Determinants of Public Health Outcomes: A Macroeconomic Perspective*

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Abstract

This paper investigates the nature of the aggregate production function of health services. We build a model to analyze the role of public policy in determining social health outcomes, taking into account households choices concerning education, health related expenditures and saving. In the model, education has a positive external effect on health outcomes. Next, we perform an empirical analysis using a data set covering 80 countries from 1961 to 1995. We find strong evidence for a dual role of education as a determinant of health outcomes. In particular, we find that society's tertiary education attainment levels contribute positively to how many years an individual should expect to live, in addition to the role that basic education plays for life expectancy at the individual household level. This finding uncovers a key externality of the educational sector on the ability of society to take advantage of best practices in the health service sector.

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JEL Classification: O30, O40

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1 Introduction

In the last few years a branch of literature has developed applying macroeconomic analysis methods to various issues related to social health status. A lot of attention has been paid to the link between health improvement and economic growth. In particular, the main focus of analysis has been to stydy the impact of health on economic growth.

This paper also applies theoretical and empirical methods from macroeconomics to examine social health status, but with a somewhat different focus. We investigate the nature of the aggregate production function of health services that determines average life-expectancy. That is, we explore the determinants of social health status paying attention to understand and quantify the role played by the different factors of an aggregate production function of health services. These factors include direct inputs such as goods and services provided in the health sector and determinants of hygienic conditions. We consider two different types of explicit inputs, one rival and another non-rival. We assume purchases of rival inputs to be mostly driven by overall purchasing power of consumers as captured by real income per capita.[1] Non-rival inputs to the health sector are pure public goods, affecting the environment in which households make their decisions. An example of this is sanitation.

Average health performance also depends on how well health-related knowledge is rooted in society. For instance, preventive behavior results from knowledge of risks incurred with hazardous behavior. For the individual this knowledge is determined by his/her education and by access to appropriate information. The availability and diffusion of this information is determined by the overall level of education in a society. Education can therefore play two different roles in the aggregate production function of health services. First, the level of education of the household's head enhances the longevity of its members. It seems reasonable in fact that education affects crucial factors such as the understanding of treatments or feeding children healthily. Second, the average level of education in the economy improves its absorption capacity for health-related technology and ideas.

[1] The correlation between per capita income and per capita health expenditure for 29 OECD countries for which the latter measure is available for 1995, is 95 percent.

These two effects play conceptually different roles. The first one operates as a rival input, benefiting only household members. The second instead determines the capacity of the heath service sector to take advantage of best practices. This sector is a high-tech sector and experiences 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 that higher education constitutes its crucial determinant. In contrast, we expect the role of education in enhancing a household's longevity to exhibit strongly diminishing returns. Thus, primary education attainment levels should suffice to capture this latter role of education. On the other hand, controlling for this, any additional effect resulting from the attainment of tertiary education can then be attributed to the second role of education discussed above.

Theoretical and empirical work has considered how human capital accumulation, health improvements and technological progress reinforce each other (e.g. van Zon and Muysken 2001, Blackburn and Cipriani 2002, Chakraborty 2004, and Howitt 2005). Kalemli-Ozcan (2002), looks at the effects of mortality rates on fertility, education, and economic growth. Some papers have underscored the possibility of multiple development paths, which may explain poverty or low life expectancy traps. For example, in Galor and Mayer (2004) a minimum level of health is a precondition for human capital accumulation and health plays a crucial role in inequality persistence. The possible role played by health in the persistence of income inequality is also studied in Deaton 2003, and Chakraborty and Das 2005 where health inequality drives persistence in income inequality through the channel of bequests to offspring and endogenous discounting. Becker, Philipson & Soares (2005) show that "full income" which includes longevity in addition to income per capita, shows much greater convergence across countries than shown for GDP per capita alone.

We present a model where rational individuals choose their educational attainment, savings, consumption of rival health-related inputs, and their children primary education level. In this model, educational choices affect future income but also have external effects on health outcomes. We find that investment in education and in health are positively related at equilibrium, and have a

reinforced impact on longevity.

We then use data from 80 countries for the period from 1961 to 1995 to test the empirical validity of the theoretical model. Using initial period averages to explain end-period life expectancy and utilizing appropriate IV estimates, allows us to alleviate the inherent endogeneity problem concerning life-expectancy and education. To further address problems with capturing the direction of causality, we also consider beginning of period changes in the explanatory variables to explain end of period changes in life expectancy.

We find that primary and tertiary education have separate positive effects on life-expectancy. Our main finding is that tertiary education has at least as great an impact as primary education on health outcomes across countries. This suggests the externality role of education in facilitating adoption of best practices in health is at least as important as the role of basic education that enhances health outcomes at the household level. The paper provides evidence of a form of increasing returns in education, concerning its role in the aggregate production function of health services. This result is particularly interesting because previous work has established that primary education is the single most important determinant of income growth, while higher education has been found to have little explanatory power for this component of welfare (see Sala-i-Martin et al. 2004). Here, tertiary education is found to be an important determinant of a second component of welfare, health status.

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

2 A model of education and health investment

Suppose that individuals can live for two periods. Everyone lives during the first period, which we refer to as youth. Each has a probability $\pi \in (0, 1)$ of surviving to the second period, i.e. adulthood. In the individual's perception her survival probability is an increasing function of health-related rival inputs,

m. We consider the isoelastic function retained by Chakraborty and Das (2005)

$$\pi_t = \mu_t m_t^{\varepsilon} \leq \bar{\pi} < 1 \tag{1}$$

Our analysis focuses on the interesting case when $m < (\bar{\pi}/\mu)^{\frac{1}{\varepsilon}}$. We consider that the following is satisfied

Assumption 1 $\varepsilon \in (0,1)$*, perceived returns on rival inputs to health are decreasing.*

The effectiveness, μ, of the agent's health investment, m, in enhancing her life expectancy, π, depends upon two non-rival factors: the average education level in her generation, $\bar{e}$, and public health policy summarized by variable H. The level of education in the labor force 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. Input H is a pure public good, affecting for instance the rate at which households are subject to diseases. Good examples are the provision of public sewage system and vaccine campaigns.

Moreover, the effectiveness of health-related investment, m, in enhancing life expectancy increases also with the individual level of basic education, e_0. The latter acts as a private input in the health production function. We assume that e_0 is entirely determined by parents at date $t-1$.[2] This is a reasonable and empirically sound assumption (e.g. Lambert and Dumas, 2005).

Using a Cobb-Douglas specification we can write

$$\mu_t \equiv \zeta H_t^{\delta} \bar{e}_t^{\kappa} e_{0t}^{\alpha} \quad \Rightarrow \quad \pi_t = \zeta H_t^{\delta} \bar{e}_t^{\kappa} e_{0t}^{\alpha} m_t^{\varepsilon} \tag{2}$$

[2]If we adopt the alternative assumption, by which each individual internalizes the effect of her educational choice on her 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 these type of reinforcing interactions to take place, while ensuring the existence of a solution to the individual problem as well as of an equilibrium solution. The assumptions that will prove sufficient for the existence of these solutions do in fact limit the extent of the above-mentioned feed-back effects between education and health-related investment.

where $\zeta > 0$ and δ, κ, $\alpha \in (0,1)$. Two different forms of education, basic and higher, are assumed.[3] This is the basic building block of our theoretical framework which we take to the data. The efficiency of rival health-related investment, m, and of basic education, e_0, in increasing life expectancy is an index of technology. As such it depends on environmental (non-rival) variables such us public investment in health, and the quality of the health service sector (i.e. average education, $\bar{e}$, and empirically tertiary education).

The representative agent in generation t chooses her education level, e, rival health-related inputs, m, savings, s, and her child's basic education level to maximize her expected intertemporal utility:[4]

$$\max_{c_{1t},c_{2t+1},s_t,m_t,e_t,e_{0t+1}} u(c_{1t}) + \rho\pi_t u(c_{2t}) + \lambda\pi_{t+1}$$

Two remarks are worthwhile at this stage. Notice first that the discount rate is composed of two factors: the rate of preference for the present, $\rho \in (0,1)$, and the (endogenous) survival probability. We have therefore set up a problem with endogenous discounting of the type analyzed by Chakraborty (2004).

Second, every agent values her child's survival probability according to a weight λ. Child's longevity is valued independetly of the parent's survival probability, differently from consumption goods.[5] The evaluation of the investment in child's basic education on his life expectancy takes into account (2).

The agent's sub-period budget constraints are:

$$w_t(1-\eta_t e_t) = c_{1_t} + p_{m_t} m_t + s_t \quad (3)$$

$$w_{t+1}(1+g(e_t)) + R_t s_t = c_{2_{t+1}} + k_{t+1} e_{0t+1} \quad (4)$$

Education is costly in terms of forgone first period income, according to the effort-cost parameter η, but it increases second period labor income by $g(e)$ percent, where $g' > 0$ and $g'' < 0$. The consumption price is the numeraire

[3] However we restrict the analysis to the case where the agent choice of own education is compatible with the level of primary education that she is endowed with.

