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Longevity and Education Externalities: A Macroeconomic Perspective
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Abstract

We argue that education exerts positive external effects on health, beyond the standard internal effects documented in the literature. We put forward a model that clarifies the links between education and longevity in the presence of such an externality. We then implement an innovative approach to control for endogeneity problems, and present evidence for the significant role played by higher education in explaining longevity across countries. Our findings provide empirical evidence in support of our hypothesis of educational externalities on health.

Keywords: Education, life expectancy, externalities.

JEL Classification: O50, O11, I0

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1 Introduction

Our purpose in writing this paper is to contribute to the understanding of the determinants of health outcomes across countries. A recent strand of the macroeconomic literature proposes various explanations for the observed evolution of life expectancy across countries over the last century. Some of these papers underscore the important role played by economic growth while others emphasize other factors not directly determined by income.¹ A related large body of research in development and health economics has long established the importance of maternal education for the health status of family members.² There is also growing evidence of a direct beneficial effect of own education on individual health (e.g. Lleras-Muney, 2005). Instead, there has not been as much emphasis on how the overall level of education in a country might lead to positive externalities for health outcomes.³ In this paper, we hypothesize a role of education that goes beyond the widely accepted role of parental education at the individual family level. We ask whether education has positive externalities on health. Does a person's education choice exert beneficial effects on the health of people outside the household? Equivalently, does the education level in a country have a positive influence on average life expectancy beyond what could be attributed to improved longevity within educated households?

Despite the interest that such a possibility arises, and although the related issue of externalities from education on income growth has been debated for over two decades (at least since Lucas, 1988), these are novel questions in economics.⁴ Several authors have emphasized the role of education in health, but they have always focused on the benefits accruing directly to the educated person or to members of his/her household. For instance, Soares (2007a) states 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." Cutler, Deaton and Lleras-

¹To the extent that income is not the sole determinant of health outcomes, one can view health as a separate component of welfare. In this case, factors driving welfare growth might well be different from those relevant for economic growth, with important policy implications. Thus, a central question in the debate on the determinants of international health outcomes is whether these are a mere by-product of economic growth (see Pritchett and Summers, 1996), or whether 'exogenous' non-income sources are in part responsible. The latter argument is made by Preston (1975, 1980, 1996) and more recently by Becker, Philipson and Soares (2005), Soares (2007a, 2007b), Papageorgiou, Savvides, and Zachariadis (2007) who argue that longevity is related to medical technology diffusion, and Galor and Moav (2007) who propose that current longevity is related to each country's distant past via the evolutionary process that got started with the increased prevalence of disease at the point in time a population experienced the Neolithic transition into farming.

²See Wolfe and Behrman (1982), Haines and Avery (1982), Merrick (1985), Barrera (1990), Hobcraft (1993), and Glewwe (1999).

³This is perhaps an artifact of the use of micro-data. Krueger and Lindahl (2001, p.1108) point out that "estimating relationships with aggregate data can capture external returns to human capital that are missed in the microeconometric literature."

⁴The related "...significant open question ... whether the social returns to human capital investment substancially exceed the private return" (Topel, 1999, p. 2973) has been raised by economists as early as Becker (1975).

⁵Similarly, Cutler, Deaton and Lleras-Muney (2006, p. 115) write that "the differential use of health knowledge

Muney (2006) review evidence from studies that compare experiences across countries and from papers that use data within a particular country. For the first group of papers, the authors do not cite any study that points to a positive impact of education on health external to the household. These papers are mainly concerned with basic education whose role is summarized in the following sentence: "to the extent that education improves an individual's ability to undertake these changes, more educated mothers will have healthier babies" (p. 110). For the second group of papers, using within-country data, the authors underscore the positive effect of higher education but their concern focuses solely on the effect that is internal to the households, whereby "educational differences [...] will maintain a gradient in health whenever there exists a mechanism or technology that more knowledgeable and educated people can use to improve their health" (p. 115). As compared to the papers reviewed in this survey, our paper puts forward an original hypothesis (the externality) and tests its empirical plausibility on cross-country data.

Our main contribution is to consider empirically and theoretically the possible existence of an externality from education on aggregate health outcomes. We examine the determinants of aggregate health outcomes with emphasis on the mechanisms through which education impacts upon longevity, with due attention paid to the role played by a number of other 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 the extent to which health-related knowledge is exploited in society. For the individual, education -whether own or parental- is crucial in facilitating access to and the understanding of health-related information.⁶ The availability of health knowledge depends in the first place 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 medical treatments, assessing the risks that hazardous behavior entails, or providing children with healthy food. Second, the aggregate level of education in the economy improves the quality of health services offered within a country. One reason for this could be that the average level of education improves a country's absorptive capacity for health-related technology and ideas. Another reason is that we would expect physicians to be more likely to adopt and implement new treatments to the general population in countries or areas where the average

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."

⁶Some effects concern spouses within the household. Cutler and Lleras-Muney (2006, p. 10) cite medical studies finding that "even controlling for own education, those who are married to more educated spouses have lower mortality rates" and better health behaviors pertaining to smoking or alcohol consumption.

⁷Kenkel (1991) emphasizes better information on health, and Grossman (1972) better decision-making by more educated individuals.

patient is more educated.⁸ Moreover, health-related information might flow from individuals with higher education to the remaining individuals⁹ enabling the latter to make better informed health decisions and maintaining a healthier lifestyle as the seeds of a new health culture take root. Finally, in the presence of a disease arrival rate which is lower for more highly educated individuals, the entire community would benefit from a lower probability of disease transmission.

The paper first considers the theoretical implications of our main hypothesis concerning the externality of education on longevity. It then sets out an empirical analysis to evaluate the pertinence of such an externality. The theory section presents a model where educational choices, investment in health, and longevity are endogenously determined. This allows us to clarify the complex relationship between education and longevity, providing guidance to the empirical investigation that follows. The latter provides evidence in support of our main hypothesis, i.e. the external effects of education on longevity. It is in this sense that we interpret the significant role played by tertiary education in explaining variations in life expectancy at birth across countries after implementing a novel approach to control for endogeneity problems. The remainder of the introduction presents an overview of the methodology used and the results obtained.

We build a theoretical model to analyze the relationship between life expectancy, educational decisions, private and public investment in health, and income. Similar to Chakraborty and Das (2005), individuals intentionally spend resources to enhance their life expectancy. 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 exists at the individual level: while improved life expectancy raises the return on education inducing greater demand, 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 $direct^{10}$ roles played by education in determining longevity. It is assumed that longevity is an increasing function of the 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 the two forms of education is mean-

⁸We expect health specialists to be more willing to learn about, explain, and implement new treatments to educated patients more receptive to new medical knowledge. Cutler and Lleras-Muney (2006, p.16) argue that "[t]he more educated ... appear to make use of new health related information first" and "are more likely to trust science."

 $^{^9}$ Mankiw (1997, p.107) notes a similar externality where "educated people generate good ideas that enter society's pool of knowledge."

¹⁰Compared to an *indirect* effect of education on longevity running through permanent income, identified in the case of the individual problem.

ingful 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. ¹¹ This allows us to identify four links between education and longevity. Three of these links are 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 the correlations between observable variables under our hypothesis on the causation from education to 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 education, basic education, private health investment and life expectancy are positively related and increasing with public health investment, per capita income and the efficiency of the educational system.

Next, we test the empirical plausibility of the supposed direct roles played by education in determining longevity, with a focus on the channel external to the household. To this end, it is necessary to address three issues. First, what is the most appropriate empirical approach? Second, how can one distinguish the two different roles played by education in determining health outcomes? Third, to what extent can we hope that our analysis uncovers causation from more education to better health rather than the other way around?

The objective of our analysis offers direct guidance in the choice of the empirical approach. Given that the externality from education can be expected to act on a geographical scale, it seems appropriate to compare data across countries. We also need a data set including sufficient variation in both life expectancy and the various measures of educational level of the population used. It is thus desirable to consider developing countries as well as industrialized economies. This comes at the cost of the time dimension of the sample since quite a few of the variables we consider

¹¹Assuming 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.

are exceedingly sparse over time, especially so for developing countries. However, we note that focusing on long-run time averages in levels and on differences over long time periods seems more appropriate due to the inherent long-run nature of the relation under study. Moreover, averaging over long periods helps alleviate potential measurement error problems. This greatly improves the reliability of the education data used as shown in previous work by Topel (1999), Krueger and Lindahl (2001) and Cohen and Soto (2007) (CS). In light of all the above, we opt to explore the cross sectional dimension of a sample of 71 countries using explanatory variables averaged over 1961-95 to explain life expectancy averaged over the period from 1995 to 2004. Moreover, we use a variety of sources for measuring higher education, that includes Barro and Lee (2001) (BL), CS, and Lutz etal (2007) (LGSS).

Turning to the issue of identifying the externality, we interpret a direct role of tertiary education on longevity as evidence of external effects of education. There are at least two reasons to do so. The first one rests on the following theoretical argument. The two direct effects of education play conceptually different roles. The first one operates as a rival input benefitting household members. We expect this role of education in enhancing a household's longevity to exhibit diminishing returns so that basic literacy rates should suffice to capture it.¹² The second effect depends in part on the ability and readiness of the health sector to take advantage of best practices. This is a hightech 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. Moreover, health-related information might flow from individuals with higher education to others. In general, and as noted by Mankiw (1997, p.107) "once we start thinking about externalities, it seems unlikely that they are the same at all levels of schooling ... educated people generate good ideas that enter society's pool of knowledge ... this externality might well flow largely from the most educated members of society ... so, differences in ... higher education would be more important ...". Controlling for literacy rates, any additional effects from higher education would then be consistent with this second external role of education.

