LONGEVITY AND EDUCATION: A MACROECONOMIC PERSPECTIVE

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Abstract

This paper investigates the determinants of longevity at a macroeconomic level, emphasizing the important role played by education. To analyze the determinants of longevity, we build a model where households intentionally invest in health and education, and where education exerts external effects on longevity. Performing an empirical analysis using data across 71 countries, we find that society's tertiary education attainment rate is important for longevity, in addition to any role that basic education plays for life expectancy at the individual level. This finding uncovers a key externality of education, consistent with the theoretical hypothesis advanced in our macroeconomic model.

Keywords: Education, life expectancy, health, externalities, absorptive capacity, welfare.

JEL Classification: O40

1   Introduction

To the extent that income is not the sole determinant of health status across the world, one can view health as a separate component of welfare, other than income. In this case, the factors driving welfare growth might well be different than those relevant for economic growth, with important policy implications.[1] Thus, a central question in the debate on the determinants of international health outcomes asks whether these are a mere by-product of economic growth (see Pritchett and Summers, 1996) or whether 'exogenous' non-income sources are largely responsible, as argued by Preston (1975, 1980, 1996).

In line with the latter, Becker, Philipson and Soares (2005), Soares (2007a, 2007b), Cutler, Deaton and Lleras-Muney (2006), and Papageorgiou, Savvides, and Zachariadis (2007) argue that improvements in life expectancy have largely occurred independently of per capita income growth and are related to new medical technology and to the accumulation and diffusion of health knowledge. Soares (2007a, p.35) emphasizes the role of education pointing out that "[*t*]echnologies related to individual-level inputs used in the production of health seem to be subject to the effectiveness with which individuals can use these inputs" so that "more educated individuals have higher survival advantage in diseases for which medical progress has been important." Similarly, Cutler, Deaton and Lleras-Muney (2006, p. 115) write that "the differential use of health knowledge and technology [*is*] almost certainly [*an*] important part of the explanation" as to why "[*t*]here is most likely a direct positive effect of education on health." As long as there exist cross-country differences in educational attainment rates, we would expect differences in international health outcomes.

This paper examines the determinants of cross-country health outcomes with emphasis on the mechanisms through which education impacts upon longevity. We attempt to understand the role played by different factors in determining life expectancy. These include private purchases of medical inputs and publicly provided health inputs affecting the environment in which households live and make decisions. The efficiency of these inputs in affecting longevity depends on how well health-related knowledge is exploited in society. For the individual, own or parental education is crucial in facilitating access to and understanding of health-related information. The availability of health-related knowledge in the first place depends on the overall level of education in the country.

Education can therefore play two direct roles in the determination of health outcomes. First, the level of education within the household enhances the longevity of its members. For example, education affects crucial factors such as understanding treatments, assessing risks incurred with

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[1]Conversely, if growth in real income per capita alone determined improvements in health status, then there would be no scope in studying health status and welfare growth as concepts distinct from economic growth.

hazardous behavior, or feeding children healthily. Second, the aggregate level of education in the economy improves the quality of health services offered within a country. One reason for this would be that the average level of education improves a country's absorptive capacity for health-related technology and ideas. Another related reason is that we would expect physicians to be more likely to implement new treatments to the general population in countries where the average patient is more educated.[2]

The two direct effects of education play conceptually different roles. The first one operates as a rival input benefiting household members. We expect this role of education in enhancing a household's longevity to exhibit diminishing returns[3] so that primary education attainment levels should suffice to capture it. The second effect depends on the ability and readiness of the health sector to take advantage of best practices. This is a high-tech sector experiencing fast technological progress. Furthermore, efficient use of new medical technologies requires understanding of scientific findings. The sophisticated character of knowledge transmission and use in this sector suggests higher education constitutes its crucial determinant. That higher education attainment constitutes a small percentage of the population during the period under study, suggests that the presence of a large effect of higher education could not be explained with a mechanism that operates just within the household[4], but should largely be due to externalities. Controlling for basic education, any additional effects from higher education would then be consistent with this second external role of education.

We build a theoretical model to analyze the relationship between life expectancy, educational decisions, private and public investment in health, and income. In our model, individuals intentionally spend resources to enhance their life expectancy similar to Chakraborty and Das (2005). The effective discount rate is therefore endogenous: by investing in health the individual chooses to become less impatient. Performing comparative statics in the case of an interior solution to the individual problem, we find that private health investment -hence life expectancy- and education are positively correlated. A bidirectional causal relationship between education and longevity ex-

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[2]Cutler and Lleras-Muney (2006) argue in page 16 that "[*t*]he more educated ... appear to make use of new health related information first" and "are more likely to trust science", so that we would expect health specialists to be more willing to learn about, explain, and implement new treatments to educated patients more receptive to new medical knowledge. Physicians practicing in regions with greater education levels for the average patient would then be more likely to implement new treatments to the general population of the region.

[3]This is consistent with evidence provided in Haines and Avery (1982) and Merrick (1985) using individual-level data from Costa Rica and Brazil respectively. These papers find female education to exhibit diminishing returns in enhancing family health output. In particular, Haines and Avery (1982, p. 43) find that "*t*he results indicate a much greater elasticity of response of child mortality to an additional year of education for women with less education than with more education (11 percent against 2 percent)", and Merrick (1985, p.6) finds that although mortality ratios fall with education attainment, "*t*he most striking difference in mortality ratios, however, is the contrast between mothers with no formal education and other groups." In fact, Merrick (1985, p.10) suggests the role of education "may be limited to such basic steps as boiling contaminated water."

[4]Unless one additional year of higher education attainment added an unrealistically large number of years to an individual's life expectancy.

ists at the individual level: while improved life expectancy raises the return on education inducing more of it, greater investment in education increases expected future labor earnings and hence incentives to keep oneself in good health.

We then study the stationary symmetric equilibrium introducing two *direct*[5] roles played by education in determining longevity. It is assumed that longevity is an increasing function of basic education supplied by parents to children and of the average level of human capital in society (i.e. higher education). For the first feature, the distinction of two forms of education is meaningful to highlight the possibility of a differential role played by education in determining longevity, as argued above and as advanced in our empirical analysis. Soares (2005) and Cervellati and Sunde (2007) also consider distinct roles of basic and higher education. As far as the second feature is concerned, by allowing for an external effect of the average level of education on the efficiency of the health sector we magnify the feedback from education to longevity at the *macroeconomic* level (see Blackburn and Cipriani, 2002).

To the best of our knowledge this is the first model that brings together private intentional investment in health and externalities from education on longevity in a unified framework.[6] This original model allows us to identify four links between education and longevity, with three of them characterized by causality running from education to life expectancy. The presence of the aggregate externality could potentially give rise to multiple equilibria (see Van Zon and Muysken, 2001). In order to predict correlations between observable variables under our hypothesis on the causation from education on longevity, we study the case with a unique interior equilibrium which prevails for relatively small external effects of education on life expectancy. We find that higher and basic education, private health investment and life expectancy are positively related, increasing with public health investment, per capita income and the efficiency of the educational system.

Next, we use data from 71 countries to test the empirical plausibility of the supposed direct roles played by education in determining longevity. Using initial period averages to explain end-period life expectancy and utilizing appropriate instrumental variables estimates, allows us to alleviate the endogeneity problem concerning longevity and education. To further address problems

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[5] Compared to an *indirect* effect of education on longevity running through permanent income, identified in the case of the individual problem.

[6] Assuming existence of this type of externality is a common method to endogenize longevity: Life expectancy is assumed to be positively affected by the average or total stock of human capital in the economy (Blackburn and Cipriani, 2002, Boucekkine et al., 2002, Lagerlöf, 2003, Chakraborty, 2004, Cervellati and Sunde, 2005), by per capita national income (Kalemli-Ozcan, 2002), or by the public provision of private health services (van Zon and Muysken, 2001, Blackburn and Cipriani, 2002, Chakraborty, 2004). Only a handful of papers consider intentional investment in health services to improve life expectancy: Blackburn and Cipriani (1998) who assume that the efficiency of health investment is decreasing in the stock of capital, Sanso and Aísa (2006) who consider age-dependant efficiency of health services, Van Zon and Muysken (2001) who restrict the analysis to the steady state solution of the social planner problem, and Galor and Moav (2005) where there is no educational choice. None of these papers considers the externality and intentional private health investment simultaneously, as we do here.

with capturing the direction of causality, we consider beginning of period changes in explanatory variables to explain end of period changes in life expectancy. Controlling for the effect of income, health spending and other health inputs, we find that tertiary education is no less important than primary education for longevity. This suggests that the aggregate externality role of education is at least as important as the role of basic education enhancing health outcomes at the household level. Evidence of a form of increasing returns in education is particularly interesting, as previous work has established that tertiary education has little explanatory power for per capita income growth while primary education is its single most important determinant. Here, tertiary education is shown to be important for another component of welfare.

The next section presents the model and theoretical results. Data are described and discussed in section 3. Section 4 describes the empirical analysis and results, while section 5 concludes.

2 A model of education and health investment

In this section, we present a model where education and health investment are chosen by individuals, and where education can exert external effects on the productivity of health investment. This framework is used to analyze the relationship between educational choices, purchases of health-related services, provision of public health services, income, and life expectancy. First, we set up the model with emphasis on the individual problem. We establish sufficient conditions for the existence of a unique interior solution to the individual problem. This solution is used to predict how changes in parameters induce adjustments in higher and basic education, as well as in health investment and thus in longevity. Next, we turn to the stationary symmetric equilibrium with externalities from education on life expectancy. We derive sufficient conditions for the existence of a unique equilibrium and use the latter to predict comovements of variables of interest.

The model shows that education and longevity are strongly and positively related to each other. Their relationship is mutually reinforcing and hinges on causation running in two directions: on the one hand, improved life expectancy increases the return on education, inducing more of it; on the other hand, more education implies longer life expectancy. In the model, the latter effect runs through three channels. First, more educated individuals expect higher future income and thus have a greater economic return on health, resulting in more health investment and in longer life expectancy. Second, wealthier individuals endow their children with more basic education, enhancing the efficiency of the child's health investment. Third, improved educational attainment in the labor force directly increases the efficiency of health investment, fostering it and leading to further improvements in life expectancy.