[4] Fertility is exogenous in our setting and each agent has one child.

[5] Also Galor and Moav (2005) assume that parental preferences are defined over the number of surving offsprings separately from utility of consumption, and distinctly from children's level of utility.

and p_m is the relative unit price of health-related inputs. Savings earn a gross return R. Investment in child's basic education takes place in the second period and requires a monetary unitary cost k.[6]

As Blanchard (1985) and Yaari (1965) we assume that annuity markets exist. The agent considers as given the return on savings, R. Free entry in the insurance market implies zero profits, i.e. fair insurance premia so that:

$$R_t = \frac{1 + r_t}{E(\pi_t)} \tag{5}$$

where r is the risk-free rate of interest and $E(\pi)$ denotes the average survival probability for the generation. In a symmetric equilibrium this price depends on the individual choice of health-related inputs and of education (via 2).

2.1 The representative agent's choice

Let us restate the problem, using (3) and (4), in the following form:

$$\begin{aligned}\max_{s_t, m_t, e_t, e_{0t+1}} & u\left[w_t\left(1 - \eta_t e_t\right) - p_{m_t} m_t - s_t\right] \\ & + \rho \pi_t u\left[w_{t+1}\left(1 + g\left(e_t\right)\right) + R_t s_t - k_{t+1} e_{0t+1}\right] + \lambda \pi_{t+1}\end{aligned}$$

the first order conditions for an interior solution are:

$$u'(c_{1t}) = R_t \rho \pi_t u'(c_{2t+1}) \tag{s}$$

$$p_{mt} u'(c_{1t}) = \rho \frac{\partial \pi_t}{\partial m_t} u(c_{2t+1}) \tag{m}$$

$$w_t \eta_t u'(c_{1t}) = w_{t+1} \frac{\partial g}{\partial e_t} \rho \pi_t u'(c_{2t+1}) \tag{e}$$

$$\rho \pi_t k_{t+1} u'(c_{2t+1}) = \lambda \frac{\partial \pi_{t+1}}{\partial e_{0t+1}} \tag{e_0}$$

Where an interior solution also requires that the agent's educational choice is unconstrained by her parent choice of basic education, i.e. $e_t > e_{0t}$.

Let us pursue the analysis for the case of isoelastic utility functions $u(c) =$

[6] In this version the parent commits to her child's basic education expenditure at the end of the first period, through an insurance device that allows e_0 to be independent of the realization parent's survival. This is not a restrictive assumption.

$c^{1-\sigma}/(1-\sigma)$ and specify $g(e) = be^{\beta}$ with $\beta \in (0,1)$, and by adopting the following[7]

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

Then the first order conditions above, taking into account (2), become:

$$c_{1t}^{-\sigma} = R_t \rho \pi_t c_{2t+1}^{-\sigma} \qquad (s')$$

$$c_{1t}^{-\sigma} = \frac{\varepsilon}{1-\sigma} \frac{1}{p_{mt} m_t} \rho \pi_t c_{2t+1}^{1-\sigma} \qquad (m')$$

$$c_{1t}^{-\sigma} = (1+\gamma_t) \frac{\beta b e_t^{\beta-1}}{\eta_t} \rho \pi_t c_{2t+1}^{-\sigma} \qquad (e')$$

$$c_{2t+1}^{-\sigma} = \frac{\alpha \lambda}{\rho k_{t+1}} \frac{1}{\pi_t} \frac{\pi_{t+1}}{e_{0t+1}} \qquad (e'_0)$$

where γ is defined as the rate of growth of wages: $1+\gamma_t = w_{t+1}/w_t$. Combining (s') and (e') we get:[8]

$$e_t = e_t^* \equiv \left(\frac{1+\gamma_t}{R_t} \frac{\beta b}{\eta_t} \right)^{\frac{1}{1-\beta}} \qquad (6)$$

Notice that education decreases with return on savings and the effort-cost of education, while it increases with the rate of growth of wages and education direct productivity. The first relationship is due to the fact that education and savings are two competing means to transfer consumption to the second period.

Combining (m') and (s') we get:

$$c_{2t+1} = \frac{1-\sigma}{\varepsilon} R_t p_{mt} m_t \qquad (7)$$

where we understand that assumption 2 is necessary for the problem to make sense. Then from (7) and (4), we have:

$$s_t = \frac{1-\sigma}{\varepsilon} p_{mt} m_t + \frac{k_{t+1}}{R_t} e_{0t+1} - w_t \frac{1+\gamma_t}{R_t} \left(1 + b e_t^{\beta}\right) \qquad (8)$$

Savings tend to increase with health-related expenditure, m, given that $\sigma \in (0,1)$, and with expenditure on child's basic education, and to decrease with

[7] Chakraborty and Das (2005) retain these same assumptions.

[8] For $e < 1/\eta$ it is necessary and sufficient that $R > (1+\gamma)\beta b$.

the level and productivity of education, be_t^{β}. Combining (s') and (7):

$$c_{1t} = \frac{1-\sigma}{\varepsilon} R_t^{-\frac{1-\sigma}{\sigma}} (\rho\pi_t)^{-\frac{1}{\sigma}} p_{mt} m_t \tag{9}$$

so that c_1 is increasing and concave in m.[9] Finally from (e_0') and (7) we get:

$$e_{0t+1} = \left[\frac{\alpha\lambda}{\rho k_{t+1}} \frac{\nu_{t+1}}{\pi_t} \left(\frac{1-\sigma}{\varepsilon} R_t p_{mt} m_t \right)^{\sigma} \right]^{\frac{1}{1-\alpha}} \tag{10}$$

where we define $\nu = \zeta H^{\delta} \bar{e}^{\kappa} m^{\varepsilon}$, such that $\pi = \nu e_0^{\alpha}$. For investment in child's basic education to be an increasing concave function of parental health-related investment it is necessary that $\sigma < 1-\alpha$, i.e. that the elasticity of life expectancy with respect to basic education, α, be small.

Now we can obtain the solution in terms of the level of health related input, m. To do this we combine the budget constraint with desired consumption, c_1 and c_2, and education, e and e_0. First substitute for c_1 and s in (3) using (9) and (8), to get:

$$\begin{aligned} w_t (1 - \eta_t e_t) &= \frac{1-\sigma}{\varepsilon} R_t^{-\frac{1-\sigma}{\sigma}} (\rho\pi_t)^{-\frac{1}{\sigma}} p_{mt} m_t + \left(1 + \frac{1-\sigma}{\varepsilon} \right) p_{m_t} m_t \\ &\quad + \frac{k_{t+1}}{R_t} e_{0t+1} - w_t \frac{1+\gamma_t}{R_t} \left(1 + be_t^{\beta} \right) \end{aligned}$$

Define the maximized permanent income, which takes into account the individually optimal investment in education (6), as

$$y_t \equiv w_t \left[1 - \eta_t e_t^* + \frac{1+\gamma_t}{R_t} \left(1 + b \left(e_t^* \right)^{\beta} \right) \right] \tag{11}$$

Substituting (10) for e_0, and using (1) and (2), the solution of the representative

[9] Although we have set up a problem with endogenous discounting, the objective function is concave in m. We know from (1) that π is concave in m, and we have established in (9) that c_1 is increasing and concave in m, in (7) that c_2 is increasing and linear in m. Thus $u(c_{1t}) + \rho\pi_t u(c_{2t+1}) + \lambda\pi_{t+1}$ is increasing and concave with respect to m.

agent's problem is therefore defined implicitly by :

$$\Gamma(m_t) \equiv \left(1+\frac{1-\sigma}{\varepsilon}\right) p_{mt} m_t + \frac{1-\sigma}{\varepsilon} R_t^{-\frac{1-\sigma}{\sigma}} (\rho\mu_t)^{-\frac{1}{\sigma}} p_{mt} m_t^{\frac{\sigma-\varepsilon}{\sigma}}$$
$$+ \frac{k_{t+1}}{R_t}\left[\frac{\nu_{t+1}}{\zeta H^{\delta} e_{0t}^{\alpha}} \frac{\alpha\lambda}{\rho k_{t+1}} \left(\frac{1-\sigma}{\varepsilon} R_t p_{mt}\right)^{\sigma} \left(\frac{1+\gamma_t}{R_t}\frac{\beta b}{\eta_t}\right)^{\frac{-\kappa}{1-\beta}}\right]^{\frac{1}{1-\alpha}} m_t^{\frac{\sigma-\varepsilon}{1-\alpha}} - y_t = 0 \tag{12}$$

Assumption 3 *Necessary and sufficient conditions for function* $\Gamma(m)$ *to be increasing and concave:*
(a) $\sigma > \varepsilon$*, the direct -privately perceived- elasticity of life expectancy with respect to rival health-related investment is not too large;*
(b) $\alpha < 1-(\sigma-\varepsilon)$*, the direct -privately perceived- elasticity of life expectancy with respect to basic education is not too strong.*

Under assumption 3 function $\Gamma(m)$ starts at $\Gamma(0) = -y_t$, then increases continuously and indefinitely in m, so that there exists a unique solution, m_t^*, to the individual problem such that $\Gamma(m_t^*) = 0$.