A complementary argument for interpreting the impact of tertiary education on average life expectancy as evidence of externalities, is based on the observation that only a small fraction of

¹²This 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 "the 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, "the 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."

the population had acquired tertiary education in our sample in conjunction with the fact that life expectancy is computed as an average across educational groups. The fact that the absence of an external effect of education suggests that only those who acquire tertiary education benefit from their actions, allows us to make an heuristic argument for the presence of an externality: If only those that choose to acquire tertiary education end up benefitting (the internal channel), then the implied increase in expected years to be enjoyed by this small group would be implausibly large based on our estimated effect of tertiary education on average life expectancy. In contrast, if we hypothesize that others benefit as well, then the implied increase in expected years to be enjoyed by this larger group of individuals is plausible. This would then be evidence of an externality for tertiary education to the extent that for a given observed estimated elasticity of average life expectancy with respect to tertiary education to be consistent with a plausible increase in expected years for different segments of society, we would have to assume that individuals other than those that choose to acquire tertiary education end up benefitting as well.

Finally, a major empirical concern arises as a result of the possibility that individuals incorporate forecasts of future longevity in making educational decisions. To address problems in capturing the direction of causality, we consider a procedure that utilizes demographic forecasts for life expectancy around 2000 as assessed by UN demographers in 1973. This is consistent with individuals making forecasts of future longevity which are as accurate as those carried out with the help of complicated demographic models. For example, individuals might simply adopt the UN longevity forecasts once these become publicly available. We use the component of education not explained by the UN forecasts¹³, to explain deviations of actual life expectancy averaged over 1995-2004 relative to its forecast. These deviations represent the unpredicted component of life expectancy that presumably does not affect educational decisions. If these deviations are explained by exogenous variation in 1961-80 education variables, we can interpret this as evidence of causality from education on life expectancy.

Controlling for the effect of female literacy, initial income, sanitation and other health inputs, we find that tertiary education is a significant determinant of average longevity in the population, consistent with an aggregate externality role for education.

The next section presents the model and theoretical results. Section 3 describes the data, the empirical analysis, and the results. The last section briefly concludes.

¹³The residual from a regression of higher education averaged over 1961-80 on the 1973 forecast of longevity and all other exogenous variables.

¹⁴This gives identical point estimates for higher education as an OLS regression of life expectancy averaged over 1995-2004, on education averaged over 1961-80 and on the 1973 forecast of end-of-period longevity.

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

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

with $\bar{\pi} \in (0,1)$, z > 0 and $m \ge 0$. 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. 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):

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

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

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

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

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.

 $^{^{15}}$ 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.

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} . ¹⁶

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.¹⁷

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.¹⁸

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.¹⁹

Remark 7 Education is the only form of investment and affects permanent income.²⁰ 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

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

¹⁶This 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.

¹⁷Only 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.

¹⁸It 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.

¹⁹If we adopted the assumption by which each individual internalized the effect of her educational choice on 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).

²⁰Previous 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.

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

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

$$\tag{5}$$

$$pc_1^{-\sigma} = \rho \mu z m^{\mu - 1} \frac{1}{1 - \sigma} \left\{ \left[w_{t+1} \left(1 + h^{\eta} \right) - \kappa b \right]^{\theta} b^{1 - \theta} \right\}^{1 - \sigma}$$
 (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. 21

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 obtain²²

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

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

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

$$\tag{10}$$

$$pc_1^{-\sigma} = \rho \frac{\mu}{1-\sigma} azm^{\mu-1} b^{1-\sigma}$$
 (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

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

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

22 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.

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

$$c_{1} = \left[\frac{1}{\rho az} \left(\frac{1-\sigma}{\mu} p \right)^{\mu} \left(\frac{kw_{t}}{\eta} \right)^{1-\mu} \left(\frac{1-\theta}{\kappa} w_{t+1} \right)^{-(1-\sigma)} h^{(1-\eta)(1-\mu)} \left(1 + h^{\eta} \right)^{\sigma-\mu} \right]^{\frac{1}{\sigma}}$$
(13)

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

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

We adopt the following

Parametric assumption 3 $\sigma \ge \mu$, 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.

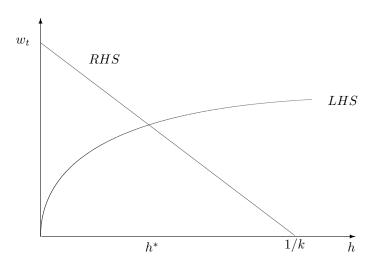


Figure 1: The equilibrium level of education: a representation of condition (14)

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

Table 1: Comparative statics for individual behavior[‡]

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

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

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).²³ For 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.²⁴ Second, more education raises expected future income increasing the agent's incentive to invest 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.

^{‡:} 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

²³We 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 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.

²⁴This 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).

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-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 $\forall t^{25}$

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

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

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

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

Parametric assumption 4 $\alpha \leq (1 - \eta)(1 - \mu)$ and $\beta \leq \sigma - \mu$, 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

 $^{^{25}}$ ¿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.

(12) for m, we have the equation defining the equilibrium level of h:

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

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

	higher	health	basic	life
	education	investment	education	expectancy
	h	m	b	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	+	+	+	+

Table 2: Comparative statics with externalities[‡]

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

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

income, w.²⁶ 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 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 expected future income, increasing the stake at surviving, thus fostering health investment, ultimately resulting in improved longevity (role of μ). Similarly, since more education implies higher permanent income, it implies greater parental investment in child's basic education which improves

^{‡ :} 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

 $^{^{-26}}$ ¿From eq. (18), life expectancy is also increasing in second period income $w(1+h^{\eta})$, which however is endogenous.

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 Empirical analysis

In this section, we present evidence of a significant role played by variables measuring higher education in explaining variation in life expectancy at birth across countries. As argued in the introduction, this finding can be interpreted as evidence of external effects of education on public health. We first introduce the data employed, then explain the methodology adopted, and finally present and interpret the results.

3.1 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, average longevity, is measured by life expectancy at birth. This measure indicates the number of years a newborn infant would live if prevailing patterns of mortality at the time of its birth were to stay the same throughout its life. The World Development Indicators (WDI) 2005 database provides data on life expectancy at birth, physicians per thousand people, sanitation²⁷, female literacy rates²⁸, higher education enrollment rates, GDP per capita in PPP dollars and real health expenditure per person²⁹. We use a measure of the incidence of AIDS, defined as number of cases per thousand persons, from Papageorgiou and Stoytcheva (2007), in an effort to control for the adverse effects of the AIDS epidemic. The geographic concentration of AIDS also means that this a bio-geographic variable that captures certain (adverse) aspects of geography. We also considered the use of geographic data on each country's latitude (proximity

²⁷Access to improved sanitation facilities refers to the percentage of the population with at least adequate access to excreta disposal facilities that can effectively prevent human, animal, and insect contact with excreta. Improved facilities range from simple but protected pit latrines to flush toilets with a sewerage connection.

²⁸The female illiteracy rate is the percentage of females ages 15-24 who cannot, with understanding, read and write a short simple statement on their everyday life. We convert these numbers to literacy rates by subtracting illiteracy percentages from 100.

²⁹Total 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, excluding 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.

to the tropics) from Hall and Jones (1999). Since this turned out to be insignificant leaving other estimates unchanged, we do not present estimates using this variable in the tables but discuss estimates utilizing this briefly in the results section. The same is true for total calories intake.³⁰

In general, we prefer to account for significant theoretically-implied control variables posing the strictest possible robustness check for our main hypothesis pertaining to the education externality on health. It is for this same reason, that we consider a micro-based measure of health spending instead of the aggregate measure of real health expenditure per person from WDI which turned out to have a statistically indistinguishable from zero impact on longevity. Real pharmaceutical imports per capita are constructed utilizing product-level imports and likely proxy for private health spending better than the aggregate WDI measure described above. Specifically, the construction of this micro-based measure utilizes pharmaceutical expenditures of each country in the sample for imports from ten "medical frontier" countries, based on data derived from the OECD International Trade by Commodity Statistics (ITCS) database, also used in Papageorgiou, Savvides, and Zachariadis (2007).

We use higher educational attainment rates and average years of higher education from BL for comparability to the vast body of previous work that has studied the relation between educational attainment and economic growth using these same data. These data is available in 5-year steps from 1960 to 2000 for 107 countries. As noted by LGSS (p. 22), average years of education are derived from attainment rates by applying additional problematic assumptions so we do not focus on these. We focus on higher educational attainment in levels averaged over long time periods (1961-95 and 1961-80), and in log differences between 1961 and 1995. One issue that arises is the measurement error that has been shown to characterize the BL data when considering changes of education rather than levels, and when considering time horizons of ten or less years long. The use of long-run average levels in our application ensures that the measurement error to signal ratio is greatly reduced, as shown in previous work by Topel (1999), Krueger and Lindahl (2001) and CS. Krueger and Lindahl (2001) compute reliability ratios suggesting that the BL education data in levels "have considerable signal" which is greater than when using differences over time, and that considering longer horizons improves reliability ratios considerably even in the case of differenced education data. Similarly, CS show (p. 59) that "if we were to use a longer time scale ... the bias would disappear." Moreover, comparing their data to BL, they state (p. 61) that "over the

 $^{^{30}}$ Total daily calorie intake per person is the sum of calorie intake from vegetable and animal sources, constructed using data on daily calorie intake from the *Food Balance Sheets* database of the Food and Agriculture Organization.