2.1 The individual problem

Suppose that individuals can live for two periods. Everyone lives during the first period but survival to the second period is dictated by probability $\pi \in (0,1)$. The survival probability is an increasing function of health-related individually purchased inputs, m. We consider an isoelastic specification

$$\pi = \min\{zm^{\mu}, \bar{\pi}\} \tag{1}$$

with $\bar{\pi} \in (0,1)$, $z > 0$ and $m \geq 0$. Our analysis focuses on the interesting case when $\pi < \bar{\pi}$. We consider that the following is satisfied

Parametric assumption 1 $\mu \in (0,1)$, *perceived returns on intentional investment in health are decreasing.*

Remark 1 *The effectiveness, z, of the agent's health investment, m, in enhancing her life expectancy, π, is perceived as being exogenously given. The value of z will be considered as being endogenous in the next subsection, where it will be affected by educational choices.*

We consider the problem of an agent in her first period of life at date t. At the beginning of the period, the individual is endowed with basic education, b_t, chosen by her parents. The agent chooses her post-basic education level, h_t (which hereafter we refer to simply as education). She chooses how to share her remaining income between consumption, c_{1t}, and purchases of health-related inputs, m_t. In our setting, fertility is exogenous and we assume that each agent has one child in the second period.[7] Conditional upon surviving to the second period, the agent chooses how to share her income between consumption, c_{2t+1}, and the purchase of her child's basic education, b_{t+1}. The agent's objective is to maximize the expected present value of the utility accruing from consumption and from providing basic education to the child, subject to two period budget constraints and survival technology (1):

$$\max_{m_t, h_t, b_{t+1}} \frac{1}{1-\sigma} c_{1,t}^{1-\sigma} + \rho \pi_t \frac{1}{1-\sigma} v_{t+1}^{1-\sigma}$$

$$v_{t+1} \equiv c_{2t+1}^{\theta} b_{t+1}^{1-\theta} \tag{2}$$

$$w_t (1 - kh_t) = c_{1t} + pm_t \tag{3}$$

$$w_{t+1} (1 + h_t^{\eta}) = c_{2,t+1} + \kappa b_{t+1} \tag{4}$$

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[7] We abstract entirely from fertility choices and population dynamics. Since each adult has one child, the population decreases at rate $1 - \pi_t$. We could assume instead an exogenous fertility rate equal to $1 + n_t = 1/\pi_t$ in order to hold population constant. This alternative assumption would make the analysis more cumbersome without carrying along any additional insight. In fact, under this assumption the cost of providing basic education per child is increasing in fertility and ultimately decreasing in longevity, a feature which reinforces our results.

All parameters and variables are non-negative and $\rho, \theta, \eta \in (0, 1)$.

According to the agent's period budget constraints (3)-(4) education, h_t, is costly in terms of forgone first period labor income, through the effort-cost parameter k. First period income is allocated to the numeraire consumption good and to purchases of medical inputs at relative price p. Second period labor income is an increasing and concave function of education. It is spent on consumption and on child's basic education at relative price κ.

Remark 2 *Second period sub-utility is discounted according to two factors: the subjective discount factor, ρ, and the endogenous survival probability. Since the agent takes into account the impact of her consumption of health-related inputs, m_t, on her life expectancy according to (1) she faces endogenous discounting.*

Remark 3 *We assume that the agent values her child's basic education in the same way as she values consumption. Second period sub-utility is a function of the "consumption bundle" of two differentiated goods: c_{2t+1} and b_{t+1}.*[8]

Remark 4 *From the individual point of view basic education, b_{t+1}, is not an investment good since it does not affect the exogenous component of the child's future income, w_{t+2}, or his effort-cost of education, k.*[9]

Remark 5 *Child's basic education is valued independently of its impact on child's income, longevity or utility. The alternative specification of assuming parents care about the child's utility would give rise to a more complex recursive problem, where parental choice of basic education takes into account its influence on child's behavior through enhanced life expectancy.*[10]

Remark 6 *We assume b_t is entirely determined by parents at $t - 1$. This is a reasonable and empirically plausible assumption. Basic education creates the intergenerational link analogous to bequests. Later on, we allow for basic education to affect the efficiency of health investment. Nevertheless, own basic education will remain out of the individual's choice set.*[11]

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[8] This approach is equivalent to the one of Galor and Weil (2000), who define parents' preferences over a bundle of two goods: the consumption good and potential aggregate income of offsprings. The latter is the product of children quantity and their per capita human capital, itself an increasing function of education supplied by parents.

[9] Only Soares(2005) and Cervellati and Sunde (2007) consider basic and higher education separately. In these papers, basic education is provided by parents as in ours, but it is productive because it reduces the effort-cost of higher education. Nevertheless, in these papers individually optimal behavior prescribes a choice of higher education independent of the endowment of basic education (Soares, 2005), or of a fixed and exogenous level of basic education (Cervellati and Sunde, 2007). These results are due to assumed linearities in technology and preferences (see section E in Soares, 2005). Because we choose to work with non linear technologies and preferences, we prefer to abstract from the productive role of basic education in facilitating the acquisition of higher education.

[10] It could be argued that this alternative approach reflects a paternalistic approach to altruism, whereas our approach instead rests on a liberal view of altruism according to which the parent perceives a moral duty to endow her child with the basic means to freely make his own choices.

[11] If we adopted the assumption by which each individual internalized the effect of her educational choice on

Remark 7 *Education is the only form of investment and affects permanent income.*[12] *Its marginal rate of return in terms of current potential consumption is $\eta w_{t+1} h_t^{\eta}/(w_t k h_t)$, i.e., an increasing function of the growth rate of wages and the educational sector's efficiency measured by η and $1/k$.*

We drop time subscripts where this does not lead to confusion, use (1)-(2) and substitute for c_1 and c_2 using (3) and (4), to write the problem as follows

$$\max_{h,m,b} \frac{1}{1-\sigma} \left[w_t (1-kh) - pm \right]^{1-\sigma} + \rho z m^{\mu} \frac{1}{1-\sigma} \left\{ \left[w_{t+1}(1+h^{\eta}) - \kappa b \right]^{\theta} b^{1-\theta} \right\}^{1-\sigma}$$

An interior solution to this problem should satisfy the following first order conditions with respect to h, m and b respectively

$$w_t k c_1^{-\sigma} = \rho z m^{\mu} w_{t+1} \eta h^{\eta-1} \theta \left(\frac{b}{c_2} \right)^{1-\theta} v^{-\sigma} \tag{5}$$

$$p c_1^{-\sigma} = \rho \mu z m^{\mu-1} \frac{1}{1-\sigma} \left\{ \left[w_{t+1}(1+h^{\eta}) - \kappa b \right]^{\theta} b^{1-\theta} \right\}^{1-\sigma} \tag{6}$$

$$c_2 = \frac{\theta}{1-\theta} \kappa b \tag{7}$$

System (3)-(7) solves for the five endogenous variables c_1, c_2, m, h and b. We adopt the following

Parametric assumption 2 $\sigma \in (0,1)$, *substitution effects dominate income effects.*

It emerges clearly from (6) that this assumption is necessary for existence of an interior solution for m, given that the marginal and absolute values of utility have the same sign only in this case.[13]

From the second period budget constraint (4) we see that the rule dictated by (7) consists in spending constant shares of income on each differentiated good $c_2 = \theta/[w_{t+1}(1+h^{\eta})]$ and $\kappa b = (1-\theta)/[w_{t+1}(1+h^{\eta})]$. Using (7) in (2) we find that

$$v = \left(\frac{\theta}{1-\theta} \right)^{\theta} \kappa^{\theta} b \tag{8}$$

Moreover, substituting for c_2 in (4) from (7) we obtain[14]

$$b = \frac{1-\theta}{\kappa} w_{t+1} (1+h^{\eta}) \tag{9}$$

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own survival probability, the problem would become non concave in general. In fact, the feature of endogenous discounting reinforces complementarity between health related investment and education. Our assumption allows for this type of feedbacks while ensuring existence of a solution to the individual problem and of an equilibrium solution (see the role of assumption 4 in the proof for existence of a unique equilibrium).

[12] Previous versions of this paper included savings. The main features of the results are not affected by the introduction of savings. However, results are more often ambiguous in that case due to wealth effects arising from redistribution of savings from non-surviving individuals.

[13] This restrictive assumption is also necessary in Chakraborty and Das (2005).

[14] Substituting b from (9) in (8) and the result in the objective function we obtain $u(c_{1t}) + \rho\pi(v_{t+1}) = [w_t - pm - w_t kh]^{1-\sigma}/(1-\sigma) + \rho z m^{\mu}[\theta^{\theta}(1-\theta)^{1-\theta} w_{t+1}(1+h^{\eta})]^{1-\sigma}/(1-\sigma)$, which is concave in h.

Taking (7)-(8) into account we rearrange (5) and (6) to get

$$w_t k c_1^{-\sigma} = \rho z m^{\mu} w_{t+1} \frac{\eta h^{\eta-1}}{\kappa} a (1-\theta) b^{-\sigma} \tag{10}$$

$$p c_1^{-\sigma} = \rho \frac{\mu}{1-\sigma} a z m^{\mu-1} b^{1-\sigma} \tag{11}$$

where we have defined $a \equiv \theta^{\theta(1-\sigma)} (1-\theta)^{-\theta(1-\sigma)} \kappa^{\theta(1-\sigma)}$. Combining (10) and (11) to eliminate c_1, then substituting for b using (9) we can write

$$m = \frac{\mu}{1-\sigma} \frac{k}{\eta p} w_t (1+h^{\eta}) h^{1-\eta} \tag{12}$$

To write c_1 as a function of h we substitute for b and m using (9) and (12) into (11) and rearrange to get

$$c_1 = \left[\frac{1}{\rho a z} \left(\frac{1-\sigma}{\mu} p \right)^{\mu} \left(\frac{k w_t}{\eta} \right)^{1-\mu} \left(\frac{1-\theta}{\kappa} w_{t+1} \right)^{-(1-\sigma)} h^{(1-\eta)(1-\mu)} (1+h^{\eta})^{\sigma-\mu} \right]^{\frac{1}{\sigma}} \tag{13}$$

Finally using (12) and (13) into the first period budget constraint (3) we obtain the equation

$$\begin{aligned} LSH(h) &\equiv \left[\frac{1}{\rho a z} \left(\frac{1-\sigma}{\mu} p \right)^{\mu} \left(\frac{k w_t}{\eta} \right)^{1-\mu} \left(\frac{1-\theta}{\kappa} w_{t+1} \right)^{-(1-\sigma)} h^{(1-\eta)(1-\mu)} (1+h^{\eta})^{\sigma-\mu} \right]^{\frac{1}{\sigma}} \\ &+ \frac{\mu}{1-\sigma} \frac{k}{\eta} w_t (1+h^{\eta}) h^{1-\eta} = w_t (1-kh) \equiv RHS(h) \end{aligned} \tag{14}$$

We adopt the following

Parametric assumption 3 $\sigma \geq \mu$, *first-period consumption is monotonically increasing in education* $(\partial c_1/\partial h > 0)$.