2.2 Macroeconomic interaction at the symmetric stationary equilibrium

We adopt a few simplifying assumptions with the aim of focusing the main subject of our analysis, that is the interaction between education, health-related investment and longevity. First we assume that every parent has one and only one child. This comes at no cost, given that fertility is considered as exogenous in our analysis. Second our equilibrium analysis is restricted to the case of an economy where the rate of return on savings, r, wages, w, and medical inputs prices, p_m, are all exogenous.[10] Notice in particular that we rule out long term growth, i.e. set $\gamma = 0$ and thus normalize wages $w = 1$. This results directly from the objective function, which would lead agents to target ever increasing child's life expectancy in front of increasing consumption flows. The result would

[10]These restrictions somehow frustrate the macroeconomic approach retained. For instance the wage could be endogenous on the number of surviving individuals. However, this type of indirect effect is out of the scope of the paper, which covers instead direct and indirect causal links between education, health-related investment and longevity.

be indefinite growth of basic education, which is implausible and incompatible with stationarity.

Let us now turn to the analysis of stationary symmetric equilibria. Symmetry implies that the average survival probability for generation t up to date $t+1$, used by insurers to compute the fair premium, equals individual survival probability

$$E_t(\pi_t) = \pi \tag{13}$$

Recall the definition of survival probability (1) and (2). Considering only unconstrained ($e > e_0$) interior solutions ($\pi < \bar{\pi}$) and taking into account symmetry and stationarity we get

$$\pi = \zeta H^{\delta} e^{\kappa} e_0^{\alpha} m^{\varepsilon} \tag{14}$$

From (5) and (13), (6) gives:

$$e = \left(\frac{1}{1+r}\frac{b\beta}{\eta}\pi\right)^{\frac{1}{1-\beta}} \tag{15}$$

Hence education depends on survival probability only to extent that the latter reduces the return on savings, favoring education investment as means to postpone consumption.

Taking into account (5) and (13) in (s') we find that consumption smoothing is independent of longevity and of education, i.e. it is exogenous, since

$$\frac{c_2}{c_1} = [\rho(1+r)]^{\frac{1}{\sigma}}$$

Using this result in (10) with (14), (5) and (13) hands:

$$e_0 = \frac{\alpha\lambda}{\rho k}\left[\frac{1-\sigma}{\varepsilon}(1+r)\,p_m\right]^{\sigma}\left(\frac{m}{\pi}\right)^{\sigma} \tag{16}$$

The system given by (14), (15) and (16) is solved in the endogenous values of basic education, overall education and life expectancy expressed as functions

of rival health-related investment, m:

$$\pi = \tilde{\pi}(m) \equiv \Omega m^{\frac{(1-\beta)(\varepsilon+\alpha\sigma)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}} \tag{17}$$

$$e = \tilde{e}(m) \equiv \left(\Omega \frac{1}{1+r}\frac{b\beta}{\eta}\right)^{\frac{1}{1-\beta}} m^{\frac{\varepsilon+\alpha\sigma}{1-\beta-\kappa+\alpha\sigma(1-\beta)}} \tag{18}$$

$$e_0 = \tilde{e}_0(m) \equiv \Omega^{1-\sigma}\frac{\alpha\lambda}{\rho(1+r)}\left[\frac{1-\sigma}{\varepsilon}(1+r)p_m\right]^{\sigma} m^{\frac{\sigma[(1-\beta)(1-\varepsilon)-\kappa]}{1-\beta-\kappa+\alpha\sigma(1-\beta)}} \tag{19}$$

where

$$\Omega \equiv \left\{\zeta H^{\delta}\left(\frac{1}{1+r}\frac{b\beta}{\eta}\right)^{\frac{\kappa}{1-\beta}}\left(\frac{\alpha\lambda}{\rho k}\left[\frac{1-\sigma}{\varepsilon}(1+r)p_m\right]^{\sigma}\right)^{\alpha}\right\}^{\frac{1-\beta}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}$$

Assumption 4 *Sufficient conditions for concavity of* $\tilde{\pi}(m)$, $\tilde{e}(m)$ *and* $\tilde{e}_0(m)$*:*[11] $\varepsilon+\beta+\kappa+\alpha<1$*, returns on investment are globally decreasing;*

Education, given by (18) is an increasing function of rival health related inputs, m, as well as of the productivity of education, $\frac{1}{1+r}\frac{b\beta}{\eta}$. Again we find that education is decreasing in the return on savings, $(1+r)$, since education and savings are two competing technologies to transfer consumption to the second period of life. All these considerations are amplified by the strength of the externality, κ, and of the efficiency of basic education in improving longevity, α. Here, there is a distinction between perceived return to education by the household and the equilibrium return which includes the external effect running through improved health services. We note here that β is the elasticity of the private return to education in terms of (ex-post) income, while α and κ are the elasticities of life expectancy with respect to education, the former of its internalized effect through basic education, the latter of its external effect on the quality of the health service sector. The total return to education is given

[11] In fact assumption 4 implies that $\kappa+\beta<1$, for which $\kappa<(1-\beta)(1+\alpha\sigma)$ follows, and hence that the denominator of the exponents of m in the three functions is positive. Assumption 4 implies that the exponent of m in (18) is positive and smaller that unity. This must also be the case for the one in (17), since $\beta<1$. Finally, concavity of $\tilde{e}_0(m)$ requires that $\kappa/(1-\beta)<[1-\sigma(1-\varepsilon-\alpha)]/(1-\sigma)$. Assumption 4 implies that the right-hand-side of the inequality id larger than unity, while it also implies $\kappa<1-\beta$, which entails that its left-hand-side is smaller than unity.

by the increase in earning if surviving (through β) times the increase in the probability of surviving weighted by second period earnings.

To determine the properties of the stationary symmetric equilibrium, we first need to study the properties of the terms of the implicit function (12) at equilibrium. Its first term $G_1(m) \equiv \left(1 + \frac{1-\sigma}{\varepsilon}\right) p_m m$ is a linear increasing function of m. Its second terms is given by $G_2(m) \equiv \frac{1-\sigma}{\varepsilon} R^{-\frac{1-\sigma}{\sigma}} (\rho\mu)^{-\frac{1}{\sigma}} p_m m^{\frac{\sigma-\varepsilon}{\sigma}}$. Taking into account (5), (13), (2) and (17) we have that

$$G_2(m) = [\rho(1+r)]^{-\frac{1}{\sigma}} \frac{1-\sigma}{\varepsilon}(1+r) p_m \Omega^{-1} m^{\frac{1-\beta-\kappa-\varepsilon(1-\beta)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}$$

which is an increasing concave function of m under assumption 4 (see appendix A.1 for the computation of first and second partial derivatives). The third term of the implicit function (12), $G_3(m) \equiv e_0 k/R$, can be rewritten at equilibrium using (5), (13), (16), the definition of ν and (17), to get:

$$G_3(m) = \frac{\alpha\lambda}{\rho(1+r)} \left[\frac{1-\sigma}{\varepsilon}(1+r) p_m\right]^{\sigma} \Omega^{1-\sigma} m^{\frac{(1-\beta)[\varepsilon+\alpha\sigma+\sigma(1-\varepsilon)]-\sigma\kappa}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}$$

which is also an increasing concave function of m if assumptions 1 and 4(b) are satisfied (see appendix A.1).

We can also rewrite permanent disposable income, (11), at the symmetric stationary equilibrium, first substituting for e using (6), next for R using (5) and (13), then exploiting the definition of e^*

$$y(m) \equiv 1 + \frac{1}{1+r}\tilde{\pi} + \frac{1-\beta}{\beta}\eta e^* \tag{20}$$

Using (15) and (17) we get:

$$y(m) = 1 + \frac{1}{1+r}\Omega m^{\frac{(1-\beta)(\varepsilon+\alpha\sigma)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}} + (1-\beta)\left(b\frac{1}{1+r}\Omega\right)^{\frac{1}{1-\beta}} m^{\frac{\varepsilon+\alpha\sigma}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}$$

which is an increasing concave function of m under assumptions 1 and 4 (see appendix A.1).