³¹Pharmaceutical imports in part capture the embodied channel of technology diffusion to importing countries. However, to the extent that the price set by the exporter for these imports reflects their full value to the importer, there should not be additional gains in terms of technological spillovers to the importing country as a result of these imports, over and above what individuals have paid for these. As such, spending on pharmaceutical imports can be treated as a private health input into the production function of health in importing countries.



Figure 2: Longevity and education (C&S dataset)

whole 1960–1980 period, both series provide a similar estimate of the total change in schooling." Finally, they report (in their Table 6) a correlation between their data and the BL education data in levels that is about 90 percent across 84 countries found in both datasets. Comparing the BL data in levels to the high quality De la Fuente and Domenech (2006) data available for 21 OECD countries, they report a "fairly high" correlation equal to 91 percent. In light of all the above, we opt to focus on estimates utilizing higher education levels averaged over long periods.

We also utilize the CS higher educational attainment data. These are available every ten years from 1960 to 2000 for 95 countries. This dataset is based on information from the OECD database on educational attainment, from UNESCO surveys and national censuses. The authors avoid using censuses based on different classification systems of education, making the series consistent for each country over time. They also account for differences in mortality rates across but not within age groups. Another potential problem, shared with other cross-country education datasets, relates to the assumption that immigrants have the same educational level as the host country population. Once again, we consider time averages over the same long periods (1961-95 and 1961-80) as with the BL data. Moreover, we consider only countries that exist in the BL dataset, subject also to availability of the other explanatory variables. Zimbabwe (shown as "x" in Figure 2) appears to be an obvious outlier in the relation of CS (log) higher educational attainment rates averaged over 1961-95 with (log) life expectancy averaged over 1995-2004. Moreover, average higher educational attainment for Zimbabwe over the period differs greatly relative to the other two datasets we consider: CS report an average value of 6.059 percent, BL 1.475 percent, and LGSS a value of 1.227 percent. For these reasons, Zimbabwe is excluded in the analysis that follows when using the CS data, leaving us with a sample of 64 countries.

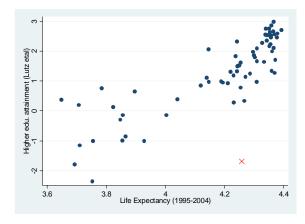


Figure 3: Longevity and education (Lutz et al. dataset)

In addition to BL and CS education data, we use educational attainment rates put together by LGSS. These data are constructed applying demographic methods that allow for a backward projection starting with 2000 as the base year and going back to 1970 in 5-year steps. Educational attainment for the base year is constructed using national censuses obtained from UNESCO and Demographic and Health Surveys. The methodology utilized to construct these allows for differential mortality rates across educational groups. Moreover, this dataset is available with age detail at each level of educational attainment, allowing us to consider the impact of tertiary education decisions taken by people at a young age. We consider age groups 20-24 and 20-29 which we view as the most relevant for tertiary education decisions, and also use an average of all age groups in the population comparable to the average higher educational attainment rate used in the case of the BL and CS data. Higher educational attainment for ages 20-24 is averaged over 1970 to 2000 to capture cohorts making educational decisions during this period. Attainment rates for ages 20-29 are averaged over 1970-2000 for estimates reported in Table 4, and for 1970-90 in the case of Tables 5 and 6 for consistency with individuals assumed to be making educational decisions and longevity forecasts around the mid seventies, a point we elaborate on later. The measure of average higher educational attainment across ages is averaged over comparable time periods as the BL and CS data (i.e. over 1970-2000 and 1970-80.) The correlation between this measure and the BL higher educational attainment rates is 85 percent (79.4 percent with the CS measure) for the common sample of 62 countries. Jordan (shown as "x" in Figure 3) appears to be an obvious outlier in the relation of the average LGSS (log) higher educational attainment rates with (log) life expectancy. Moreover, average higher educational attainment for Jordan over the period considered differs greatly relative to the other two datasets, with LGSS reporting an average value of

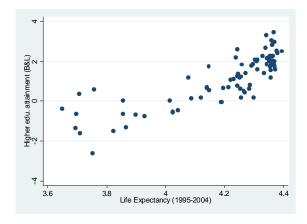


Figure 4: Longevity and education (B&L dataset)

0.185 percent contrasted with average values of 6.35 percent for BL and 16.6 percent for CS. For these reasons, Jordan is excluded in the analysis that follows when using the LGSS data, so that we are left with a sample of 61 countries.

Finally, we use 1973 UN forecasts of life expectancy for the period 1995 to 2000. These were obtained from Table 51 in Annex IV of United Nations (1977, p. 138-147.)³² These forecasts include information about a host of otherwise omitted variables that would have been excluded from our regression specifications. Thus, this measure assists us in addressing the endogeneity and omitted variables problems that haunt any such exercise.

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 per capita income, pharmaceutical imports, health spending, physicians availability and sanitation) and to education through two distinct channels (measured by basic female literacy and higher education rates).

In the dataset, end of period average life expectancy has a correlation of 84 percent with BL average years of higher education, 88 percent with higher education enrollment rates, 84 percent with CS higher educational attainment rates (see Figure 2), 81 percent with LGSS higher educational attainment rates (see Figure 3), and 83 percent with BL higher educational attainment rates (see Figure 4). Its correlation with female literacy and with physicians is also very strong at 70 and 88 percent respectively. Moreover, its correlation with sanitation is 69 percent, 65 percent with pharmaceutical imports, 77 percent with aggregate health expenditure, and minus 29 percent

³²Table titled "Estimates and projections of life expectancies at birth for both sexes, males and females, by region and country, 1950-2000, medium variant." This is the earliest ever published document containing country-specific estimates of life expectancy for the period 1995-2000.

with aids prevalence. All correlations are statistically significant at the one percent level, except the latter which is 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 aggregate health spending (93.5 percent), ³³ physicians availability (89 percent), higher education enrollment (83 percent), pharmaceutical imports (80 percent), BL and CS higher educational attainment rates (81 percent), BL average years of higher education (79 percent), and LGSS higher educational attainment rates (75 percent). Moreover, several of these inputs are highly correlated with each other raising a warning flag regarding potential collinearity problems in the regressions that follow. Notably, the correlation of BL and CS higher educational attainment rates with physicians is 86 percent (76 percent for LGSS.) We thus consider specifications both with and without the apparently highly collinear physicians-availability variable.

Finally, the 1973 UN forecast of longevity for 1995-2000 has a correlation of 85 percent with end-of-period life expectancy³⁴, suggesting that it captures a whole spectrum of factors related to future longevity outcomes. The high correlation between forecasts and outcomes implies that the former are highly correlated with our included explanatory variables, in addition to a great number of unknown omitted determinants of longevity. The forecast is highly correlated with the 1961-80 (1961-95) average of BL higher educational attainment rates at 80 (81) percent, higher education enrollment rates at 81 (83) percent, and BL average years of higher education at 76 (80) percent. The correlation of the forecast with CS higher educational attainment rates is 80 percent irrespective of the period over which we average the data. The forecast has a correlation of 75 percent with LGSS tertiary educational attainment rates over the whole period of availability 1970 to 2000, and 72 percent when the latter is averaged over 1970-80. The physicians measure averaged over 1961-80 or over 1961-95 has a correlation with the forecast of 89 percent. Finally, the correlation of the forecast with real income averaged over 1961-80 is 83 percent and its correlations with female literacy, sanitation, and pharmaceutical imports are 80, 77, and 71 percent respectively.

3.2 Methodology

We were able to put together the above series for 71 countries, shown in Table 3. The great majority of these are not available frequently over time and in some cases are exceedingly sparse in the time dimension. Since the cross-sectional dimension of the dataset is more complete and because of the inherent long-run nature of the relation under study, we opted for exploring the cross-sectional

³³This might be why health spending has insignificant longevity impact in estimations (not reported in the Tables.) Excluding income (along with physicians) renders aggregate health spending estimates positive and significant.

³⁴The pattern of unpredicted changes in life expectancy varies across countries, with forecasts turning out to be too optimistic for countries at the bottom of the life expectancy distribution but too pessimistic for countries toward the top of this distribution.

dimension of the data. Averaging over long periods helps reduce measurement error problems that haunt any exercise considering a sample of countries at different levels of development. 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. We also use lags of higher education averaged over the earlier period 1961-80, more closely corresponding to the period of availability of the UN longevity forecasts (1973), to obtain the results shown in Tables 5 and 6.

As pertaining to the issue of identifying the externality, there are at least two reasons to interpret a direct role of tertiary education on longevity as evidence of external effects of education. The first one rests on the theoretical argument that the two direct effects of education play conceptually distinct roles, with one operating as a rival input benefitting only household members and exhibiting diminishing returns so that basic literacy captures it, and the other depending on the capacity of the society and economy to absorb and use new medical technology and health knowledge. Higher rather than basic education should better proxy for this capacity. Thus, controlling for basic literacy, any effect from tertiary education would be consistent with the existence of an external role for education. A second argument for interpreting the impact of higher education on longevity as evidence of externalities is based on knowledge of the data. Only a very small fraction of the population in the sample acquired higher education. Since life expectancy is computed as an average across educational groups, the relatively large estimated elasticity of life expectancy with respect to tertiary education would imply unrealistically large gains in life years for those who choose to acquire tertiary education, if they were the only ones benefitting from their education. To make sense of the estimated elasticities, we need to allow for the possibility that others benefit as well as a result of the educational choices of the former group of individuals.

