Endogenous lifetime and economic growth

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Abstract

Endogenous mortality is introduced in a two-period overlapping generations model: probability of surviving from the first period to the next depends upon health capital that is augmented through public investment. High mortality societies do not grow fast since shorter lifespans discourage savings; development traps are possible. Productivity differences across nations result in persistent differences in capital-output ratios and relatively larger gaps in income and mortality. High mortality also reduces returns on education, where risks are undiversifiable. When human capital drives economic growth, countries differing in health capital do not converge to similar living standards, ‘threshold effects’ may also result.

JEL classification: I1; I2; O1

Keywords: Health; Life expectancy; Mortality; Growth; Human capital

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 [28]. This paper studies the effect of such pervasive ill-health, especially high mortality, on economic growth.

Health status and income are well known to systematically vary across nations. Fig. 1 displays one such relationship, between life expectancy at birth and per capita income for low- and middle-income countries in 1996. Life expectancy 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 12 times richer. Adult mortality risk, the probability that a typical 15-year old would die before reaching age 60, was three times as high in sub-Saharan Africa as in the established market economies in 1990 [28]. Conventional wisdom attributes the severity of mortality in poorer countries to widespread poverty and inadequate living conditions. We explore the possibility that high mortality distorts savings incentives and leads to poverty.

The literature on health human capital has largely focused on the implication of health and nutrition for labor market outcomes. Yet health plays a role quite unlike any other human capital: by increasing lifespans it makes individuals effectively more patient and willing to invest, and by reducing mortality risks, it raises the return on investment.

To capture this simple intuition, we construct a general equilibrium model that allows health capital and per capita income to evolve simultaneously. This is done by introducing mortality 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 poorer societies, when life expectancy is low, individuals discount the future more heavily and are less inclined to save and invest. Due to pervasive poverty, neither can they afford to spend much on public health. Low income and high mortality thus reinforce each other. During the early stages of development, a high-mortality society grows slower than what standard one-sector models predict. Poverty traps may result if the incentive to invest is particularly sensitive to health accumulation. Importantly, endogenous mortality introduces a multiplier effect through the savings rate: cross-country differences in productivity get amplified into...
persistent differences in capital-output ratios, and large differences in output per worker and longevity.

Mortality also affects investment through rates of return. Risks associated with investment in education, for instance, may not be fully diversifiable. Higher mortality would then reduce returns on such investment. Recent growth theories, Lucas [21] for example, 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. When mortality considerations affect schooling decisions, countries that differ only in health capital do not converge to similar living standards. Threshold effects of the type Azariadis and Drazen [1] analyze may also result as an economy goes through a phase of slow growth and modest health improvements before taking-off into sustained prosperity. This paper complements several pieces of work in the literature. Among others, Gersovitz [11], Ram and Schultz [24], and more recently, the World Bank [28] 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. Theoretical studies on mortality and growth have mostly considered exogenous mortality declines. Issues analyzed include the effect of longevity improvements on growth and human capital investment [8,14], and how declines in child mortality alter incentives to have children and induce a demographic transition [10,23].

The rest of the paper is organized as follows. Section 2 analyzes the effect of endogenous mortality on capital accumulation in an overlapping generations economy. The effect of mortality risk on educational investment is examined in Section 3. In Section 4, we present some cross-country evidence in support of the specific linkages stressed by the theory and conclude the discussion in Section 5.

2. Length-of-life and capital accumulation

We begin by considering the length-of-life effect. In an otherwise standard two-period overlapping generations model, the probability of surviving from the first period to the next is endogenous and depends upon health. In particular, an individual’s health status is determined by public health measures such as provision of clinical facilities, sanitation, inoculation and disease control programs.

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1 Development theories often posit a higher saving propensity for the rich (see [18,20]). Lawrance [17] finds subjective rates of time preference to be higher for poorer households, implying less willingness to save. The length-of-life effect is one way to explain both. Deaton and Paxson [7] attribute the higher savings rate in younger cohorts in Taiwan (1976–1990) to longevity improvements, among other factors.

2 Forty-five percent of deaths in developing countries occur due to infectious and parasitic diseases which create externalities [6, Table 4.3]. Hence public interventions can avoid the underinvestment that would result from private choices.

