relationship between health improvements and economic development.

Chapter III highlights the link between disease morbidity and returns to human capital investment. I develop a model that includes child survival risk and, unlike previous studies, distinguishes between the roles of infection and case fatality rates in parental decisions regarding fertility and human capital investment.[4] A changing disease burden impacts economic growth through changes in both mortality and morbidity. If a new treatment becomes available, fewer people die but they may be less healthy as a consequence of the disease. All else equal, the treatment may actually diminish the health of the population. On the other hand, a new vaccine would prevent infections, leading to a healthier population. Existing mortality-fertility studies find an unambiguously positive relationship between child mortality and fertility, but in distinguishing between disease prevalence and case fatality rates, response of fertility to child mortality becomes non-linear. The response depends on the net effect of the precautionary motive (parents have more children if the uncertainty regarding their health and survival is greater) and the average cost of children (children become more expensive as morbidity declines but less expensive as mortality

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Jones and Schoonbroodt (2007), Kalemli-Ozcan (2003, 2008), Tamura (2006) and Young (2005).

[4]Birchenall (2007) does differentiate between disease prevalence and case fatality rates.

declines). Using malaria prevalence panel data for 1985-2005 for 44 countries in sub-Saharan Africa, I confirm that birth rates decline less with a decline in malaria prevalence in areas where malaria is endemic, i.e. where reductions in prevalence reduce mortality and morbidity less per case averted relative to non-endemic malarial regions.

In Chapter IV childhood disease and adult longevity are linked. The epidemiological transition, as it occurred in many countries, is characterized by a decline in infectious disease, mostly among the very young, followed approximately one generation later by a decline in non-infectious disease, those afflicting mostly the elderly (Arora 2005). Since individuals who suffer from an infectious disease in childhood are more susceptible to other diseases later in life, this timing suggests children who avoided infectious diseases grew up better able to resist non-infectious diseases in adulthood.[5] I develop a three-period, overlapping generations model in which longevity depends on disease realizations in childhood; experiencing disease in childhood depreciates health capital, thereby reducing longevity by increasing the risk of premature mortality. The returns to human capital investment are lower for unhealthy children. When disease becomes less prevalent, fertility declines as parents engage in a quantity-quality tradeoff for children; the population becomes healthier and less likely to die prematurely, and savings and thus economic growth improve.

In Chapter II I consider the impact of disease morbidity on productivity and the role of IPR policy. In Chapters III and IV, I maintain the connection between disease and productivity and also consider how changes in the disease environment affect parental behavior and the resulting impact on economic development. The results of this dissertation contribute to the current literature analyzing the mechanisms through which the epidemiological and demographic transitions are related and provides some policy recommendations (Arora 2005, Morand 2004). If reducing childhood disease leads to a quantity-quality tradeoff in children, targeting the disease burden can stimulate economic development. However, contracting a curable disease has different consequences for fertility and human capital than contracting a fatal one, since surviving a disease may predispose a person to other disease later in life and lower the returns to quality investment. When public health initiatives have a weak effect on fertility, education subsidies, for example, should complement them. As an indirect form of health policy, in Chapter II cases are illustrated for which pharmaceutical patent reform is welfare enhancing for developing countries.

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[5]For example, bronchitis, pneumonia and whooping cough before age 5 are linked to diminished respiratory function at ages 59-70 (Barker 1991, 1994). See Chapter III for more biological evidence as well as Arora (2003) and Soares (2005).

In Chapter II morbidity, via labor productivity, plays a role in the welfare analysis of IPR reform by affecting demand for medicine. In Chapters III and IV, in addition to labor productivity, morbidity affects fertility and human capital investment by affecting longevity and the returns to human capital investment. To my knowledge there is no previous theoretical study that explores the underlying synergy between disease early and late in life, as in Chapter IV, a relationship acknowledged as important in the medical literature.

Chapter III includes material co-authored with Shankha Chakraborty.

CHAPTER II

APPROPRIATE MEDICAL INVENTIONS

II.1 Introduction

This chapter studies intellectual property rights (IPR) with respect to health innovations. In many developing countries, virulent diseases occur which are less common in developed countries. Underdevelopment prevents successful management of these diseases.[1] Developed countries present health good innovators with an effectively much larger market, both through a higher ability to pay and also through typically stronger IPR protection. Innovators are thus able to recuperate their high fixed costs more easily and quickly in these markets, and gear their research and development (R&D) resources accordingly. Only 13 of 12,000 new drugs introduced globally between 1975-1997 specifically targeted tropical diseases, which disproportionately affect developing countries.[2] Access to health goods differs markedly in importance from access to other patented goods, such as movies and computer software; health goods help fulfill the fundamental need for good health, which may directly determine survival.

In 1995 the World Trade Organization (WTO) enacted the Agreement on Trade-Related Aspects of Intellectual Property Rights (TRIPS) with the intent of achieving a near-global minimum IPR standard, and developing countries were allowed a 10-year transition period before fully adhering to its standards. As these countries come into compliance, much debate surrounds the welfare

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[1] According to the World Health Organization's (WHO) Global Burden of Disease estimates, in 2002 mortality from infectious diseases was 32 times higher in high-mortality developing countries relative to developed countries, and 4 times as high in low-mortality developing countries. Tropical-cluster diseases (Trypanosomiasis, Chagas disease, Schistosomiasis, Leishmaniasis, Lymphatic filariasis, Onchocerciasis) killed 151 individuals in developed countries and 736 and 117, respectively, times as many in high-and low-mortality developing countries. Sub-Saharan Africa is host to over 64% of all people living with HIV, and also accounts for 85-90% of the over one million malaria fatalities occurring annually worldwide.

[2] Reflecting a possible incentive response to TRIPS, the WTO treaty on trade-related aspects of IPR, the London School of Economics and Political Science's Pharmaceutical R&D Policy Project has determined that 8 or 9 new drugs for neglected diseases are expected by 2010.

consequences for them. This paper contributes to the debate by addressing the impact of IPR on the unique needs of developing countries where individuals often cannot afford medicines unless they are imitated and hence available at low cost.

Proponents of strengthening IPR in developing countries argue that, by securing returns to innovative activity, stronger IPR should increase introduction of new medicines, ultimately improving health and thereby promoting economic development. However, whether this effect would more than compensate for the accompanying increase in prices remains a contentious issue. In the older North-South models of IPR and innovation, such as Deardorff (1992) and Helpman (1993), differences in needs across regions are not accounted for, and strengthening IPR harms the South.[3] Accounting for different needs introduces a channel by which the South may gain from strengthening its IPR, through innovations geared at Southern needs. A small number of studies do acknowledge the role of different needs in IPR reform welfare analysis. See Section II.2.

The differences in disease burden between developed and developing countries cannot be explained solely by a lack of resources. Africa hosts the most virulent strain of malaria, *plasmodium falciparum*, and its tropical climate does not allow for winter frosts to weaken mosquito populations. The WHO reports that in countries with a high malaria burden, the disease accounts for as much as 40% of public health expenditure, 30-50% of inpatient admissions, and up to 60% of outpatient visits, and as resistance to antimalarial drugs spreads, demand for new treatments or a vaccine is rising.[4] While the initial denial of AIDS among policy makers in Africa contributed to the current severity of the epidemic there, unprotected sex with an HIV+ partner is 4 to 5 times more likely to result in infection in Sub-Saharan Africa than in the United States (Meredith 2005, Chakraborty *et al.* 2008). This may be due to the high prevalence of other sexually transmitted diseases there, by which open sores caused by other sexually transmitted infections facilitate the transmission of HIV (Oster 2005, Sangani *et al.* 2006). Furthermore, the interaction between HIV and malaria increases mortality rates. A recent study estimates that the interaction between malaria and HIV may have been

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[3]Branstetter et al. (2007) extend Helpman (1993) by endogenizing both imitation and FDI rates to Southern IPR policy. Both industrial development in the South and global innovation may increase when IPR is strengthened in the South, through the reallocation of labor resources which occurs as the North specializes in innovation and the South in manufacturing. When the unit labor requirement for Southern imitation rises in Branstetter *et al.* (2007), many goods become cheaper as Northern firms shift production to the South where production costs are lower, but goods that would have been imitated under a more lenient IPR regime become more expensive. This does not explicitly enter the welfare analysis, although a reduction in the relative wage gap between the North and the South presumably implies increased purchasing power of Southern consumers.

[4]WHO Media Center, http://www.who.int/mediacentre/factsheets/fs094/en/ (May 2007)

responsible for 8,500 excess HIV infections and 980,000 excess malaria episodes in Kenya (Abu-Raddad *et al.* 2006).

Table II.1: Disease burden (DALY) correlations across regions

	SSA	India	China	OAI	LAC	ME/NA	FSE	EME
Sub-Saharan Africa	1							
India	0.725	1						
China	0.168	0.460	1					
Other Asia & Islands	0.672	0.954	0.516	1				
Latin America & Caribbean	0.458	0.854	0.644	0.904	1			
Middle East & North Africa	0.716	0.985	0.444	0.955	0.862	1		
Former Socialist Economies	0.115	0.562	0.625	0.607	0.757	0.570	1	
Established Market Economies	0.096	0.521	0.769	0.585	0.791	0.518	0.921	1

Disease-adjusted life years (DALY) is a disease burden measure that accounts for both mortality and morbidity. DALY data is for 1990 and 2000 and covers 55 diseases. The eight regions above are the classifications of the WHO.

The WHO compiles regional DALY (disability-adjusted life years) data, a measure of disease burden that accounts for mortality and morbidity, measuring years of healthy life lost due to a specific disease. DALY data for 55 diseases for 1990 and 2000 exhibits a very low correlation between disease burden for high-income countries and sub-Saharan Africa, as shown in Table II.1; disease profiles vary greatly between rich and poor countries. As a proxy for innovation, the number of patents concerning the aforementioned 55 diseases for 1990 and 2000 was constructed via disease-specific keyword searches of the US Patent and Trademark online database (USPTO).[5] In Table II.2, the correlation between innovation and disease burden is quite high for developed countries and negligible for Sub-Saharan Africa, which suggests innovators respond to market size rather than just health needs. The correlation is also high for the formerly socialist economies, who in 1990 had the lowest IPR but whose health needs correlate highly with the established market economies (see Table II.1). In terms of attaining the most needed health goods, this suggests that free-riding on the innovation incentives of wealthy countries is a poor strategy for countries with unique health needs.

I continue with a review of the existing IPR reform literature that takes into account different preferences across development regions. In Section II.3 the model is presented and solved for consumption and production in both the North and the South under alternative IPR regimes; the ranges of innovations in the regimes are compared. In Section II.4, we analyze Southern welfare

[5] I would like to thank Margaret MacLeod for sharing with me some disease-specific keywords by which to search the USPTO.

Table II.2: Correlation of drug patents with regional disease burden (DALY)

Sub-Saharan Africa	0.015
India	0.231
China	0.222
Other Asia & Islands	0.242
Latin America & Caribbean	0.385
Middle East & North Africa	0.252
Former Socialist Economies	0.460
Established Market Economies	0.474

Patent data for 1990 and 2000 was gathered from the USPTO for 55 diseases using disease-specific keyword searches of all pharmaceutical and pesticide innovations in the database.

changes. Simulation results are presented, since an analytical solution for the condition under which the South benefits from enforcing IPR is beyond our present scope. The findings are discussed in the concluding Section II.5, and possible extensions to the model are indicated.

II.2 A Review of the Literature

A few studies acknowledge that needs differ across countries. The net impact of price and product variety responses to strengthening IPR in the South depends on the channels through which reforms may affect welfare, including the incentives to free-ride as determined by cooperative efforts or regional differences in needs, technological abilities, and resources limitations. In Diwan and Rodrik (1991), whose structure this paper partly adopts, the South skews Northern innovation in favor of its own needs via stronger IPR. As the gap in needs widens, free-riding on Northern innovation incentives becomes less appealing for the South, since these differences foster competition for limited R&D resources.[6]

Grinols and Lin (2006) model this effect by allowing for two, distinct R&D sectors in the North, one focusing on Southern-specific innovations and the other on globally-demanded goods. The North determines the rate at which the set of globally-consumed goods are imitated, while the South sets that rate for those goods only consumed in the South, and the patent life for each type of good is the inverse of its respective hazard rate. For certain goods the cost to the South of protecting patents far outweighs the benefit, but for others it does not. For example, a country facing an AIDS epidemic

[6]The price a Northern firm charges a Southern consumer is the limiting price that keeps a potential Southern imitator out of the market; this price reflects IPR strength in the South and is identical across innovated goods. In my model, innovators charge the monopoly price, but if IPR is not enforced, imitators charge marginal cost.

will find it more enticing to ignore IPR for HIV/AIDS medications but may still enforce patents for less consequential goods. Starting from equal IPR levels in both regions, a uniform reduction of IPR unambiguously increases Southern welfare. However, if the Southern-specific good is sufficiently important to consumers, loosening IPR only in the South causes a reduction in innovation that dominates the improvement in the South's terms of trade, reducing Southern welfare. My model suggests that drugs treating diseases primarily affecting the South are those whose IPR the South has more incentive to enforce. In these models, Northern consumers would benefit from relatively weaker IPR in the South, but from a global welfare perspective it is not clear whether IPR levels should be weaker or stronger in the South than in the North.[7]

Yang (1998) employs a game theoretic approach in a world where a single Northern country creates new technologies useful only in the multiple Southern countries, and each Southern country chooses what level of royalties it pays to the North in exchange for new technologies. Since Southern countries have an incentive to free-ride on each other, IPR is too low for the region, and the entire region gains from cooperation. Yang's results support a policy such as TRIPS, which mandates that all WTO members adhere to its IPR standards, effectively diminishing the potential group of free-riders. In my model, the number of innovated varieties increases when both the North and South protect IPR, so both benefit from Southern IPR reform, and this result extends to including multiple Southern countries in the model.

In Chen and Puttitanun (2002), where the level of Southern IPR is endogenous, the only players are a Northern innovating firm producing in the South and a Southern innovating firm, and each is paired with its own Southern imitator. Because it can also engage in innovation, the South has an incentive to reform IPR beyond merely to gain access to Northern technologies. For very poor countries, an increase in technological capabilities increases the efficiency of imitating Northern technologies more than it does the efficiency of innovation, so countries at very low levels of development benefit from weaker IPR. Further development, however, increases the efficiency of innovation relatively more, so the South prefers to then strengthen IPR. The government sets IPR to balance the trade-off between loss in consumer surplus that results from less competition for foreign innovators, and hence higher prices for imported goods, against the net benefit from quality improvement by local innovators. Optimal IPR setting by the government generates the U-shaped

[7]Simulations by Diwan and Rodrik support relatively weak IPR in the South for realistic parameter values.

relationship between IPR and development. Price reductions from imitation outweigh the loss in innovations for very poor countries, but this reverses for more developed countries. These results suggest that the least developed countries should free-ride on innovation incentives provided by wealthier nations until their technological abilities rise to a level at which they can actually benefit from stronger IPR, a result supported by my model.[8]

Both Chen and Puttitanun (2002) and Yang (1998) omit Northern consumers, and in both Diwan and Rodrik (1991) and Yang (1998) effectively no productive activity occurs in the South, excluding from the analysis effects on industrial development. Diwan and Rodrik (1991) and Chen and Puttitanun (2002) model demand as exogenous. These assumptions neither allow for resource constraints faced by individuals to be modeled, nor for optimizing trade-offs to occur between consumption of various goods, nor for Northern welfare to be analyzed.

In the model developed here, production of goods takes place in both regions, although innovation occurs only in the North. Health needs differ between the North and the South because of differing disease environments, and demand for health goods is partially endogenized through their impact on labor supply; demand for each variety of health good depends on the prevalence of the associated disease. Relative to when only the North enforces IPR, the number of innovated varieties increases when both the North and the South protect IPR, and so each can benefit. If needs differ sufficiently and health goods are effective and accessible in the South, then the imposition of IPR protection in the South stimulates innovation for health goods most needed in the South and thus Southern welfare increases. As in Chen and Puttitanun (2005), the least developed countries will free-ride on the innovation incentives of more developed countries, since even with strong IPR, the market demand they represent is too small to stimulate sufficient innovation. However, since Northern welfare unambiguously benefits from Southern IPR reform, a Pareto-improving reallocation from North to South is possible.

II.3 The Model

Suffering from a disease generates economic costs in terms of reduced ability to work. More prevalent diseases generate higher demand for the relevant treatment. Diseases, indexed by θ, are acquired with varying probabilities as determined by exogenous continuous probability density

[8]The United Nations currently designates 50 countries as least-developed (LDCs), 32 of which are WTO members.

functions $g^i(\theta)$ in regions $i = N, S$. Certain diseases are more prevalent in the South than the North: $g^S(\theta)$ is skewed to the right relative to $g^N(\theta)$.

Representative agents in the North and South each consume a variety of health goods and also non-innovated consumption goods, c. Let x_θ be consumption of the health good that treats disease θ. Each person devotes one unit of time to labor, minus the "time" taken up by each disease; being ill affects labor productivity. The unit wage rates are w^i, where $w^N > w^S$: labor in the North is more productive than labor in the South. The consumption of relevant health goods counteracts the loss in labor resources to some degree; the expected wage in region i is

$$\left(1 - \int_0^\infty g^i(\theta)[1 - \alpha^i(x_\theta^i)^\lambda]d\theta\right) w^i$$

where $\lambda \in (0,1)$ captures the effectiveness of the medicine, and α $(i = N, S)$ is such that the condition $(1 - \alpha^i(x_\theta^i)^\lambda) \geq 0$ holds: consumption of health goods cannot result in more than one unit of labor time.[9]

If we think of θ as capturing disease severity, then the South experiences more severe diseases than the North in terms of impact on labor supply. Infectious diseases target all age groups, often resulting in premature mortality during the most productive years of adulthood; these play a much greater role in developing versus developed countries. Non-infectious diseases dominate the disease environment of developed countries and tend to cause premature mortality later in life.

The representative consumer in region i chooses consumption of non-innovated goods, c, which enter utility directly, and of innovated health goods, x_θ, $\theta \in [\underline{\theta}, \overline{\theta}]$, which enter utility via the budget constraint. In addition to wage income, the Northern representative agent receives a portion Π/L^N of the profits Π accruing to Northern innovators, where L^N is the population in the North.[10] Normalizing the price of consumption goods to 1 in both regions, the utility-maximizing problem is.

$$\begin{aligned} &Max\ U^N = u(c^N) \\ &subject\ to \quad c^N + \int_{\underline{\theta}}^{\overline{\theta}} p_x^N x_\theta^N d\theta = \left(1 - \int_0^\infty g^N(\theta)(1 - \alpha^N(x_\theta^N)^\lambda)\, d\theta\right) w^N + \frac{\prod}{L^N} \end{aligned}$$

[9] For simplicity, λ is neither disease- nor region-specific in this model. A higher λ implies a more effective product.

[10] $\Pi = \int_{\underline{\theta}}^{\overline{\theta}} \pi(\theta)d\theta - (\overline{\theta} - \underline{\theta})f$, where $\pi(\theta)$ is the profit from production of x_θ and f is the fixed cost of innovation.

for the Northern representative agent, and

$$Max\ U^S = u(c^S)$$
$$subject\ to \quad c^S + \int_{\underline{\theta}}^{\overline{\theta}} (1+k) p_x^S x_\theta^S d\theta = \left(1 - \int_0^\infty g^S(\theta)(1-\alpha^S (x_\theta^S)^\lambda)\ d\theta\right) w^S$$

for the Southern representative agent, where the common utility function is increasing and concave. Prices for health goods are region- but not variety-specific, p_x^N in the North and p_x^S in the South, but Southern consumers face an additional cost, k, of acquiring health goods due, for example, to infrastructure problems or the need to bribe officials for access to medicine.[11] We refer to this additional cost as the "inefficiency tax".[12]

Substituting the budget constraints into the utility function[13] and optimizing, the optimal demands x_θ^N and x_θ^S satisfy

$$p_x^N = \lambda \alpha^N w^N g^N(\theta)(x_\theta^N)^{\lambda-1} \quad and \quad p_x^S = \frac{\lambda \alpha^S w^S g^S(\theta)(x_\theta^S)^{\lambda-1}}{1+k} \tag{1}$$

The model thus far accounts for different needs across regions. By incorporating the production sector, we determine prices, demand and innovation outcomes from market conditions.

Innovators of x_θ exist only in the North, must incur a fixed cost of innovation in order to produce a particular health good, and are monopolistic producers. While patents are perfectly protected in the North, they may or may not be enforced in the South. If patents are enforced in the South, firms collect profits in both regions. If they are not enforced in the South, firms collect only profits in the North, since imitators in the South automatically copy a new technology and price the innovator out of the market by charging the Northern marginal cost w^N $(> w^S)$. These two situations are referred to as regimes NS and N respectively. In regime N, firms choose how much to

[11] The US, for instance, has more than 83 times as many health care workers per 1,000 people as Malawi (WHO Statistical Information Systems 2004, complied by Actionaid International). Transparency International Kenya estimates that public hospitals in Kenya extorted, on average, 110 Kenyan schillings per person per month in 2001, about 5% of income.

[12] Although the inefficiency tax is modeled here as an ad valorem tax on consumption of health goods, the results would not differ importantly for a per unit inefficiency tax.

[13] In particular, $U^S = L^S u\left(\left[\int_{\underline{\theta}}^{\overline{\theta}} g^S(\theta)\alpha^S (x_\theta^S)^\lambda d\theta\right] w^S - \int_{\underline{\theta}}^{\overline{\theta}} (1+k) p_x^S x_\theta^S d\theta\right)$.

produce for the Northern market only:

$$Max\ \pi_N(\theta) = (p_x^N - mc)L^N x_\theta^N = (\lambda\alpha^N g^N(\theta)(x_\theta^N)^{\lambda-1} - 1)w^N L^N x_\theta^N$$

$$\Longrightarrow x_\theta^N = \left(\lambda^2\alpha^N g^N(\theta)\right)^{\frac{1}{1-\lambda}} \quad and \quad p_x^N = \frac{w^N}{\lambda} \quad \forall\ \theta \tag{2}$$

Southern consumers demand the same goods but they buy imitations at unit price w^N. Therefore, from (1),

$$p_x^S = w^N \quad and \quad x_\theta^S = \left(\frac{\lambda\alpha^S g^S(\theta)w^S}{(1+k)w^N}\right)^{\frac{1}{1-\lambda}} \tag{3}$$

In regime NS, patents are enforced in the South as well as the North. Northern firms supply both markets and treat them as separate, solving the following profit-maximization problem:

$$Max\ \pi_{NS}(\theta) = (p_x^N - mc)L^N x_\theta^N + (p_x^S - mc)L^S x_\theta^S$$

$$\Longrightarrow x_\theta^S = \left(\frac{\lambda^2\alpha^S g^S(\theta)w^S}{(1+k)w^N}\right)^{\frac{1}{1-\lambda}} \quad and \quad (1+k)p_x^S = \frac{(1+k)w^N}{\lambda} \tag{4}$$

where L^S is the Southern population. The values for x_θ^N and p_x^N in this regime are the same as given in (2) for regime N. Price is a constant markup over marginal cost and identical across regions since all production of x_θ occurs in the North using Northern labor, although the inefficiency tax effectively increases this marginal cost in the South. Southerners consume less of each health good when the inefficiency tax is higher, and consume more when the relative wage is higher. Both Northern and Southern consumers also demand more health goods when such goods are more effective at ameliorating the economic costs of disease, as captured by higher values of λ and α. However, a higher λ, by implying a more effective and hence valuable product, raises the monopoly price, thereby also curbing demand.