Our theoretical analysis points to potential endogeneity problems: While we expect 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. However, treating each explanatory variable as potentially endogenous and the remaining as exogenous, we fail to reject the null that any of these is exogenous at conventional levels of significance for each of the measures of higher education³⁵, female literacy, physicians per capita, pharmaceuticals per capita, initial income, sanitation, and AIDS cases per thousand population.³⁶ Moreover, treating all variables together as potentially endogenous, we fail to reject the null hypothesis that these are exogenous at the ten percent level of significance. This suggests it might be reasonable to estimate the empirical model without having to worry as much about endogeneity. However, since there is a strong

³⁵Except for enrollment rates at the ten percent significance level.

³⁶To implement these tests, we use the lag of each of the first four variables as a potential instrument for the average of that variable over the period. For the last three variables, we use social infrastructure, percentage of the population with access to clean water, and proximity to the tropics, as potential instruments.

theoretical case for the endogeneity of higher education with respect to life expectancy outcomes, we pay special attention to resolving this potential endogeneity problem.

We begin to assess the link between health inputs and life expectancy using average life expectancy for 1995-2004 as explained by the average value of the explanatory variables over 1961-95 (1970-2000 in the case of the LGSS higher educational attainment variables.) We report results from estimating this model using OLS in Table 4 and discuss them in the next subsection. This approach is an attempt to directly estimate equation (15). 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.

We then consider OLS estimation for log changes of the variables. Here, the growth rate of life expectancy between 1961 to 2004 is explained by growth rates of the explanatory variables between 1961 and 1995 (between 1970 and 2000 in the case of the LGSS higher education variables), with results presented in Table 5 and discussed in the second subsection below. All variables other than the log of the initial (1961) level of real income per capita are in log changes. This specification allows us to eliminate fixed country effects and serves as a robustness check for the relative importance of the explanatory variables.

Finally, towards the goal of addressing potential endogeneity problems and establishing some evidence of temporal causation, we consider the unpredicted component of life expectancy as the difference between actual life expectancy averaged over 1995-2004 and the 1973 forecast of life expectancy around 2000. Our test for a role of higher education as a determinant of longevity consists of an attempt to explain the unpredicted part of life expectancy at the end of the period, using the component of higher education over 1961-80 not explained by the 1973 forecast of life expectancy around 2000. We implement a procedure that provides the same point estimates for higher education as the one just described, but which is more straightforward and provides us directly with the correct standard errors. Moreover, it provides a direct test of the relation between the initial forecast and actual life expectancy at the end of the period. Specifically, we include the 1973 forecast of longevity around 2000 as an explanatory variable along with higher education averaged over 1961-80³⁷ and the remaining explanatory variables, in order to explain longevity averaged over the end of the period from 1995 to 2004. Results from this specification are reported in Tables 5 and 6 and discussed in the third subsection below.

In all cases, we consider specifications without and with the physicians measure. Physicians availability is a significant and robust determinant of longevity and is highly collinear with higher education. Thus, it is useful to verify how the explanatory power of the latter is affected by

³⁷For LGSS attainment rates across ages we consider the period 1970-80. For the LGSS attainment rates of individuals in the 20-29 age group, we consider 1970-90 for reasons explained in the third subsection below.

the presence of physicians per capita as a competing determinant of life expectancy. Moreover, there are reasons to think that physicians play two theoretically distinct roles in promoting health improvements: first, they provide rival services to patients acting as a direct rival input into the health production function; second, they are facilitators of health-related knowledge absorption and dissemination. The implication of the latter fact for our empirical analysis is that, if higher education affects longevity through an external channel as it favors the flow of knowledge in general and of health-related knowledge in particular, then its role should be partially captured by the physicians measure.

For each of the empirical approaches described above, considered in the next three subsections and presented in Tables 3-6, we use five alternative measures of higher education: BL attainment rates, BL years of education, enrollment rates, CS attainment rates, and LGSS attainment rates. For reasons exposed in the previous paragraph, we present in each Table results obtained without (columns 1-5) and with (columns 6-10) physicians availability. In column (11), we consider a measure of age-specific educational attainment rates for ages 20-24, and in column (12) for ages 20-29. In the next section, we first present results obtained using variables in levels and then their changes. We then introduce a methodology that allows us to address the endogeneity issue between education and longevity and present the relevant empirical results.

3.3 Results

Estimates for specifications in levels

In Table 4, we consider estimates from OLS regressions of end-period (1995-2004) average life expectancy on higher education and female literacy averaged over 1961-95, real income per capita averaged over the initial period from 1961 to 1980, and sanitation, pharmaceutical spending per capita, and AIDS per hundred thousand population all averaged over 1961-95 subject to data availability.³⁸ As the LGSS higher education data are available only since 1970, we use the longest possible time average (1970-2000) to alleviate measurement error problems and for comparability to the 35-years-long averages used in the case of the BL and CS data.

In columns (1)-(5), we report results without the physicians measure. In this case, irrespective of the measure used, higher education consistently has positive and strongly significant impact on life expectancy which is no less significant than that for female literacy. The elasticity of life expectancy with respect to BL higher educational attainment is 4.8 percent, with respect to CS higher educational attainment this is 5.9 percent, and 6.6 percent with respect to LGSS higher

³⁸For instance AIDS appears only since 1979.

educational attainment.³⁹ With most countries in our sample having small average values of higher educational attainment⁴⁰, these estimated effects suggest that if higher education operated only through the internal channel in improving life expectancy, the relatively small proportion of individuals acquiring higher education would have to experience unrealistically large improvements for the population average to be affected by as much as we find in the aggregate data. Based on the estimated elasticity of 4.8 percent, this would amount to an implausible 74 years of extra life⁴¹ for those acquiring tertiary education if they were the only ones benefitting from their actions. Thus, 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 female literacy ranges from 11.7 percent in column (4) down to a statistically insignificant 5.2 percent in column (5). Real per capita spending on imported medicine has a positive and significant impact close to 2 percent in columns (1)-(3), but is statistically insignificant in columns (4) and (5). On the other hand, sanitation has positive impact but never estimated to be statistically significant. Finally, the incidence of AIDS has a negative and strongly significant impact on life expectancy throughout.

We also take into account the fact that income can be a major determinant of health, by including initial period time-averages of real income per capita in the regression specifications presented in Table 4. 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. Moreover, real income per capita is likely related to the provision of rival semi-public health services, such as access to sewage systems for instance. Controlling for income helps isolate the part of the effect of each input that is unrelated to income. For the specifications reported in columns (1)-(5) of Table 4, initial income has a positive and significant impact on longevity ranging from 3.2 percent in column (4) to 6.6 percent in column (5). Thus, income comes in as a positive and significant determinant of life expectancy consistent with its role as facilitator of rival health-related inputs purchases.

In columns (6) to (12) of Table 4, we report results for specifications incorporating physicians availability in addition to the education variables, sanitation, real per capita spending on imported

³⁹The sample for CS and LGSS is constrained to countries that exist in the BL dataset. Relaxing this constraint, Madagascar, Morocco and Nigeria are added giving us a total of 67 observations for CS and 64 observations for LGSS. Using these samples, the estimates corresponding to those in columns (4) and (5) for the CS and LGSS higher educational attainment measures (not shown in Table 4) are now 6.6 and 6 percent respectively and remain strongly significant.

⁴⁰Nearly 70 percent of countries in our sample had attainment rates less than 0.02 in 1960, and about half had rates less than 0.10 even by 1995. The median across countries was 0.0218 for 1961-80 and 0.0414 for 1961-95.

⁴¹We consider a hypothetical increase of tertiary education attainment of 36.7 percent, from the cross-country median for 1961-95 which is 0.0414 (for Portugal) to 0.0599 which is the cross-country mean over the period.

⁴²Thus, including income conforms with Fogel's (1994) emphasis on nutrition as a determinant of health. We note that including total calories along with income, total calories never have a positive significant impact on longevity.

medicine, AIDS per thousand population, and real initial 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 this measure should diminish the impact otherwise captured by higher education measures. Indeed this is the case. Introducing physicians dampens the impact of higher education on life expectancy, consistent with the fact these two variables are highly collinear. Still, it remains positive and significant except when using enrollment rates in column (8). The impact of higher education is estimated to range from 1.7 percent for the BL years of education in column (7) to 5.1 percent in the last two columns, where we present estimates based on higher educational attainment rates specific to the ages 20-29 and 20-24 respectively.⁴³ This age-specific dataset is available for a somewhat different time period than the education data used so far, but has the advantage that we can consider educational decisions of individuals with the typical age profile for higher education. We note that the estimated effect of age-specific tertiary educational attainment on life expectancy is higher than the effect of overall educational attainment reported in columns (6)-(10).⁴⁴

The above estimated coefficients should be viewed as a lower bound for the importance of the knowledge externality we are focusing on in this paper. Since we are attributing all of the impact of physicians to its direct rival role in the health production function, we are understating the overall effect of health-related knowledge. Physicians availability has positive and strongly significant impact on life expectancy that is stable around 7 percent in columns (6)-(12) of Table 4. The estimated life expectancy elasticity of female literacy remains high at around 9 percent and statistically significant in columns (6)-(9), but becomes insignificant in columns (10)-(12) when it enters the regression along with the LGSS higher educational attainment rates. Sanitation and spending on pharmaceuticals retain a positive impact on life expectancy which is nevertheless statistically insignificant in all cases. The estimated impact of AIDS remains negative and strongly significant. Finally, the impact of income per capita is now estimated to be indistinguishable from zero. Collinearity might be behind the finding of an insignificant impact of income on longevity once we control for health inputs via which it impacts upon health. A clue to this effect is provided by the 89 percent correlation between income and physicians.