Two papers that incorporate endogenous mortality are Gersovitz [11] and Blackburn and Cipriani [3]. Gersovitz’s model is a partial equilibrium one where mortality risks decline with consumption early in life. Blackburn and Cipriani focus on the effects of infant mortality on fertility and capital accumulation.
2.1. The environment

Young agents of measure one are born each period with a time endowment of 1 unit which they inelastically supply to the labor market, earning a wage income \( w \). The survival probability of a young person born at \( t \), \( \phi_t \),\(^3\) depends upon her health capital, \( h_t \), and is given by a non-decreasing concave function

\[
\phi_t = \phi(h_t)
\]

that satisfies \( \phi(0) = 0 \), \( \lim_{h \to \infty} \phi(h) = \beta \leq 1 \) and \( \lim_{h \to 0} \phi'(h) = \gamma < \infty \).\(^4\)

Public health expenditure in period-\( t \) is financed through a proportional tax \( \tau_t \in (0, 1) \) on labor income so that health investment per young person equals \( \tau_t w_t \). Such investment augments private health capital through a constant returns technology:

\[
h_t = g(\tau_t w_t) = \tau_t w_t.
\]

A generation-\( t \) individual gives birth to one offspring at the end of \( t \), before she realizes her mortality shock. This new individual becomes economically active only at the beginning of \( t + 1 \), and in particular, does not inherit her parent’s health stock.\(^5\)

To abstract from the risk associated with uncertain lifetimes, we follow Blanchard [4] and Yaari [30] in assuming a perfect annuities market whereby all savings are intermediated through mutual funds.\(^6\) At the end of her youth, each individual deposits her savings with a mutual fund. The mutual fund invests these savings in capital (the sole asset) and guarantees a gross return of \( \hat{R}_{t+1} \) to the surviving old.\(^7\) If a fund earns a gross return \( R_{t+1} \) on its investment, then perfect competition ensures that in equilibrium, \( \hat{R}_{t+1} = R_{t+1}/\phi_t \).

A person born in \( t \) maximizes her expected lifetime utility,

\[
U_t = \ln c_t + \phi_t \ln c_{t+1},
\]

subject to the budget constraints

\[
c_t \leq (1 - \tau_t)w_t - z_t, \quad c_{t+1} \leq \hat{R}_{t+1}z_t,
\]

taking as given the vector of prices \( (w_t, \hat{R}_{t+1}) \). Here \( z \) denotes savings in youth. With logarithmic preferences, optimal savings takes the simple form

\[
z_t = (1 - \tau_t)\sigma_t w_t,
\]

\(^3\) Since a new-born expects to live for \( 1 + \phi \) periods, we use the terms ‘life expectancy’, ‘length of life’ and ‘survival probability’ interchangeably.

\(^4\) One example that satisfies these properties is \( \phi(h) = \beta h/(1 + h) \) with \( \gamma = \beta \).

\(^5\) Incorporating health transfers through birth adds one more dimension to the dynamical system without altering any of our basic insights.

\(^6\) If annuity markets were not perfect, a lower mortality rate would raise returns on investment and encourage savings. In that case, results that follow understate true magnitudes. But even when annuity markets are not well-developed, households can self-insure against mortality risks through interfamily transfers, substituting by more than 70% for perfect market annuities [16].

\(^7\) 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 qualitatively similar results as long as the transfers are made to surviving members of the same cohort.
where the savings propensity \( \sigma_t \equiv \phi_t / (1 + \phi_t) \) is an increasing function of the survival probability.

Final goods are produced using a technology, \( F(K, L) \), that is constant returns in aggregate capital and labor. It will be convenient to assume this technology is Cobb–Douglas, \( F(K, L) = AK^\alpha L^{1-\alpha} \), with \( \alpha \in (0, 1) \) and \( A > 0 \). Output per worker is then

\[
f(k) = Ak^\alpha,
\]

with \( k \) denoting capital per worker. Perfect competition in the final goods market implies that both labor and capital are paid their respective marginal products:

\[
w_t = w(k_t) = (1 - \alpha)Ak_t^\alpha,
\]

\[
R_t = R(k_t) = 1 + \alpha Ak_t^{\alpha-1} - \delta,
\]

where \( \delta \) is the depreciation rate on physical capital.

