Endogenous Lifetime and Economic Growth*

Shankha Chakraborty

Department of Economics, University of Oregon

Eugene, OR 97402-1285

Phone: (541) 346-4678, Fax: (541) 346-1243

shankhac@oregon.uoregon.edu

*For useful conversation and feedback, I would like to thank Costas Azariadis, Richard Barrett, Roger Farmer, Gary Hansen, Dean Jamison, Amartya Lahiri, David Meltzer, Tomas Philipson, Jia Wang and seminar participants at UCLA, UCSB, Harris School of Public Policy, and University of Oregon. All remaining errors are mine.

Endogenous Lifetime and Economic Growth

Abstract

Conventional wisdom attributes the severity of mortality in poorer countries to widespread poverty and inadequate living conditions. This paper considers the possibility that persistent poverty may arise, in turn, from a high incidence of mortality. Endogenous mortality risk is introduced in a two-period overlapping generations model: probability of survival from the first period to the next depends upon health capital that can be augmented through public investment. High mortality societies do not grow fast since shorter lifespans discourage saving and investment; multiple steady-states are possible. High mortality also reduces returns on investments, like education, where risks are undiversifiable. When human capital drives economic growth, countries differing in only health capital do not converge to similar living standards; 'threshold effects' may also result.

Keywords: Health, Life expectancy, Mortality, Growth, Human capital.

JEL Classification: I1, I2, O1

1 Introduction

More than a billion people suffer from malnutrition in developing countries, infant mortality rates there are three to four times higher than those in the richest nations and the burden of disease twice as high [World Bank, 1993]. This paper studies the effect of such pervasive ill-health, especially high mortality, on economic growth.

Health status and income levels are well known to systematically vary across nations, particularly those in low- and middle-income categories. Figure 1 displays one such relationship, that between life expectancy at birth and log GDP per capita for 1996.[1] Life expectancy at birth evidently shows a strong tendency to improve with per capita income, ranging from as low as 37 years in Sierra Leone to as high as 77 in Costa Rica, more than twelve times richer. A similar pattern is observed if we narrowed our focus to adult life expectancy, a length of time more relevant for economically active individuals. Thus, life expectancy at age 15 for the poorest nations fell $12 - 13$ years short of the 73 years prevailing in the richest ones in 1990. Adult mortality risk, the probability that a typical 15-year old would die before reaching age 60, was thrice as high in Sub-Saharan Africa as in the established market economies [World Bank, 1993, Table A.5]. Conventional wisdom attributes the severity of mortality and morbidity in poor countries to widespread poverty and inadequate living conditions. We argue here that there is as much possibility that persistent poverty results from undesirable economic incentives that high mortality creates.

Ever since economists recognized the importance of human capital, considerable atten-

[1] Data from World Bank [1998].

tion has been devoted to the various ways health improves economic outcomes.[2] But that literature has usually focused on the implications of health and nutrition for labor market outcomes. Yet health plays a role quite unlike any other human capital: by altering an individual's lifespan and mortality risks, it fosters incentives to invest in assets, especially those that realize high returns later in life. Given the central role capital accumulation plays in growth theory, it is not surprising that life expectancy would nontrivially affect the dynamics of per capita income.

With that in mind, this paper constructs a simple general equilibrium framework that allows health capital and per capita income to be determined simultaneously. This is done by introducing mortality risk in a two-period overlapping generations model. In particular, the probability of surviving from the first period to the next is *endogenously* determined through public investment in health. In low-income societies, when average lifespans are short, individuals are less willing to postpone consumption. Consequently, they are also less willing to save and invest. Due to pervasive poverty, neither can they afford substantial private health investment.

During the early stages of development, a low income - low longevity society therefore grows slower than what standard one-sector models predict. When the capital elasticity of output is sufficiently large, increase in per capita income, over a certain range, draws a large response in capital accumulation. Multiple steady-states result: societies with favorable initial conditions (income or health) grow towards a high-income steady state, less fortunate ones find high mortality a strong deterrent for saving and investment.

[2]See Srauss and Thomas [1998] for an overview of this primarily empirical literature.

Besides this length-of-life effect, mortality rates affect investment decisions through rates of return. Risks associated with investment in other forms of human capital, for instance education, may not be fully diversifiable. Higher mortality risks would then reduce returns on such investment. Recent growth theories have persuasively argued that human capital, by improving labor skills, inducing technological innovations and expanding the body of economically useful knowledge, contributes to long-run growth [for instance, Romer, 1986 and Lucas, 1988]. When mortality considerations affect schooling decisions, countries that differ only in health capital do not converge to similar living standards. 'Threshold effects' [Azariadis and Drazen, 1990] may also result with the economy going through a phase of slow growth and modest health improvements before taking-off into sustained growth.

This paper complements several pieces of work in the literature. Among others, Ram and Schultz [1979], Gersovitz [1983], and more recently, the World Bank [1993] have suggested how longevity improvements may promote economic growth. The contribution of the current paper lies in systematically tying that intuition to a general equilibrium framework with health investment.

In a model of endogenous growth, de la Croix and Licandro [1999] show that exogenous life expectancy improvements initially raise economic growth, but may be growth depressing once the average age of the workforce increases sufficiently. Kalemli-Ozcan *et al.* [1998] examine how increased life expectancy promotes human capital investment in a Blanchard-type [1985] model with constant probability of death.

The impact of prime-age and adult mortality on fertility choice and human capital accumulation is analyzed by Meltzer (1995); mortality decline initially raises incentives to

have children and discourages human capital investment, but is followed by a phase of sustained economic growth and declining population growth. Children are a source of old-age support in Ehrlich and Lui [1991], where too, a demographic transition results. In both these works, mortality declines are treated as exogenous to the development process.

Finally, several empirical works have motivated this paper to examine the health and growth causality. Fogel [1997], for instance, estimates that better health and nutrition alone may have contributed about 20-30% to British economic growth during 1780-1979. In their analyses of the economic burden of malaria, both Gallup and Sachs [1998] and McCarthy *et al.* [2000] ascribe the lackluster performance of several African nations to a severe incidence of the disease. Using cross-country regressions, Barro and Sala-i-Martin [1995] and Knowles and Owen [1995] show life expectancy to be an important determinant of growth.

The rest of the paper is organized as follows. Section 2 sets up an overlapping generations economy with endogenous mortality risk. Since low-income societies cannot invest much in health nor do they save a lot, convergence is shown to be slower during the early stages of development. Depending upon the output share of capital, the model may also deliver multiple steady-states. Section 3 studies the effect of mortality risk on schooling decision. Educational investment is assumed to improve labor-skills that are passed on from one generation to the next. Multiple equilibria result when such externalities are present. In section 4, we briefly turn to cross-country evidence that confirms the specific linkages stressed in the theory and indicates the growth effects to be significantly large. Section 5 concludes the discussion.

2 Length-of-life and Capital Accumulation

Longevity affects savings decisions through two channels: by increasing lifespans it promotes investment in assets that pay off later in life, and by reducing mortality risks, it increases the return on such investment. This section considers the length-of-life effect,[3] postponing until section 3 a discussion of the latter.

To keep the analysis simple, we shall ignore the effect of health on labor productivity and labor market participation.[4] Moreover, we assume that preferences, technologies and markets do not fundamentally differ in a low-income country from those in a high-income one.

Existing theoretical models that incorporate mortality usually assume it to be exogenous.[5] To endogenize the mortality rate, we consider a two-period overlapping generations economy where the probability of surviving from the first period to the next depends upon the health of individuals. Individuals can choose to improve this probability through appropriate investments in health.[6]

[3]Theories of development often a higher savings propensity for the rich [see Lewis, 1954 or Leibenstein, 1957]. Lawrance [1991] finds subjective rates of time preferences to be higher for poorer households, implying a less willingness to save. The length-of-life effect is one way to explain both.

Deaton and Paxson [1994] attribute the higher savings rate in younger cohorts in Taiwan (1976-90) to life expectancy improvements, among other factors.

[4]See Gersovitz [1983] for a theoretical treatment, Fogel [1999] for a historical assessment.

[5]Two exceptions are Gersovitz [1983] and Blackburn and Cipriani (1998). Gersovitz considers a partial equilibrium model where mortality risk declines with consumption early in life. Our formalization differs by assuming future survival depends upon (public) health expenditure, not past consumption.