It is therefore possible to express the equilibrium level of private health

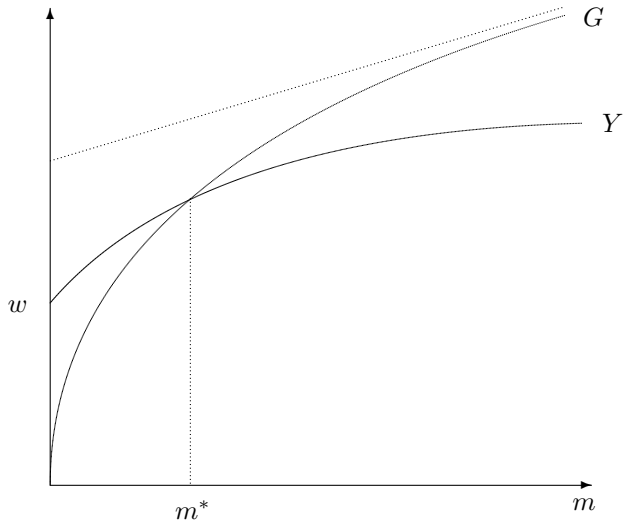

Figure 1: Determining private health investment in a symmetric equilibrium.

investment as being m^* that solves the following implicit function

$$\Gamma(m^*) \equiv G(m^*) - y(m^*) = 0 \tag{21}$$

where $G(m) \equiv G_1(m) + G_2(m) + G_3(m)$. Functions $G_1(m)$, $G_2(m)$, $G_3(m)$ and $y(m)$ are all increasing in m. We are however able to establish the following:

Proposition 1 *Equation (21) admits a unique, positive and finite, solution if assumptions 1 to 4 are satisfied.*

Proof. Under assumptions 1-4 $G_i(m)$, for $i = 1, 2, 3$, and $y(m)$ are increasing and concave, as explained in the main text. We also have that $y(0) = 1 > G_1(0) + G_2(0) + G_3(0) = 0$, but $y'(m)$ declines indefinitely towards zero, i.e. $\lim_{m\to\infty} y(m) = 0$, while $G'(m)$ never falls below a positive lower bound, since $\lim_{m\to\infty} G_2(m) = \lim_{m\to\infty} G_3(m) = 0$ while $\lim_{m\to\infty} G_1(m) = \left(1 + \frac{1-\sigma}{\varepsilon}\right) p_m > 0$. These conditions imply that the two schedules cross only once, as illustrated in figure 1. ■

We have the following result

Proposition 2 *If the elasticity of life expectancy with respect to basic education is not too strong, consumption of rival health-related inputs, higher education and basic education, and therefore life expectancy, are increasing in non-rival health-related inputs and decreasing in the relative price of medical inputs, in the effort-cost of higher education, and in the monetary cost of basic education:*

$$\begin{aligned}
\frac{dm}{dH} &> 0 \ , \ \frac{de}{dH} > 0 \ and \ \frac{de_0}{dH} > 0 \ \Rightarrow \ \frac{d\pi}{dH} > 0 \\
\frac{dm}{dp_m} &< 0 \ , \ \frac{de}{dp_m} < 0 \ and \ \frac{de_0}{dp_m} < 0 \ \Rightarrow \ \frac{d\pi}{dp_m} < 0 \\
\frac{dm}{d\eta} &< 0 \ , \ \frac{de}{d\eta} < 0 \ and \ \frac{de_0}{d\eta} < 0 \ \Rightarrow \ \frac{d\pi}{d\eta} < 0 \\
\frac{dm}{dk} &< 0 \ , \ \frac{de}{dk} < 0 \ and \ \frac{de_0}{dk} < 0 \ \Rightarrow \ \frac{d\pi}{dk} < 0
\end{aligned}$$

Proof. See appendix A.2. ■

We conclude that investment in education and in health are positively correlated, and reinforce each other in their impact on longevity.

Consider for instance a reduction in the effort-cost of education, η. It directly spurs education as an individual response (see 6). As can be seen from (11), more education increases permanent income, leading to more consumption, expenditure on child's basic education and purchase of medical inputs. These last two indirect effects reinforce at equilibrium the positive impact that increased education has on life expectancy (see 14).

3 Data description

In this section, we describe the data set we have assembled to test our main hypotheses and take a first look at the relationship of health status with each of these inputs. The focus of our study, a country's health status, is measured by the average life expectancy at birth.

We employ a number of health output and health input variables from two sources. The *World Development Indicators* (WDI) 2002 database provides data on life expectancy at birth, physicians per thousand people, adult illiteracy

Table 1: Correlations

	LIFE	EDHA	EDBA	EDH	ILLI	SAN	PHYS	INC
LIFE	1							
EDHA	0.87	1						
EDBA	0.40	0.29	1					
EDH	0.89	0.93	0.32	1				
ILLI	-0.66	-0.69	-0.22	-0.69	1			
SAN	0.75	0.69	0.52	0.67	-0.61	1		
PHYS	0.90	0.87	0.35	0.90	-0.71	0.71	1	
INC	0.77	0.76	0.21	0.79	-0.81	0.65	0.86	1

Notes: We report cross-sectional correlations after averaging life expectancy over 1990 to 1995 and all remaining (potentially explanatory) variables for the period 1961 to 1995, except income for which we use its level at the beginning of the period. The sample size used here is 71 countries. All variables are in natural logarithms. LIFE is life expectancy, EDHA is higher education attainment rate, EDBA is primary education attainment rate, EDH is tertiary education enrollment rate, ILLI is the adult illiteracy rate as a percentage of the population over 15 years of age, SAN is the percentage of the population with access to improved sanitation facilities, PHYS is number of physicians per thousand people, INC is initial GDP per capita in constant US dollars.

rates[12], and sanitation[13]. We also obtained GDP per capita in PPP dollars, and tertiary education enrollment rates from the same database. Finally, we obtained primary and higher education attainment rates from the Barro and Lee (2001) dataset.

We were able to put together all the above series for 80 countries during the period 1961-1995. The list of counties is shown in Table A1 in the appendix. However, the great majority of these series are not available annually; in some cases the data are exceedingly sparse in the time dimension. Because the cross-sectional dimension of the dataset is more complete and, more importantly, because of the inherent long-run nature of the relation under study, we chose to explore empirically the cross-sectional dimension of our dataset.

4 Empirical results

4.1 Preliminary evidence

In Table 1, we report basic correlations between our variables of interest. Our main hypothesis is that health inputs such us primary and higher education, sanitation, access to safe water, and physicians availability are related to health outcomes measured by life expectancy. Indeed, the correlations between life expectancy and higher education attainment rates or enrollment rates equal 87 and 89 percent respectively, while the correlation for basic education attainment rates equals 40 percent. An other (inverse) measure of basic education - the adult illiteracy rate - is also strongly correlated with life expectancy at minus 66 percent. All of these correlations are statistically significant with p-values below the one percent level. Sanitation and physicians are also strongly related with life expectancy with correlations of 75 and 90 percent respectively. However, nearly all of these health inputs are also strongly related to the level of real income per capita. This is especially true in the case of higher education attainment or enrollment rates and for physicians availability. Moreover, several of these inputs are highly correlated with each other raising a warning flag regarding a potential collinearity problem in the regression specifications that follow in the next subsection. Notably, the correlation between higher education attainment or enrollment rates with physicians is 87 and 90 percent respectively. As a robustness check for the importance of higher education we will thus consider specifications both with and without the apparently highly collinear physicians variable.

4.2 Cross-section regression results

We are well aware that there is a strong theoretical argument for endogeneity between life expectancy and tertiary education. While tertiary education should be expected to affect health outcomes, it can also be argued that individual decisions on tertiary education attainment depend on expected life expectancy

[12]Defined as the percentage of individuals over 15 years of age who cannot, with understanding, read and write a short simple statement on their everyday life.

[13]Defined as the percentage of the population with access to improved sanitation facilities.

so that it is plausible that longer life expectancy causes higher tertiary education levels. However, for the model we consider below, we fail to reject the null that tertiary education is exogenous with a p-value of 0.42[14] and the joint hypothesis that all explanatory variables are exogenous with a p-value of 0.72. This suggests that we could estimate the empirical model of life expectancy on secondary and primary education attainment rates, sanitation, physicians, and initial income with OLS. However, given that we have just about 70 observations and that the individual p-values for the null of exogeneity for each explanatory variable separately range from 0.13 for physicians to 0.97 for primary enrollment rates, we choose to be conservative regarding our inference of exogeneity and estimate the model using IV in addition to OLS estimation. This serves to take into account possible endogeneity problems we have been unable to detect, and also acts as a robustness check for our OLS results.