### 2.2. General equilibrium

Consider an exogenously given and constant health tax, \( \tau_t = \tau \) for all \( t \). Competitive equilibria are characterized by sequences of \( \{ (k_t, h_t) \} \) that satisfy Eqs. (8) and (9) below

\[
k_{t+1} = (1 - \tau)\sigma(\phi_t)w_t,
\]

\[
\hat{R}_{t+1} = R_{t+1}/\phi_t,
\]

together with (1), (2), (6) and (7), and given an initial capital-labor ratio \( k_0 \). Substituting equilibrium prices and health investment into (8) allows us to characterize the general equilibrium by a single first-order equation,

\[
k_{t+1} = (1 - \tau)(1 - \alpha)\sigma(k_t)Ak_t^\alpha,
\]

given \( k_0 \) and where,

\[
\sigma(k) \equiv \frac{\phi(\tau(1 - \alpha)Ak^\alpha)}{1 + \phi(\tau(1 - \alpha)Ak^\alpha)}.
\]

We are interested in examining how life expectancy affects output per worker over time and across nations. Suppose that initial cross-country income differences result solely from differences in \( k_0 \). An economy that starts out with relatively low capital realizes low levels of income so that it is unable to adequately invest in the health of its population. High mortality rates lead individuals to heavily discount the future and save less. The future stock of capital is thus low, constraining future health and economic outcomes. High mortality and low income thus tend to reinforce each other.

Whether or not differences in initial income and mortality persist, depend upon the uniqueness of positive steady states of Eq. (10). When a unique positive steady state exists, it is asymptotically stable. Initial differences in \( k_0 \) do not matter in the long-run since all economies grow toward the unique steady state \( \bar{k} \). A similar result obtains when economies differ in their initial health capital. In particular, suppose two economies start with similar income levels but, for historical and climatic
reasons, individuals in one country enjoy the higher survival probability $\beta$. Fig. 2 illustrates this scenario. The dynamic behavior of the society with better health is described by the dotted line while the solid phase-line applies to the high-mortality society. Both economies attain similar income levels in the long-run, but convergence is faster under low mortality.

Non-ergodicity of growth paths result, in contrast, in Fig. 3 where two positive steady states exist. Unless a high-mortality society starts out with a high enough capital stock (above $k_1$), it is unable to escape the vicious cycle of poverty and ill-health. Such a trap exists when the output elasticity of capital exceeds $1/2$ (see Proposition 1 below). With $\alpha > 1/2$, accumulating capital allows for a relatively large increase in wages that may be invested more extensively in mortality reduction. Hence, small changes in the capital stock result in large life expectancy gains that, in turn, provide impetus to capital accumulation.\(^8\)

Recent research, [12,15] for example, has shown technological differences to be at least as important as factor intensities in accounting for observed cross-country income disparities. Differences in the technology parameter $A$ are estimated to account for between 25% and 67% of cross-country differences in output per worker.

Suppose now that incomes differ across countries solely due to $A$. Convergence dynamics for $\alpha < 1/2$ obviously do not imply convergence in living standards any longer since differences in $A$ lead to differences in steady-state output per worker.

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\(^8\)Given existing estimates of $\alpha$ in the US, a value 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 [22], while incorporating organizational capital gives an estimate of 0.71 as in [5]. The exact restriction on $\alpha$ 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 $\alpha > 1/3$. 
But the model implies more. In the standard neoclassical model (or in a model with constant mortality), variations in \( A \) do not affect the steady-state capital-output ratio [5]. A neoclassical growth theory that seeks to understand international differences in output per worker through \( A \) thus fails to explain the observed positive correlation between income per worker and capital-output ratios across nations.

Our model provides one rationalization why inadequate social infrastructure and distortionary policies which reduce \( A \) could reduce capital-output ratios. The steady-state capital-output ratio is obtained from (5) and (10) as,

\[
\frac{k}{y} = \left( \frac{1}{C_0} \right) \left( \frac{1}{A} \right) \left( \frac{1}{C_0} \right) \left( \frac{1}{A} \right) \frac{\phi}{(1 - \phi) (1 - \phi) \sigma (1 - \phi) A k^\phi}.
\]