This additional condition is sufficient to obtain the results gathered in the following proposition.

Proposition 1 *Under assumptions 1-3 there exists a unique interior solution to the individual problem, given by the level of education h satisfying (14) and basic education, health investment, life expectancy and consumption as given by (9), (12), (1), (13) and (7) respectively.*

Changes in parameters affect individual behavior, hence endogenous variables, according to the signs reported in Table 1.

Proof. Assumption 3, together with previous assumptions, implies that the LHS of eq. (14) is increasing (up from zero) and concave, while the RHS is linear in h and decreasing from w_t down to zero for $h = 1/k$. The two sides of equation (14) cross once and only once (see figure 1).[15] For

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[15]We have $\partial c_1/\partial h > 0$ iff $h^{\eta}/(1+h^{\eta}) < \frac{1-\mu}{\mu-\sigma} \frac{1-\eta}{\eta}$ or equivalently $\sigma > \mu - (1-\mu) \frac{1-\eta}{\eta} (1+h^{\eta})/h^{\eta}$. The right-hand-side of this last inequality is increasing in h but lower than $\mu - (1-\mu) \frac{1-\eta}{\eta} (1+k^{\eta})$ for $h \leq 1/k$. Hence, another

Table 1: Comparative statics for individual behavior[‡]

	higher education	health investment	basic education	first period consumption	second period consumption	life expectancy
	h	m	b	c_1	c_2	π
efficiency of health investment, z	+	+	+	-	+	+
cost of health services, p	-	-	-	+	-	-
effort-cost of education, k	-	$-^\dagger$	-	+	-	$-^\dagger$
cost of basic education, κ	-	-	-	+	-	-

‡ : Bold signs denote reinforced effects where more than one variable act in the same direction.

† : These signs are ambiguous. As shown in appendix A.1 a sufficient condition for them to be negative is

i) $\quad \sigma > 1 - \mu$ and

ii) $\quad \left(\frac{1}{\rho a z} \left(\frac{p}{\mu} \right)^\mu \eta^{-(1-\mu)} \sigma^{-\sigma} \left(\frac{1-\theta}{\kappa} w_{t+1} \right)^{-(1-\sigma)} (1-\sigma)^{\sigma+\mu} w_t^{-(\sigma+\mu-1)} \right)^{\frac{1}{\sigma}} \left(1 + k^{-\eta} \right)^{\frac{\sigma-\mu}{\sigma}} > k^{\frac{\eta}{\sigma}(\sigma+\mu-1)}$

proof of the comparative static exercises reported in Table 1 see appendix A.1. ■

Remark 8 *From Table 1 it is apparent that we should observe positive correlations across individuals (or countries) between higher education, basic education, private health-related investment and life expectancy. This is at odds with van Zon and Muysken's result of a negative correlation between health investment and education (van Zon and Muysken, 2001, p.180).*

Remark 9 *The positive correlation between h and π is obtained without any external effect of education on longevity. There are two forces at work. First, higher longevity improves the expected payoff to education. In this case, causality runs from π to h. This is common in the literature.[16] Second, more education increases permanent income allowing the agent to invest more in health. This channel underscores causality from h to π even in this context of individual choice without externalities at work. This is an original feature of our model.*

2.2 Externalities at the stationary symmetric equilibrium

We now introduce educational externalities on health status by assuming that education affects the efficiency of health investment, i.e., parameter z. From the individual point of view, private health-

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sufficient assumption for $\partial c_1/\partial h > 0$ is $\sigma \geq \mu - (1-\mu)\frac{1-\eta}{\eta}(1+k^\eta)$ which is less restrictive that assumption 3.

[16] This same result is obtained in a large body of literature analyzing the effect of exogenous mortality reductions on economic performance. See Ehrlich and Lui (1991) where altruistic parents educate their children, de la Croix and Licandro (1999) and Kalemli-Ozcan, Ryder and Weil (2000) where increased longevity raises educational investment and reduces human capital depreciation, Blackburn and Cipriani (2002) where lower mortality pushes parents to have less children later in life and educate them more, as well as Boucekkine et al. (2002) and Chakraborty (2004).
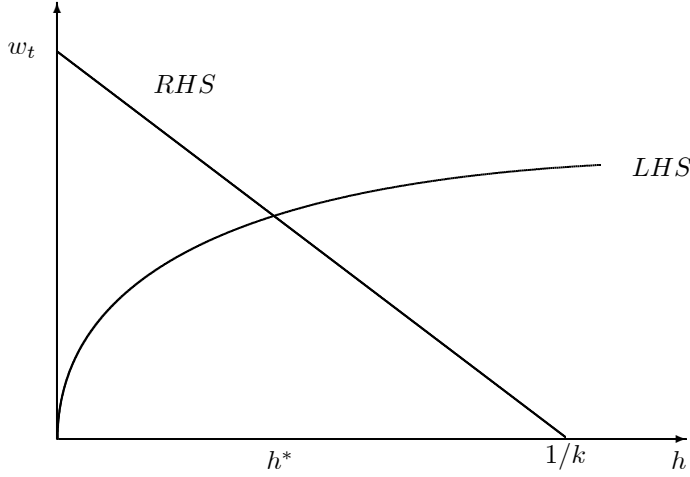
Figure 1: The equilibrium level of education: a representation of condition (14)

related investment is the more productive the greater are own basic education, public health-related services, and the average level of education in the economy.

Own basic education, b_t, enhances the individual's ability to take advantage of health services. Supply of public health services is considered exogenous and denoted by s_t. It can be interpreted as a pure public good, affecting for instance the rate at which households are subject to diseases. The average post-basic education level in the generation, $\bar{h}_t$, acts as a pure externality because it improves the quality of the health service sector by, for instance, facilitating the use and diffusion of best practices.

Using a Cobb-Douglas specification and recalling (1) we can write

$$z_t \equiv \zeta s_t^\delta \bar{h}_t^\alpha b_t^\beta \quad \Rightarrow \quad \pi_t = \zeta s_t^\delta \bar{h}_t^\alpha b_t^\beta m_t^\mu \tag{15}$$

where $\zeta > 0$ is a scale parameter and δ, α, $\beta \in (0,1)$.

By definition, at the stationary symmetric equilibrium we have that $\forall t$[17]

$$h_t = \bar{h}_t \ , \ b_t = b_{t+1} \ \text{and} \ w_t = w_{t+1}$$

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[17]From equation (14) it appears that in this model constant growth in wages can be compatible with constant educational investment only under specific assumptions concerning the dynamics of cost parameter, p, κ and k. To the extent that these costs are treated as exogenous it doesn't seem much rewarding to push the analysis in this direction to consider income growth.

Use these stationarity conditions and substitute for z in (13), then (9) to substitute for b to obtain

$$c_1 = \left[\frac{s^{-\delta}}{\rho a \zeta}\left(\frac{1-\sigma}{\mu}p\right)^\mu \left(\frac{kw}{\eta}\right)^{1-\mu}\left(\frac{1-\theta}{\kappa}w\right)^{-(1-\sigma)-\beta} h^{(1-\eta)(1-\mu)-\alpha}(1+h^\eta)^{\sigma-\mu-\beta}\right]^{\frac{1}{\sigma}} \quad (16)$$

To ensure that c_1 is increasing in h we make the additional

Parametric assumption 4 $\alpha \le (1-\eta)(1-\mu)$ *and* $\beta \le \sigma - \mu$, *the external effects of education on the efficiency of private health investment are small.*

Using expression (16) to substitute for c_1 in the first period budget constraint (3) along with (12) for m, we have the equation defining the equilibrium level of h:

$$\widetilde{LHS}(h) \equiv \left[\frac{s^{-\delta}}{\rho a \zeta}\left(\frac{1-\sigma}{\mu}p\right)^\mu \left(\frac{k}{\eta}\right)^{1-\mu}\left(\frac{1-\theta}{\kappa}\right)^{-(1-\sigma)-\beta} w^{\sigma-\mu-\beta}h^{(1-\eta)(1-\mu)-\alpha}(1+h^\eta)^{\sigma-\mu-\beta}\right]^{\frac{1}{\sigma}}$$

$$+ \frac{\mu}{1-\sigma}\frac{k}{\eta}w(1+h^\eta)h^{1-\eta} = w(1-kh) \equiv RHS(h) \quad (17)$$

which coincides with (14) for $\alpha = \beta = 0$ and $\zeta s^\delta = z$.

Finally using (9) and (12) to substitute for b and m in (15), we can write life expectancy at the symmetric stationary equilibrium as a function of the education level

$$\pi = \zeta\left(\frac{1-\gamma}{\kappa}\right)^\beta\left(\frac{\mu}{1-\sigma}\frac{k}{\eta p}\right)^\mu s^\delta[w(1+h^\eta)]^{\mu+\beta} h^{\mu(1-\eta)+\alpha} \quad (18)$$

We are now ready to state the following results:

Proposition 2 *In the presence of moderate externalities from education on life expectancy, i.e. under assumptions 1-4, there exists a unique equilibrium characterized by an interior solution $h \in (0, 1/k)$. The stronger the externalities of education on life expectancy are, i.e., the larger α and β, the greater are higher education, basic education, health related investment and life expectancy at equilibrium. These are positively correlated and react to changes in parameters as reported in Table 2.*

Proof. Under assumption 4 the $\widetilde{LHS}$ in (17) is an increasing function of h. *Mutatis mutandis* the same proof as the one of proposition 1 applies in this case.