Towards the goal of addressing potential endogeneity problems and establishing some evidence of temporal causation we consider: (i) Using lags of higher education and the other explanatory variables[15] to explain end-period averages of life expectancy. Specifically, utilizing the average value of higher education and the other explanatory variables for 1961-75 to explain the average value of life expectancy over 1990-95. This takes care of endogeneity if individual decisions about higher education in 1961-75 are independent of life expectancy at birth for individuals born between 1990 and 1995. We present results based on this specification as the "Lags" model in columns two and five in Tables 2 and 3. (ii) Instrumenting the averages of tertiary education, basic education, and physicians over 1961-95 by their average value during 1961-75 to explain the average value of life expectancy over 1990-95. In the regression of each potentially endogenous explanatory variable[16] on all exogenous variables, the lag

[14]Treating one explanatory variable at a time as potentially endogenous and the remaining as exogenous, we also fail to reject the null that initial income is exogenous with a p-value of 0.43. Similarly, we cannot reject the null that primary education attainment rates is exogenous with a p-value of 0.97. Nor, can we reject the null that the physicians measure is exogenous with a p-value of 0.13, and finally we cannot reject the null that sanitation is exogenous with a p-value of 0.91.

[15]Except for sanitation for which we often have just a single observation for each country during the end of the period.

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

of each explanatory variable is shown to be strongly significant in determining the explanatory variable's period average, with p-values always below the one percent level of significance. We present results based on the IV specification in columns three and six in Tables 2 and 3.[17] (iii) We use log changes in the explanatory variables for the period 1961-75 to explain the log change in life expectancy for the period 1976-95. We also apply IV estimation to these variables in changes, instrumenting the log change in tertiary education over 1961-95 by its 1961-75 value. Results based on this approach are reported in Table 4.

Overall, we assess the link between health inputs and life expectancy with the "Lags" and "IV" models described above, and the"Period Avg" model where we consider the average of the 1990-95 period life expectancy being explained by the 1961-95 average value of the explanatory variables. We report results for this model in columns one and four of Tables 2 and 3. In each case, we consider specifications with and without physicians, since this variable is highly collinear with higher education.[18] We also consider log changes of the variables in place of the levels and present estimation results from this exercise in Table 4. In this case, for the "Period Avg" model we consider the growth rate of life expectancy between 1976 and 1995 being explained by growth rates of the explanatory variables between 1961 and 1995, with results presented in the first and fourth columns of Table 4.

The dual effect of education on life expectancy is of primary interest to us. For this reason, we consider three different specifications with different pairs of measures for higher and basic education in Tables 2, 3, and 4. In specification one, we consider higher and primary attainment rates from the Barro and Lee database. We report the estimates from this specification in Table 2. In the second specification, results for which are reported in Table 3 we consider tertiary education enrollment rates along with the illiteracy rate,

[17]Again, for Sanitation (SAN) we typically have just a handful of observations for the whole period so we cannot instrument this variable with its lag.

[18]Physicians should have a dual role in determining health outcomes. On the one hand, this is a direct input into the health production function similar to any other medical input. On the other hand, they should have a role as vectors of knowledge facilitating medical technology absorption and the adoption of best practices. Including both tertiary education and physicians in the same specification should thus be expected to reduce the coefficient estimate of tertiary education to the extent these two variables are capturing the same concept. Thus, the coefficient estimate for tertiary education in these specifications should be seen as a lower bound of the importance of the knowledge externality we are focusing on in this paper.

Table 2: Cross-country level regressions

Specif. 1	Model 1 Period Avg	Model 1 Lags	Model 1 IV	Model 2 Period Avg	Model 2 Lags	Model 2 IV
INCOME	.033***	.039	.039**	−.013	−.008	−.004
	(1.75)	(1.51)	(2.01)	(−0.82)	(−0.36)	(−0.24)
EDHA	.072*	.051*	.063*	.038*	.027***	.036*
	(5.33)	(3.45)	(4.24)	(3.47)	(1.69)	(2.54)
EDBA	.048***	.041	.049***	.028	.019	.031
	(1.95)	(1.56)	(1.70)	(1.31)	(0.78)	(1.21)
SAN	.080***	.101**	.087**	.066***	.098**	.072**
	(1.82)	(2.13)	(2.07)	(1.84)	(2.38)	(2.12)
PHYS	—	—	—	.070*	.068*	.063*
				(4.29)	(2.61)	(3.06)
constant	3.349*	3.307*	3.278*	3.908*	3.834*	3.805*
	(20.56)	(17.76)	(18.08)	(24.47)	(17.74)	(20.43)
Adj. R^2	78.8	75.3	78.7	82.8	77.9	82.5
Obs.	72	72	72	72	71	71

Notes: * p-value less than one percent, ** p-value less than five percent, *** p-value less than ten percent. For "Period Avg" models, we consider 1990-95 averages of life expectancy being explained by 1961-95 averages for the explanatory variables. For "Lags" models, we consider again 1990-95 averages of life expectancy being explained in this case 1961-75 averages for the explanatory variables. Finally, for "IV" Models 1 and 2, we instrument the 1961-95 period averages for the explanatory variables using their beginning of period averages. All variables are in natural logs so that the reported estimates are elasticities of life expectancy with respect to each explanatory variable.

both taken from the WDI database. Finally, in Table 4, we consider log changes of education attainment levels.

In Model 1 of Table 2, we consider the impact of basic and higher education attainment rates as well as real income per capita and sanitation on the end-period (1990-95) average of life expectancy. We report results from Model 1 in the first three columns of Table 2. Irrespective of whether we consider the average value of the explanatory variables over the 1961-95 period, their average value at the beginning of the period, or instrument the former with the latter, higher education attainment rates consistently have a positive and significant impact on life expectancy which is always greater than the impact of primary education. The elasticity of life expectancy with respect to higher education is stable across the three methodologies ranging between 5.1 percent for the lags model to 6.3 percent for the instrumental variables estimation, and up to 7.2 percent for the period-averages model. Moreover, the estimated elasticity of life expectancy with respect to primary education ranges from 4.1 percent with a p-value of 0.12 for the lags model, to 4.9 and 4.8 percent and statistically significant at the ten percent level for the IV and period-averages models respectively. For

the three specifications of Model 1, sanitation has a positive and consistently significant impact on life expectancy estimated about 10 percent.

In Table 2, we also take into account the fact that income can be a major determinant of health by including the initial period value of real income per capita. To the extent to which we control for public health inputs and education, real income per capita can serve isolate the effect of private health inputs purchases as it captures the consumer's purchasing power. Thus, in the absence of a direct measure of rival inputs being available for our sample of countries, we use real income per capita as a proxy for health-related rival input purchases. We can show using data from the OECD that as of 1995, the correlation between per capita income and per capita health expenditure was actually very high at 95 percent. Finally, controlling for the effect of income helps isolate the part of the effect of each of the other inputs that is not related to income. For the specifications in the first three columns, income has a positive impact on life expectancy, slightly below the elasticity of life expectancy with respect to primary education.

In columns four to six of Table 2, we report results for Model 2 which now includes physicians availability in addition to the two education variables, sanitation, and income per capita. Since physicians and higher education are highly collinear, with a correlation of 87 percent, introducing physicians dampens the impact of higher education on life expectancy. Still, this remains positive and significant, irrespective of whether we use period-averages, initial period averages, or instrument the explanatory variables, in columns four, five, and six respectively. The impact of higher education is stable across the three methodologies ranging between 2.7 percent for the lags model to 3.6 percent for the instrumental variables estimation and 3.8 percent for the period-averages model, always above the estimated life expectancy elasticity of primary education. The latter remains positive but becomes statistically insignificant. Finally, sanitation retains a, somewhat reduced, positive and significant impact on life expectancy.

Physicians availability has a positive and strongly significant impact on life expectancy that remains stable at about seven percent in columns four to six, irrespective of the methodology pursued. To the extent that physicians facilitate the flow of health-related ideas, a component of this health input could poten-

Table 3: Cross-country level regressions

Specif 2	Model 1 Period Avg	Model 1 Lags	Model 1 IV	Model 2 Period Avg	Model 2 Lags	Model 2 IV
INCOME	.033***	.015	−.012	−.002	−.012	−.043
	(1.82)	(0.97)	(−0.44)	(−0.09)	(−0.70)	(−1.43)
EDH	.094*	.071*	.089*	.056*	.043**	.029
	(6.17)	(3.73)	(5.09)	(3.79)	(2.24)	(1.28)
ILLI	.005	−.001***	−.021***	.002	−.007	−.019
	(0.85)	(−1.69)	(−1.69)	(0.47)	(−0.96)	(−1.48)
SAN	.049**	.042***	.039***	.029	.035	.015
	(2.26)	(1.85)	(1.74)	(1.42)	(1.58)	(0.68)
PHYS	—	—	—	.062*	.062**	.088*
				(3.95)	(2.51)	(3.33)
constant	3.51*	3.83*	3.94*	3.98*	4.16*	4.48*
	(24.05)	(24.39)	(17.19)	(22.44)	(20.48)	(13.84)
Adj. R^2	83.1	80.9	79.4	85.7	83.1	83.0
Obs.	79	77	77	79	76	76

Notes: * p-value less than one percent, ** p-value less than five percent, *** p-value less than ten percent. For "Period Avg" models, we consider 1990-95 averages of life expectancy being explained by 1961-95 averages for the explanatory variables. For "Lags" models, we consider again 1990-95 averages of life expectancy being explained in this case 1961-75 averages for the explanatory variables. Finally, for "IV" Models 1 and 2, we instrument the 1961-95 period averages for the explanatory variables using their beginning of period averages. All variables are in natural logs so that the reported estimates are elasticities of life expectancy with respect to each explanatory variable.

tially be perceived as non-rival, a hypothesis that is supported by the dampening of the impact of higher education once the physicians availability variable is introduced in Model 2. Finally, once we account for physicians, income now has no impact on life expectancy.