The key here is the dependence of the effective discount rate, \( \phi \), on health. Technological differences lead to differences in steady-state capital-output ratios first because, for a given capital stock, a lower \( A \) reduces longevity through lower income and health investment, and secondly, because the steady-state capital stock is lower. Endogenous mortality thus induces a multiplier effect—differences in \( A \) are amplified through health investment into persistent differences in capital-output ratios besides reducing income levels directly. Rewriting steady-state output per worker as

\[
\bar{y} = A^{1/(1 - \phi)} (\bar{k}/\bar{y})^{\phi/(1 - \phi)},
\]

we note that the elasticity of \( \bar{y} \) with respect to \( A \) depends upon the direct productivity effect and the indirect effect operating through \( \bar{k}/\bar{y} \). An immediate implication is that, even when \( \phi < 1/2 \), small differences in technology result in relatively large differences in long-run output per worker and mortality.

A numerical example will give a better idea about the quantitative effect of this multiplier. Let \( \phi = 1/3 \), \( \phi(h) = h/(1 + h) \) and \( \tau = 0.05 \), and consider two countries \( i \).
and $j$ that differ in their $A$’s. Specifically, suppose that $A_i = 50, A_j = 25$. This two-fold productivity difference leads to a three-fold difference in output per worker through differences in mortality. If, instead productivity differed by a factor of three ($A_i = 75$), steady-state income would differ by a factor of 5.6, implying sizeable amplification effects due to life expectancy.\(^9\)

We collect these results in Proposition 1 below, the technical aspects of which are proved in Appendix A.

**Proposition 1.** (i) The dynamic system described by Eq. (10) possesses two steady states $\{0, \bar{k}\}$ when $\alpha < 1/2$, only the positive one being asymptotically stable. When $\alpha > 1/2$, three steady states $\{0, \bar{k}_1, \bar{k}_2\}$ exist, with $\bar{k}_2 > \bar{k}_1$; the two extreme steady states are asymptotically stable, the intermediate one is not.

(ii) Endogenous mortality induces a multiplier effect whereby differences in the technology parameter $A$ get amplified, through the saving rate, into persistent differences in steady-state capital-output ratio and relatively larger gaps in mortality and output per worker.

Whether Fig. 2 or 3 is a more plausible representation of high-mortality societies is worth debating. If we believe that persistent poverty and ill-health, as observed in sub-Saharan Africa, are the outcomes of a poverty trap, we also need to explain how developed societies such as Western Europe, North America and Japan, have been able to circumvent such a trap. If anything, substantial flows of medical technologies like antibiotics and vaccines, and knowledge of sanitation and personal hygiene, to developing nations over the last half-century should have made such traps less likely.

Such exogenous medical advances for poorer countries may be interpreted as parametric shifts in $\phi$, for example in $\beta$ if $\phi(h) = \beta h/(1 + h)$. An inspection of Eq. (10) together with Fig. 3 shows that such a shift would lower the threshold capital stock, $\bar{k}_1$, required to escape from the trap. One possibility is that $\beta$ depends on medical advances as also on their provision, and the latter has been lacking in poor countries. Indeed, the World Bank [28] provides extensive evidence how publicly provided health services are often ineffective in poor countries because they bypass rural areas and the urban poor who would benefit from them the most.

In conclusion, several clarifications are in order. While we have assumed a constant health tax, individuals in a low-income high-mortality environment would prefer a lower tax rate (see Appendix B). 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. Hence, besides being unable to invest enough in health, poorer economies also choose to underinvest in it.\(^{10}\)

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\(^9\)The associated mortality rates are 0.08 and 0.32 in this case, comparable to adult mortalities for the established market economies and sub-Saharan Africa in 1990 [28, Table A.5].

\(^{10}\)This may partly explain why low-income countries spend a smaller fraction of GDP on public health (see [28]). But richer countries also suffer from more expensive diseases like cancers, circulatory and respiratory ailments, which contribute to 75% of deaths [6].
Secondly, we have ignored altruism altogether. If households cared for their offspring, they could substitute for old-age consumption by leaving bequests. Even with high mortality rates, capital accumulation then might not suffer as much. Intergenerational transfers do not alter our basic results as long as individuals are not perfectly altruistic.