Blackburn and Cipriani analyze the implications of infant mortality, which depends upon health-care expenditure, on fertility and capital accumulation.

[6]It is more plausible to think of health investment here as implementing technologies and programs already available elsewhere (e.g., developed nations or through development agencies), rather than the direct cost of inventing them.

In general, a person's health status depends upon her private health choices as well as on public health facilities she can avail of. Public health intervention, particularly in developing countries, is likely to play a critical role for an important reason. Table 1, from Dasgupta [1993], divides mortality incidence in developing and developed countries according to causes of death: 45% of all deaths in developing countries occurs due to infectious and parasitic diseases, as opposed to only 5% in developed countries. Since these diseases, by their very nature, create externalities, only public intervention can circumvent the underinvestment that would result from private choices. Public health expenditure in new medical facilities, sanitation improvements, disease control and inoculation programs augments private health capital by reducing the economy-wide risk of contacting fatal diseases.

Private health inputs, on the other hand, may take various forms including nutrition and preventative medicine early in life. A majority of people in developing countries may be unable to afford these investments on a regular basis. Keeping this in mind, we ignore all forms of private health investment and expand the scope of public health interventions to include provision of subsidized nutritional supplements and clinical treatments.[7]

2.1 The Environment

Consider now an overlapping generations economy where individuals may live for two periods: they live in youth for sure, but survive into old age with probability ϕ.[8] In any period t, young agents of measure one are born, each with a time endowment of 1 unit.

[7] Although this is the preferred interpretation here, note that the model can just as well be viewed as one of private health investment by interpreting the health tax as a cost of purchasing private health inputs.

[8] Since a new-born expects to live for $1+\phi$ periods, the terms 'life expectancy', 'longevity', 'length of life' and 'survival probability' are all used interchangably in the rest of the paper.

A generation-t individual supplies this labor time inelastically, earning a wage income w_t. Public health expenditure is financed through a proportional tax $\tau_t \in (0,1)$ on labor income. Health investment, per generation-t person, therefore equals $\tau_t w_t$.

The survival probability of a generation-t person, ϕ_t, depends upon her health stock, h_t. In particular, it is given by a non-decreasing concave function

$$\phi_t = \phi(h_t) \tag{1}$$

that satisfies $\phi(0) = 0$, $\lim_{h\to\infty} \phi(h) = \beta \leq 1$ and $\lim_{h\to 0}\phi'(h) = \gamma < \infty$.[9] Public health investment augments private health capital through a constant returns technology:

$$h_t = g(\tau w_t) = \tau w_t.$$

Each generation-t person gives birth to a new individual at the end of period t, before she realizes her 'mortality shock'. This new individual becomes economically active only at the beginning of period $t+1$, and in particular, does not inherit her parent's health stock.[10]

To abstract from the risk associated with uncertain lifetimes and concentrate on the length-of-life effect, we follow Yaari [1965] and Blanchard [1985] in assuming a perfect annuities market whereby all savings are intermediated through mutual funds.[11] At the end of her youth, each agent deposits her savings with a mutual fund. The mutual fund

[9] One example that satisfies these properties is $\phi(h) = h/(1+h)$ with $\beta = \gamma = 1$.

[10] Incorporating health transfers through birth adds one more dimension to the dynamical system without altering the basic insights.

[11] If annuity markets were not perfect, a lower mortality rate would raise realized returns on investment and encourage savings. In that case, the results that follow understate true magnitudes. But a perfect annuities market assumption may not be far-fetched even for developing countries. When life-contingent annuity markets are not well-developed, the family can self-insure against mortality risks through interfamily transfers, substituting by more than 70% for perfect market annuities [Kotlikoff and Spivak, 1981].

invests these savings in capital (the sole asset) and guarantees a gross return of $\widehat{R}_{t+1}$ to the surviving old.[12] If a fund earns a gross return R_{t+1} on its investment, then perfect competition would ensure that $\widehat{R}_{t+1} = R_{t+1}/\phi_t$ holds in equilibrium.

A generation-t agent maximizes her expected lifetime utility,

$$U_t = u(c_t^t) + \phi_t u(c_{t+1}^t), \tag{2}$$

subject to the period budget constraints:

$$c_t^t \leq (1-\tau_t)w_t - z_t,$$

$$c_{t+1}^t \leq \widehat{R}_{t+1} z_t,$$

taking as given the vector of prices $(w_t, \widehat{R}_{t+1})$. Here u denotes the period utility function and z denotes savings of the youth. When u is homothetic, the savings function takes a simple form,

$$z_t = (1-\tau_t)\sigma(\widehat{R}_{t+1}, \phi_t)w_t, \tag{3}$$

σ being the savings propensity.

As in the standard neoclassical model, production is carried out using capital and labor, through a constant returns-to-scale technology, $F(K, L)$, that satisfies the usual Inada conditions. If k denotes the capital stock per worker, let $f(k) \equiv F(k, 1)$ represent the intensive-form production function. Perfect competition in the final goods market implies

[12] An alternative assumption is one where the government takes over the assets of generation-t agents who die prematurely and transfers them lump-sum to those alive. This gives qualititatively similar results as long as the transfers are made to surviving members of the same cohort. If, however, these assets are transferred to the progeny of the deceased (accidental bequests), asymptotic growth may result [Fuster, 1999].

that both labor and capital are paid their respective marginal products, so that prices are given by:

$$w_t = f(k_t) - k_t f'(k_t), \tag{4}$$

$$R_t = 1 + f'(k_t) - \delta, \tag{5}$$

where δ is the depreciation rate of physical capital.

2.2 General Equilibrium

The asset market clears when the capital stock at $t+1$ equals savings at t. When the tax rate is exogenously given and time-invariant, $\tau_t = \tau \ \forall t$, equilibria are characterized by sequences of $\{(k_t, h_t)\}$ that satisfy equations (1), (4), (5) and (6) − (8) below:

$$k_{t+1} = (1-\tau)\sigma(\widehat{R}_{t+1}, \phi_t)w(k_t), \tag{6}$$

$$h_t = \tau w(k_t), \tag{7}$$

$$\widehat{R}_{t+1} = R_{t+1}/\phi_t. \tag{8}$$

Substituting equations (1), (7) and (8) into (6) allows us to characterize the system by a single first-order difference equation in the capital stock:

$$k_{t+1} = (1-\tau)\sigma(k_{t+1}, k_t)w(k_t), \tag{9}$$

where $\sigma(k_t, k_{t+1}) \equiv \sigma\left(R(k_{t+1}), \phi(k_t)\right)$ and $\phi(k_t) \equiv \phi\left(\tau w(k_t)\right)$.

If youthful and old-age consumptions are gross substitutes, the propensity to save is increasing in the interest factor and decreasing in the future capital stock, k_{t+1}. An increase in the current capital stock k_t, on the other hand, allows society to invest more in health,

but has two opposing effects on the propensity to save. A higher capital stock allows for increased health expenditure. This raises the chance of survival, ϕ_t, and thus, the propensity to save out of lifetime income. At the same time, since it lowers the equilibrium return on savings, $\widehat{R}_{t+1}$, it tends to lower the propensity to save. As long as the direct length-of-life effect dominates the indirect interest rate effect, the savings propensity is increasing in k_t. Assuming this is true,[13] equation (9) describes a monotonically increasing phase map in (k_t, k_{t+1}) space.

It is now relatively straightforward to determine how equilibrium sequences of $\{k_t\}$behave. Consider an economy that starts out with relatively low capital per worker. Since resources are scarce, the economy is unable to invest much in the health of its population and mortality rates are high. High mortality rates make individuals effectively impatient, depressing saving and investment. The economy's future stock of capital is lower than it would otherwise be – the adverse effects of current high mortality spills into the future, constraining health choices that future generations are able to make. As long as high mortality does not severely depress savings, the economy grows by accumulating capital, only at a slower pace. When saving and investment are particularly sensitive to mortality, however, history matters: a low-income high-mortality society is unable to grow out of the 'vicious cycle' of poverty and ill-health.