Estimates for changes in variables specification

We now consider differences of the variables instead of their levels. Doing so, eliminates fixed country effects that we would otherwise be unable to handle in a cross-sectional framework, and

⁴³Using a larger sample for CS and LGSS (not constrained to countries that exist in the BL dataset) estimates corresponding to those in columns 9-12 for the CS and the three LGSS higher education measures are 4.1, 3.7, 4.5, and 4.7 percent respectively, and strongly significant in all cases.

⁴⁴The estimated impact in the respective specifications without physicians (not shown in the tables) is 6.6 and 6.4 percent and strongly significant for higher educational attainment for ages 20-29 and 20-24 respectively.

serves as a robustness check for our main finding regarding the importance of higher education for life expectancy. It should be noted that this is a particularly strict robustness check for the relation between education and longevity since differenced education data have been shown to suffer from relatively low signal to measurement error ratios biasing education coefficient estimates downwards. As noted by Topel (1999), differencing the education data magnifies the effects of measurement error and the associated downward bias in education coefficient estimates. CS (p.62) compare the BL education data in differences to the high quality De la Fuente and Domenech (2006) education data that is available for 21 OECD countries, and find that the correlation between these series falls to 10 percent for first differences, as compared to 91 percent for the levels. However, Topel (1999) points out that the estimated impact of changes in education rises as longer intervals of time are considered and that this may reflect the lower impact of measurement error for education data at lower frequencies. Similarly, Krueger and Lindahl (2001) consider the measurement error issue for changes in education and state (p.1119) that "over longer periods, true education levels are more likely to change, increasing the signal relative to the noise in measured changes." Consistent with the latter finding, CS (p.63) show that "in the case of the 30-year change over the 1960-90 period the reliability ratios" between their recently constructed education data and the BL data "are not statistically different."

Once again, we utilize the BL data for comparability to a vast body of existing work, but also use changes in higher educational attainment rates based on the more recently constructed data from CS and LGSS. For reasons exposed in the above paragraph, we consider long changes in education over the 35-year period between 1961 to 1995. For the LGSS educational attainment data, we consider the maximum possible (31-year long) change between 1970 and 2000. We report estimates from this exercise in Table 5 where we seek to explain changes in life expectancy between 1961 and 2004. We now include changes in real income per capita over the period as a proxy of the growth rate of rival health inputs. Including this reasonably good measure of purchasing power becomes necessary as we no longer include changes in sanitation and health spending. 45

The growth rate of higher educational attainment rates has positive and significant impact on longevity growth in most specifications we consider. This impact is as high as 6.5 percent for CS attainment rates in column (4), and 6 percent in specifications reported in columns (1) and (11) of Table 4 for BL attainment rates and for LGSS attainment rates for ages 20-29.⁴⁶ Instead, the

⁴⁵Changes in the share of population with access to sanitation cannot be computed because of limited data availability over time. Changes in pharmaceutical imports are not included since the estimated effect of pharmaceuticals is always statistically indistinguishable from zero and its inclusion leaves all other estimates virtually unchanged. Limited availability of pharmaceutical data over time necessitates computing their growth rate for the period as the difference between the end-of-period average and the initial fifteen year average of the period under study, which might lead to mismeasurement and likely biases estimates downwards.

 $^{^{46}}$ Excluding physicians, the coefficient estimates for ages 20-29 and 20-24 are 5.1 and 4.4 percent respectively, and

LGSS attainment rate measure across age groups is estimated to have an insignificant effect on longevity growth as shown in columns (5) and (10), and the same is true for higher education enrollment rates as shown in columns (3) and (8).

The growth rate of female literacy has an estimated longevity impact as high as 9 percent, which is typically significant as long as a measure of physicians is excluded from the specification. The growth rate of physicians has positive and strongly significant impact that is as high as 9 percent in columns (9) and (12), confirming the importance of the per capita number of physicians in determining health outcomes suggested by the estimation in levels earlier. The estimated impact of the rate of growth of AIDS is negative as we would expect, and is usually statistically significant. The growth rate of per capita income has a positive significant impact at about 7.5 percent in columns (1), (2) and (5), but is insignificant elsewhere. Finally, initial income has a negative but usually insignificant impact on changes in life expectancy between 1961 and 2004.

Explaining the unexpected component of longevity

One important concern is the extent to which life expectancy could be forecasted by individuals and this forecast then endogenized in their educational choices. We allow for individuals able to make forecasts of future longevity as accurate as those undertaken by UN demographers. We use forecasts of longevity regarding the period 1995-2000, as assessed by UN demographers in 1973.

We consider the component of education that cannot be explained by these forecasts⁴⁷, to explain the deviations of actual life expectancy relative to the 1973 forecasts. These deviations represent the unpredicted component of life expectancy around 2000, presumed not to have an impact on educational decisions made in the period from 1961 to 1980. Therefore, if these deviations are explained by exogenous variation in 1961-80 education variables, we can interpret this as evidence of causality from education on longevity.

We implement a simple procedure that provides identical point estimates for higher education to those obtained by using deviations of actual from forecasted longevity. Specifically, we apply OLS estimation of actual life expectancy at the end of the period on lagged higher education (averaged over 1961-80), including the forecast of life expectancy on the right-hand-side along with the other explanatory variables described previously.⁴⁸ In this case, the included forecast has an omitted variables interpretation where factors taken into account by UN demographers and individuals in 1973 are accounted for in the regression through the use of this proxy.

strongly significant.

⁴⁷This is the residual from a regression of higher education averaged over 1961-80 on the forecast of longevity as of 1973 and on all other exogenous variables.

⁴⁸There is also an IV interpretation of this procedure where the instrument for education is the residual from a first-stage regression of education on the forecast of longevity and all other exogenous variables. By construction, IV estimation produces the same point estimates for higher education as those reported in Table 6.

The above estimation procedure helps identify the impact of higher education on longevity as it addresses the endogeneity problem intrinsic to the relation between education and longevity and the associated omitted variables problem. We report results from this specification in Table 6. This exercise shows that higher education likely has a causal impact on longevity. This impact is statistically significant in all cases but column (9). The estimated elasticity of life expectancy with respect to BL higher educational attainment rates is 3.1 percent in column (1) and 2.3 percent in column (6). This suggests that doubling the attainment rate of higher education would increase life expectancy by as much as three percent. Compared to these, estimated elasticities are somewhat smaller in the case of years of higher education but larger in the case of enrollment rates in higher education. The estimated elasticity impact of CS higher educational attainment is 2.6 percent in column (4) but down to 1.5 percent and insignificant in column (9). The elasticity of the LGSS higher educational attainment rate across ages (averaged over the period 1970-80) is 3 percent in column (5) and 2.2 percent in column (10). Finally, the elasticity of the age-specific LGSS attainment rates is 3.3 percent for ages 20-29 (averaged over the cohorts from 1970 to 1990), and 4 percent for ages 20-24 (averaged over the cohorts from 1970 to 2000) as shown in columns (11) and (12) respectively.⁴⁹

The latter two measures of higher education serve different purposes. Higher education attainment rates for ages 20-29 averaged over 1970-90 measures the stock of human capital most likely to have been influenced by 1973 forecasts of longevity, reflecting tertiary education decisions of cohorts aged 13-22 years old between 1963 and 1983, i.e., about the time of release of the UN forecasts. Thus, for this measure, it is possible for us to use 1973 longevity forecasts to address the endogeneity issue. However, the concept closer to our theoretical one is the contemporaneous stock of human capital. In this respect, the measure of higher education attainment rates for ages 20-24 averaged over 1970-2000 may be preferable since it is similar to higher education attainment rates in 2000 averaged across ages 20-54. Its drawback is that it is more likely to suffer from endogeneity due to its contemporaneous nature in relation to longevity outcomes for 1995 to 2004, and that the 1973 forecasts seem relatively less adequate to solve the endogeneity problem as they

⁴⁹Excluding physicians, the estimates corresponding to those in columns (11) and (12) of Table 6 are 4.1 and 4.3 percent respectively (not shown in the table) and strongly significant.

⁵⁰We average attainment rates for ages 20-29 over 1970-90 to capture tertiary education decisions that were most likely to have been influenced by the 1973 forecast of longevity. We assume that the information in this forecast is relevant for individuals that are of the right age to make forward looking educational decisions within a decade prior to or after 1973. Individuals on the lower bound of the age cohort being considered, i.e., those aged 20-29 years old in 1990, were 13-22 years old in 1983. Individuals on the upper bound of the age cohort being considered, i.e., those aged 20-29 years of age in 1970, were 13-22 years of age in 1963. The remaining individuals we consider fall in the 20-29 age group in 1975, 1980, or 1985 and are even more likely to be making decisions that utilize the information in the 1973 forecast. We note that averaging these cohorts over time has the attribute of placing more weight on individuals likely to be making decisions around the middle of the period, and less weight on those making decisions at the beginning and end of the period being considered. This is desirable since the information in the 1973 longevity forecast is more relevant for the former group of individuals.

become outdated over time and especially so by the end of the period. 51

To illustrate the importance of the above estimates, we note that a 3.1 (2.3) percent estimated elasticity of life expectancy with respect to higher educational attainment, implies that those acquiring tertiary education would gain an additional 73 (54) years of life if the internal channel was the only one at work.⁵² Since this gain appears unrealistic, we interpret our estimated results as evidence for a causal external effect of higher education on average life expectancy.