Finally, it is straightforward to extend our results to general homothetic preferences. The saving propensity \( \sigma_t \) then depends upon \( k_t \) (through health investment) and \( k_{t+1} \) (through \( \bar{R}_{t+1} \)). As long as youthful and old-age consumption are gross substitutes, \( \sigma_t \) is an increasing function of \( R_{t+1} \), or a decreasing function of \( k_{t+1} \). An increase in the current capital stock \( k_t \) allows greater health investment, but has two opposing effects on \( \sigma_t \). A higher \( k_t \) increases health expenditure, which raises \( \phi_t \). At the same time, it tends to lower \( \sigma_t \) since it reduces equilibrium return on savings, \( \bar{R}_{t+1} = R_{t+1}/\phi_t \). As long as the direct length-of-life effect dominates the indirect interest rate effect, \( \sigma_t \) is increasing in \( k_t \). Assuming this is true, the general equilibrium is characterized by a monotonically increasing phase map in \((k_t, k_{t+1})\) space and our results above go through.

3. Mortality risk and investment in education

Even when perfect annuities markets exist, mortality risks in certain kinds of investment, especially in "inalienable" human capital like education [13], may be undiversifiable. Using age-specific mortality rates and earnings profiles in developing countries, Meltzer [23] demonstrates that mortality declines can have quantitatively large effects on school enrollment. When human capital is the engine of growth, mortality rates magnify initial cross-country differences in income and health into persistent differences in living standards.

This idea can be formalized with a minor modification of the previous analysis. Consider a similar setup except individuals are now endowed with 1 unit of labor time in both periods of life. Denote by \( x_t \) the average stock of skills of the workforce at \( t \), 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 as well as skills inherited by generation-\( t \) youth from their parents.

An individual born at \( t \) can improve her productivity by investing a fraction, \( s_t \), of her time attending school when young. Her future productivity relates to this investment according to:

\[
x_{t+1} = x_t \mu(s_t),
\]

\( \mu \) is the productivity function, which depends on the fraction of time \( s_t \) spent attending school. The savings propensity \( \sigma = \phi R^{\sigma - 1}/(1 + \phi R^{\sigma - 1}) \) is increasing in \( \phi \).

\( \footnote{For instance, when the period utility function is CES, } \]

\( u(c) = c^{1-\sigma}/(1 - 1/\sigma) \), the savings propensity \( \sigma = \phi R^{\sigma - 1}/(1 + \phi R^{\sigma - 1}) \) is increasing in \( \phi \).
where \( m \) is an increasing and concave function satisfying \( m(0) = 1 \). Schooling is chosen to maximize lifetime income,

\[
(1 - \tau)(1 - s_t)w_t x_t + \frac{w_{t+1} x_{t+1}}{\bar{R}_{t+1}},
\]

\( \tau \) being the health tax on youthful wage income. The first-order condition for an interior optimum equates returns on physical and human capital,

\[
(1 - \tau)\frac{w_t}{[w_{t+1}/\bar{R}_{t+1}]} = \mu'(s_t).
\]

For a strictly concave \( m \), we can express optimal schooling as a function of prices:

\[
s^*_t = s\left(\frac{\phi_t w_{t+1}}{w_t R_{t+1}}\right),
\]

where \( s \) is increasing in its argument.

Eq. (12) 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: mortality decline promotes capital accumulation through the length-of-life effect, further raising rewards to education in the form of higher future wages.

Interestingly, endogenous mortality may introduce threshold effects of the type that Azariadis and Drazen [1] elucidate. There, private returns to education depend upon the social stock of human capital through an externality. Our human capital technology does not explicitly incorporate such an externality. But, when mortality is endogenously determined through public health investment, it alters private returns to education: one form of human capital injects an externality into another. Proposition 2 below outlines this scenario.

**Proposition 2.** Threshold effects may result for the human capital technology (11) under endogenous mortality. The growth rate responds gradually to human capital accumulation as a rising stock of skills enables greater health investment and mortality reductions. For a moderately concave \( m \), sharp increases in growth occur for moderate changes in human capital.

Fig. 4 illustrates such “take-off” for a small open economy facing a constant world interest factor \( \bar{R} \) when \( \mu(s) = 1 + s^\theta, \theta \in (0, 1) \). Growth of income per worker is an increasing function of the human capital stock, \( \mu(s^*_t) - 1 = [\delta \phi(\tau \bar{R} x_t)]^{\theta/(1-\theta)}, \delta \equiv \theta/[(1 - \tau)\bar{R}] \). It is initially slow and accelerates once human capital attains a critical mass, \( \bar{x} \). In the long-run, the economy converges to the unique steady-state growth rate of \( (\delta \beta)^{\theta/(1-\theta)} \).