To prove the second part of the proposition, we compare the solution of eq. (14) to that of eq. (17). Notice that the $\widetilde{LHS}$ in (17) is flatter than the LHS in (14) since $\partial c_1/\partial h$ is smaller (the exponent of h in the first term of the $\widetilde{LHS}$ is positive under assumption 4 but smaller than the one in the first term of the LHS). Since in both cases the left-hand-side starts at zero, we have in terms of figure 1 that $\widetilde{LHS}$ lies everywhere below the LHS schedule. Given that the right-hand-sides of

Table 2: Comparative statics with externalities[‡]

	higher education h	health investment m	basic education b	life expectancy π
public health investment, s	+	+	+	+
cost of health services, p	-	**-**	-	**-**
effort-cost of education, k	-	$-$[†]	-	$-$[†]
cost of basic education, κ	-	**-**	**-**	-
exogenous comp. of income, w	+	+	+	+

[‡] : Bold signs denote reinforced effects where more than one variable act in the same direction.
[†] : These signs are ambiguous in general. As shown in appendix A.2 a sufficient condition for them to be negative is

$$iii) \quad \sigma > 1 - \mu - \alpha \text{ and}$$

$$iv) \quad \left[\frac{s^{-\delta}}{\rho a \zeta} \left(\frac{1-\sigma}{\mu} p \right)^{\mu} \left(\frac{kw}{\eta} \right)^{1-\mu} \left(\frac{1-\theta}{\kappa} w \right)^{-(1-\sigma)-\beta} \right]^{\frac{1}{\sigma}} \left(1 + k^{-\eta} \right)^{\frac{1}{\sigma}[\sigma-\mu-\beta]}$$

$$\left[\frac{\alpha}{\eta} + \left(1 - \sigma + \beta + \frac{\alpha}{\eta} \right) k^{-\eta} \right] > k^{\frac{\eta}{\sigma}(\sigma+\mu-1)}$$

equations (14) and (17) coincide, the value of h solving (17) is greater than h solving (14). Finally, b and m are increasing functions of h, while π is increasing in all of these three variables.

For the proof of the comparative static exercises reported in Table 2 see appendix A.2. ∎

Remark 10 *Externalities make the link between h and π stronger. In the data, the positive correlation between education and longevity should be stronger in the presence of the externalities (i.e. $\alpha, \beta > 0$) than in the case without externalities (i.e. the solution to the individual problem).*

Remark 11 *Longevity is increasing in public health inputs s and in the exogenous component of income, w.[18] Improved public health services have a direct positive effect on π and an indirect effect running through increased h (see eq. 19 in appendix A.2). Higher w has a similar indirect effect through increased h, and two direct effects on π (see eq. 20) due to greater purchases of private health-related inputs, m, and higher levels of basic education, b, both driven by a pure income effect.*

Remark 12 *Our theory predicts that causation between longevity and higher education runs in both directions (i) one from greater π to higher h (as suggested by the result in the previous subsection) and (ii) one from higher h to greater π in the presence of externalities. Inspection of*

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[18]From eq. (18), life expectancy is also increasing in second period income $w(1+h^{\eta})$, which however is endogenous.

equilibrium longevity (18) highlights that the second causal link hinges on the strength of externalities through the role of basic education, β, and average higher education, α, in fostering health, and on the elasticity of longevity with respect to private health investment, μ. The latter measures the importance of the second link discussed in Remark 9, according to which more education raises permanent income fostering health investment and longevity (role of μ). Similarly, since more education implies higher permanent income, it implies greater parental investment in child's basic education which improves the efficiency of the child's health investment, resulting again in enhanced longevity (role of β). Finally, higher investment in education improves the efficiency of health investment directly through the externality, leading to longer life expectancy (role of α).

The empirical relevance of our hypothesis according to which education affects life expectancy is the subject matter of the rest of the paper.

3   Data description

In this section, we describe the data set we have assembled to test our main hypotheses and take a first look at the relationship of longevity with each of the candidate health input variables. The focus of our study, a country's longevity, is measured by the average life expectancy at birth. The *World Development Indicators* (WDI) 2005 database provides data on life expectancy at birth, physicians per thousand people, real health expenditure per person[19], sanitation (defined as the percentage of the population with access to improved sanitation facilities), and GDP per capita in PPP dollars. We obtained primary and higher education attainment rates from the Barro and Lee (2001) dataset. We also use a measure of the incidence of AIDS (defined as number of cases per thousand persons) from Papageorgiou and Stoytcheva (2006) in an effort to control for the adverse effects of the AIDS epidemic on health status.

We were able to put together the above series for 71 countries, shown in Table 5 in the appendix. The great majority of these series are not available frequently over time and in some cases the data are exceedingly sparse in the time dimension. Because the cross-sectional dimension of the dataset is more complete and, more importantly, because of the inherent long-run nature of the relation under study, we opted for exploring empirically the cross-sectional dimension of our dataset. That

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[19]This is total health expenditure per capita in constant dollars. Total health expenditure is the sum of public and private expenditures as a ratio of the population and covers provision of preventive and curative health services, family planning, nutrition activities, and health-designated emergency aid. It excludes provision of water and sanitation. Private health expenditure includes direct household out-of-pocket spending, private insurance, charitable donations and direct service payments by private corporations. Public health expenditure consists of recurrent and capital spending from central and local government budgets, external borrowing and grants (including donations from international agencies and nongovernmental organizations), and social or compulsory health insurance funds. Data are in current U.S. dollars and converted to contant dollars by deflating using the US CPI.

is, we average the available data over the period 1995 to 2004 for life expectancy, and 1961 to 1995 for the explanatory variables subject to availability.

A first look at the data

Our model suggests that a country's life expectancy at birth is positively correlated to private and public health investment (measured by health spending, physicians availability and sanitation) and to education through two distinct channels (measured by primary and higher education).

In the dataset, life expectancy has a correlation of 84 percent with higher education attainment rates and 34 percent with basic education attainment rates. Physicians are also strongly correlated with life expectancy at 87 percent. Moreover, sanitation and health expenditures have correlations with life expectancy of 70 and 77 percent respectively, while aids prevalence has an unconditional correlation with life expectancy equal to minus 22 percent. All these correlations are statistically significant at the one percent level, except the latter which is statistically significant at the ten percent level.

Nearly all candidate health inputs are strongly related with real income per capita. This is especially true in the case of health spending (92 percent), physicians availability (89 percent) and higher education attainment rates (80 percent). Moreover, several of these inputs are highly correlated with each other raising a warning flag regarding a potential collinearity problem in the regression specifications that follow. Notably, the correlation of higher education attainment rates with physicians is 87 percent. We thus consider specifications both with and without the apparently highly collinear physicians-availability variable.

4   Empirical Estimation

In this section, after justifying the methodology employed, we present the empirical results obtained using variables in levels and then their changes.

We are well aware that there is a strong argument for endogeneity between life expectancy and tertiary education. In fact, this is one implication of our theoretical model. While we expect tertiary education to affect health outcomes, individual educational decisions depend on expected longevity so that it is plausible that longer life expectancy causes higher education levels.

For the empirical model we consider below, we fail to reject the null that tertiary education is exogenous with a p-value of 0.21[20] and the joint hypothesis that the two education measures, the

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[20]Treating one explanatory variable at a time as potentially endogenous and the remaining as exogenous, we also fail to reject the null that primary education attainment rates is exogenous with a p-value of 0.78. Similarly, we cannot reject the null that the physicians measure is exogenous with a p-value of 0.25. Nor, can we reject the null that initial income is exogenous with a p-value of 0.52.

physicians variable, and per capita income are all exogenous with a p-value of 0.24. This suggests it might be reasonable to estimate the empirical model with OLS. However, given that individual p-values for the null of exogeneity for each explanatory variable separately range from about 0.21 for tertiary education and physicians to 0.78 for primary enrollment rates, we choose to be conservative regarding our inference of exogeneity and use instrumental variables (IV) estimation. This helps take into account possible endogeneity problems we might have been unable to detect, and acts as a robustness check for OLS estimates.

Towards the goal of addressing potential endogeneity problems and establishing some evidence of temporal causation we consider:

(i) Using lags of higher education and the other explanatory variables[21] to explain end-period averages of life expectancy. Specifically, we utilize time averages of higher education and the other explanatory variables for 1961-75 to explain average life expectancy over 1995-2004. This takes care of endogeneity if individual decisions about higher education in 1961-75 are made independently of life expectancy at birth of the next generation of individuals born between 1995 and 2004. We present results based on this specification as the "Lags" model in columns two and five in Table 3.

(ii) Instrumenting the averages of tertiary education, basic education, real income per capita, and physicians over 1961-95 by their average value during 1961-75 to explain the average value of life expectancy over 1995-2004. In the regression of each potentially endogenous explanatory variable[22] on all exogenous variables, the lag of each explanatory variable is shown to be strongly significant in determining the explanatory variable's period average, with p-values always below the one percent level of significance. Although it is not possible to test for identification, strong rejection of the null that our instruments have no impact on the potentially endogenous explanatory variable is important for the finite sample properties of the IV estimator, as explained in Wooldridge (2002, p.86). We present results based on IV estimation in columns three and six labeled "IV" in Table 3.

(iii) We use log changes in the explanatory variables for the period 1961-75 to explain log changes in life expectancy for 1961-2004 in Table 4 (and for 1977-2004 in appendix Table 6). We report results from this exercise as the "Lags" model in columns two and five of Table 4.

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[21] We cannot use lags for sanitation for which we usually have just a single observation for each country during the end of the period, per capita health spending for which we have just a handful of time series observations per country, and aids which appears only in the second half of the period under consideration.

[22] Again, even though we fail to reject the null of exogeneity for any of these variables and jointly for all of these variables, we are being conservative in allowing for the possibility that these could be endogenous.

(*iv*) We apply IV estimation to the variables in changes, instrumenting the log change in primary and tertiary education over 1961-95 by their 1961-75 value. Results based on this approach are reported in columns three and six under the label "IV" of Table 4, and in the respective columns of Table 6 in the appendix.

In addition to the "Lags" and "IV" models described above in (i) and (ii), we assess the link between health inputs and life expectancy using the period-averages ("Period Avg") model where average life expectancy for 1995-2004 is explained by the average value of the explanatory variables over 1961-95. We report results for this model in columns one and four of Table 3. All variables considered in the regression specifications are in natural logarithms so that the reported estimates are elasticities of life expectancy with respect to each explanatory variable.