Whatever the variable used to measure higher education, its estimated effect on longevity is always positive and, in all but one case, statistically significant. Comparing these point estimates of the elasticity of life expectancy with respect to higher education with those obtained in Table 4, they appear to be somewhat smaller in most cases. It should be emphasized, however, that this approach likely minimizes any effect of higher education on longevity. For example, in the possible scenario that the average individual cannot predict future longevity as accurately as complex UN demographic models do, then the problem of reverse causality from expected longevity to education facing our estimation exercise would be less severe than we assume it to be. In this case, the true elasticity estimates of longevity with respect to higher education would lie between the OLS estimates in Table 4 and those in Table 6.

Overall, we find that higher education matters significantly for longevity and is more robust than female literacy, sanitation, spending on medicine, and per capita income. Per capita spending on imported medicine is usually significant for longevity, with an estimated elasticity typically around 1.5 percent. AIDS has a negative precisely estimated impact on longevity close to 3 percent. Female literacy, real income per capita, and sanitation are estimated to have insignificant impact on longevity once the forecast variable is included in the specification. We note that the impact of female literacy is typically positive and significant as long as we do not include the forecast variable, and the same holds for real income per capita as long as we also exclude physicians, as

⁵¹On the other hand, using more recent forecasts of longevity for year 2000 made in the 1990's would not leave much of an unpredicted component to be explained.

⁵²Based on BL data, the cross-country median tertiary educational attainment rate averaged over 1961-80 is 0.0218 and the associated life expectancy at birth over 1995-2004 is 70.695 years. We ask how much higher would life expectancy be if tertiary educational attainment was instead 0.0407 (the cross-country mean over 1961-80), representing a 60.38 percentage increase. This would imply an increase in the log of life expectancy equal to β ln(1.6038), where β is the coefficient from the regression of longevity on tertiary education attainment. Using β =0.031, implies a gain of 1.379 years so that average life expectancy increases to 72.07 years. If higher education affects life expectancy exclusively through the internal channel, then it improves only the longevity of the 1.89 percent of the population who acquired higher education. For the rest, longevity should not change. Recalling that life expectancy is an average over the whole population, we compute the increase in longevity (Δ long) that the fraction of the population concerned by the exercise should experience to be consistent with the estimated improvement in average longevity, given by: $1.379 = 0.0189 \times \Delta \log + 0.9811 \times 0$, so that $\Delta \log = 73.14$ extra life years. Those who acquire access to tertiary education would experience an implausible gain of 73 years. This is down to 54 years for the estimate of higher education in column (6) of Table 6. In either case, the elasticity of life expectancy with respect to higher education cannot be explained exclusively on the basis of an internal channel of transmission. Access to higher education for 1.89 percent of the population is associated with external benefits in terms of improved longevity for the remaining 98.11 percent.

can be seen by a simple comparison with Table 4. Physicians enters as a positive and significant determinant of longevity with estimated elasticities around 4 percent, except in the case of column (8). Finally, the forecast variable has a powerful and strongly significant impact on end-of-period longevity, suggesting that it is a rather good predictor of future longevity and that it captures a number of otherwise omitted demographic variables that were present in the mid seventies.

Next, we turn our attention to robustness checks. First, we consider the inclusion of additional variables like proximity to the tropics and total calorie intake for the specifications reported in Table 6. Adding proximity to the tropics and calorie intake one at a time or both together (not shown in the tables) leaves the point estimates for the different measures of higher education pretty much the same, while the two new explanatory variables are typically indistinguishable from zero. For example, adding proximity to the tropics gives strongly significant elasticity estimates of 3.1 and 2.3 percent for the BL measure of higher educational attainment. These estimates are identical to those currently reported in columns (1) and (6) of Table 6. Adding total calories intake alone, provides us with corresponding significant estimates of 3.1 and 1.9 percent, while adding both variables at the same time provides us with corresponding significant elasticity estimates of 3.2 and 2 percent for the BL measure of higher educational attainment.

Second, in Table 7, we consider a common sample that constrains the number of observations to 56 countries for which all measures of higher education and other explanatory variables are available. This acts as a robustness check for possible sample specificity of the findings reported above, and also renders the estimates directly comparable across different measures of higher education. As we can see in the first row of Table 7, the elasticity estimates of longevity with respect to higher education reported there are qualitatively similar to those in the first row of Table 6. The point estimates are very close to those in the previous Table even though we have now excluded a total of twelve countries in going from Table 6 to Table 7. For example, the elasticity estimate for the BL measure of higher educational attainment rates is 3.3 percent in column (1) and 2.6 percent in column (6), about the same and slightly higher than the corresponding estimates in the same columns of Table 6. All of the estimates for higher education are statistically significant except for the marginally insignificant (with p-value of 0.156) estimate of 1.9 percent reported in column (10) for the LGSS measure of higher educational attainment across ages. As before, female literacy, initial income, and public sanitation are not statistically significant. The same goes for spending on pharmaceutical imports. On the other hand, AIDS once again has a negative significant impact throughout, while the measure of physicians consistently has a positive significant impact of about 4 percent, except in the case of column (8). Finally, the forecast variable is an extremely good predictor of future longevity in this restricted group of countries as was the case in the broader

group of countries considered previously.

4 Conclusion

This paper belongs to a strand of the recent literature that addresses the determinants of longevity. As compared to this literature, our contribution is that we provide evidence for the existence of positive external effects of education on longevity.

Our theoretical analysis confirms the presence of complementarities between health and education established in the existing literature (see, for example, Becker, 2007). This complementarity is reinforced at the macroeconomic level when we consider direct externalities of education on health.

In our empirical investigation, we argue that the estimated effect of higher education on longevity provides evidence of an external role of education, if one controls for literacy rates and other common explanatory variables of longevity. In order to identify a causal link of higher education on longevity, this paper makes innovative use of forecasts made by UN demographers in 1973 regarding expected country-specific longevity around 2000. Our approach goes a long way towards taking into account the endogeneity of higher education suggested by the theory. It also allows us to control for a number of otherwise omitted factors affecting future longevity.

The main contribution of this paper is therefore the empirical finding of a causal external effect of education on life expectancy. We find a direct effect of tertiary education on longevity equal to three percent based on our benchmark specification shown in column (1) of Table 6. This result contributes to three lines of research.

First, we suggest that education may play a much more important role in public health than previously thought. As explained at some length in the introduction, education is in fact already considered an important determinant of health but only at the level of the household. While it is possible to find papers mentioning external effects of education on health, a careful reading reveals that these effects concern transmission from parents to their children. What we have established in this paper is therefore that a greater investment in education will improve health not only through this internal channel, but also through the positive external effects that average education exerts. This is a potentially important finding and at the same time calls for further scrutiny given the limitations of cross-country aggregate data.

Second, a large body of empirical work coming out of labor economics and macro growth has studied the possibility of externalities arising from education on income.⁵³ This should not come as a surprise since endogenous growth theory models dating back to Lucas (1988) and Romer

 $^{^{53}}$ This literature is summarized in Krueger and Lindahl (2001) and Psacharopoulos and Patrinos (2002).

(1990) assign a central role to human capital externalities in the process of economic growth. Moreover, "the significant open question ... whether the social returns to human capital investment substantially exceed the private return" (Topel 1999, p. 2973) has been raised by a number of economists going back to Becker (1975) and Heckman and Klenow (1997). At the same time, although the debate is still open, there appears to be accumulating evidence suggesting the absence of such externalities⁵⁴, with the exception of Rauch (1993) and Moretti (2004).⁵⁵ We note that, in assessing social returns, health status should be viewed as a separate component of welfare in addition to income, and show that education externalities for this important component of welfare can be important even when these have not been shown to matter for the other component of welfare typically studied by macroeconomists. Moreover, our findings provide evidence of a form of increasing returns to scale in education as far as longevity is concerned, whereby tertiary education appears to be no less important than basic literacy. This contrasts with evidence on the determinants of economic growth pointing to decreasing returns to scale in education, with basic 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).

Finally, 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. Non-income factors are shown to be important for explaining variations in life expectancy across countries. Policy implications may be important. For instance, our findings suggest that investing in health-related inputs might be crucial 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 authors hold that their "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, since it gives evidence of the role that education plays in influencing the health component of welfare. We propose that human capital externalities are likely to be important determinants of a broader concept of welfare growth that goes beyond the standard concept of economic growth.

⁵⁴Acemoglu and Angrist (2000) find "small social returns ... that are not significantly different from zero" and Cohen and Soto (2007, p. 72) concur stating that "the effect of schooling is close to the typical micro Mincerian return which suggests the absence of externalities to education." Pritchett (2001) documents relatively low returns to education in a growth context and so do Bils and Klenow (2000, p. 1177) who find that "the channel from schooling to growth is too weak to plausibly explain more than one-third of the observed relation between schooling and growth." Finally, Ciccone and Peri (2006, p. 407) find "no evidence of significant average-schooling externalities" for US cities.

⁵⁵Rauch (1993) estimates a large social rate of return suggesting externalities for average education on wages in the context of US cities. Moretti (2004) finds some evidence of spillovers after estimating social returns to higher education at the city level.