Multiple balanced growth paths are also possible if we admit linear human capital technologies. Suppose \( Y_t = AK_t^\alpha N_t^{1-\alpha}, N \) being efficiency labor supply, so that
Assume the following technology for skill accumulation,
\[ \mu(s) = 1 + \theta s, \quad \theta > 0. \] (13)

With logarithmic preferences, optimal savings is
\[ z_t = \sigma_t [(1 - \tau)(1 - s_t)w_t x_t - \frac{w_{t+1} x_{t+1}}{R_{t+1}}], \] (14)
with \( \sigma \) denoting the saving propensity, as before.

Depending upon the initial stock of human capital, \( x_0 \), the economy exhibits two types of dynamics: one, where no schooling investment takes place, and another with schooling. Corner equilibria with no schooling occur when skill investment yields a return no greater than that obtainable with physical capital investment, that is, when
\[ \theta \leq (1 - \tau) \frac{w_t R_{t+1}}{\phi_t w_{t+1}}. \] (15)

In this case, the social stock of human capital remains constant at \( x_0 \). Efficiency labor supply is \( N_t = (1 + \phi_{t-1})x_0 \) and market clearing requires that \( N_{t+1} k_{t+1} = z_t \).

Using (14) and equilibrium prices, competitive equilibria may be characterized by
\[ k_{t+1} = (1 - \tau)(1 - \alpha) \left[ \frac{1}{1/\sigma(k_t)(1 - \sigma(k_t)) + (1 - \alpha)/\alpha} \right] A k_t^\alpha, \] (16)
where,
\[ \sigma(k) = \frac{\phi(\tau(1 - \alpha)x_0 A k^\alpha)}{1 + \phi(\tau(1 - \alpha)x_0 A k^\alpha)}. \]

As before, as long as \( \alpha < 1/2 \), the economy converges monotonically to a unique steady-state output per worker.\(^\text{12}\)

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\(^{12}\) In general equilibrium, the no-schooling restriction becomes \( k_{t+1} \leq \alpha(1 - \tau)A[k_t^\alpha / \phi(k_t)] \equiv H(k_t) \).

Define the right-hand side of (16) as \( G(k_t) \). Since \( H(k) > G(k) \) for all \( k > 0 \), an economy that starts in a no-schooling equilibrium never switches to one with schooling.
When initial conditions admit interior schooling equilibria, they satisfy a pair of difference equations in \((s_t, k_t)\):

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

Since \(\phi_t\) converges to \(\beta\) asymptotically, a unique positive steady state \((s^*, k^*)\) exists and is a saddle-point. Given \((x_0, k_0)\), sequences of \((s_t, k_t)\) converge monotonically to the steady-state growth rate \(\theta s^*\)\(^{13}\).

Even though two countries with differing initial mortalities may converge to the same balanced growth, the low-mortality society consistently enjoys higher levels of output per worker since it invests more intensively in skill-acquisition. A growth trap may also result. The no-schooling restriction (15) may be satisfied for a high-mortality society for initial levels of \((x_0, k_0)\). For the country with higher life expectancy (say, \(\beta\)), on the other hand, (15) may not hold for the same vector \((x_0, k_0)\). As a result, it would experience sustained improvement in living standards even as the high-mortality society stagnates.

### 4. Some empirical evidence

We finally turn to some empirics, looking for evidence on the mechanisms analyzed above. In particular, we are interested in seeing how well the data supports the conjecture that mortality is a determinant of educational investment and how well mortality explains stagnation, especially in the countries of sub-Saharan Africa.

Cross-country regression analyses that incorporate measures of health status into their list of explanatory variables, for instance Sala-i-Martin [26], find health indicators to be significant predictors of future growth. The regressions here are in the spirit of those studies but are motivated by the specific linkages suggested by our theory.

Our analysis is in terms of GDP per worker, rather than per capita, to control for demographic shifts. The time horizon considered is 20 years, covering 1970–1990. The data used are for 95 countries, though the actual number of countries in each regression differs depending upon data availability. Estimation is by ordinary least squares in all cases. Appendix C provides details about data sources.

