

HEALTH, HUMAN CAPITAL FORMATION AND KNOWLEDGE PRODUCTION¹

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Abstract. Recent medical research shows that health is highly influential for learning and the ability to think laterally; however, past studies have failed to empirically examine the nexus between health, learning, schooling, ideas production and growth. This paper constructs health-adjusted educational attainment among the working age population based on their health status during the time they did their education. Using data for 21 OECD countries over two centuries it is shown that health has been highly influential for the quantity and quality of schooling and innovations, which have in turn been the main drivers of growth.

JEL classification: O1, O2, O4

Key words: ideas production, learning, schooling, productivity growth

1. Introduction

The nexus between health and per capita income remains a controversial issue. Using life expectancy as an indicator of health Barro and Sala-i-Martin (1992), Knowles and Owen (1995, 1997), Bloom and Sachs (BPEA, 1998), Bloom and Williamson (1998), Arora (2001), Soares (2005), Bloom *et al.* (2009), Aghion *et al.* (2011), and Cervellati and Sunde (2011) find a significant positive relationship between life expectancy and growth. However, in an influential paper Acemoglu *et al.* (2007), fail to find a positive relationship between improved life expectancy at birth and income growth between 1940 and 1980 for a large cross-country sample using the interaction between health invention dates and mortality for different diseases as instruments for health. They, instead, find that health innovations lead to faster population growth and, therefore, that health lowers per capita income.

The trouble associated with these estimates is that life expectancy, as an indicator of health, may not identify the timing and the channels through which health influences growth. To overcome this problem Zhang and Zhang (2005) construct a three period overlapping generations model showing that rising longevity reduces fertility, raising savings, and schooling time; however, empirically they find that these effects are not quantitatively large. Exploiting the exogenous

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variation in working age mortality across countries Lorentzen *at al.* (2008) find that lower working-age mortality leads to higher growth. Using microeconomic estimates for earnings outcomes as a result of improved health, Weil (2007) finds that the contribution to growth from improved health is quite modest. However, the channels identified by Weil (2007) may not be the most important channels through which health influences growth. A potentially important channel in which health influence growth, which is often overlooked in the literature, is the influence of diseases on cognitive development, learning, the amount of schooling and ideas production. An exception is Bleakley (2007) who shows that the eradication of the hookworm in the Southern states of the US in the early 20th century increased literacy and schooling significantly in the infected areas and was a major contributor to the income convergence between the Southern and the Northern States of the US.

This paper argues that health influences productivity growth through ideas production and schooling-induced human capital during the time the working population did their schooling. These channels have been overlooked and cannot be captured by conventional health indicators such as height and life expectancy; as, these are very coarse indicators of health and, therefore, cannot capture the multi-dimensional aspects of health, the interaction between health and other variables, and the time-lag between the health shock and the time the productivity is affected. The schooling channel illustrates this point. As the human capital of a 64-year old individual, for instance, was influenced by their health when they started learning first enrolled in school 58 years earlier. Thus, what is needed to account for the influence of health on learning is to compute a measure that summarizes the interaction between schooling and health while the working age population did their schooling; conventional health measures cannot capture this effect; even when long lags are allowed for.

As discussed in the next section the capacity to learn in school and produce new ideas is positively related to the health status of a student and/or potential innovator. A direct effect of health on learning is that malnourished and sick children are often absent from school and are even less likely to be enrolled in school. An indirect effect of health on learning is that illness can severely impair the learning capacity of students and school children due to a lack of concentration in the classroom, cognitive impairment, stigma, and coping skills. Morbidity adversely affects ideas production because it impairs creative and lateral thinking. A high morbidity compared to a low

morbidity environment will result in workers that are less entrepreneurial and innovative than workers. Furthermore, recent research shows that societies with high pathogen stress are culturally more collectivist, less open to new ideas and display introversion (Schaller and Murray, 2008; and Fincher *et al.*, 2008). An over-activation of psychological mechanisms that inhibit interaction with people who appear to pose a risk for transmission of diseases lead to xenophobia and ethnocentrism (Schaller and Murray, 2008). As a result, high disease societies tend to value collectivism while penalizing individualism, consequently, becoming less innovative and subject to rote learning (Fincher *et al.*, 2008).

The paper proceeds as follows. Based on the endogenous growth model of Howitt (2005) the next section shows that the quality and quantity of human capital and ideas production are potentially important channels through which health influences growth. To test for the influence of health on growth through the quality of learning, an optimization algorithm that finds the optimal growth effects of morbidity-adjusted educational attainment is established (Section 3). In Section 4, empirical estimates are undertaken using data over the period 1812-2009 for 21 OECD countries. Extended empirical estimates are carried out in Section 5 and Section 6 concludes the paper.

2. Growth, cognitive development and creativity

To address the qualitative aspects of health on growth through enhanced learning in school and creative production, it is instructive to consider a condensed version of the Schumpeterian model of Howitt (2005) as follows:

$$Y = \psi F[K, AS(1 - \varepsilon)], \quad \text{Production} \quad (1)$$

$$\dot{S} = \varepsilon \lambda L - \phi S, \quad \text{Skills production} \quad (2)$$

$$\frac{\dot{A}}{A} = \frac{\mu R}{L \cdot A^*}, \quad \text{Ideas production} \quad (3)$$

where Y is output; K is capital stock; A is technology; S is the stock of skills; ε is schooling; λ is learning efficiency; ψ is production efficiency; L is the labor force, ϕ is the skill-adjusted death rate; R is technology-innovation expenditure; A^* is the global technology frontier, and μ is research efficiency. Skills production, given by Eq. (2), is a positive function of the health-adjusted schooling capital of the working population, $\varepsilon \lambda L$, minus the exit of skilled workers from the labor force, ϕH .

The ideas production function given by Eq. (3) extends the first-generation models of knowledge

production function to allow for product proliferation (see Aghion and Howitt 1998, 2006; Peretto, 1998; Dinopoulos and Thompson, 1998; Peretto and Smulders, 2002; Dinopoulos and Waldo, 2005; and Ha and Howitt, 2007). Technology-innovation expenditure, R , is divided by product variety, LA^* , following the Schumpeterian paradigm in which R&D spreads more thinly across product varieties as the economy grows. Since, in steady state, product variety is growing at the same rate as population or the labor force, it follows that productivity growth remains constant as long as the fraction of researchers in the labor force remains constant; a feature that overcomes the problems associated with first-generation growth models in which the growth rate is proportional to the number of researchers. In addition to L , R is divided by A^* because the complexity of innovations increases as the economy develops (Aghion and Howitt, 1998). The restriction of scale effects in ideas production is relaxed in the empirical estimates.

In steady state, the equilibrium skills per worker in manufacturing and productivity growth are given by (see Howitt, 2005):

$$s = \frac{\varepsilon\lambda(1-\varepsilon)}{\phi+n}, \quad (4)$$

$$g = \mu\rho\psi F(k, h)(1 - a) \quad (5)$$

where n is the population growth rate; $\rho = R/Y$ is research intensity; $s = S(1 - \varepsilon)/L$ is the skill per worker in manufacturing; $k = K/AL$ is capital per unit of effective labor, and $a = A/A^*$ is the proximity to the technology frontier.