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Table 3: 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
Bolivia	63.1	Mexico	74.4
Brazil	69.7	Mozambique	42.6
Cameroon	47.2	Myanmar	59.5
Canada	78.8	Netherlands	78.0
Chile	76.9	New Zealand	78.1
Colombia	71.5	Norway	78.6
Costa Rica	77.6	Pakistan	63.2
Cyprus	78.3	Panama	74.4
Denmark	76.5	Paraguay	70.4
Ecuador	73.4	Peru	69.3
Egypt	70.1	Philippines	69.5
El Salvador	68.7	Portugal	76.2
Finland	77.5	Rwanda	40.14
France	78.8	Singapore	77.9
Ghana	56.8	Spain	78.9
Greece	78.2	Sri Lanka	73.6
Guatemala	66.2	Sudan	55.9
Haiti	50.8	Sweden	79.6
Honduras	67.4	Switzerlannd	79.7
Iceland	78.8	Tanzania	47.2
India	62.8	Thailand	69.7
Indonesia	65.9	Tunisia	72.4
Iran	69.5	Turkey	68.1
Ireland	76.9	Uganda	45.7
Israel	78.3	United Kingdom	77.6
Italy	79.2	Unites States	76.7
Jamaica	71.1	Uruguay	74.3
Japan	80.9	Venezuela	73.0
Jordan	70.7	Zaire	42.8
Kenya	49.2	Zambia	38.4
		Zimbabwe	40.7

 $[\]dagger:$ This is the end of period average life expectancy from 1995 to 2004.

Table 4: Explaining 1995-2004 average life expectancy levels

(12)	ages20-24	Lutz etal	0.051***	(0.015)	0.031	(0.054)	0.010	(0.015)	0.047	(0.043)	0.001	(0.000)	-0.018***	(0.005)	0.074***	(0.014)	3.694***	(0.197)	61	0.873
(11)	ages20-29	Lutz etal	0.051***	(0.016)	0.037	(0.053)	0.006	(0.016)	0.045	(0.043)	0.002	(0.000)	-0.020***	(0.005)	0.074***	(0.014)	3.676***	(0.198)	61	0.869
(10)	attain.	Lutz etal	0.042**	(0.016)	0.054	(0.052)	0.004	(0.016)	0.040	(0.042)	0.004	(0.00)	-0.022***	(0.005)	0.075***	(0.015)	3.645***	(0.205)	61	0.864
(6)	attain.	C&S	0.032*	(0.016)	0.104**	(0.043)	0.000	(0.017)	0.018	(0.031)	0.010	(0.00)	-0.020***	(0.004)	0.063***	(0.014)	3.565***	(0.201)	64	0.868
(8)	enrol.		0.024	(0.020)	0.083*	(0.045)	0.007	(0.017)	0.030	(0.028)	0.011	(0.00)	-0.026***	(0.000)	0.068***	(0.017)	3.507	(0.219)	71	0.861
(7)	years	B&L	0.017*	(0.010)	0.093**	(0.040)	0.010	(0.015)	0.026	(0.028)	0.011	(0.00)	-0.028***	(0.005)	0.074***	(0.014)	3.551***	(0.211)	71	0.860
(9)	attain.	B&L	0.020*	(0.011)	0.090**	(0.041)	0.009	(0.015)	0.028	(0.028)	0.011	(0.00)	-0.028***	(0.005)	0.073***	(0.014)	3.507***	(0.202)	7.1	0.861
(5)	attain.	Lutz etal	0.066***	(0.019)	0.052	(0.055)	0.066***	(0.020)	0.055	(0.042)	0.008	(0.011)	-0.024***	(0.000)			3.002***	(0.250)	61	0.824
(4)	attain.	C&S	0.059***	(0.016)	0.117**	(0.048)	0.032*	(0.018)	0.040	(0.034)	0.015	(0.010)	-0.021***	(0.005)			3.086***	(0.199)	64	0.842
(3)	enrol.		0.065***	(0.017)	0.060	(0.047)	0.038*	(0.020)	0.049	(0.030)	0.017*	(0.010)	-0.026***	(0.006)			3.142***	(0.223)	71	0.838
(2)	years	B&L	0.043***	(0.013)	0.100**	(0.040)	0.061***	(0.017)	0.038	(0.031)	0.019*	(0.010)	-0.032***	(0.005)			3.056***	(0.212)	71	0.827
(1)	attain.	B&L	0.048***	(0.014)	0.091**	(0.041)	0.056***	(0.018)	0.041	(0.031)	0.019*	(0.010)	-0.031***	(0.005)			2.967***	(0.191)	71	0.829
	Higher edu.	measure:	Higher edu.		Fem. Liter.		Initial income		Sanitation		Pharmaceut.		AIDS		Physicians		Constant		Obs	R-squared

Notes: *** p < 0.01, ** p < 0.05, * p < 0.05, * p < 0.05, * p < 0.05, * and the teroskedasticity-consistent finite sample standard errors in parentheses. All variables are in natural logarithms so that reported estimates are elasticities of life expectancy with respect to each explanatory variable. We consider 1995-2004 averages of life expectancy explained by 1961-95 averages for the explanatory variables subject to availability. For CS data, Iceland, Israel, Pakistan, Rwanda, Sri Lanka, and Zaire are missing, and Zimbabwe is excluded so that we are down to 64 countries. For LGSS data, we average over 1970-2000. In this case, we are down to 61 countries as Algeria, Iceland, Israel, Jamaica, Myanmar, Sudan, Tunisia, Venezuela and Zaire are missing, and Jordan is excluded. Initial period income is measured as the 1961-80 average real income per capita for each country.

Table 5: Explaining changes in life expectancy between 1961 and 2004

	(1)	(2)	(3)	(4)	(5)	(9)	(7)	(8)	(6)	(10)	(11)	(12)
Higher edu.	attain.	years	enrol.	attain.	attain.	attain.	years	enrol.	attain.	attain.	ages20-29	ages 20-24
measure:	B&L	B&L		C S \sim S	Lutz etal	$B\&\Gamma$	B&L		C&S	Lutz etal	Lutz etal	Lutz etal
Higher edu.	0.059***	0.054***	0.014	0.065**	0.024	0.048**	0.045	0.007	0.029	0.011	0.059**	0.052***
	(0.016)	(0.014)	(0.028)	(0.031)	(0.024)	(0.018)	(0.015)	(0.026)	(0.028)	(0.023)	(0.022)	(0.014)
Fem. Liter.	0.092**	0.091**	0.082*	0.071*	0.055	0.062*	*090.0	0.048	0.038	0.033	0.030	0.041
	(0.035)	(0.036)	(0.041)	(0.036)	(0.048)	(0.033)	(0.034)	(0.037)	(0.033)	(0.046)	(0.042)	(0.039)
Initial Income	-0.001	-0.001	-0.001	-0.001	-0.001	-0.001	-0.001	-0.001*	-0.001**	-0.001	-0.001	-0.000
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Income growth	0.074**	0.073**	0.078	0.053	0.075*	0.052	0.050	0.049	0.018	0.043	0.020	0.013
	(0.036)	(0.035)	(0.048)	(0.042)	(0.042)	(0.031)	(0.031)	(0.042)	(0.028)	(0.036)	(0.033)	(0.032)
AIDS	-0.002	-0.002	-0.003	-0.004**	-0.004**	-0.002	-0.002	-0.003	-0.003*	-0.004**	-0.003*	-0.003*
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.001)
Physicians						0.057*	0.058*	0.074**	0.091***	0.077**	0.080**	0.088***
						(0.032)	(0.031)	(0.033)	(0.032)	(0.032)	(0.031)	(0.029)
Constant	0.006	0.006	0.009*	0.010	0.010	0.007	0.007	0.010*	0.012**	0.012	0.005	0.002
	(0.005)	(0.005)	(0.002)	(0.007)	(0.008)	(0.000)	(0.006)	(0.005)	(0.000)	(0.008)	(0.007)	(0.007)
Opss	89	89	99	61	58	99	99	64	09	22	22	22
R-squared	0.316	0.321	0.189	0.328	0.193	0.360	0.367	0.277	0.434	0.284	0.345	0.408

reported estimates are elasticities of life expectancy growth with respect to each explanatory variable. We consider the growth rate of life expectancy between 1961-2004 explained by growth rates of the explanatory variables between 1961-1995. All variables other than the log of initial (1961) real income per capita are in log changes. Our sample consists of 68 countries since Bangladesh, Cameroon, Egypt, Malawi, Mozambique, Singapore, and Uganda are missing relative to the sample for Table 4, but Belgium, China, Germany, and Senegal can now be added as sanitation and pharmaceutical imports are no longer included. For CS data, seven more countries are missing. Sir Lanka, and Zaire, leaving us with 61 countries. For LGSS, we consider the growth rate over 1970-2000. In this case, we are down to 58 countries are missing: Algeria, Iceland, Israel, Janaica, Myammar, Senegal, Sudan, Tunisia, Venezuela, and Zaire. The physicians growth rate is missing for Ghana and Iceland as the level of physicians is unavailable for the early part of the period, reducing the sample for the BL data to 66 and for the CS and LGSS data to 60 and 57 observations respectively. Notes: *** p < 0.01, ** p < 0.05, * p < 0.1. Heteroskedasticity-consistent finite sample standard errors in parentheses. All variables are in natural logarithms so that the

Table 6: Explaining 1995-2004 average life expectancy levels with 1973 forecasts of longevity.