Longevity was shown to have a level effect in Section 2. To test this, we first regress GDP per worker in 1990 (\(LRGDP90\)) on average population growth during 1970–90 (\(LPOP\)), average investment-to-GDP ratio during 1965–69 (\(LINVSH\)), and secondary enrollment rates in 1970 (\(ENROLS70\)). The secondary enrollment rate is taken as a proxy for initial education capital. The result is reported in column (1) of Table 1. As the human capital augmented neoclassical model suggests, the investment rate and initial stock of human capital both significantly increase per

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13See Proposition 2 in [1].
Table 1  
Testing for the length-of-life effect

<table>
<thead>
<tr>
<th>Dependent variable: ( \text{LRGDP90} )</th>
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<tr>
<td></td>
<td>(7.59)</td>
<td>(7.59)</td>
</tr>
</tbody>
</table>

\( R^2 \) | 0.71 | 0.83 |
\( \bar{R}^2 \) | 0.70 | 0.82 |
\( \text{OBS.} \) | 86 | 84 |

capita GDP, while population growth reduces it. Column (2) adds another explanatory variable, life expectancy in 1970 (\( \text{LIFEXP70} \)), that our theory suggests should have a significant effect on capital accumulation. The results are striking: all variables except for life expectancy cease to be statistically significant. In fact, in a regression of only life expectancy on the 1990 GDP per worker, longevity explains about 81% of the cross-country variation in income levels (not reported in Table 1).

Our theory also suggests that mortality 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 1970–1990. The first regression, column (1) in Table 2, looks at the impact of enrollment in 1970 (\( \text{ENROLS70} \)) on subsequent average growth (\( \text{GR7090} \)), controlling for initial income per worker (\( \text{LGDPEA70} \)). As per the conditional convergence hypothesis, the coefficient on initial income is again negative and highly significant. The initial stock of human capital positively, and significantly, affects future growth. The second regression, in column (2), excludes human capital, and looks at the forces of convergence versus initial life expectancy (\( \text{LIFE70} \)). The convergence prediction holds, while the effect of life expectancy is high and significant. Initial income and longevity explain 42% of cross-country growth variation.

Column (3) of Table 2 includes both initial human capital and life expectancy as explanatory variables. Observe how schooling ceases to be significant at the 5% level, while GDP per worker and life expectancy have the correct signs and continue to be statistically significant. Theoretical predictions are, in other words, consistent with the evidence.

Cross-country growth regressions often incorporate a dummy for sub-Saharan Africa. A negative coefficient on this dummy is usually interpreted as geographical factors at work. To see how much of that effect is region-specific and how much the
result of high mortality, we incorporate a dummy for sub-Saharan Africa (SSAFRICA). Also included is the average share of investment in GDP between 1965 and 1969 (INVSH). Columns (1) and (2) of Table 3 examine the result of this exercise with and without initial life expectancy. Inclusion of life expectancy 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 mortality rates there are the highest in the world. The coefficient on the investment-to-GDP ratio has incorrect sign, but is 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 3. The only significant variables are initial GDP per worker, initial life expectancy and the share of primary goods in total exports. 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 [19]. 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

---

Table 2
Mortality risk, education and growth rates

<table>
<thead>
<tr>
<th>Dependent variable: GR7090</th>
<th>(t-statistics in brackets)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
</tr>
<tr>
<td><strong>CONSTANT</strong></td>
<td>6.16/2.72</td>
</tr>
<tr>
<td><strong>LGDPEA70</strong></td>
<td>−0.73/−2.36</td>
</tr>
<tr>
<td><strong>ENROLS70</strong></td>
<td>4.45/4.44</td>
</tr>
<tr>
<td><strong>LIFE70</strong></td>
<td>10.29/7.90</td>
</tr>
</tbody>
</table>

$R^2$ 0.19 0.42 0.42
$R^2$ 0.17 0.41 0.41
OBS. 94 94 93

---

14The negative association between natural resources and economic growth was pointed out in [25].
infrastructure or other policy measures do not appear to be substantive factors contributing to economic growth once a mortality measure is included. As Table 2 shows, most of the variability in growth rates comes from initial income and differences in health capital. The evidence complements what we already know from other micro- and disease-specific studies on the role of health (see [27] for example). More rigorous empirical analysis is, however, necessary to ascertain the true impact of health on growth and development.