To examine these ideas more closely, consider a version of the economy where the period utility function is logarithmic and the aggregate technology Cobb-Douglas: $u(c) = \log c$,

[13]For instance, when the period utility function is CES, $u(c) = c^{1-1/\sigma}/(1-1/\sigma)$, the savings propensity $\sigma = \phi R^{\sigma-1}/\left(1+\phi R^{\sigma-1}\right)$ is increasing in ϕ.

$f(k) = Ak^{\alpha}$, $\alpha \in (0,1)$, $A > 0$. The savings propensity is then simply $\phi/(1+\phi)$, so that

$$\sigma(k_t, k_{t+1}) \equiv \sigma(k_t) = \frac{\phi\left(\tau w(k_t)\right)}{1+\phi\left(\tau w(k_t)\right)}.$$

Equilibrium sequences of the capital stock now satisfy

$$k_{t+1} = (1-\tau)\sigma(k_t)w(k_t) \equiv J(k_t), \tag{10}$$

where,

$$J(k) \equiv (1-\tau)(1-\alpha)\left[\frac{\phi\left(Bk_t^{\alpha}\right)}{1+\phi\left(Bk_t^{\alpha}\right)}\right]Ak_t^{\alpha}, \quad B \equiv \tau(1-\alpha)A.$$

The asymptotic behavior of this economy depends upon whether or not equation (10) possesses multiple positive steady-states. When a unique positive steady-state exists, the economy converges to it asymptotically as in the standard model. When multiple steady-states exist, there are two of these. The lower one is asymptotically unstable, the higher one asymptotically stable. The proposition below specifies conditions under which this happens (refer to Appendix A for proof), while Figures 2 and 3 depict the dynamics of the economy.

Proposition 1 *The dynamical system described by equation (10) possesses three steady-states* $\{0, \overline{k}_1, \overline{k}_2\}$*, with* $\overline{k}_2 > \overline{k}_1$ *as long as* $\alpha > 1/2$*. The two extreme steady-states,* 0 *and* $\overline{k}_2$*, are asymptotically stable, while the intermediate one is not. When* $\alpha < 1/2$*, two steady states* $\{0, \overline{k}\}$ *exist, only the positive one being asymptotically stable.*

The economy converges monotonically to the positive steady-state $\overline{k}$, in Figure 2, irrespective of its initial position. Consider two countries that differ only in their initial health capital. In particular, both start with similar income levels but, for historical and

climatic reasons, individuals in one country enjoy the higher survival probability β. The dynamic behavior of the society with better health is described by the dotted line in Figure 2, whereas the solid phase-line applies to the high-mortality society. In the long run, both economies attain similar income levels. However, by virtue of having longer-lived citizens, the low mortality society grows faster during the early stages by accumulating assets faster. In the process, it is able to improve the living standards of all its transitional generations in a manner that the less healthy society cannot.

Figure 3 illustrates the starker case of history dependence. Two countries now need not converge to similar income levels even if they differ only in their initial health capitals. Here, unless a high mortality society starts out with a high enough capital stock (above $\overline{k}_1$), it is unable to escape the twin traps of poverty and ill-health. Proposition 1 suggests that such a trap exists when the output share of capital is greater than $1/2$.[14] Intuitively, this results from the dependence of the savings rate on the capital stock through health investment. The higher the capital-elasticity of wage income α, the more labor-income responds to the availability of capital: with $\alpha > 1/2$, accumulating capital allows for a relatively large increase in wages that may be invested more extensively in mortality reduction. In other words, small changes in the capital stock result in large gains in mortality reduction. In turn, this increased longevity provides impetus to capital accumulation and it is this sensitive

[14]Given existing estimates of the share of physical capital in the U.S., a value of α greater than 0.5 may be rationalized by broadening the concept of capital. By including human capital, we would expect the share to be in the range $(0.6, 0.8)$ as in Mankiw *et al.* [1992]. Incorporating business capital, on the other hand, gives an estimate of 0.71 as in Parente and Prescott [1994].

The exact restriction on α in Proposition 1 is, however, sensitive to our annuities markets assumption. If assets of the prematurely deceased were instead distributed lump-sum to existing members of that cohort, the restriction weakens to $\alpha > 1/3$.

two-way dependence that generates poverty traps.

Two clarifications are in order here. We have assumed so far that the health tax is time invariant. Appendix B shows that individuals in low-income countries also *prefer* a lower tax rate. When mortality rates are already high, the current utility cost of a higher health tax hurts more than it helps by way of improving future consumption possibilities. Besides being unable to invest enough resources in health, poorer economies also choose to underinvest in health when the gains from that investment are not realized immediately.[15]

Secondly, we have ignored the possibility that individuals may be altruistic. If households cared for their offsprings, they could substitute for their old-age consumption by bequeathing assets to their progeny. In that case, even when mortality rates are high, capital accumulation may not suffer as much since individuals save to pass on those assets to the next generation. Despite this, as long as individuals also cared for their old-age consumption or were not fully altruistic, higher mortality rates would depress economic growth [Ehrlich and Lui, 1991; Meltzer, 1995] and slow the process of convergence.

3 Mortality Risk and Investment in Education

Even when perfect annuities markets exist, mortality risks in certain kinds of investment may be undiversifiable. This is especially true of investments in "inalienable" human capital [Hart and Moore, 1994], like education.

Based on age-specific mortality rates and earnings profiles in developing countries,

[15]This may partly explain why high-mortality low-income countries tend to spend a much smaller fraction of GDP on public health [World Bank, 1993]. But as Table 1 suggests, developed countries also tend to suffer from more 'expensive' diseases.

Meltzer (1995) demonstrates that mortality declines can have large impact on school enrollment rates and significantly improve the social stock of human capital. The upshot is that when human capital is the engine of economic growth, mortality rates magnify initial cross-country differences in income and health into persistent differences in living standards.

This simple intuition is easy to capture with a minor modification of the previous analysis. Consider a similar setup except where individuals are endowed with 1 unit of labor time in youth as well as in old age. Denote by x_t the average stock of skills of the period-t workforce, now consisting of both young and old agents. Increments in x take the form of labor-augmenting technological improvements that once invented are never lost. Consequently, x_t represents the skills that old members of generation $t-1$ acquired through schooling (see below), as also the skills that the youth in period-t inherit from their preceding generation.

An individual born at t is effectively born with a labor efficiency of x_t. The individual can, however, choose to improve her productivity in the second period of life. She can do so by spending a fraction, $s_t \in [0,1]$, of her time in attending school when she is young. Her future productivity depends upon the inherited stock of skills and investment in education according to:

$$x_{t+1} = x_t \mu(s_t), \tag{11}$$

where μ is an increasing and concave function, satisfying $\mu(0) = 1$.

Time spent by a young individual in school is chosen to maximize her lifetime income,

$$(1-\tau)(1-s_t)w_t x_t + \frac{w_{t+1}x_{t+1}}{\widehat{R}_{t+1}}, \tag{12}$$

where τ is the time-invariant tax rate on youthful wage income. The first-order condition for an interior optimum equates returns on the two types of assets – physical and human capital:

$$\frac{(1-\tau)w_t}{w_{t+1}/\widehat{R}_{t+1}} = \mu'(s_t). \tag{13}$$

When μ is strictly concave, we can express optimal schooling choice as a function of prices:

$$s_t^* = s\left(\frac{\phi_t w_{t+1}}{w_t R_{t+1}}\right), \tag{14}$$

where s is increasing in its argument.

Equation (14) captures the essence of the rate-of-return argument. Through perfect market annuities, individuals can fully insure against mortality risks on physical capital investment but are unable to do so on their educational investment. Hence, a mortality decline raises the relative attractiveness of human capital. In equilibrium, another channel reinforces this effect: mortality decline promotes capital accumulation through the length-of-life effect, further raising the rewards to education in the form of higher future wages.

Interestingly, endogenous mortality risks may introduce threshold externalities of the type that Azariadis and Drazen [1990] elucidate. There, private returns to education depend upon the social stock of human capital through a technological externality. If this externality sharply rises when human capital crosses a threshold value, it elicits rapid investment in human capital and faster growth. Such externalities are one useful way to understand why developing economies may often stagnate for a while before "taking-off" into sustained growth.

Our human capital technology (11) does not explicitly allow for such externalities. Nev-

ertheless, when mortality rates are endogenously determined by *public* health investment, it alters *private* returns to education: one form of human capital injects an externality into investments in another form of human capital.