The market for the consumption good c is perfectly competitive. Both the North and the South produce for their own consumption, via the production function $c^i = A^i l^i$, where one unit of effective labor produces A^i units of c in region $i = N, S$. Since, as before, expected labor resources for the representative agent in region i are $1 - \int_0^\infty g^i(\theta)[1 - \alpha^i(x_\theta^i)^\lambda]d\theta$, we have

$$l^N = \int_{\underline{\theta}}^{\overline{\theta}} (\alpha^N g^N(\theta))^{\frac{1}{1-\lambda}} (\lambda^2)^{\frac{\lambda}{1-\lambda}} d\theta, \quad l^S_{NS} = \int_{\underline{\theta}}^{\overline{\theta}} (\alpha^S g^S(\theta))^{\frac{1}{1-\lambda}} \left(\frac{\lambda^2 w^S}{(1+k)w^N}\right)^{\frac{\lambda}{1-\lambda}} d\theta, \tag{5}$$

$$and \;\; l^S_N = \int_{\underline{\theta}}^{\overline{\theta}} (\alpha^S g^S(\theta))^{\frac{1}{1-\lambda}} \left(\frac{\lambda w^S}{(1+k)w^N}\right)^{\frac{\lambda}{1-\lambda}} d\theta \tag{6}$$

The subscripts indicate the regimes, in which, as we shall see, the ranges of integration will differ. Setting the wage per efficiency unit of labor equal to the marginal product of effective labor, we have $w^N = A^N$ and $w^S = A^S$. Consumption levels c^N_N, c^N_{NS}, c^S_N and c^S_{NS} can now be determined.

Firms enter the market for x until profits equal the fixed cost of innovation, call it f. This determines the range of innovations. Since prices are the same for each innovation, but demand changes according to prevalence of the related disease, all existing x_θ will have $\pi(\theta) > f$, except for the marginal innovations, for which profit will equal the fixed cost of innovation: $\pi(\overline{\theta}) = \pi(\underline{\theta}) = f$. Let $\pi^N(\theta)$ and $\pi^{NS}(\theta)$ be the profit functions for variety θ in regimes N and NS respectively. In regime N, the range of innovations is $\overline{\theta}_N - \underline{\theta}_N$, where $\pi^N(\overline{\theta}_N) = \pi^N(\underline{\theta}_N) = f$. If the South also enforces patents, profits to innovators increase because of the enlarged market.[14] Therefore $\pi^{NS}(\overline{\theta}_N) > \pi^N(\overline{\theta}_N) = f$ and $\pi^{NS}(\underline{\theta}_N) > \pi^N(\underline{\theta}_N) = f$. It follows that $\underline{\theta}_{NS} < \underline{\theta}_N$ and $\overline{\theta}_{NS} > \overline{\theta}_N$:

Proposition II.1. *If the South switches from regime N to regime NS, the array of existing health goods increases.*

Since profits are proportional to effective demand, which varies according to disease prevalence, we can use the exogenous probability density functions, $g^i(\theta)$ $i = N, S$, to analyze the impact of IPR reform on innovations. Figure III.1 (adapted from Diwan and Rodrik, 1991) depicts the result in Proposition II.1, assuming triangular distributions for the probabilities of getting each disease, for the case where purchasing power is equal in both regions, i.e., $L^S = L^N$ and $w^S = w^N$; the focus is on the difference in disease prevalence, i.e., $m^S \neq m^N$. Increasing the market size for innovating firms increases the range of existing innovations by loosening the free entry condition.[15] When distribution modes differ between regions, the demand faced by health goods innovators will increase more at the upper end of the distribution when the South begins enforcing IPR, but will still

[14] From (7), $\pi^{NS}(\theta) = \pi^N(\theta) + L^S\left(\frac{w^N}{\lambda} - w^N\right)\left(\frac{\lambda \alpha^S g^S(\theta) w^S}{(1+k)w^N}\right)^{\frac{1}{1-\lambda}} > \pi^N(\theta)$.

[15] Note that a higher inefficiency tax, k, by diminishing demand in the South, would dampen this innovation response of IPR reform, but the impact would remain positive.

increase somewhat at the lower end. Since profits increase more at the upper margin, the free entry constraint loosens more at the upper limit of the distribution and incentives increase more for innovations favoring Southern needs. This result holds as long as needs differ to any degree, i.e., as long as $g^N(\theta) \neq g^S(\theta)$:[16]

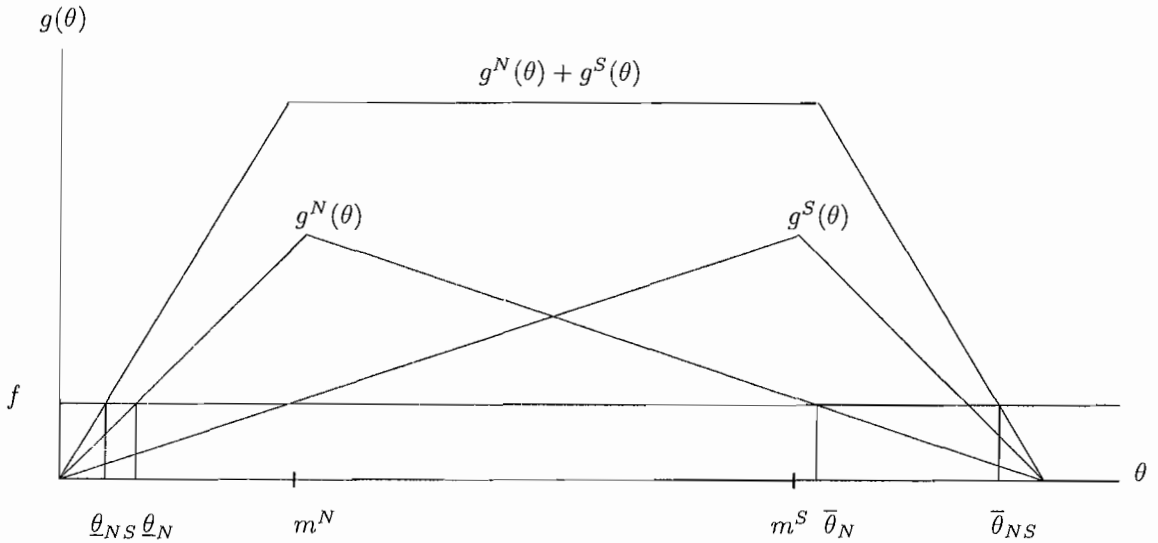

Figure II.1: Determining the range of innovated health goods under regime N and regime NS

Proposition II.2. *If needs differ between North and South ($g^N(\theta) \neq g^S(\theta)$), then when the South switches from regime N to regime NS, the range of existing health goods will increase more at the upper limit, which is closer to the mode of the disease distribution for the South than is the lower limit.*

II.4 Southern Welfare

When patents are not enforced in the South, existing innovations are available to Southern consumers but at the price $p_N^S = w^N$ charged by perfectly competitive imitators; the higher (monopoly) price if the South enforces patents is $p_{NS}^S = w^N/\lambda$. When the South moves from no patent enforcement to full patent enforcement, Southern welfare decreases due to the higher price and hence lower demand for health goods. This effectively reduces wealth. But there is a

[16]From footnote 14, $\pi^{NS}(\underline{\theta}_N) - \pi^N(\underline{\theta}_N) < \pi^{NS}(\overline{\theta}_N) - \pi^N(\overline{\theta}_N)$, which simplifies to $g^S(\underline{\theta}_N) < g^S(\overline{\theta}_N)$, and this is evident in Figure III.1.

wealth-enhancing counter effect via the consumption of additional health goods that become available. Consumption of health goods in the ranges $[\overline{\theta}_N, \overline{\theta}_{NS}]$ and $[\underline{\theta}_{NS}, \underline{\theta}_N]$ increase labor supply by combating diseases for which there was previously no treatment. Which effect dominates will determine the net impact on Southern welfare of moving from regime N to regime NS. Southern welfare in regime NS is:

$$W_{NS}^S = L^S u\left(w^S\left(1 - \int_0^\infty g^S(\theta)(1-\alpha^S(x_{\theta,NS}^S)^\lambda)d\theta\right) - \int_{\underline{\theta}_{NS}}^{\overline{\theta}_{NS}} \frac{w^N + \lambda k}{\lambda} x_{\theta,NS}^S d\theta\right)$$

and in regime N it is:

$$W_N^S = L^S u\left(w^S\left(1 - \int_0^\infty g^S(\theta)(1-\alpha^S(x_{\theta,N}^S)^\lambda)d\theta\right) - \int_{\underline{\theta}_N}^{\overline{\theta}_N} (1+k) w^N x_{\theta,N}^S d\theta\right)$$

Hence $W_{NS}^S > W_N^S$ as long as:

$$\lambda^{\frac{\lambda}{1-\lambda}} \int_{\underline{\theta}_{NS}}^{\overline{\theta}_{NS}} g^S(\theta)^{\frac{1}{1-\lambda}} d\theta > \int_{\underline{\theta}_N}^{\overline{\theta}_N} g^S(\theta)^{\frac{1}{1-\lambda}} d\theta \tag{7}$$

Welfare only increases for the South when it moves from zero to full patent protection if the gains from new technologies are sufficiently larger than the benefits of free-riding on the innovation incentives provided by the North. Using (6), we see that (7) is equivalent to effective labor in the South being higher under regime NS:

$$W_{NS}^S > W_N^S \iff l_{NS}^S > l_N^S \tag{8}$$

If needs are identical across regions, then the increase in innovations in response to an enlarged market demand under regime NS may not sufficiently benefit the South, since medications for the most prevalent diseases are already available in regime N. If needs differ, then by Proposition II.2 the new innovations will disproportionately benefit the South, since these will be medications targeting diseases that are relatively more prevalent in the South. This effect will be more pronounced the more needs differ between the North and the South. If they do not differ sufficiently, the incentives to free-ride are too great, and condition (7) will be violated. In that case, we have a return to the Deardorff (1992) result that the South is harmed by strengthening IPR.[17]

[17] If the South is sufficiently wealthy, or its population large enough, its addition to the market for monopolistically pro-

How different must needs be in order for the South to benefit from IPR protection? We cannot solve for this generally, but the question becomes tractable if we resort to the triangular distribution again. Let the cumulative distribution functions for θ be:

$$\begin{aligned} G^i(\theta) &= \frac{(\theta - a^i)^2}{(b^i - a^i)(m^i - a^i)} && for\ a^i \leq \theta \leq m^i \\ &= 1 - \frac{(b^i - \theta)^2}{(b^i - a^i)(b^i - m^i)} && for\ m^i < \theta \leq b^i \end{aligned}$$

where a^i and b^i are the bounds, or rather, the range of diseases which individuals in region $i = N, S$ have a positive probability of contracting. The modes, m^N and m^S, capture differences in need for types of health goods, or differences in disease prevalence, between the North and the South. Note that Figure III.1 depicts $\underline{\theta} < m^N$ and $\overline{\theta} > m^S$ in both regimes. For further tractability, set $a^N = a^S = 0$ and $b^N = b^S$, so that:

$$\begin{aligned} G^i(\theta) &= \frac{\theta^2}{bm^i} && for\ 0 \leq \theta \leq m^i \\ &= 1 - \frac{(b - \theta)^2}{b(b - m^i)} && for\ m^i < \theta \leq b \end{aligned} \tag{9}$$

Using (9), the condition for welfare improvement becomes

$$\lambda^{\frac{\lambda}{1-\lambda}} \left(\left(1 - \frac{(b - \overline{\theta}_{NS})^2}{b(b - m^S)} \right)^{\frac{1}{1-\lambda}} - \left(\frac{\underline{\theta}_{NS}^2}{bm^S} \right)^{\frac{1}{1-\lambda}} \right) > \left(1 - \frac{(b - \overline{\theta}_N)^2}{b(b - m^S)} \right)^{\frac{1}{1-\lambda}} - \left(\frac{\underline{\theta}_N^2}{bm^S} \right)^{\frac{1}{1-\lambda}} \tag{10}$$

and the limiting innovations are:

$$\underline{\theta}_N = \left(\frac{bm^N}{2\lambda^{1+\lambda}\alpha^N} \right) \left(\frac{f}{(1-\lambda)L^N w^N} \right)^{1-\lambda} \quad and \quad \overline{\theta}_N = b - \left(\frac{b(b - m^N)}{2\lambda^{1+\lambda}\alpha^N} \right) \left(\frac{f}{(1-\lambda)L^N w^N} \right)^{1-\lambda}$$

from $\pi^N(\underline{\theta}_N) = \pi^N(\overline{\theta}_N) = f$, using (2), and

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duced health goods may increase the range of innovations enough to improve welfare regardless of preference differences. Simulations show that this is almost never the case, however.

$$\underline{\theta}_{NS} = \left(\frac{b}{2\lambda^{1+\lambda}}\right)\left(\frac{f}{(1-\lambda)w^N\left(L^N\left(\frac{\alpha^N}{m^N}\right)^{\frac{1}{1-\lambda}}+L^S\left(\frac{\alpha^S w^S}{(1+k)w^N m^S}\right)^{\frac{1}{1-\lambda}}\right)}\right)^{1-\lambda} \quad \textit{and}$$

$$\overline{\theta}_{NS} = b-\left(\frac{b}{2\lambda^{1+\lambda}}\right)\left(\frac{f}{(1-\lambda)w^N\left(L^N\left(\frac{\alpha^N}{b-m^N}\right)^{\frac{1}{1-\lambda}}+L^S\left(\frac{\alpha^S w^S}{(1+k)w^N(b-m^S)}\right)^{\frac{1}{1-\lambda}}\right)}\right)^{1-\lambda}$$

from $\pi^{NS}(\underline{\theta}_{NS}) = \pi^{NS}(\overline{\theta}_{NS}) = f$ using (4).

$\overline{\theta}_{NS}$ is decreasing in k at an increasing rate, and $\underline{\theta}_{NS}$ is increasing in k but at a decreasing rate, i.e. $\frac{\partial\overline{\theta}_{NS}}{\partial k} < 0$, $\frac{\partial^2\overline{\theta}_{NS}}{\partial\partial k} > 0$, $\frac{\partial\underline{\theta}_{NS}}{\partial k} > 0$, and $\frac{\partial^2\underline{\theta}_{NS}}{\partial\partial k} < 0$; k does not affect the range of innovations under regime N. The South has more influence over the upper bound of the health goods distribution, while the North has more influence over the lower bound. Poorer access to medicine in the South will dim the response of innovation to IPR reform in the South, and this effect will be more prominent at the upper end of the range.

The minimum difference in modes, $m^S - m^N$, that satisfies condition (10) can be solved for if we make assumptions about other parameters. Let f = \$802 million, in accordance with the current average cost of developing a new drug (DiMasi *et al.* 2003).[18] Productivity estimates for w^S relative to the United States ($w^N = 100$) are taken from Hall and Jones (1999). We explore average productivity for Sub-Saharan Africa ($w^S = 24.6$), South America and Asia ($w^S = 51$), and Europe ($w^S = 76$).[19] We take the population sizes to be $L^N = L^S = 3$ billion (and will also explore the configurations $L^N = 1$ billion, $L^S = 5$ billion and $L^N = 2$ billion, $L^S = 4$ billion). Using these values, Figure III.2 displays the welfare effect of the South introducing IPR protection as a function of the effectiveness of the medicine, λ, and the modal difference between regions, $m^S - m^N$, when $b = 1$. Although higher values for b were explored, very large values yielded much flatter disease distributions, which detracted from the need differential focus of the model. Differences in the productivity of Southern workers generate few noticeable changes in the general welfare impact for the South. For the reasonable case of $m^N = 0.3$, it is clear in Table II.3 that the least developed countries *must* have different disease prevalence than the North in order to possibly benefit from IPR

[18] Average costs factor in losses from unsuccessful R&D endeavors.

[19] As shown in Table II.1, the correlation is high between disease burden in Latin America and much of Asia.

enforcement. This is true even for more productive regions, except when global population is concentrated in those regions.

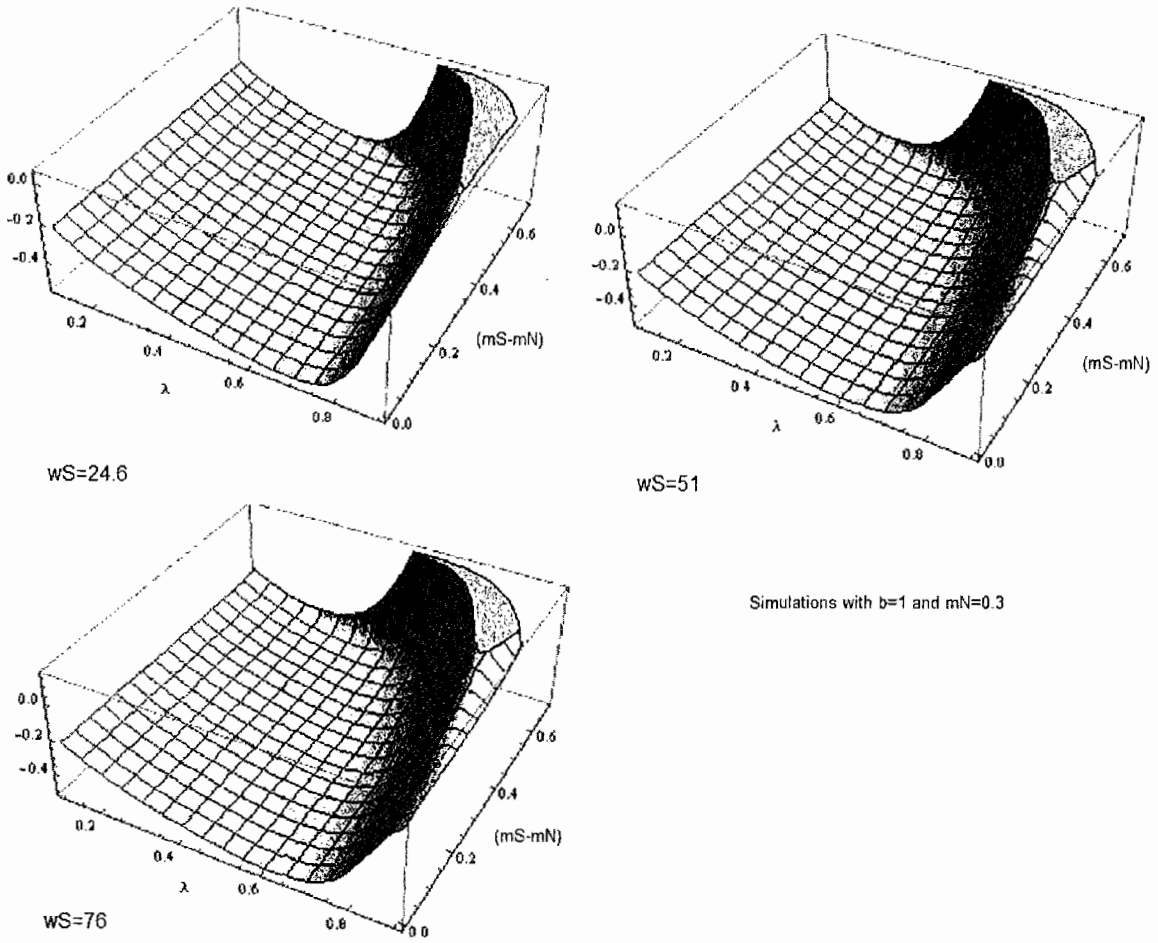

Figure II.2: Net welfare change for the South when it moves from regime N to regime NS

Health goods must be sufficiently effective in combating diseases for the South to benefit. The minimum mode differential required for a welfare improvement increases as λ decreases. A higher λ leads to a higher markup on the monopoly price that an innovator charges to consumers, which reduces consumption of health goods. However, a higher λ also increases demand for health goods, because health goods are more effective at combating disease. A higher monopoly price and the positive direct effect of λ on demand increase the profitability of innovations, thereby loosening the free entry constraint, which fosters a greater array of innovations.

When global population is concentrated in the South, welfare is more likely to increase under regime NS. This can be seen in Table II.3. Thus, while a small country may not benefit from

Table II.3: Simulation results

North's mode	Southern productivity	South's population (Total 6 billion)	Minimum mode difference to ensure positive welfare change (k=0)				
			$\lambda = 0.9$	$\lambda = 0.8$	$\lambda = 0.7$	$\lambda = 0.6$	$\lambda = 0.5$
0.3	24.6	3 billion	0.13	0.42	0.63	0.69	X
		4 billion	0.09	0.38	0.61	0.68	X
		5 billion	0.02	0.29	0.56	0.66	0.69
	51	3 billion	0.1	0.41	0.63	0.69	X
		4 billion	0.05	0.36	0.61	0.68	X
		5 billion	**0**	0.26	0.56	0.66	0.69
	76	3 billion	0.08	0.4	0.63	0.69	X
		4 billion	0.03	0.35	0.61	0.68	X
		5 billion	**0**	0.25	0.56	0.66	0.69
0.5	24.6	3 billion	0.13	0.35	0.47	X	X
		4 billion	0.1	0.32	0.45	0.49	X
		5 billion	0.06	0.27	0.43	0.48	X
	51	3 billion	0.11	0.34	0.47	X	X
		4 billion	0.08	0.31	0.45	0.49	X
		5 billion	0.03	0.26	0.43	0.48	X
	76	3 billion	0.09	0.34	0.47	X	X
		4 billion	0.06	0.31	0.42	0.49	X
		5 billion	0.02	0.25	0.43	0.48	X
0.7	24.6	3 billion	0.11	0.24	0.29	X	X
		4 billion	0.1	0.23	0.29	X	X
		5 billion	0.07	X	0.28	X	X
	51	3 billion	0.1	0.24	0.29	X	X
		4 billion	0.09	0.23	0.29	X	X
		5 billion	0.06	X	0.28	X	X
	76	3 billion	0.1	0.24	0.29	X	X
		4 billion	0.08	0.23	0.29	X	X
		5 billion	0.05	X	0.28	X	X

X indicates Southern welfare unambiguously decreases when the South enforces IPR, regardless of the degree to which needs differ.

enforcing IPR alone, a cooperative regime change, as the one orchestrated by the WTO in the form of TRIPS, is more liable to benefit the entire reforming region. Increasing Southern productivity also makes the condition for welfare improvement more apt to hold, although even if labor productivity in the South substantially exceeds that in the North, needs must generally still differ in order for the South to benefit from enforcing IPR. Also, the impact of w^S/w^N diminishes with λ.

For higher values of k, condition (7), for a welfare improvement when the South chooses to enforce IPR, is more demanding, as shown in Figure III.3.[20] That is, the less accessible are health goods to Southern consumers, the less likely is it that a welfare increase would follow the South's introduction of IPR protection. By effectively increasing the price of innovated goods in the South, a higher k reduces demand, which lowers the potential profitability of innovations. The free entry constraint becomes more binding, thereby reducing the range of innovations under the NS regime relative to if $k = 0$. A subsidy, by counteracting some portion of the inefficiency tax, would thus enhance the prospect for the South to benefit from enforcing IPR.