In this model health impacts on growth through the parameters λ , ε , ϕ , ρ and μ . The equilibrium research intensity, ρ , is a positive function of health because the pay-off from innovations is higher the easier the workforce is able to adapt to innovations (Aghion and Howitt, 1998, Ch. 6) and because healthy workers are better in dealing with stress and adapt to a new and innovative environment (Howitt, 2005). The skill-adjusted death rate, ϕ , affects growth in that skilled individuals die before they reach their retirement age. The quantity of schooling, ε , comprises of the proportion of an age cohort that is enrolled in schools (gross enrollment rate, GER) and the class attendance rate for the enrolled students. A higher life expectancy increases the present value of schooling as the dividends are discounted over a larger number of years; thus increasing the GERs. This effect can be potentially large as shown in the model of Bils and Klenow (2001) in which, there is a one-to-one relationship between life expectancy and the optimal level of schooling measured in

years. Class attendance rates are related to health as in many developing countries and in the rich countries before WWII, low attendance rates, to a large extent, can be attributed sickness (Jamison *et al.* 2006, Ch. 58). This effect has been potentially large for the sample used here because, the attendance rates in Australia, Canada, the US and Sweden, which are the only countries in this paper for which historical data are available, were on average below 50% in the 19th century (Madsen, 2012) and are even lower in the poorest countries in the world today (United Nations, 2008). If at least half of the absentee rates can be attributed to health, the quantitative effects of health on growth in the transition from a low growth regime to a modern growth regime can be potentially large.

The learning efficiency in school, λ , is related to health through concentration in the classroom, cognitive processing, and the stigma associated with illness. Surveying the literature on health and learning (Jamison *et al.*, 2006) conclude that “empirical evidence shows that good health and nutrition are prerequisites for effective learning. This finding is not simply the utopian aspiration for children to have healthy bodies and healthy minds, but also the demonstration of a systemic link between specific physical insults and specific cognitive and learning deficits, grounded in a new multisectoral approach to research involving public health and epidemiology, as well as cognitive and educational psychology.” (p. 1091).

There is strong evidence that illness impairs learning efficiency. The literature finds that temporary energy and micro-nutritional malnutrition, parasite load, infection, and untreated sensory impairment are significantly related to worsened general conventional and cognitive indicators and absenteeism and attrition (Mayer-Foulkus, 2005). Surveying the literature Watkins and Pollitt (1997) find almost unanimous support for the hypothesis that most parasitic and infectious diseases impair learning abilities. Furthermore, evidence shows that children with poor health have significantly lower educational attainment than children in good health (Mayer-Foulkus, 2005). Bloom and Canning (2009) argue that illness impairs children’s learning by contributing directly to absenteeism and inattention. For instance, vitamin A not only leads to higher mortality from other diseases but also impairs the vision and, consequently, the child’s capacity to learn.

Malnutrition, which was widespread in Europe *pre*-WWII according to the estimates of Fogel (1994), has also been found to be influential for learning and cognitive development (Grantham-McGregor, 1995). Students who suffer from malnutrition and parasitic and infectious diseases have low attendance rates, find it difficult to concentrate and focus in the classroom,

perform poorly in cognitive tests, have poor fine motor skills and delayed psychomotor development, and have poor scholastic achievements (Scrimshaw, 1998; Dickson *et al.*, 2000; Holding and Snow, 2001; and Alderman *et al.*, 2005). Furthermore, malnutrition is associated with behavioral problems among children leading to poor relationships with their peers, short attention spans, distractibility in the class, irritability, apathy, and a lack of interest in subject topics (Latham and Cobos, 1971; Grantham-McGregor, 1995; and Holding and Snow, 2001). Glewwe *et al.* (2001) find that better nourished children start earlier in school, repeat fewer grades and learn more per unit of time spent in school (see also the discussion of Mayer-Foulkus, 2005; and Jack and Lewis, 2009).

Research efficiency, μ , is influenced by health in much the same way as the efficiency of learning at school. Healthy researchers are able to concentrate for longer, are more creative, and have fewer sick days, than researchers with poor health (Howitt, 2005). Furthermore, the age-associated cognitive decline is much less pronounced for healthy than unhealthy workers (Starr *et al.*, 1997). Finally, highly infectious environments render societies more collectivist and, consequently, less innovative and less critical to learning and the established wisdom. Individualistic cultures, by contrast, value initiative, independent thinking whereas collectivist cultures expect people to identify with groups and work well in them protecting them in exchange for loyalty and compliance (Schaller and Murray, 2008). Using worldwide cross-country value surveys Fincher *et al.*, (2008) find that prevalence of pathogens is strongly positively correlated with cultural indicators of collectivism and strongly negatively correlated with individualism. These results suggest that Europe and its off-springs, presumably, would have been more collectivist and, thus, less innovative before they entered the low-mortality regime during the 20th century.

3. Health, learning and human capital

While the parameters ε , ϕ , ρ and μ in the above model are relatively straightforward to estimate, as shown in the empirical section, the growth effects of health-induced learning efficiency, λ , are much more complex to estimate as there are no historical data on learning efficiency and because it takes several years before learning at school influences productivity growth. The challenge is, therefore, to incorporate health into the learning process at each level of education and to transform health-incorporated schooling into human capital among the working age population.

The change in human capital is assumed to be a function of the interaction between formal

schooling and the health status among students in the age cohort a :

$$\dot{h}_t^a = G(GER_t^a, Health_t^a), \quad (6)$$

where h is the health-adjusted human capital among the working population, and GER^a is the gross enrollment rate of age cohort a ; that is the fraction of the population in age cohort a that is enrolled in school. The variable h is closely related to the variable s in the model in the previous section, where the principal distinction between the two variables relates to h being defined in units of the working age population while s is defined in terms of manufacturing labor. The human capital production function given by Eq. (6) can easily be extended to allow for the quality of teachers and the method of teaching. Lucas (1988), for example, assumes that the production of human capital, \dot{h} , depends on the level of human capital, h , where h can be thought of as the human capital of teachers.

To make Eq. (6) operational the health status for age cohort a , is assumed to be proportional to the survival rate, $(1 - m^a)$, at the age of a :

$$\ln Health_t^a = \Phi \cdot \ln[1 - m_t^a], \quad (7)$$

where m^a is the mortality rate at age a , which acts as a proxy for the age-dependent health status of the population and Φ is an unknown scale parameter that needs to be estimated.

Although mortality is not an ideal proxy for measuring the health status, such as morbidity among the different age cohorts, the variable relevant for leaning capacity, fitness and cognitive development, it is, however, the only age-dependent morbidity indicator that is available. Diseases that significantly impair learning capacity, such as helminth, and iron and iodine deficiency, are rarely fatal and only affect age-dependent mortalities indirectly. Iron and iodine deficiencies, however, are outcomes of malnutrition that are often associated with protein-energy malnutrition, which in turn often leads to secondary fatal diseases because protein-energy malnutrition impairs the immune system (Fogel, 1994). Furthermore, age-dependent mortality does capture the most important diseases over the past two centuries in the Western world, such as measles, tuberculosis, smallpox, and influenza; diseases that were associated with morbidity as well as mortality.