Illiteracy

Next, we consider a different (inverse) measure of basic education - the rate of illiteracy - along with tertiary education enrollment rates. In Table 3, we replicate the regression models estimated in Table 2, using now this alternative measures of basic and higher education. Conceptually, the illiteracy rate should measure an even more orthogonal component of education than primary attainment rates, relative to what is captured by our measures of tertiary education. In Table 3, tertiary education enrollment rates are shown to have a positive and statistically significant impact on life expectancy with elasticities ranging from a high of 6.9 percent down to 2.5 percent for the different models considered there. Illiteracy has a negative impact on life expectancy which is always statistically insignificant once physicians are introduced in the specification. The

Specif. 2	**Model 1** *Period Avg*	**Model 1** *Lags*	**Model 1** *IV*	**Model 2** *Period Avg*	**Model 2** *Lags*	**Model 2** *IV*
INCOME	.039**	.039***	.038**	.006	.009	.004
	(2.08)	(1.89)	(2.12)	(0.32)	(0.37)	(0.19)
EDH	.085*	.068*	.078*	.055*	.046**	.035***
	(4.98)	(3.43)	(4.36)	(3.27)	(2.21)	(1.82)
ILLI	−.028[1]	−.002***	−.045**	−.021	−.001	−.039***
	(1.41)	(−1.83)	(−1.98)	(−1.09)	(−1.22)	(−1.79)
SAN	.033[2]	.033[3]	.025	.020	.030[4]	.011
	(1.49)	(1.47)	(1.16)	(0.99)	(1.39)	(0.53)
PHYS	—	—	—	.054*	.053**	.065*
				(3.34)	(2.03)	(2.85)
constant	3.65*	3.71*	3.76*	4.03*	4.02*	4.19*
	(20.96)	(21.50)	(21.08)	(20.64)	(17.62)	(17.62)
Adj. R^2	79.8	76.9	79.8	82.0	78.6	81.9
Obs.	61	60	60	61	60	60

Notes: * p-value less than one percent, ** p-value less than five percent, *** p-value less than ten percent, p-value=0.16, p-value=0.12, p-value=0.15, 4p-value=0.17, 4p-value=0.115. Out sample here excludes 18 developed economies for which the rate of illiteracy is zero. For "Period Avg" models, we consider 1990-95 averages of life expectancy being explained by 1961-95 averages for the explanatory variables. For "Lags" models, we consider again 1990-95 averages of life expectancy being explained in this case 1961-75 averages for the explanatory variables. Finally, for "IV" Models 1 and 2, we instrument the 1961-95 period averages for the explanatory variables using their beginning of period averages. All variables are in natural logs so that the reported estimates are elasticities of life expectancy with respect to each explanatory variable.

impact of sanitation remains positive but is not significant at conventional levels of significance once physicians are introduced. Finally, the impact of physicians remains positive, significant, and of similar magnitude as previously. The estimated impact of income is statistically indistinguishable from zero in most cases.

Overall, we find that higher education matters significantly, and is more robust than primary education, sanitation, and even income. Using initial period averages to explain end-period life expectancy along with IV estimation, allows us to establish that tertiary education is a significant and robust explanatory variable of end of period health output. This approach alleviates potential endogeneity problems and provides supporting evidence of a causality link from tertiary education to health status (life expectancy).

Changes in variables specification

As an additional methodology to remedy potential endogeneity problems facing tertiary education as a determinant of health status, we consider log changes of the variables instead of their log levels. This also serves as a robustness check for our main finding regarding the dual importance of education, and

Table 4: Cross-country changes in variables regressions

Specif 3	Model 1 PeriodAvg	Model 1 Lags	Model 1 IV	Model 2 PeriodAvg	Model 2a Lags	Model 2b Lags	Model 2 IV
INCOME	−.0002	−.001**	−.0003	−.0003	−.001**	−.001**	−.001[1]
	(−0.57)	(−1.98)	(−0.65)	(−0.85)	(−2.29)	(−2.04)	(−1.63)
EDHA	.055*	.025*	.068*	.047*	.025*	.025*	.069*
	(3.98)	(3.17)	(3.82)	(3.96)	(4.42)	(2.80)	(3.56)
EDBA	.060*	.032	.042	.058*	.042**	.017	.021
	(2.77)	(1.48)	(1.18)	(2.66)	(2.14)	(0.85)	(0.72)
YGROWTH	.030	−.015	−.029	.008	−.017	−.031	−.061
	(1.03)	(−0.81)	(−0.85)	(0.34)	(−0.86)	(−1.39)	(−1.49)
PHYS				.063**	.074**	.079*	.101*
				(2.34)	(2.48)	(3.09)	(2.63)
constant	.003	.009*	.004	.003	.007**	.009**	.006
	(0.81)	(2.69)	(0.94)	(0.89)	(2.55)	(2.37)	(1.49)
Adj. R^2	31.5	22.6	25.5	42.1	41.9	29.7	32.1
Obs.	66	66	66	63	63	52	50

Notes: Notes: * p-value less than one percent, ** p-value less than five percent, *** p-value less than ten percent, p-value=0.104. All variables other than initial real income per capita are in log changes. YGROWTH is the growth rate of real income per capita. For the "Period Avg" models, we consider the growth rate of life expectancy between 1976 and 1995 being explained by growth rates of the explanatory variables between 1961 and 1995. For the "Lags" models, we consider again the growth rate of life expectancy between 1976 and 1995 being explained by growth rates of the explanatory variables between 1961 and 1975. Finally, for the "IV" Models 1 and 2, we instrument the 1961-95 period changes for the explanatory variables using their beginning of period averages. In Model 2a we do not use the beginning of the period change for physicians which allows us to use about 20comparability of the sample with the IV Model 2. All variables are in natural logs so that the reported estimates are elasticities of life expectancy with respect to each explanatory variable.

in particular the channel through which higher education affects life expectancy emphasized in this paper. We report estimates in Table 4.

The growth rate of higher education attainment levels has a positive impact on the end period growth rate in life expectancy for all seven specifications we consider. It takes its highest value of about seven percent in the IV specifications reported in columns three and seven. The growth rate of primary education also has a positive effect which is now close to that for tertiary education but is statistically insignificant in several of the models we consider.

Looking at the negative coefficient estimates for initial income levels, there appears to be some evidence for convergence in life expectancy for countries that started with low real income per capita level.[19] On the other hand, the growth

[19] From Evans (1997) we know that the coefficient estimate for initial income and the implied rate of convergence have downward bias here. As shown there, failing to account for all sources of heterogeneity across countries will have the same effect as measurement error on initial income biasing its coefficient estimate and the implied rate of convergence towards zero. Thus, we should view this evidence of convergence shown here as a lower bound and suggestive of even greater convergence rates in health status for initially poor countries.

rate of real income per capita does not seem to explain any of the gains in life expectancy. This suggests that any convergence that took place for initially low-income countries has not been the result of higher real income per capita growth, but likely due to changes in other determinants of public health in laggard countries. These other determinants would likely include changes in public inputs like sanitation (which we cannot consider in this specification directly in the absence of observations over time for this variable), and perhaps medical knowledge diffusion as emphasized in Papageorgiou, Savvides, and Zachariadis (2005).

5 Conclusion

We have presented a model where education can have external effects on life expectancy, beyond what can be expected from the impact of basic education on the individual household's health status. Our main results are as follows: a) Considering physicians per thousand inhabitants as an explanatory variable we find it extremely significant and robust. As a side effect, introducing this variable reduces the separate impact of tertiary education. b) Public health inputs such us sanitation have a positive impact on life expectancy. c) There is some evidence of convergence in life expectancy for countries that started off with low real income per capita levels in 1961 and this does not appear to be explained by faster output growth rates of initially poor countries, suggesting the possibility that faster technology absorption of initially laggard countries might actually be behind convergence. d) Education has a dual role in determining health oucomes, with both basic and higher education having positive impact on life expectancy. Moreover, the impact of higher education appears to be at least as important as the impact of basic education in determining life expectancy, suggesting the externality role of education in facilitating adoption of best practices in health is at least as important as the role of basic education enhancing health outcomes at the household level.