5. Conclusion

Widespread evidence on the strong correlation between poverty and ill-health, at both individual and aggregate levels, is often ascribed to the adverse effects of poverty. This paper has examined how better health, by improving longevity and reducing mortality risks, is conducive to growth and development.

The analysis points to health investment as a prerequisite for sustained 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 may also result in ‘development traps’. When technological efficiency differs across nations, small differences in productivity get amplified into large and persistent differences in capital-output ratios, output per worker and mortality rates. These results are broadly consistent with existing evidence and regression results indicate the growth effects of longevity to be significant and 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 to use cross-country evidence and more sophisticated econometric methods than have been used here. Since cross-country analyses suffer from a number of statistical problems, a more promising approach may be quantitative methods of the kind used in Kalemli-Ozcan et al.’s [14] 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.

Acknowledgments

I would like to thank an associate editor of this journal, Costas Azariadis, Richard Barrett, Roger Farmer, Gary Hansen, Amartya Lahiri, David Meltzer, Tomas Philipson, and seminar participants at UCLA, UCSB, Harris School of Public Policy and University of Oregon for useful comments. All remaining errors are mine.

Appendix A. Proof of Proposition 1(i)

We first establish the following lemma.

**Lemma A.1.** Define the right-hand side expression of (10) as \( J(k) \). It satisfies the following properties: (i) \( J(0) = 0 \), (ii) \( J'(k) \geq \forall k \geq 0 \), (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 \( \lim_{k \to \infty} \sigma = \beta/(1 + \beta) \). 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,

\[
J'(k) = C(\sigma k^\alpha + 2 \sigma k^{\alpha-1}),
\]
where, \( \sigma_k = \alpha \tau (1 - \alpha) A \phi'/(1 + \phi)^2 ] k^{x-1} \). Using \( \lim_{k \to 0} \phi'(\tau(1 - \alpha) A k^x) = \gamma \), we obtain

\[
\lim_{k \to 0} J'(k) = \alpha C \left[ \tau (1 - \alpha) \gamma A \lim_{k \to 0} k^{2x-1} + \tau A \gamma k^{2x-1} \right] = \infty \quad \text{iff} \quad x < 1/2.
\]

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

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

**Appendix B. Optimal choice of health tax**

Current public health expenditure benefits only the currently young, who also fund these investments out of wage income. Therefore young agents born at \( t \) choose \( \tau_t \) to maximize their expected lifetime utility. Using (1), this maximization problem becomes:

\[
\text{Max}_{\{z_t, \tau_t\}} \log[(1 - \tau_t)w_t - z_t] + \phi(\tau_tw_t)\log[\hat{R}_{t+1}z_t].
\]

The first-order condition for \( z_t \) gives:

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

while that for \( \tau_t \) equates the marginal utility cost to the (discounted) marginal utility gain from a greater chance of surviving:

\[
\frac{w_t}{c_t} = w_t \phi'(\tau_tw_t)\log[\hat{R}_{t+1}z_t]. \quad \text{(B.2)}
\]

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

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

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

Data used in Section 4 are obtained from several sources: (i) Log real GDP per capita in constant dollars ($LRGDP90$), the average ratio of real domestic investment to real GDP between 1965 and 1969 and its logarithm ($INVSH$, $LINVSH$) come from the Penn World 5.6. (ii) Total gross enrollment ratio for secondary education in 1970 ($ENROLS70$), the average black market premium between 1965 and 1969 ($BMP6590$), and terms of trade shock between 1965 and 1969 ($TOT6569$) are taken from Barro and Lee [2]. (iii) Natural logarithm of PPP-adjusted real GDP per economically active population in 1970 ($LGDPEA70$), the average annual growth in real GDP per economically active population over 1970–1989 ($GR7090$), and the share of primary product exports in GNP in 1970 ($SXP$) are from Sachs and Warner [25]. (iv) Data on the log average population growth rate for 1970–1990 ($LPOP$), life expectancy at birth in 1970 and its natural logarithm ($LIFEXP70$, $LIFE70$) are from The World Bank [29]. (v) Finally, data on log telephones per 1000 workers ($LTELPW$) are taken from Easterly and Levine [9].


References