Assuming that health and GERs influence the change in human capital multiplicatively yields the following explicit expression for the change in human capital:

$$\dot{h}_t^a = GER_t^a \cdot \exp[\Phi \cdot \ln(1 - m_t^a)]. \quad (8)$$

In order to estimate the value of Φ , the schooling flow is first converted into the stock of human capital as follows:

$$h(\Phi)_t = \frac{\sum_{i=0}^{49} \{ pop_{15+i} \sum_{j=0}^{\kappa} [GER_{t-i-j} \exp(\Phi \ln(1 - m_{t-i-j}^j))] \}}{\sum_{a=15}^{64} pop_t^a} \quad (9)$$

where h is the stock of human capital, pop^a is the size of the population at the age of a , and κ is the maximum years of schooling.

This equation computes the health-adjusted educational attainment by 1) multiplying the population, at period t , in each working-age cohort by the health-adjusted GERs in the period during which they did their education; 2) summing over all the age cohorts; and 3) dividing this sum by the working age population. This method follows the inventory perpetual principle in which the human capital depreciation rates depend on age-specific mortality rates. Age-specific mortality is, implicitly, incorporated into the estimates by following the population in each age cohort. Thus, the educational attainment for an average 64 year old person in 1870 is the sum of the health-adjusted GERs in primary school during the period 1812-1817, in secondary school during the period 1818-1822, and in tertiary education during the period 1823-1827.

Finally, the value of Φ is found through iterations that maximize the statistical significance of human capital in the following productivity growth model:

$$g_t = H[h(\Phi)_t, Z_t] + e_t, \quad 0 < \Phi < 1000, \quad (10)$$

where g is productivity growth, Z is a vector of control variables and e is a stochastic error term. The exact specification of the model and the choice of control variable are detailed in the next section. The model is iterated in the interval $0 < \Phi < 1000$. If $\Phi = 0$, health will have no effect on learning and educational attainment is estimated in the same way as the conventional human capital estimates based on the perpetual inventory method.

4. Model specifications, data and estimation method

Guided by Eqs. (3), (5) and (10) the ideas production and the productivity growth model are

stochastically specified as follows:

$$\Delta \ln Pat_{it} = \beta_0 + \beta_1 \Delta \ln \left(\frac{R\&D}{Y} \right)_{it} + \beta_2 \Delta \ln m_{it}^{wa} + \beta_3 \Delta \ln S_{it}^{Pat} + \beta_4 \left(\frac{R\&D}{Y} DTF \right)_{i,t-1} + e_{1,it}, \quad (11)$$

$$\begin{aligned} \Delta \ln y_{it} = & \alpha_0 + \alpha_1 \Delta h(\Phi)_{it} + \alpha_2 h(\Phi)_{it} + \alpha_3 h(\Phi)_{it}^2 + \alpha_4 DTF_{t-1} \\ & + \alpha_5 h(\Phi)_{i,t-1} DTF_{i,t-1} + \alpha_6 \Delta \ln Pat_t + \alpha_7 \Delta \ln S_t^f + \alpha_8 \ln(Pat/L)_{it} + CD + e_{2,it} \end{aligned} \quad (12)$$

where Pat is the number of patent applications filed by residents; S^{Pat} is patent stock; $(R\&D/Y)$ is research intensity, measured as the ratio of nominal R&D expenditures and nominal GDP; y is real GDP per hour worked, where hours worked is measured as the economy-wide employment multiplied by average annual hours worked per worker; S^f is foreign knowledge stock; CD is country dummies; m^{wa} is the working-age (15-64) mortality rate; Δ is the five-year difference operator; and $DTF = (A^* - A)/A$ is the distance to the frontier, where A^* is measured as the maximum TFP for the US and UK in purchasing power parity units. DTF_{t-1} and $h(\Phi)_{t-1} DTF_{t-1}$ are measured in the first year of the five-year period over which the first differences span. Both $h(\Phi)$ and $(R\&D/Y)$ are measured as annual averages in the five years over which the differences span. Eqs. (11) and (12) are estimated over the period 1870-2009.

The ideas production function given by Eq. (11) is more general than the ideas production function given by Eq. (3) in that the coefficient of A is allowed to vary freely and ideas production is positively related to the interaction between research intensity and the DTF . The mortality rates among the working age population are assumed to directly influence ideas production independently of research intensity in the model specification. The possibility that mortality rates affect ideas production through research intensity is investigated below. The productivity effects of the interaction between research intensity and DTF follow the predictions of the model of Howitt (2000) in which research intensity, as a measure of the absorptive capacity, aids in absorbing the technology that is developed at the frontier.

The productivity growth model extends the model presented in Section 2 to allow for international knowledge spillovers following Coe and Helpman (1995), Lichtenberg and van Pottelsberghe de la Potterie (1998), and Madsen (2007) and the possibility that the innovative activity has only temporary growth effects. Furthermore, the model allows for the interaction

between human capital and the distance to frontier, $h \cdot DTF$, following the model of Nelson and Phelps (1966) (see Benhabib and Spiegel, 1994, 2005). The philosophy behind the Nelson-Phelps model is that the further a country is behind the technological frontier, the higher is its growth potential provided that it has a sufficiently high level of human capital or absorptive capacity, to take advantage of its backwardness. DTF is included in the model to ensure that the interaction term, $h \cdot DTF$, is not picking up the effects of one of its components.

Squared educational attainment is included in the model to allow for a potential inverse U-shaped relationship between educational attainment and growth. Provided that $\alpha_2 > 0$ and $\alpha_3 < 0$, educational attainment affects growth at an increasing rate when the educational level is below $-\alpha_2/2\alpha_3$ and at a decreasing rate when it is above $-\alpha_2/2\alpha_3$. Non-linear relationships between human capital and growth have long been advocated in the theoretical and empirical literature. Theoretically, Azariadis and Drazen (1990), Redding (1996), Aghion and Howitt (1998, pp. 327-333), Bala and Sorger (2001), and Howitt (2005) show how countries with low human capital can end up in a low growth trap through various multiple equilibrium mechanisms. Empirically, Kalaitzidakis *et al.* (2001), Kruger and Lindahl (2001), Foldvari and Leeuwen (2009), and Madsen (2012) find significant non-linear growth effects of human capital.

The change in human capital, Δh , is included in the model to allow for temporary growth effects of human capital as a complement or a substitute for the level of human capital. Human capital may have only temporary growth effects if it is channeled to output through the production function as in the neoclassical model of Mankiw *et al.* (1992) or the semi-endogenous growth model discussed in Madsen (2008). Similarly, ΔPat is included in the regressions following semi-endogenous growth theory in which innovations have only temporary growth effects (see, for discussion and derivations, Ha and Howitt, 2007; and Madsen, 2008).

4.1 Data

The data cover the period 1812-2009 for the following 21 OECD countries: Canada, the US, Japan, Australia, New Zealand, Austria, Belgium, Denmark, Finland, France, Germany, Greece, Ireland, Italy, the Netherlands, Norway, Portugal, Spain, Sweden, Switzerland, and the UK. The economy-wide TFP data are based on the two-factor homogenous Cobb-Douglas production technology with labor augmenting technological progress, $Y = K^\alpha (AL)^{1-\alpha}$, where A is the TFP. Thus,

the TFP is computed as $A = (Y/L)(Y/K)^{\alpha/(1-\alpha)}$, where $(1-\alpha)$ is computed as the unweighted average of labor's income share in country i and in the US. Labor's income share for each country is in turn estimated as the average over the longest period for which the data are available (see for details, Madsen, 2007).