The last result is particularly interesting because growth regressions have established that primary education is the single most important determinant of income growth, while higher education is found to have little explanatory power

(Sala-i-Martin, Doppelhofer and Miller 2004). Also microeconomic evidence suggests that primary education is more important than tertiary education in determining growth in income (e.g. Psacharopoulos 1994). Our findings suggest that tertiary education might be important for one component of welfare, health status, even if it's less important as a determinant of an other component of welfare, income per capita.

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A Appendix

A.1 Derivation of results

Recall that

$$\Omega \equiv \left\{ \zeta H^{\delta} \left(\frac{1}{1+r} \frac{b\beta}{\eta} \right)^{\frac{\kappa}{1-\beta}} \left(\frac{\alpha\lambda}{\rho k} \left[\frac{1-\sigma}{\varepsilon} (1+r) p_m \right]^{\sigma} \right)^{\alpha} \right\}^{\frac{1-\beta}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}$$

$$G_1(m) \equiv \left(1 + \frac{1-\sigma}{\varepsilon} \right) p_m m$$

$$G_2(m) = [\rho(1+r)]^{-\frac{1}{\sigma}} \frac{1-\sigma}{\varepsilon} (1+r) p_m \Omega^{-1} m^{\frac{1-\beta-\kappa-\varepsilon(1-\beta)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}$$

$$G_3(m) = \frac{\alpha\lambda}{\rho(1+r)} \left[\frac{1-\sigma}{\varepsilon} (1+r) p_m \right]^{\sigma} \Omega^{1-\sigma} m^{\frac{(1-\beta)[\varepsilon+\alpha\sigma+\sigma(1-\varepsilon)]-\sigma\kappa}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}$$

$$y(m) = 1 + \frac{1}{1+r} \Omega m^{\frac{(1-\beta)(\varepsilon+\alpha\sigma)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}} + (1-\beta) \left[b \frac{1}{1+r} \Omega \right]^{\frac{1}{1-\beta}} m^{\frac{\varepsilon+\alpha\sigma}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}$$

$$\Gamma(m) = G_1(m) + G_2(m) + G_3(m) - y(m)$$

It will be useful to compute values for $\alpha \to 0$

$$\begin{aligned}
\hat{\Omega} &\equiv \lim_{\alpha\to 0} \Omega = \left[\zeta H^{\delta} \left(\frac{1}{1+r} \frac{b\beta}{\eta} \right)^{\frac{\kappa}{1-\beta}} \right]^{\frac{1-\beta}{1-\beta-\kappa}} \\
\hat{G}_1(m) &\equiv \lim_{\alpha\to 0} G_1(m) = G_1(m) \\
\hat{G}_2(m) &\equiv \lim_{\alpha\to 0} G_2(m) = [\rho(1+r)]^{-\frac{1}{\sigma}} \frac{1-\sigma}{\varepsilon} (1+r) p_m \hat{\Omega}^{-1} m^{\frac{1-\beta-\kappa-\varepsilon(1-\beta)}{1-\beta-\kappa}} \\
\hat{G}_3(m) &\equiv \lim_{\alpha\to 0} G_3(m) = 0
\end{aligned}$$

Differentiating with respect to m:

$$\frac{\partial \Omega}{\partial m} = 0$$

$$\frac{\partial G_1}{\partial m} = \frac{G_1}{m} > 0$$

$$\frac{\partial^2 G_1}{\partial m^2} = 0$$

$$\frac{\partial G_2}{\partial m} = \frac{1-\beta-\kappa-\varepsilon(1-\beta)}{1-\beta-\kappa+\alpha\sigma(1-\beta)} \frac{G_2}{m} > 0$$

$$\frac{\partial^2 G_1}{\partial m^2} = -\frac{(1-\beta)(\varepsilon+\alpha\sigma)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}\frac{1-\beta-\kappa-\varepsilon(1-\beta)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}\frac{G_2}{m^2} < 0$$

$$\frac{\partial G_3}{\partial m} = \frac{(1-\beta)[\varepsilon+\alpha\sigma+\sigma(1-\varepsilon)]-\sigma\kappa}{1-\beta-\kappa+\alpha\sigma(1-\beta)}\frac{G_3}{m} > 0$$

$$\frac{\partial^2 G_3}{\partial m^2} = -\frac{(1-\sigma)[1-\beta-\kappa-\varepsilon(1-\beta)]}{1-\beta-\kappa+\alpha\sigma(1-\beta)}\frac{(1-\beta)[\varepsilon+\alpha\sigma+\sigma(1-\varepsilon)]-\sigma\kappa}{1-\beta-\kappa+\alpha\sigma(1-\beta)}\frac{G_3}{m^2} < 0$$

$$\frac{\partial y}{\partial m} = \frac{(1-\beta)(\varepsilon+\alpha\sigma)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}\left\{\frac{1}{1+r}\Omega m^{\frac{(1-\beta)(\varepsilon+\alpha\sigma)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}} + \left[b\frac{1}{1+r}\Omega\right]^{\frac{1}{1-\beta}} m^{\frac{\varepsilon+\alpha\sigma}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}\right\}\frac{1}{m} > 0$$

$$\frac{\partial^2 y}{\partial m^2} = -\frac{(1-\beta)(\varepsilon+\alpha\sigma)}{[1-\beta-\kappa+\alpha\sigma(1-\beta)]^2}\left\{[(1-\beta)(1-\varepsilon)-\kappa]\frac{1}{1+r}\Omega m^{\frac{(1-\beta)(\varepsilon+\alpha\sigma)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}\right.$$
$$\left. + (1-\varepsilon-\beta-\kappa-\alpha\sigma\beta)\left[b\frac{1}{1+r}\Omega\right]^{\frac{1}{1-\beta}} m^{\frac{\varepsilon+\alpha\sigma}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}\right\}\frac{1}{m^2} < 0$$

The signs are determines using assumptions 1-4 as sufficient conditions. See proof of proposition 1 and footnote 11 for an explanation

A.2 Proof of proposition 2

Overall the marginal impact on function $\Gamma(m)$ is given by

$$\frac{\partial \Gamma}{\partial m} = \frac{\partial G}{\partial m} - \frac{\partial y}{\partial m} > 0$$

Wa can clearly sign this expression even though both $\partial G/\partial m$ and $\partial y/\partial m$ are positive, because we have demonstrated in the proof of proposition one, that at the equilibrium level of m the schedule drawn by function $G(m)$ cuts from below the schedule given by $y(m)$, i.e. at m^* $\partial G/\partial m > \partial y/\partial m > 0$ unambiguously.

Impact of a marginal increase in H

$$\begin{aligned}
\frac{\partial \Omega}{\partial H} &= \frac{\delta(1-\beta)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}\frac{\Omega}{H} > 0 \\
\frac{\partial G_1}{\partial H} &= 0 \\
\frac{\partial G_2}{\partial H} &= -\frac{G_2}{\Omega}\frac{\partial \Omega}{\partial H} < 0 \\
\frac{\partial G_3}{\partial H} &= (1-\delta)\frac{G_3}{\Omega}\frac{\partial \Omega}{\partial H} > 0 \\
\frac{\partial y}{\partial H} &= \left\{\frac{1}{1+r}m^{\frac{(1-\beta)(\varepsilon+\alpha\sigma)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}} + \left[b\frac{1}{1+r}\Omega^{\beta}\right]^{\frac{1}{1-\beta}} m^{\frac{\varepsilon+\alpha\sigma}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}\right\}\frac{\partial \Omega}{\partial H} > 0
\end{aligned}$$

Thus

$$\frac{\partial \Gamma}{\partial H} = \frac{\partial G}{\partial H} - \frac{\partial y}{\partial H} = \left(-\frac{G_2}{\Omega} + (1-\sigma)\frac{G_3}{\Omega}\right)\frac{\partial \Omega}{\partial H} - \frac{\partial y}{\partial H}$$

The sign of $\partial\Gamma/\partial H$ can be determined for the case of $\alpha = 0$, since in this case $\partial\hat{\Omega}/\partial H > 0$, implying $\partial\hat{G}_2/\partial H < 0$ but $\hat{G}_3 = 0$, so that $\lim_{\alpha\to 0} \partial\Gamma/\partial H < 0$. As a consequence

$$\lim_{\alpha\to 0} \frac{dm}{dH} = \lim_{\alpha\to 0} -\frac{\partial\Gamma/\partial m}{\partial\Gamma/\partial H} > 0$$

By continuity this is also true for sufficiently small values of α. Results on e and e_0 follow from (18) and (19).