II.5 Discussion and Extensions

This paper seeks to answer whether enforcing IPR in the South will stimulate R&D of health goods for which there is a relatively greater need in the South. The answer depends on the extent to which needs differ, relative productivity levels and population size, and accessibility of health goods in developing countries. Accounting for different needs in rich and poor countries can reverse results in previous studies in which welfare in the South diminishes in response to IPR enforcement. A larger potential market for a particular health good, as determined by population, productivity and disease prevalence, increases the probability of that good coming into existence under IPR enforcement. The more disease prevalence differs across regions, the less demanding are the conditions under which Southern consumers will gain more relative to Northern consumers.

Even though consumption of health goods is unique, the results here extend to other types of goods. Many products may be similarly desired in both developed and developing countries, but their efficient use in a developing country may necessitate an innovative adaptation.[21]

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[20] These simulation results use $w^S = 24.6$. If instead $w^S = 51$ or 76, welfare decreases in response to k more slowly.

[21] Unreliable electricity in many developing countries spurred the invention of laptop computers fitted with a wind-up, power-generating device. The laptop was developed by One Laptop per Child, a non-profit organization created by faculty members from the MIT Media Lab.

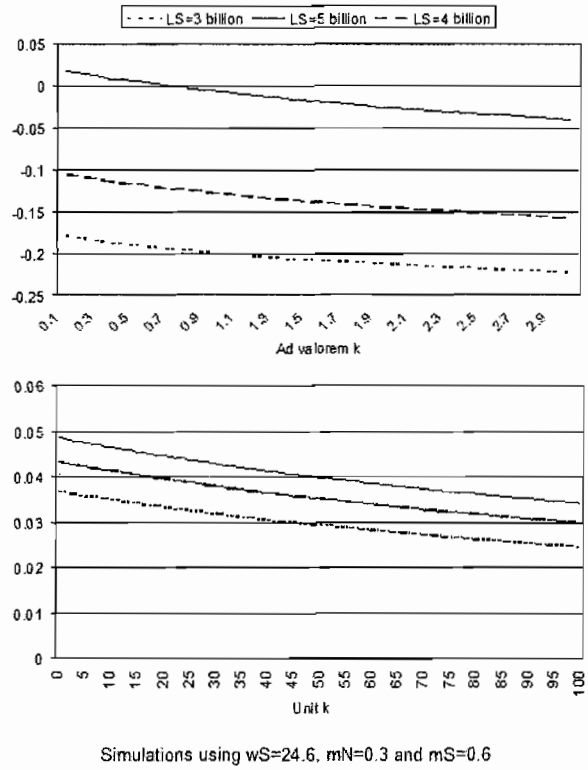

Figure II.3: Effect of inefficiency tax on net welfare change for the South when it moves from regime N to regime NS

Lack of infrastructure or corruption (i.e. higher k) can impede the South's access to medicine, and it is also in countries where such problems are most severe that enforcement of IPR is least plausible. This effect of k has similar implications as the U-shaped relationship between development and the welfare-maximizing level of IPR in the South developed and empirically supported in Chen and Puttitanun (2005), which suggests that the very poorest countries should free-ride on the innovation incentives of wealthier countries. On the other hand, the lowest correlation in Table II.1 is between disease burden in Sub-Saharan Africa and in the Established Market Economies (0.096). In this sense Sub-Saharan Africa has the least to gain from free-riding on the innovation incentives of countries with strong IPR.

Yet imitation occurs in more developed countries such as India and Brazil, so by strengthening IPR in these countries, we effectively strengthen them in the least developed countries which only consume these imitated goods.[22] The model predicts that this will be welfare reducing for

[22]Until 2005 India legislated only patents of the process used to make a drug, not the drug itself, and such patents

the least developed countries, but protection of intellectual property by the South always enhances Northern welfare[23] - via both higher profits and the additional innovations which become available to Northern consumers; therefore, a Pareto-improving redistribution of wealth from the North to the South, in the form of subsidized medicine for example, may be possible.[24] Stricter IPR may encourage modifications of existing drugs to circumvent the causes of a high k, for example, by decreasing the number of required doses or eliminating the need for refrigeration.[25] Most of the medicines deemed as minimum requirements for a basic health care system on the WHO's list of essential medicines are already available out of patent, suggesting that it is not IPR enforcement that is preventing the majority of essential treatments in developing countries. Kremer and Glennerster (2004) argue that pull incentive mechanisms, such as the promise by Western governments to purchase drugs from innovators for use in developing countries, will encourage innovations that suit the needs of the poor. Such promises may be unnecessary (if condition (7) holds), although they may anyway enhance the effects of market forces. In countries where enforcement of IPR is difficult, such a pull incentive would mimic the incentive effects of a strong IPR regime. If condition (7) fails to hold, then Kremer and Glennerster's incentive scheme would affect the condition. Similarly, foreign aid could be used to finance IPR directly so that the market force benefits of innovation stimulation can still be achieved.

Effective labor allocation is shown in (6). It is clear that if condition (7) holds, then the higher consumption of health goods in the South leads to more production and consumption of other goods c due, respectively, to a rise in effective labor supply and because individuals are wealthier. Since $w^S < w^N$ by assumption, and only the North innovates, the South has a comparative

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were also short-lived, 5-7 years compared to 14 in other countries. Even now, India does not recognize as patentable new uses or doses of existing drugs. Since 2000, generic manufacturing has brought the cost of AIDS treatment down from $10,000 per patient per year to $130, according to Médecins Sans Frontières. Indian companies provide over 80% of the drugs to fight AIDS and more than 25% of other essential drugs that Médecins Sans Frontières supplies to patients worldwide (Gentleman 2007). Oxfam reports that 95% of Uganda's drugs are imported, of which 33% originate in India, and 80% are generics. Clearly, the effects of limiting generic drug industries such as India's will be far-reaching.

[23]This is in contrast with the models of Grinols and Lin (2006) and Diwan and Rodrik (1991), in which each region competes for limited R&D resources.

[24]Such a subsidy would effectively lower the cost of health goods to Southern consumers, southern utility becoming $L^S u\left(w^S\left(1-\int_0^\infty g^S(\theta)(1-\alpha(x_\theta^S)^\lambda)d\theta\right)-\int_{\underline{\theta}}^{\bar{\theta}}(1-s)(1+k)p_x^S x_\theta^S d\theta\right)$. The subsidy would be funded by a lump-sum tax $\gamma\frac{\Pi}{L^N}$ on each Northern consumer, such that $\int_{\underline{\theta}_{NS}}^{\bar{\theta}_{NS}} sp^S x^S d\theta = \gamma\frac{\Pi}{L^N L^S}$, where $0<\gamma<1$ is a constant. To emphasize the Pareto-improving nature of this agreement, the Northern consumer could be taxed on only the additional profits under regime NS, the lump-sum tax in the North taking the form $\gamma\left(\frac{\Pi_{NS}-\Pi_N}{L^N}\right)$.

[25]Research is currently underway in India's Bhabha Atomic Research Center to imbed a hepatitis B vaccine into bananas, which could be easily administered without medical staff and at a cost of two cents a dose, rather than the current $125 (Kumar *et al.* 2005).

advantage in the production of c: both regions can benefit from trade and specialization. If trade and specialization occur, we would expect to see some income convergence across regions. Higher wages in the South would further increase demand for health goods, thereby loosening the free entry constraint and increasing innovations. As in Branstetter *et al.* (2007), the North can reallocate labor resources to R&D and meet the increased demand for innovations.[26] This additional mechanism will reinforce the positive effects of IPR.

We conclude with some remarks about what the model of this paper does *not* do. It does not allow for innovation in the South, though the trade-off between imitating foreign technologies and encouraging domestic innovation is clearly important in developing countries (Chen and Puttitanun, 2005).[27] It is reasonable to assume innovation does not occur in the least developed countries. Also, disease prevalence is exogenous in our model. It is left to future research to examine the evolution of disease prevalence and the resulting long-run impacts on health and hence growth, consequent upon Southern IPR protection.

Finally, it remains to be seen what impact compliance with TRIPS has had, and will have, on the allocation of R&D resources. An empirical analysis faces several challenges: the date when TRIPS went into effect is not concrete, since a transition period was allowed to developing countries, and the time series of accurate disease burden data remains short.

[26] Unlike Branstetter *et al.*, this paper does not address migration.

[27] China's inadequate IPR enforcement may be having significant consequences on the rate of innovation both in China and in the rest of the world by limiting incentives for innovators (Office of the US Trade Representative, 2005).

CHAPTER III

CHILDHOOD DISEASE AND THE PRECAUTIONARY MOTIVE FOR CHILDREN

This chapter, excluding the empirical section, is co-authored with Shankha Chakraborty, who contributed through analytical insights and provided editorial assistance. I was the primary contributor to the development and analysis of the theoretical model.

III.1 Introduction

Historical demographic changes were triggered by declines in mortality, generally followed by declines in fertility. In England, whose experience represents that of many Western countries, a sharp decline in deaths from infectious diseases began around 1872, followed about five years later by declines in the total and net fertility rates (Arora 2003, 2005).

England's experience suggests a positive relationship between fertility and the infectious disease burden, one that is the subject of this paper. We build on Sah (1991) and Kalemli-Ozcan's (2003, 2008) work on child mortality and the precautionary demand for children. Our key innovation is separating the childhood disease burden into child mortality and morbidity. Parents are uncertain whether their children will contract infectious diseases in early childhood and whether infected children will succumb to them. Higher are disease prevalence and case fatality, higher this uncertainty. When infected children do survive, chronic illness during the early developmental years leaves a permanent mark on their long-term health. This morbidity effect lowers the marginal return from parental investment on unhealthy children. Survivors of infectious diseases are, consequently, lower quality than children who never (significantly) contracted them in the first few years after birth.

The desired fertility of risk averse parents depends on the overall burden of infectious disease – its prevalence and its impact on child mortality and morbidity.[1] How a decline in child mortality

[1] Uncertain parents in Sah (1991) and Kalemli-Ozcan (2003) overshoot their desired fertility to insure against the

affects fertility depends on whether it is triggered by improvements in case fatality or by lower disease prevalence. The latter not only reduces uncertainty faced by parents, as does the former, it simultaneously ensures that more surviving children are of higher quality. Tackling the spread of infectious diseases, in other words, is likelier to have a stronger impact on fertility as parents substitute child quality for quantity in addition to benefiting from lower child mortality.

Under-five mortality has declined globally by 50% between 1960 and 2002 (UNICEF)[2] and this has coincided with a decline in the total fertility rate (TFR). Yet the disease burden remains high in developing countries, many of which are yet to complete, or even begin, the fertility transition. Women in industrialized countries have between one and two children and child mortality rates average 0.6%, mostly due to perinatal conditions. In developing countries child mortality rates are as high as 26% in Sierra Leone, and women in Niger have up to eight children on average (CIA World Factbook, World Bank). As Table III.1 shows, infectious diseases comprise seven of the top ten causes of child mortality in developing countries today. Pneumonia, diarrhea, malaria, measles and AIDS account for half of all deaths among children under the age of five.

Table III.1: Top 10 causes of child mortality in developing countries

Rank	Cause	Numbers (000)	% of all deaths
1	Perinatal conditions	2,375	23.1
2	Lower respiratory infections (pneumonia)	1,856	18.1
3	Diarrhoeal diseases	1,566	15.2
4	Malaria	1,098	10.7
5	Measles	551	5.4
6	Congenital anomalies	386	3.8
7	HIV/AIDS	370	3.6
8	Pertussis	301	2.9
9	Tetanus	185	1.8
10	Protein-energy malnutrition	138	1.3
	Other causes	1,437	14.0
	Total	10,263	100.0

Source: World Health Organization

possibility of too few surviving children. Hence there is little investment in child quality. The inclusion of two sources of uncertainty, in disease exposure and in survival from that exposure, amplifies the precautionary motive here for a given degree of risk aversion.

Tamura (2006) distinguishes between infant and child mortality rates but parents invest the same amount on each child at birth. Here parents invest in children after survival outcomes are known, and they can invest differently in healthy and unhealthy children.

[2] http://www.unicef.org/mdg/childmortality.html

A wealth of medical evidence suggests that acquiring infectious disease in early childhood can hamper cognitive or physical development and predispose one to other diseases later in life. Bronchitis, pneumonia and whooping cough before age 5 are linked to diminished respiratory function at ages 59-70 (Barker 1991, 1994). Acute rheumatic fever damages heart valves, and late-stage syphilis, measles and malaria can affect the functioning of the circulatory system. Typhoid shows cardiac involvement also (Khosla 1981). Infections that disrupt absorption, such as diarrheal infections, deprive the body of nutrients necessary for optimal cellular growth (Martorell 1980, Martorell and Habicht 1986, Mata 1978). In some cases, survivors of infection become susceptible to other health problems because "during infection, the biological system diverts its resources from cellular growth toward the synthesis of antibodies and repairing damaged tissue." (Arora 2005 p. 213). In other cases, an infectious disease is the direct cause of a non-infectious disease, as in the case of cervical cancer, whose primary cause is the human papillomavirus.

That infectious diseases early in life have lifelong health consequences is also borne out by more indirect evidence, for instance, adult heights. Arora (2005) reports that infectious diseases started a downward decline in 1872 in England and Wales, followed from 1890 onwards by a sharp upward trend in the height of 18-year-old British males.[3] Adult height, as a proxy for health, is of course well known to be associated with higher earning (Strauss and Thomas 1998). There is also good evidence on the complementarity between health and returns from educational investment during the early years (Behrman 1996). Hence the infectious disease burden bears upon childhood mortality as well as future productivity.

Explicitly recognizing the mortality and morbidity impact of infectious disease among children delivers both positive and negative fertility responses to disease. In Barro and Becker (1988) a decline in child mortality reduces the average fixed cost of raising a surviving child, prompting parents to have more children. This rise in the demand for children is temporary: net fertility returns to its original level (unless mortality continues to decline) but because of lower mortality rates fewer births are required to achieve that target. The Barro-Becker model consequently generates an inverted U-shaped population path when mortality declines.

Adaptations of the Barro-Becker model show an unambiguously positive response of the total fertility rate (TFR) to mortality; any negative relationship resulting from the average cost effect is

[3]Vaccination was made compulsory in 1853 and in 1871 legislation was introduced requiring all poor law unions to appoint vaccination officers and to set up a system of registration; this system, with only minor alterations, lasted until 1948 (Drake *et al.* 2001).

completely overshadowed by positive precautionary forces.[4] The model proposed below accounts for the Barro-Becker average cost effect and determines circumstances in which it dominates the forces (precautionary demand for children, quantity-quality tradeoff) contributing to a positive response of fertility to child mortality. By separating infection and case fatality rates, we introduce an additional but *positive* average cost effect that functions specifically through changes in morbidity and counteracts the negative average cost effect when infection rates decline but amplifies it when case fatality rates decline. As in Barro and Becker, the model generates an inverted U-shaped population path, but only when the decline in disease burden reduces child mortality sufficiently.[5]

The partial equilibrium model developed here has some obvious general equilibrium implications. If reducing childhood disease leads to fertility declines and rising human capital investment, targeting the disease burden can stimulate economic development. A reduction in disease burden may be achieved by reducing disease prevalence, disease severity, or both. We show that health initiatives in developing countries may have unintended consequences for fertility depending on how diseases are combated: a declining disease burden will not necessarily reduce mortality, and fertility, unless it also reduces morbidity. This partly explains why the fertility transition in sub-Saharan Africa has lagged child mortality declines – the reduction in infant mortality has been accompanied by persistent morbidity challenges (Akachi and Canning 2008).

By distinguishing between infection and case fatality rates, the model contributes to the existing mortality-fertility literature in several ways. It addresses an important reason why fertility rates remain high in many countries today: infectious disease. Modeling child mortality as caused by infectious disease allows us to analyze the unique role of morbidity. Since fertility declines with morbidity in the model, including a morbidity channel amplifies the positive relationship between mortality and fertility. By introducing a morbidity channel that links childhood disease with adult health, the model can account for the stagnation in adult health observed in sub-Saharan Africa; the stagnation is due to persistently high morbidity originating in childhood (Akachi and Canning 2008). Previous mortality-fertility models have had to assume a large precautionary motive for children to achieve a positive relationship between child mortality and fertility. The morbidity channel may reduce the role of the precautionary motive, a topic for further exploration.

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[4]Among such studies are Boldrin and Jones (2002), Cervellati and Sunde (2007), Galor and Weil (1999), Kalemli-Ozcan (2003, 2008), Soares (2005), Tamura (2006).

[5]In Doepke (2005), Kalemli-Ozcan (2003, 2008) and Boldrin and Jones (2002), the reduction in TFR when mortality rates decline is initially weak, so net fertility rises before it declines from its original level.

We present the theoretical model in Section III.2. Fertility and human capital decisions are analyzed in Section III.3 while Section III.4 offers further discussion and policy implications. Section III.6 concludes.

III.2 The Model

An individual lives for three periods – childhood, adulthood and old age – though not all children survive their early years. Children are born with an innate health capital h_0 and are exposed to infectious diseases in early childhood from which they may or may not survive. Let i denote the infection rate among these children. A fraction i of children born contract infectious diseases of whom a fraction $1-d$ survive, where $i, d \in (0,1)$. The parameter d is the case fatality rate from a disease while the product id is the child mortality rate.[6] What distinguishes our model from existing ones is the idea that exposure to infectious diseases has a morbidity effect even when children survive. To be specific, infections depreciate a child's health by the fraction $\delta \in (0,1)$.

Uncertainty about child survival resolves after the first few years of a child's life. Subsequently parents invest in the human capital of these children, for example, in their education or health. A child's health capital determines the productivity of such investment.

Surviving children are of two types. Those who were never (significantly) exposed to infectious diseases remain healthy (numbering N_1), those who survived diseases remain unhealthy (numbering N_2). Parents can invest differently in each type, h_1 on each healthy child, h_2 on each unhealthy one. A child's human capital as an adult reflects these investments and his health capital. Denoting by H' this human capital, we assume

$$H' = \begin{cases} h_0^{\alpha} h_1^{\theta} & \text{if healthy as a child,} \\ \{(1-\delta)h_0\}^{\alpha} h_2^{\theta} & \text{otherwise,} \end{cases}$$

where $\alpha, \theta \in (0,1)$. A child's consumption is not modeled explicitly and is instead subsumed in his parent's.

Adults work in youth and retire in old-age. Young adults earn a wage w per effective unit of labor which, given their human capital H (determined by their past disease experience and human

[6] In general i and d would depend on h_0 and on pre- and post-natal health inputs provided by the parent. Here both are treated as parameters.

capital investment), yields income wH. Children are valued in this economy because they financially support their elderly parents. This is particularly true in developing countries where social safety nets are most lacking. The social norm dictates that each adult contributes a $\tau \in (0,1)$ fraction of his labor earnings to his old parent and receives the same fraction of each child's earnings in old age.[7] Adults choose their consumption in youth, c, the number of children they have, n, and human capital investment in children, (h_1, h_2), that supports old-age consumption needs.

Assuming preferences over consumption are logarithmic and normalizing $h_0 = 1$, a young adult maximizes his expected lifetime utility

$$E\left[\beta \ln(c) + (1-\beta)\ln(\{N_1 h_1^{\theta} + (1-\delta)^{\alpha} N_2 h_2^{\theta}\}\tau w')\right] \tag{1}$$

subject to[8]

$$c + N_1 h_1 + N_2 h_2 \leq (1-\gamma n)(1-\tau)wH, \tag{2}$$

where w' is the efficiency wage rate faced by children in their adulthood, $\gamma \in (0,1)$ is the fixed time cost of having a child, and $\beta \in (0,1)$ is the rate of time preferences, that is, the weight attached on consumption in youth versus old-age. To conserve notation we define $z \equiv (1-\tau)wH$ and $x \equiv \tau w'$.

Adults face uncertainty in the number of surviving children and explicitly recognize the random nature of each child surviving exposure to infectious diseases. Since human capital investment (h_1, h_2) occurs after this uncertainty is resolved, we will solve the model using backwards induction. That is, we identify optimal consumption and investment decisions for the parent, conditional on N_1 and N_2. We then use the Delta Method to solve for the fertility decision, à la Kalemli-Ozcan (2008). The difference between our work and Kalemli-Ozcan's is that there are three rather than two possible outcomes for each child born.

The number and type of survivors are random draws from the discrete multinomial distribution:

$$p(N_1 = n_1, N_2 = n_2, N_3 = n_3) = \frac{n!}{n_1! n_2! n_3!} p_1 p_2 p_3 \tag{3}$$

where $\sum_{j=1}^{n} n_j = n$ and $p_j \in [0,1]$ denotes the probability of outcome n_j out of n births. On average

[7] While τ is exogenous here, Boldrin and Jones (2002) analyze endogenous donations from children that support parents in old age but there is no human capital investment in children.

[8] As in much of this literature we ignore integer constraints, that is, the constraints $n \geq 1$ and $N_1 + N_2 \geq 1$ that ensure at least one surviving offspring. We do take into account that, given a unit time endowment, fertility is bounded above $n \leq 1/\gamma$.

$\bar{N}_1 = n(1-i)$ children avoid disease and remain healthy, $\bar{N}_2 = ni(1-d)$ children survive disease but remain unhealthy, and $\bar{N}_3 = nid$ children succumb to disease.

1 Decision under Certainty

It is instructive to first consider the certainty version of the model. The number of survivors of each type is simply taken to be its expected value and the parent chooses h_1, h_2, n to maximize

$$\beta \ln\left[(1-\gamma n)z - n\{(1-i)h_1 - i(1-d)h_2\}\right] + (1-\beta)\ln\left[nx\left\{(1-i)h_1^{\theta} + i(1-d)(1-\delta)^{\alpha}h_2^{\theta}\right\}\right].$$

The first order conditions in an interior optimum yield:

$$\begin{aligned} n &= \frac{(1-\theta)(1-\beta)}{\gamma}, \\ c &= \beta z, \\ h_1 &= \frac{\theta\gamma}{(1-\theta)\left[1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}}\right]}z, \\ h_2 &= (1-\delta)^{\frac{\alpha}{1-\theta}}h_1. \end{aligned}$$

While parental income has a positive effect on child investments, fertility does not depend on it. Parents have fewer children if they value their future consumption more (lower β) and if the return to investment in child quality is higher (higher θ). But fertility is unaffected by the infection and case fatality rates, while human capital investment is *increasing* in them.[9] These results contradict historical data from demographic transitions in which declining mortality rates are associated with a decline in fertility and increases in human capital investment. This occurs because investment is inversely related to the number of survivors, which increases when either i or d declines. Since total fertility, n, does not also decline to compensate, investment per child declines.

Furthermore, net investment in children is unaffected by changes in i or d. The increase in survivors due to a decline in i or d exactly counteracts the decline in human capital investment per child. It can be shown that the net effect on total human capital investment $N_1h_1 + N_2h_2$ is exactly zero under certainty. When i decreases, fewer children die and more survivors are healthy. When d

[9]Kalemli-Ozcan (2003, 2008) also obtains this result under certainty; under CRRA, the TFR actually increases in response to lower mortality. In Boucekkine, Desbordes and Latzer (2008), who include a labor-leisure tradeoff, fertility declines with mortality despite CRRA and certainty about child survival. The TFR moves with mortality in Strulik's (2008) model with certainty and logarithmic preferences since investment in infants increases the probability of child survival and additionally provides a warm glow to the parent.

decreases, in contrast, fewer children die but more children are unhealthy for a given infection rate. Although the number of survivors, $n(1-id)$, is decreasing in i and d, $\partial N_1/\partial i = -n < 0$ while $\partial N_2/\partial i = n(1-d) > 0$, and $\partial N_1/\partial d = \partial n(1-i)/\partial d = 0$ while $\partial N_2/\partial d = \partial ni(1-d)/\partial d = -ni < 0$.