An easy way to see this is to assume that

$$\mu(s) = 1 + s^{\theta}, \quad \theta \in (0,1). \tag{15}$$

Assume also that this is a small open economy that can borrow and lend abroad at the fixed interest factor $\overline{R}$. This pins down the domestic capital stock at $\overline{k}$, and the wage rate at $\overline{w} \equiv w(\overline{k})$. Using (14), we can then solve for the economy's growth rate as,

$$g_t = \mu(s_t^*) - 1 = \left[\delta\phi(\tau\overline{w}x_t)\right]^{\theta/(1-\theta)}, \tag{16}$$

where $\delta \equiv \theta/[(1-\tau)\overline{R}]$. With constant mortality risks, say a survival probability equal to β, this economy would have immediately jumped to the steady-state growth rate of $(\delta\beta)^{\theta/(1-\theta)}$.

But when mortality is endogenous, the growth rate responds gradually to human capital accumulation since a rising stock of skills enables larger reductions in mortality rates. When μ is moderately concave ($\theta > 1/2$), sharp changes in the growth rate are observed for moderate changes in mortality rates (or, equivalently, knowledge capital).[16] Figure 4 illustrates this possibility of "take-off": starting from a low stock of human capital, the economy initially grows slowly and accelerates rapidly only when the social stock of skills attains a critical mass (near $\widehat{x}$).[17]

[16] This follows by considering the concavity of $g(x)$.

[17] This does not, however, allow multiple balanced growth paths with different (positive) rates of skill-investments to exist.

Another key implication of mortality is that small differences in health capital now imply not only large differences in income levels, but possibly in growth rates too. To examine this, let us return to the Cobb-Douglas technology and logarithmic preferences example.

Aggregate output now depends upon physical capital and efficiency labor supply (N), $Y_t = AK_t^{\alpha} N_t^{1-\alpha}$. Letting k denote capital per efficiency unit of labor, the intensive form of the production is again $y_t = Ak_t^{\alpha}$. To allow for multiple stable steady-states, we also assume a linear skill accumulation technology:

$$\mu(s) = 1 + \theta s, \quad \theta > 0. \tag{17}$$

An individual born in period-t maximizes lifetime utility (2) subject to the lifetime budget constraint (12) so that the savings function becomes

$$z_t = \sigma_t \left[(1-\tau)(1-s_t) w_t x_t - w_{t+1} x_{t+1} / R_{t+1} \right], \tag{18}$$

where $\sigma \equiv \phi/(1+\phi)$ denotes the savings propensity, as before.

Depending upon the initial stock of human capital, x_0, the economy exhibits two types of dynamic behavior: one, where no investment in schooling takes place, and another where individuals invest in skills through schooling.

Consider first corner equilibria with no schooling. From (13), this occurs when skill investment yields a return no greater than that obtainable with physical capital investment,

$$\theta \leq (1-\tau) \frac{w_t R_{t+1}}{\phi_t w_{t+1}}. \tag{19}$$

As long as (19) is satisfied, the social stock of skills remains constant at its initial value, $x_t = x_0 \ \forall t$. Efficiency labor supply is then,

$$N_t = x_t + \phi_{t-1} x_t = (1 + \phi_{t-1}) x_0.$$

Market clearing now requires that

$$N_{t+1}k_{t+1} = z_t.$$

Using (18) and equilibrium prices, this may be expressed as[18]

$$k_{t+1} = G(k_t), \tag{20}$$

where

$$\begin{aligned} G(k) &\equiv (1-\tau)(1-\alpha)\left[\frac{1}{1/[\sigma(k)(1-\sigma(k))] + (1-\alpha)/\alpha}\right] Ak^{\alpha}, \\ \sigma(k) &\equiv \frac{\phi(Bx_0k^{\alpha})}{1+\phi(Bx_0k^{\alpha})}, \quad B \equiv \tau(1-\alpha)A. \end{aligned}$$

Whether or not poverty traps occur under no-schooling depends upon the steady-states of equation (20). Not surprisingly, the dynamics here is similar to that obtained in the previous section: poverty traps result only when $\alpha > 1/2$. Otherwise, the economy converges monotonically to its unique (positive) steady-state capital stock.

Despite this similarity, there is now an important distinction. With solely physical capital investment, two economies that differed only in their initial survival probabilities converged, in the long-run, to the same steady-state whenever $\alpha < 1/2$. Better-health societies grew faster, but only temporarily. In sharp contrast, initial differences in health capital here may result in different outcomes in the long-run.

Consider, for instance, a high-mortality society for which the no-schooling restriction (19) is satisfied at initial levels of (x_0, k_0). A better-health society with the same initial

[18] In general equilibrium, the no-schooling restriction becomes $k_{t+1} \leq \alpha(1-\tau)A[k_t^{\alpha}/\phi(k_t)] \equiv H(k_t)$. Since $H(k) \geq G(k) \quad \forall k \geq 0$ (with strict equality at zero), an economy that starts at a no-schooling equilibrium never switches to one with schooling.

resources, on the other hand, may have a sufficiently higher survival probability (say, β) such that this restriction does not hold. As a result, even though both countries share similar economic conditions, the society whose citizens live longer finds it worthwhile to invest in knowledge creation and experiences positive balanced growth. Contrast this to the high-mortality society that remains stuck in a zero-growth equilibrium because its initial conditions do not favor human capital investment.

When initial conditions admit interior schooling equilibria in both countries, initial differences in mortality translate into widening income gaps, even though both converge to the same growth path.

Dynamic equilibria, in this case, satisfy the following pair of difference equations in (s_t, k_t):

$$k_{t+1} = \frac{\alpha(1-\tau)A}{\theta}\frac{k_t^{\alpha}}{\phi(k_t)}, \tag{21}$$

$$\left[1 - s_{t+1} + \phi(k_t) + \frac{1-\alpha}{\alpha}\sigma(k_t)\right](1+\theta s_t)k_{t+1} = (1-\tau)(1-\alpha)A(1-s_t)\sigma(k_t)k_t^{\alpha}. \tag{22}$$

Equation (21) follows from manipulating (13) using (17) and equilibrium prices, while substituting $N_t = [(1-s_t) + \phi_{t-1}]x_t$ into the market clearing condition gives equation (22).

Since ϕ_t converges to β asymptotically, the system described by these equations possesses a positive steady-state (s^*, k^*) that is saddle-path stable; given (x_0, k_0), all sequences of (s_t, k_t) converge monotonically to the steady-state growth rate of θs^*.[19]

Obviously, two countries with different initial mortality rates would now converge to the same balanced growth path in the long-run. But despite this convergence in growth rates,

[19]See Proposition 2 in Azariadis and Drazen (1990).

living standards in the two countries actually diverge. The low-mortality society always invests more intensively in skills at a higher rate and thereby augments its health capital at a faster pace. As a result it consistently enjoys a higher growth rate along its saddle-path than the country with higher mortality-risks.

4 Some Empirical Evidence

The theory we have developed has emphasized the importance of health and longevity in creating proper incentives for sustainable growth. But how empirically relevant is it? Can this explain why a number of countries, especially those in Sub-Saharan Africa, have languished in ill-health and poverty for so long? Is mortality indeed a significant determinant of educational investment?

To answer these questions, we turn to cross-country regression analysis in this section. The cross-country growth literature studies a gamut of issues ranging from the role of institutions, corruption, democracy, ethno-linguistic fractionalization, to the importance of capital accumulation, public infrastructure, natural resources, or international trade for economic growth. Only recently have researchers incorporated into their list of explanatory variables measures of health status. Those that have, for instance Barro and Sala-i-Martin [1995], Knowles and Owen [1995] and World Health Organization [1998], find health indicators to be significant predictors of future growth rates. The regressions in this section are in the spirit of those analyses, but are motivated by the specific linkages that our theory suggests.

The analysis is in terms of GDP per worker, rather than per capita, to control for

demographic shifts.[20] The time horizon considered is twenty years, covering 1970-90. The data used are for 95 countries, though the actual number of countries used in each regression differs depending upon data availability. Estimation is by ordinary least squares in all cases. Appendix C provides details about data sources and definitions.