Estimates based on log changes of the variables are presented in Table 4. In this case, all variables other than the log of the initial (1961) level of real income per capita are in log changes. In addition to the "Lags" and "IV" models described above in (iii) and (iv), we also consider the "Period Avg" model. In this case, the growth rate of life expectancy between 1961 to 2004 is explained by growth rates of the explanatory variables between 1961 and 1995, with results presented in the first and fourth columns of Table 4. In Table 6 of the appendix, we also present estimates obtained when explaining end-of-period average life expectancy changes between 1977 and 2004, to show robustness of the main finding regarding the importance of higher education in determining future health improvements.

As noted previously, we consider specifications without and with the physicians measure in Models 1 and 2 respectively, since this is highly collinear with higher education. Finally, we note that heteroskedasticity-consistent finite sample standard errors have been used in all estimations.

Estimates for specifications in levels

In Model 1 of Table 3, we consider the impact of basic and higher education attainment rates as well as real income per capita, sanitation, health spending per capita, and AIDS per thousand population, on end-period (1995-2004) average life expectancy. We report results from Model 1 in the first three columns of Table 3. Irrespective of whether we consider the average value of the explanatory variables over 1961-95, their average value at the beginning of the period, or instrument the former with the latter, higher education attainment rates consistently have positive and strongly significant impact on life expectancy which is at least as important as the impact of primary education. The elasticity of life expectancy with respect to higher education ranges from 3.8 percent for the lags model to 5.1 percent for IV estimation, and 5.5 percent for the period-averages model. With most countries in our sample having small average values of higher

Table 3: Explaining 1995-2004 averages of life expectancy

Specif. 1	Model 1 Period Avg	Model 1 Lags	Model 1 IV	Model 2 Period Avg	Model 2 Lags	Model 2 IV
INCOME	.048 (1.45)	.020 (0.64)	.031 (0.93)	.010 (0.30)	−.014 (−0.44)	−.003 (−0.09)
EDHA	.055*** (4.46)	.038*** (2.87)	.051*** (3.83)	.033*** (2.91)	.022* (1.79)	.033*** (2.86)
EDBA	.054* (1.86)	.044 (1.64)	.046 (1.52)	.022 (0.69)	.023 (0.82)	.022 (0.74)
SAN	.059 (1.40)	.083** (2.02)	.070* (1.83)	.074* (1.67)	.096** (2.24)	.080** (2.22)
HEALSP	.016 (1.02)	.032** (2.08)	.025 (1.51)	−.001 (0.04)	.022 (1.24)	.011 (0.61)
AIDS	−.027*** (−4.47)	−.031*** (−4.87)	−.028*** (−4.67)	−.025*** (−4.28)	−.029*** (−4.77)	−.026*** (−4.64)
PHYS	—	—	—	.073*** (3.24)	.062*** (2.59)	.062*** (2.79)
constant	3.109*** (15.1)	3.220*** (16.2)	3.181*** (16.5)	3.635*** (16.4)	3.644*** (15.4)	3.636*** (17.6)
Adj. R^2	81.3	78.9	81.2	83.8	80.1	83.5
Obs.	71	71	71	71	70	70

Notes: * p-value less than one percent, ** p-value less than five percent, *** p-value less than ten percent. Heteroskedasticity-consistent finite sample standard errors are used in constructing t-statistics. All variables are in natural logarithms so that the reported estimates are elasticities of life expectancy with respect to each explanatory variable. For "Period Avg" models, we consider 1995-2004 averages of life expectancy being explained by 1961-95 averages for the explanatory variables. For "Lags" models, we consider again 1995-2004 averages of life expectancy being explained in this case by 1961-75 averages of the education, physicians, and income variables. Finally, for "IV" Models 1 and 2, we instrument the 1961-95 period averages of the education, income, and physicians variables using their beginning of period averages.

education attainment[23], these estimated effects would then suggest that if in fact higher education operated only within the individual household in improving life expectancy, the relatively small proportion of households with higher education attainment would have to experience unrealistically large improvements in life expectancy for the population average to be effected as much as we find in the aggregate data. We interpret our estimated results as evidence for an external effect of higher education on life expectancy.

The estimated elasticity of life expectancy with respect to primary education ranges from 4.4 and 4.6 percent and marginally insignificant for the lags and IV models respectively, to 5.4 percent and marginally significant at the ten percent level for the period-averages model. Sanitation has a positive impact estimated to be significant in two of the three estimations for Model 1. The incidence of AIDS has a negative and strongly significant impact on life expectancy in all three estimations for Model 1.

We also take into account of the fact that income can be a major determinant of health by including 1961-95 and initial period time-averages of real income per capita in the regression specifications presented in the first and second columns of Table 3 respectively, and instrumenting

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[23] 69 percent of countries in our sample had attainment rates less than 2 percent in 1960, 48 percent had rates less than 5 percent in 1975, and 53 percent had rates less than 10 percent even by 1995. The average across countries was around 3 percent for the period from 1961 to 1975 and about 6 percent for 1961-1995.

the former using the latter average for the specification reported in the third column. Real income per capita largely determines individual purchasing power for rival inputs related to health. These would include medical expenditures but also spending on food.[24] Controlling for income helps isolate the part of the effect of each input that is unrelated to income. For the specifications reported in the first three columns of Table 3, income has a positive but insignificant impact on life expectancy. Collinearity might be behind the finding of an insignificant impact of income on life expectancy once we control for other health inputs through which income impacts upon health. For instance, real per capita income and real health expenditure per person have a correlation of 92 percent. When considering income without health spending in the regression (not shown in Table 3), income comes in as a positive and significant determinant of life expectancy for all three specifications of Model 1, consistent with its role as facilitator of rival health-related inputs purchases. Similarly, whereas excluding income from the regression specifications (not shown in Table 3) renders health spending positive and significant in all three specifications of Model 1, including it renders it insignificant except for the lags estimation in the second column of Table 3.

In columns four to six of Table 3, we report results for Model 2 which incorporates physicians availability in addition to the two education variables, sanitation, real per capita health spending, AIDS per thousand population, and real income per capita. To the extent that physicians help absorb and disseminate medical or health-related information across and within countries, in addition to their role as a rival health input, including it should diminish the impact otherwise captured by the measure of higher education.[25] Indeed this is the case. As physicians and higher education are highly collinear, with a correlation of 87 percent (compared to 39 percent with primary education), introducing physicians dampens the impact of higher education on life expectancy. Still, it remains positive and significant, irrespective of whether we use period-averages, lags, or instrument the explanatory variables, in columns four, five, and six respectively. This impact ranges from 2.2 percent for the lags estimation to 3.3 percent for the period-averages and IV estimations.

The estimated life expectancy elasticity of primary education remains positive but is now statistically insignificant throughout the three specifications of Model 2. Sanitation retains a positive and significant impact on life expectancy while health spending per capita is now estimated to have no impact on life expectancy. The latter result is the case irrespective of whether or

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[24]Thus, including income conforms with Fogel's (1994) emphasis on nutrition as a determinant of health.

[25]Physicians play a dual role: first, as a direct rival input into the health production function and second, as facilitators of health-related knowledge absorption and dissemination. Including both tertiary education and physicians in the same specification for Model 2 should thus be expected to reduce coefficient estimates for tertiary education to the extent these two variables capture the same concept. Thus, coefficient estimates for tertiary education in these specifications should be viewed as a lower bound for the importance of the knowledge externality we are focusing on in this paper. Here, we are attributing all of the impact of physicians to its direct role in the health production function, understating the overall effect of health-related knowledge. Alternatively, excluding physicians, leads to a considerable increase in magnitude for higher education estimates.

Table 4: Explaining changes in life expectancy between 1961 and 2004

Specif 3	Model 1 PeriodAvg	Model 1 Lags	Model 1 IV	Model 2 PeriodAvg	Model 2 Lags	Model 2 IV
INCOME	−.001** (−2.09)	−.001*** (−3.03)	−.001* (−1.68)	−.001* (−2.74)	−.001*** (−3.66)	−.001** (−2.39)
EDHA	.052*** (3.76)	.023*** (2.75)	.058*** (3.69)	.033** (2.18)	.021*** (2.85)	.051*** (3.48)
EDBA	.055** (2.43)	.046*** (2.84)	.073*** (2.88)	.038 (1.64)	.037** (2.16)	.057** (2.23)
YGROWTH	.035 (0.99)	.035 (1.21)	.042 (1.46)	.003 (0.10)	.0002 (0.01)	.010 (0.42)
AIDS	−.003* (−1.82)	−.002 (−1.04)	−.003* (−1.77)	−.003** (−2.02)	−.002 (−1.11)	−.003* (−1.93)
PHYS				.085*** (2.84)	.089*** (3.22)	.074*** (2.87)
constant	.011** (2.47)	.013* (3.39)	.008** (2.00)	.012*** (2.69)	.012*** (3.42)	.009** (2.24)
Adj. R^2	32.6	30.3	32.2	44.4	46.2	43.2
Obs.	66	66	66	63	63	63

Notes: Notes: * p-value less than ten percent, ** p-value less than five percent, *** p-value less than one percent. Heteroskedasticity-consistent finite sample standard errors are used in constructing t-statistics. All variables are in natural logarithms. Five countries, Bangladesh, Malawi, Mozambigue, Singapore and Uganda are missing relative to the sample for Table 3. In addition, the physicians measure used in the specifications reported in columns four to six is missing for Ghana, Iceland and Sierra Leone. All variables other than the log of the initial (1961) level of real income per capita are in log changes. YGROWTH is the growth rate of real income per capita. For the "Period Avg" models, we consider the growth rate of life expectancy between 1961 and 2004 being explained by growth rates of the explanatory variables between 1961 and 1995. For the "Lags" model 1, we consider again the growth rate of life expectancy between 1961 and 2004 being explained by growth rates of the education variables between 1961 and 1975. Finally, for the "IV" Models 1 and 2, we instrument the 1961-95 period changes for the education variables using their beginning of period averages.

not we include income per capita in the regression specifications. Similarly, once we include a measure for physicians, the impact of income per capita is estimated to be indistinguishable from zero irrespective of whether or not health spending is included in the estimation. Moreover, the estimated impact of AIDS remains negative and strongly significant. Finally, physicians availability has positive and strongly significant impact on life expectancy that is stable between six and seven percent, irrespective of the methodology being pursued.