2 Decision under Uncertainty

When the variance, or uncertainty, of survival outcomes is incorporated into the optimization problem, n depends on the childhood disease experience. Children are most susceptible to infectious disease during the first few years of life, and during the demographic transition the largest gains in longevity occur amongst infants and children. Therefore, we assume that uncertainty about child survival is resolved after infancy and that human capital investment occurs after this time.

Parental decisions are solved sequentially. First, given the fertility decision (n) and the number and type (N_1 or N_2) of survivors, adults choose h_1, h_2 and c to maximize utility

$$\beta \ln\left[(1-\gamma n)z - N_1 h_1 - N_2 h_2\right] + (1-\beta)\ln\left[\left\{N_1 h_1^{\theta} + (1-\delta)^{\alpha} N_2 h_2^{\theta}\right\} x\right]$$

The first-order conditions yield:

$$h_1(n) = \frac{\theta(1-\beta)(1-\gamma n)}{\{\beta + \theta(1-\beta)\}(N_1 + (1-\delta)^{\frac{\alpha}{1-\theta}} N_2)} z, \tag{4}$$

$$h_2(n) = (1-\delta)^{\frac{\alpha}{1-\theta}} h_1(n), \tag{5}$$

and

$$c(n) = \frac{\beta(1-\gamma n)}{\beta + \theta(1-\beta)} z.$$

Quality investment in children now depends negatively on the number of survivors and on total fertility. Note especially that this investment depends on child mortality even though investment decisions are made after the uncertainty of number of survivors is resolved. Moreover, even though parental investment can compensate for depreciated health capital among unhealthy children, it does so only partially ($h_1 > h_2$) unless $\delta = 0$.

Using these conditional choices $h_1(n)$, $h_2(n)$ and $c(n)$, rewrite the utility function as

$$\beta \ln\left[\frac{\beta(1-\gamma n)}{\beta + \theta(1-\beta)} z\right] + (1-\beta)\ln\left[(N_1 + (1-\delta)^{\frac{\alpha}{1-\theta}} N_2)^{1-\theta} \left\{\frac{\theta(1-\beta)(1-\gamma n)}{\beta + \theta(1-\beta)} z\right\}^{\theta} x\right]$$

which the parent then maximizes with respect to n, taking into account uncertainty regarding (N_1, N_2) as specified by (3) above. Appendix V details how this optimization leads to the quadratic first-order condition:

$$n - \frac{\gamma\{\beta + \theta(1-\beta)\}n^2}{(1-\beta)(1-\theta)(1-\gamma n)} = \frac{-i[1 - i + (1-\delta)^{\frac{2\alpha}{1-\theta}}(1-d)[1 - i(1-d)]]}{2[1 - i + i(1-\delta)^{\frac{\alpha}{1-\theta}}(1-d)]^2}. \tag{6}$$

In the section below we analyze this fertility choice in depth, in particular how it responds to changes in the prevalence and case-fatality rates.

III.3 Fertility, Quality Investment and the Disease Burden

A change in the childhood disease burden – a decline in disease prevalence and case fatalities and the ensuing changes in morbidity – affects fertility decisions in various ways. In order to understand these, consider how the three parameters (i, d, δ) affect childhood mortality, morbidity and the quantity-quality tradeoff faced by parents. We discuss their effects on the total fertility rate, the net fertility rate and human capital investment.

1 The Total Fertility Rate

The first-order condition (6) is a quadratic in n; only one of its roots is positive and is the optimal fertility choice. For the special case $i = 0$ this optimal choice becomes $n = (1-\beta)(1-\theta)/\gamma$. This is the same as under certainty since none of the children are exposed to infectious diseases and therefore all survive.

Since fertility is a highly non-linear function of the parameters in the more general case, it is best analyzed by numerically solving for n. For simplicity set $\gamma = 1$ which restricts us to $n \in (0, 1)$ instead of $n \in (0, 1/\gamma)$. To allow for more decisive interpretation we also focus on extreme values of δ which yield conclusions consistent with more general cases. Appendix V establishes these results formally.

Disease Prevalence and the TFR

The upper panels of Figures III.1 and III.2 depict the fertility decision as a function of (i, d) for $\delta = 1$ and $\delta = 0$ respectively. The two panels share the same set of values for the other parameters, $\alpha = 0.9$, $\theta = 0.9$, $\beta = 0.5$, besides $\gamma = 1$.

In Figure III.1, $\delta = 1$ implies the child mortality rate is effectively i (rather than id) since unhealthy children are completely unproductive as adults and unable to support their parents in old-age. In other words, it is irrelevant for parental investment decisions if an infected child does or does not survive.

Suppose a new vaccine is introduced that lowers overall disease prevalence and infection rates among children. The lower panel of Figure III.1 identifies the relation between n and i for various values of d. It shows that n does not depend on d: the $n(i)$ functions for different values of d coincide. Fertility is monotonically increasing in the disease prevalence rate which is also the effective child mortality rate. A decline in i, in other words, is predicted to lower the TFR. In this case our model simplifies to Kalemli-Ozcan's (2003, 2008) analysis of the relation between the TFR and child mortality, the latter being simply the prevalence rate.[10]

Turn now to Figure III.2 to gauge the role of morbidity. Since $\delta = 0$ here, all surviving children are now equally healthy regardless of childhood illnesses. But newborns are exposed to two sources of uncertainty, first whether they contract infectious diseases or not, and second, whether they survive from it. The response of fertility to the prevalence rate can be nonlinear and depends on case fatalities, as the lower panel of Figure III.2 more clearly shows. The response is strongly positive when disease prevalence is relatively low. For relatively higher disease prevalence, the disease mortality rate becomes important. At fairly high case fatalities, the relation $n(i)$ is monotonically increasing as it was for Figure III.1. At low-to-medium case fatalities and relatively high infection rates, on the other hand, disease prevalence *lowers* fertility.

Three forces determine the response of n to changes in i. First, is the precautionary motive that points to a positive relationship between fertility and disease prevalence. Faced with child survival uncertainty, parents have more children than they ultimately desire: a decline in child mortality will reduce fertility as more children survive.

Secondly, when child mortality declines, survivors become cheaper to produce, which raises desired fertility (Barro and Becker 1988). The total cost of child rearing consists of the fixed birth cost (γn) and the cost of investing in child quality after survival outcomes are realized ($N_1 h_1 + N_2 h_2$). For a given n, the average birth cost of producing a surviving child decreases as $(N_1 + N_2)/n$ rises.

[10]That said, this isomorphism depends crucially on the way we modeled parental preferences. If parents were altruistic in the Barro-Becker sense or derived warm glow from investments in each type of child, they would care about unhealthy children even if $\delta = 1$. These alternative altruism specifications complicate the model considerably without adding much to our analysis of the morbidity effect.

Investments h_1 and h_2 decrease with the number of survivors for a given level of n as (4) and (5) show. Hence the average investment cost per surviving child,

$$\frac{N_1 h_1 + N_2 h_2}{N_1 + N_2} = \left[\frac{N_1 + (1-\delta)^{\alpha/(1-\theta)} N_2}{N_1 + N_2}\right] h_1$$

decreases when $N_1 + N_2$ rises because quality investment in unhealthy children is lower and because both types of quality investment fall. Hence the combined average cost per surviving child declines with lower disease prevalence.

The third channel, a key contribution of this paper, operates through the morbidity effect. Parents substitute between the quantity and quality of children depending on the disease burden. As disease prevalence falls, more of the survivors are healthy children. That is, $N_1/(N_1 + N_2)$ increases. Since $h_1 > h_2$, this makes surviving children more expensive on average and pushes parents towards fewer but better quality children.

The first and third effects work in the same direction, the second one in the opposite. Larger is δ, the stronger is substitution towards quality. Moreover, the morbidity effect is stronger when most of the surviving children have experienced disease, that is when i is high and d is low.

The sharpest contrast between the cases $\delta = 1$ and $\delta = 0$ in Figures III.1 and III.2 occurs for high i and low d. This is because in Figure III.2, at high i and low d values, the quantity-quality tradeoff due to the morbidity effect disappears and only the first two channels are at work. But since most children born get infected, few of them die and all are equally healthy, the precautionary incentive is weak. At high values of i and low values of d, the average cost effect drives the negative relationship between fertility and disease prevalence in Figure III.2.

Case Fatality and the TFR

Now consider the relationship $n(d)$. Suppose that d declines because of the availability of an antibiotic. The antibiotic could technically reduce infection rates by reducing the time during which infected individuals are contagious, but this is a relatively unimportant effect since infection rates are generally tackled via prevention (vaccines or behavioral changes). The response of the TFR to d is charted in Figure III.3 for various values of the prevalence rate.

For $\delta > 0$, reducing fatalities leaves proportionately more unhealthy children alive and $N_1/(N_1 + N_2)$ decreases. Since parents invest less in the human capital of unhealthy children, they

have more children in order to substitute for their low quality. In contrast to the vaccine scenario above, the negative average cost effect is amplified by a decline in d when δ is high. A decline in d improves survival, thereby lowering the average fixed birth cost of surviving children, and since more unhealthy children survive, the average surviving child is also cheaper in terms of human capital investment. The maximum TFR is 0.3 in Figure 2 ($\delta = 0$) but 0.4 in Figure 1 ($\delta = 1$) where the morbidity effect is stronger.[11]

An antibiotic lowers mortality more for very prevalent and fatal diseases, and increases morbidity more for high δ diseases. Changes in case fatality rates only elicit a noticeable (and positive) fertility response when morbidity effects are low (δ low) and when the impact on child mortality is highest (i and d are both high). When both i and d are very low, fertility is already nearest its lower bound so an increase in i or d cannot decrease n anyway.[12]

When δ is low, the i- and d-specific influences on morbidity are weak, and the response of n to falling disease burden depends on the net effect of a reduction in precautionary births (n decreases when child mortality decreases) and a decline in the (traditional) average fixed cost of survivors (n increases when child mortality decreases). The positive precautionary motive dominates the negative average cost effect when mortality changes are strongest: $\partial n/\partial i > 0$ occurs when d is high and $\partial n/\partial d > 0$ when i is high (see Appendix V for calculation of the precise thresholds).[13]

Proposition III.1. *Under uncertainty, fertility falls with declining disease prevalence (i) if the disease is fatal or (ii) if disease is not fatal but causes severe long-term morbidity.*

2 The Net Fertility Rate

Kalemli-Ozcan (2003) and Galor and Weil (1999) stress a historically observed non-monotonic pattern for net fertility: as child mortality declines, initially total fertility does not decline sufficiently to lower net fertility, but eventually total fertility declines enough to lower net fertility. A similar effect is at work in this model.

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[11] As expected fertility is higher in this model when θ, the returns to quality investment, is lower.

[12] In Figure III.2, the special case of $i = 1$ yields a monotonic positive relation between n and d. Here the effective child mortality rate is d. Hence the relation $n(d)$ is again similar to Kalemli-Ozcan's model.

[13] A low i here could be as high as $i = 1/2$. See Appendix V.

On average $n(1-id)$ children survive, and

$$\frac{\partial\{n(1-id)\}}{\partial i} = (1-id)\frac{\partial n}{\partial i} - nd \quad and \quad \frac{\partial\{n(1-id)\}}{\partial d} = (1-id)\frac{\partial n}{\partial d} - ni.$$

In the case where $\partial n/\partial i > 0$ and $\partial n/\partial d > 0$, net fertility rises in response to lower i if $\partial n/\partial i < (nd)/(1-id)$, and in response to lower d if $\partial n/\partial d < (ni)/(1-id)$. These conditions are more likely to hold for high i and d. As disease prevalence and case fatality rates continue to decline, the conditions become more binding and eventually net fertility also declines.

Investment in Human Capital

Finally we turn to the impact of childhood disease burden on human capital investment (see Appendix for details):

Proposition III.2. *Under uncertainty, parental human capital investment in children rises when the disease burden falls if TFR declines sufficiently with infection and case fatality rates.*

If the TFR rises when child mortality falls, then fewer resources remain for human capital investment, and h_1 and h_2 decline. If TFR declines but not enough to counter the increase in survivors, then h_1 and h_2 still decline. In countries where the fertility response may be weak or even negative, health care initiatives should be coupled with human capital investment initiatives, such as school subsidies, because combating disease may actually raise net and even total fertility and thus reduce human capital investment.

III.4 Discussion

Infection and case fatalities were historically high in many Western countries, and it was primarily new knowledge about germs that triggered sharp declines in infectious disease mortality in the late 19th century. The new knowledge led to public sanitation reform and improvements in personal hygiene, and both mortality and morbidity fell during the epidemiological transition (McNeill 1976).

As infection rates declined, child mortality fell significantly. Our model suggests that lower disease prevalence meant that fewer children got sick and survivors became healthier. Consequently desired fertility fell and human capital investment in children rose. The model generates such a

quantity-quality tradeoff when especially morbidity declines. Historical data on stature in Western countries show that cohorts of children who experienced declining mortality rates became taller (and thus healthier) adults relative to their predecessors, suggesting morbidity declined in tandem with mortality (Arora 2005).

Worldwide reductions in child mortality over the past half century have occurred through both lower disease prevalence (improved sanitation in urban areas, vaccination) and lower case fatalities (antibiotics). But the fact that the infectious disease burden remains high in many developing countries suggests that much of the decline has come from averting deaths from illnesses rather than preventing infection. The model demonstrates that if infectious disease mortality but not morbidity declines, high fertility rates can persist as parents continue to supplement the low quality of their children with greater quantity. This is a particularly pressing problem in sub-Saharan Africa where infant mortality rates have fallen since 1960 but cohorts of children affected have not grown up to be much healthier as measured by adult height (Akachi and Canning 2008).

Depending on the types of diseases that are most prevalent and the manner in which they are combated, some developing countries may not necessarily be poised for the demographic transition and the resulting rise in human capital investment. Infectious diseases weaken individuals physically and may impair cognitive development, especially in developing countries where infections are particularly virulent and children are often under- or malnourished. Parasitic diseases are widely prevalent in developing countries, and their treatment prevents deaths but cannot reverse the damage. Effective treatments exist, for example, for diseases like leishmaniasis, which damages the spleen and liver and can cause anaemia, and schistosomiasis, a chronic disease that damages internal organs and impairs growth and cognitive development in children. Prevention is preferred to treatment in such cases. Polio, which renders its survivors paralyzed (high δ) and for which there exists no cure, is one infectious disease to have been eradicated globally.[14] The model predicts this global eradication would have had a significant impact on fertility rates, a hypothesis that deserves further attention.

Given the virulent nature of their disease burden, the average δ for childhood illnesses is clearly substantial for developing countries. Resources devoted towards lowering this burden will be more efficiently spent, in terms of lowering the TFR and raising education and its returns, when they are able to reduce both mortality and morbidity.

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[14] Led by efforts of the WHO, polio has been eradicated in all but four countries: Afghanistan, India, Nigeria and Pakistan.

Malaria

Take the case of malaria which is prominent throughout much of sub-Saharan Africa. Malaria contributes substantially to child mortality (Table III.2). It also generates substantial morbidity effects: it is the major cause of childhood anemia and maternal anemia during pregnancy, of low birth weight, and in the case of cerebral malaria, causes persistent neurological deficits (Gollin and Zimmerman 2007). The most common effect, anemia, is associated with poor school performance. In terms of the theory, malaria is a moderate δ disease in that it causes substantial long-term health problems, but it is not so severe that it renders individuals completely unproductive. Even so malaria-induced anemia is likely to have a substantial effect on child quality and returns to human capital. When children in the southern United States were treated for hookworm, a disease whose primary morbidity consequence is also anemia, teachers reported a remarkable improvement in children's scholastic performance (Bleakley 2007).[15]

Malaria tends to be more fatal in moderate prevalence areas (high d/low i) and relatively less so in high prevalence areas (low d/high i) (Marsh and Snow 1999). A study of children in Tanzania by Reyburn et al. (2005) concludes that higher case fatality rates can be attributed to a higher occurrence of the more fatal cerebral malaria in low transmission areas.[16] Thus malaria's δ parameter tends to be higher in low i/high d areas and lower in high i/low d areas, and theoretically, the fertility response to malaria is stronger for the former and weakest for the latter.

In endemic areas adults have built up some immunity since childhood, and malaria infections in children are more quickly recognized as such, and therefore patients receive prompter treatment, avoiding fatalities.[17] Delaying treatment by 5-10 days raises case fatality by a factor of 5, and delay of 10-20 days raises case fatality by a factor of 20. Despite similar levels of nutrition and health care access, in Sri Lanka, case fatality is 0.01% in endemic areas versus 1% in non-endemic areas because of quicker diagnosis in endemic areas (Alles et al. 1998). In endemic areas malaria fatalities are mostly restricted to children, while in areas that experience periodic influxes of malaria, malaria fatalities heavily affect all age groups.

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[15]The Rockefeller Sanitary Commission began a hookworm eradication campaign in 1910 after discovering that 40% of school-aged children were infected with the parasite.

[16]Lower transmission areas are those at higher altitudes where fewer mosquitoes live.

[17]Genetic adaptations have been discovered in groups of people living in malaria-intense regions; hemoglobin-related disorders and other blood cell dyscrasias are more prevalent in malaria endemic areas and are thought to provide protection from malarial disease. Acquired immunity from exposure is strain-specific and is lost if a person moves away from a malaria endemic area (Center for Disease Control).

Table III.2: Contribution of malaria to under age five child mortality (%), 2000

Country	Percentage of child deaths due to Malaria	Neonatal causes
Angola	8.3	22.2
Benin	27.2	25.0
Botswana	0	40.3
Burkina Faso	20.3	18.3
Burundi	8.4	23.3
Cameroon	22.8	24.8
Central African Republic	18.5	27.2
Chad	22.3	24.0
Comoros	19.4	37.3
Congo	25.7	30.9
Cote d'Ivoire	20.5	34.9
Dem. Rep. of the Congo	16.9	25.7
Equatorial Guinea	24.0	27.5
Eritrea	13.6	27.4
Ethiopia	6.1	30.2
Gabon	28.3	35.1
Gambia	29.4	36.6
Ghana	33.0	28.5
Guinea	24.5	28.8
Guinea-Bissau	21	24.1
Kenya	13.6	24.2
Lesotho	0	32.8
Liberia	18.9	29.1
Madagascar	20.1	25.6
Malawi	14.1	21.7
Mali	16.9	25.9
Mauritania	12.2	39.4
Mauritius	0	66.0
Mozambique	18.9	29.0
Namibia	0	38.5
Niger	14.3	16.7
Nigeria	24.1	26.1
Rwanda	4.6	21.7
Senegal	27.6	22.8
Seychelles	0	27.2
Sierra Leone	12.4	21.9
South Africa	0	35.1
Swaziland	0.2	26.8
Togo	25.3	29
Uganda	23.1	23.6
Tanzania	22.7	26.9
Zambia	19.4	22.9
Zimbabwe	0.2	28.1
Average	14.87	29.11

Source: World Health Organization

Accordingly, reducing malaria transmission will have a stronger impact on fertility in moderate transmission areas, since that is where case fatality rates are higher. Conversely, the reduction in mortality per fatality averted through treatment of malarial infections is higher in high transmission areas and lower in moderate transmission areas. More anemic children survive, for example, but the average health quality of children declines less in high transmission areas where malarial infections are relatively milder (δ is lower); parents have less incentive to increase the quantity of children to replace quality loss in high transmission areas. From a population control standpoint, reducing transmission via dissemination of bed nets or insecticide to eradicate mosquito populations is preferred to treatment of existing infections in moderate transmission settings, and the opposite holds for high transmission settings. This allocation of resources will most effectively reduce fertility and improve human capital.

III.5 Empirics

Panel regressions using country-level data on malaria incidence for 1985-2005 across 44 countries in sub-Saharan Africa confirms that the TFR response to a change in malaria prevalence is stronger in non-endemic malarial regions relative to endemic regions. Where malaria is endemic (high i/low d), malaria-caused mortality is restricted mostly to children, since surviving adults have acquired partial immunity throughout their lives. In moderate malaria prevalence areas (moderate i/high d), malaria affects both child and adult mortality. This information can be exploited to identify endemic versus non-endemic malarial regions. The following specification is tested:

$$TFR_{it} = \beta_o + \beta_1 Malaria\ prevalence_{it} + \beta_2 Malaria\ prevalence * Adult\ mortality_{it} + \beta_3 Adult\ mortality_{it} + \beta_4 Child\ mortality_{it} + \beta_5 GDP\ per\ capita_{it} + \epsilon_{it}.$$

The model predicts that the TFR response to malarial prevalence will be stronger and more positive in non-endemic regions, where adult mortality associated with malaria is higher. The expected sign for β_2 is positive.

Using the same data, one can test whether the response of fertility to child mortality is amplified (more positive) where morbidity moves in the same direction as mortality. A decline in case fatality rates may actually raise morbidity, since more children survive disease and may suffer long-term health problems. Thus a reduction in child mortality will have a larger downward impact on TFR if morbidity falls also, that is, if disease prevalence also falls. The following specification is

tested:

$$TFR_{it} = \alpha_o + \alpha_1 Child\ mortality_{it} + \alpha_2 Child\ Mortality * Malaria\ prevalence_{it} + \alpha_3 Malaria\ prevalence_{it} + \alpha_4 Adult\ mortality_{it} + \alpha_5 GDP\ per\ capita_{it} + \mu_{it}.$$

The expected sign for α_2 is positive. If disease prevalence declines, then so does morbidity, and this magnifies the positive TFR response to child mortality.

Since human capital and fertility are inversely related, replacing TFR with a measure of education in the two regressions above should yield the opposite coefficient signs. If a decline in disease prevalence lowers TFR more where case fatalities are higher, then education should rise more in response to a decline in disease prevalence in non-endemic regions where case fatality rates are higher (and where malaria-related adult mortality is higher). A negative coefficient on the interaction of malaria prevalence and adult mortality is expected in the first specification above when education replaces TFR as the dependent variable. If a decline in child mortality lowers TFR more if morbidity, or disease prevalence, falls also, then a reduction in child mortality should increase education more if disease prevalence falls more. A negative coefficient on the interaction of child mortality and malaria prevalence is expected in the second specification above when education replaces TFR as the dependent variable.

GDP per capita is included in each regression as a control variable. GDP per capita does not affect parents' fertility choice in the model, but human capital investment is increasing in income. In reality wealthier parents can devote more resources to protecting their children from disease, so more children survive and they tend to be healthier; parents have fewer children since the precautionary motive is weaker and because parents replace quantity of children with quality. A negative coefficient on GDP per capita is expected in the fertility regressions, although theoretically the coefficient is zero. A positive coefficient is expected in the education regressions.

An increase in adult mortality is equivalent to an increase in β in the model developed above; parents place more emphasis on current consumption and less on future consumption. Theoretically, both fertility and human capital investment in children decline, since parents benefit from these only in retirement.[18]

Country effects account for cultural, institutional and other unobserved, time-invarying country characteristics that affect fertility rates, and year effects account for shocks affecting all

[18]However, children could also be modeled as consumption goods (see Chapter IV); parents gain utility from having a family, so fertility rises with adult mortality as parents consume more resources now and invest less for the future.

countries simultaneously. In countries with weak institutions, health and education infrastructure are less adequate, so disease prevalence and mortality rates may be higher. With less effective dissemination of contraceptives and knowledge about family planning, TFR may be higher. Over the sample period, TFR and child mortality rates generally decline while adult mortality and GDP per capita generally follow an upward trend. Thus in regressions without country fixed effects, the coefficients on mortality rates may be biased upwards, and in regressions omitting time effects, the coefficients on child mortality may be biased upward while those on adult mortality and GDP per capita may be biased downward.