Longevity was shown to have a level effect in Section 2. To test this we first regress the end-of-period per worker GDP, i.e., GDP per worker in 1990 (*LRGDP90*), on the average population growth rate (*LPOP*), the initial investment-to-GDP ratio (*LINVSH*), and the initial enrollment rate in secondary education (*ENROLS70*). The secondary enrollment rate is taken as a proxy for the initial stock of education capital. The result is reported in column (1) of Table 2. As the human capital augmented neoclassical model suggests, the investment rate and initial stock of human capital both significantly increase per capita GDP, while population growth reduces it. Column (2) adds another explanatory variable, life expectancy in 1970 (*LIFEXP70*) that the theory suggests has a significant effect on capital accumulation. The results are striking — all variables except for life expectancy, cease to be statistically significant. Indeed, in a regression of only life expectancy on the end-of-period GDP per worker, life expectancy explains about 81% of the cross-country variation in income levels (not reported in Table 2).

Our theory also suggests that longevity has a growth effect through human capital accumulation. The first step in testing this is to consider the relative effects of initial human capital and initial life expectancy on the average growth rate for 1970-90. The first regres-

[20]Demographic shifts are usually controlled for using fertility rates in GDP per capita regressions. However, since both mortality and fertility depend upon the underlying health stock, they are likely to be collinear.

sion, column (1) in Table 3, looks at the effect of the enrollment rate in 1970 (*ENROLS70*) on the subsequent average growth rate (*GR7090*), controlling for initial income per worker (*LGDPEA70*). Once again, as per the conditional convergence hypothesis, the coefficient on initial income is negative and highly significant. The initial stock of human capital positively and significantly affects the future growth rate.

The second regression, in column (2), excludes human capital, and looks at the forces of convergence versus initial life expectancy (*LIFE70*). The convergence prediction holds, while the effect of life expectancy is high and significant. Moreover, initial per capita income and life expectancy explain 42% of cross-country growth variation.

Column (3) of Table 3 includes both initial human capital and life expectancy as explanatory variables. Observe how *ENROLS70* ceases to be significant at the 5% level, while *LGDPEA70* and *LIFE70* have the correct signs and continue to be statistically significant. In other words, the theoretical predictions are consistent with the evidence.

Cross-country growth regressions often incorporate a dummy for Sub-Saharan Africa. The coefficient for this dummy is usually negative, suggesting geographical factors at work. To see how much of that is region-specific and how much simply the result of high mortality, we next include a dummy for Sub-Saharan Africa (*SSAFRICA*). Also included is the average share of investment in GDP between 1965-69 (*INVSH*). Columns (1) and (2) of Table 4 examine the result of this exercise with and without initial life expectancy. Inclusion of life expectancy in the regression makes the initial stock of human capital insignificant. It also takes away the influence of the regional dummy variable, suggesting that Sub-Saharan countries are poor not due to region-specific characteristics, but because life expectancies

there are the lowest in the world. Moreover, the coefficient on the investment-to-GDP ratio has the incorrect sign, although statistically not different from zero. Here too, a large part of the effect of investment-to-GDP ratio on the growth rate seems to be explained by its dependence on longevity.

Finally, as a crude sensitivity check, the regression model is extended to include several other variables commonly used in cross-country regressions. These are the black-market premium for exchange rates (*BMP6590*) as an indicator of corruption, terms of trade effects (*TOT6590*), share of primary goods in exports (*SXP*) as an index of natural resources, the number of telephones per worker as an index of public infrastructure (*LTELPW*), and the initial total fertility rate (*TFR70*), to control for fertility induced demographic shifts. The results appear in column (3) of Table 4. The only significant variables are initial GDP per worker, initial life-expectancy and the share of primary goods in total exports.[21] All other variables have the correct signs but none is highly significant.

Cross-country regressions are well known for their sensitivity to the list of variables included [Levine and Renelt, 1992]. It is therefore encouraging to note that the only two variables that strongly predict future growth rates in the regressions presented here are initial income and longevity. In particular, human capital, in the form of education, public infrastructure or other policy measures do not appear to be substantive factors contributing to economic growth once a mortality measure is included. As Table 3 shows, most of the variability in growth rates comes from initial incomes and initial differences in health.

[21]The negative association between natural resources and economic growth was pointed out in Sachs and Warner [1995]. Asea and Lahiri [1999] suggest that this results from the effect of natural resource abundance on human capital investment.

The evidence complements what we already know from other micro- and disease-specific studies on the role of health. More rigorous econometric methods, however, are necessary to ascertain the true magnitude of health's contribution to economic growth.

5 Conclusion

Widespread evidence, at both individual and aggregate levels, points to a strong correlation between poverty and ill-health. This is often ascribed to the adverse impact of poverty on health, but recent evidence suggests that health could equally well be an important determinant of economic welfare. This paper has examined how better health, by improving longevity and reducing mortality risks, may be conducive to growth and development.

The analysis points to health investment as a prerequisite for sustainable economic growth. In particular, savings and investment rates are systematically low in high mortality societies since low life expectancy raises the effective impatience of individuals and decreases returns on human capital investment. The implication is that such societies would not grow as fast as standard theory predicts even when technology is not the bottleneck. Mortality risks may indeed be so growth-depressing as to result in 'development traps'. These results are broadly consistent with existing evidence on cross-country growth and regression results indicate the growth effects of longevity improvements to be significantly large.

However, to better gauge the importance of health, one needs to quantify its contribution better. There are two ways of doing this. One approach is use cross-country evidence and more sophisticated econometric methods than have been used here. Since cross-country analyses suffer from a variety of problems, a more promising approach may be the use

of quantitative methods, as have been used in McGrattan and Schmitz's [1998] analysis of cross-country growth or Kalemli-Ozcan *et al.*'s [1998] study of the effects of (exogenous) mortality decline. If the theory presented here is any indication, health capital could explain a surprisingly large portion of cross-country income and growth differentials.

Appendix A: Proof of Proposition 1

To prove Proposition 1, we establish the following lemma.

Lemma 1

The function $J(k)$ defined in equation (10) satisfies the following properties:

(i) $J(0) = 0$,

(ii) $J'(k) \geq \quad \forall\ k \geq 0$,

(iii) $\lim_{k\to\infty} J(k)/k < 1$, and

(iv) $\lim_{k\to 0}\ J'(k) = \infty$ iff $\alpha < 1/2$.

Proof. The first two properties are easy to check. To prove (iii), note that $\lim_{h\to\infty} \sigma = \beta/(1+\beta)$. Therefore,

$$\lim_{k\to\infty} \frac{J(k)}{k} = C \lim_{k\to\infty} \frac{\sigma(k)}{k^{1-\alpha}} = C\left(\frac{\beta}{1+\beta}\right) \lim_{k\to\infty} \frac{1}{k^{1-\alpha}} = 0,$$

where $C \equiv (1-\alpha)(1-\tau)A$.

Now,

$$J'(k) = C(\sigma_k k^{\alpha} + \alpha\sigma k^{\alpha-1}),$$

where,

$$\sigma_k = \alpha\tau(1-\alpha)A\frac{\phi'}{(1+\phi)^2}k^{\alpha-1},$$

using the chain rule. Therefore, using $\lim_{k\to 0}\phi'(\tau(1-\alpha)Ak^{\alpha})=\gamma$, we obtain

$$\begin{aligned}\lim_{k\to 0}J'(k) &= C\lim_{k\to 0}\left[\alpha\tau(1-\alpha)A\frac{\phi'}{(1+\phi)^2}k^{2\alpha-1}+\alpha\sigma k^{\alpha-1}\right]\\ &= C\left[\alpha\tau(1-\alpha)\gamma A\lim_{k\to 0}k^{2\alpha-1}+\alpha\lim_{k\to 0}\frac{\sigma_k}{(1-\alpha)k^{-\alpha}}\right]\ \text{(using L'Hopital's Rule)}\\ &= \alpha C\left[\tau(1-\alpha)\gamma A\lim_{k\to 0}k^{2\alpha-1}+\tau\alpha\gamma A\lim_{k\to 0}k^{2\alpha-1}\right]\\ &= \infty \text{ iff } \alpha<1/2.\end{aligned}$$

When $\alpha>1/2$, on the other hand, $\lim_{k\to 0}J'(k)=0$. ■

Proposition 1 follows immediately from these results. By (i), 0 is always a steady-state of (10). By (ii) and (iii), the phase map is monotonically increasing and eventually falls below the 45^o line, so that at least one positive steady-state exists. Whether or not multiple such steady-states exist depends upon the stability of 0. By (iv), 0 is a stable steady-state *iff* $\alpha>1/2$, intersecting the 45^o line from below. In that case, $J(k)$ intersects the 45^o line from below at least once before falling below it. Hence, at least one positive steady-state separates 0 from the asymptotically stable highest one.