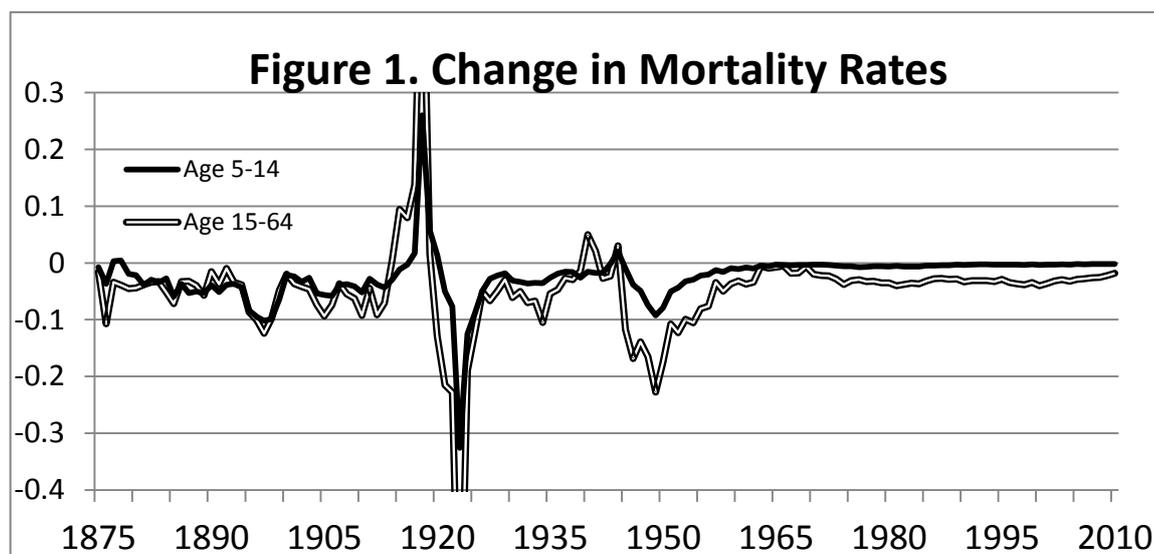
The research intensity data, $(R\&D/Y)$, are extrapolated for most of the countries before the 1960s using data for Australia, Germany, Japan, Spain and the US as templates as detailed in the data appendix. Clearly, this renders the research intensity data less reliable than the other data and is the main reason for measuring research intensity by (Pat/L) in the productivity growth model. GERs are estimated for primary, secondary and tertiary education and the construction of the data are discussed in depth in Madsen (2012). As discussed in Madsen (2012) the schooling systems are comparable over the past two centuries across the countries in this sample; even Japan, which is often considered culturally distinct from the West, adapted the Western schooling system in the early 19th century.

Knowledge spillovers through the channel of imports of intermediate products that contain new technology from country j to country i are computed from the following weighting scheme suggested by Lichtenberg and van Pottelsberghe de la Potterie (1998) and modified by Madsen (2007) to smooth out erratic movements in import ratios:

$$S_{it}^f = \sum_{j=1}^{21} \left(\frac{M_{ijt}^s}{Y_{jt}^{s,n}} \right) S_{jt}^d, \quad i \neq j. \quad j = 1, 2, \dots, 21.$$

where M_{ij}^s is the smoothed nominal imports of goods of high-technology products from country j to country i , $Y_j^{s,n}$ is country j 's smoothed nominal income and S_j^d is country j 's stock of domestic knowledge, which is estimated using the perpetual inventory method and a 15% depreciation rate (see, for details, Madsen, 2007). The 21 countries used to estimate knowledge spillovers are the same as those considered in this paper.

Historical age-dependent mortality rates are only available in five-year age intervals (5-9, 10-14, ..., 60-64), and measure the probability of dying at a certain age. These data are available for most countries from 1870 to 2009. Mortality data for the school-age population that are required back to 1812 in order to compute the health-adjusted GERs, are available for quite a few countries, which in turn are used as templates for other countries that have missing data, as discussed in the



Notes. The figures are the unweighted averages for the 21 OECD countries considered in this study and are measured in five-year differences and are multiplied by 100. The spikes in 1918 and 1923 have the values of 0.64 and -0.75, respectively.

Figure 1 displays the five-year changes in mortality rates in the 5-14 and 15-64 age cohorts, where the former is dominated by the school-age population and the latter reflects mortality among the working age population. The figures are in absolute and not relative changes because mortality is already measured relative to population in the relevant age cohorts. The spike in 1918 is almost entirely due to the Spanish influenza pandemic during which the mortality rate was higher by 57% compared to 1913. Using mortality rates in the period 1914-1917 as a guide for war casualties in 1918, WWI contributed to less than 20% of the mortality growth in 1918, suggesting that the Spanish flu was the dominant cause of the mortality hike in 1918.

Up until the end of WWII the changes in mortality rates were almost identical for the two age groups. Thereafter, the decline has been substantially more pronounced in the 15-64 age-cohort than its younger counterpart; predominantly because of the decrease in non-communicable diseases and because of reduced smoking since 1970, predominantly affecting the working-age cohort (Preston, 1996). Common for both graphs is that the fastest mortality decline occurred before 1960, a time at which the epidemiologic transition was complete in the advanced countries. The marked decline immediately after WWII is due to the high mortality rates during WWII and the commercialization of antibiotics around 1944 (Preston, 1996).

4.2 Instruments

Instruments are used for the working age mortality in the ideas production function to cater for the possibility that excluded variables impact simultaneously on mortality and ideas production. Accumulated numbers of worldwide significant medical innovations and the growth in infant mortality are used as instruments for the growth in the working-age mortality. The number of significant medical innovations is accumulated because it is the stock of medical knowledge and not its change that is essential for mortality growth. Medical innovations are unlikely to be influenced significantly by contemporaneous economic circumstances in each individual country and, as such, can be considered exogenous. Furthermore, it is unlikely that the number of patents is affected directly by significant medical inventions through channels other than health status since the most significant medical inventions are not patentable (germ theory, for example, is probably the most significant medical innovation in human history). Finally, if significant medical innovations were influenced by mortality then they should have been high well before they started gaining momentum during the second half of the 19th century, because diseases have been a serious issue since the nihilistic revolution because the population concentration became sufficiently high for pathogens to survive. Thus, significant medical inventions are likely to be exogenous.

Infant mortality is used to capture infectious disease cycles and outbreaks that impact on mortality of all ages such as measles, cholera, typhoid, influenza, etc. Although infant mortality cannot be considered completely exogenous it nevertheless benefits from not affecting ideas production directly. As shown in the next section, ideas production is independent of infant mortality, confirming the prior that ideas production is independent of infant health and that only factors that impact simultaneously on infant and working age mortalities affect ideas production. Furthermore, infant mortality is caused by factors that are often independent of factors that are responsible for mortality among the working age population. Lee (2007), for example, argues that the main factor behind the strong decline in infant mortality at the turn of the last century was essentially pasteurization of cow milk; a factor that would not have been that influential for adult mortality.