1 Data

Table III.3 summarizes the data collected on 44 African countries for 1985-2005.[19] Data for each country and year is not always available, but panel regression analysis allows maximum exploitation of all variation across countries and years that are available. Data is interpolated to maximize observations, and Table III.3 shows descriptive statistics for both original and interpolated variables. Data on adult, infant and child mortality rates and GDP per capita are from the World Bank's World Development Indicators. Children's education statistics are from the World Health Organization (WHO). Reported malaria cases for 1982-1997 from the WHO's Weekly Epidemiological Record are supplemented with reported malaria cases for 1990-2007 from the WHO's Global Malaria Programme, and these proxy for malaria prevalence.

As seen in Table III.3, malaria prevalence rates vary substantially both between countries and across time, with malaria incidence rates as high as 95%. Most of the variation in mortality rates is between countries rather than across time. Mortality rates are as high as 19% for infants, 32% for children under five years of age and 74% for adults, where adult mortality is defined as the probability of death between age 15 and 60. TFR varies from 1.91 to 8.35 births per woman.

Children's education is regressed on malaria prevalence using three different measures of education: primary school completion rate and primary school gross and net enrollment rates. Gross enrollment rates measure the number of children in primary school as a proportion of total children

[19]Countries: Angola, Benin, Botswana, Burkina Faso, Burundi, Cameroon, Central African Republic, Chad, Comoros, Congo Democratic Republic, Congo Republic, Cote D'Ivoire, Equatorial Guinea, Eritrea, Ethiopia, Gabon, Gambia, Ghana, Guinea, Guinea-Bissau, Kenya, Lesotho, Liberia, Madagascar, Malawi, Mali, Mauritania, Mauritius, Mozambique, Namibia, Niger, Nigeria, Rwanda, Senegal, Seychelles, Sierra Leone,[20] South Africa, Sudan, Swaziland, Tanzania, Togo, Uganda, Zambia, Zimbabwe.

of primary school age, so the gross enrollment rate could be above 100%. The net enrollment rate counts only children of primary school age, so the maximum net enrollment rate possible is 100%. In regressions of education, year fixed effects are included because, for example, an increase in foreign aid one year could increase education expenditures and thereby increase school enrollment. Country fixed effects account for country differences such as institutions, which can affect the efficacy and influence of the education system.

2 Regression Results

Table III.4 presents the results for regressions of TFR on malaria according to regional epidemiology. The response of TFR to malaria prevalence is stronger and more positive where adult mortality is higher. This confirms the model's prediction that reducing malaria prevalence reduces TFR more in moderate prevalence areas. The result is significant only when country fixed effects are omitted. Malaria depends partially on health interventions (mosquito nets, pesticide) but also largely on geographical characteristics such as climate and elevation, factors that change little, or not at all, within a region over time. Including country fixed effects introduces a large degree of collinearity between malaria prevalence and country fixed effects into the model. Given the data limitations, there is not sufficient variation within countries across time in the malaria prevalence measure to get significant results.[21] What is of interest is how malaria prevalence, not geography or climate, affects TFR. Plotting the predicted TFR for each country across time in Figure III.4 shows the specification to be quite accurate even without country fixed effects.

Table III.5 presents the results for regressions of TFR on child mortality according to malarial morbidity changes. The coefficients on child mortality and its interaction with malaria prevalence are both positive, suggesting that declines in child mortality have a greater downward impact on TFR when accompanied by declines in morbidity. Once again, the result is not robust to including country fixed effects, as explained above. Figure III.5 compares actual and predicted TFR.

Tables III.6 and III.7 present results for the education regressions. When primary school completion rate is substituted for TFR as the dependent variable in the same right-hand-side specifications, the coefficient estimates change signs, supporting a negative relationship between TFR and human capital investment. Furthermore, where TFR responds more strongly and positively to

[21] A random effects model does not pass the Hausman test for consistency and is thus inappropriate here.

child mortality and morbidity, education does so as well, as evidenced by the negative coefficient on the interaction of malaria prevalence and adult mortality in Table III.6 and on the interaction of malaria prevalence and child mortality in Table III.7. The results are not robust to different measures of human capital investment, particularly gross and net enrollment rates, the results of which are available upon request.

III.6 Conclusion

By building on the mortality-fertility literature, this paper explores the consequences for fertility of reducing disease burden in developing countries. The results suggest that health initiatives can have different effects on fertility depending on the morbidity and mortality associated with the disease in question. The strongest positive response of fertility to disease prevalence occurs where both mortality and morbidity rates change in the same direction.

That reducing disease burden may raise fertility rates does not contradict the existing consensus that fertility follows child mortality. Rather it highlights that health initiatives are most effective at reducing fertility when they also tackle morbidity. Nor do the results suggest that disease burden should not be tackled if it risks raising fertility. Health policies that increase fertility or cause a response so weak that population growth actually rises should be complemented with education subsidies, for instance, so that parents can afford to educate their larger families.

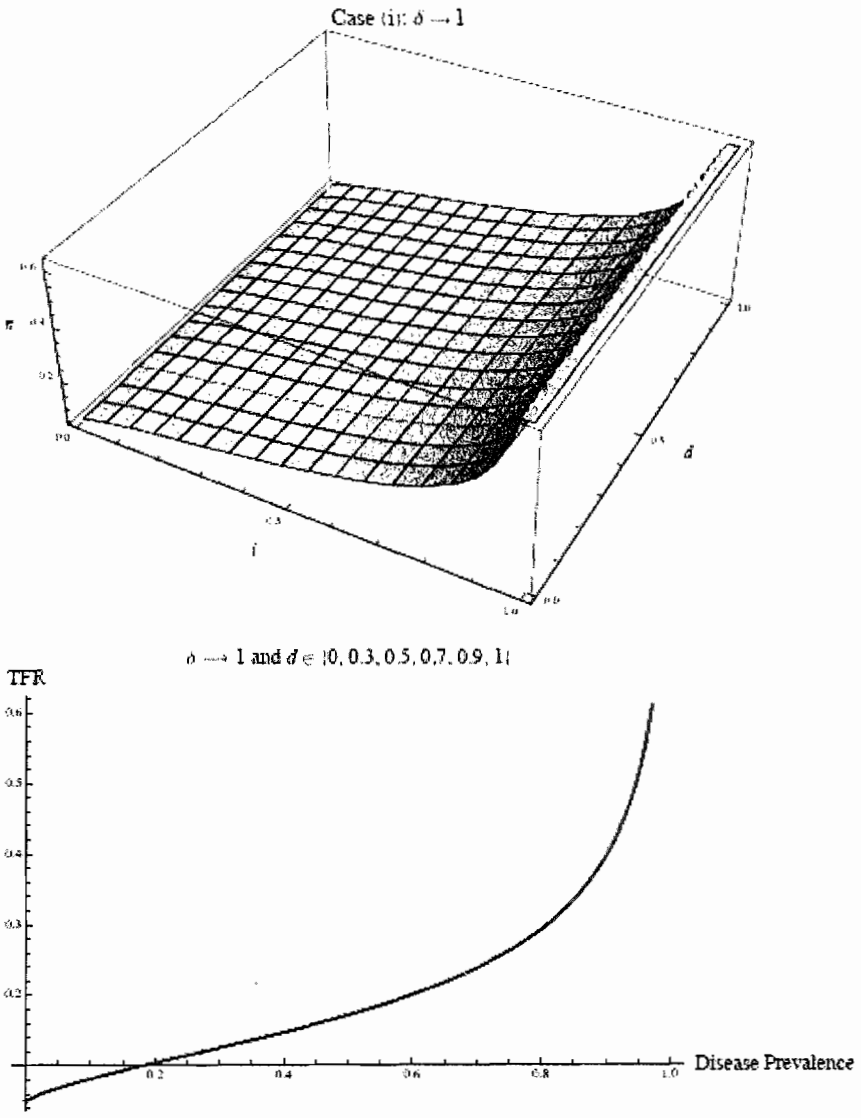

Figure III.1: Fertility response to disease burden when $\delta = 1$

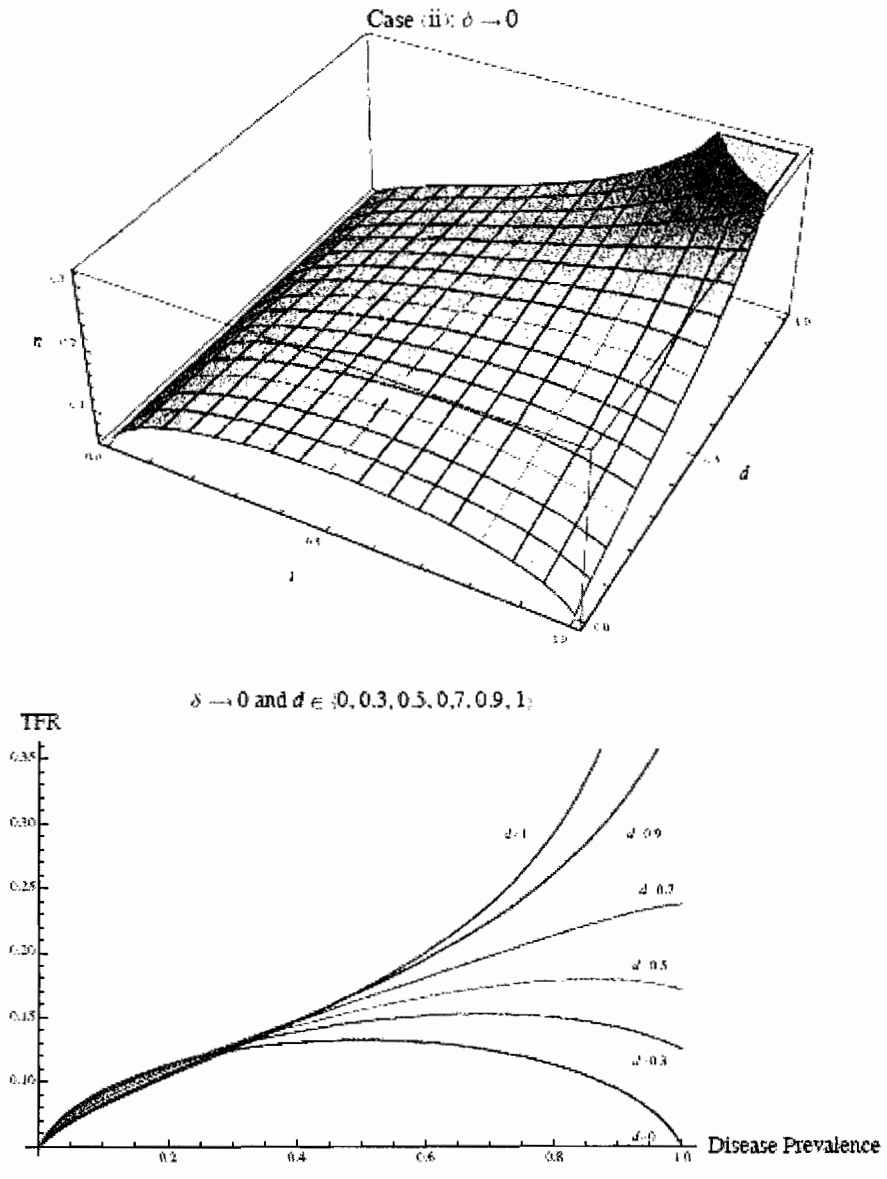

Figure III.2: Fertility response to disease burden when $\delta = 0$

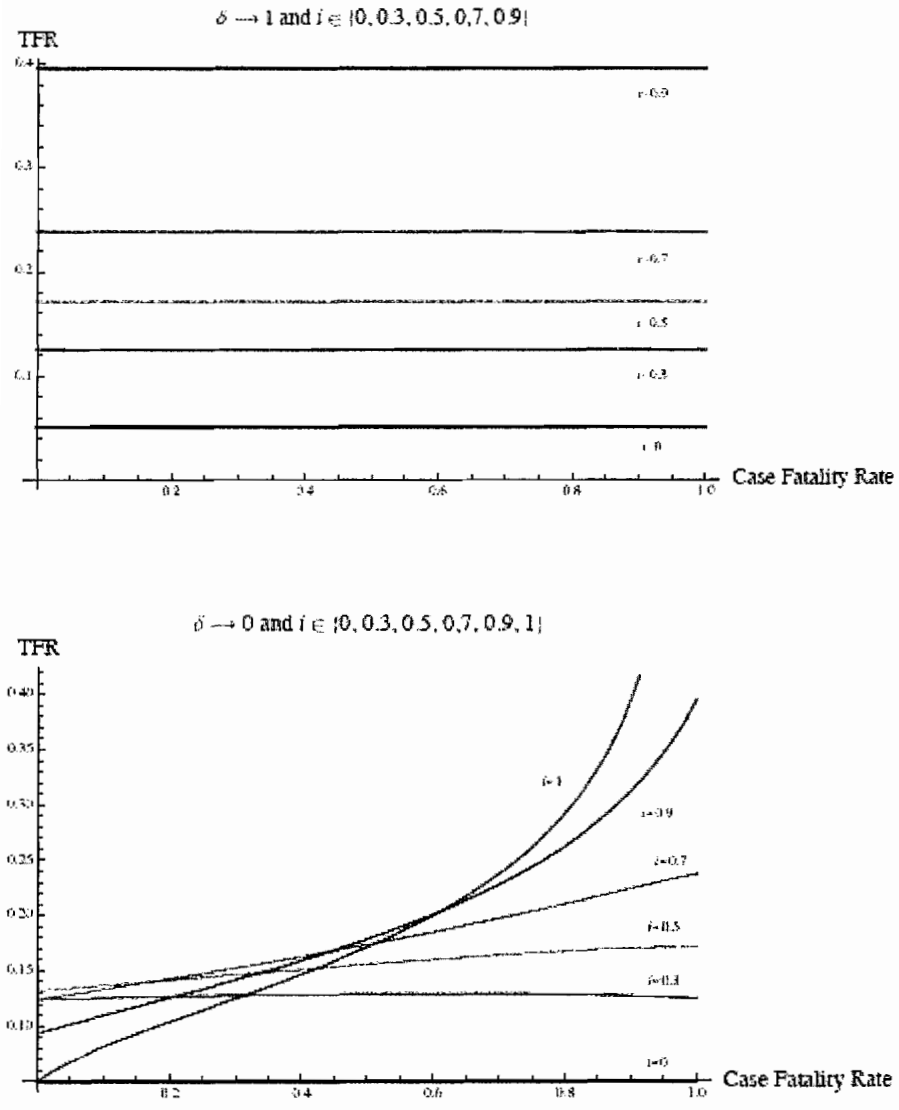

Figure III.3: Fertility response to the case fatality rate

Table III.3: Descriptive statistics

Variable		Mean	Std. Dev.	Min	Max	Observations
Malaria rate	overall	9.80	11.40	0.003	94.58	N=663
	between		9.23	0.006	37.64	n=42
	within		7.56	-15.52	78.49	T-bar=15.79
iMalaria rate	overall	9.68	11.31	0.003	94.58	N=795
	between		8.69	0.006	35.71	n=42
	within		7.40	-15.94	79.55	T-bar=18.93
GDP per capita	overall	919.71	1508.71	62.89	15549.69	N=903
	between		1322.65	154.94	6327.57	n=44
	within		731.66	-2902.11	13939.72	T-bar=20.52
iGDP per capita	overall	912.30	1501.10	62.89	15549.69	N=914
	between		1322.57	154.94	6327.57	n=44
	within		727.24	-2909.52	13932.31	T-bar=20.77
Infant mortality	overall	9.56	3.51	1.22	19.10	N=682
rate	between		3.46	1.42	16.83	n=43
	within		0.71	6.57	12.33	T=15.86
iInfant mortality	overall	9.56	3.49	1.22	19.10	N=688
rate	between		3.46	1.42	16.83	n=43
	within		0.71	6.57	12.33	T=16
Under 5 mortality	overall	15.65	6.41	1.33	32.00	N=217
rate	between		6.20	1.76	29.22	n=44
	within		1.75	9.40	21.38	T-bar=4.93
iUnder 5 mortality	overall	15.60	6.35	1.33	32.00	N=919
rate	between		6.24	1.73	29.30	n=44
	within		1.51	9.31	21.29	T-bar=20.89
Adult mortality	overall	38.95	10.36	16.28	74.15	N=682
rate	between		9.15	17.78	56.27	n=43
	within		5.04	11.28	62.05	T=15.86
iAdult mortality	overall	38.87	10.35	16.28	74.15	N=688
rate	between		9.15	17.78	56.27	n=43
	within		5.02	11.20	61.96	T=16
Total fertility	overall	5.53	1.47	1.91	8.35	N=422
rate	between		1.22	2.10	7.68	n=44
	within		0.52	4.03	7.48	T-bar=9.59
iTotal fertility	overall	5.69	1.29	1.91	8.35	N=924
rate	between		1.21	2.10	7.68	n=44
	within		0.49	4.23	7.50	T=21
iPrimary school	overall	49.14	23.48	10.47	120.70	N=548
completion rate	between		23.78	18.51	114.98	n=42
	within		7.21	14.20	80.67	T-bar=13.05
iPrimary school	overall	83.64	27.89	19.50	172.86	N=622
enrollment rate	between		25.76	34.27	150.61	n=44
(gross)	within		10.91	36.95	133.50	T-bar=14.14
iPrimary school	overall	60.52	19.57	14.71	99.40	N=510
enrollment rate	between		17.92	29.38	95.52	n=43
(net)	within		7.90	26.87	93.04	T-bar=11.86

Prefix "i" denotes interpolated variables. The negative minimum for GDP per capita within occurs because it is showing the variation of GDP per capita within country around the global mean of 919.713.

Table III.4: Panel regressions of TFR response by epidemiology to malaria prevalence

	Expected	1	2	3	4	5	6
Log(Malaria Prevalence)	?	-0.897***	-0.362	-0.733***	-0.740***	-0.0179	-0.0171
		(0.214)	(0.562)	(0.219)	(0.215)	(0.403)	(0.397)
Log(Malaria Prevalence)*Log(Adult Mortality)	+	0.255***	0.0826	0.216***	0.218***	0.0062	0.0061
		(0.059)	(0.151)	(0.0611)	(0.0601)	(0.108)	(0.107)
Log(Adult Mortality)	-	-1.298***	-2.450***	-0.823**	-0.852**	-0.687*	-0.685*
		(0.328)	(0.418)	(0.384)	(0.375)	(0.39)	(0.381)
Log(Child Mortality)	+	2.302***	2.280***	1.999***	2.028***	0.486	0.489
		(0.234)	(0.57)	(0.282)	(0.272)	(0.442)	(0.438)
Log(GDP per capita)	-	-0.331***	-0.0954	-0.379***	-0.367***	-0.0374	-0.0448
		(0.064)	(0.0842)	(0.0708)	(0.0683)	(0.096)	(0.0749)
Year	-				-0.0502***		-0.0560***
					(0.00833)		(0.00825)
Country FE		No	Yes	No	No	Yes	Yes
Year FE		No	No	Yes	No	Yes	No
Observations		613	613	613	613	613	613
R^2		0.854	0.965	0.886	0.884	0.982	0.982

Robust standard errors clustered by country (*** p<0.01, ** p<0.05, * p<0.1)

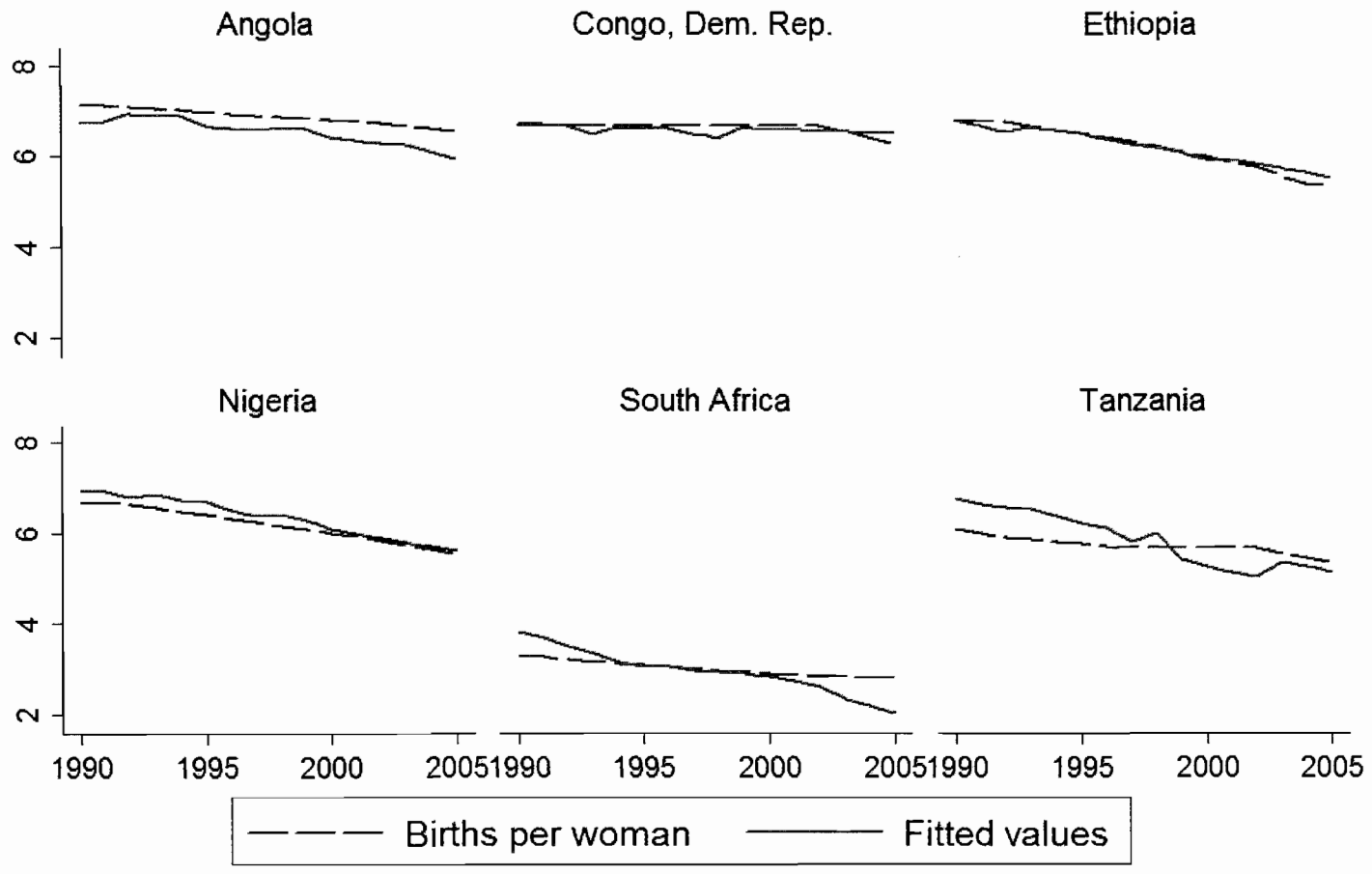

Figure III.4: Predicted TFR response to malaria prevalence according to epidemiology by country