Appendix B: The 'Optimal' Tax Rate

Current public health expenditure benefits only the currently young, who also bear the cost of funding these investments. Therefore young agents born at t choose τ_t to maximize their expected lifetime utility. Using the probability function (1), this maximization problem becomes:

$$\text{Max}_{\{z_t,\tau_t\}}\log\left[(1-\tau_t)w_t-z_t\right]+\phi(\tau_t w_t)\log\left[\widehat{R}_{t+1}z_t\right].$$

The first-order condition for z_t gives:

$$z_t = (1 - \tau_t) \left[\frac{\phi_t}{1 + \phi_t} \right] w_t, \tag{B.1}$$

while that for τ_t equates the marginal utility cost to the (discounted) marginal utility gain from a greater possibility of surviving:

$$\frac{w_t}{c_t^t} = w_t \phi'(\tau_t w_t) \log \left[\widehat{R}_{t+1} z_t \right] \tag{B.2}$$

(B.1) can be used to simplify (B.2) and obtain an equation solely in the tax rate τ_t, given the price vector $(w_t, \widehat{R}_{t+1})$:

$$\Omega(\tau_t) \equiv \frac{1 + \phi(\tau_t w_t)}{1 - \tau_t} = w_t \phi'(\tau_t w_t) \log \left[\widehat{R}_{t+1} (1 - \tau_t) w_t \frac{\phi(\tau_t w_t)}{1 + \phi(\tau_t w_t)} \right] \equiv \Lambda(\tau_t).$$

Ω is monotonically increasing in τ, while Λ is inverted U-shaped satisfying $\lim_{\tau \to 0} \Lambda = -\infty = \lim_{\tau \to 1} \Lambda$. When $\Omega(\tau)$ and $\Lambda(\tau)$ intersect, they do so twice; the optimal tax rate τ^* is obtained at the higher point of intersection. Moreover, τ^* depends positively upon wage income, w, as well as on the interest factor, $\widehat{R}$.

Appendix C: Data Sources

The data used in Section 4 are from several sources. The list of variables, their definitions and sources are listed below. The Sachs-Warner [1995] dataset is available at

"http://www.nuff.ox.ac.uk/Economics/Growth/datasets.htm",

the Easterly-Levine [1997] dataset at

"http://www.worldbank.org/html/prdmg/grthweb/datasets.htm",

and the Barro-Lee [1993] dataset at both sites.

LRGDP90: Real GDP per capita in constant dollars (expressed in international prices, base 1985). *Source:* The Penn World 5.6 [see Summers and Heston, 1991]

ENROLS70: Total gross enrollment ratio for secondary education in 1970, from UNESCO. *Source*: Barro-Lee [1993].

LPOP: Log of average population growth rate between 1970-90. *Source*: World Bank [1998].

INVSH: Average ratio of real domestic investment (private plus public) to real GDP between 1965 and 1969. *Source*: The Penn World Table 5.6.

LINVSH: Natural logarithm of *INVSH*.

LIFEXP70: Life expectancy at birth in 1970. *Source:* World Bank [1998]

LIFE70: Natural logarithm of *LIFEXP70*.

LGDPEA70: Natural logarithm of real (purchasing power parity adjusted) GDP per economically active population in 1970. GDP data are from the Penn World Tables, Mark 5.6, and are in constant 1985 international prices. The economically active population is defined

as the population between the ages 15-64 and is taken from the World Data CD-ROM, 1995, World Bank. *Source:* Sachs-Warner [1995].

GR7090: Average annual growth in real GDP per economically active population between 1970 and 1989. Based on the same original sources as *LGDPEA70*. *Source:* Sachs-Warner [1995].

SSAFRICA: Dummy variable for Sub-Saharan Africa.

BMP6590: Average black market premium between 1965-69. BMP = (black market exchange rate/official exchange rate) -1, where the exchange rate = local currency per dollar. *Source*: Barro-Lee [1993].

TOT6569: Terms of trade shock (growth rate of export prices minus growth rate of import prices) between 1965-69. *Source:* Barro-Lee [1993].

SXP: Share of exports of primary products in GNP in 1970. *Source:* Sachs-Warner [1995].

LTELPW: Log of telephones per 1000 workers. *Source*: Easterly-Levine [1997].

Bibliography

1. Asea, Patrick and Amartya Lahiri, "The Precious Bane", *Journal of Economic Dynamics and Control,* **23** (1999), 823-849.

2. Azariadis, Costas and Allan Drazen , "Threshold Externalities in Economic Development", *Quarterly Journal of Economics*, **105** (1990), 501-26.

3. Barro, Robert J. and Jong-Wha Lee, "International Comparisons of Educational Attainment", *Journal of Monetary Economics*, **32** (1993), 363-94.

4. Barro, Robert J. and Xavier Sala-i-Martin, *Economic Growth,* McGrawHill, (1995).

5. Blackburn, Keith and Giam Pietro Cipriani, "Endogenous Fertility, Mortality and Growth", *Journal of Population Economics*, 11 (1998), 517-34.

6. Blanchard, Olivier J., "Debt, Deficits and Finite Horizons", *Journal of Political Economy,* **93** (1985), 223-47.

7. Dasgupta, Partha, *An Inquiry into Well-Being and Destitution*, Oxford University Press, New York, NY (1993).

8. Deaton, Angus and Christina Paxson, "Saving, Growth, and Aging in Taiwan", in Wise, David A., ed.. *Studies in the Economics of Aging,* NBER Project Report series, University of Chicago Press (1994), 331-57.

9. de la Croix, David and Omar Licandro, "Life expectancy and endogenous growth", *Economics Letters* **65** (1999), 255-263.

10. Easterly, William and Ross Levine, "Africa's Growth Tragedy: Policies and Ethnic Divisions," *Quarterly Journal of Economics,* **112** (1997), 1203-50.

11. Ehrlich, Isaac and Francis T. Lui, "Intergenerational trade, Longevity and Economic growth," *Journal of Political Economy* **99**(1991), 1029-1059.

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

13. Fuster, Luisa, "Effects of Uncertain Lifetime and Annuity Insurance on Capital Accumulation and Growth", *Economic Theory* **13** (1999), 429-445.

14. Gallup, John Luke and Jeffrey D. Sachs, "The Economic Burden of Malaria: Cross-Country Evidence", in World Health Organization (1998).

15. Gersovitz, Mark, "Savings and Nutrition at Low Incomes", *Journal of Political Economy* **91**(1983), 841-55.

16. Hart, Oliver and John Moore (1994), "A Theory of Debt Based on the Inalienability of Human Capital", *Quarterly Journal of Economics* **109**, 841-79.

17. Kalemli-Ozcan, Sebnem, Ryder, Harl E. and David N. Weil, "Mortality Decline, Human Capital Investment and Economic Growth", *mimeo*, Brown University (1998).

18. Knowles, Stephen and P. Dorian Owen, "Health Capital and cross-country variation in income per capita in the Mankiw-Romer-Weil Model", *Economics Letters* **48** (1995), 99-106.

19. Kotlikoff, Laurence J. and Avia Spivak, "The Family as an Incomplete Annuities Market", *Journal of Political Economy* **89** (1981), 372-91.

20. Lawrance, Emily, "Poverty and the Rate of Time Preference: Evidence from Panel Data", *Journal of Political Economy* **99** (1991), 54-77.

21. Leibenstein, Harvey, *Economic Backwardness and Economic Growth*, John Wiley & Sons, New York (1957).

22. Levine, Ross and David Renelt, "A Sensitivity Analysis of Cross-Country Growth Regressions", *American Economic Review* **82** (1992), 942-963.