Instruments are also used for health-adjusted educational attainment; however, only in the robustness checks because it is not clear whether health-adjusted educational attainment is exogenous given it is determined by health and schooling at the time at which the working population undertook their education. Furthermore, numerous research projects have shown that

mortality is at best influenced only weakly by income, and therefore it is health insults in the period at which the students did their education is likely to have been independent of income (see, for example, Preston, 1996; Haines and Steckel, 2000; Arora, 2005; Falcao and Soares, 2008; and Bloom and Canning, 2009).

Accumulated significant medical innovations, adjusted years of compulsory schooling, and crude birth rates are used as instruments for the health-adjusted GERs at the primary, secondary and tertiary levels, separately. Unlike other studies, except for Madsen (2012), health-adjusted GERs are instrumented here and not education attainment, because the decision to invest in human capital is taken prior to or during the time at which the individual is enrolled in the educational system. Education is undertaken during the schooling age and no factor can influence educational attainment among the working age population. Significant international medical inventions serve as potentially good instruments because they influence mortality rates directly and, as discussed above, they are likely to be independent of mortality and economic factors for each individual country.

Fertility is used as an instrument for the health-adjusted GERs as it proxies for the quantity-quality tradeoff in the fertility decision. An exogenous shock that changes the relative returns from investing in quality (human capital of each child) and quantity (number of children) will simultaneously impact on fertility and the GERs. It is likely that technical progress over past two centuries has been human capital using and, therefore, has increased the returns to investment in human capital as opposed to the number of children (see, for theory and evidence Galor and Weil, 2000, and Galor, 2005). This has induced parents to have fewer children and, instead, spend more resources on each individual child.

The length of compulsory schooling is used as an instrument for the health-adjusted GERs because it influences directly the length of schooling, possibly without any changes in economic incentives. The imperfection of this instrument is that compulsory schooling laws were not always enforced in the 19th century (see, for example, Green, 1997). However, the change in the compulsory years of schooling is likely to reflect a political desire to support education financially and politically. The differentiated adjustment of compulsory years of education is used for the health-adjusted GERs at different levels of schooling. Denoting YCS as the years of compulsory schooling, $\Omega^P = YCS$ if $YCS \leq 7$; otherwise, $\Omega^P = 7$, where Ω^P is the adjusted length of compulsory primary education. This measure adjusts for the fact that GERs in primary education are only affected by up to seven

years of compulsory schooling. The adjusted length of secondary education, Ω^S , is computed as $\Omega^S = YCS - 7$ if $YCS \geq 7$; and 0, otherwise, where Ω^S is the adjusted length of compulsory secondary education. This adjustment ensures that GERs in secondary education are only affected by the compulsory length of schooling beyond year seven. Since compulsory schooling laws, thus far, have not applied to tertiary education, Ω^S is also used as an instrument for tertiary GERs under the view that the longer Ω^S is the more likely it is that the student will enter tertiary education. Furthermore, an increase in the length of compulsory education is a signal from the government that they support further education.

4.3 Estimation results: Ideas production

The first-round IV regressions are displayed in Table 1. The coefficients of accumulated significant medical inventions as well as infant mortality are highly significant determinants of working age mortality and have the expected signs regardless of whether they are included jointly or individually. The F -tests of excluded restrictions are well in excess of 10, suggesting that the instruments are potentially good.

Table 1. First round regressions. Working age mortality rates instrumented.

Dep. Var.	$\Delta \ln m_{it}^{wa}$	$\Delta \ln m_{it}^{wa}$	$\Delta \ln m_{it}^{wa}$
Med_t	-0.0003(4.25)		-0.0003(4.50)
Δm_{it}^0		0.0017(5.78)	0.0018(5.97)
$F(r,566)$	18.0	33.4	27.4

Notes: the numbers in parentheses are absolute t -statistics, Med = accumulated significant medical innovations; and $F(r,566)$ = F -test for excluded instruments, where r is the number of excluded instruments. Fixed effect dummies are included in the regressions.

The results of estimating the ideas production function are presented in Table 2. The coefficients of knowledge stock are highly significant and are sufficiently close to one to conclude that there are constant returns to knowledge production. Statistically, the null hypothesis of scale effect is strongly rejected because the coefficients of A are highly significant; however, increasing returns to knowledge is extremely implausible in that it would imply increasing productivity growth over time. The coefficients of A are probably biased upward because of omitted variables and measurement errors. The interaction between DTF and research intensity is positive as predicted by theory but only significant at the five percent level.

The coefficients of research intensity are all highly significantly positive as predicted by Schumpeterian theory and the working age mortality rates are significantly negative at the 1% level regardless of which instrument set is used in the regressions in the first four columns. Coupled with the finding of scale effects in the ideas production, these results suggest that mortality and research intensity have permanent growth effects. If R&D expenditure are kept in a constant proportion to nominal GDP, productivity will grow at a constant positive rate along the balanced growth path. Similarly, positive mortality rates will constantly exert negative pressure on productivity growth rates and, thus, for mortality rates that are sufficiently high to override positive growth effects from R&D and other factors, productivity growth will become negative.

Table 2. Parameter estimates of ideas production function (Eq. (11)).

Dep. Var.	$\Delta \ln \dot{A}_{it}$	$\Delta \ln \dot{A}_{it}$	$\Delta \ln \dot{A}_{it}$				
	1	2	3	4	5	6	7
Instr.	None	<i>Med</i>	m^0	<i>Med, m⁰</i>	<i>Med, m⁰, m^{wa}</i>	<i>Med</i>	<i>Med</i>
$\Delta \ln(R/Y)_{it}$	0.05(2.43)	0.09(5.11)	0.07(4.01)	0.07(4.08)	1.22(12.5)	1.54(16.6)	1.19(11.9)
Δm_{it}^{wa}	-0.39(13.2)	-1.20(4.47)	-0.29(2.24)	-0.49(4.11)	-0.21(1.93)		
Δm_{it}^0							-0.00(1.65)
$\Delta \ln(Lexp)_{it}$						-0.93(7.48)	
$\Delta \ln A_{it}$	1.07(58.2)	1.09(61.3)	1.05(56.5)	1.07(58.8)	1.07(60.8)	1.06(62.1)	1.07(59.5)
$\left(\frac{R\&D}{Y} DTF\right)_{i,t-1}$	1.65(2.00)	1.65(2.00)	1.65(2.00)	1.65(2.00)	1.65(2.00)	1.52(1.90)	1.66(2.01)
$R^2(Buse)$	0.90	0.91	0.89	0.90	0.91	0.92	0.90
DW	2.04	2.05	2.03	2.02	2.03	2.04	2.04

Notes: the numbers in parentheses are absolute t -statistics, *Med* = accumulated significant medical innovations; *Lexp* = life expectancy at birth; and m^0 = infant mortality rate. Fixed effect dummies are included in the regressions. In the regression in the 5th column the log of the mortality rate at working age is used as an “instrument” for $\ln(R/Y)$ (the F -test for overall significance in the first-round regression is $F(1,566) = 23.3$). The SUR estimator, which is used in all the regressions in this table, weights the covariance matrix by the correlation of the residuals using the variance-covariance structure as follows: $E\{\varepsilon_i^2\} = \sigma_i^2$, $i = 1, 2, \dots, N$, $E\{\varepsilon_{it}, \varepsilon_{jt}\} = \sigma_{ij}$, $i \neq j$, $\varepsilon_{it} = \rho \varepsilon_{i,t-1} + v_{it}$, where σ^2 is the variance of the disturbance terms for country $i = 1, 2, \dots, N$, σ_{ij} is the covariance of the disturbance terms across countries i and j ; ε are the residuals; and v is an *iid* disturbance term. The variance, σ^2 , is assumed to be constant over time but to vary across countries and the error terms are assumed to be mutually correlated across countries, σ_{ij} . The parameters σ_i^2 , ρ and σ_{ij} are estimated using feasible generalized least squares.