Table III.5: Panel regressions of TFR response by morbidity change to malaria mortality

	Expected	1	2	3	4	5	6
Log(Malaria Prevalence)	?	-0.207***	-0.0279	-0.164**	-0.164**	-0.0931	-0.0935
		(0.0706)	(0.124)	(0.075)	(0.0728)	(0.103)	(0.097)
Log(Malaria Prevalence)*Log(Child Mortality)	+	0.0952***	-0.0106	0.0882***	0.0884***	0.0356	0.0358
		(0.0257)	(0.0452)	(0.0276)	(0.0269)	(0.0386)	(0.0362)
Log(Child Mortality)	+	2.101***	2.247***	1.826***	1.854***	0.424	0.427
		(0.235)	(0.553)	(0.274)	(0.265)	(0.428)	(0.423)
Log(Adult Mortality)	-	-0.863**	-2.343***	-0.443	-0.469	-0.642**	-0.643**
		(0.342)	(0.28)	(0.376)	(0.369)	(0.298)	(0.292)
Log(GDP per capita)	-	-0.362***	-0.0998	-0.404***	-0.391***	-0.0398	-0.0444
		(0.0679)	(0.0854)	(0.0735)	(0.0711)	(0.0952)	(0.074)
Year	-				-0.0522***		-0.0565***
					(0.00865)		(0.00816)
Country FE		No	Yes	No	No	Yes	Yes
Year FE		No	No	Yes	No	Yes	No
Observations		613	613	613	613	613	613
R^2		0.849	0.965	0.884	0.882	0.982	0.982

Robust standard errors clustered by country (*** p<0.01, ** p<0.05, * p<0.1)

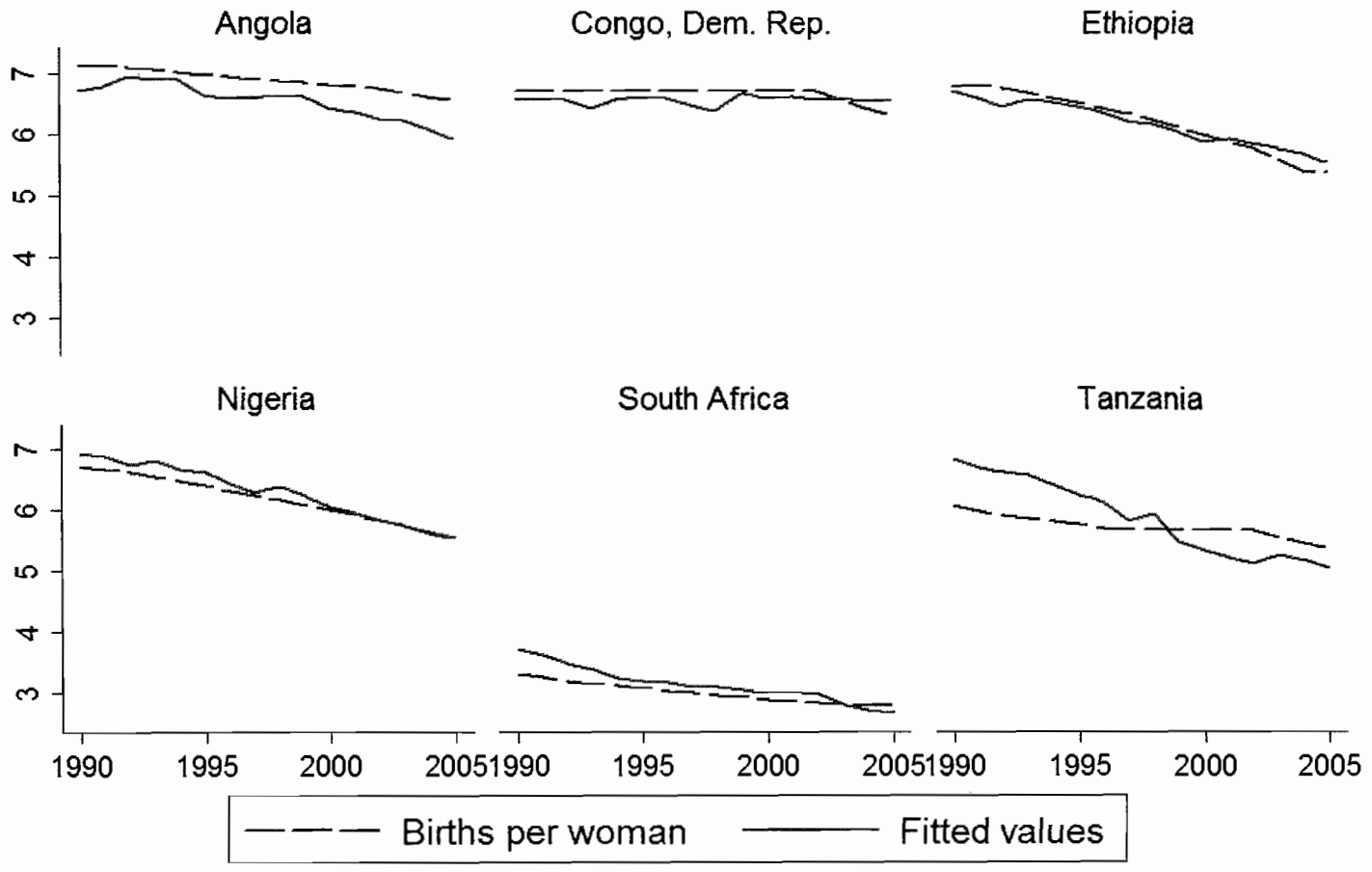

Figure III.5: Predicted TFR response to malaria mortality according to morbidity change by country

Table III.6: Panel regressions of education response by epidemiology to malaria prevalence

	Primary school completion rate						
	Expected	1	2	3	4	5	6
Log(Malaria Prevalence)	+	13.00**	25.40***	14.19**	13.29**	18.43**	17.82**
		(5.4)	(6.317)	(5.831)	(5.765)	(7.61)	(7.385)
Log(Malaria Prevalence)*Log(Adult Mortality)	-	-2.990*	-6.601***	-3.321*	-3.063*	-4.944**	-4.789**
		(1.57)	(1.679)	(1.677)	(1.654)	(2.051)	(1.995)
Log(Adult Mortality)	+	45.32***	23.03***	47.45***	45.93***	-2.974	-4.191
		(4.354)	(6.21)	(5.387)	(5.401)	(9.123)	(9.424)
Log(Child Mortality)	-	-51.69***	-42.35***	-53.59***	-52.14***	-16.28	-16.25
		(4.75)	(12.25)	(5.356)	(5.474)	(12.1)	(11.98)
Log(GDP per capita)	+	4.674**	8.070**	4.068**	4.592**	7.202*	7.970**
		(1.817)	(3.106)	(1.913)	(1.932)	(4.02)	(2.945)
Year	+				-0.0645		0.859***
					(0.284)		(0.231)
Country FE		No	Yes	No	No	Yes	Yes
Year FE		No	No	Yes	No	Yes	No
Year trend		No	No	No	Yes	No	Yes
Observations		489	489	489	489	489	489
R^2		0.783	0.923	0.791	0.783	0.934	0.933

Robust standard errors clustered by country (*** p<0.01, ** p<0.05, * p<0.1)

Table III.7: Panel regressions of education response by morbidity change to malaria mortality

	Primary school completion rate						
	Expected	1	2	3	4	5	6
Log(Malaria Prevalence)	+	4.699***	7.274**	4.835***	4.729***	9.209**	8.654**
		(1.648)	(3.299)	(1.681)	(1.696)	(3.402)	(3.25)
Log(Malaria Prevalence)*Log(Child Mortality)	-	-1.022	-2.210*	-1.084	-1.029	-3.290**	-3.102**
		(0.822)	(1.285)	(0.828)	(0.83)	(1.316)	(1.261)
Log(Adult Mortality)	+	40.05***	14.30*	41.16***	40.22***	-14.43	-14.98
		(4.174)	(7.884)	(4.749)	(4.745)	(8.862)	(9.041)
Log(Child Mortality)	-	-48.96***	-36.23***	-50.15***	-49.10***	-6.351	-6.824
		(4.04)	(12.06)	(4.424)	(4.523)	(10.14)	(10.15)
Log(GDP per capita)	+	5.132***	8.343**	4.675**	5.107***	7.574*	8.035**
		(1.76)	(3.184)	(1.817)	(1.839)	(4.035)	(3.021)
Year	+				-0.0233		0.959***
					(0.277)		(0.217)
Country FE		No	Yes	No	No	Yes	Yes
Year FE		No	No	Yes	No	Yes	No
Year trend		No	No	No	Yes	No	Yes
Observations		489	489	489	489	489	489
R^2		0.781	0.92	0.787	0.781	0.934	0.933

Robust standard errors clustered by country (*** p<0.01, ** p<0.05, * p<0.1)

CHAPTER IV

THE DYNAMICS OF DISEASE AND ECONOMIC DEVELOPMENT

IV.1 Introduction

Once in a stagnant state of high and volatile mortality, short lifespan and negligible population growth, today's industrialized countries experienced an "age of receding pandemics"; mortality declined substantially while longevity and population grew, converging to a state of relatively little and stable infectious disease (Omran 1971). During the epidemiological transition of England and Wales, which epitomizes the experience of many industrialized countries, the decline in infectious diseases, those overwhelmingly afflicting the young, began approximately 30 years before did the decline in non-infectious, those afflicting primarily the elderly (Figure IV.1). These events imply that the decline in non-infectious diseases began when the first cohort of children experiencing reduced exposure to infectious disease reached adulthood. This chapter models the link between disease experience in childhood and adulthood in order to capture the timing structure of the epidemiological transition as it occurred historically and to better understand how persistent disease morbidity prevents many countries from completing and benefiting from a similar transition.

Considerable biological evidence shows that surviving infection early in life increases susceptibility to other diseases later in life; the introduction in Chapter III provides specific examples. Anthropometric measures of health, in particular height, have been used as an objective measure of lifelong or cumulative health. Average annual growth in height is greatest during infancy, slowing until adolescence, during which its velocity peaks at one-half of infancy rates, before slowing to zero at maturity; stunting during infancy has the most adverse consequences on stature (Fogel 1986). Historical data on stature in Western countries reveal that cohorts of children who experienced declining mortality rates became taller (and thus healthier) adults relative to their predecessors, suggesting morbidity declined in tandem with mortality (Arora 2005). Having escaped much of the childhood infectious disease burden experienced by their predecessors, it is presumably

these taller and healthier adults who then more effectively avoided non-infectious diseases, those afflicting primarily older age groups.

Many developing regions have been experiencing a rising trend in adult height, but sub-Saharan Africa is a large exception (Akachi and Canning 2008). Despite substantial improvements in child survival probabilities since 1960, children in sub-Saharan Africa are not growing up to be taller than their parents (at least not at the expected rate), suggesting that morbidity remains high: fatalities from disease have declined among children, but disease remains a common experience. This may be stalling the demographic transition there and thus the quantity-quality tradeoff for children.

In England and Wales fertility rates began to decline approximately five years after did deaths from infectious diseases (Arora 2005). When child mortality rates are high, parents compensate by having additional children. Higher fertility rates leave fewer resources for quality investment in children, and quality investment in any individual child is risky anyways, since there exists a substantial probability that the child will not reach adulthood and bring that investment to fruition. Experiences of malnutrition and disease during infancy especially, may have stunting impacts, and stunting in the first two years of life is associated significantly with poor cognitive performance in later childhood and reduced work capacity in adulthood, which depreciates wages and economic productivity (Akachi and Canning 2008, Schultz 2002, Schultz 2005). Thus when either mortality or morbidity is high, the returns to quality investment in children are lower, and parents have more children but of lower quality. Non-infectious diseases affect primarily older age groups, but infectious diseases affect the young also, so in less developed countries the high disease burden stifles human capital accumulation and labor productivity.

I extend the model from Chapter III and develop a three-period, overlapping generations model that endogenizes adult longevity to disease outcomes during childhood. As before, I continue to distinguish between infection and case fatality rates. I model the generational lag between the decline in infectious and non-infectious diseases by introducing a function, $\phi \in (0,1)$, which represents the fraction of late adulthood that individuals live for. Longevity, ϕ, is increasing in health capital, which depreciates by fraction $\delta \in (0,1)$ during bouts of infection in childhood. Parental investment reduces the probability of infection during early childhood and therefore disease later in life.

As childhood disease burden declines, the population becomes healthier and less likely to die prematurely, so savings and thus economic growth improve. The epidemiological transition from high

to low disease prevalence triggers a quantity-quality tradeoff for children: fertility declines with disease burden, and quality investment in children rises. Rising wealth, however, raises fertility rates.

Previous studies employ a variety of modeling techniques in order to study fertility and mortality. Sah (1991), Kalemli-Ozcan (2003, 2008) and Tamura (2006) approach the problem by introducing uncertainty into parents' utility maximization problems, so that the variance about child survival outcomes rather than just average outcomes affect fertility and human capital investment decisions. Given the log-utility preference structure in which children are investment goods, incorporating uncertainty achieves the empirically observed positive relationship between child mortality and fertility. This is the approach followed in Chapter III of this dissertation. In Chapter IV I adopt the preference structure of Strulik (2008), which assumes certainty about parents' expectations of their children's health and survival outcomes, and also parental investment affects survival outcomes and provides a "warm glow" to parents. Children are not investment goods as in Chapter III but rather consumption goods, and this preference structure generates the desired positive relationship between child mortality and utility. Unlike Strulik (2008) the model below distinguishes between infection and case fatality rates and addresses the role of morbidity.

Birchenall (2007) also distinguishes between infection and case fatality rates and the resulting impact on labor productivity, but to my knowledge no study explores how the link between disease in childhood and disease later in life affects fertility and human capital investment choices. In Morand (2004) human capital passes from parents to children, but there exists no physiological transmission of health across generations, and human capital grows because adults invest in themselves.[1] In the model below, human capital improves naturally as childhood disease morbidity declines, and since improved health raises the expected returns to human capital investment, parents invest more in the human capital of their children in low disease environments. Morand (2004) also allows health capital to influence longevity, but as there is no fertility choice, and adults invest in themselves rather than in their children, there is no quantity-quality tradeoff, as in the model below.

I present the general equilibrium model in Section IV.2. Simulation results are discussed in Section IV.3, and Section IV.4 concludes and proposes some extensions.

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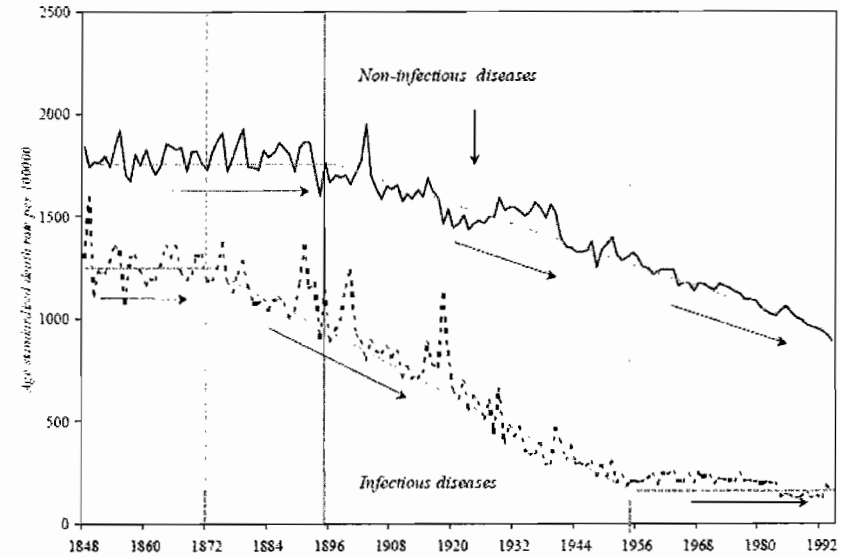
[1] Agents invest in human capital only once physical capital has accumulated to the level at which returns to physical capital are sufficiently low.



Source: Arora (2003)

Figure IV.1: Infectious and non-infectious disease-caused mortality in England

IV.2 The Model

1 Household Preferences

Unlike Chapter III there is no uncertainty regarding child health and survival outcomes; i fraction of children born contract a disease, and of those children d fraction do not survive. Parental investment, h, affects survival outcomes of children directly by reducing the chance of infection according to

$$i_t = \overline{i_t} - \psi h_t \tag{1}$$

where $\overline{i_t}$ is the exogenous infection rate at time t, $\psi > 0$ is a parameter determining the effectiveness of h in reducing i, and $i_t \in (0,1)$ for $\psi(\overline{i_t} - 1) \leq h_t \leq \overline{i_t}/\psi$.[2] For example, proper nutrition during pregnancy and infancy and vaccination during infancy improve child survival and later school attainment and income earnings (Behrman and Rosenzweig 2001). Since investment occurs at the beginning of a child's life before survival outcomes are realized, parents invest the same amount in the quality of both healthy and unhealthy children, unlike Chapter III. Health quality depreciates by a fraction δ for those children who experience but survive a disease. With probability $(1 - i)$, a child

[2] A specification of $i(h)$ that is inherently bounded between zero and one, such as $i = \bar{i}/(1 + \psi h)$, would be preferred and would likely yield smoother dynamics, but it greatly complicates the algebra.

enters adulthood with health capital stock h^θ and with probability $i(1-d)$ a child enters adulthood with health capital stock $(1-\delta)h^\theta$, where d is the case fatality rate, and $\theta \in (0,1)$. This stock determines labor productivity during working-age adulthood, as well as longevity, ϕ, during late adulthood; longevity is increasing in health capital.

During early adulthood parents work, consume c, have n children, invest h in each child and save s for consumption during retirement. Unlike Chapter III there is no fixed time cost per child born. Working-age adults will consume their savings during retirement, so unlike Chapter III parents do not depend on their children for financial support during old age. Working-age adults expect to live for ϕ fraction of late adulthood, so they make these decisions based on their own expected longevity. Children with higher health capital earn more as working-age adults and also live longer, so in addition to h improving child survival probabilities, parents receive a "warm glow" from maximizing the health capital of their children. The unconsumed savings of those adults who die prematurely are transferred to those remaining in the form of a higher return on savings.[3]

Working-age adults at time t choose c_t, n_t, h_t and s_t to maximize utility

$$U_t = ln(c_t) + \beta ln(w_{t+1} n_t h_t^\theta [1 - i_t(h_t) + i_t(h_t)(1-d)(1-\delta)]) + \phi_t ln(c_{t+1}) \tag{2}$$

subject to

$$c_t = w_t H_t - h_t n_t - s_t \tag{3}$$

$$c_{t+1} = \frac{R_{t+1}}{\phi_t} s_t \tag{4}$$

where w_t is the wage per worker, and R_{t+1} is the interest rate, although R_{t+1}/ϕ_t is the effective interest rate, and it is increasing in adult mortality. Healthy parents, those who avoided disease as children, earn more income;

$$H_t = h_{t-1}^\theta \quad if\ healthy \tag{5}$$

$$= (1-\delta) h_{t-1}^\theta \quad if\ unhealthy \tag{6}$$

[3]This method is followed in Chakraborty (2004), following Blanchard (1985) and Yaari (1965).

and healthy parents also live longer;

$$\phi_t = \frac{H_t}{1 + H_t} \tag{7}$$

Degenerative diseases such as cancer and diseases affecting the circulatory system are responsible for much of old-age mortality, and individuals who experience infectious disease early in life are predisposed to non-infectious diseases later in life (Arora 2003). For example, bronchitis, pneumonia and whooping cough before age five are linked to diminished respiratory function at ages 59-70 (Barker 1991, 1994). Acute rheumatic fever damages heart valves, and late-stage syphilis, measles and malaria can affect the functioning of the circulatory system. (See the introduction in Chapter III for more examples.) In the model, children who experience but survive infectious disease grow up with lower health capital and are more likely to die from non-infectious disease during retirement: longevity, ϕ, is lower.

First-order conditions yield:

$$n_t = \frac{\beta w_t H_t}{(1 + \beta + \phi_t) h_t} \tag{8}$$

$$s_t = \frac{\phi_t w_t H_t}{1 + \beta + \phi_t} \tag{9}$$

$$h_t = \frac{(1 - \theta)[1 - (\delta + d(1 - \delta))\bar{i}_t]}{\theta\psi(\delta + d(1 - \delta))} \tag{10}$$

Total fertility, n_t, is increasing in $\bar{i}_t$, d, δ, ψ and θ, and parental investment, h_t, is decreasing in these; parents have fewer children and invest more in each if disease is less prevalent, less fatal and less debilitating. Returns to quality investment, h, are lower when risk of mortality or morbidity are higher. Surprisingly, parents have fewer children and invest more in each if h is less effective at reducing the probability of infection (low ψ) and if the quality returns to investment are lower (low θ); however, total expenditure on children, nh, increases in both ψ and θ.[4] The saving rate is increasing in longevity, ϕ, and decreasing in β, the preference parameter for large and high quality families.

Parents optimally invest the same amount h in their children regardless of parental health status. Healthy parents have more children because they can afford to do so (and they also consume more in both periods). A decline in the aggregate infection rate improves the average health of the population, directly through fewer infections and indirectly through higher h and thus even fewer

[4]On a technical note, that parental investment, h, is decreasing in θ and ψ, which capture the returns to h, is consistent with problems encountered in Strulik (2008). However unlike Strulik, although n falls and h rises when $\bar{i}d \to 0$, they do not go to zero and ∞, respectively (unless $\delta \to 0$).

infections. A reduction in case fatality rates will increase subsequent infection rates, all else equal, although a lower case fatality rate raises h, which reduces infections.

Given the optimal solution for h, $i_t(h_t) \leq 1$ holds always, but to ensure a non-negative infection rate, i must be bounded from below in the simulations below.[5]

2 Infection dynamics

The working-age population each period consists of L_t young adults. I assume that children who survive disease remain a source of infection during their working-age years, but for simplicity retired adults are not a source of infection. This implies that contact with adults for young children is limited to working-age adults such as parents, friends of parents, teachers, doctors, etc. At time t there are $L_t n_t$ susceptible children and each encounters β_A adults, of whom $i_{t-1}(1-d)$ fraction are contagious. The aggregate risk of infection is

$$\overline{i_t} = \frac{L_t n_t \beta_A i_{t-1}(1-d)\tau}{L_t n_t} = \beta_A i_{t-1}(1-d)\tau \tag{11}$$

where τ is the disease transmission rate from contact with an infective adult.[6] Then Equation 1 becomes

$$i_t = \beta_A i_{t-1}(1-d)\tau - \psi h_t \tag{12}$$

3 The Economy

Output at time t, Y_t, is produced via a Cobb-Douglas production function using physical capital, K_t, and effective labor in perfectly competitive markets.

$$Y_t = K_t^{\eta}(H_t L_t)^{1-\eta} \tag{13}$$

where $\eta \in (0,1)$. Each working-age adult supplies one unit of labor time inelastically times their human capital, and the aggregate stock of effective labor is

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[5] $i_t(h_t) \geq 0$ holds if $1 - \theta \leq \overline{i_t}(\delta + d(1-\delta))$, that is, if δ is sufficiently small and d and $\overline{i_t}$ are sufficiently large, or if δ and $\overline{i_t}$ are both sufficiently large. To ensure a non-negative infection rate, h_t must be bounded from above by $\overline{i_t}/\psi$; this is smaller the smaller is $\overline{i_t}$, so that the upper bound on h falls as i falls, yet $\partial h/\partial \overline{i_t} < 0$. It is more intuitive for i to be bounded from below than for h to be bounded from above, since h affects utility other than just through the rate of infection.