Overall, we find that higher education matters significantly and is more robust than basic education, sanitation, health spending, and income. Using initial period averages to explain end-period life expectancy along with IV estimation, we establish that tertiary education is a significant and robust determinant of end of period health status. Our approach alleviates potential endogeneity problems and provides supporting evidence of a causality link from tertiary education to longevity.

Estimates for changes in variables specification

As an additional methodology to remedy potential endogeneity problems facing tertiary education as a determinant of future improvements in life expectancy, we consider log changes of the variables instead of their log levels. This serves as a robustness check for our main finding regarding the relative importance of higher education for life expectancy. When considering changes,

we have to exclude our measures of public sanitation and health spending per capita because of limited data availability over time for these health inputs. We now include changes in real income per capita over the period as a proxy of the growth rate of private health inputs, given that income per capita is a reasonably good measure of purchasing power.

We report estimates from this exercise in Table 4 where we seek to explain changes in life expectancy between 1961 and 2004. The growth rate of higher education attainment levels has positive impact on end-period growth rates in life expectancy for all specifications we consider. It takes its highest value of about six percent in the IV specification reported in column three. The growth rate of primary education has a positive effect higher than that for tertiary education and is statistically significant except for the specification in column four where it is marginally insignificant.[26] It takes its highest value of about seven percent in the IV specification reported in column three. The estimated impact of AIDS is negative as we should expect, and statistically significant in most specifications. The growth rate of physicians comes in positive and strongly significant confirming the importance of the per capita number of physicians in determining health outcomes suggested by the estimation in levels earlier.

Initial income has a negative statistically significant impact on changes in life expectancy between 1961 and 2004 across the board. This is consistent with convergence in life expectancy for countries that started with low real income per capita levels in 1961.[27] The growth rate of real income per capita does not explain any of these gains in life expectancy, suggesting that any gains in life expectancy occurring for initially low-income countries have not been due to higher real income per capita growth in poor countries but likely due to changes in non-income determinants of public health in laggard countries. For example, faster technology absorption (including implementation of public health technologies) of initially laggard countries might actually be behind observed improvements in life expectancy.

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[26]The result about the relative magnitude and statistical significance of basic education is not robust when estimating the impact of the same explanatory variables on end-of-period life expectancy changes between 1977 and 2004. In this case, the estimated coefficients for basic education fall and are statistically insignificant in all three specifications for Model 2. In contrast, the estimated coefficients for higher education are nearly unchanged and remain statistically significant across the board in explaining end-of-period life expectancy changes, suggesting that higher education is an important determinant of improvements in future health outcomes. Table 6 in the appendix reports the results of this estimation exercise.

[27]Evans (1997) shows the coefficient estimate for initial income and the implied rate of convergence to be biased downwards; failing to account for all sources of heterogeneity across countries has the same effect as measurement error, biasing the coefficient estimate of initial income and the implied rate of convergence towards zero. Thus, evidence of convergence shown here is likely a lower bound.

5 Conclusion

This paper presents the results of a macroeconomic analysis of the role of education and other determinants of life expectancy in order to shed some light on the factors responsible for international differences in health performance.

We have presented a theoretical model where intentional investment in health by individuals and external effects of education on the efficiency of this investment, are considered in a unified framework. This allows us to illustrate the complexity of the relation between education and longevity. The causation is bidirectional: longer life expectancy encourages more education by increasing its rate of return and, on the other hand, more education causes longevity. We have advanced three reasons for the latter to be the case. First, better educated agents expect to earn higher future income and have a greater economic return on health, so that they choose to invest more on their own health. Second, these same agents can provide their children with more or better basic education, enhancing the efficiency of these children's investment in health. Third, the efficiency of health investment may directly benefit from the average education level in the economy by enhancing, for instance, the economy's absorptive capacity.

Our empirical results confirm the importance of educational attainment rates in explaining cross-country variation in life expectancy at birth. In addition to the well established health effect of primary education, we find a *direct external* effect of tertiary education on longevity which is as high as six percent. This estimated effect along with the observed small proportion of households having attained higher education, indicates that the estimated effect of higher education attainment rates is indeed largely due to an externality. Our findings then provide evidence of a form of increasing returns to scale in education as far as longevity is concerned. This contrasts with evidence on the determinants of economic growth pointing to decreasing returns to scale in education, with primary education being the single most important factor for income growth and higher education having little or no explanatory power (see Sala-i-Martin, Doppelhofer and Miller 2004).

These findings are robust with respect to our attempts to take into account the endogeneity of higher education. Moreover, results are obtained controlling for other well-established explanatory variables of longevity: real per capita income, real per capita health expenditure, sanitation, AIDS prevalence, and physicians availability. It is noteworthy that the important role of physicians availability comes to the expense of the role of the higher education attainment rate. This supports the idea that the educational externality captures society's absorptive capacity, given that physicians are not just another rival health input but constitute a main carrier of health-related informa-

tion across and within countries. Public health inputs, measured by sanitation, have a positive impact on life expectancy, consistent with Soares (2007b) in relation to Brazil and with evidence summarized in the review articles of Cutler, Deaton, and Lleras-Muney (2006) and Soares (2007a).

Finally, we find that the growth rate of real income per capita does not explain improvements in life expectancy between 1961 and 2004. This is consistent with non-income explanations of gains in longevity. These include changes in tertiary education attainment rates related to the direct external effect interpretation of our theoretical model, but could also include changes in public inputs as suggested by Soares (2007a), and different medical knowledge diffusion channels as in Papageorgiou, Savvides, and Zachariadis (2007).

The results presented in this paper suggest that there is scope for studying the determinants of welfare growth as a concept that is (closely related but) distinct from economic growth. In fact, non-income factors are shown to be important for explaining variation of life expectancy across countries. Policy implications may be important. For instance, the findings of this paper suggest that investing in health inputs might be important for welfare growth even if the effect of health on economic growth is small as in Weil (2007), or non-existent as in Acemoglu and Johnson (2008). While the latter's "estimates exclude any positive effects of life expectancy on GDP per capita" (p. 3), they acknowledge that consistent with Becker, Philipson, and Soares (2005), "[*health*] interventions have considerably improved overall welfare" (p. 4). Our analysis highlights the crucial role that educational policies may play in enhancing welfare, by pointing out to the role education plays in influencing the health component of welfare.

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A Appendix

A.1 Proof of proposition 1

Preliminary. Recall that from (14)

$$LHS\left(h\right) \equiv c_1\left(h\right) + pm\left(h\right)$$

where $c_1\left(h\right)$ and $m\left(h\right)$ are given by (13) and (12) respectively. Consider (14) and write it in implicit form as

$$F\left(h,x\right) \equiv c_1\left(h\right) + pm\left(h\right) + w_t kh - w_t = 0$$

where x denotes a parameter with respect to which comparative statics exercises are performed. The we can compute

$$\frac{dh}{dx} = -\frac{\frac{\partial F}{\partial x}}{\frac{\partial F}{\partial h}} = -\frac{\frac{\partial c_1}{\partial x} + \frac{\partial pm}{\partial x} + \frac{\partial w_t kh}{\partial x} - \frac{\partial w_t}{\partial x}}{\frac{\partial c_1}{\partial h} + \frac{\partial pm}{\partial h} + \frac{\partial w_t kh}{\partial h} - \frac{\partial w_t}{\partial h}}$$

Impact of z. An exogenous increase in z shifts downward the LHS schedule but leaves the RHS unchanged. As a result h must increase to restore individual optimality. As a consequence from (12) $\frac{dm}{dz} = \frac{\partial m}{\partial h}\frac{dh}{dz} > 0 \Rightarrow$ from (1) $\frac{d\pi}{dz} = \frac{\partial \pi}{\partial z} + \mu\frac{\pi}{m}\frac{dm}{dz} >> 0$, from (9) $\frac{db}{dz} = \frac{\partial b}{\partial h}\frac{dh}{dz} > 0$, from (7) $\frac{dc_2}{dz} = \frac{\theta}{1-\theta}\kappa\frac{db}{dz} > 0$. Finally from (13) $\frac{dc_1}{dz} = \frac{\partial c_1}{\partial z} + \frac{\partial c_1}{\partial h}\frac{dh}{dz}$, yet the sign is determined through graphical analysis: at the new individual solution it must be that $\frac{dLHS}{dz} < 0$ which requires that $\frac{dc_1}{dz} << 0$ since $\frac{dpm}{dz} = p\frac{dm}{dz} > 0$.

Impact of p. An exogenous increase in p shifts upward the LHS schedule but leaves the RHS unchanged. As a result h must decrease to restore individual optimality (more so the greater is μ/σ). As a consequence from (12) $\frac{dm}{dp} = \frac{\partial m}{\partial p} + \frac{\partial m}{\partial h}\frac{dh}{dp} << 0 \Rightarrow$ from (1) $\frac{d\pi}{dp} = \mu\frac{\pi}{m}\frac{dm}{dp} << 0$, from (9) $\frac{db}{dp} = \frac{\partial b}{\partial h}\frac{dh}{dp} < 0$, from (7) $\frac{dc_2}{dp} = \frac{\theta}{1-\theta}\kappa\frac{db}{dp} < 0$, from (13) $\frac{dc_1}{dp} = -\frac{\partial c_1}{\partial h}\frac{dh}{dp} > 0$ (moreover at the new individual solution $\frac{dLHS}{dp} > 0$, while expenditure in health-related inputs, pm, falls according to (12), implying an important increase in c_1).

Impact of k. An exogenous increase in k shifts upward the LHS schedule and downwards the RHS (which pivots on its vertical intercept). As a result h must decrease to restore individual optimality. As a consequence from (9) $\frac{db}{dk} < 0$, from (7) $\frac{dc_2}{dk} < 0$. The other effects are less straightforward.

From (12) $\frac{dm}{dk} = \frac{\partial m}{\partial k} + \frac{\partial m}{\partial h}\frac{dh}{dk} = \frac{m}{k}\{1 + \left[1 - \eta\left(1 - \frac{h^\eta}{1+h^\eta}\right)\right]\frac{dh}{dk}\frac{k}{h}\}$.