Mortality has been assumed to impact directly on ideas production in the regressions in the first four columns of Table 2. The regression in column 5 allows mortality to influence ideas production directly as well as indirectly through research intensity. In this regression research intensity is first regressed on working age mortality and then the predicted values from this regression are used in the

structural regression in column 5. The results show that mortality significantly impacts on ideas production through research intensity, as indicated by the high significance of the coefficient of research intensity. While these results should not be interpreted as mortality being the only major driving force behind research intensity and, thus, ideas production they, nevertheless, underscore the importance of health for ideas production.

Life expectancy at birth, $Lexp$ is included instead of working age mortality in the regressions in column 6 to gain insight into the sensitivity of the results to health measure. The coefficient of $Lexp$ is of the wrong sign and highly significant, however, it is rendered insignificant at any conventional level if research intensity is not instrumented (the latter results are not shown). These results suggest that life expectancy at birth, as a proxy for health in the growth regressions, does not capture the influence of health on the most important drivers of growth, namely innovations. The problem associated with life expectancy at birth as an indicator of the working age survival probability is that most of its variations are due to changes in child and old-age mortality rates, which are not always echoed in working age mortality rates.

Finally, health is proxied by infant mortality instead of working age mortality in the regression in the last column to shed light on whether it is the disease environment in general that impacts on ideas production or, alternatively, that there is a third factor affecting simultaneously mortality and ideas production. The coefficient of infant mortality is insignificant; it is only negative and significant at the 5% level if research intensity is not instrumented (the latter results are not shown). This suggests that 1) it is specifically the health among the adult population that is essential for ideas production; 2) it is unlikely that the working-age mortality is capturing the effects of a third variable that influences ideas production and the working-age mortality simultaneously; and 3) that infant mortality is a good instrument because it captures the essential features of working-age mortality that are important for ideas production.

4.4 Estimation results: Productivity growth

The results of estimating Eq. (12) are presented in Table 3. Consider first the regression in the first column in which educational attainment is unadjusted, that is $\Phi = 0$. The coefficients of h , h^2 and $h \cdot DTF$ are all statistically significant at the one percentage level, suggesting that educational has significant and permanent growth effects; however, the relationship between growth and human

capital is inverted U-shaped as the coefficient of h is positive and the coefficient of h^2 is negative. The estimated coefficients of h and h^2 imply that the productivity growth effect of h peaks at 10.6 years of education it contributes 2% to annual growth. Its contribution to growth is 1% when h is equal to 3 and 18 years of education. The finding of a significant interaction effect is consistent with the Nelson-Phelps model in which education enhances absorptive capacity and enables off-frontier countries to tap into the knowledge that is created at the frontier.

Turning to the innovation-based variables the coefficients of growth in S^f and patent counts, and the log of (Pat/L) are consistent with the predictions of endogenous growth theory as they are all positive and highly significant. The domestic innovative activity has permanent growth effects and as long as the innovative activity, measured by the number of patent applications, is kept in a fixed proportion to employment, the innovation-driven growth effect will remain constant; a result that is consistent with the ideas production regressions. The temporary growth effects of an increase in the innovative activity exceed the permanent growth effects through the $\Delta \ln(Pat)$ -term. The stock of foreign knowledge has only temporary productivity growth effects as the domestic knowledge stock has to increase to further productivity. This result is consistent with the results of Coe and Helpman (1995) and Madsen (2007). Finally, the coefficient of DTF is positive and significant, indicating that DTF has growth effects that are independent of the level of educational attainment.

Table 3. Parameter estimates of productivity growth model (Eq. (12)).

	1	2	3	4	5	6	7
Φ	$\Phi = 0$	$\Phi = 195$	$\Phi = 500$	$\Phi = 0$	$\Phi = 0$	$\Phi = 418$	$\Phi = 191$
Estimator	SUR	SUR	SUR	SUR	GLS	GLS	SUR/IV
Δh_t^x				0.120(5.01)			
h_t^x				0.085(14.4)			
$(h_t^x)^2$				-0.004(7.68)			
Δh_t	0.009(1.03)	0.048(3.77)	0.033(1.99)	0.049(4.03)	0.003(0.09)	0.062(1.30)	0.000(0.01)
h_t	0.019(4.24)	0.036(9.35)	0.034(6.66)	0.021(4.13)	0.035(2.13)	0.045(2.14)	0.131(8.68)
h_t^2	-0.0009(3.24)	-0.0031(10.5)	-0.0040(8.80)	-0.0029(8.83)	-0.0018(1.83)	-0.0051(3.26)	-0.0491(7.39)
DTF_{t-1}	0.025(2.94)	0.044(7.07)	0.056(11.0)	0.009(1.06)	0.036(0.96)	0.107(4.28)	0.021(4.31)
$(h_t^d * DTF)_{t-1}$	0.012(8.02)	0.026(11.8)	0.038(11.7)	0.023(13.1)	0.018(2.57)	0.066(5.21)	0.023(15.7)
$\Delta \ln S_t^f$	0.086(8.97)	0.112(8.57)	0.088(7.55)	0.114(9.16)	-0.045(1.55)	-0.023(0.75)	0.167(13.8)
$\Delta \ln Pat_t$	0.025(10.0)	0.032(10.6)	0.088(7.54)	0.028(9.22)	-0.011(0.77)	0.009(0.76)	0.035(11.6)
$\ln(Pat/L)_t$	0.023(11.0)	0.015(8.22)	0.020(11.3)	0.020(9.52)	0.033(3.03)	0.031(3.57)	0.010(7.26)
Differences	5-Year	5-Year	5-Year	5-Year	10-Year	10-Year	5-Year
DW	2.01	1.89	2.01	1.89	1.76	1.85	1.93

$\chi^2(4) = 0$	230	357	271	-	21	128	537.9
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Notes: The numbers in parentheses are absolute t -statistics and the numbers in squared brackets are p -values. $\Delta h_t^x = \Delta h_t(\Phi = 195) - \Delta h_t(\Phi = 0)$; $h_t^x = h_t(\Phi = 195) - h_t(\Phi = 0)$; $(h_t^x)^2 = (h_t^x)^2(\Phi = 195) - (h_t^x)^2(\Phi = 0)$. The following years are included in the 5-year difference estimates: 1875, 1880, 1885, 1890, 1895, 1900, 1905, 1910, 1915, 1920, 1925, 1930, 1935, 1940, 1945, 1950, 1955, 1960, 1965, 1970, 1975, 1980, 1985, 1990, 1995, 2000, 2005 and 2009. $\chi^2(4)$ = test for joint significance of the coefficients of Δh_t , h_t , h_t^2 , and $(h^d \cdot DTF)_{t-1}$, and is distributed as $\chi^2(4)$ under the null hypothesis that the coefficients are zero. See the Notes to Table 2 for details on the SUR estimator. The GLS estimator corrects for first-order serial correlation and cross-country heteroscedasticity (see Notes to Table 2). IV is the instrument variable estimator where accumulated significant medical innovations, crude birth rates and the adjusted years of compulsory education are used as instruments for GERs at the primary, secondary and tertiary level individually.