	(1)	(3)	(3)	(4)	(2)	(9)	(-)	(8)	(6)	(10)	(11)	(12)
Higher edu.	attain.	years	enrol.	attain.	attain.	attain.	years	enrol.	attain.	attain.	ages20-29	ages20-24
measure:	B&L	B&L		CSS	Lutz etal	B&L	B&L		C&S	Lutz etal	Lutz etal	Lutz etal
Higher edu.	0.031***	0.024**	0.046***	0.026**	0.030**	0.023**	0.017*	0.039**	0.015	0.022*	0.033**	0.040***
	(0.011)	(0.010)	(0.013)	(0.011)	(0.014)	(0.000)	(0.00)	(0.017)	(0.012)	(0.013)	(0.016)	(0.014)
Fem. Liter.	0.029	0.031	0.004	0.076	0.015	0.037	0.039	0.012	0.077	0.033	0.018	0.005
	(0.047)	(0.044)	(0.058)	(0.057)	(0.054)	(0.048)	(0.045)	(0.060)	(0.055)	(0.055)	(0.061)	(0.062)
Initial Income	0.017	0.022	0.004	0.015	0.035*	0.002	0.004	-0.001	0.001	0.011	0.008	0.012
	(0.018)	(0.017)	(0.020)	(0.018)	(0.018)	(0.017)	(0.016)	(0.019)	(0.019)	(0.017)	(0.017)	(0.016)
Sanitation	0.006	0.007	0.020	0.004	0.017	0.006	0.007	0.019	-0.003	0.018	0.022	0.029
	(0.028)	(0.028)	(0.033)	(0.026)	(0.038)	(0.029)	(0.029)	(0.034)	(0.025)	(0.040)	(0.041)	(0.042)
Pharmaceut.	0.018**	0.018**	0.018**	0.015*	0.010	0.015*	0.014*	0.016**	0.012	0.007	0.006	0.004
	(0.008)	(0.008)	(0.007)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.007)	(0.008)	(0.008)	(0.008)
AIDS	-0.028***	-0.029***	-0.026***	-0.026***	-0.026***	-0.027***	-0.028***	-0.026***	-0.024***	-0.025***	-0.023***	-0.020***
	(0.004)	(0.005)	(0.004)	(0.004)	(0.005)	(0.004)	(0.005)	(0.004)	(0.004)	(0.005)	(0.005)	(0.005)
Lon. forecast	0.874***	0.904***	0.820***	0.832***	0.882***	0.707***	0.715***	0.750***	0.649**	0.642***	0.616***	0.551***
	(0.187)	(0.189)	(0.187)	(0.206)	(0.204)	(0.191)	(0.186)	(0.198)	(0.243)	(0.204)	(0.208)	(0.204)
Physicians						0.034**	0.036***	0.016	0.039**	0.042***	0.043***	0.046***
						(0.014)	(0.013)	(0.018)	(0.017)	(0.014)	(0.013)	(0.012)
Constant	0.061	-0.031	0.388	0.088	-0.086	0.882	0.884	0.716	1.031	1.078	1.240	1.524*
	(0.734)	(0.786)	(0.696)	(0.752)	(0.843)	(0.764)	(0.782)	(0.745)	(0.936)	(0.852)	(0.861)	(0.822)
Obs	89	89	89	63	59	89	89	89	63	59	59	59
R-squared	0.865	0.862	0.877	0.881	0.858	0.870	0.868	0.876	0.888	0.866	0.869	0.874

Notes: *** p < 0.01, ** p < 0.05, * p < 0.0.5. Heteroskedasticity-consistent finite sample standard errors in parentheses. All variables are in natural logarithms so that the reported estimates are elasticities of life expectancy with respect to each explanatory variable. We estimate an OLS regression of average life expectancy over 1995-2004 on higher education averaged over the period from 1961 to 1980. The sample is down to 68 countries as forecast data are not available for El Salvador, Rwanda, and Saire. For CS data, the sample is 63 countries. Iceland, Israel, Pakistan, and Sri Lanka are missing, and Zimbabwe is excluded. For LGSS attainment data across ages we average over 1970-80, for ages 20-29 over 1970-90, and for ages 20-24 over 1970-2000. The sample is 59 countries: Algeria, Iceland, Israel, Jamaica, Myanmar, Sudan, Tunisia and Venezuela are missing, and Jordan is excluded. Initial income is measured as the 1961- 80 average real income per capita for each country, and the remaining explanatory variables, are average levels over 1961-95.

Table 7: Explaining 1995-2004 average life expectancy levels with 1973 forecasts of longevity.

(12)	ages20-24	Lutz etal	0.033**	(0.013)	0.045	(0.061)	0.011	(0.015)	0.002	(0.031)	0.002	(0.007)	-0.018***	(0.005)	0.557**	(0.266)	0.048***	(0.014)	1.470	(1.074)	26	0.897
(11)	ages20-29	Lutz etal	0.025*	(0.015)	0.060	(0.060)	0.008	(0.018)	-0.005	(0.030)	0.004	(0.007)	-0.022***	(0.005)	0.625**	(0.269)	0.044***	(0.015)	1.157	(1.107)	56	0.891
(10)	attain.	Lutz etal	0.019	(0.013)	0.069	(0.057)	0.010	(0.018)	-0.008	(0.029)	0.005	(0.008)	-0.023***	(0.005)	0.650**	(0.264)	0.043***	(0.015)	1.023	(1.091)	56	0.890
(6)	attain.	C&S	0.022*	(0.013)	0.092	(0.059)	-0.001	(0.020)	0.001	(0.032)	0.008	(0.008)	-0.023***	(0.004)	0.550**	(0.260)	0.041**	(0.018)	1.384	(1.047)	26	0.891
(8)	enrol.		0.031**	(0.015)	0.062	(0.062)	0.006	(0.018)	-0.003	(0.032)	0.009	(0.008)	-0.022***	(0.004)	0.652**	(0.266)	0.027	(0.018)	1.001	(1.056)	26	0.895
(7)	years	B&L	0.022**	(0.010)	0.064	(0.055)	0.003	(0.017)	-0.014	(0.027)	0.008	(0.008)	-0.024***	(0.004)	0.666**	(0.250)	0.041***	(0.015)	1.130	(1.072)	56	0.894
(9)	attain.	B&L	0.026**	(0.010)	0.067	(0.058)	0.001	(0.018)	-0.015	(0.028)	0.009	(0.008)	-0.023***	(0.004)	0.632**	(0.261)	0.040**	(0.016)	1.193	(1.076)	56	0.895
(5)	attain.	Lutz etal	0.026*	(0.015)	0.057	(0.058)	0.032	(0.019)	-0.008	(0.028)	0.006	(0.008)	-0.024**	(0.005)	0.918***	(0.228)			-0.263	(0.918)	26	0.882
(4)	attain.	C&S	0.031**	(0.013)	0.089	(0.062)	0.017	(0.021)	0.004	(0.034)	0.011	(0.008)	-0.024***	(0.004)	0.769***	(0.214)			0.284	(0.814)	26	0.883
(3)	enrol.		0.041***	(0.013)	0.050	(0.061)	0.015	(0.018)	-0.001	(0.032)	0.011	(0.008)	-0.022***	(0.004)	0.788***	(0.237)			0.361	(0.922)	56	0.894
(2)	years	B&L	0.027**	(0.012)	0.056	(0.055)	0.024	(0.018)	-0.015	(0.027)	0.010	(0.008)	-0.025***	(0.004)	0.925***	(0.217)			-0.103	(0.914)	56	0.887
(1)	attain.	B&L	0.033**	(0.013)	0.060	(0.058)	0.021	(0.019)	-0.017	(0.027)	0.011	(0.008)	-0.025***	(0.004)	0.875***	(0.222)			0.017	(0.885)	56	0.887
	Higher edu.	measure:	Higher edu.		Fem. liter.		Initial income		Sanitation		Pharmaceut.		AIDS		Lon. forecast		Physicians		Constant		Ops	R-squared

Notes: *** p < 0.01, ** p < 0.05, * p < 0.1. Heteroskedasticity-consistent finite sample standard errors in parentheses. We now exclude countries missing for either of the BL, CS, or LGSS higher educational attainment data, so that we are down to a common sample of 56 countries. The twelve countries excluded relative to Table 6 are: Algeria, Iceland, Israel, Jamaica, Jordan, Myanmar, Pakistan, Sri Lanka, Sudan, Tunisia, Venezuela, and Zimbabwe.

A Appendix

A.1 Proof of proposition 1

Preliminary. Recall that from (14)

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

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

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

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) $\frac{d\pi}{dz} = \frac{\partial \pi}{\partial z} + \mu \frac{m}{m} \frac{dm}{dz} >> 0$, 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 $\frac{dpm}{dz} = p\frac{dm}{dz} > 0$.

graphical analysis: at the new individual solution is linear as $\frac{dc_1}{dz} <<0 \text{ since } \frac{dpm}{dz} = p\frac{dm}{dz} > 0.$ 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 k 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)
$$\frac{dm}{dk} = \frac{\partial m}{\partial k} + \frac{\partial m}{\partial h} \frac{dh}{dk} = \frac{m}{k} \left\{ 1 + \left[1 - \eta \left(1 - \frac{h^{\eta}}{1 + h^{\eta}} \right) \right] \frac{dh}{dk} \frac{k}{h} \right\}.$$

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

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

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

The impact on expenditure on education, $w_t k h$, is given by

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

Therefore

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

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 $\left[1-\eta\left(1-\frac{h^{\eta}}{1+h^{\eta}}\right)\right]\in(0,1)$

(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

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

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

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

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

Both sides of this inequality are increasing and concave in h if

Moreover the left-hand-side of the inequality starts above zero with finite slope, while the righthand-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:

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

This is a sufficient condition for the inequality implied by dm/dk < 0 to be satisfied for any

 $h \le 1/k$. 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) 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

Impact of s. 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)

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

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

Impact of p. An exogenous increase in p shifts upward the \widehat{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).

Impact of k. 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

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

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

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

that is

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

Next substituting for c_1 using (16) we get

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

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

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

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 \widetilde{RHS} unchanged. As a result h must decrease to restore individual optimality. The shift in the \widetilde{LHS} , $\frac{\partial \widehat{LHS}}{\partial \kappa} = (1 - \sigma + \beta)c_1/\kappa$, 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)

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