[6] Another option is to make τ rather than i a function of parental investment h_t, such that $\tau_t(h_t) = \overline{\tau_t} - \psi h_t$. Then $\overline{i_t} = \beta_A(\overline{\tau_t} - \psi h_t)(1-d)i_{t-1}$.

$H_t L_t = L_{t-1} n_{t-1} h_{t-1}^{\theta}(1 - i_{t-1} + i_{t-1}(1-d)(1-\delta))$. Output per worker is

$$y_t = k_t^{\eta} \left(\frac{h_{t-1}^{\theta}(1 - i_{t-1} + i_{t-1}(1-d)(1-\delta))}{1 - i_{t-1}d} \right)^{1-\eta} \tag{14}$$

In competitive market, the wage per worker is equal to the marginal product of labor, or

$$w_t = \frac{\partial Y_t}{\partial L_t} = (1-\eta) k_t^{\eta} \left(\frac{h_{t-1}^{\theta}[1 - i_{t-1} + i_{t-1}(1-d)(1-\delta)]}{(1 - i_{t-1}d)} \right)^{1-\eta} \tag{15}$$

Income per working-age adult is $w_t H_t$ where H_t is δ fraction lower for infective working-age adults. The wage per worker is increasing in the aggregate level of human capital in the economy. Also, the wage is increasing in the child mortality rate last period, $i_{t-1}d$, because the marginal product of labor rises as labor becomes more scarce. On net $\partial w_t / \partial d > 0$, because a higher case fatality rate reduces the infection rate but also the size of labor supply, while $\partial w_t / \partial i_{t-1} < 0$, because more sick people means less productive workers (unless $\delta = 0$).

Let subscripts U and I denote uninfective and infective individuals, respectively. Capital evolves according to

$$\begin{aligned} K_{t+1} &= L_{U,t} s_{U,t} + L_{I,t} s_{I,t} && (16) \\ &= L_{t-1} n_{t-1} [(1 - i_{t-1}) s_{U,t} + i_{t-1}(1-d) s_{I,t}] && (17) \\ &= \frac{L_t [(1 - i_{t-1}) s_{U,t} + i_{t-1}(1-d) s_{I,t}]}{1 - i_{t-1}d} && (18) \end{aligned}$$

where I have used $L_t = (1 - i_{t-1}d) L_{t-1} n_{t-1}$. Then given

$$\begin{aligned} s_{U,t} &= \frac{\phi_t w_t H_t^U}{1 + \beta + \phi_t} = \frac{w_t h_{t-1}^{2\theta}}{1 + \beta + (2+\beta) h_{t-1}^{\theta}} && (19) \\ s_{I,t} &= \frac{\phi_t w_t H_t^I}{1 + \beta + \phi_t} = \frac{w_t h_{t-1}^{2\theta}(1-\delta)^2}{1 + \beta + (2+\beta)(1-\delta) h_{t-1}^{\theta}} && (20) \end{aligned}$$

capital per worker is

$$\begin{aligned} k_{t+1} &= \frac{L_t((1 - i_{t-1}) s_{U,t} + i_{t-1}(1-d) s_{I,t})}{(1 - i_{t-1}d) L_{t+1}} && (21) \\ &= \frac{w_t h_{t-1}^{2\theta}}{(1 - i_{t-1}d)(1 - i_t d_t) n_t} * && (22) \\ &\quad \left(\frac{1 - i_{t-1}}{1 + \beta + (2+\beta) h_{t-1}^{\theta}} + \frac{i_{t-1}(1-d)(1-\delta)^2}{1 + \beta + (2+\beta)(1-\delta) h_{t-1}^{\theta}} \right) && (23) \end{aligned}$$

Capital per worker is increasing in the quality of workers in previous periods; more productive workers earn more so they can save more, and healthier workers live longer so they also have more incentive to save.

4 Dynamics

The dynamics of the model are characterized by equations 23 and, substituting optimal h_t into Equation 12 for infection risk,

$$i_t = \frac{\beta_A \tau i_{t-1}(1-d)(\delta + d(1-\delta)) - (1-\theta)}{\theta(\delta + d(1-\delta))} \tag{24}$$

The steady state infection rate,

$$i^{ss} = \frac{1-\theta}{(\beta_A \tau(1-d) - \theta)(\delta + d(1-\delta))} \tag{25}$$

is positive for $\theta < \beta_A \tau(1-d)$, which is more likely to hold for low fatality rates, d, and a high rate of contagion, β_A and τ. However, this is precisely the condition for which the steady state is unstable. A stable steady state requires $\theta > \beta_A \tau(1-d)$, so the stable steady state infection rate is negative. In the simulations below i is bounded from below to at 2%. If i is instead bound by 0%, the decisions of working-age adults are affected by the case fatality rate, d, and the degree of morbidity, δ, even when disease prevalence is zero. Moreover, a positive long-run level of infection is consistent with empirical observation.

IV.3 Simulation Results

In order to discover how the fertility and investment decisions of parents affect the economy over time, I simulate the model across several generations. Its behavior depends on parameter assumptions and initial conditions.

The number of adults a young child encounters is set to $\beta_A = 0.9$.[7] The disease transmission rate is a fraction $\tau = 0.9$, where the closer the fraction is to one, the more persistent is infection. The returns to human capital investment, h, must be less than one, as a linear human capital production

[7] One could allow β_A, the number of adults that a child encounters, to rise with population.

function renders the model indeterminate; θ is set to 0.8. The effectiveness of h in reducing the chance of infection is determined by ψ, which is set to 0.1, and this parameter functions primarily as a scaling factor in the simulations. As seen below, this level of ψ causes birth rates to remain below the population replacement level, while lower values of ψ lead to exponential growth of population, and this threshold is reached at higher levels of ψ for low δ and d. I assume that physical and human capital contribute equally to production, or $\eta = 0.5$. Finally, $\beta = 0.5$; parents value their children moderately, relative to their own consumption.

The initial stock of physical capital per worker is one, and the initial working-age population is 10, all of whom are healthy. In the first period a new disease is introduced into the population to which only children are susceptible. This initial infection rate is 0.9, and parents initially choose to invest $h(i = 0.9)$ in their children to help protect against this infection.

Figures IV.2-IV.6 present simulation results for different values of the case fatality rate, d, and disease severity, δ, across time, where each time period represents a generation. The behavior of each variable generally follows a monotonic pattern with regards to d and δ; the steady state value of each variable is decreasing in d and δ. This pattern reflects the strong role of the wealth effect in the model, as wealth is also decreasing in d and δ.

1 Quantity-quality tradeoff

In Figure IV.2 disease prevalence declines over time. Except for very fatal diseases, disease prevalence declines more quickly for less morbid diseases, since infection-reducing parental investment is higher then.[8] When disease is very fatal, infection dies out so quickly that morbidity plays little role. The infection rate approaches its lower bound of 2% in one to three generations; in England and Wales, deaths from infectious diseases began an abrupt decline around 1870 and reached a low, stable level in approximately 75 years, or two and one-half generations.

As disease prevalence declines, parental investment in children and longevity both rise. Parents invest h in their children in order to prevent infection, and the decline in disease prevalence in turn increases parental investment ($\partial h/\partial i < 0$).[9] The rise in longevity lags that in parental

[8] Infection declines, although more slowly, even if parents choose to invest nothing in children, and there exist parameter values for which infection explodes. If children can catch disease from both young and old adults, rather than just from young adults, then infection should be more persistent and may experience a brief resurgence as longevity rises.

[9] Where i takes longer to reach its long-run level of 2%, h also takes longer to reach its steady state; h reaches its steady state more slowly for low d and high δ, since infection dies out more slowly when fatalities are low and h, which reduces i, is lower for higher δ.

investment; as children experience less disease, parents invest more in them, and both of these effects improve longevity. Returning to the case of England and Wales, in Figure IV.1 deaths from non-infectious diseases began an abrupt decline approximately 30 years, or one generation, after did infectious diseases. Since infectious diseases affect the young and non-infectious diseases primarily the elderly, this lag suggests that children experiencing reduced disease grew up healthier and thus lived longer by avoiding non-infectious diseases. In comparing Figures IV.2 and IV.5, we see that the improvements in human capital investment and longevity continue for one and two generations, respectively, after the decline in disease prevalence stabilizes.

In addition to an improvement in human capital corresponding to the disease decline, in Figure IV.3 the total fertility rate declines initially with disease prevalence where disease burden is substantial (high d and δ).[10] Parents engage in a quantity-quality tradeoff by having fewer children and investing more in the human capital of each. In addition, in equation 8 fertility is decreasing in longevity, which rises over time in Figure IV.5; the transmission of human capital across generations perpetuates the quantity-quality tradeoff.

2 Economic development and population dynamics

Improvements in longevity increase the incentive to save, and in Figure IV.5 the average saving rate in the economy follows average longevity precisely. The economy grows as disease becomes less prevalent (Figures IV.6 and IV.7), but to a lower level where disease burden remains greater; both labor productivity and longevity (and thus savings) are lower where d and δ are higher, so less capital accumulates.[11]

Since children are normal goods, economic growth raises fertility rates, and this effect eventually dominates the quantity-quality tradeoff described above. Fertility in Figure IV.3 is always higher for healthy, uninfected parents relative to unhealthy, infected parents, because the income earnings of the former are greater. Also, lower adult mortality would increase the size of the labor

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[10] After the initial decline in disease prevalence, the wealth effect dominates for all values of d and δ, and n rises to its steady state, but wealth rises less for higher d and δ, so steady state fertility is lower here. $n^U > n^I$ due to infected parents earning less income, and this difference diminishes for low δ, seen in Figure IV.3. In Table IV.1 the difference in steady state fertility is negligible across high d because the mortality effect dominates the morbidity effect, and similarly across high δ.

[11] In comparing Figures IV.5 and IV.6 one sees that k follows the saving rate beginning at $t = 2$ with a lag of one generation, and k dips down initially for the cases where the saving rate dips down the most. The downward dip in k occurs later for $\delta = 0.1$, especially for low d; the uninfected labor force, L^U, briefly resurges during this time, which reduces k for this case.

force, thereby reducing wages and thus the opportunity cost of having children (diverting time from earning income); however, although the wage is decreasing in the labor force size $(1 - i_{t-1}d)$, the model does not impose a time cost to rearing children, so labor supply is inelastic, and fertility rises only because of the income effect of rising human capital.

Despite an overall rise in fertility, fertility remains below replacement level for the specified parameter values; population declines over time to its steady state value. Since birth rates are highest for low d and δ, population declines most slowly here, and to a higher steady state; children are normal goods and higher d and δ translate into lower earned income, wH. If the parameter values were set such that fertility rose above one, population would grow exponentially. In countries that have completed the demographic transition, an inverted $\bigcup$-shaped population path occurred, since total fertility rates take time to adjust to improved survival probabilities among children. A lack of this pattern in Figure IV.4 is due to the absence of a fixed birth cost in the model (Barro and Becker 1989). In the presence of a fixed birth cost, a reduction in child mortality reduces the average fixed cost per surviving child, making children more affordable; fertility initially rises before this effect wears off and the rise in survival probabilities dominates, bringing net fertility rates down below their original level. In Figure IV.4 net fertility is considerably lower than total fertility when d is high, and the decline in disease prevalence immediately brings the two rates closer together.

3 Morbidity

Morbidity hinders the quantity-quality tradeoff for children. In Figure IV.2 parental investment remains lower where disease remains more burdensome (where d and δ are higher), since the returns to investing in children is lower, in terms of potential income earnings.[12] Human capital and thus longevity improve less, hindering economic growth. In equation 8 parents have more children if those children are likely to be of lower quality or are less likely to survive to adulthood, and parents have more children if their expected lifespan is shorter. On the other hand, parents have *fewer* children if they earn less, and morbidity reduces income earnings.

Steady state longevity is decreasing in δ, because morbidity directly reduces human capital through the experience of illness and indirectly because parents invest less when facing more morbidity. The direct consequence of morbidity on longevity is less important when case fatality

[12]The difference in steady state h across δ and d is weaker for very high d and δ, respectively; as d or δ approach one, h_t approaches $(1 - \theta)(1 - \overline{i_t})/\theta\psi$, which is independent of d and δ.

rates are high, since most infected children do not survive to adulthood (Table IV.1). However, the rise from initial to steady state longevity is greatest in more morbid environments: as childhood disease declines, longevity improves more where the disease burden was greater.[13]

When morbidity is high, a reduction in the disease burden improves wage per worker. The wage is increasing in physical capital per worker, k, and in quality of the labor force, $h^0(1-i+i(1-d)(1-\delta))$, and decreasing in the average child survival rate, $1-id$. Thus as disease becomes less prominent, wage per effective worker rises via quality and declines via population (survival rate). Overall, steady state capital and income per worker are lower where morbidity is more persistent.

IV.4 Conclusion

The model above aims to duplicate the epidemiological-demographic transition as it occurred historically. It captures several of the transition's defining characteristics. In particular, as the disease burden diminishes, parents substitute quantity with quality of children. As child quality rises, children grow up healthier and live longer, which fosters more savings; there are improvements in labor productivity and rises is physical capital and income per worker.

The timing of the epidemiological transition in simulations closely mimics that of the transition as it occurred in England and Wales. Infectious disease reaches its long-run low level in one to three generations, more quickly for more fatal diseases and more slowly for more morbid diseases, compared with two to three generations, or approximately 75 years, in Figure IV.1. In England and Wales declines in non-infectious diseases, those diseases affecting primarily the elderly, began approximately one generation, or 30 years, after did declines in infectious diseases. In the simulations improvements in parental investment in child quality and longevity lag the decline in childhood disease by one and two, respectively, generations. See Figures IV.2 and IV.5.

Morbidity restricts the improvements in human capital and longevity and thus economic development. Child quality rises more slowly where morbidity is greater (higher δ), and longevity, ϕ,

[13] Average ϕ $((L^U\phi^U + L^I\phi^I)/(L^U + L^I))$ initially dips down in some cases in Figure IV.5 due to rapid changes in population as disease becomes less prevalent (Figure IV.4). The infected population grows initially as the disease is introduced but then declines with disease prevalence, and the healthy population falls with disease prevalence also. Average ϕ is decreasing in unhealthy population ($\partial\phi/\partial L^I = L^U(\phi^I - \phi^U)/(L^U + L^I)^2 \leq 0$), and more so for higher δ. ϕ decreases due to the rise in L^I, which is bigger for low δ, and the drop in L^U, which is bigger for high δ. The latter effect, combined with the amplifying impact of δ on $\partial\phi/\partial L^I < 0$ and $\partial\phi/\partial L^U > 0$, dominates and so the initial dip in ϕ is greater for high δ. The infected population grows less initially if disease is very fatal so that disease never claims a large fraction of the population, and this is reflected by a milder dip in ϕ for high case fatalities.

is lower for adults who experienced disease as children. The model provides an explanation for why in sub-Saharan Africa adult health has not improved despite declines in child mortality; morbidity declines have not accompanied observed mortality declines, and the lag in morbidity in this region may be stalling the demographic transition. The data for sub-Saharan Africa suggests that debilitating childhood disease remains a common experience and that declines in mortality can be largely attributed to reductions in case fatalities, d, rather than in disease prevalence, i.

There remain some inconsistencies between the model and historical evidence on the epidemiological-demographic transition. Although net fertility rates rise in Figure IV.4 due to a wealth effect, they never decline again, yet historically we observe an inverted $\cup$-shaped population path when disease burden declines. Adding a fixed time cost per birth would resolve this, although it complicates the model. While the duration of the decline in infectious disease mimics the story of England and Wales, it does not achieve a proper representation of the disease environment prior to the epidemiological transition. For a long time prior to the transition, infectious disease was both very prevalent and volatile, but in simulations disease declines immediately once it is introduced. For other parameter values, disease prevalence is volatile and does not reach a stable low level. It remains a task for future work to find a combination of parameters that generate a path of disease more consistent with historical evidence; a threshold is reached that triggers a rapid transition from a volatile state of devastating pandemics to low, stable levels of disease. Finally, while we observe sustained economic growth in England and other countries that have completed the epidemiological-demographic transition, in Figure IV.7 the economy stabilizes at zero growth because human capital plateaus shortly after infection reaches its long-run level. Endogenizing health capital at birth to parental health capital will prolong the rise in human capital; one might also incorporate educational investment, as in Chapter III. Despite some inconsistencies with the empirical evidence, the current model does succeed in generating several defining characteristics of the epidemiological-demographic transition.

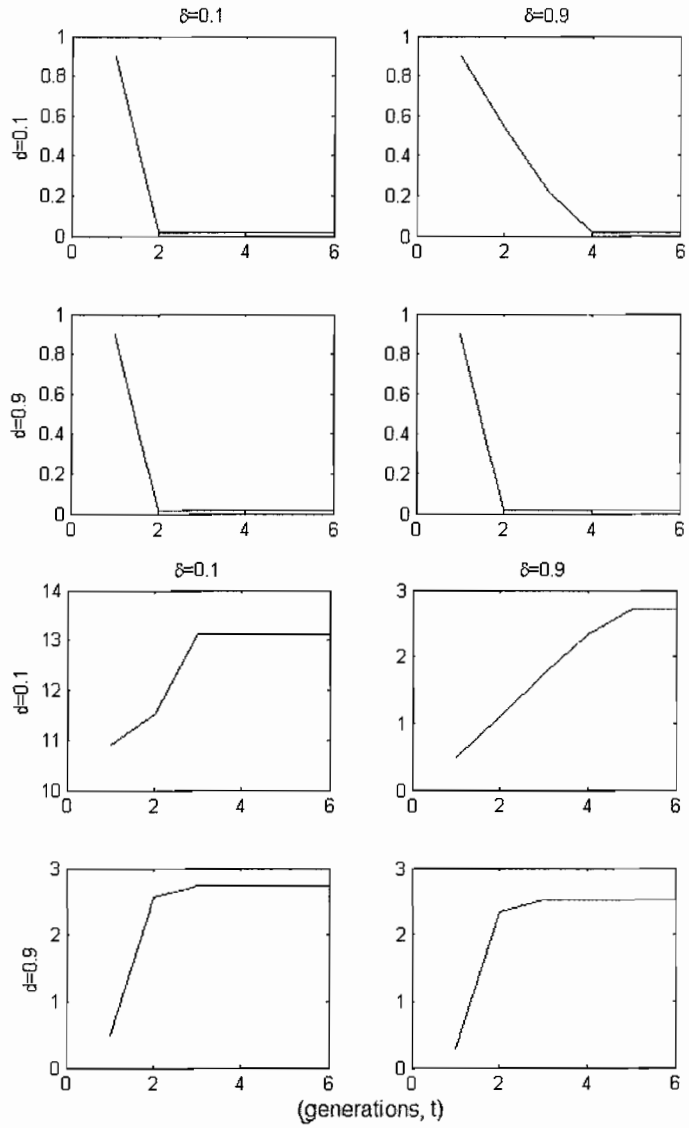

Figure IV.2: Infection rate (top) and parental investment (bottom) across time

An exogenous initial disease prevalence rate of 90% declines within three generations. Parental investment in children continues to improve in response to declining disease prevalence for one generation after disease stabilizes at its lower bound.

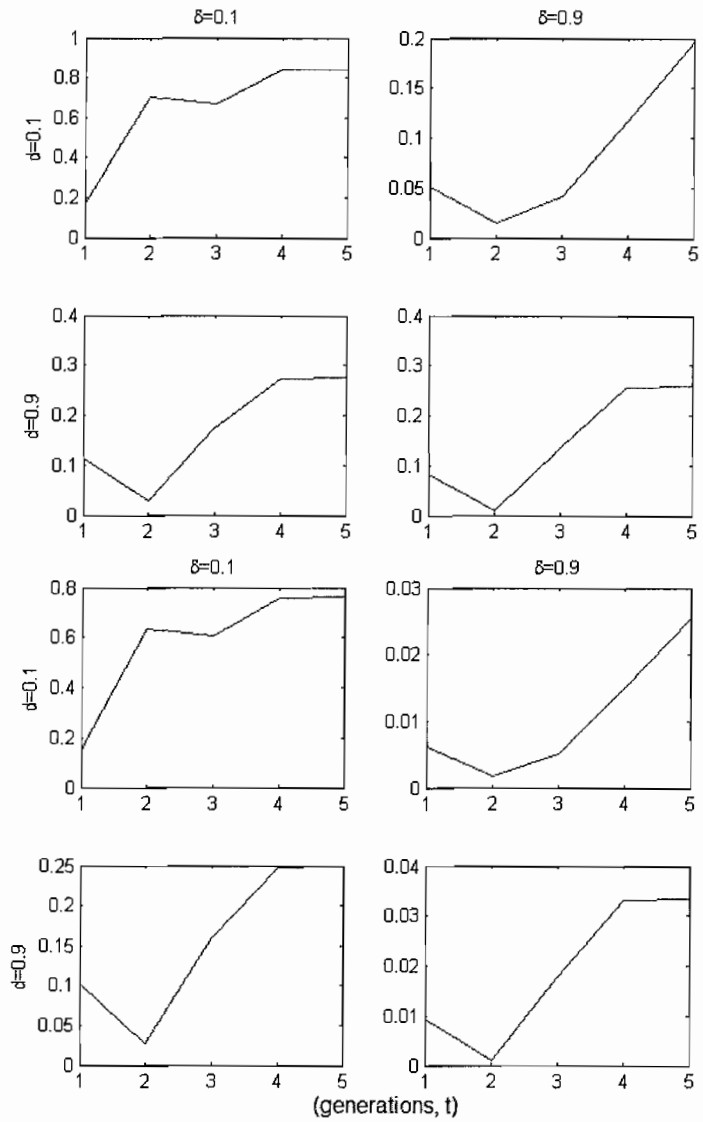

Figure IV.3: Total fertility rates for uninfected (top) and infected (bottom) individuals across time

As disease becomes less prevalent, the total fertility rate declines where disease burden was highest (high d or δ); parents substitute quantity with quality of children. After the initial tradeoff, the wealth effect dominates: fertility rises with economic growth and is higher among healthy parents, since healthy parents earn more income.