The regression in the second column shows the results from the simulations that give the value of Φ that maximizes the joint significance of Δh , h , h^2 and $h \cdot DTF$. The joint significance of Δh , h , h^2 and $h \cdot DTF$ is maximized for $\Phi = 195$ with $\chi^2(4) = 357$, which not only strongly rejects the null hypothesis of zero joint productivity effects of human capital but, is also well in excess of $\chi^2(4) = 230$ for $\Phi = 0$ (column 1). This result suggests that good health is essential for students' learning.

The quantitative effects of mortality on productivity growth were significant before WWII. Using the coefficients of h and h^2 in the second column, in which $\Phi = 195$ we get the following scenario. In 1875 the average health-adjusted educational attainment was 1.01 years for $\Phi = 195$ and 3.59 years for $\Phi = 0$, where $\Phi = 0$ is the zero mortality scenario. The growth effect is 0.66% for a health-adjusted educational attainment of 1.01 years and 1.79% for a health-adjusted educational attainment of 3.59 years. Thus, the productivity growth rate would have been 1.13 percentage points higher back in 1875 if the mortality rate was zero when the workforce undertook their education. In 1920 the growth effects from h and h^2 would have been 0.65 percentage points higher if mortality rates were zero; the gap gradually narrows thereafter. These mortality-induced growth effects are even higher if the contribution from the terms Δh and $h \cdot DTF$ is added to the simulations. Thus, the health-induced reduction in the health-adjusted human capital before WWII was a large drag on growth and partly accounts for the low growth rates experienced during the Second Industrial Revolution despite it being probably the most innovative period in human history.

Finally, health-adjusted educational attainment is decomposed into the GER^a -term and the $\exp[\Phi \ln(1-m^a)]$ -term (see Eq. (8)) in the regression in column 4, where $h = h(\Phi = 0)$ and $h^x = h(\Phi = 195) - h(\Phi = 0)$. The coefficients of Δh^x , h^x , $(h^x)^2$ and $h^x DTF$ are statistically highly significant and of the expected sign; thus, reinforcing the other results that health is highly influential for the quality of learning and later enhances the productivity of the labor force when the graduates

join the labor market.

5 Extensions and alternative tests

5.1 Instrument variable growth regressions

IV regressions are presented in the last column of Table 3. The health parameter at which the significance of the human capital related variables is maximized, Φ^{\max} , is estimated to 191, which is close to the iterated value of 195 in the un-instrumented regressions. The coefficients of all the instruments are significant and have the expected sign in the first-round regression; also the F -tests for excluded restrictions are well in excess of the benchmark level of 10 (the results are not shown). The coefficients of h , h^2 and $h \cdot DTF$ remain highly significant in the structural estimates; again reinforcing that human capital has permanent growth effects. The coefficient of Δh is insignificant, which is consistent with most of the other regressions in Table 3; thus, it is not entirely clear whether the short-run effects of a change in human capital are stronger than the medium and long run effects. Finally, all the innovation driven variables remain statistically significant.

5.2 Estimates in 10-year differences

Columns 5 and 6 of Table 3 present 10-year difference estimates. The joint significance of Δh , h , h^2 and $h \cdot DTF$ is maximized for $\Phi = 410$ with $\chi^2(4) = 127$, which is well in excess of $\chi^2(4) = 21$ when $\Phi = 0$. The lower significance here compared to the 5-year estimates reflects that the effective number of observations and the contemporaneous correlations between the residuals, as in the SUR model, are not allowed for in the 10-year estimates. The estimated value of Φ^{\max} of 410 is substantially higher than the Φ^{\max} of 195 found in the 5-year estimates; a difference that underscores the uncertainty that is attached to estimates in which health effects are estimated indirectly. The high weight given to health in human capital accumulation in the 10-year regressions suggests that the growth effects of health relative to the quantity of schooling may be underestimated in the 5-year difference estimates. IN regards to the innovation variables, knowledge transfers and growth in patents are insignificant in the 10-year regressions; however, research intensity, (Pat/L) , is highly significant and, therefore, consistent with the ideas production function regressions and the Schumpeterian growth model of Howitt (2005). Thus, innovations remain an important channel through which health is affecting growth.

5.3 Educational progression and health-adjusted GERs

As an alternative test to examine whether health influences the quality of learning, the unadjusted GERs for secondary education is regressed on the health-adjusted GERs for primary education lagged seven years to allow for the time-lag between primary and secondary education. The higher is the health-induced learning quality, the higher is the likelihood that the pupil will proceed to secondary education. Furthermore, GER regressions can be used to test whether mortality has a direct effect on the schooling decision.

The following two models are regressed:

$$\Delta GER_{it}^S = \nu_0 + \nu_1 \Delta GER_{i,t-7}^P + \nu_2 \Delta \ln m_{it}^{wa} + \nu_3 \Delta \ln y_{it} + \nu_3 \Delta \Omega_{it}^S + CD + e_{3,it} \quad (13)$$

$$\Delta GER_{it}^S = w_0 + w_1 \Delta GER_{i,t-7}^P + w_2 \Delta \ln m_{it}^{wa} + w_3 \Delta \ln y_{it} + w_3 \Delta \Omega_{it}^S + CD + e_{4,it} \quad (14)$$

where GER^P is the health-adjusted GERs for primary education based on Eq. (8) for $\Phi = 195$; GER^P is the unadjusted GERs for primary education; GER^S is GERs for secondary education; Ω^S is the adjusted years of compulsory secondary education; and Δ is the five-year difference operator. The only difference between the two models is that GER^P is a regressor in Eq. (13) while GER^P is a regressor in Eq. (14). As discussed in the instrument section, Ω^S is measured as the years of compulsory school (YCS) beyond primary schooling, $\Omega^S = YCS - 7$ if $YCS \geq 7$; and 0, otherwise. The OLS estimator with the t -ratios based on robust standard errors is used instead of the SUR estimator because the SUR estimator was not able to sufficiently correct for the first-order serial correlation.

The model is *not* iterated to get the Φ that yields the highest significance of GER^P since, as shown below, GER^P is not positive and significant, suggesting that it is health that is driving force behind the results that the model seeks to uncover. Instead, Φ is set to 195 as found in the previous section. The full model is estimated over the period from 1875 to 1960 as well as over the period 1830-1960 with the working-age mortality excluded from the regression. The estimation period terminates in 1960 since the GER^P by 1960 had reached 100% for all countries in the sample (although the results are almost identical if the model is regressed over the full estimation period). The regressions are limited to GER^S because the GERs for tertiary education in the 19th century were

well below 1%, except for the US and Canada, and, thus, do not give sufficient identifying variations. Note, however, that the results for tertiary GERs are significantly stronger than those obtained for secondary education in this sub-section.

Eqs. (12) and (13) essentially follow the predictions of the schooling model of Bils and Klenow (2001) in which the returns to schooling are a positive function of the expected productivity growth and life expectancy. Productivity growth may also impact positively on GERs under the assumption that schooling is a normal good, which implies that education is an increasing function of income. Working age mortality is used instead of life expectancy because the relevant metric is the likelihood of surviving from the time the pupil enrolls in school until the retirement at the age of 64.