23. Lewis, W. Arthur, "Economic Development with Unlimited Supplies of Labour", *Machester School Econ. and Soc. Sci.* **22** (1954), 139-191.

24. Lucas, Robert, "On the Mechanics of Economic Development", *Journal of Monetary Economics* **22** (1988), 3-42.

25. Mankiw, Gregory N., David Romer and David N. Weil, "A Contribution to the Empirics of Economic Growth", *The Quarterly Journal of Economics* **107** (1992), 407-437.

26. McCarthy, Desmond, Wolf, Holger and Yi Wu, "The Growth Costs of Malaria", *NBER Working Paper* No. W7541 (2000).

27. McGrattan, Ellen R., and James A. Schmitz, "Explaining Cross-Country Income Differences", *Research Department Staff Report 250*, Federal Reserve Bank of Minneapolis (1998).

28. Meltzer, David, "Mortality Decline, the Demographic Transition and Economic Growth", *mimeo*, University of Chicago (1995).

29. Parente, Stephen L. and Edward C. Prescott, "Barriers to Technology Adoption and Development," *Journal of Political Economy* **102** (1994), 298-321.

30. Ram, Rati and Theodore W. Schultz, "Life Span, Health, Savings and Productivity", *Economic Development and Cultural Change* **27** (1979), 394-421.

31. Romer, Paul, "Increasing Returns and Long Run Growth, *Journal of Political Economy* **98** (1986), 1002-37.

32. Sachs, Jeffrey and Andrew M. Warner, "Natural Resource Abundance and Economic Growth", *NBER Working Paper 5398* (1995).

33. Strauss, John and Duncan Thomas, "Health, Nutrition, and Economic Development", *Journal of Economic Literature* **XXXVI** (1998), 766-817.

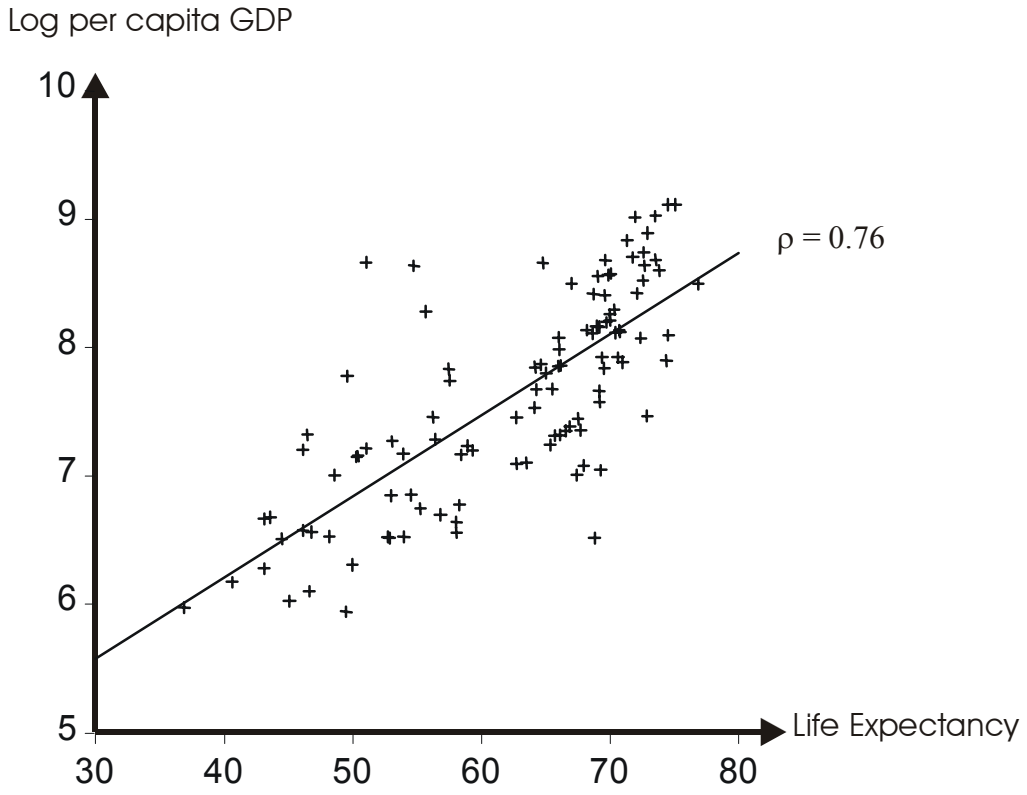
34. Summers, Robert and Alan Heston, "The Penn World Table (Mark 5): An Expanded Set of International Comparisons, 1950-1988", *Quarterly Journal of Economics* **106** (1991), 327-368.

35. World Bank, *World Development Report: Investing in Health*, Oxford University Press, New York, (1993).

36. World Bank, *World Development Indicators 1998 on CD-ROM*, World Bank, Washington D.C, (1998).

37. World Health Organization, *Health, Health Policy, and Economic Outcomes*, prepared by the Health and Development Satellite, WHO Transition Team (1998).

38. Yaari, Menahem E., "Uncertain Lifetime, Life Insurance, and the Theory of the Consumer", *Review of Economic Studies* **32** (1965), 137-50.



Source: World Bank (1998). Per capita GNP is measured in PPP$.
These 109 low- and middle-income countries are those
in which 1996 per capita GNP was $9,635 or less.

Figure 1: Life Expectancy at Birth and Log Per Capita GDP

Cause of Death (%)	**Developing Countries**	**Developed Countries**
Infectious and parasitic diseases	45	5
Diarrhoeal	(13)	-
Tuberculosis	(8)	-
Acute respiratory illness	(17)	3
Perinatal diseases	8	1
Cancers	7	21
Circulatory and degenerative diseases	17	54
Injury	6	7
Other causes	17	12

Source: Dasgupta (1993), Table 4.3

Table 1: Causes of Deaths in Developing and Developed Countries

Dependent Variable: *LRGDP90* (t-statistic in brackets)		
	(1)	**(2)**
CONSTANT	6.77	3.62
	(13.23)	(6.05)
LPOP	-0.33	-0.09
	(-2.87)	(-0.87)
LINVSH	0.39	0.06
	(3.19)	(0.62)
ENROLS70	1.81	0.55
	(4.31)	(1.39)
LIFEXP70		0.07
		(7.59)
R^2	0.71	0.83
$\overline{R}^2$	0.70	0.82
OBS.	86	84

Table 2: Testing for the Length-of-Life Effect

Dependent Variable: *GR7090* (t-statistics in brackets)			
	(1)	**(2)**	**(3)**
CONSTANT	6.16	-25.85	-21.00
	(2.72)	(-7.44)	(-4.23)
LGDPEA70	-0.73	-1.70	-1.80
	(-2.36)	(-5.53)	(-5.63)
ENROLS70	4.45		1.36
	(4.44)		(1.35)
LIFE70		10.29	9.17
		(7.90)	(5.94)
R^2	0.19	0.42	0.42
$\overline{R}^2$	0.17	0.41	0.41
OBS.	94	94	93

Table 3: Mortality Risk, Education and Growth Rates

Dependent Variable: *GR7090* (t-statistic in brackets)			
	(1)	**(2)**	**(3)**
CONSTANT	9.14	-18.59	-9.69
	(3.90)	(-2.63)	(-1.10)
LGDPEA70	-1.14	-1.79	-2.89
	(-3.75)	(-5.53)	(-5.71)
ENROLS70	3.11	1.43	0.27
	(3.07)	(1.40)	(0.24)
INVSH	1.65	-0.06	0.45
	(1.85)	(-0.07)	(0.45)
LIFE70		8.58	8.34
		(4.12)	(3.71)
SSAFRICA	-1.40	-0.33	
	(-2.93)	(-0.63)	
BMP6590			-0.52
			(-0.78)
TOT6569			-5.02
			(-1.52)
SXP			-5.63
			(-2.62)
LTELPW			0.55
			(1.89)
R^2	0.31	0.42	0.55
$\overline{R}^2$	0.28	0.39	0.48
OBS.	94	93	73