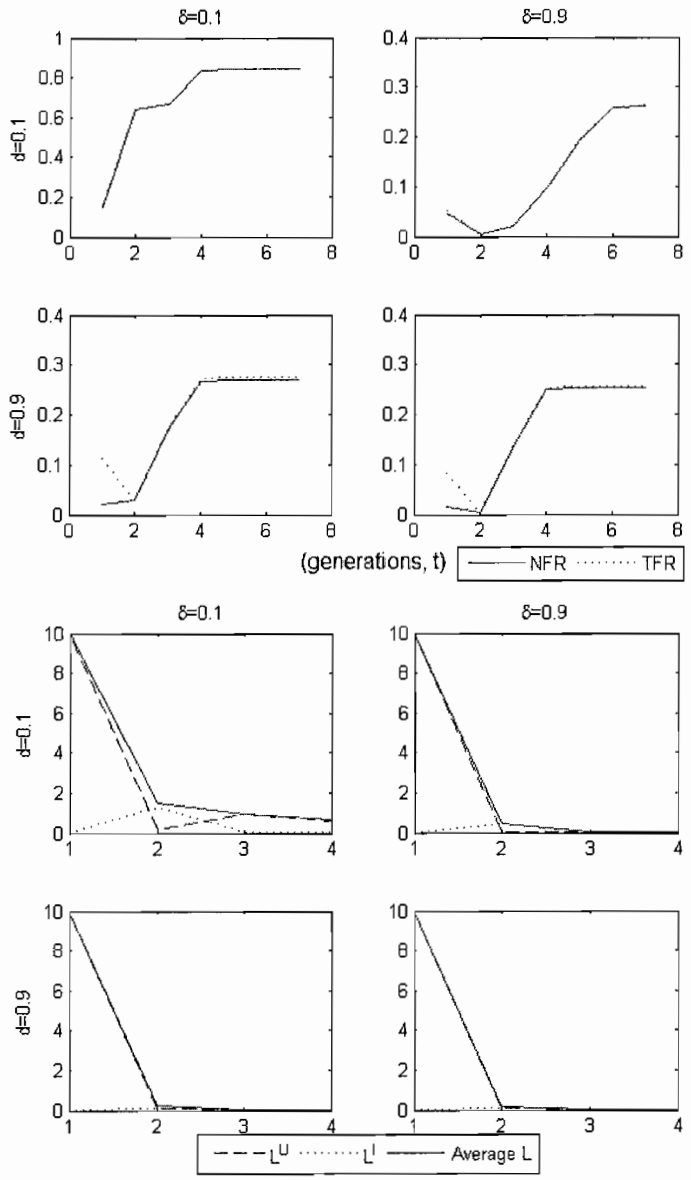

Figure IV.4: Average net versus total fertility rates (top) and labor force (bottom) across time

The disparity between total and net fertility rates diminishes with disease prevalence. The infective population initially surges and then recedes with disease prevalence. The labor force declines over time since fertility falls below replacement level, but the labor force stabilizes at a positive steady state, which is higher where morbidity is lower.

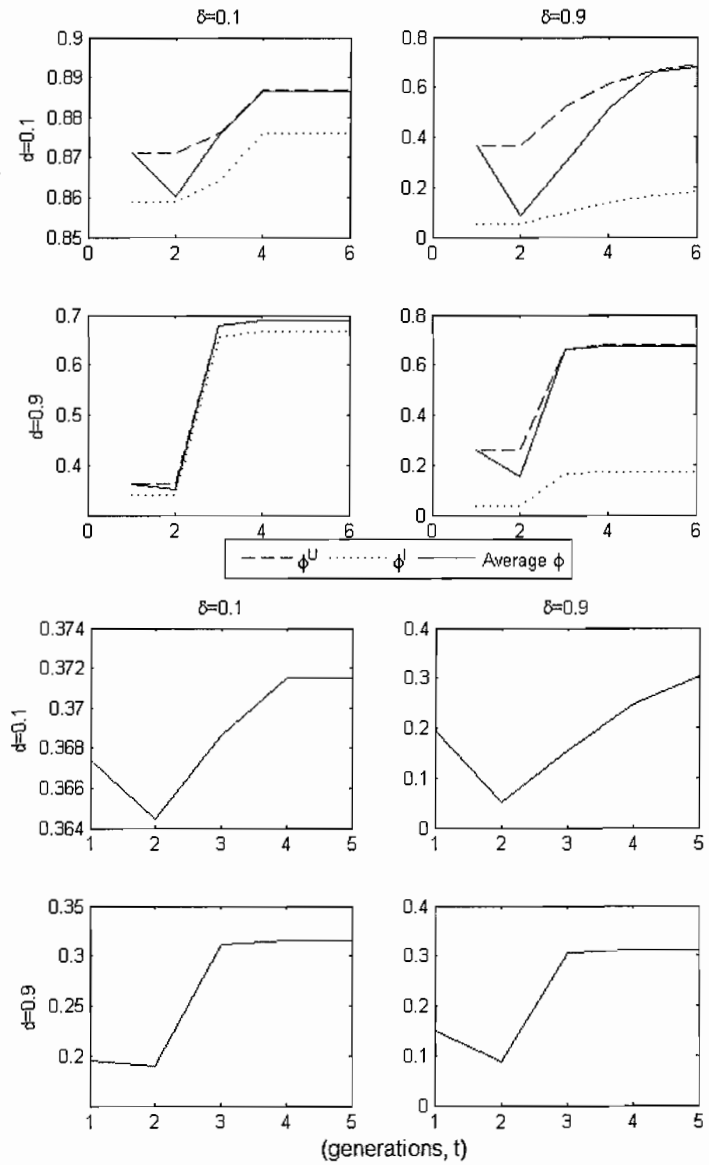

Figure IV.5: Longevity (top) and the saving rate (bottom) across time

The average saving rate follows average longevity: individuals save more if they expect to live longer. Longevity is lower where disease burden remains greater (high d and δ).

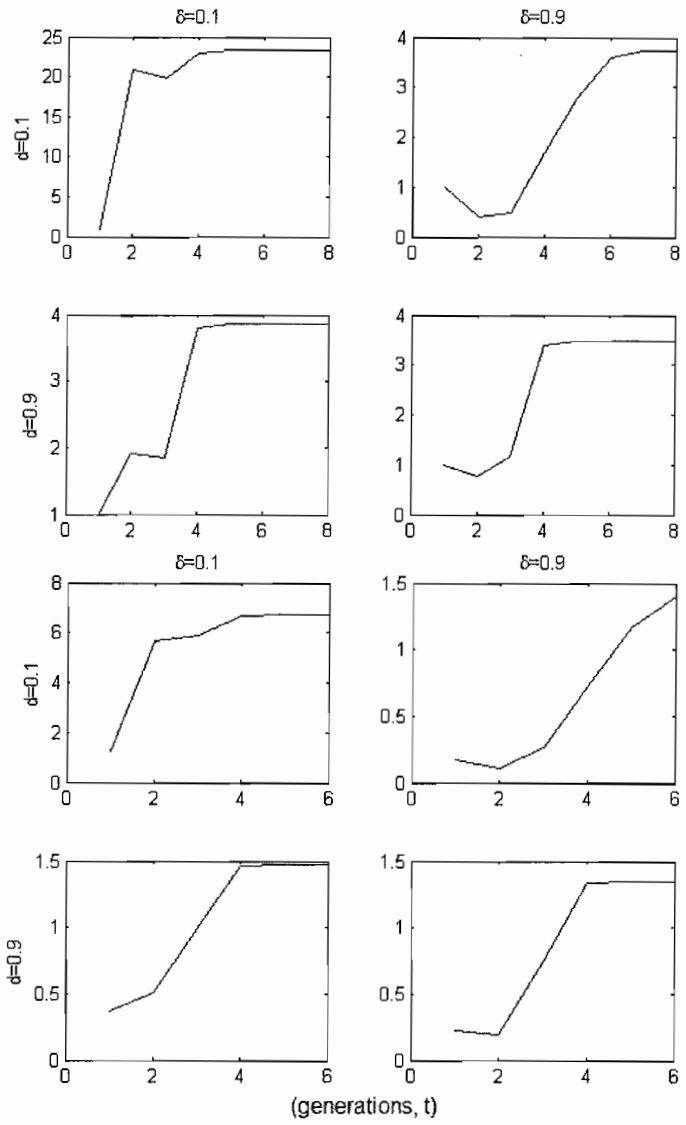

Figure IV.6: Average physical capital (top) and wage (bottom) per worker across time

The economy grows over time until a steady state is reached. The steady state of physical capital per worker is lower where disease disease burden is greater (high d and δ).

Table IV.1: Steady state values for different values of d and δ

		$\delta = 0.1$	$\delta = 0.9$
Quality, h	d=0.1	13.12	2.71
	d=0.9	2.74	2.52
Longevity, ϕ	d=0.1	0.89	0.68
	d=0.9	0.69	0.68
ϕ^U	d=0.1	0.89	0.69
	d=0.9	0.69	0.68
ϕ^I	d=0.1	0.88	0.18
	d=0.9	0.67	0.17
Total fertility, n	d=0.1	0.84	0.26
	d=0.9	0.27	0.26
n^U	d=0.1	0.85	0.27
	d=0.9	0.27	0.26
n^I	d=0.1	0.76	0.03
	d=0.9	0.25	0.03
Net fertility, $n(1-id)$	d=0.1	0.84	0.26
	d=0.9	0.27	0.25
Labor force, L	d=0.1	2.02E-30	9.09E-236
	d=0.9	2.69E-229	1.38E-241
L^U	d=0.1	1.98E-30	8.92E-236
	d=0.9	2.68E-229	1.38E-241
L^I	d=0.1	3.65E-32	1.64E-237
	d=0.9	5.47E-232	2.81E-244
Consumption, c	d=0.1	22.15	1.43
	d=0.9	1.50	1.30
Wage per worker, w	d=0.1	6.75	1.43
	d=0.9	1.47	1.35
Saving rate, s	d=0.1	0.37	0.31
	d=0.9	0.32	0.31
Physical capital per worker, k	d=0.1	23.32	3.74
	d=0.9	3.86	3.48

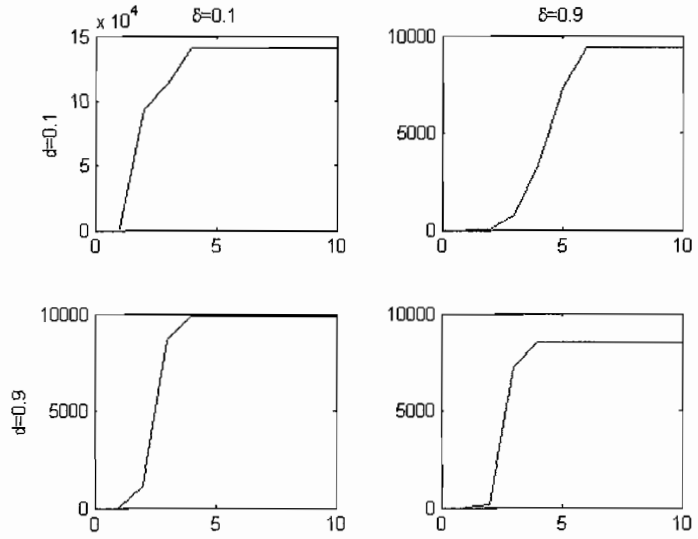

Figure IV.7: Income per worker across time

Income per worker rises as human capital and wages both grow with the decline in disease burden. Where disease burden persists, income per worker is lower.

CHAPTER V

CONCLUSION

The defining feature of this dissertation is its focus on morbidity. In Chapter II I focus on how disease morbidity directly reduces labor productivity. In Chapters III and IV I distinguish between disease-caused mortality and morbidity and the role each plays in fertility and human capital decisions. In Chapter III morbidity reduces productivity directly by depleting health and indirectly by reducing the returns to human capital investment. In Chapter IV morbidity also reduces human capital, and in addition provides a link between childhood disease and adult mortality, a feature of the epidemiological transition that has been described in previous studies but received little attention in economic models (Arora 2003, 2005).

Historically, sanitation infrastructure and technological change have instigated the epidemiological-demographic transition from high to low mortality and fertility. In England and Wales the innovation of the germ theory coupled with sanitation reform triggered the transition (Arora 2003). Great Britain set up improved sanitation and water management in 1848-54, and the United States set up a Board of Health in New York City in 1866. This vastly reduced disease and drastically reduced infant mortality. Asia, Africa and Latin America did not impose the same systems, but knowledge trickled down, so people there at least boiled water and periodically tested water sources for bacteria. Across Europe and Asia, coordinated efforts emerged to contain epidemics, and when epidemics erupted, the West would often send medical experts to quell them, similar to the World Health Organization today (McNeill, 1976).

Many countries have yet to complete the epidemiological-demographic transition. Medical knowledge continues to trickle down from more to less developed regions, which has allowed for large gains in child survival probabilities since the 1950s. Yet while the technology to avoid many diseases exists today, it continues to elude certain regions where infectious disease remains widespread and virulent. Policy changes such as the intellectual property rights (IPR) reform discussed in Chapter II

may encourage innovation of health goods that function well in the inadequate health care infrastructure characteristic of less developed regions. If IPR reform helps people protect themselves from debilitating diseases by encouraging appropriate technological innovations, it could foster economic growth. A declining disease burden improves productivity directly by reducing morbidity, which should also reduce fertility, thus increasing the resources available for quality investment; by improving health and increasing life expectancy, reducing disease morbidity also raises the returns to, and thus incentives for, quality investment. Chapter III illustrates how disease morbidity reduces the returns to education, and it is the first study to analyze how child morbidity, not just child mortality, affects the quantity-quality tradeoff for children which occurs as disease burden declines.

It is unclear whether a modern epidemiological-demographic transition would mimic historical ones. Much of the gains in child survival in sub-Saharan Africa since the 1950s can be attributed to avoiding fatalities from infection rather than avoiding infection itself, so morbidity remains high. A major conclusion in Chapters III and IV is that despite improving child survival, health interventions may have undesirable effects in some cases, in particular, exacerbating morbidity, thus raising fertility and hampering education. While reducing mortality is clearly worthwhile, this alone seems to be insufficient to generate a quantity-quality tradeoff for children and stimulate much needed economic growth, as evidenced by the case of Africa.

Understanding the consequences of changing infection rates versus case fatality rates is important when allocating scarce health care resources. Different types of health interventions may be more effective in different scenarios, as illustrated with the example of malaria in Chapter III. Chapter III highlights the possibility that changes in the disease environment could have unintended consequences, such as increased fertility and therefore reduced human capital investment. In such cases policies aimed at reducing disease burden should be accompanied by policies that directly foster human capital investment or lower fertility.

Chapter IV highlights the link between disease in childhood and adulthood, a little studied feature of the epidemiological transition. If disease burden among adults can be reduced through health interventions early in life, then public health policy will be more efficient if it is aimed at combating diseases that affect the young. Vaccination campaigns have already made much progress on this front, although in sub-Saharan Africa morbidity remains high despite improvements in child survival probabilities; adult disease burden remains high in part because childhood morbidity persists. The model in Chapter IV provides an explanation for the regional differences observed in

the response of adult health to falling child mortality rates. Understanding why morbidity remains high in Africa despite successful vaccination efforts remains a goal for future work.

Disease burden remains high in developing regions today, and understanding its consequences is important in formulating public health and economic policies. This dissertation makes some progress towards this goal by emphasizing the importance of morbidity versus mortality as components of the disease burden. There remain some inconsistencies between the models of Chapters II, III and IV. The disease environment is assumed to be completely exogenous in Chapter II, while Chapters III and IV assume the disease environment changes along with economic development; the truth is probably somewhere in the middle, since certain tropical regions are likely to always be plagued by more prevalent and virulent parasites. Although empirical evidence shows the direction of causality between disease and economic development to run predominantly from disease to economic development, malnutrition is an important factor contributing to a high disease burden, especially among children (Arora 2003, Soares 2005). In this respect the specification in Chapter IV, in which parents invest in all children born, is appropriate. However, morbidity reduces the returns to human capital investment, and this case is made explicitly through parental investment in surviving children in Chapter III. The most realistic components of the models developed in this dissertation should be combined to create a more cohesive model of the epidemiological-demographic transition and economic growth.

APPENDIX

CHAPTER III CALCULATIONS

Optimal Fertility using the Delta Method in Chapter III:

Let $E(N_j) = \bar{N}_j$ for $j = 1, 2, 3$ and $\bar{\mathbf{N}} = (\bar{N}_1\ \bar{N}_2\ \bar{N}_3)$ where

$$\bar{N}_1 = n(1-i),\ \bar{N}_2 = ni(1-d),\ \bar{N}_3 = nid.$$

A second-order Taylor expansion around the means gives us

$$\begin{aligned} E(U(N_1, N_2, N_3)) \cong\ & U(\bar{\mathbf{N}}) + E(N_1 - \bar{N}_1)U_{N_1}(\bar{\mathbf{N}}) + \frac{E(N_1 - \bar{N}_1)^2}{2!}U_{N_1N_1}(\bar{\mathbf{N}}) \\ & + E(N_2 - \bar{N}_2)U_{N_2}(\bar{\mathbf{N}}) + \frac{E(N_2 - \bar{N}_2)^2}{2!}U_{N_2N_2}(\bar{\mathbf{N}}) \\ & + E(N_3 - \bar{N}_3)U_{N_3}(\bar{\mathbf{N}}) + \frac{E(N_3 - \bar{N}_3)^2}{2!}U_{N_3N_3}(\bar{\mathbf{N}}). \end{aligned}$$

Since $E(N_j - \bar{N}_j) = 0$ for $j = 1, 2, 3$, this simplifies to

$$\begin{aligned} E(U(N_1, N_2, N_3)) \cong\ & U(\bar{\mathbf{N}}) + \frac{E(N_1 - n(1-i))^2}{2!}U_{N_1N_1}(\bar{\mathbf{N}}) \\ & + \frac{E(N_2 - ni(1-d))^2}{2!}U_{N_2N_2}(\bar{\mathbf{N}}) + \frac{E(N_3 - nid)^2}{2!}U_{N_3N_3}(\bar{\mathbf{N}}). \end{aligned}$$

From the first and second derivatives of the utility function

$$\begin{aligned} U_{N_1} &= \frac{(1-\beta)(1-\theta)}{N_1 + N_2(1-\delta)^{\frac{\alpha}{1-\theta}}}, \qquad U_{N_1N_1} = -\frac{(1-\beta)(1-\theta)}{(N_1 + N_2(1-\delta)^{\frac{\alpha}{1-\theta}})^2} \\ U_{N_2} &= \frac{(1-\beta)(1-\theta)(1-\delta)^{\frac{\alpha}{1-\theta}}}{N_1 + N_2(1-\delta)^{\frac{\alpha}{1-\theta}}}, \qquad U_{N_2N_2} = -\frac{(1-\beta)(1-\theta)(1-\delta)^{\frac{2\alpha}{1-\theta}}}{(N^1 + N^2(1-\delta)^{\frac{\alpha}{1-\theta}})^2} \\ U_{N_3} &= 0 \end{aligned}$$

we have

$$U_{N_1N_1}(\bar{\mathbf{N}}) = -\frac{(1-\beta)(1-\theta)}{n^2(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^2},$$

$$U_{N_2N_2}(\bar{\mathbf{N}}) = -\frac{(1-\beta)(1-\theta)(1-\delta)^{\frac{2\alpha}{1-\theta}}}{n^2(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^2}.$$

For the multinomial distribution, $V(N_j) = np_j(1-p_j)$ for $j = 1, 2, 3$, which implies

$$\begin{aligned} E[N_1 - \bar{N}_1]^2 &= ni(1-i), \\ E[N_2 - \bar{N}_2]^2 &= ni(1-d)[1-i(1-d)]. \end{aligned}$$

Making these substitutions yields

$$\begin{aligned} E(U) \simeq\ & \beta \ln\left[\frac{\beta(1-\gamma n)z}{\beta+\theta(1-\beta)}\right] \\ & +(1-\beta)\ln\left[wn^{1-\theta}(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^{1-\theta}\left(\frac{\theta(1-\beta)(1-\gamma n)z}{\beta+\theta(1-\beta)}\right)^{\theta}\right. \\ & \left.-\frac{(1-\beta)(1-\theta)i}{2n(1-i+i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^2}[1-i+(1-\delta)^{\frac{2\alpha}{1-\theta}}(1-d)(1-i(1-d))]\right] \end{aligned}$$

The optimality condition (6) is obtained by setting to zero the partial of this expression with respect to n.

Fertility Response to i and d in Chapter III:

Define

$$\Phi(n) \equiv n - \frac{\gamma(\beta+\theta(1-\beta))n^2}{(1-\beta)(1-\theta)(1-\gamma n)} = -\frac{i[1-i+(1-\delta)^{\frac{2\alpha}{1-\theta}}(1-d)[1-i(1-d)]]}{2[1-i+i(1-\delta)^{\frac{\alpha}{1-\theta}}(1-d)]^2} \equiv \Gamma(i,d)$$

which implicitly solves for n as a function of i and d. Evidently $dn/di = \Gamma_i/\Phi_n$ and $dn/dd = \Gamma_d/\Phi_n$. The results for extreme values of δ are consistent with the general results.

$\Gamma_i > 0$ if $i > i_L$, where

$$i_L \equiv \frac{1+(1-\delta)^{\frac{2\alpha}{1-\theta}}(1-d)}{1+(1-\delta)^{\frac{\alpha}{1-\theta}}(1-d)[1+(1-\delta)^{\frac{\alpha}{1-\theta}}(1-2d+(1-\delta)^{\frac{\alpha}{1-\theta}}(1-d))]}$$

is increasing in δ and d. As d or δ increase, $\Gamma_i > 0$ becomes less likely. If $\delta = 1$, $\Gamma_i < 0$ for all i and d. If $\delta = 0$, $\Gamma_i > 0$ if $i > 1/2$ when $d = 0$, and $\Gamma_i < 0$ when $d = 1$.

$\Gamma_d > 0$ if $i < i_U$, where

$$i_U \equiv \frac{1}{4}\left[2 + (1-\delta)^{\frac{\alpha}{1-\theta}} - \left\{\frac{4 - (1-\delta)^{\frac{\alpha}{1-\theta}}[4d + (1-\delta)^{\frac{\alpha}{1-\theta}}\{-5 + 4d - (1-\delta)^{\frac{\alpha}{1-\theta}}(1-d)\}]}{1 + (1-\delta)^{\frac{\alpha}{1-\theta}}(1-d)}\right\}^{1/2}\right]$$

is decreasing in δ and increasing in d. $\Gamma_d > 0$ is more likely as d increases and less likely as δ increases. If $\delta = 0$, $\Gamma_d > 0$ when $d = 0$ and if $i < 1/2$ when $d = 1$. If $\delta = 1$, $\Gamma_d < 0$.

Since $\Gamma(i, d) < 0$, it must be that $n > [(1-\beta)(1-\theta)]/\gamma \equiv \overline{n}$, fertility choice under certainty. $\Phi_n > 0$ if $n > \underline{n} \equiv [1 - \{\beta + \theta(1-\beta)\}^{1/2}]/\gamma$. Since we are restricted by $n < 1/\gamma$ we have $\underline{n} \equiv [1 - \{\beta + \theta(1-\beta)\}^{1/2}]/\gamma > \overline{n}$. While this means it is not always the case that $\Phi_n < 0$, given the response of n to i and d in Figures 1 and 2, one can conclude that $\Phi_n < 0$ for the parametric assumptions we made about β and θ. Note that $\underline{n} = 0$ when $\beta = 1$ or $\theta = 1$.

Human Capital Response to i and d in Chapter III:

Differentiating

$$h_1 = \frac{\theta(1-\beta)(1-\gamma n)z}{(\beta + \theta(1-\beta))(N_1 + N_2(1-\delta)^{\frac{\alpha}{1-\theta}})}$$

we get

$$\frac{\partial h_1}{\partial i} = \frac{-\frac{\partial n}{\partial i}}{n(1 - i + i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})} - \frac{(1-n)[n\{-1 + (1-d)(1-\delta)^{\frac{\alpha}{1-\theta}}\} + \{1 - i + i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}}\}\frac{\partial n}{\partial i}]}{n^2(1 - i + i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})^2}$$

which is negative if

$$\frac{\partial n}{\partial i} > \frac{n(1-n)(1 - (1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})}{(2-n)(1 - i + i(1-d)(1-\delta)^{\frac{\alpha}{1-\theta}})} > 0.$$

Similarly for $\partial h_1/\partial d$.

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