Using the implicit function formulation of (14) we can write

$$\frac{dh}{dk} = -\frac{\frac{1-\mu}{\sigma}\frac{c_1}{k} + p\frac{m}{k} + w_t h}{\frac{1}{\sigma}\left[(1-\eta)(1-\mu) + \eta(\sigma-\mu)\frac{h^\eta}{1+h^\eta}\right]\frac{c_1}{h} + \left[(1-\eta) + \eta\frac{h^\eta}{1+h^\eta}\right]\frac{pm}{h} - w_t k}$$

$$\frac{dh}{dk}\frac{k}{h} = -\frac{\frac{1-\mu}{\sigma}c_1 + pm + w_t kh}{\frac{1-\mu}{\sigma}c_1\left[1 - \eta\left(1 - \frac{\sigma-\mu}{1-\mu}\frac{h^\eta}{1+h^\eta}\right)\right] + pm\left[1 - \eta\left(1 - \frac{h^\eta}{1+h^\eta}\right)\right] + w_t kh}$$

The impact on expenditure on education, $w_t kh$, is given by

$$\frac{d\left(w_t kh\right)}{dk} = w_t h\left(1 + \frac{dh}{dk}\frac{k}{h}\right)$$

Therefore

$$\frac{d\left(kh\right)}{dk} < 0 \quad \Leftrightarrow \quad \left|\frac{dh}{dk}\frac{k}{h}\right| > 1$$

which is always satisfied given that $\left[1 - \eta\left(1 - \frac{\sigma-\mu}{1-\mu}\frac{h^\eta}{1+h^\eta}\right)\right] \in (0,1)$ and $\left[1 - \eta\left(1 - \frac{h^\eta}{1+h^\eta}\right)\right] \in$

$(0,1)$.

Moreover from (12) we have that

$$\frac{dm}{dk}\frac{k}{m} = 1 + \left[1 - \eta\left(1 - \frac{h^\eta}{1+h^\eta}\right)\right]\frac{dh}{dk}\frac{k}{h}$$

Therefore

$$\frac{dm}{dk} < 0 \quad \Leftrightarrow \quad \left|\frac{dh}{dk}\frac{k}{h}\right| > \left[1 - \eta\left(1 - \frac{h^\eta}{1+h^\eta}\right)\right]^{-1}$$

According to $\left|\frac{dh}{dk}\frac{k}{h}\right|$ computed above the last inequality is equivalent to

$$\left[\frac{1-\mu}{\sigma}c_1 + pm + w_t kh\right]\left[1 - \eta\left(1 - \frac{h^\eta}{1+h^\eta}\right)\right]$$
$$> \frac{1-\mu}{\sigma}c_1\left[1 - \eta\left(1 - \frac{\sigma-\mu}{1-\mu}\frac{h^\eta}{1+h^\eta}\right)\right] + pm\left[1 - \eta\left(1 - \frac{h^\eta}{1+h^\eta}\right)\right] + w_t kh$$

$$\frac{1-\sigma}{\sigma}c_1 > w_t kh^{1-\eta}$$

Next substituting for c_1 using (13) we get

$$\frac{1-\sigma}{\sigma}\left(\left(\frac{1-\sigma}{\mu}p\right)^\mu\frac{1}{\rho az}\left(\frac{k}{\eta}w_t\right)^{1-\mu}\left(\frac{1-\theta}{\kappa}w_{t+1}\right)^{\sigma-1}\right)^{\frac{1}{\sigma}}h^{\frac{1}{\sigma}(1-\eta)(1-\mu)}(1+h^\eta)^{\frac{\sigma-\mu}{\sigma}} > w_t kh^{1-\eta}$$

$$\left(\frac{1}{\rho az}\left(\frac{p}{\mu}\right)^\mu\eta^{-(1-\mu)}\sigma^{-\sigma}\left(\frac{1-\theta}{\kappa}w_{t+1}\right)^{-(1-\sigma)}(1-\sigma)^{\sigma+\mu}(kw_t)^{-(\sigma+\mu-1)}\right)^{\frac{1}{\sigma}}(1+h^\eta)^{\frac{\sigma-\mu}{\sigma}} > h^{(1-\eta)\frac{\sigma+\mu-1}{\sigma}}$$

Both sides of this inequality are increasing and concave in h if
$(i)\ \sigma > 1 - \mu$.
Moreover the left-hand-side of the inequality starts above zero with finite slope, while the right-hand-side starts at zero with infinite slope. Under (i) for low levels of h we have $\frac{dm}{dk} < 0$ implying $\frac{d\pi}{dk} < 0$.
This is the case for any admissible value of h if the following inequality holds:

$$(ii)\ \left(\frac{1}{\rho az}\left(\frac{p}{\mu}\right)^\mu\eta^{-(1-\mu)}\sigma^{-\sigma}\left(\frac{1-\theta}{\kappa}w_{t+1}\right)^{-(1-\sigma)}(1-\sigma)^{\sigma+\mu}w_t^{-(\sigma+\mu-1)}\right)^{\frac{1}{\sigma}}(1+k^{-\eta})^{\frac{\sigma-\mu}{\sigma}} > k^{\frac{\eta}{\sigma}(\sigma+\mu-1)}$$

This is a sufficient condition for the inequality implied by $dm/dk < 0$ to be satisfied for any $h \leq 1/k$. This restriction on parameters configurations is satisfied for sufficiently low values of k.

From (13) $\frac{dc_1}{dk} = -w_t\frac{dkh}{dk} - p\frac{dm}{dk}$. Under (i)-(ii) the second term on the right-hand-side is positive. Furthermore according to $\frac{dh}{dk}\frac{k}{h}$ computed above we have that $\left|\frac{dh}{dk}\frac{k}{h}\right| > 1$. It follows that $\frac{dkh}{dk} < 0$, implying that also the first term on the right-hand-side is positive. Overall $\frac{dc_1}{dk} > 0$ under conditions (i)-(ii).

Impact of κ. An exogenous increase in κ shifts upward the LHS schedule but leaves the RHS unchanged. As a result h must decrease to restore individual optimality. As a consequence from (12) $\frac{dm}{d\kappa} = \frac{\partial m}{\partial h}\frac{dh}{d\kappa} < 0 \Rightarrow$ from (1) $\frac{d\pi}{d\kappa} = \mu\frac{\pi}{m}\frac{dm}{d\kappa} < 0$. Given that at the new individual solution $\frac{dLHS}{d\kappa} > 0$, but expenditure in health-related inputs, pm, falls while income increases (as kh falls), c_1 must increase substantially. Moreover from (9) $\frac{db}{d\kappa} = -\frac{b}{\kappa} + \frac{\partial b}{\partial h}\frac{dh}{d\kappa} << 0$, implying $\frac{dc_2}{d\kappa} = \frac{\theta}{1-\theta}\frac{d\kappa b}{d\kappa} = \theta w_{t+1}\eta h^{\eta-1}\frac{dh}{d\kappa} < 0$.

A.2 Proof of proposition 2

<u>Impact of s.</u> An exogenous increase in s shifts downward the $\widetilde{LHS}$ schedule but leaves the RHS unchanged since $\frac{d\widetilde{LHS}}{ds} = -\delta c_1/s$ with c_1 given by (16). As a result h must increase to restore individual optimality. As a consequence from (12) $\frac{dm}{ds} = \frac{\partial m}{\partial h}\frac{dh}{ds} > 0$ and from (9) $\frac{db}{ds} = \frac{\partial b}{\partial h}\frac{dh}{ds} > 0$. Overall the impact on life expectancy is computed using (15)

$$\frac{d\pi}{ds}\frac{s}{\pi} = \delta + \left[\mu(1-\eta) + \eta(\mu+\beta)\frac{h^\eta}{1+h^\eta} + \alpha\right]\frac{dh}{ds}\frac{s}{h} \tag{19}$$

which is increasing in the strength of the externalities α and β.

<u>Impact of p.</u> An exogenous increase in p shifts upward the $\widetilde{LHS}$ schedule but leaves the RHS unchanged. As a result h must decrease to restore individual optimality. As a consequence from (12) $\frac{dm}{dp} = \frac{\partial m}{\partial p} + \frac{\partial m}{\partial h}\frac{dh}{dp} << 0$, from (9) $\frac{db}{dp} = \frac{\partial b}{\partial h}\frac{dh}{dp} < 0$. The total effect on π is reinforced by the fact that m, b and h move in the same direction (see eq. 15).

<u>Impact of k.</u> An exogenous increase in k shifts upward the $\widetilde{LHS}$ schedule and downwards the RHS (which pivots on its vertical intercept). As a result h must decrease to restore equilibrium. As a consequence from (9) $\frac{db}{dk} = (1-\theta)(w/\kappa)\eta h^{\eta-1}dh/d\kappa < 0$. Differentiating (12) $\frac{dm}{dk} = \frac{\partial m}{\partial k} + \frac{\partial m}{\partial h}\frac{dh}{dk} = \frac{m}{k}\{1 + \left[1 - \eta\left(1 - \frac{h^\eta}{1+h^\eta}\right)\right]\frac{dh}{dk}\frac{k}{h}\}$. Therefore

$$\frac{dm}{dk} < 0 \quad \Leftrightarrow \quad \left|\frac{dh}{dk}\frac{k}{h}\right| > \left[1 - \eta\left(1 - \frac{h^\eta}{1+h^\eta}\right)\right]^{-1}$$

We compute $\left|\frac{dh}{dk}\frac{k}{h}\right|$ applying the same method as in the proof of proposition 1 taking into account (16) for $\partial c_1/\partial k$ and $\partial c_1/\partial h$, and get

$$\frac{dh}{dk}\frac{k}{h} = -\frac{\frac{1-\mu}{\sigma}c_1 + pm + wkh}{\frac{1-\mu}{\sigma}c_1\left[1 - \eta\left(1 - \frac{\sigma-\mu-\beta}{1-\mu}\frac{h^\eta}{1+h^\eta}\right) - \frac{\alpha}{1-\mu}\right] + pm\left[1 - \eta\left(1 - \frac{h^\eta}{1+h^\eta}\right)\right] + wkh}$$