Table 4. Parameter estimates of GERs for secondary education (Eqs. (13) and (14)).

	1	2	3	4	5	6
$\Delta GERH_{i,t-7}^P$	0.097(4.10)		0.073(3.14)		0.075(3.24)	
$\Delta GER_{i,t-7}^P$		-0.08(3.09)		-0.08(2.58)		-0.08(2.70)
$\Delta \ln m_{it}^{wa}$			-4.79(3.28)	-4.79(3.27)		
$\Delta \ln y_{it}$	1.01(1.60)	1.05(1.65)	3.92(2.36)	3.80(2.30)	4.85(3.04)	4.69(2.96)
$\Delta \Omega_{it}^S$	2.21(2.17)	2.06(2.07)	1.85(1.98)	1.69(1.84)	1.70(1.88)	1.53(1.73)
$\Delta \ln Lexp_{it}$					8.10(1.63)	8.57(1.72)
Est. Per.	1820-1960	1820-1960	1875-1960	1875-1960	1875-1960	1875-1960

Notes. *Lexp* = life expectancy at birth. The OLS estimator is used and the *t*-ratios are based on serial correlation and heteroscedasticity consistent standard errors.

The results of regressing Eqs. (12) and (13) are presented in Table 4. The regressions in columns (2) and (4) show that the unadjusted GERs are negative and significant in both cases, suggesting that high enrolment in primary education does not automatically lead to higher enrolment in secondary education. Using, instead, the health-adjusted GERs for primary education, $GERH^P$, yields positive and highly significant results (columns 1 and 3). These results give further support to the hypothesis that good health improves scholastic achievements and enhances the potential of students to further their education.

More supportive evidence for the health-insult hypothesis is found by the estimated coefficients of the working-age mortality rates, which are all negative and highly significant. Based on the coefficients of the working age mortality in the regressions in columns (3) and (4) (which are the same), the decrease in the working-age mortality rate over the period 1875-2009, on average, explains 15% of the increase in the GER^S . Thus, health improvements have not only boosted

educational attainment because they have enabled further education; but also they have given an economic incentive for further education.

Finally, life expectancy at birth, $Lexp$, is included in the regressions in the last two columns instead of the working age mortality to gain insight into the sensitivity of the results in regards to measure of health ($GERH^P$ is still adjusted for mortality rates in the primary school age groups). The regressions in the last two columns show that the coefficients of $Lexp$ are only marginally significant, suggesting that the results are critically dependent on how health is measured.

6. Concluding remarks

The contribution of this paper has been to show that health is potentially influential for growth through learning, ideas production, and schooling, as predicted by the model of Howitt (2005). An algorithm was incorporated into a morbidity-induced learning model, and it was argued that health -adjusted learning may be better at explaining growth than raw schooling enrolment data. Furthermore an ideas production function was established to show that mortality may influence ideas production, directly, as well as indirectly through research intensity. Using data covering the period 1812-2009 the models were tested for 21 OECD countries; thus, covering the high-morbidity *post*-Malthusian era, the transition to the modern growth regime in the first half of the 20th century, and the low-morbidity modern growth regime.

The regression results showed that health has been highly influential for the growth anatomy of 21 OECD countries considered here since 1870 through human capital and innovations, the main drivers of technological progress. First, it was shown that health-adjusted educational attainment was statistically, a much more significant determinant of productivity growth than the unadjusted educational attainment. Model simulations indicated that the productivity growth in the 21 OECD countries would have been more than one percentage point higher back in 1870 if the mortality rates, at the time the working age population undertook their education, were zero; an effect that remained significant until WWII. The declining mortality in addition to enhanced human capital through the quality of learning also increased the class attendance rates that started from a very low base in the early 19th century. Second, the regressions revealed that working age mortalities are significant determinants of ideas production, directly, as well as indirectly through R&D. Third, it was shown that health-adjusted primary education, in addition to the working age survival rates, are influential for

secondary school enrolment, suggesting that health does not only affect learning but also affects enrolment rates and, therefore, the relationship between health and human capital.

Since growth, along the balanced growth path, is driven by human capital and innovations the results in this paper show that declining mortality, as a proxy for morbidity, has been a major force behind the transition from the post-Malthusian to the modern growth regime in the 21 OECD countries considered here. While it cannot be ruled out that the composite productivity growth effects of the improved health in the OECD countries over the past two centuries have been exaggerated in the regressions as a result of the growth effects of omitted variables having been captured by the health-adjusted variables, the results are, nevertheless, consistent with the psychological literature findings of massive IQ gains over the past century.

In his survey on the time-series evidence of IQ gains, Flynn (1999) concludes that “data are now available for 20 nations, and there is not a single exception to the finding of massive IQ gains over time.... Recent data show that IQ gains in Britain began no later than the last decade of the 19th century.... All nations but Norway have shown gains at a rate of about 20 IQ points per generation (30 years)” (pp. 26-27). While the psychological literature has not come to an agreement about the approximate causes of the IQ gains, schooling and nutrition are the two leading explanations for the IQ gain (Lynn, 1990; and Flynn, 1999). Furthermore, Lynn and Mikk (2007) find a correlation between IQ and scholastic achievement of 0.92-1, suggesting that enhanced learning during the past two centuries, in the OECD countries, has contributed to the IQ increase.

The results in this paper have important implications for growth theory and policy. First, while the empirical macro literature has established that productivity growth is driven by human capital and innovations, very little research has tried to explain the increase in human capital and in innovations since the first Industrial Revolution. This paper is one of the first steps to empirically attempt to shed light on their increase and suggest that morbidity has played a much larger role in their increase than is normally incorporated into growth models. Second, conventional measures of health, such as life expectancy at birth, are unlikely to capture the myriad of channels through which health affects growth. The regressions revealed that neither schooling nor ideas production are positively affected by life expectancy; on the contrary, ideas production is significantly related to life expectancy at birth.

Third, productivity growth will be reduced significantly in an environment with high mortality and may even be negative. Thus, we get the reverse result of the Malthusian prediction where the

exogenous adverse mortality shocks are associated with increasing per capita income due to diminishing returns introduced by land as a fixed factor of production. Thus, the Malthusian mechanism is likely to be less active in an innovation-driven economy compared to an economy with little technological progress, with land being an important factor of production. This result is consistent with the finding of Crafts and Mils (2008) in which the Malthusian mechanism operates only through preventive checks (fertility) and not through the positive check (mortality). Fourth, the difficulties associated with finding a positive relationship between growth and educational attainment for non-OECD countries may be related to the finding in this paper where it is the health-adjusted educational attainment that is an essential driver of growth and that conventional educational attainment measures fail to capture this mechanism (see, for a literature review, Delgado *et al.*, 2011).

Data Appendix

To be finalized

Significant medical innovations. Frances Adlington and Chris Humphries (eds.), 1999, *Philip's Science and Technology: People, Dates and Events*, London: George Philip Limited.

TFP, Employment, real and nominal GDP, annual hours worked, patents, GERs, population distributed on ages, patents, see Madsen (2007, 2008, 2010, and 2012).

Age dependent mortality rates.

R&D.

Crude birth rates.

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