Impact of a marginal increase in p_m

$$\begin{aligned}
\frac{\partial\Omega}{\partial p_m} &= \frac{\alpha\sigma\,(1-\beta)}{1-\beta-\kappa+\alpha\sigma\,(1-\beta)}\frac{\Omega}{p_m} > 0 \\
\frac{\partial G_1}{\partial p_m} &= \frac{G_1}{p_m} \\
\frac{\partial G_2}{\partial p_m} &= \left(\frac{1}{p_m} - \frac{1}{\Omega}\frac{\partial\Omega}{\partial p_m}\right) G_2 > 0 \\
\frac{\partial G_3}{\partial p_m} &= \left(\sigma\frac{1}{p_m} + (1-\sigma)\,\frac{1}{\Omega}\frac{\partial\Omega}{\partial p_m}\right) G_3 > 0 \\
\frac{\partial y}{\partial p_m} &= \left\{\frac{1}{1+r} m^{\frac{(1-\beta)(\varepsilon+\alpha\sigma)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}} + \left[b\frac{1}{1+r}\Omega^{\beta}\right]^{\frac{1}{1-\beta}} m^{\frac{\varepsilon+\alpha\sigma}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}\right\} \frac{\partial\Omega}{\partial p_m} < 0
\end{aligned}$$

Thus

$$\begin{aligned}
\frac{\partial\Gamma}{\partial p_m} &= \frac{\partial G}{\partial p_m} - \frac{\partial y}{\partial p_m} \\
&= \frac{G_1}{p_m} + \left(\frac{1}{p_m} - \frac{1}{\Omega}\frac{\partial\Omega}{\partial p_m}\right) G_2 + \left(\sigma\frac{1}{p_m} + (1-\sigma)\,\frac{1}{\Omega}\frac{\partial\Omega}{\partial p_m}\right) G_3 - \frac{\partial y}{\partial p_m} > 0
\end{aligned}$$

As a consequence

$$\frac{dm}{dp_m} = -\frac{\partial\Gamma/\partial m}{\partial\Gamma/\partial p_m} < 0$$

Concerning we get from (18)

$$\begin{aligned}
\frac{\partial e}{\partial p_m} &= \left(\frac{1}{1+r}\frac{b\beta}{\eta}\right)^{\frac{1}{1-\beta}} \Omega^{\frac{1}{1-\beta}} m^{\frac{\varepsilon+\alpha\sigma}{1-\beta-\kappa+\alpha\sigma(1-\beta)}} \left(\frac{1}{1-\beta}\Omega^{-1}\frac{\partial\Omega}{\partial p_m} + \frac{\varepsilon+\alpha\sigma}{1-\beta-\kappa+\alpha\sigma\,(1-\beta)}\frac{1}{m}\frac{\partial m}{\partial p_m}\right) \\
&=
\end{aligned}$$

implying

Impact of a marginal increase in η

$$\begin{aligned}
\frac{\partial \Omega}{\partial \eta} &= \frac{-\kappa}{1-\beta-\kappa+\alpha\sigma\left(1-\beta\right)}\frac{\Omega}{\eta} < 0 \\
\frac{\partial G_1}{\partial \eta} &= 0 \\
\frac{\partial G_2}{\partial \eta} &= -\frac{G_2}{\Omega}\frac{\partial \Omega}{\partial \eta} > 0 \\
\frac{\partial G_3}{\partial \eta} &= \left(1-\delta\right)\frac{G_3}{\Omega}\frac{\partial \Omega}{\partial \eta} < 0 \\
\frac{\partial y}{\partial \eta} &= \left\{\frac{1}{1+r}m^{\frac{(1-\beta)(\varepsilon+\alpha\sigma)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}} + \left[b\frac{1}{1+r}\Omega^{\beta}\right]^{\frac{1}{1-\beta}} m^{\frac{\varepsilon+\alpha\sigma}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}\right\}\frac{\partial \Omega}{\partial \eta} < 0
\end{aligned}$$

Thus

$$\frac{\partial \Gamma}{\partial \eta} = \frac{\partial G}{\partial \eta} - \frac{\partial y}{\partial \eta} = \left(-\frac{G_2}{\Omega} + \left(1-\sigma\right)\frac{G_3}{\Omega}\right)\frac{\partial \Omega}{\partial \eta} - \frac{\partial y}{\partial \eta}$$

The sign of $\partial\Gamma/\partial\eta$ can be determined for the case of $\alpha = 0$, since in this case $\partial\hat{\Omega}/\partial\eta < 0$ implies $\partial\hat{G}_2/\partial\eta > 0$ but $\hat{G}_3 = 0$, so that $\lim_{\alpha\to 0}\partial\Gamma/\partial\eta > 0$. As a consequence

$$\lim_{\alpha\to 0}\frac{dm}{d\eta} = \lim_{\alpha\to 0} -\frac{\partial\Gamma/\partial m}{\partial\Gamma/\partial\eta} < 0$$

By continuity this is also true for sufficiently small values of α. Results on e and e_0 follow from (18) and (19).

Impact of a marginal increase in k

$$\begin{aligned}
\frac{\partial \Omega}{\partial k} &= \frac{-\alpha\left(1-\beta\right)}{1-\beta-\kappa+\alpha\sigma\left(1-\beta\right)}\frac{\Omega}{k} < 0 \\
\frac{\partial G_1}{\partial k} &= 0 \\
\frac{\partial G_2}{\partial k} &= -\frac{G_2}{\Omega}\frac{\partial \Omega}{\partial k} > 0 \\
\frac{\partial G_3}{\partial k} &= \left(1-\delta\right)\frac{G_3}{\Omega}\frac{\partial \Omega}{\partial k} < 0 \\
\frac{\partial y}{\partial k} &= \left\{\frac{1}{1+r}m^{\frac{(1-\beta)(\varepsilon+\alpha\sigma)}{1-\beta-\kappa+\alpha\sigma(1-\beta)}} + \left[b\frac{1}{1+r}\Omega^{\beta}\right]^{\frac{1}{1-\beta}} m^{\frac{\varepsilon+\alpha\sigma}{1-\beta-\kappa+\alpha\sigma(1-\beta)}}\right\}\frac{\partial \Omega}{\partial k} < 0
\end{aligned}$$

Thus

$$\frac{\partial \Gamma}{\partial k} = \frac{\partial G}{\partial k} - \frac{\partial y}{\partial k} = \left(-\frac{G_2}{\Omega} + \left(1-\sigma\right)\frac{G_3}{\Omega}\right)\frac{\partial \Omega}{\partial k} - \frac{\partial y}{\partial k}$$

The sign of $\partial\Gamma/\partial k$ can be determined for the case of $\alpha = 0$, since in this case $\partial\hat{\Omega}/\partial k < 0$ implies $\partial\hat{G}_2/\partial k > 0$ but $\hat{G}_3 = 0$, so that $\lim_{\alpha\to 0}\partial\Gamma/\partial k > 0$. As a consequence

$$\lim_{\alpha\to 0}\frac{dm}{dk} = \lim_{\alpha\to 0} -\frac{\partial\Gamma/\partial m}{\partial\Gamma/\partial k} < 0$$

By continuity this is also true for sufficiently small values of α. Results on e and e_0 follow from (18) and (19).

A.3 Table A1: List of countries in the dataset

Country	**Avg Life Expectancy**[1]
Algeria	68.5
Argentina	72.1
Australia	77.5
Austria	76.3
Bangladesh	56.4
Belgium	76.6
Bolivia	59.4
Brazil	66.2
Cameroon	54.2
Canada	77.8
Chile	74.3
Colombia	68.9
Costa Rica	75.8
Cote d'Ivoire	48.8
Cyprus	76.8
Denmark	75.0
Ecuador	67.4
Egypt	64.0
El Salvador	67.1
Ethiopia	44.8
Finland	75.7
France	77.3
Germany	75.6
Ghana	58.1
Greece	77.3
Guatemala	62.5
Haiti	53.4
Honduras	65.3
Iceland	78.3
India	60.2
Indonesia	62.9
Iran	65.8
Iraq	59.6
Ireland	75.5
Israel	76.6
Italy	77.4
Jamaica	73.8
Japan	79.3
Jordan	69.5
Kenya	55.5

Table A1: List of countries cont.

Country	**Avg Life Expectancy**
Korea	71.0
Madagascar	53.0
Malawi	43.4
Malaysia	71.2
Mali	44.3
Mauritius	70.1
Mexico	71.4
Morocco	64.6
Mozambique	43.8
Myanmar	55.3
Netherlands	77.1
New Zealand	75.9
Nigeria	49.5
Norway	77.2
Pakistan	59.9
Panama	73.0
Paraguay	68.6
Peru	66.8
Philippines	66.6
Portugal	74.1
Rwanda	37.7
Sierra Leone	35.2
Singapore	75.5
Spain	76.8
Sri Lanka	70.8
Sudan	53.1
Sweden	78.1
Switzerlannd	77.9
Tanzania	49.3
Thailand	69.1
Tunisia	70.8
Turkey	67.2
Uganda	45.4
United Kingdom	76.2
Unites States	75.5
Uruguay	73.0
Venezuela	71.8
Zambia	47.8
Zimbabwe	53.6

[1]This is the end of period average life expectancy from 1990 to 1995.