Therefore $dm/dk < 0$ if

$$\left[\frac{1-\mu}{\sigma}c_1 + pm + wkh\right]\left[1 - \eta\left(1 - \frac{h^\eta}{1+h^\eta}\right)\right]$$
$$> \frac{1-\mu}{\sigma}c_1\left[1 - \eta\left(1 - \frac{\sigma-\mu-\beta}{1-\mu}\frac{h^\eta}{1+h^\eta}\right) - \frac{\alpha}{1-\mu}\right] + pm\left[1 - \eta\left(1 - \frac{h^\eta}{1+h^\eta}\right)\right] + wkh$$

that is

$$c_1\left[h^\eta\left[1-\sigma+\beta\right] + \frac{\alpha}{\eta}(1+h^\eta)\right] > \sigma wkh$$

Next substituting for c_1 using (16) we get

$$\frac{\alpha}{\eta}\left[\frac{s^{-\delta}}{\rho a\zeta}\left(\frac{1-\sigma}{\mu}p\right)^\mu\left(\frac{kw}{\eta}\right)^{1-\mu}\left(\frac{1-\theta}{\kappa}w\right)^{-(1-\sigma)-\beta}\right]^{\frac{1}{\sigma}}(1+h^\eta)^{\frac{1}{\sigma}[\sigma-\mu-\beta]}$$

$$+ \left[\frac{s^{-\delta}}{\rho a\zeta}\left(\frac{1-\sigma}{\mu}p\right)^\mu\left(\frac{kw}{\eta}\right)^{1-\mu}\left(\frac{1-\theta}{\kappa}w\right)^{-(1-\sigma)-\beta}\right]^{\frac{1}{\sigma}}(1+h^\eta)^{\frac{1}{\sigma}[\sigma-\mu-\beta]}\left[1-\sigma+\beta+\frac{\alpha}{\eta}\right]h^\eta$$

$$> \sigma wkh^{\frac{1}{\sigma}[\sigma-(1-\eta)(1-\mu)+\alpha]}$$

Under assumption 4 the right-hand-side of this inequality is increasing in h, up from

$\alpha/\eta[\frac{s^{-\delta}}{\rho a \zeta}(\frac{1-\sigma}{\mu}p)^\mu(\frac{kw}{\eta})^{1-\mu}(\frac{1-\theta}{\kappa}w)^{-(1-\sigma)-\beta}]^{\frac{1}{\sigma}} > 0$ for $h = 0$. The left-hand-side is increasing and concave up from zero for $h = 0$, if $\sigma - (1-\eta)(1-\mu) + \alpha > 0$. A sufficient condition for the latter to be satisfied is that

(iii) $\sigma > 1 - \mu - \alpha$

which is less restrictive than corresponding condition (i) for the case of the individual solution.

Hence the inequality is satisfied for any admissible $h \leq 1/k$ if (iii) and the following condition are satisfied:

$$(iv) \quad \left[\frac{s^{-\delta}}{\rho a \zeta}\left(\frac{1-\sigma}{\mu}p\right)^\mu\left(\frac{kw}{\eta}\right)^{1-\mu}\left(\frac{1-\theta}{\kappa}w\right)^{-(1-\sigma)-\beta}\right]^{\frac{1}{\sigma}}\left(1+k^{-\eta}\right)^{\frac{1}{\sigma}[\sigma-\mu-\beta]}$$

$$\left[\frac{\alpha}{\eta}+\left(1-\sigma+\beta+\frac{\alpha}{\eta}\right)k^{-\eta}\right] > \sigma w k^{\frac{1}{\sigma}[(1-\eta)(1-\mu)-\alpha]}$$

This condition is satisfied, for instance, for low enough values of k.

Impact of κ. An exogenous increase in κ shifts upward the $\widetilde{LHS}$ schedule but leaves the $\overline{RHS}$ unchanged. As a result h must decrease to restore individual optimality. The shift in the $\widetilde{LHS}$, $\frac{\partial \widetilde{LHS}}{\partial \kappa} = (1-\sigma+\beta)c_1/\kappa$, is relatively large as compared to that of the LHS of (14), $\frac{\partial LHS}{\partial \kappa} = (1-\sigma)c_1/\kappa$, which implies that the adjustment of h is larger with externalities than without. It follows from (12) that $\frac{dm}{d\kappa} = \frac{\partial m}{\partial h}\frac{dh}{d\kappa} = [1-\eta(1-\frac{h^\eta}{1+h^\eta})]\frac{dh}{d\kappa}\frac{m}{h} < 0$ and from (9) that $\frac{db}{d\kappa} = -\frac{b}{\kappa} + \frac{\partial b}{\partial h}\frac{dh}{d\kappa} = (-1+\eta\frac{h^\eta}{1+h^\eta}\frac{dh}{d\kappa}\frac{\kappa}{h})\frac{b}{\kappa} << 0$ (this adjustment is much larger with externalities than without since b is higher as results from proposition 2 and equation 9, and at the same time $dh/d\kappa$ is greater as argued above). Differentiating (15)

$$\frac{d\pi}{d\kappa}\frac{\kappa}{\pi} = -\beta + \left\{\alpha + \beta\eta\frac{h^\eta}{1+h^\eta} + \mu\left[1-\eta\left(1-\frac{h^\eta}{1+h^\eta}\right)\right]\right\}\frac{dh}{d\kappa}\frac{\kappa}{h}$$

where only the last term is plays a role in the case without externalities.

Impact of w. To determine the direction of adjustment of h, first divide both sides of (17) by w, so as to see that an increase in w shifts downwards only the first term of the (modified) LHS. This implies that h must increase to restore equilibrium. Moreover from (9) $\frac{db}{dw} = (1+\eta\frac{h^\eta}{1+h^\eta}\frac{dh}{dw}\frac{w}{h})\frac{b}{w}$ and from (12) $\frac{dm}{dw} = \{1+[1-\eta(1-\frac{h^\eta}{1+h^\eta})]\frac{dh}{dw}\frac{w}{h}\}\frac{m}{w}$, implying from (15)

$$\frac{d\pi}{dw}\frac{w}{\pi} = \mu + \beta + \left\{\alpha + \beta\eta\frac{h^\eta}{1+h^\eta} + \mu[1-\eta(1-\frac{h^\eta}{1+h^\eta})]\right\}\frac{dh}{dw}\frac{w}{h} \quad (20)$$

The complete impact on life expectancy of an increase in the exogenous component of income is much stronger in the presence of externalities than without them.

Table 5: List of countries in the dataset

Country	Avg Life Expectancy†	Country	Avg Life Expectancy†
Algeria	70.3	Korea	75.7
Argentina	73.9	Malawi	40.8
Australia	78.9	Malaysia	72.7
Austria	78.0	Mali	47.7
Bangladesh	61.5	Mauritius	71.3
Belgium	77.7	Mexico	74.4
Bolivia	63.1	Mozambique	42.6
Brazil	69.7	Myanmar	59.5
Cameroon	47.2	Netherlands	78.0
Canada	78.8	New Zealand	78.1
Chile	76.9	Norway	78.6
Colombia	71.5	Pakistan	63.2
Costa Rica	77.6	Panama	74.4
Cyprus	78.3	Paraguay	70.4
Denmark	76.5	Peru	69.3
Ecuador	73.4	Philippines	69.5
El Salvador	70.1	Portugal	76.2
Finland	77.5	Sierra Leone	40.3
France	78.8	Singapore	77.9
Germany	77.7	Spain	78.9
Ghana	56.8	Sri Lanka	73.6
Greece	78.2	Sudan	55.9
Guatemala	66.2	Sweden	79.6
Haiti	50.8	Switzerlannd	79.7
Honduras	67.4	Tanzania	47.2
Iceland	78.8	Thailand	69.7
India	62.8	Tunisia	72.4
Indonesia	65.9	Turkey	68.1
Iran	69.5	Uganda	45.7
Ireland	76.9	United Kingdom	77.6
Israel	78.3	Unites States	76.7
Italy	79.2	Uruguay	74.3
Jamaica	71.1	Venezuela	73.0
Japan	80.9	Zambia	38.4
Jordan	70.7	Zimbabwe	40.7
Kenya	49.2		

$\dagger$: This is the end of period average life expectancy from 1995 to 2004. Iceland is missing for the lags and IV models.

Table 6: Considering end-of-period life expectancy changes

Specif 3	Model 1 PeriodAvg	Model 1 Lags	Model 1 IV	Model 2 PeriodAvg	Model 2 Lags	Model 2 IV
INCOME	.0002 (0.27)	−.0002 (−0.35)	.0003 (0.46)	−.0001 (−0.16)	−.0003 (−0.54)	.0000 (0.08)
EDHA	.050*** (2.84)	.024** (2.05)	.060*** (2.72)	.033* (1.69)	.023** (2.19)	.056*** (2.75)
EDBA	.056** (1.98)	.040** (2.07)	.061** (2.12)	.034 (1.24)	.027 (1.35)	.037 (1.31)
YGROWTH	.062 (1.33)	.058 (1.49)	.061* (1.67)	.028 (0.75)	.022 (0.75)	.026 (0.90)
AIDS	−.004 (−1.62)	−.003 (−1.14)	−.004* (−1.76)	−.003 (−1.55)	−.002 (−1.01)	−.003* (−1.65)
PHYS				.099** (2.42)	.105*** (2.75)	.089** (2.48)
constant	.0001 (0.01)	.004 (0.67)	−.001 (−0.20)	.001*** (0.13)	.002 (0.37)	−.001 (−0.23)
Adj. R^2	16.7	14.1	16.5	29.3	30.7	28.3
Obs.	66	66	66	63	63	63

Notes: * p-value less than ten percent, ** p-value less than five percent, *** p-value less than one percent. Heteroskedasticity-consistent finite sample standard errors are used in constructing t-statistics. All variables are in natural logarithms. Five countries, Bangladesh, Malawi, Mozambigue, Singapore and Uganda are missing relative to the sample for Table 5. In addition, the physicians measure used in the specifications reported in columns four to six is missing for Ghana, Iceland and Sierra Leone. All variables other than the log of the initial (1961) level of real income per capita are in log changes. YGROWTH is the growth rate of real income per capita. For the "Period Avg" models, we consider the growth rate of life expectancy between 1977 and 2004 being explained by growth rates of the explanatory variables between 1961 and 1995. For the "Lags" model 1, we consider again the growth rate of life expectancy between 1977 and 2004 being explained by growth rates of the education variables between 1961 and 1975. Finally, for the "IV" Models 1 and 2, we instrument the 1961-95 period changes for the education variables using their beginning of period averages.