Table 4: Effect of Longevity on Cross-country Growth

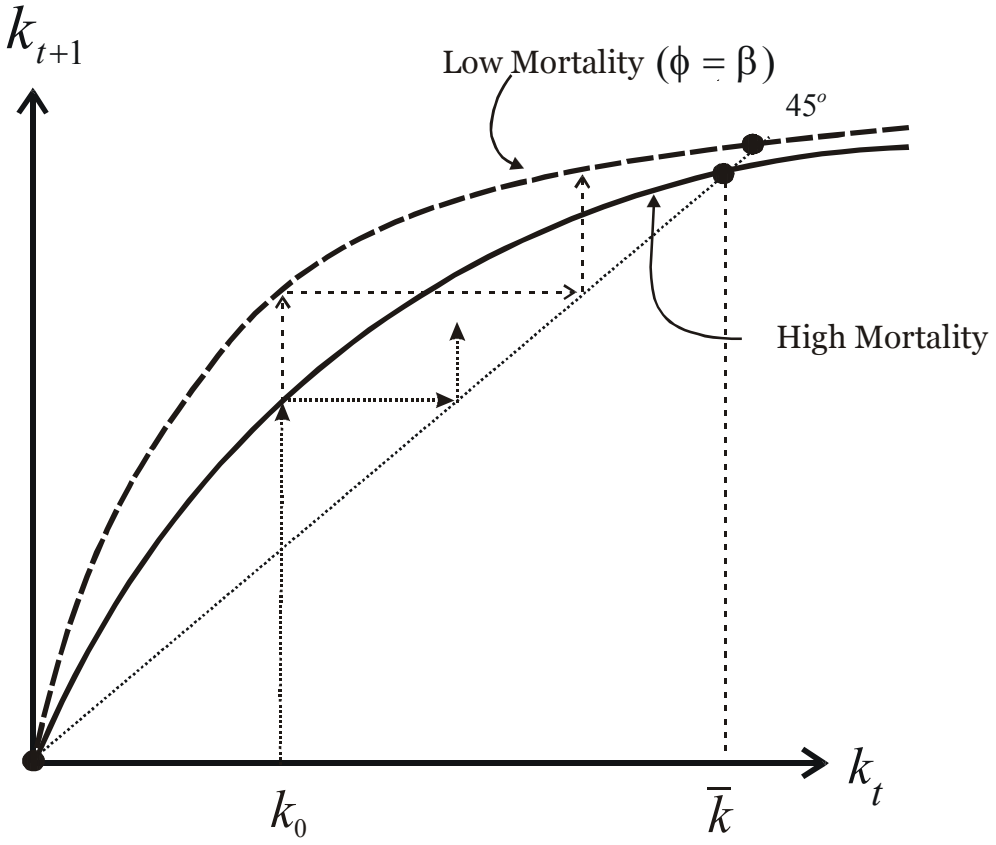


Figure 2: Convergence under the Length-of-Life Effect

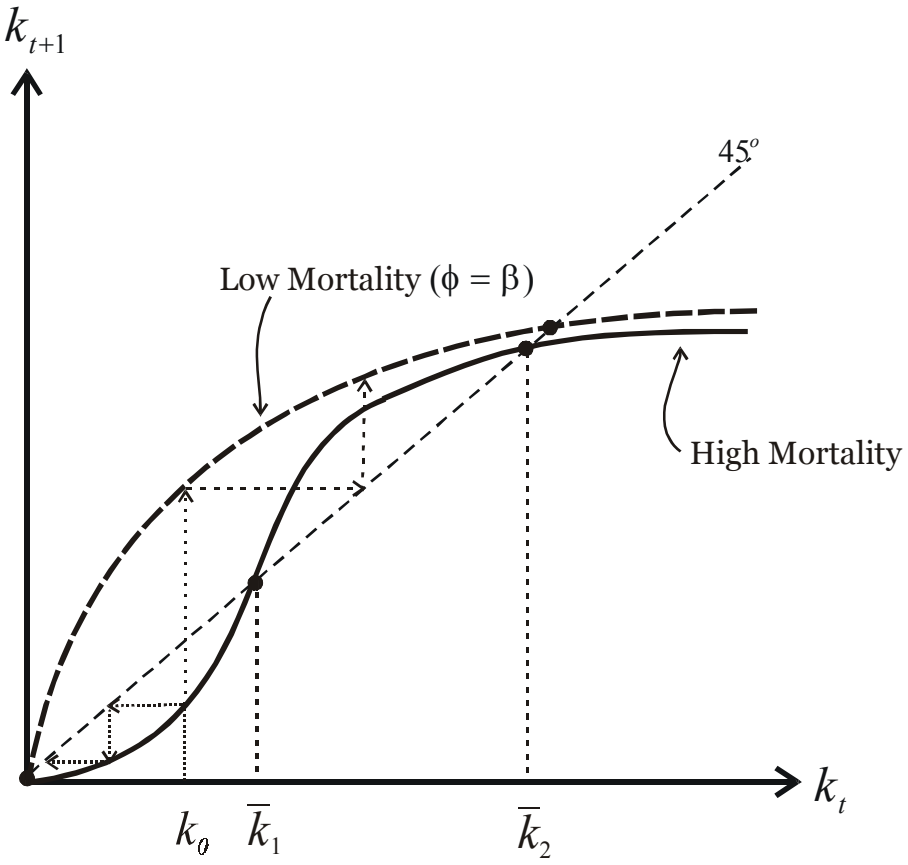


Figure 3: Non-convergence under the Length-of-Life Effect

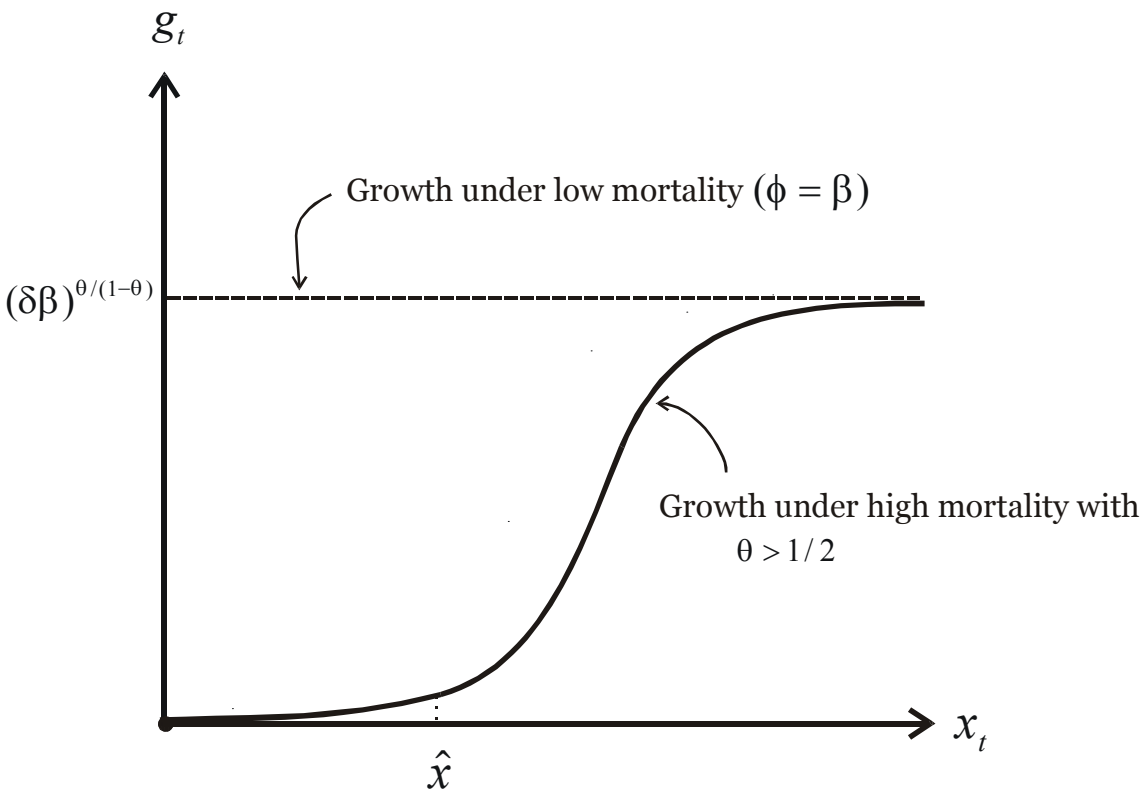


Figure 4: Threshold Effects and Mortality Risk